

**Early Social Experiences and Living Well:  
A Longitudinal View of Adult Physical Health**

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### **Abstract**

A foundational component of a good life is the quality of one's physical health. Although a great deal is known about how current life events (e.g., stress, social support, access to resources) are associated with physical health outcomes, few prospective studies have examined whether or how early-life events—especially interpersonal ones—are related to physical health outcomes across time, extending into middle adulthood. In this chapter, we describe a series of studies based on a 40-year longitudinal project—the Minnesota Longitudinal Study of Risk and Adaptation (MLSRA). The goal of this research is to identify specific early-life events that prospectively predict better versus worse physical health outcomes in middle adulthood, controlling for current life events also known to be associated with health outcomes. In addition, we discuss how one important interpersonal experience early in life—the quality of care received from one's mother—appears to protect (buffer) individuals who experienced early-life stress from having health problems later in life. We conclude by discussing a few promising future directions for research.

## **Early Social Experiences and Living Well:**

### **A Longitudinal View of Adult Physical Health**

Many of the chapters in this volume highlight the importance of psychological well-being for living the good life. Physical well-being, however, is also important in order to enjoy and appreciate life, especially as people age. Aristotle suggested that bodily excellences were constituent parts of happiness, and research now supports this assertion. Perceptions of physical health, for example, are positively associated with reports of subjective well-being (Okun & George, 1984), especially among older individuals (Okun & Stock, 1987). In order to live the good life, therefore, individuals need to have and maintain good physical health.

The seeds of good health may be planted much earlier in life than was once presumed. Our work with the Minnesota Longitudinal Study of Risk and Adaptation (MLSRA; Sroufe, Egeland, Carlson, & Collins, 2005), a 40-year ongoing prospective study of development, is finding that certain social experiences encountered in early childhood not only have lasting effects on our minds, but also on our bodies. Building on the well-established literature showing the importance of close relationships for mental well-being across the lifespan (Reis, Collins, & Berscheid, 2000), we are examining whether and how higher quality functioning in the first close relationship—the early mother-child relationship—has lasting effects on different markers of physical health measured decades later in adulthood. Our research is grounded on the concept of biological programming, which suggests that exposure to certain kinds of events during sensitive periods early in development program the body to react more vigorously to potential health threats, resulting in more health-related problems later in life.

We begin the chapter by describing the biological programming model that has guided our research and by indicating the type of evidence that is needed to test whether certain early

life variables may have produced biological programming effects. We then discuss three physical health studies that have been conducted with the MLSRA sample. In doing so, we highlight the roles that early attachment security, the quality of early caregiving, and the amount of early life stress play in forecasting health outcomes approximately three decades later in adulthood. We also highlight the crucial role that the quality of early maternal care assumes in protecting individuals who experienced higher levels of stress earlier in life from experiencing health problems years later. We conclude by pointing out some promising avenues for future research.

### **A Biological Programming Model**

In recent years, attention has turned to the role that childhood socioeconomic disadvantage may have in setting up health problems years later in adulthood. One of the most prominent models, shown in Figure 1, is the biological programming model proposed by Miller and Chen (2013). According to this model, greater socioeconomic disadvantage in childhood generates both “programming effects” and “accentuating effects,” each of which may influence vulnerability to chronic disease later in life. Miller and Chen (2013) propose that programming effects result from exposure to certain events or agents during sensitive periods early in life, such as high levels of family instability, poor parental care, witnessing intense conflict or violence, receiving poor nutrition, or being exposed to toxins in the home, school, or neighborhood. Over time, exposure to these events/agents produces a “pro-inflammatory phenotype” whereby individuals develop hyper-aggressive responses to microbial agents (e.g., bacteria, viruses) and become resistant to signals that typically down-regulate such responses in most people. The net result is chronic inflammation, which over time causes cell damage and, ultimately, vulnerability to chronic disease.

The second pathway in the model involves “accentuating effects,” which emerge in response to the behavioral tendencies commonly associated with childhood socioeconomic

disadvantage. Children who grow up disadvantaged, for instance, often display tendencies or engage in behaviors that may further compromise their long-term health, such as being hyper-vigilant to threats, having interpersonal problems and chronically low social support, seeking immediate, health-damaging rewards (e.g., smoking, excessive alcohol use, drug use), having poor self-control, or living an unhealthy lifestyle (e.g., having a poor diet, irregular sleep patterns) (Miller, Chen, & Parker, 2011). Each of these tendencies and behaviors should also increase pro-inflammatory phenotypes, eventually resulting in greater vulnerability to chronic disease. This biological programming model also assumes that genetic liabilities and other environmental factors might exacerbate (or in some cases attenuate) these effects and outcomes.

Our research focuses on the possible programming effects of early exposure to certain social experiences, such as greater family stress, less parental nurturance, and heightened family instability/conflict. What kind of data and evidence are needed to document early programming effects? First, one needs to follow individuals prospectively across their lives, ideally starting at birth. Second, one must collect theoretically-relevant measures at multiple periods of development. Third, one should attempt to demonstrate that the programming effects of certain early life variables forecast adult health outcomes years later in adulthood while statistically controlling for variables also known to correlate with health outcomes, such as current levels of life stress and other potential confounds.

