



**California Center for Population Research**  
**University of California - Los Angeles**

# **Social Conflict and Cortisol Regulation**

Esther M. Friedman  
Arun Karlamangla  
David M. Almeida  
Teresa E. Seeman

**PWP-CCPR-2010-010**

**August 2010**

***California Center for Population Research  
On-Line Working Paper Series***

## Social Conflict and Cortisol Regulation

Esther M. Friedman<sup>a</sup>, Arun Karlamangla<sup>a</sup>, David M. Almeida<sup>b</sup>, Teresa E. Seeman<sup>a</sup>

<sup>a</sup>University of California, Los Angeles

<sup>b</sup>Pennsylvania State University

August 10, 2010

This research was supported, in part, by Grant P01-AG020166 from the National Institute on Aging to conduct a longitudinal follow-up of the MIDUS (Midlife in the United States) investigation. The original study was supported by the John D. and Catherine T. MacArthur Foundation Research Network on Successful Midlife Development. Support was also provided to the first author by the UCLA Interdisciplinary Relationship Science Program sponsored by the National Science Foundation. We are grateful to Rob Mare, Judy Seltzer, and the members of the UCLA demography, family, and stratification research group for helpful advice as we developed the paper. A previous version of this paper was presented at the 2008 meeting of the Gerontological Society of America (GSA), National Harbor, MD. Please direct all correspondence to Esther M. Friedman, Department of Sociology, University of California – Los Angeles, 264 Haines Hall, Los Angeles, CA 90095-1551, USA; email: efriedman@ucla.edu.

## **ABSTRACT**

Chronic stress has been implicated in a variety of adverse health outcomes, from compromised immunity to cardiovascular disease to cognitive decline. The hypothalamic pituitary adrenal (HPA) axis has been postulated to play the primary biological role in translating chronic stress into ill health. Stressful stimuli activate the HPA axis and cause an increase in circulating levels of cortisol. Frequent and long-lasting activation of the HPA axis, as occurs in recurrently stressful environments, can in the long run, compromise HPA axis functioning and ultimately affect health. Negative social interactions with family and friends may be a significant source of stress in daily life, constituting the type of recurrently stressful environment that could lead to compromised HPA functioning and altered diurnal cortisol rhythms. We use data from two waves of the Midlife in the U.S. Survey to explore the diurnal cortisol rhythm and its relationship with histories of social conflict. We find that for both men and women, reported levels of social conflict were significantly associated with their diurnal cortisol rhythm. These effects were slightly more pronounced for individuals with a history of social conflict across both waves.

**Keywords:** Biological markers; Cortisol; Diurnal rhythm; Social relationships, Social conflict

The importance of social relationships for physical and psychological health is well known (see Seeman, 1996; Seeman and McEwen, 1996 for a review). Less clear are the proximate biological mechanisms underlying these effects, although recent studies have found significant relationships between the social environment and such biomarkers as blood pressure, cholesterol, cortisol, and overall allostatic load (Seeman et. al, 2002; Seeman, et al., 2004; Seplaki, et al, 2006; Uchino, 2006; Vogelzangs et al, 2007; Ryff, Singer, and Love, 2004; Sjogren, Leanderson, and Kristenson, 2006).

The hypothalamic-pituitary-adrenal (HPA) axis is a key component of the body's physiological regulatory machinery that orchestrates patterns of physiological adaptation to the stimuli and conditions we confront throughout the course of our lives. The HPA axis plays a central role in managing stress and has been hypothesized to serve as a central mediator of social influences on health (McEwen and Seeman 1999). Recurring stressful events, including social conflict, may directly influence health by affecting HPA axis function and altering diurnal cortisol rhythms. Although short-term activations of the HPA axis are necessary for everyday functioning, frequent or chronic activation, as occur in recurrently stressful environments, can compromise HPA axis regulation and ultimately health. From a demographic perspective, HPA axis dysregulation with its attendant changes in cortisol reactivity and recovery (as seen in altered diurnal rhythms) is hypothesized to serve as a mediating mechanism, linking features of the social environment to downstream health outcomes such as disease, disability and mortality.

We use data from two waves of the Midlife in the U.S. (MIDUS) study and from the National Study of Daily Experiences (NSDE) and piecewise growth curve models to investigate relationships between histories of social conflict and patterns of diurnal cortisol rhythms. These data are unparalleled for an analysis of this sort as they include multiple cortisol samples across

multiple days for a large sample of U.S. respondents. We also use rich information on conflict with spouses, family, and friends across both waves to determine the role of chronic social stress on cortisol rhythms for the overall sample and separately for men and women. In so doing, we hope to extend past work on the association between social stress and health by explicitly examining associations between histories of social conflict across relationship types and a key biological mechanism involved in translating social stress into long-term health outcomes, namely the HPA axis.

## **BACKGROUND**

### **Social Relationships and Health**

The role of social support in a variety of health outcomes is, by now, well-documented in the demographic, sociological, psychological, and epidemiological literatures (see, for example, Anderson and Armstead 1995; House et al. 1994; House, Umberson, and Landis 1988; Mendes de Leon et al. 1999; Seeman, Seeman, and Sayles 1985; Seeman and McEwen, 1996; Uchino, Cacioppo, and Keicolt-Glaser 1996). Though attention has focused more heavily on the protective influences of social contact and support, a smaller literature has also documented the negative health consequences of adverse social interactions. Stressful relationships with family and friends, for example, is related to a variety of health outcomes, including decreased immunity (Seeman, 1996), lower self-reported health (Krause, 1996; Levenson and Gottman, 1985), functional limitations (Newsom, Mahan, Krause, 2008), cardiovascular disease (Coyne et al., 2001; Ewart et al., 1991; Orth-Gomer et al., 2000), and even mortality (Seeman et al., 1987).

Importantly, the health effects of social relationships may persist over time. Stressful or conflicted early-life relationships with primary caregivers or high levels of cumulative social

disadvantage over the life course, for example, may carry with them long-term consequences for health (Evans, 2003, Evans et al, 2007; Gunnar, 2000, Krienechin, Seagart, and Evans, 2001; O'Rand and Hamil-Luker, 2005). In this paper, we examine relationships between histories of social conflict and cortisol regulation as one key biological pathway through which social interactions may influence health.

### **Why Salivary Cortisol?**

Large-scale surveys have increasingly sought to include salivary assessments because of the centrality of the HPA axis in regulating multiple aspects of human physiology that are critical to health and well-being, and the hypothesized links between such HPA axis activity and cognitive-emotional responses to the world around us, including importantly our social worlds. Stimuli that activate the HPA function cause an increase in cortisol which triggers downstream physiological responses that help provide the energy and physiological resources needed to adapt to that stimulus. Activation of cortisol also helps to contain other components of the physiological stress response such as increases in inflammatory processes which, if unchecked, can themselves have negative health consequences. Thus, short-term activation of the HPA axis is necessary for everyday functioning. However, recurrent or chronic activation of this system has been linked to increased risks for a variety of adverse health outcomes, including cardiovascular disease, diabetes, cancer, cognitive decline, and reduced immune function (for a review, please see McEwen and Seeman , 1999). In addition, the diurnal rhythm has repeatedly been found to be sensitive to and altered by a variety of stressful situations (Adam and Gunnar, 2001; Steptoe et al., 2003; Vedhara et al., 2000).

