Published in final edited form as:

Stress Health. 2022 August ; 38(3): 509–521. doi:10.1002/smi.3110.

Adolescent effects on mothers' bedtime cortisol: Cognitive interference as a mediating mechanism

Melissa A. Lippold[1], **Peter Molenaar**[2], **Kelly D. Chandler**[3], **Soomi Lee**[4], **David M. Almeida**[2]

[1]The School of Social Work, The University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA

[2]Human Development and Family Studies, The Pennsylvania State University, University Park, Pennsylvania, USA

[3]Human Development and Family Sciences, Oregon State University, Corvallis, Oregon, USA

[4]School of Aging Studies, The University of South Florida, Tampa, Florida, USA

Abstract

Prior studies have shown that parent and adolescent cortisol are associated across days and that this covariation may be adolescent-driven. This study extends this literature by (a) testing whether parents' cognitive interference (i.e., distracting and ruminative thoughts potentially due to worry) mediates the linkages between adolescent and next-day parent cortisol and (b) whether these linkages were moderated by parent gender or warmth. Daily diary data, including bedtime cortisol, were collected on two samples of employees and their adolescent-aged children (N= 318 dyads, $M_{\text{youth age}}$ = 13.18 years, 74% mothers). We tested mediation with autoregressive cross-lagged models. Moderated mediation by parent gender was found in our bedtime cortisol models. Higher adolescent bedtime cortisol levels were associated with higher next-day levels of mothers' cognitive interference. In turn, higher levels of mothers' cognitive interference were linked to higher mothers' same-day bedtime cortisol levels. These linkages were not significant for fathers. Cognitive interference did not mediate the associations between child and parent area under the curve or cortisol awakening response. No moderation was evident for parental warmth. Results suggest that mothers' cognitions play a key role in the transmission of elevated bedtime cortisol levels from adolescents to their mothers.

Keywords

child effects; cognitive interference; cortisol; cortisol synchrony; daily diary study; parenting

Correspondence: Melissa Lippold, The School of Social Work, The University of North Carolina at Chapel Hill, Tate Turner Kuralt Building, CB #3550, Chapel Hill, NC 27599, USA. mlippold@unc.edu.

CONFLICT OF INTEREST

The authors have no conflicts of interest.

1 | INTRODUCTION

Theory and research suggest that children have important effects on their parents and that these effects become stronger as children become older (Lansford et al., 2018): The emotions, interactions and behaviour of adolescents have implications for how parents behave towards them (Pardini, 2008). Recent work suggests that adolescents may also have effects on parents' daily physiology, in particular, the functioning of their physiological stress-response system, the hypothalamic–pituitary–adrenal (HPA) axis (Davis et al., 2018; Pendry & Adam, 2009; Saxbe et al., 2014). Parent and their adolescent-aged children's physiological stress-response systems are indeed linked in day-to-day life and several studies have now found evidence that when one member of the parent–child dyad's cortisol increases, so does the other's (Pendry & Adam, 2009; Saxbe et al., 2014). A recent study found evidence that adolescent cortisol predicted parent cortisol the following day but not vice versa (Lippold et al., 2020). Such adolescent-driven effects on parent cortisol are important, given that unhealthy patterns of cortisol are linked to a host of health problems, including lower immunity, increased risk for depression and cardiovascular disease, and increased mortality (Cacioppo, 1994; Juster et al., 2010). Identification of factors that may explain adolescent effects on parent cortisol can help inform interventions to reduce the transmission of physiological stress responses from adolescents to their parents. Here, we build explicitly on the prior findings to unpack mechanisms of *adolescent effects* on parent cortisol. In this study, we test whether parents' cognitive interference (i.e., intrusive and ruminative thoughts potentially due to stress and worry) mediates the associations between adolescent and next-day parent cortisol. We also test whether these processes differ by parent gender and parental warmth. Thus, this paper builds off our prior work both conceptually (by expanding on the role of parent cognitions) and analytically (by testing for mediation and moderation). By considering a potential mechanism and moderators, this study aims to improve understanding of how adolescent cortisol affects parent physiology and under what circumstances.

Cortisol is an important hormone produced by the HPA axis to mobilize energy during times of stress (Adam & Kumari, 2009; Piazza et al., 2010). At the daily level, cortisol exhibits a diurnal pattern: Cortisol levels rise 30–45 min after waking and then decline over the course of the day, reaching the lowest levels at bedtime (Lovallo & Thomas, 2000; Sapolsky et al., 2000). Deviations to this diurnal rhythm, such as high bedtime levels of cortisol and a blunted rise of cortisol in the morning have been linked to negative health outcomes (for review, see Piazza et al., 2010). In order to provide a comprehensive picture on adolescent effect on parent cortisol, we include three cortisol measures that have been linked to health outcomes: (1) bedtime cortisol levels, which capture the ability to recover from daily stressors and be in a relaxed state at the end of the day; (2) the cortisol awakening response (CAR), which captures the ability to mobilize energy in the morning; and (3) total cortisol output throughout the day assessed as the area under the curve (AUC).

We hypothesize cognitive interference will mediate the effects of adolescent bedtime cortisol on parent bedtime cortisol specifically for two reasons. First, bedtime cortisol levels are more strongly linked to daily contextual factors (such as child stress) than other diurnal cortisol indicators such as the cortisol awakening response (CAR; Fries et al., 2009; Stadler

et al., 2015). Indeed prior studies have found daily stressful experiences with children were associated with flatter declines in parent cortisol across the day, which would result in high parent cortisol levels at bedtime (Barker et al., 2012; Seltzer et al., 2009, 2010). Second, rumination, one aspect of cognitive interference, has been associated with elevated bedtime cortisol levels specifically (Cropley et al., 2015), suggesting parents' cognitions may play a key role in whether parents' are able to calm down and recover from stress at the end of the day. Although included in our analyses, we do not hypothesize mediation effects for CAR or AUC.

1.1 | Adolescent effects on parents' HPA functioning

Child characteristics have been found to affect the HPA axis of parents (Dykens & Lambert, 2013) in studies on parents of children with disabilities or behaviour problems. Parents of children with disabilities experience more daily stress and have less healthy patterns of diurnal cortisol compared to parents with children without disabilities (Barker et al., 2012; Seltzer et al., 2009, 2010). Studies on younger children have also found some evidence that parent and child diurnal cortisol and cortisol during laboratory tasks are linked (Pratt et al., 2017; Ruttle et al., 2011; Sethre-Hofstad et al., 2002).

Less is known about how children affect parents' HPA functioning among non-clinical populations during adolescence. A small number of daily diary (Papp et al., 2009; Williams et al., 2013) and lab-based studies (Saxbe et al., 2014) have found synchrony between adolescent and parent cortisol, such that when one experiences rises in cortisol so does the other. Such studies on cortisol synchrony do not shed light on the direction of effect between adolescent and parent cortisol. A recent study analysed data across days to examine the direction of effect between parent and child cortisol. Lippold et al. (2020) found that even when controlling for daily stressors, adolescent cortisol was associated with parents' cortisol on the following day, but parents' cortisol did not predict adolescent cortisol on the following day. Although replication of these findings is needed, they provide preliminary evidence that adolescent cortisol may be an important predictor of their parents' next-day cortisol.

Adolescent effects on parent cortisol may reflect an empathic stress response where parents experience stress and HPA activation from observing a stress, even if they do not experience it directly themselves (Buchanan et al., 2012). Empathic stress has been found to be stronger in closer relationships than with strangers, thus it may be especially salient in parent-child relationships (Engert et al., 2014). Physiological transmission of stress from adolescents to parents may occur in several ways. When adolescents experience high bedtime cortisol levels, parents may observe that their children are distressed and having difficulty calming down at night and recuperating from the day's events. Second, children may disclose to their parents about their daily stressful experiences (Lippold et al., 2014). Either parents' observations or children's self-disclosure may affect a parent's physiology, and a parents' ability to calm down at the end of the day. Further, adolescents with high cortisol levels may have difficulty falling and staying asleep, thus keeping parents awake later—which may affect parent cortisol on the following day (Omisade et al., 2010). Thus, although adolescent stress may affect parent cortisol the same day—it can also affect parent cortisol

the following day. In our prior work, we found next-day associations: higher levels of adolescent bedtime cortisol were associated with higher levels of parent bedtime cortisol levels the following day (Lippold et al., 2020). Yet little is known about how adolescent cortisol may affect next-day parent cortisol and the underlying mediating mechanisms, which would inform interventions to stop the physiological transmission of stress from adolescents to their parents.

