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Relative Influences: Patterns of HPA Axis Concordance During Triadic Family Interaction

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Abstract

Objective—Within-family concordance in physiology may have implications for family system functioning and for individual health outcomes. Here, we examine patterns of association in cortisol within family triads.

Methods—A total of 103 adolescents and their parents sampled saliva at multiple timepoints before and after a conflict discussion task. We explored whether within-family associations existed and were moderated by stepparent presence and youth gender, and whether within-family patterns of influence correlated with individuals' aggregate cortisol.

Results—Across the laboratory visit, the cortisol levels of fathers, mothers, and youth were positively associated. In time-lagged models, mothers' cortisol predicted fathers' cortisol levels sampled at the following timepoint, whereas fathers' predicted youths' and youths' predicted mothers' cortisol. These patterns appeared stronger in families not including stepparents. Youth gender moderated some associations: in the aggregate, youth were more strongly linked with their same-gender parent. In time-lagged models, girls were more closely linked to their mothers than boys, and both parents were more linked to girls. Youth showed higher aggregate cortisol output if they were more linked with their mothers, and lower output if more linked with their fathers; parents had higher output if they were more linked with their spouses and lower output if more linked with their children.

Conclusions—These results suggest that family members' physiological activation may be linked during shared interaction, and that these patterns may be affected by family role and by youth gender. Our findings identify specific patterns of physiological influence within families that may inform family systems theories.

Keywords

cortisol; HPA axis; coregulation; family systems; family conflict

Social interactions unfold in a complex dance in which speech, gestures, and affect states become mutually coordinated among interaction partners. This process takes on additional meaning among intimately connected individuals like parents, children, and spouses, where

shared history, as well as situational factors, inform patterns of correspondence. Social baseline theory (Beckes & Coan, 2011) suggests that the tasks of regulating emotions and responding to stress can be distributed across social groups. In other words, while an isolated individual must expend energy resources toward self-regulation of affect and vigilance to threat, an individual in a group can “offload” some of those tasks to others. This account suggests that synchrony in physiological activation might appear in groups of closely related individuals such as families that jointly orient to stressors. However, when individuals’ stress responses match each other too closely, the group function of helping to balance or ameliorate stress responses becomes compromised. Family conflict situations might present one type of occasion in which intercorrelations in family members’ stress responses can be detected.

The hypothalamic-pituitary-adrenal (HPA) axis, which releases the hormone cortisol, reacts to both chronic and momentary stressors and is specifically responsive to social inputs; for example, social evaluative threat (Dickerson & Kemeny, 2004). Dysregulation of this system has been linked with mental and physical health outcomes, including depression, diabetes, and disease progression (McEwen, 1998; Sепhton, Sapolsky, Kraemer & Spiegel, 2000). Therefore, family members’ influences on each others’ stress physiology might be a pathway by which the family environment affects health outcomes. This study examines intercorrelations in cortisol between adolescents and their parents participating in a conflict discussion, looking both in the aggregate (overall cortisol output over the laboratory visit), and at change over time (a family members’ cortisol level at a particular timepoint driving changes in the subsequent cortisol of other family members).

Studies examining within-family correspondences in physiology have generally found modest positive correlations between family members. For example, afternoon basal cortisol levels were correlated among siblings, parents and children, and spouses (Schreiber et al., 2006). Spouses’ cortisol levels were positively correlated over 3 days, and these correlations appeared stronger among more partially dissatisfied partners, and also at times that partners were more likely to be in a shared environment (Saxbe & Repetti, 2010). Mothers’ and adolescents’ cortisol levels were correlated over 2 days, and these correlations were heightened when families were more cohesive (e.g., engaged in more shared activities) or when mothers or adolescents reported more momentary negative affect (Papp, Pendry, & Adam, 2009). Mother–child correlations in cortisol also appeared in mothers and preschoolers participating in a home visit, although only during more challenging portions (Ruttle et al., 2011). Finally, a study of triadic family interaction that included a subsample of the participants in the current study found mother–adolescent symmetry in salivary alpha-amylase, a biomarker of sympathetic arousal. Fathers and adolescents also showed symmetry, but only in family triads with a history of interparental aggression (Gordis, Margolin, Spies, Susman, & Granger, 2010). Taken together, these studies suggest that concordance in physiological activation occurs within families; that concordance may be strengthened during the experience of challenge or negative affect; and that proximity, family cohesion, or family conflict history may also moderate the strength of linkages between couples, parents, and children.

