TRAJECTORIES, ANTECEDENTS, AND OUTCOMES OF CHILDHOOD SOMATIZATION IN SCHOOL-AGE BOYS

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The current study had three goals that were designed to extend our understanding of childhood somatization (SOM). The first goal was to use a group-based trajectory analysis to plot developmental pathways of SOM. The second goal was to use a developmental psychopathology framework to identify risk factors from multiple domains that discriminated SOM trajectories. The third and final goal was to examine the relationship between child maladjustment, including functional impairment, and SOM trajectory group status. These goals were conducted with a sample of 310 ethnically diverse, low-income boys followed longitudinally from ages 2 to 12, using multiple methods and informants. Similar to research using broadly-defined internalizing behaviors, three developmental trajectories were identified: No, Low Increasing, and Moderate Increasing. The majority of the boys (i.e., 82%) were reported as displaying at least low to moderate levels of SOM across childhood. In addition to these three trajectories, a small group of boys (n = 5) demonstrated a distinct pattern of SOM, called the Moderate to High (MTH) group. Follow-up analyses indicated that the Moderate Increasing group was differentiated from the No group by higher levels of maternal depressive symptoms and parent-child conflict, whereas the MTH group demonstrated lower levels of social skills than the other three groups. Higher levels of both child negative affectivity and parent-child conflict discriminated the Low Increasing group from the No group. Differences in child outcomes at ages 11 and 12 were not found. The implications of the results for clinical intervention and future research are discussed.

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PREFACE

This project never would have seen the light of day had it not been for the inspiration and invaluable clinical experience I gained from working with Drs. Lin Ewing and Anna Marsland in the area of pediatric pain. I am grateful to my advisor, Dr. Daniel Shaw, for his support and guidance in my pursuit in studying this novel subject. My committee members, Drs. Susan Campbell, John Campo, Daniel Nagin, and Robert Noll, also have provided much knowledge, support, and encouragement over the years. I also want to thank Dr. Bobby Jones for his consultation on the TRAJ analyses. I am absolutely indebted to my parents, Richard and Elizabeth Beck, who have provided every imaginable type of support over the course of my graduate career. My sister, Jennifer Beck, and my fiancée, Douglas Hackworth, were always there to cheer me up and to "spur me on" during the rough times. Thank you. Finally, I am grateful to the families of the Pitt Mother and Child Project for their participation and dedication to the study of boys' development.

1.0 INTRODUCTION

Somatization (SOM), the tendency to experience medically unexplained physical symptoms, affects 10-30% of children and adolescents in the United States (Campo & Fritsch, 1994). In addition to their high prevalence, recurrent somatic complaints are associated with excessive health care utilization as well as high levels of functional impairment (e.g., frequent school absenteeism) and internalizing and externalizing problems (Campo & Garber, 1998). Despite these costs and consequences to children, their families, and society, little is known about the etiology, course, and prognosis of chronic somatization.

While a number of theories have been posed to address the development of SOM in children, most focus on one central and causal mechanism. Moreover, empirical studies have been primarily correlational and cross-sectional in nature, limiting the inferences that can be drawn pertaining to the stability and progression of SOM over time and to potential risk factors (e.g., stressful life events) of SOM. Hence, research to date has been insufficient in explicating a developmental model of somatization. In the past 20 years, a developmental psychopathology perspective (DPP) has offered a new approach to the study of abnormal child development. Unlike the developmentally static, Western medical model of disease, the crux of the DPP is to investigate multifactorial causation and the dynamic processes that foster the development of psychopathology throughout childhood and adolescence. Also central to the DPP is the identification of risk factors and interactive processes that differentiate individuals who progress

to various adaptive and maladaptive outcomes depending upon risk status (Cicchetti & Sroufe, 2000; Cummings, Davies, & Campbell, 2000). Guided by the DPP and the extant literature, the present research will investigate how risk factors from multiple domains (i.e., child, parent-child relationship, family, sociodemographic, and school) distinguish developmental pathways of SOM and related maladaptive behavior and functional impairment in early adolescence.

Specifically, the goals of this study were to extend our understanding of SOM by (1) using latent growth modeling (LGM) procedures to plot developmental trajectories of SOM, (2) identifying risk factors associated with divergent developmental trajectories, and (3) examining the incidence of co-occurring child maladjustment and functional impairment, namely internalizing and externalizing behaviors and school absenteeism, respectively. These goals will be undertaken with a sample of 310 ethnically diverse, low-income boys followed longitudinally from ages 2 to 12, using multiple methods and informants. The findings of the present research should improve the understanding of the antecedents and course of SOM and clarify whether histories and correlates of SOM can discriminate SOM from other internalizing and externalizing behaviors. Such data are vital in identifying model-based intervention strategies for children with SOM across different periods of childhood.

1.1 LITERATURE REVIEW

This paper will first discuss the definition, conceptualization, and prevalence of childhood SOM. Second, problems facing the child somatization literature will be explicated. Third, the developmental psychopathology perspective (DPP) will be introduced and offered as a method of addressing the aforementioned problems. Fourth, the DPP will be applied to

childhood SOM. Finally, research supporting the developmental model of SOM will be reviewed.

1.2 DEFINITION AND CONCEPTUALIZATION OF CHILD SOMATIZATION

Somatization has been defined as the tendency to experience and communicate physical complaints that have an unknown or unfound pathological origin, to ascribe these symptoms to a physical disease, and to seek medical attention for them (Lipowski, 1988). Despite this straightforward definition, somatization can be conceptualized in a number of ways. For instance, it can be viewed as a continuum of severity or as a categorical construct. Typically, somatization has been used as a descriptive term, where physical symptoms are experienced by the individual, but medical evaluation reveals no discernable physical pathology (Campo & Fritsch, 1994). Often in the childhood literature, somatization is defined by the presence of frequent and recurrent physical complaints, such as headaches, abdominal pain or discomfort, musculoskeletal pain, nausea and vomiting, chest pain, fatigue, dizziness, and pseudoneurological symptoms, such as pseudoseizures and unexplained falls. These somatic complaints are investigated either individually or in combination. Both ways are considered workable definitions of somatization in children and adolescents.

The dimensional definition of somatization described above is distinctly different from the categorical definition of somatization disorder outlined by the *Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition* (DSM-IV, American Psychiatric Association, 1994). According to the *DSM-IV*, the diagnosis of somatization disorder is given when multiple physical symptoms are present over a period of several years. Specifically, an individual must

experience eight symptoms from four domains, namely *pain* (4), *gastrointestinal* (2), *sexual* (1), and *pseudoneurological* (1). The symptoms cannot be explained by a medical condition, or if physical disease is present, the symptoms and associated impairment are deemed excessive. The criteria for somatization, particularly the sexual and pseudoneurological symptoms, are not applicable or inappropriate for prepubescent children. As a result, somatization disorder is a rare entity in children and adolescents, and empirical work on the disorder in children and adolescents is all but nonexistent (Campo & Fritsch, 1994). For these reasons, the present study focused on a descriptive definition, or syndrome, of SOM as opposed to the categorical diagnosis of somatization disorder.

1.3 PREVALENCE OF SOMATIC COMPLAINTS

As stated above, recurrent somatic complaints affect 10-30% of children and adolescents in the United States (Campo & Fritsch, 1994). Research shows that the prevalence of somatization has risen dramatically from 17% in 1984 to 24.2% in 1996 for children and adolescents (Berntsson & Koehler, 2001). Alfven (1993) found that 47% of 1,333 Swedish schoolchildren reported one or more somatic symptoms. The most commonly reported symptom is headache with a prevalence of 10-30%, followed by recurrent abdominal pain (RAP) with a prevalence of 10-25%. Muscle and limb pain has been reported to affect 21% of children, and fatigue affects 15%. Other common complaints are dizziness (15%) and chest pain (7-15%) (Campo & Fritsch, 1994). Somatoform disorders, including somatization disorder, are rare in childhood and adolescence. For example, in a community sample of 637 adolescents, only 3 (0.5%) were diagnosed with a *DSM-IV* somatoform disorder (Dhossche, Ferdinand, van der

Ende, & Verhulst, 2001). Polysymptomatic presentation, however, is common, especially in adolescence (Alfven, 1993; Campo & Fritsch, 1994). Garber, Walker, and Zeman (1991) conducted a study on 540 school age children and adolescents in grades 3 –12. They found that in a previous 2-week period, 13% of the children experienced one symptom; 15% reported four or more symptoms, and 1% reported 13 or more symptoms. Findings from the Ontario Child Health Study of 12- to 16-year-olds also support polysymptomatic presentation. The researchers found that 4.5% of boys and 10.7% of girls were identified with multiple somatic complaints (Offord et al., 1987).

1.4 PROBLEMS WITH THE CHILDHOOD SOMATIZATION LITERATURE

Due to the physical nature of their symptoms, somatizing children are frequently seen in pediatric settings. In fact, it has been reported that 2-4% of all pediatric visits are due to unexplained somatic complaints (Campo & Reich, 1999). This high level of medical attention is not only costly, but often it leads to unnecessary and dangerous medical procedures such as surgery (Campo & Fritsch, 1994; Campo & Reich, 1999). Moreover, researchers and practitioners note that the uncertainty of a diagnosis, inadequate medical advice, and excessive reassurance appear to encourage somatizing in children (Campo & Fritsch, 1994). Conceptualizing, and hence treating, somatizing children within a medical framework is ill-advised for two reasons. First, recurrent somatic complaints have been hypothesized as one of the more common ways for emotional and behavioral difficulties to present in a primary care setting (Campo & Reich, 1999). There is an accumulation of evidence from multiple disciplines and research populations that childhood somatization frequently co-occurs with other psychiatric

symptoms, and the frequency of somatic symptoms tends to increase with the severity of anxiety and depression symptoms (Bernstein, Massie, Thuras, Perwien, & et al., 1997; Dhossche et al., 2001; Garber, Zeman, & Walker, 1990; Last, 1991; Livingston, Taylor, & Crawford, 1988; McCauley, Carlson, & Calderon, 1991; Walker & Greene, 1989). However, children's frequent physical symptoms may encourage physicians to investigate areas of their own medical expertise, which then steers them from considering psychological factors as the source of the problem (Campo & Garber, 1998). Second, researchers and pediatricians alike note that headaches and RAP are ascribed to psychosocial and environmental variables over physiological variables, such as intracranial pathological conditions or a dietary disorder (Chu & Shinnar, 1992; Scharff, 1997). The British pediatrician William Henry Day stated some 125 years ago that "headaches in the young are for the most part due to bad arrangements in the lives of children" (Rothner, 1993). This viewpoint is still held today by physicians. For instance, Chu and Shinnar (1992) note that even migraines occurring in young children rarely have a serious underlying intracranial pathological condition. Rothner (1993) concurs that most headaches in children are not associated with organic structural disease, but nevertheless, parents often seek medical help for fear of a brain tumor.

It follows that two problems facing this field of study are that (1) childhood somatization (SOM) may be an early marker for later psychopathology and (2) any number of child and/or environmental risk factors may contribute to the development of SOM. Inherent in these statements and as illustrated above, many children with recurrent somatic complaints may not be receiving appropriate treatment for their problems. The treatment of childhood SOM has been limited, at least in part, by the way in which childhood SOM has been conceptualized and studied. For one, previous theories of SOM have been adevelopmental; therefore, few studies

investigating these theories are truly longitudinal. Some may include a one- to two-year follow-up, but this is not adequate to illustrate the unfolding of SOM over time, including the timing of co-occurring maladjustment (e.g., internalizing and externalizing problems). Second, most theories of SOM emphasize only one domain of risk as contributing to the development of childhood SOM. For example, family systems approaches focus on the family environment, regarding children's frequent physical complaints as a way for family members to avoid conflict (Terre & Ghiselli, 1997). Focusing on one aspect of the child or environment does not permit the interplay among factors or represent the breadth of the child's experience. By applying a developmental psychopathology perspective to the extant literature, issues concerning the antecedents, course, and outcome of SOM can be addressed simultaneously.

1.5 THE DEVELOPMENTAL PSYCHOPATHOLOGY PERSPECTIVE (DPP)

A model based on the developmental psychopathology perspective (DPP) is unique in that it describes the transactional processes between a child and his environment over time. As described by Cicchetti and Sroufe (2000), the central focus of DPP is to "discover processes of development with the goal of comprehending the emergence, progressive unfolding, and transformation of patterns of adaptive and maladaptive behaviors over time" (pp. 258-9). The DPP stands apart from traditional theories, because it goes beyond naming correlates or finding predictors of a maladaptive behavior. The DPP inspires a complex model that incorporates multiple contexts, investigates qualitative change in processes over time, and relies on the interdisciplinary collaboration of social, psychological, and biological scientists to describe the emergence and course of disturbed and well-adjusted behaviors (Cicchetti & Sroufe, 2000;

Cummings et al., 2000). The fact that childhood SOM has been a topic of interest and concern of multiple disciplines implies that the DPP is an appropriate, indeed necessary, method to use in the study of this problematic and maladaptive behavior.

1.5.1 The active organism.

At the crux of the DPP is the conceptualization of the child as an active organism, a proactive processor of experience instead of a passive bystander/recipient of events. Children are described as having their own needs, desires, and ways of interpreting the environment. Viewed as active contributors to their environment, children are capable of evoking various reactions from people, thereby taking a hand in shaping their environment. This idea is known as "niche picking" (Cummings et al., 2000). Although niche picking can promote adaptive behaviors, it can also propel maladaptive development. For instance, a child who has shown an early propensity for antisocial behavior is more likely to seek out a deviant peer group at school and in the neighborhood, which would consequently increase his exposure to risky situations and illicit acts. As a result, this child will develop a reputation, such that adults may come to expect little from him and his nondeviant peer group may reject him, which could drive him to further interactions with deviant peers (Patterson, DeBaryshe, & Ramsey, 1989).

1.5.2 Contextualism.

DPP promotes an integrative model. It not only observes the contribution of the active individual, but it also examines the dynamic processes and complex interplay between the individual and multiple contextual influences in the child's ever-changing environment (Cummings et al., 2000). Developmental psychopathologists describe this dynamic

environmental exchange in terms of "contextualism." Each level of the child's ecological context is considered and synthesized as a part of the child's experience. For instance, a proximal context is the parenting a child receives; whereas, a more distal sphere of influence is the child's neighborhood.

1.5.3 Multifinality and equifinality.

The DPP transcends the notion that one causal factor has a singular outcome and anticipates that organism-environmental transactions lead to a wide array of cause and effect relationships depending upon risk and protective factors. *Multifinality* is the term used to describe the phenomenon where one cause can result in many outcomes (Cummings et al., 2000). For example, being raised in an adverse family environment in early childhood is a risk factor for both internalizing and externalizing behaviors (Shaw, Vondra, Hommerding, Keenan, & Dunn, 1994; Shaw, Winslow, Owens, & Hood, 1998). *Equifinality* is the idea that multiple causes result in a single outcome (Cummings et al., 2000). For example, genetics, delivery complications, or head injury are all separate causes of mental retardation.

1.5.4 The role of risk and protective factors.

Since finding a definite and exclusive cause in child psychopathology literature is rare, the purpose of the DPP is to define a number of risk and protective factors that contribute to a child's development. Establishing a process or condition as a risk factor is complex by nature. For instance, sometimes one condition may serve as a risk or a protective factor for different outcomes: being male is a risk factor for conduct disorder but is a protective factor for anorexia nervosa (Cicchetti & Sroufe, 2000). Moreover, a condition normally conceived as an outcome

may also serve as both a protective factor and a risk factor for other maladjusted behavior. For example, anxiety is a risk factor for depression in girls but may be a protective factor for conduct disorder in boys.

1.5.5 Developmental pathways.

A prototypical question posed by developmental psychopathologists is whether different risk factors and pathways can distinguish cases of disordered behavior. The DPP advocates investigating differential patterns of predictors and pathways of a disorder, so as to increase the power of predicting adult outcome. The timing of symptom manifestation and the identification of combinations of risk and protective factors have helped guide developmental psychopathologists in delineating trajectories of childhood disorders (Cicchetti & Sroufe, 2000), such as early-onset antisocial behavior (Moffitt, 1993). While there has been some outstanding research contributing to our improved understanding of childhood disorders, we are still in the early stages of developmental analysis for these disorders, with more questions being raised than answered. It is time to start raising these same questions with respect to childhood SOM.

1.6 A DEVELOPMENTAL PSYCHOPATHOLOGY MODEL OF SOMATIZATION

The child literature does not provide extensive evidence for the contribution of one particular factor to somatization. However, when taken together, there is enough evidence to advance a model when viewed from the DPP (see Figure 1). Below is a dynamic, developmental model that highlights the interplay between previous theories of SOM (noted in parentheses) and DPP constructs (*italicized in parentheses*). Empirical support for the model follows.

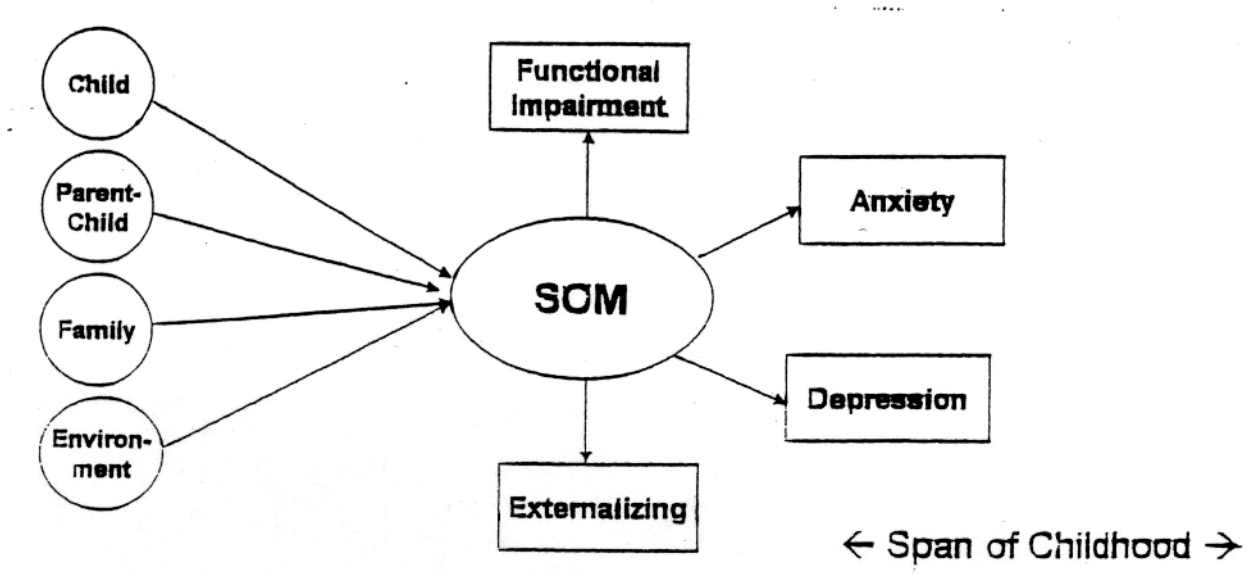

Figure 1: A DPP model of childhood somatization

The DPP starts with a sensitive and/or emotionally reactive child, who perceives more threats and dangers, be they real or imagined, in the environment. A sensitive or reactive child may be more likely to use somatic complaints in signaling caregivers to help cope with distress (attachment theory). The quality of the parent-child relationship may shape the way a sensitive or emotionally reactive child copes with stress, which in turn, may affect the course of somatization. For example, having a harmonious and open parent-child relationship may minimize impairment associated with somatic complaints, insofar as the responsive parent may be helpful in alleviating the child's fears or provide alternative and more adaptive coping strategies (*risk/ protective factor*), which could then lower the child's reactivity and somatic distress. On the other hand, a sensitive and/or anxious child may feel rejected by the parent or may be more prone to get upset and emotionally overaroused in a parent-child relationship

ridden by hostility and conflict. In such an acrimonious climate, the child may begin to internalize feelings and express somatic problems more frequently, as it is the only outlet for an anxious child's feelings. A child's internalizing may perpetuate self-isolation and the experience of somatic distress, further exacerbating the intensity of the pain (*active organism-environment dynamic*). These behaviors might be maintained by family conflict (family systems theory), and this effect might be stronger in socially disadvantaged homes with few financial and social resources, which could potentially increase stress and conflict in the home (*contextualism*).

If a child has a high level of anxiety (*a potential risk factor*), the child may eventually feel helpless and hopeless, which increases the risk for a later mood disorder and continued somatization (*pathway/ progression*). With the onset of puberty, girls may be at greater risk for comorbid internalizing/ somatization than boys, perhaps due to hormones or socialization practices. Somatizing boys may follow a much different trajectory than girls, where early concentration difficulties and behavior problems prevent boys from forming synchronous parent-child relationships and from learning appropriate coping styles. Social and academic failure and low self-esteem may lead to anger and acting out. If taken to an extreme, these boys may evidence an externalizing disorder, and to a lesser extent, somatic complaints in later childhood and adolescence (Aromaa, Rautava, Helenius, & Sillanpaeae, 1998; Egger, Angold, & Costello, 1998; Egger, Costello, Erkanli, & Angold, 1999).

In the next section, the literature pertaining to the course and progression of SOM from childhood to adulthood will be reviewed in order to inform individual trajectories of SOM. TRAJ, a latent growth modeling statistical technique (Nagin, 1999) will be introduced as a means by which to delineate multiple developmental courses of SOM. Finally, literature pertaining to the risk factors, co-occurring problem behaviors, and functional impairment associated with childhood SOM will be reviewed within the DPP.