1.2 | Cognitive interference as a mediator between adolescent and parent cortisol

Parents' cognitions may be an important intermediate mediating mechanism underlying the empathic stress response system and may explain the effects of adolescent cortisol on parent next-day cortisol (Figure 1). Cognitive interference captures the extent to which an individual experiences intrusive, ruminating thoughts as well as attempts to suppress, avoid, and control these thoughts (Brosschot et al., 2006; Sarason & Sarason, 1996). Cognitive interference includes elements of both ruminating thoughts, as well as thought control and suppression potentially due to lingering stress (Sarason et al., 1986; Wegner & Zanakos, 1994; Wells & Davies, 1994). Parents may experience increased cognitive interference when their adolescents have high bedtime levels of cortisol (Figure 1, Path A). On days after observing their adolescents' physiological stress responses (e.g., having difficulty calming down at night due to high bedtime cortisol levels), parents may ruminate or worry about the source of or solution to adolescent stress. Such thoughts may be overwhelming to parents, and they may have difficulty controlling these worries.

Parents who experience more cognitive interference may subsequently demonstrate high bedtime levels of cortisol themselves (Figure 1, Path B). According to the perseverative cognition hypothesis, the HPA axis is activated longer when individuals experience cognitive interference (Brosschot et al., 2006), which may have important implications for bedtime cortisol levels specifically (Cropley et al., 2015). Prior studies have found that individuals who experience more cognitive interference, such as rumination, are more likely to have high cortisol levels at the end of the day (Cropley et al., 2015; Gianferante et al., 2014), which may reflect higher initial cortisol activation to a stressor and prolonged cortisol activation (Zoccola & Dickerson, 2015). Thus, parents who experience cognitive interference in response to their adolescent's physiological stress may experience subsequent high levels of cortisol at bedtime.

1.3 | The moderating role of parent gender and parental warmth

Parent gender may be an important moderator affecting whether cognitive interference mediates the linkages between adolescent and parent cortisol and may potentially affect both Paths A and B in Figure 1 (Kudielka & Kirschbaum, 2005). Studies on the role of parent gender have been mixed. In terms of Path A (adolescent cortisol affecting parents' cognitive interference), there is some evidence that women are more likely to experience aspects of cognitive interference (e.g., rumination) in response to stressors, such as parental concerns over their children (Nolen-Hoeksema, 2001). Mothers may be more involved and more knowledgeable about child activities than fathers (McHale et al., 2003), which may increase potential worries about children. Mothers may also exhibit more empathy or a

stronger empathic response to stress in others than men (Rueckert & Naybar, 2008; Singer et al., 2006), although other studies have not found gender differences (Engert et al., 2014).

In terms of the path from parents' cognitive interference to parent cortisol (Path B), findings regarding gender differences have also been inconsistent. Some studies have found stronger linkages between cognitive interference and HPA activation in men (Zoccola et al., 2010), whereas others have only found effects for women (Shull et al., 2016). One prior study on parent–child cortisol associations found some evidence that the linkages between child cortisol and parent cortisol may differ between mothers and fathers (Saxbe et al., 2014), but another study found no parent gender differences (Lippold et al., 2020). Most prior studies on parent–child cortisol associations have focused on mothers only (Papp et al., 2009; Pratt et al., 2017; Ruttle et al., 2011), making gender differences impossible to test.

Parental warmth may also moderate the mediational pathways that link adolescent cortisol to cognitive interference and parent cortisol. The literature on these linkages is somewhat mixed. Adolescent cortisol may have a stronger relationship to parents' cognitive interference (Path A) and subsequently with parents' cortisol (Path B) when parents are closer to their children. There is some evidence that close relationships engender a stronger empathic stress response (Engert et al., 2014); thus, parents who are closer to their adolescent may be more strongly affected by them. However, a few studies have found that parent–child cortisol synchrony is stronger in the context of a strained parent–child relationship (Han et al., 2019; Papp et al., 2009; Williams et al., 2013). Parents who have a close relationship with their children may also know more about the child's stressors and thus may be more likely to worry about their children (Lippold et al., 2014; Yun et al., 2016).

1.4 | This study

In this study, we tested whether parents' cognitive interference mediated the linkages between daily adolescent and parent bedtime levels of cortisol. We hypothesized that high levels of bedtime cortisol in adolescents will be associated with higher next-day levels of parents' cognitive interference (Path A). Subsequently, parents' cognitive interference will be positively associated with higher levels of same-day parent bedtime cortisol (Path B). We also tested these models using the AUC and CAR[1] but hypothesized there would be no evidence of mediation by cognitive interference. In a second step, we examined whether these mediational processes were moderated by parent gender and warmth. Given mixed findings in the literature, we did not posit specific moderation hypotheses.

2 | METHOD

2.1 | Participants and procedure

We used data from parent-child dyads who participated in the daily diary component of the Work, Family, Health Study (WFHS). The study included two samples: employees in an information technology (IT) division of a U.S. Fortune 500 company and workers in a

[1]In our CAR models, parents' cognitive interference is assessed on the same day as child cortisol (i.e., child CAR predicts same-day cognitive interference and cognitive interference predicts next-day parent CAR).

nursing home (NH; Bray et al., 2013). Employees who were the parent of an adolescent aged 9–17 who lived at home for at least 4 days a week were recruited for a home interview and, if both parent and adolescent completed the home interview, were eligible to participate in a daily diary study involving eight consecutive evening telephone calls. During the daily diary study, parents and their adolescent provided information on their daily activities, emotions and experiences.

On four diary study days (Days 2, 3, 4 and 5), parents 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, interviewers distributed saliva collection kits. Each kit contained 16 salivettes for collecting adolescent cortisol (4 salivettes/day for 4 days) along with a DVD that demonstrated saliva collection procedures. Parents and youth were instructed to roll a cotton swab across their tongue for 2 min 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. 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). Assays were run on a rolling basis throughout the entire study period. The assay had a lower limit of sensitivity of 0.003 μg/dl, with average inter- and intra-assay covariances of less than 7% and 4%, respectively. Outliers were winsorized such that cortisol values below 0.003 μg/dl were designated as off-the-curve low and were set to the lowest level of sensitivity to the assay. In addition, based on previous studies (Almeida et al., 2016; Stawski et al., 2013), cortisol values greater than 82.77 μg/dl were rerun on a 1.8 dilution. Assayed samples that remained high were considered invalid and removed from the sample (Almeida et al., 2016).

In order to maximize statistical power and to test our models on a diverse sample, we combined our sample to include participants in both the IT and NH samples for a total of 318 parent–adolescent participants. In the IT sample (n = 132), 45% of employees were women and 55% of the children were girls. Among parents, 78% were college graduates and the average annual income was $116,900 (SD = $26,396). The majority of children were Caucasian (74%), 16% Asian, 2% African American, 2% Pacific Islander, 4% more than one race and 3% marked other. Of these, 9% indicated they were of Hispanic heritage. For the NH subsample (n = 186), 96% of the employees were women and 51% of the children were girls. Among parents, 64% were college graduates and the average annual income was $56,660 (SD = $31,759). Among children in the NH sample, 60% were Caucasian, 14% African American, 3% Asian, 1% American Indian, 7% more than one race and 15% marked other. Of these, 15% indicated they were of Hispanic heritage. In our combined sample, we have 318 dyads that includes 233 mothers and 80 fathers (5 were missing gender data). Children ranged from 9 to 17 years of age (M = 13.40 in IT sample, 13.03 in NH sample), 167 were daughters and 151 were sons. To assess potential differences in study findings between our samples, we tested for moderation by industry in all analyses. Because we found no differences, we present results for the combined sample. Further, because our

sample contained youth ages 9–17, we conducted sensitivity analyses on a subsample of youth ages 12 and above. Because results from this subsample based on child age were the same, we present findings from the full sample here.