Family systems theory suggests that families are interdependent groups that interact in set patterns that may be influenced by family roles and by the distribution of power within the family—for example, a hierarchical pattern in which parents ally to exercise authority over children (Broszormenyi-Nagy & Spark, 1973). Emotion contagion research has explored how family roles and power contribute to the direction of effects from one family member to another (Larson & Almeida, 1999). For example, parents are more likely to transmit emotions to their children than vice versa, and this transmission appears especially strong from fathers to children (e.g., Almeida, Wethington, & Chandler, 1999; Larson & Richards, 1994). The literature is mixed as to whether husbands are more likely to transmit emotions to their wives or wives to their husbands. Some studies have found husbands to be more receptive to changes in wives' emotions (Schoebi, 2008), while others suggest that wives' emotions are shaped more by their husbands than vice versa (e.g., Bolger, DeLongis, Kessler, & Wethington, 1989; Larson & Richards, 1994). This study's triadic design allows us to directly compare the impact of two family members' cortisol levels on the levels of the third family member and detect whether one or both family members contribute uniquely to the third family members' cortisol over and above the effects of shared variance. We also explore the possible adaptive significance of these effects by examining whether patterns of “relative influence” are associated with the magnitude of cortisol excreted by each family member during the discussion.

Research on within-family linkage in cortisol leaves many unanswered questions. Many of the above-cited studies focused on basal or diurnal cortisol, and most sampled dyads. To our knowledge, this is the first study to examine concordance in cortisol levels during triadic family interaction, and to use time-lagged analyses of cortisol to understand which family members are the “leaders” and which are the “followers” in predicting change in physiological activation over time. In addition, stepparent families might show different or weaker patterns of within-family influence, due perhaps to differences in shared history. Our analyses will explore the possible moderating role of stepparent presence on within-family associations in cortisol.

Research Questions

(1) Within-Family Associations

We hypothesize that family members will generally show positive associations in cortisol, both for momentary time-lagged cortisol levels (1a) and aggregate cortisol output (1b).

(2) Patterns of Independent Influence

We will compare the “relative influence” of each family member; for example, are mothers' cortisol levels independently predictive of children's cortisol levels when fathers' levels are included in the same model? For adults, does one's spouse or one's child have stronger independent influence? We expect patterns of influence to emerge, based on evidence from the emotion contagion literature, but do not have specific hypotheses about directionality.

(3) Moderators

We will examine two potential moderators of within-family correspondence in cortisol: stepparent presence (3a), and youth gender (3b). We expect that families including a stepparent will show weaker linkages in cortisol. We also expect youths' gender to moderate within-family concordance, given evidence that gender may affect transmission of emotions between parents and children (e.g., Gore, Aseltine, & Cohen, 1992).

(4) Patterns of Influence and Aggregate Output

Finally, we will examine whether the direction of influence shown in time-lagged models (parent to child, spouse to spouse, child to parent) is linked with the magnitude of cortisol excreted during the lab visit. That is, how are patterns of within-family intercorrelation linked with overall output for each family member?

Method

Participants

One hundred three family triads consisting of a mother, father, and youth participated in a laboratory-based family conflict discussion, with all procedures approved by the University of Southern California's Institutional Review Board. Participating youth included 50 girls and 53 boys, average age 15.31 ($SD = 0.75$, range = 13.68–18.60). Families were originally recruited to participate in a longitudinal study of the impact of aggressive family conflict behavior on youth development. The sample's ethnic composition reflected the diversity of metropolitan Los Angeles, with 34.7% of the youth identifying as Hispanic/Latino; in addition, 12.5% identified as Asian, 23.4% as African American, 48.4% as Caucasian American, and 15.6% as multiracial or "other." Families were recruited in two cohorts: the first cohort entered the study when the target child was 9–10 years of age, and participated in the conflict discussion as part of their fourth wave of data collection (waves were scheduled 1–3 years apart). The second cohort entered the study when the target child was 12–13 years of age and participated in the discussion as part of their second wave of data collection. Families from both cohorts were recruited from the greater Los Angeles area through newspaper ads, fliers, and referrals from other participants. Eligible families had lived together for the past 3 years and were able to complete the study measures in English at the time of study enrollment; see Margolin, Vickerman, Oliver, & Gordis, 2010, for further details. The two cohorts did not differ in terms of their age at the time of participation, gender, ethnicity, family income, or the starting time of the laboratory visit.

