The Impact of Early Adversity on Health

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Abstract

A harsh early environment has adverse affects on health not only in childhood but across the

lifespan. Chief among the toxic aspects of early environment that have been related to poor

health are low childhood socioeconomic status and a harsh family environment, including

physical and sexual abuse as well as more modest family dysfunction, such as family conflict,

neglect, and a cold, non-nurturant environment. These characteristics of the early environment

interact with genetic predispositions to affect socioemotional resources and coping skills; these

include such resources as optimism, a sense of mastery or control, self-esteem, and social

support. On the negative side, a harsh early environment affects a propensity for chronic

negative affect, including depressive symptomatology, anxiety, and hostility. Health habits, such

as substance use, diet, and exercise, are also affected by the early environment. Jointly, these

factors predict alterations in biological stress regulatory systems. Together in interaction with

genetic factors, these pathways help to account for the lifelong affects of early adversity on

health.

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Good health begins early in life. Unfortunately, so does poor health. Low socioeconomic status (SES) in childhood and a harsh early family environment can adversely influence trajectories of health outcomes long into adulthood. It may come as no surprise that childhood physical and sexual abuse have negative mental and physical health consequences, both immediately and over the lifespan. These effects are well documented (e.g., Springer et al., 2003). What may be more surprising is that relatively modest family dysfunction that numerous people routinely experience can lead to adverse outcomes as well.

Both animal and human research conclusively documents that warm, nurturant contact early in life exerts permanent beneficial effects on the functioning of biological stress regulatory systems and on socioemotional skills that affect responses to stress across the lifespan (Francis et al., 1999; Liu et al., 1997; Repetti et al., 2002; Repetti et al., 2007). When this contact is lacking, both biological stress regulatory systems and behavioral skills for managing stress are compromised. Adverse downstream consequences include problems in emotion regulation, social skills deficits, poor health habits, and exacerbation of biological stress responses prognostic for poor mental and physical health.

1.   Early family environment

This review will be guided by the model pictured in Figure 1. The model maintains that a stressful or harsh early environment, in conjunction with genetic or acquired risks, is linked to

adverse health outcomes in adulthood via its impact on the (in)ability to develop effective socioemotional skills; through a propensity to experience chronic negative affect; by affecting health habits adversely; and by influencing the neural pathways that ultimately regulate neuroendocrine stress responses. Drawing on the concept of allostatic load (McEwen, 1998), we suggest that these pathways contribute to the experience of chronic or recurring stress which, in interaction with genetic predispositions and acquired risks such as poor health habits, lead to the accumulating damage to biological systems that ultimately results in risks for mental and physical health disorders.

2.   Childhood socioeconomic status

The point of departure for the model is low childhood SES. Low childhood SES has been related to exposure to a broad array of early stressful events. These include neighborhood conflict, violence exposure, noise, poor housing, exposure to pathogens, and other chronic stressors (Adler et al., 2000). Substantial research also links economic adversity (low SES) early in life to mental and physical health disorders (Adler et al., 2000).

Research has consistently tied low childhood SES to all of the downstream variables in the model (Figure 1). It is associated with poor or deteriorating quality of parenting, including higher levels of family conflict, a harsh, restrictive parenting style, and chaotic or neglectful parenting (McLoyd, 1998). Low childhood SES has also been related to chronic negative affective states (Gallo and Matthews, 2003) and to problems in the development or use of socioemotional resources (Adler et al., 2000; Taylor and Seeman, 2000; Repetti et al., 2002). Low SES has been tied to unhealthful habits, including smoking (Winkleby et al., 1999), obesity (e.g., Wardle et al., 2002), poor sleep (Van Cauter and Spiegel, 2000), and drug abuse (Spooner,

1999). Low socioeconomic status has been tied to a broad array of diseases and all-cause

mortality as well (e.g., Adler et al., 2000; Hemingway et al., 2003; Kivimäki et al., 2004; Lawlor,

and Smith, 2005; Owen et al., 2003). In summary, there is substantial evidence that adverse

health outcomes in adulthood have origins in SES, including SES in childhood, via pathways

implicating socioemotional resources, chronic negative affect, and health behaviors.

