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Cortisol Reactions During Family Conflict Discussions: Influences of Wives' and Husbands' Exposure to Family-of- Origin Aggression

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Abstract

Objective—Though family-of-origin aggression (FOA) is a known risk for later emotional and physical problems in adulthood, little is known about how early exposure to aggression influences physiological reactivity in the domain-specific context of family conflict experienced as an adult. This study investigates whether report of FOA influences spouses' hypothalamic-pituitary-adrenal (HPA) axis responses during conflict discussions with their family-of-procreation and also whether current hostilities, observed during a family discussion, moderate those responses.

Method—In a sample of 91 families, we measured parents' HPA responses through salivary cortisol total output and discussion-related increase surrounding 15-minute hot-topic discussions that trained observers coded for family members' hostility. Partners' also reported on eight items assessing parent-to-child and interparental FOA.

Results—In models testing within-partner and across-partner influences, wives' higher FOA was linked with increases in their own and their husbands' cortisol. Spouses' own FOA showed significant interactions with the partners' hostility to affect total cortisol output, although in an attenuated direction for wives and a heightened direction for husbands.

Conclusion—The results suggest that HPA responsiveness can elucidate links between family-of-origin experiences and adult intimate relationships and may be a factor in risk and resilience over time.

Keywords

cortisol; dyadic; family aggression; family conflict discussion; family-of-origin; HPA axis

There is increasing evidence that growing up in a hostile and aggressive family can impair physical and psychological health well into adulthood (e.g., Anda et al., 2006; Taylor, Way, & Seeman, 2011). Though it is not fully understood what biological regulatory systems are disrupted by early exposure to family hostility, the study of biosocial interactions provides compelling evidence that the hypothalamic-pituitary-adrenocortical (HPA) axis stress system is particularly vulnerable to early adversity and also plays an important role in wide-ranging

behavioral and health outcomes (Repetti, Robles, & Reynolds, 2011; Tarullo & Gunnar, 2006). When individuals experience severe or prolonged stress, their HPA axis responsiveness may be either too high or too low to adaptively meet environmental demands (Repetti et al., 2011; Shirtcliff, Peres, Dismukes, Lee, & Phan, 2014). However, evidence linking early stressful and threatening experiences with later dysregulated HPA axis activity comes primarily from having adults participate in stressful speech or mental arithmetic tasks (e.g., Carpenter et al., 2007; Goldman-Mellor, Hamer, & Steptoe, 2012), which do not embody the types of adversity salient to the participants' past experiences or the current psychosocial stressors that they normally encounter. Because sensitivity to stress may vary by the nature of challenge task (Obradovi, Bush, & Boyce, 2011), the present study was designed to investigate long-term effects of early exposure to family-of-origin aggression (FOA) on adults' HPA activity within the domain-specific context of family stress. Consistent with calls for biosocial approaches that consider family processes at different developmental stages (D'Onofrio & Lahey, 2010), the overriding aim of this study is to investigate the combined influences of early family adversity and current family hostilities to understand biological responses during stressful family interactions.

Family Stress and HPA Responsiveness

The HPA axis, which follows a distinct diurnal pattern, also sets into motion a set of physiological reactions in response to immediate stressors (Hostinar, Sullivan, & Gunnar, 2014). The perception of a stressful situation mobilizes the HPA system to ready the body for action through the release of adrenocorticotrophic hormone, which then causes the release of cortisol—a process that takes approximately 20 minutes to initiate and another 40-60 minutes for recovery (Dickerson & Kemeny, 2004). Whereas the short-term activation of the HPA axis to acute stressors is adaptive, repeated activation to recurrent stressors can result in allostatic load, where the HPA system is chronically set too high or too low and, most importantly, no longer is responsive to fluctuations in environmental demands. Family conflict or aggression is a unique type of environmental stress because it typically repeats daily or weekly within the same family system and, additionally, may recur with different family members at a much later developmental stage. That is, parents' patterns of aggressive, power-assertive behavior in the family-of-origin may be replicated—albeit with different people and in different forms—once adult children have their own families of procreation.

One means of measuring physiological reactivity to short-term family stress has been the collection of salivary cortisol before and after laboratory-based stressful couple discussions. Cortisol reactivity in this assessment paradigm appears responsive to characteristics of the discussion as well as the relationship more generally. For example, cortisol samples taken before and after couples discuss a high conflict topic in their relationship tend to show post-discussion increases, particularly if couples exhibit high levels of hostility or withdrawal (Heffner et al., 2006; Kiecolt-Glaser et al., 1996; Kiecolt-Glaser et al., 1997). Using the same sample as the present study, Rodriguez and Margolin (2013) reported that husbands showed heightened HPA reactivity associated with marital criticism in the conflict discussion whereas wives showed heightened HPA reactivity only if the discussion criticism was accompanied by marital aggression within the past year. The findings, however, are not

necessarily consistent or robust. As reviewed by Robles and Kiecolt-Glaser (2003), not all couples show an increase in cortisol in conflict discussions. Moreover, recent relationship context in combination with gender appears to contribute variability to HPA response during couples' discussions. For example, there is evidence of HPA hypoactivation, particularly for female partners, when relationship characteristics such as demand-withdraw patterns and partners' avoidance are considered (Brooks, Robles, & Dunkel Schetter, 2011; Laurent et al., 2013).

