PSYCHOSOCIAL, COGNITIVE, AND PHYSIOLOGICAL PROTECTIVE FACTORS AND THE ABSENCE OF ANTISOCIAL BEHAVIOR IN A LONGITUDINAL STUDY OF LOW INCOME BOYS

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The present study sought to advance our understanding of resilience by examining the timing and continuity of multiple domains of protective factors (i.e., psychosocial, cognitive, physiological) measured in early childhood and the transition to adolescence as they related to low antisocial behavior in adolescence among an ethnically diverse cohort of 310 low-income boys. While antisocial behavior was the main outcome of interest, given its huge societal and personal costs (Kazdin, 1996), the study also examined positive functioning across domains (i.e., internalizing, school achievement) to address issues of cross-domain adjustment. Although the entire sample could be considered at high risk due to low socioeconomic status, levels of risk were further differentiated by investigating the accumulation of other risk factors (e.g., single parent status, neighborhood disadvantage, overcrowding in the home) as they related to outcomes across domains. Furthermore, in addition to more ubiquitously researched psychosocial and cognitive protective factors such as child IQ and parenting, this study also included measures of physiological variables (i.e., vagal tone, sleep, testosterone).

In line with hypotheses, several parenting protective factors, as well as child IQ and sleep, were significantly associated with low antisocial behavior at ages 15 and 17. With the

exception of vagal tone, the relations between protective factors and antisocial behavior did not vary across levels of cumulative risk. Hypotheses regarding the importance of continuity and timing of protective factors were generally not supported, in that it was equally helpful to have a protective factor present at one or two time periods, in either early childhood or the transition to adolescence. Finally, in line with hypotheses, there did appear to be some fluctuation across positive outcomes; while youth who had low antisocial behavior at ages 15 and 17 were more likely than youth high on antisocial behavior to being doing well in school, they were not more likely to have low internalizing symptoms. Both cumulative risk and cumulative protective factors were related to the number of positive outcomes that youth had across domains. Results highlight the importance of both cumulative risk and protection in the development of positive adaptation.

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1.0 INTRODUCTION

Over the past several decades, the concept of resilience has gained prominence as a way to study the processes and mechanisms through which exposure to risk factors may be associated with children's positive and negative outcomes. The term resilience has been defined as a positive outcome in the context of adversity (Luthar, Cicchetti, & Becker, 2000), and centers on the study of various child, family, and community protective factors that may be associated with positive adjustment despite exposure to risk factors. Resilience has become a popular topic both in the context of developmental research and in the media, and has implications for understanding child development, and for prevention and intervention efforts aimed at guiding public policy and social programs to improve outcomes for children at risk (Masten, 2001).

When the concept of resilience was first introduced in the 1970s, it was conceptualized as a stable personal characteristic; at-risk children who appeared to be doing well were thought to be "invulnerable" (Pines, 1975). This perspective that certain children, due to some internal characteristic (e.g., IQ) or positive feature of their environment (e.g., strong relationship with a caregiver), could 'beat the odds' and demonstrate positive adjustment in the context of adversity, led to a search for protective factors that could explain such associations. As research in the area of resilience has developed over time, the conceptualization of resilience has been refined, such that most researchers now recognize it as a dynamic process that results from ongoing transactions between a child and the environment, rather than a stable, internal characteristic of

the child (Luthar & Zelazo, 2003). Few researchers now view children with positive outcomes as "invulnerable," and there is increasing recognition that the effects of risk persist over time or emerge in unexpected ways (Luthar, 2006; Vanderbilt-Adriance & Shaw, 2008a).

However, despite improvements in the conceptualization of resilience, a number of important challenges remain. First, the variability in the establishment of 'high risk' environments has hindered our ability to determine the prevalence of resilience. For example, some studies of resilience have utilized predominantly European American, middle-class children who, although experiencing a significant life event (e.g., divorce), have been exposed to a qualitatively lower level of adversity than children growing up in the context of inner city or rural poverty. Because of the comparatively low level of risk in the former context, such studies may obtain misleadingly high rates of positive adjustment compared to children living in more chronic and severe settings (Vanderbilt-Adriance & Shaw, 2008a). Relatedly, there is also a need for more studies comparing children at differing levels of high risk in order to make fine grain distinctions between protective factors that operate in the context of high but not extreme risk (e.g., Kliewer et al., 2004; Miller et al., 1999).

Second, there are also data to suggest that when positive adjustment is identified among children living in adverse contexts, it may vary across domains. For example, children who may be doing well in one area, such as low antisocial behavior, may demonstrate problems in other areas, such as internalizing symptoms or school achievement (Loeber, Pardini, Stouthamer-Loeber, & Raine, 2007; Moffitt, Caspi, Harrington, & Milne, 2002). Thus, resilience may not be generalized, but rather specific, with children showing strengths and weaknesses depending on the domain in question.

Finally, the majority of studies investigating resilience have focused solely on

psychosocial or cognitive protective factors (e.g., child IQ, parenting). Within the literature, there has been increasing recognition of the importance of a multilevel perspective on resilience, focusing on multiple domains of protective factors, including physiological/biological and psychosocial protective factors (Cicchetti & Curtis, 2007). As has been pointed out, integrating biological and physiological factors (e.g., testosterone, sleep, vagal tone) into resilience research may help to inform models of plasticity and/or constraints (Curtis & Cicchetti, 2003; Greenberg, 2006; Luthar, 2006). Biological processes affect many aspects of behavior, emotion, and cognition, and likely mediate and/or moderate the associations between risk, protection, and outcome (Greenberg, 2006; Silk et al., 2007). Given the dearth of studies that examine multiple levels of protective factors, there is a clear need for studies that investigate both physiological and psychosocial processes.

The present study aims to advance our understanding of resilience by examining multiple domains of protective factors (i.e., psychosocial, cognitive, physiological) in early childhood and the transition to adolescence as they relate to low levels of antisocial behavior in late adolescence among an ethnically diverse cohort of 310 low-income children. Protective factors in early childhood and the transition to adolescence were selected as a focus because these developmental periods are associated with extraordinary change and transformation in multiple areas, including emotion, cognition, and biology (Shaw & Gross, 2008; Steinberg et al., 2006). Such important developmental transitions may prove to be key points for both increased vulnerability and positive change as children adapt to new challenges and demands (Steinberg et al., 2006; Vanderbilt-Adriance & Shaw, 2008a). Thus, early childhood and the transition to adolescence may present ideal developmental periods for intervention, working either to augment and support already successful adaptation or to alter potentially negative trajectories.

While antisocial behavior was the main outcome of interest in the current study, given its huge societal and personal costs (Kazdin, 1996), positive functioning across domains (i.e., internalizing symptoms, school achievement) was also examined to address issues of cross-domain adjustment. Although the entire sample could be considered at high risk due to low socioeconomic status, levels of risk were further differentiated by investigating the accumulation of other risk factors (e.g., single parent status, neighborhood disadvantage, overcrowding in the home) as they related to outcomes across domains. As discussed above, there is a clear need for more studies of children at severe risk, particularly ones that examine continuity and discontinuity in adaptation across domains of functioning. Furthermore, in addition to more ubiquitously researched psychosocial and cognitive protective factors such as child IQ and parenting, this study also included measures of physiological variables (i.e., vagal tone, sleep, testosterone). In general, child and parenting protective factors measured in early childhood and the transition to adolescence were expected to be associated with low antisocial behavior in late adolescence, although these relations were expected to be attenuated in the context of high cumulative risk. Furthermore, it was expected that there would be considerable fluctuation in positive outcomes across domains, with few youth demonstrating adaptive outcomes on antisocial externalizing behavior *and* internalizing symptoms or school achievement.

1.1 RESILIENCE

Resilience is currently conceptualized as a dynamic process consisting of a series of ongoing, reciprocal transactions between the child and the environment (Luthar & Zelazo, 2003; Masten, 2001). Importantly, this conceptualization rejects the notion of resilience as a personal or

individual trait. In fact, researchers have warned against using such terms as “resiliency” because they connote a stable characteristic, and may foster perspectives that blame the individual for their negative outcomes (Luthar et al., 2000). Although personal traits (e.g., IQ, temperament) can influence outcomes in the context of adversity, they are also often strongly affected by both genetic and contextual factors, and are thus not fully attributable to the child (Luthar & Cicchetti, 2000).

Resilience has been operationalized in many ways, but it is most commonly defined as a positive outcome in the context of risk, or factors known to be associated with negative outcomes (Luthar et al., 2000). Explicit within this definition is the requirement of risk, in addition to a positive outcome; thus high functioning children in situations of low adversity would not be considered resilient.

1.2 RISK

Resilience research has operationalized “risk” in a number of different ways, utilizing such factors as parental psychopathology (Conrad & Hammen, 1993; Luthar & Sexton, 2007), socioeconomic disadvantage (Buckner, Mezzacappa, & Beardslee, 2003; Kim-Cohen, Moffitt, Caspi, & Taylor, 2004), urban poverty and community violence (Gorman-Smith, Henry, & Tolan, 2004; Hammack et al., 2004), negative life events (D'Imperio, Dubow, & Ippolito, 2000; Masten et al., 1999), child maltreatment (Cicchetti & Rogosch, 1997; Jaffee et al., 2007), and cumulative risk indices (Seifer, Sameroff, Baldwin, & Baldwin, 1992). While all of these factors are associated with negative outcomes in children, it is important to note that they are not necessarily equivalent in severity; rather severity depends upon both the risk factor and the

population in question. For example, some researchers have utilized normative middle class samples exposed to varying levels of negative life events (e.g., Masten et al., 1999), while others have utilized ethnically diverse samples of children growing up in low-income neighborhoods (e.g., Gorman-Smith et al., 2004). Low socioeconomic status (SES) is associated with a substantial number of stressors and adversities, including community violence, crowding, poor quality schools, and inadequate housing (McLoyd, 1998; Sampson, Morenoff, & Earls, 1999). Arguably, children growing up in low SES environments are exposed to a wide array of risks that are both qualitatively and quantitatively more adverse than those experienced by most children living in middle-class environments. It is not clear that results from middle-class, predominantly Caucasian samples can be generalized to low SES, ethnically diverse samples; thus results from the former studies may be overestimating the degree to which resilience exists in situations of chronic, severe risk (see Vanderbilt-Adriance & Shaw, 2008, for a review).

In addition to using low SES, other researchers have used a cumulative risk approach to index a child's risk status. The cumulative risk approach typically involves summing a range of dichotomized community- and family-level risk factors (e.g., neighborhood disadvantage, overcrowding) to generate an index of risk. Risk factors, such as neighborhood disadvantage or harsh parenting, which may not have set cut-offs are generally classified as "present" if scores are 1 SD above the mean (e.g., Trentacosta et al., 2008) or in the top quartile (e.g., NICHD Early Child Care Research Network, 2004). Other risk factors have more clinically- or culturally-meaningful cut-offs; for example, "low" maternal education is generally classified as less than a high school diploma or GED (Ackerman et al., 1999; Gerard & Buehler, 2004; Trentacosta et al., 2008). In addition to the commonly used approach described above (i.e., summing dichotomized risk factors), several studies have also entered continuous risk factors into hierarchical regression

models to test both the importance of individual risks and their cumulative impact, as measured by total R^2 (Deater-Deckard, Dodge, Bates, & Pettit, 1998; Greenberg et al., 1999). Both methods examine the influence of cumulative risks, but have slightly different rationales; summing risk factors emphasizes the *number* of risks, while entering multiple risks into a hierarchical regression enables the researcher to test theories about the relative importance of specific risk factors relative to others (e.g., harsh parenting versus maternal education). As the focus of the current study was the overall severity of risk, rather than the relative strength of specific risk factors, the method of summing dichotomized risk factors was used.

A diverse range of distal contextual risk factors have been included in cumulative risk indices, including birth complications (Deater-Deckard et al., 1998), mother's age at first birth (Greenberg et al., 1999; Lengua, Honorado, & Bush, 2007), low maternal education (NICHD Early Child Care Research Network, 2004), single parent status (Deater-Deckard et al., 1998; Sameroff, Seifer, Zax, & Barocas, 1987), parental criminality (Ackerman, Brown, & Izard, 2004; Rutter, 1979; Trentacosta et al., 2008), neighborhood quality (Jaffee et al., 2007; Jones, Forehand, Brody, & Armistead, 2002), and overcrowding (Shaw, Winslow, Owens, & Hood, 1998). More proximal factors are often measured as well, such as harsh parenting (Kaplow, Curran, Dodge, & Conduct Problems Prevention Research, 2002), parental marital conflict (Shaw et al., 1998), negative life events (Lengua et al., 2007), and troubled sibling relationships (Jaffee et al., 2007).

Many studies have demonstrated that cumulative risk is highly associated with negative child outcomes, and that the probability of a negative outcome increases with exposure to the number of risk factors in a linear or multiplicative manner (Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Fergusson & Lynskey, 1996; Gerard & Buehler, 2004; Jones et al., 2002; Rutter,

2000; Seifer et al., 1992). For example, in one study of a sample of four-year-old children, an index of cumulative risk explained three times the variation in outcomes compared to individual risk factors (Sameroff et al., 1987). In fact, cumulative risk scores predicted outcome even after SES, minority status, and maternal IQ were partialled out, suggesting that the type of risk factor matters less than the number of risk factors present (Sameroff, Seifer, Baldwin, & Baldwin, 1993). Perhaps even more startling, another study found that rates of crime recidivism increased drastically as the number of risk factors increased, from 11% recidivism with no family risk to 47% with five risk factors (Stattin, Romelsjo, & Stenbacka, 1997).

An important methodological consideration when computing a cumulative risk score is whether to use risk factors more proximal to the child's development (e.g., harsh parenting) or more distal (e.g., neighborhood disadvantage, parental education). While each method has its strengths, a cumulative risk index that includes more proximal risks precludes the possibility of examining interactions between broader contextual risk and proximal protective factors (Trentacosta et al., 2008). These interactions are important to consider because the probability of a positive outcome in the context of risk may depend not only upon the presence or absence of a protective factor, but also upon the number of risks a child is exposed to (Jaffee et al., 2007). At high levels of cumulative risk, protective factors may be less beneficial, and positive outcomes may become increasingly unlikely (Vanderbilt-Adriance & Shaw, 2008a). For example, a study of a nationally representative sample of 1,116 twin pairs in the UK examining maltreatment found that the protective effects of high IQ and positive temperament disappeared once exposure to cumulative risk was accounted for (Jaffee et al., 2007). Thus children's exposure to cumulative risk can be important to take into consideration, even within the context of other risk factors, such as maltreatment or low SES.

Based on the research reviewed above, the current study examined whether cumulative risk moderated the relation between child and parenting protective factors and low antisocial behavior in a sample of boys already at risk due to low SES. Because the focus was on *proximal* child and parenting protective factors, more distal socio-demographic risk factors were selected to facilitate investigation of interactions between proximal protective factors and distal cumulative risk (Trentacosta et al., 2008). These risks included: 1) teen parent status; 2) single parent status; 3) household overcrowding; 4) low maternal education; 5) household member or biological parent criminal conviction; 6) perinatal complications; and 7) neighborhood disadvantage. These particular risks were selected because they represent distal socio-demographic factors that have been investigated in previous cumulative risk research, and are associated with negative child outcomes (e.g., Beck & Shaw, 2005; NICHD Early Child Care Research Network, 2004; Trentacosta et al., 2008).

1.3 POSITIVE OUTCOME

Positive outcome has been operationalized as either the absence of a negative outcome (e.g., psychopathology) or the presence of a positive outcome, such as academic competence, social competence, or meeting appropriate developmental milestones (Luthar et al., 2000). Arguments can be made for each method, but perhaps the most important consideration is to select an outcome that is relevant to the specific risk factor in question (Luthar & Zelazo, 2003). For example, both low SES and high cumulative risk have been demonstrated as significant risk factors for externalizing behavior (Fergusson, Horwood, & Lynskey, 1994; McLoyd, 1998), thus examining protective factors in the context of these risk factors may yield important information

on mechanisms specific to the development and prevention of externalizing behavior.

However, resilience is not an "all-or-nothing" phenomenon and it should not be assumed that because an individual is doing well in one domain (e.g., antisocial behavior) they are also doing well in other domains (Luthar & Zelazo, 2003). Particularly in the context of severe or high cumulative risk, resilience tends to fluctuate across domains (Vanderbilt-Adriance & Shaw, 2008a). While not all children exposed to high levels of risk have disastrous outcomes, it is also rare for them to completely "escape" the negative effects of risk altogether (D'Imperio et al., 2000; Radke-Yarrow & Brown, 1993; Werner & Smith, 1992).

For example, in two separate samples of ethnically diverse, inner city adolescents exposed to high levels of negative life events, "resilient" youth who were doing well in terms of school-based social competence were also found to have high rates of internal distress (Luthar, 1991; Luthar, Doernberger, & Zigler, 1993). Another study of a similar low-SES sample found that peer-rated sociability prospectively predicted lower indices of school functioning, and that low anxiety in girls was related to decreased performance in school over a six month period (Luthar, 1995). The author concluded that although there was some continuity across domains for academic achievement and teacher-rated classroom behavior, it was also true that adolescents with the best interpersonal or emotional adjustment may also be those who are not doing well in aspects of instrumental functioning.

Longitudinal community studies of antisocial behavior in boys have also demonstrated discontinuity across domains of functioning (Farrington et al., 1988a; Farrington et al., 1988b; Loeber et al., 2007; Moffitt et al., 2002; Stouthamer-Loeber, Wei, Loeber, & Masten, 2004). A longitudinal study of a New Zealand birth cohort found that although there was a group of boys termed "recoveries" because they ceased to exhibit antisocial behavior in adolescence, they were

characterized by higher rates of internalizing disorders in adulthood, with 1/3 formally diagnosed with depressive or anxiety disorders (Moffitt et al., 2002). They tended to be neurotic and socially isolated, obtained lower rates of education and lower-status occupations, and were also more likely to engage in drug or alcohol use. Similarly, the Pittsburgh Youth Study found that even among those who desisted from serious crime in early adulthood, there still appeared to be detrimental effects in the realms of educational attainment, cigarette and marijuana use, unemployment, and anxiety (Loeber et al., 2007; Stouthamer-Loeber et al., 2004).

As these studies show, individuals who are "resilient" in one domain, such as antisocial behavior, may exhibit difficulties in other domains, such as internal distress or educational outcomes. Rutter (2000) has pointed out that a certain amount of discontinuity across domains is to be expected, given that risks and protective factors may be specific to particular outcomes. For example, we would not expect that because someone has avoided cancer they would be protected against coronary artery disease (Rutter, 2000). However, it is also true that the likelihood of discontinuity across domains appears to increase as the level of risk increases, such that middle class children experiencing negative life events are more likely to have positive outcomes across domains than low-income minority children (see Vanderbilt-Adriance & Shaw, 2008a, for a review). In line with previous research, it was hypothesized in the current study that there would be fluctuation in positive outcomes across domains, such that youth who were low on antisocial behavior would not necessarily have positive outcomes on internalizing symptoms or school achievement.

1.4 PROTECTIVE FACTORS

Protective factors are defined as characteristics of the child, family, and wider environment that reduce the negative effects of adversity on child outcome (Masten & Reed, 2002). A wide range of factors, including youth IQ, temperament, parent-child relationship quality, safe neighborhoods, and advantaged SES are associated with positive outcomes in the context of high risk (Masten & Reed, 2002). More recently, researchers have begun examining biologically-oriented protective factors, including physiological and genetic factors, but the number of studies that have accounted for such protective factors or integrated them with more ubiquitously studied psychosocial factors is still quite small (Cicchetti & Curtis, 2007). As Curtis and Cicchetti (2003) have pointed out, a multilevel approach to resilience, investigating both physiological and psychosocial protective processes, has the potential to increase our understanding of the ways in which these systems interact to influence outcome.

Another important issue is to look at differences in how protective factors work across levels of high risk. Examining different patterns of adjustment within a high risk group can help to elucidate the processes that contribute to positive outcomes by highlighting the variation in protective factors and associated outcomes that might be otherwise obscured in a between-group design (Seidman & Pedersen, 2003). Furthermore, comparisons of children at differing levels of high risk can also lead to fine grain distinctions between protective factors that operate in the context of high risk, but not extreme risk.

Indeed, there is evidence that some protective factors may not be beneficial across all levels of risk. For example, there are several studies of children living in poverty that identified protective factors that were only helpful for children who had been exposed to low levels of community violence (Hammack et al., 2004; Kliewer et al., 2004; Li, Nussbaum, & Richards,

2007; Miller et al., 1999; Sullivan, Kung, & Farrell, 2004). Thus, although all of the children in these studies could be considered high risk because of poverty, some were at more extreme risk due to high levels of violence exposure. If these children had been grouped together and compared to a low risk sample of children, the differential benefits of the protective factors within this high risk group would most likely have been missed.

Studies examining neighborhood disadvantage have also found that some protective factors are diminished at higher levels of risk (Silk et al., 2007; Stouthamer-Loeber et al., 2002; Vanderbilt-Adriance & Shaw, 2008b), with differences emerging even between low-income urban neighborhoods and inner city 'project' neighborhoods (Gorman-Smith et al., 2004; Shaw, Criss, Schonberg, & Beck, 2004). Finally, one study took a novel approach of further differentiating a high risk sample of maltreated children based on levels of cumulative risk, finding that the protective effects of high IQ and positive temperament disappeared once cumulative family stressors were examined (Jaffee et al., 2007). This suggests that while certain protective factors may be associated with resilience at low and moderate levels of risk, the magnitude of such associations may be attenuated in the context of high risk.

In short, there is significant evidence that protective factors do not always buffer children from negative outcomes across levels of risk. Importantly, this does not mean that there are no protective factors that benefit children exposed to severe levels of risk; indeed, many of the studies above also found evidence of main effects (i.e., protective factors beneficial at all levels of risk). However, it is important to note that there do appear to be limits to the protective value of some protective factors at extreme levels of risk (e.g., low SES, multiple risks), which suggests that it may be difficult for children exposed to severe adversity to demonstrate a broad pattern of positive outcomes.