Of 169 families recruited into either the first or second cohort and invited to participate in the current wave, 139 participated in the family discussion task; of these, 16 chose not to provide saliva, 16 families participated in the discussion task with only two members (e.g., the youth and one parent), and four families provided saliva that had not been assayed when this manuscript was prepared. The median combined family income was \$80,000 ($SD = \$66,705$), and 18.6% of families reported an income below \$40,000. Mothers' mean education level was 14.79 years ($SD = 2.72$) and fathers' mean education level was 15.09 years ($SD = 2.41$). Most (88%) of participating families contained two biological parents, but 11 families participating in the discussion included a stepparent.

Procedures

Families visited the lab for a 3- to 4-hour visit, scheduled after 10:30 a.m. to avoid the morning cortisol peak; please see Figure 1 for an overview of the visit and saliva collection times. Parents gave informed consent and youth gave assent. Before their visit, families were informed about all aspects of the discussion task and given the option to decline saliva collection while participating in the discussion. They were instructed not to eat or drink for an hour before the appointment and not to consume tobacco, alcohol, or caffeine for 24 hr prior to the appointment. Compliance was assessed before the saliva collection began and, if families had eaten, saliva collection was postponed to allow for an hour interval after eating. Before beginning the discussion task, families participated in a relaxation induction, viewing a 10-min video with calming images and music, to establish a baseline for cortisol. The first saliva sample was collected (via passive drool or salivettes) after the induction.

Next, the conflict portion of the task began. Each family member was given a questionnaire of 33 common family conflict topics, with the option to write in additional topics, and asked to rate the amount of conflict they typically elicited. Each family member then met with an experimenter in an individual priming interview to identify and describe conflict topics of greatest concern. Three experimenters met separately and simultaneously with one of the three family members in separate rooms. The experimenters then met briefly to identify the three greatest conflict-provoking areas of discussion for each family. Families were seated together in a room and given 15 min to discuss at least one of the three identified topics, starting with the most contentious. Families were instructed to discuss the topic as they would at home, and to “make sure that each of you gets your point across.”

Six saliva samples were collected: at baseline, immediately postdiscussion (baseline + 40 min), and at four postdiscussion timepoints (baseline + 50 min, baseline + 60 min, baseline + 80 min, and baseline + 100 min). The mean baseline sample collection time was 2:10 p.m. ($SD = 2$ hours).

Cortisol Measures

Saliva samples were frozen immediately after the family session and were later shipped in dry ice to Salimetrics, LLC, to be assayed for concentrations of free salivary cortisol, using an enzyme immunoassay with a lower limit of sensitivity of 0.003 $\mu\text{g/dl}$, and intraassay and interassay coefficients of variation of 3.5% and 5.1%, respectively. Each saliva sample was assayed twice, and analyses were repeated if any pair of results differed by $>7\%$. Fifteen samples were dropped because of insufficient quantity of saliva to analyze as determined by Salimetrics.

A conservative approach was taken to handling outliers for two reasons: (1) this study used cortisol as both a predictor and an outcome variable, potentially exaggerating the impact of extreme values; and (2) many factors (compliance, medication use) can affect cortisol values in community samples, and these factors may not vary independently across families. Therefore, we elected to truncate, rather than winsorize or transform, extreme values. Raw cortisol values were examined and any samples that were out of range (>3 SD s above the sample mean) were dropped. This procedure resulted in $<5\%$ of samples being dropped from

analyses. In addition, detailed questionnaires administered at the start of the lab visit assessed medication use and other sources of alterations in cortisol, such as blood contamination. Three dummy variables were created: one indicated the presence of any medication known to affect cortisol, such as inhalers, steroid medications, or antirejection medications; one indicated possible blood contamination, including questions about bleeding gums while flossing, tongue or mouth piercing, and cuts or sores in the mouth; and one indicated consumption of tobacco, alcohol or caffeine within 24 hr of the laboratory visit. One participating mother indicated current pregnancy and was dropped from analyses.

The current study uses two approaches to modeling cortisol: first, a time-lagged multilevel model to examine how two family members' cortisol levels at one sampling timepoint influence the third family member's cortisol at the following timepoint (controlling, also, for within-person autocorrelation, e.g., one's own cortisol sampled during the prior timepoint). This approach reveals how each family member's cortisol level influences other family members' cortisol at the following occasion. Next, area under the curve (AUCg) statistics are used to see whether aggregate cortisol output is correlated among mothers, fathers, and children over the course of a laboratory visit including a family discussion, showing whether family members show similar levels of overall activation over the entire visit.

