Cumulative Risk, Maternal Responsiveness, and Allostatic Load Among Young Adolescents

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The purpose of this study was to examine the impact of cumulative risk exposure in concert with maternal responsiveness on physiological indicators of chronic stress in children and youth. Middle-school children exposed to greater accumulated psychosocial (e.g., family turmoil, poverty) and physical (e.g., crowding, substandard housing) risk factors manifested higher levels of allostatic load, a physiological marker of cumulative wear and tear on the body caused by the mobilization of multiple physiological response systems. This effect was longitudinal, residualizing allostatic load 3–4 years earlier when the youth were in elementary school. This effect, however, occurred only among adolescents with mothers low in responsiveness. Cumulative risk was also associated with dynamic cardiovascular processes in response to an acute stressor (mental arithmetic). Higher risk was associated with muted reactivity and slower, less efficient recovery in blood pressure. These dynamic cardiovascular effects occurred irrespective of maternal responsiveness.

Keywords: cumulative risk, stress, maternal responsiveness, allostatic load

Elementary-school children exposed to accumulated psychosocial (e.g., family turmoil, violence) and physical (e.g., noise, substandard housing) risk factors manifest higher allostatic load relative to children with lower levels of cumulative risk (Evans, 2003). Allostatic load is a physiological marker of cumulative wear and tear on the body caused by the mobilization of multiple physiological systems in response to environmental demands (McEwen, 2002; McEwen & Seeman, 1999; McEwen & Stellar, 1993; Sterling & Eyer, 1988). This study extends developmental research on cumulative risk in several respects. We incorporate a longitudinal design examining risk and allostatic load among a young adolescent sample. Most cumulative risk research has examined preadolescent children, and only Evans (2003) examined physiological sequelae of cumulative risk exposure. We also incorporate a protective factor—maternal responsiveness—that potentially could buffer the adverse effects of cumulative risk on allostatic load. There is very little work on protective factors and cumulative risk exposure in adolescents and no work on protective factors and allostatic load among people of any age. We also investigate dynamic cardiovascular functioning (i.e., blood pressure reactivity and recovery to an acute stressor) in relation to cumulative risk exposure. This is the first study to examine this topic and only the second study of cardiovascular recovery among a sample of children or youth. All prior studies of recovery except one used adult samples.

Overexposure to a combination of physiological mobilizations across different systems in response to adaptive demands over time alters the ability of the body to respond efficiently to environmental demands (Karlamangala, Singer, McEwen, Rowe, & Seeman, 2002; McEwen, 1998, 2002; Seeman, McEwen, Rowe, & Singer, 2001; McEwen, Rowe, & Singer, 2000). In contrast to homeostasis, allostatic load posits a more dynamic and interactive set of multiple physiological systems (i.e., neuronal, endocrinological, cardiovascular, and immunological) of equilibrium maintenance. Operation of these dynamic interactive systems causes the body to continuously adjust its operating range, producing downward regulation of metabolic activities in order to maintain internal stability congruent with shifting environmental demands (McEwen, 1998, 2000, 2002; McEwen & Seeman, 1999). Allostatic load has generated considerable interest in both the neuro-science and medical communities. Whereas alterations in singular physiological risk factors (e.g., blood pressure) produced by environmental demands reveal modest consequences for morbidity and eventually mortality, the combination of singular changes across multiple physiological indicators captured by allostatic load portends marked elevations in physical and possibly psychological morbidity (Karlamangala et al., 2002; McEwen, 1998, 2002; Seeman et al., 1997, 2001, 2004). Moreover, allostatic load also appears to provide a cogent model of how physiological changes accompanying adaptive demands from the environment can, in turn, alter neurological activity responsible for mental and cognitive disorders (McEwen, 2000, 2002).

In addition to scant research on young adolescents and cumulative risk exposure, and no data on allostaticity among adolescents, this is an important age group to consider in its own right. Children...
between age 11 and 13 years undergo rapid dramatic changes in physical growth and endocrinological activity. These physiological alterations coincide with marked social changes, particularly enhanced autonomy, more intensive peer associations, and onset of romantic relationships, that have obvious implications for cumulative risk exposure (Compas, Hinden, & Gerhardt, 1995; Grotevant, 1998; Steinberg & Morris, 2001). This age period also marks a major transition point from the comforts of a relatively small, often homogeneous elementary school, with most of the day spent with one adult in one place along with the same group of same-age peers, to a larger, often more heterogeneous middle school with greater variability in where and with whom time is spent during the day. Lengthening of the school day and, especially in rural areas, much longer bus rides can also dramatically increase the amount of time one is away from home.

Nearly all studies of cumulative risk have relied on urban samples, and most have focused on low-income children of color. The major limitations of such sample restrictions include (a) most high risk families in America are White (U.S. Census Bureau, 2005), (b) both the degree and persistence of material deprivation is considerably worse in rural areas compared with urban centers (Auchincloss & Hadden, 2002; Blank, 2005; Mathemtica Policy Research, 2005; Sherman, 1992), and (c) by focusing on low-income samples, the degree of variance in cumulative risk exposure is truncated; thus, estimates of cumulative risk and developmental disarray may be downwardly biased.

