



Daily parent-adolescent cortisol associations: Unpacking the direction of effects

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ABSTRACT

Prior studies suggest bidirectional relationships between parent and adolescent behavior. This study examined how parents and their adolescent child's cortisol patterns are associated across days and if there are bidirectional associations between parent and child cortisol. Participants included two samples of employees and their children who participated in a daily diary study where diurnal salivary cortisol was collected on four study days ($N = 318$ dyads, $M_{\text{youth age}} = 13.18$ years, 52 % female). Autoregressive cross-lagged models were used to estimate parent-driven effects (parent cortisol effects on adolescent cortisol) and adolescent-driven effects (adolescent cortisol effects on parent cortisol). Adolescents' steeper cortisol awakening response (CAR) was significantly associated with parents' steeper CAR the following day. Adolescents' higher bedtime cortisol levels were also significantly associated with parents' higher bedtime cortisol levels the following day. Parents' cortisol did not predict their children's next-day cortisol. Results support a primarily adolescent-driven process of stress transmission in families. These results suggest that interventions to reduce adolescent stress, as well as to reduce parents' reactivity to adolescents, may be warranted.

1. Introduction

Studies on parenting and adolescent behavior provide evidence that parents and adolescents have a bidirectional influence on one another (Pardini, 2008). Little is known about the extent to which parents and adolescents affect each other's physiological functioning, however. Cortisol, the hormonal byproduct of the hypothalamic-pituitary-adrenal (HPA) system, is an important physiological marker of stress and arousal that is linked to a range of health outcomes (Piazza et al., 2010). Cortisol exhibits a diurnal pattern, with levels rising in the first 30–45 min after waking and then declining until reaching a nadir in the evening hours (Lovallo and Thomas, 2000). This pattern, however, is prone to deviations, and unhealthy cortisol patterns, such as blunted (lower) awakening responses and high bedtime levels, have been linked to adverse psychological and physical health outcomes (Adam et al., 2017). Research indicates that family members' cortisol patterns are associated within a day (Papp et al., 2009), but the associations across days and the direction of effect is unclear (i.e., whether parents affect their adolescents' cortisol, if adolescents affect their parents' cortisol, or both). Understanding the direction of effect has important implications

for who to target in preventive interventions to reduce physiological stress responses in families. In this study, we test the direction of effects between parents' and their adolescent-age children's cortisol patterns across a four-day study period.

1.1. Transactional models of family relationships

Transactional models posit that relationships within the family are dynamic and bidirectional. Studies on parenting and child behavior have demonstrated that parents affect children and vice versa (Sameroff, 2009). Child and adolescent behavior problems and temperament have bidirectional linkages with a range of parenting behaviors, such as parental monitoring, warmth and involvement, and discipline (Burke et al., 2008; Fite et al., 2006; Gross et al., 2008; Laird et al., 2003; Lengua and Kovacs, 2005; Lee et al., 2013; Pardini et al., 2008) and parental depression (Gross et al., 2008).

Although research highlights bidirectionality in behavior between family members, little empirical work has been done regarding bidirectionality in physiology between family members. Parents and children exhibit attunement during infancy: Parents and infants engage in

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an interactive process, where the behavior, affect and physiology of one affects the other. Prior studies have found that parents and infants sync up and match each other in physiological responses such as cortisol and vagal tone (See Feldman, 2007). However, we know very little about how parents and children affect each other's physiological processes in adolescence, including diurnal patterns of cortisol.