For multilevel modeling analyses, cortisol concentrations for each sampling point were natural log transformed to correct for positive skew. Because of statistical interdependence between members of the family triad, three separate models were run predicting cortisol for fathers, mothers, and youth. Total cortisol output during the conflict task was evaluated by calculating area under the curve with respect to ground (AUCg; Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) using a trapezoidal formula that included the first five cortisol values and the time elapsed between these measurements.

Results

Time-Lagged Results

To Test Hypotheses 1a (within-family associations in cortisol) and 2 (directions of influence), Hierarchical Linear Modeling (HLM) software (Raudenbush, Bryk, & Congdon, 2004) was used to run time-lagged models for youth, mothers, and fathers. Results are shown in Table 1; for this and for Tables 2 and 3, note that although the three models (predicting youth, mother, and father) are presented together to save space, they were run separately. Each model includes a Level 2 predictor of starting time of the lab visit and four Level 1 coefficients: time between samples, and the three family members' cortisol levels at the prior sampling time-point (the participants' own prior cortisol level was included to control for within-person autocorrelation from the previous time-point). The predictors include the first five cortisol values collected (baseline, baseline + 40 min, baseline + 50 min, baseline + 60 min, baseline + 80 min), and the outcome variable includes five cortisol values that were lagged by one sample collection time-point behind the predictors (baseline + 40 min, baseline + 50 min, baseline + 60 min, baseline + 80 min, baseline + 100 min). Participants' race, cohort (1 or 2), age, and sampling issues (medications, possible blood contamination, and alcohol/caffeine/nicotine consumption) were entered into regression analyses but were not significantly linked with cortisol (whether entered separately or

together) and did not affect any of the results reported below; these variables are therefore not included in final HLM or AUCg analyses.

All three family members showed positive autocorrelation in their own cortisol, such that one's prior cortisol level was significantly linked with cortisol sampled at the following timepoint. In addition, youth cortisol levels were positively and uniquely associated with fathers' prior cortisol levels. Mothers' cortisol levels were positively and uniquely associated with youths' prior cortisol levels. Fathers' cortisol levels were positively and uniquely associated with mothers' prior cortisol levels. Figure 2 depicts cortisol from six families chosen to illustrate some of the possible patterns that emerged within family triads.

Moderators of time-lagged associations—To test Research Question 3a, the same time-lagged models were rerun, but with stepparent presence introduced as a Level 2 predictor on the Level 1 intercept and Level 1 slope coefficients corresponding to the influence of the two other family members. Results are shown in Table 2. Given the fairly small number of families (11 families) participating with a stepparent, stepparent status was calculated at the family level rather than the individual parent level to maximize power. The same overall patterns emerged as in the prior analysis: fathers influenced youth cortisol at the following timepoint, youths influenced mothers, and mothers influenced fathers. These patterns appeared to be weaker when both parents participating in the discussion were not biologically related to the youth, as shown by the negative Level 2 coefficients moderating fathers' Level 1 effects on youth, youths' Level 1 effects on mothers (at a marginal level of significance) and mothers' Level 1 effects on fathers.

Next, to test Research Question 3b, youth gender was introduced as a Level 2 predictor on the Level 1 intercept and Level 1 slope coefficients corresponding to the influence of the two other family members, with results shown in Table 3. Again, main effect results remained similar, such that fathers' cortisol predicted youths' subsequent cortisol, youths' cortisol predicted mothers', and mothers' cortisol predicted fathers'. Gender affected the slope of mothers' cortisol on youths' subsequent cortisol, such that girls were linked more strongly with their mothers. Gender had a marginally significant effect on the slope of youths' cortisol predicting both mothers' and fathers' subsequent cortisol; in both cases, parents were more strongly linked to daughters than to sons. Gender also had a marginally significant effect on the intercept of mothers' cortisol, suggesting that mothers of daughters had higher starting values of cortisol (because time-lagged outcome variables were used, the starting value would correspond to the level of cortisol sampled immediately after the discussion task).