### **The Minnesota Longitudinal Study of Risk and Adaptation**

To test for possible biological programming effects, we have examined the lives of approximately 170 individuals who have been continuously studied from before birth into middle adulthood as part of the Minnesota Longitudinal Study of Risk and Adaptation (MLSRA: see Sroufe et al., 2005, for a description of the project). Between 1975 and 1977, a sample of low-

income women who were receiving free health care in Minneapolis, Minnesota were recruited into the study during their third trimester of pregnancy. Their first-born child (the current participants, all of whom have been followed longitudinally) were thus born into lower socioeconomic and potentially higher risk environments. Approximately half of the participants in the MLSRA are male and half are female. The racial composition is roughly 65% white, 11.0% African-American, 18% mixed race, and 6% undetermined (due to missing father information).

Beginning with their mothers prior to birth, the lives of participants have been assessed approximately every 3-4 years, with more frequent assessments occurring during the first six years of life. Up until the participants were 17 years old, many of the assessments were focused on the birth mothers, concentrating on variables that could affect the development of their child (e.g., SES, life stress). However, we also assessed the nature and quality of the relationship between each mother and her child by videotaping each dyad at different ages as the two interacted in well-validated structured tasks that were designed to assess the attachment pattern of each dyad and the quality of care each mother gave to her child. From age 17 onward, all of the assessments focused on the participants themselves. Some of the assessments focused on each participant's attachment representations of childhood (i.e., memories and interpretations of being raised by his/her parents). When they were 32 and 37 years old, we assessed each participant's health status by measuring his or her self-reported quality of physical health, whether she/he had experienced any diagnosed health problems in the recent past, and a set of standard biomarkers (e.g., BMI, inflammation in the blood) indexing his/her current health status.

We now overview three recent MLSRA studies that have investigated whether and how three theoretically-relevant early life events—being securely versus insecurely attached to one’s mother in infancy, receiving higher versus lower maternal care in childhood, and being exposed to more versus less life stress while growing up—longitudinally predict various health outcomes in middle adulthood (at ages 32 and 37).

### **Study 1: Child Attachment Insecurity and Adult Health**

In the first study (Puig, Englund, Simpson, & Collins, 2013), we examined whether being securely versus insecurely attached to one’s mother during the first 12 to 18 months of life uniquely predicts having more versus less health problems in middle adulthood. Several prior studies have indicated that exposure to a larger number of adverse life experiences across development is associated with poorer health outcomes during adulthood (e.g., Barker et al., 1993; Felitti et al., 1998). In addition, retrospective studies have found that people who report having had higher quality relationships earlier in life experience better health outcomes as adults than people who report having had lower quality relationships (e.g., Coan, Schaefer, & Davidson, 2006; Kiecolt-Glaser, Glaser, Cacioppo, & Malarkay 1998).

Very little longitudinal research, however, has investigated links between the quality of early relationships and adult physical health status. Nearly all of the existing research on relationship functioning and adult health has focused on concurrent measures taken at a single time-point. Furthermore, the small number of longitudinal studies addressing this topic (e.g., Christakis & Fowler, 2007; House, Landis, & Umberson, 1988) have examined time-periods only in adulthood. Incorporating prospective assessments of relationship functioning from very early in development may provide both a less biased and more enriched understanding of how the quality of relationships in the opening years of life impacts adult health.

Bowlby (1969) and Boyce (1985) were among the first to hypothesize that the nature of the attachment relationship between primary caregivers and their children should play a pivotal role in shaping health outcomes, not only during childhood, but across the life-course as well. When early parent-child relationships are secure, parents serve as a safe haven within which the child feels protected and learns to regulate his/her emotions constructively when they are distressed and as a secure base from which the child can confidently explore the surrounding world. Whether early attachment relationships are secure or insecure depends in part on the quality of care that infants receive from their caregivers (Ainsworth, Blehar, Waters, & Wall, 1978). More supportive and responsive care usually results in secure parent-child attachments, whereas inconsistent or rejecting care typically culminates in insecure attachments (van IJzendoorn, 1995).

Across development, the nature and quality of care that a child receives is internalized into secure or insecure working models (i.e., schemas), which then guide his/her interpersonal functioning during adolescence and adulthood (Bowlby, 1973; Simpson & Rholes, 2012). Based on their positive interpersonal experiences, securely attached children become adept at giving and receiving high quality care and support from their significant others (Waters, Merrick, Treboux, Crowell, & Albersheim, 2000), which helps them regulate negative emotions more effectively. Insecurely attached children, on the other hand, do not give and receive care and support well, which impedes their emotion regulation abilities in later relationships (e.g., Simpson, Collins, Tran, & Haydon, 2007). Although secure and insecure attachment patterns can and sometimes do change as individuals encounter new events and new attachment figures later in life (Fraley & Brumbaugh, 2004), early attachment security versus insecurity is believed to lay the groundwork for the functioning of later relationships (Bowlby, 1973).