3.   Early family environment

Similar patterns are found when an early family environment is assessed directly. A

harsh family upbringing has been related to poor health behaviors (Repetti et al., 2002), to high

levels of depression, hostility, and anxiety (Repetti et al., 2002); to preclinical risk factors for

physical health disorders, including elevated autonomic and cortisol responses to threatening

circumstances (e.g., Roisman et al., 2009; Taylor et al., 2004); to risk factors for disease,

including compromised metabolic functioning (Lehman, Taylor, Kiefe, & Seeman, 2005) and C-

reactive protein (Taylor et al., 2006); and to diagnosed health disorders, including ischemic heart

disease, some cancers, and depression, among many others (Felitti et al., 1998). Socioemotional

resources that affect the ability to regulate emotional states effectively and to develop social

competencies are implicated in these pathways. For example, offspring from harsh early

environments experience difficulty in managing emotions in challenging circumstances (Repetti

et al., 2002; Repetti et al., 2007).

4.   Genes and gene-environment interactions

As the model indicates and as the theory of allostatic load maintains, a harsh early

environment contributes to lifespan risk for health disorders, not only directly, but also via gene-

environment interactions (Repetti et al., 2002). Recently, what some of those genes may be has

come to light.

One such gene is the serotonin transporter gene, certain alleles of which may predispose

to anxiety and/or depression. People with two copies of the 5-HTTLPR short allele (short/short)

who have experienced childhood maltreatment are more likely to be diagnosed with major

depressive disorders than individuals with one or two copies of the long allele who have

experienced similarly harsh environments (Caspi et al., 2003; Kaufmann et al., 2004). Taylor

and colleagues (Taylor et al., 2006) found that the short allele may not only function as a risk

allele for depression in the face of an adverse early environment but as a general sensitivity

allele, providing protection from symptoms of depression if the early environment is nurturant.

Using a non-clinical sample of 118 adults, we found that people with two copies of the short

allele had greater depressive symptomatology if they had experienced early familial adversity

compared to people with the short/long or long/long genotypes, but significantly less depressive

symptomatology if they reported a supportive early environment. Another gene whose

functioning is affected by early environment is the gene that regulates monoamine oxidase

(MAO-A). Men with the low-expressing alleles of the MAOA-uVNTR are more likely to

engage in aggressive and antisocial behavior than men with high expressing alleles; these

outcomes appear to be especially likely if the men have also been exposed to maltreatment as

children (Caspi et al., 2002; Kim-Cohen, 2006). Recent evidence suggests that the long allele of

the DRD4 receptor gene, involved in the regulation of dopamine, may similarly interact with

family environment; that is, the long allele may increase sensitivity to both negative and positive

parental influences (e.g., Bakermans-Kranenburg and van IJzendoorn, 2007; Bakermans-

Kranenburg et al., 2008).

Expression of the glucocorticoid receptor gene is also affected by the early environment. In animal studies, Meaney and colleagues have shown that rat pups exposed to highly nurturant mothering exhibit less emotionality in novel circumstances and more normative social behavior, including mothering in adulthood, compared to recipients of normal mothering (Francis et al., 1999; Weaver et al., 2004). These long-term effects of maternal care appear to be the result of epigenetic structural alteration (methylation) to the glucocorticoid receptor gene that affect its expression throughout the lifespan (Meaney and Szyf, 2005). This mechanism may help to explain why early family environment has such enduring effects on biological responses to stress and ultimately on long-term health outcomes, via altered glucocorticoid receptor expression that affects adult reactivity to stress.

Since the study of gene-by-environment interactions is in its early stages, especially those involving the early environment, these findings are more tantalizing than they are definitive. The coming decades will no doubt identify other genes and other ways in which early environment may interact to affect propensities for risk for illness.

5.   Emotion regulation

Problems in the regulation of emotional states are implicated in the pathways linking early adversity to adverse health outcomes. Emotion regulation is a broad term that includes skills for recognizing one's own and others' emotions, controlling one's emotional reactions to potentially stressful or challenging situations, and expressing one's emotions in socially appropriate ways (Eisenberg and Spinrad, 2004).