Evans (2003) is the only published documentation of a link between cumulative risk exposure and allostatic load in children. One study has uncovered similar results in older adults. B. H. Singer and Ryff (1999) found that retrospective reports of cumulative risk exposure over the life course among 59-year-old adults predicted allostatic load. A handful of studies suggest that exposure to singular rather than cumulative risk factors has the potential to elevate allostatic load in children, although none of these studies assessed allostatic load, instead monitoring a single physiological marker of stress. Adolescents in more hostile, demanding, and noisy environments had higher blood pressure throughout the day (Southard et al., 1986). Experimental exposure to simulated interpersonal conflict among adults elevated acute blood pressure in children (El-Sheik, Cummings, & Goetesch, 1989). Chronic exposure to high ambient noise levels as well as crowded homes correlates with elevated blood pressure and catecholamines in children (Evans, 2001).

Adverse environmental circumstances early in childhood, which include neglect, abuse, or severely deprived physical and social stimulation, influence the hypothalamic–pituitary axis (HPA) in children. This has been documented among children growing up in abusive families (Cicchetti & Rogosch, 2001; Hart, Gunnar, & Cicchetti, 1996) or in severely deprived institutional settings such as Romanian orphanges (Gunnar, 2000). Furthermore, children chronically exposed to loud ambient noise manifest elevated, basal cortisol levels (Evans, 2006; Ising & Braun, 2000).

One of the important objectives of the present article is to build upon and extend prior studies of risk exposure and physiological activity among children by examining cumulative rather than singular risk exposure. Most if not all of the childhood risk factors identified above as correlates of HPA or sympathetic–adrenal–medullary (SAM) impacts do not occur independently of one another. A unique and potentially key aspect of childhood poverty is the confluence of psychosocial and physical environmental risk factors that low-income children must contend with in their daily lives (Evans, 2004; Repetti, Taylor, & Seeman, 2002; Schell, 1997; Taylor, Repetti, & Seeman, 1997). Risks rarely occur in isolation, instead clustering among families living in poverty. Moreover, singular risk exposure pales in comparison with cumulative risk exposure in accounting for adverse socioemotional and cognitive outcomes in children (Ackerman, Kogos, Youngstrom, Schoff, & Izard, 1999; Evans, 2003; Gutman, Sameroff, & Cole, 2003; Lengua, 2002; Liaw & Brooks-Gunn, 1994; Rutter, 1983; Sameroff, 1998).

Another reason why it is valuable to examine cumulative risk exposure and allostatic load during earlier periods in the life course is because of the possibility that cumulative risks may accumulate to affect changes in cardiovascular dynamics as the body mobilizes in response to specific acute demands. Under normal conditions when confronted with an acute environmental demand, the body mobilizes metabolically to meet the requirements imposed by the immediate circumstances. Such energy mobilization is manifested by a sharp, rapid rise in cardiovascular activity, termed reactivity. As soon as the demand is over, the body rapidly returns it its prior resting state (Haynes, 1991; Krantz & Falconer, 1995; Linden, Earle, Gerin, & Christenfeld, 1997; McEwen, 1998, 2000). This latter process is called recovery. However, if the demands from the environment are excessive and/or occur over a prolonged period of time, allostatic load increases, and the system becomes overloaded. Typical healthy patterns of cardiovascular reactivity and recovery in response to an acute stressor no longer occur. Instead, one witnesses cardiovascular hyper-reactivity to acute environmental demands coupled with prolonged recovery to baseline levels (Diestbier, 1989; McEwen, 1998, 2000). Animal research (Diestbier, 1989) in addition to case reports of traumatized children suggest that very high levels of stressor exposure depress cardiovascular reactivity (Perry & Pollard, 1998). Exposure to violence and harsh parenting in public housing projects has also been associated with depressed cardiovascular reactivity among children (Kernichyn, Saegert, & Evans, 2001). Murali and Chen (2005) found similar relations between the experience of violence and muted cardiovascular reactivity among adolescents. Adolescents with a history of a high number of life events manifested muted cardiovascular reactivity to several acute stressors (Boyce & Chesterman, 1990). Analogous results were uncovered by Mengel et al. (1992) in a study of diabetic adolescents. In a fascinating series of studies of children residing in rural Caribbean villages, Flinn (1999) has shown that chronically stressed children (e.g., persistent family conflict) evidence blunted cortisol responses to physical demands that evoke increased cortisol in children without chronic stress.