Some research suggests that infant attachment relationships are associated with certain aspects of health during childhood, which in turn could be associated with health later in life (e.g., Anderson & Whitaker, 2011). However, prospective studies testing the actual association between the security of early attachment relationships and adult physical health have not been conducted. To fill this gap, Puig and her colleagues (2013) prospectively examined the link between the security of mother-child attachment relationships during the first two years of life (assessed in the Strange Situation) and the health problems of participants in adulthood from the MLSRA sample. The principle hypothesis was that individuals who were rated as securely attached in the Strange Situation during infancy would be less likely to report physical health problems in middle adulthood than those rated as insecurely attached in infancy.

**Measures and Methods.** Early attachment status was assessed by the Strange Situation Procedure (SSP) when each MLSRA participant was 12 and 18 months old. The SSP is a 20-minute videotaped laboratory procedure in which young children are exposed to a series of stressful separations from, followed by reunions with, their primary caregiver (Ainsworth et al., 1978). Raters classified each participant's attachment relationship with his or her primary caregiver (always the mother in the MLSRA) at 12 and 18 months. Classifications were made according to how each participant responded to the separations and reunions. At both 12 and 18 months, approximately 60% of the participants were secure and 40% were insecure. To create a more reliable attachment measure, we summed the number of times each participant was rated as secure in the 12 and 18-month SSP assessments. Children with a score of 2 were rated as being secure at both time-points (41% of the sample), children with a score of 1 were secure at one time-point (33%), and those with a score of 0 were insecure at both time-points (26%).

Thirty years later, when participants were 32 years old, they completed the Adult Health Survey (Blum, Resnick, & Bergeisen, 1989). They were asked to indicate whether they had experienced any of several chronic diagnosed physical illnesses within the preceding year. Because 60% of the sample reported *no* chronic physical illness within the past year, we created a binary variable on which participants who reported having one or more illnesses were coded 1, and those who reported no illnesses were coded 0.

To rule out the possibility that the hypothesized early attachment security → better adult health link was confounded with participants' current life circumstances, we also statistically controlled for their gender, current socioeconomic status, body mass index (BMI), current observer-rated life stress, current self-reported neuroticism, and current perceived support that was available, all of which were assessed at age 32 with well-validated measures. Each of these variables tend to correlate with adult health outcomes (Puig et al., 2013).

**Findings.** As hypothesized, the security of early mother-child attachment relationships significantly predicted the likelihood that individuals reported having a chronic physical illness within the past year, even controlling for the possible confounds listed above. Specifically, individuals who had been securely attached to their mothers early in life (at 12 and 18 months) were significantly *less* likely to report having a chronic illness compared to those who were insecurely attached during infancy. The effect sizes were small-to-moderate in magnitude.

These findings are among the first to document the important role that early attachment status may play in setting the stage for physical health outcomes in adulthood. In related research, Miller and colleagues (2011) have found that retrospective reports of the quality of maternal nurturance early in life mediate the relation between lower SES and more health problems in middle adulthood. Early attachment security could affect adult health through any of

several possible pathways, such as by facilitating higher quality relationships with romantic partners in adulthood (Simpson et al., 2007), increasing medical treatment adherence (Ceichanowski, Walker, Katon, & Russo, 2002), or maintaining better health-promoting behaviors (Scharfe & Eldredge, 2001).

### **Study 2: Early Maternal Care, Adult Attachment Insecurity, and Adult Health**

Research indicates that a primary precursor of attachment security is the quality of care that children receive from their primary caregivers in the home (Ainsworth et al., 1978; van IJzendoorn, 1995). Realizing this fact, we next turned our attention to how the quality of early maternal care forecasts health outcomes in middle adulthood.

Growing evidence suggests that “risky” family environments can impact both biological functioning and health quality in adulthood (e.g., Repetti, Taylor, & Seeman, 2002). Risky families tend to have greater conflict and lower quality caregiving, which ought to disrupt psychosocial and biological functioning, leading to elevated risk for early onset of diseases (Miller & Chen, 2013). Consistent with this view, children who receive less warmth, support, and responsiveness from their parents tend to have higher inflammation, higher blood pressure, and greater overall allostatic load than children who receive more warmth, support, and responsiveness. Allostatic load reflects the general amount of wear and tear on the body in response to encountering repeated or chronic stressors. More specifically, it represents the physiological consequences of chronic exposure to high or fluctuating neural or neuroendocrine responses owing to repeated or chronic stress. Such health outcomes are precursors to cardiovascular problems (Bell & Belsky, 2008; Carroll et al., 2013; Lehman et al., 2009; Tobin et al., 2015). Brody and colleagues (2014) have recently shown that these effects extend beyond

childhood, with harsher parenting in childhood predicting higher levels of inflammation in adolescence.