A variety of investigations have tied a harsh family environment to children's reactions to emotionally charged circumstances, understanding of emotions, and abilities to regulate their

emotions (Repetti et al., 2002). Offspring from harsh family environments may overreact to threatening circumstances, responding aggressively to situations that are only modestly stressful (Reid and Crusafulli, 1990), but may also tune out or avoid stressful circumstances, as through behavioral escape/avoidance or substance abuse (O'Brien et al., 1991; Valentiner et al., 1994; Johnson and Padina, 1991). Deficits in emotion regulation skills related to early family environment may appear in early childhood and compromise the development and use of socioemotional skills in adulthood (Repetti et al., 2002).

A harsh family environment also predicts an incomplete understanding of emotional experience in others. Investigations with young children have found those who were maltreated or whose homes were marked by high levels of anger and distress are less accurate in their understanding of emotions compared to their peers (Camras et al., 1998; Dunn and Brown, 1994). Relatedly, Laible and Thompson (1998) found that children with insecure attachments showed less emotion understanding and less accurate appraisals of emotion in others.

In short, growing up in a risky family environment appears to interfere with the development of skills for processing emotional information in self and others.

6.   Social skills

A harsh family environment has been tied to fewer social skills for facilitating successful interactions with peers (Crockenberg and Lourie, 1996; Pettit et al., 1988). Children from risky families who evidence emotion regulation difficulties are more likely to behave in an aggressive or antisocial manner with their peers (Repetti et al., 2002), undermining their ability to develop friendships. Several studies attest to the peer rejection and even victimization experienced by children from harsh families (Dishion, 1990; Schwartz et al., 1997). Similarly, children whose

parents are unresponsive, cold, and insensitive are less likely to initiate social interactions, and they demonstrate more aggression and criticism in social relationships (see Repetti et al., 2007). Children of parents who are cold, unsupportive, or neglectful show deficits in social relationships throughout their lives, with more problematic and less supportive social networks (Repetti et al., 2007).

Inadequate social support networks may translate into some of the adverse health effects of a harsh early environment. More than 100 investigations have shown that social support reduces health risks of all kinds, affects the initial likelihood of illness, influences the course of recovery among people who are already ill, and affects mortality risk more generally (House et al., 1988; Seeman, 1996; see Taylor, 2009 for a review).

7.   Chronic negative affect

Deficits in socioemotional skills may ultimately stabilize into enduring risks for emotional disorders, such as anxiety, depression, and other chronic negative emotional states. These states may act as predisposing factors for adverse physical health outcomes (Hemingway et al., 2003). For example, hostility has been tied to the development of metabolic syndrome among children and adolescents (Dembroski et al., 1985) and to an increased risk for coronary heart disease (CHD) and hypertension (Julkunen et al., 1994). Major depression, depressive symptoms, and history of depression have all been identified as predictors of cardiac events (Frasure-Smith et al., 1995), and depression is a risk factor for mortality following myocardial infarction, independent of cardiac disease severity (Frasure-Smith et al., 1995). State depression, as well as clinical depression, have been related to sustained suppressed immunity (Herbert and Cohen, 1993). Anger appears to play a significant role in the development of coronary artery

disease and hypertension, at least among some individuals (e.g., Julkunen et al., 1994; Smith, 1992). Depression and anxiety are implicated in numerous health risks, including all-cause mortality (Martin et al., 1995), and evidence points to a dose-response relation between anxiety and coronary heart disease (Kubzansky, et al., 1998).

Links between negative emotional states and health outcomes may result from chronic or recurring engagement of biological stress regulatory systems. Negative emotional states have been tied to heightened biological stress responses, including evidence of stronger autonomic response to stressful circumstances (e.g., Matthews et al., 1996) and stronger hypothalamic pituitary adrenocortical (HPA) responses to stress (e.g., Flinn and England, 1997; Chorpita and Barlow, 1998). Studies also suggest links between negative emotions and reduced heart rate variability (e.g., Kawachi et al., 1995), implicating potential compromises in parasympathetic functioning in these relations. Intense, chronic, or recurring biological responses to stress may, thus, represent one pathway by which a harsh early environment exerts adverse effects on adult health outcomes (Repetti et al., 2002; McEwen, 1998), effects that may be mediated, at least in part, by negative emotional states.