Earlier work on the relationships between cortisol and health has focused on average cortisol measures with an interest in cortisol levels over the entire day. This is the approach used, for example, when collecting urinary cortisol, which involves one cortisol sample that is an aggregate measure of, typically, 12 - 24 hours of cortisol (Seeman et al. 2002). With the advent of salivary cortisol protocols, research has examined patterns of cortisol activity at multiple times of the day for one or more days. Such data capture what is typically referred to as the “cortisol diurnal rhythm.” The diurnal rhythm is characterized by a rapid increase in cortisol over the first 30-45 minutes after waking, followed by a rapid decline over approximately the next two hours and then a slower decline through the late afternoon and evening. Younger, healthier individuals show a more pronounced diurnal rhythm with a higher morning peak and a lower nighttime nadir and less healthy and older individuals have a flatter curve (for more details on salivary cortisol and its diurnal rhythm, please see Adam and Kumari, 2009).

Examining salivary cortisol over the course of the day provides a more complete picture of cortisol regulation (or dysregulation). In fact, one of the primary advantages of salivary cortisol samples over urinary or blood samples is that they allow for repeated and unobtrusive measurement of cortisol over multiple times of the day.

### **Prior Research on Social Relationships and Cortisol**

The influence of social relationships on cortisol response has been a topic of interest in both human and animal research. Animal research has long suggested that contact with others of the same species plays a critical role in successful development, and animals even demonstrate the potential for both positive and negative effects of the social environment (Cassel, 1976; Henry, Meehan, Stephens, 1967; Levine, 1993). For instance, among male primates, dominant

social status in a stable social environment is associated with lower levels of cortisol, while in less stable environments cortisol levels of dominant primates are higher than that of those of lower social status (Sapolsky, 1989). Social isolation also negatively impacts HPA axis activity in primates (Levine, 1993). Interestingly, the magnitude of the increase in HPA axis activity among socially isolated monkeys is also associated with the availability of social support in the broader environment (for a review, see Seeman and McEwen, 1996).

To date, research examining social support and cortisol in human populations has largely taken experimental approaches. Experimental manipulations provide strong evidence that social contact or support from a friend or partner during challenge tests (such as math or public speaking tasks) *decreases* neuroendocrine responses, including cortisol (Seeman and McEwen, 1996, Floyd et al, 2007; Gruwen et al, 2003; Uchino, Cacioppo, and Kiecolt-Glaser, 1996). In contrast, reported inadequate support and/or social conflict have been linked to *greater* physiological reactivity, again including cortisol responses, to laboratory-based challenge tests (Nausheen et al, 2007; for review Seeman and McEwen 1996; Uchino et al, 1996).

Community-based and, more recently, population-level studies have also begun to focus on associations between aspects of the social environment and cortisol regulation. For instance, one community-based study of social strain and urinary cortisol found that increased frequency of demands and criticism was positively related to overnight urinary cortisol levels for men but not women (Seeman et al., 1994). Greater reports of hostility and cynicism are related to higher levels of cortisol in the daytime (but not the overnight period), suggesting that the subjects' greater HPA activity is in response to daytime interactions with others in their environment (Pope and Smith, 1991; Ranjit et al, 2009). In addition, social relationships with parents in childhood may have lasting effects on cortisol levels well into middle and later life (Repetti,

Taylor, and Seeman, 2002; Taylor et al, forthcoming). Although prior work has documented associations between social relationships and cortisol activity in experimental settings, and between conflict and static measures of overnight cortisol (Seeman et al, 2002) in community-based studies, the relationship between adverse social relationships on the diurnal rhythm of cortisol has not yet been explored in a large national study of the U.S. population.

### **The Role of Gender**

Though social relationships have been related to health in both men and women, there are reasons to think that the effect of the social environment on cortisol may differ for men and women. Men and women pursue social relationships through different strategies and with different expectations. Men, on average, tend to focus on broader, but less intimate relationships, while women choose a smaller set of friends with whom they have closer bonds (Taylor et al., 2000, Baumeister and Sommer, 1997). Interestingly, several studies have found that the relationships between social support and health tend to be stronger for men than for women (House, Robbines, Metzner, 1982; Kaplan et al, 1988, Seeman, 1996, Seeman et al, 1994, Seeman et al, 2002). There is also experimental evidence that shows that the presence of a supportive companion during the waiting period just before a public speaking task was associated with lower cortisol responses, but only for men (Kirschbaum et al., 1995). Among the women, presence of one's partner was associated with a trend toward *increased* cortisol responses.

Other work suggests that both men's and women's health risks are affected by social interactions, but that the relative impact of different types of relationships differs for men and women. While men are psychologically affected by stressors involving their family members,

women are reactive to stressors affecting both family and friends. In fact, one study examining social support in later life finds that family relationships play a particularly prominent role in older men's lives, while women depend on both family and friends for support in older ages (McIlvane and Reinhardt, 2001). In addition, women are more often the ones others turn to for help, and caring for others may pose a cost when it comes to social relationships. Women do, in fact, report providing support to a broader array of network members (Wethington, McLeod, and Kessler, 1987).

In addition, while there is some evidence (such as that cited above) that men benefit more from social support than women do, women exhibit greater physiological and psychological reactivity to *negative* social interactions than do men (Taylor et al., 2000, Seeman et al, 1994, Seeman and Crimmins, 2001). In a population-based study in Taiwan, Goldman and colleagues find that level of perceived stress and physiological reactivity (based on an overall score on 16 biomarkers, including cortisol) is stronger for women than men (Goldman et al, 2005). In addition, one experimental study shows that women may generally be more responsive to the downside of social relationships: to wit, social rejection. In this study, men showed significantly greater cortisol responses to the achievement challenges, whereas women showed greater cortisol responses to the social rejection challenges (Stroud, Salovey, and Epel, 2002). Despite their potentially greater vulnerability to family and friends' stressors, though, there is at least some evidence that women also enjoy greater health benefits from their friendships than do men (Walen and Lachman, 2000, Seeman et. al, 2002).

## **Current Analyses**

In this paper, we use data from the National Survey of Midlife Development in the United States (MIDUS) study. We assess the diurnal cortisol rhythm using relatively new methodology and salivary cortisol information based on sampling across four time points over the course of each of 4 days, estimating slopes for the morning rise as well as both early and later afternoon/evening declines. In addition, we use model-based estimates to calculate measures of the highest cortisol value of the day (peak), the lowest cortisol value of the day (nadir) and the waking-day area under the curve (for an overall measure of cumulative cortisol secreted over the day, when awake). We examine associations between social conflict and diurnal cortisol rhythms in several ways, drawing on the rich longitudinal data on histories of social conflict from the MIDUS study. We examine both cross-sectional associations between current levels of conflict and current cortisol rhythms as well as examining how longer-term histories of social conflict covering approximately a decade relate to current diurnal cortisol rhythms. In light of prior work suggesting potential sex differences in the physiological sequelae of social interactions, we also examine the possibility that social conflict may be differentially related to cortisol rhythms in men and women. In the coming sections we describe our data, methods, measures, and analysis in detail, report the findings of this study, and conclude with a discussion of the implications of this work for broader health outcomes.

## **DATA AND METHODS**

The National Survey of Midlife Development in the United States (MIDUS) study was initiated in 1995 to determine how social, psychological, and behavioral factors interrelate to influence mental and physical health. The first wave collected socio-demographic and

psychosocial data on 7,108 Americans, ages 25 to 74 years, from a representative sample of English-speaking, non-institutionalized adults residing in the contiguous 48 states, with oversampling of 5 metropolitan areas, twin pairs, and siblings. Eighty nine percent of the sample (N=6,329) completed both a phone interview and a detailed self-administered questionnaire.

In the second wave of data collection, a random sub-sample also completed short telephone interviews about their daily experiences over eight consecutive days and collected saliva (for cortisol assessments) on four of the eight days. This National Study of Daily Experiences (NSDE) subsample and the MIDUS sample from which it was drawn had very similar distributions for age, marital status, and parenting status (for a complete description, see Almeida, Wethington, and Kessler, 2002). Importantly for this analysis, as part of the NSDE study, respondents were asked to take salivary cortisol samples four times a day (i.e. at waking, half hour after waking, lunchtime, and at bedtime) for four random days over the week. These daily cortisol data are used in the construction of the dependent variable for this study.

