



RESEARCH ARTICLE

Prenatal mother–father cortisol linkage predicts infant executive functions at 24 months

Stephen H. Braren¹  | Rosemarie E. Perry¹ | Andrew Ribner¹ |
Annie Brandes-Aitken¹ | Natalie Brito¹  | Clancy Blair¹ | the New Fathers and
Mothers Study (NewFAMS) Team^{1,2,3}

¹ Department of Applied Psychology, New York University, USA

² Centre for Family Research, University of Cambridge, UK

³ Faculty of Social Sciences, University of Leiden, The Netherlands

Correspondence

Stephen H. Braren, 627 Broadway, 8th Floor, New York, NY, 10012, USA.

E-mail: stephen.braren@nyu.edu

Abstract

The present study investigated associations between prenatal mother–father cortisol linkage and infant executive functions. Data come from an international sample ($N = 358$) of predominantly white and middle- to upper-class first-time parents. During late pregnancy, parents collected diurnal salivary cortisol samples and reported on levels of psychological stress. At 24 months, children completed a battery of executive function tasks. Parent cortisol linkage was operationalized as the time-dependent, within-dyad association between maternal and paternal diurnal cortisol. Results indicated that prenatal linkage was positively related to infant executive functions, suggesting that stronger mother–father cortisol linkage was associated with higher executive function scores. Additionally, this relation was moderated by paternal average cortisol levels such that executive function scores were lower when fathers had higher average cortisol levels and linkage was weak. This association suggests that elevated paternal cortisol amplifies the negative relation between lower cortisol linkage and lower infant executive function scores. Importantly, these findings were observed while controlling for observational measures of caregiving and self-report measures of psychosocial functioning and infant social-emotional behavior. These results suggest that prenatal linkage of mother's and father's stress physiology plays a potentially important part in programming and regulating infant neurocognitive development.

KEYWORDS

co-regulation, self-regulation, executive function, cortisol, fathers

1 | INTRODUCTION

The capacity to self-regulate cognition, emotion, physiology, and behavior develops rapidly during the first few years of life. The development of early self-regulation is not a simple, individual process of maturation but rather depends greatly on the social co-regulation of infant functioning by caregivers (Blair & Ursache, 2011; Calkins et al., 2016; Feldman et al., 1999; Fogel, 1993; Kopp, 1982; Vygotsky, 1978). Traditionally, caregiver–infant co-regulation in early development has been

examined on the behavioral level postnatally, although co-regulation on the physiological level occurs even during pregnancy via maternal regulation of the fetal environment (Bobin-Bègue, 2019; Gitau et al., 1998; Seckl & Meaney, 2004). Research shows that pregnancy is a sensitive period of development that is shaped by social and environmental factors and the regulation of stress physiology (Glynn et al., 2018; Saxbe et al., 2018). In particular, fetal programming via maternal cortisol may impact neurocognitive development and executive functions—a set of core cognitive skills comprised of inhibitory control, working

memory, and attention shifting that are recruited in the volitional pursuit of goals (Bridgett et al., 2015; Perry et al., 2019; Zijlmans et al., 2015).

Understandably, much of the research on prenatal predictors of infant developmental outcomes has focused on the role of the mother. But fathers are also key players in supporting and co-regulating their partners during pregnancy and potentially shaping fetal development (Brumberg & Shah, 2020; Hughes et al., 2020; Plantin et al., 2011; Saxbe et al., 2018). However, research on the role of the father during (and following) pregnancy is understudied (Samdan et al., 2020). In particular, little is known about how mother–father co-regulation relates to infant developmental outcomes such as self-regulation and executive functions. Specifically, more work is needed to understand the complex, dynamic, and intergenerational processes linking prenatal caregiver co-regulation and infant self-regulation. Of particular interest is whether a couple's ability to co-regulate stress physiology during pregnancy might affect the prenatal environment and impact infant development. In line with this, some studies suggest that correlated, or “linked,” physiological activity between partners in close relationships may reflect partners' ability to mutually influence or co-regulate each other's physiological and psychological stress (Meyer & Sledge, 2020; Palumbo et al., 2017; Saxbe et al., 2018; Timmons et al., 2015). In the present study, we examined the potential role of prenatal mother–father cortisol linkage in the development of infant executive functions.

1.1 | Cortisol linkage in close relationships: Theory and evidence

Close interpersonal relationships are complex, dynamic systems in which people are constantly engaged in a reciprocal exchange of bio-behavioral cues that can influence each other on different levels of functioning, from the physiological and behavioral to the cognitive and emotional (Butler, 2011; Cox & Paley, 1997; Field, 2012; Fogel, 1993; Sbarra & Hazan, 2008). Research has repeatedly shown that partners in close relationships mutually influence or co-regulate each other directly and indirectly, consciously and non-consciously, to impact mental and physical health and functioning for better and for worse (Butler, 2011; Field, 2012; Hofer, 1994; Kiecolt-Glaser & Wilson, 2017; Meyler et al., 2007; Pietromonaco et al., 2013; Robles et al., 2014; Sbarra & Hazan, 2008).

One process by which interpersonal processes of co-regulation may occur is via the time-dependent association between partners' physiological activity—also known as physiological linkage. Although there is still no consensus on how to define them, processes of co-regulation are sometimes investigated through constructs such as synchrony, linkage, covariation, or attunement. Each of these terms describes either similarity or dissimilarity between two (or more) people's psychophysiological activity. Theoretically, physiological linkage may reflect one's sensitivity to send and receive subtle, non-conscious social-emotional cues, and the capacity to adapt to or influence one another (Butler, 2011; Feldman et al., 1999; Harrist & Waugh, 2002; Meyer & Sledge, 2020; Timmons et al., 2015). Theory and research indicate that link-

age processes are fundamental to the organization of development and social-emotional processes, such as empathy and attachment, across the lifetime (Butler & Randall, 2013; Field, 2012; Sbarra & Hazan, 2008; Timmons et al., 2015). Furthermore, some researchers suggest that our ability to physiologically connect with others may have evolved specifically for the purpose of responding adaptively to threats to promote survival and maintain individual homeostasis (Beckes & Coan, 2011).

Much of the research on physiological linkage has focused on the hypothalamic-pituitary-adrenal (HPA) axis and its primary output, the hormone cortisol. Cortisol linkage is likely important because the HPA axis mediates the response to threat and stress and thus is essential to basic survival and functioning. Moreover, the HPA axis is highly susceptible to social and environmental influence and, consequently, cortisol is a likely mediator of physiological linkage and co-regulation (Gunnar & Donzella, 2002; Saxbe et al., 2018). Several studies have suggested that couples in close romantic relationships have linked diurnal cortisol activity and cortisol reactivity to stressors (Meyer & Sledge, 2020; Timmons et al., 2015). Although the vast majority of this research has been with couples in the postnatal period, physiological linkage as it pertains to children's early development may be especially important during pregnancy.

Cortisol is an essential hormone during pregnancy that regulates the growth and maturation of fetal organ systems (Busada & Cidlowski, 2017; Morsi et al., 2018). Relatedly, cortisol is also important for parenting behaviors and, during pregnancy, may support the transition to parenthood for both mothers and fathers (Abraham & Feldman, 2018; Almanza-Sepulveda et al., 2020; Bos, 2017; Gettler, 2014; Storey & Ziegler, 2016; Wynne-Edwards, 2001). Indeed, research has shown that maternal and paternal cortisol in and around pregnancy are related to caregiving behaviors and parental involvement (Barrett & Fleming, 2011; Bos et al., 2018; Kuo et al., 2018; Monk & Hane, 2014; Zijlmans et al., 2015).

Over the course of pregnancy, daily maternal cortisol output along with other hormones, including estradiol, progesterone, and testosterone, exhibit drastic changes (Glynn & Sandman, 2011; Saxbe et al., 2018). Moreover, cross-species research with human and non-human primates shows that like their partner, expectant fathers also display similar changes in diurnal hormone activity over the course of pregnancy (Gettler, 2014; Storey & Ziegler, 2016). In light of these correlated hormonal changes between partners during pregnancy, hormonal linkage between expectant parents may generally be an important adaptive and normative process that facilitates the transition to parenthood while also supporting fetal development via co-regulation of fetal cortisol (Abraham & Feldman, 2018; Gettler, 2014; Storey & Ziegler, 2016). Theoretically, hormonal linkage between expectant parents during pregnancy may reflect the couple's connectedness, commitment to each other, and investment in their relationship (Saxbe et al., 2017). Thus, more broadly, cortisol linkage during pregnancy may reflect an adaptive process that indicates healthy relationship functioning.