At present, we do not know whether these effects extend beyond adolescence into adulthood. However, we do know that adults who report having received lower quality parenting during childhood tend to have higher allostatic load and more health problems as adults (e.g., Russek & Schwartz, 1997; Slopen et al., 2015). Relying on retrospective reports of parenting is problematic, as individuals may not accurately remember their childhood experiences (Rubin, Rahhal, & Poon, 1998), especially those early in life when maternal care might be more impactful (cf. Meaney & Szyf, 2005). Moreover, confounding third variables may lead people to exaggerate either their early life experiences or the severity of their current health problems, artificially inflating associations between the two.

Only one study to our knowledge has investigated the link between early parenting quality and adult health outcomes prospectively. Using the data from the Dunedin Multidisciplinary Health and Development Study, Danese and colleagues (2007) found that childhood maltreatment (indexed by maternal rejection, harsh discipline, changes in primary caregivers, and physical/sexual abuse before age 10) predicts higher incidence of age-related disease risk in adulthood, including elevated inflammation and other biological health-risk markers. The Dunedin study, however, did not begin assessing participants until they were 3 years old, and their maltreatment composite contains a mix of coder ratings and retrospective self-reports.

If poorer parenting early in life is systematically tied to more health problems in adulthood, we need to understand how these early experiences “get under the skin” and are carried forward to affect health decades later. Chen and her colleagues (2011) have proposed that

more nurturant caregiving communicates to children that the world is a relatively safe place and that people can typically be trusted and counted upon. This realization may allow children—even those from disadvantaged socioeconomic backgrounds—to perceive less threat in their daily lives, which ought to reduce the wear-and-tear that chronic vigilance can have on endocrine and other health-relevant systems. Nurturant care might also help children—even disadvantaged ones—learn better coping skills and more effective emotion-regulation strategies, which, over time, should reduce stress and aggressive biological responses to it. This emphasis on instilling feelings of safety and trust, reducing threat, and developing more constructive emotion-regulation strategies insinuates that the attachment system should be a key psychosocial mediator of the purported link between the quality of early parenting and adult health outcomes.

There is, in fact, some preliminary evidence supporting this possibility. As we have seen, higher quality parenting early in life is associated with attachment security in childhood and beyond (e.g., van IJzendoorn, 1995; Zayas et al., 2011). Moreover, some of the cardinal features of attachment insecurity—over-perception of stressors, poorer self-regulation, less effective support-seeking, and poorer relationship functioning—typically produce over-activation of the biological stress system and, therefore, elevated health risks due to repeated exposure to stress hormones (Farrell & Simpson, 2017; Pietromonaco, Uchino, & Dunkel-Schetter, 2013). Indeed, across different types of relationships, insecure attachment is associated with greater biological stress reactivity (Fagundes et al., 2011), more inflammation (Gouin et al., 2008; Kidd et al., 2014), and higher incidence of cardiovascular disease (McWilliams & Bailey, 2010).

Given this backdrop, Farrell and her colleagues (2017) decided to examine links between early parenting quality, adult attachment security, and a measure of cardiometabolic risk during adulthood within the MLSRA. They hypothesized that higher quality early parenting would

longitudinally forecast lower cardiometabolic risk in middle adulthood, and that adult attachment security (i.e., memories and interpretations of how one was treated by parents during childhood) would mediate this link.

**Measures and Methods.** To assess the quality of early care, mother–child interactions were videotaped during semi-structured tasks and then coded for maternal sensitivity when MLSRA participants were 3, 6, 24, and 42 months old. At three months, each mother and child were observed in the home during a routine feeding situation. When infants were six months old, two feeding situations and one play interaction were observed in the home on two different days. For these early assessments, maternal sensitivity was operationalized using Ainsworth's sensitivity scale (Ainsworth et al., 1978), which assesses each mother's ability to perceive and accurately interpret her infant's signals and respond appropriately and promptly. When participants were 24 and 42 months old, they and their mothers were observed in a laboratory setting while attempting to solve several problem-solving and teaching tasks. At each age, the tasks gradually increased in complexity, eventually becoming too difficult for the child to complete on his/her own. Mothers were instructed to initially allow their child to try to solve each task independently, and then to give their child any help they thought was needed. Maternal sensitivity at 24 and 42 months was evaluated with a rating of each mother's supportive presence, which assessed the extent to which each mother provided a secure base for her child (i.e., helped the child feel comfortable with the task) as well as each mother's positive involvement during the interaction. A single composite measure of early maternal sensitivity (i.e., quality of maternal caregiving) was then created by standardizing and averaging all four maternal sensitivity ratings.

Adult attachment security was assessed by secure base script knowledge coded from Adult Attachment Interviews (AAIs), which were conducted twice when participants were 19

and 26 years old. The AAI is a semi-structured audiotaped interview that assesses adults' state of mind with respect to their attachment relationships with their primary caregivers (Main, Goldwyn, & Hesse, 2003). It contains questions that elicit recollections of experiences with caregivers early in life, typically between ages 5 to 12. Each participant's secure base script knowledge was rated from his/her interview on a scale that assessed the extent to which each narrative followed or implied knowledge of the secure base script (Waters, 2016; Waters et al., 2013). Raters focused on: (a) explicit or implied expectations consistent with the secure base script (e.g., caregivers were available, responsive, and/or provided comfort effectively), and (b) recall of specific autobiographical memories that follow the secure base script. Narratives receiving high scores contained several events that followed the secure base script structure. Those receiving a low score had several events that directly violated secure base script structure (e.g., the caregiver was rejecting or did not offer help when the participant reported being hurt, ill, or afraid in childhood) or reflected other relationship expectations (e.g., recurring abuse).