8.   Health habits

Health habits are also implicated in these pathways (e.g., Repetti et al., 2002). A harsh early environment has been tied to increased rates of smoking, alcohol abuse, drug use, and risky sexual behaviors in adolescence and adulthood (e.g., Wagner, 1997). Both cross-sectional and longitudinal investigations have found that neglect, abuse, and conflict in the early environment predict poor health habits in adulthood (Repetti et al., 2002). Prospective studies have found increased rates of substance abuse and risky sexual behaviors among offspring of families

lacking cohesion or in offspring of parents who are neglectful and unsupportive (e.g., Baumrind, 1991; Shedler and Block, 1990), relations found in people who have been followed for many years after exposure to the initial environment (e.g., Repetti et al., 2002; Repetti et al., 2007). Moreover, there is evidence for non-genomic intergenerational transfer of these and related adverse outcomes (e.g., Noll, Trickett, Harris & Putnam, 2009). By contrast, a nurturant early family environment is associated with beneficial health behaviors, including maintaining a good diet, a propensity to exercise, good dental care and flossing, and obtaining regular checkups and immunizations (see Repetti et al., 2002, for a review).

The relation between a harsh early environment and poor health habits in adolescence may result from several factors. One is parental knowledge about and supervision of adolescent activities in homes. With less monitoring and more permissiveness, adolescents seek out more frequent sexual activity and are more likely to smoke and abuse other substances (see Repetti et al., 2002, for a review). Another potential route is the fact that substance abuse and risky sexual behavior may compensate for deficiencies in the social, emotional, and even biological functioning of offspring from risky families. That is, these adverse health-related behaviors may represent self-soothing behaviors that compensate for the absence of socioemotional resources that have been tied to a nurturant family environment (see Repetti et al., 2002, for a review). Unfortunately, poor health habits in adolescence can extend into adulthood and to future generations (Noll et al., 2009).

9.   Neural regulation of stress responses

The difficulties that offspring from harsh early environments have with developing effective socioemotional skills and self-regulatory behaviors may be evident in neural activity

that affects downstream neuroendocrine stress responses. The socioemotional skills described earlier have reliable effects on neural responses to threat cues, which in turn regulate downstream biological responses to stress. Brain regions implicated in threat detection and responses to emotional stimuli may mediate the relation between a harsh early family environment and elevated biological responses to stress.

A region consistently associated with threat detection and affective processing is the amygdala. The amygdala responds to a variety of emotion-related stimuli, including pictures depicting physical threats (Hariri et al., 2002) and faces depicting fear and anger (Hariri et al., 2000). Once activated, the amygdala sets in motion a cascade of responses to threat via projections to the hypothalamus and prefrontal cortex (LeDoux, 1996). A neural region that is critical for regulating responses to emotional stimuli is the ventrolateral prefrontal cortex (VLPFC; Hariri et al., 2002). Studies have shown that the labeling of negative affective states activates the right VLPFC and that increased activity in right VLPFC is associated with decreased activity in the amygdala (Hariri et al., 2000, 2002; Lieberman et al., 2005). This pattern of increased right VLPFC activity and decreased amygdala activity may be implicated in emotion regulation.

To test these ideas, we (Taylor et al., 2006) recruited participants who had previously completed assessments of family background. We conducted an fMRI investigation that examined amygdala reactivity to the observation of fearful and angry faces; amygdala and right VLPFC reactivity to labeling the emotions displayed in those faces; and the relation between right VLPFC and amygdala activity during the labeling task. We found that offspring from nurturant families showed expected amygdala activity in response to observing the fearful and angry faces and expected activation of right VLPFC while labeling the emotions. The relation

between right VLPFC and amygdala reactivity was significantly negative, consistent with the idea that right VLPFC activity inhibits amygdala responses to the threatening faces. Offspring from harsh families, however, showed a different pattern. During the observation of fearful and angry faces, they showed little activation of the amygdala. During the labeling task, they showed expected activation of right VLPFC; however, they also showed amygdala activation and a strong positive correlation between right VLPFC and amygdala activation, the opposite of what was seen in offspring from nurturant families. Thus, offspring from risky families exhibit atypical responses to emotional stimuli that are evident at the neural level (Taylor et al., 2006). Of interest, this pattern of neural responses to threat cues maps onto behavioral research showing maladaptive coping among offspring from harsh families. That is, offspring from risky families may avoid threat-relevant stimuli with which they need not engage, but overreact to and demonstrate an inability to regulate emotional responses to emotional stimuli with which they must engage. These responses are evident at both the behavioral and the neural levels.

