Parent pathology and family environment as correlates of child separation anxiety disorder

Courtney Ryan Pursell

University of Nevada, Las Vegas

Follow this and additional works at: https://digitalscholarship.unlv.edu/rtds

Repository Citation

https://digitalscholarship.unlv.edu/rtds/1258

This Thesis is brought to you for free and open access by Digital Scholarship@UNLV. It has been accepted for inclusion in UNLV Retrospective Theses & Dissertations by an authorized administrator of Digital Scholarship@UNLV. For more information, please contact digitalscholarship@unlv.edu.
INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

ProQuest Information and Learning
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
800-521-0600

UMI®

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
NOTE TO USER

Page missing in number only; text follows. Microfilmed as received.

50

This reproduction is the best copy available.

UMI
PARENT PATHOLOGY AND FAMILY ENVIRONMENT AS CORRELATES OF
CHILD SEPARATION ANXIETY DISORDER

by

Courtney Ryan Pursell

Bachelor of Arts
Franklin and Marshall College
1993

A thesis submitted in partial fulfillment
of the requirements for the

Master of Arts Degree
Department of Psychology
College of Liberal Arts

Graduate College
University of Nevada, Las Vegas
May 2001
The Thesis prepared by

Courtney Ryan Pursell

Entitled

Parent Pathology and Family Environment as Correlates of Child Separation Anxiety Disorder

is approved in partial fulfillment of the requirements for the degree of

Master of Arts in Psychology

Examination Committee Chair

Dean of the Graduate College

Examination Committee Member

Graduate College Faculty Representative
ABSTRACT

Parent Pathology and Family Environment as Correlates of Child Separation Anxiety Disorder

by

Courtney Ryan Pursell

Dr. Christopher Kearney, Examination Committee Chair
Professor of Psychology
University of Nevada, Las Vegas

Forty-four families whose children were tested for Separation Anxiety Disorder three years ago were again contacted for a follow-up study focusing on stability of the diagnosis over time and the associative characteristics of parent pathology and aspects of their family environment. Children were diagnosed with either no symptoms of separation anxiety, subclinical symptoms of separation anxiety (1 or 2 symptoms), or clinical Separation Anxiety Disorder (3 or more symptoms). Parent pathology focused on depression, obsessive/compulsive, phobic anxiety, and somatization. Family environment examined parental control and level of expression in the family. Current levels of parental pathology were found to be associated with both current and previous levels of Separation Anxiety. However, there was a decrease in diagnosis severity over time, so the hypothesis that Separation Anxiety Disorder is a stable disorder was not supported.
# TABLE OF CONTENTS

ABSTRACT .......................................................................................................................... iii

LIST OF FIGURES ............................................................................................................. v

ACKNOWLEDGEMENTS ................................................................................................. vi

CHAPTER 1  INTRODUCTION ......................................................................................... 1
  History of Separation Anxiety ..................................................................................... 3
  Parental Psychopathology .......................................................................................... 7
    Depression ................................................................................................................ 7
    Agoraphobia .......................................................................................................... 9
    Parental Anxiety ...................................................................................................... 10
  Family Environment .................................................................................................. 14
  The Current Study ...................................................................................................... 19
  Hypotheses ................................................................................................................ 19

CHAPTER 2  METHODS ................................................................................................. 21
  Participants ............................................................................................................... 21
  Measures ..................................................................................................................... 22
  Procedure ................................................................................................................... 25
  Data Analysis ............................................................................................................. 27

CHAPTER 3  RESULTS ................................................................................................. 32
  Change in Clinical Status from Time One to Time Two .......................................... 32
  Associations with Clinical Status at Time One and Time Two .............................. 33

CHAPTER 4  DISCUSSION AND CONCLUSIONS ..................................................... 38

APPENDICES ............................................................................................................... 51
  Appendix I  Consent Form ....................................................................................... 51
  Appendix II  Demographic Information Sheet ......................................................... 53

REFERENCES ................................................................................................................. 56

VITA ................................................................................................................................. 62
LIST OF TABLES

Table 1  Breakdown of Diagnostic Measures Across Time.................................24
Table 2  Factor Loadings for the Twelve Dependent Variables..............................29
Table 3  Chi Square Analysis of SAD Status at Time One and Time Two ...............34
Table 4  Breakdown of SAD Diagnosis Change Over Time....................................35
Table 5  Stepwise Regression Results with Five Factors and Change in Clinical Status and Diagnoses at Time One and Time Two .....................36
Table 6  Average SCL-90-R Scores Separated by Change in Clinical Status ............42
Table 7  Average FES Time One Scores Separated by Change in Clinical Status ......45
Table 8  Average FES Time Two Scores Separated by Change in Clinical Status ......46
ACKNOWLEDGEMENTS

I would like to thank Dr. Christopher Kearney, my committee chair and research advisor. Without his support, guidance, and patience, I would not have been able to achieve even half of this research. In particular, I’d like to thank Dr. Kearney for his sense of humor in dealing with the many mishaps throughout my thesis and his insistence that I never give up, no matter how difficult the thesis seemed. Thank you for getting me through this.

In addition, I’d like to thank my thesis committee, Dr. Jeffrey Kern, Dr. Marta Laupa, and Dr. Alice Corkill, not only for their assistance throughout my thesis, but also for treating me more like a colleague and less like a student struggling through research.

Finally, I need to thank my parents and my brother. Without their support I would not have made it this far.
CHAPTER 1

INTRODUCTION

Separation anxiety is a condition long viewed by researchers and clinicians as a relatively normal and healthy process and a basic human disposition. The term refers to child or parent concerns regarding loss or absence of significant others in one's life (Hock & Lutz, 1998). When looking at separation anxiety in children, attention primarily focuses on children's anxiety of separation from their mothers, although separation from any central attachment figure can produce distress (Bernstein & Borchart, 1991; Hock, McBride, & Gnezda, 1989). At times, normal childhood separation anxiety may become excessive and disruptive in a child's life. This condition is referred to as Separation Anxiety Disorder (SAD), a childhood anxiety disorder recognized by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994).

According to Crowell and Waters (1990), separation anxiety usually does not occur during the first six months of an infant's life. After age six months, separation anxiety may appear with 50-75% of children showing signs around 18 months. Signs of separation anxiety begin to wane around three years of age, with only 20-40% of children at this age showing distress. However, separation anxiety is not restricted to infants and toddlers. Researchers have found that, during childhood, phobias and anxiety about
separation are common. During adolescence, separation anxiety is more often replaced by anxiety of social evaluations and overanxiousness; however, separation anxiety may still be seen in teenagers and even adults (Bauer, 1976; Kashani & Orvaschel, 1990). Infant and toddler anxiety is typically considered by clinicians as age-appropriate and thus would not merit a diagnosis of SAD. It is when the symptoms are excessive or are not age appropriate (beyond the age of three or four years), that a diagnosis may be warranted.

For a child to obtain a DSM-IV diagnosis of SAD, there must be an onset of developmentally inappropriate and excessive symptoms before age 18 years. Specifically, the child must exhibit at least three of the following eight symptoms: 1) recurrent excessive distress when separation from home or major attachment figures occurs or is anticipated, 2) persistent and excessive worry about losing, or about possible harm befalling, major attachment figures, 3) persistent and excessive worry that an untoward event will lead to separation from a major attachment figure, 4) persistent reluctance or refusal to go to school or elsewhere because of fear of separation, 5) persistent and excessive fearful or reluctance to be alone or without major attachment figures at home or without significant adults in other settings, 6) persistent reluctance or refusal to go to sleep without being near a major attachment figure or to sleep away from home, 7) repeated nightmares involving the theme of separation, and 8) repeated complaints of physical symptoms when separation from major attachment figures occurs or is anticipated (p. 113, American Psychiatric Association, 1994). These symptoms must last at least four weeks and cause significant impairment in a child’s academic or social functioning.

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
Brief History of Separation Anxiety

The concept of separation anxiety has appeared in literature as far back as Sigmund Freud's works in 1926. He described general anxiety as a warning of impending danger and categorized the construct into three types (Hergenhahn, 1992).

*Objective anxiety* emanates from an actual or perceived threat to the individual's well being. *Neurotic anxiety* occurs when the ego feels it may be overwhelmed by the irrational id. *Moral anxiety* is much like guilt in that it arises when an internal moral is violated. Freud explained that the ego aims to decrease our anxiety, either by dealing with the environment or through defense mechanisms. These mechanisms serve to decrease anxiety by distorting reality and operate at an unconscious level. Freud combined these theories on anxiety with his theories on separation. He believed that attachments (both child-adult and adult-adult attachments) were formed from mental bonds that throughout our lives are never completely broken. Thus, any threats to these attachment bonds are invariably painful and will trigger self-protective measures in the form of defense mechanisms (Freud, 1926/1961). Separation experiences are dealt with via these defense mechanisms (separation anxiety behaviors) which, according to Freud, have a lasting impact on future bonds and patterns in relationships and may ultimately affect an individual's personality structure.

