Early Environment, Emotions, Responses to Stress, and Health

Shelley E. Taylor
University of California, Los Angeles
Jennifer S. Lerner
Carnegie Mellon University
Rebecca M. Sage
Loyola Marymount University
Barbara J. Lehman and Teresa E. Seeman
University of California, Los Angeles

ABSTRACT A harsh early family environment is related to mental and physical health in adulthood. An important question is why family environment in childhood is associated with these outcomes so long after its initial occurrence. We describe a program of research that evaluates a model linking these variables to each other. Specifically, we hypothesize that low social competence and negative emotional states may mediate relations between a harsh early family environment and physiological/neuroendocrine responses to stress, as well as long-term health outcomes. We report evidence that the model characterizes self-rated health, cortisol responses to stress, and, in males only, elevated cardiovascular responses

This research was supported by NIMH grant MH056880 and by funds from the MacArthur Foundation's SES and Health Network. The first and third authors were supported by NIMH grant MH62376-01 and by a grant from the Fetzer Foundation. The second, third, and fourth authors were supported, in part, by NIMH training grant 15750. We are grateful to Marisa Callaghan, Sara Fernandes-Taylor, Jennifer Harmon, Matthew Loew, Justin Malakhow, Nina McDowell, Urvi Patel, Regan Roby, and Heidi Stayn for their assistance on this project.

Correspondence concerning this article should be addressed to Shelley E. Taylor, UCLA Department of Psychology; 1282A Franz Hall; Los Angeles, CA 90024 or to taylors@psych.ucla.edu.

Journal of Personality 72:6, December 2004.
Blackwell Publishing 2004

to stress. We discuss how the social context of early life (such as SES) may affect the family environment in ways that precipitate adverse health consequences. Perspectives on comorbidities in physical and mental health are discussed.

Early family environment affects the development of emotional, social, and biological mechanisms that underlie the ability to regulate stress. The goal of this article is to articulate and test a model of these processes. We describe our concept of risky families, namely families in which offspring are exposed to cold, conflict-ridden, or neglectful parenting, and discuss the relation of family environment to the development of emotion regulation and social competence skills. Using downstream markers of these variables—chronic negative emotions and (lack of) social support—we evaluate the fit of our model to autonomic and neuroendocrine stress responses and to self-rated health. In doing so, we point out how the social environment, specifically how stressful it is, can take a toll on family functioning.

Development of Stress-Regulatory Systems

As is true of all animals, humans begin life with emerging abilities to monitor the environment for potential threats. Areas in the amygdala are activated any time there is something new or unexpected in the environment, especially if there are signs of danger. Early in life, the amygdala sends many messages of alarm. Any loud noise, for instance, will upset an infant, and a few months later, a stranger typically provokes distress. Over time, as the prefrontal cortex develops, children learn ways to moderate the signals that they get from the amygdala and become increasingly sophisticated in ways that provide finely tuned information about both the threatening and the comforting aspects of the world. As such, the brain and its emotional underpinnings fundamentally represent a system for managing threat.

How this system develops is critically affected by early family life. Through relationships, infants learn to form ties with others, which may become comforting and, in turn, give rise to emotion-regulation skills and social skills that ultimately enable them to manage potentially threatening events on their own. By emotion-regulation skills, we refer to the experience, control, and expression of emotion, particularly in emotionally arousing situations (Repetti, Taylor, &

Seeman, 2002). By social skills, we refer to how socially competent children are at managing the frustrating and challenging experiences they have with family and peers, such as whether they demonstrate a socially constructive response versus an antisocial, aggressive, or withdrawn response to such circumstances (see Repetti et al., 2002). In family environments where the information from others is not soothing and comforting, these vital skills may be deficient.

How do socioemotional skills contribute to long-term change in stress-regulatory systems? When a threat arises, the amygdala sends messages to the hypothalamus, which, in turn, engages stress responses. There are two main stress systems of the body. One is the sympathetic nervous system; the body releases the catecholamines epinephrine and norepinephrine with concomitant arousal. A second system is the hypothalamic pituitary adrenocortical axis, which involves the release of corticosteroids, 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 recurrent or chronic activation, they can lead to adverse effects with poor implications for health. That is, if these stress systems are continuously or frequently activated, their elasticity can be compromised, with the result that their parameters may change, and stress responses may not be as well regulated over time (McEwen & Stellar, 1993; McEwen, 1998).

Socioemotional skills develop early in life and are critical to whether challenging circumstances are experienced as stressful or not. As such, they are implicated in the evolving functioning of these biological stress-regulatory systems. Early nurturant experiences not only help people to manage challenging circumstances throughout life but also moderate the frequency and extremity of biological stress responses. Early experiences in nonnurturant families lead to a very different pattern and may consequently accelerate the accumulating biological and psychological damage that recurrent or chronic exposure to stress may produce (Repetti et al., 2002).

Consistent with this reasoning, families characterized by overt conflict and aggression and/or by a cold and unaffectionate interaction style have children with an enhanced risk for a wide variety of emotional and behavioral problems (Repetti et al., 2002). A broad array of cross-sectional and prospective investigations has shown that conditions ranging from living with irritable and quarreling parents to being exposed to violence and abuse at home show

associations with mental health problems in childhood and lasting effects into the adult years (Repetti et al., 2002). Mounting evidence suggests that children from these "risky families" are at risk for a broad array of physical health problems across the life span as well (Felitti, et al., 1998; Repetti et al., 2002). The objective of our research program has been to build and test a theoretical perspective that may enlighten the relations between early family environment and adult health outcomes.

Our model focuses on the pathways by which risky family characteristics influence both childhood and adult health. We maintain that these families create a cascade of risks that exacerbate or lead to vulnerabilities and deficits in children's emotional and social skills for meeting the social environment and that may also produce or exacerbate disturbances in physiological and neuroendocrine responses to stress. Specifically, we maintain that risky families create deficits in children's control of and expression of emotional states, as well as their ability to interact with others in an effective manner. These deficits can lead to disturbances in physiological and neuroendocrine responses that, over time, have adverse implications for health.

Our thinking has been guided, in part, by the theory of allostatic load (McEwen, 1998; Seeman, Singer, Horwitz, & McEwen, 1997). McEwen and colleagues maintain that chronic or recurrent stress can lead to cascading, potentially irreversible changes in biological stress-regulatory systems. Such changes may assume the form of main effects of stress or interactions between stress exposure and genetic or acquired risks. Over time, these effects can lead to large individual differences in biological markers of the cumulative effects of stress and in stress-related physical and mental disorders. Thus, exposure to a risky family environment may produce physical wear-and-tear on the body that, over the lifetime, is associated with less healthy stress-related biological profiles and trajectories.

Animal Studies of Nurturant Contact

Some of the earliest evidence for the impact of early environment on offspring responses to stress has come from animal studies. Meaney and associates (Liu, et al., 1997; Francis, Diorio, Liu, D. & Meaney, 1999), for example, have employed a paradigm in which infant rats are separated from the mother and removed from the nest, stroked,

and then subsequently returned. The maternal response to being reunited is to immediately begin vigorous licking and grooming and arched-back nursing of the returned offspring. Offspring who are the recipients of this nurturant attention get lifelong protection against stress as a result. The immediate effect of these maternal behaviors is to reduce corticosterone responses and sympathetic activity in the offspring. The long-term effects of maternal nurturance involve regulation of emotional and biological responses to stress. Specifically, in these studies, rat pups who were the recipients of warm maternal care had more hippocampal glucocorticoid receptors, lower hypothalamic corticosteroid releasing factor, and less glucocorticoid secretion during a stressor, and faster recovery to baseline afterwards. They also showed more open-field exploration, suggesting less anxiety. They were less likely to show age-related onset of HPA-axis dysregulation in response to challenge and were less likely to exhibit age-related cognitive deficits.