One final conceptual issue that has been discussed in the literature is the seemingly arbitrary labeling of various factors as either "risk" or "protective factors." For example, a low IQ may increase the likelihood of a negative outcome, but a high IQ may increase the likelihood of a positive outcome. Is it a risk or a protective factor then? In fact, there are likely very few "pure" risk or protective factors; that is, factors that are only associated with a positive or negative outcome (Masten, 2001). Rather, most factors are on a continuous bipolar scale, in which one end is associated with positive outcomes, while the other is associated with negative outcomes (Masten, 2001; Stouthamer-Loeber et al., 1993; Stouthamer-Loeber et al., 2002). Thus, it is left to the individual researcher to determine how such variables will be classified. Because proximal child and family factors may be more accessible targets for intervention than distal socio-demographic factors, the current study focused on the *protective* function of child and parenting factors. Risk, in turn, was defined by more distal socio-demographic factors such as single parent status and maternal education.

1.5 CHILD PROTECTIVE FACTORS

Child attributes that have been found to be associated with positive outcomes include intelligence, emotion regulation, temperament, coping strategies, locus of control, attention, and genetic influences (Masten & Powell, 2003). As noted above, it is important to keep in mind that although child attributes can be protective in the context of adversity, they are also influenced by external factors, such as family environment and the overall context in which the child lives. As such, they are not entirely "personal" traits. The current study focused on two commonly studied child characteristics, child IQ and the emotion regulation system (e.g., behavioral and

physiological), as well as sleep and testosterone level, which have been less frequently studied as protective factors, but are both associated with externalizing behavior.

1.5.1 Child IQ

IQ is one of the most widely researched and validated protective factors in the child domain (Masten & Coatsworth, 1998). Child IQ has consistently been found to predict a range of positive outcomes, including academic achievement, prosocial behavior, and peer social competence (Masten et al., 1988; Masten et al., 1999), as well as the absence of antisocial behavior (Kandel et al., 1988; Kolvin, Miller, Fleeting, & Kolvin, 1988; White, Moffitt, & Silva, 1989) and other types of psychopathology (Radke-Yarrow & Brown, 1993; Tiet et al., 1998; Tiet et al., 2001; Werner & Smith, 1982, 1992). There are several reasons why IQ may be important in high risk contexts. First, children with high IQs may be more likely to possess effective information-processing and problem-solving skills, which enable them to contend with the stresses and challenges they encounter. Children with higher intellectual skills should also perform better at school; increased academic success is associated with the adoption of social norms and integration into prosocial peer groups (Masten & Coatsworth, 1998). Although some studies have found that IQ was more important in the context of high risk (Kandel et al., 1988; Kolvin et al., 1988; Masten et al., 1988; Masten et al., 1999; Tiet et al., 2001), one study of inner city adolescents found that high intelligence was only related to positive outcomes in the context of *low* negative life events (Luthar, 1991). Thus in this particular study, IQ seemed to lose its ability to protect children once external stress became too high.

1.5.2 Emotion Regulation

Emotion regulation refers to monitoring, evaluating, and modifying the intensity and duration of emotional reactions to accomplish one's goals (Eisenberg et al., 1997a; Thompson & Calkins, 1996). Research demonstrates that a lack of control over emotion is associated with problem behaviors (Calkins & Fox, 2002; Eisenberg et al., 1996), while the ability to manage one's emotional expression predicts more positive social functioning in middle childhood both contemporaneously and longitudinally (Buckner et al., 2003; Eisenberg et al., 1997a; Eisenberg et al., 1997b). Furthermore, studies of resilience have found that factors associated with emotion regulation (e.g., self-help skills, ego control, and ego resiliency) are related to positive adjustment across risk status, and that such factors appear to be especially important in the context of adversity (Cicchetti & Rogosch, 1997; Cicchetti, Rogosch, Lynch, & Holt, 1993; Werner & Smith, 1982, 1992). Children who are adept at managing their emotions may be better able to proactively cope with stressors (Buckner et al., 2003) and thereby decrease the impact of associated negative effects. They may also be less likely to engage in oppositional behavior such as hitting or throwing a tantrum because of their ability to modulate negative emotion. Such children may be less likely to become involved in coercive cycles with their caregivers, and, therefore, may receive more support from their social environment. Across contexts of risk, such children should function better in school and in social relationships because they are able to modulate negativity and emotional expression. The current study focused on the use of active distraction as a coping strategy and low expressions of anger and frustration in a delay-of-gratification context. These factors represent just a couple of the many aspects of emotion regulation, namely the ability to distract oneself and to sustain regulation of negative emotions,

and were selected because of their previously hypothesized and validated associations with positive outcomes in later childhood (Silk et al., 2006; Trentacosta & Shaw, in press).

More recently, in addition to the more ubiquitously studied behavioral measures, researchers have begun investigating physiological markers of emotion reactivity and regulation, such as *vagal tone*. Vagal tone is the variability in heart rate that corresponds to respiration rate (respiratory sinus arrhythmia, RSA), and is a measure of parasympathetic nervous system functioning that is thought to reflect stress vulnerability and reactivity (Porges, 1992; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996). More specifically, polyvagal theory (Porges, 2001, 2003) posits that vagal regulation mediates emotion regulation and enables social engagement. In support of this hypothesis, several studies have found links between vagal tone and aspects of emotion regulation and reactivity (Calkins, 1997; Calkins & Dedmon, 2000; Calkins & Keane, 2004; Eisenberg et al., 1995; Santucci et al., 2008).

High baseline vagal tone is conceptualized as an index of an individual's reactivity and capacity for coping with challenge, while vagal withdrawal (decreases in vagal tone) in response to a challenge or stress allows the individual to respond flexibly to affectively challenging situations (Porges, 2001, 2003; Porges, Doussard-Roosevelt, & Maita, 1994). Indeed, high baseline vagal tone and vagal withdrawal are both associated with lower rates of externalizing behavior (Beauchaine, Gatzke-Kopp, & Mead, 2007; Calkins & Dedmon, 2000; El-Sheikh, 2005; El-Sheikh, Harger, & Whitson, 2001; Katz & Gottman, 1997), internalizing symptoms (Calkins, Blandon, Williford, & Keane, 2007; El-Sheikh, 2005; El-Sheikh et al., 2001; Shannon et al., 2007; Srinivasan, 2006), and physical health problems (Katz & Gottman, 1997). Vagal tone is also associated with adaptive functioning as well, including increased positive social functioning (Eisenberg et al., 1995), adaptive behavior (Sheinkopf et al., 2007), social skills

(Calkins & Keane, 2004) and academic achievement (Katz & Gottman, 1997). Although the majority of studies have examined vagal tone from a risk perspective, there are several studies that have found high baseline vagal tone and vagal withdrawal to be associated with positive outcomes in the context of risks such as marital conflict (El-Sheikh et al., 2001; Katz & Gottman, 1997), cumulative risk (Calkins et al., 2007; Sheinkopf et al., 2007), parental problem drinking (El-Sheikh, 2005), and parental depression (Forbes et al., 2006).

Although vagal tone also has been shown to be important in childhood, it may be particularly important in the transition to adolescence when youth face new challenges and stressors. The transition into adolescence is also marked by an increase in emotional arousal and reactivity, and a shift towards more autonomous regulation as parents and other adults in the environment provide less structuring of emotional response (Steinberg et al., 2006). At the same time, many physiological/biological systems, including those associated with self-control, have not yet fully matured (Steinberg et al., 2006). Investigating the role of one of these systems associated with emotion regulation (i.e., vagal tone) may provide valuable information on the individual differences of youth in this important developmental transition.

1.5.3 Sleep

Sleep is an important physiological process that affects many cognitive, physical, behavioral, and emotional outcomes (Dahl, 1996; Millman, Working Group on Sleepiness in Adolescents and Young Adults, & AAP Committee on Adolescence, 2005). National surveys indicate that the vast majority of youth in middle school and high school do not get adequate sleep (>8 hours), but that youth who do receive adequate sleep are much less likely to endorse fatigue, irritability, depressed mood, and falling asleep in school (National Sleep Foundation, 2006). Other studies

conducted with children and adolescents have found associations between sleep and depressed mood (Roberts, Roberts, & Chen, 2002; Silk et al., 2007), interpersonal functioning (Roberts et al., 2002), school functioning (Millman et al., 2005), inattention and conduct disorder (Morrison, McGee, & Stanton, 1992), and various aspects of antisocial behavior including risky behavior (O'Brien & Mindell, 2005) and substance use (Bootzin & Stevens, 2005; Tynjala, Kannas, & Levalahti, 1997; Wong, Brower, Fitzgerald, & Zucker, 2004). In experimental research studies, associations have also been found between sleep and cognitive functioning among adults (Pilcher & Huffcutt, 1996; Stickgold, 2005). Of interest, one longitudinal study that followed 22 children (ages 6-11) at high familial risk for depression found that ease and quickness of falling asleep and a greater amount of time in deep Stage 4 sleep protected against the development of internalizing disorders into adulthood (Silk et al., 2007).

There are several explanations for these associations between sleep and the outcomes listed above. Sleep appears to be related to cognitive functioning and emotional lability and reactivity (Dinges et al., 1997), such that youth who are receiving adequate amounts of sleep may have more cognitive and emotional resources for coping with stressful or challenging situations. The association between children's sleep quality and youth problem behavior may also be a function of the quality and consistency of the home environment, including caregiving attributes like parental involvement, such that parents who are more involved with their children may be more likely structure bedtimes and create environments that constrain the development of problem behavior (e.g., antisocial activities, affiliation with deviant peers).

While receiving adequate sleep is important across the lifespan, it may be particularly important during the transition to adolescence when biological and social influences combine to increase vulnerability to sleep problems (Millman et al., 2005; Steinberg et al., 2006). While

adolescents appear to actually require *more* sleep during this developmental period of rapid physical, emotional and cognitive change, maturational changes also result in lighter nighttime sleep that is more easily disrupted (Dahl, 1996; Millman et al., 2005). Furthermore, decreased parental control over bedtimes and access to stimulating activities at night (e.g., watching TV, talking with friends, computer time) create the conditions for increasingly delayed bedtimes (Millman et al., 2005; National Sleep Foundation, 2006). In conjunction with early school start times, these factors result in limited time for sleeping, and high rates of inadequate sleep. However, if youth are receiving adequate sleep in the transition to adolescence, they may be less likely to develop such negative sleep trajectories as they continue through adolescence.

1.5.4 Testosterone

Although testosterone has been investigated most commonly as a risk factor for externalizing problems, it is possible that testosterone may serve a protective function (Haglund et al., 2007), with low levels reducing the probability of externalizing problem behavior, particularly in high-risk contexts. Testosterone levels may impact a range of behavioral responses, including responses to threat and stress (Haglund et al., 2007). Arguing from an evolutionary perspective, Archer (2006) suggests that high levels of testosterone are associated with aggressive and dominance-seeking behaviors that may increase reproductive fitness. However, he also points out that such behaviors simultaneously interfere with the ability to maintain long-term relationships and force the individual to deal with the societal and social consequences of engaging in antisocial behavior (e.g., criminal prosecution, getting seriously injured or killed in a fight). Conversely, low levels of testosterone are associated with greater marital satisfaction, higher quality parental relationships, and nurturance (Archer, 2006). Thus while testosterone

does serve an adaptive function, it is also possible that lower levels of testosterone might be associated with some positive outcomes, particularly in relation to low levels of externalizing behavior.

Reviews of the literature show modest positive relations between testosterone and antisocial behavior in adulthood (Archer, 2006; van Goozen, Fairchild, Snoek, & Harold, 2007). Although studies in childhood and adolescence have been somewhat more inconsistent in their results (Raine, 2002), several studies have found support for a similar association during these time periods. For example, one study of 14 year olds found that testosterone in boys was linked with externalizing outcomes, in particular delinquency (Maras et al., 2003), while another study of low income boys found associations between testosterone levels at age 16 and measures of proactive and reactive aggression and self-reported delinquency (van Bokhoven et al., 2006). Furthermore, the latter study found that boys who had consistently high levels of testosterone throughout adolescence were at the highest risk for becoming delinquent convicts (van Bokhoven et al., 2006). Studies of slightly younger children have found similar results regarding aggressive outcomes (Chance, Brown, Dabbs, & Casey, 2000), as well as other outcomes such as social withdrawal (Chance et al., 2000) and moodiness (Strong & Dabbs, 2000). Overall, a review of studies of testosterone and aggression in prepubertal boys found a mean weighted r value of .28 (Archer, 2006).

Thus there is some evidence that testosterone is associated with a range of externalizing outcomes in childhood and adolescence, albeit relatively modestly. However, this relation may not always be direct. Several studies have found that testosterone levels were only related to externalizing outcomes in the context of risk factors, such low SES (Dabbs & Morris, 1990), poor parent-child relationship quality (Booth et al., 2003), or deviant peers (Rowe et al., 2004).

Similar interactions might be evident for low levels of testosterone functioning as a protective factor, as it might only serve this role in the context of high levels of family- and/or community-level adversity.

While testosterone appears to be important in childhood, it may be particularly salient as boys move into adolescence. For example, the challenge hypothesis suggests that given the cost-benefit ratio of high testosterone, testosterone may be selectively increased during times of competition or challenge to status (Archer, 2006; Wingfield, Lynn, & Soma, 2001). The transition to adolescence coincides with pubertal development and subsequent interest in the opposite sex, as well as differentiation of the self and attempts to gain status and recognition within the peer group (Steinberg et al., 2006), all of which may lead to an increase in potential "challenge" situations. Thus, as youth move into adolescence, there may be more variability in testosterone than in childhood.

1.6 PARENTAL INVOLVEMENT AND RELATIONSHIP QUALITY PROTECTIVE FACTORS

Researchers agree that one of the most important resources for normal development is the presence of a caregiver to provide both material resources, such as nutrition and shelter, and more abstract resources, such as love, nurturance, and a sense of safety and security (Masten, 2001). If the caregiving system is functional, this can help children to overcome considerable adversity (Masten, 2001). Resilience research clearly demonstrates the importance of the caregiving system. The current study investigates multiple facets of the parenting environment, including parental nurturance, parental knowledge, and parent-child relationship quality. Parental

nurturance during early childhood and parental knowledge during adolescence both reflect heterotypic attributes of parents who are actively and positively engaged in their children's lives, while parent-child relationship quality provides an overall appraisal of the quality of the parent-child relationship parent.

1.6.1 Parental Warmth and Involvement

Theory suggests that children whose parents are warm and responsive to their offspring's emotional needs, and at the same time firm in setting limits on inappropriate behavior, are better able to self-regulate and explore their environment, particularly during early childhood when physical dependence on parents is greatest (Baumrind, 1971; Thompson, 1998). In early childhood, and to a gradually lesser extent thereafter, parents explicitly teach their children the skills they need to succeed in later developmental tasks, set guidelines for acceptable behavior, and provide opportunities for cognitive and social stimulation (Masten & Coatsworth, 1998). Although some have attributed the association between parenting and child outcome to genetic covariation (e.g., Rowe, 1994), research on parenting interventions supports the conclusion that parenting can play a protective role (Collins et al., 2000).

A wide variety of parenting attributes have been investigated, including warmth, consistent discipline, responsiveness, structure, and monitoring or parental knowledge (Masten & Reed, 2002). One of the factors most consistently associated with positive outcomes, especially in early childhood, is *nurturant*, responsive parenting. Across risk status, various aspects of nurturant or responsive parenting have been associated with lower levels of externalizing/internalizing symptoms (Kim-Cohen et al., 2004; Masten et al., 1988; Masten et al., 1999; Werner & Smith, 1982, 1992) and delinquency (Kolvin et al., 1988), as well as higher

levels of peer social competence (Masten et al., 1999; Wyman et al., 1999) and school achievement (Masten et al., 1999). Few studies have examined the interaction of parenting with risk status, but there is some evidence that parenting may be more strongly associated with child outcomes in the context of high risk (Masten et al., 1999).

Parental knowledge of a child's friends, activities, and whereabouts is another protective factor that has been investigated in relation to externalizing behavior, particularly in older children and adolescents. Parental knowledge can be gained through a variety of methods including child self-disclosure (Kerr & Stattin, 2000; Smetana, 2008; Stattin & Kerr, 2000), as well as parent behaviors such as limit setting or monitoring (Lahey et al., in press; Soenens, Vansteenkiste, Luyckx, & Goossens, 2006). Thus, parental knowledge may reflect parental efforts to monitor and structure their child's environment, and may reduce negative outcomes by reducing exposure to deviant peers or antisocial activities (Dishion & McMahon, 1998). It may also reflect child disclosure, indicating an open family environment where children feel comfortable sharing information with their parents and may also be more willing to accept their influence and direction (Kerr & Stattin, 2000; Soenens et al., 2006). It is likely that both pathways are active (Fletcher, Steinberg, & Williams-Wheeler, 2004; Soenens et al., 2006), and that this construct reflects important aspects of earlier parenting, such as warmth and involvement.

Although the majority of research has focused externalizing or delinquent outcomes (Dishion & McMahon, 1998; Lahey et al., in press; Laird, Pettit, Bates, & Dodge, 2003; Stattin & Kerr, 2000), several studies have also found that high levels of parental monitoring are also associated with decreased likelihood of psychiatric diagnoses (Tiet et al., 1998; Tiet et al., 2001), lower internalizing symptomatology (Kerr & Stattin, 2000; Lansford et al., 2006), and higher

rates of school achievement (Crouter, MacDermid, McHale, & Perry-Jenkins, 1990; Graber et al., 2006; Kerr & Stattin, 2000). Among studies that have examined parental knowledge across different levels of risk, evidence of differential effects has been mixed, with one study finding that parental knowledge was equally helpful across levels of neighborhood risk (Lahey et al., in press), and others finding that it was more important in the context of high neighborhood risk (Beyers, Bates, Pettit, & Dodge, 2003; Vanderbilt-Adriance, Shaw, & Moilanen, 2008). Finally, one study of low SES children found that parental knowledge did not seem to be protective in the context of high levels of community violence exposure (Sullivan et al., 2004).

1.6.2 Parent-Child Relationship Quality

In addition to nurturance and parental knowledge, having a good relationship with a parent may be another important protective factor, preparing the child to engage in healthy, productive relationships with other people in the social environment. In support of this idea, Ingoldsby and colleagues (Ingoldsby, Shaw, & Garcia, 2001) found that having a good relationship with at least one parent was associated with less conflictual relationships with siblings, teachers, and peers. Even among materially privileged children, the absence of a close parent-child relationship is linked with negative outcomes (Luthar & Latendresse, 2005). Indeed, a high quality relationship with at least one parent, characterized by high levels of warmth and openness and low levels of conflict, is associated with positive outcomes across levels of risk and stages of development (Luthar & Latendresse, 2005; Luthar & Sexton, 2007; Owens & Shaw, 2003; Radke-Yarrow & Brown, 1993; Stouthamer-Loeber et al., 2002; Vanderbilt-Adriance & Shaw, 2008b; Werner & Smith, 1982). Several studies, however, have found that qualities of the parent-child relationship are not always related to positive outcomes for youth living in the worst neighborhoods

(Gorman-Smith, Tolan, & Henry, 1999; Shaw et al., 2004), or for low-SES youth exposed to high levels of community violence (Hammack et al., 2004; Kliewer et al., 2004), suggesting that there may be limits to the benefits of this protective factor at the highest level of risk.

2.0 STATEMENT OF PURPOSE

In combination with research on vulnerability, research on positive outcomes in the context of risk can inform theories of psychopathology and competence, as well as guide prevention and intervention efforts (Masten, 2001). Along with increased recognition of the persistent effects of risk and the difficulty of demonstrating positive outcomes in the context of severe risk comes the need for more studies examining within-group differences in high risk samples, the likelihood of positive adaptation across domains, and the role of physiological protective factors in addition to psychosocial and cognitive factors.

The current study aims to advance our understanding of positive adaptation by examining multiple domains of protective factors in two important developmental periods, early childhood (i.e., child IQ, emotion regulation, parental nurturance, parent-child relationship quality) and the transition to adolescence (i.e., child IQ, vagal tone, sleep, testosterone, parental knowledge, parent-child relationship quality), as they relate to low antisocial behavior in adolescence. The sample consists of 310 low income boys followed longitudinally from 1.5 to 17 years and employs a combination of observational, physiological, and questionnaire measures from multiple informants. Although the entire sample could be considered high risk due to socioeconomic conditions, risk was further differentiated via a cumulative risk index of distal socio-demographic factors (e.g., neighborhood disadvantage, single parent status, household overcrowding) from age 1.5 to 12. Antisocial behavior was the main outcome of interest, given

its huge societal and personal costs (Kazdin, 1996); however, the study also examined positive functioning across the domains of internalizing symptoms and school achievement to address the issue of cross-domain adjustment. In addition, as many constructs were measured in both early childhood and the transition to adolescence (i.e., IQ, emotion regulation, parental warmth and involvement, parent-child relationship quality), the study also investigated the benefits of continuity in protective factors in relation to low antisocial behavior during adolescence.

Analyses focused on associations between child and parenting protective factors in both early childhood and the transition to adolescence and a dichotomous measure of low antisocial behavior at ages 15 and 17 (i.e., below median youth-reported delinquency, absence of externalizing diagnoses, *and* the absence of court records). It was hypothesized that protective factors that are present in both early childhood and the transition to adolescence would be more highly associated with low antisocial behavior, but that the benefits of protective factors would be attenuated in the context of high cumulative risk. Finally, it was expected that there would be fluctuation on positive outcomes across domains, such that even youth who were low on antisocial behavior at ages 15 and 17 would not necessarily show positive adaptation in the domains of internalizing symptoms and school achievement.

3.0 HYPOTHESES

3.1 HYPOTHESIS 1A: DIRECT ASSOCIATIONS BETWEEN PROTECTIVE FACTORS IN EARLY CHILDHOOD AND LOW ANTISOCIAL BEHAVIOR

Based on previous research, it was hypothesized that child (i.e., IQ, emotion regulation) and parenting (i.e., parental nurturance, parent-child relationship quality) protective factors measured during early childhood (EC) would be associated with low youth antisocial behavior in adolescence.