Subsequent to Freud's separation and attachment theories came a barrage of alternative theories, including Rank's separation anxiety as reliving the trauma of birth and Klein's theory of separation anxiety as actions against the mother (Crowell et al., 1990). However, none of these newer theories of separation anxiety were as influential.
as Freud’s until the 1960s, when John Bowlby began examining separation, attachment, and loss.

Bowlby examined attachment and separation through a combination of evolutionary development and personal experience (Bowlby, 1973). He explained that attachment, separation, and reunion responses are learned as infants develop, but our evolutionary heritage makes us more likely to learn certain behaviors over others. The theory states that separation anxiety occurs when a parent leaves and the child’s crying is reinforced when the parent returns. This is where the early experiences are learned. In addition, there is the evolutionary development seen in children’s actions of attachment and exploration. When a child wanders away from a caregiver it is considered exploration and the child shows few signs of anxiety or distress. However, when the parent walks away from the child, the child’s access to the parent is reduced. The child then begins to display distress at being alone and engages in proximity seeking (following the parent to be able to stay with them).

Bowlby described three distinct phases of separation. The first phase is protest, where the child displays overt distress during separation from a caregiver. According to Bowlby, this phase is synonymous with SAD. The second phase is the despair phase. Here the child displays sadness and withdrawal if the separation continues. The final phase is detachment where the child is indifferent to the reappearance of the caregiver. Intense anger and anxious clinging accompany this indifference on the part of the child. Often, any attempt on the part of the caregiver to separate from the child will result in separation anxiety. Bowlby states that these phases may or may not become psychopathological. If the intense feelings of the child are not expressed, then the
behaviors and feelings may become distorted by the repressed emotions. The detachment phase involves repression of emotions. These three phases are often seen in children aged 6 months to 4 years and tend to be more pronounced if the child does not find a substitute attachment figure.

Throughout this time and until the 1970s, separation anxiety was often not considered a distinct behavioral pattern but was rather combined with school phobia into an encompassing childhood anxiety description. School phobia was first introduced to the literature by Johnson and colleagues (1941). Although the concept of school phobia focused on extreme anxiety about attending school, theorists then believed that this anxiety was precipitated by an underlying fear of separation from the child’s mother and home (Last, Francis, Hersen, Kazdin, & Strauss, 1987a). In fact, more emphasis was placed on the anxiety of separation than the fear of school. In 1956, the term “separation anxiety” was used for school phobias to focus the attention on the so-called underlying problem (Last et al., 1987a). This focus on separation rather than fear of school was supported by John Bowlby, who believed that loss of an attachment figure or security base (i.e., home) was the central fear of children with school phobia, rather than fear of the actual school environment (Last et al., 1987a).

Around 1969, clinicians began steering away from attachment and separation as the underlying cause of school phobia and began examining the school environment (Last et al., 1987a). However, the two anxieties were still enmeshed with one another, as seen with the publication of the DSM-III. Here, for the first time, SAD was described as an anxiety disorder distinct from a general category of Anxiety Disorders of Childhood and Adolescence (APA, 1980). However, the confusion over distinguishing separation
anxiety from school phobia had not ended with this new category: one of the criteria for SAD is reluctance or refusal to go to school.

Recent studies have also focused on the defining features of SAD and school refusal behavior. While children with separation anxiety show fear of leaving an attachment figure in a variety of situations (Last et al., 1987a), the behaviors of children with school refusal tend to focus around the school environment, ranging from avoidance of peers to anxiety of tests and teachers (Hansen, Sanders, Massaro, & Last, 1998). Unfortunately, these central features are not always clear-cut and there are still cases in the current literature where separation anxiety and school refusal are treated as the same. Greater attention needs to be paid to the distinguishing factors of the two anxieties to promote more accurate conceptualization, assessment, and treatment of the problems.

The most recent theories of separation anxiety center around genetics and the heritability of psychological disorders. Relatively few studies have been conducted on the heritability of anxiety disorders among twins and, of those available, extremely few focus on separation anxiety. Torgersen (1983) reported that there was a 34.0% concordance rate of anxiety disorders in general among monozygotic twins and a 17.0% rate among dizygotic twins. However, contradicting results were found when Topolski and colleagues (1997) conducted a twin study looking specifically at the heritability of manifest anxiety, SAD, and overanxious disorder. They found an overall (regardless of gender) SAD concordance of .35 for monozygotic twins and .33 for dizygotic twins.

These results led to speculation of the influence of shared environmental effects in the development of SAD (Topolski et al., 1997). The speculation that environmental factors may increase a child’s likeliness of SAD is a relatively new one. Two prevalent
factors (family environment and parental psychopathology) appear central to this theory, yet severely under researched. It is this unanswered question of the effects of the family environment and parent pathology on SAD that is the focus of the current study. A review of the current literature will show what efforts have been taken to examine the factors associated with anxiety disorders in general and what further research needs to be done in the area of SAD.

Parental Psychopathology

Many studies indicate a relationship between parental psychopathology and general childhood psychological disorders. For example, Warner and colleagues (1999) found that in families where both grandparents and parents had depression, 49% of the grandchildren had some type of psychopathology. Zahn-Waxler and colleagues (1988) found in families with at least one parent with bipolar disorder, that children were at greater risk for developing depression and antisocial behaviors. Orvaschel, Walsh-Allis and Ye (1988) also found that, in general, parents with psychiatric disorders are twice as likely to see emotional and behavioral problems in their children than parents without any psychiatric illness. These are just a sample of the many studies examining parental psychopathology and childhood disorders. Unfortunately, relatively few studies have been conducted on childhood anxiety in general and fewer on SAD in particular.

Depression and SAD

Due to the high comorbidity rates of anxiety and depression, some researchers have examined the correlation among parental anxiety and depression and anxiety disorders in children. In a study by Weissman and her colleagues (1984a), first-degree
relatives (parents, siblings, and children) of people with depression and an additional anxiety disorder were more likely to have major depression, panic, phobias, and alcoholism than relatives of people with just depression or people with no emotional or anxiety disorder (Weissman, Leckman, Marikangas, Gammon, & Prusoff, 1984a). In particular, children's diagnoses tended to follow those of their parents; thus, parents with depression and panic disorder had a greater likelihood of having a child with depression and panic disorder. In addition, Weissman found, with regard to parents with depression and panic disorder, that over one-third of their children met requirements for separation anxiety disorder.

Another study by Weissman and colleagues (1984b) not only examined the children of parents with major depression but also examined differences in the child's diagnosis depending on whether one or both parents had major depression. They found that the primary diagnosis in the children was major depression (13.0%), followed by attention deficit disorder (10.0%) and separation anxiety (10.0%). They also found that, in families where both parents had major depression, the child was more likely to have any diagnosis than in families where only one parent had major depression.

Mufson, Weissman, and Warner (1992) examined the transmission of depression and panic disorder from parents with both of these traits to their children. Results indicated that children were at a greater risk to develop depression and an anxiety disorder when their parents had depression and panic disorder. Another study by Turner and his colleagues compared children of parents with depression to children of parents with an anxiety disorder (Turner, Beidel, & Costello, 1987). Children of parents with anxiety disorders were more likely than normative children and children of parents with...
depression to be anxious, depressed, fearful, introverted, and worried (specifically about their families and themselves). In addition, they were more likely to have problems in school and somatic complaints. These children were seven times more likely to be diagnosed with a psychological disorder than children of parents with no diagnosis, and twice as likely to have a DSM-III diagnosis as children of parents with depression.

Agoraphobia and SAD

There has been some research on agoraphobia in parents and resulting psychological disorders in their children. Capps and her colleagues found that children of parents with agoraphobia had a greater number of psychological and emotional problems than children from a comparison group (Capps, Signam, Sena, Henker, & Whalen, 1996). Sixty-eight percent of children with a parent with agoraphobia met criteria for one or more DSM-III diagnoses, particularly depression and anxiety. The most common diagnosis for these children (56.0%) was separation anxiety disorder. In addition, these children reported more fears and anxieties than comparison children and saw themselves as more vulnerable.

Research indicates that children of parents with agoraphobia have the greatest amount of behavioral problems compared to children of parents with phobias and children of parents with panic disorder (Silverman, Cerny, Nelles, & Burke, 1988). Silverman and her colleagues concluded that child behavior problems are highly associated with the presence of parental phobias or avoidant behavior. Although the reasons for these findings are unknown, they hypothesized that a parent with an anxiety disorder may model overly cautious and fearful behaviors for the child, thus indirectly teaching them anxious behaviors.
Capps, however, describes another theory of the relationship between child and parent pathologies. He states that anxious people inherit a heightened responsiveness to stress. When this characteristic is presented with experiences that undermine a child’s perception of their control on the environment, this combination may lead to the development of negative affective adjustment and high levels of anxiety (Capps et al., 1996).