This compelling animal model suggests that nurturant stimulation by the mother modulates offspring responses to stress in early life in ways that have permanent effects on the offspring's HPA-axis responses to stress. Conceptually related studies with macaque monkeys have shown similar effects. Rosenblum, Coplan, and colleagues (Rosenblum, et al., 1994), for example, manipulated the environments in which mother macaque monkeys raised their offspring by altering how easy or difficult it was for them to find food. The purpose of the study was to see if harsh or difficult conditions influenced the mother's care giving towards her infants and to examine how the infants' development was affected as a result. In one environment, food was readily available and in those environments, the mother monkeys were attentive to their offspring, whose development proceeded normally. In a second environment, finding food required a lot of effort, but the mothers still raised their offspring with attentiveness, and normal development of the offspring ensued. In the third environment, however, food was sometimes plentiful and sometimes not, and under these "variable foraging" conditions, the mothers became harsh and inconsistent in their mothering. The offspring of variable foraging mothers showed clear biological signs of being under intense stress. Infants exhibited sustained clinging to the mother, low levels of social play and exploration, and high levels of affective disturbance. The authors concluded that when mothers are psychologically unavailable to their infants due to ongoing stress

in the environment, the resulting attachments will be less secure, normal emotional and social development will be disrupted, and psychopathology will be more likely to develop (Rosenblum & Paully, 1984; see also Coplan, et al., 1996). Even in adulthood, the offspring raised in the variable foraging environments had more extreme HPA-axis responses to stress, and they were fearful and socially maladept as well. As adults, they had more dominance struggles and lower levels of grooming, suggesting long-term deficits in social behavior. The important role that early nurturance plays in normal growth and socioemotional development is now well documented in animal studies.

Conceptualization of Risky Families

Although our research program began without benefit of these animal studies, the convergences with that evidence are striking. We have focused on a variable that is conceptually related to that studied in the animal research, which we call "risky families." As noted, it refers to families lacking in nurturance, characterized instead by overt conflict and aggression; by a cold, unaffectionate interaction style; or by neglect (Repetti et al., 2002). Although risky families may include extreme cases of physical or sexual abuse, in many cases, the dysfunction observed in risky families is well within normal bounds, and, as such, the goals of our research program have been to elucidate how relatively normal and common family dysfunction may, nonetheless, have an impact on offspring's emotional, social and biological development.

Considerable research suggests that children from risky families have an enhanced risk for a broad array of emotional and behavioral problems as well as health problems. For example, in a study of 13,494 adults, Felitti and colleagues (Felitti et al., 1998) found a strong, graded relationship between exposure to abuse or household dysfunction during childhood and risk for a variety of adult health disorders, including ischemic heart disease, some cancers, chronic lung disease, skeletal fractures, and liver disease (see also Russek & Schwartz, 1997; Walker, et al., 1999). They also found a similar graded relationship for certain mental health outcomes, including a depressive episode and whether suicide had ever been attempted. These findings converge with other research, both retrospective and prospective, that has documented similar effects (Repetti et al., 2002).

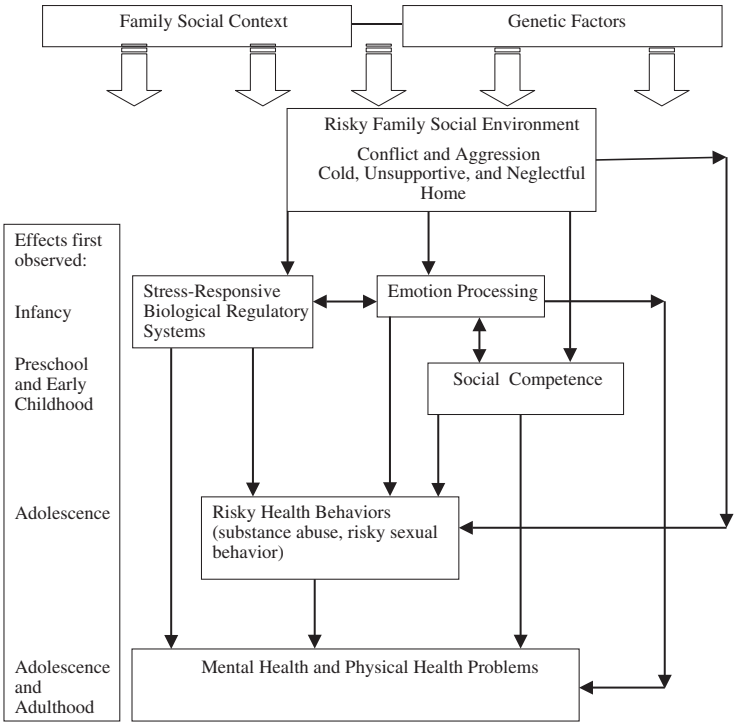

Figure 1
Risky families model (*Source:* Repetti, Taylor, & Seeman, 2002).

The important question that arises is why an adverse family environment in childhood is associated with such a broad array of mental and physical health disorders so long after its initial occurrence, that is, in adulthood. Our hypothesis is that exposure to harsh or chaotic parenting during childhood affects these diverse health and mental health outcomes in adulthood via their impact on emotion-regulation skills, social competencies, and ultimately, biological responses to stress. Figure 1 portrays this model.

The family social and biological context, including such factors as the family's socioeconomic resources and genetic factors, act as input to a risky family social environment. Low socioeconomic status (SES) has been argued to be an input to a risky family environment

(Dodge, Pettit, & Bates, 1994; McLoyd, 1998; Emery & Laumann-Billings, 1998; Repetti et al., 2002). Specifically, SES may be thought of, in part, as a marker for the chronic stressfulness of the environment (see Adler, Marmot, & McEwen, 2000), and chronic stress takes a toll on relationships, including those in the family. Consistent with this point, low SES has been tied to all of the risky family characteristics noted above, and reductions in SES have been associated with an increase in risky family characteristics. Poor children are at heightened risk for physical mistreatment or abuse (e.g., McLoyd, 1998; Reid, Macchetto, & Foster, 1999) and are more likely to be in family relationships lacking in warmth and support (Bradley, Corwyn, McAdoo, & Coll, 2001; McLeod & Shanahan, 1996). Sustained poverty and economic problems move parenting in a more harsh, punitive, irritable, inconsistent and coercive direction (e.g., McEwen, 1998; Wahler, 1990).

Genetic factors also act as an input to risky families. Some of the characteristics that increase the likelihood of a risky family environment, such as hostility in the family, may have genetic origins (Plomin, DeFries, Craig, & McGuffin, 2003). In addition, children who are genetically predisposed to particular problems (such as children with overly reactive or overly inhibited temperaments) may be more adversely affected by a risky family environment than children without such preexisting vulnerabilities. To date, our research has not explicitly addressed these genetic bases for risky family environments and their consequences. But the fact that the same family characteristics appear to fuel such a diverse array of adverse physical and mental health outcomes suggests that the impact of a risky family environment might be to exacerbate preexisting risks as well as, or perhaps instead of, creating risks that would not otherwise exist.