2.2 | Measures

2.2.1 | Cortisol measures—

Bedtime levels of cortisol were assessed as levels of cortisol at the end of the day. The CAR was calculated as the difference in cortisol levels at wake and 30 min post-wake. Cortisol values were converted to nmol/L and natural log transformed before analysis (Adam & Kumari, 2009). Given the diurnal rhythm of cortisol (i.e., cortisol levels peak after waking and decline over the day), unhealthy patterns of cortisol include high bedtime levels of cortisol and blunted CAR (Piazza et al., 2010). We also assessed the AUC with respect to the ground over the day (AUC-G; Pruessner et al., 2003) which accounts for time intervals between samples (McHale et al., 2012).

2.2.2 | Cognitive interference—

Parents' cognitive interference was measured with a nine-item Likert-type scale that assessed parental rumination, intrusive thoughts, thought control and suppression (Lee et al., 2019; Stawski et al., 2011). Items included how often that day parents thought about personal worries, thought about something they did not mean to think about, had difficulty concentrating, had thoughts that kept jumping in their head, had thoughts they could not stop, tried to avoid certain thoughts, did things to distract themselves from their thoughts, and stayed busy just to keep thoughts form entering their mind (1 = *never* and 5 = *very often*). Cronbach's alpha across days averaged 0.89 (range 0.82–0.92).

2.2.3 | Moderators and control variables—

Moderators include parent gender (0 = *father*, 1 = *mother*) and parental warmth (assessed by the child report of an eight-item parental behaviour inventory CRPBI; Schaefer, 1965; α = 0.85, dichotomized to be 0 = *low warmth*; 1 = *high warmth* based on median split). Following previous studies (Almeida et al., 2017), we controlled for demographics such as adolescent age, gender (0 = *boys*, 1 = *girls*), and race/ethnicity (0 = *White*, 1 = *non-White*), as well as parent education (0 = *no college education*, 1 = *college education*). Additional cortisol-related control variables included whether adolescents and parents were taking steroid medications that interfere with cortisol (0 = *no medication use*; 1 = *medication use*), time of cortisol sample collection and a variable for cortisol protocol compliance (0 = *compliant sample*; 1 = *non-compliant sample*). Participants received a 1 for non-compliance if the time difference between samples at wake and 30 min after waking was less than 15 min and more than 60 min, the participant woke up later than noon, or if the participant was awake for less than 12 h or more than 20 h. Other control variables included type of day (0 = *weekday*, 1 = *weekend*) and average daily stressors (calculated as the average number of reported daily stressors across the week using the Daily Inventory of Stressful Experiences; Almeida et al., 2002).

2.3 | Data analysis plan

Autoregressive longitudinal mediation models were run across 4 days of cortisol measurement using *Mplus*. Models included two mediating paths: from adolescent bedtime cortisol to parents' cognitive interference the following day (Path A; Figure 1) and from

parents' cognitive interference to parents' same-day bedtime cortisol levels (Path B). Models also included direct paths from adolescent to parent cortisol across days (solid grey lines) as well as stability paths (dotted lines). Stability paths control for previous levels of cortisol variables and cognitive interference. Additional non-predicted variables collected in the same day were correlated (e.g., all control variables at Day 1, same-day correlations between parent and adolescent cortisol). Control variables included demographics (age, gender, race/ethnicity, parent education), cortisol specifications (medication use, time of sample, protocol compliance), type of day (weekend or weekday) and average stressors.

Model fit proceeded through a series of steps to assess the best fitting, most parsimonious model. In Step 1, we fit our baseline model, in which all paths were freely estimated. In Step 2, 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., Path A across days) 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. Invariance tests were conducted separately for each type of path.

Model goodness-of-fit was assessed according to strict likelihood ratio principles and included chi-square tests and practical indices of model fit (root-mean-square error of approximation [RMSEA], Rho and the comparative fit index [CFI]). The following criteria indicated acceptable model fit: CFI and Tucker–Lewis Index (TLI) values of 0.90 or higher, and an RMSEA value of 0.08 or lower (Chen et al., 2008; West et al., 2012). Missing data were handled using full information maximum-likelihood procedures (Enders, 2010). Bootstrapping was used to assess model estimates (Cheung & Lau, 2008). When significant mediation was found, we estimated the percentage of variance in parent cortisol explained by the mediator (cognitive interference). To do this, we calculated the change in variance explained by the model (r-squared) when the mediator was and was not included in the model.

In Step 3, we tested whether our models differed based on parent gender and parental warmth. In terms of parent gender, first, we ran a series of nested models that compared the fit of a model with the substantive model paths constrained to be equal between genders to a model where the paths are freely estimated. If there was not a significant difference in chi-square between models when these paths were and were not constrained, we concluded that there was no evidence of moderation by gender. If there was a significant difference in chi-square between these models, we split our sample to run models separately for mothers and fathers. When running models separately by gender, we followed the same process as Step 2, again testing if any substantive model paths could be constrained to be equal across time for each subgroup (e.g., fathers only) in order to obtain the best model for each gender. We assessed moderation by parental warmth using the same steps. Our measure of parental warmth was dichotomized based on a median split (0 = *low*; 1 = *high*). Each cortisol indicator was run separately.

3 | RESULTS

3.1 | Descriptive statistics

Descriptive statistics are shown in Table 1. Moderate correlations were found between adolescent and parent bedtime cortisol levels ($r = 0.37$), AUC ($r = 0.24$) and CAR ($r = 0.23$). Parents' cognitive interference was not significantly correlated with any cortisol indicators. Parent gender was correlated with adolescent bedtime cortisol ($r = 0.14$), and parent CAR ($r = 0.12$).

3.2 | Mechanism of parent cognitive interference linking child cortisol and parent cortisol

Results on model invariance across days for bedtime cortisol, AUC and CAR are presented in Table 2. Below, we present results from final models.

3.3 | Bedtime cortisol

In our final model, adolescent bedtime cortisol stability paths were allowed to vary from each other across the time points. Final model fit was good, $\chi^2 = 260.84$, $df = 228$, $p = 0.06$; RMSEA = 0.02, NNFI = 0.96, and CFI = 0.95. Same-day correlations between parent and adolescent bedtime cortisol ranged from 0.02 to 0.03 across days and all were not significant ($p > 0.05$). In our final models, Paths A and B were significant: Adolescent bedtime cortisol predicted parents' cognitive interference on the following day ($B = 0.07$, SE = 0.04, $p = 0.04$) and parents' cognitive interference predicted parents' same-day bedtime levels of cortisol ($B = 0.07$, SE = 0.03, $p = 0.03$). Direct paths from adolescent to parent bedtime cortisol (Path C′) were also significant ($B = 0.13$, $SE = 0.03$, $p = 0.00$). Thus, parents' cognitive interference was a statistically significant partial mediator of the associations between adolescent and parent bedtime cortisol. The change in *r*-squared for the outcome when the mediator was in the model compared to a model without the mediator was 0.006 for parent cortisol on Day 2, 0.003 for Day 3, and 0.005 for Day 4. Thus, parent cognitive interference had a small mediation effect.

3.4 | AUC and CAR

For AUC, model fit was good, $\chi^2 = 266.75$, $df = 228$, $p = 0.04$; RMSEA = 0.03, NNFI = 0.95, and CFI = 0.92. Same day correlations between parent and adolescent AUC were significant for Day 1 ($r = 0.18$, SE = .01, $p = .02$) but not for Day 2 ($r = 0.13$, SE = 0.08, $p = 0.08$), Day 3 ($r = -0.03$, SE = 0.23, $p = 0.91$), or Day 4 ($r = -0.02$, SE = 0.22, $p = 0.91$). Adolescent AUC did not predict parents' cognitive interference on the following day (Path A: $B = -0.004$, SE = 0.003, $p = 0.25$). Parents' cognitive interference predicted parents' same-day AUC (Path B: $B = 0.65$, SE = 0.31, $p = 0.03$). Direct paths from adolescent to next-day parent AUC (Path C′) were also not significant ($B = 0.01$, SE = 0.04, $p = 0.79$). In CAR models, fit was also good, $\chi^2 = 290.85$ $df = 228$, $p < 0.05$. RMSEA = 0.02, NNFI = 0.92, and CFI = 0.94. Same day correlations between mother and adolescent cortisol were not significant (range 0.02–0.10, all $p > 0.05$). In our final models for CAR, Path A was not significant: Child CAR did not predict parents' same-day cognitive interference ($B = -0.01$, SE = 0.04, $p = 0.72$). Path B was inconsistent: Parents' cognitive interference on Day 2 ($B = -0.01$, SE = 0.05, $p = 0.87$) and Day 3 ($B = 0.01$, SE = 0.05, $p = 0.88$) did not predict

parent CAR on the following day. However, cognitive interference on Day 4 did predict parent CAR on the following day ($B = -0.21$, SE = 0.06, $p = 0.001$). Direct effects (Path C′) were also significant ($B = 0.10$, SE = 0.04, $p = 0.003$). Therefore, parents' cognitive interference was not a mediator of adolescent and parent AUC or CAR.