1.7 COURSE AND PATHWAYS OF SOMATIZATION

Somatization in adults is associated with high health care utilization, high costs, and lower productivity. Adults with somatization often report that they have suffered similar symptoms or a physical illness in childhood, and polysymptomatic adults report suffering symptoms before age 20 (Campo & Garber, 1998; Craig, Boardman, Mills, Daly-Jones, & Drake, 1993). These reports suggest that adult somatization has its roots in childhood and may be continuous over time (Campo & Garber, 1998; Fritz, Fritsch, & Hagino, 1997). Work in the area of RAP has shown that between one-third and one-half of pediatric patients with RAP continue to show some degree of abdominal pain or discomfort in adulthood and additional somatic complaints, such as headaches (Apley, 1975; Apley & Hale, 1973; Christensen & Mortensen, 1975). In a more recent study, Walker, Garber, Van Slyke, and Greene (1995) found that children with RAP demonstrated higher levels of abdominal discomfort, other somatic symptoms, and functional disability (such as school or work absences) than healthy controls at a 5-6 year follow-up. Similarly, in an epidemiological study of adolescents, Dhossche et al. (2001) found that adolescents with a specific somatic complaint tended to report the same symptom along with other symptoms at a six-year follow-up.

Studies on the course and prognosis of childhood somatization are equivocal due to inconsistent definitions of outcome (Zwaigenbaum, Szatmari, Boyle, & Offord, 1999). That is, the outcome in some studies has been defined as the presence of somatic symptoms, whereas others have employed such criterion as functional impairment, psychiatric diagnosis, or medical help-seeking, which are other indicators of severity (Campo & Fritsch, 1994; Walker, Smith, Garber, & Van Slyke, 1997). For example, in their study of a British birth cohort, Hotopf, Carr, Mayou, Wadsworth, and Wessely (1998) found that children experiencing abdominal pain at

ages 7, 11, and 15 were more likely to suffer psychiatric disorders in adulthood than children with less persistent or no abdominal pain at those ages, but they did not necessarily continue to experience physical complaints in adulthood.

Some developmental trends for the type and frequency of somatic complaints have emerged from the literature. First, the pattern of symptom presentation appears to change as a function of the child's developmental status (Achenbach, Conners, Quay, Verhulst, & Howell, 1989; Offord et al., 1987). For example, RAP is the most common complaint around 9 years of age, and headache is the most frequent complaint around age 12. Before the age of 6, pseudoseizures are rare; however, they become most apparent during adolescence (Campo & Fritsch, 1994). Second, the incidence of somatic complaints tends to be low in early childhood but increases with age, especially for polysymptomatic presentation (Campo, Jansen-McWilliams, Comer, & Kelleher, 1999; Egger et al., 1998). Two studies have demonstrated that in early childhood 8-9% of preschoolers have recurrent stomachaches, and 2-3% have recurrent headaches (Domenech-Llaberia et al., 2004; Zuckerman, Stevenson, & Bailey, 1987). Berntsson and Koehler's (2001) epidemiological study across the five Nordic countries illustrates how the prevalence of somatic complaints increases with age. The authors report that 17.6% of 2- to 6-year-olds, 25% of 7- to 12-year-olds, and 30.6% of 13- to 17-year-olds reported at least one somatic complaint occurring every or every other week. In the study of a British birth cohort, Fearon and Hotopf (2001) found the prevalence of headaches to be 8.2% (n = 811) and 15.4% (n = 1511) for 7- and 11-year-olds, respectively. Although these epidemiological studies demonstrate that the occurrence of SOM increases with age, the results should be interpreted with caution due to the measurement issues. For instance, two of the six "somatization" items used in the Berntsson and Koehler study were more indicative of depression (i.e., sleeplessness

and loss of appetite) than of physical complaints, and Fearon and Hotopf asked the child's parent only one question concerning the presence of headaches.

Regarding gender, epidemiological research has shown that before puberty there is no difference in the prevalence of somatization for boys and girls (Berntsson & Koehler, 2001; Campo & Reich, 1999). In adolescence, though, girls tend to report over twice as many somatic complaints as boys (Achenbach et al., 1989; Offord et al., 1987). In a longitudinal study of 90 Finnish school children, no sex differences emerged in the number of somatic complaints at age 11 or 13; however, girls reported more somatic symptoms than boys at ages 15 and 18 (Rauste-von Wright & von Wright, 1981). Both boys and girls demonstrated the same general pattern of somatic complaints: increasing from age 11 to 13, then decreasing from age 13 to 15, and stabilizing from ages 15 to 18. Across all ages, girls were more consistent in their reporting (rs = .40 to .65, all $p < .01$) than boys (rs = .19 to .42, $p < .01$ for ages 13 vs. 15 and 15 vs. 18). In an epidemiological study, 637 children and adolescents ages 11-18 completed the Somatic Complaints subscale of the *Youth Self-Report* (YRS, Achenbach, 1991b) at baseline and 8 years later (Dhossche et al., 2001). Again sex differences emerged, such that adolescent girls with multiple somatic complaints had a nine times higher risk of having multiple somatic symptoms in young adulthood than girls with fewer symptoms; adolescent boys with multiple somatic symptoms were six times as likely to have multiple symptoms in young adulthood than adolescent boys with fewer symptoms.

In summary, research shows that somatic symptoms increase over time and peak at age 13 and that there is modest support for the stability of SOM, particularly for girls (Aro, Paronen, & Aro, 1987; Dhossche et al., 2001; Rauste-von Wright & von Wright, 1981). However, the continuity of SOM is not universal. For instance, in a pediatric sample of RAP children ages 6-18, the correlation between the number of somatic symptoms at baseline and one-year follow-up

was .29 ($p < .01$), which, to the authors, indicated a rather unstable course of SOM (Walker, Garber, & Greene, 1994). This finding may be due to the potentially more severe cases in this pediatric sample, the inclusion of a wide age range, and analyzing boys and girls together.

Developmental trajectories of SOM have not been delineated. However, preliminary work on broadly defined child internalizing behaviors provides some basis for hypothesizing individual trajectories of SOM. For example, Gilliom and Shaw (2004) examined trajectories of internalizing problems in disadvantaged boys followed from ages 2 to 11, using maternal report on six internalizing *CBCL* items (i.e., "Too fearful or anxious," "Self-conscious or easily embarrassed," "Shy or timid," "Unhappy, sad, or depressed," "Withdrawn," and "Worries"). Three trajectories of internalizing problems emerged: a persistent low group (comprising 77% of the sample), an increasing group (20%), and a persistent high group (3%). For both boys and girls ages 6 to 12, Cote and colleagues found three relatively stable trajectories for fearful, anxious behaviors, namely low (16%), moderate (75%), and high (9%) groups (Cote, Tremblay, Nagin, Zoccolillo, & Vitaro, 2002). Taken together, these findings provide some basis for hypothesizing three pathways for SOM in school-age boys.

No study to date has been able to chart the progression of the symptomatic prevalence over time, especially in children younger than age 11. Accordingly, the proposed study will use a broad syndrome of SOM comprised of multiple somatic symptoms to capture the range of symptoms presented in boys from ages 6 to 12. This measurement of SOM also will allow for investigation of the prevalence of individual symptoms across time, just before the age SOM has been shown to peak.

To facilitate an investigation of childhood trajectories of SOM, Nagin's (1999) semiparametric LGM approach was used. This procedure, using a software program referred to

as TRAJ within SAS, provides objective criteria for deciding how many types of trajectories exist within a population and for estimating the proportion of individuals who follow each trajectory. The TRAJ technique was expected to identify different patterns of SOM, including those who are persistently low or high on symptoms and those with increasing scores over time. Based on the two existing studies in this area (Cote et al., 2002; Gilliom & Shaw, 2004), and epidemiological data on gender and age effects on SOM (e.g., Rauste-von Wright & von-Wright, 1981), three groups are expected to emerge: persistent high, increasing, and persistent low groups.

Applying a developmental lens and using a person-based, multilevel modeling procedure was proposed to expand the way SOM has been conceptualized and studied. Ultimately, this approach also has the potential to inform the development of model-driven intervention approaches. This study aims to take an initial step in this process by addressing the following three questions: "What are the developmental patterns of SOM for boys across childhood?," "What risk factors are important in the development and maintenance of SOM?," and "What is the relation between persistent patterns of SOM and other related externalizing and internalizing problems, such as anxiety and depression, in school-age boys?"

1.8 RISK FACTORS

Rather than postulating a definite and exclusive cause of childhood SOM, which tends to be rare in the area of childhood psychopathology, the DPP presumes that there are a number of risk factors from many domains that contribute to the onset and maintenance of SOM. This paper now turns to the review of the five risk areas pertinent to this study; namely, child, parent-

child, family, sociodemographic, and school, in relation to the development of SOM in childhood (see Figure 2). By applying a developmental psychopathology perspective to SOM, the individual significance of these factors and how they might additively and interactively contribute to the developmental trajectories of SOM will be highlighted. Finally, I will review the literature pertaining to co-occurring adjustment problems (e.g., depression) and functional impairment (e.g., school absenteeism) that have been associated with recurrent somatic complaints and that may differentiate individual SOM trajectories.

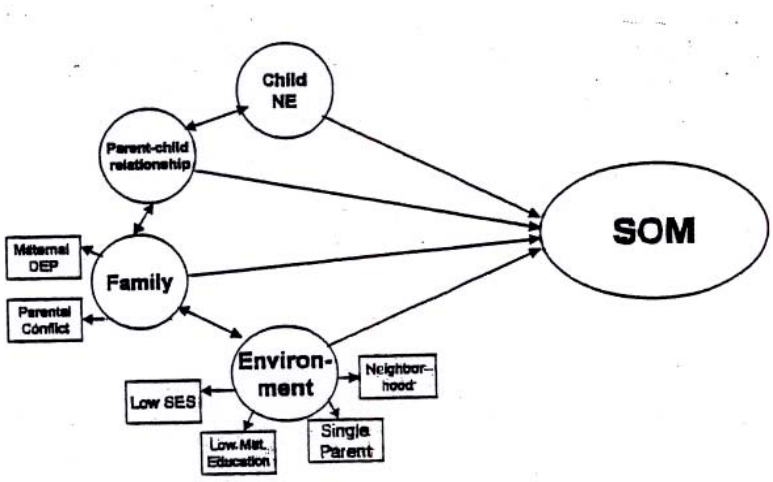

Figure 2: Risk factors of childhood somatization.

1.8.1 Child Risk.

In clinical settings, somatizing children tend to be described as conscientious or obsessive, sensitive, insecure, and anxious (Garralda, 1996; Kowal & Pritchard, 1990). Thus,

children with attributes such as negative emotionality and/or difficult temperament are hypothesized to be at-risk for developing SOM (Dorn et al., 2003). In fact, RAP children with high levels of trait negative affectivity (i.e., a component of difficult temperament) have been shown to demonstrate more SOM in the face of negative daily stressors than RAP children with lower negative affect (Walker, Garber, Smith, Van Slyke, & Claar, 2001). Empirical evidence suggests that somatizing children and adolescents have fewer adaptive coping strategies and, to some extent, a heightened emotional response to stress than organically ill and community samples of children (Aromaa, Sillanpaa, Rautava, & Helenius, 2000; Bandell-Hoekstra et al., 2002; Rauste-von Wright & von Wright, 1981; Rocha, Prkachin, Beaumont, Hardy, & Zumbo, 2003; Ruchkin, Eisemann, & Haeggloef, 2000; Thomsen et al., 2002; Walker et al., 1997). Aromaa and colleagues found that 6-year-olds with headaches demonstrated behaviors indicating a greater sensitivity to pain (e.g., showed avoidance reactions during playtime for fear of getting hurt), complained of more somatic symptoms in stressful situations, and reacted more negatively during stressful times than children without headaches. These findings also may be partially explained by poor emotion regulation skills, such that children who display negative emotionality may get upset more easily in the face of stressors, after which it is harder for them to return to baseline levels of arousal (Calkins, 1994). For these reasons, one of the hypotheses of the current study is that children who display high levels of negative emotionality in early childhood may be at a greater risk for SOM. The present study investigated a child's negative emotionality and difficult temperament and their interaction with early social and environmental risk in relation to childhood SOM in order to discriminate children with SOM and co-occurring internalizing and/or externalizing problems or functional impairment.

1.8.2 Parent-Child Relationship Risk.

Somatizing children have been described as having poor parent-child relationships (Aro, Haenninen, & Paronen, 1989; Aro et al., 1987; Rauste-von Wright & von Wright, 1981), and their families have been described as less supportive, cohesive, and adaptive than other families (Walker & Greene, 1987; Walker, McLaughlin, & Greene, 1988). Moreover, research on childhood anxiety and depression has shown that parents who are overcontrolling (hostile/rejecting), intrusive, or unresponsive are likely to exacerbate children's worries, fears, and sadness (Zahn-Waxler, Klimes-Dougan, & Slattery, 2000). One way children's fears and anxieties may be intensified are through **acrimonious parent-child relationships**. It is plausible that sensitive and/or anxious children are more prone to get upset and emotionally overaroused in parent-child relationships ridden by conflict. In such an acrimonious climate, children may begin to internalize their feelings and express somatic problems more frequently. Therefore, it was hypothesized that boys in acrimonious parent-child relationships would be more likely to be in the increasing or high SOM trajectories, especially if the boys also demonstrated high negative emotionality/difficult temperament.

1.8.3 Family Risk.

Two proximal within-family variables were evaluated as risk factors for SOM, namely **maternal depressive symptoms** and **interparental conflict,** both of which have been previously related to boys' internalizing problems (Shaw & Emery, 1987). Many studies show that anxiety, depression, and antisocial behavior are more common in families of children and adolescents with somatic complaints than in healthy families or families with an organic illness (Campo &

Fritsch, 1994; Garralda, 1996; Gulhati & Minty, 1998). This could be due to social learning or familial processes, such as parental modeling of internalizing symptoms or a lack of responsiveness to a child's bids for attention, found among depressed mothers. In addition, research has shown that high levels of perceived conflict and stress are reported in families of somatic patients (Aro, 1987, 1989; Zuckerman et al., 1987). Frequently occurring stressors in the home, such as marital conflict and poor family functioning, have been shown to increase somatizing in community and clinical samples of children (Boey & Goh, 2001; Terre & Ghiselli, 1997; Walker, Garber, & Greene, 1993; Walker et al., 1994; Walker et al., 2001; Zuckerman et al., 1987). Similar to acrimonious parent-child relationships, having a depressed parent and/or frequent conflictual exchanges between parents may inundate children with stress, with which they are unable to cope. Therefore, it was hypothesized that boys in families characterized by high levels of family risk would more likely be in the increasing or high SOM trajectories, especially if the boys also demonstrated high negative emotionality/ difficult temperament.

1.8.4 Sociodemographic Adversity.

Findings have been inconsistent pertaining to markers of **sociodemographic adversity** (e.g., single parent, low socioeconomic status) and SOM. Studies using cross-sectional data with large samples of older children and adolescents have found support for an association between high SOM and measures of low socioeconomic status (SES), including low income, low parental education, single parent status, and disadvantaged school districts (Alfven, 1993; Berntsson & Koehler, 2001; Fearon & Hotopf, 2001). However, two prospective studies with younger children did not find that measures of SES, such as parental education and social class status, were related to SOM (Aromaa et al., 1998; Zuckerman et al., 1987). This inconsistency may be

due to unidentified moderating effects or the exclusion of multiple co-occurring sociodemographic stressors. That is, sociodemographic adversity appears to affect SOM in children older than 7 years, and perhaps, when occurring with other social and environmental risk factors. This assertion is supported by a cumulative risk hypothesis that appears in the child psychopathology literature. The hypothesis states that the number of environmental stressors rather than the particular combination of stressors is associated with child behavior problems both concurrently and longitudinally (Rutter, Cox, Tupling, Berger, & Yule, 1975; Rutter, Yule et al., 1975; Sameroff, Seifer, Zax, & Barocas, 1987; Shaw et al., 1994; Shaw et al., 1998). To improve upon previous studies of childhood SOM, this study used a cumulative index of four sociodemographic risk factors, namely low SES, low maternal education, single parent status, and neighborhood dangerousness, as a measure of sociodemographic adversity.

1.8.5 School Risk.

Walker and her colleagues (Walker, Claar, & Garber, 2002; 1994; 2001) provide some of the only research investigating interactive effects between stressors and school functioning on childhood and adolescent SOM. This group has found that the relationship between stressors and SOM is amplified in the context of **poor social and academic skills**. Taken together, Walker's studies describe children who are incompetent in at least one area of school functioning as being at-risk for greater somatization in the face of stressful situations. These findings are consistent with a "secondary gain" hypothesis, such that children who may fear failure in social or academic realms are less apt to cope with negative life stressors and consequently express somatic symptoms. The expression of these symptoms may benefit them by allowing them to avoid the feared situation or because they receive attention, thereby reinforcing the expression,

and likely the experience, of the somatic distress. The main limitation to Walker's studies is the sole reliance on pediatric populations already experiencing RAP, a potentially more severe group of somatizers. The current study aims to extend and potentially replicate Walker's findings in a community sample of school-age children with the purpose of broadening the generalizability of these results. It was hypothesized that boys who were less socially skilled and who demonstrated lower levels of academic competence, especially in the face of proximal environmental stressors, would be more likely to show higher levels of SOM across time and demonstrate more maladjustment and functional impairment in early adolescence.

In summary, data suggest that conflict and stress in parent-child relationship and family domains are associated with increased SOM in children, especially when children are characterized by high negative emotionality/difficult temperament or deficits in social or academic functioning. Further, multiple sociodemographic risks may compound these effects, particularly in older children. To clarify the complex relationship across risk domains, the proposed research will study the additive and interactive effects of child, parent-child relationship, family, sociodemographic, and school stressors on the development of SOM.

1.9 CO-OCCURRING MALADJUSTMENT

The prognosis can be worse for child-onset problems when they are persistent and/or comorbid, as is the case for antisocial behavior with attention-deficit/hyperactivity disorder (Moffitt, 1990, 1993). Due to the nascence of the literature on this subject, there has been no evidence to demonstrate a similar pattern with child-onset somatization. However, it is well-cited across disciplines that children suffering from recurrent somatic complaints often display

elevated levels of psychiatric symptoms, and the frequency of somatic symptoms tend to increase with the severity of anxiety and depression symptoms (Bernstein et al., 1997, Dhossche et al., 2001; Garber et al., 1990; Last, 1991; Livingston et al., 1988; McCauley et al., 1991; Walker & Greene, 1989). This is particularly salient given that symptoms such as generalized anxiety, thoughts of death, and those typical of externalizing behaviors, including hyperactivity, oppositionality, and conduct problems, have been found to co-occur with recurrent somatic complaints in children between the ages of 3 and 6 (Aromaa et al., 2000; Domenech-Llaberia et al., 2004; Stevenson, Simpson, & Bailey, 1988; Zuckerman et al., 1987). For example, a significantly higher occurrence of conduct problems has been found in boys with headaches and in somatizing children age 6 and younger compared to healthy children (Egger et al., 1998; Zuckerman et al., 1987).

1.9.1 Internalizing Problems

Significant correlations between children's somatic complaints and self-reported anxiety and depression (with somatic symptoms removed from questionnaires) have been found to be .43 and .37, respectively (Garber et al., 1991). In a longitudinal study of adolescents, somatic complaints were significantly correlated with self-reports of anxiety in boys (*r*s = .30-.42) and girls (*r*s = .31-.36) at ages 13, 15, and 18 (Rauste-von Wright & von Wright, 1981). Longitudinal data also demonstrate some support for high somatization in children predicting a later psychiatric disorder (Egger et al., 1999; Zwaigenbaum et al., 1999). For example, Zwaigenbaum and colleagues found that high somatization at baseline was associated with major depressive disorder (MDD) four years later, independent of gender, baseline emotional disorder, and sociodemographic factors. In addition, the authors found that having an emotional disorder

at baseline moderated the relationship between somatization and later MDD. Specifically, the authors found that the adolescents at the greatest risk for MDD were those with high somatizing but no evidence of an emotional disorder at baseline compared to peers with low somatization and no emotional disorder. In fact, these high somatizing teens were at the same risk as teens with an emotional diagnosis at baseline, and the authors note that there were no differences between girls and boys. The authors interpret their findings as evidence indicating somatization as an early expression of depressive feelings. However, in their longitudinal study, Dhossche and colleagues (2001) did not find that high somatizers were at a greater risk for a psychiatric disorder at follow-up 6 to 8 years later, despite using similar measurements of somatizing and psychiatric disorders to those used in the Zwaigenbaum study. However, Dhossche and colleagues did not investigate the moderating effects of emotional disorders in their study. Hence, at this point the data are mixed concerning whether or not a chronic course of SOM is a risk factor for later psychopathology, specifically clinical depression.

1.9.2 Externalizing Problems

Besides having early psychological problems, another potential moderator of the association between somatization and psychological problems may be gender. In Egger's (1998, 1999) longitudinal studies, data suggest that the type of somatic symptom may predict a particular psychiatric diagnosis (as defined by DSM-III-R criteria, American Psychological Association, 1987), depending on the child's sex. For example, musculoskeletal pains predicted depression in boys and both depression and anxiety disorders in girls. The combination of headaches and RAP also predicted anxiety disorders in girls, whereas RAP predicted ODD and ADHD in boys (Egger et al., 1999). Moreover, 30.6% girls with one or more psychiatric

diagnosis reported concurrent chronic headaches compared to 9.3% of girls without a psychiatric diagnosis, but this difference was not found in boys.