The most immediate products of a risky family environment are difficulties in emotion regulation. Children from risky families do not do well at recognizing their own emotions, recognizing the emotional states of others, and managing their emotional responses to social situations (Repetti et al., 2002). In developmental psychopathology parlance, these children may exhibit high levels of internalizing (e.g., social withdrawal, anxiety) or externalizing (e.g., aggression, hyperactivity) problems. In adolescence, young adulthood, and extending into adulthood, these problems may stabilize into chronic negative emotional states, including chronic anxiety, depression, or hostility (see Repetti et al., 2002).

Repetti et al. (2002) reviewed research linking early environments marked by harsh and chaotic parenting to deficits in emotion-regulation skills and to the development of negative emotional states. The fact that deficits in emotion regulation are seen quite early in offspring from risky families, coupled with the relation of chronic negative emotional states to disease states later in life, makes negative emotions potential markers of disruptions in emotion-regulation skills and candidates for mediating the relation between early family environment and health outcomes. For example, hostility has been tied to risk for coronary heart disease (e.g., Dembroski, MacDougall, Williams, Haney, & Blumenthal, 1985); epidemiological evidence points to a dose-response relationship of anxiety to coronary heart disease (e.g., Kubanski, Kawachi, Weiss, & Sparrow, 1998); major depression, depressive symptoms, history of depression, and anxiety have been identified as predictors of cardiac events (Frasure-Smith, Lesperance, & Talajic, 1995); and depression is a risk factor for mortality following myocardial infarction, independent of disease severity (Musselman & Nemeroff, 2000). Clinical depression has been related to sustained suppressed immunity (Herbert & Cohen, 1993). Both anxiety (Chorpita & Barlow, 1998) and depression (Chrousos & Gold, 1992) have been tied to atypical HPA-axis diurnal rhythms and responses to stress. Thus, deficits in emotion-regulation skills and their relation to chronic negative emotions may well represent vital pathways that link early family environment to health and mental health outcomes later in life.

Risky families also produce children lacking in social competence. Studies that document this relation often involve researchers going into the home, observing the family social environment, and then obtaining ratings from teachers and/or peers of a target child's social competence. The common finding is that risky family environments produce children who are unpopular, in some cases, highly aggressive, and in other cases, socially withdrawn (Repetti et al., 2002). These deficits in social skills may also represent risk factors for disease later in life, because individuals with deficits in social skills may have difficulty attracting or maintaining social relationships. As such, their ability to gain social support may be compromised. Social support is increasingly recognized as an important contributor to health. The lack of social support is a predictor of all-cause mortality in humans and animals (House, Landis, & Umberson, 1988). A lack of social support has also been tied to poor immune function in

response to stress (e.g., Cacioppo, et al., 1998), to a heightened risk of infectious disorders (e.g., Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997), and to poor recovery from illness (Seeman, 1996).

As noted, our hypothesis has been that these socioemotional variables contribute to poor health by exacerbating the accumulating damage that may be done to stress systems over the life span. That is, when offspring lack the emotional and social skills for managing their reactions and responses to stressful events, their biological responses to stress may be stronger, chronic, or recurrent, and slower to return to normal than is the case for those with better social and emotional skills for managing stress. As such, these biological stress-regulatory systems may become overly reactive, unresponsive, and/or less resilient (see McEwen, 1998). Accordingly, we hypothesized that by taking a "snapshot" of these systems during late adolescence/young adulthood, we might be able to see signs of this accumulating damage, in advance of the pathology and extant illness that has been so widely documented later in life as a function of risky families.

Investigations of the Risky-Families Model

We recently completed several investigations that have assessed this model. In the first study, the participants (ages 18–25) were prescreened for medical and psychological problems so as to avoid confounding the neuroendocrine assessments. In a three-part investigation, participants first completed self-report measures of personality and individual differences, including those that assessed family background, chronic emotional states, and social relations. Approximately a week later, they participated in an interview that included an extensive assessment of early family environment. Following the interview, in the late afternoon, they participated in an acute-stress laboratory challenge, patterned, in part, after the arithmetic portion of the Trier Social Stress Task (Kirschbaum, Pirke, & Hellhammer, 1993), specifically, counting backwards as rapidly as possible by 7s from 9,095 and by 13s from 6,233 under harassing conditions. These tasks were supplemented by other stressful laboratory tasks (Shedler, Mayman, & Manis, 1993), such that the entire challenge period was approximately 25 minutes in length, a sufficient period of time for stress-related increases in cortisol to be detected (Dickerson & Kemeny, in press). Heart rate and blood pressure were assessed throughout the protocol, and saliva was collected prior to the

interview, following completion of the stress task, and following a 30-minute recovery period for the assessment of cortisol levels.

To address the early family environment, we used two procedures. The first was a questionnaire based on the measure employed by Felitti and colleagues (1998), which includes such questions as: How often did a parent or other adult in the household make you feel that you were loved, supported, and cared for? Swear at you, insult you, put you down, or act in a way that made you feel threatened? Express physical affection for you, such as hugging or other physical gesture of warmth and affection?

The second assessment was an interview in which many questions were asked about the nature of the family environment. From the interview transcripts, trained coders rated the family dynamics that had been reported by the participants on a broad array of rating scales. Examples are: Did the family argue and fight a lot? Was the participant exposed to fighting between parents? Was there physical violence in the family? Was the participant verbally abused? Was there physical affection? Coders of the interviews were able to achieve a reliability of 0.91. The full text of the interview is available from the authors.

Studies that require participants to reconstruct their early family environment and then relate those answers to mental and physical health have the potential to confound actual childhood environment with response biases and/or preexisting psychopathology. For example, it is possible that people who are ill in adulthood reconstruct their childhood environment negatively, or that people who are chronically high in negative affect see both their childhood and their current physical health in a negative light. Even with young adults who have recently left home, there is the possibility that their reconstructions of what their family life was like may be colored by factors that may also affect health; such confounding would establish a relation not between risky families and health outcomes, but between a variable correlated with both risky family assessments and stress responses, such as neuroticism. Validating reports of early childhood is difficult, inasmuch as other people with the potential to reconstruct the environment, such as the mother, father, or siblings, may both see the early environment differently than the participant and/or have issues of self-presentation that would lead them to provide unreliable or biased reports.

Consequently, we obtained multiple assessments of the early family environment, and we used the interview data to help validate the

questionnaire. Factor analysis demonstrated one factor common to both instruments, which we termed "cold and unaffectionate family." The interview and questionnaire assessments of conflict-ridden families, however, were less highly related. Inspection of the instruments revealed that, although the interview had tapped whether or not participants were exposed to conflict and fighting within the family (without being personally involved), the questionnaire had assessed only whether a participant had been involved in fighting or abuse. Because one of the most common stressors in risky families is being exposed to family conflict, we needed to capture this component. Taken together, the two instruments tap a broad range of risky family characteristics, and so we combined the questionnaire and interview ratings into a single risky families measure, using factor analysis with promax rotation. We address the question of whether there may be sub-types of risky families associated with different outcomes later in the article.

Risky Families and HPA-Axis Functioning

Our theory maintains that a risky family background will be associated with signs of potential dysregulation in HPA axis responses to stress and that these relations are mediated by emotional distress and social competencies, as noted above. An analytic technique that examines such a model in its entirety is structural equation modeling. We examined whether our theoretical model could account for participant variation in baseline cortisol, cortisol responses to the stress challenge, and in cortisol recovery following the challenge, as indicators of HPA-axis activity.[1] Figure 2 models those relationships, showing all significant paths and, for illustrative purposes, also retains all theoretically predicted paths that border on significance ($p < .10$). Maximum likelihood estimators were used to calculate parameter estimates in the model.