Potential linkages between cardiovascular recovery and chronic stressor exposure have not been examined among children or youth. We were able to uncover only one study of cardiovascular recovery in children. Jackson, Treiber, Turner, Davis, and Strong (1999) reported that African American, 11–15-year-old males took longer to recover from exposure to acute stressors compared with White children and females, respectively. These authors did not examine chronic stress as a factor in their study. Among adults, slower cardiovascular recovery to an acute stressor is associated with higher levels of chronic stress (Fleming, Baum, Davidson, Rectanus, & McArule, 1987; Lepore, Miles, & Levy, 1997; Pardine & Napoli, 1983; Schaubroeck & Ganster, 1993). Cardiovascular recovery to an acute stressor has also been linked to socioeconomic status (SES) in the Whitehall II study of British civil servants. Lower SES was correlated with delayed recovery (Steptoe et al., 2002).
We were also interested in scrutinizing maternal responsiveness as a potential protective factor for the impact of cumulative risk exposure on allostatic load. Although there is much literature on the protective effects of maternal responsiveness on socioemotional development (Bornstein, 1989; Bradley, Corwyn, Burchinal, McAdoo, & Garcia-Coll, 2001; Demo & Cox, 2000; Shonkoff & Phillips, 2000), no research has examined maternal responsiveness and allostatic load. There are, however, a few studies that have explored the potential buffering effects of maternal responsiveness on SAM and HPA activity levels in relation to stress. Children in families with lower levels of social support (Woodall & Matthews, 1989) and those who receive harsher, more punitive parenting (Gump, Matthews, & Raikkonen, 1999) evidence higher cardiovascular reactivity to an acute stressor. Children in families with more punitive parenting also evidence higher levels of resting blood pressure (Wright, Treiber, Davis, Bunch, & Strong, 1998). Children with less responsive parents and less secure maternal attachment show stronger HPA reactivity to acute stressors (Gunnar, 2000; Gunnar & Donzella, 2002). Children with more negative parent–child interactions also manifest elevated basal cortisol levels (Flinn & Englund, 1997; Luecken, 1998). Thus, although there is evidence that responsive parenting can influence physiological stress responses in relation to childhood exposure to singular stressors, none of this work has investigated the potential of parental responsiveness to buffer cumulative risk exposure among children or examined allostatic load outcome measures. Furthermore, the parenting research on physiological stress responses in children has examined basal or reactivity patterns only, neglecting the investigation of recovery from acute stressor exposure.

In sum, the primary objective of the present study was to replicate and extend the one existing study on cumulative risk and allostatic load in children with a longitudinal assessment of young adolescents. We also wanted to explore whether dynamic cardiovascular activity (i.e., reactivity and recovery to an acute stressor) would be influenced by cumulative risk. Finally, we were interested in the potential ameliorative effects of a well-documented resource for adolescents—maternal responsiveness—on cumulative risk and allostatic load.

We hypothesized that cumulative risk would elevate allostatic load over time. We expected that cumulative risk would also influence dynamic SAM activity, dampening cardiovascular reactivity and inhibiting recovery to an acute stressor. Finally, both of the above sets of physiological outcomes to cumulative risk were expected to be moderated by maternal responsiveness. We predicted that youth with more responsive mothers would be less adversely impacted by cumulative risk exposure.

Method

Participants

Participants for this second wave of a longitudinal study of rural poverty and child development were 207 seventh and eighth grade children (mean age 13.37 years, 52% male, 48% female) who had physiological data. In Wave 1 there were 339 elementary-school children (mean age 9.20 years).1 All of these youth lived in rural areas in upstate New York. They were recruited from public schools, New York State Cooperative Extension programs, and various antipoverty programs (< 5% Wave 1 refusal rate). Low-income families (income-to-needs ratio ≤ 1) were over sampled (53%) because this research program is focused on poverty and human development. Only one child per household participated in the study. The sample was predominantly White (94%), reflecting upstate, rural New York demographics. Attrition analyses revealed that children with higher levels of cumulative risk at Wave 1 (mean cumulative risk index = 3.57) were less likely to remain in the sample at Wave 2 (mean cumulative risk index = 1.79), t(336) = 8.82, p < .001. The individual components of cumulative risk that significantly reflected this pattern of selective attrition included poverty, housing quality, family turmoil, child separation from family, exposure to violence, and maternal high school drop out. There was no selective attrition, however, as a function of allostatic load.

Procedure

A standard protocol was used in data collection in each youth’s home at Wave 1 when the participants were in third through fifth grade (Evans, 2003) and then again when the youth was in seventh or eighth grade. The target youth and his or her mother were interviewed independently by two interviewers. The gender of the interviewer was matched to the youth’s gender in Wave 2.

Cumulative risk assessment. Nine domains of risk factors for young adolescents were assessed. Residential density was determined by dividing the number of people living in the household by the number of rooms (including bathrooms). Anyone sleeping in the home three or more nights per week was deemed a resident. Only rooms regularly used by family members were counted. Garages, hallways, attics, and basements were not counted unless regularly occupied (e.g., basement converted into a family room). Noise levels were estimated by a 2-hr monitoring of decibel levels (Leq, dBA) in the primary social space (typically the living room) in the home. Housing quality was assessed with a standardized, observer rating scale consisting of subscales measuring structural quality, maintenance, cleanliness and clutter, safety hazards, children’s resources, and climatic conditions (Evans, Wells, Chan, & Saltzman, 2000). The housing quality instrument has undergone extensive psychometric development. Exposure to family turmoil and child–family separation were determined by combining maternal and youth reports. Maternal responses came from the Life Events and Circumstances Checklist (Work, Cowen, Parker, & Wyman, 1990; Wyman, Cowen, Work, & Parker, 1991), which consists of multiple dichotomous (yes/no) item subscales for each of these stressor domains. Sample items include “Our child has been involved in serious family arguments” and “A close family member was away from home a lot.” The time period was the time between the initial wave of data collection and the present one, typically 2–3 years later. A calendar that the mother and interviewer filled in together with important personal and family events was used as a mnemonic device. Youth were also asked to indicate the occurrence of stressors (again with the aid of personally constructed calendar to facilitate recall) with a life event scale based on a revised Adolescent Perceived Events Scale (Compa, 1997) supplied to us by Bruce Compa. Youth indicated “yes” or “no” to whether each stressful event or circumstance had occurred. Sample items in each of the domains of family turmoil and child–family separation included “Pretty serious arguments or

1 See Evans (2003) for further details on Wave 1.
fights between parents” and “Parents getting divorced or separated.” From mothers, information about exposure to violence was obtained (e.g., “Our neighborhood has been unsafe”). For each of these stressor domains, risk was defined dichotomously (as 0 or 1) based upon a statistical criterion with 1 equal to a value greater than one standard deviation above the mean for the distribution of the specific risk factor across the entire sample of youth. A value of 0 was assigned for levels of exposure less than the cutoff. In addition to these six continuous risk factors, three additional categorical risks 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). Thus, the cumulative risk index could vary from each participant from 0 to 9.

