

## CHAPTER 3

# Family Relationships and Cortisol in Everyday Life

RICHARD B. SLATCHER

When asked to describe what comes to mind when hearing the word *family*, it is not uncommon for people to conjure up images of happy experiences, relaxing times, and a place of refuge. But in reality, time with family is not always so harmonious. Even in generally happy and well-adjusted families there can be arguments, yelling, crying, and other negative emotions. Everyday lives of families can be stressful for both parents and children. There are also outside forces that exert a daily push and pull on the family environment, including work, peer relationships, financial difficulties, and even traffic hassles. When these outside forces are brought home by family members in the form of bad moods, emotional withdrawal, impatience, and/or less time spent with family, the ways in which other family members react to these outside forces—and how the person bringing them home reacts to them—is critically important for the mental and physical health of family members. In the best of cases, relationships in the home can provide a restorative place that buffers or diffuses the effects of outside stressors. In other cases, relationships at home—between spouses, children, siblings, and parents—can be stressors themselves.

Research has shown that the type of social environment that family members come home to has important consequences for the body's stress physiology and, ultimately, physical health. In this chapter, I present an overview of the links between family relationships in everyday life and the production of cortisol, the primary stress hormone produced by the body. The chapter begins with a description of cortisol—what it is, how it can be measured in naturalistic settings, and its implications for physical health. I then review key findings linking marital relationships to cortisol production before turning to a discussion of how family relationships impact children's cortisol production. Throughout the chapter, I will emphasize key

findings from the past 5 years relating to associations between family relationships and cortisol that have not yet been reviewed elsewhere (to the author's knowledge). The final section of the chapter will cover cutting-edge work that is attempting to answer critical mechanistic questions of how family relationships "get under the skin" to affect cortisol, and, ultimately, physical health. The chapter will conclude with a preview of some of the work that our research group is conducting to tie together various aspects of links between family relationships, stress physiology, and health outcomes in order to clarify stress-health links.

## CORTISOL RESPONSES TO STRESS

How might the relationships that we have at home translate into between-person differences in how our immune systems operate, how often we get sick, and how long we live? In the 50 years since Selye's (1956) pioneering rat studies on the physiological affects of stress, scientists across a variety of disciplines have sought to understand the biological mechanisms through which stress adversely impacts physical health. The allostatic load model suggests that chronic strain leads to alterations in the body's regulatory "set points," speeding up eventual wear and tear (McEwen, 1998b). Frequent activation of the body's stress response that accompanies chronic stress can lead to greater "load" on a variety of organs and tissues, including the cardiovascular system. It is this overload on the body's various biological systems that is thought to lead to serious health problems if chronic stress is not reduced. Extending this model to everyday family life, it is believed that biological systems involved in the stress response are chronically activated in families in which there are consistently high stress levels (due to a variety of factors described later in this chapter). This chronic activation, in turn, results in greater allostatic load and poorer health outcomes among family members within and across generations (Kiecolt-Glaser & Newton, 2001; Miller, Chen, & Parker, 2011; Repetti, Robles, & Reynolds, 2011; Repetti, Wang, & Saxbe, 2011; Slatcher, 2010).

A key biological system implicated in allostatic load is the HPA (hypothalamic-pituitary-adrenal) axis, a complex cascade network in which the hypothalamus, pituitary gland, and adrenal glands work in concert to produce the stress hormone cortisol (ultimately secreted by the adrenal cortex). The HPA axis has attracted particular attention from researchers due to its sensitivity to psychological stress and its effects on multiple biological systems throughout the body. This system is present in organisms ranging from birds to humans and can be engaged by an array of psychological and physical stressors (McEwen, 1998a; McEwen & Stellar, 1993; Müller, Chen, & Zhou, 2007; Sapolsky, Romero, & Muncck, 2000). Activation of the HPA occurs when neurons in the paraventricular nucleus of the hypothalamus secrete corticotropin-releasing hormone (CRH). CRH then travels to the anterior pituitary gland, which responds to its presence by secreting a burst of adrenocorticotropin hormone (ACTH). ACTH then is carried through peripheral circulation

to the adrenal glands, which proceed to synthesize and release cortisol. It is hard to overstate the biological reach of cortisol, for which receptors exist in virtually every cell of the human body. Cortisol is a regulatory hormone, involved in learning, memory, and emotion in the central nervous system; regulation of glucose storage and utilization in the metabolic system, particularly in times of threat (the fight-or-flight response); and regulation of the magnitude and of the inflammatory response and maturation of lymphocytes in the immune system (Miller et al., 2007; Sapolsky et al., 2000).

Cortisol production has a diurnal rhythm, with levels peaking approximately 30 minutes after a person wakes, declining sharply in the morning hours, then slowly decreasing over the afternoon and evening to its nadir shortly before bedtime. Flattened diurnal cortisol slope (less decline in cortisol throughout the day) is thought to be a marker of a dysregulated stress response system because it indicates that people's stress hormones are remaining unusually high as the day progresses, thus contributing to greater allostatic load (McEwen, 1998a). Dysregulation of the HPA axis—including flatter cortisol slopes but also greater total cortisol output (assessed through area under the curve analysis, or AUC)—is associated with a number of poor health outcomes, including compromised immune functioning (Cohen et al., 2012), inflammation (DeSantis et al., 2012; Rueggeberg, Wrosch, Müller, & McDade, 2012), diabetes (Schoorlemmer, Peeters, van Schoor, & Lips, 2009), lung disease (Schoorlemmer et al., 2009), hippocampal atrophy (Sapolsky, 2000), and cardiovascular disease (Matthews, Schwartz, Cohen, & Seeman, 2006). Recent evidence suggests that flatter diurnal cortisol slopes especially are indicative of poorer future health, with links to mortality in two studies (Kumari, Shipley, Stafford, & Kivimaki, 2011; Sephton, Sapolsky, Kraemer, & Spiegel, 2000). In one study, flatter cortisol slopes were associated with lowered cell counts and activity of circulating natural killer cells and greater mortality among patients with metastatic breast cancer (Sephton et al., 2000). Further, in a prospective cohort analysis of 4,047 civil servants from the Whitehall II study (Kumari et al., 2011), flatter cortisol slopes predicted greater all-cause mortality and were especially predictive of an increased risk of cardiovascular death.