3.2 HYPOTHESIS 1B: DIRECT ASSOCIATIONS BETWEEN PROTECTIVE FACTORS IN THE TRANSITION TO ADOLESCENCE AND LOW ANTISOCIAL BEHAVIOR

Based on previous research, it was hypothesized that child (i.e., IQ, vagal tone, sleep, testosterone) and parenting (i.e., parental knowledge, parent-child relationship quality) protective factors measured during the transition to adolescence (TA) would be associated with low youth antisocial behavior in adolescence.

3.3 HYPOTHESIS 2: CONTINUITY AND TIMING OF PROTECTIVE FACTORS IN EARLY CHILDHOOD AND THE TRANSITION TO ADOLESCENCE

It was expected that youth for whom a protective factor was present at two time periods (i.e., EC and the TA) would be more likely to have low antisocial behavior in adolescence than youth for whom a protective factor was absent or only present at one time period. The study also explored whether there are protective factors that appear to be more important during specific developmental time periods (EC vs. TA).

3.4 HYPOTHESIS 3A: INTERACTION BETWEEN PROTECTIVE FACTORS IN EARLY CHILDHOOD AND CUMULATIVE RISK

Based on research pointing to the diminishing benefits of child and parenting protective factors at the highest levels of risk, it was hypothesized that the magnitude of association between protective factors measured in EC (i.e., child IQ, emotion regulation, parental nurturance, parent-child relationship quality) and low antisocial behavior in adolescence would be attenuated in the context of high cumulative risk.

3.5 HYPOTHESIS 3B: INTERACTION BETWEEN PROTECTIVE FACTORS IN THE TRANSITION TO ADOLESCENCE AND CUMULATIVE RISK

Based on research pointing to the diminishing benefits of child and parenting protective factors at the highest levels of risk, it was hypothesized that the magnitude of association between protective factors measured in the TA (i.e., child IQ, vagal tone, sleep, testosterone, parental knowledge, parent-child relationship quality) and low antisocial behavior in adolescence would be attenuated in the context of high cumulative risk.

3.6 HYPOTHESIS 4A: POSITIVE ADJUSTMENT ACROSS DOMAINS

Given evidence within the existing literature that resilience tends to be domain specific, rather than general, it was hypothesized that there would be fluctuation in positive outcomes across domains (i.e., antisocial behavior, internalizing symptoms, school achievement). It was expected that fewer than 50% of youth who were low on antisocial behavior would also demonstrate positive adaptation in the domain of internalizing symptoms or school achievement.

3.7 HYPOTHESIS 4B: THE ROLE OF RISK AND PROTECTIVE FACTORS IN POSITIVE ADJUSTMENT ACROSS DOMAINS

Because research demonstrates that achieving positive outcomes in the context of severe or chronic risk is particularly difficult, it was hypothesized that youth experiencing a higher level of

cumulative risk or a lower number of protective factors would be less likely to demonstrate positive outcomes across domains than those youth with lower levels of cumulative risk or higher levels of protective factors.

4.0 METHOD

4.1 PARTICIPANTS

Participants in this study were part of the Pitt Mother and Child Project (PMCP), a longitudinal study of child vulnerability and resilience in low-income families (Vanderbilt-Adriance & Shaw, 2008b). In 1991 and 1992, 310 infant boys and their mothers were recruited from Allegheny County Women, Infant, and Children (WIC) Nutrition Supplement Clinics when the boys were between 6 and 17 months old. The sample was restricted to boys to increase the likelihood of emergent conduct problems and more serious forms of antisocial behavior during adolescence. At the time of recruitment, 53% of the target children in the sample were European American, 36% were African American, 5% were biracial, and 6% were of other races (e.g., Hispanic American or Asian American). Two-thirds of mothers in the sample had 12 years of education or less. The mean per capita income was $241 per month ($2,892 per year), and the mean Hollingshead SES score was 24.5, indicative of a working class sample. Thus, a large proportion of the families in this study could be considered high risk due to their low socioeconomic status.

Retention rates have generally been high at each of the 12 time points from age 1.5- to 17-years old, with 90-94% of the initial 310 participants completing assessments at ages 5 and 6, some data available on 89% or 275 participants at ages 10, 11, or 12, and some outcome data available on 86% or 268 participants at ages 15 or 17. At the time of the present analyses, five

additional age 17 visits were expected to be scheduled. Age 18 phone calls are currently on-going, and will continue through May 2010. When compared with those who dropped out at earlier time points, participants who had outcome data at ages 15 and/or 17 showed no differences on the CBCL Externalizing or Internalizing factors at ages 2, 3.5, or 5; or on maternal age, income, or educational attainment (*p*s = .21 to .73). To be included in the present analyses, participants needed to have some data available on at least two of three antisocial behavior outcome measures (i.e., self-reported delinquency, externalizing diagnoses, court records).

4.2 PROCEDURES

Target children and their mothers were seen for two- to three-hour visits when the children were ages 1.5, 2, 3.5, 5, 5.5, 6, 8, 10, 11, 12, 15, and 17 years old. Data were collected in the laboratory (ages 1.5, 2, 3.5, 6, 11) and/or at home (ages 2, 5, 5.5, 8, 10, 12, 15, 17). The adolescents were interviewed over the phone for the age 16 and 18 assessments. During home and lab assessments, parents completed questionnaires regarding sociodemographic characteristics, family issues (e.g., parenting, family member's relationship quality), and child behavior. Children were interviewed regarding their own adjustment starting at age 5.5. In addition, parents, other family members (siblings, alternative caregivers), and friends of the target child were videotaped interacting with each other and/or the target child in age-appropriate tasks, including mother-son clean-up tasks in early childhood, sibling play or discussion tasks during preschool and school-age periods, and peer discussion of problematic topics at age 15 and 17. Physiological measures (i.e., vagal tone, testosterone) were collected at the age 12

assessment. Beginning at age 15, court and school records were obtained for participants residing within Allegheny County and the Pittsburgh School District, respectively. During the age 18 phone interview, participants were asked to report their school GPA and school status (e.g., dropped out, graduated, GED). Participants were reimbursed for their time at the end of each assessment.

4.3 MEASURES

To form more generalizable constructs, efforts were made to aggregate across periods close in time and/or informants whenever possible (Patterson, Reid, & Dishion, 1992). In cases in which data for a composited measure was missing at one of two time points or for one of two informants, data from the one data point was used to minimize missing data. When data were missing for a variable, I took a conservative approach and did not impute data, using a list-wise method of deletion to ensure that only subjects with complete data on the analysis variables were entered into that specific analysis.

4.3.1 Child Protective Factors

4.3.1.1 Child IQ

Child intellectual skills were evaluated at the age 5.5 and 11 laboratory assessments using a four-subtest short form of the Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R, Wechsler, 1989) and a two-subtest short form of the Wechsler Intelligence Scale for Children-III (WISC-III, Wechsler, 1991), respectively. The Block Design, Geometric Design,

Information, and Vocabulary subtests from the WPPSI-R and the Block Design and Vocabulary subtests from the WISC-III were selected because of their high average correlation with overall Full Scale IQs (FSIQ) and the high test-retest reliability and internal consistency coefficients of these subtests (Sattler, 1990, 1992). Full Scale IQ (FSIQ) scores at each age were derived according to prorating procedures described by Tellegen and Briggs (1967, cited in Sattler, 1990, 1992).

4.3.1.2 Emotion Regulation

Behavioral observation of emotion regulation. During the age 3.5 visit, mothers and sons engaged in a blocked-goal waiting task, in which the child was required to wait for a cookie for 3 minutes (Gilliom et al., 2002; Marvin, 1977). This task was designed to measure children's coping strategies and ability to regulate affect in a delay-of-gratification context. During the 3 minutes, children had to wait for the cookie with little stimulation to occupy their time. All toys and activities were removed from the room, and the mother was instructed to complete questionnaires. Mothers were also told not to allow the child to have the cookie until the end of the waiting period. At the end of 3 minutes, the examiner signaled to the mother to give the cookie to the child.

The primary objective was to assess child emotion regulation strategies that have previously been associated with positive outcomes in later childhood using this measure, including sustained regulation of negative emotions and the ability to distract oneself (Gilliom et al., 2002; Silk et al., 2006; Trentacosta & Shaw, in press). Thus, the following previously-coded strategies and affective states were coded to generate a factor that differentiates regulated versus dysregulated coping styles (e.g., active distraction versus displays of anger and frustration).

Specifically, strategies were coded based on a system created by Grolnick and colleagues

(Grolnick, Bridges, & Connell, 1996) and adapted by Gilliom and colleagues (Gilliom et al., 2002). The presence or absence of child active distraction was scored in 10-second intervals. Active distraction was defined as purposeful behaviors in which the focus of attention was shifted from the delay object or task, including fantasy play, exploration of the room, singing, talking with mother, or turning lights on and off. Percent agreement with a master coder was 92.5% and kappa was .72. Displays of child anger were also coded from videotape using procedures adapted by Cole and colleagues (Cole, Zahn-Waxler, & Smith, 1994) that identify basic emotions through facial action and vocal quality cues. Intensity of anger was rated in seconds on a scale of 0-3, with 0 indicating "none," 1 indicating "mild," 2 indicating "moderate," and 3 indicating "high". The number of seconds that the child demonstrated mild to high anger was summed to arrive at the total amount of time that the child exhibited some form of anger. Agreement with a master coder was 88% and kappa was .76. There was no coder membership overlap between the active distraction and affect coding teams. Coders were unaware of the study hypotheses. To generate a composite factor that accounted for both strategy use and regulation of anger, the standardized anger expression score (total time) was subtracted from the standardized active distraction score to generate an emotion regulation variable (r = -.39, p < .01).

Physiological measure of emotion regulation. Cardiac vagal activity was assessed at the age 12 home assessment as a measure of physiological emotion reactivity and regulation. Heart rate was recorded using the 3992/2-ER Biolog system, which provides ambulatory recording of ECG sampled at 1000 Hz. and respiration sampled at 5 Hz. Three electrodes were attached to the right and left shoulders, and the left abdomen near the bottom of the ribs, respectively. A Pneumotrace gauge was used to measure the respiration, and it was placed across the front of the

abdomen. Physiological data were recorded continuously throughout a five minute baseline period (i.e., reading a magazine) and the first five minutes of a stress task (i.e., "hot topics" discussion task of frequent issues of disagreement with the primary caregiver). After examining the ECG signals and correcting artifactual R-wave occurrences, the data were processed via the point-process analytic approach used by the PSPAT program (Weber, Molenaar, & van der Molen, 1988). Following standard recommendations, oscillations in heart period occurring within the range of .15-.40 Hz (9-24 cycles per minute), or high-frequency bandwidth (HF), were used as an estimate of parasympathetic (vagal) activity (Berntson et al., 1997; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

As both baseline vagal tone and vagal withdrawal (decreases in vagal tone) during stressful tasks have been found to be predictive of various child outcomes, both were examined. Baseline vagal tone was measured when boys are occupied with the magazine. To create a measure of vagal withdrawal, stress task HF was subtracted from baseline HF to create a measure of change in vagal activity. Positive change scores indicate a decrease from baseline and the presence of vagal withdrawal, while negative change scores indicate an increase from baseline and the absence of vagal regulation (e.g., Calkins et al., 2007). Recent research suggests that it is not necessary to control for individual variation in respiration rate (Denver, Reed, & Porges, 2007), but to ensure that respiration was within the normal range, respiration rates during the baseline condition were counted for 15% of the participants with vagal tone data (n = 34). The average respiration rate per minute was 16.6, which is within the normal range for individuals in this age range (Bates, 1995), and is also within the HF bandwidth (.15 to .4 Hz, or 9-24 breaths per minute).

Baseline vagal tone was controlled for in all analyses involving vagal withdrawal because initial levels may influence the amount of change that is possible (Santucci et al., 2008).

4.3.1.3 Sleep

Sleep amount and sleep quality were assessed via mother report at the age 11 home visit using a modified 8-item version of the Pittsburgh Sleep Quality Index (Buysse et al., 1989). The Pittsburgh Sleep Quality Index demonstrates acceptable test-retest reliability and validity (Buysse et al., 1989). Mothers answered questions on their child's bedtime, amount of sleep, daytime fatigue, difficulty sleeping, and sleep quality over the past month. Sleep amount was calculated by subtracting the number of minutes awake during the night (i.e., initial and middle insomnia, early morning awakening) from the number of minutes spent in bed during the night. Sleep quality was based on an item asking about the child's overall sleep quality in the past month, ranging from 1) very bad to 4) very good.

4.3.1.4 Testosterone

Saliva samples were obtained from the target child at the beginning of the age 12 home assessment, 45 minutes after arrival (immediately prior to discussion task), and 45 minutes after the end of the discussion task. Saliva samples were immediately placed in a cooler with ice for the remainder of the visit, and then transferred to a freezer until they could be sent for processing. Saliva samples were then assayed for testosterone, and testosterone levels were averaged across the three samples.

Time of visit and pubertal development were controlled for in all analyses involving testosterone. Pubertal development was rated by examiners after the visit using an adaptation of the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1988). Examiners rated

youth on a scale from 1 (child-like) to 5 (adult-like) for the following markers of pubertal development: facial development (e.g., facial hair, acne), body proportion (e.g., proportioned vs. disproportioned, muscle development), coordination (e.g., coordinated child-like vs. gawky vs. coordinated adult-like), and voice (e.g., deepening). Based on these items, examiners then assigned a global rating of pubertal development ranging from 1 (prepubertal) to 5 (late pubertal). The global rating was used as the index of pubertal development in all analyses involving testosterone.

4.3.2 Parental Involvement and Relationship Quality Protective Factors

4.3.2.1 Parental Warmth and Involvement

Parental Nurturance. Maternal levels of nurturant, responsive parenting were assessed via observation at age 2 using the Home Observation for Measurement of the Environment (HOME, Caldwell & Bradley, 1984). This commonly used measure combines the use of observational ratings and data gathered from an interview with the parent to generate indices of maternal behavior and quality of the home environment. Each item of the HOME is rated as '0' or '1' based on the item's absence or presence, respectively. Two of the six subscales were aggregated in the present study to create a single measure of Nurturant Parenting (Vanderbilt-Adriance & Shaw, 2008b). The first subscale, the Acceptance subscale, is comprised of eight items assessing maternal response to child misbehavior or distress (e.g., "Parent does not shout at child," "Parent neither slaps nor spanks child during visit"). Two items regarding the family home (i.e., "At least ten books are present and visible," "Family has a pet") were omitted from the scale in the current study because they do not reflect parent-child interactions about misbehavior, rendering this a 6–item scale. The 11-item Emotional/Verbal Responsivity subscale

rates communicative and affective parent-child interactions (e.g., "Parent caresses or kisses child at least once during visit," "Parent responds verbally to child's verbalizations"). Past research has demonstrated inter-observer agreement of .80 and above, as well as internal consistency of subscales in the moderate range (Bradley, 1993). To generate a scale of Maternal Nurturance, items from the 6-item Acceptance and 11-item Emotional/Responsivity scales will be summed. Internal consistency for the Nurturance variable was found to be adequate in the present sample ($\alpha = .74$).

Parental Knowledge. Interviewers asked youth and their primary caregivers a series of questions about parental knowledge and discipline (Dishion, Patterson, Stoolmiller, & Skinner, 1991) at ages 10, 11, and 12 (Moilanen, Shaw, Criss, & Dishion, in press). Both youth report and parent report were used in order to capture potentially different perceptions. The knowledge factor was based on 5 youth-report items and 4 parent-report items that focused on the degree to which parents were informed of boys' whereabouts, plans, and interests. Sample items included "How often does at least one of your parents know where you are after school?" and "In an average week, how often do you talk with your child about what he does with his friends or kids at school?" These items were rated on a five-point response scale, ranging from 1 (*Never or almost never*) to 5 (*Always or almost always*). The scale demonstrated low to acceptable internal consistency at each time point (youth report: $\alpha = .58$ - .71; parent-report: $\alpha = .33$ - .59). Scale scores were computed by summing and averaging responses across assessments, and finally informants ($r = .22$, $p < .01$). In all cases, a higher score indicates greater perceived parental knowledge.

4.3.2.2 Parent-Child Relationship Quality

Parent-child relationship quality (PCRQ) was measured in childhood (age 5) and the transition to adolescence (ages 10, 11 and 12) using the Adult-Child Relationship Scale, an adaptation of the Student-Teacher Relationship Scale (Pianta, Steinberg, & Rollins, 1995). The original questionnaire, which focused on teacher-child relationship quality, was modified to assess maternal perception of openness and conflict in the relationship with their child. The Openness scale consists of 5 items (e.g., "This child likes telling me about himself"; "It's easy to be in tune with what this child is feeling"), and the Conflict scale consists of 10 items (e.g., "This child and I always seem to be struggling with each other"; "This child feels I am unfair to him"). A composite of these two scales (age 5: r = -.39, p < .001; ages 10-12: r = -.50, p < .001) was used to assess parent-child relationship quality. For the early childhood measurement at age 5, the conflict score was subtracted from the openness score. An average of the openness and conflict scores from ages 10-12, respectively, was used to create an overall score for each scale; then the conflict score was subtracted from the openness score to obtain the final score for PCRQ. Internal consistency for the Openness and Conflict scales was .69 and .83, respectively, at age 5, and ranged from .75-.77 and .88-.89, respectively, at ages 10-12.

4.3.3 Cumulative Risk

The cumulative risk index was generated from seven distal socio-demographic indicators, with adversity scores ranging from 0 to 7 depending on how many of the following risk factors were present: 1) teen parent status; 2) single parent status; 3) household overcrowding; 4) low maternal education; 5) household member or biological parent criminal conviction; 6) perinatal complications; 7) neighborhood disadvantage. These risk factors were chosen because they

represent more distal socio-demographic factors that have been investigated in previous cumulative risk research, and are associated with negative child outcomes (Beck & Shaw, 2005; Lengua et al., 2007; NICHD Early Child Care Research Network, 2004; Rutter, 1979; Trentacosta et al., 2008). With the exception of teen parent status and perinatal complications, which would not change over time, risk factors were assessed at each of 10 assessments from age 1.5 to 12.

Primary caregivers reported on *teen parent status* at the 1.5 year assessment, and received a score of '1' scored if they were under 18 years of age at the first child's birth. At each of the ten assessments between age 1.5 and 12, primary caregivers also reported on *single parent status* ('1' = single adult in the home at ≥ 5 assessments); *household overcrowding* ('1' = 4 or more children in the home *or* fewer rooms than people at ≥ 5 assessments); *low maternal education* ('1' = less than a high school degree or no GED at ≥ 5 assessments); and *criminal conviction of household member or biological parent* ('1' = at least one household resident with a criminal conviction since the child's birth). As noted above, a cut-off of ≥ 5 assessments was used for single parent status, household overcrowding, and low maternal education to ensure that the risk factor was present for a significant portion of the child's lifetime.

Neighborhood disadvantage was ascertained by geocoding addresses at each assessment from age 1.5 to 12, and obtaining U.S. census data at the block group level (Vanderbilt-Adriance & Shaw, 2008b). Block group is the smallest unit for which all census data are available, and thus provides the best representation of the proximal neighborhood context a child is exposed to. Addresses were collected from 1991-2004, so both 1990 and 2000 census data were used. For data from assessments collected between 1990 and 1995, the 1990 census data were used; for data from assessments collected between 1996 and 2004, the 2000 census data were used. Based

on methods devised by Wikström and Loeber (2000) and adapted by Winslow (2001), a composite variable of neighborhood disadvantage was generated using the following census block group level variables: 1) median family income, 2) percent families below poverty level, 3) percent households on public assistance, 4) percent unemployed, 5) percent single-mother households, 6) percent African American, 7) percent Bachelor degree and higher. Wikström and Loeber (2000) selected these variables based on previous research investigating neighborhood census structural characteristics associated with antisocial behavior. Support was found for combining these items via factor analysis (Wikstrom & Loeber, 2000). In the present study, these individual variables were standardized, summed, and then averaged (after reverse scoring median family income and percent Bachelor's degree) to create an overall neighborhood disadvantage score for each block group. Neighborhood disadvantage scores at each time point were then averaged, and families were assigned an overall risk score of '1' if their average neighborhood disadvantage score was ≥ 1 SD above the sample mean.

Information on *perinatal complications* was obtained from hospital medical records in the Pittsburgh metropolitan area (Beck & Shaw, 2005). The present study employed the same weighted scale system for perinatal complications used in previous research studies (Beck & Shaw, 2005; Kandel & Mednick, 1991; Raine, Brennan, & Mednick, 1994). This weighted-severity scale was developed by the collaboration of American and Danish obstetricians and pediatric neurologists and was derived from a "logical and clinical approach" as opposed to factor or cluster analysis techniques. For the current study, participants were assigned a score of "1" if they had a weighted severity score of 4 or 5. This cut-off was selected to ensure that significant risk had occurred. Examples of complications that would receive a weighted severity score of 4 include severe preeclampsia and breech fetal presentation, while a weighted severity

score of 5 includes complications such as a ruptured uterus and incomplete fetal extraction. (For a complete list of complications and their associated scores, please see Beck & Shaw, 2005.)

Criterion scores from each risk factor were summed to create the cumulative risk index. To be included in the risk index calculation, participants had to have data available for at least half of the risk factors. Youth with four or more risk factors were collapsed into one group since there were very few participants with more than four risk factors.

4.3.4 Positive Outcomes

4.3.4.1 Low Antisocial Behavior

Youth antisocial behavior was measured via a combination of youth report of delinquency, parent- and youth-reported psychiatric externalizing diagnoses, and court records in late adolescence. Youth were deemed to have positive outcomes within this domain if they meet all of the following three criteria: 1) below median youth report of delinquency at ages 15 and 17; 2) no diagnoses of externalizing disorders at age 15 and 17; *and* 3) no court record. In order to be included in the analyses, youth needed to have some data available on at least two measures.

Youth-Reported Delinquency. Youth-reported delinquency was assessed at age 15 and 17 using the Self-report of Delinquency Questionnaire (SRD, Elliott, Huizinga, & Ageton, 1985). The SRD is a questionnaire that contains 62 items which assess the frequency with which an individual has engaged in aggressive and delinquent behavior, alcohol and drug use, and related offenses during the prior year. Using a 3-point rating scale (1 = never, 2 = once/twice, 3 = more often), youth rated the extent to which they engaged in different types of antisocial activities (e.g., stealing, throwing rocks at people, drug use). Scores were summed and averaged across

assessments. Internal consistency was .91 and .93 at ages 15 and 17, respectively. Youth who were at or below the sample median on the SRD were considered to exhibit positive functioning on this measure.