1.2. Associations between parent and adolescent cortisol

Prior research supports a transactional perspective: The cortisol of family members are linked. Studies on adults in romantic relationships reveal synchrony in cortisol between partners (Liu et al., 2013; Saxbe and Repetti, 2010). When one marital partner's cortisol rises, so does the other partner's. There is a small, but growing, body of research suggesting synchrony between the cortisol of parents and their children (Papp et al., 2009; Saxbe et al., 2014; Williams et al., 2013), with some studies showing this synchrony is stronger when there are high levels of negative affect (Papp et al., 2009), challenge, or stress (Pratt et al., 2017; Ruttle et al., 2011a,b; Saxbe et al., 2014). However, many of these studies have focused on young children (Pratt et al., 2017; Ruttle et al., 2011a,b; Sethre-Hofstad et al., 2002) or cortisol in response to a lab-induced stressor, rather than daily cortisol patterns in participants' naturalistic settings (Saxbe et al., 2014; Sethre-Hofstad et al., 2002). Only one prior study has addressed the direction of effect between parents' and children's cortisol, and this study assessed short-term reactivity in cortisol to a lab-based stressor (Saxbe et al., 2014). Adolescence is a time of higher stress and stressor reactivity than earlier developmental periods; making it a critical period to understand parent-adolescent cortisol associations (Lippold et al., 2016; Romeo, 2010). We know little about the direction of effect between parents and their adolescent children's daily patterns of cortisol (i.e., whether parents affect their adolescents' cortisol, if adolescents affect their parents' cortisol, or both).

1.3. The moderating roles of gender, age, and contextual factors

Adolescent age, gender, and family contextual factors such as negative parent-child interactions, negative affect, time together, and levels of family stress may moderate the associations between adolescent and parent cortisol (Davis et al., 2018). Compared to younger children, adolescents have more autonomy and independence in structuring their daily lives (Wray-Lake et al., 2010). There is some evidence that parent effects on adolescent behavior may become weaker and adolescent-driven effects on parenting may become stronger as adolescents become older (Lansford et al., 2018). Although not yet studied, it is possible that as youth become older, the effects of parent cortisol on adolescent cortisol become weaker. In addition, the effects of adolescent cortisol on parent cortisol may become stronger. Findings on gender are mixed. Some studies have found stronger associations between parents and their adolescent daughters' cortisol compared to their sons' (Saxbe et al., 2014), but other studies have found no moderation by gender (Papp et al., 2009). Most studies on parent-child cortisol associations have focused on mothers only (Papp et al., 2009; Pratt et al., 2017; Ruttle et al., 2011a,b), and it is unknown if the same process occurs with fathers. One study (Saxbe et al., 2014) found differences by parent gender: Fathers', but not mothers', cortisol predicted child cortisol. However, youths' cortisol predicted mothers', but not fathers', cortisol. Overall, findings are mixed, and gender effects are unclear. Prior studies on cortisol synchrony suggest that the associations between adolescent and parent cortisol may vary based on contextual factors. For example, studies show stronger cortisol synchrony between husbands and wives (Papp et al., 2013) and mothers and adolescents (Papp et al., 2009) when they spend more time together. Other studies have found that the associations between romantic partners' cortisol is stronger under conditions of marital strain and dissatisfaction (Laws et al., 2015; Liu et al., 2013; Saxbe and Repetti, 2010) with some evidence that

parent-adolescent synchrony may also be stronger under conditions of high negative affect (Papp et al., 2009). Thus, the bidirectional links between parent and adolescent cortisol may also be stronger when parents and adolescents spend more time together, have lower relationship quality marked by more negative interactions, and experience greater negative affect and stress.

1.4. This study

In this study, we tested (a) the direction of effects between parents' and their adolescents' cortisol across four days of measurement and (b) whether the associations were moderated by adolescent age or gender, parent gender, and family contextual factors (time together, negative affect, negative parent-child interactions, and levels of family stress). We hypothesized that (a) there would be bidirectional linkages between daily parent and adolescent cortisol (e.g., adolescent cortisol would predict parent cortisol the next day and vice versa) and (b) parent-driven effects (e.g., parent effects on adolescent cortisol) will be weaker and adolescent-driven effects (e.g., adolescent effects on parent cortisol) will be stronger for younger, compared to older, adolescents. We also hypothesize that both the parent-driven and adolescent-driven effects of cortisol will be stronger when parents and adolescents spend more time together and have higher levels of negative parent-child interactions, negative affect, and stress. We do not posit hypotheses regarding gender.