Of the initial NSDE sample of 1,605 participants, 1,589 participants had usable cortisol and sampling time data for 6,071 days. Missing data on model variables brought the final sample for these analyses to 1,498.

### **Salivary Cortisol**

Salivary cortisol was measured four times per day over four random days, at: (1) awaking (2) half an hour after waking (3) before lunch, and (4) at bedtime. Respondents took samples in their own home, by placing a roll of cotton in their mouths, chewing on it for approximately 30 seconds, and placing it in a tube called a salivette, which respondents stored at room temperature until they were returned to the clinic the next day. Data on the exact time of

each saliva sample were obtained from nightly telephone interviews by study staff and on a paper-pencil log sent with the collection kit. In addition, a quarter of the respondents receive a “Smart Box” to store their salivettes, with a computer chip that records the time of box opening and closing. In our analyses, we used information on collection time from the home collection sheet times, unless they are missing, in which case the times reported in the interview are used instead. Salivettes were frozen (at  $-60^{\circ}\text{C}$ ) for storing and shipping. Cortisol concentrations (in nmol/L) were measured with a commercially available luminescence immunoassay. For all analyses, cortisol is recoded as the  $\ln(\text{cortisol}+1)$ , in order to account for outlying cases and some small cortisol values.

We began with an initial NSDE sample of 1,605 participants with 6,383 days of cortisol data. We dropped data from 130 days when participants awoke before 4 am, 104 days when the 3rd cortisol sample was 10 nmol/L or more higher than the 2nd sample (since this might reflect a time-recording error for one of the saliva samples or saliva sample contamination with food), 49 days when respondents woke after 11am, and an additional 28 days for respondents who were awake more than 20 hours on a given day, based on our observation that cortisol patterns during these days were distinct from that in days with less extreme wake times. This left us with 1,589 participants with 6,071 days of cortisol data and 24,284 saliva measurements. After excluding those with missing predictor/covariate data or cortisol values outside of the normal range (i.e.  $>60$  nmol/L), we were left with a final analytic sample of 1,498 people, 5,462 days and 21,265 total saliva measurements. Respondents in the final sample had at least one valid salivary sample of cortisol, with some respondents providing as many as sixteen samples (four on each of four days).

## History of Social Conflict

Adult social conflict with family and friends were assessed from items in the self-administered mail questionnaires in both MIDUS I and MIDUS II. Frequency of social conflict was queried with respect to spouse/partner (6 items), friends (4 items), and other family members (4 items). The items include the following: “How often do your *friends/spouse/family* make too many demands on you?”; “How often do they criticize you?”; “How often do they let you down when you are counting on them?”; “How often do they get on your nerves?” For the spouse/partner scale, two additional items are included: “How often does he or she argue with you?”; and “How often does he or she make you feel tense?”.

All items are measured on a four point scale indicating whether this occurs 1 Often; 2 Sometimes; 3 Rarely; or 4 Never. The mean of all items was calculated for each relationship type (i.e. spouse, family, friends), with items recoded so that higher scores reflect higher conflict. We then averaged the three scales into one global score measuring the respondent’s average level of social conflict from all sources. Although our summary measure of primary interest is mean conflict across relationship, for comparative purposes, we also took the maximum score across the three sources for a measure of highest level of conflict. We did this separately for both Wave 1 and Wave 2.

In addition to constructing separate conflict scores for each wave, we also combined the scores for the two waves by dividing each wave’s scores into quartiles and constructing a score across the two waves using the following coding:

- (1) Not in the highest quartile of conflict in either Wave 1 or 2
- (2) Highest quartile conflict in Wave 1
- (3) Highest quartile conflict in Wave 2
- (4) Highest quartile conflict in both waves

We do this for mean conflict, maximum conflict, and for conflict with each relationship type. This construction allows us to distinguish different “histories” of social conflict based on MIDUS I and II data and, in particular, to identify those reporting the high levels of conflict at both waves – a group we hypothesize would be at highest risk for the biological consequences of social conflict.

### **Control Variables**

Multivariable analyses also include controls for race, age, sex, and education. Both race and sex were coded as indicator variables, with the first indicating whether a respondent was white or nonwhite and the latter, whether male or female. A three category age variable was included in the models with age coded as <50 years old, 50-64, and 65+. Education was included in all models as a three-category variable indicating whether the respondent completed high school or less schooling; some college; or college or more.

### **ANALYSES**

Because previous studies indicate that cortisol rhythms are driven by time elapsed since awakening and less by clock time (van Couter, 1990; Steptoe et al., 2003; Clow et al., 2004; Fries et al., 2009; Kumari et al. 2010), we examined cortisol trajectories as a function of time since waking. Based on visual examination of average cortisol rhythms in the sample and in demographic strata defined by age, gender, race, and socioeconomic status (Taylor et al, forthcoming; Karlamangla et al., in preparation) and in line with other work (Ranjit et al. 2005), we modeled the diurnal cortisol trajectories as piecewise linear growth curves, using four linear splines with three knots, fixed at 0.5 hours, 4.5 hours, and 15 hours after waking. The four spline pieces represent four phases of the day, with the first piece representing the morning rise

(waking– half hour after waking); the second, a steep early decline (0.5-4.5 hours after waking); the third, a more gradual late afternoon through evening decline (4.5-15 hours after waking); and the final piece representing a later night plateau (15-20 hours after waking).

The intercept (representing the waking value) and all four spline slopes were modeled as functions of the primary predictor (social conflict) and covariates. In order to remove any bias that may arise from differences in sleep patterns, all models include controls for the average number of hours the respondent is awake (averaged across all days of diary data provided and centered at the median waking time of the sample), and several variables that vary from day to day, including: whether the respondent woke before the median wake up time of the sample (6:40am) on the days the cortisol sample were taken ; whether the respondent slept fewer than 6 hours or more than 8 hours the night before, and whether the sample was taken on a weekend day (for employed respondents only). For further information on the choice of model covariates to account for differences sleep patterns, please see (Karlmanangla et al, in preparation).

We used hierarchical, 3-level, linear mixed effects models to fit the cortisol growth curves and to account for within-individual and within-family clustering. To account for the correlation between repeated measures of cortisol in the same individual (between 1 and 16 measurements per person), we included random effects for the intercept (wakening value of cortisol) and all four slopes. To allow for correlation between members of the same family (twin pairs and siblings), we included an additional hierarchical level with random intercept. Model-predicted intercept and slopes were used to estimate mean values for other trajectory parameters,

such as the magnitude of the daily peak, the nightly nadir, and the total exposure over 16 hours since waking<sup>1</sup>, or area under the curve (AUC). These were calculated as:

$$\begin{aligned}\text{Peak} &= \text{Intercept} + 0.5 * \text{slope}_1 \\ \text{Nadir} &= \text{Peak} + 4 * \text{slope}_2 + 10.5 * \text{slope}_3 \\ \text{AUC} &= 0.25 * (\text{Intercept} + \text{Peak}) + 2 * (2 * \text{Peak} + 4 * \text{slope}_2) + 5.25 * (\text{Peak} + 4 * \text{slope}_2 + \text{Nadir}) + 0.5 * (2 * \text{Nadir} + 1 * \text{slope}_4)\end{aligned}$$

Slope<sub>1</sub>, Slope<sub>2</sub>, Slope<sub>3</sub>, and Slope<sub>4</sub> refer to the model-estimated mean slopes (per hour) for the four piece-wise linear segments of the trajectory.