1.10 FUNCTIONAL IMPAIRMENT

In addition to psychological problems, somatizing children have problems in social and school realms. Research shows that children with headache and other somatic complaints have more problems in daycare, fewer hobbies, and report a greater impact of their symptoms on daily life and leisure activities (Aromaa et al., 2000; Bandell-Hoekstra et al., 2002). Somatic complaints are often associated with frequent and prolonged daycare and school absences in pediatric, psychiatric, and community samples (Bernstein et al., 1997, Domenech-Llaberia et al., 2004; Rothner, 1993; Walker et al., 1995). The relationship among school attendance, somatic complaints, and psychiatric problems (i.e., anxiety and depressive symptoms) is important to delineate because missing school may have adverse consequences for youth, including strain on or loss of peer relationships, social isolation, and academic difficulties (Bernstein et al., 1997, Vannatta, Gartstein, Short, & Noll, 1998). Moreover, children with frequent school absenteeism (i.e., school refusers) are often referred to a social worker rather than to a psychiatrist, perhaps due to perceived delinquency (Stickney & Miltenberger, 1998). Empirical work from the pediatric and school refusal literature suggests that many children with somatic complaints are not receiving the appropriate treatment for their problems due to the complexity of their presentation.[1]

[1] A number of papers address school refusal and related topics (see Bernstein et al., 1997; Honjo et al., 2001; King & Bernstein, 2001; Last, 1991).

In conclusion, although many studies have investigated the co-occurrence of SOM and internalizing and/or externalizing behaviors, few explicate possible mechanisms for this association. At this point, it is not possible to determine whether the comorbidity of SOM, anxiety, and depression represents a single underlying phenomenon or distinct disorders. One of the aims of the current research is to address the question of whether SOM is a syndrome that precedes certain behavior difficulties in school-age boys. Moreover, school-age boys with high levels of somatic complaints may have more functional impairment and consequently a worse prognosis, at least in the short-term. Determining co-occurring maladjustment and functional impairment associated with chronic somatic complaints may have direct clinical implications for intervention for somatizing children (Gladstein & Holden, 1996).

2.0 STATEMENT OF PURPOSE

Childhood SOM has received much attention across disciplines, however, little is known about the etiology, course, and prognosis of chronic SOM. This results from the fact that most theories addressing the development of SOM in children focus on one central and causal mechanism. Moreover, empirical studies investigating potential risk factors (e.g., stressful life events) have been primarily correlational and cross-sectional in nature. Not without merit, these efforts to establish risk factors are insufficient in explicating a developmental model of somatization. Using a developmental psychopathology perspective (DPP) as a guide, the present research seeks to extend current knowledge by investigating the multiple risk factors and the dynamic processes that foster the development of childhood SOM. To achieve this goal, the study incorporates theoretically and empirically derived risk factors from child, parent-child relationship, family, sociodemographic, and school domains to predict differential longitudinal pathways of childhood SOM. The aim of this developmental approach is to identify risk factors and interactive processes that differentiate individuals who progress to various adaptive and maladaptive outcomes depending upon risk status (Cicchetti & Sroufe, 2000; Cummings et al., 2000).

Further, according to the DPP, the prognosis of child-onset problems can be worse when they are persistent and/or comorbid (Moffitt, 1990, 1993). Due to the nascence of the literature on this subject, there has been no evidence to demonstrate a similar pattern with child-onset

SOM. In addition to broadening the knowledge of the course and antecedents of developmental pathways of SOM, this research also investigates how different developmental pathways of SOM are associated with different forms of maladaptive behavior (e.g., anxiety and depressive symptomatology) and functional impairment in early adolescence.

The investigation of potential risk factors, trajectories, and outcomes are examined in a sample of 310 low-income boys followed longitudinally from ages 2 to 12. This research study has several unique features. First, few empirical studies have utilized longitudinal data sets to study SOM, and no studies to date have attempted to chart its developmental pathways. Due to an exclusive reliance on cross-sectional designs, findings are limited pertaining to causation and comorbidity. The present research design allows for delineating the progression of somatic complaints (and their relation to other internalizing symptoms) throughout childhood into early adolescence, a critical period before the emergence of gender differences of internalizing behaviors. Second, the study's design is novel in its measurement of risk factors in early childhood, when the first signs of SOM emerge. Finally, multiple domains of child maladjustment are evaluated in relation to SOM during early adolescence, and the relationship between SOM and risk factors are assessed using multiple methods and informants. The findings should improve our understanding of the antecedents and course of SOM and clarify whether histories and correlates of SOM can discriminate SOM from other internalizing problems. Such data could prove instructive in formulating model-based intervention strategies for children with SOM across different periods of childhood.

3.0 HYPOTHESES

Based on the research reviewed, the following three (3) groups of hypotheses were tested.

3.1 IDENTIFYING TRAJECTORIES OF SOM FOR SCHOOL-AGE BOYS.

On the basis of recent research that has documented three distinct groups for broadly defined internalizing problems (Cote et al., 2002; Gilliom & Shaw, 2003) and developmental trends that have emerged from studies investigating SOM in community samples of children and adolescents (Rauste-von Wright & von Wright, 1981), three SOM groups were expected in the proposed study: persistent high, increasing, and persistent low groups. Cumulatively, it was hypothesized that 30% of the entire sample would be classified in the persistent high and increasing groups; whereas, the remainder (i.e., the majority) of the sample would be classified in the persistent low group.

3.2 IDENTIFYING ANTECEDENTS OF TRAJECTORY GROUPS.

Based on the literature cited above, it was hypothesized that high levels of variables from the following domains will differentiate the persistent high and increasing SOM trajectory groups from the persistent low SOM group: (1) child risk – difficult temperament and child

negative emotionality, (2) parent-child relationship risk – acrimonious parent-child relationship; (3) family risk - maternal depressive symptoms and interparental conflict; (4) sociodemographic adversity, and (5) school risk – low social skills and academic incompetence. In addition, based on recent research that suggests that children who are characterized by difficult and irritable temperament or high negative emotionality may be more sensitive to aversive conditions in their environment (Aromaa et al., 2000; Walker et al., 2001), boys who show elevated levels of risk factors across child and environmental domains (e.g., child negative emotionality x acrimonious parent-child relationship; difficult temperament x maternal depression) are expected to more likely follow a persistent high or increasingly high trajectory versus a persistent low trajectory of SOM. Table 1 lists the combinations of risk factors that are expected to differentiate the high and increasing SOM groups from the low group.

In addition, based on Walker's and colleagues' (1994, 2001, 2002) studies, it was hypothesized that boys in the increasing SOM group would have lower social and academic skills than boys in the persistent low SOM group in early childhood, but comparable to the persistent high SOM group. Lack of competence in social and academic areas may first become noticeable and prove problematic at school, which makes it more difficult for boys in the two more elevated SOM groups than boys in the persistent low SOM group to make the transition to school. Repeated failure at school, be it social or academic failure, may lower the child's self-esteem and propel somatizing and other internalizing and externalizing problems over time. Hence, it was hypothesized that boys demonstrating lack of competence in social or academic skills in addition to high levels of stress in their proximal environment would be a greater risk for

following the high or increasingly high SOM trajectories compared to the low trajectory. Table 1 shows the interactions tested involving school risk and environmental stress variables.

Finally, in line with the cumulative risk hypothesis (Rutter et al., 1975a, 1975b; Shaw et al., 1994, 1998), it was expected that the persistent high and increasing SOM groups would be differentiated by the quantity of risk factors across domains, such that persistent high group will be characterized by risk from one or more domains and from more domains than the increasing group. It follows that the increasing SOM group is expected to demonstrate risk from more domains than the persistent low SOM group.

3.3 TESTING THE ASSOCIATION OF TRAJECTORY GROUP STATUS AND LATER MALADJUSTMENT AND FUNCTIONAL IMPAIRMENT IN EARLY ADOLESCENCE.

Psychiatric problems have been associated with high SOM in community, psychiatric, and pediatric samples of children and adolescents. In a community sample of children and adolescents, Egger and colleagues (1998, 1999) have shown that somatic complaints predicted anxiety and depression in girls and depression and externalizing disorders in boys. Moreover, studies also demonstrate boys with high levels of SOM demonstrate high levels of anxiety symptoms compared to boys with lower levels of SOM (Rauste-von Wright & von Wright, 1981). For these reasons, boys in the persistent high and increasing groups are expected to show higher levels of depressive, anxiety, and externalizing problems than boys in the persistent low group. Finally, based on research suggesting severe levels of SOM is associated with frequent school absenteeism (i.e., functional impairment) (Bernstein et al., 1997; Walker et al., 1995), it

was hypothesized that boys in the high and increasing SOM groups would show higher levels of school absenteeism compared boys in the low SOM group.

In order to examine these issues, the present study applies SMSS (Nagin, 1999) to maternal reports of SOM collected at ages 6 through 12 and related the resulting trajectory groups to risk factors and later youth maladjustment. To ensure that the groups generalized beyond maternal report, the study incorporated observational measures of child negative emotionality and teacher report of social and academic incompetence in early childhood as well as teacher- and self-report measures of youth internalizing and externalizing behaviors and school records of school absenteeism in early adolescence.

4.0 METHOD

4.1 SUBJECTS

The current study investigated the development of childhood SOM in an ongoing longitudinal study conducted in the Pittsburgh metropolitan area, namely the Pitt Mother and Child Project (PMCP). The PMCP was started in 1991 on a cohort of 310 boys, who were recruited between the ages of 6 to 17 months of age and have been followed to age 12. Recruitment for the PMCP was conducted over the course of two years at Women, Infants, and Children (WIC) Nutritional Supplement Program Clinics in Allegheny County, PA. WIC provides nutritional aid for income-eligible families in the United States. The sample is an ethnically diverse cohort of low-income families. Fifty-four percent of the target children were Caucasian, 40% were African-American, and 6% were from other races. At the time of the initial assessment, the mean annual income across families was $12,552 (*sd* = $7,681), and the mean Hollingshead SES score was 24.5, indicative of a working class sample. Retention rates have been consistently high across the 12-year span of the study. An average of 85% or 264 participants were seen for assessments at ages 10 or 11. A slightly lower but similar percentage (approximately 78%) was seen for the age 12 assessments.

4.2 PROCEDURES

The PMCP participants and their mothers were seen for two- to three-hour visits between the ages of 1.5 and 12 years of age, for which data from the following assessment points will be used in the present study: ages 2, 3.5, 5, 6, 8, 10, 11, and 12. During these visits, mothers and sons participated in a number of parent-child interaction tasks, and mothers completed a series of questionnaires about their child's behavior, their own adjustment, and their family functioning. Beginning at the age-8 assessment, children were interviewed about similar topics. The visits at ages 3.5, 6, and 11 were conducted at the laboratory, with the remaining visits conducted at the participants' homes. Teachers also completed a packet of questionnaires via postal mail on boys' behaviors and competence annually between ages 6-12. Participants were reimbursed for their time after each assessment.

4.3 MEASURES

In this section, the measures used for SOM trajectories and later child maladjustment and functional impairment are described and summarized in Table 2. Next, the predictor variables of SOM trajectories are described and summarized in Table 1. The importance of risk factors from five domains was assessed in this study: child, parent-child relationship, family, sociodemographic, and school. As a means of minimizing shared-method variance, reports from different informants of SOM, risk factors, and outcomes were employed when available. A second priority was to measure risk factors during the initial stages of when trajectories of SOM are being assessed (i.e., ages 2, 3.5, 5, and 6). When risk factors are measured later, it is difficult

to determine whether they precede or follow the onset of problem behavior (Shaw, Gilliom, Ingoldsby, & Nagin, 2003).

4.3.1 Child Adjustment Variables

Child Behavior Checklist (CBCL, Achenbach, 1991a) and *Teacher Report Form* (TRF, Achenbach & Edelbrock, 1986). The *CBCL* and the *TRF* are instruments that assess behavior problems exhibited by children for parent and teachers, respectively. Parents and teachers rate the items on a three point scale ranging from "Not True" to "Very True" of the child in the past 6 or 2 months, respectively. From this behavior checklist, corresponding profiles are designed to assess children's behavior problems. These instruments have been used extensively with clinical and research samples and have been shown to be valid and reliable with nationally normed data (Achenbach, 1991; Achenbach & Edelbrock, 1986).

4.3.1.1 Maternal-Reported Trajectories.

To map trajectories of SOM from ages 6 to 12 (i.e., assessment periods include ages 6, 8, 10, 11, and 12), boys' somatic complaints were assessed using maternal report on the Somatic Complaints subscale on the *CBCL* (Achenbach, 1991a). The Somatic Complaints subscale includes the items *feels dizzy* and *overtired* in addition to the following medically unexplained physical problems: *aches and pains, headaches, nausea (feels sick), problems with eyes, rashes or other skin problems, stomachaches or cramps*, and *vomiting*, which are each scored 0, 1, or 2 depending on the symptom's frequency in the past 6 months. The *CBCL* Somatic Complaints subscale has been used in extensively in community, psychiatric, and pediatric populations (e.g.,

Andrasik, Kabela, Quinn, Attanasio, & et al., 1988; Bernstein et al., 1997; Cunningham, McGrath, Ferguson, Humphreys, & et al., 1987; Dorn et al., 2003; McCauley et al., 1991; Walker et al., 1993) and has excellent psychometric properties (Achenbach et al., 1989). For the present sample, alpha coefficients for the Somatic Complaints subscale range from .59 to .66 across ages 6 to12.

4.3.1.2 Teacher-Reported Outcomes.

Teacher reports of child adjustment were used for two purposes: (1) to corroborate maternal-report trajectories of SOM and (2) to determine whether these trajectories are predictive of broader adjustment problems. Specifically, the Somatic Complaints subscale, the Externalizing broad-band factor, and a composite of the Withdrawn and Anxious/Depressed syndromes of the *TRF* (Achenbach & Edelbrock, 1986) were used to measure co-occurring child problems associated with maternal-reported trajectories of SOM in early adolescence. To maximize the frequency of teacher reports, *TRF* scores from ages 11 and 12 were used. Scores for the Somatic Complaints subscale, the Externalizing broad-band factor, and the composite of the Withdrawn and Anxious/Depressed syndromes were averaged across time-points if both were available; otherwise, only one score was used.

4.3.1.3 School Reports.

School absenteeism was assessed at ages 10 through 12 to measure functional impairment that may be associated with maternal-reported SOM trajectories. School records were obtained from one school district with the highest percentage of children in the study (i.e., Pittsburgh Public Schools, n = 130) and were used to determine the total number of school days for which boys were absent during the school year. To maximize the frequency of school reports, school

absenteeism from ages 11 and 12 were used; however if one of those time points was missing, absenteeism from age 10 was substituted for the missing value. Absenteeism was averaged for the two time points when available; otherwise, only one time point was used. Boys who were enrolled for less than 100 days of the school year were not included in analyses involving school absenteeism.

4.3.1.4 Youth-Reported Outcomes.

In addition to teacher reports of child adjustment difficulties, boys' self-reports of depressive and anxiety symptoms and antisocial activities were assessed at age 12 to measure co-occurring child problems and to predict a wider range of adjustment difficulties that may be associated with maternal-reported SOM trajectories. Boys' self-reports of depressive symptoms were measured with a 10-item short form of the *Child Depression Inventory* (CDI; Kovacs, 1992; $\alpha = .66$). For the items on the *CDI*, boys were presented with a group of three statements and were asked to choose the sentence that best described their feelings in the past two weeks. Boys' anxiety symptoms were measured with a 10-item short form of the *Multidimensional Anxiety Scale for Children* (MASC, March, Parker, Sullivan, Stallings, & Conners, 1997). For the items on the *MASC*, boys were presented with a series of statements indicating anxiety-arousing situations (e.g., "I'm afraid that other kids will make fun of me") and were asked to rate how true each statement was for him "recently" on a 4-point scale. For the present study, the two items "I get dizzy or faint feelings" and "I feel sick to my stomach" were removed due to their overlap with items on the *CBCL* Somatic Complaints subscale. The resulting 8-item short form of the *MASC* has an alpha coefficient of .65. Finally, boys' self-reports of delinquency were assessed with an age-appropriate adaptation of the 40-item *Self-Report of Delinquency* (SRD; Elliot, Huizinga, & Ageton, 1985, $\alpha = .84$), a semi-structured interview. Using a 3-point

rating system (1 = "never," 2 = "once/twice," 3 = "more often"), boys rated the extent to which they engaged in different types of antisocial behaviors (e.g., stealing, throwing rocks or bottles at people) within the last year. The *CDI*, *MASC*, and *SRD* have been shown to have adequate reliability and validity (Elliot et al., 1985; Kazdin, French, Unis, Esveldt-Dawson, & Sherick, 1983; March et al., 1997).

4.3.2 Child Risk

Negative emotionality. A composite of two observational methods from the age-5 home assessment were used as a measure of child negative emotionality. First, at the end of the age-5 assessment, interviewers rated their impressions of boys' negative affectivity (i.e., oversensitive, grouchy, or cranky) on a 4-point scale. Second, global ratings of child negative reactivity were made during a one-hour sibling interaction. Ratings were coded from videotapes using the Sibling Conflict Coding System (SCCS, Garcia, Shaw, Winslow, & Yaggi, 2000), an observational coding system for sibling and parent/child interaction adapted from a system developed by Volling and Belsky (1992). The SCCS was designed to capture the amount and quality of sibling conflict with molecular and global codes. The global rating of child negative reactivity reflects how much the target child whined, reacted negatively, or overreacted when provoked by his sibling, relative to how much the sibling was intentionally provoking the child. A consensus procedure was utilized for this rating. Scores from both observational measures were standardized and then averaged to form a negative emotionality score ($r = .44$, $p < .01$).

Difficult and irritable temperament. Observations of child negative emotionality at age 5 were supplemented by earlier maternal reports of negative emotionality, using the Difficulty factor of the *Infant Characteristics Questionnaire* (ICQ, Bates, Freeland, & Lounsbury, 1979).

During the age-2 assessment, mothers rated their toddlers on seven items indexing persistent difficult and irritable behavior based on a 7-point Likert scale. The *ICQ* meets customary psychometric standards for maternal reports on infant temperament, has the advantage of brevity, and has shown longitudinal relations with preschool behavior problems (Bates, Maslin, & Frankel, 1985).

4.3.3 Parent-Child Relationship Risk

Parent-child conflict. To assess the conflict in the parent-child relationship, the conflict factor of the *Adult-Child Relationship Scale* (ACRS) was used. The ACRS is an adaptation of the *Student-Teacher Relationship Scale* (Pianta & Steinberg, 1991), a 30-item questionnaire that was originally designed to assess teacher's perceptions of their relationship with children. Issues related to attachment and the adult's feelings about the child and his behavior are ascertained. The ACRS was adapted for use with mothers by slightly changing the wording of items to reflect the parent-child relationship and was used at the age 5 home visit. For the proposed study, because of the interest in conflictual dyadic relationships, the "conflict" factor was utilized, comprised of eight items (e.g., "This child and I seem to always struggling with one another", "This child feels I am unfair to him") assessing the frequency of conflict on a 5-point Likert scale. Coefficient alpha for this measure in a sample of preschool children was .90 (Pianta & Steinberg, 1991). In the PMCP sample, Cronbach's alpha was .81 for mother-reported data at age 5.

4.3.4 Family Risk

Two measures of proximal within-family risk were evaluated, both of which have been previously related to boys' internalizing problems: maternal depressive symptomatology and parental conflict.

Maternal depressive symptoms. Maternal depressive symptoms were measured with the 21-item *Beck Depression Inventory* (BDI, Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961), which was administered at the age 3.5, 5, and 6 assessments. The *BDI* has been shown to have good reliability and validity (Beck, Steer, & Garbin, 1988). Instructions on the *BDI* were altered to cover depressive symptoms during the previous six months (instead of the last two weeks) to provide a more stable indication of maternal affect throughout her child's life. *BDI* total scores were aggregated across the three time-points and averaged to form a composite score. When one or two assessment points were missing, scores from the remaining period(s) was substituted.

Interparental conflict. Strategies of interparental conflict resolution were measured with the *Conflict Tactics Scales* (CTS-Form N, Straus, 1979), administered at the age 3.5 and 6 visits. This 26-item questionnaire is designed to measure the use of reasoning, verbal aggression, and violence within the family, and how often the child witnessed the acts over the past year. A composite of the Verbal and Physical Aggression factors was used to generate a measure of conflict in the relationship. The composite scores from ages 3.5 and 6 were averaged (α = .77-.88). Again, when only one time point was available, it was used as the final score to minimize missing data.

4.3.5 Sociodemographic Adversity

Cumulative adversity index. A cumulative index of four sociodemographic risk factors was devised to measure sociodemographic adversity. Adversity scores will range from 0 to 4 depending upon how many of the following risk factors are present: 1) low family income, 2) low maternal education, 3) single parent status, and 4) high neighborhood dangerousness. The first three constructs were assessed from an interview administered at the age 3.5-, 5-, and 6-visits, reflecting a 2.5-year period. Neighborhood dangerousness was measured with the *Neighborhood Questionnaire* (NQ; Pittsburgh Youth Study, 1991), a 17-item measure of problematic and dangerous activities within a family's neighborhood as perceived by the parent at the age-5 visit. The items were summed to create one factor score for neighborhood dangerousness (α = .94-.95; Shaw et al., 2003). To create a cumulative index score, each of the four measures listed above were assigned a score of one (1) if they meet the following criteria (indicating greater adversity), and zero (0) if they do not. If the family was living in poverty, as defined by the U.S. Census, at any of the three time points, the family received a score of 1 for low income. Low maternal education was defined as present if the mother did not complete high school or equivalent (e.g., GED). If a child was living with a single parent at any of the three time points, a score of 1 was given. Finally, for the *NQ*, criterion was set at or greater than one standard deviation above the sample mean. The criterion scores were summed and then averaged (in the case where one of the four risk factors was missing, e.g., *NQ*) to create the cumulative index of sociodemographic adversity with scores ranging from 0 to 1.

4.3.6 School Risk

Social skills. Teacher ratings on the Social Skills domain of the *Social Skills Rating System* (SSRS, Gresham & Elliott, 1990) were used to measure boys' social skills in the classroom at ages 6 and 7. The Social Skills domain measures positive child behaviors, including cooperation, assertion, responsibility, and self-control. The *SSRS* is an instrument that provides a broad, multi-rater assessment of student social behaviors that can affect teacher-student relations, peer acceptance, and academic performance. Teachers rate the frequency of child behaviors on a three-point scale ranging from "Never" to "Very Often." To maximize the frequency of teacher reports, *SSRS* scores from both ages 6 and 7 were averaged when available; otherwise, only one score was used. The Social Skills scale demonstrates good reliability and validity (Gresham & Elliott, 1990).