At age 37, four biomarkers of cardiometabolic risk were assessed. During a laboratory assessment, participants' blood pressure was measured at the start and end of the assessment. Participants' Body Mass Index (BMI) was also calculated from their height and weight measurements. Their Waist-to-Hip ratio (WHR) was assessed by dividing the measurement of each participant's waist at the narrowest point from the measurement of his/her hips at the widest point. Their level of C-reactive protein (CRP), a marker of inflammation in the blood, was assayed from blood samples. A composite measure of cardiometabolic risk was then created by standardizing and averaging these four biomarker measures.

We also measured variables that could be confounds. They included participants' gender, race, and the life stress experienced by the mother during the early life of her child.

**Findings.** In line with our expectations, individuals who received higher quality care early in life from their mothers (rated by observers) had greater attachment security in early adulthood (based on secure base script knowledge coded from the AAI) and lower levels of cardiometabolic risk at age 37. Moreover, being securely attached in adulthood proved to be a significant mediator of this connection (see Figure 2). These effects also held when the potential confounds of sex, race, and mother's stress were controlled. These mediation findings are noteworthy because they identify one of the psychological variables that may carry the impact of early parenting forward to eventually impact adult health. However, attachment did not fully mediate the link between maternal sensitivity and adult cardiometabolic risk (i.e., a significant direct path between maternal sensitivity and cardiometabolic risk remained, despite the significant mediational path). This suggests that other psychological or biological variables may also serve as important mechanisms that need to be identified.

### **Study 3: Life Stress, Early Caregiving, and Adult Health**

According to Miller and Chen (2013), being socially or economically disadvantaged early in life should render people more susceptible to early onset health problems later in life. A great deal of cross-sectional research has focused on one major consequence of being disadvantaged—experiencing high levels of stress (e.g., Cohen et al., 2007). As we discuss below, some more recent evidence suggests that *when* stress is encountered during life might also uniquely impact later health outcomes (e.g., Lupien et al., 2009; Miller, Chen, & Parker, 2011). Moreover, there are compelling reasons to believe that certain experiences, such as receiving better maternal care early in childhood, may protect (buffer) individuals from the adverse effects of life stress. Little longitudinal research, however, has examined the impact of stress at different life stages, and very few studies have explored protective factors.

Being aware of these gaps in our knowledge, Farrell, Simpson, Englund, Carlson, and Sung (2017) sought answers to two questions: (1) When during life does stress more strongly predict later adult health problems? and (2) Does receiving better maternal care early in life reduce the negative effects that stress typically has on adult health outcomes? Some prior research indicates that there could be sensitive periods during development when stress has a somewhat stronger impact on later adult health (e.g., Fagundes & Way, 2014; Miller, Chen, & Parker, 2011). Miller and colleagues (2011), for example, propose that early life stress affects adult health by programming the immune system to be hyperactive to potential threats, which produces chronic inflammation and associated health problems later in life (e.g., high blood pressure, poor immune functioning). Consistent with this idea, exposure to higher levels of stress early in life prospectively forecasts a myriad of deleterious health outcomes, including coronary heart disease (Dong et al., 2004) and premature death (Galobardes et al., 2006).

Life stages other than early childhood, however, might also impact long-term health. Experiencing greater life stress during adolescence, for example, is associated with higher levels of inflammation (Ehrlich et al., 2016) and more chronic health problems (Gustafsson et al., 2012). In addition, experiencing greater current life stress is also associated with poorer health outcomes (DeLongis et al., 1988; Uchino, Cacioppo, Malarkey, & Glaser, 1995). Complementing these ideas, animal studies have shown that early life and adolescence are two critical periods for the development of chronic health issues (e.g., Meaney & Szyf, 2005; McCormick, Matthews, Thomas, & Waters, 2010). The long-term effects of stress beyond early childhood on health in humans, however, remain largely unknown.

To our knowledge, two prospective studies have investigated the impact of early life stress on later physical health. Essex and colleagues (2011) followed children from infancy to

adolescence and found that greater stress in infancy and preschool (operationalized as having depressed parents and being exposed to anger in the family) resulted in atypical diurnal cortisol patterns in middle childhood and adolescence, which usually are associated with health problems. Raposa and colleagues (2014) investigated how young adult health outcomes were affected by early adversity (measured by mother's psychopathy, parental discord, harsh discipline, family income, and parental criminal behavior during the first five years of life). Children who experienced greater adversity in early in life had higher levels of inflammation, BMI, and smoking at age 20. These two studies, however, examined the effects of stress only from early childhood.