**Parental Anxiety and SAD**

Mancini, Van Ameringen, Szatmari, Fugere, and Boyle (1996) found that the number of anxiety disorders in parents positively correlated with the number of current anxiety disorders in children. The most common diagnoses in these children were overanxious disorder (30.0%), social phobia (23.0%) and separation anxiety (19.0%). Mancini hypothesized that children raised by parents with psychological problems may learn the problem behavior, indicating that the greater the parental problem, the greater the influence that problem has on the development of the child.

Kashani and colleagues (1990) examined the relationship of anxiety in parents and their psychiatrically hospitalized children. They divided the children into three groups: those with severe anxiety (defined when both the child and the parent reported the child having an anxiety disorder), those with possible anxiety (where either the parent or the child reported the child having an anxiety disorder), and those with no anxiety disorders. Anxiety disorders included overanxious disorder, phobias, and SAD. In addition to screening the children, the parents completed the SCL-90-R to examine any parental disturbances. The researchers reported a relationship between the parent’s and child’s anxiety disorders, with parent report of higher anxiety correlating with children
with more severe anxieties. Unfortunately, parental anxieties were never specifically defined. Parent scores on the SCL-90-R revealed that parents of children with anxiety (both severe and possible anxiety) scored higher on somatization, phobic anxiety, depression, and hostility than parents of children with no anxiety. Although this study may not be generalized to the non-hospitalized population, there is evidence that parents of children with SAD, phobias, and/or overanxious disorder report greater amounts of anxiety and other symptoms than parents of children with no anxiety.

The studies presented thus far examined specific disorders in parents (e.g. agoraphobia or social phobia) and reported on the range of disorders found in their children. However, there have been some studies that specifically examined SAD in children and reported on correlating disorders found in parents. Last et al. (1987a) examined maternal psychopathologies associated with child SAD and school phobia. In this study, the majority of mothers of SAD and school phobia children had a history of at least one anxiety disorder, with 49.0% of the mothers of children with SAD meeting criteria for generalized anxiety disorder. The most frequent diagnosis of mothers of children with school phobia was simple phobia (21.0%). In addition, mothers of children with SAD were four times more likely to have a history of affective disorders than mothers of children with school phobia. Eighty-nine percent of the SAD mothers who had a history of affective disorders also met criteria for major depression. At the time of their interview, 57.0% of the mothers of SAD children had an anxiety disorder at the same time as their children. The researchers suggested that more severe forms of childhood anxiety are correlated with more severe parental pathologies. However,
participants were recruited from an outpatient anxiety disorders clinic and the findings may not generalize to those with subclinical levels of separation anxiety.

Last and her colleagues (1987b) also examined psychiatric illnesses in mothers of children with SAD and/or overanxious disorder. They reported that 83.0% of the mothers reported a history of anxiety disorders throughout their lives, although the authors did not report on subtypes. In addition, they found that a majority of mothers with a history of affective disorders also met criteria for major depression (91.0% of the mothers of SAD children, 82.0% of the mothers of SAD/overanxious children, and 64.0% of the mothers of overanxious children). Results also showed that approximately 50.0% of the mothers presented with an anxiety disorder at the same time as their child. Although the results suggest that children of parents with anxiety disorders are at a higher risk for developing their own anxiety disorders, this study was conducted on a sample of psychiatrically referred children and thus may not represent all children with SAD.

Martin and colleagues (1999) examined anxiety and depression in parents of children with anxiety-based school refusal. They divided the children into two groups on the basis of their anxiety diagnosis: those with SAD and those with phobic disorders. Parents were examined individually for anxiety and depression. Results showed that 56.0% of mothers of SAD children had a history of panic disorder and agoraphobia, compared to 19.2% of phobic mothers. Both sets of mothers had high rates of depression (48.0% and 53.8%, respectively) but this difference was not statistically significant. In addition, maternal history of any anxiety disorder was high in both groups (80.0% and 76.9%, respectively) but, again, these differences were not significantly different. Paternal history of panic disorder with agoraphobia was also high with SAD.
Like the mothers, many fathers (56.0%) had a history of some anxiety disorder but again this percentage was not statistically different from the fathers with phobias. Although panic disorder with agoraphobia may be a parental pathology related to childhood SAD, there are three limitations in this study. First, children were recruited from a psychiatric hospital and so these results may not be found in the general population. Second, the authors examined children with SAD and school refusal, which also limits the applicability of the study. Third, there was no control group.

These studies have provided a greater understanding of children of parents with psychological disorders. Results indicate that children of adults with anxiety disorders are more likely to have an anxiety disorder themselves than children of adults with no diagnosis. Evidence appears to indicate that certain disorders (e.g., agoraphobia and depression) in parents are likely to produce certain anxiety disorders in children (e.g., overanxious disorder and SAD). Research generally indicates that children of parents with anxiety disorders are more likely to have a similar disorder to their parents than children of parents with other psychiatric disorders such as schizophrenia (Mufson et al., 1992). When examining depression, agoraphobia, or social phobia in parents, SAD often appears among the disorders found in their children. While these studies have laid the groundwork for understanding the heritability of anxiety disorders, more specific research is needed to provide definitive conclusions.

Despite the implications of these studies, their applicability to the population of children with SAD is limited. A majority of these studies used inpatient participants, which provides little evidence that their results can carry over into the general population. In addition, while many studies reported that increased anxiety in parents was highly
correlated with increased anxiety in children, they did not specify which anxiety disorders applied. Many studies also compared two clinical groups and did not use a nonclinical control group. Current research needs to focus on looking at specific disorders, not anxiety in general, and work on applying findings to a non-inpatient population.

Family Environment

In examining environmental factors that correlate with SAD in children, it is important to establish a basis for defining types of family environments. In a review by Kearney and Silverman (1995), five types of family environments were seen as characteristic of children with school refusal behavior. Although these five groupings may not directly apply to SAD, they will provide a basis for understanding different family subtypes that are seen in children with anxiety.

First, the *enmeshed family* is characterized by overindulgent parents and a dependent parent-child relationship. Parents here often do not recognize their child’s need for independence and are overinvolved in details of the child’s life. Second, the *conflictive family* is characterized by higher rates of aggression, hostility, conflict, and coercion. Views regarding this type of family conflict include the psychodynamic perspective, where conflict is an expression of an unsteady mother-child relationship, and the family systems perspective, where aggression is seen as a symptom of inappropriate boundaries between parents and children. The *detached family* is another categorization, defined by a lack of familial interactions and attentiveness to family members. The authors describe this as withdrawal within the family, where the mother seeks more independence from her child.
The fourth family environment is the *isolated family*, where contact outside of the family is minimized. This is the type of family that is rarely seen in therapy, as they often avoid activities outside of the home. Finally, the *healthy family* is marked by low levels of conflict and normal levels of expression and coercion. This type of family has strong support and relationships both within and outside the family. Many families also display characteristics from two or more environments, which may cause complications in assessment and diagnosis. However, these five family dyads will serve as a basis for describing family environments in children with SAD.

Chorpita and Barlow (1998) examined the role of parental control in the development of childhood anxiety. They examined two dimensions of the family environment (warmth and overprotection) that are consistently tied to childhood anxiety. They defined overprotection as a parent’s excessive attempts to protect a child from real or imagined aversive situations. This often has the effect of limiting the child’s opportunities to act independently in the environment. They reported on one study where four parenting styles (rejection, emotional warmth, overprotection, and favoring the child) were used to distinguish among anxious and nonanxious patients. They found that the overprotective parenting style was the best at discriminating the two groups. The other factor, warmth, is the parent’s responsiveness to their child. Low responsiveness or warmth is thought to teach the child that their actions are independent of the environment around them. Thus, a combination of high overprotection and low warmth in parents (control without the emotional bonds) may serve to produce anxiety in their children.

Silove and colleagues (1991) examined adults with panic disorder or generalized anxiety disorder and asked them to recall the parenting styles used when they were
children. They were asked to classify their parents into the following groups: high care and low overprotection, high care and high overprotection, low care and low overprotection, and low care and high overprotection. The researchers found that low care and overprotection correlated highly with anxiety in the participants. In particular, while high overprotection correlated with both panic disorder and generalized anxiety disorder, low care correlated only with panic disorder. However, the limits of this study include the fact that it was retrospective and so memories of parenting styles may not have been accurate. In addition, the study categorized the participants by their current anxiety status, not any diagnoses as children. Thus, while this overprotection/warmth model appears to apply to some anxiety disorders, it is not yet proven to be relevant to SAD.