3.5 | Moderation by parent gender

3.5.1 | Bedtime cortisol—First, overall significance tests were run to assess if any of the bedtime cortisol model paths differed by parent gender. The difference between a model where all substantive model paths were constrained to be equal with a model where paths were free to vary between genders was statistically significant ($\Delta\chi^2 = 31.9$, $df = 18$, $p = 0.02$), suggesting at least one path differed by gender. Then models were fit separately for mothers and fathers. For fathers (See Table 2), nested models revealed that four paths were invariant across days (adolescent cortisol stability paths, mediator stability paths, Paths A and C′) and could be constrained to be equal. For mothers, all paths except adolescent stability paths were invariant across days.

As seen in Figure 2, mother but not father cognitive interference was a significant mediator of the associations between adolescent and parent bedtime cortisol. For mothers, higher levels of adolescent bedtime cortisol were significantly associated with higher mothers' cognitive interference on the following day (Path A for mothers; $B = 0.09$, SE = 0.04, $p = 0.03$) but these linkages were not significant for fathers (Path A for fathers; $B = 0.01$, SE = 0.06, $p = 0.90$). Similarly, for mothers, higher mother cognitive interference was associated with higher same-day bedtime levels of mother's cortisol (Path B for mothers; $B = 0.08$, SE = 0.04, $p = 0.03$) but these associations were not significant for fathers for Day 2 (Path B for fathers; $B = -0.04$, SE = 0.12, $p = 0.73$), Day 3 (Path B; $B = 0.28$, SE = 0.21, $p = 0.20$) or Day 4 ($B = -0.03$, SE = 0.14, $p = 0.78$). Direct effects from adolescent to parents' bedtime cortisol (Path C′) were significant for both mothers ($B = 0.12$, SE = 0.04, $p = 0.002$) and fathers ($B = 0.20$, SE = 0.07, $p = 0.003$). Same-day correlations between parent and adolescent bedtime cortisol levels (not shown) were also significant for mothers (ranging from 0.03 to 0.04 across days, all $p < 0.05$) but were not significant for fathers (ranging from −0.01 to 0.03 for fathers, all $p > 0.05$). Because Paths A, B and C′ were significant for mothers, cognitive interference was a statistically significant partial mediator of the associations between adolescent and mother bedtime levels of cortisol. The change in r-squared for the outcome when the mediator was in the model compared to a model without the mediator was 0.007 for mother cortisol on Day 2, 0.002 for Day 3 and 0.006 for Day 4.

3.5.2 | AUC and CAR—The difference between a model where all substantive AUC model paths were constrained to be equal with a model where paths were free to vary between genders was statistically significant ($\Delta\chi^2 = 38.01$, $df = 18$, $p = 0.004$), suggesting differences by parent gender. Same day correlations between father and adolescent AUC were significant for Day 1 ($r = 0.39$, SE = 0.08, $p = 0.00$) and Day 4 ($r = 0.25$, SE = 0.0–9, $p = 0.01$) but were not significant for Day 2 ($r = 0.21$, SE = 0.13, $p = 0.11$) or Day 3 ($r = 0.03$, SE = 0.13, $p = 0.81$). Same-day correlations between mother and adolescent AUC were significant for Day 1 ($r = 0.20$, SE = 0.08, $p = 0.01$) but not significant for Day 2 ($r = 0.08$, $SE = 0.09$, $p = 0.35$), Day 3 ($r = 0.09$, SE = 0.33, $p = 0.78$) or Day 4 ($r = -0.07$, SE

= 0.33, $p = 0.78$). Cognitive interference did not mediate the associations between child and parent AUC for either mothers or fathers. Path A was not significant: Child AUC was not associated with next-day cognitive interference for fathers ($B = -0.006$, SE = 0.04, $p = 0.18$) or mothers ($B = -0.001$, SE = 0.005, $p = .79$). For Path B, fathers' cognitive interference was not associated with fathers' same-day AUC ($B = 0.87$, SE = 0.54, $p = 0.11$). For mothers, Path B was inconsistent with mothers' cognitive interference predicting mothers' same-day AUC on Day 4 ($B = 2.22$, SE = 0.72, $p = 0.002$) but not for Day 2 ($B = -0.28$, SE = 0.65 $p = 0.66$) or Day 3, ($B = 0.62$, SE = 0.59 $p = 0.47$); Direct effects (Path C′) from adolescent to parent AUC were significant for fathers ($B = 0.87$, SE = 0.54, $p = 0.11$) but not mothers ($B = -0.02$, SE = 0.04, $p = 0.55$). Thus, there is no evidence that cognitive interference mediated the linkages between adolescent and parent AUC for mothers or fathers. In our CAR models, there was no evidence of moderation by parent gender: The difference between a CAR model where all substantive model paths were constrained to be equal with a model where paths were free to vary between genders ($\Delta\chi^2 = 16.47$, $df = 18$, $p = 0.55$) was not statistically significant.

3.6 | Moderation by parental warmth

3.6.1 | Bedtime cortisol—The difference between a bedtime cortisol model where all substantive model paths were constrained to be equal with a model where paths were free to vary between based on parent–child warmth was not significant ($\Delta\chi^2 = 22.61$, $df = 18$, $p = 0.21$). Thus, there is no evidence that parental warmth moderated these linkages.

3.6.2 | AUC and CAR—For both AUC and CAR, there was no evidence of moderation by parental warmth. The difference in models where all substantive model paths were constrained to be equal with a model where paths were free to vary between based on parent–child warmth was not significant (AUC: $\Delta\chi^2 = 22.57$, $df = 18$, $p = 0.20$; CAR $\Delta\chi^2 = 25.82$, $df = 18$, $p = 0.11$).

4 | DISCUSSION

Prior studies have found that behaviours and experiences of children have implications for parenting behavior and parental well-being (Pardini, 2008). Recently, evidence has found that child effects may apply not only to behavioral indicators, but also to physiological ones as well: Adolescent-aged children's cortisol patterns are associated with parent's cortisol the following day (Lippold et al., 2020). The purpose of this study was to examine parent's cognitive interference—intrusive thoughts and efforts to control those thoughts—as a mediating factor that may link adolescent cortisol to parent cortisol.

We found evidence that cognitive interference partially mediated the associations between adolescent and mother bedtime levels of cortisol. Adolescent bedtime cortisol predicted increases in mothers' cognitive interference the next day, which subsequently led to increases in mothers' own cortisol levels at bedtime. When adolescents experience stressors and have trouble calming down at night before bedtime, mothers may worry about them the following day, experiencing intrusive and potentially ruminating thoughts, which in turn, may affect mothers' own ability to calm down at bedtime. Our findings may reflect an empathic stress response, whereas mothers react to adolescent stress even if they do

not experience it themselves (Buchanan et al., 2012; Engert et al., 2014). However, it also suggests that mothers' own cognitive responses the following day may play an important role in whether adolescent physiological stress responses affect mothers' physiological stress responses at bedtime. This finding supports the perseverative cognition hypothesis, in that repetitive thoughts and mentally reliving stressors lead to extended activation of the HPA axis (Brosschot et al., 2006). Yet, it extends this work to suggest that their adolescent-age children's own bedtime cortisol levels may be an important trigger for such maternal cognitive interference.