2. Material and methods

2.1. Participants and procedure

Parent-adolescent dyads participated in the daily diary component of the Work, Family Health Study (WFHS; Bray et al., 2013). The study included two samples: employees in (a) an IT division (IT) of a U.S. Fortune 500 company and (b) a nursing home (NH). Employees who were the parent of a child age 9–17 who lived at home for at least 4 days a week were recruited for a home interview, and if both employee and adolescent completed the home interview, they were eligible to participate in a daily diary study involving eight consecutive evening telephone calls and saliva collection. On the calls, the target parent and adolescent provided information on their daily activities, emotions, and experiences. Consent was obtained from study participants. All procedures were approved by the university Institutional Review Board. Authors had no conflicts of interest.

On four diary study days (Days 2, 3, 4, and 5), employees and adolescents collected saliva samples at four time points: upon awakening, 30 min after waking, before dinner, and before going to bed. During the home interviews, saliva collection kits with instructions were distributed. Each kit contained 16 salivettes for collecting participant cortisol (4 salivettes/day for 4 days) along with a DVD that demonstrated saliva collection procedures. Parents and adolescents were instructed to roll a cotton swab across their tongue for two minutes and then return the swab to the tube without touching it and were told not to eat, drink, or brush their teeth within 30 min prior to collection. Instructions for saliva collection and completing the data collection sheet were reviewed with parents and adolescents during the first phone interview, and participants were reminded about the saliva collection on the evenings prior to scheduled collections. Participants recorded the time of each saliva sample (using an electronic time stamper) and any medications they were taking on a separate data collection sheet. Participants refrigerated saliva samples after collection and, at the end of the saliva collection period, mailed the samples to the laboratory using prepaid overnight delivery. Upon receipt at the laboratory, saliva samples were weighed and frozen at -80°C until later assay of cortisol in the Biomarker Core Laboratory at The Pennsylvania State University lab using commercially available EIA kits (Salimetrics, LLC, State College, PA). Assays were run on a rolling basis throughout

the entire study period. The assay had a lower limit of sensitivity of 0.003 ug/dL, with average inter- and intra-assay covariances of less than 7% and 4%, respectively. Cortisol values below 0.003 ug/dL were designated as off-the-curve low and were set to the lowest level of sensitivity to the assay. We converted cortisol values to nmol/l and log transformed them for analyses.

In order to maximize statistical power and to test models on a diverse sample, we combined participants in both the IT ($n = 132$) and NH ($n = 186$) samples for a total of 318 parent-adolescent dyad participants. Samples were similar in adolescent age (ranged 9–17 years, $M = 13.40$ IT; 13.03 NH) but were different regarding gender (45 % IT, 96 % NH mothers; 55 % IT, 51 % NH girls), education (78 % IT, 64 % NH college graduates), income (\$116,900 average income, $SD = \$26,396$ IT; \$56,660, $SD = \$31,759$ NH) and race (Caucasian 74 % IT, 60 % NH; Asian 16 % IT, 3% NH; African American 2% IT, 14 % NH; Pacific Islander 2% IT, < 1% NH; more than one race 7% IT, 4% NH; other 3% IT, 15 % NH; Of these 9% IT and 15 % NH identified as Hispanic). In our combined sample, we have 233 mothers and 80 fathers (5 were missing gender data) with 167 daughters and 151 sons. To assess potential differences in findings between our samples, we tested for moderation by industry in all analyses. Because we found no differences in our findings between our two samples, we present results for the combined sample.

2.2. Measures

2.2.1. Cortisol measures

We used two indicators of the HPA diurnal rhythm: morning rise in cortisol and bedtime levels of cortisol. Cortisol Awakening Response (CAR; morning rise) was calculated as the difference in cortisol levels at wake and thirty minutes post-wake. Bedtime levels were assessed as levels of cortisol at the end of the day. Cortisol values were converted to nmol/l and natural log transformed before analysis (Adam and Kumari, 2009). Prior studies suggest these two indicators are important for health and that on a daily level, a steep CAR and low bedtime levels of cortisol are indicative of a healthy diurnal rhythm (Adam and Kumari, 2009; Piazza et al., 2010).