Despite attention to the ongoing relationship context, no prior investigations to our knowledge have examined the influence of distal relationship contexts such as aggression in the family-of-origin on biological reactions during current family interactions. Incorporating information about FOA aggression when interpreting adults' cortisol reactivity during present-day conflictual family discussions can inform our understanding of lingering influences of early exposure to aggression. In general, adults who report early life adversity are said to experience stress sensitivity. However, a fundamental question is whether this sensitization is evidenced in exaggerated HPA activity when confronted with new stressful situations, or whether over time the HPA axis resets to a lower level of activity and responsivity (Miller, Chen, & Zhou, 2007).

As others have cautioned (e.g., Del Giudice, Ellis, & Shirtcliff, 2011; Hostinar & Gunnar, 2013), high and low cortisol reactions have been known to have both adaptive and maladaptive functions in response to environmental challenges. On the one hand, heightened HPA response to stress might be adaptive for those who experienced the family as threatening at a young age. Responsive cortisol secretion could help such individuals mount resources to deal with current family stressors. Conflict sensitization theories posit that early exposure to family aggression threatens a child's security, which is then exhibited in physiological hyperreactivity, i.e., elevated cortisol levels in response to repeated additional interpersonal stresses (Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; Koss et al., 2013). Although this theory relates primarily to children's reactivity, conflict sensitization also suggests heightened cortisol reactivity to interpersonal challenges as an anticipated reaction for adults (Shirtcliff et al., 2014). For example, a history of sexual abuse, aggression, and family maltreatment are associated with elevated cortisol levels in response to a standardized psychosocial laboratory stressor, particularly among those who currently do not experience psychological problems (Goldman-Mellor et al., 2012; Heim et al., 2000). Thus, individuals with aggressive early family histories may be hypervigilant to later hostile interactions, may more readily perceive everyday family conflict as dangerous, and also may be biologically conditioned to respond with heightened physiological reactivity, even when facing relatively low-intensity family conflicts.

Alternatively, considerable literature suggests that the adversities associated with heightened HPA response in childhood might later contribute to attenuated HPA activity in adulthood (see Repetti et al., 2011 for more details). Early negative family environment has been associated with attenuated cortisol responses to simulated challenges, such as public speaking and standardized role plays during adulthood (e.g., Carpenter et al., 2007; Luecken, Kraft, & Hagan, 2009) suggesting the downward recalibration in physiological set-points often found in response to prolonged stress.

While adverse early experiences appear to be linked with later HPA dysregulation, findings vary. Though blunted cortisol is commonly recognized as a hallmark of chronic stress and adversity, some individuals show the opposite pattern of heightened cortisol reactivity, compared to controls, when they face stress (Heim, Newport, Mletzko, Miller & Nemeroff, 2008). In addition, gender differences, not uncommon in this research, are poorly understood and studies frequently lack direct comparisons of males and females (Hostinar et al., 2014).

Moreover, little is known about whether the link between early family adversity and adults' dysregulated HPA reactivity extends to more common early life stressors and more true-to-life current stressors. In light of evidence that exposure to FOA increases the risk for aggression in the family-of- procreation (Fritz, Slep, & O'Leary, 2012), a particularly relevant concern is how adults with negative childhood experiences respond physiologically to commonplace challenges that arise through conflict and hostility with family members.

Present Study

The aim of the present study is to extend what is known about the long-term physiological consequences of childhood exposure to family aggression in a 'real-life' domain-specific stressful situation of a conflictual family discussion. We address two specific questions regarding HPA activity and family aggression. First, does childhood exposure to FOA have pervasive and prolonged effects that contribute to adult HPA reactivity during family conflict experiences? Based on anticipated influences of conflict sensitization, we hypothesize that FOA will be related to heightened cortisol activity during the family discussions (Hypothesis 1). Second, does observed hostility of couples in combination with childhood exposure to aggression influence HPA activity during a conflictual family discussion? We hypothesize that high current family hostility, in combination with FOA, possibly represents an echoing of adverse experiences in the family-of-origin and thereby could lead to dysregulated cortisol activity (Hypothesis 2) though we are not hypothesizing the direction of dysregulation. We investigate these questions by measuring HPA activity during a three-person conversation involving two parents and their teenage child as they discuss family conflicts that are personally relevant and current. Although we focus on the hostility of each partner as representations of adult negativity, we also control for the youth hostility. We test both of these hypotheses through multi-level modeling so that we can investigate gender differences and cross-partner influences, and also account for within-couple dependencies.