Socioemotional skills are themselves related to the neural regulation of threat responses, and thus, constitute an indirect route whereby early environment is implicated in the regulation of biological stress responses. In a recent study (Eisenberger et al., 2007), participants completed a signal-contingent daily diary experience sampling procedure over a nine day period in which each time they were signaled, they rated how supportive their most recent social interaction had been. At the end of this period, participants took part in an fMRI investigation of neural responses to threat, specifically a virtual social rejection task (cyberball) that has previously been shown to evoke psychological distress (Eisenberger et al., 2003). At a third time point, participants experienced laboratory stress challenges (the Trier Social Stress Task (TSST), Kirschbaum et al., 1993) to assess autonomic and neuroendocrine reactivity to social stressors.

People who reported frequent supportive interactions showed lower dorsal anterior cingulate cortex (dACC) and Brodmann's area 8 (BA 8) reactivity to social rejection. These are brain regions whose activity has previously been tied to social distress. They also showed lower cortisol reactivity to the laboratory challenges. Moreover, individual differences in dACC and BA 8 activity mediated the relationship between social support and cortisol reactivity. Thus, positive socioemotional contact may influence downstream biological stress responses by modulating neurocognitive reactivity to social stressors, which in turn attenuates neuroendocrine stress responses.

A second study (Taylor et al., 2008) also examined whether socioemotional resources modulate reactions to threat cues. We tested two hypotheses, namely, whether psychosocial resources, including optimism, a sense of control, and high self-esteem, are tied to decreasing sensitivity to threat or whether they are associated with enhanced prefrontal inhibition of threat responses during threat regulation. In an fMRI investigation, participants responded to the threatening faces task described earlier. Socioemotional resources were associated with greater right ventrolateral prefrontal cortex activity and less amygdala activity during a threat regulation task. Participants had also gone through laboratory stress tasks, and meditational analyses suggested that the relation of socioemotional resources to low cortisol reactivity was mediated by lower amygdala activity during threat regulation. These findings suggest that socioemotional resources downregulate biological stress responses by means of enhanced inhibition of threat responses during threat regulation, rather than by decreasing sensitivity to threat.

10.   Impact of early environment on biological stress responses

The neural regulation of responses to threat ultimately affects downstream biological stress regulatory systems. What are these systems? During times of stress, the body releases the catecholamines epinephrine and norepinephrine with concomitant sympathetic nervous system arousal. Stress may also engage the HPA (hypothalamic-pituitary-adrenocortical) axis, involving the release of corticosteriods including cortisol. These responses have short-term protective effects under stressful circumstances, because they mobilize the body to meet the demands of pressing situations. However, with chronic or recurrent activation, they can be associated with deleterious long-term implications for health (e.g., Seeman and McEwen, 1996; Uchino et al., 1996, See chapter xx). For example, excessive or repeated discharge of epinephrine or norepinephrine can lead to the suppression of cellular immune function, produce hemodynamic changes such as increases in blood pressure and heart rate, provoke abnormal heart rhythms such as ventricular arrhythmias, and produce neurochemical imbalances that may relate to psychiatric disorders. Intense, rapid, and/or long-lasting sympathetic responses to repeated stress or challenge have been implicated in the development of hypertension and coronary artery disease (McEwen and Stellar, 1993).