2.2.2. Moderators and control variables

Similar to previous cortisol studies (Almeida et al., 2017), we also controlled for demographics [adolescent age, race/ethnicity (0 = *White*, 1 = *non-White*), parent education (0 = *no college education*, 1 = *college education*)] and cortisol-related variables [whether adolescents were taking any medications (0 = *no medication use*; 1 = *medication use*), time of cortisol sample collection, cortisol protocol compliance (0 = *compliant sample*; 1 = *non-compliant sample*), tobacco use (1 = *never use*; 2 = *use some days*; 3 = *use most days*), exercise (number of times exercised for 20+ min in past 4 weeks), type of day (0 = *weekday*, 1 = *weekend*) and average daily stressors (average number of reported daily stressors across the week using the Daily Inventory of Stressful Experiences (DISE; Almeida et al., 2002). Tests of moderation included adolescent and parent gender (0 = *male*, 1 = *female*), and age. Additional family contextual variables were averaged across reporter and study days including average weekly stressors (using DISE), negative parent-child experiences (7 items assess frequency of negative parent-child interactions such as yelling and criticism; Lippold et al., 2016), negative affect (11 items adapted from the Positive and Negative Affect Scale (PANAS) to capture daily mood; Watson et al., 1988), and parent-child time together (time parents and adolescents spent doing 5 activities such as schoolwork or hanging out; Almeida and McDonald, 2005). Each day between 89.5–92.3 % of adolescents and 93.6–95.5 % of parents were compliant with cortisol procedures.

2.3. Data analysis plan

Autoregressive longitudinal cross-lagged models (one-day lag) were

run across four days of measurement using Mplus. Models assessed parent-driven paths (i.e., how parent cortisol affects adolescent cortisol the following day) and adolescent-driven paths (i.e., how adolescent cortisol affects parent cortisol the following day). Models also included stability paths for parent and adolescent cortisol, which allows the cross-lagged paths to be estimated while controlling for previous levels of cortisol. Variables in the same day were correlated. A series of steps were used to assess the best fitting, most parsimonious model. First, we fit our baseline model, in which all cross-lagged and stability paths were freely estimated. Next, nested models were used to test whether model paths could be constrained to be equal across days. We tested if model paths of the same type (e.g., parent-driven cross-lagged paths) can be constrained to be equal by using invariance tests, comparing the fit of a model in which specific paths are constrained to be equal to the fit of a model in which paths are freely estimated. Separate tests were conducted for parent and adolescent cross-lagged and stability paths. Model goodness-of-fit was assessed according to strict likelihood ratio principles and included chi-square tests and fit indices. The following criteria indicated acceptable model fit: Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) values of .90 or higher and a root mean square error of approximation (RMSEA) value of .08 or lower (Chen et al., 2008; Hu and Bentler, 1999; West et al., 2012). Missing data were handled using full information maximum likelihood procedures (Enders, 2010).

Lastly, we tested whether our models differed based on parent or adolescent gender, adolescent age, or family contextual factors using two-group autoregressive cross-lagged models. For gender, we ran a series of nested models that compared the fit of a model with the adolescent-driven and parent-driven cross-lagged paths were constrained to be equal between genders to a model where the paths are freely estimated. We tested moderation by parent and adolescent gender separately. We tested for moderation by age by comparing the fit of a model with the model paths constrained to be equal between older and younger adolescents (based on a median split) to a model where these paths were freely estimated. We tested for moderation by family contextual factors by comparing the fit of a model with the model paths constrained to be equal between families high and low on time together, negative experiences, negative affect, and levels of family stress (based on median split).