Method

Sample

This study includes 91 couples who participated with their adolescent child in a lab-based conflict discussion and who provided saliva samples surrounding the discussion. These families were part of a larger longitudinal study that examines stress processes in families across time. Eligibility for the larger study required that both parents lived with the child for three or more years at study entry, all three family members were able to complete the procedures in English, and the child was either age 9–10 (original cohort) or in middle school (a second cohort). Out of a total of 139 families who participated in some part of this wave of data collection, 102 families were eligible for this study based the criterion of

having coded interaction data from lab-based triadic discussions. The 37 were ineligible due to the following reasons: 14 only provided questionnaire data from home; in 17 families, only one parent participated in the lab-based video recording with the child; 6 families had technical video-recording problems. Missing FOA data for 4 couples further reduced our sample to 98. We then assessed the remaining 98 couples' reports for unusable cortisol data due to medications (e.g., antidepressants or steroid medication), health conditions (e.g., diabetes), or behaviors (smoking, not sleeping the prior night) likely to alter cortisol concentrations (See Granger, Hibel, Fortunato, & Kapelewski, 2009) or for having systematically elevated cortisol (i.e., every sample > 3 SD above the mean) for unknown reasons. Seven couples were excluded because we could not use the cortisol data from both partners, resulting in a sample of 91. In an additional 10 couples that we retained, we dropped the cortisol data from one partner (6 wives and 4 husbands) due to medications and/or out-of-range scores.

Couples had been married, on average, 15.78 years ($SD = 5.77$). Reflective of the metropolitan community from which they were drawn, 14.3% of couples self-reported being Latino/Hispanic, 28.6% Caucasian/White, 16.5% African American, 9.9% Asian/Pacific Islander, and 30.8% multiple races within the couple. Included spouses had an average age of 46.90, $SD = 6.09$) and participated in this study with their youth (mean age = 15.25, $SD = 0.69$). Wives averaged 14.67 ($SD = 2.43$) years of education and husbands averaged 14.96 years ($SD = 2.62$). Annual family income varied considerably: 23.0% \$50,000, 27.6% \$50,000 – 75,000, 16.1% \$75,000 – 100,000, and 33.3% \$100,000 ($Mdn = \$75,000$). No significant differences emerged in comparisons between the 91 participating families and the 48 non-participating families in parents' age, family income, years of marriage, marital adjustment and exposure to FOA.

Procedure

Data for this study were collected during a 4-hour laboratory session in which couples participated in a three person (mother, father, and youth) conflict discussion task, contributed saliva samples for cortisol assay, and completed self-report questionnaires. The university's Institutional Review Board approved the study procedures, and all participants provided written informed consent or, for youth, assent. Lab-based sessions were scheduled to begin after 10:30 am to avoid the morning diurnal cortisol peak, and participants were instructed to avoid activities (smoking, eating or drinking one hour prior to the meeting) that could compromise saliva assays (Kudielka, Hellhammer, & Wüst, 2009). After 15 minutes of a standardized relaxing activity (watching a video of nature scenes) to establish baseline cortisol levels, family members rated 33 common family conflict issues to identify topics salient to the family. Next, each family member met with a clinically-trained experimenter for a priming interview to identify the most conflictual topics and to prepare them to communicate their personal perspectives on the topics. Based on the ratings and interviews, the experimenters conferred with one another to identify the three most conflictual topics for each triad and then instructed the family to discuss for 15 minutes as many of the three topics as they wished, beginning with the most contentious topic. Immediately after the discussion, family members rated how similar the discussion was to other conflict

discussions outside the lab (0 = *not at all similar* to 4 = *very similar*). The mean rating was 2.87 for wives, and 2.74 for husbands, indicating moderate similarity.

We collected four saliva samples: (a) one following the relaxing video and before the priming interviews and discussion (T1) representing the baseline cortisol level; (b) the second following the priming interviews and immediately after the 15-minute discussion (T1+40); (c) the third at 10 minutes post-discussion (T1+50); and (d) the fourth at 20 minutes post-discussion (T1+60); participants filled out questionnaires concurrent with samples 3 and 4. Saliva samples were frozen immediately following the laboratory visit and later shipped on dry ice to Salimetrics, LLC (State College, PA) where they were assayed for concentrations of free salivary cortisol using an enzyme immunoassay with a lower limit of sensitivity of 0.003 µg/dL, and intra-assay and inter-assay coefficients of variation of 3.5% and 5.1%, respectively. All samples were assayed in duplicate and assays were repeated if any pair of results differed by > 7%. Averages of the two scores were used in the analyses.