To check that a median split provided meaningfully separate groups, a logistic regression was computed in which the presence or absence of externalizing diagnoses on the K-SADS was regressed on the median split group described above. There was a significant and positive relation between dichotomized self-reported delinquency group status and the presence of externalizing diagnoses ($B = 1.58$, OR = 4.83, $p < .001$), such that youth in the high self-reported delinquency group were 4.83 times more likely to have received at least one externalizing diagnosis on the K-SADS.

Externalizing Diagnoses. During the age 15 and 17 home visits, primary caregivers and their sons were administered the *Schedule for Affective Disorders and Schizophrenia* (KSADS, Kaufman et al., 1997) by a trained examiner (Trentacosta, Hyde, Shaw, & Cheong, in press). The K-SADS is a semi-structured interview that assesses DSM-IV (American Psychiatric Association, 2000) child psychiatric symptoms over the last year. Examiners privately interviewed the primary caregiver and then the youth about both internalizing (e.g., depression) and externalizing diagnoses (e.g., conduct disorder) and made a clinical judgment about the presence or absence of each symptom. The same examiner interviewed the mother and the youth, with diagnoses made through consensus. To establish reliability, clinical interviewers participated in an intensive training program at the Western Psychiatric Institute and Clinic or were trained by doctoral-level clinical psychology students who had attended this training. All examiners were observed multiple times by more experienced examiners before administering the interview. Additionally, every case in which a youth approached or met diagnostic criteria

was discussed at regularly held interviewing team meetings, which included all other interviewers and a licensed clinical psychologist with 18 years of experience using the K-SADS. For the current study, youth who did not meet criteria for any externalizing disorders (i.e., Attention-Deficit/Hyperactivity Disorder, Oppositional Defiant Disorder, or Conduct Disorder) at either age 15 or 17 were considered to exhibit positive functioning on this measure.

Court Records. To assess each youth's involvement with the legal system, after receiving written permission from primary caregivers, court records were obtained from the primary county where the participants resided (Allegheny, PA) and when available, other counties where participants lived (Trentacosta et al., in press). The court records were obtained on an annual basis and have been most recently collected when all youth were at least 16 years old. Given the two year range of the youths' birthdays, court records were last collected when the youth were between 16.1 and 18.7 years old (mean = 17.6 years). Youth with no petitions against them (equivalent to charges against the youth in this state) were considered to exhibit positive functioning on this measure. Given the lag in time between petition date and disposition hearing (similar to a verdict and sentencing), dispositions could not be used because many cases are still being processed. If court records could not be obtained for a youth (i.e., he lived in a county where data was not obtainable), these data were considered missing (87% of the youth had data). Of those youth with data (i.e., they lived in counties that provided access to court records in response to written permission from parents), 36% had at least one petition against them with a range of 0 – 8 petitions per youth.

Analysis to verify differential group status. As a final check, a one-way ANOVA was computed to determine whether the final low antisocial behavior group significantly differed from the high antisocial behavior group on their average scores on the Self-Report of

Delinquency, number of externalizing diagnoses on the K-SADS, and number of court petitions. The analysis was significant and in the predicted direction for all antisocial variables (*F*s ranged from 48.86 to 113.87, all *p*s < .001), indicating that youth in the low antisocial behavior group had significantly lower scores on all measures.

4.3.4.2 Low Internalizing Symptomatology

Youth internalizing symptomatology was measured at ages 15 and 17 via a combination of youth report of depression and anxiety symptomatology, and parent- and youth-reported psychiatric internalizing diagnoses. Youth were deemed to have positive outcomes in the domain of internalizing symptoms if they met all of the following three criteria: 1) youth-reported depressive symptomatology at or below the median; 2) youth-reported anxiety symptomatology at or below the median; *and* 3) no internalizing diagnoses on the KSADS. In order to be included in analyses involving internalizing behavior, youth needed to have some data available on at least two measures.

Youth-Reported Depressive Symptomatology. Youth self-report of depressive symptoms at ages 15 was measured with a 10-item short form of the Child Depression Inventory (CDI, Kovacs, 1992), and at age 17 with the 21-item Beck Depression Inventory (Beck et al., 1961). For the items on the CDI, youth were presented with a group of three statements and asked to choose the sentence that best describes their feelings in the past two weeks. The CDI has been shown to have adequate reliability and validity (Kazdin et al., 1983), and had a Cronbach's alpha of .66 in the current sample. Similarly, for items on the BDI, youth rated the intensity of depressive symptoms on a 0 (no symptomatology) to 3 (severe symptomatology) scale, and a score is derived by summing these ratings. Reliability and external validity of the BDI are high (Beck, Steer, & Garbin, 1988). In this sample, the internal consistency of the BDI was .82.

The scores from the CDI at age 15 and the BDI at age 17 were standardized and then averaged ($r = .28$, $p < .01$) to create a more generalizable measure of depressive symptomatology. Youth were considered to display positive functioning in this domain if their score was at or below the sample median. To check that a median split provided meaningfully separate groups, a logistic regression was computed in which the presence or absence of internalizing diagnoses on the K-SADS was regressed on the median split group described above. There was a significant and positive relation between dichotomized self-reported depressive symptomatology group status and the presence of internalizing diagnoses ($B = .96$, OR = 2.62, $p < .01$), such that youth in the high self-reported internalizing group were 2.62 times more likely to have received at least one internalizing diagnosis on the K-SADS.

Youth-Reported Anxiety Symptomatology. Youth-reported anxiety symptoms were measured with a 10-item short form of the Multidimensional Anxiety Scale for Children (MASC, March et al., 1997). For the items on the MASC, youth 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 asked to rate how true each statement is for him "recently" on a 4-point scale. The MASC has been shown to have adequate reliability and validity (March et al., 1997). The internal consistency was .76 and .78 at ages 15 and 17, respectively. The scores from age 15 and 17 were averaged to create a more generalizable measure of anxiety symptomatology. Youth were considered to display positive functioning in this domain if their score was at or below the sample median.

To check that a median split provided meaningfully separate groups, a logistic regression was computed in which the presence or absence of internalizing diagnoses on the K-SADS was regressed on the median split group described above. The relation between dichotomized self-

reported depressive symptomatology group status and the presence of internalizing diagnoses approached significance (B = .63, OR = 1.88, $p < .10$), such that youth in the high self-reported internalizing group were almost two times more likely to have received at least one internalizing diagnosis on the K-SADS.

Internalizing Diagnoses. As discussed above, during the age 15 and 17 home visits, primary caregivers and their sons were administered the *Schedule for Affective Disorders and Schizophrenia* (KSADS, Kaufman et al., 1997) by a trained examiner (Trentacosta et al., in press). For the current study, youth who did not meet criteria for any internalizing disorders (i.e., Major Depressive Disorder, Dysthymic Disorder, Generalized Anxiety Disorder, Panic Disorder, Specific Phobia, Social Phobia, Post-Traumatic Stress Disorder, Separation Anxiety Disorder, Obsessive-Compulsive Disorder) at either age 15 or 17 were considered to exhibit positive functioning on this measure.

Analysis to verify differential group status. As a final check, a one-way ANOVA was computed to determine whether the final low internalizing group significantly differed from the high internalizing group on their average scores on the Child Depression Inventory, Beck Depression Inventory, MASC, and number of internalizing diagnoses on the K-SADS. The analysis was significant and in the predicted direction for all internalizing variables (Fs ranged from 19.67 to 68.70, all ps < .001), indicating that youth in the low internalizing group had significantly lower scores on all measures.

4.3.4.3 School Achievement

High school achievement was assessed via official school records and/or self- or parent-report of school retention and cumulative GPA when youth were between 15 and 18 years old. Participants' and their parents provided consent to contact schools for official GPA data. Due to

the difficulty with collecting school data from multiple school districts, data were only obtained from the Pittsburgh Public School District, where the majority of participants reside (collected summer 2008). In addition, youth and their parents were asked to report on youth school retention and cumulative GPA during the age 18 phone interviews (n = 148, as not all participants had reached age 18 by the completion of the current project). Official school data was used to measure GPA when available, with self- or parent-report as an alternate measure for youth who lived outside the Pittsburgh School District or did not have school data available. Official school data were significantly correlated with self- and parent-report GPA (r = .52, p < .001).

Youth who remained in school (or graduated/obtained a GED) *and* had a cumulative GPA of 2.5 or higher were considered to exhibit positive functioning on this measure. A GPA cut-off of 2.5 was selected as this corresponds to average grades in the C+ to B- range, which could be considered "average." It is also a pragmatic cut-off in that it corresponds to the eligibility requirements for the Pittsburgh Promise. The Pittsburgh Promise is a city-wide program which provides scholarships up to $5000 per year to city youth who are attending a technical, two year, or four year college. As many of the youth in the present sample are from low-income families, participation in this scholarship program could enable them to attend college when it would not otherwise be possible.

There were 37 participants who had data on GPA but not school status. According to the criteria above, youth who had a GPA below the 2.5 cut-off would fall into the low school achievement group regardless of their school status, thus they were assigned on the basis of their GPA alone. The remaining 12 participants with missing school status data had GPAs above the

2.5 cut-off, and were assigned to the positive outcome group with the assumption that they would continue to display similar outcomes, and were consequently likely to remain in school.

Analysis to verify differential group status. As a final check, a one-way ANOVA was computed to determine whether the final low school achievement group significantly differed from the high school achievement group on their average school-reported and self/parent-reported GPA. The analysis was significant and in the predicted direction for all school achievement variables (Fs ranged from 50.26 to 121.89, all ps < .001), indicating that youth in the high school achievement group had significantly higher GPAs, regardless of the measure.

5.0 DATA ANALYTIC PLAN

The primary goal of the proposed research was to investigate the relations among child (i.e., IQ, behavioral and physiological emotion regulation, sleep, testosterone) and parenting (i.e., parental nurturance, parental knowledge, parent-child relationship quality) protective factors measured in early childhood (EC) and the transition to adolescence (TA), and low antisocial behavior at ages 15 and 17 (i.e., at or below the median on youth-reported delinquency, no externalizing diagnoses, *and* absence of a court record). Analyses focused on the direct associations among protective factors and low antisocial behavior, the continuity of protective factors over time, the interaction between protective factors and cumulative risk, and the likelihood of positive outcomes across domains (i.e., antisocial behavior, internalizing symptoms, and school achievement). A more detailed description of the analytic strategies that were used to test the hypotheses follows.

6.0 RESULTS

Results are presented in the following sequence: 1) descriptive statistics and intercorrelations for study variables; 2) direct associations between child and parenting protective factors and low antisocial behavior; 3) continuity and timing of protective factors as they predict to low antisocial behavior; 4) interactions between protective factors and cumulative risk; 5) examination of positive outcomes across domains and prediction by cumulative risk and cumulative protective factors.

6.1 DESCRIPTIVE STATISTICS AND BIVARIATE CORRELATIONS

Descriptive statistics for all study variables appear in Tables 1-2. Many of the measures used in the present study were either constructed for the purpose of this study or modified from their original format, making direct comparisons with other samples difficult. However, such comparisons are discussed when possible. For example, the mean IQ at both time points for boys in the present study (Table 1) was approximately 1/3 a SD lower than normative scores (M = 100, SD = 15) (Wechsler, 1989, 1991). Mothers reported that their sons were sleeping an average of 9.10 hours per night at age 11, which is slightly higher than a recent national survey of youth in middle school (M = 8.6 hours) (National Sleep Foundation, 2006). On outcome measures, 32% and 18% of the sample had externalizing and internalizing diagnoses, respectively, at ages

15 and 17 (Table 3). This is higher than other surveys of DSM-IV psychiatric disorders in adolescence, which report overall prevalence rates between 11.5% and 18% (Ford, Goodman, & Meltzer, 2003; Roberts, Roberts, & Xing, 2006). Youth-report of depressive symptomatology on the Beck Depression Inventory at age 17 was in the "minimal depression" range (< 10) (Beck et al., 1988). Finally, both school- and self-reports of GPA were low, with average grades in the C range.

Descriptives on the dichotomized outcome groups for antisocial behavior, internalizing symptomatology, and school achievement are presented in Table 3. Thirty-seven percent of the sample met criteria for low antisocial behavior, while 26% and 39% met the criteria for positive outcomes on internalizing symptomatology and school achievement, respectively.

Table 1: Descriptive Statistics for Predictor Variables

	N	Mean	*SD*	Range
Early Childhood				
WPPSI-R (short)[1]	234	93.81	12.76	56 - 127
Emotion Regulation Task	247	.05	1.56	-6.99 - 1.86
HOME[2] Subscales	291	13.54	3.15	1 - 18
ACRS[3] age 5	279	-2.91	9.26	-40 - 14
Transition to Adolescence				
WISC-III (short)[4]	231	94.94	18.69	51 - 138
Vagal Tone (baseline)	173	.24	.24	0 - 1.56
Vagal Withdrawal	160	.00	.19	-1.24 - 1.05
Sleep (amount in minutes)	186	545.95	68.66	240 - 745
Sleep Quality	195	3.52	.66	1 - 4
Testosterone	150	84.15	60.35	16.57 – 397.77
Parental Knowledge	269	4.06	.46	2.61 - 5
ACRS[3] age 10-12	268	1.06	9.19	-34 – 15
Cumulative Risk 1.5-12	313	1.58	1.19	0 - 4

[1] Wechsler Preschool and Primary Scale of Intelligence – Revised
[2] Home Observation for Measurement of the Environment
[3] Adult-Child Relationship Scale
[4] Wechsler Intelligence Scale for Children – III

Table 2: Descriptive Statistics for Outcome Variables

	N	Mean	*SD*	Range
Self-Report of Delinquency Scale	268	11.21	10.30	0 - 55
Court Petitions	272	.76	1.39	0 - 8
K-SADS[1] externalizing diagnoses	268	.43	.67	0 - 2
Child Depression Inventory (short)	257	1.27	1.79	0 - 10
Beck Depression Inventory	241	4.96	5.69	0 - 35
MASC[2] (short)	268	5.98	4.23	0 - 24
KSADS[2] internalizing diagnoses	268	.20	.45	0 - 2
School-reported GPA	90	1.86	1.07	0 - 3.75
Participant-reported GPA	95	2.46	.75	0 - 3.82

[1] Schedule for Affective Disorders and Schizophrenia
[2] Multidimensional Anxiety Scale for Children

Table 3: Descriptive Statistics for Dichotomized Outcome Groups

	Negative Outcome n (%)	Positive Outcome n (%)
Antisocial Behavior		
Self Report of Delinquency	133 (50%)	135 (50%)
Court Records	99 (36%)	173 (64%)
K-SADS externalizing diagnosis	86 (32%)	182 (68%)
Overall antisocial behavior	168 (63%)	100 (37%)
Internalizing Symptomatology		
Self-report of depressive symptoms	134 (50%)	134 (50%)
MASC (anxiety)	134 (50%)	134 (50%)
K-SADS internalizing diagnoses	48 (18%)	220 (82%)
Overall internalizing symptomatology	199 (74%)	69 (26%)
School Achievement		
GPA at or above 2.5	111 (60%)	74 (40%)
School retention	15 (10%)	133 (90%)
Overall school achievement	113 (61%)	72 (39%)

Bivariate correlations between protective factors, cumulative risk, and ethnicity are presented in Tables 4-5. For protective factors in early childhood, there were significant correlations between child IQ at age 5.5 and maternal nurturance (age 2; $r = .32$, $p < .001$), and between parent-child relationship quality (PCRQ; age 5) and emotion regulation (ER; age 3.5; $r = .17$, $p < .05$) and maternal nurturance ($r = .32$, $p < .001$). Cumulative risk assessed at ages 1.5-12 was significantly and negatively related to child IQ ($r = -.26$, $p < .001$), maternal nurturance ($r = -.21$, $p < .001$), and PCRQ ($r = -.23$, $p < .001$). Ethnicity was significantly related to child IQ ($r = -.26$, $p < .001$), maternal nurturance ($r = -.30$, $p < .001$), and cumulative risk ($r = .32$, $p < .001$).

For protective factors in the transition to adolescence, there were significant correlations between vagal withdrawal at age 12 and child IQ at age 11 ($r = .20$, $p < .05$) and baseline vagal tone ($r = .37$, $p < .001$); between sleep amount and sleep quality at age 11 ($r = .39$, $p < .001$); between parental knowledge at ages 10-12 and child IQ ($r = .16$, $p < .05$); and between parent-child relationship quality at ages 10-12 and sleep amount ($r = .29$, $p < .001$), sleep quality ($r = .21$, $p < .01$), and parental knowledge ($r = .37$, $p < .001$). In contrast with associations in early childhood, cumulative risk at ages 1.5-12 was only significantly related to child IQ ($r = -.30$, $p < .001$). Ethnicity was significantly related to child IQ ($r = -.41$, $p < .001$), testosterone at age 12 ($r = .21$, $p < .05$), and cumulative risk ($r = .32$, $p < .001$).

Given that both cumulative risk and a number of the protective factors in early childhood and the transition to adolescence were significantly associated with ethnicity, all study analyses were computed with and without including ethnicity as a covariate. In the majority of analyses, the results were the same, thus for ease of interpretation results are present without controlling for ethnicity. It is noted in the text, however, when differences emerged after controlling for ethnicity.

Table 4: Intercorrelations Among Protective Factors in Early Childhood, Cumulative Risk, and Ethnicity

	1	2	3	4	5	6
1. Child IQ age 5	.--	.13[a]	.32***	.12[a]	-.26***	-.26***
2. Emotion Regulation age 3.5	.--	.--	.09	.17*	-.05	-.09
3. Maternal Nurturance	.--	.--	.--	.32***	-.211***	-.30***
4. Parent-Child Relationship Quality age 5	.--	.--	.--	.--	-.23***	-.11[a]
5. Cumulative Risk	.--	.--	.--	.--	.--	.32***
6. Ethnicity	.--	.--	.--	.--	.--	.—

[a] $p<.10$; *$p<.05$;**$p<.01$;***$p<.001$

Table 5: Intercorrelations Among Protective Factors in the Transition to Adolescence, Cumulative Risk, and Ethnicity

	1	2	3	4	5	6	7	8	9	10
1. Child IQ age 11	.--	.00	.20*	-.05	-.12	-.10	.16*	-.04	-.30***	-.41***
2. Vagal Tone (baseline)	.--	.--	.37***	-.11	-.06	-.08	.09	-.06	.00	.11
3. Vagal Withdrawal	.--	.--	.--	-.04	-.04	-.03	-.06	-.08	-.02	.03
4. Testosterone	.--	.--	.--	.--	-.01	-.10	-.14[a]	.01	.09	.21*
5. Sleep Amount	.--	.--	.--	.--	.--	.39***	.13[a]	.29***	-.13[a]	.07
6. Sleep Quality	.--	.--	.--	.--	.--	.--	.03	.21**	.01	.04
7. Parental Knowledge	.--	.--	.--	.--	.--	.--	.--	.37***	-.12[a]	-.11
8. P-C Relationship Qual.	.--	.--	.--	.--	.--	.--	.--	.--	-.11[a]	.05
9. Cumulative Risk	.--	.--	.--	.--	.--	.--	.--	.--	.--	.32***
10. Ethnicity	.--	.--	.--	.--	.--	.--	.--	.--	.--	.—

[a] $p<.10$; *$p<.05$;**$p<.01$;***$p<.001$

6.2 HYPOTHESES 1A AND 1B: DIRECT ASSOCIATIONS BETWEEN PROTECTIVE FACTORS AND LOW ANTISOCIAL BEHAVIOR

A series of separate point biserial correlations were computed to test the hypotheses that child and parenting protective factors in early childhood and the transition to adolescence would be directly associated with a dichotomous measure of low antisocial behavior at ages 15 and 17. In line with hypotheses, univariate analyses revealed that maternal nurturance and PCRQ in early childhood (EC) and child IQ, parental knowledge, PCRQ, sleep amount, and sleep quality in the transition to adolescence (TA) were significantly associated with low antisocial behavior in late adolescence (Table 6). The association between child IQ in early childhood and low antisocial behavior was a nonsignificant trend. In contrast with hypotheses, emotion regulation, baseline vagal tone, vagal withdrawal, and testosterone were not significantly related to low antisocial behavior. When partial correlations controlling for ethnicity were run for each protective factor, IQ was no longer a significant predictor of low antisocial behavior at either age, and testosterone became significant.

As a follow-up to the point biserial correlations, three separate multiple logistic regressions were computed to account for overlapping variance between protective factors. When all protective factors in EC were simultaneously entered, PCRQ remained a significant predictor of low antisocial behavior (B = -.04, OR = .96, $p < .05$), but maternal nurturance became nonsignificant (B = -.03, OR = .97, p = ns). When all protective factors in the TA were simultaneously entered (controlling for time of visit and pubertal development, which were included in all analyses involving testosterone), child IQ approached significance (B = -.03, OR

= .97, $p < .10$), but all other protective factors became nonsignificant. These results are not necessary surprisingly given the level of multicollinearity among protective factors (see Tables 4-5 for intercorrelations among these factors). Finally, when all protective factors from both time periods were simultaneously entered (controlling for time of visit and pubertal development), child IQ at age 5.5 was significant (B = -.23, OR = .80, $p < .05$), and emotion regulation (B = 1.55, OR = 4.71, $p < .10$), PCRQ age 5 (B = -.26, OR = .77, $p < .10$), and sleep quality (B = -5.24, OR = .01, $p < .10$) all approached significance. When ethnicity was included in the multiple logistic regressions, results remained the same except that child IQ in the TA was no longer a trend.