Although the stress-to-health link is robust, one protective factor may be the quality of early maternal. It is well-established that social support promotes better health, partly because it buffers the deleterious effects of stress (Cohen & Smye, 1985; Uchino, Cacioppo, & Kiecolt-Glaser., 1996). In a similar manner, higher quality parenting serves a protective factor for many non-health-relevant outcomes linked with child adversity because it reduces children's stress responses. Thus, higher quality parenting ought to have a buffering effect on the relation between early stress and adult health outcomes (Cicchetti & Blender, 2006; Gunnar & Quevedo, 2007). Consistent with this view, greater self-reported maternal warmth and nurturance early in life buffers the impact of low SES on inflammation and metabolic syndrome in adulthood (Chen et al., 2011; Miller, Chen, & Parker, 2011). Moreover, parenting interventions tend to be effective in improving child health (Miller, Brody, Yu, & Chen, 2014).

Similar to research on stress, however, virtually all prior research on parenting as a protective factor has relied on retrospective accounts, typically by asking adults to report on their mother's warmth and nurturance when they were young (e.g., Chen et al., 2011; Miller,

Lachman, et al., 2011). As with retrospective reports of stress, there may be disparities between what actually happened during childhood and adult perceptions of those experiences (Roisman et al., 2002). To complicate matters, past studies have conflated parenting quality and life stress by including parenting or parenting-relevant issues (e.g., maternal depression; Essex et al., 2011) in life stress measures.

Working with the MLSRA, Farrell and her colleagues (2017) hypothesized that exposure to greater stress at three life stages—early in life, during adolescence, and currently—should have somewhat stronger prospective effects on adult health. They also explored whether experiencing greater stress at two or more of these life stages revealed a “dual-risk” pattern, with higher stress at two or more stages resulting in worse health outcomes than the main effects of each stage alone. Most importantly, they also anticipated that higher quality maternal care would have a buffering effect on adult health, with higher quality parenting reducing or perhaps eliminating the negative effects of earlier life stress on adult health.

**Measures and Methods.** Three health measures were collected on MLSRA participants when they were 32 years old: (1) a self-rating of their overall physical health (on a scale from *Excellent* to *Poor*); (2) their body mass index (BMI), calculated from their height and weight; and (3) self-reports as to whether they had any one of several chronic illnesses within the past year, assessed by the Adult Health Survey (coded 1 if they reported one or more illnesses, and 0 if they had none).

Life stress was assessed at 16 time-points when participants were between 12 months and 32 years old. At each time-point, life stress was measured by an interview (the Life Events Schedule [LES]; Egeland, Breitenbucher, & Rosenberg, 1980), which assessed whether and the extent to which several potentially stressful life events had occurred during the prior year (e.g.,

financial issues, conflict with romantic partners, being a victim of crime, losing a job). The audio-recorded answers were then rated by coders for how disruptive each potential stressor was on a scale ranging from 0 (*no disruption*; e.g., minor changes in job responsibilities) to 3 (*extreme disruption*; e.g., having a parent commit suicide). Each mother completed the LES until her child (the participant) was 17.5 years old, after which each participant completed the LES. Stress ratings for each potential stressor were summed within each assessment period to create a total stress score for each participant. Assessments were then grouped into four life stages: early childhood (ages 0-5), middle childhood (ages 6-12), adolescence (ages 13-19), and young adulthood (ages 21-31). Total life stress scores were standardized at each assessment and were then averaged within each life stage to create a total stress composite for that stage. Life stress at age 32 was also standardized to measure current life stress.

As described earlier, ratings of the quality of maternal caregiving earlier in life were based on several observational assessments of mother-child interactions that were conducted in the home and in the lab.

The control measures included the sex and race/ethnicity of each participant (white = 1; non-white = 0) and his or her self-reported level of neuroticism, which was assessed at age 32 by the Berkeley Personality Profile (John & Srivastava, 1999). Neuroticism was controlled because it correlates with self-reported health problems (Watson & Pennebaker, 1989).

**Findings.** When the three health outcomes at age 32 were examined, nearly all of the significant stress→adult health correlations were found for the three hypothesized life stages: early childhood (ages 0-5), adolescence (ages 13-19), and currently (age 32). Further analyses revealed that experiencing higher levels of stress during certain stages yielded interaction effects. For example, early childhood and adolescent stress significantly interacted to predict

participants' BMI (see Figure 3, panel B), such that individuals who encountered greater stress at both of these life stages had the highest BMI at age 32. A marginally significant interaction was also found involving early childhood and adolescent stress predicting overall self-rated physical health (see Figure 3, panel A). Specifically, individuals who experienced higher stress at both life stages rated their physical health as somewhat worse than those who experienced higher stress at only one or neither life stage.

Tests of the buffering effects of maternal sensitivity on life stress-adult health connections revealed a significant three-way interaction predicting BMI (see Figure 4, panels B<sub>1</sub> and B<sub>2</sub>) and a marginal three-way interaction predicting chronic illness counts (see Figure 4, panels A<sub>1</sub> and A<sub>2</sub>). In both cases, experiencing higher stress at both life stages as well as lower maternal sensitivity predicted poorer adult health outcomes, whereas experiencing lower life stress at both life stages along with higher maternal sensitivity predicted lower BMI and fewer illnesses. As hypothesized, however, individuals who experienced higher life stress during both early childhood and adolescence but also experienced higher maternal sensitivity had *equally good* health outcomes as those with no risk factors (i.e., lower life stress at both life stages and higher maternal sensitivity). The majority of these effects held when we controlled for participants' sex, ethnicity, and self-reported neuroticism (see Farrell et al., 2017).