Measures

HPA response (salivary cortisol)—Out of a total of 688 cortisol samples from the included spouses, three were above 4 µg/dL and were dropped as values this high may indicate sampling error (Granger et al., 2009). We winsorized 11 cortisol samples (across 7 couples) to $M + 3 SD$ to minimize the impact of outliers. Two indices of cortisol output were calculated using the trapezoidal formulae described by Pruessner, Kirschbaum, Meinlschmid, and Hellhammer (2003): (a) area under the curve with respect to ground (*AUCG*), a measure of total cortisol secretion that includes baseline levels of cortisol output and reflects the overall set point of cortisol; and (b) area under the curve with respect to increase (*AUCI*), a measure of change from the baseline value to assess reactivity or sensitivity to the discussion.

FOA—During their first wave of data collection, participants reported on FOA exposure during childhood (i.e., prior to age 18). Table 1 shows the endorsement rates on the eight FOA items: four assessing parent-to-child physical and verbal aggression, and four assessing inter-parental verbal and physical aggression. Spouses responded on a 7-point scale ranging from 0 (*never*) to 6 (*> 50 times*). For analyses, we used each participant's mean across the eight items. Cronbach's α were .83 for wives and .88 for husbands. Husbands and wives did not differ in their reported level of FOA, $t(90) = -0.32$, n.s.

Wives' and husbands' hostility in the family discussion—A score for each family member's observed hostility was derived from trained coders' ratings of four behaviors: 1) direct criticism of another person or implied rejection of the person; 2) criticism of another person's idea; 3) dismissiveness conveyed through nonverbal behaviors that the other person's communication is not important (e.g. waving off with hands); and 4) disregard conveyed through verbal behaviors that the other person's communication is not important (e.g. failing to address the other's comments). Two coders rated each 3-minute interval of the 15-minute discussion on a 4-point scale (0 = *not at all*; 1 = *a little*; 2 = *moderate*; and 3 = *a lot*). Coders watched and rated the discussions three times (one for each family dyad in counterbalanced order across coders). Then we summed the ratings across dyads to capture

each person's total hostile behavior directed to the two other family members (e.g., wives' hostility included behaviors directed to the husband and youth). For all analyses here, we used the average of the two coders' independent ratings and the mean across the four coded behaviors per 3-minute interval. The summary score was internally consistent (Cronbach's $\alpha = .73$ for wives, .80 for husbands and .84 for youth) and inter-rater reliability was high (ICC = .90 for wives, .91 for husbands, and .90 for the youth).

Control variables—All analyses included; 1) time of day of the first cortisol sample to control for typical diurnal variability of cortisol output, 2) partner's FOA to control for the impact of assortative mating on family history as well as partners' probable awareness and possible sensitivity to one another's FOA, and 3) marital adjustment, assessed by average of partners' scores on the Dyadic Adjustment Scale (DAS; Spanier, 1976), to control for possible effect of the ongoing marital context. When testing hypothesis 2, youths' hostile behavior, rated and calculated in the same way as parents', was entered as the youths' behaviors may be related to parents' hostile behaviors as well as to the parents' cortisol activity. The following other variables were tested for their potential influence in these models due to their possible association with the predictor or outcome variables: each partner's depression symptoms assessed by the Beck Depression Inventory-II (BDI-II) (Beck, Steer, R., & Brown, 1996), participants' age, and couple household income.

Analytic Strategy

To account for the dependency between partners when examining the effects of partners' FOA on cortisol, we used Hierarchical Linear Modeling version 7 (HLM 7; Raudenbush et al., 2011) and modeled our data at the dyadic level by utilizing a two-intercept model (Kenny, Kashy, & Cook, 2006) that allows for the simultaneous modeling of wives' and husbands' cortisol. The following equations specify our models for testing hypothesis 2; for testing hypothesis 1, the Level-1 equation is identical, and only the Level-2 equation includes the terms γ_{11i} , γ_{16i} , γ_{18i} , and γ_{19i} .

$$\text{Level-1: } Y_i = \pi_{0i} \text{HUSB} + \pi_{0i} \text{WIFE} + e_i.$$

where Y_i is the cortisol AUC value for individual i ; *HUSB* and *WIFE* are dummy variables representing husbands' and wives' data, with π_{0i} representing spouses' respective intercepts for the model; and e_i representing within-couple error.