Table 6: Correlations Between Protective Factors and Antisocial Behavior

Protective factors	Antisocial behavior
Early Childhood	
Child IQ age 5.5	-.12[a]
Emotion Regulation	.02
Maternal Nurturance	-.15*
Parent-Child Relationship Quality age 5	-.20**
Transition to Adolescence	
Child IQ age 11	-.15*
Vagal Tone (Baseline)	.10
Vagal Withdrawal (controlling for vagal tone baseline)	-.03
Testosterone (controlling for time of visit and pubertal development)	-.14
Parental Knowledge ages 10-12	-.15*
Parent-Child Relationship Quality age 10-12	-.20**
Sleep (amount in minutes)	-.17*
Sleep Quality	-.16*

[a] ***p* < .10; * *p* < .05; ** *p* < .01**

6.3 HYPOTHESIS 2: CONTINUITY AND TIMING OF PROTECTIVE FACTORS IN EARLY CHILDHOOD AND THE TRANSITION TO ADOLESCENCE

To examine the question of whether there is an added value of having continuity in protective factors over time, and whether there are protective factors during specific developmental time periods that appear to be more strongly related to low antisocial behavior in adolescence than others (i.e., EC vs. the TA), protective constructs that were measured at both time points (i.e., IQ, behavioral and physiological emotion regulation, parental warmth and involvement, parent-child relationships quality) were classified as present (75^{th} percentile) or absent (below the 75 percentile) in EC and the TA (Stouthamer-Loeber et al., 2002). Contrast codes were then constructed for each protective construct (Cohen, Cohen, West, & Aiken, 2003) to compare youth with a protective factor at 0 time periods to youth with a protective factor at 1 or 2 time periods (contrast code 1), and to compare youth with a protective factor at 1 versus 2 time periods (contrast code 2). Contrast codes were also constructed for each protective construct to compare youth with a protective factor at 0 or 1 time periods versus youth with a protective factor at 2 time periods (contrast code 3), and to compare youth with a protective factor at 0 versus 1 time period (contrast code 4).

A series of separate logistic regressions predicting antisocial behavior were computed using contrast codes 1 and 2 for each respective protective construct (Table 7). Another series of separate logistic regressions predicting antisocial behavior were computed using contrast codes 3 and 4 for each respective protective construct (Table 7). For the child IQ construct, there was a .22 reduction in the likelihood of membership in the high antisocial group for youth who had this

protective factor at either one or two time periods compared to youth who did not have this protective factor at either time period (B = -.25, OR = .78, $p < .05$). There was also a trend for youth who had higher IQs at both time periods to be more likely to be in the low antisocial group than youth who either did not have this protective factor or only had it at one time period (B = -.24, OR = .79, $p < .10$). For the parent-child relationship quality construct, there was a .19 reduction in the likelihood of membership in the high antisocial group for youth who had this protective factor at either one or two time periods compared to youth who did not have this protective factor at either time period (B = -.22, OR = .81, $p < .05$). None of the other analyses were significant, suggesting that the number of time periods youth had protective factors present did not matter.

Finally, to address the question of whether it matters *which* time period a protective factor is present (EC vs. the TA), dummy codes were created for each protective construct comparing the subgroup of youth who had a protective construct in either EC or the TA but not both (Table 8). There were no significant differences for any of the protective constructs, suggesting that it does not matter whether a protective factor is present in EC or the TA. It should be noted, however, that the *n* for these analyses was substantially smaller than for other analyses as they only included youth who had a protective factor at one time period, limiting the statistical power to detect any differences between groups (i.e., cell sizes ranged from 20 to 64 in these analyses).

When the analyses were rerun controlling for ethnicity, results were similar, with the notable exception of the child IQ analyses which became nonsignificant.

Table 7: Logistic Regression Analyses Predicting Antisocial Behavior from the Number of Time Periods a Protective Factor is Present

Independent Variables	*B*	*SE*	Wald	OR
Child IQ				
1. Contrast Code 1 (0 vs *1or 2* time periods)	-.25	.11	5.60*	.78
Contrast Code 2 (1 vs 2 time periods)	-.23	.24	.93	.79
2. Contrast Code 3 (*0 or 1* vs 2 time periods)	-.24	.14	3.06[a]	.79
Contrast Code 4 (0 vs 1 time period)	-.26	.19	1.91	.78
ER and Vagal Tone (baseline)				
1. Contrast Code 1 (0 vs *1or 2* time periods)	.08	.17	.23	1.09
Contrast Code 2 (1 vs 2 time periods)	.13	.45	.08	1.14
2. Contrast Code 3 (*0 or 1* vs 2 time periods)	.11	.30	.13	1.11
Contrast Code 4 (0 vs 1 time period)	.06	.18	.11	1.06
ER and Vagal Withdrawal				
1. Contrast Code 1 (0 vs *1or 2* time periods)	.00	.17	.00	1.00
Contrast Code 2 (1 vs 2 time periods)	.40	.44	.81	1.49
2. Contrast Code 3 (*0 or 1* vs 2 time periods)	.20	.29	.47	1.22
Contrast Code 4 (0 vs 1 time period)	-.21	.19	1.20	.82
Parental Warmth and Involvement				
1. Contrast Code 1 (0 vs *1or 2* time periods)	-.04	.10	.14	.96
Contrast Code 2 (1 vs 2 time periods)	-.02	.23	.01	.98
2. Contrast Code 3 (*0 or 1* vs 2 time periods)	-.03	.14	.04	.97
Contrast Code 4 (0 vs 1 time period)	-.05	.15	.09	.96
Parent-Child Relationship Quality				
1. Contrast Code 1 (0 vs *1or 2* time periods)	-.22	.09	5.26*	.81
Contrast Code 2 (1 vs 2 time periods)	-.11	.19	.35	.89
2. Contrast Code 3 (*0 or 1* vs 2 time periods)	-.17	.12	1.99	.85
Contrast Code 4 (0 vs 1 time period)	-.27	.15	3.05[a]	.77

[a] $p<.10$; *$p<.05$;**$p<.01$

Table 8: Logistic Regression Analyses Predicting Antisocial Behavior from the Time Period a Protective Factor is Present (Early Childhood vs. Transition to Adolescence)*

Independent Variables	*B*	*SE*	Wald	OR
Child IQ				
Time period	-.61	.64	.92	.54
Emotion Regulation and Vagal Tone (baseline)				
Time period	.66	.56	1.42	1.94
Emotion Regulation and Vagal Withdrawal				
Time period	-.11	.58	.04	.90
Parental Warmth and Involvement				
Time period	.11	.47	.05	1.11
Parent-Child Relationship Quality				
Time period	.16	.53	.10	1.18

* Early childhood dummy code = 0; Transition to adolescent dummy code = 1

6.4 HYPOTHESIS 3A AND 3B: INTERACTION BETWEEN PROTECTIVE FACTORS AND CUMULATIVE RISK

To examine the hypothesis that protective factors would be less beneficial at the highest level of cumulative risk, a series of logistic regressions was computed. Prior to running the main analyses, a series of separate regressions was computed using each individual protective factor and the cumulative risk index to check for problems of multicollinearity. All *VIF* were below 10 (1 - 1.18) and all tolerance statistics were above .10 (.85 - 1), indicating that multicollinearity was not a substantial issue for these data (Cohen et al., 2003).

In each regression equation, one of the centered protective factors was entered first, followed by cumulative risk, and the interaction between the protective factor and cumulative risk. Cumulative risk was not centered because "0" was a meaningful value for this variable (Cohen et al., 2003). For the analysis involving testosterone, the time of day that the visit took place and the youth's pubertal development were entered in the first step as control variables. In the analysis involving vagal withdrawal, baseline vagal tone was entered in the first step as a control variable.

Contrary to study hypotheses, none of the interactions between protective factors in early childhood and cumulative risk were significant (Table 9). In the transition to adolescence, only the interaction between baseline vagal tone and cumulative risk was significant (Table 10; B = 1.80, OR = 6.03, $p < .05$), indicating that the log odds of having antisocial behavior, as predicted by vagal tone, differed according to the level of cumulative risk. Following Hosmer and Lemeshow (2000), the simple slopes of the relation between vagal tone and antisocial behavior

were calculated and graphed at each level of cumulative risk (Figure 1). As can be seen, there is a negative relation between vagal tone and antisocial behavior at the lowest level of risk, essentially no relation when cumulative risk was equal to 1, and increasingly positive relations as cumulative risk rises. Thus, contrary to study hypotheses, high vagal tone is associated with high antisocial behavior for youth at the highest level of risk.

Also of note is that with the exception of testosterone, none of the protective factors were significantly associated with low antisocial behavior when cumulative risk was included in the regression equation. This raises questions about the strength of these protective factors in the context of cumulative risk.

Results did not change when controlling for ethnicity.

Table 9: Logistic Regression Analyses Predicting Antisocial Behavior from Early Childhood Protective Factors with Cumulative Risk as a Moderator

Independent Variables	*B*	*SE*	Wald	OR
Child IQ age 5.5				
Child IQ	-.02	.02	1.08	.98
Cumulative Risk	.27	.14	3.60[a]	1.31
IQ x Cumulative Risk	.01	.01	.28	1.01
Parental Nurturance				
Parental Nurturance	-.01	.11	.00	1.00
Cumulative Risk	.32	.14	5.51*	1.38
Nurturance x Cumulative Risk	-.04	.05	.78	.96
Emotion Regulation				
Emotion Regulation	.07	.14	.27	1.00
Cumulative Risk	.28	.13	5.04*	1.32
Emotion Regulation x Cumulative Risk	-.03	.09	.12	.97
Parent-Child Relationship Quality age 5				
Parent-Child Relationship Quality	-.04	.03	2.03	.96
Cumulative Risk	.24	.12	3.86*	1.27
Parent-Child Relationship Quality x Cum. Risk	.00	.01	.00	1.00

[a] $p<.10$; *$p<.05$

Table 10: Logistic Regression Analyses Predicting Antisocial Behavior from Transition to Adolescence Protective Factors with Cumulative Risk as a Moderator

Independent Variables	*B*	*SE*	Wald	OR
Child IQ age 11				
Child IQ	-.02	.01	2.57	.98
Cumulative Risk	.29	.14	4.38*	1.33
IQ x Cumulative Risk	.01	.01	.80	1.01
Vagal Tone (baseline)				
Vagal Tone (baseline)	-.82	1.24	.44	.44
Cumulative Risk	.53	.16	10.67**	1.70
Vagal Tone x Cumulative Risk	1.80	.90	3.94*	6.03
Vagal Withdrawal				
Vagal Tone (baseline)	1.04	.87	1.45	2.84
Vagal Withdrawal	-.75	1.23	.37	.47
Cumulative Risk	.51	.16	10.11**	1.67
Vagal Withdrawal x Cumulative Risk	.42	.84	.25	1.53
Testosterone				
Time of Visit	.25	.14	3.11[a]	1.29
Pubertal Development	.25	.31	.67	1.28
Testosterone	-.02	.01	4.40*	.98
Cumulative Risk	.67	.19	11.86**	1.95
Testosterone x Cumulative Risk	.01	.00	1.69	1.01
Sleep Amount				
Sleep Amount	.00	.01	.54	1.00
Cumulative Risk	.30	.14	4.38*	1.35
Sleep Amount x Cumulative Risk	.00	.00	.15	1.00
Sleep Quality				
Sleep Quality	-.77	.48	2.58[a]	.46
Cumulative Risk	.34	.14	5.73*	1.41
Sleep Quality x Cumulative Risk	.10	.24	.18	1.11
Parental Knowledge				
Parental Knowledge	-.24	.53	.21	.79
Cumulative Risk	.33	.12	7.27**	1.39
Parental Knowledge x Cumulative Risk	-.25	.28	.81	.78

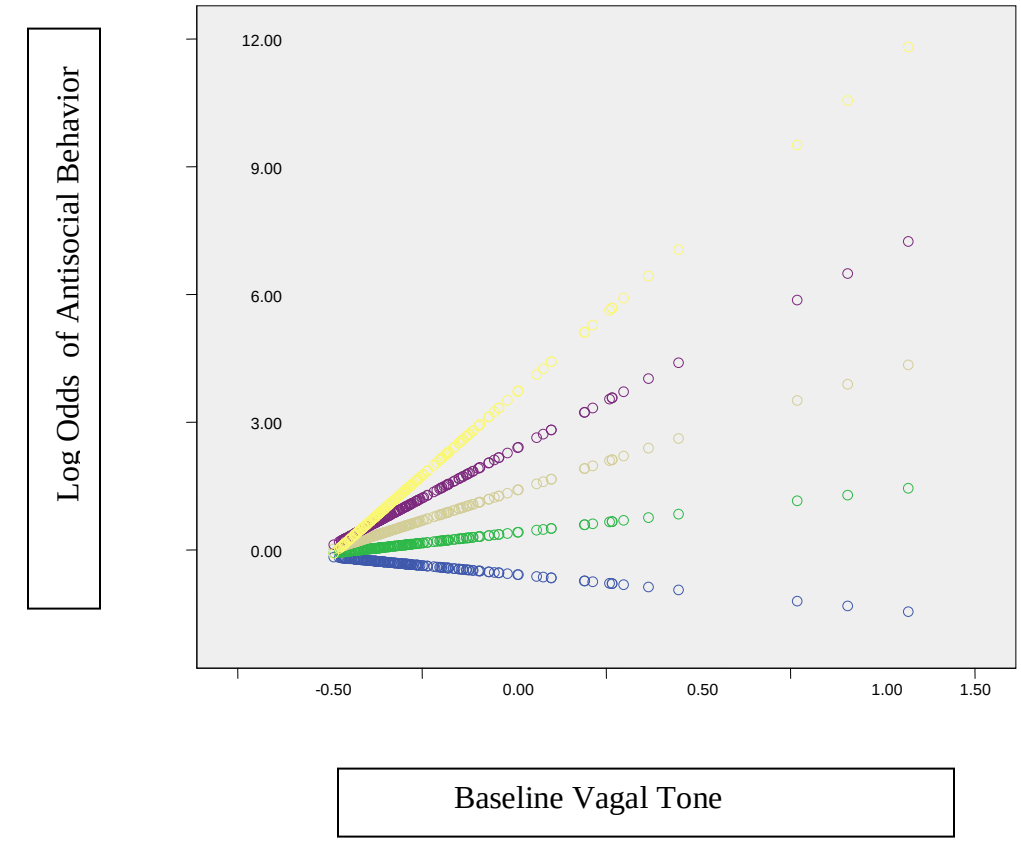
[a] $p<.10$; *$p<.05$;**$p<.01$

Table 10 continued:

Independent Variables	*B*	*SE*	Wald	OR
<u>Parent-Child Relationship Quality age 10-12</u>				
Parent-Child Relationship Quality	-.02	.03	.54	.98
Cumulative Risk	.30	.12	5.77*	1.35
Parent-Child Relationship Quality x Cum. Risk	-.02	.02	1.23	.98

[a] $p<.10$; *$p<.05$;**$p<.01$

Figure 1: Log Odds of Antisocial Behavior Predicted by Vagal Tone and Increasing Levels of Cumulative Risk



6.5 HYPOTHESIS 4A: POSITIVE ADJUSTMENT ACROSS DOMAINS

To examine the hypothesis that positive adaptation would fluctuate across domains, youth were divided into dichotomous groups on their antisocial behavior (at or below the median on self-reported delinquency, no externalizing diagnoses, *and* the absence of a court record), internalizing symptomatology (at or below the median on self-reported depression and anxiety *and* no internalizing diagnoses), and school achievement outcomes (school retention or graduation/GED *and* GPA $\geq$ 2.5), respectively. Cross-tabs were calculated to determine the number of youth who had positive outcomes on antisocial behavior and internalizing problems or school achievement. In line with hypotheses, only 30% of the youth with positive outcomes on antisocial behavior also had positive outcomes on internalizing symptomatology. In contrast with hypotheses, however, 60% of youth with positive outcomes on antisocial behavior also had positive outcomes on school achievement.

Two separate logistic regressions were then computed to determine whether antisocial behavior significantly predicted outcomes on internalizing symptomatology and school achievement, respectively (Table 11). Results supported the findings above, in that antisocial behavior did not significantly predict internalizing symptomatology (B = .35, OR = 1.42, p > .05), but did significantly predict school achievement (B = -1.50, OR = .22, p < .001). In other words, youth in the high antisocial behavior group were no more or less likely to be doing well on internalizing symptomatology, but had a .78 reduction in their likelihood of being in the high school achievement group when compared to the low antisocial behavior group. Results did not change when controlling for ethnicity.

Table 11: Logistic Regression Analyses Predicting Internalizing Symptoms and School Achievement from Antisocial Behavior

Independent Variables	*B*	*SE*	Wald	OR
Predicting Internalizing Problems				
Antisocial Behavior	.35	.29	1.50	1.42
Predicting School Achievement				
Antisocial Behavior	-1.50	.33	21.11***	.22

***$p<.001$

6.6 HYPOTHESIS 4B: THE ROLE OF CUMULATIVE RISK AND PROTECTION IN POSITIVE ADJUSTMENT ACROSS DOMAINS

As a follow-up to Hypothesis 4a, the impact of cumulative risk and cumulative protective factors on cross-domain adaptation was also investigated. First, youth identified as low on antisocial behavior at ages 15 and 17 were further divided into groups based on their outcomes on internalizing problems and school achievement. Four groups were created: 1) negative outcome on antisocial behavior (n = 112); 2) positive outcome on antisocial behavior only (n = 22); 3) positive outcome on antisocial behavior and *either* internalizing or school achievement (n = 33); 4) positive outcome on antisocial behavior, internalizing, and school achievement (n = 15). Second, following Stouthamer-Loeber and colleagues (2002), a cumulative protective factor index was created by summing protective factors classified as present if the score was above the 75th percentile (or below the 25th percentile for testosterone since the low testosterone was

posited as a protective factor). In order to be included in the cumulative protective factor index, youth needed to have data available for at least half of the protective factors. Youth with five or more protective factors were collapsed into one group as there were very few participants with more than five protective factors. Third, nested dichotomy outcome groups were created comparing groups (i.e., groups 1, 2, 3 vs. 4; groups 1, 2 vs. 3, 4; group 1 vs. 2, 3, 4; group 1 vs. 2, 3). Finally, a separate logistic regression was computed for each of the nested dichotomy outcome groups listed above (dependent variable), using either cumulative risk or cumulative protective factors as a predictor.

Cumulative risk significantly differentiated between youth who had a negative outcome on antisocial behavior (group 1) or a positive outcome on antisocial behavior only (group 2) versus youth who had positive outcome on antisocial behavior and at least one other outcome (groups 3 and 4; Table 12; B = -.39, OR = .68, $p < .05$). In other words, for each one unit increase in cumulative risk, youth had a .32 reduction in their likelihood of having a positive outcome on antisocial behavior and at least one other outcome. There were trends for cumulative risk differentiating between youth who had a negative outcome on antisocial behavior (group 1) and all other groups (B = -.24, OR = .79, $p < .10$), and between youth who had a positive outcome on antisocial behavior only versus multiple positive outcomes (B = -.40, OR = .67, $p < .10$).

Cumulative protective factors significantly differentiated between youth who had a negative outcome on antisocial behavior and all other groups (Table 13; B = .30, OR = 1.34, $p < .01$). That is, for each unit increase in cumulative protective factors, youth had a 1.34 increase in their likelihood of having a positive outcome on antisocial behavior (group 2) or antisocial behavior and at least one other outcome (groups 3 and 4). There was a trend, albeit in the

unexpected direction, for cumulative protective factors differentiating between youth who had a positive outcome on antisocial behavior only and those who had multiple positive outcomes (B = -.37, OR = .69, $p < .10$).

The way in which the outcome groups were classified meant that youth in group 1 (negative outcome on antisocial behavior) might actually have a positive outcome on internalizing problems and/or school achievement. Therefore, the analyses above were also rerun using groups classified on the total number of positive outcomes without the emphasis on antisocial behavior: 1) no positive outcomes in any of the three outcome domains (n = 66); 2) one positive outcome (n = 60); 3) two positive outcomes (n = 41); 4) positive outcomes on all three outcome domains (n = 15). Results were similar except that two of the three trends became nonsignificant.

Results were also similar, although attenuated in most cases, when ethnicity was entered as a control variable. For the cumulative risk analyses, the significant finding was reduced to a trend and the trends became nonsignificant. For the cumulative protective factor analyses, the significant finding remained, and the trend was reached significance when controlling for ethnicity.

Table 12: Nested Dichotomy Logistic Regression Analyses Predicting the Number of Positive Outcomes from Cumulative Risk[1]

Independent Variables	*B*	*SE*	Wald	OR
<u>Predicting group 1, 2, 3 vs. 4</u>				
Cumulative Risk	-.24	.24	1.03	.79
<u>Predicting Groups 1, 2 vs. 3, 4</u>				
Cumulative Risk	-.39	.15	6.52*	.68
<u>Predicting Groups 1 vs. 2, 3, 4</u>				
Cumulative Risk	-.24	.13	3.37[a]	.79
<u>Predicting Groups 2 vs. 3, 4</u>				
Cumulative Risk	-.40	.22	3.24[a]	.67

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

[1] 1 = negative outcome on antisocial behavior; 2 = positive outcome on antisocial behavior only; 3 = positive outcome on antisocial behavior and *either* internalizing or school achievement; 4 = positive outcome on antisocial behavior, internalizing, and school achievement

Table 13: Nested Dichotomy Logistic Regression Analyses Predicting the Number of Positive Outcomes from Cumulative Protective Factors[1]

Independent Variables	*B*	*SE*	Wald	OR
Predicting group 1, 2, 3 vs. 4				
Cumulative Protective Factors	.16	.18	.81	1.17
Predicting Groups 1, 2 vs. 3, 4				
Cumulative Protective Factors	.10	.11	.85	1.11
Predicting Groups 1 vs. 2, 3, 4				
Cumulative Protective Factors	.30	.11	7.54**	1.34
Predicting Groups 2 vs. 3, 4				
Cumulative Protective Factors	-.37	.20	3.55[a]	.69

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

[1] 1 = negative outcome on antisocial behavior; 2 = positive outcome on antisocial behavior only; 3 = positive outcome on antisocial behavior and *either* internalizing or school achievement; 4 = positive outcome on antisocial behavior, internalizing, and school achievement

7.0 DISCUSSION

The purpose of the present study was to examine the relations between psychosocial, cognitive, and physiological protective factors in two important developmental periods, early childhood (EC) and the transition to adolescence (TA), as they relate to low antisocial behavior in adolescence in a sample of urban, low SES boys exposed to differing levels of cumulative risk. Support was found for some, but not all, hypotheses. First, in line with hypotheses, several parenting protective factors in EC and the TA, as well as child IQ and sleep amount and quality in the TA, were significantly associated with low antisocial behavior at ages 15 and 17. Second, hypotheses examining the importance of continuity and timing of protective factors were generally not supported by analyses, in that when significant findings emerged they indicated that the presence of a protective factor at *either* time point was associated with low antisocial behavior. In other words, it was equally helpful to have a protective factor present at one or two time periods, in either EC or the TA. Third, when interactions between individual protective factors and cumulative risk were investigated to test whether associations varied according to risk, only baseline vagal tone was found to interact with cumulative risk. Contrary to predictions, *high* vagal tone was associated with high antisocial behavior for youth with two or more cumulative risks. Fourth, in line with hypotheses, youth who were in the low antisocial behavior group at ages 15 and 17 were no more likely to being doing well on internalizing problems than youth in the high antisocial behavior group. Conversely, youth low on antisocial behavior *were*

more likely than youth in the high antisocial behavior group to be doing well on school achievement. Finally, in partial support of hypotheses, cumulative risk and cumulative protective factors appeared to differentiate between youth who had a very low number of positive outcomes (0 or 1) and youth who had multiple positive outcomes (2 or 3). Results were generally similar, albeit attenuated, when ethnicity was entered as a covariate in analyses.

