CUMULATIVE RISK AND BEHAVIORAL PROBLEMS IN EARLY ADOLESCENCE: TESTING THE MEDIATION AND MODERATION ROLES OF DYSREGULATION IN MULTIPLE BIOLOGICAL STRESS SYSTEMS

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ABSTRACT

In an attempt to understand how cumulative risk influences behavioral problems in early adolescence, this study focuses on the mediating and the moderating roles of dysregulation in multiple biological stress systems. In a sample of 223 seventh- and eighth-grade children, cumulative risk included psychosocial factors (family turmoil, parent-child separation, exposure to violence) and physical factors (noise, crowding, housing quality) and sociodemographic characteristics of the adolescents' families (maternal high school drop out, single parent, and poverty). Physiological markers of biological stress dysregulation were cortisol, epinephrine, norepinephrine, fat deposition, resting systolic and diastolic blood pressure, and systolic and diastolic reactivity and recovery. There were adverse effects of cumulative risk on both internalizing and externalizing behaviors. Cumulative risk and biological stress dysregulation had a curvilinear relationship. We found that biological stress dysregulation may have an indirect effect on the relation between cumulative risk and internalizing behaviors. Further, older children were more likely to develop internalizing behaviors when they were exposed to cumulative risk. Biological stress dysregulation moderated the effects of cumulative risk on externalizing behaviors; the inefficient stress regulation in multiple biological systems made children more vulnerable to externalizing behavioral problems when they were living in cumulative risk environment. The importance of understanding both mediating and moderating roles of biological stress dysregulation for behavioral problems was discussed.

BIOGRAPHICAL SKETCH

Pilyoung Kim was born in Pusan, South Korea and grew up in Seoul, South Korea. She received her bachelor's degree from Korea University in Seoul, South Korean in 2002 majoring in English literature and Psychology. She received her master's from Harvard Graduate School of Education in 2003 majoring in Human Development and Psychology with a concentration in Mind, Brain and Education. Since 2004, she has been enrolled in the MA/PhD Developmental Psychology program in the department of Human Development at Cornell University.

To my parents,

the best parents

in the world.

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CHAPTER 1

Introduction

Risk factors are aspects of the child and his or her environment that contribute to difficulties in socioemotional adjustment and the development of behavioral problems in children and adolescents (Garmezy & Rutter, 1983; Lewis & Feiring, 1998). A number of risk factors such as family conflict (Davis & Cummings, 1994), parent-child separation (Emery & Forehand, 1994), violence (Cooley-Quille, Boyd, Frantz, & Walsh, 2001), poverty (McLoyd, 1998), noise and crowding (Evans, 2001) have been associated with children's behavioral problems. Typically, investigators have distinguished between two broad categories of behavioral problems – internalizing (e.g., anxiety, depression, social withdrawal, and psychosomatic complaints) and externalizing behaviors (e.g., delinquency and aggressive behavior) among children and adolescents (Achenbach, 1991a, 1991b; Cicchetti & Toth, 1991; Compas, 1987; Grant et al., 2003; Haggerty, Sherrod, Garmezy, & Rutter, 1994).

For children and adolescents with behavioral problems, studies have often linked behavioral problems to a particular risk factor. However, the attempt to explain behavior problems by a single risk factor has limitations. Some children and adolescents, despite being subject to the same risk factor, exhibit little or no behavioral problems (Liaw & Brooks-Gunn, 1994; Sameroff, Bartko, Baldwin, Baldwin, & Seifer, 1998; Sameroff, Seifer, & Bartko, 1997). One explanation of this inconsistency is that the development of behavioral problems may involve exposure to more than one risk factor. Moreover, risk factors often occur in multiples; having one risk factor increases the chance of exposure to another risk factor (Bronfenbrenner & Evans, 2000; Coie et al., 1993; Rutter, 1990; Sameroff et al., 1997). For example, children and adolescents in households with family conflicts are more likely to witness domestic violence and suffer separation from the family (Repetti, Taylor, & Seeman,

2002). Moreover, children and adolescents who live in poverty tend to receive less responsive parenting than more affluent children (McLoyd, 1998), and their living conditions tend to be crowded, noisy and of poor quality (Evans & Saegert, 2000; Evans, Wells, Chan, & Saltzman, 2000). Furthermore, risk factors in various aspects of one's life − individual, family, or neighborhood − may interact with each other as they influence development (Bronfenbrenner, 1979).

Because of the limitations of single-factor explanations of behavior problems, a measure that can encompass multiple risk factors, such as cumulative risk, may be more desirable. Studies have demonstrated that when multiple risk factors are considered, even though each singular factor may not be sufficient to cause behavioral problems, their cumulative effect can lead to serious behavioral problems. In his attempt to explain the relations between cumulative risk and psychiatric disorders among children, Rutter (1979) measured six risk factors—high marital distress, low socioeconomic status, large family size, paternal criminality, maternal psychiatric disorder, and foster care placement. Twenty percent of the children in families with more than four risk factors exhibited psychiatric disorders (Rutter, 1979). In contrast, only 2% of the children developed psychiatric disorders when confronted by one risk factor (Rutter, 1979). In the Rochester Longitudinal Study (Sameroff, Seifer, Zax, & Barocas, 1987), ten risk factors were assessed; maternal chronic mental illnesses, severe maternal anxiety, rigid parental perspectives on child development, inadequate maternal interactions, poor maternal education, unskilled occupation, minority status, single parenthood, stressful life event, and large family size. The cumulative number of the risk factors was positively correlated with the number of psychiatric symptoms in preschoolers (Sameroff et al., 1987) as well as longitudinally in 13 and 18 year-old adolescents (Sameroff, et al., 1998).

Other studies have shown that cumulative risk provides a potentially powerful explanation of behavioral problems among children and adolescents. In a study using similar cumulative risk coding scheme as described in Sameroff et al. (1987), only 7% of children in families with less than two risk factors had behavioral problems, whereas 40% of children in families with eight or more risk factors had behavioral problems (Williams, Anderson, McGee, & Silva, 1990). Family adversity including parental history of mental disorders, antisocial problems, and single parenthood were positively correlated with both internalizing and externalizing behavioral problems in six to seven-year-old children (Ackerman, Izard, Schoff, Youngstrom, & Kogos, 1999). In a study by Deater-Deckard and his colleagues, cumulative risk including socioeconomic status (SES), marital status, stressful life events, isolation, parent conflict, and violence was associated with more externalizing problems throughout middle childhood (age 5 to10) (Deater-Deckard, Dodge, Bates, & Pettit, 1998). Twenty percent of adolescents at age 16 from the most disadvantaged home environment developed behavioral problems (Fergusson, Horwood, & Lynskey, 1994). In comparison, among those who lived in the most-advantaged home environment, only one adolescent showed behavioral problems (Fergusson, Horwood, & Lynskey, 1994). Furthermore, cumulative risks in early childhood have predicted both internalizing and externalizing problems in adolescence (Appleyard, Egeland, van Dulmen, & Sroufe, 2005).

An important limitation of existing studies on cumulative risk has been their focus on psychosocial components of risks, such as single parenthood, family conflict, or violence (Ackerman, et al., 1999; Sameroff et al., 1987), while showing little interest in the physical aspects of the environment, such as crowding and noise. It is important to consider physical aspects of cumulative risk because in a typical environment where a child grows up, these aspects not only work alone but also often

interact with psychosocial characteristics to influence the child's development (Bronfenbrenner, 1979). Several studies have associated various physical conditions in the environment with negative psychosocial outcomes among children. For example, crowding or high residential density has been shown to increase children's psychological distress (Evans & Saegert, 2000; Evans, Saegert, & Harris, 2001; Saegert, 1982). More people per room in a house is positively correlated with more internalizing and externalizing behavioral problems among children living in rural families (Evans et al., 2001). Crowding is also further related to psychosocial risk factors, such as less responsive parenting and more family turmoil (Evans & Saegert, 2000; Wachs & Camli, 1991). High residential noise levels contribute to helplessness and increased psychological distress (Evans, 2001). Poor housing quality can also impact children's psychosocial adjustment. Children living in houses with poor structure, hazards, little privacy, and limited resources showed more psychological distress (Evans et al., 2000). Poverty can influence children's behavioral problems as it interacts with other risk factors in the physical environment of the children. Low SES children are more likely to live in crowded and noisy houses (Evans, 2001). While these studies did not specifically deal with cumulative risk, they nevertheless highlight the importance of physical factors in relation to behavioral problems. Among the few studies that incorporated physical environment factors into cumulative risk scales, Evans and English (2002) found that cumulative risk including crowding, noise, housing qualities as well as family turmoil, family separation, and violence increased behavioral problems among low SES children. Therefore, the cumulative risk construct provides a more comprehensive explanation of behavioral problems when physical factors are also considered.