Although the relationship between SAD and family environmental factors is severely under researched, examining studies conducted on families and children with anxiety disorders may provide insight into the family dyads of this population. Stark, Humphrey, Crook, and Lewis (1990) compared the family environments of children with depression and children with anxiety disorders. They found they were able to predict whether or not the child was in a diagnostic category based on two family environment functions. The first function was the amount of support, closeness, and conflict in the family. The second encompassed concerns about morality and religion, social skills, conflict, enmeshment, and support. They found that children with depression and anxiety, as compared to a control group, perceived their home environments as less supportive and more conflictive. Their families were also seen as more disengaged from
others in general, as they participated in fewer religious, social, or recreational activities than did control families.

Comparing depressive, anxious, and depressive/anxious children, these researchers found that the anxious and depressive children perceived very different family environments from the depression/anxiety children. Stark and colleagues believe this was due to higher levels of symptomology reported in the anxiety and depression groups. In addition, they found that children with depression and some children with anxiety reported less influence in family decision-making than did the depression/anxiety or control children. The researchers hypothesized that this may have prevented the children from developing problem solving skills or may have led to a sense of helplessness. However, this study had two major limitations. First, it did not distinguish between anxiety disorders. Second, it used family environment as a predictor of clinical and nonclinical status and not between clinical diagnoses.

Caster, Inderbitzen, and Hope (1999) examined family environment in youths with social anxiety. Adolescents with high levels of social anxiety perceived their parents as more socially isolated than adolescents with low levels of social anxiety or none at all. In addition, those with higher levels also saw their parents as more ashamed of the child’s shyness and poor performances and more concerned about the opinions of others. The high anxiety group also reported themselves as being less socially active than the other two groups. The researchers hypothesized that continuous parental criticism and overconcern about the opinions of others may be a leading factor in the development of social anxiety. Their data also lends support to the theory that children often model their parents’ social anxiety behaviors. However, this study was conducted on
adolescents, which may not generalize to younger children. In addition, causes of social anxiety may not be the same as causes of separation anxiety.

Messer and Beidel (1994) examined the psychosocial and environmental factors that correlate with anxiety disorders in children. They found that children with anxiety disorders, when compared to children who only had test anxiety and a control group, described their family environment as less conducive to promoting independence through higher family control. In particular, children with overanxious disorder or social phobia reported more rigidity in their homes. The researchers believe this indicates that they are uncomfortable with changes in their environment and thus more resistant to any change. Children with anxiety disorders also reported lower self-competence and higher temperamental rigidity, which researchers believe correlate highly with greater control and inflexibility in these families.

Research on childhood anxiety has discovered various patterns in the family environment. The most prominent theme appears to be decreased amounts of social and family activities. A majority of the studies report lower participation in social activities than control groups and less parental encouragement of these activities to the children. The children also appear to have lower feelings of independence and less influence in family decision-making. They report that their families tend to be more rigid and have greater conflict. Another common theme found in these studies is the children’s feelings that they do not receive much support from their parents and, in fact, often feel that their parents are ashamed or embarrassed by them. These findings appear to point to three of the previously discussed family dyads. There are some characteristics of the enmeshed family (lack of independence on the part of the children), the isolated family (minimal
amounts of social activity condoned by the parents), and the conflictive family (higher rigidity and fighting). However, as none of the studies looked specifically at SAD in children, and one focused on adolescents instead of children, more research needs to be conducted on specifying the family environments of this specific population.

The Current Study

This study extended the research on SAD in children and addressed factors that are missing in previous research. Specifically, this study examined participants not recruited from inpatient or day treatment facilities, which few of the previous studies attempted. In addition, this was a longitudinal study, examining the children’s diagnoses and family environments over a three-year period. This study also utilized a “subclinical” category of SAD, where the child had one or two symptoms of the disorder but did not meet full criteria, and a SAD nonclinical control group. An important strength of the study is its specific focus on child SAD and not any childhood anxiety disorder broadly defined. In addition, there were two independent sources of information: child and parent. These improvements on previous research provided data to help reveal substantial parental/familial predictors of childhood SAD.

Hypotheses

Hypothesis #1: Within-Group/Longitudinal

Children will maintain their clinical status from their first assessment three years ago. Specifically, children previously diagnosed with clinical SAD will again be diagnosed with clinical SAD. Children previously assigned a subclinical diagnosis of
SAD will maintain a subclinical diagnosis. Children previously assessed as nonclinical will remain so.

However, if a change in diagnosis occurred from time one to time two, several factors will correlate with this change. These factors include parental health, parental control, and level of expression in the family. Parental health is defined as the SCL-90-R scales of phobic anxiety, obsessive/compulsive, depression, and somatization. Parental control is defined as the FES scales of independence, control, and cohesion. Level of expression in the family is defined as the FES scale of expression.

**Hypothesis # 2: Between-Group**

The constructs of parental health, parental control, and level of expression in the family will correlate with the child’s clinical status at time one and again at time two.
CHAPTER 2

METHODS

Participants

Participants consisted of 44 children aged 6-7 years. In 1996, 60 original participants were recruited from seven day care settings in Las Vegas: Creative Kids (4 locations), First Presbyterian Preschool, University of Nevada, Las Vegas Preschool, Children's Oasis at the Lakes, Community College Child Development Laboratory School, and Fellowship Family Daycare. The children were initially assessed and divided into three groups of 20: one group was diagnosed with separation anxiety disorder (clinical), one group had one or two symptoms of separation anxiety disorder (subclinical), and one group did not have any symptoms of separation anxiety disorder (nonclinical).

Forty-four of the original 60 participants were re-evaluated three years after their initial evaluation. Sixteen original participants did not participate because they could not be reached. The racial breakdown included: 40 subjects who were Caucasian, 1 who was Hispanic, and 3 who were multiracial. There were an equal number of males and females.
Measures

Diagnostic Interview

The Anxiety Disorders Interview Schedule for Children (ADIS-C; Silverman & Albano, 1996) is a semi-structured interview based on DSM-IV criteria. The ADIS-C screens for 18 DSM-IV disorders and contains sections of specific, criterion-related questions that may eventually allow for a diagnosis of that disorder. Two interviews are available: one for the parent (ADIS-P) and one for the child (ADIS-C). When examining parent and child reports at time two, there was no significant difference in the diagnosis obtained. Therefore, only the parent report results were used in the analyses. In this study, only the section regarding separation anxiety disorder was utilized.

Reliability of the ADIS-C is adequate, with an overall interviewer-observer Kappa of .75 and individual diagnosis Kappas ranging from .64 for overanxious disorder to 1.00 for specific phobia (Silverman & Nelles, 1988). Silverman and Eisen (1992) reported a two-week test-retest reliability of .75 for the ADIS-C. Individual diagnosis Kappas for test-retest reliability ranged from .64 (overanxious disorder) to .84 (specific phobia). Silverman and Rabian (1995) conducted test-retest reliability on each of the specific symptoms for Separation Anxiety Disorder and Overanxious Disorder, with Kappas ranging from .42 to 1.00.

Family Measure

The Family Environment Scale (FES; Moos & Moos, 1986) is a 90-item true-false questionnaire administered to parents. The purpose of the FES is to assess family systems on three levels: system maintenance dimensions, interpersonal relationships, and personal growth. Subscales on the FES include cohesion, expressiveness, conflict,
independence, achievement-orientation, intellectual-cultural orientation, active-
recreational orientation, moral-religious emphasis, organization, and control. For this
study, only the subscales of cohesion, expressiveness, independence, and control were
used from both time one and time two (see Table 1).

The test-retest reliability for the FES after a year ranges from .52 to .89. Internal-
consistency (KR20) for the subscales ranges from .64 to .79 (Moos & Moos, 1981). In
addition, average subscale intercorrelations range from .01 (low) to .46 (moderate).
Bloom (1985) also found moderate internal consistency (.65-.85) for 8 of the 10
subscales.