7.1 DIRECT ASSOCIATIONS BETWEEN PROTECTIVE FACTORS AND LOW ANTISOCIAL BEHAVIOR

Corroborating other research studies on protective factors (e.g., Kandel et al., 1988; Masten et al., 1999; Tiet et al., 2001), both child IQ and parenting (i.e., age 2 maternal nurturance, age 5 and age 10-12 parent-child relationship quality, age 10-12 parental knowledge) in EC and the TA were significantly associated with low antisocial behavior at ages 15 and 17, although IQ only attained trend status. In fact, child IQ and parenting are two of the most widely reported protective factors in the literature (Yates, Egeland, & Sroufe, 2003). Nurturant, supportive parenting and a positive, close relationship with a parent may help children to navigate a stressful environment by providing them with valuable interpersonal and social resources (Masten & Coatsworth, 1998), as well as foster internal working models of trust in relationships with peers and adults outside of the family (Thompson, 1998). Similarly, parental knowledge of a youth's friends, activities, and whereabouts, reflecting a high level of engagement and involvement, likely reduces a youth's exposure to deviant peers and promotes youth comfort in disclosing information to parents (Soenens et al., 2006). That parenting protective factors assessed in EC were associated with low antisocial behavior ten to fifteen years later points to the importance of

parenting in EC. In fact, although parenting is important at any stage of child development, it may be particularly important in EC when parents serve as the primary socializing agents and children have less access to influences outside of the home (Dishion & Patterson, 2006).

Sleep amount and quality in the TA were also associated with low antisocial behavior later in adolescence. Sleep is an important physiological process that has the capacity to impact multiple biological and behavioral systems in the body (Dahl, 1996; Millman et al., 2005). As such, it is not surprising to find that it is related to antisocial behavior in the present study. The current results are in line with other research studies showing links between sleep and various aspects of antisocial behavior including risky behavior (O'Brien & Mindell, 2005) and drug and alcohol use (Bootzin & Stevens, 2005; Tynjala et al., 1997). There are several possible explanations for this association. First, lack of quality sleep is associated with impaired cognitive functioning (Pilcher & Huffcutt, 1996) and emotional lability and reactivity (Dinges et al., 1997). Thus, youth who are not obtaining adequate sleep may be more prone to engage in risky behavior or to respond in reactive or aggressive ways in stressful interpersonal situations. Conversely, youth who obtain adequate sleep may be more capable in stressful situations of taking advantage of prosocial coping skills that result in more adaptive and less adverse outcomes.

A second possible explanation is that a third variable may be associated with both sleep and antisocial behavior. For example, youth who are high on sensation seeking may be more likely to engage in delinquent or risky behavior, and may also be less likely to engage in regular sleep routines (O'Brien & Mindell, 2005). It is also possible that parents who provide greater structure and who are more involved in their children's daily routines may influence both antisocial behavior and sleep, with parents high on structure and involvement having children

who show lower levels of antisocial behavior (i.e., via monitoring) and better quality sleep (i.e., from providing structured bedtimes). Additionally, sleep may mediate the relation between family structure and positive child outcomes. For example, parents who provide high levels of structure for their offspring may be more likely to create an environment that promotes good sleep quality, in turn protecting and restoring brain function and development and facilitating a variety of positive outcomes.

7.2 CONTINUITY AND TIMING OF PROTECTIVE FACTORS IN EARLY CHILDHOOD AND THE TRANSITION TO ADOLESCENCE

Contrary to expectations, children with the same or a very similar protective factor present at two time periods did not seem to be any more likely to be low on antisocial behavior at ages 15 and 17 than children with the same protective factor present at only one time period. For child IQ and parent-child relationship quality, results indicated that what mattered most was whether a child had a protective factor during at least one time period, although there was a nonsignificant trend suggesting that children who had high IQs at two time periods were more likely to have low antisocial behavior than those who with a high IQ at none or only one of the time periods. Similarly, it did not seem to matter *when* a protective factor was present (EC vs. the TA). Taken together, these results suggest that having a protective factor at any time point seems to be more important than the continuity or timing of the protective factor. There are very few studies that have even measured protective factors at more multiple time periods (Masten et al., 2004), and it does not appear that any have explicitly examined the issue of continuity and timing in protective factors. Consequently, the current result certainly requires replication and examination with both

similar protective factors and those not available in the present study (e.g., attention, locus of control, family cohesion, school quality). However, if this finding is corroborated, it would provide hopeful news for intervention and prevention projects seeking to increase childhood protective factors because it implies that it is the mere fact of having had a protective factor at all, regardless of whether it occurs during EC or the TA, rather than whether or not there is continuity over time. For example, a close relationship with a parent in EC may continue to have benefit in adolescence even if that relationship deteriorates over time. Conversely, a successful intervention to ameliorate a conflictual parent-child relationship in the TA could have a substantial impact on an adolescent's later functioning, even though the early environment was less than optimal.

This finding also suggests the importance of protective factors in EC. Given the length of time between the measurement of protective factors in EC and outcomes at age 15 and 17, we might expect the magnitude of the relation to be smaller than for protective factors in the TA, which are much closer in time to the assessment of antisocial behavior. In fact, in the current study, protective factors measured in the EC were found to be comparably predictive of adolescent antisocial behavior as protective factors measured during the TA.

7.3 INTERACTION BETWEEN PROTECTIVE FACTORS AND CUMULATIVE RISK

The interaction between baseline vagal tone and cumulative risk was the sole significant interaction, indicating that all other protective factors operated similarly across levels of risk. Although this pattern was results was inconsistent with study hypotheses, it is positive news

because it demonstrates that child IQ, parenting, and sleep are equally beneficial for youth at the highest level of cumulative risk as they are for youth at lower levels of risk. This finding highlights an important role for intervention projects seeking to increase levels of protective factors for children experiencing high levels of risk.

The results also indicate the importance of intervening to reduce cumulative risk. First, in line with several other studies (Dubow, Edwards, & Ippolito, 1997; Farber & Egeland, 1987), youth in this study who were exposed to higher levels of cumulative risk were generally less likely to have protective factors present (see Tables 4-5). Second, cumulative risk was a significant, or near significant, predictor of antisocial behavior in every multiple regression analysis, even after controlling for variance associated with the individual protective factor and the interaction term between cumulative risk and the individual protective factor (see Tables 9-10). In contrast, most individual protective factors were *not* significant after controlling for cumulative risk and the interaction term, highlighting cumulative risk's prominent role in predicting outcomes. This finding is consistent with other research demonstrating stronger effects for cumulative risk than protective factors (Gerard & Buehler, 2004; Jaffee et al., 2007; Sameroff, 1998). From a clinical perspective, the consistency with which cumulative risk was associated with antisocial behavior supports the premise of ecologically-based interventions that target multiple domains of children's ecologies, including child, family, and community-level risk factors (e.g., Dishion et al., 2008; Henggeler, 1999).

On the other hand, the association between baseline vagal tone and low antisocial behavior was found to be moderated by the level of cumulative risk. Contrary to hypotheses, however, the direction of the association was positive for youth at higher levels of cumulative risk (≥ 2), indicating that higher levels of vagal tone were associated with higher levels antisocial

behavior. While the majority of studies have found a negative association between baseline vagal tone and antisocial behavior or aggression (e.g., Beauchaine, 2001; Beauchaine et al., 2007; Calkins & Dedmon, 2000; El-Sheikh, 2005), several studies have found a positive association for certain subgroups (Beauchaine, Gartner, & Hagen, 2000; Dietrich et al., 2007; Scarpa, Fikretoglu, & Luscher, 2000; Scarpa & Ollendick, 2003; Scarpa, Tanaka, & Haden, 2008). For example, Beauchaine and colleagues (2000) found that high vagal tone was associated with increases in aggression among preadolescent males admitted for inpatient hospitalization with comorbid diagnoses of conduct disorder and ADHD. Scarpa and colleagues (2008) found that high vagal tone was associated with higher rates of reactive aggression among preadolescent children who had witnessed community violence.

Although there are fewer studies demonstrating a positive association between vagal tone and antisocial or aggressive behavior, this association may be consistent with findings of low resting heart rate among aggressive individuals (Scarpa & Raine, 2006). In conjunction, these two findings could support the hypothesis of vagotonia (i.e., increased vagal tone) in antisocial individuals (Venables, 1988), as a low heart rate could reflect increased parasympathetic mediation or vagal control. Behaviorally, vagotonia may indicate a reduced fear response and low emotional reactivity, which are posited to be related to antisocial behavior (Scarpa & Raine, 2006; Venables, 1988). Because the direction of the relation between vagal tone and antisocial behavior is somewhat inconsistent, Scarpa and Raine (2006) have suggested that there may be two subgroups of antisocial individuals, one characterized by high vagal tone and low reactivity, and one characterized by low vagal tone and high reactivity.

Indeed, the description of vagotonia above resembles characterizations of highly antisocial or psychopathic individuals, who are differentiated from other antisocial individuals by

physiological underarousal, fearlessness, and lack of empathy (Scarpa & Raine, 2006). In childhood, such individuals are thought to display callous-unemotional traits (e.g., lack of guilt, absence of empathy, shallow and constricted emotions), which are associated with particularly severe forms of antisocial behavior and other features resembling psychopathy (Barry et al., 2000; Frick & White, 2008). To explore the possibility of a relation between psychopathic characteristics and high vagal tone in the current sample, a one-way ANOVA was computed comparing levels of callous-unemotional traits at age 12 in youth with two or more cumulative risks who were either above or below the median on vagal tone at age 12. Results indicated that there was trend for youth high on vagal tone to be more likely to have high levels of callous-unemotional traits than youth who were low on vagal tone, $F(1, 86) = 3.56$, $p < .10$. Although the difference only approached significance, the effect size was $d = .40$, suggesting a moderate effect, and providing some support for a link between callous-unemotional traits and high vagal tone. No such difference was found among youth low on cumulative risk.

While there may be a subgroup of highly antisocial individuals who exhibit high vagal tone, the question remains as to why high vagal tone would only be positively associated with antisocial behavior in the context of high risk. In fact, the interaction between vagal tone and high risk supports a biosocial transactional perspective in which antisocial behavior is heightened in the context of both biological and social risk (Raine, 2002). In support of this perspective, Raine (2002) has reviewed a range of biological factors including genetics, heart rate, prenatal and birth complications, brain function, and hormones that appear to be most strongly associated with antisocial behavior in the context of social risk (e.g., harsh parenting, poverty). In part, this interaction may reflect a "self-righting" tendency, or an ability for individuals to continue to demonstrate fairly positive outcomes in the face of a single risk factor. From this perspective, the

negative effects of risk can be compensated for by positive influences in other areas (e.g., a strong relationship with a parent), and it may be only under circumstances of multiple disadvantage across domains that negative outcomes are commonly seen (Masten, 2001). Thus for youth who may already be at risk for antisocial behavior due to physiological underarousal, high cumulative risk may be the tipping point.

Because there are very few studies examining vagal tone in relation to antisocial behavior in high risk adolescent samples, particularly in the context of cumulative risk, these explanations should be considered tentative at present. Given the somewhat inconsistent findings regarding the direction of the relation between vagal tone and antisocial behavior, future research should continue to investigate this issue, with particular attention to potential moderators such as cumulative risk.

7.4 POSITIVE OUTCOMES ACROSS DOMAINS AND THE ROLE OF CUMULATIVE RISK AND CUMULATIVE PROTECTIVE FACTORS

There was partial support for the hypothesis that there would be fluctuations in positive outcomes across domains; youth in the low antisocial behavior group were no more likely to be low on internalizing problems than youth in the high antisocial behavior group, but they were more likely to be high on school achievement. In some ways, the finding of modest continuity in the presence of low symptoms of antisocial behavior and low levels of internalizing symptoms may seem counterintuitive given that comorbidity between externalizing and internalizing problems tends to be moderate to high across developmental periods of childhood and adolescence (Oland & Shaw, 2005). However, when this finding is considered within the

framework of resilience among youth at high levels of risk, it makes sense such youth would be unlikely to have multiple positive outcomes. In particular, two prominent longitudinal studies of antisocial behavior have found high rates of internalizing problems among individuals with positive outcomes on antisocial behavior (Loeber et al., 2007; Moffitt et al., 2002). Given the inconsistency in positive outcomes across domains among high risk samples, it has been suggested that the literature may benefit from a reconceptualization of resilience as domain-specific (see Vanderbilt-Adriance & Shaw, 2008a, for a review). This perspective recognizes the difficulty of attaining multiple positive outcomes in the context of high risk, and may also increase our understanding of the mechanisms at work for specific outcomes (e.g., low antisocial behavior vs. low internalizing problems).

Interestingly, there was moderate continuity between low antisocial behavior and positive school achievement, such that youth who showed relatively low levels of antisocial behavior were also more likely to be doing well in school. This is consistent with studies that have examined cascading effects between antisocial behavior, school achievement, and internalizing problems (Masten et al., 2005; Moilanen & Shaw, 2009). The authors of these cascade studies have suggested several pathways in which externalizing problems could lead to later academic problems. For example, a youth who demonstrates high levels of antisocial behavior may be more likely to skip school or get into trouble at school, detracting from his ability to attend to classroom instruction, complete assignments, or form strong bonds with either high achieving peers or teachers, both of whom might facilitate learning and investment in academic success. Conversely, a youth low on antisocial behavior may find it easier to obtain help from teachers or make friendships with prosocial peers, leading to better grades and more engagement with the school environment. It is also possible that school achievement could contribute to decreases in

antisocial behavior (Moilanen & Shaw, 2009). Underachieving students are likely to have an early history of antisocial behavior and because such children are often placed in the same classroom, the potential for peer contagion effects can easily occur, resulting in increased rates of antisocial behavior (Dishion, McCord, & Poulin, 1999; Dishion et al., 1991).

Regarding the frequency of *multiple* positive outcomes during adolescence, levels of cumulative risk and cumulative protective factors were both associated with the number of positive outcomes across domains. Cumulative risk significantly differentiated youth who were doing well on antisocial behavior plus at least one other outcome versus youth who were either not doing well on antisocial behavior or were *only* doing well on antisocial behavior. On the other hand, cumulative protective factors significantly differentiated youth who had a negative outcome on antisocial behavior versus those who were doing well on antisocial behavior or antisocial behavior plus at least one other outcome. Thus while risk predicted the presence of *multiple* positive outcomes, cumulative protective factors predicted the presence of any positive outcome (1, 2, or 3 positive outcomes). Levels of cumulative protective and risk factors did not distinguish between youth who had two positive outcomes and youth who had positive outcomes on all three measures, but this may have been because of limited power to detect differences, as the cell size was very small for those with positive outcomes across all three domains (n = 15). Although the small number of youth with positive outcomes across all three domains limited the study's ability to test differences between youth with two versus three positive outcomes, it is also in keeping with the overall expectation that very few youth in this high risk sample would be doing well across domains.

In summary then, youth who had lower levels of cumulative risk were more likely to have multiple positive outcomes than other youth, and youth who had higher levels of protective

factors were more likely to have a positive outcome on antisocial behavior or antisocial behavior plus another positive outcome. These findings are consistent with other studies demonstrating that the probability of a negative outcome increases with the number of risk factors (Fergusson & Lynskey, 1996; Jones et al., 2002; Stattin et al., 1997) and decreases with the number of protective factors (Bradley et al., 1994). Cumulative protective factors have been examined much less frequently than cumulative risk, but one study of premature infants with multiple risks found that children at the highest level of risk needed at least three protective factors to be categorized in the "resilient" group at age 3 (i.e., meeting developmentally appropriate cognitive, health, and behavioral milestones; Bradley et al., 1994). Just as a single risk factor does not always lead to a negative outcome, a single protective factor may not be powerful enough to counteract high levels of risk; an accumulation of protective factors may be necessary, particularly for positive outcomes across domains (Luthar & Zigler, 1992).

7.5 FUTURE DIRECTIONS AND LIMITATIONS

As with any study, there were questions of interest that could not be addressed by the current data set. One of the overall goals of this study was to examine the nature of positive outcomes at high levels of risk. The available data presented the opportunity to examine the frequency of positive outcomes across domains, and the relation between cumulative risk and protection to positive outcomes, both of which are important issues. However, it would have also been interesting to examine fluctuations in positive outcome over time to determine the likelihood of its continuity and to examine the factors related to persistent positive outcomes. As this sample moves into adulthood, it will be interesting to chart their trajectories, and to investigate the

factors that predict continuity and discontinuity in both positive and negative outcomes (e.g., occupational attainment, relationship quality/stability with significant others, antisocial behavior and substance use).

On a related note, two of the three outcomes for positive functioning in the current study were based on the "absence" of problems; although an absence of antisocial behavior or internalizing symptoms is certainly indicative of positive functioning, it would have been preferable to have included more truly "positive" outcomes, such as social competence, close peer relations, or other indicators of positive social or instrumental functioning. Furthermore, although there were several protective factors that were measured in both early childhood and the transition to adolescence, it would have been ideal if this were the case for all factors. Finally, there are many other potential biological or genetic protective factors that would have been interesting to consider, but were not available in the present study. Follow-ups with this sample as they move into young adulthood have been designed to include measures tapping genetics and brain functioning (i.e., fMRI), as well as measures of social (e.g., romantic relationships) and instrumental (e.g., higher education, employment) functioning. Future studies with this sample will likely explore the direct and moderating roles of genetics and brain functioning in influencing positive outcomes in the context of past and ongoing individual and contextual factors.

In addition to the issues listed above, there were a number of limitations in the present study that should be noted. First, the sample was comprised of urban, low income males, and it is unknown whether these findings would generalize to other samples. For example, there is some evidence that pathways to antisocial behavior may be different in girls (Pepler & Craig, 2005),

and as such the relations between protective factors and antisocial behavior may differ by gender.

Second, because this study focused on a low income sample, it is not known whether similar relations between protective factors and antisocial behavior would be found, or whether results regarding the timing and continuity of protective factors or the prevalence of positive outcomes across domains would be similar. Based on other research in lower risk samples, there does seem to be at least some evidence suggesting that cross-domain adaptation might be more frequent in lower risk samples (see Vanderbilt-Adriance & Shaw, 2008a, for a review). Although the current study was purposefully focused on a high risk sample to examine within-group heterogeneity in the trajectories of at-risk youth, it is also possible that more interactions would have been found if lower risk individuals had also been included.

Third, the power to detect differences between groups was limited for certain analyses. In some cases, there were higher rates of missing data for certain variables (i.e., vagal tone, testosterone), while in other cases youth were unevenly distributed across groups. For example, the number of youth who had four or more cumulative risks (n = 21), or five or more protective factors (n = 25) was relatively small, as was the number of youth who had multiple positive outcomes. While this did limit statistical power, the low ns in certain groups are not irrelevant to the focus of the study. In fact, these small group sizes are meaningful in that they are either consistent with theory (i.e., a low number of youth with multiple positive outcomes, decreased likelihood of protective factors in the context of risk), or reflect classification criteria (i.e., a strict definition of cumulative risk, measured over a 10 year time span).

Fourth, official school-reported GPA was only available for 90 participants, so self- and parent-report GPA was also used to create the GPA measure. Self-reported GPA might be less

reliable than school-reported GPA due to recall difficulties, or to desirability effects (i.e., participants not wanting to report low GPAs). As previously noted, however, these two measures of GPA were significantly, albeit modestly, correlated, which provided some support for combining them into a single factor.

Finally, the cut-offs for several of the antisocial behavior and internalizing problem measures were necessarily arbitrary as there are no established norms for positive functioning on these measures (e.g., SRD, CDI short form). For the purposes of this study, positive functioning was defined as at or below the median on these measures, but it is possible that this cut-off could have been either too lenient or too conservative. However, as described in the methods section, these median cut-off groups were compared on externalizing and internalizing diagnoses, respectively, to ensure that these classifications created sufficiently differentiated and valid groups. Indeed, youth who were at or below the median on the SRD were less likely to have externalizing diagnoses, and youth who were at or below the median on the CDI and MASC were less likely to have internalizing diagnoses than youth who were above the median on those measures, providing some support for using the median as a valid cut-off point.

Relatedly, it could be argued that the cut-offs for protective factors (i.e., 75^{th} percentile) were too conservative, eliminating children who had above average scores on protective factors but still below the 75^{th} percentile. A conservative threshold of the 75^{th} percentile was used because of the lack of established criteria for generating cut-offs for protective factors and because this criterion has been used in previous research (Stouthamer-Loeber et al., 1993; Stouthamer-Loeber et al., 2002), permitting comparisons between the current study and prior research.