### **Conclusions and Future Directions**

Viewed together, the current findings provide some of the most direct evidence to date that certain early life experiences leave long-term, enduring effects on physical health. Consistent with biological programming ideas (e.g., Boyce, 1985; Miller & Chen, 2013), our studies using the MLSRA document that being securely attached to one's mother very early in life, receiving higher quality maternal care during childhood, and encountering less life stress all

predict better health outcomes in middle adulthood. Moreover, the association between receiving better maternal care during childhood and having better health in middle adulthood is mediated (i.e., carried forward) by having more secure attachment representations of childhood in early adulthood. Corroborating animal models and research (e.g., Meaney & Szyf, 2005), we have also found that stress encountered at certain life stages—especially early in life (ages 0-5), during adolescence (ages 13-19), and concurrently (at the time of the health assessment in adulthood)—has interactive effects on adult health outcomes. Importantly, however, receiving higher quality maternal care appears to protect (buffer) individuals who experienced higher levels of stress early in life from experiencing health problems in middle adulthood.

Future research should head in several different directions. Returning to Miller and Chen's (2013) biological programming model (see Figure 1), we need to learn more about how accentuating effects (e.g., threat vigilance, social difficulties, poor self-regulation) affect adult health and whether certain accentuating effects statistically interact with certain programming effects (e.g., family instability, low nurturance, exposure to conflict) to impact health outcomes years later. If, for example, individuals grow up in low nurturance homes and become highly vigilant to threats later in life, does the combination of low nurturance and threat vigilance result in especially poor adult health outcomes? Alternatively, instead of operating in parallel, the programming effects of social experiences stemming from early adversity may be the cause of accentuating behavioral proclivities, making the path to a pro-inflammatory phenotype more linear. Testing causal mechanisms between each construct in this model can clarify the pathways linking early adversity to adult health problems.

We also need to learn more about whether and how genetic liabilities in combination with other harmful (or protective) environmental factors influence adult health outcomes via specific

programming or accentuating effects. If, for instance, children are born with genetically compromised immune systems and are raised in environments containing more pathogens, how does this interface with their nutritional status or health-related lifestyle to affect their health status in adulthood? Finally, we need to better understand how different early life variables “get under the skin” to affect or perhaps reprogram biological systems known to regulate health outcomes (see, for example, Farrell & Simpson, 2017). Our recent findings from the MLSRA open the door to addressing each of these important future issues in order to help people live healthier and more fulfilling lives.