Maternal responsiveness. Maternal responsiveness was measured by combining the youth’s perception of maternal responsiveness with observations of mother–child interaction during a cooperative game. A scale consisting of 11 items tapping both instrumental (e.g., help with homework) and emotional (e.g., willing to talk to me when needed) responsiveness was constructed. Youth answered each question on a 5-point scale (never, hardly ever, sometimes, fairly often, very often). The scale has good internal consistency (α = .84) and strong test–retest reliability (r = .92) over a 3-month period. Evidence for validity includes confirmatory factor analysis (two moderately correlated subscales of instrumental and emotional responsiveness), a nomological network of associations with other constructs (significant but modest negative correlations with income, household crowding, and a positive higher correlation with Moos’ Family Cohesion scale (Moos & Moos, 1986). We also rated maternal sensitivity to the child during a game of Jenga (Hasbro; Pawtucket, RI). Mothers and the target child took turns working together to build a tower of blocks as high as possible. The Jenga game was videotaped and then coded for maternal responsiveness. Interrater reliability for the sensitivity ratings was good (Ebel r = .95; Ebel, 1951). Raters were blind to the child’s cumulative risk and perceived maternal responsiveness scores. Sensitivity was coded for each 2.5-min period over the 15-min duration of Jenga. Sensitivity ratings ranged from 1 (Not at all characteristic. There are almost no signs of parent sensitivity. The parent rarely responds appropriately to the child’s cues.) to 5 (Highly sensitive/responsive. The parent displays consistent sensitivity to the child throughout the rating period.). Behavioral and affective indicators of sensitivity were operationalized in a coders’ manual summarized in Belsky, Crnic, and Woodworth (1995). These six observational ratings (α = .97) were then averaged for the overall observational rating. Perceived maternal responsiveness was significantly correlated (r = .68) with observer codings of maternal parental responsiveness to the child during the game. The perceived maternal responsiveness scale and the mean observational rating were each converted to z scores and then added to develop our index of maternal responsiveness.

Allostatic load. Resting blood pressure was monitored with a Critikon Dinamap Pro 100 (Critikon; Tampa, FL) while the youth sat and read quietly. Seven readings were taken every 2 min and the average of the second through seventh reading was used as the resting index. This procedure yields highly reliable indices of chronic resting blood pressure (Kamarck et al., 1992). Overnight (8 p.m. to 8 a.m.) urinary catecholamines and cortisol were assayed from the evening of the interview with the youth. All urine voided during this time was stored on ice in a container with a preservative (metabisulfite). Total volume was recorded, and four 10-ml samples were randomly extracted and then deep frozen at −80°C until subsequent assays were completed. The pH of two of the 10-ml samples was adjusted to 3 to further inhibit oxidation of catecholamines. Total unbound cortisol was assayed with a radio-immune assay (Contreras, Hane, & Tyrrell, 1986). Epinephrine and norepinephrine were assayed with high-pressure liquid chromatography with electrochemical detection (Riggins & Kissinger, 1977). Creatinine was assayed to control for differences in body size and incomplete urine voidings (Tietz, 1976). The samples were assayed by technicians blind to the respondent’s status.

Allostatic load (0–6) was calculated by summing the number of singular physiological indicators on which each child scored in the top quartile of risk. Allostatic load included overnight cortisol, epinephrine, and norepinephrine, resting diastolic and systolic blood pressure, and an index of fat deposition (body mass index = kg/m²). Prior studies of allostatic load in adults (Kubzansky, Kawachi, & Sparrow, 1999; Seeman et al., 2002; B. H. Singer & Ryff, 1999) and in children (Evans, 2003) have used similar metrics, combining multiple physiological indicators of risk into one total allostatic load index.

We did not incorporate cardiovascular reactivity and recovery into the allostatic load index for several reasons. First, prior studies of allostatic load have not included reactivity or recovery measures. Second, unlike the other measures included in prior allostatic load indices, reactivity and recovery have not yet been shown to be independent risk factors for morbidity. Third, dynamic SAM or HPA processes in response to an acute stressor likely reflect tertiary outcomes, which are caused by chronically elevated SAM and HPA levels (McEwen, 2000; McEwen & Seeman, 1999). Fourth, because there is only one prior study of cardiovascular recovery in children and no studies of cumulative risk and reactivity, we felt it was important to examine these impacts separately from allostatic load.