7.6 SUMMARY AND CLINICAL IMPLICATIONS

The present study advances our understanding of risk and resilience within a high risk sample of urban, low income boys by examining multiple domains of protective factors in two developmental periods as they relate to low antisocial behavior in late adolescence. The findings highlight the importance of "basic human adaptational systems," (Curtis & Cicchetti, 2003; Masten, 2001) such as child IQ, sleep, and parenting protective factors, in protecting against the development of antisocial behavior. The findings also emphasize the inherent difficulty in achieving positive outcomes, particularly across domains, in the context of high risk.

Consistent with prior research, the current findings illustrate the importance of both cumulative risk and protective factors. While it is obviously important to design intervention programs aimed at increasing protective factors within at-risk samples, it is also clear that is perhaps even more important to work towards decreasing initial levels of risk. Contrary to hypotheses, the current study did not find that protective factors were less beneficial at the highest levels of risk. However, analyses did show that the likelihood of having a protective factor was decreased in the context of risk. Moreover, protective factors seemed to lose some of their strength after accounting for the contribution of cumulative risk. Taken together, these results may indicate that while protective factors are important, cumulative risk may be a more powerful predictor of outcome. Consistent with this idea, Stouthamer-Loeber and colleagues (2002) found that the balance of risk and protective factors predicted outcome, but that youth in disadvantaged neighborhoods were still more likely to have negative outcomes even if they had a high number of protective factors. In essence, there is no "magic bullet" that can eliminate the effects of exposure to chronic high risk, and we must work as a society to decrease risk in the first place. The dual goal of decreasing risk and increasing protective factors offers multiple

opportunities to intervene in the lives of at-risk youth, and suggests that targeting multiple domains is more likely to improve outcomes than focusing on single risk or protective factors (e.g., Dishion et al., 2008; Henggeler, 1999).

This study also highlights the importance of examining the interactions between physiological and social factors (e.g., cumulative risk), even if the current findings were unexpected. Physiological and biological factors are only just beginning to be incorporated into research on resilience (Cicchetti & Curtis, 2007), but they hold promise for increasing our understanding of mediators and moderators at work in both positive and negative trajectories of development (Curtis & Cicchetti, 2003). Examining biosocial interactions may help to elucidate which specific protective factors work for whom and under what circumstances. In addition, future research should continue to examine the dual roles of cumulative risk and protective factors in both chronic high risk and lower risk samples, as they relate to positive and negative adaptation across time and domains.

REFERENCES

Ackerman, B. P., Brown, E. D., & Izard, C. E. (2004). The relations between contextual risk, earned income, and the school adjustment of children from economically disadvantaged families. *Developmental Psychobiology, 40*, 204-216.

Ackerman, B. P., Izard, C. E., Schoff, K., Youngstrom, E. A., & Kogos, J. (1999). Contextual risk, caregiver emotionality, and the problem behaviors of six- and seven-year-old children from economically disadvantaged families. *Child Development, 70*, 1415-1427.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th, text revision ed.). Washington, D.C.: American Psychiatric Association.

Appleyard, K., Egeland, B., van Dulmen, M. H., & Sroufe, L. (2005). When more is not better: The role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry, 46*, 235-245.

Archer, J. (2006). Testosterone and human aggression: An evaluation of the challenge hypothesis. *Neuroscience & Biobehavioral Reviews, 30*, 319-345.

Barry, C. T., Frick, P. J., DeShazo, T. M., McCoy, M., Ellis, M., & Loney, B. R. (2000). The importance of callous-unemotional traits for extending the concept of psychopathy to children. *Journal of Abnormal Psychology, 109*, 335-340.

Bates, B. (1995). *A guide to physical examination and history taking* (6th ed.). Philadelphia, PA: Lippincott.

Baumrind, D. (1971). Current patterns of parental authority. *Developmental Psychology, 4*, 1-103.

Beauchaine, T. P. (2001). Vagal tone, development, and Gray's motivational theory: Toward an integrated model of autonomic nervous system functioning in psychopathology. *Development and Psychopathology, 13*, 183-214.

Beauchaine, T. P., Gartner, J., & Hagen, B. (2000). Comorbid depression and heart rate variability as predictors of aggressive and hyperactive symptom responsiveness during inpatient treatment of conduct-disordered, ADHD boys. *Aggressive Behavior, 26*, 425-441.

Beauchaine, T. P., Gatzke-Kopp, L., & Mead, H. K. (2007). Polyvagal Theory and developmental psychopathology: Emotion dysregulation and conduct problems from preschool to adolescence. *Biological Psychology, 74*, 174-184.

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*, 77-100.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 40*, 561-571.

Beck, J. E., & Shaw, D. S. (2005). The influence of perinatal complications and environmental adversity on boys' antisocial behavior. *Journal of Child Psychology and Psychiatry, 46*, 35-46.

Berntson, G. G., Bigger, J. T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M., et al. (1997). Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology, 34*, 623-648.

Beyers, J. M., Bates, J. E., Pettit, G. S., & Dodge, K. A. (2003). Neighborhood structure, parenting processes, and the development of youths' externalizing behaviors: A multilevel analysis. *American Journal of Community Psychology, 31*, 35-53.

Booth, A., Johnson, D. R., Granger, D. A., Crouter, A. C., & McHale, S. (2003). Testosterone and child and adolescent adjustment: The moderating role of parent-child relationships. *Developmental Psychology, 39*, 85-98.

Bootzin, R. R., & Stevens, S. J. (2005). Adolescents, substance abuse, and the treatment of insomnia and daytime sleepiness. *Clinical Psychology Review, 25*, 629-644.

Bradley, R. H. (1993). Children's home environments, health, behavior, and intervention efforts: A review using the HOME inventory as a marker measure. *Genetic, Social, and General Psychology Monographs, 119*, 437-490.

Bradley, R. H., Whiteside, L., Mundfrom, D. J., Casey, P. H., & et al. (1994). Early indications of resilience and their relation to experiences in the home environments of low birthweight, premature children living in poverty. *Child Development, 65*, 346-360.

Buckner, J. C., Mezzacappa, E., & Beardslee, W. R. (2003). Characteristics of resilient youths living in poverty: The role of self-regulatory processes. *Development and Psychopathology, 15*, 139-162.

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193-213.

Caldwell, B. M., & Bradley, R. H. (1984). *Home Observation for Measurement of the Environment*. Little Rock, AR: University of Arkansas at Little Rock.

Calkins, S. D. (1997). Cardiac vagal tone indices of temperamental reactivity and behavioral regulation in young children. *Developmental Psychobiology, 31*, 125-135.

Calkins, S. D., Blandon, A. Y., Williford, A. P., & Keane, S. P. (2007). Biological, behavioral, and relational levels of resilience in the context of risk for early childhood behavior problems. *Development and Psychopathology, 19*, 675-700.

Calkins, S. D., & Dedmon, S. E. (2000). Physiological and behavioral regulation in two-year-old children with aggressive/destructive behavior problems. *Journal of Abnormal Child Psychology, 28*, 103-118.

Calkins, S. D., & Fox, N. A. (2002). Self-regulatory processes in early personality development: A multilevel approach to the study of childhood social withdrawal and aggression. *Development and Psychopathology, 14*, 477-498.

Calkins, S. D., & Keane, S. P. (2004). Cardiac vagal regulation across the preschool period: Stability, continuity, and implications for childhood adjustment. *Developmental Psychobiology, 45*, 101-112.

Chance, S. E., Brown, R. T., Dabbs, J. M., Jr., & Casey, R. (2000). Testosterone, intelligence and behavior disorders in young boys. *Personality and Individual Differences, 28*, 437-445.

Cicchetti, D., & Curtis, W. (2007). Multilevel perspectives on pathways to resilient functioning. *Development and Psychopathology, 19*, 627-629.

Cicchetti, D., & Rogosch, F. A. (1997). The role of self-organization in the promotion of resilience in maltreated children. *Development and Psychopathology, 9*, 797-815.

Cicchetti, D., Rogosch, F. A., Lynch, M., & Holt, K. D. (1993). Resilience in maltreated children: Processes leading to adaptive outcome. *Development and Psychopathology, 5*, 629-647.

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences*. Mahwah, NJ: Lawrence Erlbaum.

Cole, P. M., Zahn-Waxler, C., & Smith, K. (1994). Expressive control during a disappointment: Variations related to preschoolers' behavior problems. *Developmental Psychology, 30*, 835-846.

Collins, W., Maccoby, E. E., Steinberg, L., Hetherington, E., & Bornstein, M. H. (2000). Contemporary research on parenting: The case for nature and nurture. *American Psychologist, 55*, 218-232.

Conrad, M., & Hammen, C. (1993). Protective and resource factors in high- and low-risk children: A comparison of children with unipolar, bipolar, medically ill, and normal mothers. *Development and Psychopathology, 5*, 593-607.

Crouter, A. C., MacDermid, S. M., McHale, S. M., & Perry-Jenkins, M. (1990). Parental monitoring and perceptions of children's school performance and conduct in dual- and single-earner families. *Developmental Psychology, 26*, 649-657.

Curtis, W., & Cicchetti, D. (2003). Moving research on resilience into the 21st century: Theoretical and methodological considerations in examining the biological contributors to resilience. *Development and Psychopathology, 15*, 773-810.

D'Imperio, R. L., Dubow, E. F., & Ippolito, M. F. (2000). Resilient and stress-affected adolescents in an urban setting. *Journal of Clinical Child Psychology, 29*, 129-142.

Dabbs, J. M., & Morris, R. (1990). Testosterone, social class, and antisocial behavior in a sample of 4,462 men. *Psychological Science, 1*, 209-211.

Dahl, R. (1996). The regulation of sleep and arousal: Development and psychopathology. *Development and Psychopathology, 8*, 3-27.

Deater-Deckard, K., Dodge, K. A., Bates, J. E., & Pettit, G. S. (1998). Multiple risk factors in the development of externalizing behavior problems: Group and individual differences. *Development and Psychopathology, 10*, 469-493.

Denver, J. W., Reed, S. F., & Porges, S. W. (2007). Methodological issues in the quantification of respiratory sinus arrhythmia. *Biological Psychology, 74*, 286-294.

Dietrich, A., Riese, H., Sondeijker, F. E., Greaves-Lord, K., van Roon, A. M., Ormel, J., et al. (2007). Externalizing and internalizing problems in relation to autonomic function: A population-based study in preadolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 46*, 378-386.

Dinges, D. F., Pack, F., Williams, K., Gillen, K. A., Powell, J. W., Ott, G. E., et al. (1997). Cumulative sleepiness, mood disturbance and psychomotor vigilance performance decrements during aweek of sleep restricted to 4-5 hours per night. *Sleep: Journal of Sleep Research & Sleep Medicine, 20*, 267-277.

Dishion, T. J., McCord, J., & Poulin, F. (1999). When interventions harm: Peer groups and antisocial behavior. *American Psychologist, 54*, 755-764.

Dishion, T. J., & McMahon, R. J. (1998). Parental monitoring and the prevention of child and adolescent problem behavior: A conceptual and empirical formulation. *Clinical Child and Family Psychology Review, 1*, 61-75.

Dishion, T. J., & Patterson, G. R. (2006). The development and ecology of antisocial behavior in children and adolescents. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (2nd ed., Vol. 3, pp. 503-541). Hoboken, NJ: John Wiley & Sons.

Dishion, T. J., Patterson, G. R., Stoolmiller, M., & Skinner, M. L. (1991). Family, school, and behavioral antecedents to early adolescent involvement with antisocial peers. *Developmental Psychology, 27*, 172-180.

Dishion, T. J., Shaw, D., Connell, A., Gardner, F., Weaver, C., & Wilson, M. (2008). The Family Check-Up with high-risk indigent families: Preventing problem behavior by increasing parents' positive behavior support in early childhood. *Child Development, 79*, 1395-1414.

Dubow, E. F., Edwards, S., & Ippolito, M. F. (1997). Life stressors, neighborhood disadvantage, and resources: A focus on inner-city children's adjustment. *Journal of Clinical Child Psychology, 26*, 130-144.

Eisenberg, N., Fabes, R. A., Guthrie, I. K., Murphy, B. C., & et al. (1996). The relations of regulation and emotionality to problem behavior in elementary school children. *Development and Psychopathology, 8*, 141-162.

Eisenberg, N., Fabes, R. A., Murphy, B., Maszk, P., & et al. (1995). The role of emotionality and regulation in children's social functioning: A longitudinal study. *Child Development, 66*, 1360-1384.

Eisenberg, N., Fabes, R. A., Shepard, S. A., Murphy, B. C., Guthrie, I. K., Jones, S., et al. (1997a). Contemporaneous and longitudinal prediction of children's social functioning from regulation and emotionality. *Child Development, 68*, 642-664.

Eisenberg, N., Guthrie, I. K., Fabes, R. A., Reiser, M., Murphy, B. C., Holgren, R., et al. (1997b). The relations of regulation and emotionality to resiliency and competent social functioning in elementary school children. *Child Development, 68*, 295-311.

El-Sheikh, M. (2005). Does poor vagal tone exacerbate child maladjustment in the context of parental problem drinking? A longitudinal examination. *Journal of Abnormal Psychology, 114*, 735-741.

El-Sheikh, M., Harger, J., & Whitson, S. M. (2001). Exposure to interparental conflict and children's adjustment and physical health: The moderating role of vagal tone. *Child Development, 72*, 1617-1636.

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

Farber, E. A., & Egeland, B. (1987). Invulnerability among abused and neglected children. In E. J. Anthony & B. J. Cohler (Eds.), *The invulnerable child* (pp. 253-288). New York, NY: Guilford.

Farrington, D. P., Gallagher, B., Morley, L., St, & et al. (1988a). Are there any successful men from criminogenic backgrounds? *Psychiatry: Journal for the Study of Interpersonal Processes, 51*, 116-130.

Farrington, D. P., Gallagher, B., Morley, L., St. Ledger, R. J., & West, D. J. (1988b). A twenty-four year follow-up of men from vulnerable backgrounds. In R. L. Jenkins (Ed.), *The abandonment of delinquent behavior* (pp. 156-173). New York, NY: Praeger.

Fergusson, D. M., Horwood, L., & Lynskey, M. T. (1994). The childhoods of multiple problem adolescents: A 15-year longitudinal study. *Journal of Child Psychology and Psychiatry, 35*, 1123-1140.

Fergusson, D. M., & Lynskey, M. T. (1996). Adolescent resiliency to family adversity. *Journal of Child Psychology and Psychiatry, 37*, 281-292.

Fletcher, A. C., Steinberg, L., & Williams-Wheeler, M. (2004). Parental influences on adolescent problem behavior: Revisiting Stattin and Kerr. *Child Development, 75*, 781-796.

Forbes, E. E., Fox, N. A., Cohn, J. F., Galles, S. F., & Kovacs, M. (2006). Children's affect regulation during a disappointment: Psychophysiological responses and relation to parent history of depression. *Biological Psychology, 71*, 264-277.

Ford, T., Goodman, R., & Meltzer, H. (2003). The British Child and Adolescent Mental Health Survey 1999: The prevalence of DSM-IV disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 42*, 1203-1211.

Frick, P. J., & White, S. F. (2008). Research review: The importance of callous-unemotional traits for developmental models of aggressive and antisocial behavior. *Journal of Child Psychology and Psychiatry, 49*, 359-375.

Gerard, J. M., & Buehler, C. (2004). Cumulative environmental risk and youth maladjustment: The role of youth attributes. *Child Development, 75*, 1832-1849.

Gilliom, M., Shaw, D. S., Beck, J. E., Schonberg, M. A., & Lukon, J. L. (2002). Anger regulation in disadvantaged preschool boys: Strategies, antecedents, and the development of self-control. *Developmental Psychology, 38*, 222-235.

Gorman-Smith, D., Henry, D. B., & Tolan, P. H. (2004). Exposure to community violence and violence perpetration: The protective effects of family functioning. *Journal of Clinical Child and Adolescent Psychology, 33*, 439-449.

Gorman-Smith, D., Tolan, P. H., & Henry, D. (1999). The relation of community and family to risk among urban-poor adolescents. In P. Cohen, C. Slomkowski & L. N. Robins (Eds.), *Historical and geographical influences on psychopathology* (pp. 349-367). Mahwah, NJ: Lawrence Erlbaum.

Graber, J. A., Nichols, T., Lynne, S. D., Brooks-Gunn, J., & Botvin, G. J. (2006). A longitudinal examination of family, friend, and media influences on competent versus problem behaviors among urban minority youth. *Applied Developmental Science, 10*, 75-85.

Greenberg, M. T. (2006). Promoting resilience in children and youth: Preventive interventions and their interface with neuroscience. *Annals of the New York Academy of Sciences, 1094*, 139-150.

Greenberg, M. T., Lengua, L. J., Coie, J. D., Pinderhughes, E. E., Bierman, K., Dodge, K. A., et al. (1999). Predicting developmental outcomes at school entry using a multiple-risk model: Four American communities. *Developmental Psychology, 35,* 403-417.

Grolnick, W. S., Bridges, L. J., & Connell, J. P. (1996). Emotion regulation in two-year-olds: Strategies and emotional expression in four contexts. *Child Development, 67,* 928-941.

Haglund, M., Nestadt, P., Cooper, N., Southwick, S., & Charney, D. (2007). Psychobiological mechanisms of resilience: Relevance to prevention and treatment of stress-related psychopathology. *Development and Psychopathology, 19,* 889-920.

Hammack, P. L., Richards, M. H., Luo, Z., Edlynn, E. S., & Roy, K. (2004). Social support factors as moderators of community violence exposure among inner-city African American young adolescents. *Journal of Clinical Child and Adolescent Psychology, 33,* 450-462.

Henggeler, S. W. (1999). Multisystemic therapy: An overview of clinical procedures, outcomes, and policy implications. *Child Psychology and Psychiatry Review, 4,* 2-10.

Hosmer, D. W., & Lemeshow, S. (2000). *Applied Logistic Regression* (2nd ed.). New York, NY: John Wiley & Sons.

Ingoldsby, E. M., Shaw, D. S., & Garcia, M. M. (2001). Intrafamily conflict in relation to boys' adjustment at school. *Development and Psychopathology, 13,* 35-52.

Jaffee, S. R., Caspi, A., Moffitt, T. E., Polo-Tomas, M., & Taylor, A. (2007). Individual, family, and neighborhood factors distinguish resilient from non-resilient maltreated children: A cumulative stressors model. *Child Abuse & Neglect, 31,* 231-253.

Jones, D. J., Forehand, R., Brody, G., & Armistead, L. (2002). Psychosocial adjustment of African American children in single-mother families: A test of three risk models. *Journal of Marriage and Family, 64,* 105-115.

Kandel, E., & Mednick, S. (1991). Perinatal complications predict violent offending. *Criminology, 29,* 519-529.

Kandel, E., Mednick, S. A., Kirkegaard-Sorensen, L., Hutchings, B., & et al. (1988). IQ as a protective factor for subjects at high risk for antisocial behavior. *Journal of Consulting and Clinical Psychology, 56,* 224-226.

Kaplow, J. B., Curran, P. J., Dodge, K. A., & Conduct Problems Prevention Research, G. (2002). Child, parent, and peer predictors of early-onset substance use: A multisite longitudinal study. *Journal of Abnormal Child Psychology, 30,* 199-216.

Katz, L. F., & Gottman, J. M. (1997). Buffering children from marital conflict and dissolution. *Journal of Clinical Child Psychology, 26,* 157-171.

Kaufman, J., Birmaher, B., Brent, D., Rao, U., & et al. (1997). Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*, 980-988.

Kazdin, A. E. (1996). *Conduct disorders in childhood and adolescence* (2nd ed.). Thousand Oaks, CA: Sage.

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*, 504-510.

Kerr, M., & Stattin, H. (2000). What parents know, how they know it, and several forms of adolescent adjustment: Further support for a reinterpretation of monitoring. *Developmental Psychology, 36*, 366-380.

Kim-Cohen, J., Moffitt, T. E., Caspi, A., & Taylor, A. (2004). Genetic and environmental processes in young children's resilience and vulnerability to socioeconomic deprivation. *Child Development, 75*, 651-668.

Kliewer, W., Cunningham, J. N., Diehl, R., Parrish, K. A., Walker, J. M., Atiyeh, C., et al. (2004). Violence exposure and adjustment in inner-city youth: Child and caregiver emotion regulation skill caregiver-child relationship quality, and neighborhood cohesion as protective factors. *Journal of Clinical Child and Adolescent Psychology, 33*, 477-487.

Kolvin, I., Miller, J. W., Fleeting, M., & Kolvin, P. A. (1988). Risk/protective factors for offending with particular reference to deprivation. In M. Rutter (Ed.), *Studies of psychosocial risk: The power of longitudinal data* (pp. 77-95). Cambridge: Cambridge University Press.

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

Lahey, B. B., Van Hulle, C. A., D'Onofrio, B. M., Rodgers, J. L., & Waldman, I. D. (in press). Is parental knowledge of their adolescent offspring's whereabouts and peer associations spuriously associated with offspring delinquency? *Journal of Abnormal Child Psychology*.

Laird, R. D., Pettit, G. S., Bates, J. E., & Dodge, K. A. (2003). Parents' monitoring-relevant knowledge and adolescents' delinquent behavior: Evidence of correlated developmental changes and reciprocal influences. *Child Development, 74*, 752-768.

Lansford, J. E., Malone, P. S., Stevens, K. I., Dodge, K. A., Bates, J. E., & Pettit, G. S. (2006). Developmental trajectories of externalizing and internalizing behaviors: Factors underlying resilience in physically abused children. *Development and Psychopathology, 18*, 35-55.

Lengua, L. J., Honorado, E., & Bush, N. R. (2007). Contextual risk and parenting as predictors of effortful control and social competence in preschool children. *Journal of Applied Developmental Psychology, 28*, 40-55.

Li, S. T., Nussbaum, K. M., & Richards, M. H. (2007). Risk and protective factors for urban African American youth. *American Journal of Community Psychology, 39*, 21-35.

Loeber, R., Pardini, D. A., Stouthamer-Loeber, M., & Raine, A. (2007). Do cognitive, physiological, and psychosocial risk and promotive factors predict desistance from delinquency in males? *Development and Psychopathology, 19*, 867-887.

Luthar, S. S. (1991). Vulnerability and resilience: A study of high-risk adolescents. *Child Development, 62*, 600-616.