Despite the importance of cortisol during pregnancy, only a few studies have examined diurnal cortisol linkage between expectant parents in humans (Berg & Wynne-Edwards, 2002; Braren et al., 2020;

Edelstein et al., 2015; Storey et al., 2000). These studies have all found correlations between partners' diurnal cortisol activity, suggesting that, in general, cortisol linkage may be a normative process during pregnancy that may influence infant development. Yet no studies that we are aware of have examined the developmental consequences of linkage for the developing child. Thus, more research is needed to understand how physiological linkage functions during pregnancy and potentially relates to postnatal infant development.

1.2 | Moderators of cortisol linkage

Cortisol linkage is not necessarily a simple, linear, all-or-none phenomenon but demonstrates considerable variation that depends on various factors. For instance, in close adult relationships, linkage is associated with both risk and protective factors (Meyer & Sledge, 2020; Timmons et al., 2015). Some studies have found that the magnitude of diurnal cortisol linkage and cortisol reactivity in non-expectant couples is often greater in relationships that report heightened levels of marital strain, aggression, conflict, or lack of support (Ha et al., 2016; Laws et al., 2015; Liu et al., 2013; Saxbe & Repetti, 2010; Saxbe et al., 2015; Schneiderman et al., 2014). Other studies, however, have found that greater cortisol linkage is related to physical closeness and time spent together (Laws et al., 2015; Papp, Pendry, Simon, & Adam, 2013; Saxbe & Repetti, 2010). In general, these studies have also found that cortisol linkage is present on average across couples. Taken together, these studies suggest that some degree of cortisol linkage is normative and may reflect healthy relationship functioning but that in some relationships, higher linkage may indicate negative relationship functioning. More generally, as these studies and others suggest, the context within which physiological linkage occurs is essential to understanding how linkage functions (M. Davis et al., 2018; Palumbo et al., 2017; Timmons et al., 2015).

Because pregnancy is a unique context, it is not clear whether previous findings generalize to expectant parents during pregnancy. Pregnancy is a transformative time in a couple's relationship, especially for first-time parents (as we investigate in the present analysis; Glynn et al., 2018; Saxbe et al., 2018). Given that during pregnancy, as discussed above, cortisol is a key hormone supporting fetal development and parenting and that it undergoes drastic changes in both mothers and fathers, cortisol linkage may serve a unique function during pregnancy. Furthermore, in light of the idea (also discussed above) that hormonal linkage between expectant parents during pregnancy may be a normative and adaptive process reflecting healthy relationship functioning, a greater magnitude of hormonal linkage in expectant parents may function as a supportive process that confers benefit on the developing fetus. Thus, more research on moderators is needed to understand how linkage functions during pregnancy.

Only one previous study that we are aware of has assessed moderators of parent linkage during pregnancy. In a previous analysis with the present sample, we found that cortisol linkage was stronger when mothers reported higher levels of psychological stress (Braren et al., 2020). When maternal psychological stress was low, there was no

relation between maternal and paternal cortisol. Whether this finding reflects an adaptive or maladaptive process, however, is not clear partially because this analysis was cross-sectional, and thus we could not infer whether linkage was influencing stress or vice versa. As noted in the paper, cortisol linkage may indicate a risk or a protective factor. For instance, on one hand, higher psychological stress in the presence of higher cortisol linkage may facilitate the transmission of stress between partners. On the other hand, higher linkage may present an opportunity for the partner to down-regulate or buffer the mother's elevated stress. In fact, we found that when maternal psychological stress was high and paternal cortisol was low, maternal cortisol was also low. Conversely, when maternal psychological stress was high and paternal cortisol was high, maternal cortisol was also high. In this way, cortisol linkage may be good or bad depending on the partner's cortisol level. Because pregnancy and the transition to parenthood are periods of immense change, some degree of increased stress during pregnancy is normative and likely even adaptive. Excessively high levels of parent stress during pregnancy, however, may dysregulate maternal-fetal cortisol activity and impact fetal development (Bowers & Yehuda, 2016; Seckl & Meaney, 2004). Moreover, levels of parent psychosocial stress and relationship risk may be highest during late gestation, just prior to childbirth (Philpott et al., 2017; Saxbe et al., 2018). For these reasons, psychological stress is likely an important factor during pregnancy that may be associated with cortisol linkage and infant development.

Similar to psychological stress, physiological stress may also be an important moderator of cortisol linkage, especially during late gestation when maternal-fetal cortisol levels reach their highest levels (Glynn & Sandman, 2011; Saxbe et al., 2018). However, most studies on physiological linkage overlook the role of each individual person's activity of the physiological system being measured. Yet a person's overall level of the physiological activity of a given system may influence whether linkage in the same physiological system occurs (Butler, 2011). For instance, cortisol linkage may be engaged only when a person's cortisol level is sufficiently high or low. Conversely, linkage may be disrupted when a person's cortisol output is too high or too low. Conceptually, this relation makes sense inasmuch as a person's average level of physiological activity is indicative of physiological self-regulation. Thus, it seems logical that dyadic physiological linkage might depend on each person's physiological self-regulation. Indeed, one of the hypothesized functions of linkage is to maintain or restore individual homeostatic balance, thus supporting self-regulation. Surprisingly, however, most studies examining within-dyad linkage have not concurrently assessed individual levels of physiological activity.

1.3 | Linkage and the development of self-regulation and executive functions

One of the most notable gaps in research on physiological linkage is that most studies have assessed associations between linkage and child or parent outcomes cross-sectionally, at a single time point. Yet a pressing question in this area of research is how might earlier parent physiological linkage shape subsequent development of their

offspring? More specifically, how might mother–father physiological linkage affect the development of infant self-regulation and executive functions? In answering these questions, longitudinal studies are needed to help us understand such developmental trajectories.

In recent years, strides have been made in understanding how self-regulation capacities such as executive functions develop early in life. Hierarchical and bidirectional models of self-regulation emphasize how reciprocal relations between social and biological factors contribute to executive function development (Blair & Ursache, 2011; Calkins et al., 2016; Kopp, 1982; Sameroff, 2009). For instance, on one hand, studies have shown that environmental and social factors, such as parenting and peer relations, play an important role in shaping early executive functions (Bridgett et al., 2015; Deater-Deckard, 2014; Finegood & Blair, 2017; Hughes & Ensor, 2009; Perry et al., 2018). On the other hand, executive functions rely heavily on the growth and functioning of the brain and supporting physiology—namely, the HPA axis in conjunction with the limbic system (amygdala and hippocampus) and the prefrontal cortex (PFC; Arnsten, 2009; Arnsten & Li, 2005; Perry et al., 2019). Thus, nature and nurture work hand-in-hand to shape the development of executive functions as experience affects physiological processes that direct brain growth and development.

Most research on the development of executive functions has been on postnatal functioning, but cognitive development begins even before birth. Based on studies on the developmental origins of health and disease, we now know that the prenatal period is an especially sensitive period of development and susceptible to influences from the environment (Glynn et al., 2018; Saxbe et al., 2018). The fetal programming hypothesis posits that exposure to maternal risk factors such as psychosocial stress can affect fetal and infant development (Bowers & Yehuda, 2016; Gitau et al., 1998; Seckl & Meaney, 2004). In support of this, several studies have found that heightened maternal stress during pregnancy is negatively related to infant and child general cognitive functioning (Graignic-Philippe et al., 2014; Talge et al., 2007) and, in particular, executive functions (Buss et al., 2011; Camerota & Willoughby, 2020; El Marroun et al., 2017; Neuenschwander et al., 2018).

A key mechanism in fetal programming is thought to be the HPA axis and glucocorticoid activity (Glynn et al., 2018; Seckl & Meaney, 2004; Zijlmans et al., 2015). Indeed, cross-species studies show that prenatal maternal cortisol regulates the growth and development of the HPA axis, and brain areas rich in glucocorticoid receptors such as the hippocampus, amygdala, and PFC (Brunton & Russell, 2011; Seckl & Meaney, 2004; Ulrich-Lai & Herman, 2009). However, exposure to excessive cortisol in utero may have long-term effects on the development of cognitive processes including executive functions (Bridgett et al., 2015; Moisiadis & Matthews, 2014; Monk et al., 2019). Given the centrality of the HPA axis in regulating executive functions, it is plausible that maternal and, by extension, paternal cortisol during pregnancy might be related to executive function in the child. Indeed, a growing body of research has shown associations between diurnal or resting maternal cortisol during pregnancy and child outcomes, such as HPA axis functioning (Davis et al., 2011; O'Connor et al., 2013; Rash et al., 2016), and neurocognitive development (Buss et al., 2012; Davis &

Sandman, 2010; Davis et al., 2017; Graignic-Philippe et al., 2014; Nazzari et al., 2020).