Stress can also suppress immune functioning in ways that leave a person vulnerable to opportunistic diseases and infections. Corticosteroids such as cortisol have immunosuppressive effects, and stress-related increases in cortisol have been tied to decreased lymphocyte responsivity to mitogenic stimulation and to decreased lymphocyte cytotoxicity. Such immunosuppressive changes may be associated with increased susceptibility to infectious disorders and to destruction of neurons in the hippocampus as well (McEwen and Sapolsky, 1995). Chronic stress can also diminish the immune system's sensitivity to glucocorticoid

hormones that normally terminate the inflammatory cascade that occurs during stress (Miller et al., 2002).

Extensive evidence suggests that these systems - the HPA axis, the immune system and the sympathetic nervous system - influence each other and thereby affect each other's functioning. To the extent, then, that early environment influences the affective states and socioemotional skills that can keep sympathetic nervous system and HPA axis responses to stress low, it may have a beneficial impact on other systems as well (Seeman and McEwen, 1996; Uchino et al., 1996). In turn, these may beneficially affect health.

Correspondingly, a lack of supportive contacts in early childhood has been tied to higher autonomic responses to stress in children (e.g., El-Sheikh et al., 1989), and to higher HPA axis responses to stressors in children (Gunnar et al., 1992). Studies of young adults reveal that a harsh early family environment is tied to elevated autonomic responses to a laboratory stressor, and to an elevated flat cortisol response to laboratory stressors (Taylor et al., 2004). Thus, the existing literature provides a strong basis for pathways linking a stressful early childhood to high reactivity of biological stress regulatory systems.

11.   Early adversity and health outcomes: tests of the model

The association of early adversity with adverse health outcomes is clear. Not only does outright abuse impact health across the lifespan, but low SES in childhood and a harsh early family environment also affect health. To this point, we have identified socioemotional skills, chronic negative affective states, and health behaviors as among the mediators to health outcomes. As yet, however, no tests of the entire model have been presented to suggest that these are indeed the routes by which early environment has adverse health effects.

We accordingly undertook several collaborative studies with the Coronary Artery Risk Development in Young Adults Study (CARDIA), an ongoing, prospective, epidemiologic investigation of risk factors for coronary artery disease involving more than 3,000 participants at four different recruitment sites (Lehman et al., 2005). The samples were approximately evenly balanced between African American and white participants and between men and women. At the initial examination, participants were between the ages of 18 and 25. There have been five follow-up studies since that time, most recently at year 15 (2000-2001). Our investigations with CARDIA used structural equation modeling to determine whether the model in Figure 6.3.1 can account for individual differences in adult metabolic functioning, C-reactive protein (CRP), and blood pressure.

Metabolic functioning is a complex of risk factors for coronary artery disease and diabetes, and is typically defined by fasting glucose, cholesterol, triglycerides, and abdominal obesity, among other indicators. High levels of these variables contribute to metabolic syndrome, which is prognostic for heart disease, diabetes, inflammatory disorders, and all-cause mortality. The prevalence of metabolic syndrome in the United States is approximately 22% (McEwen and Seeman, 1999), making it an important contributor to chronic illness.

We had included an assessment of early family environment during the year 15 CARDIA data collection and tested our model on this sample with a composite index of indicators of metabolic functioning as an outcome variable. Socioemotional functioning was assessed by depression-hostility and the positivity/negativity of social contacts. The model fit the data very well, with early family environment strongly related to socioemotional functioning, which in turn, was significantly related to metabolic functioning (Lehman et al., 2005). When each of the race-sex subgroups was examined separately, the model continued to be an acceptable fit. These

findings suggest that early environment is significantly related to dysregulation in socioemotional functioning, which in turn, leads to alterations in metabolic functioning.

A second investigation related the model to CRP (Taylor et al., 2006). CRP is a biomarker of inflammatory processes which has been reliably related to depression (e.g., Suarez, 2004) and to enhanced risk for cardiovascular disease (King, Mainous, & Taylor, 2004), among other diseases. As was true of metabolic functioning, the model was a good fit to the data, suggesting that the model helps to explain differences in CRP. Since CRP is related to risks for both mental and physical health disorders, it may be important for understanding the comorbidities observed between mental and physical health disorders (e.g., Martin et al., 1995).