$$\begin{aligned} \text{Level 2: } \pi_{0i} = & \gamma_{10i} + \gamma_{11i} (\text{OwnFOA}) + \gamma_{12i} (\text{OwnHosBeh}) + \gamma_{13i} (\text{PartnerHosBeh}) + \gamma_{14i} \\ & (\text{OwnFOA} \times \text{OwnHosBeh}) + \gamma_{15i} (\text{OwnFOA} \times \text{PartnerHosBeh}) + \gamma_{16i} (\text{PartnerFOA}) + \gamma_{17i} \\ & (\text{YouthHosBeh}) + \gamma_{18i} (\text{MarAdj}) + \gamma_{19i} (\text{Time}) + u_{2i}. \end{aligned}$$

where π_{0i} is predicted by the following: γ_{10} , a constant term representing the mean AUC value across individuals from the Level 1 model; the variables of interest, which included γ_{11} and γ_{12} , the spouse's own FOA and own hostile behaviors (respectively); γ_{13} , the partner's hostile behaviors; γ_{14} and γ_{15} , interactions between own FOA and own and

partner's hostile behaviors; three covariates: γ_{16} - partner's FOA, γ_{17} - youth's hostility, γ_{18} - overall marital adjustment, and γ_{19} - time of day; and u_{2i} , the between-couple error term. The other three covariates (participants' depressive symptoms, age, and family income) were entered one at a time for their possible inclusion as control variables in our models. Including these variables did not change the significance, magnitude, or direction of our other effects, and thus we only report results of parsimonious models excluding these variables. Prior to the main analyses, all predictors and covariates were mean-centered for the purpose of reducing error related to multicollinearity.

Results

Descriptive Statistics and Inter-Correlations

Table 2 displays means and standard deviations for all study variables and their inter-correlations. Wives and husbands differed on only two variables: wives exhibited significantly more hostile behaviors than their husband during the conflict task $t(90) = 2.45$, $p = .018$, and husbands had significantly higher total cortisol AUCG, $t(80) = -2.56$, $p = .010$. Wives' FOA was positively associated with both wives' and husbands' cortisol reactivity (AUCI) during the conflict task. Wives' hostile behaviors were associated with lower total cortisol output (AUCG) for husbands, but husbands' hostile behaviors were not significantly associated with wives' cortisol measures. FOA was not related to hostile behaviors in the discussion for either partner. Wives and husbands' hostile behaviors during the conflict discussion were positively correlated as were wives and husbands' total cortisol output (AUCG).

Multilevel Modeling: Effects of FOA and Hostile Behaviors on Couples' Cortisol

In our initial test of FOA on couples' cortisol (Hypothesis 1), wives' FOA was positively associated with their own cortisol reactivity (AUCI) during the conflict discussion, $B = 28.60$, $t(160) = 2.66$, $p = .007$, but not with their total cortisol output (AUCG), $B = 12.38$, $t(160) = 0.79$, $p = .43$. Although we did not hypothesize cross-partner effects, wives' FOA also was associated with husbands' AUCI, $B = 28.01$, $t(160) = 2.66$, $p = .009$. Husbands' FOA was not significantly associated with their own AUCI, $B = -9.17$, $t(160) = -0.85$, $p = .400$, or AUCG, $B = 13.50$, $t(160) = 1.26$, $p = .211$. Cross partner effects for husbands' FOA also did not emerge.

Results of the model testing interactive effects of spouses' own FOA and hostile behaviors during the conflict discussion (Hypothesis 2) appear in Table 3. For both wives and husbands, spouses' own FOA interacted significantly with partners' hostile behavior to affect total cortisol output AUCG, albeit in opposite directions.

As shown in Figure 1, we decomposed the significant interactions for wives and husbands through follow-up simple slopes analyses testing the associations between partners' own FOA and AUCG when exposed to high (+1 *SD* above the mean) levels of the partner's hostile behavior versus when the partner exhibited no hostile behaviors. For wives who experience high levels of the husbands' hostile behaviors, higher FOA was associated with lower AUCG, $B = -35.70$, $t(150) = -2.97$, $p = .003$. When husbands' exhibited no hostility

during the discussion, wives FOA was positively linked to their AUCG, $B = 44.99$, $t(150) = 2.20$, $p = .003$. When wives exhibited high levels of hostile behaviors, husbands' high FOA was associated with high AUCG, $B = 52.89$, $t(150) = 3.19$, $p = .002$. However, when wives exhibit no hostility during the discussion, there was no relationship between husbands' FOA and AUCG, $B = -16.25$, $t(150) = 0.90$, $p = .371$.

Although no interactions emerged for AUCI, the main effects for wives' FOA and their own heightened AUCI and the husbands' heightened AUCI persisted, regardless of level of hostility exhibited in the discussion. A significant main effect emerged for husbands' FOA, and husbands' higher AUCG, but is best explained through the interaction with wives' hostility. A significant main effect also emerged showing that husbands' hostility was associated with wives' lower AUCI. Partners' own level of hostility did not contribute unique variance to these models.