Socioeconomic status is recognized to be an input to the risky family process, as noted earlier; this was also the case in this dataset. Risky family environment was, in turn, associated with significantly higher levels of depression and anxiety and with somewhat elevated hostility as well. As the model also reveals, participants who came from risky family backgrounds and who sustained high levels of

1. Significant increases in heart rate, blood pressure, and cortisol were found in response to the stress tasks.

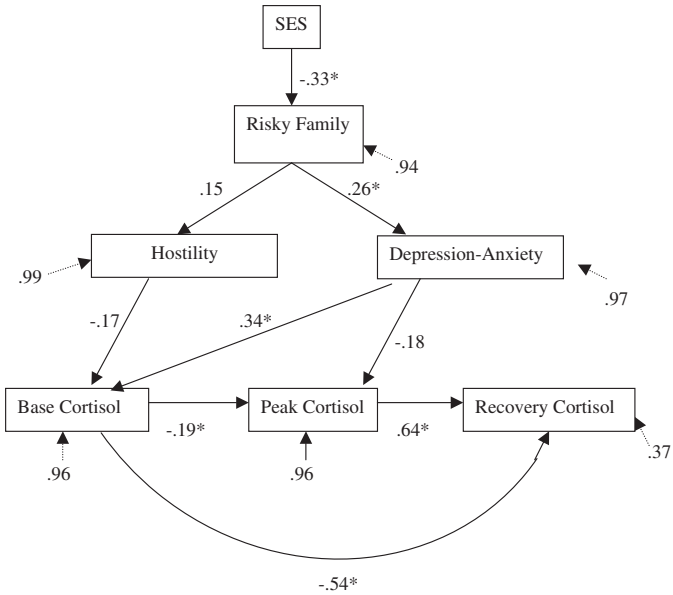

Figure 2
Structural equation modeling relating theoretical model to cortisol responses to stress. Entries represent path coefficients. $*p<.05$. ($\chi^2 = (11) = 5.68$, $df = 11$, $p<.89$); Bentler-Bonett Normed Fit index = .98; Bentler-Bonnett Nonnormed Fit index = 1.00, and, CFI = 1.00. (The error term for the combined anxiety-depression score was correlated .50 with hostility).

anxiety and depression showed cortisol levels that were more likely to be elevated at baseline and flat throughout the protocol. Those participants who perceived their early home life to be warm and nurturant were more likely to exhibit a lower baseline cortisol and the expected increase in cortisol in response to stress. To assess the fit of the model to the observed data and modeled covariance matrix, the chi-square statistic and several fit indices (CFI) were calculated. The model provided a good fit to the data, as indicated by these fit indices (see Figure 2).

It is important to note that the relation of anxiety/depression to elevated baseline cortisol was seen, even though participants had been prescreened to exclude anyone under treatment for an anxiety-related or depressive disorder. Consequently, these findings suggest that, even among individuals with subclinical levels of anxiety and depression, HPA-axis activity may show signs of potential dysregulation.

Risky Families and Autonomic Responses to Stress

A second goal of our research program has been to examine the relation of risky families to sympathetic responses to stress. The sympathetic nervous system is the second major stress system which, together with the HPA axis, mobilizes the body for action in response to threat. Accordingly, like the HPA axis, any accumulating damage to stress systems conferred by growing up in a risky family should be evident in autonomic functioning, as well as in HPA-axis functioning.

We tested whether our causal model provided a good fit to heart rate and blood pressure responses to the stressful tasks. Although the fit of the model was poor, there are a priori reasons to believe that these effects would be found only for males. Prior investigations of responses to laboratory stressors have found that elevated heart rate and blood pressure are evident largely for boys from families with adverse family dynamics, but not for girls (e.g., Allen, Matthews, & Sherman, 1997; Woodall & Matthews, 1989). Such a pattern is, of course, also consistent with the reliably earlier onset of CVD for males, compared to females.

Consequently, we examined the relation of risky family background to indicators of autonomic function (heart rate, blood pressure) separately for males and females. Because the additional variable precludes the use of structural equation modeling, we divided the sample into quartiles on the risky families variable, with the fourth quartile indicating the harshest, most chaotic families and the first quartile, the most stable and nurturant families. The patterns were highly similar for both heart rate and blood pressure (heart rate is pictured in Figure 3), and show that males (only) from the riskiest families had significantly higher heart rate at baseline, immediately following the laboratory stress challenges, and at recovery. Marginally significant patterns that mirror these significant effects were seen for both systolic and diastolic blood pressure as well, but again only in males.

Risky Families and Health

Ultimately, one would anticipate that these dysregulations in HPA-axis and autonomic activity (as well as potentially in other systems) could lead to health risks. Although our young adult sample is

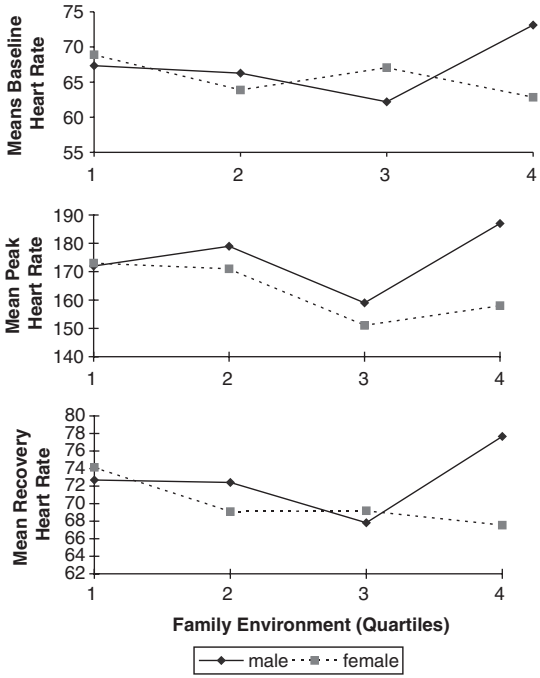

Figure 3
Relation of gender and family environment to baseline, peak, and recovery heart rate. Males (only) from quadrant 4 (riskiest families) had significantly higher heart rate at baseline, after the stress challenge, and at recovery.

unlikely to show these effects at such early ages, we did examine whether risky families affected self-rated health, testing the entire model. The model pictured in Figure 4 shows all significant pathways and any additional marginally significant theoretically predicted pathways ($p<.10$). As can be seen, lower SES is significantly related to growing up in a risky family; growing up in a risky family is related to the negative effects of depression, anxiety, and, marginally, to hostility; and depression is related to poorer subjective health. Anxiety is marginally related to poor health, as is hostility, but only by virtue of its (negative) relation to Relations with Others, which was positively, albeit marginally, related to subjective health. (Correlations among the error terms for the emotion variables were 0.70 for anxiety and depression; 0.47 for hostility and anxiety; and 0.55 for hostility and depression). There was no significant direct

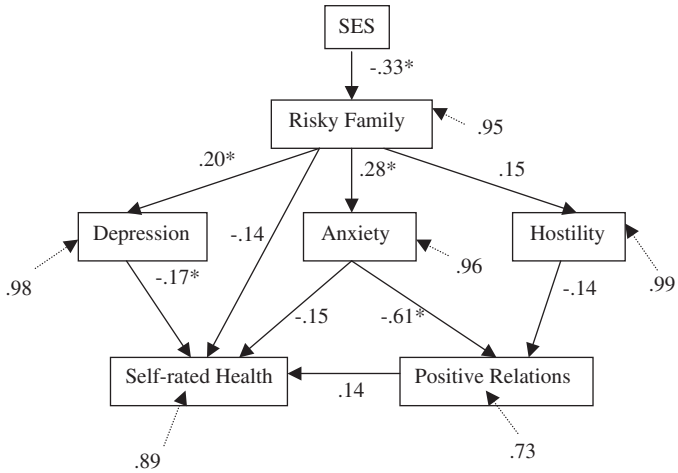

Figure 4
Structural equation model for self-related health. Entries represent path coefficients. *$p<.05$. (χ^2 (8) = 3.63, $p<.89$); Bentler-Bonett Normed fit index = .98; Bentler-Bonett Nonnormed fit index = 1.00; CFI = 1.00.

path from SES to health or from family environment to health, although the latter relation was marginally significant.