In addition to inclusion of physical qualities of the child's living environment, there are other factors that can influence the relationship between cumulative risk and

behavioral problems. As suggested by the ecological model of human development (Bronfenbrenner, 1979), various characteristic of the child including dispositions and resources may affect how an individual reacts to multiple stressors in the environment. One individual characteristic that may influence behavioral problems is biological stress regulatory systems. Thus, in the present study, we examined how function of the regulatory systems interacted with cumulative risk to increase behavioral problems.

A few studies have suggested an association between biological stress dysregulation and behavioral problems in childhood and adolescence. Stressor-induced biological responses are essential to coping with stressors (Nelson, 1999). When the crucial biological mechanisms are impaired, serious behavioral and emotional problems may occur. Experiences of stress trigger the brain to send signals to the body in order to generate appropriate responses. Examples of these signals are various hormones such as cortisol, epinephrine and norepinephrine. These hormones trigger a series of physiological responses—increase in heart rate and blood pressure to help prompt behavioral responses (Nelson, 1999). When stress is no longer present, hormones and cardiovascular activities restore to the basal level through a negative feedback mechanism induced by the brain (Nelson, 1999). Abnormalities in biological regulatory systems, such as overly heightened or dampened reactivity in response to stress might impair emotional and behavioral regulation among children and adolescents (Bauer, Quas, & Boyce, 2002; Grant et al., 2003; Repetti, Taylor, & Seeman, 2002). Aberrant cortisol levels can adversely influence psychological functions (Gunnar & Vazquez, 2004). For example, low cortisol levels were found among adolescents who exhibited disruptive behaviors (Van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000) and high internalizing behavioral problems (Gunnar & Vazquez, 2004), whereas high cortisol levels were found among clinically depressed adolescents (Goodyer, Herbert, Tamplin, & Altham, 1996). Similarly,

heightened resting cardiovascular activity and dampened cardiovascular stress reactivity have both been associated with behavioral problems (Bauer et al., 2002). Higher resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) have been associated with delinquent behavioral problems among six to ten year olds (Pine et al., 1996), and higher resting heart rate (HR) and dampened HR reactivity positively related to aggression (Schneider, Nicolotti, & Delamater, 2002). These findings underscore the potential implications biological regulatory systems of emotional and behavioral responses of children and adolescents.

Biological stress regulation could influence the relationship between cumulative risk and behavioral problems in two primary ways (Lorber, 2004; Raine, 2002; Repetti, Taylor, & Seeman, 2002). First, biological stress regulation could serve as a *moderator*. The biological stress regulatory system can be conceptualized as a genetically based biological predisposition that alters the likelihood that the individual will develop behavioral problems in response to cumulative risk (Wadsworth, Raviv, Compas, & Connor-Smith, 2005). As suggested by the diathesis-stress model (Hammen, 2005) and the bioecological model (Bronfenbrenner & Evans, 2000), biological vulnerability can interact with risks to affect behavioral problems. Despite living in similar high-risk environments, individuals with this genetic vulnerability are more likely to develop psychological disorders than those without the genetic component (Caspi et al., 2002; Caspi et al., 2003). As an example, low resting HR, was associated with more delinquent behavioral problems in adulthood when boys had poor relationships with parents and lived in a large family in adolescence (Farrington, 1997). However, whether biological stress regulatory systems can act as a moderator of cumulative risk on internalizing and externalizing behaviors has not been studied.

Alternatively, biological stress regulation can function as a *mediator*, helping to explain the negative effect of cumulative risk on behavioral problems. Acting as a mediator, biological stress dysregulation specifies the mechanism through which cumulative stress may affect behavioral problems (Wadsworth et al., 2005). In the moderator model, the effects of cumulative risk on behavioral problems are influenced by what levels of physiological dysregulation. In contrast, a mediator model examines whether the impact of cumulative risk on behavioral problems is caused by physiological dysregulation. Figure 1a depicts the moderation model whereas Figure 1b illustrates the mediation perspective. Thus, in the mediation model, biological stress dysregulation is affected by cumulative risk which, in turn, influences children's behavioral problems directly. This is in contrast to the moderation model that suggests the biological stress dysregulation is a stable characteristic independent of cumulative risk.

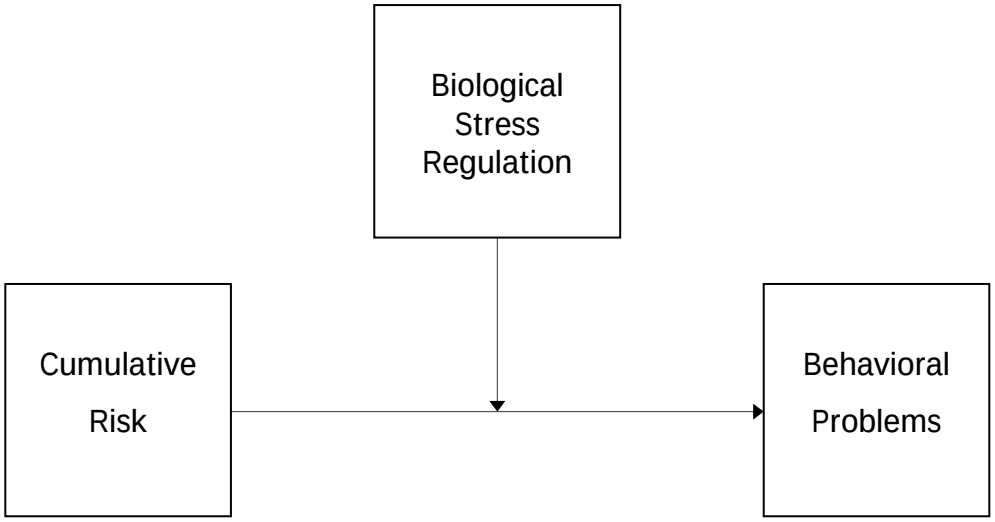

Figure 1a. Moderation Model of Biological Stress Regulation

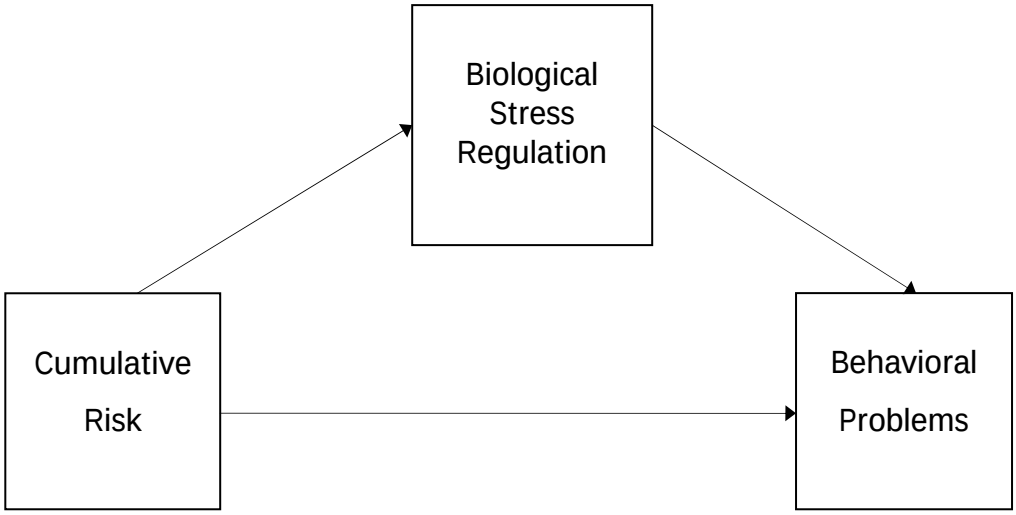

Figure 1b. Mediation Model of Biological Stress Regulation

While our biological stress regulatory system enables us to cope with short-term stressors (Nelson, 1999), its effectiveness can be compromised if it is constantly activated due to multiple and repeated stressors in the environment (McEwen, 2000). Adverse effects on multiple regulatory systems are possible if such activation is prolonged due to cumulative stress, a scenario referred as "allostatic load" (McEwen, 2000). Having been impaired as a result of the wear and tear on both the brain and the body due to cumulative risk exposure, regulatory systems either can not turn off properly or can not generate adequate responses to stress (McEwen, 2000, 2003). For example, cumulative risk leads to constantly elevated levels of neuroendocrine activities both in hypothalamic-pituitary-adrenocortical (HPA) axis and in the sympathetic nervous system. These alterations in neuroendocrine activities can lead to heightened basal levels of cortisol, epinephrine and norepinephrine (McEwen, 2000, 2003). Continual exposure to cumulative risk may also result in inefficient cardiovascular regulatory functions such as dampened reactivity and slower, inefficient recovery to basal levels (McEwen, 2000, 2003). Heightened fat deposition

is another indicator of biological stress dysregulation (McEwen, 2000). Furthermore, dysregulation in multiple biological stress systems due to cumulative stress exposure can increase difficulties in behavioral and emotional regulation, which in turn causes more behavioral problems among children and adolescents (Evans, 2003).