Our bedtime cortisol results were moderated by parent gender, such that the partial mediation by parents' cognitive interference was significant for mothers but not fathers. However, findings were not moderated by parental warmth, suggesting similar effects regardless of parent–child relationship quality. Mothers may experience cognitive interference in response to their adolescent's HPA functioning and subsequently have high levels of bedtime cortisol. This finding supports prior studies that have found children's behaviour to have stronger effects on mothers' behaviour when compared to fathers (Moes et al., 1992) and suggests gender differences may extend beyond behavioral child-effects to physiological ones as well. These findings may reflect socialization differences: Mothers may be more involved with their adolescents (McHale et al., 2003) and more aware of their adolescent's activities and well-being. Therefore, mothers may be more likely to notice and be affected by their adolescent's physiological responses to stress. Some studies have found that mothers are more strongly affected by children's behaviour than fathers (Elam et al., 2017; Moes et al., 1992), and also that women may experience a stronger empathic stress response (Rueckert & Naybar, 2008; Singer et al., 2006). Other work has also found evidence that women experience more rumination—one aspect of cognitive interference—in response to stressors than men (Nolen-Hoeksema, 2001). Given mixed findings regarding gender differences in prior literature, more studies with fathers and replication in other samples are clearly needed.

We found no evidence that parents' cognitive interference mediated the relationship between adolescent and parent AUC or CAR. This was not surprising given that prior studies have found that bedtime cortisol levels may be affected more strongly by contextual factors than the cortisol awakening response, which would also be reflected in the AUC (Fries et al., 2009; Stadler et al., 2015). It is possible that CAR indicators capture chronic stress responses that are less affected by child characteristics, such as their stress physiology (Fries et al., 2009). Indeed adolescent AUC and CAR did not predict parent cognitive interference in our models. Further, there was inconsistent evidence that parent cognitive interference predicted their own AUC or CAR. This supports other studies that have found cognitive interference to be linked to bedtime cortisol levels specifically (Cropley et al., 2015). There was some evidence that adolescent AUC and CAR may have direct effects on parent cortisol —which are not explained by cognitive interference. It raises questions as to whether other processes—perhaps on other timescales—may be at work.

4.1 | Strengths and limitations

This study has several strengths and limitations. First, our study used daily diary data with biomarkers, which allowed us to assess individuals in their natural context increasing ecological validity (Lippold & McNamee, 2014). Daily assessments can capture day-to-day variability in cognitive interference and minimize recall bias. However, our measure of cognitive interference combines multiple cognitive processes into one. Studies with more fine-grained measures are needed to understand the specific role of rumination versus thought control, or suppression or other cognitions, such as parental efficacy or parental attributions about their children. Second, our study provides information about the day-to-day associations between adolescent and parent cortisol and cognitive interference, but it cannot inform us about shorter (e.g., moment-to-moment) or longer term associations (e.g., over months, years). Importantly in our study, we tested whether child bedtime cortisol levels predicted *next-day* parent cognitive interference and bedtime cortisol. We were not able to assess same-day mediation given that parents' cognitive interference was assessed before many adolescents go to bed. Note that, in our study, the same-day correlations between child and parent bedtime cortisol were often not significant. Third, our study did not employ a community-based sample that represent general families in the United States, which may limit generalizability. Our findings may be specific to parents who work in department similar to those included in this study (i.e., IT departments, nursing home workers). However, we tested our model on two different samples with different demographics, and the lack of differences between them lends support for the potential application of our findings among different populations. Fourth, our study does not shed light on the implications of this process for other aspects of parental well-being. Several studies have shown important linkages between stress, cognitive interference, and memory and learning (Sarason et al., 1996; Stawski et al., 2006). Cognitive interference can reduce the attention and resources available for other cognitive tasks, thereby reducing performance in other aspects of learning and work, and potentially affecting other parenting behaviour. Cognitive interference has also been associated with other aspects of well-being, namely sleep (Thomsen et al., 2003; Lee et al., 2019). Future studies are needed to explore the role of sleep especially. Fifth, our study controlled for stressors individually experienced by parents and children. However, we were unable to assess collective family stress or shared stressors, which may underlie these findings. Future studies with more fine-grained assessments of shared family stressors would shed light on the various types of stressors families may experience and how they affect adolescent effects on parent cortisol. Further, our mediation effects although significant, were small, suggesting other mediators are likely at work. Further, we included three indicators of cortisol in our analyses to be comprehensive, however increasing the number of statistical tests can increase the risk of Type I error. Future studies should examine additional processes—including sleep—that may explain parent–child cortisol linkages.

5 | CONCLUSIONS

Our study suggests that cognitive interference may play a key role in the transmission of physiological stress (indicated by bedtime cortisol levels) from adolescents to their mothers. Interventions to help reduce mothers' rumination and intrusive thoughts about their

children may have important implications for mothers' own stress physiology at bedtime. Approaches such as mindfulness or cognitive-based therapies may give mothers tools to observe and detach from intrusive thoughts and reduce the need for effort to suppress and manage such thoughts (Lippold & Duncan, 2018; Lippold et al., 2021) and may help them calm down at bedtime specifically. Mindfulness-based programs for both mothers and adolescents may also reduce the physiological transmission of stress from adolescents to their mothers.

ACKNOWLEDGEMENTS

This study was funded by a grant from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (R03 HD 087611). Additional funding was provided to the parent grant (Work Family Health Study and Work, Family and Health Network) by the Eunice Kennedy Shriver National Institute of Child Health and Human Development (U01HD051217, U01HD051218, U01HD051256, U01HD051276), National Institute on Aging (U01AG027669), Office of Behavioral and Science Sciences Research, National Institute for Occupational Safety and Health (U01OH008788, U01HD059773), the William T. Grant Foundation, Alfred P Sloan Foundation, and the Administration for Children and Families. The contents of this publication are solely the responsibility of the authors and do not necessarily represent the official views of these institutes and offices.

Funding information

Eunice Kennedy Shriver National Institute of Child Health and Human Development, Grant/Award Numbers: R03 HD 087611, U01HD051217, U01HD051218, U01HD051256, U01HD051276; William T. Grant Foundation; National Institute on Aging, Grant/Award Number: U01AG027669; Administration for Children and Families; National Institute for Occupational Safety and Health, Grant/Award Numbers: U01HD059773, U01OH008788; Alfred P Sloan Foundation

DATA AVAILABILITY STATEMENT

To request access for the publicly available Work, Family, Health data, please visit https://workfamilyhealthnetwork.org/data.

REFERENCES

Adam EK, & Kumari M (2009). Assessing salivary cortisol in largescale, epidemiological research. Psychoneuroendocrinology, 34(10), 1423–1436. 10.1016/j.psyneuen.2009.06.011 [PubMed: 19647372]

Almeida DM, Davis KD, Lee S, Lawson KM, Walter KN, & Moen P (2016). Supervisor support buffers daily psychological and physiological reactivity to work-to-family conflict. Journal of Marriage and Family, 78(1), 165–179. [PubMed: 26778857]

Almeida DM, Lee S, Walter KN, Lawson KN, Kelly EL, & Buxton OM (2017). The effect of a workplace intervention on employees' cortisol awakening response. Community, Work & Family, 21(2), 151–167. 10.1080/13668803.2018.1428172

Almeida DM, Wethington E, & Kessler RC (2002). The daily inventory of stressful events: An interview-based approach for measuring daily stressors. Assessment, 9, 41–55. 10.1177/2F1073191102091006 [PubMed: 11911234]

Barker ET, Greenberg JS, Seltzer MM, & Almeida DM (2012). Daily stress and cortisol patterns in parents of adult children with a serious mental illness. Health Psychology, 31(1), 130–134. 10.1037/a0025325 [PubMed: 21895369]

Bray JW, Kelly EL, Hammer LB, Almeida DM, Dearing JW, King RB, & Buxton OM (2013). An integrative, multi-level, and transdisciplinary research approach to challenges of work, family, and health. RTI Press. RTI Press Publication No. MR-0024–1303.