Given the association between HPA dysregulation and physical health problems, links between family relationships—which can be both stress inducing and stress buffering—and cortisol production have begun to receive increased attention from researchers. Although most cortisol researchers have investigated the effects of acute stressors on cortisol in lab settings (for a review, see Dickerson & Kemeny, 2004; Halpern, Campbell, Agnew, Thompson, & Udry, 2002), it is currently believed that chronic, naturalistic stressors have a more profound impact on physical health via the HPA than do acute stressors (Miller et al., 2007). Cortisol studies conducted in daily life are beginning to provide a picture of how everyday stressors impact cortisol production in situ and, unlike laboratory studies, allow for examination of how real-life stressors impact diurnal cortisol patterns. Field studies of links between everyday life and HPA axis functioning have become

more common since the introduction of salivary cortisol assays in the late 1980s. In these studies, participants generally provide between three and seven saliva samples per day (either by passively drooling into a tube for approximately 2 minutes or by chewing on a cotton swab, which then becomes soaked with saliva), usually over 1 to 7 days. The samples then either can be analyzed with commercially purchased assay kits or sent to a laboratory for analysis, which generally charges between \$5 and \$10 per sample. Although there is a learning curve for those who have not conducted salivary cortisol studies, the expense is quite low by biological research standards and collecting assays is relatively straightforward (for an excellent review and field guide to conducting salivary cortisol studies in everyday life, see Saxbe, 2008).

Understanding the specific psychological processes and events in everyday family life that lead to HPA axis dysregulation is key to clarifying how stress impacts physical health. Such an understanding adds to the allostatic load literature by defining the emotional, cognitive, and behavioral responses that family members have around one another when coping with the daily hassles and chronic stressors of daily life. While the allostatic load model takes into account the repeated "hits" on biological systems, the psychological literature has begun to outline what constitutes those hits (Repetti, Robles, et al., 2011). Next, I review the literature linking everyday family life to daily cortisol production, first focusing on how adults' cortisol levels are impacted by daily experiences in the home and then turning to how everyday family experiences impact children's production of cortisol.

#### EVERYDAY FAMILY ENVIRONMENTS AND ADULT CORTISOL

Social and emotional experiences in the family—particularly within the couple relationship—impact adults' daily cortisol production. For instance, couples' daily physical intimacy (including holding hands, touching, hugging, kissing, and sexual intercourse) is associated with lower cortisol production, and this link is mediated by greater levels of daily positive affect (Ditzen, Hoppmann, & Klumb, 2008). In a recent naturalistic study of 31 dual-earner married couples with children in the United States, wives who were more satisfied in their marriages had steeper (more "healthy") cortisol slopes throughout the day (Saxbe, Repetti, & Nishina, 2008). Further, marital satisfaction moderated the links between cortisol during the day (while women were at work) and cortisol at night; among wives high in marital satisfaction, the links between daily cortisol and nighttime cortisol were attenuated. These findings are in line with cross-sectional work demonstrating associations between aspects of marriage and daily cortisol patterns. Those studies have shown that less positive marital relationships and greater marital role concerns are linked to flatter diurnal cortisol slopes (Adam & Gunnar, 2001; Barnett, Steptoe, & Gareis, 2005), whereas greater marital satisfaction is linked to steeper cortisol slopes (Vedhara, Stra, Miles, Sanderman, & Ranchor, 2006).

The influence that family dynamics have on adult family members' HPA axis activity is not limited to the marital relationship. Stress related to household chores, for instance, has been shown to impact cortisol levels at home. In a recent study of dual-earner German couples, time devoted to housework was linked to greater cortisol production over the course of the day (Klumb, Hoppmann, & Staats, 2006). Housework, not surprisingly, also appears to be detrimental to unwinding after work: Husbands and wives who spend a higher percentage of their time at home on housework have higher evening cortisol levels and weaker declines in cortisol from afternoon to evening (Saxbe, Repetti, & Graesch, 2011). Interestingly, how people describe their homes—as either restorative or stressful—is linked to HPA axis functioning. In a recent study, spouses gave home tours to interviewers that were videotaped and then linguistically analyzed for words like "clutter," "unfinished," and "restful" (Saxbe & Repetti, 2010a). Over the 3 days following the home tours, wives with higher stressful home scores showed flatter cortisol slopes, whereas wives with higher restorative home scores had steeper cortisol slopes.

The effect of parenthood on cortisol functioning has received only limited attention, but studies suggest that from child delivery onward, having children impacts HPA functioning. In a study of women who recently gave birth, there were significant differences in postnatally depressed and postnatally nondepressed mothers: Those who were depressed showed a smaller morning rise in cortisol (CAR) than those not depressed (Taylor, Glover, Marks, & Kammerer, 2009), an effect similar to that seen in people reporting posttraumatic stress disorder. The mere presence of children in the home appears to attenuate recovery from daily stressors; among mothers of 2-year-olds, the greater the number of children in the home, the flatter the mother's cortisol slope (Adam & Gunnar, 2001). Similarly, working mothers have been shown to secrete greater cortisol over a 24-hour period than working women without children (Luecken et al., 1997).

Stress biology also is affected by the worries that people bring home with them from work. For instance, negative mood when people are at work is associated with day-to-day increases in angry marital behavior (Schulz, Cowan, Pape Cowan, & Brennan, 2004), and greater work-related stress is associated with less responsiveness to spouses and increased distraction after the work day ends (Story & Repetti, 2006). It is also the case that work stress impacts cortisol levels at home and that being in a highly satisfying marriage attenuates this effect (Saxbe et al., 2008). Further, increased work hours are linked to greater cortisol output over the course of the day—impacting both one's own and spouses' cortisol levels (Klumb et al., 2006).

When one comes home from work, it can be difficult to unwind. Even on the weekends, when we are spending time with family engaging in leisure pursuits, worries about work can sometimes stay with us. We recently investigated the physiological impact of work worries at home in a sample of dual-earner couples with young children (Slatcher, Robles, Repetti, & Fellows, 2010). Men's increases in work worries at home not only led to greater increases in their own cortisol levels (an actor effect) but also were linked to increases in their wives' cortisol levels (a partner

effect). Interestingly, wives' work worries impacted their own cortisol levels but had no effect on their husbands' cortisol levels. Why might husbands' work worries impact their wives' stress biology, but not vice versa? Previous laboratory findings suggest that wives are more biologically sensitive to changes in marital behaviors than husbands are (Robles & Kiecolt-Glaser, 2003; Smith, Gallo, Goble, Ngu, & Stark, 1998). This also is in line with more recent work showing that couples' cortisol levels covary (Saxbe & Repetti, 2010b).