Discussion

This study illustrates subtle, under-the-skin phenomena indicating that a childhood history of FOA can influence adult's HPA responsiveness during conflict discussions with their current family members. Moreover, through actor-partner couple analyses, the results highlight differences between females and males in their current HPA activity as a function of past and current experiences with family hostility. In partial support of the hypothesized sensitization effect (Hypothesis 1), wives' experiences of FOA were associated with their heightened cortisol reactivity during a conflictual family discussion; this association was not found for husbands. Unexpectedly, however, wives' FOA also was related to husbands' AUCI. In support of the anticipated combined effect of FOA and the partner's current hostilities (Hypothesis 2), wives showed attenuation whereas husbands' showed elevated total cortisol output. Higher FOA along with hostility from the husband was associated with low total cortisol output (AUCG) for wives, but higher FOA along with wives' hostility was associated with high AUCG for husbands. Thus, FOA experiences appear to have some influence on HPA stress regulatory processes during current conflictual family discussions for both men and women, though the nature of the response also depends upon the level of hostility exhibited in the discussion and varies across men and women.

Overall Influence of FOA and HPA Responses (Hypothesis 1)

Two converging results in these data demonstrate that wives' FOA experiences have an enduring impact on their biological responses even when their current family situation is reasonably 'safe': first, women with high FOA show heightened AUCI and, second, when husbands do not exhibit hostility, these women show heightened AUCG. This heightened cortisol response may signify women's enhanced risk detection, even when no objective threat is present, i.e., the husband is not hostile. Previous research has linked the effects of severe early life stress with heightened cortisol activation in children (e.g., Essex, et al., 2011), and also has shown somewhat robust cortisol dysregulation from childhood to adulthood, even beyond current risk (Evans & Kim, 2012; Taylor, et al., 2011). Wives in this study with low hostile husbands still appear to mount a physiological readiness for risk, but

overall their HPA axis may have the opportunity to recover and they thus may avoid a chronic down-regulation.

Though the data do not explain why the heightened reactivity is expressed only in women and not in men who have difficult family-of-origin experiences, it is possible that memory traces of aversive childhood experiences may hold more relevance for women than for men during the family discussions. With cognitive appraisals associated with distinct HPA reactions (Denson, Spanovic, & Miller, 2009), it is possible that men and women anticipate the family discussion with different expectations, which, in turn, could be associated with different HPA responses. Unexpectedly, however, men do show heightened cortisol activity (AUCI) when married to women who report more FOA experiences. We speculate that the men might be showing heightened reactivity in protection of their wives. That is, attuned husbands might be highly aware of what circumstances are upsetting for their wives and might be on alert to ameliorate those situations. There also is growing attention to the biological co-regulation between spouses, which has been associated with the ability to identify the partners' emotions (Reuf, 2001) and to respond in a similar fashion. To more fully understand these findings, it would be helpful to assess what each partner knows of the other's family history.

Interaction between FOA, Partners' Hostility, HPA Responses (Hypothesis 2)

Significant interactions for both women and men between FOA and hostility from the other spouse indicate that childhood experiences can exert long-term influences in combination with current experiences of aggression. With this as the first study to investigate both current and childhood family influences, there is little to guide our explanations of the gender differences in the directions of this effect—overall attenuated cortisol levels for women and overall heightened cortisol levels for men. In prior investigations (Rodriguez & Margolin, 2013), men's HPA responses had been highly connected to the actual conflict discussions. The present findings further qualify men's reactivity as a function of the combination of both past and present experiences as well as their wives' past experiences. For women, whereas FOA alone was associated with heightened cortisol, FOA in combination with husbands' hostility shows a pattern of down-regulation, a pattern that others have interpreted as possibly related to disengagement from the discussion (Feinberg, Jones, Granger, & Bontempo, 2011). Perhaps for women with greater reactivity to FOA overall, the additional dimension of the husbands' hostility is the tipping point to more chronic, negative feedback mechanisms in the HPA axis.

Research Implications

The different HPA patterns between men and women demand more attention in future research. Male-female differences also have emerged in a handful of previous studies, e.g., in dating couples where men with anxious attachment styles showed particularly high reactivity during conflict resolution discussions (Brooks et al., 2011; Powers, Pietromonaco, Gunlicks, & Sayer, 2006) and in couples expecting a first child where intimate partner aggression was associated with men's persisting cortisol reactivity while women showed down-regulation (Feinberg, et al, 2011). In general, the emerging pattern of sex differences in cortisol reactions may suggest that this 'challenge task' of engaging in conflict-based

couple and family discussions holds different meaning for women and men. The cortisol attenuation that women exhibit makes sense if the hostility received from the partner is a marker of repeating and chronic stress stretching from childhood into adulthood. For the men, there may be specific features of these family discussions that are more threatening and evoke more performance pressure: These discussions are semi-public in that they take place in the presence of the child and in front of a team of experimenters, which also might magnify the men's cortisol reactions. Again, greater attention to partners' expectations about how such conversations typically unfold between them and the partner and how those expectations are influenced by early family experiences might help explain the varying gender patterns of cortisol reaction.