Brosschot JF, Gerin W, & Thayer JF (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. Journal of Psychosomatic Research, 60(2), 113–124. 10.1016/j.jpsychores.2005.06.074 [PubMed: 16439263]

Buchanan TW, Bagley SL, Stansfield RB, & Preston SD (2012). The empathic, physiological resonance of stress. Social Neuroscience, 7(2), 191–201. [PubMed: 21777106]

Cacioppo JT (1994). Social neuroscience: Autonomic, neuroendocrine, and immune responses to stress. Psychophysiology, 31(2), 113–128. [PubMed: 8153248]

Chen F, Curran PJ, Bollen KA, Kirby J, & Paxton P (2008). An empirical evaluation of the use of fixed cutoff points in RMSEA test statistic in structural equation models. Sociological Methods & Research, 36(4), 462–494. 10.1177/0049124108314720 [PubMed: 19756246]

Cheung GW, & Lau RS (2008). Testing mediation and suppression effects of latent variables: Bootstrapping with structural equation models. Organizational Research Methods, 11(2), 296–325. 10.1177/2F1094428107300343

Cropley M, Rydstedt LW, Devereux JJ, & Middleton B (2015). The relationship between work-related rumination and evening and morning salivary cortisol secretion. Stress and Health, 31(2), 150–157. 10.1002/smi.2538 [PubMed: 24166947]

Davis M, West K, Bilms J, Morelen D, & Suveg C (2018). A systematic review of parent–child synchrony: It is more than skin deep. Developmental Psychobiology, 60(6), 674–691. 10.1002/dev.21743 [PubMed: 29900545]

Dykens EM, & Lambert W (2013). Trajectories of diurnal cortisol in mothers of children with autism and other developmental disabilities: Relations to health and mental health. Journal of Autism and Developmental Disorders, 43(10), 2426–2434. 10.1007/s10803-013-1791-1 [PubMed: 23468069]

Elam KK, Chassin L, Eisenberg N, & Spinrad TL (2017). Marital stress and children's externalizing behavior as predictors of mothers' and fathers' parenting. Development and Psychopathology, 29(4), 1305–1318. [PubMed: 28065187]

Enders CK (2010). Applied missing data analysis. Guildford Press.

Engert B, Plessow F, Miller R, Kirschbaum C, & Singer T (2014). Cortisol increase in empathic stress is modulated by emotional closeness and observation modality. Psychoneuroendocrinology, 45, 192–201. 10.1016/j.psyneuen.2014.04.005 [PubMed: 24845190]

Fries E, Dettenborn L, & Kirschbaum C (2009). The cortisol awakening response (CAR): Facts and future directions. International Journal of Psychophysiology, 72, 67–73. 10.1016/j.ijpsycho.2008.03.014 [PubMed: 18854200]

Gianferante D, Thoma MV, Hanlin L, Chen X, Breines JG, Zoccola PM, & Rohleder N (2014). Post-stress rumination predicts HPA axis responses to repeated acute stress. Psychoneuroendocrinology, 49, 244–252. 10.1016/j.psyneuen.2014.07.021 [PubMed: 25127082]

Han ZR, Gao MM, Yan J, Hu X, Zhou W, & Li X (2019). Correlates of parent-child physiological synchrony and emotional parenting: Differential associations in varying interactive contexts. Journal of Child and Family Studies, 28(4), 1116–1123.

Juster RP, McEwen BS, & Lupien SJ (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. Neuroscience & Biobehavioral Reviews, 35(1), 2–16. [PubMed: 19822172]

Kudielka BM, & Kirschbaum C (2005). Sex differences in HPA axis responses to stress: A review. Biological Psychology, 69(1), 113–132. 10.1016/j.biopsycho.2004.11.009 [PubMed: 15740829]

Lansford JE, Rothenberg WA, Jensen TM, Lippold MA, Bacchini D, Bornstein MH, Deater-Deckard K, Di Giunta L, Dodge KA, Malone PS, Oburu P, Pastorelli C, Skinner AT, Sorbring E, Steinberg L, Tapanya S, Uribe Tirado LM, Alampay LP, & Al-Hassan SM (2018). Bidirectional relations between parenting and behavior problems from age 8 to 13 in nine countries. Journal of Research on Adolescence, 28(3), 571–590. 10.1111/jora/12381 [PubMed: 30515947]

Lee S, Buxton OM, Andel R, & Almeida DM (2019). Bidirectional associations of sleep with cognitive interference in employees' work days. Sleep Health, 5, 298–308. 10.1016/j.sleh.2019.01.007 [PubMed: 30905693]

Lippold MA, & Duncan LD (2018). Mindful parenting. In Levesque R (Ed.), Encyclopedia of adolescence (pp. 2343–2349). Springer.

Lippold MA, Graham J, Greenberg MT, & Feinberg ME (2014). Unpacking the effect of parental monitoring on early adolescent problem behavior: Mediation by parental knowledge and moderation by parent–youth warmth. Journal of Family Issues, 35, 1800–1823. [PubMed: 25382891]

Lippold MA, Jensen TM, Duncan LG, Nix RL, Coatsworth JD, & Greenberg MT (2021). Mindful parenting, parenting cognitions, and parent-youth communication: Bidirectional linkages and mediational processes. Mindfulness, 12(2), 381–391. [PubMed: 33737985]

Lippold MA, & McNamee C (2014). Capturing the complexity of families using innovative methods. In McHale SM, Amato P, & Booth A (Eds.), Emerging methods in family research (pp. 263–280). Urban Institute.

Lippold MA, Molenaar P, Lee S, Chandler KD, & Almeida DM (2020). Daily parent- adolescent cortisol associations: Unpacking the direction of effects. Psychoneuroendocrinology, 116, 104652. [PubMed: 32272359]

Lovallo WR, & Thomas TL (2000). Stress hormones in psychophysiological research: Emotional, behavioral, and cognitive implications. In Cacioppo JT, Tassinary LG, & Berntson GG (Eds.), Handbook of psychophysiology (pp. 342–367). Cambridge University Press.

McHale SM, Blocklin MK, Walter KN, Davis KD, Almeida DM, & Klein LC (2012). The role of daily activities in youths' stress physiology. Journal of Adolescent Health, 51(6), 623–628.

McHale SM, Crouter AC, & Whiteman SD (2003). The family contexts of gender development in childhood and adolescence. Social Development, 12(1), 125–148. 10.1111/1467-9507.00225

Moes D, Koegel RL, Schreibman L, & Loos LM (1992). Stress profiles for mothers and fathers of children with autism. Psychological Reports, 71, 1272–1274. 10.2466/2Fpr0.1992.71.3f.1272 [PubMed: 1480714]

Nolen-Hoeksema S (2001). Gender differences in depression. Current Directions in Psychological Science, 10(5), 173–176.

Omisade A, Buxton OM, & Rusak B (2010). Impact of acute sleep restriction on cortisol and leptin levels in young women. Physiology & Behavior, 99(5), 651–656. [PubMed: 20138072]

Papp LM, Pendry P, & Adam EK (2009). Mother-adolescent physiological synchrony in naturalistic settings: Within-family cortisol associations and moderators. Journal of Family Psychology, 23(6), 882–894. [PubMed: 20001147]

Pardini DA (2008). Novel insights into longstanding theories of bidirectional parent–child influences: Introduction to the special section. Journal of Abnormal Child Psychology, 36(5), 627–631. 10.1007/s10802-008-9231-y [PubMed: 18437550]

Piazza JR, Almeida DM, Dmitrieva NO, & Klein LC (2010). Frontiers in the use of biomarkers of health in research on stress and aging. Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 65B(5), 513–525. 10.1093/geronb/gbq049

Pratt M, Apter-Levi Y, Vakart A, Kanat-Maymon Y, Zagoory-Sharon O, & Feldman R (2017). Mother-child adrenocortical synchrony; Moderation by dyadic relational behavior. Hormones and Behavior, 89, 167–175. 10.1016/j.yhbeh.2017.01.003 [PubMed: 28131596]

Pruessner JC, Kirschbaum C, Meinlschmid G, & Hellhammer DH (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. Psychoneuroendocrinology, 28(7), 916–931. [PubMed: 12892658]

Rueckert L, & Naybar N (2008). Gender differences in empathy: The role of the right hemisphere. Brain and Cognition, 67(2), 162–167. [PubMed: 18295950]

Ruttle PL, Serbin LA, Stack DM, Schwartzman AE, & Shirtcliff EA (2011). Adrenocortical attunement in mother–child dyads: Importance of situational and behavioral characteristics. Biological Psychology, 88, 104–111. 10.1016/j.biopsycho.2011.06.014 [PubMed: 21767597]

Sapolsky RM, Romero LM, & Munck AU (2000). How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocrine Reviews, 21(1), 55–89. 10.1210/edrv.21.1.0389 [PubMed: 10696570]

Sarason IG, Sarason BR, Keefe DE, Hayes BE, & Shearin EN (1986). Cognitive interference: Situational determinants and traitlike characteristics. Journal of Personality and Social Psychology, 51, 215–226.

Sarason P, & Sarason (1996). Cognitive interference: Theories, methods and findings. Routledge.