Parent Measure

The Symptom Checklist-90-R (SCL-90-R; Derogatis, 1994) is a 90-item self-
report multidimensional symptom profile. The SCL-90-R assesses how frequently
certain psychological (not personality) symptoms occurred in the past week. Nine
symptom dimensions include somatization, obsessive-compulsiveness, interpersonal
sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and
psychoticism. In addition, three global scales furnish summary levels of psychological
distress: global severity index (GSI), positive symptom distress index, and positive
symptom total. For this study, only somatization, obsessive-compulsive, depression, and
phobic anxiety symptom dimensions were used. The SCL-90-R is scored on a 5-point
Likert-type scale of distress ranging from 0 (not at all) to 4 (extremely) and is used with
persons aged 13 years and older.
<table>
<thead>
<tr>
<th>Table 1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Breakdown of Diagnostic Measures Across Time</strong></td>
</tr>
<tr>
<td><strong>Time One</strong></td>
</tr>
<tr>
<td>FES</td>
</tr>
<tr>
<td>- Cohesion</td>
</tr>
<tr>
<td>- Expressiveness</td>
</tr>
<tr>
<td>- Control</td>
</tr>
<tr>
<td>- Independence</td>
</tr>
<tr>
<td>ADIC-C</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>ADIS-C</td>
</tr>
</tbody>
</table>
Horowitz, Rosenberg, Baer, Ureno and Villasenor (1988) calculated internal consistency (coefficient alpha) for the SCL-90-R ranging from .79 (paranoid ideation) to .90 (depression). Horowitz and colleagues (1988) also found ten-week test-retest reliability to range from .68 (somatization) to .83 (paranoid ideation). Derogatis, Rickels, and Rock (1976) found that test-retest reliability after one week ranged from .78 (hostility) to .90 (phobic anxiety). They also correlated the SCL-90-R with scales on the MMPI and found good convergent-discriminant validity that ranged from .41 (phobia/anxiety) to .75 (depression).

Consent forms (Appendix I) and demographic information sheets (Appendix II) were administered to parents. Information was solicited with respect to age and gender of both child and parents, socioeconomic status, ethnicity, occupation, and sibling and grandparent status. Consent was obtained from the parents.

Procedure

The setting for data collection was at the discretion of the participants, either at the University of Nevada, Las Vegas School Refusal Clinic or at the homes of the participants. The researcher first interviewed the child, while parents completed the consent form, demographic information sheet, and questionnaires. When possible, parents completed the questionnaires together. After the child’s interview, the researcher interviewed the parents. When their interview was completed, parents finished any questionnaires they did not complete. All information was then placed in an envelope and coded numerically to ensure the confidentiality of the participants. The entire data collection process took approximately two hours. Each participant was paid $50 for
participating. To limit bias, the participant's original diagnosis was not compared to their new diagnosis until after the follow-up data were collected.

To establish reliability for the interviewer's ADIS diagnoses, the interviewer first observed a clinical psychologist interview a child and his parents with the ADIS. The interviewer and psychologist then compared their diagnoses and recorded whether they agreed or disagreed on each of the parent and child interviews. The interviewer observed at least three interviews before calculating the reliability of the diagnoses. The reliability was calculated by dividing the total number of agreements by the total number of agreements and disagreements. It was decided that if an 80.0% reliability had been reached after the first three interviews, then the interviewer was considered reliable in her diagnoses. If reliability was not met, then the interviewer would continue to interview with the clinical psychologist until reliability was met. Reliability was met after three interviews, with a reliability of 96.0%. During the subsequent interviews, there were random diagnosis reliability checks by having a second trained interviewer sit in on the interview session and later compare their diagnosis with those of the first interviewer. Again, reliability was calculated by dividing the total number of agreements by the total number of agreements and disagreements. Initially, there were 16 random reliability checks. If the total reliability exceeded 80.0%, then no further random reliability checks would be conducted. However, if reliability was not met, then random reliability checks would continue until reliability is met. After 16 random checks, reliability was met at 92.0%

Only completed packets were used for data analysis. A packet was considered complete if it contained both the parent and child versions of the ADIS, the Family
Environment Scale, the Symptom Checklist-90-R, the consent form, and demographics sheets. All packets were returned completed.

Data Analysis

A stepwise regression analysis was used to identify significant predictors of clinical status. Specifically, twelve variables were chosen as possible predictors. These twelve variables included FES expression, cohesion, independence, and control at time one and time two (N= 8 variables), and SCL-90-R depression, obsessive/compulsive, phobic anxiety, and somatization at time two. Because the number of subjects was too small to support this many dependent variables in a regression, the twelve variables were subjected to factor analysis with varimax rotation. When the variables were forced into three or four factors, variables from time one and time two were mixed into the same factor. Thus, the resulting matrix used five factors. Factor one included FES time one cohesion and time one expression. Factor two included FES time one independence and time one control. Factor three included FES time two control. Factor four included FES time two independence, time two cohesion, and time two expression. Factor five included all four SCL-90-R scales: depression, obsessive/compulsive, phobic anxiety, and somatization.

This study stated that three constructs (parental control, parental health, and level of expression in the family) would associate with clinical status at time one, at time two, and change in clinical status from time one to time two. These constructs encompassed twelve dependent variables which, after factor analysis, were supposed to be grouped into three factors, each one corresponding with one construct. This was not the case. Neither a three factor or four factor analysis was possible, as at both times FES scales from time
one and time two were in the same factors. Thus, five factors were produced, but few of them matched with the three constructs as originally expected. While factor number five (containing the four SCL-90-R scales) did match the construct of parental health, the other four factors did not match as easily. It had been expected that the time one and time two FES scales of expression would be grouped as one factor, which they were not. In addition, the FES scales of independence, control, and cohesion did not factor together to produce the construct of parental control. Thus, the constructs that were originally expected were not produced.

Each of the 5 factors produced factor loadings for each individual variable (see Table 2). The factor loadings were used for weighting purposes in that each individual score of a variable was multiplied by that variable's factor loading. The weighted scores were then summed to derive a single score per subject for each factor. These five factor scores were the predictors that were entered into the regression analyses.

**Change in Clinical Status from Time One to Time Two**

Hypothesis one stated that there would be no change in SAD diagnosis from time one to time two. Three chi-square analyses were performed. The Wilcoxon Matched-Pairs Signed-Ranks Test was performed to test the normality of the underlying distribution in the number of SAD diagnosis changes from time one to time two. This statistic tested the null hypothesis that two related samples drawn from symmetrical populations will have the same mean. Thus, the analysis examined the number of cases where the diagnosis stayed the same from time one to time two, how many cases increased in severity, and how many decreased. As the null hypothesis expects that the
Table 2

Factor Loadings for the Twelve Dependent Variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>Factor 1</th>
<th>Factor 2</th>
<th>Factor 3</th>
<th>Factor 4</th>
<th>Factor 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor One</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Express. (Time 1)</td>
<td>.87993</td>
<td>-.12083</td>
<td>-.01463</td>
<td>.22390</td>
<td>.04487</td>
</tr>
<tr>
<td>-Cohesion (Time 1)</td>
<td>.75270</td>
<td>.27628</td>
<td>-.01534</td>
<td>.17883</td>
<td>.09537</td>
</tr>
<tr>
<td>Factor Two</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Control (Time 1)</td>
<td>-.11522</td>
<td>.90304</td>
<td>.00341</td>
<td>.02627</td>
<td>.00244</td>
</tr>
<tr>
<td>-Indepen. (Time 1)</td>
<td>-.16764</td>
<td>-.49293</td>
<td>.00581</td>
<td>-.04211</td>
<td>.14126</td>
</tr>
<tr>
<td>Factor Three</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Control (Time 2)</td>
<td>.11031</td>
<td>-.07874</td>
<td>.91787</td>
<td>-.13947</td>
<td>-.08295</td>
</tr>
<tr>
<td>Factor Four</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Express. (Time 2)</td>
<td>.00562</td>
<td>.01178</td>
<td>-.18568</td>
<td>.78828</td>
<td>-.20192</td>
</tr>
<tr>
<td>-Indepen. (Time 2)</td>
<td>.20825</td>
<td>-.16399</td>
<td>.17772</td>
<td>.66806</td>
<td>.07350</td>
</tr>
<tr>
<td>-Cohesion (Time 2)</td>
<td>.16893</td>
<td>.13323</td>
<td>-.17939</td>
<td>.75906</td>
<td>.01376</td>
</tr>
<tr>
<td>Factor Five</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Depression</td>
<td>-.11974</td>
<td>.06743</td>
<td>.13275</td>
<td>-.07583</td>
<td>.86717</td>
</tr>
<tr>
<td>-Obsessive/Comp.</td>
<td>-.00732</td>
<td>-.15639</td>
<td>.13561</td>
<td>.14498</td>
<td>.87387</td>
</tr>
<tr>
<td>-Phobic Anxiety</td>
<td>-.01373</td>
<td>-.17603</td>
<td>-.32905</td>
<td>-.15676</td>
<td>.70254</td>
</tr>
<tr>
<td>-Somatization</td>
<td>-.16554</td>
<td>.07871</td>
<td>-.27386</td>
<td>-.03750</td>
<td>.75538</td>
</tr>
</tbody>
</table>
distribution of difference score will be symmetrical to zero, a significant Wilcoxon test means that the centers for time one and time two data are unequal. In addition to the Wilcoxon, a chi-square was also performed to examine the breakdown of SAD diagnoses at time one, and another was performed to examine the breakdown of the SAD diagnoses at time two.