Academic incompetence. To generate a measure of academic incompetence and underachievement in the classroom, the following three items were selected from the *TRF*: 'Has difficulty learning'(item 49), 'Poor school work' (item 61), and 'Underachieving, not working up to potential'(item 92). Scores from both ages 6 and 7 were averaged when available (α = .88 and .83, respectively); otherwise, only one score was used.

4.4 DATA ANALYTIC PLAN

Analyses proceeded in three stages. In the first stage, semi-parametric mixture modeling was applied to mothers' reports of SOM from ages 6 to 12 to determine whether groups with distinct longitudinal trajectories for school-age boys (e.g., low, middle increasing, high) could be

identified. In the second stage, binary logistic regressions were performed to identify early child, parent-child, family, sociodemographic, and school risk factors that might distinguish membership among trajectory groups. In the third stage, univariate analyses (i.e., ANOVAs) were performed to examine the covariation of trajectory group status and later child maladjustment. To carry out the data analytic plan, a strategy for data analysis that captures children's developmental histories of SOM was needed. Nagin's (1999) semi-parametric group-based approach for analyzing developmental trajectories (TRAJ) was well-suited for this purpose. TRAJ provides objective criteria for deciding how many trajectories exist within a population and for estimating the proportion of individuals who follow each trajectory. Although some of the groups created by TRAJ were expected to be small, previous research conducted using this technique has had sufficient power to detect group differences despite having cell sizes as small as 14 (e.g., Laub, Nagin, & Sampson, 1998; Shaw et al., 2003). Below is a brief overview of TRAJ.

4.4.1 Semi-Parametric Mixture Modeling

To study SOM trajectories, this investigation used semi-parametric mixture modeling (SPMM; Nagin, 1999). SPMM is designed to identify groups of individuals with distinct trajectories. This approach provides empirical bases for determining (1) the number of groups in the population and (2) the optimal shapes of the trajectories for repeated measures of a single outcome variable (Nagin, 1999; Nagin & Tremblay, 1999). Specifically, each individual's *i*'s score on the variable of interest *y* (e.g., SOM) at a specific time *t*, given membership in a specific group *j*, is approximated with:

$$y_{it}^{j} = \beta_0^{j} + \beta_1^{j} \text{Age}_{it} + \beta_2^{j} \text{Age}^2{}_{it} + \varepsilon_{it} \quad (1)$$

where the parameters β_0^j (intercept, or level when Age = 0), β_1^j (slope, or growth rate), and β_2^j (slope^2, or change in growth rate), determine the shape of the trajectory and are superscripted by *j* to indicate that they are free to vary across groups. The residual error of each individual's score at a given time is denoted by ε_{it}. The estimation procedure yields two additional parameters: the proportion of the population belonging to each group, π_j, and the conditional probability of individual *i*'s longitudinal sequence of Y_i, given membership in group *j*, $P(Y_i|j)$.

To determine the optimal number of trajectories for SOM from ages 6 to 12, models with two to four groups were estimated, and the model that showed the most optimal Bayesian Information Criterion (BIC) was chosen for use in the remaining analyses. Unlike cluster analysis and other ad hoc person-oriented methods, BIC uses empirical methods for finding the best model. That is, the BIC index rewards parsimony in the model specification by imposing penalty functions on the log likelihood for increasing the number of the model parameters to be estimated. For example, for two models with equivalent likelihoods, the model with fewer groups is preferred.

4.4.2 Model Estimation

Model estimation produces two main outputs: parameter estimates that demarcate the shape of the trajectories and posterior probabilities of group membership for each individual in

the estimation sample. The posterior probabilities estimate the probability of belonging to each of the trajectory groups. For instance, if an individual who receives high SOM ratings throughout childhood, then the probability of belonging to the persistent high group would be high whereas the probability of belonging to the low trajectory group would be close to zero. The posterior probabilities provide a basis for assigning individuals to trajectory groups: The largest probability for each individual indicates the trajectory that best conforms to that individual's behavior over time.

5.0 RESULTS

The presentation of results follows the analytic plan outlined above. First, descriptive statistics and bivariate correlations among variables are reported. Next, SOM trajectory groups identified by SPMM are presented, followed by their relations with risk factors from the child, parent-child relationship, family, sociodemographic, and school domains. Interactions between child or school risk and proximal environmental variables (i.e., stressors) are also presented. Finally, the relationships between child maladjustment, including functional impairment, and trajectory group status are examined.

5.1 DESCRIPTIVE STATISTICS AND BIVARIATE CORRELATIONS

Table 3a displays the descriptive statistics for individual somatic complaints and total scores on the *CBCL Somatic Complaints* subscale as reported by mothers at ages 6, 8, 10, 11, and 12. A more meaningful representation of the prevalence of each symptom across ages, however, is presented in Table 3b. For each age, the percentage of boys for which each symptom was "somewhat/ sometimes true" or "often/ very true" within the previous six months is presented, and in parentheses, the actual number of boys is represented. Across ages, the average percentage of boys who were reported as experiencing at least one somatic symptom "sometimes" or "often" was 45%, which is consistent with Alfven's (1993) findings with

Swedish school children (i.e., 47%). For polysymptomatic presentation, 7.7% of boys, averaged across time points, received a score of 4 or higher (e.g., two symptoms being "very true" or four different symptoms being "sometimes" true). Using a slightly more stringent definition of polysymptomatology, 6% received a score of 5 or higher, indicating that the boys experienced at least three symptoms. This result is slightly higher than Offord and colleagues' (1987) findings from the Ontario Child Health Study (i.e., 4.5% of boys); however, boys in that study were older – between the ages of 12 and 16 – which is when somatic complaints tend to decrease in prevalence for boys.

Consistent with the literature, this study found that the prevalence of SOM, be it defined as mono- or poly-symptomatic, was low at age six and generally increased over time (Berntsson & Koehler, 2001; Campo et al., 1999; Egger et al., 1998). In this particular sample of boys, the prevalence of SOM peaked at age 11 (52%) and then dropped slightly at age 12 to the same percentage reported at age 10 (48% and 47%, respectively).

The prevalence for individual symptoms was also consistent with the extant literature. For example, across ages, the most commonly reported symptom was *headache* with a prevalence of 8-25%, followed by *aches and pains* affecting 9-24%, and *stomachaches or cramps* 10-17%. Other common complaints were *rashes or other skin problems* (11-17%), *overtired*, more commonly referred to as "fatigue," (9-14%), and *nausea, feels sick* (5-13%). *Feels dizzy* was somewhat lower in prevalence compared to that which has been reported in the literature (5-13% vs. 15%; Campo & Fritsch, 1994), and *vomiting* was the least common symptom reported across time (3-7%).

The symptoms with the highest prevalence at each age are as follows: *stomachaches* (or RAP) and *rashes* at age 6, *headaches* and *stomachaches* at age 8, and *headaches* (followed by

stomachaches and *aches or pains*) at ages 10, 11, and 12. These findings are also in keeping with the literature, such that RAP has been found to be the most common complaint around age 9, and headache is the most frequent complaint around age 12 (Achenbach et al., 1989; Campo & Fritsch, 1994; Offord et al., 1987). Pseudoneurological symptoms have been reported as being rare before the age of 6, and in fact, less than 0.5% of boys in the present study were reported as *feeling dizzy* at age 6.

Bivariate intercorrelation coefficients among maternal report of SOM (total score) at ages 6, 8, 10, 11, and 12 appear in Table 3c. SOM was moderately correlated across time points. The highest correlation was between SOM at ages 11 and 12 ($r = .58$, $p < .01$); whereas, the lowest was between SOM at ages 6 and 12 ($r = .29$, $p < .01$). In general, correlations became stronger over time, except for age 6 (see row), where the opposite trend was found. That is, the more time that elapsed between age 6 and the other time point, the lower the correlation became. Previous literature concerning the stability of SOM, particularly for boys, has been equivocal (Aro et al., 1987; Dhossche et al., 2001; Rauste-von Wright & von Wright, 1981; Walker et al., 1994). However, the current findings suggest a moderately stable course of SOM across childhood for boys.

Descriptive statistics for risk factors and outcome variables appear in Tables 4a and 5a. Means and standard deviations for difficult temperament, negative emotionality, parent-child conflict, maternal depressive symptoms, interparental conflict, sociodemographic adversity, social skills, and academic incompetence are presented in Table 4a. One-way ANOVAs demonstrated that minority boys had higher mean scores than Caucasian boys on parent-child conflict (26.36 vs. 24.10, $p < .02$), maternal depressive symptoms (8.31 vs. 6.52, $p < .01$), the adversity index (.46 vs. .23, $p < .001$), and academic incompetence (2.14 vs. 1.45, $p < .01$), and

lower mean scores for social skills (35.43 vs. 38.72, $p < .02$). In Table 5a, means and standard deviations are shown for the outcome variables: Externalizing, Anxiety/Depression, and Somatic Complaints by teacher report; days absent from school obtained from school records; and depressive and anxiety symptoms and antisocial behaviors by youth self-report. Again, differences between minority and Caucasian boys were found. Teacher- and youth-reported externalizing scores were significantly higher for minority boys compared to Caucasian boys (*p*s < .05), whereas, teachers reported that Caucasian boys demonstrated more internalizing behaviors than minority boys ($p < .05$). Direct comparison of these descriptive statistics with those of other studies is hindered by the fact that many of the variables are unique to this study (e.g., child risk variables, sociodemographic adversity index, and youth self-report outcome variables) or the PMCP (e.g., observational measures). However, mothers reported an average score of 7.38 on the *BDI*, which indicates mild depressive symptomatology (Beck et al., 1988), and the average scores on the *SSRS* Social Skills and *TRF* Externalizing were in the average range for boys at ages 6/7 and 11/12, respectively (Achenbach & Edelbrock, 1986; Gresham & Elliott, 1990).

Bivariate correlation coefficients among risk factors and with SOM across ages are presented in Table 4b and 4c, respectively. Table 4b shows that, in general, parent-child conflict was positively related to all other risk factors across domains (*r*s = .20 - .34, $p < .01$), particularly to other maternal-report variables. The one exception was with teacher-reported social skills, where the relationship was negative ($r = -.27$, $p < .01$), indicating that higher parent-child conflict was related to lower social skills. In addition to parent-child conflict, social skills were negatively related to maternal depressive symptoms and the sociodemographic adversity index (*r*s = -.22 and -.17, respectively; $p < .01$). For child risk variables, difficult temperament was

positively, yet modestly, correlated with family risk variables, namely, parent-child conflict, maternal depression, and interparental conflict (*r*s = .29, .13, and .17, respectively; $p < .05$); whereas, child negative emotionality was related to parent-child conflict and the adversity index (*r*s = .21 and .14, respectively; $p < .05$).

Table 4c shows that maternal depressive symptoms were significantly, yet modestly, related to SOM at ages 6, 8, and 11. As expected, teacher-reported social skills were inversely related to SOM at ages 8, 10 , and 11 (*r*s = -.14 to -.16, $p < .05$), that is, as somatic complaints increased, socials skills declined. No other significant relationships were found between the risk factors and SOM, except for academic incompetence, which was modestly related to SOM at age 11 ($r = .20$, $p < .01$).

Bivariate correlation coefficients among outcome variables and with SOM are presented in Table 5b and 5c, respectively. All teacher-reported outcomes were related to one another (*r*s = .29 and .52, respectively; $p < .01$). School absenteeism was related to both teacher and youth report of externalizing/delinquency (*r*s = .29 and .32, respectively; $p < .01$) and to teacher report of somatic complaints ($r = .23$, $p < .05$). Youth self-reports were related to some outcomes within and across reporters. For instance, youth-reported depression was positively correlated with youth and teacher reports of anxiety/withdrawal (*r*s = .21 and .32, respectively; $p < .01$) and externalizing/delinquency (both *r*s = .20; $p < .01$). Reports of externalizing problems across informants (i.e., youth and teacher) were also correlated ($r = .37$, $p < .01$).

Table 5c shows that two outcome variables were consistently related to SOM over time. Teacher-reported SOM at ages 11 and 12 were significantly related to maternal report of SOM at ages 8-12, though the association tended to decrease in strength over time (i.e., *r*s = .22 - .18 from ages 8 to 12, respectively, $p < .05$). Similarly, school absenteeism was positively correlated

with maternal report of SOM across all ages, and the strength of the relationship also decreased over time (i.e., *rs* = .36 to .18 from age 6 to age 12, respectively). No other teacher- or youth-reported outcome variables were significantly related to SOM.

5.2 IDENTIFIED TRAJECTORIES OF MATERNAL-REPORTED SOM FOR SCHOOL-AGE BOYS

To investigate the number of SOM trajectories from ages 6 to 12, Nagin's semi-parametric mixture modeling was employed using the steps described above. Maternal reports on the *CBCL* Somatic Complaints subscale at ages 6, 8, 10, 11, and 12 were used as the outcome variable *y* (i.e., SOM) in these analyses. As stated above, two to four models were estimated (see Table 6). Ultimately, it was found that seven (7) cases were "outliers," in the sense that they did not belong to the groups found using the trajectory group-based models. Two cases, which started off high on SOM and rapidly dropped to zero for the last two time points, were particularly troublesome. Hence, it was decided best to exclude these seven cases from the TRAJ modeling procedure. Without the seven cases, the three-group model was selected as the best fitting model, as it had the least negative BIC score, -1549.89 (D'Unger, Land, McCall, & Nagin, 1998). The censored normal (CNORM) model was chosen because it is useful for modeling the distribution of psychometric scale data. A distribution allowing for censoring is used because the data tend to cluster at the minimum of the scale and at the scale maximum (Jones, Nagin, & Roeder, 2001).

Figure 3 plots the three SOM trajectory groups, showing both the observed and predicted trajectories by group. The observed trajectories reflect mean SOM scores for boys assigned to

each group on the basis of their posterior probabilities. Visual inspection shows good agreement between the observed and predicted curves. Despite the slight drop in SOM at age 12, linear trends were deemed sufficient to describe the data. Quadratic and cubic functions were tried, and the coefficients of the higher order terms were not significant (Bobby Jones, personal communication on May 20, 2005).

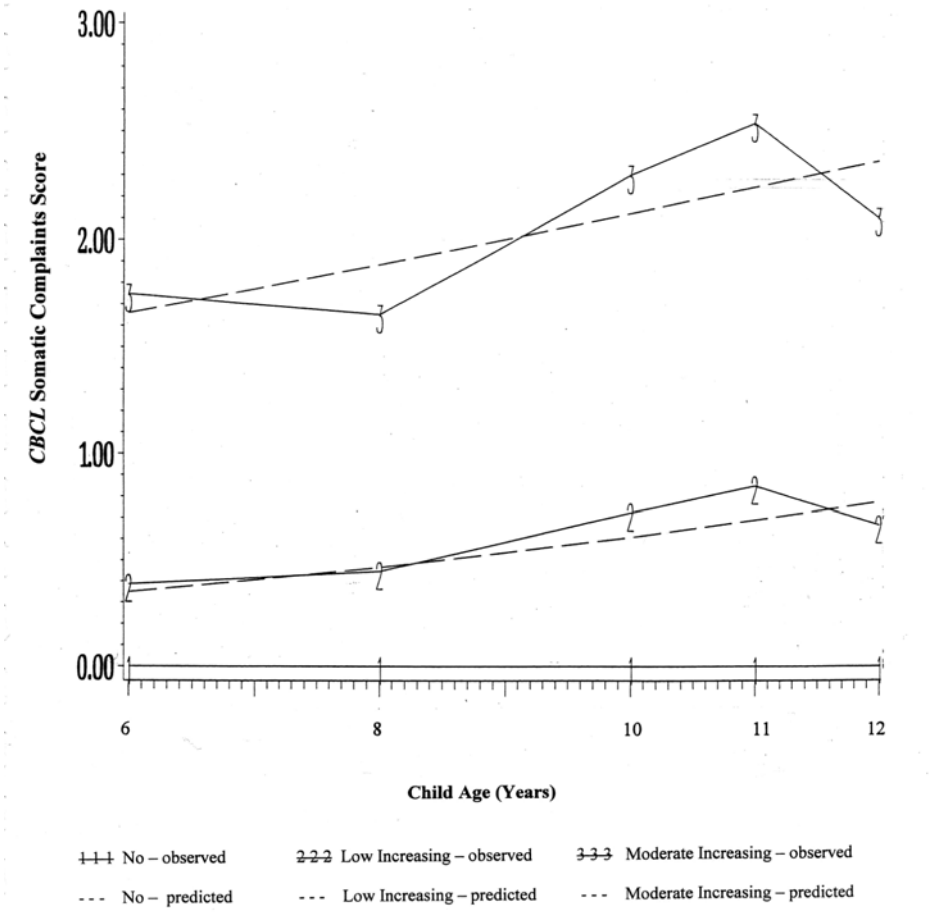

Figure 3: Trajectories of somatization in school-age boys.

Table 7 shows the means and standard deviations for SOM by age and trajectory group status. Overall, the model-based trajectories reveal increases in SOM for the majority of boys between the ages of 6 and 12. One group, accounting for 17% of the sample, exhibited no somatic complaints across time and will be referred to as the No group (constant = -12.13, *SE* = 802.74, $p = .99$). A second, larger group comprising 54% of the sample, may be described as exhibiting low SOM initially which then gradually increased over time (constant = -0.63, *SE* = .24, $p < .01$; slope = 1.88, *SE* = .55, $p < .001$). This group will be referred to as the Low Increasing group. On average, mothers reported slightly less than one somatic symptom ($x = .91$, see Table 3a) at age 11 for boys in this group. The third and final group accounted for 28% of the sample and is referred to as the Moderate Increasing group (constant = 1.79, *SE* = .23, $p < .0001$; slope = 1.47, *SE* = .63, $p = .02$). The Moderate Increasing group showed a similar pattern to the Low Increasing group, except that the mean SOM score at each time point was reported at a level three times greater than that of the Low Increasing group.

While examining the pattern of scores for the excluded cases, five of the seven boys followed a trajectory that was similar in shape to those of the latter two groups but at a much higher level. Although these boys initially demonstrated a mean SOM score equivalent to that of the Moderate Increasing group at age 6, their trajectory ended with two very high end points at ages 11 and 12 (see Figure 4). Technically, this group of boys was not a TRAJ-identified group. Nevertheless, this small yet distinct group, comprising 2% of the entire sample, were used in some of the following analyses, as these select boys may constitute a group of children at extreme risk for SOM and other associated risks. This fourth "group" will be referred to as the Moderate to High (MTH) group.

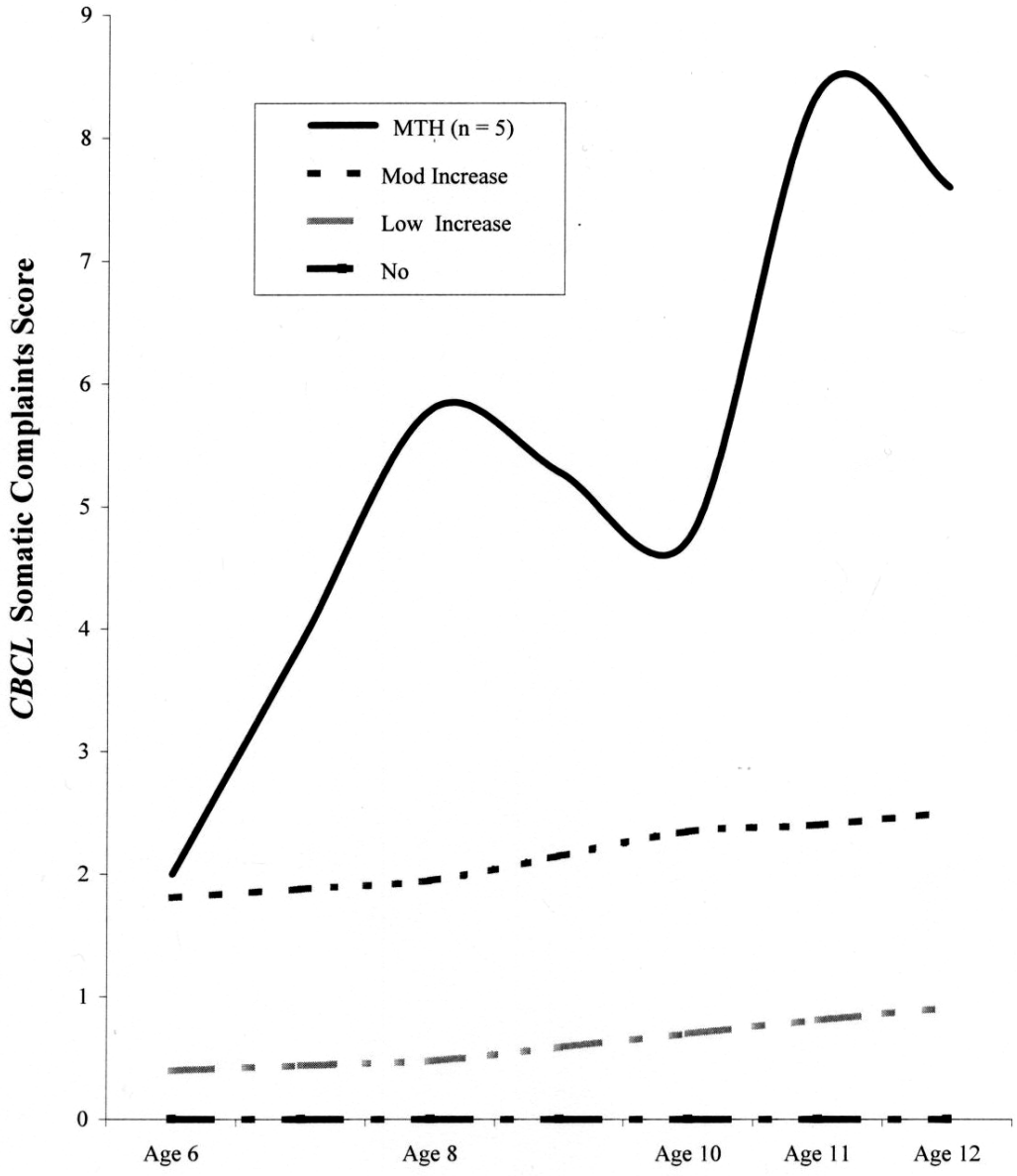

Figure 4: Actual mean values of SOM for the Moderate to High (MTH) group compared to the three predicted trajectories of SOM.