Saxbe DE, Margolin G, Spies Shapiro L, Ramos M, Rodriguez A, & Iturralde E (2014). Relative influences: Patterns of HPA axis concordance during triadic family interaction. Health Psychology, 33(3), 273–281. 10.1037/a0033509 [PubMed: 23914815]

Schaefer ES (1965). Children's reports of parental behavior: An inventory. Child Development, 36, 417–424.

Seltzer MM, Almeida DM, Greenberg JS, Savla J, Stawski RS, Hong J, & Taylor JL (2009). Psychosocial and biological markers of daily lives of midlife parents of children with disabilities. Journal of Health and Social Behavior, 50(1), 1–15. 10.1177/2F002214650905000101 [PubMed: 19413131]

Seltzer MM, Greenberg JS, Hong J, Smith LE, Almeida DM, Coe C, & Stawski RS (2010). Maternal cortisol levels and behavior problems in adolescents and adults with ASD. Journal of Autism and Developmental Disorders, 40(4), 457–469. [PubMed: 19890706]

Sethre-Hofstad L, Stansbury K, & Rice MA (2002). Attunement of maternal and child adrenocortical response to child challenge. Psychoneuroendocrinology, 27(6), 731–747. [PubMed: 12084665]

Shull A, Mayer SE, McGinnis E, Geiss E, Vargas I, & Lopez-Duran NL (2016). Trait and state rumination interact to prolong cortisol activation to psychosocial stress in females. Psychoneuroendocrinology, 74, 324–332. 10.1016/j.psyneuen.2016.09.004 [PubMed: 27716572]

Singer T, Seymour B, O'Doherty JP, Stephan KE, Dolan RJ, & Frith CD (2006). Empathic neural responses are modulated by the perceived fairness of others. Nature, 439(7075), 466–469. [PubMed: 16421576]

Stadler T, Kirschbaum C, Kudielka BM, Adam EK, Wüst S, Dockray S, Smyth N, Evans P, Hellhammer DH, Miller R, Wetherell MA, Lupien SJ, & Clow A (2015). Assessment of the cortisol awakening response: Expert consensus guidelines. Psychoneuroendocrinology, 63–414.

Stawski RS, Cichy KE, Piazza JR, & Almeida DM (2013). Associations among daily stressors and salivary cortisol: Findings from the national study of daily experiences. Psychoneuroendocrinology, 38, 2654–2665. [PubMed: 23856186]

Stawski RS, Mogle J, & Sliwinski MJ (2011). Intraindividual coupling of daily stressors and cognitive interference in old age. Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 66(suppl_1), i121–i129. [PubMed: 21743045]

Stawski RS, Sliwinski MJ, & Smyth JM (2006). Stress-related cognitive interference predicts cognitive function in old age. Psychology and Aging, 21(3), 535–544. 10.1037/0882-7974.21.3.535 [PubMed: 16953715]

Thomsen DK, Mehlsen MY, Christensen S, & Zachariae R (2003). Rumination—relationship with negative mood and sleep quality. Personality and Individual Differences, 34(7), 1293–1301. 10.1016/S0191-8869(02)00120-4

Wegner DM, & Zanakos S (1994). Chronic thought suppression. Journal of Personality, 62, 615–640. 10.1111/j.1467-6494.1994.tb00311.x

Wells A, & Davies MI (1994). The Thought Control Questionnaire: A measure of individual differences in the control of unwanted thoughts. Behaviour Research and Therapy, 32, 871–878. 10.1016/0005-7967(94)90168-6 [PubMed: 7993332]

West SG, Taylor AB, & Wu W (2012). Model fit and model selection in structural equation modeling. In Hoyle RH (Ed.), Handbook of structural equation modeling (pp. 209–231). Guilford Press.

Williams SR, Lush E, Daup M, Geronimi EM, Sephton SE, & Woodruff-Borden J (2013). Exploring patterns in cortisol synchrony among anxious and nonanxious mother and child dyads: A preliminary study. Biological Psychology, 93(2), 287–295. 10.1016/j.biopsycho.2013.02.015 [PubMed: 23511898]

Yun HJ, Cui M, & Blair BL (2016). The mediating roles of adolescent disclosure and parental knowledge in the association between parental warmth and delinquency among Korean adolescents. Journal of Child and Family Studies, 25(8), 2395–2404.

Zoccola PM, & Dickerson SS (2015). Extending the recovery window: Effects of trait rumination on subsequent evening cortisol following a laboratory performance stressor. Psychoneuroendocrinology, 58, 67–78. 10.1016/j.psyneuen.2015.04.014 [PubMed: 25965871]

Zoccola PM, Quas JA, & Yim IS (2010). Salivary cortisol responses to a psychosocial laboratory stressor and later verbal recall of the stressor: The role of trait and state rumination. Stress: The International Journal on the Biology of Stress, 13(5), 435–443. 10.3109/10253891003713765 [PubMed: 20666646]

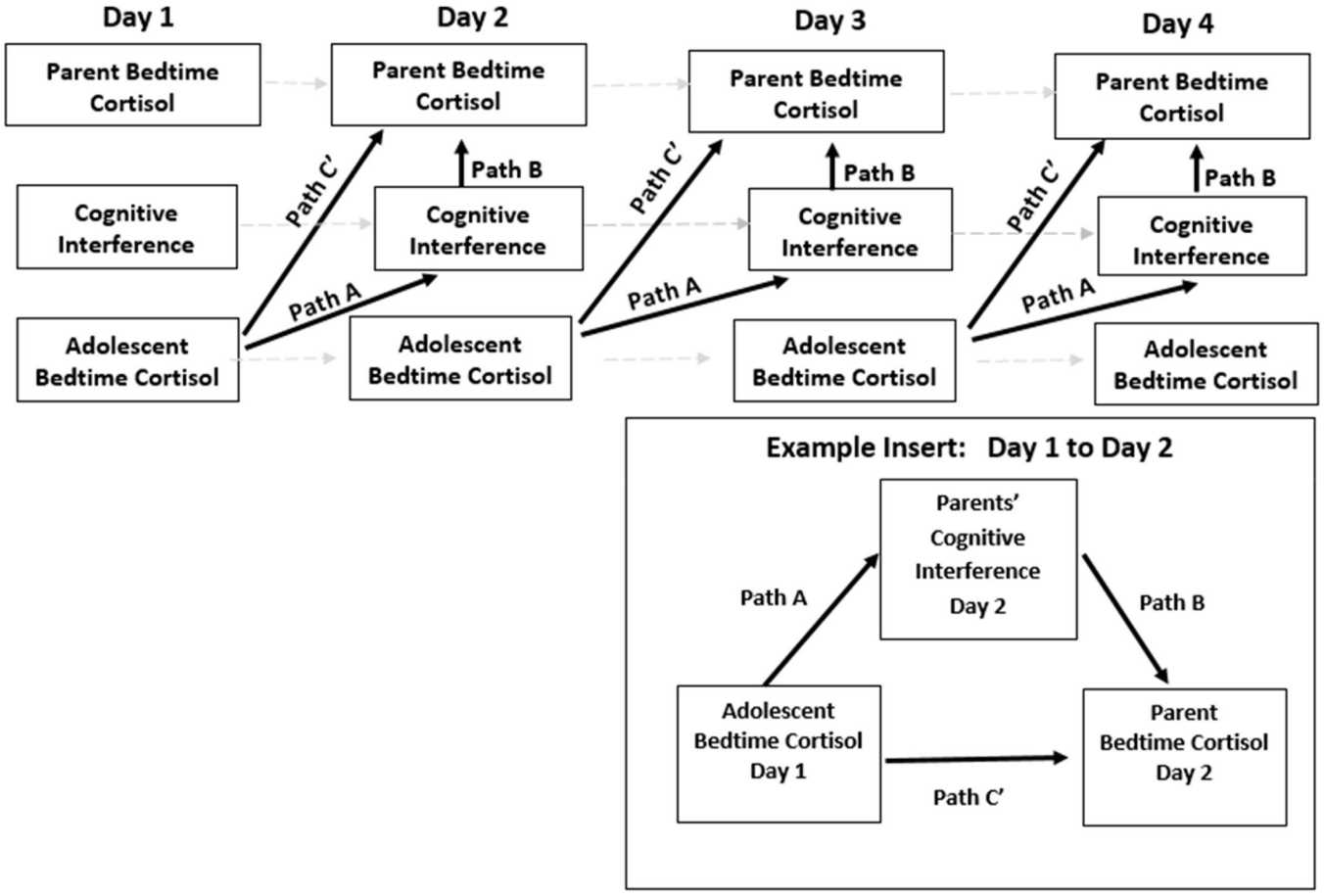

FIGURE 1.