Cardiovascular reactivity and recovery. Immediately following the physiological resting period, the youth was given a “math test” without warning to assess cardiovascular reactivity and recovery. Blood pressure monitoring continued during and immediately following the mental arithmetic task. The youth’s task was to mentally subtract a two-digit number from a four-digit number continuously and report aloud the correct answer. The youth did this task for 12 min without writing down any calculations. Every 4 min, the number to be subtracted was changed. Thirty seconds after the test was completed, the first of five blood pressure readings was taken while the youth again sat quietly and read for a period of 10 min. This constituted the cardiovascular recovery procedure. At the end of the math test the participant was assured that no more tests would be given and he or she could relax and read while the blood pressure monitoring continued for a little longer. Mental arithmetic is a common stress induction stimulus used in reactivity protocols for both children and adults (Gump & Matthews, 1999; Krantz & Manuck, 1984; Matthews, Gump, Block, & Allen, 1997).

Results

Cumulative Risk Exposure

Table 1 provides descriptive information on each of the singular risk factors as well as the total cumulative risk index. Thirty
percent of the sample had zero risk factors, 24% had one risk, 17% two risks, 14% had three risks, 9% had four risks, and 6% had five or more risks. For the analyses of cumulative risk and allostatic load, cumulative risks of five or more were combined into one category, given small sample sizes at the upper end.

Allostatic Load

Longitudinal analyses with orthogonal least squares regression examined the relations between cumulative risk, maternal responsiveness, and the interaction of cumulative risk and maternal responsiveness on allostatic load. The analysis incorporated Wave 1 allostatic load as an initial control variable in the regression model. Gender was also included as a control given its significant relations with many of the physiological measures. We examined potential interactive effects for gender and cumulative risk but found none. The statistical controls, each of the main effects, and their multiplicative term were also centered to reduce multicollinearity (Aiken & West, 1991).

As indicated in Table 2 and shown in Figure 1, there was an interaction of maternal responsiveness and cumulative risk on allostatic load at Wave 2, $b = -0.08$ ($SE = .04$), $p < .05$, $f^2 = .02$, statistically controlling for Wave 1 allostatic load and gender. Neither of the main effects for cumulative risk or maternal responsiveness were significant (see Table 2). The regression plots depicted in Figure 1 were calculated at one standard deviation above and below the mean of maternal responsiveness, as well as at the mean. This was done for descriptive purposes only; the orthogonal least squares analysis maintained the continuous nature of cumulative risk, maternal responsiveness, and their multiplicative interaction term. Analyses of the simple slopes revealed that as cumulative risk increased, allostatic load rose but only for those youth with mothers who were low in responsiveness, $t(169) = 1.85$, $p < .05$. The simple slopes at the mean, $t(169) < 1.00$, and at one standard deviation above the mean, $t(169) < 1.00$, were not significant.

Cardiovascular Reactivity and Recovery

There were main effects of cumulative risk on both cardiovascular reactivity and recovery. Maternal responsiveness and its interaction with cumulative risk had no effect on dynamic cardiovascular functioning. Reactivity was assessed by subtracting the baseline level (i.e., the mean of Readings 2–7 [Reading 1 was discarded] while sitting quietly and reading) from the mean of the six blood pressure readings during the mental arithmetic task. This is the standard procedure for the calculation of cardiovascular reactivity (Krantz & Falconer, 1995; Matthews et al., 1987, 1997). Gender was also included as a control. Gender did not interact with cumulative risk to impact either reactivity or recovery. Because we did not assess dynamic cardiovascular parameters in Wave 1 of this study, the reactivity and recovery data are cross-sectional and not longitudinal, as is the allostatic load analysis above. As cumulative risk increased, both systolic reactivity, $b = -0.08$ ($SE = .33$), $p < .05$, $f^2 = .04$, and diastolic reactivity, $b = -0.08$ ($SE = .24$), $p < .05$, $f^2 = .03$, were muted (see Table 3). As can be seen in Figure 2, participants exposed to more cumulative risk exhibited lower levels of reactivity in response to the acute stressor, mental arithmetic.

Recovery, the efficiency with which physiological function approached basal levels, was analyzed with multilevel (time nested within cumulative risk) hierarchical linear modeling (J. D. Singer & Willett, 2003). This analytic approach allows one to calculate interindividual heterogeneity in change over time: in the present

<table>
<thead>
<tr>
<th>Measure</th>
<th>$M$</th>
<th>$SD$</th>
<th>Proportion of sample with risk factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cumulative risk factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crowding (no. of people per room)</td>
<td>0.56</td>
<td>0.19</td>
<td>.02</td>
</tr>
<tr>
<td>Noise (Leq, dBA)</td>
<td>60.57</td>
<td>6.46</td>
<td>.11</td>
</tr>
<tr>
<td>Housing problems (0–2)</td>
<td>0.59</td>
<td>0.31</td>
<td>.07</td>
</tr>
<tr>
<td>Family separation (0–12)</td>
<td>2.23</td>
<td>1.73</td>
<td>.24</td>
</tr>
<tr>
<td>Family turmoil (0–9)</td>
<td>2.78</td>
<td>2.04</td>
<td>.14</td>
</tr>
<tr>
<td>Violence (0–5)</td>
<td>0.68</td>
<td>0.88</td>
<td>.11</td>
</tr>
<tr>
<td>Poverty line (0 or 1)</td>
<td></td>
<td></td>
<td>.18</td>
</tr>
<tr>
<td>Single parent (0 or 1)</td>
<td></td>
<td></td>
<td>.43</td>
</tr>
<tr>
<td>Maternal high school dropout (0 or 1)</td>
<td></td>
<td></td>
<td>.07</td>
</tr>
<tr>
<td>Cumulative risk (0–9)</td>
<td>1.67</td>
<td>1.55</td>
<td></td>
</tr>
</tbody>
</table>