The previously reviewed research clearly suggests that the mother is an important regulator of the fetus. Yet fathers likely also play a role in co-regulating the prenatal environment indirectly through the relationship with their partners (Brumberg & Shah, 2020; Plantin et al., 2011; Saxbe et al., 2018). However, the prenatal programming hypothesis and related research have by and large neglected the role that fathers might play in influencing the intrauterine environment and fetal development. To the extent that research has investigated the father's role during pregnancy in relation to child outcomes, some studies have assessed the father's potential influence indirectly through measures of maternal-reported social support, marital conflict, and relationship quality during pregnancy (Brumberg & Shah, 2020; Hanington et al., 2012; Plantin et al., 2011; Stapleton et al., 2012; Thomas et al., 2018). In general, these studies suggest that paternal support and involvement may confer some benefit on the pregnant mother and the developing fetus to positively shape infant outcomes.

Additionally, studies have also found direct relations between prenatal psychological paternal factors, such as paternal depression and stress and postnatal child outcomes (Brumberg & Shah, 2020; Letourneau et al., 2019; Velders et al., 2011). These results may also be partially mediated prenatally via the mother–father relationship. Alternatively, prenatal paternal characteristics may extend into the postpartum and directly impact the child through father–child interactions, for example. Taken together, these studies suggest that paternal psychological stress may play a part in co-regulating the fetal environment and, along with the mother, potentially programming infant development. However, we are not aware of any studies that have explicitly investigated the prenatal physiological linkage between parents in relation to postnatal child cognitive outcomes related to self-regulation or executive functions.

1.4 | The current study

In order to address some of the limitations of prior research, in the current analysis, we used data from a large, longitudinal international sample of families in three countries to investigate the extent to which mother–father diurnal cortisol linkage during pregnancy longitudinally predicted infant executive function performance. We operationalized linkage as the within-dyad correlation between mother's and father's diurnal cortisol activity measured across two typical days. Our main aim was to assess whether prenatal mother–father cortisol linkage was associated with infant executive functions at 24 months, over and above the main effects of mother's and father's average cortisol levels and reports of psychological stress. Our second aim was to assess whether linkage was moderated by mother's or father's overall cortisol levels or self-reported levels of psychological stress. Based on previous literature suggesting that hormonal linkage between expectant parents may be a normative and adaptive process (Berg & Wynne-Edwards, 2002; Edelstein et al., 2015; Gettler, 2014; Saxbe et al., 2017; Storey & Ziegler, 2016; Storey et al., 2000), we hypothesized that

cortisol linkage would be positively related to infant executive functions on average. Additionally, based on research suggesting that higher parent psychological and physiological stress is generally negatively associated with child developmental outcomes (Bowers & Yehuda, 2016; Brumberg & Shah, 2020; Glynn et al., 2018; Monk et al., 2019; Seckl & Meaney, 2004; Talge et al., 2007), we hypothesized that higher mother or father cortisol and/or higher self-reported stress would be associated with lower infant executive functions in the presence of lower cortisol linkage. Furthermore, we included several covariates in our analyses to assess the robustness of our hypothesized results. Previous research suggests that parent involvement, caregiving sensitivity, social support, relationship quality, and child social-emotional behavior may be associated with either cortisol activity and/or child outcomes and thus may account for these relations (Bridgett et al., 2015; Brumberg & Shah, 2020; Hanington et al., 2012; Hughes et al., 2020; Perry et al., 2019; Plantin et al., 2011; Stapleton et al., 2012). We included these self-report and observational measures to assess whether cortisol linkage explains unique variation in infant executive functions over and above them. By controlling for these factors, we can infer that the association between parents' diurnal cortisol is a uniquely important and robust interpersonal phenomenon that likely contributes to infant cognitive development. Additionally, it is theorized that physiological linkage and co-regulation often occur outside of conscious awareness or intentional behavior and thus may be mediated by subtle and indirect mechanisms (Field, 2012; Harrist & Waugh, 2002; Meyer & Sledge, 2020; Sbarra & Hazan, 2008). This idea is consistent with Hofer's (1994) "hidden regulators" hypothesis. In line with this idea, by including measures of subjective psychosocial functioning and global observations of behavior, we can test whether cortisol linkage may function as a hidden regulator of infant development.

2 | METHODS

2.1 | Participants and procedure

Participants for this analysis come from the New Fathers and Mothers Study, a prospective, international, longitudinal study investigating the transition to parenthood and infant development from late pregnancy through infancy (Hughes et al., 2018). Expectant couples in heterosexual partnerships ($N = 484$) were recruited from prenatal classes, hospitals, and clinics in the United States ($N = 131$), the United Kingdom ($N = 221$), and the Netherlands ($N = 132$). Couples were eligible to participate in the study if they were cohabiting first-time parents, expecting a healthy singleton baby, had no history of severe mental illness or substance use, and planning to speak English (or Dutch in the Netherlands) as the primary language. Table 1 contains descriptive statistics for those included in the current analysis.

For this analysis, we used data collected at three time points: When mothers were in the last trimester of pregnancy at approximately 36 weeks gestation (T1), when infants were approximately 14 months old (T2), and when infants were approximately 24 months old (T3). At each time point, parents completed online surveys reporting vari-

ous socioeconomic and demographic information, as well as questionnaires on psychosocial functioning and mental health. At T1, parents collected diurnal saliva samples, which were later assayed for cortisol. When children were 14 and 24 months old, families participated in a home visit that included parent-infant interactions, parent questionnaires, and infant executive function tasks.

All parents provided written informed consent at all time points. The study protocol was approved by the National Health Service Research Ethics Committee in the United Kingdom, the Institutional Review Boards at New York University in the United States, and Leiden University in the Netherlands.

2.2 | Measures

2.2.1 | Cortisol

At the T1 prenatal time point, parents were given supplies and instructions to collect saliva at home three times on two consecutive, typical days. Samples were to be taken immediately upon waking, 30 min after waking and just before sleep. The procedure required parents to place a 30 mm cotton swab beneath their tongue for 2 min after which the swab was immediately placed in a storage tube in their home freezer to be later picked up by a research assistant. Upon collection, samples were transported on ice and stored in a freezer (-20°C) until shipped on dry ice for processing at Universität Trier, Germany. All samples were assayed in duplicate and the average of both for each sample was used in analyses. Inter- and intra-assay coefficients of variation were on average 6% and 5%, respectively. Parents were given detailed instructions on how to properly collect saliva and were instructed for each sample to not consume alcohol at least 12 h before, to not eat at least 1 h before, to not consume dairy at least 20 min before, and to not consume caffeine or brush their teeth at least 45 min before sample collection. Last, parents were given a saliva diary and asked to record for each sample the date and time of sampling, as well as other relevant information that may influence cortisol activity (outlined above, i.e., whether each parent had followed the instructions provided).

2.2.2 | Executive functions

During the T3 home visit at 24 months, the children completed a battery of three executive function tasks. The infant sat in the parent's lap at a table across from the research assistant. Parents were asked to not talk to or assist their child in any way during the tasks. The tasks were video recorded and later scored offline.