Aggregate Cortisol Results

To test Research Question 1b (within-family correspondence in overall cortisol output) correlations between family members' AUCg values were calculated. Overall cortisol outputs were positively associated among mothers and youth in the same family, $r(87) = .40$, $p < .001$, fathers and mothers, $r(84) = .26$, $p = .02$, and, at a marginal level of significance, fathers and youth, $r(86) = .19$, $p = .08$.

Next, regression analyses including family members' AUCgs were used to examine Hypothesis 2 (directions of influence). The starting time of the lab visit was also entered as a covariate. Because youth gender moderated time-lagged results, the sample was split by gender. All results are shown in Table 4. First, fathers and mothers were entered together to predict youth AUCg. Mothers' AUCg was a unique predictor of girls' and fathers' AUCg a unique predictor of boys' AUCg. Next, when youth and spouse AUCg were entered together to predict mothers' AUCg, both the spouse and child uniquely contributed to the AUCg of mothers of daughters; neither spouse nor child predicted mothers of boys' AUCg. Finally, when youth and wife AUCg were entered to predict fathers' AUCg, fathers of girls showed stronger associations with wives' cortisol, and fathers of boys showed stronger associations with sons' cortisol.

Associations between overall cortisol output and patterns of within-family influence—In a follow-up analysis, the above-described time-lagged models were rerun in HLM (without moderators) and the Empirical Bayesian (EB) estimates of the time-lagged Level 1 coefficients for each model (youth, mother, and father) were extracted from the Level 2 residual file. These estimates represent the extent to which each family member's cortisol is affected by the other family members' prior cortisol levels. Simple correlations were then calculated using these Level 1 slope estimates and the overall cortisol output shown by each family member across the laboratory visit. In other words, this analysis allows us to see whether the absolute magnitude of cortisol excreted by each individual is linked to his or her degree of linkage with other family members. Patterns for boys and girls did not differ, so we report results for the full sample. Youths showed higher overall cortisol output, (AUCg), $r(89) = .36, p < .001$, if they were more strongly linked with their mothers' prior cortisol level in time-lagged analyses, whereas youths who were more strongly linked to their fathers' cortisol showed lower overall output, $r(89) = -.47, p < .001$. Mothers showed higher overall cortisol if they were more strongly linked to fathers' prior cortisol, $r(83) = .26, p = .02$, and lower overall cortisol if they were more strongly linked with their children, $r(83) = -.26, p = .02$. Fathers showed higher cortisol output if they were more strongly linked to mothers' prior cortisol, $r(87) = .26, p = .01$, and lower output if they were more strongly linked with their children, $r(87) = -.46, p < .001$.

Discussion

This study reported on patterns of within-family intercorrelation in cortisol during a laboratory visit that included six saliva sampling occasions. In time-lagged models that tested the influence of each family member's momentary cortisol on the other family members' cortisol levels at the following timepoint, youths appeared to be particularly strongly associated with their fathers, mothers with their children, and fathers with their wives. Associations appeared weaker when the participating family included a stepparent. Moreover, youth gender was associated with the direction of intercorrelations within families at both the aggregate and the time-lagged level of analysis. At the aggregate level, same-gender children and their parents appeared more closely aligned: girls were more strongly linked with their mothers and boys with their fathers; mothers of daughters were more strongly linked with their children and spouses than mothers of sons; fathers of

daughters were more strongly linked with their wives, and fathers of sons were more strongly linked with their sons. At the time-lagged level, girls seemed to drive patterns of activation more than boys: both mothers and fathers appeared more strongly associated with daughters than with sons. Moreover, girls were more strongly associated with their mothers than sons were. Finally, for youths, being more closely synchronized with mothers and less synchronized with fathers was associated with higher overall cortisol output across the laboratory visit, while parents had higher cortisol output if they were more synchronized with their spouses and less synchronized with their children.

Our findings both dovetail with, and suggest new directions for, the existing literature on transmissions of mood and physiology within families. Youth cortisol had unique effects on mothers' cortisol when fathers' cortisol was controlled, in both aggregate and time-lagged analyses. Attunement between mothers and children may have particular evolutionary significance and evidence for physiological and emotional synchrony has been found among mother-child pairs in infancy and early childhood (Sbarra & Hazan, 2008). Fewer studies have described intercorrelations between mothers and adolescents, although one study did detect concordance in cortisol that was linked to both positive and negative relationship phenomena (family cohesion and momentary negative affect; Papp, Pendry, & Adam, 2009). Mothers whose cortisol was more closely linked with their children also had lower overall cortisol across the discussion relative to mothers who were more strongly linked with their husbands; this finding suggests that mothers' physiological synchronization with their children might be adaptive, at least in the context of short-term family conflict. Interestingly, mothers with daughters appeared to be more closely synchronized with their children (in both aggregate and time-lagged models) and with their spouses (in aggregate models) than mothers of sons. These results may be because of greater behavioral involvement, and potentially greater social evaluative threat, in the family conflict discussion among mothers of daughters: the mother-daughter relationship may be both more contentious, but also more intimate, than the mother-son relationship.