The concept of allostatic load provides a useful framework for examining the mechanisms behind the development of depression (McEwen, 2003, 2004). Exposure to a stressor activates the amygdala in the brain, that interprets the nature of the stressor. However, repeated exposures to multiple stressors cause the amygdala to become hyperactive, rendering the person more sensitive to negative environmental and interpersonal stimuli and more prone to interpret neutral stimuli as negative. The hyperactive amygdala produces excessive stress hormones, which can destabilize hormonal and cardiovascular activities and eventually damage neuronal and physiological regulatory systems (McEwen, 2003, 2004). As a result of long-term imbalances in stress regulatory systems, the brain areas related to anxiety, fear and emotional regulation experience long-term changes. This may increase an individual's vulnerability to depression and anxiety, and may lead to problems in social interactions with others (McEwen, 2004).

While most studies have examined the link between singular risk exposure and a specific biological regulatory system in children (Cicchetti & Toth, 1991; Gunnar & Vazquez, 2004; Regecova & Kellcrova, 1995; Repetti et al., 2002), a few studies have empirically examined cumulative risk and multiple biological stress systems. In a study of 8 to 10 year-old children, Evans and English (2002) found that cumulative risk, including physical stressors (poor housing quality, noise and crowding) and psychosocial (family turmoil, separation, and violence) stressors, partially mediated the negative effect of poverty on hormonal levels (overnight cortisol and epinephrine levels) and basal cardiovascular processes (resting blood pressure) (Evans & English,

2002). In a subsequent study, Evans (2003) demonstrated a direct association between cumulative risk and higher allostatic load, using a cumulative index involving several physiological markers to measure stress dysregulation in childhood. For cumulative risk, he added three factors, poverty, single parenthood and maternal high school dropout, to the previous list of cumulative risks in the study of Evans and English (2002). The coding of the allostatic load involved six physiological markers to denote various abnormalities in stress regulation— cortisol, epinephrine, norepinephrine, resting SBP and DBP, and fat deposition (Evans, 2003). Evans and his colleagues (in press) found a similar prospective association between cumulative risk in early childhood and allostatic load in early adolescence. Cumulative risk was associated with allostatic load in 12 to14 year-olds. This main effect was qualified by an interaction with maternal responsiveness (Evans, Kim, Ting, Tesher, & Shannis, in press). This is, cumulative risk led to allostatic load only when the children experienced low maternal responsiveness. Furthermore, in the same study, Evans and his colleagues (in press) found that cumulative risk was also associated with dampened cardiovascular reactivity to an acute stressor and slower recovery to basal levels (Evans et al., in press). However, whether dysregulation in multiple stress systems due to exposure to cumulative risk can lead to behavioral problems among children and adolescents has never been studied.

In summary, biological stress regulation systems may contribute to an understanding of the adverse effects of cumulative risk on psychosocial adjustment. However, because most studies in this area have investigated relations among singular risk factors and either the biological stress system or behavioral problems, we know very little about how stress dysregulation in multiple biological systems might influence affect behavioral problems. To our knowledge, no studies have directly tested the link between cumulative risk, multiple biological stress systems, and

behavioral problems in childhood and adolescence. Early adolescence is an especially important developmental period to study behavioral problems. It is a transitional period from childhood to adolescence, and during this period, children typically experience various challenges due to rapid biological changes associated with puberty as well as radical changes in family and school environments (Steinberg, 2005). As a result, the rate of internalizing and externalizing problems dramatically increases in early adolescence (Loeber & Farrington, 2000; Twenge & Nolen-Hoeksema, 2002).

The present study examines the role of dysregulation in multiple biological stress systems in the link between cumulative risk and internalizing and externalizing behavioral problems in early adolescence. First, we test whether cumulative risk in both psychological and physical environments, predicts internalizing and externalizing behavior problems. Second, we test whether dysregulation in multiple stress systems moderates the relations between cumulative risk and internalizing and externalizing behavioral problems. Third, we test whether the relations between cumulative risk and internalizing and externalizing behavioral problems are mediated by dysregulation in multiple stress regulatory systems. The mediation model of biological stress dysregulation consists of two major steps. We test (a) whether cumulative risk predicts dysregulation in multiple stress systems. Then, (b) we evaluate whether dysregulation in multiple stress systems predicts behavioral problems.

CHAPTER 2

Method

Participants

Participants were 223 seventh- and eighth-grade students and their families. They were originally recruited from public schools, New York State Co- Operative Extension programs, Head Start Programs, and other state and federal programs for low-income families in five rural upstate New York counties. The children were in third- through fifth grade at initial recruitment (see Evans & English, 2002 for more information).

The average age of the children was 13.4 years (SD = 1.0) and 58% of the children were girls. Consistent with census data, 97% of the children were Caucasian. The mean income-to-needs ratio was 2.35 and 28% of the families were living under the poverty line, an income-to-needs ratio equal to or less than 1 (the federal per capita poverty line). Only one child per household participated in the study and families were paid for their participation.

Measures

Cumulative Risk

There were nine risk factors in the cumulative risk index. Three psychosocial risk factors – family turmoil, child-family separation, and exposure to violence - were reported by both mothers and children. Mothers completed the Life Events and Circumstances Checklist (LEC; Wyman, Cowen, Work, & Parker, 1991) which includes 32 stressful events and circumstances. The events and circumstances are related to chronic stress. Mothers indicated events and circumstances experienced by the family or the target child within the past six month. Mothers answered "Yes" to events that happened to the family or the child. The number of "Yes's" was summed

for each subscale. Sample questions of three subscales of LEC are “Your child has been involved in serious family arguments” (for family turmoil), “A close family member was away from home a lot” (for child-family separation), and “Your child had to deal with people whose behavior was frightening” (for exposure to violence). Children also reported stressful events by completing a revised Adolescent Perceived Events Scale (Compas, 1997). The scale includes 30 stressful life events. Children answered "Yes" to events that happened to them within the past six months. The number of "Yes’s" was tallied for each subscale. Sample questions for subcategories were “Pretty serious arguments or fights between parents” (for family turmoil), “Parents getting divorced/separated” (for child-family separation). To combine the two measurements obtained from the mothers and the children respectively, the scores from each measure were added together for each subscale. However, if an identical event appeared in both measurements, it was counted only once.

Three physical risk factors were noise, residential density, and housing quality. Noise levels were assessed by measuring decibel levels (Leq, dBA) in the primary social place (a living room in most households) in the house over a two-hour period. Residential density was estimated by dividing the number of residents by the number of rooms in the house. Resident was defined as anyone who sleeps in the house more than three nights per week. Only the rooms that were used regularly by residents including washrooms were counted. Housing quality was evaluated by a rater who was trained on a standardized instrument. The instrument includes six subscales with three-point ratings of quality. The instrument assessed structural quality (e.g. the worst ceiling/wall surface in the room), privacy (e.g., walking through the bedroom to get to another room), indoor climatic conditions (e.g., heat has broken down), hazards (e.g., stairs are potentially dangerous), cleanliness/clutter (e.g., much clutter in the kitchen),

and child resources (e.g., toys are accessible to the child in more than one room) (see Evans, Wells, Chan, & Saltzman, 2000 for more information).

Six continuous risk factors, consisting of the three psychosocial risk factors and the three physical risk factors, were then coded dichotomously by assigning 1 if the value was greater than one standard deviation above the mean of the entire sample, or 0 if otherwise. In addition to these six continuous risk factors, three categorical sociodemographic risk factors were included in the cumulative risk index: maternal high school drop out, single parent, and household income at or below the poverty line (income-to-needs ratio < 1.0). The three categorical risk factors were coded dichotomously. The final cumulative risk index was obtained by summing over the values of the risk factors. Thus, the cumulative risk index could vary for each participant from 0 to 9.