Table 2

Regression Results for Allostatic Load, Cumulative Risk, and Maternal Responsiveness, Controlling for Wave 1 Allostatic Load and Gender

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Total $R^2$</th>
<th>$\Delta R^2$</th>
<th>$F$</th>
<th>df</th>
<th>$b$</th>
<th>SE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cumulative risk</td>
<td>.12</td>
<td>.00</td>
<td>&lt;1.00</td>
<td>1,171</td>
<td>.06</td>
<td>.07</td>
</tr>
<tr>
<td>Maternal responsiveness</td>
<td>.12</td>
<td>.00</td>
<td>&lt;1.00</td>
<td>1,170</td>
<td>-.11</td>
<td>.08</td>
</tr>
<tr>
<td>Cumulative Risk × Maternal Responsiveness</td>
<td>.14</td>
<td>.02</td>
<td>4.11*</td>
<td>1,169</td>
<td>-.08*</td>
<td>.04</td>
</tr>
</tbody>
</table>

* $p < .05$. 

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case, how intraindividual blood pressure scores recover as a function of cumulative risk exposure. Figure 3 shows millimeters of mercury for diastolic blood pressure recovery and the timing of the blood pressure measures taken every 2 min throughout the recovery period. The initial recovery reading occurred 30 s after cessation of the mental arithmetic task and every 2 min after that for a total of five readings. Recovery was analyzed by setting the intercept for each participant at their value for the last reading during the reactivity period. The three line graphs in Figure 3 depict exposure to 0–1, 2–4, and 5 or more cumulative risks. This is for descriptive purposes only. The actual analyses maintained the continuous nature of the cumulative risk metric (i.e., 0–5).

As can be seen in Figure 3, diastolic blood pressure recovery is less efficient (i.e., slower), with higher levels of cumulative risk exposure, \( b = .13 \) (SE = .05), \( p < .01 \). This significant effect is for the quadratic function, controlling for the linear function, which was also significant. No effect size estimates are available for hierarchical linear modeling parameters. The 95% confidence interval for the diastolic recovery quadratic \( b \) weight is .04–.22 (see Table 4). Although the pattern was similar for systolic blood pressure recovery, suggesting less efficient recovery with greater cumulative risk exposure, this effect was not significant for either the linear or quadratic terms.

Discussion

Childhood exposure to singular social and physical environmental risk factors is associated with adverse developmental outcomes including depression, behavioral conduct problems, poorer academic achievement, dysregulated HPA and SAM, and premature morbidity and mortality in adulthood (Evans, 2001; Gallo & Matthews, 2003; Repetti et al., 2002; Taylor et al., 1997). One explanatory construct that may explain some of the linkage between early childhood risk and adverse outcomes both in childhood and subsequently over the life course is allostatic load. Allostatic load is indicative of changes in activity levels across multiple physiological systems. Each specific physiological change, although modest when aggregated across different systems, appears to be an important precursor to long-term morbidity and possibly premature mortality (Karlamangala et al., 2002; McEwen, 1998, 2002; Seeman et al., 1997, 2001, 2004).

The first objective of this study was to replicate and extend earlier work in younger children, showing a cross-sectional relation between cumulative risk exposure and allostatic load. In previous research, allostatic load was associated with higher levels of cumulative risk exposure among 8–10-year-olds (Evans, 2003). Inspection of Figure 1 reveals that greater cumulative risk is also related to elevated allostatic load among young adolescents. Furthermore, this relation holds longitudinally, controlling for levels of allostatic load 3–4 years prior, when these adolescents were in elementary school. However, the main effect of cumulative risk on allostatic load is qualified by a significant interaction with maternal responsiveness. Cumulative risk elevates allostatic load in young adolescents only when maternal responsiveness is low.

Significant longitudinal relations between cumulative risk exposure and allostatic load may help illuminate why early childhood risk exposure is associated with increased morbidity and premature mortality later in life (Felitti et al., 1998; Power, Manor, & Fox, 1991; Power & Matthews, 1998; Repetti et al., 2002; Taylor et al., 1997). B. H. Singer and Ryff (1999) documented that similar, early childhood elevated risk profiles, as shown herein, were associated with allostatic load in older adults. Elevated allostatic load early in life could be a viable process to explain how childhood exposure to excess risk causes morbidity later in life. In the MacArthur studies of successful aging, allostatic load explained 35% of the SES-related variance in mortality (Seeman et al., 2004). Clearly, longer term data collection over the life course is required to directly test the role of elevated allostatic load in linking early

Figure 1. Regression plots showing the relationship between cumulative risk exposure, maternal responsiveness, and allostatic load (as measured by indices described in the text).
cumulative risk exposure to morbidity. Our results are assessed too early in the life course to uncover disease endpoints.