The *Multilocation Search Task* (Miller & Marcovitch, 2015) is a measure of working memory. In the task, children are required to find five cars hidden in five colored boxes with a 5 s pause in between each search trial. Testing was discontinued when children made three consecutive errors. Children scored correctly on a trial if they pointed to a box that contained a car. Thus, on the first trial, any response was correct. Once a car was "found," the experimenter removed the car and clearly showed to the infant that the car was placed in a bag behind

TABLE 1 Descriptive statistics

| | N | M or % | SD | Min | Max |
|--|-----|--------|-------|--------|--------|
| Mother's age T1 (years) | 354 | 32.36 | 3.80 | 22.66 | 43.68 |
| Father's age T1 (years) | 341 | 34.02 | 4.54 | 23.00 | 50.07 |
| Mother ethnicity (% White) | 340 | 89.7% | - | - | - |
| Father ethnicity (% White) | 338 | 91.7% | - | - | - |
| Child's sex (% male) | 359 | 50.7% | - | - | - |
| Child age T3 | 337 | 24.46 | 0.81 | 19.43 | 26.97 |
| Gestational age T1 (weeks) | 352 | 36.36 | 1.76 | 31.00 | 41.00 |
| Gestational age at birth (weeks) | 350 | 39.82 | 1.27 | 33.00 | 46.00 |
| Child birth weight (lbs.) | 331 | 3.68 | 1.10 | 1.98 | 10.60 |
| Mother education T1 | 358 | 5.36 | 1.19 | 2.00 | 7.00 |
| Father education T1 | 344 | 5.09 | 1.43 | 1.00 | 7.00 |
| Mother employment status T1 (% full time) | 354 | 79.4% | - | - | - |
| Father employment status T1 (% full time) | 353 | 92.9% | - | - | - |
| Household income T1 | 345 | 2.70 | 1.25 | 0.74 | 8.63 |
| Father psychological stress T1 (factor score) | 342 | 0.05 | 1.02 | -1.42 | 3.95 |
| Mother psychological stress T1 (factor score) | 358 | 0.05 | 0.98 | -1.58 | 4.35 |
| Father social support T1 | 340 | 66.48 | 12.94 | 12 | 84 |
| Mother social support T1 | 357 | 71.00 | 11.02 | 12 | 84 |
| Father relationship quality T1 (factor score) | 342 | -0.04 | 0.98 | -4.02 | 1.92 |
| Mother relationship quality T1 (factor score) | 357 | -0.02 | 1.02 | -6.67 | 1.85 |
| Parental involvement T2 | 315 | 4.07 | 1.01 | 1.25 | 7.75 |
| Maternal sensitivity T2 | 348 | 6.15 | 1.54 | 2.00 | 9.00 |
| Paternal sensitivity T2 | 334 | 5.78 | 1.62 | 2.00 | 9.00 |
| Maternal BITSEA T2 | 327 | 12.62 | 1.88 | 9.00 | 17.00 |
| Paternal BITSEA T2 | 304 | 11.34 | 1.19 | 9.00 | 16.00 |
| Multilocation Search Score T3 (empirical Bayes estimate) | 359 | 0.01 | 0.38 | -1.08 | 0.83 |
| Ball run score T3 (empirical Bayes estimate) | 359 | 0.01 | 0.37 | -0.91 | 0.68 |
| Baby Stroop score T3 (empirical Bayes estimate) | 359 | 0.00 | 0.38 | -0.79 | 0.92 |
| Father cortisol T1 (AUCg) | 339 | 232.24 | 92.21 | 25.87 | 600.64 |
| Mother cortisol T1 (AUCg) | 343 | 358.69 | 99.39 | 139.59 | 700.86 |
| Mother-father cortisol linkage T1 (empirical Bayes estimate) | 359 | 0.11 | 0.08 | -0.15 | 0.34 |

Note: Household income was computed as couples' average income proportional to the median income of the smallest area for which data were available (i.e., New York State, Cambridgeshire, and the Netherlands). T1: prenatal time point; T2: 14-month time point; T3: 24-month time point; BITSEA: Brief Infant-Toddler Social and Emotional Assessment; AUCg: area under the curve with respect to ground.

the experimenter. If the child pointed to a box that had already been chosen, it was coded as an error response. Children were given feedback on each trial. For this task, children engage working memory to remember the box chosen on the previous trial and inhibitory control to resist choosing the same location on the subsequent trial. The scoring procedure for this task was based on the total number of searches to successfully find a car on each trial except the first (i.e., 0 = did not find the car; 1 = 3 searches; 2 = 2 searches; 3 = 1 search).

The *Ball Run Task* (Devine et al., 2019) is a measure of attention shifting. In the task, children are shown a toy with three colored holes in the top through which a corresponding colored ball could be inserted and would then roll down to the bottom. At the bottom of the toy, there was

a speaker that could be activated by the rolling ball, that would play 5 s of a children's song (*The Wheels on the Bus*). Two movable metal brackets were fixed to the top allowing the experimenter to block any two of the three holes, leaving one hole open (the middle hole was always closed). The infant was shown which holes were open and closed. In the learning phase, the experimenter demonstrated how the toy worked by inserting either the red ball into the red hole or the green ball into the green hole and letting it roll to the bottom. The ball was then handed to the infant who was given six chances to place the ball in the open hole. If the child successfully completed four of the six trials, the task continued to the second phase. In the reversal phase, the researcher closed the previously used hole and opened a different hole. Again,

the researcher demonstrated the toy by placing a different colored ball into the correspondingly colored open hole (e.g., the green ball into the green hole). The experimenter then handed the ball to the child who again completed another six trials. A trial was scored as correct if the infant placed the ball into the open hole and incorrect if placed in either of the two closed holes. Infants were given feedback on each trial. In this task, children must shift attention to match the color of the hole with the color of the ball and shift attention during the reversal learning phase. Scoring for this task consisted of performance (0 = incorrect; 1 = correct) on each of the six trials of each phase for a total of 12 trials.

The *Baby Stroop Task* (Hughes & Ensor, 2005) is a measure of inhibitory control. In the task, children are shown two spoons, a large spoon (a standard soup spoon) and a small spoon (a child-sized, rubberized spoon). After correctly identifying the large spoon as the “mommy spoon” and the small spoon as the “baby spoon,” the child is told they are going to play a “silly game” in which the large spoon was referred to as the “baby spoon” and a small spoon referred to as “mommy spoon.” After children demonstrated understanding of the game, they completed six trials in which the experimenter asked the infant to point to either the “baby spoon” or “mommy” spoon. A correct response was coded if the infant pointed to the large spoon when asked to indicate the “baby” spoon or if the infant pointed to the small spoon when asked to indicate the “mommy” spoon. The scoring procedure was based on performance on each of the six trials (0 = incorrect; 1 = correct). Again, children were given feedback on each trial. For this task, children inhibit a highly learned response based on typically learned naming conventions (e.g., that the “mommy” spoon is the large spoon).

Previous analyses with this dataset and others show support for the validity and reliability of these tasks. In particular, Hughes et al. (2020) found that executive functions measured with these tasks at 14 and 24 months were correlated ($r = 0.13$, $p < 0.05$). Similarly, McHarg et al. (2020) found that this set of tasks was moderately correlated at 24 and 36 months ($r = 0.15$, $p = 0.05$). Latent factor scores of these tasks at 14 months have also shown high degrees of reliability (Devine et al., 2019). Furthermore, these tasks have been used in other studies and have demonstrated expected associations with other related constructs (e.g., language, Miller & Marcovitch, 2015; theory of mind, Hughes & Ensor, 2005). Additional details on this battery can be found in Devine et al. (2019) and Hughes et al. (2020).

2.2.3 | Psychological stress

As done previously (Braren et al., 2020; Hughes et al., 2020), we calculated latent factor scores for maternal and paternal psychological stress variables using self-reported Center for Epidemiological Studies–Depression (CES-D; Radloff, 1977), the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983), and the General Health Questionnaire (GHQ; Goldberg et al., 1997) at the T1 prenatal time point. Internal reliability for each measure was adequate (CES-D: mother $\alpha = 0.80$, father $\alpha = 0.83$; STAI: mother $\alpha = 0.77$, father $\alpha = 0.73$; GHQ: mother $\alpha = 0.74$, father $\alpha = 0.80$). Results from a confirmatory factor analysis without rotation fit the data well ($\chi^2(10) = 19.44$,

$p = 0.0351$; RMSEA (Root Mean Square Error of Approximation) = 0.045 [0.012, 0.074] $p = 0.571$, CFI (Comparative Fit Index) = 0.989, SRMR (Standardized Root Mean Square Residual) = 0.028), and each indicator loaded significantly in the expected direction (CES-D: mother $\beta = 0.93$, father $\beta = 0.94$; STAI: mother $\beta = 0.56$, father $\beta = 0.60$; GHQ: mother $\beta = 0.68$, father $\beta = 0.71$; all $ps < 0.001$).

2.2.4 | Covariates

We included several demographic and socioeconomic variables in our analysis to control for potentially confounding factors. These included the mother’s and father’s highest level of education, household income, child and parent age, employment status, race/ethnicity, gestational age at the prenatal time point, gestational age at birth, infant birth weight, data collection site, and child sex. Additionally, we included several other covariates (described below) that could confound relations between cortisol, stress, linkage, and executive functions.