The model was a good fit (See Figure 4). The model chi square was nonsignificant, indicating that the model predicts the covariances among the model variables well. Although not all of the hypothesized pathways in the model were significant, the model required no post-hoc modifications to achieve a good fit. Overall, the model explained 21% of the variance in self-related health and 35% of the variance in positive relations with others.

Tests of alternative models. Negative affectivity or neuroticism can influence both the reporting of physical symptoms and subjective estimates of health (Watson & Pennebaker, 1989), and so it represents a potential alternative explanation of these results. Simply removing the contribution of neuroticism from the key variables in the model would be one solution to this concern, were it not for the fact that negative affect (a key component of neuroticism) represents a significant set of variables in the model (depression, anxiety, hostility).

Accordingly, to assess the ability of neuroticism to account for these effects, we ran alternative models using neuroticism as the first

step in the model. Specifically, we ran a model in which SES was removed from the model, and neuroticism was substituted as the first step. This model tests whether neuroticism accounts for reports of growing up in a "risky family," which, in turn, influences negative affective states, absence of social support, and poor subjective health. This model chi square was significant, indicating that this alternative model was a poor fit to the data (χ^2 (9) = 91.61; $p < .001$). The model fit indices were exceedingly low, indicating that the model was poorly specified (NFI = 0.67, NNFI = 0.24, CFI = 0.67). A second alternative model retained self-rated SES on the grounds that a subjective sense of SES may be influenced by neuroticism (Ostrove & Adler, 1998), but gave neuroticism causal priority in the model again to see if it might account for the subjective SES as well as for the subsequent variables in the model. This model, too, yielded a significant chi square and small fit indices, indicating that it was a poorly specified model (χ^2 (15) = 104.42; NFI = 0.64, NNFI = 0.35, CFI = 0.65). Neuroticism, as a causally prior variable, does not explain the relationships among the variables.

Although controls like these do not entirely remove concerns that might be expressed about self-rated health, they go some distance in suggesting that the primary potential confounding factor, namely neuroticism, does not appear to be the determinant of reports of risky families, or the emotional states, social relationships, and biological stress responses that may result. Moreover, although subjective health status is a self-report, and not a direct indicator of health, it is important to note that manifold evidence links self-rated health to concrete health outcomes, including mortality (Idler & Benyamini, 1997).

Risky Families: Current Explorations

Our current work explores the viability of the Risky Families model in a large representative sample that is well characterized biologically and medically and that has been followed over the past 15 years (CARDIA). CARDIA is an ongoing prospective epidemiologic study. The baseline data were collected in 1985–1986 at four sites: Minneapolis, Minnesota; Birmingham, Alabama; Chicago, Illinois; and Oakland, California. The total sample consists of approximately 5,115 participants (2787 women and 2328 men), approximately balanced at each center for race (African American, White), gender, and

socioeconomic status (SES). At the initial examination, approximately half of the participants were between the ages of 18 and 25, while the other half were between 26 and 30 years of age. There have been five follow-up examinations at year 2 (1987–1988), year 5 (1990–1991), year 7 (1992–1993), year 10 (1995–1996), and most recently at year 15 (2000–2001).

CARDIA has many advantages for studying pathways that may connect family environment to social, psychological, and biological properties and to health outcomes in later adulthood. The large sample has a substantial SES range, sufficient numbers to evaluate the model in four gender/ethnic groups (black men, black women, white men, white women), a wide range of both longitudinally and cross-sectionally evaluated biological parameters, and a (current) age range (33–45) that includes a substantial number of disease outcomes including hypertension, diabetes, and assessments of coronary calcification. At year 15, we received permission to include our risky family questionnaire measure on the CARDIA protocol, and data collection from that time point is now complete. Our analyses are still in progress, but we highlight some advantages and preliminary analyses of the CARDIA dataset.

Among the many opportunities afforded by this longitudinal sample is the ability to look at health behaviors as a pathway that intervenes between risky families and health outcomes. As pictured in Figure 1, the likelihood that a person will engage in risky health behaviors, such as substance abuse, risky sexual behavior, poor diet, lack of exercise, and other risk-related behaviors, appears to be increased by growing up in a risky family. Specifically, as reviewed in Repetti et al. (2002), we documented relations between the characteristics of risky families and an increased likelihood of smoking, alcohol abuse, drug abuse, and risky sexual behavior. Although the evidence for this association is based primarily on cross-sectional analyses and retrospective descriptions of problems in the family, stronger research designs have also documented increased rates of poor health behaviors in conflict-ridden homes. For example, in one study, interpersonal conflict in the adoptive homes of female adoptees interacted with a biological background of alcoholism (having at least one alcoholic biological parent) to increase the probability of alcohol abuse or dependence, although neither genetic risk nor family environment alone predicted problems with alcohol (Cutrona, et al, 1994; see also Widom & White, 1997). A large

number of prospective studies also demonstrate an association between growing up in a cold, unsupportive, or neglectful home and health risk behaviors, including cigarette smoking, increased drug and alcohol abuse, and an increased likelihood of an unwanted pregnancy (e.g., Baumrind, 1991; see Repetti et al., 2002 for a review).

The reasons underlying the relation of risky family characteristics to poor or risky health behaviors may be several. One possibility supported by existing research is that in neglectful homes there is inadequate parental knowledge about and supervision of adolescent activities in which these behaviors begin to develop. The direct effect of neglectful parents on these behaviors may be compounded by a concomitant increase in the influence exerted by peers. Most intriguing is the possibility that substance abuse, risky sexual behavior, and other such activities may represent a form of self-medication to compensate for biological dysregulations that result from growing up in risky families. For example, significant evidence relates smoking, alcohol abuse, and drug abuse to enhancement of serotonergic activity; dysregulations of the serotonergic system have been tied to several of the health outcomes related to risky families. Moreover, evidence from animal studies (Suomi, 1999) and human studies (Kaufman, et al., 1998) suggests that dysregulations of serotonergic functioning may be evident in at least some risky families and/or risky family characteristics may exacerbate genetic risks for lower than normal circulating levels of serotonin (see Repetti et al., 2002, for a discussion of this issue).