5.3 PREDICTORS OF TRAJECTORY GROUPS

To examine the hypothesis that risk factors from child, parent-child relationship, family, sociodemographic, and school domains would discriminate SOM trajectory groups, analyses of variance (ANOVAs) were conducted to examine the relationship between individual risk factors and trajectory group membership. One-tailed tests of significance were employed because hypotheses with directionality of findings had been specified. In these analyses, the Moderate to High (MTH) group was included for reasons mentioned in the section above. In Table 8, scores on risk factors by trajectory group and statistical differences between individual groups are presented. The *F*-statistics for parent-child conflict, maternal depressive symptoms, and child's social skills were significant (*p*s < .05), while the *F*-statistic tended toward significance for child's academic incompetence ($p < .10$). Post hoc comparisons using Tukey's tests indicated significantly higher scores on parent-child conflict ($p < .05$) and maternal depression ($p < .01$) for the Moderate Increasing group compared to the No group. Significantly lower social skills were found for the MTH group compared to the other three groups and higher academic incompetence scores discriminated the MTH group from the Moderate and Low Increasing groups. A nonsignificant trend for higher academic incompetence scores also was found between the MTH and No groups in the expected direction ($p < .10$). Nonsignificant results were found for between-group comparisons computed for difficult temperament, child negative emotionality, interparental conflict and sociodemographic adversity. However, consistent with hypotheses, risk factor scores tended to increase with increasing rates of SOM.

Tables 9-11b contain coefficients and their associated statistics estimated in binary logit analyses. Logit coefficients indicate the direction, strength, and reliability of the relationship between predictor variables. The exponentiated value of the coefficient measures the odds ratio

– the relative change in the risk factor. To identify factors that significantly discriminate trajectory group status, all independent variables listed in Table 2 (e.g., ACRS, child negative emotionality) were entered into a binary logit model to account for the influence of all risk factors simultaneously (see Table 9). Out of all the comparisons among the No, Low Increasing, and Moderate Increasing groups, only one risk factor, maternal depressive symptoms, was found to discriminate trajectory group status. Specifically, greater maternal depression distinguished the Moderate Increasing group from the Low Increasing ($p = .06$) and the No ($p < .05$) groups. Significant results may have been underestimated in these analyses because of the high multicollinearity of the predictor variables, especially those that relied on maternal report. Therefore, regressions were performed again, but this time risk factors were limited to those that discriminated SOM trajectory group status within a univariate framework.

Using the variables identified in the previous ANOVAs, parent-child conflict, maternal depressive symptoms, social skills, and academic incompetence were entered into binary logit models to predict group membership, the results of which are summarized in Table 10. The results were similar to those in the initial ANOVA. That is, higher levels of both parent-child conflict and maternal depression increased the likelihood of following the Moderate Increasing trajectory versus the No trajectory by 7% ($p < .05$) and 8% ($p < .07$), respectively. Trends for these variables also emerged when the Moderate Increasing group was compared to the Low Increasing group, such that a one unit increase in each of these risk factors (1 point on the *ACRS* and *BDI*) increased the odds of being in the Moderate Increasing group by 4% and 5% (for parent-child conflict and maternal depression, respectively).

5.4 INTERACTIONS PREDICTING TRAJECTORY GROUPS

The next set of analyses examined whether interactions between child risk factors (i.e., difficult temperament and negative emotionality) x proximal environmental stress (e.g., maternal depressive symptoms, interparental conflict) would discriminate trajectory group membership. Interaction terms involving these factors were entered into binary logit models after accounting for the direct effects of individual risk factors (see Table 1 for list of interactions that were tested). Three interactions, namely difficult temperament x adversity index, difficult temperament x interparental conflict, and child negative emotionality x parent-child conflict, significantly differentiated the Low Increasing group from the No group ($p < .05$, for each interaction). The interaction between child negative emotionality x parent-child conflict also significantly differentiated the Moderate Increasing group from the Low Increasing group ($p < .05$). Tables 11a and 11b summarize the coefficients and associated statistics for these significant interactions.

To explore the nature of these effects, the probability of being in the higher of the two trajectories being compared was modeled with the following logistic regression equation:

$$log\ (p/(1-p)) = \beta_0 + \beta_1 V_1 + \beta_2 V_{2q} + \beta_3 (V_1 * V_2) \qquad (2)$$

where p is the probability of a boy belonging to the Low Increasing group versus the No group as the reference group (i.e., three models), or the probability of the boy belonging to the Moderate Increasing group versus the Low Increasing group as the reference group (i.e., one model). Three equations were computed for scores on the first variable (V_1) within each interaction, while holding the second variable's score (V_{2q}) constant (at the first, second, or third quartile, q, where the adversity increases from quartile 1 to quartile 3). That is, a separate equation was computed for each quartile, where $q = 1$, 2, and 3, for V_{2q}. Each risk factor was V_1

in one set of equations and V_{2q} in another. Therefore, for each interaction, six (6) equations were run. Risk factors were not dichotomized into "high" and "low" groups on the basis that one loses power, despite gaining easier interpretability.

Figures 5-8 illustrate the effects of the predictor variables on the probability of group membership from the logistic model.[2] In Figures 5-7, the graph represents the probability of a child being in the Low Increasing group compared to No group (y-axis) when one predictor variable is held constant at its 1st, 2nd, and 3rd quartiles, which are represented by the continuous, dashed, and dotted lines, respectively, as the other variable increases (shown on the x-axis). For each graph, greater risk is represented by higher x-axis values. Figure 8 was generated in the same fashion but gives the probability of a boy being in the Moderate Increasing group versus the Low Increasing group (shown on the y-axis).

Figure 5 represents the effects of difficult temperament and adversity index predicting membership to the Low Increasing group compared to the No group. The curves show that the interaction is occurring at low levels of each risk factor. For example, only when the adversity index is low does a child's temperament have a pronounced effect on probability of the expected direction. That is, as temperament scores become more adverse, the probability of being in the Low Increasing group increases from approximately .60 to .90. The same can be said for the interaction when temperament scores are low; as the adversity index score increases the probability of being in the Low Increasing group increases from approximately .70 to .80. Consistent with a ceiling effect, when both risk factors are at high levels, the probability of being in the Low Increasing group compared to the No group decreases.

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[2] The logistic regressions were evaluated for overall fit using the Hosmer-Lemeshow test. The presence of problematic outliers and influential cases was also investigated. In all the models, there was adequate fit to the data and no evidence that outliers or influential cases were biasing the results.

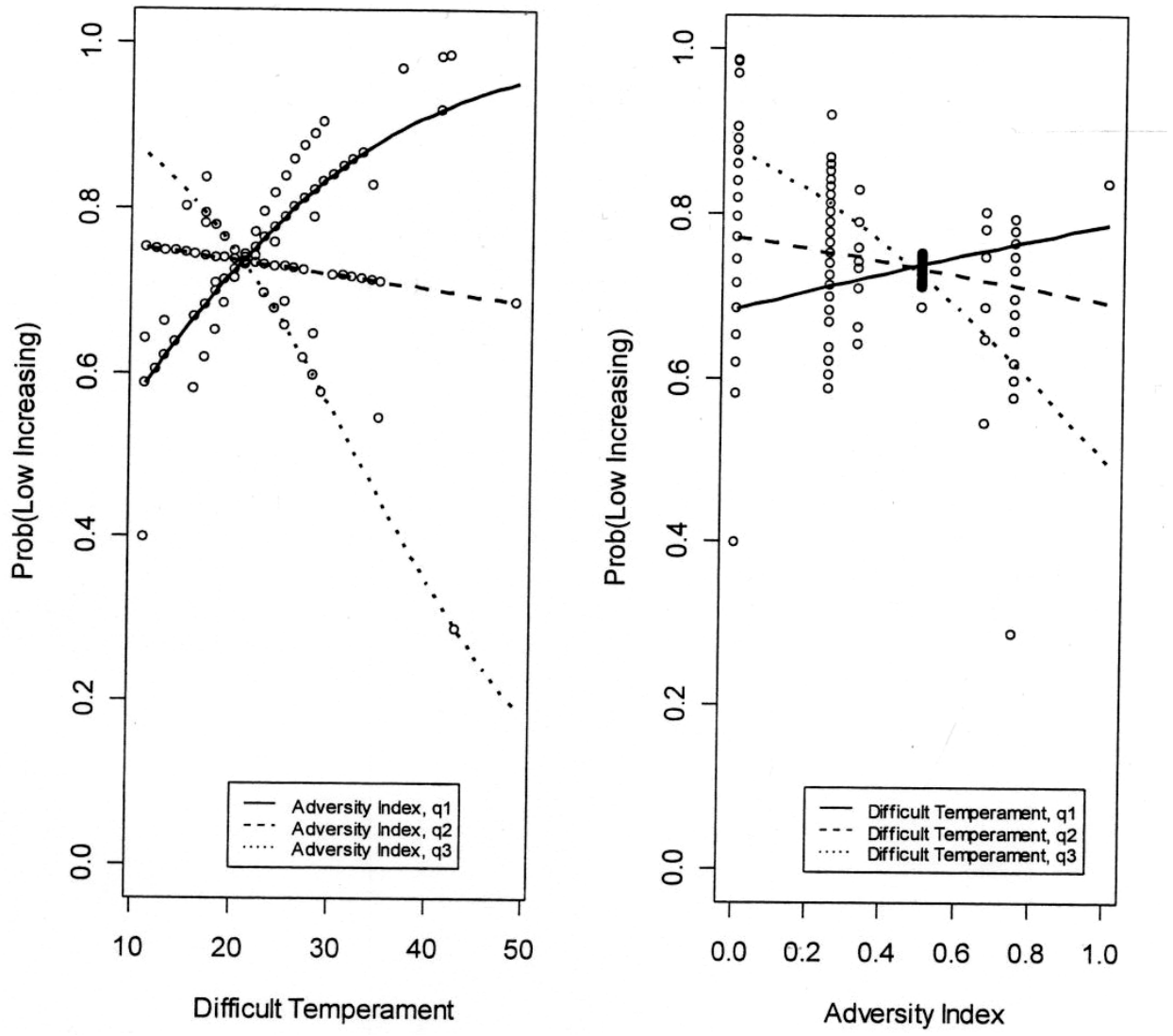

Figure 5. The effects of difficult temperament and the adversity index predicting membership to the Low Increasing SOM group compared to No SOM group.

Figure 6 shows the effects of difficult temperament and interparental conflict predicting membership in the Low Increasing group compared to the No group. Similar results were obtained for the temperament x adversity interaction, as the potentiation effect occurred only when the second variable was at low or mean levels, rather than at high levels. Specifically, when difficult temperament was in the low or average range ($x \leq 23$) and interparental conflict was high, the probability of being in the Low Increasing group increased up to .95.

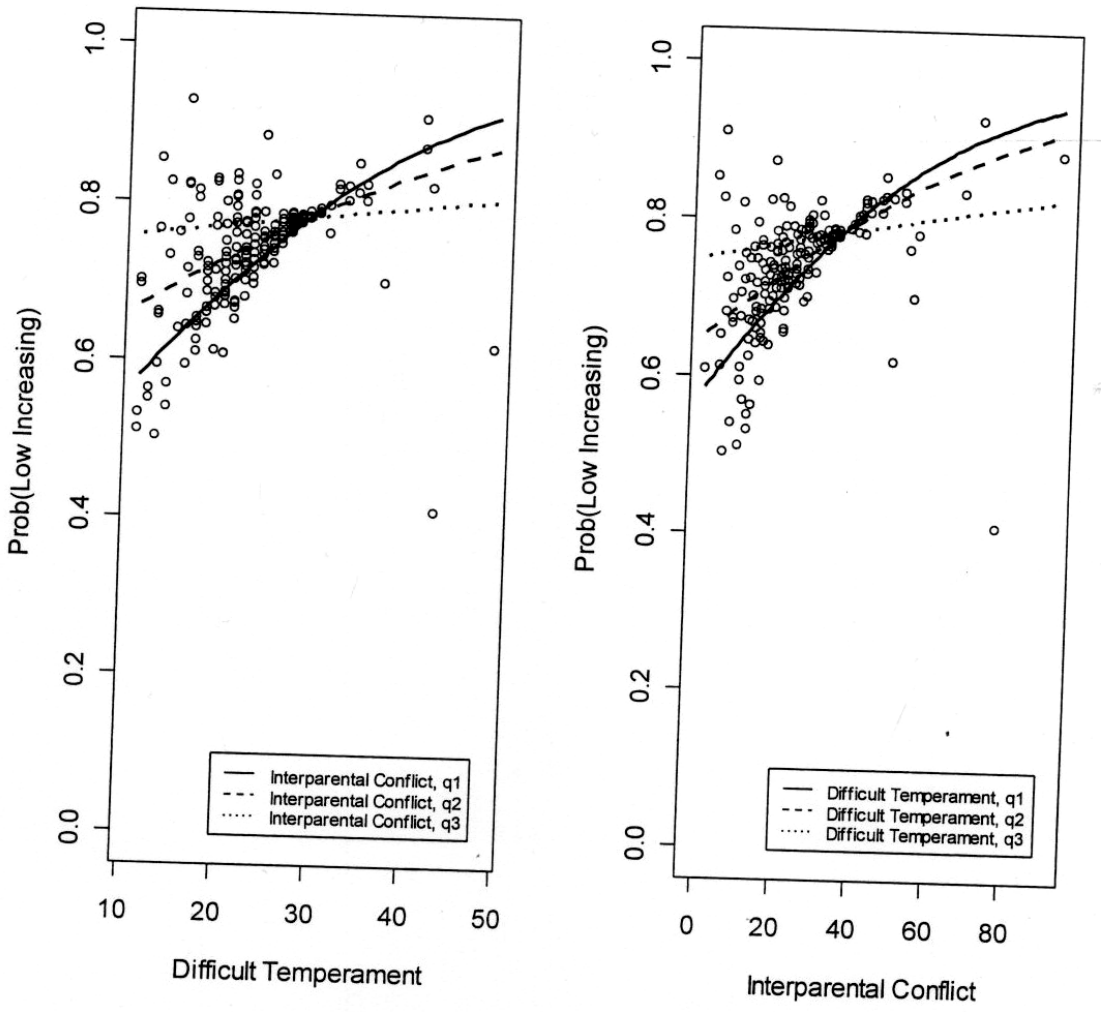

Figure 6: The effects of difficult temperament and interparental conflict predicting membership to the Low Increasing SOM group compared to No SOM group.

Figure 7 shows the effects of child negative emotionality (NE) and parent-child conflict predicting the probability of being in the Low Increasing group versus the No group. Results supported the hypothesis that boys with high levels of both child (i.e., child NE) and proximal environmental (i.e., acrimonious parent-child relationship) risk were more likely to be in the Low Increasing group than the No group.

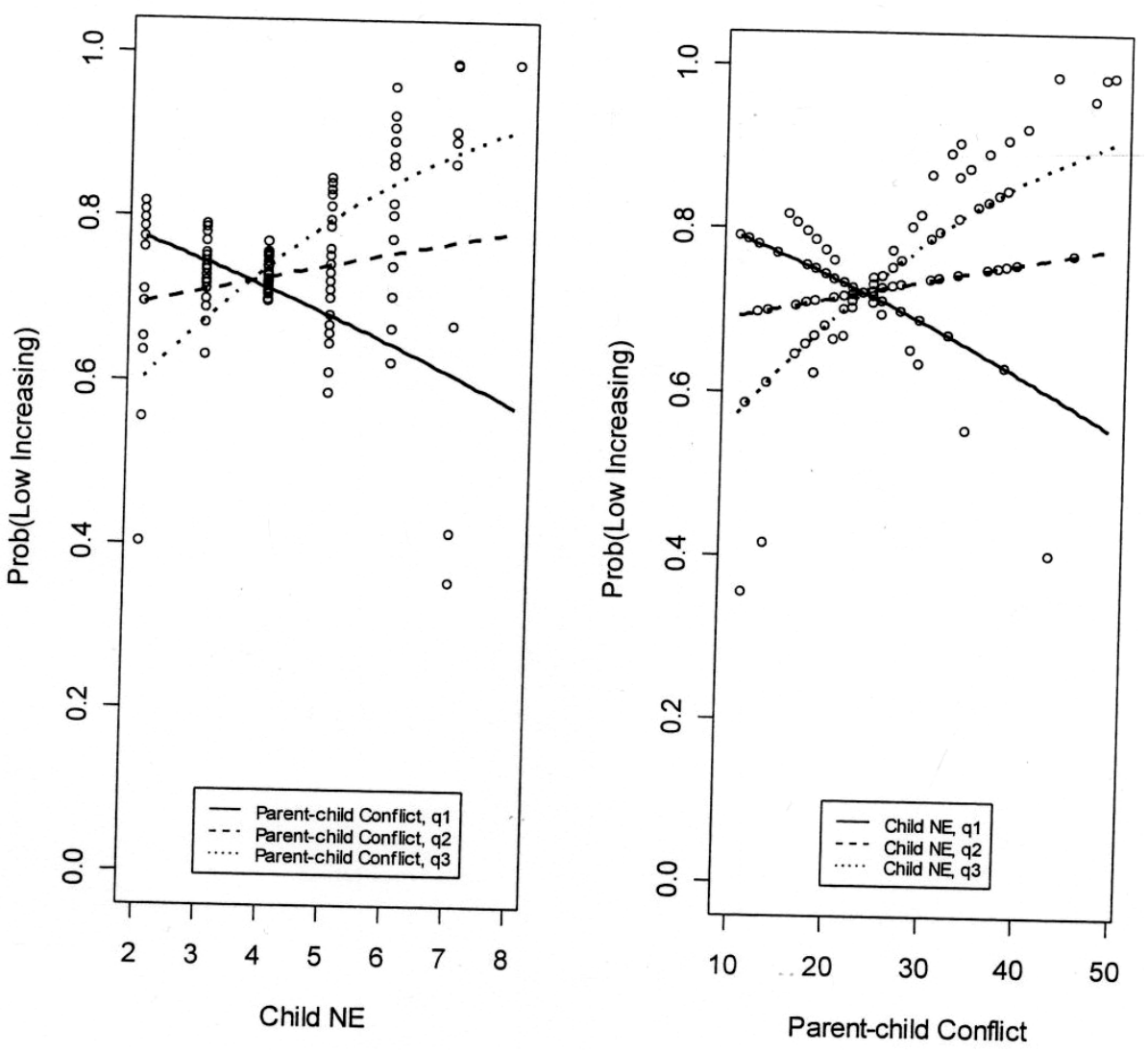

Figure 7: The effects of child negative emotionality (NE) and parent-child conflict predicting the probability of being in the Low Increasing group versus the No SOM group.

Figure 8 shows the effects of child NE and parent-child conflict predicting membership to the Moderate Increasing group compared to the Low Increasing group. As with the first two cases described above, the effects for this interaction were more pronounced in the low range. That is, only when one variable was at a high level, and the other low, did the probability increase. However, the effects may be more salient pertaining to parent-child conflict: when negative emotionality was held constant, parent-child conflict appeared to increase the probability of membership in the Moderate Increasing group as boys' scores demonstrated greater risk. It is also important to note that for this interaction, most of the probabilities for being in the Moderate Increasing group were below the .50 level.

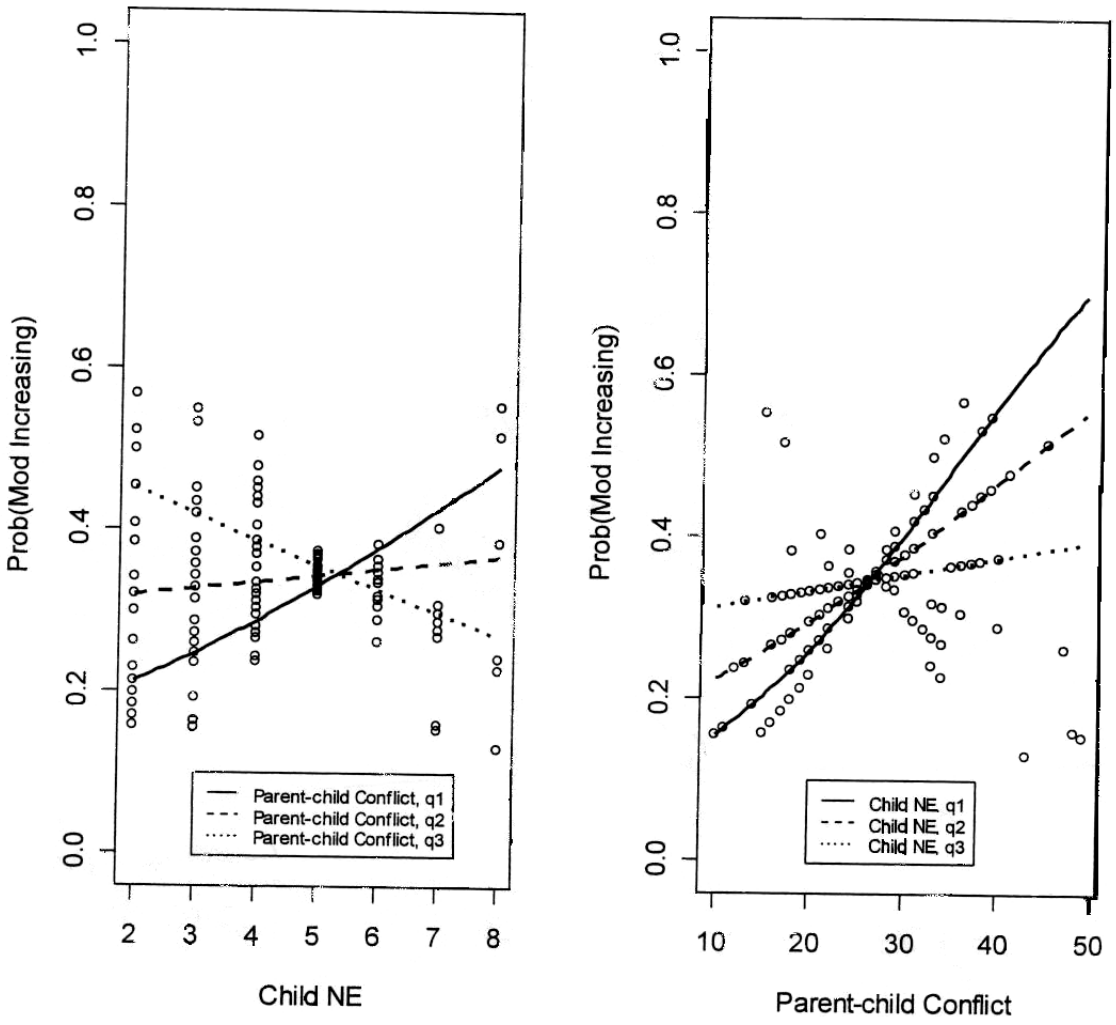

Figure 8: The effects of child negative emotionality (NE) and parent-child conflict predicting membership to the Moderate Increasing SOM group versus the Low Increasing SOM group.