By and large, the presumption when interpreting cortisol data is that HPA dysregulation, that is, patterns that veer from the norm whether too high or too low, are problematic, particularly when evidenced in individuals with prior extreme stress. What we do not know, based on prior literature and the results here, is whether the 'dysregulation' might actually signify resilience, particularly if not exhibited at extreme levels. For example, for women with high FOA but low current stress, the heightened AUCI reactions might help prepare them for a potentially challenging situation. For women with high FOA and high husband hostility, attenuated HPA axis activity may indicate that they have built up the psychological resources to cope with repeated stress without initiating a HPA axis stress reaction (Taylor et al., 2008). The link between wives' FOA and husbands' cortisol reactivity may reflect a subtle attunement to their wives' experiences but we do yet know what function it may ultimately serve in the relationship. Future research is needed to identify the implications of physiological deviations from the norm for the partners' mental and physical health and relationship adjustment.

Finally, this study also highlights the importance of timing and context when considering the influence of historical factors and later HPA reactivity (Shirtcliff et al., 2014). For example, whereas the combination of FOA and husbands' observed hostility was associated with wives' attenuated AUCG here, prior analyses with this same sample showed that the combination of marital aggression within the past year and couples' observed conflict was associated with wives' heightened AUCG (Rodriguez & Margolin, 2013). It thus appears that there may be meaningful distinctions if family aggression occurs in the distant past versus more proximal past. Moreover, the spouse being the source of both past and current hostilities may have different implications as wives mobilizes HPA resources for the current marital challenge as contrasted with past hostilities emanating from persons not currently in the room. Further study is recommended to better understand these nuanced differences among historical stressful experiences as well as in the ways that people respond to various ongoing salient social cues.

Limitations

Some limitations deserve notice when considering the study results. First, with the cross-sectional design, the FOA data are based on retrospective reports of each spouse, which could be influenced by more proximal events. We also do not have information about whether the FOA aggression occurred in early childhood, adolescence, or throughout the

relationship with the participants' parents, even into adulthood. The timing of the FOA is worthy of study in future investigations due to growing attention to developmental points of HPA axis calibration (Del Giudice et al., 2011). Second, we do not have information about stressful events intervening between the childhood FOA aggression and the current relationship. Thus, although we look at a repeating pattern of family conflict from childhood to the present, overall HPA activity could be affected by a persistent life-long pattern of cumulative family adversity and stress or by other serious adverse experiences unrelated to the family. Third, our assessment of total FOA aggression does not allow us to disentangle the unique contributions of verbal versus physical FOA family aggression, which are said to have distinct effects on brain function, partially through the mediation of the HPA axis activity (Frodl & O'Keane, 2013). Fourth, our coded data did not simultaneously test for supportive couple interactions, which have been shown to affect HPA activity, particularly for men (Laurent et al., 2013). Fifth, although depression didn't emerge as a significant covariate in our analyses, it is possible that psychological symptoms moderate our pattern of results but, due to power constraints, cannot be tested. Sixth, results from these long married couples who are raising children together may not generalize to other couples. Moreover, the stressor task of a family discussion with the child present is different from dyadic couple conflicts. Nonetheless, for parents at this life stage, conflicts about teenage children and conflicts that actually involve the children themselves are highly common (Papp, Cummings, & Goeke-Morey, 2009).

Clinical and Policy Implications

This study highlights the importance of considering each partner's past family experiences with aggression when interpreting responses to immediate and ongoing conflict. Considerable attention has been paid to ways that FOA is embedded in cognitive, affective and behavioral indices of present day couple and family interactions; results from this study expand that notion to physiological indices. These findings are the clearest indication to date of the importance of assessing both FOA along with current hostilities and psychophysiological reactions to understand the phenomenology of couple interaction. Whereas several theoretical approaches to clinical work with couples emphasize how couples' observed communication patterns might reveal sensitivities related to prior life experiences, the results here illustrate that the sensitivities may actually be logged in biological reactions. Further understanding of the role of FOA in partners' psychophysiology, and the interface between psychophysiology and ongoing interactions are highly relevant to multidimensional explanations of conflict processes.