Of less immediate clinical concern, but, nonetheless, ultimately prognostic of later disease, are health habits themselves. In families that are poorly regulated, in which there is conflict, neglect, and disorganization, the likelihood that children will grow up practicing regular health habits may be lessened. In particular, one may expect to see a poorer diet, lack of exercise, poor dental hygiene, and other health habits that may be prognostic for chronic disease in adulthood. To date, our samples have not had the statistical power or variability in these behaviors needed to model the relation of health habits to signs of potential biological dysregulation in stress-regulatory systems. However, the CARDIA study has assessed health behaviors, including exercise, diet, smoking, alcohol consumption, and other health behaviors potentially prognostic for the development of heart disease over 15 years and the size of the dataset provides

ample variability and statistical power to see the potential relations of these risk-related behaviors to health and to examine whether they are implicated as pathways from risky families to biological outcomes.

At present, we are focusing on tests of the Risky Families model with respect to indicators of metabolic functioning in the CARDIA sample. The indicators include body mass index, low-density lipoprotein level (LDL), high-density lipoprotein level (HDL), fasting glucose, and fasting insulin. Preliminary analyses suggest that the Risky Families model is supported with respect to these outcome variables (Taylor, Lehman, Kiefe, & Seeman, 2003).

Risky Families: Where Do We Stand?

Our evidence to date suggests that the Risky Families model is a promising direction for understanding how early childhood environment is related to mental health and health outcomes in adulthood. More specifically, the tests of the model implicate emotional functioning (Repetti et al., 2002), as manifested in the chronic negative emotional states of anxiety, depression, and possibly hostility as intermediate steps in understanding these relations. Specifically, negative affective states, coupled with poor social relations, are linked to poorer self-perceived health, and chronic negative affective states are related to HPA-axis activity (Bugental, Martorell, & Barraza, 2003). As such, the results are consistent, both with a large literature from developmental psychology that relates early family dysfunction to emotional, social, and health problems in adulthood, and also with a large animal literature that has provided manifold evidence concerning the mechanisms that underlie these relations. As noted, that body of evidence from animal studies has heavily implicated HPA-axis functioning and affective states involving fear and depression-like symptoms as important outcomes of nonnurturant behavior early in life.

Concerns may be raised about the assessment of risky families. Although the measures employed in our model makes use of data from two different time points, by two different raters (self-report and judges coding narrative interviews), using two different types of material, nonetheless, the measures depend critically on participants' recollections of their early family environment. Unfortunately, reports that might provide validating evidence, such as accounts by

other family members, may themselves be colored by biases in the need to represent the family in a particular way. Although this remains an enduring concern, it should be recalled that measures very much like our own (e.g., Felitti et al., 1998) show dose-response relationships to a variety of hard health outcomes, including cancer, heart disease, liver disease, and skeletal fractures, and response biases alone are highly unlikely to account for such findings. Nonetheless, testing the model in circumstances in which early family environment characteristics are well validated is important.

What are the relations of early family environment and the process model we have detailed with health? In the research reported here, we focus primarily on self-rated health and pre-illness states (such as heightened SNS responses to stress), rather than on extant diseases. This is because our interest has been focused on the mechanisms that underlie developing dysregulations of stress systems, rather than on disease outcomes per se. These processes, however, may ultimately enhance risk for such disorders as hypertension, heart disease, and adult-onset diabetes, among others (e.g., Seeman, McEwen, Rowe, & Singer, 2001; Seeman, Singer, Rowe, Horwitz & McEwen, 1997), outcomes that may become evident as people age.

Discussion of the contributing role of socioeconomic status (SES) to these relations merits additional attention. SES has long been identified as a consistent predictor of all-cause mortality and several major causes of death (Adler Boyce, Chesney, Folkman, & Syme, 1993). In addition, it has been reliably related to poor mental and physical health in adulthood (Adler et al., 1994). Although differential access to health care and poor health habits explain some of this gradient, the residual variance is substantial (Adler, et al,, 1993). Some researchers have suggested that chronic stress is distributed across SES in a manner that may contribute to this gradient in health and mortality.

Our risky families model suggests several important implications for this issue. The first is that, as a source of chronic stress, low SES has adverse effects on parenting. Consistent with animal research using the variable foraging paradigm, environments that are inconsistent, chaotic, uncontrollable, and stressful may contribute to harsh maternal or parental behavior, which, in turn, is associated with the adverse emotional, social, and biological outcomes that

have been so persuasively documented in animal studies and which are now being documented in the human literature as well.

A second implication derives from the fact that childhood exposure to stress as a function of low SES may provide a partial mechanism for understanding the SES gradient in health and mortality. In the models that appear in Figures 2 and 4, there is no significant direct path from SES to the outcome variables of self-rated health or cortisol responses to stress, respectively. This means that the relation of SES to these outcome variables is entirely explained by exposure to a risky family environment in this particular sample. Although risky family environment is unlikely to be a sufficient explanation for understanding socioeconomic differences in mental health and health outcomes, it appears to be a contributing factor: early exposure to nonnurturant, harsh, conflict-ridden, or neglectful parenting that is exacerbated by low SES, can affect a young child at precisely the time that these important biological stress-regulatory systems are developing, and thus, the damage may be sustained over the long term. From animal studies, it is evident that maternal nurturance sculpts the HPA axis in ways that have profound implications for health and behavior across the life span. Very possibly, the same mechanisms may account for some of the SES-related health and mental health outcomes that have been tied to socioeconomic status. The fact that socioeconomic status is a consistent predictor of chronic negative emotional states, including anxiety, depression, and hostility (Gallo & Matthews, 2003) not only adds weight to these conjectures, but suggests that, as in the evidence reported here and in the animal studies, emotional mechanisms represent vital components of these pathways.

A notable feature of our evidence to date is the fact that we have documented signs of chronic negative emotional states and potential dysregulation in autonomic and HPA-axis responses to stress in a nonclinical sample; individuals who were either chronically ill or who were under treatment for anxiety or depressive disorders were screened out of the protocol. As such, these potential signs of dysregulation have been noted in a young and very healthy sample. In addition, few of the signs of "risky families" observed in the interview protocols would be classified as abusive, but rather as indicative of chaos, neglect, conflict, and other common family occurrences. Thus, the results suggest that "normal" family strain, well within the bounds of typical, everyday, family problems, may nonetheless relate

reliably to signs of potential dysregulation of biological stress-regulatory systems in offspring.[2]

The pathways predicting the outcomes in self-rated health and stress- regulatory systems were not direct but were indirect via negative emotions and lower levels of social support. In an earlier paper (Repetti et al., 2002), we reviewed evidence that the impact of an adverse home life appears early in high rates of internalizing and externalizing symptoms, which may later stabilize into elevated levels of anxiety, depression, and/or hostility; these states have been shown, in turn, to be related to difficulty forming and sustaining social relationships. We found evidence largely consistent with these pathways in young adult offspring, suggesting not only that stress system dysregulation may be seen as early as young adulthood but that the intermediate products that may exacerbate those dysregulations, namely negative emotional states and difficulties with social relationships, are present, to a degree, as well. As such, these findings may be relevant to understanding the substantial comorbidities that have been documented among mental health disorders (Kessler, 1997) and between mental and physical health disorders (Repetti et al., 2002).