Within the emotion transmission literature, fathers have been found to exert particularly strong influences on children (Larson & Richards, 1994), and this finding was echoed by the results of this study: in time-lagged models, fathers' prior cortisol levels had unique effects on youths' cortisol when also controlling for mothers' cortisol and youths' own prior cortisol. In aggregate analyses, gender appeared to play a moderating role: girls were more strongly linked with their mothers, and boys with their fathers. Gender also moderated the Level 1 coefficient for mothers in time-lagged analyses, such that girls were more linked with their mothers. As mentioned, the mother-daughter relationship may be especially fraught; similarly, boys may be more closely aligned with their fathers, at least during family conflict. A study of attachment in middle childhood found that girls reported stronger attachment to mothers, boys to fathers (Diener, Isabella, Behunin, & Wong, 2007).

In time-lagged models, mothers' cortisol had unique effects on fathers' cortisol when youth cortisol was also controlled. Fathers are typically less directly involved in parenting than mothers, even in adolescence (Paulson & Spota, 1996), and some studies suggest that cues within the marital relationship, such as wives' approval of their husbands' parenting practices and confidence in husbands' parenting, predict father involvement with children

(McBride & Rane, 1998). In other words, fathers' family involvement may be shaped by the marital relationship to a greater extent than mothers' involvement (Belsky, Youngblade, Rovine, & Volling, 1991). It is possible that fathers were more vigilant to signs of stress shown by their wives than by their children during the triadic discussion.

Patterns of intercorrelation appeared weaker in families including a stepparent. Although genetic factors might contribute to stronger interconnections between biologically related parents and children, they do not explain linkages between spouses. Family conflict discussions in nonstepparent families might reflect more shared family history, possibly changing the dynamic of these discussions or the degree to which family members are aware of each others' fluctuating moods and stress states. Youth in stepparent families might have lived with only one parent for a period of time, or might have survived the death or departure of the other parent, possibly leading to stronger connectedness within a particular parent-child dyad. These results are preliminary, because we did not have power to calculate stepparent status at the individual level, but suggest that the type or length of a family relationship might moderate physiological linkage.

We found that individuals' overall cortisol output was associated with directions of influence within families. Parents showed higher output if they were more strongly associated with each other and lower output if they were more strongly associated with their children, and youth showed lower cortisol output if they were more associated with their fathers and higher output if they were more associated with their mothers. In general, the time-lagged patterns that emerged as more normative within our data (youths linked with fathers, mothers linked with youth) were associated with lower cortisol excretion, although not for fathers who were typically more strongly linked with their wives than their children. Mothers were the "drivers" or "senders" linked with higher output in both fathers and youth, suggesting that mothers might "set" others' physiology to a higher level of arousal. These findings offer evidence that patterns of within-family influence may have adaptive significance in family conflict situations.

The study's limitations include the fact that we sampled salivary cortisol, an end-product of the HPA axis response, which takes some time (20–30 min; Kirschbaum & Hellhammer, 1989) to reflect the experience of a stressor. This complicates the interpretation of our time-lagged results, particularly because our sampling occasions were not evenly spaced. Future studies may use more frequent or more evenly spaced sampling timepoints and should seek to replicate our findings using other measures of physiological arousal. Additionally, families' shared routines—for example, a shared breakfast or lunch prior to study participation, the shared experience of traveling together to our laboratory, even coordinated sleep/wake patterns - might have strengthened intercorrelations within families. Finally, behavioral characteristics of the discussion, for example, family members' engagement with each other or withdrawal from each other, were not explored in this study but represent an important direction for future research. A related future direction is the need to identify the mechanisms linking changes in one family member's cortisol with changes in another member's cortisol. One possibility is that individuals' HPA axis activation leads to behavioral changes that elicit responses from other individuals, subsequently affecting those other individuals' HPA axis activation. Another possibility is that family members'

physiological responses become coordinated through the release of chemosignals that can be detected by olfaction. While the literature on chemosignals in social communication is very small, there is some evidence that emotions such as fear and disgust can be communicated to others via sweat odor (de Groot, Smeets, Kaldewaij, Duijndam, & Semin, 2012).