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Figure 1. Wives' and Husbands' Family-of-Origin Aggression (FOA) and Total Cortisol Output (AUCG) Plotted at High and Low Levels of the Partner's Hostility During Family Discussions

Table 1

Descriptive Statistics for Family-of-Origin Items

Items	Wives (<i>n</i> =91)		Husbands (<i>n</i> =91)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
How often did your parents... before the age of 18?				
Scream or yell at you	3.16	1.94	89	3.19
Insult, swear at, or criticize you	2.24	2.18	66	2.63
Hit or slap you	2.15	1.85	74	2.63
Physically injure or bruise you	0.66	1.73	28	0.69
Scream or yell at each other	2.97	2.16	84	2.73
Insult, swear at, or criticize each other	2.63	2.04	79	2.33
Hit or slap each other	0.64	1.21	27	0.93
Physically injure or bruise each other	0.33	0.75	17	0.49

Note. The anchors for the ratings were: 0 = *never*, 1 = *once*, 2 = 2 to 5 times, 3 = 5 to 10 times, 4 = 10 to 20 times, 5 = 20 to 50 times, 6 = more than 50 times.

^aPercent of participants who endorsed that item at > once.

Table 2

Descriptives and Correlations for All Study Variables

Variable	<i>n</i>	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. FOA-W	91	2.82	1.25															
2. FOA-H	91	2.88	1.43	.06														
3. Hostile behaviors-W	91	0.92	0.81	.16	-.09													
4. Hostile behaviors-H	91	0.71	0.77	.11	.17	.45***												
5. AUCI-W	86	-3.51	89.12	.39***	-.01	-.08	-.20											
6. AUCI-H	90	7.79	121.82	.30**	-.09	.14	.14	-.01										
7. AUCG-W	86	276.23	140.60	.10	-.02	-.19	-.04	.20	.16									
8. AUCG-H	90	325.88	152.98	.05	.09	-.28**	-.16	.02	.04	.40***								
9. Hostile behaviors-Y	90	0.76	0.81	.05	.19	.55**	.31*	-.09	.14	-.16	-.19							
10. Age-W	91	45.66	5.95	.03	-.06	-.07	-.30*	.13	.19	-.02	-.08	.10						
11. Age-H	91	48.26	7.13	.03	.02	-.11	-.37*	.11	.01	.01	.04	.02	.74**					
12. Income ^a	90	92.42K	64.53K	.01	.01	-.19	-.20	.00	-.04	-.13	.05	.06	.25*	.22*				
13. BDI-W	91	8.85	7.86	.17	.07	.02	.24*	-.14	.00	-.05	.03	.01	-.23*	-.23*	-.09			
14. BDI-H	89	7.66	7.47	.16	.24*	.07	.13	.01	.11	-.03	-.05	.03	.01	.01	-.04	.19		
15. DAS	90	107.48	15.37	-.24*	-.11	-.22*	-.38**	-.01	-.08	.04	.09	-.09	.05	.15	.07	-.34**	-.31**	
16. Time ^b	91	2.30	2	.03	-.18	.09	.05	-.23*	-.16	-.17	-.10	.00	.01	.04	-.02	-.04	-.04	-.01

Note: Correlations that included cortisol measures controlled for sampling time. W=Wives, H=Husbands, Y=Youth, FOA = Family-of-origin aggression. AUCI = Area under the curve with respect to increase. AUCG = Area under the curve with respect to ground. BDI = Beck Depression Inventory. DAS = Dyadic Adjustment Scale.

^a Annual household income.

^b Time of first cortisol sample.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

Family-of-Origin Aggression (FOA) and Observed Hostility on Wives' and Husbands' Cortisol Activity: Interactions and Main Effects

Table 3

Variable	Wives (<i>n</i> = 86)				Husband (<i>n</i> = 90)			
	AUCI ^a		AUCG ^b		AUCI		AUCG	
	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>	<i>B</i>	<i>SE</i>
Intercept	139.00	71.31	444.39	100.47	181.49	122.05	414.80	100.74
Own FOA	31.14 *	9.61	5.63	11.64	-11.07	10.80	19.88 *	9.22
Own hostility	-9.58	16.25	-28.84	20.81	5.16	1.89	-0.90	23.83
Partner's hostility	-24.24 *	10.22	30.40	18.90	3.92	19.30	-11.27	22.22
FOA x own hostility	11.07	14.97	-8.55	16.11	9.80	10.44	-16.67	12.91
FOA x partner's hostility	7.18	13.16	-52.99 **	16.05	3.77	11.77	39.76 *	16.87
Partner's FOA	0.20	5.81	-1.32	7.74	27.33 *	11.29	10.18	12.62
Youth's hostility	0.26	11.50	-13.05	18.25	11.90	17.93	-42.83	22.39
Time of first cortisol sample	-10.40	4.95	-11.53	6.20	-12.30	8.69	-5.05	7.11
Marital adjustment	0.11	0.71	-0.24	-0.74	-0.01	0.95	1.63	1.13

Note: *B*= Unstandardized Coefficient. *SE*= Standard Error.

^a Cortisol area under the curve with respect to increase.

^b Cortisol area under the curve with respect to ground.

* $p < .05$.

** $p < .01$.

*** $p < .001$.