In addition to studying how outside stressors impact spouses at home, we are investigating the aspects of marriage that may help buffer the effects of those stressors. One relationship process that we believe may be especially important is self-disclosure—the opening up to another person about one's thoughts, feelings, and emotions. It is now well known that self-disclosure has positive benefits for romantic relationships (Laurenceau, Barrett, & Rovine, 2005; Reis & Shaver, 1988; Slatcher & Pennebaker, 2006). Although there are a large number of experimental studies showing that self-disclosure through expressive writing is associated with better physical health (Frattaroli, 2006; Slatcher & Pennebaker, 2005) and stress biology (Smyth, Hockemeyer, & Tulloch, 2008), surprisingly few studies have investigated the effect of self-disclosure in one's close relationships—the context in which most self-disclosure naturally occurs—on health-related outcomes. Our research has shown that wives who report low levels of self-disclosure to their husbands show a much stronger association between the worries about work that they carry home with them and cortisol levels at home than those who are highly disclosing (Slatcher et al., 2010). Wives low in self-disclosure who also reported low levels of marital satisfaction showed especially strong links between work worries and cortisol levels. These findings suggest that self-disclosure may be a way in which women "inoculate" themselves against the physiological effects of work stress.

Having shown that family relationships impact adults' cortisol levels, researchers are now beginning to test the effectiveness of couples interventions on HPA axis activity. The results of these studies so far are mixed. In one study, married couples were randomly assigned to either a behavioral monitoring control group or participated in a 4-week "warm touch" support enhancement intervention (Holt-Lunstad, Birmingham, & Light, 2008). The intervention led to increases in salivary oxytocin (a hormone that promotes pair bonding) and reductions in salivary alpha amylase (an index of sympathetic nervous system activity), but no changes in cortisol. More recently, the effects of couples relationship education (CRE) on cortisol have been tested. CRE, which is geared toward improving positive communication and conflict management (Markman, Rhoades, Delaney, & White, 2010), is effective in boosting relationship quality and stability in married couples (Carroll & Doherty, 2003). Couples who go through a CRE intervention show significantly reduced cortisol responses during couple conflict discussions compared to preintervention levels (Ditzen, Hahlweg, Fehm-Wolfsdorf, & Baucom, 2011). These data suggest that improving relationship functioning through interventions may buffer the harmful effects of repeated conflict on stress physiology.

Although more research is still needed, the past decade has seen a growing number of studies investigating how family relationships impact adults' cortisol production in daily life. This work has shown that the quality of family relationships—especially couple relationships—impacts HPA functioning and can buffer the effects of outside stressors such as the worries about work that people bring home with them. However, the effects of family relationships on cortisol are not limited to adult family members. Next, I review findings suggesting that family relationships can have potent effects on children's cortisol levels as well.

## EVERYDAY FAMILY ENVIRONMENTS AND CHILD CORTISOL

Alterations of the HPA axis are considered a primary mechanism through which early-life experiences impact adult health (McEwen, 1998a; Repetti, Taylor, & Seeman, 2002). There have been a great number of studies examining the effects of stress on cortisol production in childhood, which have been exhaustively reviewed elsewhere (e.g., Gunnar & Quevedo, 2007). This review focuses specifically on family experiences and naturalistic assessments of cortisol in daily life throughout child development.

### Prenatal Development

The impact of stress on human stress physiology begins in the earliest stages of life. The effects of stress on mothers' cortisol levels in pregnancy appear to have important implications for the long-term health of the child. Low birth weight remains one of the greatest concerns of perinatal health, and maternal stress is linked to clinically meaningful reductions in birth weight (Maina et al., 2008). Glucocorticoids may be a common link between prenatal stressors, intrauterine growth, and birth weight (Seckl & Meaney, 2004), but the evidence so far is mixed. In one study, mothers' higher CARs were associated with lower birth weight, but mothers' distress was unrelated to cortisol or birth weight (Bolten et al., 2011). In another study, higher cortisol concentrations at awakening and throughout the day and flatter CARs were associated with shorter length of gestation; negative affect was associated with higher cortisol throughout the day, but not to gestational length. Although this type of work in humans is only just starting, a recent review provides suggestive evidence that prenatal stress or anxiety in the mother is associated with raised basal cortisol or raised cortisol reactivity in offspring, from infancy through adolescence (Glover, O'Connor, & O'Donnell, 2010).

### Infancy and Early Childhood

In the first year of life, external regulation of infants' arousal by primary caregivers is needed to protect infants from excessive stress. Caregivers who respond sensitively

to the infant help to regulate this arousal, providing the infant with a secure base (Bowlby, 1969). Insensitive or intrusive parenting behaviors are believed to be stressful for newborns because of their uncontrollable nature (Ainsworth, Blehar, Waters, & Wall, 1978). Among 3-month-olds, maternal sensitivity is associated with faster cortisol recovery from the mild everyday stress of being bathed (Albers, Riksen-Walraven, Sweep, & de Weerth, 2008). Additionally, infants of mothers experiencing parenting stress show higher daily cortisol output (Saridjan et al., 2010).

This pattern continues into the preschool years. Although relatively few studies have examined how everyday family behaviors impact young children's diurnal cortisol, research has shown that mothers who report higher maternal parenting quality have kindergarteners who show steeper ("healthier") cortisol slopes in daily life (Pendry & Adam, 2007). In a recent study, we investigated the links between everyday conflict at home—assessed with a naturalistic observation tool called the Electronically Activated Recorder (EAR)—and preschoolers' diurnal cortisol patterns (Slatcher & Robles, 2012). Typically, participants carry the EAR (in a pocket, purse, etc.) for 1–4 days, unobtrusively capturing 30- to 50-second audio snippets every 9–12 minutes (Mehl, Robbins, & Deters, 2012). Essentially, the EAR provides an acoustic window into people's daily behaviors. Preschoolers in our study wore a child EAR inside a "special magic shirt" with a pocket to hold the EAR that had different cartoon characters sewn onto it; this helped to build children's enthusiasm for wearing the EAR, while at the same time helping to protect the EAR and make it more unobtrusive. Greater EAR-assessed conflict at home with parents and siblings was linked to children having higher initial morning cortisol levels and flatter cortisol slopes across the day (e.g., less "healthy" diurnal cortisol rhythms). Although it is now commonplace for studies of adolescents to include momentary self-reports of daily behaviors (Conner, Tennen, Fleeson, & Barrett, 2009), the inability of young children to complete these type of reports (and the inherent biases of parent reports) make everyday studies of family life and early childhood health less common. Studying actual behaviors at home offers a potential way of getting around some of these stubborn methodological difficulties. Emerging technologies such as the EAR offer a fairly low-cost way of studying the effects of early family environments on HPA axis functioning (the most current version of the EAR is now iPod touch based). Toward the end of this chapter I describe a new study being conducted at Wayne State University that is using the EAR to examine how everyday family behaviors are linked to diurnal cortisol patterns and health outcomes in youth with asthma.