At T1, parents completed the Conflict Tactics Scale (CTS; Straus, 1979) and the Couple Satisfaction Index (CSI; Funk & Rogge, 2007). Internal reliability for each measure was adequate (CTS: mother $\alpha = 0.66$, father $\alpha = 0.65$; CSI: mother $\alpha = 0.96$, father $\alpha = 0.94$). Similar to the psychological stress variables, we calculated latent factors scores with both of these measures to create variables of maternal and paternal of relationship quality. We used confirmatory factor analysis without rotation. Based on modification indices, we correlated mother’s and father’s CTS scores, which resulted in a fully saturated model ($\chi^2(0) = 0.00$, RMSEA = 0.00, CFI = 1.00, SRMR = 0.00). Each indicator loaded significantly in the expected direction (CTS: mother $\beta = 0.45$, father $\beta = 0.37$; CSI: mother $\beta = 0.93$, father $\beta = 0.98$; all $ps < 0.001$).

At T1, parents also completed the Multidimensional Scale of Perceived Social Support (Zimet et al., 1988). This measure demonstrated adequate internal reliability (mother $\alpha = 0.75$, father $\alpha = 0.72$). Summed scores of this scale were calculated for both mothers and fathers and used as indicators of mother and father social support.

At 14 months (T2), parents completed the Who Does What questionnaire (Cowan & Cowan, 1990), which measures parents’ perceptions of their contribution to the household, family, and child-related tasks. This measure demonstrated adequate internal reliability (mother $\alpha = 0.94$, father $\alpha = 0.92$). As both mother’s and father’s scores for this scale were highly correlated, we averaged these together and included this combined score as an indicator of postnatal parental involvement.

At T2, both parents also completed the Brief Infant-Toddler Social and Emotional Assessment (BITSEA; Briggs-Gowan et al., 2004). We used the problems subscale of the BITSEA, which comprised 31 items assessing a range of social-emotional problems, internalizing behaviors, externalizing behaviors, and behavioral dysregulation. The measure demonstrated adequate internal reliability (mother $\alpha = 0.78$, father $\alpha = 0.67$). We averaged together mother’s and father’s scores on the problems subscale to use as a measure of infant social-emotional behavior.

Mothers and fathers also participated separately in semi-structured free play interactions with their infants at the T2 home assessment. Each parent was given a standardized set of toys (different for each parent) and asked to play with their child as they normally would for 5 min. The interactions were video-recorded and globally coded for parental sensitivity using an adapted version of Ainsworth's Observation Scales for Sensitivity versus Insensitivity and Cooperation versus Interference (Ainsworth, Bell, & Stayton, 1974).

2.3 | Analytic strategy

2.3.1 | Executive functions

As previously described (Devine et al., 2019), a latent variable framework was used to obtain empirical Bayes estimates for each of the three executive function tasks. Briefly, we used confirmatory factor analysis with a Bayesian estimator to simultaneously model performance on the three executive function tasks for each child. For each respective task, performance on each trial was used as latent factor indicators. Results indicated that the model fit the data adequately based on the Bayesian posterior predictive $p = 0.404$ (95% Confidence Interval (CI) = $-103.69, 142.83$; Muthén & Asparouhov, 2012). Each indicator loaded significantly in the expected direction and all p s < 0.001. We extracted the empirical Bayes estimates for performance on each task for each child to use in our main analyses. More specifically, in the main models, we modeled executive function as a single latent factor with the three empirical Bayes estimates as factor indicators. To control for any potential bias due to missing data, we used multiple imputations with 20 datasets.¹

2.3.2 | Cortisol indices

Following previous recommendations (Gunnar & Talge, 2007; Khoury et al., 2015), we used the raw, untransformed cortisol values to calculate the area under the curve with respect to ground (AUCg) for each day. Following the calculation of each cortisol index, we averaged the indices across both days. AUCg was calculated for each day using the trapezoid formula (Pruessner et al., 2003). This index reflects total cortisol output across the day (Fekedulegn et al., 2007).

2.3.3 | Linkage

To estimate mother–father cortisol linkage, we used a similar approach as previously done with these data (Braren et al., 2020). Specifically, we used a two-level mixed (i.e., multilevel) model with random intercepts in which cortisol samples were nested within persons. At level 1,

for each dyad, mother's six cortisol samples were regressed on father's six cortisol samples. Thus, for each dyad, linkage was operationalized as the linear association between mother's cortisol at one time point with father's cortisol at the same time point. We used a conservative approach by excluding morning samples that were not collected either within 15 min of waking (for sample 1) and/or within 15–45 min of waking (for sample 2). Additionally, prior to analysis, for each person, cortisol was time-detrended by regressing cortisol on person-mean centered time. Last, several other cortisol covariates (described above) were examined in relation to mother's and father's cortisol.

From this mixed model, we extracted empirical Bayes estimates of the level 1 slopes for each couple, which we used as linkage coefficients for subsequent analyses. To facilitate the interpretation of coefficients, cortisol samples were centered on each person's cortisol mean (person-mean centered). Thus, the level 1 slope coefficient for each dyad reflected the within-dyad association between each partner's cortisol relative to each person's cortisol mean. Thus, for example, a positive linkage coefficient indicates that when the father's cortisol is higher (or lower) than his average, the mother's cortisol is also higher (or lower) than her average. Conceptually, this approach specifies linkage as a concurrent process of mutual linkage and, as such, does not assume directionality in who is influencing whom. In short, these linkage coefficients represent the magnitude of the linear association between mother and father cortisol for each dyad. That is, larger values indicate stronger within-couple linkage, and smaller values indicate weaker linkage. This approach is similar to that of other studies investigating interpersonal linkage or synchrony (Kalomiris & Kiel, 2018; Saxbe & Repetti, 2010; Saxbe et al., 2017; Thorson, West, & Mendes, 2018). To control for any potential bias due to missing data, we used multiple imputations with 20 datasets.²

2.3.4 | Main analyses

We used structural equation modeling to address our primary aims. Specifically, to address our research questions we ran two models. In the first model, we tested the direct effects of cortisol linkage, maternal and paternal cortisol, and maternal and paternal psychological stress in their relation to infant executive functions while controlling for all of our covariates. In the second model, we added four interactions between linkage and average maternal cortisol, average paternal cortisol, maternal psychological stress, and paternal psychological stress. To interpret interactions, we evaluated simple slopes at high (+SD) and low levels (–SD) of each variable, one SD above and one SD below the mean (Aiken & West, 1991). We conducted sensitivity analyses to ensure the robustness of our results when including multiple interactions, which entailed running the secondary models with all four interactions simultaneously and with each interaction separately. Further, to correct for any potential biases due to multiple comparisons in testing these four interactions, we applied the Benjamini–Hochberg procedure to adjust p -values (Benjamini & Hochberg, 1995). Results

¹ We chose to use empirical Bayes estimates given that Bayesian estimation has been shown to yield more precise and reliable factor score estimates than traditional frequentist-derived estimates for ordinal/count variables and multilevel models (Asparouhov & Muthén, 2010; Raudenbush & Bryk, 2002). As an additional sensitivity analysis, we also estimated factor scores using full information maximum likelihood (FIML) estimation. Results from the FIML analysis were nearly identical to results using Bayesian estimation with multiple imputation.

² See footnote 1.

of sensitivity analyses and adjusting for multiple comparisons did not significantly alter the results.

Families were included in these analyses if they had both a cortisol linkage score and completed all three executive function tasks, yielding a sample size of $n = 358$. All models were estimated in Mplus using maximum likelihood with robust standard errors. To control for any potential bias due to missing data, we also used FIML.³ All variables were visually and statistically evaluated to ensure normality and to check for outliers. For the main analyses, all variables were grand-mean centered. All model results were examined to ensure residuals were normally distributed and that there were no points of significant leverage or influence. There were no significant points of leverage or influence.

2.3.5 | Missing data

Of the 484 families initially recruited, 445 were visited at T1 (36 weeks prenatal). At T1, 23 families withdrew from the study or declined participation, six were unable to participate in data collection due to scheduling issues, and 10 became ineligible due to changes in status regarding inclusion criteria. At T2 (14 months), 422 families were seen. At T2, 13 families became ineligible after T1 due to moving out of the country, six families declined participation, and six families who missed appointments at T1 returned. Last, at T3 (24 months), 404 families participated. At T3, 12 were ineligible due to changes in status regarding inclusion criteria, 16 declined to participate, and 10 who missed appointments at T2 returned.