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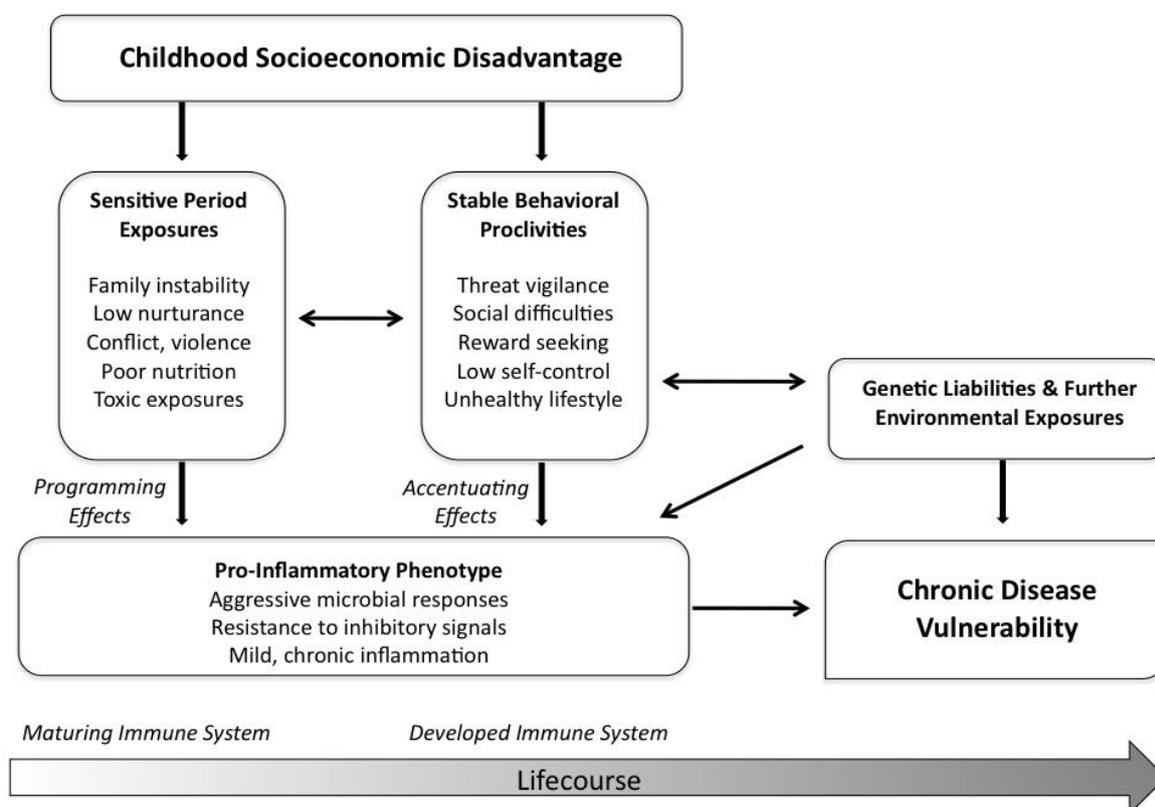
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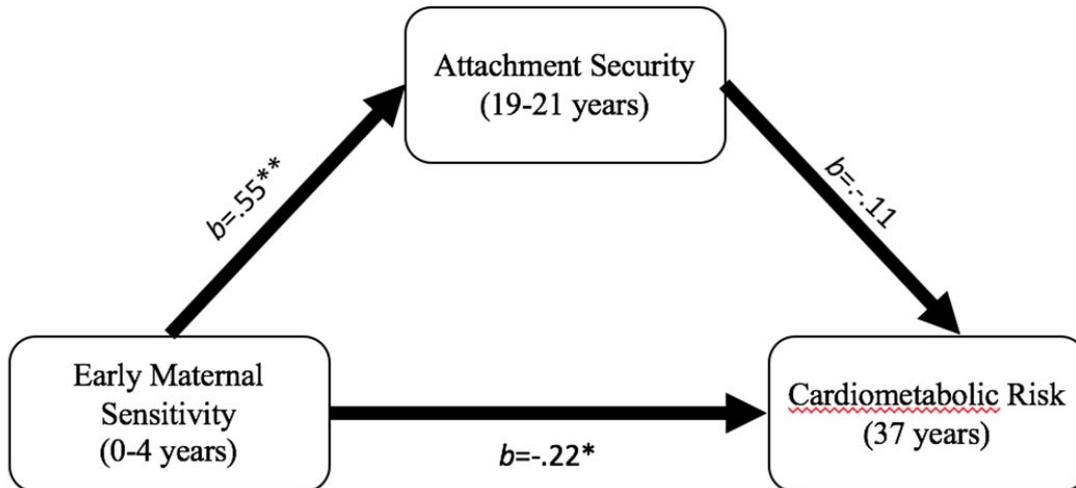
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**Figure 1. Miller and Chen's (2013) Biological Programming Model.**

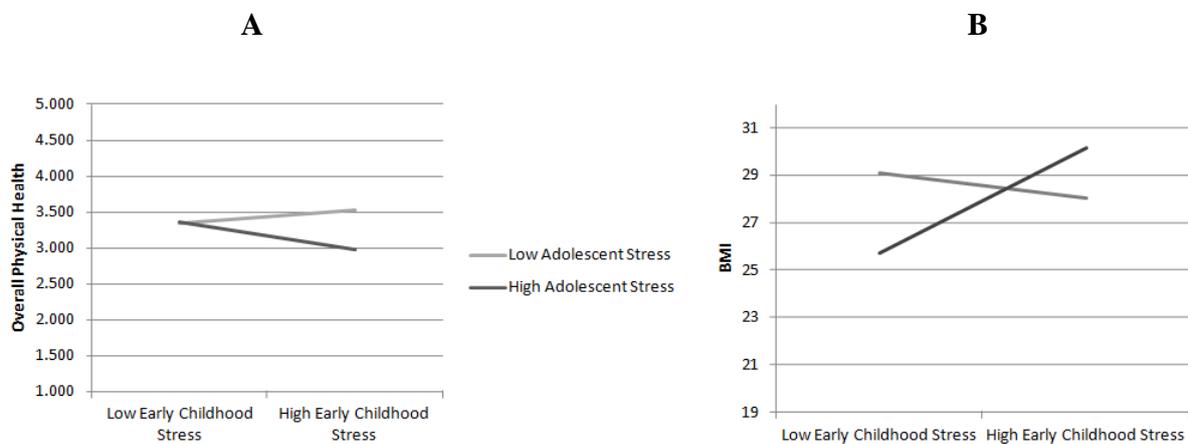


**Figure 2.** A mediation model showing that the effect of higher quality maternal care early in life on health outcomes in middle adulthood is mediated by secure base representations of childhood in early adulthood.



Indirect path:  $b=-.06$ , Bootstrapped CI:  $-.01$  to  $-.15$   
\* $p<.05$ , \*\* $p<.01$

**Figure 3. Interaction effects of early childhood and adolescent life stress on subjective ratings of overall physical health (A) and BMI (B) (from Farrell et al., 2017).**



**Figure 4. Buffering effects of maternal supportive care on life stress on illness/symptom counts (A) and BMI (B) (from Farrell et al., 2017).**