### Middle Childhood

The time of middle childhood is a period of relative calm in terms of HPA axis development (Gunnar & Quevedo, 2007), but the family continues to exert an influence on daily cortisol production. The pattern of findings generally is

consistent with what we see in earlier childhood, with recent stressful events and parents' lower marital satisfaction being associated with greater overall cortisol output and flatter diurnal slopes (Bevans, Cerbone, & Overstreet, 2008; Pendry & Adam, 2007; Wolf, Nicholls, & Chen, 2008). Much of the work with this age group has looked at the extreme end of adverse family environments, investigating the effects of maltreatment and neglect. Cicchetti and Rogosch (2001a, 2001b), for example, have reported that children who experienced multiple, severe, prolonged abuse had greater daily cortisol output. A major challenge in interpreting the existing literature is that it remains unclear whether altered cortisol levels are due to ongoing adversity or prior-experienced adversity. In well-known studies of children in Romanian orphanages characterized by extreme neglect, children still in the orphanages showed a lack of diurnal rhythm in cortisol production during the daytime hours (Carlson & Earls, 1997), whereas after an average of 6.5 years post adoption, children exhibited the normal expected decrease in cortisol from wakeup to bedtime (Gunnar, Morison, Chisholm, & Schuder, 2001). More recently, it was shown that preadoption deprivation among internationally adopted children—as reported by adoptive parents—was associated with higher morning cortisol levels and larger diurnal cortisol decreases, but only among those children who had physical growth delays (e.g., shorter than expected height).

Current negative experiences at home also affect daily cortisol production at this age. These effects appear to be strongest during the evening, when cortisol levels are typically at their lowest and when children are at home with their families. Living with chronic stress at home (Bevans et al., 2008; Schreier & Evans, 2003; Wolf et al., 2008), marital dysfunction (Pendry & Adam, 2007), and family strife, crowding, and noise (Evans, 2003; Evans & Kim, 2007) all are associated with higher evening cortisol. A few recent studies have investigated the long-term effects of home-life adversity, showing that maltreatment in childhood is associated with greater daily cortisol output among adult women with chronic pain (Nicolson, Davis, Kruszewski, & Zautra, 2010), while low parental care is associated with increased CAR and increased afternoon/evening cortisol levels in healthy men and women (Engert, Efanov, Dedovic, Dagher, & Pruessner, 2011). Recent intervention research is encouraging, showing, for example, that atypical diurnal cortisol patterns of children in foster care can be altered—becoming comparable to nonfoster children—following a family-based treatment intervention (Fisher, Stoolmiller, Gunnar, & Burraston, 2007), and a poverty-alleviation intervention has led to reductions in daily cortisol production among low-income children with depressed mothers in Mexico (Fernald & Gunnar, 2009).

### Adolescence

Adolescence is a critical time to study links between family environment and HPA axis activity, as transitions into early, middle, and late adolescence are each

characterized by significant changes in family contexts (Graber, Brooks-Gunn, & Petersen, 1996; Lerner, 2002). The quality of youths' family environments both shapes and is shaped by these transitions, with important implications for the HPA axis and, more broadly, for physical health (Holmbeck et al., 2010; Poulin & Heckhausen, 2007). For example, transitions from elementary to middle school and from middle to high school are each associated with greater family conflict, particularly among high-stress families (Gutman & Eccles, 2007; Herrenkohl et al., 2010; Obradović & Hipwell, 2010; Seidman, Lambert, Allen, & Aber, 2003). Within families, parental monitoring shifts as youth assert greater behavioral autonomy (Barber, Stolz, & Olsen, 2005; Darling, Cumsille, & Martinez, 2008). Links between low family support, decreased monitoring, and precocious behavioral autonomy suggest that family environments may be crucial to supporting youths' behavioral autonomy in ways that influence health-related outcomes (Dishion, Poulin, & Skaggs, 2000; Laird, Criss, Pettit, Dodge, & Bates, 2008; Ryan, Deci, Grohnick, & La Guardia, 2006).

HPA axis activity increases substantially during adolescence (Gunnar & Quevedo, 2007; Oskis, Loveday, Hucklebridge, Thorn, & Clow, 2009). Although fewer studies of family effects on daily cortisol have been conducted in adolescence than in earlier developmental periods, the current evidence suggests that parental warmth has an inverse, linear association with basal cortisol levels (Marsman et al., 2012). Further, adolescents living in homes where mothers and their partners report poorer marital functioning have significantly higher average cortisol levels than adolescents in homes where parents report higher marital functioning (Pendry & Adam, 2007). There also appears to be significant synchrony between parent and child cortisol at this age. In a recent study (Papp, Pendry, & Adam, 2009), family members' cortisol levels were measured seven times a day on 2 typical weekdays. After accounting for the effects of time of day and relevant demographic and health control variables on cortisol levels, multilevel modeling indicated the presence of significant covariation over time in mother-adolescent cortisol. Importantly, mother-adolescent cortisol synchrony was strengthened among dyads characterized by mothers and adolescents spending more time together, and in families rated higher on levels of parent-youth shared activities and parental monitoring or supervision.

In a new study funded by the National Institutes of Health (NIH), we are using the EAR (described previously) to gain a fly-on-the-wall perspective on how everyday family interactions impact youth health via their effects on the HPA axis. This project, Asthma in the Lives of Families Today (ALOFT), takes a multimethod biosychosocial approach by investigating the links between everyday family behaviors and asthma morbidity in a sample of 180 youth aged 10-15 years in Detroit, Michigan. With the ALOFT study, we are collecting longitudinal data over three waves in a 2-year period, having participants in the study wear EAR, and collecting parent and child daily diaries of family functioning, biological measures (including diurnal cortisol, gene expression, and epigenetic markers), and clinical asthma

evaluations. Asthma is a chronic inflammatory disease of the respiratory tract that stems from a complex interaction of environmental, psychological, and genetic factors (Howard, Meyers, & Bleecker, 2003). Because asthma symptoms are modulated by cortisol—not only cortisol produced by the body but also synthetic cortisol administered via oral steroids (e.g., prednisone) to control asthma—scientists are beginning to look to asthma as a target disease for understanding how stress “gets under the skin” to affect health. Next, I conclude this chapter with a discussion of the biological mechanisms through which cortisol dysregulation stemming from family-related stressors may ultimately impact physical health and how these are being tested with the ALOFT study.

### HOW DO THE EFFECTS OF FAMILY ENVIRONMENTS ON CORTISOL ULTIMATELY IMPACT HEALTH?

The biological mechanisms through which families exert their influence on physical health via cortisol remain poorly understood. Among cortisol's physiological functions is modulation of the immune system—its primary role in this regard is to suppress inflammation. Based on this role, one would predict that the high levels of cortisol that accompany stress would lead to a dampening of inflammation in inflammatory diseases such as asthma, similar to what occurs when corticosteroids are exogenously administered (e.g., when one applies hydrocortisone to an itchy bug bite or takes prednisone for more serious inflammation). In reality, empirical evidence suggests just the opposite: Stress *exacerbates* inflammation (Miller, Rohleder, & Cole, 2009; Pervanidou, Margeli, Lazaropoulou, Pappasotiropou, & Chrousos, 2008).