In a third investigation (Lehman et al., in press), we related the model to blood pressure and to changes in blood pressure across the longitudinal occasions with the CARDIA sample. We found that a harsh family environment was related to negative emotions and to obesity, which in turn, predicted blood pressure as well as change in blood pressure. Low childhood SES directly predicted change in systolic blood pressure as well. The strength of these pathways did not vary by race or gender. Thus, the findings suggest that socioemotional factors contribute to biological mechanisms that may underlie the impact of early family environment on the development of elevated blood pressure.

Two important caveats deserve mention. First, the effects revealed in these tests of the model were modest in size. One reason is that genetic factors are strong contributors to these outcomes, and they could not be measured in this dataset. Second, the fact that participants reconstructed their early family environment and that these studies were retrospective rather than prospective raises the possibility that negative emotions themselves color reconstruction of family environment. Accordingly, for all three of these investigations, we evaluated an

alternative model that gave chronic negative affect causal priority in the model to see if it affected reconstruction of family environment. In all three cases, this model was a significantly poorer fit to the data. Moreover, there is parallel evidence from studies relating documented childhood maltreatment to adverse mental and physical health outcomes (Danese, Pariante, Caspi, Taylor, & Poulton, 2007; Danese et al., 2008). As such, we conclude that, although negative affect may color how people regard their families, reconstructive biases do not account for the relation of early family environment to adverse health outcomes.

12.   Conclusions

Growing up in a stressful early environment marked by low SES and/or harsh parenting has effects on socioemotional skills, chronic negative affect, and health behaviors that are implicated in downstream adverse health outcomes. The evidence is, thus, consistent with our theoretical model, namely that the failure to learn emotion recognition and regulation skills in early childhood due to a harsh early environment may interfere with the ability to manage potentially threatening stimuli. Compromises in the regulation of stress responses may ultimately produce changes in biological stress regulatory systems, which, in turn, confer a broad array of mental and physical health risks. Findings such as these underscore the vital importance of developing methods for identifying children at risk for maltreatment early and for developing interventions to offset or attenuate adverse costs to socioemotional regulation incurred from maltreatment.

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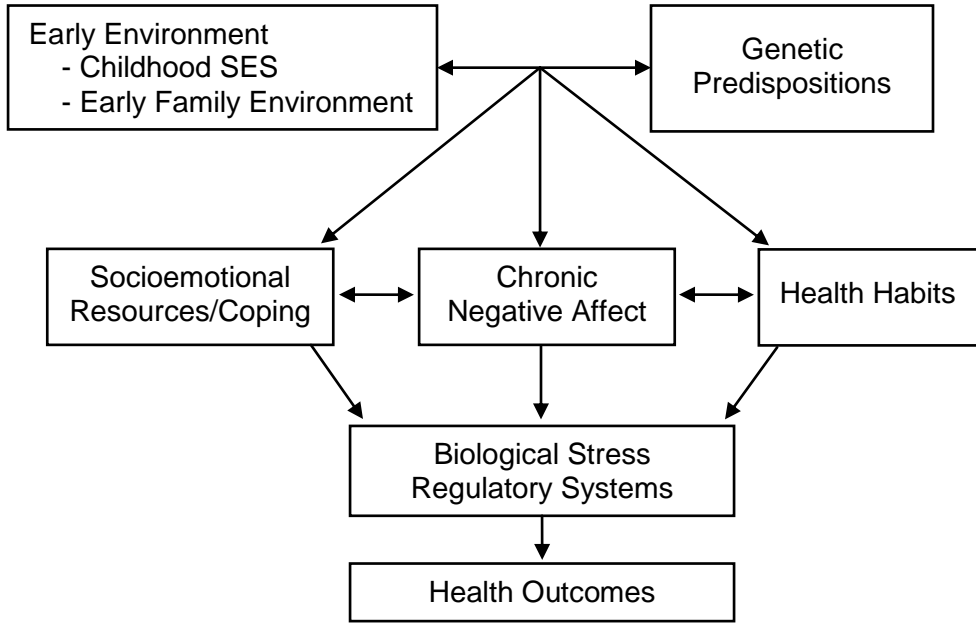
Figure 1. A model of the impact of early environment on biological risk profiles and health

outcomes