For any significant change in clinical diagnosis over time, an additional stepwise regression analysis was used to test the hypothesis that several constructs would significantly correlate with any change in SAD status. For the regression analysis, each diagnosis was assigned a number: clinical SAD was 1, subclinical SAD was 2, and nonclinical SAD was 3. Clinical status at time two was subtracted from clinical status at time one, thereby producing scores ranging from 2 to −2. A positive two signified that the participant’s diagnosis worsened two levels, or from nonclinical status to a diagnosis of clinical SAD. A positive one signified that the diagnosis worsened one level: either from nonclinical status to subclinical SAD or from subclinical SAD to clinical SAD. A zero signified that there was no change in clinical status from time one to time two. A negative one signified that the participant’s diagnosis improved one level: either from clinical SAD to subclinical SAD, or from subclinical SAD to nonclinical status. A negative two signified that the diagnosis improved two levels: from clinical SAD to nonclinical status. Thus, a positive score of one or two meant that the condition had gotten worse, while a negative number meant that the condition had improved. The stepwise regression analysis was performed to see if any of the five factor scores (variables) significantly correlated with change in clinical status.
Prediction of Clinical Status at Time One and at Time Two

The second hypothesis stated that several constructs would significantly correlate with clinical status in children at time one and at time two. These constructs include parental control (defined as the FES scales of independence, control, and cohesion), parental health (defined as the SCL-90-R scales of phobic anxiety, obsessive/compulsive, depression, and somatization), and level of expression in the family (defined as the FES scale of expression). The first multiple regression examined whether the five factors significantly predicted SAD diagnosis at time one. A second regression analysis examined whether the five factors significantly associated with SAD diagnosis at time two.
CHAPTER 3

RESULTS

Change in Clinical Status from Time One to Time Two

A Wilcoxon Matched-Pairs Signed-Ranks Test was performed to examine the normality of the underlying distribution of the change in SAD category from time one to time two. Results indicated that the change in SAD categories was not equal across time. An increased number of participants had improved diagnoses at time two; Wilcoxon T = 3.82, p < .01.

A chi-square analysis examined the number of cases for each SAD diagnosis at time one. As expected, no significant difference was found in the proportion of clinical, subclinical, and nonclinical SAD diagnoses at time one, Chi Square (2) = 3.32, ns.

Using time two SAD diagnoses, there were significant differences between the expected number of cases and the actual number of cases observed, Chi Square (2) = 30.05, p < .01. Thus, comparing these results to those from time one, there was a statistically significant change in diagnostic status among the 44 participants from time one to time two (see Table 3). A more complete breakdown of how the children's diagnoses changed over time can be seen in table 4.
Since clinical status was not stable over time, a stepwise regression analysis was conducted involving the five factors and SAD clinical status change. The first variable that was entered was Factor 5 with a significant overall effect, $F(1, 42) = 8.85, p<.01$. Thus, factor five did correlate with overall SAD status change from time one to time two. None of the other four factors were entered into the equation. The $R^2$ was .17412 with an adjusted $R^2$ of .15446. There was a Multiple $R$ of .41728. Table 5 shows the Beta values of each of the five factors.

Association with Clinical Status at Time One and at Time Two

Time One

The second hypothesis stated that several factors would correlate with the clinical status of the participants. To test this, a stepwise multiple regression was conducted involving the five factors and SAD diagnosis at time one. Factor 5 was the only factor entered, $F(1, 42) = 2.69, p<.05$. Thus, factor five did predict SAD status at time one. None of the other four factors were significant (see Table 5). The $R^2$ was .26191 with an adjusted $R^2$ of .16479. There was a Multiple $R$ of .51177.

Time Two

A stepwise multiple regression was then conducted involving the five factors and SAD diagnosis at time two. Factor 5 was again the only factor entered into the equation, $F(1, 42) = 2.89, p<.05$. Once again, the factor five did predict SAD status at time two. None of the other four factors were significant (see Table 5). The $R^2$ was .27559 with an adjusted $R^2$ of .18027. There was a Multiple $R$ of .52497.
**Table 3**

**Chi Square Analysis of SAD Status at Time One and Time Two***

<table>
<thead>
<tr>
<th>SAD Status</th>
<th>Time One</th>
<th>Time Two</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical</td>
<td>17</td>
<td>2</td>
</tr>
<tr>
<td>Subclinical</td>
<td>18</td>
<td>11</td>
</tr>
<tr>
<td>Nonclinical</td>
<td>9</td>
<td>31</td>
</tr>
</tbody>
</table>

*The expected number of cases for each diagnosis at time one and time two is 14.67.*
# Table 4

## Breakdown of SAD Diagnosis Change Over Time

<table>
<thead>
<tr>
<th></th>
<th>Nonclinical</th>
<th>Subclinical</th>
<th>Clinical</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Time Two</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Time One</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonclinical</td>
<td>2</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>Subclinical</td>
<td>13</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Clinical</td>
<td>16</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>
Table 5

Stepwise Regression Results with Five Factors and Change in Clinical Status and SAD Diagnosis at Time One and Time Two

<table>
<thead>
<tr>
<th>Change in Clin. Stat.</th>
<th>Beta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor 1</td>
<td>-.236950</td>
</tr>
<tr>
<td>Factor 2</td>
<td>-.176667</td>
</tr>
<tr>
<td>Factor 3</td>
<td>-.046249</td>
</tr>
<tr>
<td>Factor 4</td>
<td>-.214150</td>
</tr>
<tr>
<td>Factor 5</td>
<td>.417276*</td>
</tr>
</tbody>
</table>

Diagnosis at Time One

| Factor 1              | -.185797   |
| Factor 2              | -.218544   |
| Factor 3              | .103294    |
| Factor 4              | -.271502   |
| Factor 5              | .373096**  |
Table 5 (con’t)

<table>
<thead>
<tr>
<th>Diagnosis at Time Two</th>
<th>Beta</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor 1</td>
<td>.112455</td>
</tr>
<tr>
<td>Factor 2</td>
<td>.223370</td>
</tr>
<tr>
<td>Factor 3</td>
<td>.182574</td>
</tr>
<tr>
<td>Factor 4</td>
<td>.195825</td>
</tr>
<tr>
<td>Factor 5</td>
<td>-.387352*</td>
</tr>
</tbody>
</table>

*- significant at the .01 level

** * - significant at the .05 level
CHAPTER 4

DISCUSSION

The purpose of this study was to empirically and longitudinally examine the association between parental variables, family environment, and status of children’s Separation Anxiety Disorder (SAD). Two hypotheses were examined. The first hypothesis predicted that there would be no change in the children’s clinical SAD status from time one to time two. The data did not support this hypothesis. While the results showed no significant difference in the proportion of clinical SAD, subclinical SAD, and nonclinical diagnoses at time one, there was a change among the diagnoses at time two. Specifically, there was a significant increase in the number of nonclinical diagnoses and a decrease in both subclinical and clinical diagnoses. This may indicate that a diagnosis of SAD is relatively unstable over time and that most changes in the diagnoses represent an improvement in SAD status. No participants obtained any psychological counseling from time one to time two, so it may be argued that a diagnosis of SAD is usually temporary over time and remission on some level may be expected.

One of the main possibilities as to why the current findings did not reveal stability in SAD diagnoses may be that this hypothesis was based on studies where the authors concluded that young children with anxiety tended to remain anxious as they matured. However, these studies did not specify the type of anxiety the children developed, nor did they examine the stability of SAD over a few years. Thus, while it may be true that
anxiety disorders usually persist throughout childhood, it may not necessarily be true for SAD.

There are various implications of SAD not being a stable disorder. Parents may choose not to seek treatment if their young children display signs of SAD. If the disorder tends to lessen over time, they may choose to wait and hope their children’s symptoms will remiss over time. However, if the young children are too anxious to leave their parents, they may not go to school or socialize with friends. This deficiency early in life may have long-lasting effects on the child, even if their anxiety recedes later. These are just some of the implications of SAD not being a stable disorder.

The second hypothesis in this study stated that three constructs (parental control, parental health, and level of expression in the family) would associate with clinical status at time one, at time two, and change in clinical status from time one to time two. These constructs encompassed twelve dependent variables which, after factor analysis, were supposed to be grouped into three factors, each one corresponding with one construct. This was not the case. Not only were five factors produced, but few of them matched with the three constructs as originally expected. While factor number five (containing the four SCL-90-R scales) did match the construct of parental health, the other four factors did not match as easily. It had been expected that the time one and time two FES scales of expression would be grouped as one factor, which they were not. In addition, the FES scales of independence, control, and cohesion did not factor together to produce the construct of parental control. Thus, the constructs that were originally expected were not produced.
The most reasonable explanation for the unexpected division of the dependent variables lies in the difficulty in trying to define and measure an abstract concept. As there was no one scale where the definition of ‘parental control’ matched this study’s definition, it was difficult to find an appropriate measure. Thus, although the definitions of parental control may have had similarities, either the measure used was not the most appropriate one to use or the study’s definitions of the abstract family constructs were not measurable.