Over the past decade, Miller and colleagues (Miller, Cohen, & Ritchey, 2002) have proposed a theory of glucocorticoid resistance (immune system resistance to the regulatory effects of cortisol) that offers a plausible explanation for why chronic stress is linked to both high levels of circulating cortisol and high levels of inflammation. The basic idea behind the glucocorticoid resistance model is that chronic stress interferes with the immune system's responsiveness to cortisol, which normally should dampen the body's inflammatory immune response. In support of this idea, new evidence shows that the persistent secretion of cortisol associated with chronic stress leads to a downregulation of glucocorticoid receptor expression and functioning (Cohen et al., 2012; Miller & Chen, 2006; Miller et al., 2002; Miller, Gaudin, Zysk, & Chen, 2009). Among those with chronic inflammatory diseases (which include diseases such as asthma, arthritis, and cardiovascular disease), this downregulation can lead to increased inflammation and, in the case of asthma, reduced efficacy of glucocorticoid therapy (including both inhaled and oral steroids)—a mainstay of asthma treatment (Lee, Brattsand, & Leung, 1996). For example, among children with asthma, high levels of chronic family stress are associated with a 5.5-fold reduction in leukocyte (white blood cell) glucocorticoid

receptor mRNA. This indicates that immune cells' receptors—which normally respond to cortisol signals by turning “off” inflammatory immune processes—are not being produced in sufficient numbers to work as they should (Miller & Chen, 2006). This is akin to a strong radio signal being sent out from a radio station (the body producing large amounts of cortisol under stress) but having very few receivers in people's homes (the glucocorticoid receptors in immune cells) to pick up the signal. There are functional implications for these cellular processes that drive asthma: peripheral blood mononuclear cells (PBMCs) harvested from asthmatic children with greater family stress are more resistant to hydrocortisone's effects on pro-inflammatory cytokine expression (e.g., IL-5 and IF- $\gamma$ ) relative to asthmatic patients reporting lower family stress (Miller, Gaudin, et al., 2009).

The possibility that epigenetic changes may play a contributing role to the development of inflammation has received significant attention in the past few years from human and animal researchers (Bjornsson et al., 2008; McGowan et al., 2009; Weaver et al., 2005). In classic terms, epigenetics refers to the heritable, but reversible, regulation of genetic functions. A major mechanism of epigenetic regulation that has been identified is DNA methylation. DNA methylation is a modification of DNA in which methyl groups are added to certain positions on the nitrogen bases, typically leading to the inhibiting or “silencing” of gene expression. With the ALOFT study, we are focusing on methylation of the glucocorticoid receptor gene NR3C1, a gene responsible for gene expression of the glucocorticoid receptors in immune cells (e.g., the “radio receivers” described previously). Recent work has shown that DNA methylation profiles can change over time (Bjornsson et al., 2008) and that such profiles can differ in monozygotic twins (Fraga et al., 2005). The relative instability of these epigenetic signatures makes them attractive candidates for studying the underlying mechanisms of complex disease such as asthma. Recent studies suggest that epigenetic regulation may in part mediate the complex gene-environment interactions that can lead to disease (Miller & Ho, 2008). These exciting new findings point to potential biological pathways through which family stress exacerbates physical health problems. The ALOFT study is investigating whether stressful family relationships foster epigenetic changes in immune cells—specifically hypermethylation (greater methylation) of glucocorticoid receptor promoter sites—and gene expression (e.g., reduced glucocorticoid receptor expression) that, in turn, explain how the tonically higher levels of cortisol associated with chronic stress may begin to shut down the immune system's “normal” response to cortisol, thus leading to greater incidence of inflammatory diseases such as asthma, cardiovascular disease, and arthritis.

## CONCLUSION

The number of naturalistic studies of family relationships and cortisol is only beginning to catch up to the decades of cortisol studies conducted in the lab. However,

the studies reviewed in this chapter hint at the huge strides that have been made in investigating how family interactions are linked to HPA axis functioning in daily life. Although this type of work can be challenging in terms of expense and both researcher and participant burden, the knowledge gained has led to a number of critical advances in our understanding of family relationships, stress, and health. On the psychological side, emerging technologies such as EAR are allowing us access to the inner workings of everyday family life that was never before possible. On the health side, advances in the biological sciences and a growing “team science” approach is bringing scientists from a variety of disciplines together to understand the effects of psychological factors on HPA functioning at a molecular level. What is most exciting is not what we have learned about these processes over the past 10 years, but what is still left to be learned about how family relationships “get under the skin” to impact physical health.

## REFERENCES

- Adam, E. K., & Gunnar, M. R. (2001). Relationship functioning and home and work demands predict individual differences in diurnal cortisol patterns in women. *Psychoneuroendocrinology, 26*, 189–208.
- Ainsworth, M., Blehar, M., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale, NJ: Erlbaum.
- Albers, E. M., Riksen-Walraven, J. M., Sweep, F. C. G. J., & Weerth, C. (2008). Maternal behavior predicts infant cortisol recovery from a mild everyday stressor. *Journal of Child Psychology and Psychiatry, 49*, 97–103.
- Barber, B. K., Stolz, H. E., & Olsen, J. A. (2005). Parental support, psychological control, and behavioral control: Assessing relevance across time, culture, and method. *Monographs of the Society for Research in Child Development, 70*, 1–137.
- Barnett, R. C., Steptoe, A., & Gareis, K. C. (2005). Marital-role quality and stress-related psychobiological indicators. *Annals of Behavioral Medicine, 30*, 36–43.
- Bevas, K., Cerbone, A., & Overstreet, S. (2008). Relations between recurrent trauma exposure and recent life stress and salivary cortisol among children. *Development and Psychopathology, 20*, 257–272.
- Bjornsson, H. T., Sigurdsson, M. I., Fallin, M. D., Itzary, R. A., Aspelund, T., Cui, H., . . . Feinberg, A. P. (2008). Intra-individual change over time in DNA methylation with familial clustering. *Journal of the American Medical Association, 299*, 2877–2883.
- Bolten, M. I., Wurmser, H., Buske-Kirschbaum, A., Papoušek, M., Pirke, K.-M., & Hellhammer, D. (2011). Cortisol levels in pregnancy as a psychobiological predictor for birth weight. *Archives of Women's Mental Health, 14*, 33–41.
- Bowlby, J. (1969). *Attachment*. New York, NY: Basic Books.
- Carlson, M., & Earls, F. (1997). Psychological and neuroendocrinological sequelae of early social deprivation in institutionalized children in Romania. *Annals of the New York Academy of Sciences, 807*, 419–428.
- Carroll, J. S., & Doherty, W. J. (2003). Evaluating the effectiveness of premarital prevention programs: A meta-analytic review of outcome research. *Family Relations, 52*, 105–113.
- Cicchetti, D., & Rogosch, F. A. (2001a). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology, 13*, 677–693.