Behavioral Problems

The Youth Self Report (YSR; (Achenbach, 1991b) was used to assess children's self-reported behavioral adjustment. All the items were rated on a 3-point Likert scale of 0 (not true), 1 (somewhat or sometimes true), 2 (very true or often true). The measure has two empirically derived categories of behavioral problems - internalizing and externalizing behaviors. The score of internalizing category was obtained by summing three specific subscales of anxious/depressed behaviors (e.g., "I feel worthless or inferior"), social withdrawal behaviors (e.g., "I rather be alone than with others"), and somatic complaints (e.g., "I feel overtired"). Internal reliability of internalizing behaviors was high, $\alpha = .90$. Internal reliability of the three subscales were α (anxious/depressed behaviors) = .85; α (social withdrawal behaviors) = .65, α (somatic complaints) = .74. The score of externalizing category was obtained by summing two specific scales of delinquent Behavior (e.g., "I steal from home") and

aggressive Behavior (e.g., "I fight a lot"). Internal reliability of externalizing behaviors was also high, α = .89. Internal reliability of the two subscales were α (delinquent behaviors) = .76; α (aggressive behaviors) = .86.

Dysregulation in multiple biological stress systems

To measure the dysregulation in multiple biological stress systems, we considered ten physiological makers concerning both hormonal and cardiovascular stress regulatory systems. The ten physiological markers were overnight urinary cortisol, epinephrine, and norepinephrine, resting systolic blood pressure (SBP) and diastolic blood pressure (DBP), SBP and DBP reactivity and recovery to an acute stressor, and an index of fat deposition.

Hormonal markers--- cortisol, epinephrine and norepinephrine, were measured based on overnight urinary samples, collected from 8pm in the evening of the home interview to 8am in the morning of the next day. The urine samples were immediately stored on ice in a container with a preservative (metabisulfite) by the participants. Then, the container was picked up in the same morning and total volume was recorded. Four 10-ml samples were randomly extracted, and then deep frozen at - 80° C until subsequent biochemical assays by technicians blind to the participants' cumulative risk. To further inhibit oxidation of catecholamines, the pH of two of the 10 ml samples was adjusted to 3. Total unbound cortisol was assayed with a radioimmune assay (Contreras, Hane, & Tyrrell, 1986) and epinephrine and norepinephrine were assayed with high pressure liquid chromatography with electrochemical detection (Riggin & Kissinger, 1977). To control for differences in body mass and incomplete urine voidings, creatinine was also assayed (Tietz, 1976).

Resting SBP and DBP was recorded with a Critikon Dinamap Pro 100 blood pressure monitor while the child seated quietly and read a magazine. Physical exercise

was prohibited for one hour prior to the recordings. Both systolic and diastolic blood pressures were measured every two minutes, seven consecutive times. Dropping the first reading, the second to the seventh readings were averaged. This procedure provided high reliablity (Kamarck et al., 1992). SBP and DBP reactivity were assessed by SBP and DBP changes when the child was exposed to a stressor. Immediately after sitting and reading quietly for the resting blood pressure assessment, the child was asked to carry out a mental arithmetic test. The test was a surprise. The test was proved to be a valid acute stressor for both children and adults (Gump & Matthews, 1999; Matthews, Gump, Block, & Allen, 1997). During the task, the interviewer read two numbers, a two-digit number and a four-digit number. The child was asked to subtract the smaller number from the larger number, without using a pen, paper or a calculator, and then spoke the answer out loud back to the interviewer. The task was continued for 12 minutes (6 readings – each reading every 2 minutes). SBP and DBP reactivity was calculated as a slope of the values of 6 readings for each child with Hierarchical Linear Modeling (HLM). After the end of the task, the child was asked to relax and read a magazine quietly again for 10 minutes (5 readings– each reading every 2 minutes) to assess his/her SBP and DBP recovery. The initial BP reading during the recovery phase occurred 30 seconds after cessation of the math task. SBP and DBP recovery was calculated as a slope of the values of 5 readings for each child with HLM.

The last marker of multiple biological stress systems in this study, an index of fat deposition was estimated according to body mass index (kg/m2).

Each physiological marker was dichotomously coded. A value of 1 indicates dysregulation in the stress system and a value of 0 indicates no risk. For SBP and DBP reactivity, since dysregulation is associated with dampened cardiovascular reactivity and slow recovery in previous study (Evans et al., in press), the lowest quartile in the

distribution of the entire sample data received 1 to indicate dysregulation. For all the other markers, the top quartile in the distribution received 1 to indicate dysregulation. After each physiological marker was dichotomously coded, an overall scale of biological stress dysregulation was calculated for each child by summing across all dichotomized markers (0-10).

Procedures

All data including demographic information were collected following a standardized protocol in the participant's residence. The target child and his/her mother were interviewed independently by two interviewers. The gender of the interviewer was matched to the child's gender.

CHAPTER 3

Results

Cumulative risk

Descriptive statistics and intercorrelations are provided in Table 1. The generally high correlation between risk factors suggested a concurrence of multiple risk factors. However, noise and crowding were not correlated with the other psychosocial stressors.

Table 2 shows the mean and the standard deviation of the cumulative risk exposure variable. The percentages of the sample with zero to three risk factors are 30%, 22%, 15% and 16% respectively. There are relatively fewer children with four or more risk factors. The percentages of the sample with four to six risk factors are 5%, 2% and 1% respectively.

Behavioral Problems

Table 2 shows the means, standard deviations and the zero-order correlations among the primary variables. Cumulative risk was positively correlated with both internalizing behaviors ($r = .23$) and externalizing behaviors ($r = .37$). Internalizing and externalizing behaviors were positively correlated ($r = .69$).

Ordinary least squares (OLS) regression was performed to test whether cumulative risk predicts behavioral problems in early adolescence. Gender and age were introduced as control variables and interactions between cumulative risk and control variables were tested. Results of OLS regression indicated that cumulative risk predicted internalizing behaviors, $b = 1.14$ (.33), $p < .01$, $f2 = .05$. Girls had significantly higher internalizing problems than boys, $b = 2.32$ (1.11), $p < .05$, $f2 = .02$. There was no significant interaction found between cumulative risk and gender. To further investigate, OLS was performed on the three subscales of internalizing

Table 1.
Mean, Standard Deviations and Correlations of Cumulative Risk Factors

Measure	Mean	SD	Proportion of sample with risk factor (%)	1	2	3	4	5	6	7	8
1. Family turmoil (0-9)	2.79	2.05	23	--							
2. Family separation (0-12)	2.23	1.72	23	0.51***	--						
3. Violence (0-5)	0.68	0.88	17	0.32***	0.33***	--					
4. Noise (Leq, dBA)	60.56	6.49	18	0.28***	0.11	0.09	--				
5. Crowding (# of people/ room)	0.56	0.19	11	0.12	0.13	-0.02	0.19*	--			
6. Housing problems (0-2)	0.59	0.31	14	0.38***	0.34***	0.21**	0.21**	0.29**	--		
7. Poverty line (0/1)			28	0.38***	0.19**	0.19**	0.29***	0.37***	0.35***	--	
8. Single parent (0/1)			4	0.16*	0.20**	0.10	0.13	0.35***	0.18*	0.23**	--
9. Maternal high school dropout (0/1)			43	0.31***	0.15*	0.18**	0.08	-0.19*	0.24**	0.28***	0.10

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

Table 2.
Mean, Standard Deviations and Correlations of the Primary Variables

Variables	Mean	SD	1	2	3	4	5	6	7	8	9	10
1. Cumulative risk (0-9)	1.81	1.71	--									
2. Biological stress dysregulation (0-10)	2.44	1.88	0.07	--								
3. Age (year)	13.36	0.99	0.06	0.00	--							
4. Gender (0=male, 1=female)	--	--	-0.05	-0.14	-0.06	--						
5. Internalizing behaviors (0-32)	14.09	8.52	0.23**	-0.00	0.08	0.12	--					
5.1. Anxious/Depressed behaviors (0-16)	6.50	5.02	0.25***	0.02	0.08	0.11	0.93***	--				
5.2. Social withdrawn behaviors (0-7)	3.15	2.04	0.13	0.06	0.09	0.12	0.75***	0.63***	--			
5.3. Somatic complaints (0-9)	4.45	3.01	0.13	-0.09	0.01	0.09	0.77***	0.55***	0.38***	--		
6. Externalizing behaviors (0-30)	12.46	8.14	0.37***	0.08	0.11	0.01	0.69***	0.68***	0.45***	0.51***	--	--
6.1. Aggressive behaviors (0-19)	9.33	5.97	0.32***	0.05	0.08	0.05	0.70***	0.69***	0.46***	0.51***	0.96***	
6.2. Delinquent behaviors (0-11)	3.13	2.85	0.39***	0.12	0.16	-0.10	0.52***	0.52***	0.32***	0.38***	0.84***	0.66***

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

behaviors. Cumulative risk were significantly associated with all of the three specific scales: anxious/depressed behaviors (b = .74 (.19), $p < .001$, $f2 = .07$), social withdrawn behaviors (b = .24 (.09), $p < .05$, $f2 = .03$), and somatic complaints (b = .24 (.12), $p < .05$, $f2 = .02$). However, there was no gender effect on any of these scales alone. There was no significant interaction found between cumulative risk and gender. Curvilinear relations of cumulative risk and internalizing behavior were tested, but found to be nonsignificant.