3. Results

3.1. Descriptive statistics

Study variable means and correlations (calculated using the average of each cortisol indicator across 4 days) are shown in Table 1. Moderate correlations were found between average adolescent and parent CAR ($r = .19$) and average adolescent and parent bedtime cortisol levels ($r = .37$). Adolescents' age was correlated with adolescent cortisol variables but not parent cortisol variables. Parent gender was correlated with parents' CAR and adolescents' bedtime levels, whereas youth gender was only correlated with adolescents' CAR.

3.2. Main effect models

3.2.1. Cortisol awakening response (CAR) models

Nested models revealed that adolescent stability paths and adolescent-driven and parent-driven cross-lagged paths for CAR could be constrained to be equal across time points: The difference in chi-square between the model where these paths were constrained to be equal were not significantly different from those where they were freely estimated (See Table 2). Significant differences did emerge in models where the parent stability paths were constrained to be equal from a model where they were freely estimated. In our final model, only the parent stability paths were allowed to vary across the time points. The overall chi-square of the model was not significant ($\chi^2 = 170.2$, $df =$

Table 1
Descriptive Statistics.

	1.	2.	3.	4.	5.	6.	7.
1. Child Cortisol Awakening Response							
2. Parent Cortisol Awakening Response	.19**						
3. Child bedtime cortisol level	-.14*	.02					
4. Parent bedtime cortisol level	-.13*	.04	.37***				
5. Child gender	.09**	-.05	.09	.01			
6. Parent gender	.14	.13*	.14*	.07	-.03		
7. Child age	.04*	.04	.22*	.08	.05	-.01	
Mean (SD)	.23 (.41)	.26 (.44)	.86 (.51)	1.02 (.44)	52 % girls	74 % mothers	13.18 (2.28)

* $p < .05$, ** $p < .01$, *** $p < .001$.

Note: Correlations were calculated using the average of cortisol variables across four days.

Table 2
Testing Nested Models.

	$\Delta \chi^2$	df	Invariant Paths
Cortisol Awakening Response			
Parent-driven cross-lag paths	1.19	2	yes
Child-driven cross-lag paths	5.00	2	yes
Parent stability paths	7.46*	2	no
Child stability paths	1.41	2	yes
Bedtime Level			
Parent-driven cross-lag paths	0.10	2	yes
Child-driven cross-lag paths	0.80	2	yes
Parent stability paths	0.10	2	yes
Child stability paths	8.63**	2	no

* $p < .05$, ** $p < .01$, *** $p < .001$.158, $p = .24$). Model fit was good (RMSEA = .02, NNFI = .94, CFI = .92).

Fig. 1 shows results of final cross-lagged model for CAR. Adolescent-driven but not parent-driven effects for CAR were significant. When controlling for prior levels, steeper adolescent CAR was significantly associated with steeper parents' CAR the following day ($B = .10$, $SE = .04$, $p = .005$). However, parents' CAR was not associated with adolescents' CAR the following day ($B = -.02$, $SE = .04$, $p = .67$).

3.2.2. Bedtime cortisol models

Nested models revealed that parent stability paths and adolescent-driven and parent-driven cross-lagged paths for bedtime cortisol levels could be constrained to be equal across time points: The model where these paths were constrained to be equal were not different from those where they were freely estimated (see Table 2). However, significant differences did emerge in models where the adolescent stability paths were constrained to be equal from a model where they were freely estimated. In our final model, only the adolescent stability paths were allowed to vary from each other across the time points. The overall chi-square of the model was not significant ($\chi^2 = 162.46$, $df = 158$, $p = .39$). Model fit was good (RMSEA = .02, NNFI = .94, CFI = .93).

Consistent with the prior model, adolescent-driven but not parent-

driven effects were significant. Higher adolescents' bedtime cortisol levels were significantly associated with higher parent bedtime cortisol levels the following day ($B = .14$, $SE = .08$, $p = .000$). By contrast, parents' bedtime cortisol levels were not associated with adolescents' bedtime cortisol levels the next day ($B = .03$, $SE = .04$, $p = .33$).