- Cicchetti, D., & Rogosch, F. A. (2001b). The impact of child maltreatment and psychopathology on neuroendocrine functioning. *Development and Psychopathology, 13*, 783-804.
- Cohen, S., Janicki-Deverts, D., Doyle, W. J., Miller, G. E., Frank, E., Rabin, B. S., & Turner, R. B. (2012). Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk. *Proceedings of the National Academy of Sciences USA, 109*, S995-S999.
- Conner, T. S., Tennen, H., Fleeson, W., & Barrett, L. F. (2009). Experience sampling methods: A modern idiographic approach to personality research. *Social and Personality Psychology Compass, 3*, 292-313. doi: 10.1111/j.1751-9004.2009.00170.x
- Darling, N., Cumsille, P., & Martinez, M. L. (2008). Individual differences in adolescents' beliefs about the legitimacy of parental authority and their own obligation to obey: A longitudinal investigation. *Child Development, 79*, 1103-1118.
- DeSantis, A. S., DiezRoux, A. V., Hajjat, A., Aiello, A. E., Golden, S. H., Jenny, N. S., ... Shea, S. (2012). Associations of salivary cortisol levels with inflammatory markers: The Multi-Ethnic Study of Atherosclerosis. *Psychoneuroendocrinology, 37*, 1009-1018.
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130*, 355-391.
- Dishion, T. J., Poulin, F., & Skaggs, N. M. (2000). The ecology of premature autonomy in adolescence: Biological and social influences. In K. Kerns, J. Contreras, & A. Neal-Barnett (Eds.), *Family and peers: Linking two social worlds* (pp. 27-45). Westport, CT: Praeger/Greenwood.
- Ditzen, B., Hahlweg, K., Fehm-Wolfsdorf, G., & Baucom, D. (2011). Assisting couples to develop healthy relationships: Effects of couples relationship education on cortisol. *Psychoneuroendocrinology, 36*, 597-607.
- Ditzen, B., Hoppmann, C., & Klumb, P. (2008). Positive couple interactions and daily cortisol: On the stress-protecting role of intimacy. *Psychosomatic Medicine, 70*, 883-889.
- Engert, V., Efanov, S. I., Dedovic, K., Dagher, A., & Pruessner, J. C. (2011). Increased cortisol awakening response and afternoon/evening cortisol output in healthy young adults with low early life parental care. *Psychopharmacology, 214*, 261-268.
- Evans, G. W. (2003). A methodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology, 39*, 924.
- Evans, G. W., & Kim, P. (2007). Childhood poverty and health-Cumulative risk exposure and stress dysregulation. *Psychological Science, 18*, 953-957.
- Fernald, L. C. H., & Gunnar, M. R. (2009). Poverty-alleviation program participation and salivary cortisol in very low-income children. *Social Science and Medicine, 68*, 2180-2189.
- Fisher, P. A., Stoolmiller, M., Gunnar, M. R., & Burraston, B. O. (2007). Effects of a therapeutic intervention for foster preschoolers on diurnal cortisol activity. *Psychoneuroendocrinology, 32*, 892-905. doi: 10.1016/j.psypneuen.2007.06.008.
- Fraga, M., Ballestar, E., Paz, M., Ropero, S., Setien, F., Ballestar, M., ... Esteller, M. (2005). Epigenetic differences arise during the lifetime of monozygotic twins. *Proceedings of the National Academy of Sciences USA, 102*, 10604-10609.
- Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin, 132*, 823-865.
- Glover, V., O'Connor, T. G., & O'Donnell, K. (2010). Prenatal stress and the programming of the HPA axis. *Neuroscience and Biobehavioral Reviews, 35*, 17-22.
- Graber, J., Brooks-Gunn, J., & Petersen, A. C. (Eds.). (1996). *Transitions through adolescence: Interpersonal domains and context*. Hillsdale, NJ: Erlbaum.
- Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology, 58*, 145-173. doi: 10.1146/annurevpsych.58.110405.085605.
- Gunnar, M. R., Morison, S. J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Development and Psychopathology, 13*, 611-628.
- Gutman, L. M., & Eccles, J. S. (2007). Stage-environment fit during adolescence: Trajectories of family relations and adolescent outcomes. *Developmental Psychology, 43*, S22-S37.
- Halpern, C. T., Campbell, B., Agnew, C. R., Thompson, V., & Udry, J. R. (2002). Associations between stress reactivity and sexual and nonsexual risk taking in young adult human males. *Hormones and Behavior, 42*, 387-398.
- Herrenkohl, T. I., Kosterman, R., Mason, W. A., Hawkins, J. D., McCarty, C. A., & McCauley, E. (2010). Effects of childhood conduct problems and family adversity on health, health behaviors, and service use in early adulthood: Tests of developmental pathways involving adolescent risk taking and depression. *Development and Psychopathology. Special Issue: Developmental Cascades: Part 1, 22*, 655-665.
- Holmbeck, G. N., DeLucia, C., Essner, B., Kelly, L., Zembracki, K., Friedman, D., & Jandasek, B. (2010). Trajectories of psychosocial adjustment in adolescents with spina bifida: A 6-year, four-wave longitudinal follow-up. *Journal of Consulting and Clinical Psychology, 78*, 511-525.
- Holt-Lunstad, J., Birmingham, W. A., & Light, K. C. (2008). Influence of a "warm touch" support enhancement intervention among married couples on ambulatory blood pressure, oxytocin, alpha amylase, and cortisol. *Psychosomatic Medicine, 70*, 976-985. doi: 10.1097/PSY.0b013e318187ae7f.
- Howard, T., Meyers, D., & Bleecker, E. (2003). Mapping susceptibility genes from allergic diseases. *Chest, 123*, 365S-368S.
- Kiecolt-Glaser, J. K., & Newton, T. L. (2001). Marriage and health: His and hers. *Psychological Bulletin, 127*, 472-503.
- Klumb, P., Hoppmann, C., & Staats, M. (2006). Work hours affect spouse's cortisol secretion: For better and for worse. *Psychosomatic Medicine, 68*, 742-746.
- Kumari, M., Shipley, M., Stafford, M., & Kivimaki, M. (2011). Association of diurnal patterns in salivary cortisol with all-cause and cardiovascular mortality: Findings from the Whitehall II study. *Journal of Clinical Endocrinology and Metabolism, 96*, 1478-1485. doi: 10.1210/jc.2010-2137.
- Laird, R. D., Criss, M. M., Pettit, G. S., Dodge, K. A., & Bates, J. E. (2008). Parents' monitoring knowledge attenuates the link between antisocial friends and adolescent delinquent behavior. *Journal of Abnormal Child Psychology, 36*, 299-310.
- Laurenceau, J. P., Barrett, L. F., & Rovine, M. J. (2005). The interpersonal process model of intimacy in marriage: A daily-diary and multilevel modeling approach. *Journal of Family Psychology, 19*, 314.
- Lee, T. H., Brattsand, R., & Leung, D. Y. M. (1996). Corticosteroid action and resistance in asthma. *American Journal of Respiratory Cell and Molecular Biology, 154* (Suppl), S1-S79.
- Lerner, R. M. (2002). *Adolescence: Development, diversity, context, and application*. Upper Saddle River, NJ: Prentice Hall/Pearson Education.
- Luecken, L. J., Suarez, E. C., Kuhn, C. M., Barefoot, J. C., Blumenthal, J. A., Siegler, I. C., et al. (1997). Stress in employed women: Impact of marital status and children at home on neurohormone output and home strain. *Psychosomatic Medicine, 59*, 352-359.
- Maina, G., Saracco, P., Gholito, M. R., Danelon, D., Bogetto, F., & Todros, T. (2008). Impact of maternal psychological distress on fetal weight, prematurity and intrauterine growth retardation. *Journal of Affective Disorders, 111*, 214-220.
- Markman, H. J., Rhoades, G., Delaney, R., & White, L. (2010). Extending the reach of research-based couples interventions: The role of relationship education. In K. Hahlweg, M. Grawe-Gerber & D. H. Baucom (Eds.), *Enhancing couples: The shape of couple therapy to come* (pp. 128-141). Göttingen, Germany: Hogrefe.
- Marsman, R., Nederhof, E., Rosmalen, J. G. M., Oldehinkel, A. J., Ormel, J., & Buitelaar, J. K. (2012). Family environment is associated with HPA-axis activity in adolescents: The TRAILS study. *Biological Psychology, 89*, 460-466.