Results of OLS regression revealed that cumulative risk also predicted externalizing behaviors, b = 1.76 (.30), $p < .001$, $f2 = .14$. Cumulative risk was significantly associated with two subscales of externalizing problems: aggressive problems (b = 1.13 (.22), $p < .001$, $f2 = .10$) and delinquent problems (b = .63 (.10), $p < .001$, $f2 = .15$). Only for delinquent behaviors, age was found to be significant: older youths were more likely to have delinquent behaviors, b = .03 (.02), $p < .05$, $f2 = .02$. There were no gender effect on externalizing behaviors and the two subscales, and no significant interaction between cumulative risk and gender. Curvilinear relations of cumulative risk with externalizing behavior were not significant.

Dysregulation in multiple biological stress systems

Table 3 provides the descriptive statistics and the zero-order correlations of each biological stress system. High correlations were found among three stress hormones: cortisol, epinephrine, and norepinephrine, as well as among six indicators of cardiovascular activities: resting SBP, resting DBP, SBP reactivity, DBP reactivity, SBP recovery, and DBP recovery. In addition, higher levels of norepinephrine were correlated with lower SBP reactivity and slower DBP recovery was correlated with high fat deposition among children (see Table 3). However, cardiovascular activities and cortisol were not significantly correlated.

Table 3.
Mean, Standard Deviations and Correlations of Biological Stress Regulation Systems

Variables	Mean	SD	Proportion of sample with risk (%)	1	2	3	4	5	6	7	8	9
1. Cortisol	0.17	0.06	21	--								
2. Epinephrine	7.80	7.01	22	0.41***	--							
3. Norepinephrine	33.13	17.58	22	0.50***	0.65***	--						
4. Resting systolic BP	4.29	7.56	24	-0.01	0.01	-0.10	--					
5. Resting diastolic BP	4.28	8.09	23	0.04	0.07	-0.05	0.47***	--				
6. Systolic BP reactivity	0.38	0.66	24	-0.03	-0.09	-0.17*	0.68***	0.22**	--			
7. Diastolic BP reactivity	0.36	0.49	24	-0.07	-0.03	-0.10	0.43***	0.47***	0.60***	--		
8. Systolic BP recovery	-0.93	2.04	24	0.02	0.09	0.08	-0.30***	-0.19**	-0.35***	-0.29***	--	
9. Diastolic BP recovery	-0.39	1.61	24	-0.02	0.04	0.03	-0.20***	-0.24***	-0.21**	-0.42***	0.36***	--
10. Body mass index (kg/m2)	21.99	6.24	15	-0.09	-0.12	-0.13	-0.08	-0.07	-0.11	-0.13	0.23**	0.11

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

Table 2 presents the mean and standard deviation of the cumulative index of biological stress dysregulation plus its correlation with cumulative risk and behavioral problems. The majority of the sample was fairly evenly distributed among people with 0 to 4 indicators of stress dysregulation. The percentages of the sample having zero to four indicators are 14%, 17%, 18%, 13%, and 14% respectively. There are relatively fewer people with five or more indicators. The percentages of the sample having five to eight indicators are 7%, 7%, 4% and 2% respectively.

Moderational Analyses

Moderational analyses were conducted to examine the possibility that dysregulation in multiple biological stress systems might moderate the relation between cumulative risk and behavioral problems. We tested whether the interaction term of biological stress dysregulation (the moderator) and cumulative risk (the independent variable) significantly predict behavioral problems (the dependent variable). The moderation model included cumulative risk, biological stress dysregulation, and the multiplicative term of cumulative risk and biological stress dysregulation. Gender and age were included as control variables in the model.

As shown in Table 4, the interaction term between biological stress dysregulation and cumulative risk was significant for externalizing behaviors, $b = .31$ (.15), $p < .05$, $f2 = .02$. Children with poor biological stress regulatory systems were more likely to develop externalizing behaviors when they were living in high risk environments than children with well-functioning biological stress regulatory systems (see Figure 2) Age and gender did not interact with cumulative risk and biological stress dysregulation. Stronger moderational relations were found in delinquent behaviors, a subscale of externalizing behaviors. As shown in Table 4, the interaction term between biological stress dysregulation and cumulative risk was significant for

delinquent behaviors, b = .13 (.05), p < .01, f2 = .04. The main effect of cumulative risk became nonsignificant, b = .25 (.17), p = n.s. In terms of gender, boys showed more delinquent behaviors than girls, b = -.78 (.33), p < .05, f2 = .03. However, interactions of gender with cumulative risk and biological stress dysregulation were not significant. No moderational relations were found for aggressive behaviors, another subscale of externalizing behaviors.

Table 4.
Moderational Analysis of Externalizing Behaviors, Cumulative Risk and Biological Stress Dysregulation, Statistically Controlling for Age and Gender

Predictor	Total R^2	ΔR^2	$F \Delta R^2$	*b* (*SE*)
1. Externalizing Behavior				
Cumulative risk	.16	.15	33.97***	.78 (.53)
Biological Stress Dysregulation	.16	.00	.55	-.34 (.38)
Cumulative risk*Biological Stress Dysregulation	.18	.02	4.16*	.31 (.15)*
1.1. Delinquent Behavior				
Cumulative risk	.24	.19	45.91***	.25 (.17)
Biological Stress Dysregulation	.24	.00	1.05	-.14 (.12)
Cumulative risk*Biological Stress Dysregulation	.27	.03	7.11**	.13 (.05)**
1.2. Aggressive Behavior				
Cumulative risk	.107	.107	7.542***	.535 (.415)
Biological Stress Dysregulation	.109	.002	.345	-.179 (.305)
Cumulative risk*Biological Stress Dysregulation	.119	.101	2.058	.171 (.119)

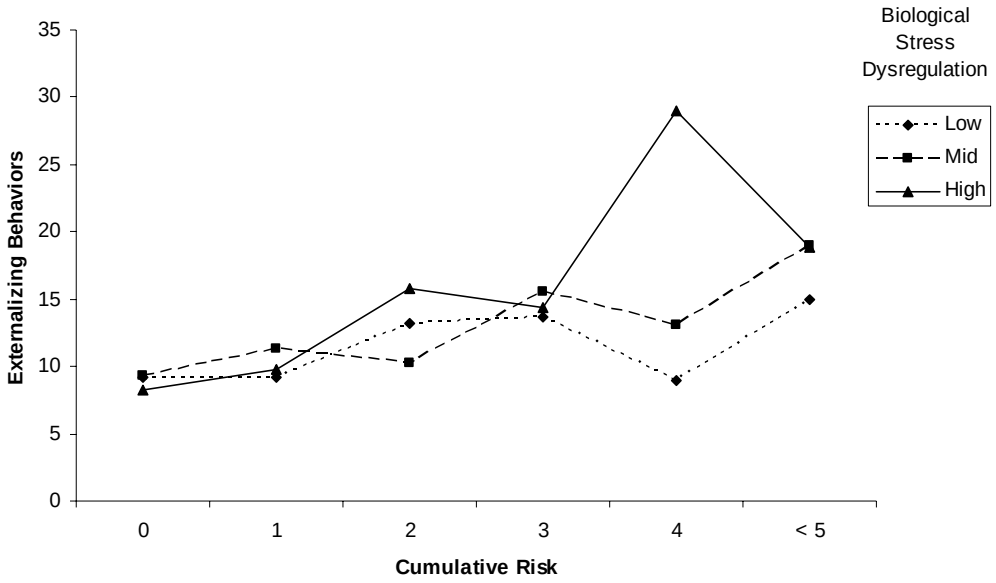

Figure 2. Relation of Cumulative Risk to Externalizing Behaviors for Different Levels Biological Stress Dysregulation

The moderation analysis was also conducted for internalizing behaviors. No significant interactions between cumulative risk and biological stress dysregulation were found.