Of the 445 families visited at T1, 384 (86%) had at least one saliva sample collected according to the protocol guidelines (outlined above) on either day and were thus included in the analyses. Of the 384 included in the analysis, 28% of mothers had all six saliva samples, 56% had at least five samples, 80% had at least four samples, and 93% had at least three samples. Regarding fathers, 31% had all six saliva samples, 56% had at least five samples, 81% had at least four samples, and 92% had at least three samples.

Regarding missingness on the executive functions tasks, of the 404 children who participated in the T3 visit, 387 (95.8%) completed the multilocation search task, 391 (96.8%) completed the ball run task, and 363 (89.9%) completed the baby Stroop task. Additionally, 364 (90.1%) children completed at least one task, 289 (71.5%) completed at least two tasks, and 153 (37.9%) completed all three tasks.

3 | RESULTS

3.1 | Descriptives

Tables 1 and 2 show descriptive statistics and correlations for the primary variables used in the current analysis. As reported at the prenatal

time point, parents were predominantly white (mothers: 90%, fathers: 92%), had obtained at least an undergraduate degree (mothers: 86%, fathers: 79%), and were working full time (mothers: 78%, fathers: 93%). Couples' annual household income was on average more than twice (mean = 2.7, median = 2.4, $SD = 1.25$) the median income of each couple's respective smallest geographical area for which data were available (i.e., New York state, Cambridgeshire, and the Netherlands). On average across the sample, there was significant positive within-dyad linkage between mother and father cortisol ($b = 0.12$, $\beta = 0.20$, $p < 0.001$).

Regarding our main variables of interest, cortisol linkage was negatively correlated with maternal education and positively correlated with paternal sensitivity at 14 months as well as prenatal maternal cortisol output (AUCg). Maternal cortisol was also positively associated with maternal psychological stress. Paternal cortisol was not associated with any of the main analysis variables. Father's and mother's psychological stress levels were positively correlated. Paternal stress was also positively related to both mother's and father's education levels.

3.2 | Main analyses: Direct effects of linkage, stress, and cortisol

To address our main research questions, we ran two models predicting the latent factor representing infants' executive function. In the first model, we addressed our first hypothesis by testing the main effects of cortisol linkage, maternal and paternal cortisol, and maternal and paternal psychological stress. This model fit the data well, $\chi^2(52) = 59.76$, $p = 0.21$, CFI = 0.97, RMSEA = 0.02 [0.00, 0.04] $p = 0.99$, SRMR = 0.02. As shown in Table 3 in the model 1 column, there was a significant positive association between cortisol linkage and infant executive functions, indicating that stronger prenatal cortisol linkage was associated with better performance on the executive function tasks at 24 months. Neither maternal nor paternal cortisol output (AUCg) was related to executive functions. Similarly, neither maternal nor paternal psychological stress was associated with executive functions.

3.3 | Main analyses: Interactions between linkage, stress, and cortisol

To test our second hypothesis that prenatal maternal and paternal cortisol (AUCg) and psychological stress would moderate the association between cortisol linkage and executive function, we tested interactions between each of these four variables and linkage. This model fit the data well, $\chi^2(60) = 62.46$, $p = 0.39$, CFI = 0.99, RMSEA = 0.01 [0.00, 0.03] $p = 0.99$, SRMR = 0.02. The results for this model are shown in the model 2 column in Table 3. Neither maternal nor paternal psychological stress moderated the relation between linkage and infant executive function. However, paternal cortisol, but not maternal cortisol, did moderate the relation between linkage and executive functions.

As shown in Figure 1, the linkage was positively associated with infant executive functions but only when father cortisol AUCg was high

³ As an additional sensitivity analysis, we also ran the main analyses using Bayesian estimation and multiple imputation. The pattern of results from these models was nearly identical to the results using FIML estimation. We note that although there is generally not agreement on whether FIML or multiple imputation is better at handling missing data, we agree with Allison (2012) that FIML has some advantages over multiple imputation, and thus we present results from the models using FIML.

TABLE 2 Bivariate correlations

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 |
|---------------------------------------|---------|--------|--------|---------|---------|---------|--------|--------|-------|-------|--------|--------|--------|-------|-------|-------|--------|
| 1. Mother education T1 | - | | | | | | | | | | | | | | | | |
| 2. Father education T1 | 0.39** | - | | | | | | | | | | | | | | | |
| 3. Household income T1 | 0.24** | 0.28** | - | | | | | | | | | | | | | | |
| 4. Father psychological stress T1 | 0.13* | 0.15** | 0.06 | - | | | | | | | | | | | | | |
| 5. Mother psychological stress T1 | -0.08 | -0.03 | -0.06 | 0.18** | - | | | | | | | | | | | | |
| 6. Father social support T1 | 0.060 | 0.083 | -0.043 | -0.134* | -0.074 | - | | | | | | | | | | | |
| 7. Mother social support T1 | 0.076 | 0.039 | 0.036 | -0.015 | -0.131* | 0.197** | - | | | | | | | | | | |
| 8. Father relationship quality T1 | -0.02 | -0.01 | -0.03 | -0.23** | -0.14* | 0.28** | 0.08 | - | | | | | | | | | |
| 9. Mother relationship quality T1 | 0.03 | 0.05 | 0.00 | -0.11* | -0.24** | 0.16** | 0.23** | 0.55** | - | | | | | | | | |
| 10. Parental involvement T2 | 0.12* | 0.10 | 0.00 | -0.04 | 0.03 | -0.01 | -0.05 | 0.01 | 0.14* | - | | | | | | | |
| 11. Maternal sensitivity T2 | 0.02 | -0.01 | 0.01 | -0.04 | -0.03 | -0.08 | 0.04 | 0.05 | 0.06 | 0.05 | - | | | | | | |
| 12. Paternal sensitivity T2 | 0.10 | 0.14* | -0.11 | -0.11 | -0.06 | 0.10 | -0.05 | -0.01 | 0.01 | 0.12* | 0.15** | - | | | | | |
| 13. Multilocation search score T3 | 0.03 | 0.03 | 0.05 | 0.00 | 0.04 | 0.02 | -0.12* | 0.07 | 0.02 | 0.03 | 0.10 | 0.08 | - | | | | |
| 14. Ball run score T3 | 0.07 | 0.09 | 0.04 | 0.09 | 0.05 | 0.01 | 0.06 | -0.08 | -0.04 | -0.02 | 0.02 | -0.01 | 0.24** | - | | | |
| 15. Baby Stroop score T3 | 0.06 | 0.02 | 0.04 | 0.03 | 0.04 | 0.06 | -0.04 | 0.00 | 0.04 | 0.03 | 0.07 | 0.18** | 0.31** | 0.08 | - | | |
| 16. Father cortisol T1 (AUCg) | -0.09 | -0.05 | -0.10 | 0.03 | 0.08 | 0.02 | -0.01 | 0.05 | 0.03 | 0.02 | 0.07 | 0.05 | -0.05 | -0.09 | 0.04 | - | |
| 17. Mother cortisol T1 (AUCg) | -0.08 | -0.02 | -0.08 | 0.09 | 0.15** | -0.10 | 0.04 | 0.01 | -0.03 | 0.04 | 0.09 | 0.02 | 0.03 | 0.01 | -0.04 | 0.15* | - |
| 18. Mother-father cortisol linkage T1 | -0.18** | -0.04 | -0.05 | -0.06 | 0.03 | 0.02 | -0.05 | 0.04 | 0.08 | -0.03 | -0.01 | 0.11* | 0.06 | -0.03 | 0.09 | 0.04 | 0.15** |

Note: Household income was computed as couples' average income proportional to the median income of the smallest area for which data were available (i.e., New York State, Cambridgeshire, and the Netherlands); T1: prenatal time point; T2: 14-month time point; T3: 24-month time point; AUCg: area under the curve with respect to ground.

* $p < 0.05$; ** $p < 0.01$.