## RESULTS

Table 1 provides descriptive information on the model variables for the analytic sample. Approximately 30 percent of participants were less than 50 years old at the time of the MIDUS 2 data collection, 40 percent were between 50 and 65 and nearly 30 percent were 65 and older. The sample was largely White and relatively well-educated, with over 40 percent having a college degree. There was substantial variance in the social conflict scales. For Wave 1, the average level of social conflict across all relationship types was 2.05 (equivalent to responses of “rarely”), while a measure of the maximum conflict across relationships was higher at 2.40 (moving closer of responses of “sometimes”). In Wave 2 the corresponding values were 1.98 and 2.32, respectively. The lowest levels of mean conflict were for friends and the highest for spouse. The 10-year history of social conflict in quartiles shows that although most people do not experience extreme levels of conflict, just over 13 percent of respondents are in the highest quartile of mean conflict in both waves.

---

<sup>1</sup> We do not include the late night plateau in our calculations of the area under the curve as this is only available for select respondents who stay up more than 15 hours and is most likely capturing the beginning of their next day's diurnal rhythm.

## TABLE 1 HERE

Figure 1 shows the model-predicted diurnal cortisol rhythm as a function of time since waking in hours for the null model, with inflexions at 0.5 hours, 4.5 hours, and 15 hours. These results are from a model controlling for early waking (waking before median wake-up time), sleeping fewer than 6 hours the night before, sleeping more than 8 hours the night before, average hours awake, and weekend vs. weekday (if employed).

## FIGURE 1 HERE

This figure shows the expected morning rise from waking until about half an hour after waking, followed by a rapid decline until 4.5 hours after waking, followed by a more gradual late afternoon decline until 15 hours after waking and finally, for respondents who are awake more than 15 hours we find a flattening of their cortisol rhythm to a late night plateau. The first three slopes are statistically significantly different from zero at  $p < 0.01$ ; slope 4 was not statistically different from zero. Model-predicted mean wakening value, peak and nadir, are 13.88 nmol/L (or  $2.7 \ln(\text{nmol/L}+1)$ ), 19.29 nmol/L, and 2.00 nmol/L respectively. Estimated area under the log-cortisol curve (AUC) was  $29.2 \ln(\text{nmol/L}+1)$  hours.

Table 2 shows the results of our fully adjusted models predicting Wave 2 salivary cortisol over the day as a function of Wave 2 social conflict scores. This table displays the effects of concurrent social conflict on the waking value (intercept) and the four slopes, as well as the model estimated peak (highest point), nadir (lowest point) and the full area under the curve for the day (AUC). We present results of two summary measures of social conflict: mean conflict (averaged across spouse, family, friends) and maximum conflict (maximum conflict reported from any source). Both measures are treated as continuous in these models (range: 1 to 4).

These results provide a cross-sectional snapshot of the relationship between the cortisol rhythm and social conflict.

#### TABLE 2 HERE

As Table 2 reveals, respondents with higher levels of social conflict have a less rapid late day decline (more positive slope), suggesting that their cortisol levels do not come down as much in the evening hours as they do for their counterparts with lower levels of social conflict. Higher levels of social conflict are also associated with a lower morning peak value (statistically significant for maximum conflict, marginally significant for average conflict) and a marginally higher Nadir (for average conflict only). Taken in combination, these results suggest that for respondents with higher levels of social conflict, cortisol values do not go up as much in the early morning hours, do not come down as much later in the evening before bedtime, and do not reach as low a point as they do for individuals with lower levels of social conflict.

Interestingly, although the coefficients are in the same direction regardless of whether we use a mean or a maximum construction of the social conflict variable, the magnitude of the coefficients for cortisol measures capturing the earlier part of the day (i.e. first slope and peak) are greater when we consider maximum conflict. The converse appears to be true for mean conflict, where cortisol measures capturing the latter part of the day show a greater response to conflict. Nonetheless, regardless of the measure used, we see that cortisol rhythms are flatter for those with higher reported conflict than for their counterparts with lower level of reported conflict with family and friends.

We can visually observe these differences in trajectories by level of conflict in the graph depicted in Figure 2. Figure 2 shows the predicted cortisol trajectories over the course of the day

for individuals with a mean conflict score of 1 (“never”), 2 (“sometimes”), and 3 (“often”). The average of the mean conflict score for the analytic sample was 2.0 (see Table 1). Although all three groups show clear diurnal rhythms with a morning rise and a later day decline, the most pronounced trajectory is apparent for the lowest conflict group, the flattest trajectory is for highest conflict group, and those with mean levels of reported conflict fall directly in between the other two groups.

#### FIGURE 2 HERE

Comparing the coefficients in Table 2 with those produced by the null unadjusted model and displayed in Figure 1 gives us a sense of the magnitude of the effect of low social conflict on cortisol levels throughout the day. For instance, in Table 2 we see that each unit increase in the score of maximum social conflict is associated with waking cortisol value that is 0.029 ln(nmol/L) smaller, which translates to scaling of waking cortisol by 0.97 per unit increase in maximum conflict score. On the other hand, the evening decline slope (slope 3) is 0.012 ln(nmol/L) per hour higher per unit increase in average conflict score, which is a fairly large effect (>10%) relative to the average slope in the sample (-0.09 ln(nmol/L) per hour).

In results not shown here, we reran the model described above, with the addition of a difference score measuring the change in conflict between Waves 1 and Wave 2. The coefficients for the change score were not statistically significant. In addition, the significant values for the Wave 2 cortisol slopes remained strong and were sometimes even strengthened when the difference score was added. This suggests that it is Wave 2 levels of social conflict that are related to Wave 2 cortisol, and that the change between the two waves does not explain the relationship between Wave 2 social conflict and cortisol rhythm.

## History of Social Conflict

Another way to bring in information from both waves of social conflict is by classifying respondents into categories based on whether they were in the highest quartile of social conflict in Wave 1 only, Wave 2 only, both waves, or in neither wave. Looking only at individuals in the highest quartile of conflict allows us to assess how much experiencing extreme levels of social conflict is related to cortisol dysregulation over the course of the day. By bringing together the two waves of information, this also gives us a richer 10-year history of social conflict for each respondent. This allows us to examine whether the association between conflict and cortisol is stronger if the social conflict occurred more recently, and whether individuals who experienced extreme levels of conflict in both waves also experience more cortisol dysregulation than those who only recently experienced high levels of social conflict for the first time or those who may have been in the highest quartile of conflict in the past but are no longer experiencing it to the same extent.

Table 3 shows the results of spline models predicting the cortisol diurnal rhythm as a function of 10-year history of social conflict, assessing whether respondents were in the highest quartile of conflict in Wave 1, Wave 2, both waves, or neither wave (reference category). This table shows the coefficients and standard errors for the waking values, the four slopes during the day, and the model estimated peak, nadir, and AUC.

TABLE 3 HERE

If we focus in on the results for the first panel (mean conflict) and look at the evening decline slope and the morning peak, both of which were significant or at least marginally for both the mean and maximum conflict measures in the cross-sectional Wave 2 analysis above, we

see a similar trend to that found in Table 2; i.e., that waking and peak values are lower, and evening declines are slower in those with high levels of conflict. This is true for those in the highest quartile of conflict at only Wave 1 and only Wave 2, although it is more pronounced for the Wave 1 only group<sup>2</sup>. However, we see that a history of conflict does take its toll when it comes to the waking values and morning peak. Individuals with a history of two waves of conflict have a marginally significantly lower waking and peak values. Although these results are modest, there is at least some evidence that cortisol functioning is more impaired for those with a history of conflict than those with conflict in only one wave of data.