3.3. Moderation by gender, adolescent age, and contextual variables

There was no evidence of moderation by youth gender, parent gender, adolescent age or family contextual variables. Nested models revealed that there were no significant differences between a model where cross-lagged model paths were freely estimated from a model where they were constrained to be equal between boys and girls (bedtime models: $\Delta \chi^2 = 2.76$, $df = 6$, $p = .83$; CAR models: $\Delta \chi^2 = 3.80$, $df = 6$, $p = .70$), mothers and fathers (bedtime models: $\Delta \chi^2 = 8.36$, $df = 6$, $p = .22$; CAR models: $\Delta \chi^2 = 3.80$, $df = 6$, $p = .70$), and younger and older children (bedtime models: $\Delta \chi^2 = 2.62$, $df = 6$, $p = .85$; CAR models: $\Delta \chi^2 = 7.6$, $df = 6$, $p = .27$). There were no differences in nested models comparing paths between families high and low in negative parent-child interactions (bedtime models: $\Delta \chi^2 = 4.59$, $df = 6$, $p = .59$; CAR models: $\Delta \chi^2 = 4.33$, $df = 6$, $p = .63$), negative affect (bedtime models: $\Delta \chi^2 = 5.01$, $df = 6$, $p = .54$; CAR models: $\Delta \chi^2 = 3.48$, $df = 6$, $p = .75$), parent-adolescent time together (bedtime models: $\Delta \chi^2 = 8.66$, $df = 6$, $p = .19$; CAR models: $\Delta \chi^2 = 9.99$, $df = 6$, $p = .13$), and levels of weekly stressors (bedtime models: $\Delta \chi^2 = 3.04$, $df = 6$, $p = .80$; CAR models: $\Delta \chi^2 = 4.71$, $df = 6$, $p = .58$).

4. Discussion

Theories have increasingly focused on bidirectional relationships between family members (Sameroff, 2009). Studies on behavior have supported these theoretical developments: parenting affects adolescent behavior during adolescence and adolescent behavior affects parenting behavior (Pardini, 2008). Studies on bidirectional relationships between parent and adolescent physiology, such as HPA functioning, have been limited. In this paper, we assessed the direction of effect between parent and their adolescent-age children's daily cortisol patterns. Given

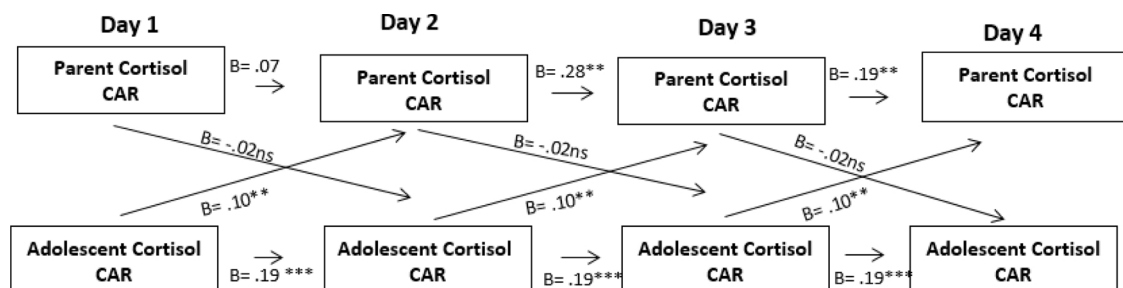


Fig. 1. Covariation between daily adolescent and parent Cortisol Awakening Response (CAR). Steeper adolescents' CAR was significantly associated with steeper parents' CAR the following day but parents' CAR was not associated with adolescents' CAR the following day. Control variables include child age, gender, race, time of cortisol sample, medication use, cortisol compliance, type of day, and daily stressors. Parent and child cortisol collected in the same day were correlated.

transactional models of development (Sameroff, 2009) and prior studies on behavioral indicators such as conduct problems and depression (Pardini, 2008), we expected bidirectional associations between parent and adolescent cortisol.