Luthar, S. S. (2006). Resilience in development: A synthesis of research across five decades. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology: Risk, disorder, and adaptation* (2nd ed., Vol. 3, pp. 739-795). Hoboken, NJ: John Wiley & Sons, Inc.

Luthar, S. S., & Cicchetti, D. (2000). The construct of resilience: Implications for interventions and social policies. *Development and Psychopathology, 12*, 857-885.

Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development, 71*, 543-562.

Luthar, S. S., Doernberger, C. H., & Zigler, E. (1993). Resilience is not a unidimensional construct: Insights from a prospective study of inner-city adolescents. *Development and Psychopathology, 5*, 703-717.

Luthar, S. S., & Latendresse, S. J. (2005). Comparable "risks" at the socioeconomic status extremes: Preadolescents' perceptions of parenting. *Development and Psychopathology, 17*, 207-230.

Luthar, S. S., & Sexton, C. C. (2007). Maternal drug abuse versus maternal depression: Vulnerability and resilience among school-age and adolescent offspring. *Development and Psychopathology, 19*, 205-225.

Luthar, S. S., & Zelazo, L. B. (2003). Research on resilience: An integrative review. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 510-549). New York, NY: Cambridge University Press.

Luthar, S. S., & Zigler, E. (1992). Intelligence and social competence among high-risk adolescents. *Development and Psychopathology, 4*, 287-299.

Maras, A., Laucht, M., Gerdes, D., Wilhelm, C., Lewicka, S., Haack, D., et al. (2003). Association of testosterone and dihydrotestosterone with externalizing behavior in adolescent boys and girls. *Psychoneuroendocrinology, 28*, 932-940.

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*, 554-565.

Marvin, R. S. (1977). An ethological-cognitive model for the attenuation of mother-infant attachment behavior. In T. M. Alloway, L. Krames & P. Pliner (Eds.), *Advances in the study of communication and affect* (Vol. 3: The development of social attachments, pp. 25-60). New York, NY: Plenum.

Masten, A. S. (2001). Ordinary magic: Resilience processes in development. *American Psychologist, 56*, 227-238.

Masten, A. S., Burt, K. B., Roisman, G. I., Obradovic, J., Long, J. D., & Tellegen, A. (2004). Resources and resilience in the transition to adulthood: Continuity and change. *Development and Psychopathology, 16*, 1071-1094.

Masten, A. S., Garmezy, N., Tellegen, A., Pellegrini, D. S., & et al. (1988). Competence and stress in school children: The moderating effects of individual and family qualities. *Journal of Child Psychology and Psychiatry, 29*, 745-764.

Masten, A. S., Hubbard, J. J., Gest, S. D., Tellegen, A., Garmezy, N., & Ramirez, M. (1999). Competence in the context of adversity: Pathways to resilience and maladaptation from childhood to late adolescence. *Development and Psychopathology, 11*, 143-169.

Masten, A. S., & Powell, J. L. (2003). A resilience framework for research, policy, and practice. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 1-25). New York, NY: Cambridge University Press.

Masten, A. S., & Reed, M. J. (2002). Resilience in development. In C. R. Snyder & S. J. Lopez (Eds.), *Handbook of positive psychology* (pp. 74-88). New York, NY: Oxford University Press.

Masten, A. S., Roisman, G. I., Long, J. D., Burt, K. B., Obradovic, J., Riley, J. R., et al. (2005). Developmental Cascades: Linking Academic Achievement and Externalizing and Internalizing Symptoms Over 20 Years. *Developmental Psychology, 41*, 733-746.

McLoyd, V. C. (1998). Socioeconomic disadvantage and child development. *American Psychologist, 53*, 185-204.

Miller, L. S., Wasserman, G. A., Neugebauer, R., Gorman-Smith, D., & Kamboukos, D. (1999). Witnessed community violence and antisocial behavior in high-risk, urban boys. *Journal of Clinical Child Psychology, 28*, 2-11.

Millman, R. P., Working Group on Sleepiness in Adolescents and Young Adults, & AAP Committee on Adolescence. (2005). Excessive sleepiness in adolescents and young adults: causes, consequences, and treatment strategies. *Pediatrics, 115*, 1774-1786.

Moffitt, T. E., Caspi, A., Harrington, H., & Milne, B. J. (2002). Males on the life-course-persistent and adolescence-limited antisocial pathways: Follow-up at age 26 years. *Development and Psychopathology, 14*, 179-207.

Moilanen, K. L., & Shaw, D. S. (2009). Developmental cascades: Externalizing, internalizing, and academic competence from middle childhood to early adolescence. *Manuscript submitted for publication*.

Moilanen, K. L., Shaw, D. S., Criss, M., M., & Dishion, T. J. (in press). Growth and predictors of parental knowledge of youth behavior during early adolescence. *Journal of Early Adolescence*.

Morrison, D. N., McGee, R., & Stanton, W. R. (1992). Sleep problems in adolescence. *Journal of the American Academy of Child & Adolescent Psychiatry, 31*, 94-99.

National Sleep Foundation. (2006). *Sleep in America Poll*. Retrieved March 15, 2009, from http://www.sleepfoundation.org/atf/cf/%7BF6BF2668-A1B4-4FE8-8D1A-A5D39340D9CB%7D/2006_summary_of_findings.pdf.

NICHD Early Child Care Research Network. (2004). Trajectories of physical aggression from toddlerhood to middle childhood. *Monographs of the Society for Research in Child Development, 69*, 1-129.

O'Brien, E. M., & Mindell, J. A. (2005). Sleep and Risk-Taking Behavior in Adolescents. *Behavioral Sleep Medicine, 3*, 113-133.

Oland, A. A., & Shaw, D. S. (2005). Pure versus co-occurring externalizing and internalizing symptoms in children: The potential role of socio-developmental milestones. *Clinical Child and Family Psychology Review, 8*, 247-270.

Owens, E. B., & Shaw, D. S. (2003). Poverty and early childhood adjustment. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 267-292). New York, NY: Cambridge University Press.

Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *Antisocial boys*. Eugene, OR: Castalia.

Pepler, D. J., & Craig, W. M. (2005). Aggressive girls on troubled trajectories: A developmental perspective. In D. J. Pepler, K. Madsen, C. Webster & K. S. Levene (Eds.), *The development and treatment of girlhood aggression* (pp. 3-28). Mahwah, NJ: Lawrence Erlbaum.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence, 17*, 117-133.

Pianta, R. C., Steinberg, M., & Rollins, K. (1995). The first two years of school: Teacher-child relationships and deflections in children's classroom adjustment. *Development and Psychopathology, 7*, 295-312.

Pilcher, J. J., & Huffcutt, A. J. (1996). Effects of sleep deprivation on performance: A meta-analysis. *Sleep: Journal of Sleep Research & Sleep Medicine, 19*, 318-326.

Pines, M. (1975, Dec). In praise of "invulnerables". *APA Monitor,* 7.

Porges, S. W. (1992). Vagal tone: a physiologic marker of stress vulnerability. *Pediatrics, 90*, 498-504.

Porges, S. W. (2001). The polyvagal theory: Phylogenetic substrates of a social nervous system. *International Journal of Psychophysiology, 42*, 123-146.

Porges, S. W. (2003). The polyvagal theory: Phylogenetic contributions to social behavior. *Physiology & Behavior, 79*, 503-513.

Porges, S. W., Doussard-Roosevelt, J. A., & Maita, A. K. (1994). Vagal tone and the physiological regulation of emotion. *Monographs of the Society for Research in Child Development, 59*, 167-186, 250-283.

Porges, S. W., Doussard-Roosevelt, J. A., Portales, A., & Greenspan, S. I. (1996). Infant regulation of the vagal "brake" predicts child behavior problems: A psychobiological model of social behavior. *Developmental Psychobiology, 29*, 697-712.

Radke-Yarrow, M., & Brown, E. (1993). Resilience and vulnerability in children of multiple-risk families. *Development and Psychopathology, 5*, 581-592.

Raine, A. (2002). Biosocial studies of antisocial and violent behavior in children and adults: A review. *Journal of Abnormal Child Psychology, 30*, 311-326.

Raine, A., Brennan, P., & Mednick, S. A. (1994). Birth complications combined with early maternal rejection at age 1 year predispose to violent crime at age 18 years. *Archives of General Psychiatry, 51*, 984-988.

Roberts, R. E., Roberts, C. R., & Chen, I. G. (2002). Impact of insomnia on future functioning of adolescents. *Journal of Psychosomatic Research, 53*, 561-569.

Roberts, R. E., Roberts, C. R., & Xing, Y. (2006). Prevalence of Youth-Reported DSM-IV Psychiatric Disorders Among African, European, and Mexican American Adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 45*, 1329-1337.

Rowe, R. (1994). *The limits of family influence: Genes, experience, and behavior*. New York, NY: Guilford Press.

Rowe, R., Maughan, B., Worthman, C. M., Costello, E., & Angold, A. (2004). Testosterone, antisocial behavior, and social dominance in boys: Pubertal development and biosocial interaction. *Biological Psychiatry, 55*, 546-552.

Rutter, M. (1979). Protective factors in children's responses to stress and disadvantage. In M. W. Kent & R. E. Rolf (Eds.), *Primary prevention in psychopathology* (Vol. 8: Social competence in children, pp. 49-74). Hanover, NH: University Press of New England.

Rutter, M. (2000). Resilience reconsidered: Conceptual considerations, empirical findings, and policy implications. In J. P. Shonkoff & S. J. Meisels (Eds.), *Handbook of early childhood intervention* (2nd ed., pp. 651-682). New York, NY: Cambridge University Press.

Sameroff, A. (1998). Environmental risk factors in infancy. *Pediatrics, 102*, 1287-1292.

Sameroff, A., Seifer, R., Baldwin, A., & Baldwin, C. (1993). Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. *Child Development, 64*, 80-97.

Sameroff, A., Seifer, R., Zax, M., & Barocas, R. (1987). Early Indicators of Developmental Risk: Rochester Longitudinal Study. *Schizophrenia Bulletin, 13*, 383-394.

Sampson, R. J., Morenoff, J. D., & Earls, F. (1999). Beyond social capital: Spatial dynamics of collective efficacy for children. *American Sociological Review, 64*, 633-660.

Santucci, A. K., Silk, J. S., Shaw, D. S., Gentzler, A., Fox, N. A., & Kovacs, M. (2008). Vagal tone and temperament as predictors of emotion regulation strategies in young children. *Developmental Psychobiology, 50*, 205-216.

Sattler, J. M. (1990). *Assessment of children* (3rd ed.). San Diego, CA: Jerome M. Sattler.

Sattler, J. M. (1992). *Assessment of children* (Revised and updated 3rd edition ed.). San Diego, CA: Jerome M. Sattler.

Scarpa, A., Fikretoglu, D., & Luscher, K. (2000). Community violence exposure in a young adult sample: II. Psychophysiology and aggressive behavior. *Journal of Community Psychology, 28*, 417-425.

Scarpa, A., & Ollendick, T. H. (2003). Community violence exposure in a young adult sample: III. Psychophysiology and victimization interact to affect risk for aggression. *Journal of Community Psychology, 31*, 321-338.

Scarpa, A., & Raine, A. (2006). The psychophysiology of human antisocial behavior. In R. J. Nelson (Ed.), *Biology of aggression* (pp. 447-461). New York: Oxford.

Scarpa, A., Tanaka, A., & Haden, S. C. (2008). Biosocial bases of reactive and proactive aggression: The roles of community violence exposure and heart rate. *Journal of Community Psychology, 36*, 969-988.

Seidman, E., & Pedersen, S. (2003). Holistic contextual perspectives in risk, protection, and competence among low-income urban adolescents. In S. S. Luthar (Ed.), *Resilience and*

vulnerability: Adaptation in the context of childhood adversities (pp. 318-342). New York, NY: Cambridge University Press.

Seifer, R., Sameroff, A., Baldwin, C. P., & Baldwin, A. L. (1992). Child and family factors that ameliorate risk between 4 and 13 years of age. *Journal of the American Academy of Child & Adolescent Psychiatry, 31*, 893-903.

Shannon, K. E., Beauchaine, T. P., Brenner, S. L., Neuhaus, E., & Gatzke-Kopp, L. (2007). Familial and temperamental predictors of resilience in children at risk for conduct disorder and depression. *Development and Psychopathology, 19*, 701-727.

Shaw, D. S., Criss, M. M., Schonberg, M. A., & Beck, J. E. (2004). The development of family hierarchies and their relation to children's conduct problems. *Development and Psychopathology, 16*, 483-500.

Shaw, D. S., & Gross, H. E. (2008). What we have learned about early childhood and the development of delinquency. In A. M. Liberman (Ed.), *The long view of crime: A synthesis of longitudinal research* (pp. 79-127). New York, NY: Springer.

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*, 545-553.

Sheinkopf, S. J., Lagasse, L. L., Lester, B. M., Liu, J., Seifer, R., Bauer, C. R., et al. (2007). Vagal tone as a resilience factor in children with prenatal cocaine exposure. *Development and Psychopathology, 19*, 649-673.

Silk, J. S., Shaw, D. S., Forbes, E. E., Lane, T. L., & Kovacs, M. (2006). Maternal depression and child internalizing: The moderating role of child emotion regulation. *Journal of Clinical Child and Adolescent Psychology, 35*, 116-126.

Silk, J. S., Vanderbilt-Adriance, E., Shaw, D. S., Forbes, E. E., Whalen, D. J., Ryan, N. D., et al. (2007). Resilience among children and adolescents at risk for depression: Mediation and moderation across social and neurobiological context. *Development and Psychopathology, 19*, 841-865.

Smetana, J. G. (2008). "It's 10 o'clock: Do you know where your children are?" Recent advances in understanding parental monitoring and adolescents' information management. *Child Development Perspectives, 2*, 19-25.

Soenens, B., Vansteenkiste, M., Luyckx, K., & Goossens, L. (2006). Parenting and adolescent problem behavior: An integrated model with adolescent self-disclosure and perceived parental knowledge as intervening variables. *Developmental Psychology, 42*, 305-318.

Srinivasan, K. (2006). Childhood anxiety disorder and heart rate variability. In *Panic disorders: New research.* (pp. 65-81). Hauppauge, NY: Nova Biomedical Books.

Stattin, H., & Kerr, M. (2000). Parental monitoring: A reinterpretation. *Child Development, 71*, 1072-1085.

Stattin, H., Romelsjo, A., & Stenbacka, M. (1997). Personal resources as modifiers of the risk for future criminality: An analysis of protective factors in relation to 18-year-old boys. *British Journal of Criminology, 37*, 198-223.

Steinberg, L., Dahl, R., Keating, D., Kupfer, D. J., Masten, A. S., & Pine, D. S. (2006). The study of developmental psychopathology in adolescence: Integrating affective neuroscience with the study of context. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (2nd ed., Vol. 2: Developmental neuroscience, pp. 710-741). Hoboken, NJ: John Wiley & Sons.

Stickgold, R. (2005). Sleep-dependent memory consolidation. *Nature, 437*, 1272-1278.

Stouthamer-Loeber, M., Loeber, R., Farrington, D. P., Zhang, Q., & et al. (1993). The double edge of protective and risk factors for delinquency: Interrelations and developmental patterns. *Development and Psychopathology, 5*, 683-701.

Stouthamer-Loeber, M., Loeber, R., Wei, E., Farrington, D. P., & Wikstrom, P.-O. H. (2002). Risk and promotive effects in the explanation of persistent serious delinquency in boys. *Journal of Consulting and Clinical Psychology, 70*, 111-123.

Stouthamer-Loeber, M., Wei, E., Loeber, R., & Masten, A. S. (2004). Desistance from persistent serious delinquency in the transition to adulthood. *Development and Psychopathology, 16*, 897-918.

Strong, R. K., & Dabbs, J. M., Jr. (2000). Testosterone and behavior in normal young children. *Personality and Individual Differences, 28*, 909-915.

Sullivan, T. N., Kung, E. M., & Farrell, A. D. (2004). Relation between witnessing violence and drug use initiation among rural adolescents: Parental monitoring and family support as protective factors. *Journal of Clinical Child and Adolescent Psychology, 33*, 488-498.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: standards of measurement, physiological interpretation and clinical use. *Circulation, 93*, 1043-1065.

Thompson, R. A. (1998). Early socio-personality developmetn. In N. Eisenberg (Ed.), *Handbook of child psychology* (5th ed., Vol. 3, Social, emotional, and personality development, pp. 25-104). New York, NY: Wiley.

Thompson, R. A., & Calkins, S. D. (1996). The double-edged sword: Emotional regulation for children at risk. *Development and Psychopathology, 8*, 163-182.

Tiet, Q. Q., Bird, H. R., Davies, M., Hoven, C., Cohen, P., Jensen, P. S., et al. (1998). Adverse life events and resilience. *Journal of the American Academy of Child & Adolescent Psychiatry, 37*, 1191-1200.

Tiet, Q. Q., Bird, H. R., Hoven, C. W., Wu, P., Moore, R., & Davies, M. (2001). Resilience in the face of maternal psychopathology and adverse life events. *Journal of Child and Family Studies, 10*, 347-365.

Trentacosta, C. J., Hyde, L. W., Shaw, D. S., & Cheong, J. (in press). Adolescent dispositions for antisocial behavior in context: The roles of neighborhood dangerousness and parental knowledge. *Journal of Abnormal Psychology*.

Trentacosta, C. J., Hyde, L. W., Shaw, D. S., Dishion, T. J., Gardner, F., & Wilson, M. (2008). The relations among cumulative risk, parenting, and behavior problems during early childhood. *Journal of Child Psychology and Psychiatry, 49*, 1211-1219.

Trentacosta, C. J., & Shaw, D. S. (in press). Emotional self-regulation, peer rejection, and antisocial behavior: Associations from early childhood to early adolescence. *Journal of Applied Developmental Psychology*.

Tynjala, J., Kannas, L., & Levalahti, E. (1997). Perceived tiredness among adolescents and its association with sleep habits and use of psychoactive substances. *Journal of Sleep Research, 6*, 189-198.

van Bokhoven, I., van Goozen, S. H., van Engeland, H., Schaal, B., Arseneault, L., Seguin, J. R., et al. (2006). Salivary testosterone and aggression, delinquency, and social dominance in a population-based longitudinal study of adolescent males. *Hormones and Behavior, 50*, 118-125.

van Goozen, S. H., Fairchild, G., Snoek, H., & Harold, G. T. (2007). The Evidence for a Neurobiological Model of Childhood Antisocial Behavior. *Psychological Bulletin, 133*, 149-182.

Vanderbilt-Adriance, E., & Shaw, D. S. (2008a). Conceptualizing and re-evaluating resilience across levels of risk, time, and domains of competence. *Clinical Child and Family Psychology Review, 11*, 30-58.

Vanderbilt-Adriance, E., & Shaw, D. S. (2008b). Protective factors and the development of resilience in the context of neighborhood disadvantage. *Journal of Abnormal Child Psychology, 36*, 887-901.

Vanderbilt-Adriance, E., Shaw, D. S., & Moilanen, K. L. (2008). *The protective role of parental monitoring in urban neighborhoods during adolescence*. Paper presented at the Society for Research on Adolescence, Chicago IL.

Venables, P. H. (1988). Psychophysiology and crime: Theory and data. In T. E. Moffit & S. A. Mednick (Eds.), *Biological contributions to crime causation* (pp. 3-13). Dordrecht: Nijhoff.

Weber, E. J. M., Molenaar, P. C. M., & van der Molen, M. W. (1988). PSPAT: A program for spectral analysis of point events including a test for stationarity. In L. J. M. Mulder, F. J. Maarse, W. P. B. Sjouw & A. E. Akkerman (Eds.), *Computers in psychology:*

Applications in education, research, and psychodiagnostics. Amsterdam: Swets and Zeitlinger.

Wechsler, D. (1989). *Wechsler Preschool and Primary Scale of Intelligence - Revised*. San Antonio, TX: The Psychological Corporation.

Wechsler, D. (1991). *Wechsler Intelligence Scale for Children-III*. San Antonio, TX: The Psychological Corporation.

Werner, E. E., & Smith, R. S. (1982). *Vulnerable, but invincible: A longitudinal study of resilient children and youth*. New York, NY: McGraw-Hill.

Werner, E. E., & Smith, R. S. (1992). *Overcoming the odds: High risk children from birth to adulthood*. Ithaca, NY: Cornell University Press.

White, J. L., Moffitt, T. E., & Silva, P. A. (1989). A prospective replication of the protective effects of IQ in subjects at high risk for juvenile delinquency. *Journal of Consulting and Clinical Psychology, 57*, 719-724.

Wikstrom, P. O., & Loeber, R. (2000). Do disadvantaged neighborhoods cause well-adjusted children to become adolescent delinquents? A study of juvenile serious offending, individual risk and protective factors, and neighborhood context. *Criminology, 38*, 1109-1142.

Wingfield, J. C., Lynn, S., & Soma, K. K. (2001). Avoiding the 'costs' of testosterone: ecological bases of hormone-behavior interactions. *Brain, Behavior & Evolution, 57*, 239-251.

Winslow, E. B. (2001). Development of boys' early conduct problems in a low-income, urban sample: Implications of neighborhood context and maternal parenting (Doctoral Dissertation, University of Pittsburgh, 2001). *Dissertation Abstracts International, 62*, 2509.

Wong, M. M., Brower, K. J., Fitzgerald, H. E., & Zucker, R. A. (2004). Sleep problems in early childhood and early onset of alcohol and other drug use in adolescence. *Alcoholism: Clinical and Experimental Research, 28*, 578-587.

Wyman, P. A., Cowen, E. L., Work, W. C., Hoyt-Meyers, L., Magnus, K. B., & Fagen, D. B. (1999). Caregiving and developmental factors differentiating young at-risk urban children showing resilient versus stress-affected outcomes: A replication and extension. *Child Development, 70*, 645-659.

Yates, T. M., Egeland, B., & Sroufe, L. A. (2003). Rethinking resilience: A developmental process perspective. In S. S. Luthar (Ed.), *Resilience and vulnerability: Adaptation in the context of childhood adversities* (pp. 243-266). New York, NY: Cambridge University Press.