A word should be said about potential genetic input to these processes. It is likely that shared genetic inheritance contributes to negative emotional states, family environment, child temperament, and poor health. We regard shared genetic heritage as a potential source of input to the model rather than as an alternative explanation. That is, genes are expressed in phenotypes that can be strongly influenced by characteristics of the environment. A nonnurturant environment has been found to influence genetic expression in animal studies (Liu et al., 1997), and even in animal populations that are well characterized with respect to genetic risks, meaningful alterations in exposure to nurturant parenting heavily influences the behavioral characteristics that emerge, especially in at-risk offspring (e.g., Suomi, 1999). As we have argued elsewhere, the impact of

2. Are these risky family processes related to more extreme stress disorders, such as post-traumatic stress disorder (PTSD)? Certainly, the elevated and flat cortisol response to stress associated with a risky family background is suggestive of such a link (e.g., LeMieux & Coe, 1995; Yehuda, Southwick, Krystal, & Bremne, 1993). We would urge caution, however, in making such a connection, given that important diagnostic criteria for documenting PTSD are not present in the study and participants under treatment for mental health disorder were explicitly excluded from the study.

harsh parenting on the development of poor mental and physical health in offspring may commonly assume the form of gene-environment interactions (Repetti et al., 2002).

Future directions. On the biological side, documenting changes in other stress responsive systems, such as serotonergic functioning (Repetti et al., 2002) and dopaminergic functioning, may elucidate pathways to mental health disorders, such as depression and anxiety disorders. A focus on the immune system may help to further enlighten ties to disease; this is a focus of our current work, which addresses the relation of risky family background to proinflammatory cytokine responses to stress. Continued attention to autonomic functioning, including potential alterations in parasympathetic functioning is another future direction. Charting the risky family model developmentally from early family environment through the mediating pathways to disease outcomes is the most important overarching goal. On the personality side, we have thus far focused on chronic negative emotions rather than on emotion-regulation skills. Charting the explicit pathways from the experience, control, and expression of emotion to stable representation in chronic negative emotions is an important link, suggested by considerable research that itself necessitates closer attention (see Repetti et al., 2002, for a discussion of this issue). Similarly, how social skills stabilize into the ability to attract and maintain social support similarly merits focused attention. Understanding the health implications of other emotional styles that may be tied to risky families, such as emotional lability, emotional repression, and emotional suppression, would be another important next step. In addition, a detailed examination of the interaction between generic risks and early environmental determinants of individual differences in emotion-regulation skills and in chronic negative emotions represents an important future direction as well. With respect to family environment, an important next direction will be to see whether particular types of risky families (e.g. conflict-ridden versus cold and unaffectionate) are associated with particular patterns of socioemotional skills and/or biological outcomes.

Conclusions

Researchers have known for decades that experiences such as abuse in the family are toxic for both the psychological and the physical

development of young children. Every year, thousands of children are murdered or disabled through violence in the family, and many children who survive abuse may show dysregulations in their responses to stress (especially HPA-axis responses) throughout childhood and into adulthood (e.g., Kaufman et al., 1998; Kaufmann, Plotsky, Nemeroff, & Charney, 2000). However, the dose-response relations found between exposure to family dysregulation and adult physical and mental health outcomes (Felitti et al., 1998) suggest the need for attention to the potential adverse effects of everyday "normal" family pathology. No family is ideal, and, as such, even in the best of families, children may be exposed to some degree of risky family characteristics. Our research suggests that, to the degree this is true, these subclinical sources of family dysfunction may, nonetheless, produce more modest versions of the same kind of damage that has previously been tied largely to more extreme cases of abuse or maltreatment.

The problems associated with risky families are serious ones, and they include coronary heart disease, cancers, and liver disorders, among other illness. Over the past 30 years, there has been a two- to three-fold increase in suicide and homicide rates in children—outcomes that have been reliably tied to adverse family characteristics (e.g., Malinoski-Rummell & Hansen, 1993). Emotional problems, including depression and anxiety disorders, are at very high levels as well. Risky families may be an important piece of the puzzle represented by these rampant social and public health problems.

Moreover, with respect to these problems, risky family environments may be a valuable point for intervention. Focusing on family characteristics that represent risk factors for major physical and mental health problems can provide the basis for early interventions that could, at least partially, offset the potential for cascading risk that may accumulate over the life span. Targeting emotion-regulation skills and social competencies as additional intervention points may similarly have beneficial effects, inasmuch as these pathways appear to be implicated in the sustaining effects that risky families have across the life span. Interventions that help parents learn behaviors that may shape effective behavioral and self-regulatory skills in children, especially those that affect emotional regulation and social competencies, may be especially valuable.

REFERENCES

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, C., Folkman, S., & Kahn, R. L., et al. (1994). Socioeconomic status and health: The challenge of the gradient. *American Psychologist*, **49**, 15–24.

Adler, N. E., Boyce, W. T., Chesney, M. A., Folkman, S., & Syme, L. (1993). Socioeconomic inequalities in health: No easy solution. *Journal of the American Medical Association*, **269**, 3140–3145.

Adler, N., Marmot, M., & McEwen, B. (Eds.). (2000). *Socioeconomic status and health in industrial nations: Social, psychological, and biological pathways*. New York: New York Academy of Sciences.

Allen, M. T., Matthews, K. A., & Sherman, F. S. (1997). Cardiovascular reactivity to stress and left ventricular mass in youth. *Hypertension*, **30**, 782–787.

Baumrind, D. (1991). The influence of parenting style on adolescent competence and substance use. *Journal of Early Adolescence*, **11**, 56–95.

Bradley, R. H., Corwyn, R. F., McAdoo, H. P., & Coll, C. G. (2001). The home environments of children in the United States: I. Variations by age, ethnicity, and poverty status. *Child Development*, **72**, 1844–1867.

Bugental, D. B., Martorell, G. A., & Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones and Behavior*, **43**, 237–244.

Cacioppo, J. T., Poehlmann, K. M., Kiecolt-Glaser, J. K., Malarkey, W. B., Burleson, M. H., & Berntson, G. G., et al. (1998). Cellular immune responses to acute stress in female caregivers of dementia patients and matched controls. *Health Psychology*, **17**, 182–189.

Chorpita, B. F., & Barlow, D. H. (1998). The development of anxiety: The role of control in the early environment. *Psychological Bulletin*, **124**, 3–21.

Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders: Overview of physical and behavioral homeostasis. *Journal of the American Medical Association*, **267** (9), 1244–1252.

Cohen, S., Doyle, W. J., Skoner, D. P., Rabin, B. S., & Gwaltney, J. M. Jr. (1997). Social ties and susceptibility to the common cold. *Journal of the American Medical Association*, **277**, 1940–1944.

Coplan, J. D., Andrews, M. W., Rosenblum, L. A., Owens, M. J., Friedman, S., & Gorman, J. M. et al. (1996). Persistent elevations of cerebrospinal fluid concentrations of corticotropin-releasing factor in adult nonhuman primates exposed to early life stressors: Implications for the pathophysiology of mood and anxiety disorders. *Proceedings of the National Academy of Sciences*, **93**, 1619–1623.

Cutrona, C. E., Cadoret, R. J., Suhr, J. A., Richards, C. C., Troughton, E., & Schutte, K., et al. (1994). Interpersonal variables in the prediction of alcoholism among adoptees: Evidence for gene-environment interactions. *Comprehensive Psychiatry*, **35**, 171–179.

Dembroski, T. M., MacDougall, J. M., Williams, R. B., Haney, T. L., & Blumenthal, J. A. (1985). Components of Type A, hostility, and anger-in: Relationship to angiographic findings. *Psychosomatic Medicine*, **47**, 219–233.

Dickerson, S. S., & Kemeny, M. E. (in press). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*.