TABLE 3 Results from models predicting infant executive functions at 24 months

| | Model 1 | | | Model 2 | | |
|----------------------------------|-------------|-------------|-----------------|--------------|--------------|-----------------|
| | β | <i>b</i> | <i>p</i> | β | <i>b</i> | <i>p</i> |
| Child sex (0 = male) | 0.19 | 0.09 | 0.01 | 0.18 | 0.09 | 0.01 |
| Child age T3 | 0.24 | 0.08 | <0.01 | 0.24 | 0.07 | <0.01 |
| Mother education T1 | 0.03 | 0.00 | 0.78 | 0.03 | 0.00 | 0.79 |
| Father education T1 | -0.03 | 0.00 | 0.90 | -0.03 | 0.00 | 0.98 |
| Household income T1 | 0.18 | 0.10 | 0.03 | 0.19 | 0.11 | 0.03 |
| Parental involvement T2 | 0.07 | 0.02 | 0.29 | 0.06 | 0.01 | 0.53 |
| Mother psychological stress T1 | 0.11 | 0.05 | 0.17 | 0.10 | 0.05 | 0.13 |
| Father psychological stress T1 | 0.05 | 0.03 | 0.50 | 0.06 | 0.03 | 0.45 |
| Mother relationship quality T1 | 0.06 | 0.01 | 0.89 | 0.04 | 0.01 | 0.91 |
| Father relationship quality T1 | 0.01 | 0.02 | 0.49 | 0.01 | 0.01 | 0.39 |
| Mother social support T1 | -0.04 | 0.01 | 0.83 | -0.03 | -0.01 | 0.93 |
| Father social support T1 | 0.02 | 0.01 | 0.51 | 0.02 | 0.01 | 0.59 |
| Maternal sensitivity T2 | 0.10 | 0.02 | 0.14 | 0.09 | 0.01 | 0.25 |
| Paternal sensitivity T2 | 0.14 | 0.02 | 0.12 | 0.13 | 0.02 | 0.10 |
| Maternal BITSEA T2 | 0.08 | 0.01 | 0.32 | 0.07 | 0.01 | 0.38 |
| Paternal BITSEA T2 | -0.13 | -0.03 | 0.11 | -0.13 | -0.03 | 0.11 |
| Mother cortisol T1 (AUCg) | -0.01 | -0.01 | 0.84 | -0.08 | -0.02 | 0.22 |
| Father cortisol T1 (AUCg) | -0.04 | -0.02 | 0.61 | -0.26 | -0.08 | 0.05 |
| Cortisol linkage T1 | 0.14 | 0.42 | 0.03 | 0.14 | 0.45 | 0.04 |
| Linkage × mother stress | - | - | - | -0.11 | -0.36 | 0.45 |
| Linkage × father stress | - | - | - | 0.05 | 0.16 | 0.76 |
| Linkage × mother cortisol (AUCg) | - | - | - | 0.02 | 0.09 | 0.70 |
| Linkage × father cortisol (AUCg) | - | - | - | 0.28 | 0.56 | 0.01 |
| <i>R</i> ² | 0.18 | - | 0.01 | 0.23 | - | <0.01 |

Note: Bold values are $p \leq 0.05$; T1: prenatal time point; T2: 14-month time point; T3: 24-month time point; BITSEA: Brief Infant-Toddler Social and Emotional Assessment; AUCg: area under the curve with respect to ground.

(relative to other fathers). Specifically, low cortisol linkage was associated with lower infant executive functions but only for children whose fathers had higher cortisol AUCg. Conversely, high cortisol linkage was related to higher executive functions in the presence of higher father cortisol. At low father cortisol, the linkage was not associated with executive functions. This interaction remained significant even after adjusting for multiple comparisons.

4 | DISCUSSION

Fathers play an important role during pregnancy by regulating maternal emotions, stress, and well-being, which can impact the fetal environment (Brumberg & Shah, 2020; Hughes et al., 2020; Plantin et al., 2011; Saxbe et al., 2018). Yet most research on prenatal predictors of infant developmental outcomes has focused solely on the role of the mother. In particular, regulating cortisol activity during pregnancy is important for facilitating healthy fetal brain development, which may impact postnatal self-regulation and executive functions abilities

via fetal programming (Glynn et al., 2018; Monk et al., 2019; Seckl & Meaney, 2004; Talge et al., 2007). Cortisol linkage between expectant parents may be an important process of social co-regulation (Butler & Randall, 2013; Field, 2012; Timmons et al., 2015). However, the extent to which parent cortisol linkage may influence child developmental outcomes is not known. As a first step in investigating the developmental consequences of parental physiological linkage, we evaluated whether mother–father prenatal cortisol linkage longitudinally predicted infant executive functioning at 24 months of age.

Conceptually, physiological linkage describes the concurrent covariation (similarity or difference) of physiological activity between two people. Technically, in our analysis, we operationalized linkage as the correlation between a mother's and father's cortisol levels at the same point in time. Thus, larger, positive linkage coefficients indicate that a couple's diurnal cortisol activity is more strongly linked or synchronous. More strongly linked cortisol profiles theoretically suggest a couple's ability to mutually influence and/or flexibly adapt to each partner's stress physiology for better or for worse (Butler & Randall, 2013; Field, 2012; Timmons et al., 2015).

Cortisol Linkage & Father Cortisol Predict Infant Executive Functions

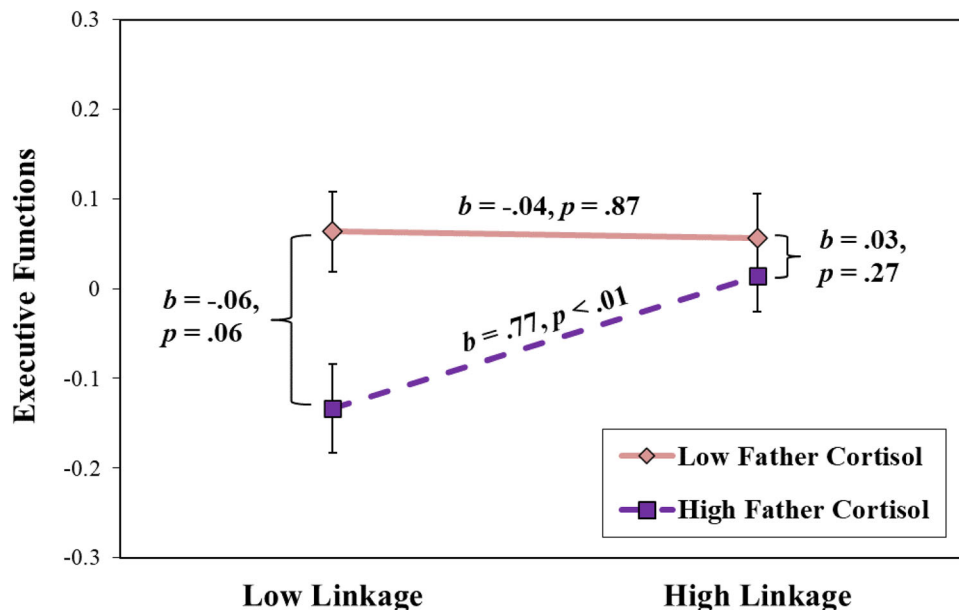


FIGURE 1 Mother–father prenatal cortisol linkage interacted with father cortisol (area under the curve with respect to ground) in predicting infant executive function performance at 24 months. Low cortisol linkage was associated with lower executive functions but only among children whose fathers had higher cortisol (relative to other fathers). Specifically, high cortisol linkage was related to higher executive functions in the presence of high father cortisol ($\beta = 0.29$, 95% CI [0.11, 0.46]). Conversely, at low father cortisol, linkage was not associated with executive functions ($\beta = -0.02$, 95% CI [-0.20, 0.17]). At low linkage, father cortisol was marginally associated with executive functions ($\beta = -0.21$, 95% CI [-0.42, -0.01]). Conversely, at high linkage, father cortisol was not associated with executive functions ($\beta = 0.09$, 95% CI [-0.08, 0.27]). Note: Error bars represent standard errors; *b*-values represent unstandardized coefficients.

4.1 | Cortisol linkage predicts infant executive functions

In support of our first hypothesis, we found that greater mother–father diurnal cortisol linkage was positively associated with higher infant executive function scores. Specifically, a stronger linear association between mother and father diurnal cortisol activity was associated with better infant performance on executive function tasks at 24 months of age. Conversely, weaker prenatal cortisol linkage was related to lower infant executive function scores. This result suggests that higher prenatal parent cortisol linkage may support the development of infant executive function, whereas lower linkage may compromise infant executive function development.