In addition, with such a small sample size, the factor structure may be unstable. A larger sample size may have produced different results. However, as this was a longitudinal study, the sample size was not one that could be manipulated and thus the analysis and results were performed taking into consideration that it is a small sample size.

A further breakdown of the scores aided in the interpretation of the results. It was necessary to examine the change in clinical status (where scores ranged from 2 [the child’s diagnosis worsened two levels] to –2 [the child’s diagnosis improved two levels]) and the individual parental scores from the twelve variables. For the SCL-90-R data, the parents with lower scores on all four variables (somatization, depression, phobic anxiety, and obsessive/compulsive) tended to have children whose diagnosis improved one or two levels (see Table 2). Their scores were consistently below or at the test mean of 50. Parents of children whose diagnoses worsened one or two levels, however, consistently had higher scores on the SCL-90-R variables (see Table 6). Their scores were usually above the mean and often in the critical range (above 60). This breakdown shows a
relationship between lower parental psychopathology scores and an improvement in the child’s clinical diagnosis.

In particular, two of the SCL-90-R variables (obsessive/compulsive and depression) had the greatest range in scores, going from average levels (at the mean) of depression and obsessive/compulsive in parents of children whose diagnoses improved two levels to clinical levels of depression and obsessive/compulsive (above 60) to those who worsened two levels. A speculation may be that these parental pathologies are most related to the development of SAD in children and thus they may be the best parental pathology predictors of future SAD status in children. This supports such research as Weissman and colleagues (1984a, 1984b) where rates of anxiety in children were high if the parent was diagnosed with depression. It also supports theories that higher anxiety in parents (such as the obsessive/compulsive variable and possibly the phobic anxiety variable) correlate with more severe anxiety in children (Kashani et al., 1990; Last et al., 1987a).

Thus, factor five appears to have strong links as predictors of SAD change. Possibly, if a child has been exposed to low levels of depression and anxiety in their parents, their SAD will decrease over time. There are two possible explanations for this. First, the child may be modeling their parent’s behavior. For example, if a parent remains calm in an anxiety-provoking situation (e.g., the child leaving for their first day of school), a child may learn to model the parent’s response instead of showing separation anxiety symptoms. Conversely, if a parent is anxious before the child leaves for their first day of school, a child may then become anxious. The second possibility is
<table>
<thead>
<tr>
<th>Clinical Status Change</th>
<th>-2</th>
<th>-1</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCL-90-R</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>51.00</td>
<td>50.36</td>
<td>53.33</td>
<td>62.43</td>
<td>63.00</td>
</tr>
<tr>
<td>Somatization</td>
<td>46.69</td>
<td>46.14</td>
<td>50.17</td>
<td>50.71</td>
<td>55.00</td>
</tr>
<tr>
<td>Obsess./Compul.</td>
<td>50.93</td>
<td>52.57</td>
<td>55.17</td>
<td>58.86</td>
<td>64.00</td>
</tr>
<tr>
<td>Phobic Anxiety</td>
<td>46.00</td>
<td>48.07</td>
<td>50.00</td>
<td>53.00</td>
<td>58.00</td>
</tr>
</tbody>
</table>

*Clinical Status Change:
-2 – child’s diagnosis improved two levels
-1 – child’s diagnosis improved one level
0 – child’s diagnosis did not change
1 – child’s diagnosis worsened one level
2 – child’s diagnosis worsened two levels

*These scales have a mean of 50 and a clinical status of 60+
that children with SAD are having their anxiety symptoms reinforced by parents with pathology. If a child says he is afraid to go to school because something bad could happen on the way, an anxious parent may then agree with the child and walk him to school every day to protect him. This would reinforce the child’s fear of separation. In contrast, if a non-anxious parent praises a child for walking to school by himself despite his fear, then this parent would be reinforcing the child’s attempts to conquer his fear. However, these are just theories. As this is not a causal study, it cannot be stated that parent pathology causes SAD; only that they are associated.

When the same breakdown of change in clinical status and family scores was performed on FES data, the results are not as clear as those of the SCL-90-R data. In table 7 (time one) and table 8(time two) there are only a few instances of steady increase or decrease in scores across clinical status change. In time one, cohesion tends to be higher in the SAD status improvement groups. In addition, independence appears to be much lower for the groups that worsened in SAD status. In addition, there is a general increase in level of control as the child’s diagnosis improves at time two (table 4). Thus, there are trends of higher levels of cohesion and independence for those children with low SAD status at time one and an increase in control for children whose diagnosis improved at time two. While these trends follow the idea of a healthy family environment (including higher levels of expression, cohesion, and independence, and lower levels of control), they are not strong enough to support the hypothesis that parental control can predict SAD status. In addition, as none of the factors with FES variables were ever factored into a multiple regression equation, it can be assumed that they do not associate with SAD. Perhaps cohesion, independence, control, and
expression were not the most reliable constructs to define parental control. Another likely explanation would be that parents who scored higher on levels of pathology may not be able to make accurate assessments of the family environment and thus data from the FES was not as reliable as hoped.

There are various possible explanations for the emphasis on using parental health as a predictive value. It could be simply that specific parental psychological constructs such as depression or phobic anxiety correlate highly with anxiety disorders in children while family constructs such as communication do not. This theory of parental psychopathologies and disorders in their children is supported in many other studies (e.g., Walsh-Allis et al., 1988, Capps et al., 1996, Mancini et al., 1996). Although none of these studies performed a longitudinal study of SAD, the theory that parents with depression or anxiety disorders are more likely to have children with similar disorders can be applied here.

Another explanation of why parental health was the most predictive for both time one and time two clinical status may involve how these constructs were measured. The scales used from the SCL-90-R were based upon one parent’s self-examination. They were required to answer various questions about how they had felt during the past week. Thus, as the questionnaire was only about one parent and what they experienced, there was no need to consult someone else about the answers. In addition, the questions were straightforward about certain disorders, such as depression or anxiety. There were few, if any, abstract concepts being tested. The FES, however, required the parent to consider situations that involved the entire family, how they interact, and the level of family
Table 7

Average FES Time One Scores Separated by Change in Clinical Status*

<table>
<thead>
<tr>
<th>Clinical Status Change</th>
<th>-2</th>
<th>-1</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>FES</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cohesion</td>
<td>59.94</td>
<td>48.78</td>
<td>63.00</td>
<td>50.71</td>
<td>38.00</td>
</tr>
<tr>
<td>Independence</td>
<td>46.94</td>
<td>46.00</td>
<td>46.00</td>
<td>52.00</td>
<td>36.00</td>
</tr>
<tr>
<td>Expressiveness</td>
<td>58.44</td>
<td>55.86</td>
<td>61.17</td>
<td>47.14</td>
<td>66.00</td>
</tr>
<tr>
<td>Control</td>
<td>54.25</td>
<td>52.57</td>
<td>54.83</td>
<td>49.00</td>
<td>54.00</td>
</tr>
</tbody>
</table>

*Clinical Status Change:
-2 – child’s diagnosis improved two levels
-1 – child’s diagnosis improved one level
0 – child’s diagnosis did not change
1 – child’s diagnosis worsened one level
2 – child’s diagnosis worsened two levels

*These scales have a mean of 50 and a clinical status of 60+
Table 8

**Average FES Time Two Scores Separated by Change in Clinical Status***

<table>
<thead>
<tr>
<th>Clinical Status Change</th>
<th>-2</th>
<th>-1</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cohesion</td>
<td>59.37</td>
<td>61.93</td>
<td>52.83</td>
<td>57.00</td>
<td>60.00</td>
</tr>
<tr>
<td>Independence</td>
<td>49.12</td>
<td>52.07</td>
<td>37.80</td>
<td>50.86</td>
<td>45.00</td>
</tr>
<tr>
<td>Expressiveness</td>
<td>59.56</td>
<td>59.50</td>
<td>51.33</td>
<td>49.14</td>
<td>60.00</td>
</tr>
<tr>
<td>Control</td>
<td>56.06</td>
<td>57.64</td>
<td>57.50</td>
<td>53.85</td>
<td>48.00</td>
</tr>
</tbody>
</table>

*Clinical Status Change:
- -2 – child’s diagnosis improved two levels
- -1 – child’s diagnosis improved one level
- 0 – child’s diagnosis did not change
- 1 – child’s diagnosis worsened one level
- 2 – child’s diagnosis worsened two levels

*These scales have a mean of 50 and a clinical status of 60+*
functioning as a whole. The parents were allowed to consult over the answers, which may have caused the answers to be modified and more restricted than if only one parent had responded. In addition, this scale questioned the actions of all the individuals in the family, not just the respondent, and asked about more difficult constructs, such as communication, rather than something more straightforward like depression. Thus, the different levels of personal experience reflected in the two questionnaires may account for why one appears to play a more important role in predicting SAD status.