Despite these limitations, this study advances research on families and health in several important ways. First, it offers proof of concept that family members' physiology is coordinated during family interaction. And, as suggested by the time-lagged results, this coordination unfolds over time, so that family members contribute to changes in each other's cortisol levels that are not merely "set" by within-person intercorrelation. Learning that families show synchrony not just in terms of behavior, attitudes, and emotions but also in physiology may open up fruitful new directions for research on families and health. Moreover, discovering who "leads" and who "follows" in family conflict situations may have implications for family therapy and for theoretical development in family systems research. For example, to what extent are these patterns entrenched within families, and can they be changed through intervention? How do patterns of relative physiological influence change across the lifecycle, and may they have long-term implications for development, health, and mortality? Future studies that pursue these and other questions can help continue to refine our understanding of how close relationships shape health and well-being.

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Figure 1.
Overview of procedures and cortisol collection timepoints.

Figure 2.

Cortisol patterns of six selected family triads. (A) A rise in the mother's cortisol is followed by an increase in the father's and then the youth's cortisol. (B) A decline in the mother's cortisol is followed by a decline in the father's and then the youth's cortisol. (C) The youth and mother show a decline in cortisol that is followed by a decline in the father's cortisol. (D) A drop in the youth's cortisol is followed by a drop in the mother's and then the father's cortisol. (E) The mother's cortisol rises slightly and falls, followed by a rise and fall in the father's and youth's cortisol. (F) A small rise in the father's cortisol is followed by a rise in the youth's and then the parents' cortisol.

Table 1

Time-Lagged Analyses Predicting Each Family Member's Log-Transformed Cortisol Level From All Three Family Members' Cortisol Levels at the Previous Timepoint

	Intercept: Baseline sample	Level 1 predictors			
		Sampling time (centered around starting time)	Mothers' cortisol (prior timepoint)	Fathers' cortisol (prior timepoint)	Youth cortisol (prior timepoint)
Youth Cortisol	-1.09 (.02); -4.81 ***	-0.01 (.06); -.19	0.06 (.04); 1.53	0.13 (.05); 2.47 *	0.43 (.09); 4.83 ***
Level 2 effect, starting time of lab visit	-0.02 (.01); -2.04 *				
Mothers' Cortisol	-0.70 (.18); -3.97	0.07 (.04); 2.09 *	0.64 (.05); 12.11 ***	0.02 (.03); 0.67	0.11 (.05); 2.08 *
Level 2 effect, starting time of lab visit	-0.02 (.01); -1.80				
Fathers' Cortisol	-0.46 (.17); -2.64 *	-0.02 (.05); -0.49	0.15 (.05); 2.89 **	0.58 (.06); 10.46 ***	0.06 (.05); 1.34
Level 2 effect, starting time of lab visit	-0.01 (.01); -1.09				

*
 $p < .05$.

**
 $p < .011$.

 $p < .001$.

Table 2

Time-Lagged Analyses With Stepparent Presence Included as a Level 2 Moderator

	Intercept: Baseline sample	Level 1 predictors			
		Sampling time (centered around starting time)	Mothers' cortisol (prior timepoint)	Fathers' cortisol (prior timepoint)	Youth cortisol (prior timepoint)
Youth Cortisol	-1.13 (.22); -5.07 [*]	-0.01 (.06); -0.16	0.06 (.04); 1.42	0.12 (.05); 2.31 [*]	0.43 (.09); 4.97 [*]
Level 2, starting time	-0.03 (.02); -1.59				
Level 2, stepparent presence	-0.46 (.24); 1.94 ⁺		-0.03 (.06); 0.49	-0.15 (.07); 2.05 [*]	
Mothers' Cortisol	-0.77 (.17); -4.58 [*]	0.08 (.04); 2.18 [*]	0.65 (.06); 11.13 [*]	0.02 (.03); 0.73	0.12 (.05); 2.46 [*]
Level 2, starting time	-0.02 (.01); -1.62				
Level 2, stepparent presenCe	0.56 (.36); 1.56			0.01 (.04); -0.10	-0.20 (.11); 1.85 ⁺
Fathers' Cortisol	-0.48 (.17); -2.82 ^{**}	-0.02 (.05); -0.46	0.17 (.05); 3.18 [*]	0.57 (.06); 10.10 [*]	0.05 (.05); 1.08
Level 2, starting time	-0.01 (.01); -0.95				
Level 2, stepparent presenCe	-0.38 (.15); 2.62 ^{**}		-0.15 (.04); 3.47 [*]		0.01 (.03); -0.28