5.5 TRAJECTORY GROUP STATUS AND LATER CHILD MALADJUSTMENT AND FUNCTIONAL IMPAIRMENT

To examine the hypothesis that SOM trajectory group membership would be related to higher levels of child maladjustment and functional impairment in early adolescence, a series of ANOVAs were computed to determine group differences on the child outcome variables listed in Table 1. Average scores on child outcomes by trajectory group membership are presented in Table 12. Results demonstrate that higher levels of teacher-reported SOM at ages 11 and 12 were significantly associated with the MTH group compared to both the Low Increasing and No groups (*x*s = 2.5 vs. .55 and .59, respectively, $p < .05$). A nonsignificant trend was found for functional impairment, or days absent from school ($p < .10$). The number of days absent from school increased in the expected direction, such that the Moderate Increasing group demonstrated more days missed from school than the Low Increasing and No group (*x*s = 18.78 vs. 14.13 and 13.33, respectively). No other differences among outcome variables were found to differentiate group membership.

5.6 CONTROLLING FOR CHILD'S RACE AND FAMILY SOCIOECONOMIC STATUS (SES)

Finally, follow-up ANCOVAs were conducted controlling for child's race and family SES at age 5 for analyses demonstrating significant differences between trajectory groups.[3] These covariates did not influence the results of the univariate or multivariate analyses reported in Tables 8 and 9, respectively. However, a few of the significant multivariate results presented

[3] Note: Child's race and family SES at age 5 were modestly correlated ($r = .14$, $p < .05$).

in Tables 10 and 11a were affected. For instance, referring to Table 10, child's race had a slight reduction of effect on the importance of parent-child conflict (Wald statistic changed from 2.73 to 2.60, $p <$. 11 for the latter) and maternal depressive symptoms (reducing Wald statistic from 2.85 to 2.67, $p < .11$ for the latter), when the Moderate Increasing was compared to the Low Increasing group. For both risk factors, however, the odds ratios did not change. When controlling for family SES in the regressions comparing the Moderate Increasing to the Low Increasing group, parent-child conflict was no longer a trend ($B = .31$, $SE = .02$, Wald = 1.95, OR = 1.03, $p = .16$); however, maternal depression remained a nonsignificant trend ($B = .05$, $SE = .03$, Wald = 3.12, OR = 1.06, $p < .08$). Referring to Table 11a, the addition of SES to the equation for temperament x interparental conflict reduced the interaction from significance to a trend ($B = -.003$, $SE = .001$, Wald = 3.21, OR = .99, $p = .07$).

6.0 DISCUSSION

By applying a developmental psychopathology framework and a semi-parametric mixture modeling (SPMM) technique to a longitudinal dataset of low-income boys, the present study provided novel data on developmental trajectories of SOM from middle childhood to early adolescence. The current study has also begun to fill significant holes in our understanding of the stability, prevalence, risk factors, and outcomes of SOM.

6.1 PREVALENCE AND COURSE OF SOM

Mothers' ratings of SOM were modestly stable and consistent across time. This finding is congruent with other studies investigating the stability of SOM in community samples of children (Aro et al., 1987; Dhossche et al., 2001; Rauste-von Wright & von Wright, 1981). Although previous studies have suggested that SOM is more stable in girls, the present study found a similar pattern for boys aged 6-12. As described in the Results section, the current findings are also consistent with prior research on the prevalence of individual somatic complaints. As in previous studies, the most common symptoms were *headaches*, *stomachaches or cramps* (RAP), and *aches or pains* (Campo & Fritsch, 1994; Garralda, 1996); whereas, the symptom *overtired*, or fatigue, was slightly less than what has been reported in the literature (9-14% vs. 15%). Unlike other studies, the symptoms *rashes and other skin problems* were also

frequently endorsed (Campo & Fritsch, 1994). The one *CBCL* item that can be described akin to a "pseudoneurological" symptom, '*feels dizzy'*, had an extremely low incidence at ages 6 and 8, which is also congruent with the existing literature (Campo & Fritsch, 1994). In the current study, 37% - 58% of boys experienced at least one somatic symptom across childhood, which is similar to other research on community samples of schoolchildren (e.g., Alfven, 1993). The results of the current study also support epidemiological findings illustrating that the prevalence of SOM increases with age (Berntsson & Koehler, 2001; Fearon & Hotopf, 2001), especially for polysymptomatic presentation (Campo et al., 1999; Egger et al., 1998). This study extends the current literature by examining multiple somatic complaints (as opposed to one symptom, such as headache; Fearon & Hotopf, 2001) and by excluding symptoms that may be more indicative of depression than somatization (e.g., loss of appetite; Berntsson & Koehler, 2001).

6.2 DELINEATING DISTINCT PATHWAYS OF SOM

The present findings supported the first hypothesis, and the existing literature, that three trajectories of SOM would emerge and that SOM would increase over time (Cote et al., 2002; Gilliom & Shaw, 2004; Rauste-von Wright & von Wright, 1981). Specifically, this study found three distinct pathways of SOM: one that demonstrated no somatic complaints across time (No group = 17% of the sample), a second presenting a low score of SOM initially that gradually increased over time (Low Increasing group = 54%), and a third that followed the same pattern as the second pathway, but showed higher mean levels at the outset and increased at a greater rate over time (Moderate Increasing = 28%). This finding also supports Hypothesis 1, in that, 28% of the entire sample (compared to the predicted 30%) was classified in this top group. The results

of the study suggest that in this urban high-risk sample it was more common than not for boys to experience some, albeit modest, somatic symptoms from ages 6-12. In fact, boys who were reported as experiencing no somatizing across childhood comprised the least prevalent TRAJ group. The results suggest that somatization increases for the majority of boys throughout childhood. That is, for most boys (i.e., 82%), there was an increase of somatic symptoms from ages 6 to 12. However, it is important to point out that rates of SOM for all three groups tended to be modest based on the number of items included on the *CBCL* Somatic Complaints factor (*N* = 9, with a total possible score of 18), as means ranged from only 1.81 to 2.78 for the Moderate Increasing group.

Apart from these three groups, a fourth group, not identified by TRAJ, was comprised of five boys (Moderate to High group = 2% of the sample). This group demonstrated a similar starting point as the Moderate Increasing group, but SOM scores increased to a very high level at age 11 (i.e., *x*s = 8.40 vs. 2.78). This small percentage of boys may represent a more clinically significant group and therefore was included in many of the analyses despite its small size. However, as this Moderate to High (MTH) group was extremely small, it was challenging to detect between-group differences on risk factors or later child outcomes.

Using the same longitudinal sample of boys as the current study, Gilliom and Shaw (2004) studied broadly-defined internalizing problems (no somatic symptoms were included) from ages 2 to 11 and found three trajectories: a persistent low group (comprising 77% of the sample), an increasing group (20%), and a persistent high group (3%). The percentages of boys in each of the three groups were quite similar across studies: both had a group with no or few symptoms (17% (No) + 54% (Low Increasing) = 71% vs. 77%), a middle group with symptoms increasing over time (28% vs. 20%), and a small group demonstrating a higher level of

symptoms over time (2% vs. 3%). Cote and colleagues (2002) investigated fearful and anxious behaviors in 930 Canadian boys aged 6 to 12 and found three relatively stable trajectories, namely low (16%), moderate (75%), and high (9%) groups. Again, these percentages were close to those in the present study and the Gilliom and Shaw study. There were some similarities between the current study and the Cote study. For instance, boys in both samples were the same ages and similar measures were used (*Social Behaviour Questionnaire* in the Cote study). In Cote's study, however, both urban and rural children in Quebec were included, less than 10% were living with a single mother (compared to 30% in the PMCP), and trajectories were based on teacher report at seven time points, as opposed to maternal report at only five time points. Nonetheless, this lends some evidence that pathways and incidence of SOM are quite similar to those of more broadly-defined patterns of internalizing symptoms (e.g., worrying, anxiety, and depression). This assertion is especially persuasive given that this finding holds for multiple reporters (mothers vs. teachers), SES levels, and countries (the United States and Canada).

6.3 RISK FACTORS AND TRAJECTORIES OF SOM

The second major objective of the study was to examine risk factors in early childhood (i.e., ages 2 - 6) that might differentiate membership in SOM trajectory groups. The research on pediatric, psychiatric, and community samples suggests that a number of risk factors from multiple domains contribute to or are associated with the development of SOM in childhood and adolescence (e.g., Aro et al., 1987; Campo & Fritsch, 1994; Walker et al., 1994). Hence, looking across disciplines, cross-sectional and longitudinal research supports a developmental psychopathology framework in studying the development of SOM. The present study is the first

of its kind to look at risk from five different domains using multiple informants. Moreover, these risk factors were assessed in early childhood, at or before the measurement of SOM trajectories.

At the level of individual risk factors, the results corroborated previous research that has demonstrated associations between SOM and risk in the parent-child relationship (Aro et al., 1987; 1989; Rauste-von Wright & von Wright, 1981), family (Campo & Fritsch, 1994; Gulhati & Minty, 1998), and school (Walker et al., 1994; 2001; 2002) domains. Specifically, when examined in a univariate framework, acrimonious parent-child relationships and maternal depressive symptoms discriminated membership in the Moderate Increasing group compared to the No and Low Increasing groups, and lower social skills discriminated MTH group from all other SOM groups. However, when all risk factors were entered into the same logit regression, only maternal depressive symptoms significantly predicted membership in the Moderate Increasing group compared to the No group. Another set of follow-up regressions were then computed with only the risk factors identified through the univariate analyses due to high multicollinearity among variables. Within this model, only acrimonious parent-child relationships continued to be a significant discriminator of boys in the Moderate Increasing group from the No group.

These findings indicate that both conflictual parent-child relationships and maternal depressive symptoms can predict, under certain circumstances, boys who will demonstrate moderate levels of SOM versus boys who will not demonstrate any SOM across childhood. For instance, a mother who shows moderate levels of depressive symptoms is more likely to be withdrawn, irritable, and/or unavailable to her son. This type of maternal behavior could be a stressor for the son, especially when he is making the transition from spending more time at home to greater time at school. Parent-child relationships characterized by high levels of

conflict and low levels of synchrony are also likely to be a stressor for a young boy. Thus, both variables, parent-child conflict and maternal depressive symptomatology, can be viewed as increasing the stress within the child's most proximal context (i.e., the primary caregiver). This unharmonious relationship between mother and son, accompanied by increasing levels of SOM, may set the standard for the child's future relationships to be maladaptive, which then may lead to problems during the school-age years. The data, in fact, showed some support for highly somatizing boys to demonstrate lower social skills and more school problems at ages 6 and 7 than boys demonstrating lower levels of SOM across childhood.

In line with previous studies (Aromaa et al., 2000; Walker et al., 1994; Walker et al., 2001) and the DPP, four biosocial interactions predicted SOM group membership, where child risk factors (i.e., difficult temperament and child NE) represented the biological component and proximal environmental stressors (i.e., parent-child conflict, interparental conflict, and sociodemographic adversity) represented the social component of the model. However, in only one instance, child NE x parent-child conflict, were the effects of the variables associated with group membership in the expected direction. Specifically, when high levels of both child NE and parent-child conflict were present, the probability of being in the Low Increasing group compared to the No group increased. It should be noted that no interactions were found between the Moderate Increasing and No groups. Hence, concerning interactive effects, there were only modest differences among SOM groups with subclinical levels of symptoms.

6.4 CHILD MALADJUSTMENT AND TRAJECTORIES OF SOM

The final objective of the study was to determine associations between SOM groups and later maladjustment in early adolescence. Outcome variables were selected to include multiple facets of the child's emotional (e.g., anxiety and depression) and behavioral (e.g., externalizing and school absence) experiences using multiple informants, including school records, teacher, and youth reports. Maternal-reported trajectories were discriminated by teacher reports of SOM at age 12 in the expected direction. Overall, the findings of co-occurring maladjustment in the present study did not replicate existing literature showing that children with higher levels of SOM have higher levels of psychological problems in early adolescence (Bernstein et al., 1997; Dhossche et al., 2001; Garber et al., 1990; Last, 1991; Livingston et al., 1988; McCauley et al., 1991; Walker & Greene, 1989). Boys reported as having higher levels of SOM did not appear to be at greater risk for emotional or behavioral problems. That is, significant between-group differences were not found for either teacher- or youth self-report externalizing and internalizing behaviors. However, MTH group showed the highest means on teacher-reported outcomes, but as mentioned earlier, significant effects were difficult to detect due to the small group size (n = 5). The reverse pattern was found for youth-reported outcomes, such that the MTH group showed lower, or equivalent, mean scores compared to the No group. This is not surprising because boys in the present sample did not endorse many internalizing symptoms, especially depression (i.e., on average boys reported less than one symptom on the *CDI).*

The results from the present study did not support the finding that a relationship exists between SOM and co-occurring internalizing and/or externalizing. Prior studies using youth self report have found significant correlations between SOM and anxiety (rs = .30 - .42) and depression (r = .37, Garber et al., 1991; Rauste-von Wright & von Wright, 1981), but no

significant correlations were found in the present study between youth reports of internalizing or externalizing and maternal- or teacher-reported SOM. However, the studies mentioned above used only one reporter, the child, while the current study used multiple informants.

6.5 CONCLUSIONS AND CLINICAL IMPLICATIONS

The present study has sought to expand the way SOM has been conceptualized and studied with the intention to inform the development of model-driven intervention approaches. Three questions were posed at the outset of the study, namely: "What are the developmental patterns of SOM for boys across childhood?," "What risk factors are important in the development and maintenance of SOM?," and "What is the relation between persistent patterns of SOM and other co-occurring problems, such as anxiety and depression, in school-age boys?"

SOM displayed similar developmental patterns to those of other broadly-defined internalizing behaviors, but these trajectories were not successful in identifying groups of boys at-risk for later maladjustment. However, maternal-reported trajectories of SOM were validated by teacher-reports of SOM at ages 11 and 12. Results pertaining to risk factors of SOM partially support a DPP model that starts with a difficult or sensitive child who may be more likely to use negative affect and somatic complaints to signal caregivers to help cope with distress. In a home environment characterized by conflict either between the parent and child or both parents, the child may begin to internalize feelings and express somatic problems more frequently. Somatic behaviors might also emerge in the presence of maternal depression. Before any definitive conclusions can be drawn, however, these results must be replicated by other research studies

with larger community samples of boys who demonstrate higher levels of SOM (i.e., levels similar to those found in the MTH group in the present study).

6.6 FUTURE DIRECTIONS

There is still much to be learned in the area of childhood somatization. The notion that SOM is related to later psychopathology was not supported by the current research. Larger longitudinal samples are needed to determine whether a MTH group exists and whether this is a "clinically" at-risk group. For the current sample, adding more time points by extending the study into adolescence may help clarify whether there is a fourth pathway of SOM. Moreover, examining children's diagnoses and/or medical and psychotropic medications may also add insight into the ambiguous relationship between SOM and internalizing/externalizing.

It is also possible that we as researchers are not yet adept at selecting appropriate predictors of child and adolescent somatization. We may need to broaden our lens by utilizing biological variables, such as physiological reactivity or pubertal timing/ development. For example, more physiologically-based studies are needed to confirm a hyper-responsive reaction to stress in children and adolescents with SOM. In addition, longitudinal samples charting pubertal development of boys and girls may clarify gender differences seen in the co-occurrence of childhood SOM and psychopathology during adolescence.

6.7 LIMITATIONS

The sample chosen for the current study had some inherent limitations for studying the development of childhood SOM. First, the PMCP is a sample comprised of all boys. Therefore, the results of this study may not be applicable to girls, particularly those regarding co-occurring internalizing problems during early adolescence when sex differences in depression emerge. Further, the majority of the families in the sample are from a low socioeconomic background, including those in poverty, representing a constrained range of SES. For example, the mean annual income at the initial assessment for PMCP families was $12,553 and per capita income was approximately $2,900 per family member. Due to the constrained range of SES, we would expect SES to play a larger role in discriminating patterns of SOM in samples that included greater variability on this factor. Also, there are likely limits in the generalizability of the findings to nonurban, less socioeconomically deprived populations.

The PMCP was designed to study the development of antisocial behaviors in boys, not SOM; therefore, there were limitations with the measurement of certain constructs. First, as somatic complaints were assessed solely by a questionnaire and only from one informant, it was not possible to establish whether the somatic complaints had a medical origin. However, as stated above, the *CBCL* and other comparable measures (e.g., *Somatic Complaints Inventory*; Walker, et al., 1991) have been frequently used in the field of psychology to assess this construct. In the pediatric literature, as few as one question pertaining to one symptom at one time point has been used to identify "somatizers" (Fearon & Hotopf, 2001). In this light, the design and measurement of the current study, despite its limitations, actually represents a methodological advance in the field compared to the bulk of research in this area. Second, it would have been optimal to have an observational measure of acrimonious parent-child

relationships or rejecting parenting. Finally, part of the DPP is to investigate biological factors in addition to environmental factors, inasmuch as they can be separated. Measures of pubertal and physiological constructs were not included due to the scope of the present project but are nonetheless worthy areas of study. Despite these limitations, the current research is the only study of its kind, investigating risk factors across multiple domains in longitudinal sample of boys.

APPENDIX

TABLES

Table 1: Independent (Predictor) Variables

Type of Variable	Age	Reporter	Construct	Instrument	Factor
<u>Risk Factors</u>					
Child Risk	2	Mother	Difficult Temperament	*ICQ*	
	5	Observer	Child Negative Emotionality	Interviewer impressions, SCCS global ratings	Negative affectivity + Negative reactivity
Parent-Child Relationship Risk	5	Mother	Parent-Child Conflict	*ACRS*	Conflict Factor
Family Risk	3.5, 5, 6*	Mother	Maternal Depressive Symptomatology	*BDI*	
	3.5, 6*	Mother	Interparental Conflict	*CTS*	Verbal + Physical Aggression Factors
Socio-demographic Adversity†	3.5, 5, 6*	Mother	Low SES, low maternal education, single parent	Demographic Interview	
	5	Mother	Neighborhood Dangerousness	*NQ*	
School Risk	6, 7*	Teacher	Social Skills	*SSRS*	Social Skills domain: assertion, cooperation, responsibility, & self-control
	6, 7*	Teacher	Academic Incompetence	*TRF*	Composite of item numbers: 49, 61, & 92

* Scores from all time points were composited (i.e., summed and then averaged).

† A *cumulative adversity index* was comprised of low SES, low maternal education, single parent status, and neighborhood dangerousness.

Table 1, con't
Predictor Variables: Interaction Terms

Interactions

1. Difficult Temperament x Parent-Child Conflict
2. Difficult Temperament x Maternal Depression
3. Difficult Temperament x Interparental Conflict
4. Difficult Temperament x Sociodemographic Adversity
5. Child Negative Emotionality x Parent-Child Conflict
6. Child Negative Emotionality x Maternal Depression
7. Child Negative Emotionality x Interparental Conflict
8. Child Negative Emotionality x Sociodemographic Adversity

9. Social Skills x Parent-Child Conflict
10. Social Skills x Maternal Depression
11. Social Skills x Interparental Conflict
12. Social Skills x Sociodemographic Adversity
13. Academic Incompetence x Parent-Child Conflict
14. Academic Incompetence x Maternal Depression
15. Academic Incompetence x Interparental Conflict
16. Academic Incompetence x Sociodemographic Adversity

Table 2: Child Adjustment Variables for Hypotheses 1 and 3

Type of Variable *(Source)*	Age	Construct	Instrument	Factor
Trajectory Group *Maternal Report*	6, 8, 10, 11, 12*	Somatic Complaints (SOM)	*CBCL*	Somatic Complaints subscale
Outcome Variables				
Teacher Report	11, 12**	Depression and Anxiety	*TRF*	Composite of Anxious/Depressed and Withdrawn subscales
	11, 12**	Externalizing	*TRF*	Externalizing broadband
	11, 12**	Somatization	*TRF*	Somatic Complaints subscale
School Records	10,11, 12***	Functional Impairment/ School absenteeism	School Records	Number of days absent during the school year
Boys' Self-Report	12	Depression	*CDI*	
	12	Anxiety	*MASC*	
	12	Antisocial Behavior	*SRD*	10-item short form 8-item short form (somatic items removed)

* Scores from each age will be used individually.
** Scores from all time points will be composited (i.e., summed and then averaged).
*** Days absents were taken from ages 11 and 12. However, if one of those data points were missing, absences from age 10 were used. Two time points, then, were summed and averaged.