Mediational analyses

Mediational analyses were performed to examine whether dysregulation in multiple biological stress systems helped account for the relation between the cumulative risk and behavioral problems. Test of mediation was conducted following procedures described by Baron and Kenny (1986). The requirements for mediation are (a) the independent variable must predict the dependent variable; (b) the independent variable must predict the mediator; and (c) when the mediator is included in the model

with the independent variable, the mediator must predict the dependent variable (d) the relation between the independent variable and the dependent variable is substantially reduced (Baron & Kenny, 1986). Under these requirements, if the independent variable becomes nonsignificant, full mediation is demonstrated. In other words, the relation between the independent variable and the dependent variable is fully explained by the mediator. If the mediator variable is significant and the effect of the independent variable on the dependent variable is reduced, partial mediation is demonstrated.

Because we found that (a) cumulative risk (the independent variable) predicted behavioral problems (see Behavioral problems in the results section), next, we tested (b) whether cumulative risk (the independent variable) predicts dysregulation in multiple biological stress systems (the mediator) with ordinary least squares (OLS) regression analyses. Gender and age were included as control variables and dysregulation in multiple biological stress systems at the previous wave was incorporated as a control. Interactions between cumulative risk and control variables were also tested in the model. The significant relationship between cumulative risk and biological stress dysregulation was curvilinear. The quadratic term of cumulative risk was significant, $b = .11$ (.04), $p < .01$, $f2 = .04$ and the linear term of cumulative risk was also significant, $b = -.48$ (.22), $p < .05$, $f2 = .03$. Thus, children's biological stress regulatory systems were less affected by low levels of cumulative risk, while their regulatory systems were more greatly impaired by higher levels of cumulative risk. In terms of the gender difference, boys had dysregulation in significantly more biological stress systems than girls, $b = -.58$ (.27), $p < .05$, $f2 = .02$. However, no significant interaction was found between cumulative risk and gender.

Next, we tested (c) whether dysregulation in multiple biological stress systems (the mediator) predicts behavioral problems (the dependent variable) and (d) whether

the effect of cumulative risk (the independent variable) on behavioral problems (the dependent variable) is reduced. We used only a linear term of cumulative risk for these analyses because the relationship between cumulative risk and internalizing behaviors was linear. A quadratic term of cumulative risk was not significant for internalizing behaviors and all three subscales of internalizing behaviors.

Table 5 summarizes findings from two models for internalizing behaviors. The first model tested the main effect of cumulative risk. The second model is the mediation model; it includes cumulative risk and biological stress dysregulation. Gender was included as a control variable in both models and age was included as a control variable in the first model. Interactions between the control variables and the mediator were explored in the both models. As shown in Table 5, biological stress dysregulation significantly predicted internalizing behaviors when children were older, $b = .07$ (.03), $p < .01$, $f2 = .04$. The older children (age late 13 to 16) were more likely to develop internalizing behaviors when they had dysregulation in multiple biological stress systems (see Figure 3). The main effect of cumulative risk on internalizing behavior was still significant after biological stress dysregulation was introduced in the model, $b = 1.01$ (.34), $p < .01$, $f2 = .05$. However, the value of b of cumulative risk, indicating the main effect of cumulative risk on internalizing behaviors, was reduced by 12% by including biological stress dysregulation in the model. This was a significant reduction in the b, $t (192) = 2.33$, $p < .01$. No significant gender effect or interaction between gender and biological dysregulation were found.

Similar results were found among subscales of internalizing behaviors. As shown in Table 5, biological stress dysregulation significantly predicted anxious/depressed behaviors when children were older, $b = .04$ (.02), $p < .01$, $f2 = .04$. The main effect of cumulative risk was significant, $b = .64$ (.20), $p < .01$, $f2 = .05$.

Table 5.
Mediational Analysis of Internalizing Behaviors, Cumulative Risk and Biological Stress Dysregulation, Controlling for Age and Gender

	Total R^2	ΔR^2	$F \Delta R^2$	*b* (*SE*)
1. Internalizing Behaviors				
Model 1				
Cumulative risk	.07	.05	12.40**	1.14 (.33)**
Model 2				
Biological Stress Dysregulation	.02	.00	.04	-10.71 (4.07)**
Age	.02	.00	.61	-.14 (.08)
Biological Stress Dysregulation*Age	.06	.04	7.80**	.07 (.03)**
Cumulative risk	.10	.04	9.10**	1.01 (.34)**
1.1. Anxious/Depressed Behaviors				
Model 1				
Cumulative risk	.08	.06	15.18***	.74 (.19)***
Model 2				
Biological Stress Dysregulation	.01	.00	.23	-6.32 (2.40)**
Age	.01	.00	.66	-.08 (.05)
Biological Stress Dysregulation *Age	.05	.04	7.91**	.04 (.02)**
Cumulative risk	.10	.05	10.35**	.64 (.20)**
1.2. Social Withdrawn Behaviors				
Model 1				
Cumulative risk	.05	.03	6.78*	.24 (.09)*
Model 2				
Biological Stress Dysregulation	.02	.01	1.10	-2.01 (1.18) †
Age	.03	.01	1.29	-.02 (.02)
Biological Stress Dysregulation *Age	.05	.02	4.33*	.01 (.01) †
Cumulative risk	.07	.02	4.44*	.21 (.10)*
1.3. Somatic Complaints				
Model 1				
Cumulative risk	.03	.02	4.10*	.24 (.12) *
Model 2				
Biological Stress Dysregulation	.01	.01	.96	-2.56 (1.46) †
Age	.01	.00	.01	-.04 (.03)
Biological Stress Dysregulation *Age	.03	.02	3.22	.02 (.01) †
Cumulative risk	.05	.02	3.70	.23 (.12) †

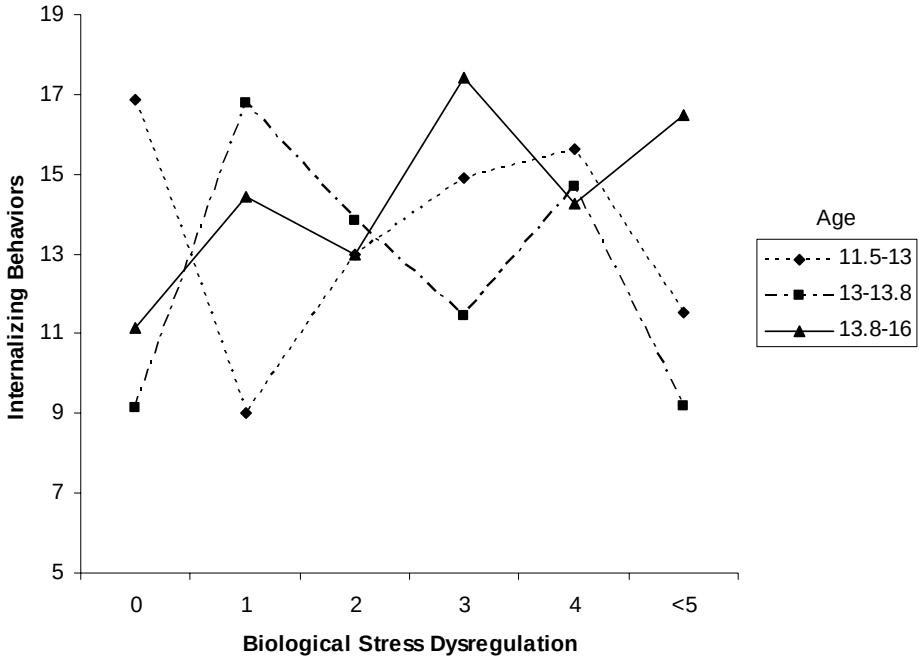

Figure 3. Relation of Biological Stress Dysregulation to Internalizing Behaviors for Different Age Groups

However, the value of b of cumulative risk was reduced by 14% by including biological stress dysregulation in the model, and this was a significant reduction in the b, $t(192) = 2.0$, $p < .01$. Thus, the older children exhibited more anxious/depressed behaviors when they had dysregulation in multiple biological stress systems. Similar trends between biological stress dysregulation and social withdrawal behaviors and somatic complaints were found (see Table 5). Biological stress dysregulation was marginally associated with social withdrawal behaviors among older children, b = .01 (.01), $p < .10$, $f2 = .02$; and with somatic complaints when children were older, b = .02 (.01), $p < .10$, $f2 = .02$. The main effect of cumulative risk was significant for social withdrawal behaviors, b = .21 (.10), $p < .05$, $f2 = .02$. The value of b of cumulative risk was reduced by 12% when biological stress dysregulation is included in the model for social withdrawal behaviors. The significant main effect of cumulative risk on

somatic complaints became nonsignificant when biological stress dysregulation was included in the model for somatic complaints. There were no significant gender effects on interactions between gender and biological dysregulation.