The FES tested constructs that were more difficult to define and break down into questions, which may be another reason parental health appeared to have greater predictive value. While depression may have similar definitions across measures, a construct such as ‘parental control’ undoubtedly has very different definitions across questionnaires. Thus, it was harder to find a questionnaire that tested the study’s operational definition of ‘parental control’ than it was to find a questionnaire that tested the definition of ‘phobic anxiety.’ These different definitions may account for why the FES scales did not play a role in predicting clinical status. Perhaps they did not test the construct the study was trying to define or perhaps family constructs are, in general, harder to evaluate than anxiety disorders. This is yet another possible explanation for the higher predictive value of the SCL-90-R scores for both time one and time two data.

Although this study did provide a great deal of information for future studies, there were some limitations. First, a larger sample size would have been useful. Sixteen of the original 60 subjects were not retested. Not only would having all of the original 60 subjects have improved the power of the experiment, but also a great deal of information was lost by not having them. Of the 44 participants who did volunteer to be retested,
however, there were approximately an equal amount in all three original diagnostic categories. Nonetheless, the information provided by the other 16 would have been helpful.

Examining the 16 families who did not participate in this follow up study, at time one, 3 had SAD, 2 had subclinical SAD, and 11 were given a nonclinical status. There were an approximately equal number of males and females (7 males and 9 females). The majority of the children (12) were Caucasian. It does not appear that these 16 were dramatically different from the 44 who agreed to be re-evaluated.

A second limitation of the study involved the number of dependent variables. This relates back to the small sample size, as very few dependent variables could be used with forty-four subjects. If this study had a greater number of subjects, there could have been more questionnaires testing parental health and family constructs. This could have provided stronger evidence that parental health is in fact the most significant predictor of SAD status, instead of having to consider the possibility that the one questionnaire used to measure parental health is simply more valid than the one questionnaire used to measure family variables.

A third limitation of the study involves the restricted information provided by the children. Because the children were too young to complete questionnaires on their family environment, the study had to depend on the parents’ view of the family dynamics. In addition, it was the parent’s SAD diagnosis that was used in the analyses, not the children’s. The reason for this is that the parent’s diagnosis was used three years ago, and for sake of the analyses the parent’s was again used in this study. If the children were again followed up in three years, they would then be old enough to complete
questionnaires on family environment and this would provide a more in depth and reliable view of the family dynamics.

Future research should also focus on more specific parental psychopathological disorders, such as just depression or just anxiety, when studying SAD. Following much of the previous research done, the parental pathologies in this study were not specified, but instead grouped into one construct. It would be better to examine one or two pathologies in particular or to concentrate on only one family construct. This study may have attempted to examine too much for the limited number of subjects tested. A more focused longitudinal study would probably provide greater information on the predictors of SAD in children.

In conclusion, Separation Anxiety Disorder does not appear to be a stable disorder over time in young children, with most children showing some improvement in three years. One of the best predictors of both past and present levels of SAD is the parent’s levels of pathology. The higher the levels of parental pathology, the more likely it was that the child’s diagnosis would worsen over time. Family environment, however, was not as strong a predictor as parent pathology, as there was no strong relationship between higher levels of expression, independence, and cohesion and an improvement in the child’s diagnosis. Thus, to predict what changes may occur to a child’s SAD status, or to define their current levels of SAD, an examination of the parent’s levels of pathology would be beneficial.
Dear Parents:

As part of an effort with the Department of Psychology and the UNLV Child Anxiety Disorders and School Refusal Clinic at the University of Nevada, Las Vegas, we are interested in having you and your child participate in a research study looking at children's development. Changes during growth often create anxiety in youngsters, and it is this level of anxiety and related factors that we are interested in examining.

Specifically, we are asking permission to interview you and your child separately, to have you complete several questionnaire regarding you, your child, and your family, and to conduct a brief observation of you and your child. This should require about 90 minutes of your family's time. A payment of $50.00 will be sent to you at the completion of the session. In addition, permission is sought to send a questionnaire to you child's teacher.

All information obtained in this study will be kept confidential. All information collected will be encoded numerically to ensure confidentiality. The coded data will only be examined by researchers and will not be used for any purpose other than the scientific goals of the study. Furthermore, at no time during the study will your name or your child's name be associated with the responses you or your child gives. There are no known hazards from participating in this study. Once collected, the information you and your child provide will be summarized into one, large data pool. General results of the study will be made available to parents upon request at its conclusion.

If you wish your child to participate, please sign this consent form. Of course, you or your child may feel free to withdraw from this study at any time, even after you have consented to participate. No loss of benefit will occur. If you have any questions regarding this study at any time, please speak with Courtney Pursell or Cheryl Tillotson, the graduate coordinators of this project (895-3305) or Dr. Christopher Kearney, the principal investigator (895-0183). For questions regarding the rights of research subjects, please contact the UNLV Office of Sponsored Programs at 895-1357. Thank you for your consideration.

_________________________     __________________
Child's name                     Date

_________________________
Parent signature

Christopher A. Kearney, Ph.D.     Courtney Pursell and Cheryl Tillotson
Associate Professor, Psychology M.A. Candidates, Psychology
University of Nevada, Las Vegas University of Nevada, Las Vegas

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
APPENDIX II

DEMOGRAPHIC INFORMATION SHEET
Information sheet

This sheet is to be filled out by one or both parents (or guardians). Once again, the information you provide will be coded numerically and will in no way be associated with you or your child. Please feel free to skip an item if you don’t feel comfortable answering, however it is hoped that you will respond honestly to all items.

1. Child’s name: __________________________

2. Child’s birth date: ______________________

3. Child’s gender: (circle one) M F

3. Child’s race: (circle one)
   - Asian
   - African-American
   - Caucasian
   - Hispanic
   - Multiracial
   - Native American
   - Other _____________

Please indicate whether you are the child’s parent or guardian by circling one.

4. Mother’s / Guardian’s name: __________________________ Age: __________

5. Father’s / Guardian’s name: __________________________ Age: __________

6. Did mother / guardian graduate from high school? yes no
   How many years, if any, did mother/guardian attend school after high school? _____

7. Did father / guardian graduate from high school? yes no
   How many years, if any, did father/guardian attend school after high school? _____

8. Mother’s / Guardian’s occupation: __________________________

9. Father’s / Guardian’s occupation: __________________________

10. Number of hours mother / guardian works outside the home per week? __________

11. Number of hours father / guardian works outside the home per week? __________

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
12. Age (in years) and gender of all siblings:

<table>
<thead>
<tr>
<th>Age</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>F</td>
</tr>
<tr>
<td></td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>F</td>
</tr>
</tbody>
</table>

13. Marital status of parents / guardians? (circle one)

- Married
- Never married
- Separated
- Divorced

14. If parents / guardians are separated or divorced, circle one of the following:

- Joint custody
- Mother had custody
- Father has custody

15. Is one or both of the custodial parents remarried?  yes  no

If yes, circle one of the following:

- Both are remarried
- Only mother is remarried
- Only father is remarried

16. If parents do not have joint custody, how many hours per month does the noncustodial parent spend with the child?  _____________

17. Do any of the child’s grandparents live in town?  yes  no

18. How many hours per month does the child spend with his / her grandparents?  ___

19. Is your child adopted?  yes  no

20. What is your family’s average annual income?  _____________

In the future, the researcher may want to make brief contact with you again as a follow up. Of course, your cooperation would be entirely voluntary at this time again. Please provide the following information if it is all right that someone contact you.

Mailing address:  

______________________

______________________

______________________

Telephone Number:

home:  ___________

work:  ___________

Reproduced with permission of the copyright owner. Further reproduction prohibited without permission.
REFERENCES


VITA

Graduate College
University of Nevada, Las Vegas

Courtney Ryan Pursell

Local Address:
2121 E. Warm Springs Rd. Ap. #2177
Las Vegas, NV 89119

Degree:
Bachelor of Arts, Psychology and English, 1997
Franklin and Marshall College

Special Honors and Awards:
Graduate Student Summer Research Assistantship
Graduate Student Research Award

Publications:

Thesis Title: Parent Pathologies and Family Environment as Correlates of Child Separation Anxiety Disorder

Thesis Examination Committee:
Chairperson, Dr. Christopher Kearney, Ph.D
Committee Member, Dr. Marta Laupa, Ph.D
Committee Member, Dr. Jeffrey Kern, Ph.D
Graduate Faculty Representative, Dr. Alice Corkill, Ph.D