Table 3: Descriptive Statistics for Somatic Complaints

Table 3a: Descriptive Statistics for Individual Items and Total Score on the CBCL Somatic Complaints Subscale Across Ages

Somatic Complaint Item (*CBCL* item no.)		**Age 6** (n = 262)	**Age 8** (n = 247)	**Age 10** (n = 226)	**Age 11** (n = 235)	**Age 12** (n = 222)
Feels dizzy	Mean	0	.02	.04	.02	.04
(Item 51)	*SD*	.062	.167	.218	.144	.227
Overtired	Mean	.11	.10	.11	.12	.16
(Item 54)	*SD*	.322	.340	.350	.337	.399
Medically Unexplained Physical Problems:						
Aches or pains	Mean	.09	.11	.16	.16	.13
(Item 56a)	*SD*	.303	.387	.400	.404	.348
Headaches	Mean	.08	.15	.26	.27	.26
(Item 56b)	*SD*	.292	.411	.487	.479	.467
Nausea, feels sick	Mean	.08	.06	.11	.14	.07
(Item 56c)	*SD*	.291	.264	.339	.371	.267
Problems with eyes	Mean	.08	.08	.12	.18	.15
(Item 56d)	*SD*	.353	.335	.404	.503	.444
Rashes or other skin problems	Mean	.16	.15	.15	.21	.16
(Item 56e)	*SD*	.453	.446	.427	.493	.453
Stomachaches or cramps	Mean	.13	.14	.16	.18	.13
(Item 56f)	*SD*	.374	.400	.379	.420	.373
Vomiting	Mean	.05	.04	.07	.07	.04
(Item 56g)	*SD*	.210	.217	.283	.251	.208
Total	Mean	**.75**	**.83**	**1.11**	**1.34**	**1.11**
(Sum of items)	*SD*	1.26	1.52	1.59	1.87	1.70

Table 3b: Prevalence of Individual Somatic Complaints on the CBCL *Across Ages*

Individual Complaints by Frequency	**Age 6**	**Age 8**	**Age 10**	**Age 11**	**Age 12**
Feels dizzy					
Somewhat/sometimes true = 1	0.4 (1)	1.2 (3)	3.1 (7)	2.1 (5)	3.5 (8)
Often/very true = 2	0 (0)	0.4 (1)	0.4 (1)	0 (0)	0.4 (1)
Overtired					
Somewhat/sometimes true	10 (26)	7.7 (19)	8 (18)	11 (26)	12.9 (29)
Often/very true	0.4 (1)	1.2 (3)	1.3 (3)	0.4 (1)	1.3 (3)
Aches or pains					
Somewhat/sometimes true	8.4 (22)	6.5 (16)	13 (29)	13.9 (33)	11.9 (27)
Often/very True	0.4 (1)	2.4 (6)	1.3 (3)	1.3 (3)	0.4 (1)
Headaches					
Somewhat/sometimes true	7.7 (20)	10.9 (27)	21.4 (48)	23.2 (55)	23 (52)
Often/very true	0.4 (1)	2 (5)	2.2 (5)	1.7 (4)	1.3 (3)
Nausea, feels sick					
Somewhat/sometimes true	7.6 (20)	4 (10)	9 (20)	12.2 (29)	5.8 (13)
Often/very true	0.4 (1)	0.8 (2)	0.9 (2)	0.8 (2)	0.3 (1)
Eyes, problems with					
Somewhat/sometimes true	3.9 (10)	3.6 (9)	6.8 (15)	8.1 (19)	7.6 (17)
Often/very true	2.3 (6)	2 (5)	2.7 (6)	5.1 (12)	3.6 (8)
Rashes or other skin problems					
Somewhat/sometimes true	9.2 (24)	7.3 (18)	9.8 (22)	13.4 (32)	8.8 (20)
Often/very true	3.4 (9)	3.7 (9)	2.7 (6)	3.8 (9)	3.5 (8)
Stomachaches or cramps					
Somewhat/sometimes true	11.1 (29)	9.7 (24)	15 (34)	16 (38)	10.2 (23)
Often/very true	1.1 (3)	2 (5)	0.4 (1)	1.3 (3)	1.3 (3)
Vomiting					
Somewhat/sometimes true	0 (0)	3.2 (8)	4.9 (11)	6.7 (16)	2.7 (6)
Often/very true	0 (0)	0.4 (1)	0.9 (2)	0 (0)	0.4 (1)
At Least One Symptom Present	37 (96)	41 (100)	48 (108)	52 (122)	47 (104)
Score of 2 or Higher Across Symptoms	22 (57)	22 (55)	30 (67)	35 (82)	28 (62)
Score of 4 or Higher Across Symptoms	6 (15)	5 (12)	10 (22)	11 (25)	8 (17)
Score of 5 or Higher Across Symptoms	3.4 (9)	3.2 (8)	5.3 (12)	6 (14)	6 (13)

Note: Numbers listed are percentages. The numbers in parentheses are the number of boys constituting that percentage.

Table 3c:
Correlations of Somatic Complaints across Ages and with Trajectory Group Assignment

CBCL Somatic Complaints		Age 6	Age 8	Age 10	Age 11	Age 12	Traj Grp
Age 6	Pearson *r*	1	.456**	.368**	.322*	.293**	.556**
	N	262	229	209	220	203	260
Age 8	Pearson *r*		1	.413**	.414**	.425**	.565**
	N		247	208	216	207	245
Age 10	Pearson *r*			1	.483**	.425**	.613**
	N			226	200	191	224
Age 11	Pearson *r*				1	.575**	.700**
	N				235	198	233
Age 12	Pearson *r*					1	.672**
	N					222	220
Trajectory Group	Pearson *r*						1
	N						307

Note: All comparisons are two-tailed.
* $p < .05$, ** $p < .01$

Table 4: Descriptive Statistics for Risk Factors

Table 4a: Means and Standard Deviations for Risk Factors

Risk Factors (Questionnaire)	Child's Age(s)	Reporter	Mean	Standard deviation	Range	Sample size
Difficult and Irritable Temperament (ICQ)	2	Maternal	23.03	6.14	11 – 49	292
Child Negative Emotionality	5	Observed	4.08	1.54	2 – 8	212
Parent-Child Conflict (ACRS)	5	Maternal	25.18	7.96	10 – 49	279
Maternal Depressive Symptomatology (BDI)	3.5, 5, 6	Maternal	7.38	5.77	0 – 30	300
Interparental Conflict (CTS)	3.5, 6	Maternal	24.23	13.12	0 – 92	293
Sociodemographic Adversity Index	3.5, 5, 6	Maternal	.3408	.2495	0 – 1	302
Social Skills (SSRS)	6, 7	Teacher	37.23	10.28	3 – 55	232
Academic Incompetence (TRF)	6, 7	Teacher	1.76	1.93	0 – 6	233

Table 4b: *Pearson Correlations Among Risk Factors*

Predictors		ICQ	Child NE	ACRS	BDI	CTS	Index	SSRS	TRF
Difficult Temperament (ICQ)	Pearson *r*	1	.043	.291**	.126*	.174**	.022	-.026	.042
	N	292	201	264	283	277	285	221	222
Child Negative Emotionality (NE)	Pearson *r*		1	.214**	.105	.012	.144*	-.036	.056
	N		212	212	211	209	212	167	167
Parent-Child Conflict (ACRS)	Pearson *r*			1	.344**	.223**	.228**	-.272**	.203**
	N			279	278	271	279	222	223
Maternal Depressive Symptoms (BDI)	Pearson *r*				1	.333**	.153**	-.221**	.157*
	N				300	293	300	232	233
Interparental Conflict (CTS)	Pearson *r*					1	.004	-.107	.086
	N					293	293	228	228
Adversity Index	Pearson *r*						1	-.169**	.221**
	N						302	232	233
Social Skills (SSRS)	Pearson *r*							1	-.607**
	N							232	232
Academic Incompetence (TRF)	Pearson *r*								1
	N								233

Note: All comparisons are two-tailed.
* $p < .05$, ** $p < .01$

Table 4c: *Correlations Among Predictor Variables and Maternal-Report Somatization (SOM)*

Predictors		Age 6 SOM	Age 8 SOM	Age 10 SOM	Age 11 SOM	Age 12 SOM
Difficult Temperament	Pearson *r*	.02	.06	.05	.03	-.07
	N	250	235	219	222	208
Child Negative Emotionality	Pearson *r*	.05	-.01	-.09	.03	.00
	N	189	178	165	171	164
Parent-Child Conflict	Pearson *r*	.11	.13	.04	.11	.08
	N	250	230	212	222	211
Maternal Depressive Symptoms	Pearson *r*	.15*	.16*	.12	.14*	.10
	N	262	245	224	300	220
Interparental Conflict	Pearson *r*	.09	.10	.09	.09	.09
	N	259	242	221	231	216
Adversity Index	Pearson *r*	-.01	.01	.04	.12	.00
	N	262	245	224	234	220
Social Skills	Pearson *r*	.03	-.14*	-.16*	-.15*	-.08
	N	217	204	185	198	190
Academic Incompetence	Pearson *r*	-.05	.09	.09	.20**	.05
	N	217	204	185	198	190

Note: All comparisons are two-tailed.
* $p < .05$, ** $p < .01$

Table 5: Descriptive Statistics for Outcome Variables

Table 5a: Means and Standard Deviations for Outcome Variables

Outcome Variables by Reporter and Age	Mean	Standard deviation	Range	Sample size
Teacher Report – Ages 11 & 12				
TRF Externalizing broadband factor*	25.91	20.07	0 – 91	207
TRF Withdrawn + Anxious/Depressed* syndromes	5.22	5.27	0 – 26	206
TRF Somatic Complaints subscale*	.6990	1.38	0 – 10	206
School Records – Ages 10-12				
Functional Impairment, school absenteetism (e.g., days absent)	15.33	10.57	0 – 52	130
Youth Self Report – Age 12	.9646	1.40	0 – 6	226
Child Depression Inventory (CDI)	8.06	4.15	0 – 19	224
Multidimensional Anxiety Scale for Children (MASC)	3.60	4.01	0 – 26	226
Self-Report of Delinquency (SRD)				

* Raw scores were used.

Table 5b: *Pearson Correlations Among Outcome Variables by Reporter*

		Teacher Report			**School**	**Youth-Self Report**		
Teacher Report *(TRF;* Ages 11, 12)		EXT	Anx/Dep	SOM	Absent	CDI	MASC	SRD
Externalizing	Pearson *r*	1	.524**	.285**	.293**	.204**	-.067	.372**
	N	206	206	205	94	178	177	177
Anxious/ depressed	Pearson *r*		1	.459**	.162	.322**	.089	.125
	N		207	206	94	179	178	178
Somatic complaints	Pearson *r*			1	.229*	.107	-.002	.032
	N			206	93	179	178	178
School Records (Ages 10-12)								
Absenteeism	Pearson *r*				1	.044	.076	.324**
	N					96	96	96
Youth Report (Age 12)								
Depression symptoms (CDI)	Pearson *r*					1	.214**	.197**
	N					226	224	225
Anxiety symptoms(MASC)	Pearson *r*						1	.011
	N						224	223
Delinquency (SRD)	Pearson *r*							1
	N							226

Note: All comparisons are two-tailed.
* $p < .05$, ** $p < .01$

Table 5c: *Correlations Between Outcome Variables and Maternal-Report Somatization (SOM)*

Outcomes		Age 6 SOM	Age 8 SOM	Age 10 SOM	Age 11 SOM	Age 12 SOM
Teacher Report						
Externalizing	Pearson *r*	.05	.08	-.01	.11	.06
	N	186	190	177	178	175
Anxious/depressed	Pearson *r*	.01	.10	.01	.04	.10
	N	187	191	178	179	176
Somatic complaints	Pearson *r*	.13	.22**	.19**	.19**	.18*
	N	186	190	177	178	176
School Records						
School absenteeism	Pearson *r*	.36**	.20*	.25*	.19^{+}	.18^{+}
	N	108	103	96	100	98
Youth Self-Report						
Depression symptoms	Pearson *r*	-.04	.02	.10	-.03	-.02
	N	206	212	194	201	218
Anxiety symptoms	Pearson *r*	.01	-.02	.03	-.07	-.11
	N	204	210	192	199	217
Delinquency	Pearson *r*	.06	-.01	.01	.07	.04
	N	206	212	195	201	218

Note: All comparisons are two-tailed.
$^{+}$ $p < .08$, * $p < .05$, ** $p < .01$

Table 6: Bayesian Information Criteria (BIC) by Model Type for School-Age Somatization with Entire Sample and Excluding Five and Seven Cases (Outliers)

Model	Order[a]	BIC
Full Data		
a. One group	1	-1768.43
b. Two group	1 1	-1689.61
c. Three group	1 1 1	-1679.89
Full Data Minus Five (5) Outlier Cases		
a. Two group	1 1	-1586.51
b. Three group	0 1 0	-1581.57
c. Four group	1 1 1 1	-1592.98
Full Data Minus Seven (7) Outlier Cases		
a. Two group	1 1	-1550.04
b. Three group	**0 1 1**	**-1549.89**

[a] Entries in second column represent the parameters used to approximate each group's trajectory. For instance, a two group model with the order 0 1 would indicate that the first group's trajectory is approximated by a zero-order polynomial. That is, it is defined only by an intercept. The second group's trajectory, however, is approximated by a first-order polynomial that includes (by definition) an intercept and a linear growth term.

Table 7: Means and Standard Deviations for Somatic Complaints by Age and Trajectory Group Status

Maternal Report on the Somatic Complaint subscale *Child Behavior Checklist* (CBCL)	Mean	Standard deviation	Range	Sample size
Age 6				
No Somatic Complaints (SOM)	0	0	0	53
Low Increasing SOM	.40	.754	0 – 4	136
Moderate Increasing SOM	1.95	1.65	0 – 6	66
MTH[+]	2.00	1.41	0 – 4	5
Age 8				
No SOM	0	0	0	51
Low Increasing SOM	.48	.787	0 – 3	126
Moderate Increasing SOM	1.81	1.47	0 – 7	63
MTH	5.80	5.45	1 – 14	5
Age 10				
No SOM	0	0	0	49
Low Increasing SOM	.81	1.11	0 – 5	116
Moderate Increasing SOM	2.47	1.80	0 – 7	55
MTH	4.75	2.36	3 – 8	4
Age 11				
No SOM	0	0	0	54
Low Increasing SOM	.91	1.08	0 – 4	114
Moderate Increasing SOM	2.78	1.76	0 – 7	60
MTH	8.40	.894	7 – 9	5
Age 12				
No SOM	0	0	0	50
Low Increasing SOM	.70	.863	0 – 3	110
Moderate Increasing SOM	2.35	1.72	0 – 6	55
MTH	7.60	2.07	5 – 10	5

Note: Across ages, 72% (age 12) to 85% (age 6) of all boys had scores for the Somatic Complaints subscale.

[+] MTH = Moderate to High

Table 8: Risk Factors by Somatization Trajectory Group

Risk Factor	No (n = 53)	Low Increase (n = 145)	Moderate Increase (n = 64)	MTH (n = 5)	F
Difficult Temperament	22.09 (5.65) 11 – 43	23.07 (6.24) 11 – 49	23.94 (6.48) 13 – 42	21.00 (3.54) 15 – 24	1.08
Child Negative Emotionality	3.95 (1.40) 2 – 7	4.12 (1.44) 2 – 8	4.19 (1.86) 2 – 8	3.25 (.957) 2 – 4	.590
Parent-Child Conflict	23.77_{b} (8.07) 11 – 43	24.64 (8.13) 10 – 49	27.28_{b} (7.45) 15 – 46	26.60 (4.56) 19 – 31	2.45^{*}
Maternal Depression	$5.27_{\mathbf{a}}$ (5.23) 0 – 22	7.36 (5.85) 0 – 26	$8.82_{\mathbf{a}}$ (5.91) 0 – 30	6.13 (2.12) 3 – 8	3.19^{*}
Interparental Conflict	22.88 (13.18) 0 – 77	24.07 (13.65) 2 – 92	25.98 (12.07) 0 – 57	24.50 (6.05) 18 – 32	.837
Adversity Index	.356 (.259) 0 – 1	.327 (.238) 0 – .75	.348 (.263) 0 – .75	.450 (.326) 0 – .75	.569
Social Skills	$38.29_{\mathbf{b}}$ (9.43) 17 – 53	37.29_{b}(10.79) 3 – 55	37.21_{b} (9.42) 17 – 54	24.38_{b} (12.86) 8 – 37	2.28^{*}
Academic Incompetence	1.72_{c} (1.89) 0 – 6	1.74_{b} (1.97) 0 – 6	1.68_{b} (1.86) 0 – 6	$4.00_{b,c}$ (1.58) 3 – 6	1.86^{+}

Note: MTH = Moderate to High Group. Entries in the first four columns are means with standard deviations in parentheses. The range of scores appear below the mean and SD. Means with subscript "a" are significantly different at the $p < .01$ level; means with subscript "b" indicate significance at the $p < .05$ level, and the subscript "c" indicates a trend ($p < .10$), all based on Tukey's honestly significant post hoc comparisons.
$^{+}p < .10$. $^{*}p < .05$, one-tailed.

Table 9: Binary Logistic Regressions: Predicting Membership in Moderate Increasing versus No and Low Increasing SOM Groups

Variable	No Group				Low Increasing Group			
	B	*SE*	Wald	Odds ratio	*B*	*SE*	Wald	Odds ratio
Difficult temperament	.07	.06	1.41	1.07	.01	.03	.19	1.01
Child NE	.16	.18	.82	1.18	-.05	.13	.14	.95
Parent-child conflict	.06	.04	2.18	1.07	.01	.03	.22	1.01
Maternal depression	.13	.07	3.93*	1.14	.07	.04	3.61+	1.07
Interparental conflict	.00	.03	.00	1.00	-.00	.02	.06	1.00
Adversity Index	.02	1.32	.00	1.02	.95	.85	1.25	2.58
Child social skills	-.01	.04	.07	.99	.01	.03	.05	1.01
Child academic incompetence	.00	.19	.00	1.00	-.09	.13	.49	.92

* $p < .05$; + $p < .10$.

Table 10: Binary Logistic Regressions: Predicting Membership in Moderate Increasing versus No and Low Increasing SOM Groups

Variable	No Group				Low Increasing Group			
	B	*SE*	Wald	Odds ratio	*B*	*SE*	Wald	Odds ratio
Parent-child conflict	.06	.03	4.52*	1.07	.04	.02	2.73+	1.04
Maternal depression	.08	.04	3.37+	1.08	.05	.03	2.85+	1.05
Child social skills	-.01	.03	.04	.99	-.00	.02	.00	1.00
Child academic incompetence	-.04	.14	.09	.96	-.07	11	.47	.93

* $p < .05$; + $p < .10$.

Table 11: Binary Logistic Regressions Testing Interactions

Table 11a:
Predicting Membership in Low Increasing SOM Group Versus No SOM Group

Risk Factor	*B*	*SE*	Wald	Odds ratio	*p*
Regression 1					
Difficult Temperament	.148	.058	6.44	1.16	.011
Adversity Index	6.48	2.94	4.85	652.0	.028
Temperament x Index	-.312	.129	5.86	.732	.015
Regression 2					
Difficult Temperament	.097	.047	4.26	1.10	.039
Interparental Conflict	.081	.038	4.59	1.09	.032
Temperament x Conflict	-.003	.001	3.98	.997	.046
Regression 3					
Child negative emotionality (NE)		-.903	.454	3.96	.405	.047
Parent-child conflict		-.146	.078	3.49	.864	.062
Child NE x parent-child conflict		.039	.018	4.70	1.04	.030

Note: Low Increasing group is coded as "1," and No group is coded as "0" for the three separate regressions.

Table 11b:
Predicting Membership in Moderate Increasing Versus Low Increasing SOM Groups

Risk Factor	*B*	*SE*	Wald	Odds ratio	*p*
Child negative emotionality (NE)	.733	.376	3.79	2.08	.051
Parent-child conflict	.149	.064	5.36	1.16	.021
Child NE x parent-child conflict	-.028	.014	4.20	.972	.040

Note: Moderate Increasing group is coded as "1," and Low Increasing group is coded as "0" for the regression.