Cumulative risk predicted internalizing behaviors and the three subscales of internalizing behaviors. Biological stress dysregulation also predicted internalizing behaviors and subscales of internalizing behaviors among older children. However, whereas cumulative risk had a curvilinear relationship with biological stress dysregulation, we found that cumulative risk and biological stress dysregulation had a linear relationship with internalizing behaviors. Thus, the results were not substantiated to show whether biological stress dysregulation mediated the relations between cumulative risk and internalizing behaviors. Nevertheless, the effect of cumulative risk on the behavioral problems was significantly reduced when biological stress dysreugaltion was included in the mediation model. This finding suggests that both cumulative risk and biological stress dysregulation have significant effects on internalizing behaviors. Furthermore, cumulative risk may have an indirect effect on internalizing behaviors through biological stress dysregulation.

Parallel mediational analyses were tested for externalizing behavioral problems. However, no significant mediating effects of biological stress dysregulation were found. Interaction between biological stress dysregulation and control variables and curvilinear relations of biological stress dysregulation were tested. None of them was proved to be significant.

CHAPTER 4

Discussion

This study investigated the potential role of dysregulated multiple stress systems in cumulative risk and behavioral problems in early adolescence. As predicted, cumulative risk influences behavioral problems. Biological stress dysregulation appeared to play a two-part role. First, biological stress dysregulation was associated with internalizing behaviors when the children were older. Biological stress dysregulation may have an indirect effect on the relationship between cumulative risk and internalizing behavioral problems among older children. Second, biological stress dysregulation moderated the effect of cumulative risk on externalizing behavioral problems. This interaction occurred irrespective of the child's age. Negative impacts of cumulative risk on externalizing behavioral problems appeared to be aggravated by biological stress dysregulation.

Consistent with prior research in this area (Forehand, Biggar, & Kotchick, 1998; Rutter, 1979; Sameroff et al., 1987), children exposed to higher levels of cumulative risk showed more behavioral problems in early adolescence. Exposure to cumulative risk was also associated with dysregulation in multiple biological stress systems. Most prior research has only included familial or psychosocial factors in cumulative risk. Using a more comprehensive measure of cumulative risk incorporating environmental risk factors such as noise, crowding at home, and housing quality, we found multiple psychosocial and physical risk factors may have detrimental effects on behavioral regulation as well as biological stress regulation in early adolescence.

The strong linear relationship between cumulative risk and behavior problems in the current study supports the theory that cumulative risk has an additive effect on the severity of behavioral problems—the greater the number of risks, the more severe

behavioral problems become (Appleyard et al. , 2005; Sameroff et al. , 1998). A competing theory suggests a threshold effect of cumulative risk on behavioral problems – behavioral problems dramatically increase when children are exposed to more than a certain number of risk factors (Biederman et al., 1995; Forehand et al., 1998; Rutter, 1979). To test this theory, I examined the curvilinear relationship of cumulative risk on behavioral problems. However, the results were non-significant for both internalizing and externalizing behavioral problems.

In testing the mediation model of dysregulation in multiple biological stress systems, significant curvilinear links were found between cumulative risk and biological stress dysregulation. This suggests that multiple biological stress systems became deteriorated when a child is exposed to more than a certain numbers of risk factors. In his study with children age 8-10, Evans (2003) also found that both linear and quadratic terms of cumulative risk predicted allostatic load, indicative of more severe biological stress dysregulation. The curvilinear findings between cumulative risk and biological stress regulation provide more evidence for the importance of studying cumulative risk and multiple stress biological stress systems. By studying relations between one single risk factor and a specific biological stress outcome, we may miss or under appreciate stress and health dynamics since a. stressors tend to covary, especially among the disadvantaged children; b. the adverse impacts of cumulative risk exposure far exceed qa singular impact.

The analyses of the three subscales of internalizing behaviors, the physiological stress response system predicts anxious/depressed behaviors when children were older. This finding suggests that exposure to cumulative risk may exhaust capacities of stress regulatory systems. When multiple biological stress systems are impaired, children have more difficulties in emotional and behavioral

regulation and suffer from high anxiety, depression, or social withdraw (Bauer, Quas, & Boyce, 2002; Grant et al., 2003; Repetti, Taylor, & Seeman, 2002).

Findings of the current study also suggest that biological stress dysregulation interacts with age in its effect on internalizing behavioral problems. Age alone does not predict internalizing behavioral problems. Instead, the dysregulation in multiple biological stress systems among older children (age late 13 to16) led to internalizing problems, whereas, for younger children (age 11 to mid 13), this did not occur. One potential explanation for this phenomenon is that the transition to high school generates higher stress and place higher demands on stress regulation systems, making them more vulnerable to internalizing problems. School transition is a significant risk factor for emotion instability and depression, especially for girls (Robinson, Garber, & Hilsman, 2004). Hormonal changes as a result of puberty may also interact with biological stress systems to increase vulnerability to internalizing problems. Age 13-15 is also a time for most children to begin experiencing puberty. Hormones (estrogens and androgens) responsible for the development of secondary sex characteristics increase during this age period (Petersen & Taylor, 1980), and these hormonal changes were related to depression and negative affect (Angold, Costello, Erkanli, & Worthman, 1999). The stronger impact of biological dysregulation in older adolescents could also reflect greater duration of exposure to cumulative risk. Chronic exposure to risks may undermine children's ability for coping and alter the efficiency of biological stress regulatory systems (McEwen, 2003).

With regard to externalizing behavioral problems, however, the data suggest a different pattern. Dysregulation in multiple biological stress systems moderates the impact of cumulative risk on externalizing behaviors. This finding supports the diathesis-stress model (Hammen, 2005). Dysregulation in multiple biological stress systems moderated the negative effect of cumulative risk only on externalizing

behavioral problems, specifically delinquent behaviors. There are few studies that have directly tested the moderating role of biological stress regulation. Recent behavioral genetic studies may help us understand genetic effects as a moderator of the relationship between family environment and delinquent behavioral problems. A functional polymorphism in the gene encoding the monoamine oxidase A (MAOA), moderated the effect of maltreatment on antisocial behaviors among children (Caspi et al., 2002). Low-activity MAOA genotype did not independently predict antisocial problems (Caspi et al., 2002), consistent with my finding that allostatic load alone did not predict delinquent behaviors. However, low-activity MAOA genotype increased the likelihood of developing antisocial problems among maltreated children. The MAOA gene encodes the MAOA enzyme, an important inhibitor of three kinds of neurotransmitters: norepinephrine, dopamine, and serotonin. As has been demonstrated in mice, low activity of MAOA genotype is linked to elevated norepinephrine, dopamine and serotonin levels (Cases et al., 1995). Thus, low MAOA activity may affect behaviors through elevated reactivity of multiple biological stress systems, related to increased levels of the neurotransmitters in humans (Caspi et al., 2002). A similar gene-environment interaction was found for conduct disorders among males aged 8 to 17 (Foley et al., 2004). Males with low-activity MAOA genotype were more likely to develop conduct disorders when family adversity (parental neglect, inconsistent parental discipline, and marital conflict) in childhood was high (Foley et al., 2004). In sum, despite the lack of empirical evidence directly targeting early adolescence, results of studies on other age groups provide some insight into the moderating role of biological stress dysregulation on the relationship between cumulative risk and delinquent behaviors in early adolescence.