A second objective of this study was to examine the role of cumulative risk exposure in dynamic cardiovascular activity. Youth exposed to greater cumulative risk had muted cardiovascular reactivity to an acute stressor (see Figure 2). These effects match several adult studies showing that higher chronic stressor exposure leads to muted cardiovascular reactivity (Gump & Matthews, 1999). A much smaller body of work shows similar trends among children (Boyce & Chesterman, 1990; Krenichyn et al., 2001; Mengel et al., 1992; Perry & Pollard, 1998). None of these studies, however, examined reactivity to an acute stressor in relation to cumulative risk exposure. Moreover, there is only one prior study of cardiovascular recovery to an acute stressor in children (Jackson, Treiber, Turner, Davis, & Strong, 1999), and these investigators did not examine chronic stressor exposure. Adult studies (Gump & Matthews, 1999) reveal slower, less efficient recovery in conjunction with greater chronic stress. To our knowledge, this is the first study with children or youth to reveal the same pattern. As shown in Figure 3, exposure to higher levels of cumulative risk impedes cardiovascular recovery to an acute stressor in young adolescents. The nonlinearity of the data was unexpected and is difficult to interpret. One speculative explanation might be the toughening hypothesis: Some modest amount of exposure to challenges over time, somewhat analogous to physical exercise, enables the body to improve its efficiency in dealing with an acute demand (Dienstbier, 1989). What is clear is that at higher levels of cumulative risk exposure, cardiovascular recovery is inhibited.

A less efficient stress response (i.e., muted reactivity, slower recovery) to an acute stressor in relation to cumulative risk exposure is potentially important for at least two reasons. First, there is growing evidence that inefficient physiological mobilization and recovery rather than resting indices of physiological stress are particularly important precursors of morbidity (Gunnar & Vasquez, 2001; Haynes, 1991; Heim, Newport, Heit, & Graham, 2000; Linden et al., 1997; McEwen, 1998, 2002; Seeman et al., 2001). Changes in the patterns of responsiveness to demands rather than resting, basal levels may be more indicative of developing pathology. Second, the pattern of hyporeactivity and delayed recovery uncovered in relation to cumulative risk exposure is in accord with allostatic load theory. One of the costs of accumulated wear and tear on the body is loss of a fast acting, robust stress response to acute demands (McEwen, 1998, 2002; Seeman et al., 1997, 2001). One limitation of our study was the inability to also examine HPA reactivity and recovery to an acute stressor. There is very little work on this in children, and none has examined cumulative risk exposure. Research in adults (Cohen et al., 2006; Heim et al., 2000) and in children (Flinn, 1999; Gunnar, 2000; Gunnar & Donzella, 2002; Gunnar & Vasquez, 2001; Watamura, Donzella, Alwin, & Gunnar, 2003) suggests that experiences of chronic stress lead to a flattening of the daytime diurnal cortisol rhythm, particularly the absence of a sharp afternoon decline in cortisol levels. Flinn (1999) has also found evidence for muted cortisol reactivity to physical demands that typically elevate cortisol among chronically stressed children. These downward-regulated, daytime diurnal rhythms could be a byproduct of the sustained overnight activity of the HPA as indexed in the allostatic

![Figure 2](image-url) Cumulative risk exposure (as measured by the cumulative risk index) and cardiovascular reactivity level.

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Regression Results for Cardiovascular Reactivity and Cumulative Risk, Controlling for Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outcome</td>
<td>Predictor</td>
</tr>
<tr>
<td>Diastolic reactivity</td>
<td>Cumulative risk</td>
</tr>
<tr>
<td>Systolic reactivity</td>
<td>Cumulative risk</td>
</tr>
</tbody>
</table>

* $p < .05$. † $p < .01$.
load metric (e.g., higher levels of overnight cortisol; McEwen, 2000). Increasingly, evidence points toward the importance of not only examining mean levels of SAM and HPA activity and other markers of chronic stress but scrutinizing the temporal patterns of activity throughout the day.

The null results for maternal-responsiveness buffering of cardiovascular reactivity and recovery could reflect temporal aspects of allostatic load development. Changes in resting, basal levels of multiple physiological systems are theorized to reflect secondary outcomes caused by primary allostatic load mechanisms at the cellular level, including downward regulation of neurotransmitters, trafficking of immune cells, and excitatory amino acids. Reactivity and recovery as tertiary processes may take longer to occur, resulting from wear and tear on the body produced over time by elevated blood pressure levels, higher levels of circulating catecholamines, and elevated cortisol levels (McEwen, 2000; McEwen & Seeman, 1999).

The buffering of cumulative risk impacts on allostatic load by maternal responsiveness uncovered herein is in accord with a few studies that revealed protective effects of parenting practices on cumulative risk and socioemotional and cognitive development. Socioemotional difficulties associated with elevated cumulative risk exposure were attenuated by better social relationships between adolescents and other adults, including parents (Jessor, Van Den Bos, Vandersynn, Costa, & Turbin, 1995), and for adolescents whose mothers expressed less negative affect toward them (Seifer, Sameroff, Baldwin, & Baldwin, 1992). Similar protective effects were shown for parental monitoring among elementary-school-age children exposed to more cumulative risk (Klein, Forehand, & Group, 2000). The adverse consequences of cumulative risk exposure on adolescent academic achievement have also been shown to be moderated by high parental expectations for success (Reynolds, 1998), consistent discipline (Gutman, Sameroff, & Eccles, 2002), and granting of lower autonomy (Gutman et al., 2002). We show a similar buffering pattern for allostatic load, an index of chronic physiological stress. Some caution is warranted, however, in interpreting these findings solely as a reflection of parental responsiveness. It is difficult to disentangle maternal responsiveness from at least two other constructs that might also affect reactions to cumulative risk exposure: (a) Maternal responsiveness might be a reflection of a more general positive youth disposition. Happier and better adjusted youths likely see their parents as more responsive. (b) It is difficult to determine whether parental responsiveness is the key process or simply a marker for better parent–child relationships in general.