Pervanidou, P., Margeli, A., Lazaropoulou, C., Papassotiou, I., & Chrousos, G. P. (2008). The immediate and long-term impact of physical and/or emotional stress from motor vehicle accidents on circulating stress hormones and adipo-cytokines in children and adolescents. *Stress: The International Journal on the Biology of Stress*, *11*, 438-447.

Poulin, M. J., & Heckhausen, J. (2007). Stressful events compromise control strivings during a major life transition. *Motivation and Emotion*, *31*, 300-311.

Reis, H. T., & Shaver, P. (1988). Intimacy as an interpersonal process. In S. Duck (Ed.), *Handbook of personal relationships* (pp. 367-389). Chichester, UK: Wiley.

Repetti, R. L., Robles, T. F., & Reynolds, B. M. (2011). Allostatic processes in the family. *Development and Psychopathology*, *23*, 921-938.

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. doi: 10.1037/0033-2909.128.2.330.

Repetti, R. L., Wang, S.-W., & Saxbe, D. E. (2011). Adult health in the context of everyday family life. *Annals of Behavioral Medicine*, *42*, 285-293.

Robles, T. F., & Kiecolt-Glaser, J. K. (2003). The physiology of marriage: Pathways to health. *Psychology and Behavior*, *79*, 409-416.

Rueggeberg, R., Wrosch, C., Miller, G. E., & McDade, T. W. (2012). Associations between health-related self-protection, diurnal cortisol, and C-reactive protein in lonely older adults. *Psychosomatic Medicine*, *74*, 937-944. doi: 10.1097/PSY.0b013e3182732dc6.

Ryan, R. M., Deci, E. L., Grolnick, W. S., & La Guardia, J. G. (2006). The significance of autonomy and autonomy support in psychological development and psychopathology. In D. Cicchetti & D. Cohen (Eds.), *Developmental psychopathology, Vol 1. Theory and method* (2nd ed., pp. 795-849). Hoboken, NJ: Wiley.

Sapolsky, R. M. (2000). Glucocorticoids and hippocampal atrophy in neuropsychiatric disorders. *Archives of General Psychiatry*, *57*, 925-935.

Sapolsky, R. M., Romero, M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. *Endocrine Reviews*, *21*, 55-89.

Sardjian, N. S., Huizink, A. C., Koetsier, J. A., Jaddoe, V. W., Mackenbach, J. P., Hofman, A., ... Tiemeir, H. (2010). Do social disadvantage and early family adversity affect the diurnal cortisol rhythm in infants? The Generation R study. *Hormones and Behavior*, *57*, 247-254.

Saxbe, D. E. (2008). A field (researcher's) guide to cortisol: Tracking HPA axis functioning in everyday life. *Health Psychology Review*, *2*, 163-190.

Saxbe, D. E., & Repetti, R. (2010a). No place like home: Home tours correlate with daily patterns of mood and cortisol. *Personality and Social Psychology Bulletin*, *36*, 71-81.

Saxbe, D. E., & Repetti, R. L. (2010b). For better or worse? Coregulation of couples' cortisol levels and mood states. *Journal of Personality and Social Psychology*, *98*, 92-103.

Saxbe, D. E., Repetti, R. L., & Graesch, A. P. (2011). Time spent in housework and leisure: Links with parents' physiological recovery from work. *Journal of Family Psychology*, *25*, 271-281.

Saxbe, D. E., Repetti, R. L., & Nishina, A. (2008). Marital satisfaction, recovery from work, and diurnal cortisol among men and women. *Health Psychology*, *27*, 15-25. doi: 10.1037/0278-6133.27.1.15.

Schoorlemmer, R. M., Peeters, G. M., van Schoor, N. M., & Lips, P. (2009). Relationships between cortisol level, mortality and chronic diseases in older persons. *Clinical Endocrinology*, *71*, 779-786. doi: 10.1111/j.1365-2265.2009.03552.x.

Schreier, A., & Evans, G. W. (2003). Adrenal cortical response of young children to modern and ancient stressors. *Current Anthropology*, *44*, 306-309.

Matthews, K., Schwartz, J., Cohen, S., & Seeman, T. (2006). Diurnal cortisol decline is related to coronary calcification: CARDIA study. *Psychosomatic Medicine*, *68*, 657-661. doi: 10.1097/01.psy.000244071.42939.0e.

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

McEwen, B. S. (1998b). Stress, adaptation and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, *840*, 33-44.