Surprisingly, when we look at the maximum conflict across relationship types, we do not see an added burden for those who have experienced a longer history of conflict. In fact, the coefficients for Wave 2 conflict only and the ten-year history of conflict are nearly identical. This may suggest that when using a more extreme measure of conflict (maximum as opposed to mean) we are already capturing such extreme cases that introducing a second wave of conflict adds little predictive power to these models.

### **Are There Differences by Gender?**

The literature on social relationships and health suggests that women's health may be more sensitive to the social environment than men's health outcomes, and that women may be especially sensitive to conflict with friends, more so than men (Taylor et al., 2000; Seeman and McEwen, 1996). In Table 4, we examine whether there are gender differences in the associations between social conflict and salivary cortisol broken down by source of conflict (i.e., from spouse, family, or friends). The model used to produce Table 4 is identical to that used for

---

<sup>2</sup> This may suggest that there is a lag in the amount of time it takes for conflict to be translated into dysregulated neuroendocrine functioning. However, it should be noted that Wave 2 conflict and cortisol were not measured concurrently. Cortisol samples were obtained later than the initial survey.

Table 3, except in this analysis the model is run separately for men and women, and only results comparing the group with the highest quartile of conflict in both waves to those not in the highest conflict quartile in either wave are displayed.

#### TABLE 4 HERE

Table 4 shows the cortisol trajectories stratified by gender and type of conflict. For men, being in the highest quartile of conflict with family in both waves results in a marginally significant lower waking value and a statistically significant lower peak value, as compared to those who are never in the highest quartile of conflict. However, for women, it is conflict with friends, not family, that appears to be most strongly related to cortisol rhythms. High levels of conflict with friends in both waves result in significantly lower waking values, a significantly more positive evening decline slope (comes down slower), a marginally significant lower peak, and a marginally significant higher nadir for women. Although these results are suggestive of gender differences in the effect of type of conflict on cortisol reactivity, most of the differences between men and women are not statistically significant. In fact, of the significant results in these tables, the only coefficient that shows a statistically significant difference for men and women is that of the waking cortisol value for conflict with friends. Women who report being in the highest quartile of conflict with friends in both waves have significantly lower waking values than their male counterparts. However, women's trajectories over the rest of the day are not significantly different from those of men.

## **DISCUSSION**

Conflict with spouse, family, and friends has significant implications for health. We look at social conflict at a point in time, as a ten year cumulative history of conflict, and broken down into conflict with family, friends, and spouse to get a complete picture of the role of conflict in cortisol dysregulation. We find evidence that individuals with higher levels of social conflict have dysregulated cortisol rhythms. Their cortisol levels do not go up as much in the early morning hours, do not come down as much later in the evening before bedtime, and do not reach as low a point as they do for individuals with lower levels of conflict. All in all, their rhythms are much flatter than those of their counterparts. These effects are slightly more pronounced for those individuals with a history of social conflict over a ten year period.

We also find modest (though not statistically significant) evidence that there may be a gender component to the importance of social relationships for stress. Men are more affected by stress from family, while cortisol dysregulation is most pronounced for women with frequent conflict with friends. Although most of the differences between men and women are not statistically significant, the direction and magnitude of the effects support prior smaller scale and experimental studies showing gender differences in the extent to which different social relationships are associated with health outcomes.

This work has several limitations. Although we use a large survey dataset of the U.S. population, it is not national representative and is, in fact, primarily White. This makes it difficult to know if these results generalize to other race-ethnic subpopulations. In addition, although we have information on social conflict over two waves of data, we only have cortisol measures at one point in time. Without longitudinal data on cortisol, it is impossible to

determine whether there is a causal relationship between social relationships and cortisol diurnal rhythm.

Despite these limitations, this work has important implications for the growing body of work showing a link between social relationships and health. However, this paper goes beyond social relationships at a point in time and indicates that cumulative social conflict over the long run is more detrimental than recent conflict. This study suggests that a longer-run perspective on social relationships and health is necessary for understanding the lasting effects of negative social relationships and to get a more complete picture of how health inequalities develop within societies. We only look at social relationships over a 10-year period, future work should examine how social relationships over the life course – from childhood on– translate into adverse health effects in midlife and beyond.

## REFERENCES

- Adam E.K. and M. R. Gunnar. 2001. "Relationship Functioning and Home and Work Demands Predict Individual Differences in Diurnal Cortisol Patterns in Women." *Psychoneuroendocrinology*, 26: 189-208.
- Adam E.K. and M. Kumari. 2009. "Assessing Salivary Cortisol in Large-Scale, Epidemiological Research." *Psychoneuroendocrinology*, 34:1423-1436.
- Almeida D.M., E. Wethington, and R. C. Kessler. 2002. "The Daily Inventory of Stressful Events: An Interview-Based Approach for Measuring Daily Stressors." *Assessment*, 9:41-55.
- Anderson N. and C. Armstead. 1995. "Toward Understanding the Association of Socioeconomic Status and Health: A New Challenge for the Biopsychosocial Approach." *Psychosomatic Medicine*, 57:213-225.
- Baumeister R.F. and K. L. Sommer. 1997. "What Do Men Want? Gender Differences and Two Spheres of Belongingness: Comment on Cross and Madson." *Psychological Bulletin*, 122:38-44.
- Cassel J. 1976. "The Contribution of the Social Environment to Host Resistance." *American Journal of Epidemiology*, 104:107-123.
- Clow A., L. Thorn, P. Evans, and F. Hucklebridge. 2004. "The Awakening Cortisol Response: Methodological Issues and Significance." *Stress: The International Journal on the Biology of Stress*, 7:29-37.
- Coyne J.C., M.J. Rohrbaugh, V. Shoham, J. S. Sonnega, J. M. Nicklas, and J.A. Cranford. 2001. "Prognostic Importance of Marital Quality for Survival of Congestive Heart Failure." *The American Journal of Cardiology*, 88: 526-529.

- Evans G.W. 2003. "A Multimethodological Analysis of Cumulative Risk and Allostatic Load Among Rural Children." *Dev Psychol*, 39:924-933.
- Evans G.W., P. Kim, A. H. Ting, H. B. Teshler, and D. Shannis. 2007. "Cumulative Risk, Maternal Responsiveness, and Allostatic Load Among Young Adolescents." *Dev Psychol*, 43:341-51.
- Ewart C.K., C. B. Taylor, H. C. Kraemer, W. S. Agras. 1991. "High Blood Pressure and Marital Discord: Not Being Nasty Matters More than Being Nice." *Health Psychology*, 10:155–163.
- Floyd K., A. C. Mikkelsen, M. A. Tafoya, et al. 2007. "Human Affection Exchange: XIII. Affectionate Communication Accelerates Neuroendocrine Stress Recovery." *Health Communication*, 22:123 - 132.
- Fries E., L. Dettenborn, and C. Kirschbaum. 2009. "The Cortisol Awakening Response (CAR): Facts and Future Directions." *International Journal of Psychophysiology*, 72:67-73.
- Goldman N., D. Gleib, C. Seplaki, I. Liu, and M. Weinstein. 2005. "Perceived Stress and Physiological Dysregulation." *Stress*, 8: 95-105
- Grewen K.M., B. J. Anderson, S. S. Girdler, and K. C. Light. 2003. "Warm Partner Contact is Related to Lower Cardiovascular Reactivity." *Behavioral Medicine*, 29:123-130.
- Gunnar M.R. 2000. "Early Adversity and the Development of Stress Reactivity and Regulation", in: C. A. Nelson (Ed.), *The Effects of Early Adversity on Neurobehavioral Development* Erlbaum: Mahwah, NJ. 163-200.
- Henry J. P., J. P. Meehan, and P. M. Stephens. 1967. "The Use of Psychosocial Stimuli to Induce Prolonged Systolic Hypertension in Mice." *Psychosomatic Medicine*, 29:408-432.