⁺
 $p < .10$.^{*}
 $p < .05$.^{**}
 $p < .011$.^{*}
 $p < .001$.

Table 3

Time-Lagged Analyses With Youth Gender Included as a Level 2 Moderator

	Intercept	Sampling time (Centered around starting time)	Mothers' Cortisol (prior timepoint)	Fathers' Cortisol (prior timepoint)	Youth Cortisol (prior timepoint)
Youth Cortisol	-1.20 (.23); -5.23 ***	-0.02 (.06); -0.35	0.04 (.04); 1.12	0.15 (.05); 2.74 **	0.40 (.09); 4.37 ***
Level 2, starting time of lab visit	-0.03 (.02); -1.86 +				
Level 2, youth gender	-0.22 (.29); -0.75		-0.21 (.07); -3.20 ***	0.16 (.11); 1.48	
Mothers' Cortisol	-0.81 (.17); -4.66 ***	0.07 (.04); 2.05 *	0.63 (.05); 11.75 ***	0.02 (.03); 0.63	0.11 (.05); 2.06 *
Level 2, starting time of lab visit	-0.03 (.01); -2.14 *				
Level 2, youth gender	-0.51 (.27); 1.85 +			-0.01 (.07); -0.08	-0.16 (.10); -1.68 +
Fathers' Cortisol	-0.48 (.17); -2.81 **	-0.03 (.05); -0.55	0.14 (.05); 2.87 **	0.59 (.05); 10.74 ***	0.05 (.04); 1.26
Level 2, starting time of lab visit	-0.02 (.01); -1.24				
Level 2, youth gender	-0.45 (.31); -1.46		-0.02 (.09); -0.28		-0.13 (.07); -1.84 +

+ $p < .10$.* $p < .05$.** $p < .011$.*** $p < .001$.

Table 4

Within-Family Intercorrelations in Aggregate (Area Under The Curve With Respect to Ground [AUCg])
Cortisol

<u>Girls' Cortisol AUCg $R(35, 3) = .60, F = 5.98, p = .000$</u>				<u>Boys' Cortisol AUCg $R(40, 3) = .56, F = 5.46, p = .000$</u>			
	Beta	<i>T</i>		Beta	<i>T</i>		
(Constant)		1.87 ⁺			3.23 ^{***}		
Starting time of lab visit	-.11	-.72		-.31	-2.15 [*]		
Mothers' Cortisol AUCg	.59	3.47 ^{***}		.21	1.42		
Fathers' Cortisol AUCg	-.12	-.76		.34 [*]	2.45 [*]		
<u>Mothers of girls' Cortisol AUCg $R(35, 3) = .70, F = 10.43, p = .000$</u>				<u>Mothers of boys' Cortisol AUCg $R(40, 3) = .38, F = 2.07, p = .12$</u>			
	Beta	<i>T</i>		Beta	<i>T</i>		
(Constant)		1.31			1.85 ⁺		
Starting time of lab visit	-.19	-1.38		-.19	-1.17		
Youth Cortisol AUCg	.47	3.47 ^{***}		.25	1.42		
Fathers' Cortisol AUCg	.32	2.49 [*]		.03	0.16		
<u>Fathers of girls' Cortisol AUCg $R(35, 3) = .47, F = 2.98, p = .05$</u>				<u>Fathers of boys' Cortisol AUCg $R(40, 3) = .38, F = 2.07, p = .12$</u>			
	Beta	<i>T</i>		Beta	<i>T</i>		
(Constant)		1.27			0.12		
Starting time of lab visit	-.08	-0.45		-.15	0.88		
Youth Cortisol AUCg	-.15	-0.76		.41	2.45 [*]		
Mothers' Cortisol AUCg	.50	2.49 [*]		.03	0.16		

⁺ $p < .10$.

^{*} $p < .05$.

^{***} $p < .001$.