Interestingly, we found that the association between adolescent and parent cortisol is primarily an adolescent-driven process. Counter to our hypothesis, there was no bidirectional association between parent and adolescent cortisol. Adolescent cortisol predicted parent cortisol (CAR, bedtime levels) the following day. However, parent cortisol did not predict adolescent cortisol the following day. These adolescent-driven effects were significant and invariant across days and emerged even when controlling for average daily stressors. Thus, adolescent-driven effects found in our study may be indicative of a relatively stable process that emerges in families. The presence of adolescent effects on parent cortisol is not surprising, given prior theory and a growing literature that finds adolescent behavior to affect parenting behavior in general (Lansford et al., 2018; Pardini, 2008). Our study supports other work on parent-adolescent cortisol synchrony, in that parent and adolescent cortisol were indeed linked during adolescence (Papp et al., 2009; Saxbe et al., 2014). However, it extends this line of work by revealing that the direction of effect between adolescent and parent cortisol across days is primarily adolescent-driven.

It is possible that our findings reflect parental reactivity to child temperament during adolescence. That is, parents may react to underlying physiological dispositions in their adolescents, such as irritability, impulsivity, sociability, adaptability, and activity level (Chess and Thomas, 1991; Putnam et al., 2002). Child temperament is a relatively stable characteristic of children. Although its manifestations may change depending on environmental conditions and age, several studies have found nervous system and genetic differences underlie many temperamental differences (Wachs, 2006). Child temperament has been linked to differential responses to parenting, with certain temperamental characteristics (e.g., sociability) eliciting more positive parental responses (Putnam et al., 2002; Wachs, 2006). Increased cortisol reactivity has been associated with child characteristics such as fearfulness, anger-proneness, and negativity during earlier developmental periods (Fortunato et al., 2008; Talge et al., 2008; van Bakel and Riksen-Walraven, 2004). Thus, in our study, changes in parents' cortisol patterns may be emerging in response to child temperamental characteristics, which are reflected in their adolescent-aged children's cortisol patterns.

A second possibility is that our study findings reflect differences in social roles, responsibilities, and goals between parents and adolescents (Burton, 2007). According to the generational stake theory, adolescent behavior may focus on the goal of obtaining their own independence and autonomy, whereas parents' goals are oriented towards intergenerational continuity and caring for their offspring (Bengtson, and Kuypers, 1971). Parents, given their role as caretaker, may be more attuned and sensitive to the needs of their adolescents and more responsive to adolescent stress and cortisol patterns than vice versa. For example, when adolescents have high bedtime cortisol levels, their parents may engage in efforts to help their adolescents calm down to prepare for sleep. As a result of their efforts to be responsive to their child's needs, parents' own bedtime levels of cortisol may increase the following day. Similarly, parents may worry about their adolescents' stressors and well-being, and it may impact their own cortisol levels on the following day (McBride et al., 2002; Seginer et al., 2002). In contrast, adolescents may be less aware of their parents' energy levels, needs, and stresses. In fact, adolescents may not be aware of their parents' unhealthy cortisol levels, such as difficulty activating energy in the morning or calming down at night before bed. Even if aware, adolescents may not necessarily be responsive or engage in activities to aid their parents, given their focus on obtaining their own separate identity and autonomy (Bengtson, and Kuypers, 1971). Thus, parents' cortisol may be affected by their adolescents' cortisol due to their attunement and responsiveness to their adolescents' well-being.

Given prior theory, it was surprising that parent-effects were not found in our data. The lack of parent-driven effects may be due to the age of adolescents in our sample, who ranged in age from 9 to 17. Although we found no evidence of moderation by the age, it is possible differences would have emerged if our study included younger children. Parents' effects on children's cortisol may be significant or stronger when children are younger than age 9. Indeed, one prior study on bidirectional effects between parenting and youth behaviors across ages 8–12 found more parent-effects on child behavior when children were younger (Lansford et al., 2018). The same may be true for physiological reactivity and HPA functioning, in that parent effects on children may be stronger earlier in development. Youth cortisol during adolescence may be more strongly predicted by environmental factors outside of the family, such as school or peers, rather than by experiences with their parents (Blackhart et al., 2007).