Dodge, K. A., Pettit, G. S., & Bates, J. E. (1994). Socialization mediators of the relation between socioeconomic status and child conduct problems. *Child Development*, **65**, 649–665.

Emery, R. E., & Laumann-Billings, L. (1998). An overview of the nature, causes, and consequences of abusive family relationships. *American Psychologist*, **53**, 121–135.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Apitz, A. M., & Edwards, et al. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine*, **14**, 245–258.

Francis, D., Diorio, J., Liu, D., & Meaney, M. J. (1999). Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science*, **286**, 1155–1158.

Frasure-Smith, N., Lesperance, F., & Talajic, M. (1995). The impact of negative emotions on prognosis following myocardial infarction: Is it more than depression? *Health Psychology*, **14**, 388–398.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and health: Do negative emotions play a role? *Psychological Bulletin*, **129**, 10–51.

Heim, C., Ehlert, U., & Hellhamer, D. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuronendocrinology*, **25**, 1–35.

Herbert, T. B., & Cohen, S. (1993). Depression and immunity: A meta-analytic review. *Psychological Bulletin*, **113**, 1–15.

House, J. S., Landis, K. R., & Umberson, D. (1988). Social relationships and health. *Science*, **241**, 540–545.

Idler, E. L., & Benyamini, Y. (1997). Self-rated health and mortality: A review of twenty-seven community studies. *Journal of Health and Social Behavior*, **38**, 21–37.

Kaufman, J., Birmaher, B., Perel, J., Stull, S., Brent, D., & Trubnick, L., et al. (1998). Serotonergic functioning in depressed abused children: Clinical and familial correlates. *Biological Psychiatry*, **44**, 973–981.

Kaufman, J., Plotsky, P. M., Nemeroff, C. B., & Charney, D. S. (2000). Effects of early adverse experiences on brain structure and function: Clinical implications. *Society of Biological Psychiatry*, **48**, 778–790.

Kessler, R. C. (1997). The prevalence of psychiatric comorbidity. In S. Wetzler & W. C. Sanderson (Eds.), *Treatment strategies for patients with psychiatric comorbidity*. New York: John Wiley & Sons.

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'—a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, **28**, 76–81.

Kubzansky, L. D., Kawachi, I., Weiss, S. T., & Sparrow, D. (1998). Anxiety and coronary heart disease: A synthesis of epidemiological, psychological, and experimental evidence. *Annals of Behavioral Medicine*, **20**, 47–58.

Lemieux, A. M., & Coe, C. L. (1995). Abuse-related post-traumatic stress disorder: Evidence for chronic neuroendocrine activation in women. *Psychosomatic Medicine*, **57** (2), 105–115.

Liu, D., Dorio, J., Tannenbaum, B., Caldji, C., Francis, D., & Freedman, A. et al. (1997). Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. *Science*, **277**, 1659–1662.

Malinosky-Rummell, R., & Hansen, D. J. (1993). Long-term consequences of childhood physical abuse. *Psychological Bulletin*, **114**, 68–79.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, **338**, 171–179.

McEwen, B. S., & Stellar, E. (1993). Stress and the individual: Mechanisms leading to disease. *Archives of Internal Medicine*, **153**, 2093–2101.

McLeod, J. D., & Shanahan, M. J. (1996). Trajectories of poverty and children's mental health. *Journal of Health and Social Behavior*, **37**, 207–220.

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

Musselman, D. L., & Nemeroff, C. B. (2000). Depression really does hurt your heart: stress, depression, and cardiovascular disease. *Progress in Brain Research*, **122**, 43–59.

Ostrove, J. M., & Adler, N. E. (1998). The relationship of socio-economic status, labor force participation, and health among men and women. *Journal of Health Psychology*, **3**, 451–463.

Plomin, R., DeFries, J. C., Craig, I. W., & McGuffin, P. (Eds.). (2003). *Behavioral genetics*. Washington DC: American Psychological Association.

Rosenblum, L. A., Coplan, J. D., Friedman, S., Bassoff, T., Gorman, J., & Andrews, M. (1994). Adverse early experiences affect noradrenergic and serotonergic functioning in adult primates. *Biological Psychiatry*, **35**, 221–227.

Reid, J., Macchetto, P., & Foster, S. (1999, January). *No safe haven: Children of substance-abusing parents*. Report from the Center on Addiction and Substance Abuse, Columbia University.New York: CASA Publications.

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin*, **128**, 330–366.

Rosenblum, L. A., & Paully, G. S. (1984). The effects of varying environmental demands on maternal and infant behavior. *Child Development*, **55**, 305–314.

Russek, L. G., & Schwartz, G. E. (1997). Feelings of parental caring can predict health status in mid-life: A 35-year follow-up of the Harvard Mastery of Stress study. *Journal of Behavioral Medicine*, **20**, 1–13.

Seeman, T. E. (1996). Social ties and health: The benefits of social integration. *Annals of Epidemiology*, **6**, 442–451.

Seeman, T. E., McEwen, B. S., Rowe, J. W., & Singer, B. H. (2001). Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proceedings of the National Academy of Sciences of the United States of America*, **98**, 4770–4775.

Seeman, T. E., Singer, B., Horwitz, R., & McEwen, B. S. (1997). The price of adaptation—allostatic load and its health consequences: MacArthur studies of successful aging. *Archives of Internal Medicine*, **157**, 2259–2268.

Shedler, J., Mayman, M., & Manis, M. (1993). The illusion of mental health. *American Psychologist*, **48** (11), 1117–1131.

Suomi, S. J. (1999). Attachment in rhesus monkeys. In J. Cassidy & P. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 181–197). New York: Guilford Press.

Taylor, S. E., Lehman, B. J., Kiefe, C. I., & Seeman, T. E. (2003). The relation of early family environment to metabolic functioning in the CARDIA study. Manuscript in preparation.

Taylor, S. E., Sage, R. M., & Lerner, J. S. (2003). Pathways from early family environment to stress regulatory systems and subjective health. Manuscript submitted for publication.

Taylor, S. E., & Seeman, T. E. (2000). Psychosocial resources and the SES-health relationship. In N. Adler, M. Marmot, & B. McEwen (Eds.), *Socioeconomic status and health in industrial nations: Social, psychological, and biological pathways* (pp. 210–225). New York: New York Academy of Sciences.

Wahler, R. G. (1990). Some perceptual functions of social networks in coercive mother-child interactions. *Journal of Social and Clinical Psychology*, **9**, 43–53.

Walker, E. A., Gelfand, A., Katon, W. J., Koss, M. P., Von Korff, M., & Bernstein, D., et al. (1999). Adult health status of women with histories of childhood abuse and neglect. *The American Journal of Medicine*, **107**, 332–339.

Watson, D., & Pennebaker, J. W. (1989). Health complaints, stress, and distress: Exploring the central role of negative affectivity. *Psychological Review*, **96**, 234–254.

Widom, C. S., & White, H. R. (1997). Problem behaviours in abused and neglected children grown up: prevalence and co-occurrence of substance abuse, crime, and violence. *Criminal Behaviour and Mental Health*, **7**, 287–310.

Woodall, K. L., & Matthews, K. A. (1989). Familial environment associated with Type A behaviors and psychophysiological responses to stress in children. *Health Psychology*, **8**, 403–426.

Yehuda, R., Southwick, S. M., Krystal, J. H., & Bremne, D. (1993). Enhanced suppression of cortisol following dexamethasone administration in post-traumatic stress disorder. *American Journal of Psychiatry*, **150** (1), 83–86.