Table 12: Youth Outcomes by Somatization Trajectory Group

Outcomes	SOM Group (*n*)	Mean	SD	Range	*F*
Teacher-Report Form (Ages 11 & 12)					
Externalizing broadband factor	No (40)	27.33	16.98	0 – 80	1.32
	Low Increasing (108)	24.04	18.83	0 – 91	
	Moderate Increasing (51)	27.06	20.74	0 – 70	
	MTH (5)	40.60	16.32	25 – 61	
Internalizing (Anx/Dep +Withdrawal)	No (40)	4.93	5.67	0 – 22	.799
	Low Increasing (108)	5.06	5.31	0 – 26	
	Moderate Increasing (52)	5.50	5.04	0 – 23	
	MTH (5)	8.60	4.16	4 – 14	
Somatic Complaints subscale	No (39)	$.590_a$	1.17	0 – 5	4.09**
	Low Increasing (108)	$.546_a$	.987	0 – 6	
	Moderate Increasing (52)	.952	1.65	0 – 10	
	MTH (5)	2.50_a	5.29	0 – 10	
Functional Impairment (Ages 10-12)					
Days Absent from School (from school records)	No (24)	13.33	11.45	3 – 52	2.14^+
	Low Increasing (68)	14.13	10.25	1 – 46	
	Moderate Increasing (34)	18.78	10.50	4 – 45	
	MTH (2)	22.67	2.36	21 – 24	
Youth Self-Report (Age 12)					
Depressive Symptoms – (*CDI*)	No (50)	1.16	1.60	0 – 6	.731
	Low Increasing (112)	.848	1.38	0 – 6	
	Moderate Increasing (57)	1.03	1.32	0 – 6	
	MTH (5)	.600	0.89	0 – 2	
Anxiety Symptoms – (*MASC*)	No (50)	8.56	4.46	2 – 19	.859
	Low Increasing (110)	8.05	3.97	0 – 18	
	Moderate Increasing (57)	7.75	3.93	0 – 18	
	MTH(5)	5.80	5.23	2 – 12	
Delinquency – *(SRD)*	No (51)	3.04	4.08	0 – 23	.539
	Low Increasing (111)	3.86	4.36	0 – 26	
	Moderate Increasing (57)	3.47	3.28	0 – 14	
	MTH (5)	3.00	2.65	0 – 7	

Note: MTH = Moderate to High Group. Means with the subscript "a" are significantly different based on Tukey's honestly significant post hoc comparisons. Means with the subscript "b" indicate a trend towards significance.
$^+p < .10$, $^{**}p < .01$.

BIBLIOGRAPHY

Achenbach, T. M. (1991a). *Manual for the Child Behavior Checklist 4/18 and 1991 Profile*. Burlington: University of Vermont, Department of Psychiatry.

Achenbach, T. M. (1991b). *Manual for the Youth Self-Report and 1991 Profile.* Burlington: University of Vermont, Department of Psychiatry.

Achenbach, T. M., Conners, C., Quay, H. C., Verhulst, F. C., & Howell, C. T. (1989). Replication of empirically derived syndromes as a basis for taxonomy of child/adolescent psychopathology. *Journal of Abnormal Child Psychology, 17*(3), 299-323.

Achenbach, T. M., & Edelbrock, C. (1986). *Manual for the Teacher's Report Form and Teacher Version of the Child Behavior Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.

Alfven, G. (1993). The covariation of common psychosomatic symptoms among children from socio-economically differing residential areas. An epidemiological study. *Acta Paediatrica, 82*(5), 484-487.

American-Psychiatric-Association. (1987). *Diagnostic and statistical manual of mental disorders, 3rd edition-revised (DSM-III-R)*. Washington, DC: American Psychiatric Association.

American-Psychiatric-Association. (1994). *Diagnostic and statistical manual of mental disorders, 4th edition (DSM-IV)*. Washington DC: American Psychiatric Association.

Andrasik, F., Kabela, E., Quinn, S., Attanasio, V., & et al. (1988). Psychological functioning of children who have recurrent migraine. *Pain, 34*(1), 43-52.

Apley, J. (1975). *The child with abdominal pains*. London: Blackwell.

Apley, J., & Hale, B. (1973). Children with recurrent abdominal pain: How do they grow up? *British Medical Journal, 3*, 7-9.

Aro, H. (1987). Life stress and psychosomatic symptoms among 14 to 16-year old Finnish adolescents. *Psychological Medicine, 17*(1), 191-201.

Aro, H. (1989). Stress, development and psychosomatic symptoms in adolescence: A comparison of the sexes. *Psychiatria Fennica, 20*, 101-109.

Aro, H., Haenninen, V., & Paronen, O. (1989). Social support, life events and psychosomatic symptoms among 14-16-year-old adolescents. *Social Science & Medicine, 29*(9), 1051-1056.

Aro, H., Paronen, O., & Aro, S. (1987). Psychosomatic symptoms among 14-26 year old Finnish adolescents. *Social Psychiatry, 22*(3), 171-176.

Aromaa, M., Rautava, P., Helenius, H., & Sillanpaeae, M. (1998). Factors of early life as predictors of headache in children at school entry. *Headache, 38*(1), 23-30.

Aromaa, M., Sillanpaa, M., Rautava, P., & Helenius, H. (2000). Pain experience of children with headache and their families: A controlled study. *Pediatrics, 106*(2), 270-275.

Bandell-Hoekstra, I. E., Abu-Saad, H. H., Passchier, J., Frederiks, C. M., Feron, F. J., & Knipschild, P. (2002). Coping and Quality of Life in relation to headache in Dutch schoolchildren. *European Journal of Pain: Ejp, 6*(4), 315-321.

Bates, J. E., Freeland, C. A., & Lounsbury, M. L. (1979). Measurement of infant difficultness. *Child Development, 50*(3), 794-803.

Bates, J. E., Maslin, C. A., & Frankel, K. A. (1985). Attachment security, mother-child interaction, and temperament as predictors of behavior-problem ratings at age three years. *Monographs of the Society for Research in Child Development, 50*(1-2), 167-193.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8*(1), 77-100.

Beck, A. T., Ward, C. H., Mendelsohn, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561-571.

Bernstein, G. A., Massie, E. D., Thuras, P. D., Perwien, A. R., & et al. (1997). Somatic symptoms in anxious-depressed school refusers. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(5), 661-668.

Berntsson, L. T., & Koehler, L. (2001). Long-term illness and psychosomatic complaints in children aged 2-17 years in the five Nordic countries. Comparison between 1984 and 1996. *European Journal of Public Health, 11*(1), 35-42.

Boey, C. C. M., & Goh, K.-L. (2001). The significance of life-events as contributing factors in childhood recurrent abdominal pain in an urban community in Malaysia. *Journal of Psychosomatic Research, 51*(4), 559-562.

Boyce, W., Quas, J., Alkon, A., Smider, N. A., Essex, M. J., Kupfer, D. J., et al. (2001). Autonomic reactivity and psychopathology in middle childhood. *British Journal of Psychiatry, 179*, 144-150.

Calkins, S. D. (1994). Origins and outcomes of individual differences in emotion regulation. *Monographs of the Society for Research in Child Development, 59*(2-3), 53-72, 250-283.

Campo, J. V., & Fritsch, S. L. (1994). Somatization in children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 33*(9), 1223-1235.

Campo, J. V., & Garber, J. (1998). Somatization. In R. T. Ammerman & J. V. Campo (Eds.), *Handbook of pediatric psychology and psychiatry: Psychological and psychiatric issues in the pediatric setting* (pp. 137-161). Boston: Allyn & Bacon.

Campo, J. V., Jansen-McWilliams, L., Comer, D. M., & Kelleher, K. J. (1999). Somatization in pediatric primary care: Association with psychopathology, functional impairment, and use of services. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(9), 1093-1101.

Campo, J. V., & Reich, M. D. (1999). Somatoform disorders. In S. D. Netherton & D. Holmes (Eds.), *Child & adolescent psychological disorders: A comprehensive textbook* (pp. 320-343). New York: Oxford University Press.

Christensen, M. F., & Mortensen, O. (1975). Long-term prognosis in children with recurrent abdominal pain. *Archives of Disease in Childhood, 50*(2), 110-114.

Chu, M. L., & Shinnar, S. (1992). Headaches in children younger than 7 years of age. *Archives of Neurology, 49*(1), 79-82.

Cicchetti, D., & Sroufe, L. (2000). The past as prologue to the future: The times, they've been a-changin'. *Development & Psychopathology, 12*(3), 255-264.

Compas, B. E., & Boyer, M. C. (2001). Coping and attention: Implications for child health and pediatric conditions. *Journal of Developmental & Behavioral Pediatrics, 22*(5), 323-333.

Compas, B. E., Connor-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin, 127*(1), 87-127.

Cote, S., Tremblay, R. E., Nagin, D., Zoccolillo, M., & Vitaro, F. (2002). The development of impulsivity, fearfulness, and helpfulness during childhood: Patterns of consistency and change in the trajectories of boys and girls. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 43*(5), 609-618.

Craig, T., Boardman, A., Mills, K., Daly-Jones, O., & Drake, H. (1993). The South London Somatisation Study: I. Longitudinal course and the influence of early life experiences. *British Journal of Psychiatry, 163*, 579-588.

Cummings, E. M., Davies, P. T., & Campbell, S. B. (2000). *Developmental psychopathology and family process: Theory, research, and clinical implications*. New York: Guilford Press.

Cunningham, S., McGrath, P., Ferguson, H., Humphreys, P., & et al. (1987). Personality and behavioural characteristics in pediatric migraine. *Headache, 27*(1), 16-20.

Dhossche, D., Ferdinand, R., van der Ende, J., & Verhulst, F. (2001). Outcome of self-reported functional-somatic symptoms in a community sample of adolescents. *Annals of Clinical Psychiatry, 13*(4), 191-199.

Domenech-Llaberia, E., Jane, M. C., Canals, J., Ballespi, S., Esparo, G., & Garralda, E. (2004). Parental reports of somatic symptoms in preschool children: Prevalence and associations in a Spanish sample. *Journal of the American Academy of Child & Adolescent Psychiatry, 43*(5), 598-604.

Dorn, L. D., Campo, J. C., Thato, S., Dahl, R. E., Lewin, D., Chandra, R., et al. (2003). Psychological comorbidity and stress reactivity in children and adolescents with recurrent abdominal pain and anxiety disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*(1), 66-75.

D'Unger, A., Land, K., McCall, P., & Nagin, D. S. (1998). How many latent classes of delinquent/criminal careers? Results from mixed Poisson regression analyses of the London, Philadelphia, and Racine cohort studies. *American Journal of Sociology, 103*, 1593-1630.

Egger, H. L., Angold, A., & Costello, E. (1998). Headaches and psychopathology in children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*(9), 951-958.

Egger, H. L., Costello, E., Erkanli, A., & Angold, A. (1999). Somatic complaints and psychopathology in children and adolescents: Stomach aches, musculoskeletal pains, and headaches. *Journal of the American Academy of Child & Adolescent Psychiatry, 38*(7), 852-860.

Elliot, D. S., Huizinga, D., & Ageton, S. S. (1985). *Explaining delinquency and drug use*. Thousand Oaks, CA: Sage Publications.

Fearon, P., & Hotopf, M. (2001). Relation between headache in childhood and physical and psychiatric symptoms in adulthood: National birth cohort study. *British Medical Journal, 322*(7295), 1145-1148.

Fritz, G. K., Fritsch, S., & Hagino, O. (1997). Somatoform disorders in children and adolescents: A review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(10), 1329-1338.

Garber, J., Walker, L. S., & Zeman, J. (1991). Somatization symptoms in a community sample of children and adolescents: Further validation of the Children's Somatization Inventory. *Psychological Assessment, 3*(4), 588-595.

Garber, J., Zeman, J., & Walker, L. S. (1990). Recurrent abdominal pain in children: Psychiatric diagnoses and parental psychopathology. *Journal of the American Academy of Child & Adolescent Psychiatry, 29*(4), 648-656.

Garcia, M. M., Shaw, D. S., Winslow, E. B., & Yaggi, K. E. (2000). Destructive sibling conflict and the development of conduct problems in young boys. *Developmental Psychology, 36*(1), 44-53.

Garralda, M. (1996). Somatisation in children. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 37*(1), 13-33.

Gilliom, M., & Shaw, D. S. (2004). Early trajectories of child internalizing problems. *Submitted to Developmental Psychopathology*.

Gladstein, J., & Holden, E. (1996). Chronic daily headache in children and adolescents: A 2-year prospective study. *Headache, 36*(6), 349-351.

Gresham, F. M., & Elliott, S. N. (1990). *Social Skills Rating Scales Manual*. Circle Pines, MN: American Guidance Service.

Gulhati, A., & Minty, B. (1998). Parental health attitudes, illnesses and supports and the referral of children to medical specialists. *Child: Care, Health & Development, 24*(4), 295-313.

Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology, 27*(1-2), 199-220.

Honjo, S., Nishide, T., Niwa, S., Sasaki, Y., Kaneko, H., Inoko, K., et al. (2001). School refusal and depression with school attendance in children and adolescents: Comparative assessment between the Children's Depression Inventory and somatic complaints. *Psychiatry & Clinical Neurosciences, 55*(6), 629-634.

Hotopf, M., Carr, S., Mayou, R., Wadsworth, M., & Wessely, S. (1998). Why do children have chronic abdominal pain, and what happens to them when they grow up? Population based cohort study. *British Medical Journal, 316*(7139), 1196-1200.

Jones, B. L., Nagin, D. S., & Roeder, K. (2001). A SAS procedure based on mixture models for estimating developmental trajectories. *Sociological Methods and Research, 29*, 374-393.

Kazdin, A. E., French, N. H., Unis, A. S., Esveldt-Dawson, K., & Sherick, R. B. (1983). Hopelessness, depression, and suicidal intent among psychiatrically disturbed inpatient children. *Journal of Consulting & Clinical Psychology, 51*(4), 504-510.

King, N. J., & Bernstein, G. A. (2001). School refusal in children and adolescents: A review of the past 10 years. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*(2), 197-205.

Kovacs, M. (1992). *The Children's Depression Inventory Manual*. North Tonawanda, NY: Multi-Health Systems.

Kowal, A., & Pritchard, D. (1990). Psychological characteristics of children who suffer from headache: A reasearch note. *Journal of Child Psychology and Psychiatry, 31*(4), 637-649.

Last, C. G. (1991). Somatic complaints in anxiety disordered children. *Journal of Anxiety Disorders, 5*(2), 125-138.

Laub, J. H., Nagin, D. S., & Sampson, R. J. (1998). Trajectories of change in criminal offending: Good marriages and the desistance process. *American Sociological Review, 63*(2), 225-238.

Lipowski, Z. J. (1988). Somatization: The concept and its clinical application. *American Journal of Psychiatry, 145*(11), 1358-1368.

Livingston, R., Taylor, J., & Crawford, S. (1988). A study of somatic complaints and psychiatric diagnosis in children. *Journal of the American Academy of Child & Adolescent Psychiatry, 27*(2), 185-187.

March, J. S., Parker, J. D., Sullivan, K., Stallings, P., & Conners, C. K. (1997). The Multidimensional Anxiety Scale for Children (MASC): Factor structure, reliability, and validity. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*(4), 554-565.

McCauley, E., Carlson, G. A., & Calderon, R. (1991). The role of somatic complaints in the diagnosis of depression in children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 30*(4), 631-635.

Moffitt, T. E. (1990). Juvenile delinquency and attention-deficit disorder: Developmental trajectories from age 3 to 15. *Child Development, 61*, 893-910.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review, 100*(4), 674-701.

Nagin, D. S. (1999). Analyzing developmental trajectories: A semiparametric, group-based approach. *Psychological Methods, 4*(2), 139-157.

Nagin, D. S., & Tremblay, R. E. (1999). Trajectories of boys' physical aggression, opposition, and hyperactivity on the path to physically violent and nonviolent juvenile delinquency. *Child Development, 70*(5), 1181-1196.

Offord, D. R., Boyle, M. H., Szatmari, P., Rae-Grant, N. I., Links, P. S., Cadman, D. T., et al. (1987). Ontario Child Health Study: II. Six-month prevalence of disorder and rates of service utilization. *Archives of General Psychiatry, 44*(9), 832-836.

Patterson, G., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist, 44*(2), 329-335.

Pianta, R. C., & Steinberg, M. (1991). *Relationship between children and kindergarten teachers: Associations with home and classroom behavior.* Paper presented at the Society for Research in Child Development, Seattle, WA.

Pittsburgh_Youth_Study. (1991). *Neighborhood dangerousness scale*: Unpublished measure, University of Pittsburgh.

Rauste-von Wright, M., & von Wright, J. (1981). A longitudinal study of psychosomatic symptoms in healthy 11-28 year old girls and boys. *Journal of Psychosomatic Research, 25*(6), 525-534.

Rocha, E. M., Prkachin, K. M., Beaumont, S. L., Hardy, C. L., & Zumbo, B. D. (2003). Pain reactivity and somatization in kindergarten-age children. *Journal of Pediatric Psychology, 28*(1), 47-57.

Rothner, A. D. (1993). Diagnosis and management of headaches in children and adolescents. In N. L. Schechter, C. B. Berde & M. Yaster (Eds.), *Pain in infants, children, and adolescents* (pp. 547-554). Baltimore: Williams & Wilkins.

Ruchkin, V., Eisemann, M., & Haeggloef, B. (2000). Coping styles and psychosomatic problems: Are they related? *Psychopathology, 33*(5), 235-239.

Rutter, M., Cox, A., Tupling, C., Berger, M., & Yule, W. (1975). Attainment and adjustment in two geographical areas: 1. The prevalence of psychiatric disorder. *British Journal of Psychiatry, 126*, 493-509.

Rutter, M., Yule, B., Quinton, D., Rowlands, O., Yule, W., & Berger, M. (1975). Attainment and adjustment in two geographical areas: 3. Some factors accounting for area differences. *British Journal of Psychiatry, 126*, 520-533.

Sameroff, A. J., Seifer, R., Zax, M., & Barocas, R. (1987). Early indicators of developmental risk: Rochester longitudinal study. *Schizophrenia Bulletin, 13*(3), 383-394.

Scharff, L. (1997). Recurrent abdominal pain in children: A review of psychological factors and treatment. *Clinical Psychology Review, 17*(2), 145-166.

Shaw, D. S., & Emery, R. E. (1987). Parental conflict and other correlates of the adjustment of school-age children whose parents have separated. *Journal of Abnormal Child Psychology, 15*(2), 269-281.

Shaw, D. S., Gilliom, M., Ingoldsby, E. M., & Nagin, D. S. (2003). Trajectories leading to school-age conduct problems. *Developmental Psychology, 39*(2), 189-200.

Shaw, D. S., Vondra, J. I., Hommerding, K. D., Keenan, K., & Dunn, M. (1994). Chronic family adversity and early child behavior problems: A longitudinal study of low income families. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 35*(6), 1109-1122.

Shaw, D. S., Winslow, E. B., Owens, E. B., & Hood, N. (1998). Young children's adjustment to chronic family adversity: A longitudinal study of low-income families. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*(5), 545-553.

Stevenson, J., Simpson, J., & Bailey, V. (1988). Research note: recurrent headaches and stomachaches in preschool children. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 29*(6), 897-900.

Stickney, M. I., & Miltenberger, R. G. (1998). School refusal behavior: Prevalence, characteristics, and the schools' response. *Education & Treatment of Children, 21*(2), 160-170.

Straus, M. A. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics (CT) Scales. *Journal of Marriage and the Family, 41*(1), 75-88.

Terre, L., & Ghiselli, W. (1997). A developmental perspective on family risk factors in somatization. *Journal of Psychosomatic Research, 42*(2), 197-208.

Thomsen, A. H., Compas, B. E., Colletti, R. B., Stanger, C., Boyer, M. C., & Konik, B. S. (2002). Parent reports of coping and stress responses in children with recurrent abdominal pain. *Journal of Pediatric Psychology, 27*(3), 215-226.

Vannatta, K., Gartstein, M. A., Short, A., & Noll, R. B. (1998). A controlled study of peer relationships of children surviving brain tumors: Teacher, peer, and self ratings. *Journal of Pediatric Psychology, 23*(5), 279-287.

Volling, B. L., & Belsky, J. (1992). The contribution of mother-child and father-child relationships to the quality of sibling interaction: A longitudinal study. *Child Development, 63*, 1209-1222.

Walker, L. S., Claar, R. L., & Garber, J. (2002). Social consequences of children's pain: When do they encourage symptom maintenance? *Journal of Pediatric Psychology, 27*(8), 689-698.

Walker, L. S., Garber, J., & Greene, J. W. (1993). Psychosocial correlates of recurrent childhood pain: A comparison of pediatric patients with recurrent abdominal pain, organic illness, and psychiatric disorders. *Journal of Abnormal Psychology, 102*(2), 248-258.

Walker, L. S., Garber, J., & Greene, J. W. (1994). Somatic complaints in pediatric patients: A prospective study of the role of negative life events, child social and academic

competence, and parental somatic symptoms. *Journal of Consulting & Clinical Psychology, 62*(6), 1213-1221.

Walker, L. S., Garber, J., Smith, C. A., Van Slyke, D. A., & Claar, R. L. (2001). The relation of daily stressors to somatic and emotional symptoms in children with and without recurrent abdominal pain. *Journal of Consulting & Clinical Psychology, 69*(1), 85-91.

Walker, L. S., Garber, J., Van Slyke, D. A., & Greene, J. W. (1995). Long-term health outcomes in patients with recurrent abdominal pain. *Journal of Pediatric Psychology, 20*(2), 233-245.

Walker, L. S., & Greene, J. W. (1987). Negative life events, psychosocial resources, and psychophysiological symptoms in adolescents. *Journal of Clinical Child Psychology, 16*(1), 29-36.

Walker, L. S., & Greene, J. W. (1989). Children with recurrent abdominal pain and their parents: More somatic complaints, anxiety, and depression than other patient families? *Journal of Pediatric Psychology, 14*(2), 231-243.

Walker, L. S., McLaughlin, F., & Greene, J. W. (1988). Functional illness and family functioning: A comparison of healthy and somaticizing adolescents. *Family Process, 27*(3), 317-325.

Walker, L. S., Smith, C. A., Garber, J., & Van Slyke, D. A. (1997). Development and validation of the pain response inventory for children. *Psychological Assessment, 9*(4), 392-405.

Zahn-Waxler, C., Klimes-Dougan, B., & Slattery, M. J. (2000). Internalizing problems of childhood and adolescence: Prospects, pitfalls, and progress in understanding the development of anxiety and depression. *Development & Psychopathology, 12*(3), 443-466.

Zuckerman, B., Stevenson, J., & Bailey, V. (1987). Stomachaches and headaches in a community sample of preschool children. *Pediatrics, 79*(5), 677-682.

Zwaigenbaum, L., Szatmari, P., Boyle, M. H., & Offord, D. R. (1999). Highly somatizing young adolescents and the risk of depression. *Pediatrics, 103*(6 Pt 1), 1203-1209.