- House J.S., C. Robbins, and H. L. Metzner. 1982. "The Association of Social Relationships and Activities With Mortality: Prospective Evidence From The Tecumseh Community Health Study." *American Journal of Epidemiology*, 116:123-140.
- House J.S., D. Umberson, K. R. Landis. 1988. "Structures and Processes of Social Support." *Annual Review of Sociology*, 14:293-318.
- House J.S., J. M. Lepkowski, A.M. Kinney, R. P. Mero, Kessler R.C., and A. R. Herzog. 1994. "The Social Stratification of Aging and Health." *Journal of Health and Social Behavior*, 35:213-234.
- Kaplan G.A., J. T. Salonen, R. D. Cohen, R. J. Brand, S. S. Leonard, and P. Puska. 1988. "Social Connections and Mortality from All Causes and From Cardiovascular Disease: Prospective Evidence from Eastern Finland" *American Journal of Epidemiology*, 128:370-380.
- Karlamangla, A., E.M. Friedman, R. Stawski, and T. E. Seeman. In preparation. "Modeling Salivary Cortisol in the National Study of Daily Experiences."
- Kirschbaum C., T. Klauer, S. Filipp, and D. Hellhammer. 1995. "Sex-Specific Effects of Social Support on Cortisol and Subjective Responses to Acute Psychological Stress." *Psychosomatic Medicine*, 57:23-31.
- Krause N. 1996. "Neighborhood Deterioration and Self-Rated Health in Later Life." *Psychology and Aging*, 11:342-352.
- Krenichyn K., S. Saegert, G. W. Evans. 2001. "Parents as Moderators of Psychological and Physiological Correlates of Inner-City Children's Exposure to Violence." *Journal of Applied Developmental Psychology*, 22:581-602.

- Kumari M., E. Badrick, and T. Chandola, et al. 2010. "Measures of Social Position and Cortisol Secretion in an Aging Population: Findings From the Whitehall II Study." *Psychosomatic Medicine*, 72: 27-34.
- Levenson R.W., J. M. Gottman. 1985. "Physiological and Affective Predictors of Change in Relationship Satisfaction." *Journal of Personality and Social Psychology*, 49:1, 85-94.
- Levine S. 1993. "The Influence of Social Factors on the Response to Stress." *Psychosomatic Medicine*, 60: 33-38.
- McEwen B.S. and T.E. Seeman. 1999. "Protective and Damaging Effects of Mediators of Stress: Elaborating and Testing the Concepts of Allostasis and Allostatic Load." *Annals of the New York Academy of Sciences*, 896: 30-47.
- McIlvane J., J. P. Reinhardt. 2001. "The Interactive Effect of Support from Families and Friends in Visually Impaired Elders." *Journal of Gerontology: Psychological Sciences*, 56B: 374-382.
- Mendes de Leon C.F., T.A. Glass, L. A. Beckett, T.E. Seeman, D. A. Evans, L. F. Berkman. 1999. "Social Networks and Disability Transitions across Eight Intervals of Yearly Data in the New Haven EPESE." *Journal of Gerontology: Social Sciences*, 54B:S162-S172.
- Nausheen B., Y. Gidron, A. Gregg, H.S. Tissarchondou, R. Peveler. 2007. "Loneliness, Social Support and Cardiovascular Reactivity to Laboratory Stress." *Stress: The International Journal on the Biology of Stress*, 10:37-44.
- Newsom J.T., T. L. Mahan, K. S. Rook, N. Krause. 2008. "Stable Negative Social Exchanges and Health." *Health Psychology*, 27:78-86.

- O'Rand A.M., and J. Hamil-Luker. 2005. "Processes of Cumulative Adversity: Childhood Disadvantage and Increased Risk of Heart Attack Across the Life Course." *Journal of Gerontology B Psychological Sciences and Social Sciences*, 60: 117-24.
- Orth-Gomer K., S. P. Wamala, M. Horsten, K. Schenck-Gustafsson, N. Schneiderman, and M. A. Mittleman. 2000. "Marital Stress Worsens Prognosis in Women with Coronary Heart Disease: The Stockholm Female Coronary Risk Study." *Journal of the American Medical Association*, 284:3008–3014.
- Pope M., and T. Smith. 1991. "Cortisol Excretion in High and Low Cynically Hostile Men." *Psychosomatic Medicine*, 53:386-392.
- Ranjit N., A. Diez-Roux, B. Sanchez, T. Seeman, S. Shea, S. Shrager, K. Watson. 2009. "Association of Salivary Cortisol Circadian Pattern With Cynical Hostility: Multi-Ethnic Study of Atherosclerosis." *Psychosomatic Medicine*, 71:748-755.
- Repetti R.L., S. E. Taylor, and T. E. Seeman. 2002. "Risky Families: Family Social Environments and the Mental and Physical Health of Offspring." *Psychological Bulletin*, 128: 330-336.
- Ryff C.D., B. H. Singer, G. D. Love. 2004. "Positive Health: Connecting Well-Being with Biology." *Philosophical Transactions of the Royal Society Lond B Biological Sciences*, 359:1383-94.
- Sapolsky RM. 1989. "Hypercortisolism among Socially Subordinate Wild Baboons Originates at the CNS Level." *Archives of General Psychiatry*, 46:1047-1051.
- Seeman M., T. Seeman, and M. Sayles. 1985. "Social Networks and Health Status: A Longitudinal Analysis." *Social Psychology Quarterly*, 48:237-248.

- Seeman T., and B. McEwen. 1996. "Impact of Social Environment Characteristics on Neuroendocrine Regulation." *Psychosomatic Medicine*, 58:459-471.
- Seeman T., D. Gleib, N. Goldman, M. Weinstein, B. Singer, and Y.-H. Lin. 2004. "Social Relationships and Allostatic Load in Taiwanese Elderly and Near Elderly." *Social Science and Medicine*, 59:2245-2257.
- Seeman T.E. 1996. "Social Ties and Health: The Benefits of Social Integration." *Annals of Epidemiology*, 6:442-451.
- Seeman T.E. and E. Crimmins. 2001. "Social Environment Effects on Health and Aging: Integrating Epidemiologic and Demographic Approaches and Perspectives." *Annals of the New York Academy of Science*, 954:88-117.
- Seeman T.E., L. F. Berkman, D. Blazer, and J. W. Rowe. 1994. "Social Ties and Support and Neuroendocrine Function: The MacArthur Studies of Successful Aging." *Annals of Behavioral Medicine*, 16: 95-106.
- Seeman T.E., G. A. Kaplan, L. Knudsen, R. Cohen, and J. Guralnik. 1987. "Social Network Ties and Mortality Among the Elderly In the Alameda County Study." *American Journal of Epidemiology*, 126:714-723.
- Seeman T.E., B. H. Singer, C. D. Ryff, G. Diener, Love, L. Levy-Storms. 2002. "Social Relationships, Gender, and Allostatic Load Across Two Age Cohorts." *Psychosomatic Medicine*, 64:395-406.
- Seplaki C.L., N. Goldman, M. Weinstein, Y.-H. Lin. 2006. "Measurement of Cumulative Physiological Dysregulation in an Older Population." *Demography*, 43:165-183.