There was no evidence of moderation in our study. Our study findings did not vary by gender of the parent or child, and cortisol associations were similar for mothers and fathers with boys and girls. These findings support some other cortisol studies during adolescence that found no evidence of adolescent gender effects (Papp et al., 2009) but differs from other studies that have found gender differences (Saxbe et al., 2014). Given prior mixed findings in the literature and the dearth of studies that include fathers, more replication of these findings are needed. Findings also did not differ based on family contextual factors such as the level of negative parent-child experiences, negative affect, family stress and time together. These findings were surprising given some prior studies on cortisol synchrony (Liu et al., 2013; Papp et al., 2009; Saxbe and Repetti, 2010) have found contextual family factors to be important moderators. It is unclear whether differences in findings reflect differences in measures, methods, or samples and further replication is needed. Given the lack of moderation, this study suggests the adolescent effects on parent cortisol are universal, at least across the constructs measured here.

4.1. Strengths and limitations

This study has strengths and limitations that are important to consider. This study utilized daily diary data, allowing us to examine the diurnal rhythm of cortisol. Aspects of the diurnal rhythm, such as the morning awakening response or bedtime levels have important long-term health implications (Adam et al., 2017) and may be particularly important for health and well-being. However, our study does not inform shorter or longer-term linkages between adolescent and parent cortisol, such as moment-to-moment or year-to-year physiological linkages. Studies with multiple timescales may further improve our understanding on the direction of effect between parent and adolescent cortisol and how they may manifest differently on different time scales. Our cross-lagged models allowed us to test two competing directions of cortisol covariation in the same model (adolescent → parent and parent → adolescent) while controlling for prior levels of cortisol. Thus, the strength of our models increases our confidence that parent-adolescent cortisol covariation is an adolescent-driven process. However, our study does not shed light on the underlying processes of how adolescent cortisol is linked to parent cortisol the following day (e.g. mediators). More studies that unpack the underlying mediating and moderating processes that link adolescent cortisol to parent cortisol are clearly needed, including studies that include measures of child temperament. Lastly, our study was conducted with two different samples with different demographics, lending support that our findings are similar across different populations. However, our samples were derived based on a workplace study and were not reflective of the community or population in general. Replication in larger community-based samples is needed.

4.2. Intervention implications

Our findings have important implications for interventions. First, future interventions that address parental reactivity to adolescent characteristics may be an important avenue to promote parent well-being. Mindful parenting interventions that give parents tools to have more compassion for themselves and their adolescent and to pause and interrupt automatic emotional and behavioral reactions to their adolescents may hold promise (Lippold and Duncan, 2018). Training in mindful parenting may reduce the extent to which parents are physiologically reactive to challenging adolescent behaviors and characteristics. Second, interventions for adolescents may have important implications for their parents. Some parenting interventions, such as Attachment and Biobehavioral Catch-Up and Multidimensional Treatment Foster Care, have shown promise for promoting healthy adolescent cortisol patterns (Dozier et al., 2008; Fisher et al., 2007). Findings suggest that promoting healthy cortisol in adolescence will likely promote healthy cortisol patterns in their parents. Future studies that examine the effects of interventions on parents' cortisol are warranted.

5. Conclusions

In sum, our study suggests that parent and adolescent HPA functioning are linked but that this process is primarily adolescent-driven: Parents' cortisol, an important physiological marker of arousal and the stress response system, is predicted by cortisol in their adolescent. This study is part of a growing body of evidence that adolescents have important effects on their parents and extends this to include parents' cortisol patterns. Interventions to reduce adolescents' stress and parents' reactivity to their children may benefit parents' health.

Declaration of Competing Interest

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