The current study has found that biological stress dysregulation was a moderator only for delinquent behaviors but not for aggressive behaviors. This finding

seems to be contrary to the traditional belief that aggressive behaviors and delinquent behaviors tend to co-occur (Tremblay, Masse, Perron, LeBlanc, Schwartzman, & Ledingham, 1992), and share similar underlying mechanisms (Lorber, 2004). One possible explanation for the contradictory finding is that the co-occurrence of aggressive behaviors and delinquent behaviors may be more observable among people with more severe, clinical cases of behavior problems. Indeed, as shown by an earlier study examining more than 2500 children aged 4-18, the comorbidity rate between aggressive and delinquent behaviors was found to be 45% among clinical samples. But the rate dropped to nearly half, 28%, among people in the general population (McConaughy & Achenbach, 1994). This implies that the underlying process and risk factors involved in the development of aggressive and delinquent behaviors at moderate levels might be different from that at more severe levels. This is consistent with our finding that biological stress dysregulation affects these two types of externalizing behaviors differently.

Other evidence suggesting differences between aggressive and delinquent behaviors is the fact that these two behavior types follow separate developmental trajectories (Stanger, Achenbach & Verhulst, 1997). For boys and girls, aggressive behaviors decrease as they grow from early childhood to adolescence, whereas delinquent behaviors increase as they grow from late childhood to late adolescence (Loeber, 1982; Stanger, et al., 1997). Children in the current study were in their early adolescence, the period when the frequency of delinquent behaviors is on the rise. During this period, the biological predisposition that influences biological stress regulation plays a more critical role in the development of delinquent behaviors. Considering that aggressive behavior is relatively stable throughout early childhood until adolescence, biological predisposition in early childhood may be more critical for aggressive behaviors. These different developmental changes over time may explain

why biological system acts as a significant moderator only for delinquent behaviors but not for aggressive behaviors among early adolescence (12-15 years).

Although the current study has found some evidence of gender differences in the prevalence of behavioral problems (e.g., internalizing behaviors are more common among girls than boys), it uncovered no gender difference in the mediation and moderation role of biological stress dysregulation. Other studies have found that, during early adolescence, internalizing behaviors become more common among girls, whereas externalizing behaviors become more comment among boys (Loeber & Farrington, 2000; Twenge & Nolen-Hoeksema, 2002). The findings from the current study suggest that the biological stress system plays a similar role in both genders. However, different environmental factors or other individual risk factors, such as coping strategies or types of peer groups, could contribute to the gender differences in the distribution of various behavioral problems among girls and boys.

The findings on mediation and moderation models of biological stress regulatory systems suggest a new perspective to understand dysregulation in multiple biological stress systems. According to allostatic load theory (McEwen, 2003), environmental risks are the primary factors that affect allostatic load, in turn, lead to causes behavioral problems. However, other inherited factors can also affect stress regulatory systems, which, by interacting with environmental risks, influence behavioral problems.

Hence, understanding of the interceding roles of biological stress regulation as a moderator and a mediator may be important in order to explain how heritable and environmental factors contribute to behavioral problems. Both heritable and environmental factors influence the development of internalizing and externalizing behavioral problems. Previous studies examining the genetic and environmental contributions to children's behavioral problems found that among children age

between 10 and 15, the continuity of externalizing behaviors was influenced mostly by genetic factors, whereas the continuity of internalizing behaviors was best explained by environmental factors (Van der Valk, Verhulst, Neale, & Boomsma, 1998). From age seven to twelve, the contribution of genetic factors to externalizing behaviors ranged between 43% and 62%, whereas the contribution of genetic factors to internalizing behaviors ranged between 28% and 48% for both boys and girls (Haberstick, Schmitz, Young, & Hewitt, 2005).

As we discussed earlier, biological stress regulation systems can be also affected by both genetic and environmental variables. Thus, the current study's findings on the significant relations between internalizing behaviors and biological stress dysregulation when the biological stress dysregulation was a product of environmental variables − cumulative risk, may suggest a reason why internalizing behaviors are more influenced by environmental factors than by genetic factors. On the other hand, we found that biological stress dysregulation was independent of environmental variables − cumulative risk, however, it made children more apt to externalizing behaviors when they lived in cumulative risk environment. This may explain why externalizing behaviors are predicted by genetic factors or inherited biological predisposition better than environmental factors alone.

Thus, testing both the mediational and moderational model of biological stress regulation for behavioral problems is important in order to capture both influencing pathways. In addition, the measurement of biological risk requires the inclusion of several physical indicators. Then, the comprehensive indicator of biological stress regulatory systems can account for not only environmental risks but also genetic factors.

The findings of the current study on the mediation and moderation roles of behavioral problems should be interpreted with caution. First, the current study has

tested the mediating and moderating effects of biological stress systems on behavioral problems in two separate sets of analyses. However, the influence of the biological stress regulator system on a particular behavioral problem may not be explained as either exclusively a mediating or a moderating effect. Exposure to cumulative risks reduces the efficiency of the biological stress regulatory systems, which can further make behavioral regulation more difficult. This phenomenon demonstrates how biological stress regulatory systems can act as a mediator between cumulative risks and behavioral problems. Furthermore, the inefficient biological stress system can impair children's ability to regulate their behaviors during later developmental stages when they live in an environment with higher risks. It implies that the biological stress system can also have moderating effects on the same behavioral problems when children are older. Indeed, studies suggest that the mediating and the moderating roles of dysregulation in multiple biological stress systems might not be restricted to only internalizing or only externalizing behavioral problems. For instance, a gene-environment interaction has been uncovered for depression (Caspi et al., 2003; Kaufman et al., 2006). Therefore, in order to understand the dynamic process in which behavioral problems develop, longitudinal studies should be carried out to measure how these problems relate to the biological stress regulatory system and cumulative risk over time.

Second, the statistically significant relations between cumulative risk and biological stress dysregulation reflect low correlations and should be interpreted with caution. The significant association between cumulative risk and biological stress regulatory systems depended on functions of different levels of maternal responsiveness. That is, cumulative risk significantly predicted biological stress dysregulation only when children lived with less responsive mothers.

Third, in addition to the factors considered by the current study, there may be other factors, such as cognitive and socioemotional factors, that may affect behavioral problems. Temperament (Morris et al., 2002), effortful control (Eisenberg et al., 2003), coping strategies (Compas, Connor-Smith, & Jaser, 2004), and appraisal styles (El-Sheikh & Harger, 2001) are examples of factors known to mediate or moderate the relationship between risks and behavioral problems. These factors may interact with allostatic load or biological stress regulation to affect behavioral outcomes (Repetti et al. , 2002). Moreover, other physiological systems may also influence behavioral problems. For example, testosterone levels are on the one hand significantly associated with aggressive behaviors among boys (Raine, 2002), and on the other hand, moderate the effect of risks on delinquent behaviors (Dabbs & Morris, 1990).

Fourth, the current study has defined higher morning basal cortisol level as a risk factor for biological stress regulatory systems. Literature on allostatic load—a well-documented index of dysregulation in multiple physiological stress regulatory systems—has consistently used higher cortisol level to indicate dysregulation of cortisol (Seeman et al., 2004; Evans, 2003; McEwen, 2003). However, recent studies have shown that low and dampened cortisol levels in response to stress are also related to more behavioral problems (Shirtcliff et al., 2005; McBurnett, Lahey, Rathouz,& Loeber, 2000). Exposure to excessive stress such as living with abusive parents was also related to flattening circadian rhythm of cortisol (Gunnar & Vazquez, 2004). Thus, it is important to investigate whether cumulative risk does dampen cortisol reactivity and affect circadian rhythm, which further hampers behavioral regulation in early adolescence.

Lastly, the current study is based on a sample mostly composed of rural and white children. Therefore, it is a possibility that its finding may not be generalized to children of other demographic backgrounds.

In conclusion, the results of this study contribute to our understanding of underlying biological process of the development of behavioral problems in early adolescence, particularly, the role of multiple biological stress systems in this process. Cumulative risk predicts more internalizing and externalizing behaviors in early adolescence. Regulatory functions in multiple biological stress systems were impaired by cumulative risk and they may cause children to be more vulnerable to internalizing behaviors. Compared to children age 11-13, older children age 13-15 developed internalizing behaviors when their biological stress systems were dysregulated. On the other hand, dysregulation in multiple biological stress systems moderates the effects of cumulative risk on externalizing behaviors. The results of this study contribute to our understanding of the underlying processes involved in the development of behavioral problems in early adolescence. In particular, we studied the role of multiple biological stress systems in this process. Findings also provide insight on unanswered research questions worth future investigation—how biological, cognitive, and socioemotional factors mediate and moderate the adverse effect of cumulative risk on behavioral problems. A longitudinal study would help us understand the long-term consequence of dysregulation in multiple biological stress systems in early adolescence on behavioral problems and psychiatric disorders.

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