- Sjögren E., P. Leanderson, M. Kristenson. 2006. "Diurnal Saliva Cortisol Levels and Relations to Psychosocial Factors in a Population Sample of Middle-Aged Swedish Men and Women." *International Journal of Behavioral Medicine*, 13:193-200.
- Steptoe A., S. Kunz-Ebrecht, N. Owen, P. J. Feldman, G. Willemsen, C. Kirschbaum, and M. Marmot. 2003. "Socioeconomic Status and Stress-Related Biological Responses Over the Working Day." *Psychosomatic Medicine*, 65:461-470.
- Stroud L.R., P. Salovey, E. S. Epel. 2002. "Sex Differences in Stress Responses: Social Rejection Versus Achievement Stress." *Biological Psychiatry*, 52:318-327.
- Taylor S. E. , L. Cousino Klein, B. P. Lewis, T. L. Gruenewald, R. A. R. Gurung, and J. A. Updegraff. 2000. "Biobehavioral Responses to Stress in Females: Tend-and-Befriend, Not Fight-or-Flight." *Psychological Review*, 107:411-429.
- Taylor S.E., A.S. Karlamangla, E. M. Friedman, T. E. Seeman. forthcoming. "Early Environment Affects Neuroendocrine Regulation in Adulthood." *Social Cognitive and Affective Neuroscience: Special Issue on Aging Research*.
- Uchino B. 2006. "Social Support and Health: A Review of Physiological Processes Potentially Underlying Links to Disease Outcomes." *Journal of Behavioral Medicine*, 29:377-387.
- Uchino B.N., J. T. Cacioppo, J. K. Kiecolt-Glaser. 1996. "The Relationship Between Social Support and Physiological Processes: A Review with Emphasis on Underlying Mechanisms and Implications for Health." *Psychological Bulletin*, 119:488-531.
- van Couter E. 1990. "Diurnal and Ultradian Rhythms in Human Endocrine Function: A Mini Review." *Hormone Research*, 34:45-53.
- Vedhara K., J. Hyde, I. D. Gilchrist, M. Tytherleigh, S. Plummer. 2000. "Acute Stress, Memory, Attention and Cortisol." *Psychoneuroendocrinology*, 25:535-549.

- Vogelzangs N., A. Beekman, S. Kritchevsky, A., et al. 2007. "Psychosocial Risk Factors and the Metabolic Syndrome in Elderly Persons: Findings from the Health, Aging and Body Composition Study." *Journal of Gerontology A Biol Sci Med Sci*, 62:563-569.
- Walen H.R., and M. E. Lachman. 2000. "Social Support and Strain from Partner, Family, and Friends: Costs and Benefits for Men and Women in Adulthood." *Journal of Social and Personal Relationships*, 17:5-30.
- Wethington E., J. D. McLeod, and R. C. Kessler. 1987. "The Importance of Life Events in Explaining Sex Differences in Psychological Distress", in: R.C. Barnett, L. Biener, and G.K. Baruch (Ed.), *Gender and Stress*. Basic Books, New York. 144-156.

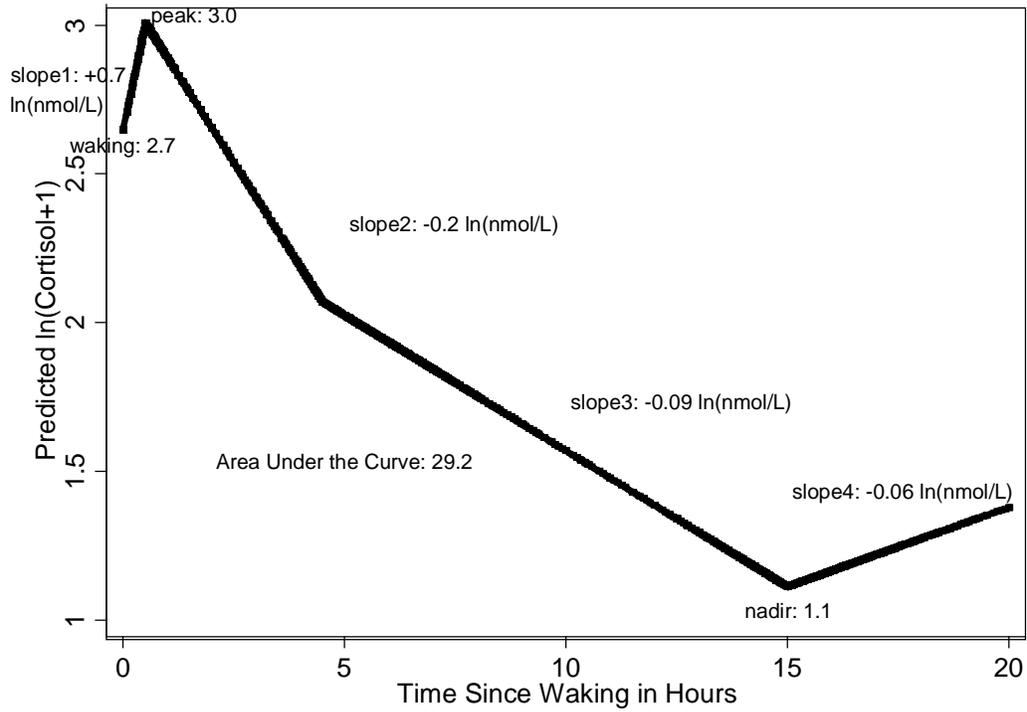
## TABLES AND FIGURES

**Table 1: Descriptive Statistics for Analytic Sample (n=1,498)**

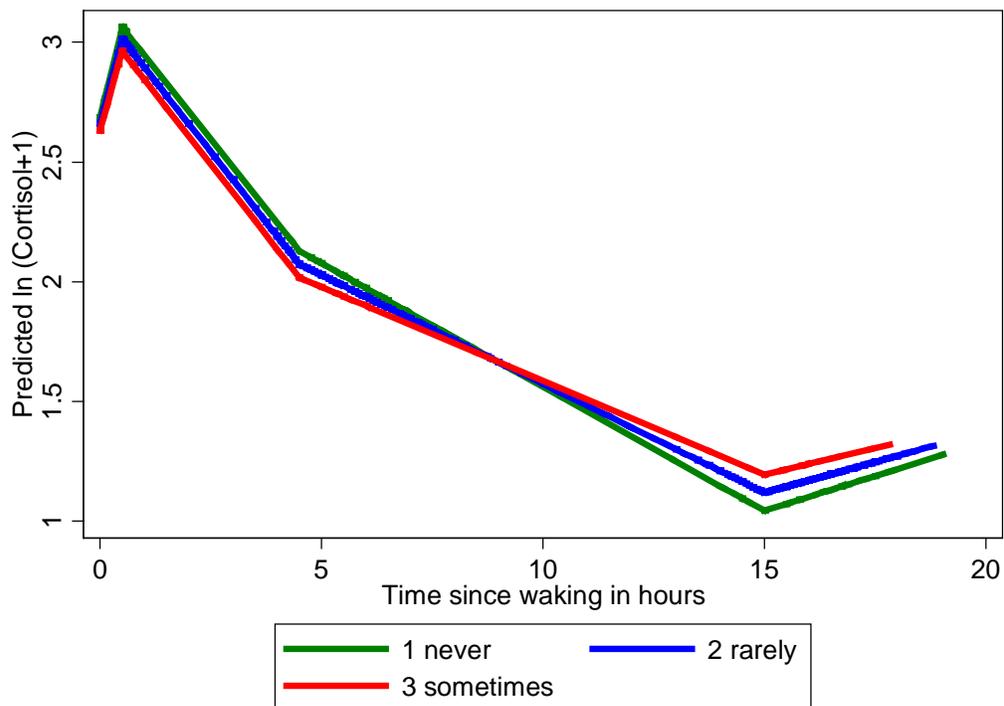
Variable	Unit or Category	Mean (sd) or %
Age	<50	30.84
	50-65	40.72
	65+	28.44
Sex	Male	44.13
	Female	55.87
Race	White	93.06
	Non-white or mixed race	6.94
Education	High school degree or less	29.17
	Some college	29.44
	College degree+	41.39
<b>Wave 1 Social Conflict</b>		
Spouse only (n=1,116)	Range, 1-4	2.22 (0.60)
Friends only (n=1,488)	Range, 1-3.75	1.90 (0.48)
Family only (n=1,491)	Range, 1-4	2.08 (0.58)
Mean Conflict (n=1,498)	Range, 1-3.58	2.05 (0.43)
Max Conflict (n=1,498)	Range, 1-4	2.40 (0.56)
<b>Wave 2 Social Conflict</b>		
Spouse only (n=1,116)	Range, 1-4	2.15 (0.61)
Friends only (n=1,488)	Range, 1-3.50	1.81 (0.49)
Family only (n=1,491)	Range, 1-4	2.01 (0.58)
Mean Conflict (n=1,498)	Range, 1-3.64	1.98 (0.44)
Max Conflict (n=1,498)	Range, 1-4	2.32 (0.57)
<b>10-Year History of Social Conflict</b>		
Spouse Only (n=1,116)	Never highest quartile conflict	66.67
	Highest quartile conflict wave 1	9.68
	Highest quartile conflict wave 2	13.98
	Highest quartile conflict both waves	9.68
Friends Only (n= 1,488)	Never highest quartile conflict	71.84
	Highest quartile conflict wave 1	6.25
	Highest quartile conflict wave 2	14.65
	Highest quartile conflict both waves	7.26
Family Only (n=1,491)	Never highest quartile conflict	69.68
	Highest quartile conflict wave 1	6.91
	Highest quartile conflict wave 2	14.08
	Highest quartile conflict both waves	9.32