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

McGowan, P. O., Sasaki, A., D'Alessio, A. C., Dymov, S., Labonté, B., Szyf, M., ... Meaney, M. J. (2009). Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. *Nature Neuroscience*, *12*, 342-348.

Mehl, M. R., Robbins, M. L., & Deters, F. (2012). Naturalistic observation of health-relevant social processes: The electronically activated recorder (EAR) methodology in psychosomatics. *Psychosomatic Medicine*, *74*, 410-471.

Miller, G. E., & Chen, E. (2006). Stressful experience and diminished expression of genes encoding the glucocorticoid receptor and b2-adrenergic receptor in children with asthma. *Proceedings of the National Academy of Sciences USA*, *103*, S496-S501.

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin*, *137*, 959-997.

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin*, *133*, 25-45.

Miller, G. E., Cohen, S., & Ritchey, A. K. (2002). Chronic psychological stress and the regulation of pro-inflammatory cytokines: A glucocorticoid-resistance model. *Health Psychology*, *21*, S31-S41.

Miller, G. E., Gaudin, A., Zysk, E., & Chen, E. (2009). Parental support and cytokine activity in childhood asthma: The role of glucocorticoid sensitivity. *Journal of Allergy and Clinical Immunology*, *123*, 824-830.

Miller, G. E., Rohleder, N., & Cole, S. W. (2009). Chronic interpersonal stress predicts activation of pro- and anti-inflammatory signaling pathways 6 months later. *Psychosomatic Medicine*, *71*, 57-62.

Miller, R., & Ho, S. (2008). Environmental epigenetics and asthma: Current concepts and call for studies. *American Journal of Respiratory and Critical Care Medicine*, *177*, S67-S73.

Nicolson, N. A., Davis, M. C., Kruzewski, D., & Zutra, A. J. (2010). Childhood maltreatment and diurnal cortisol patterns in women with chronic pain. *Psychosomatic Medicine*, *72*, 471-480.

Obradović, J., & Hipwell, A. (2010). Psychopathology and social competence during the transition to adolescence: The role of family adversity and pubertal development. *Development and Psychopathology Special Issue: Developmental Cascades: Part 1*, *22*, 621-634.

Oskis, A., Loveday, C., Hucklebridge, F., Thorn, L., & Clow, A. (2009). Diurnal patterns of salivary cortisol across the adolescent period in healthy females. *Psychoneuroendocrinology*, *34*, 307-316.

Papp, L. M., Pendry, P., & Adam, E. K. (2009). Mother-adolescent physiological synchrony in naturalistic settings: Within-family cortisol associations and moderators. *Journal of Family Psychology*, *23*, 882-894.

Pendry, P., & Adam, E. K. (2007). Associations between parents' marital functioning, maternal parenting quality, maternal emotion and child cortisol levels. *International Journal of Behavioral Development*, *31*, 218-231. doi: 10.1177/0165025407074634.

- Schulz, M. S., Cowan, P. A., Pope Cowan, C., & Brennan, R. T. (2004). Coming home upset: Gender, marital satisfaction, and the daily spillover of workday experience into couple interactions. *Journal of Family Psychology, 18*, 250-263.
- Seckl, J. R., & Meaney, M. J. (2004). *Glucocorticoid programming. In Biobehavioral stress response: Protective and damaging effects* (pp. 63-84). New York, NY: New York Academy of Sciences.
- Seidman, E., Lambert, L. E., Allen, L., & Aber, J. L. (2003). Urban adolescents' transition to junior high school and protective family transactions. *Journal of Early Adolescence, 23*, 166-193.
- Selye, H. (1956). *The stress of life*. New York, NY: McGraw-Hill.
- Sephton, S. E., Sapolsky, R. M., Kraemer, H. C., & Spiegel, D. (2000). Diurnal cortisol rhythm as a predictor of breast cancer survival. *Journal of the National Cancer Institute, 92*, 994-1000. doi: 10.1093/jnci/92.12.994.
- Slatcher, R. B. (2010). Marital functioning and physical health: Implications for social and personality psychology. *Social and Personality Psychology Compass, 4*, 455-469. doi: 10.1111/j.1751-9004.2010.00273.x.
- Slatcher, R. B., & Pennebaker, J. W. (2005). Emotional processing of traumatic events. In C. L. Cooper (Ed.), *The handbook of stress medicine and health* (pp. 293-307). Boca Raton, FL: CRC Press.
- Slatcher, R. B., & Pennebaker, J. W. (2006). How do I love thee? Let me count the words: The social effects of expressive writing. *Psychological Science, 17*, 660-664.
- Slatcher, R. B., & Robles, T. F. (2012). Preschoolers' everyday conflict at home and diurnal cortisol patterns. *Health Psychology, 31*, 834-838.
- Slatcher, R. B., Robles, T. F., Repetti, R., & Fellows, M. D. (2010). Momentary work worries, marital disclosure and salivary cortisol among parents of young children. *Psychosomatic Medicine, 72*, 887-896.
- Smith, T. W., Gallo, L. C., Goble, L., Ngu, L. Q., & Stark, K. A. (1998). Agency, communion, and cardiovascular reactivity during marital interaction. *Health Psychology, 17*, 537-545.
- Smyth, J. M., Hockemeyer, J. R., & Tulloch, H. (2008). Expressive writing and post-traumatic stress disorder: Effects on trauma symptoms, mood states, and cortisol reactivity. *British Journal of Health Psychology, 13*, 85-93. doi: 10.1348/135910707x250866.
- Story, L. B., & Repetti, R. (2006). Daily occupational stressors and marital behavior. *Journal of Family Psychology, 20*, 690-700.
- Taylor, A., Glover, V., Marks, M., & Kammerer, M. (2009). Diurnal pattern of cortisol output in postnatal depression. *Psychoneuroendocrinology, 34*, 1184-1188.
- Vedhara, K., Stra, J. T., Miles, J. N. V., Sanderman, R., & Ranchor, A. V. (2006). Psychosocial factors associated with indices of cortisol production in women with breast cancer and controls. *Psychoneuroendocrinology, 31*, 299-311.
- Weaver, I. C. G., Champagne, F. A., Brown, S. E., Dymov, S., Sharma, S., Meaney, M. J., ... Szyf, M. (2005). Reversal of maternal programming of stress responses in adult offspring through methyl supplementation: Altering epigenetic marking later in life. *Journal of Neuroscience, 25*, 11045-11054.
- Wolf, J. M., Nicholls, E., & Chen, E. (2008). Chronic stress, salivary cortisol, and alpha-amylase in children with asthma and healthy children. *Biological Psychology, 78*, 20-28. doi: 10.1016/j.biopsycho.2007.12.004.