The principal limitation of the present study is its research design. Although we have longitudinal data for one of our outcome measures, allostatic load, our design is still correlational, and thus, causal conclusions are not warranted. We also lack measures of cardiovascular dynamics in Wave 1, so the reactivity and recovery data are cross-sectional. As noted above, there is emerging research suggesting that chronic stressor exposure can influence HPA patterns of response to acute stressors as well. A valuable adjunct to the present physiological protocol of basal cardiovascular and neuroendocrine assessments and cardiovascular dynamic assessment would have been the inclusion of HPA reactivity and recovery to an acute stressor. It is also possible that children experiencing more risk did not perceive or react to the task, mental arithmetic, in the same manner as those exposed to less risk. Evans (2003), for example, found that cumulative risk exposure was

![Figure 3](cumulative_risk_diastolic_blood_pressure_recovery.png)

**Figure 3.** Cumulative risk exposure (as measured by the cumulative risk index) and diastolic blood pressure recovery (in millimeters of mercury). The x-axis depicts the timing of the blood pressure measures taken every 2 min throughout the recovery period.

**Table 4**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Coefficient</th>
<th>SE</th>
<th>t Ratio</th>
<th>df</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>66.07**</td>
<td>1.03</td>
<td>64.44</td>
<td>246</td>
</tr>
<tr>
<td>Gender</td>
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<td>0.92</td>
<td>−0.26</td>
<td>212</td>
</tr>
<tr>
<td>Cumulative risk</td>
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<td>1.05</td>
<td>0.04</td>
<td>222</td>
</tr>
<tr>
<td>Cumulative risk quadratic</td>
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<td>0.23</td>
<td>−0.06</td>
<td>223</td>
</tr>
<tr>
<td>Trials</td>
<td>−0.10</td>
<td>0.19</td>
<td>−0.55</td>
<td>211</td>
</tr>
<tr>
<td>Cumulative Risk × Trials</td>
<td>−0.58**</td>
<td>0.22</td>
<td>−2.68</td>
<td>212</td>
</tr>
<tr>
<td>Cumulative Risk Quadratic × Trials</td>
<td>0.13**</td>
<td>0.05</td>
<td>2.74</td>
<td>215</td>
</tr>
</tbody>
</table>

**p < .01.**
associated with greater learned helplessness in an achievement task (solving puzzles) among 8–10-year-olds. Thus, a valuable addition to the reactivity and recovery physiological protocol would be assessments of perceived challenge, stress, engagement, and so on with the task itself.

The sample of the present study is not representative of young adolescents. We over sampled low-income children given our interest in risk and poverty, and all of the participants were from rural areas in upstate New York and predominantly Caucasian, reflecting that geographic area. At the same time, it is worth reiterating the marked paucity of research on rural poverty and low-income, high-risk White children. Given that the majority of children in America who are poor are White (U.S. Census Bureau, 2005) and that rural poverty constitutes greater and more persistent material deprivation than urban poverty (Auchincloss & Hadden, 2002; Blank, 2005; Mathematica Policy Research, 2005; Sherman, 1992), more work on populations like the present one are needed. Research by Conger and Elder (Conger & Elder, 1994; Elder & Conger, 2000) on Midwestern families struggling with the farm crisis of the 1980s and research by Brody, Flor, and Gibson (1999) on low-income, African-American families in the Southeast stand out as excellent exceptions to the dominance of research on urban high-risk populations conducted to date. Another limitation of the present study is selective attrition. Children with higher levels of cumulative risk exposure in Wave 1 were less likely to remain in the sample. The net effect of this selective attrition is probably to underestimate the impacts of cumulative risk on allostatic load and dynamic cardiovascular processes.

Early childhood risk exposure has consistently been associated with concurrent, and in a smaller number of studies, subsequent, socioemotional and cognitive difficulties (Ackerman et al., 1999; Evans, 2003; Gutman et al., 2003; Lengua, 2002; Liaw & Brooks-Gunn, 1994; Rutter, 1993; Sameroff, 1998). We know much less about cumulative risk exposure and adolescent development and almost nothing about physiological processes such as allostatic load that may be associated with early exposure to accumulated psychosocial and physical environmental risk factors. We have shown that cumulative risk exposure is positively associated with allostatic load in middle-school children, after controlling for prior levels of allostatic load in elementary school. This is especially true for those with mothers who are less responsive. Middle-school children exposed to higher levels of cumulative risk also evidence muted cardiovascular reactivity to a cognitive task and are slower to recover physiologically thereafter. These effects, however, are not moderated by maternal responsiveness. There is a growing body of evidence documenting links between early childhood risk exposure and adult morbidity and premature mortality. Most of this work has examined specific risk factors, largely ignoring the fact that many childhood risk factors tend to covary. Allostatic load may play a critical role in understanding how early childhood exposures to cumulative risk factors translate into poorer adjustment and elevated morbidity across the lifespan.

References


poor diabetic control and dysfunctional family dynamics. Family Systems Medicine, 10, 5–33.

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