Mean Conflict (n=1,498)	Never highest quartile conflict	64.62
	Highest quartile conflict wave 1	11.15
	Highest quartile conflict wave 2	10.81
	Highest quartile conflict both waves	13.42
Max Conflict (n=1,498)	Never highest quartile conflict	67.36
	Highest quartile conflict wave 1	11.88
	Highest quartile conflict wave 2	9.95
	Highest quartile conflict both waves	10.81

**Figure 1: Cortisol Diurnal Rhythm for the Null Model (n=1,498)**



**Figure 2: Predicted  $\ln(\text{Cortisol}+1)$  Over the Day by Wave 2 Mean Social Conflict Score (n=1,498)**



**Table 2: Coefficients and Standard Errors from Spline Models of ln(cortisol+1) as a Function of Wave 2 Social Conflict, (n=1,498)**

	Waking Value	Slope 1: Morning Rise	Slope 2: First Decline	Slope 3: Late Day Decline	Slope 4: Late Night Plateau	Peak	Nadir	AUC
Mean Conflict	-0.029 (0.025)	-0.043 (0.043)	-0.001 (0.007)	0.012** (0.004)	-0.007 (0.020)	-0.050+ (0.027)	0.075+ (0.042)	-0.062 (0.459)
Max Conflict	-0.049* (0.019)	0.012 (0.033)	-0.003 (0.005)	0.006+ (0.003)	-0.002 (0.015)	-0.044* (0.021)	0.006 (0.033)	-0.468 (0.356)

Notes: Other model covariates defined in text. Standard errors in parentheses

+ p<.10, \* p<.05, \*\* p<.01

**Table 3: Coefficients and Standard Errors from Spline Models of ln(cortisol+1) as a Function of 10-yr History of Social Conflict (n=1,498)**

	Waking Value	Slope 1: Morning Rise	Slope 2: First Decline	Slope 3: Late Day Decline	Slope 4: Late Night Plateau	Peak	Nadir	AUC
Mean Conflict (reference: never highest quartile conflict)								
Highest Quartile Wave 1	-0.025 (0.035)	0.070 (0.060)	-0.001 (0.010)	0.010* (0.005)	-0.059* (0.027)	0.010 (0.034)	0.114+ (0.059)	0.736 (0.636)
Highest Quartile Wave 2	0.058 (0.035)	-0.057 (0.060)	-0.008 (0.010)	0.006 (0.005)	0.016 <sup>1</sup> (0.026)	0.029 (0.037)	0.055 (0.060)	0.411 (0.647)
Highest Quartile Both Waves	-0.057+ <sup>2</sup> (0.033)	-0.020 (0.057)	0.008 (0.009)	0.013* (0.005)	0.011 <sup>1</sup> (0.026)	-0.067+ <sup>2</sup> (0.035)	0.096+ (0.055)	0.184 (0.601)
Max Conflict (reference: never highest quartile conflict)								
Highest Quartile Wave 1	-0.032 (0.034)	0.073 (0.057)	-0.004 (0.009)	0.003 (0.005)	0.008 (0.025)	0.004 (0.036)	0.014 (0.056)	0.002 (0.614)
Highest Quartile Wave 2	-0.071+ (0.036)	-0.024 (0.062)	0.003 (0.010)	0.006 (0.006)	-0.007 (0.027)	-0.083* (0.038)	-0.007 (0.062)	-0.772 (0.668)
Highest Quartile Both Waves	-0.086* (0.036)	0.006 (0.061)	0.002 (0.010)	0.007 (0.005)	0.020 (0.029)	-0.083* (0.038)	0.000 (0.060)	-0.741 (0.651)

Notes: Other model covariates defined in text.

Standard errors in parentheses

+ p<.10, \* p<.05, \*\* p<.01

<sup>1</sup> Differs significantly from 'highest quartile wave 1' group at p<0.05

<sup>2</sup> Differs significantly from 'highest quartile wave 2' group at p<0.05

**Table 4: Coefficients and Standard Errors from Spline Models of ln(cortisol+1) as a Function of 10-yr History of Social Conflict, Highest Quartile Conflict, By Type of Conflict and Gender of Respondent**

	Highest Quartile Conflict in Both Waves					
	Men			Women		
	Spouse (n=527)	Friends (n=656)	Family (n=657)	Spouse (n=589)	Friends (n=832)	Family (n=834)
Waking Value	0.015 (0.066)	0.033 <sup>1</sup> (0.069)	-0.133+ (0.072)	-0.072 (0.055)	-0.158** <sup>1</sup> (0.053)	-0.079+ (0.045)
Slope 1: Morning Rise	-0.041 (0.115)	-0.150 <sup>1</sup> (0.118)	-0.040 (0.122)	-0.018 (0.092)	0.129 <sup>1</sup> (0.094)	0.006 (0.078)
Slope 2: First Decline	-0.004 (0.019)	0.008 (0.019)	0.027 (0.020)	-0.007 (0.015)	0.014 (0.016)	0.007 (0.013)
Slope 3: Late Day Decline	-0.007 (0.010)	0.018+ (0.011)	0.001 (0.011)	0.012 (0.008)	0.018* (0.008)	0.003 (0.007)
Slope 4: Late Night Plateau	0.052 (0.054)	-0.077 (0.053)	0.020 (0.047)	0.025 (0.044)	-0.022 (0.046)	-0.003 (0.035)
Peak	-0.005 (0.070)	-0.042 (0.073)	-0.153* (0.076)	-0.081 (0.057)	-0.093+ (0.057)	-0.076 (0.048)
Nadir	0.096 (0.114)	0.172 (0.115)	-0.041 (0.121)	0.019 (0.090)	0.147+ (0.089)	-0.019 (0.077)
AUC	-0.728 (1.216)	0.917 (1.248)	-0.967 (1.305)	-0.844 (1.000)	0.383 (0.967)	-0.666 (0.831)

Notes: Results are from six separate models predicting the diurnal rhythm as a function of 10-yr history for conflict as a four category variable: (1) highest quartile of conflict both waves (displayed above), (2) highest quartile of conflict wave 1, (3) highest quartile of conflict wave 2, and (4) never highest quartile of conflict (reference category). Other model covariates defined in text.

Standard errors in parentheses

+ p<.10, \* p<.05, \*\* p<.01

<sup>1</sup> Significant difference between men and women at p<0.05