Conceptual model. Parents' cognitive interference mediates the day-to-day linkages between child and parent cortisol. We hypothesize that child bedtime levels of cortisol will predict higher levels of parents' cognitive interference on the following day (Path A). Higher levels of parents' cognitive interference will predict higher same-day levels of parents' bedtime cortisol levels (Path B). Models control for the direct effects of child cortisol on next-day parent cortisol (Path C′) as well as child cortisol, parent cortisol, and mediator stability paths (dotted lines). Models control for demographics, daily stressors, type of day, and cortisol specifications. Variables collected at the same time are also correlated (not shown)

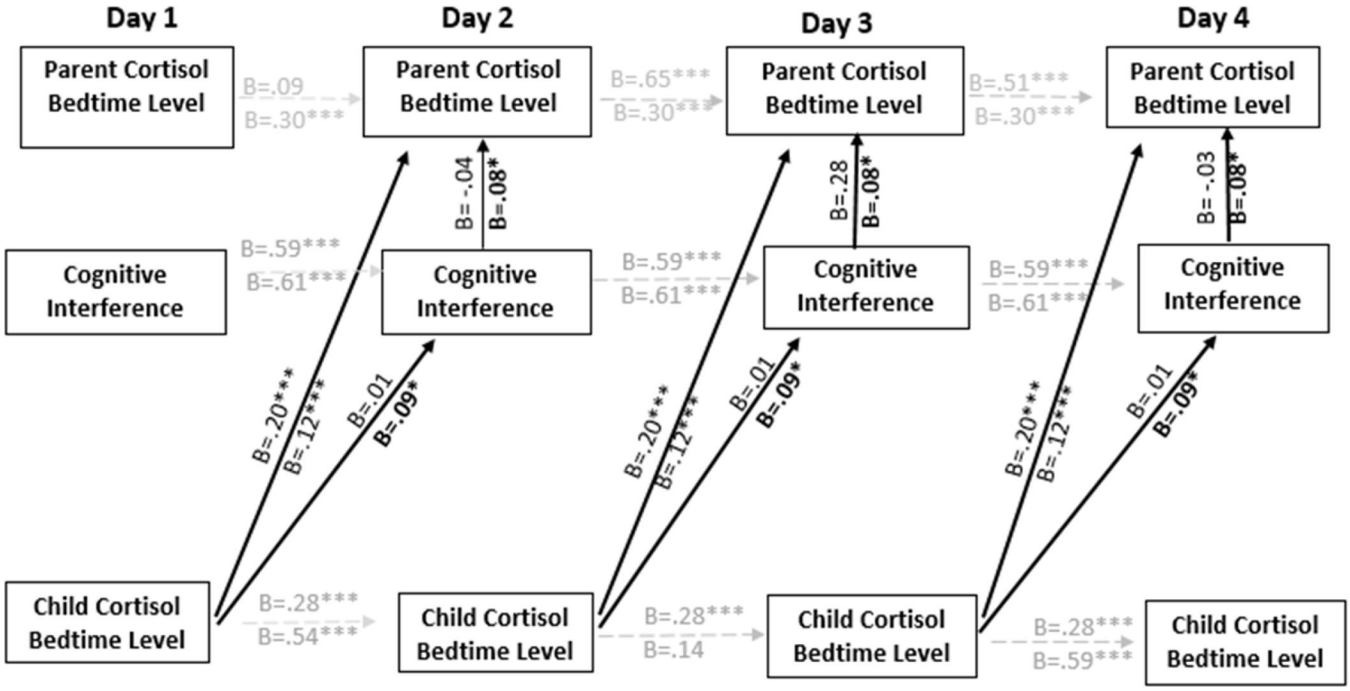

FIGURE 2.

Moderation by parent gender. Cognitive interference mediates the linkages between adolescent and parent cortisol for mothers but not fathers. For mothers, higher levels of adolescent cortisol at bedtime were associated with higher levels of mothers' cognitive interference (B = 0.09*) the following day. Subsequently, higher levels of cognitive interference were associated with higher levels of cortisol at bedtime for mothers (B = 0.08*). Unstandardized coefficients for fathers are above the line. Unstandardized coefficients for mothers are below the line. Models include additional control variables and correlations, including same day correlations between parent and adolescent cortisol (not shown in figure)

TABLE 1

Descriptive statistics

		1	2	3	4	5	6	7	8	9	10	11
1	Child bedtime cortisol	1										
2	Parent bedtime cortisol	0.37**	1									
3	Child AUC	0.40**	0.17**	1								
4	Parent AUC	0.30**	0.61**	0.24**	1							
5	Child CAR	−0.14*	−0.12*	0.35**	0.05	1						
6	Parent CAR	0.02	0.04	−0.06	0.30**	0.23**	1					
7	Parent cognitive interference	0.04	0.10	−0.07	0.06	−0.05	−0.01	1				
8	Parent gender	0.14*	0.07	−0.07	−0.09	0.05	0.12*	0.09	1			
9	Parental warmth	0.05	0.11	0.07	0.12	−0.00	−0.02	0.02	0.02	1		
10	Child gender	0.09	0.02	0.14*	0.05	0.16*	−0.05	−0.08	−0.03	−0.18*	1	
11	Child age	0.22**	0.08	0.36*	0.07	0.09	0.04	0.01	−0.01	0.09	0.05	1
Mean (SD)		0.86 (0.51)	1.02 (0.44)	21.58 (5.33)	24.11 (5.09)	0.23 (0.45)	0.26 (0.44)	1.61 (0.69)	74% mothers	3.71 (1.48)	52% girls	13.18 (2.28)

Note: Correlations were calculated using the average of cortisol variables and cognitive interference across 4 days.

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$.

TABLE 2

Testing invariance across days

	Main effect models			Subgroup models					
				$\Delta\chi^2$	df	Invariant paths	$\Delta\chi^2$	df	Invariant paths
	$\Delta\chi^2$	df	Invariant paths	Fathers			Mothers		
Bedtime levels of cortisol									
Adolescent cortisol to mediator (Path A)	0.12	2	Yes	1.34	2	Yes	0.77	2	Yes
Mediator to parent cortisol (Path B)	2.24	2	Yes	6.46*	2	No	1.06	2	Yes
Direct effect from adolescent to parent cortisol (Path C′)	0.42	2	Yes	5.55	2	Yes	0.94	2	Yes
Parent cortisol stability paths	0.57	2	Yes	10.30*	2	No	1.30	2	Yes
Mediator (cognitive interference) stability paths	0.12	2	Yes	0.87	2	Yes	0.21	2	Yes
Adolescent cortisol stability paths	17.43**	2	No	1.53	2	Yes	19.11**	2	No
Area under the curve (AUC)									
Adolescent cortisol to mediator (Path A)	0.24	2	Yes	1.34	2	Yes	0.44	2	Yes
Mediator to parent cortisol (Path B)	3.12	2	Yes	4.74	2	No	6.09*	2	Yes
Direct effect from adolescent to parent cortisol (Path C′)	1.82	2	Yes	3.51	2	Yes	3.73	2	Yes
Parent cortisol stability paths	3.22	2	Yes	1.24	2	No	1.32	2	Yes
Mediator (cognitive interference) stability paths	0.14	2	Yes	2.51	2	Yes	0.01	2	Yes
Adolescent cortisol stability paths	13.27**	2	No	0.85	2	Yes	8.18*	2	No
Cortisol awakening response (CAR)									
Adolescent cortisol to mediator (Path A)	3.80	2	Yes	-			-		
Mediator to parent cortisol (Path B)	11.64**	2	Yes	-			-		
Direct effect from adolescent to parent cortisol (Path C′)	0.96	2	Yes	-			-		
Parent cortisol stability paths	7.84*	2	Yes	-			-		
Mediator (cognitive interference) stability paths	1.79	2	Yes	-			-		
Adolescent cortisol stability paths	0.02	2	No	-			-		

Note: Because there was no evidence of moderation by parent gender in CAR models, subgroup models for mothers and fathers are not presented.

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$.