No studies that we are aware of have explicitly investigated physiological linkage in parents during pregnancy in relation to infant outcomes. However, a handful of studies in humans and nonhuman primates have shown that expectant parents display correlated hormonal activity during pregnancy and suggest that this linkage may reflect a supportive process that facilitates the transition to parenthood (Berg & Wynne-Edwards, 2002; Edelman et al., 2015; Storey & Ziegler, 2016; Storey et al., 2000). In support of this idea, a related study found that a stronger correlation between prenatal mother and father testosterone was associated with greater postnatal father relationship investment, commitment, and satisfaction (Saxbe

et al., 2017). Relatedly, inasmuch as cortisol linkage reflects supportive relationship functioning, our finding is similar to other studies that have shown that increased prenatal partner support enhances infant self-regulation, including lower levels of infant emotional distress and reactivity (Stapleton et al., 2012), and decreased infant cortisol reactivity to a laboratory stressor (Thomas et al., 2018).

There are at two least potential developmental mechanisms mediating parent linkage with infant cognitive development. First, there may be a direct effect through fetal programming. Second, there may be an indirect effect of linkage through the postnatal caregiving environment. As to the first potential mechanism, research has shown that prenatal maternal cortisol activity may be a key mechanism in programming fetal development, with potent effects on the HPA axis, amygdala, hippocampus, and PFC—key regions that support executive functions (Bowers & Yehuda, 2016; Bridgett et al., 2015; Glynn et al., 2018). Stronger parent cortisol linkage may help to co-regulate and maintain healthy maternal and fetal cortisol activity, thereby supporting healthy brain development and executive functions. On the other hand, lower cortisol linkage may dysregulate maternal and fetal cortisol activity and negatively impact neurocognitive development.

Notably, we did not find a direct effect of diurnal maternal cortisol output on infant executive functions unlike some prior studies on prenatal programming and child outcomes (Glynn et al., 2018; Seckl & Meaney, 2004; Zijlmans et al., 2015). However, other prenatal

programming studies have found inconsistent associations between maternal cortisol and child outcomes, indicating that these associations are dependent on other factors, such as the timing of fetal cortisol exposure (Zijlmans et al., 2015). Maternal cortisol activity may have more or less programming potential at different stages of gestation. However, there is some disagreement as to when during gestation the effects of fetal programming may be more or less pronounced (Zijlmans et al., 2015). For instance, some studies have found that elevated cortisol earlier in gestation but not later was related to child neurocognitive development (e.g., Buss et al., 2012), whereas others have found the opposite pattern (e.g., E. P. Davis & Sandman, 2010; E. P. Davis et al., 2017). Unfortunately, we assessed cortisol activity at only one time point during pregnancy and thus cannot know whether maternal cortisol would be associated with executive functions at a different stage of gestation.

Alternatively, given that maternal cortisol output changes over the course of pregnancy to support fetal development (Glynn & Sandman, 2011; Saxbe et al., 2018), the pattern of how this change unfolds across pregnancy may be an important regulator of neurocognitive growth. For instance, one noteworthy study that measured maternal cortisol repeatedly over the course of pregnancy found that the trajectory of maternal cortisol levels across gestation was a stronger predictor of infant cognitive development at 15 months than any of the individual cortisol levels (E. P. Davis & Sandman, 2010). This suggests that there may not necessarily be a simple relation in which only too much or too little maternal cortisol at a single time point may impact fetal development. Rather, the pattern of cortisol activity across gestation may be more important for fetal programming than cortisol at individual time points. Thus, future studies should assess parent cortisol linkage across pregnancy and examine how it relates to fetal programming and infant development.

Additionally, as other studies have shown, the effects of fetal programming involve other neurophysiological mediators, such as the autonomic nervous system (Beijers et al., 2014). The HPA axis and autonomic nervous system both mediate and regulate the biological stress response and operate in a coordinated, interdependent fashion (Kaltsas et al., 2007; Ulrich-Lai & Herman, 2009). Thus, in our sample, it is possible that cortisol linkage may facilitate co-regulation of autonomic nervous system activity in couples. In turn, this regulation of autonomic nervous system activity may support healthy fetal neurocognitive development. During pregnancy, multiple independent and interactive aspects of stress physiology likely program fetal development (Rash et al., 2016). Thus, future studies should employ a multi-system approach to examine how interactions between the HPA axis and autonomic nervous system relate to physiological linkage.

A second potential mediating pathway connecting prenatal cortisol linkage and infant executive functions may be through the postnatal environment. To the extent that cortisol linkage is stable and adaptive, its advantages may carry over into the postnatal period and have indirect effects on the home environment and caregiving quality. For instance, parent cortisol linkage may support sensitive parent-infant interactions and a positive home environment. Indeed, others have shown that supportive relationships during the transition to par-

enthood are related to caregiving quality (Goldstein et al., 1996; Shin et al., 2006). In turn, higher caregiving quality during infancy may scaffold the development of infant executive functions (Blair & Ursache, 2011; Bridgett et al., 2015; Samdan et al., 2020). Thus, parents who are better at co-regulating each other physiologically may also better co-regulate their infant, thereby supporting the development of cognitive self-regulation.

It should be noted that we also included postnatal global observational measures of maternal and paternal sensitivity in our analysis—neither of which was associated with infant executive functions. However, this does not necessarily rule out the possibility that caregiving sensitivity impacts executive function development in our sample. This finding may be due to an inherent limitation of global measures of behavior in that they do not fully capture the full range of variation or subtle behaviors that can occur within a caregiver-child interaction. This idea is in line with Hofer's (1994) "hidden regulators" hypothesis, which emphasizes how caregiving factors that regulate infant functioning are often not easily observable or apparent and are thus "hidden." Indeed, a great deal of cues exchanged during face-to-face interactions occurs outside of immediate awareness (Bargh et al., 2012; Harrist & Waugh, 2002; Papoušek & Papoušek, 2002). Thus, as others suggest, micro-coding of behaviors may be an important method in elucidating hidden behaviors that influence child development (Beebe & Steele, 2013; M. Davis et al., 2018; Leclere et al., 2014). Furthermore, a limited range in the distribution of sensitivity scores does not seem to be a likely cause of these null results, as both measures showed substantial variability.

4.2 | Father cortisol moderates the association between cortisol linkage and infant executive functions

The second aim of our analysis was to assess whether linkage was moderated by mother's or father's total diurnal cortisol output and/or self-reported stress. In partial support of our hypothesis, we found that the association between cortisol linkage and infant executive functions was dependent on the father's diurnal cortisol. Mother-father cortisol linkage was related to lower infant executive function scores when fathers had higher diurnal cortisol output (AUCg; relative to other fathers) and lower linkage with their partner. Specifically, the combined effect of low linkage coupled with high father cortisol was associated with lower executive functions. Conversely, high father cortisol in the presence of high cortisol linkage was related to higher executive functions. This finding suggests that elevated paternal cortisol amplifies the negative relation between lower cortisol linkage and lower infant executive function scores. Alternatively, this moderation suggests that higher cortisol linkage may buffer or normalize the negative effects of higher father cortisol in relation to executive functions.

One interpretation of this relation is that couples with higher linkage may be better able to co-regulate each other's physiological stress and thus buffer the potentially damaging effects of elevated paternal cortisol. This relation also may be mediated by fetal programming. As

previously discussed regarding the main effect of linkage, the combination of low cortisol linkage and high father cortisol may result in dysregulated maternal cortisol or increased maternal physiological stress, which may impact fetal neurocognitive development. Relatedly, high cortisol linkage may protect against the negative effects of high paternal cortisol by maintaining healthy levels of physiological stress and maternal–fetal cortisol.

It is also notable that paternal cortisol, but not maternal cortisol, moderated the association between cortisol linkage and infant executive functions. Given that pregnancy is a sensitive period for the pregnant mother, mothers may generally be more likely attuned to a partner's stress cues (Glynn et al., 2018). Indeed, research has shown that during pregnancy, mothers are more responsive to emotion- and stress-related cues from others and themselves (de Carli et al., 2019; Pearson et al., 2009; Raz, 2014; Senese et al., 2018). In turn, a mother's enhanced sensitivity to her partner's stress cues may translate to dysregulation of her own stress. However, couples who display stronger physiological linkage may adaptively co-regulate each other's stress despite the mother's potentially heightened sensitivity to her partner's stress. Relatedly, this enhanced maternal sensitivity to stress cues may explain why the mother's reported stress was correlated with her total cortisol output, but the father's reported stress was not related to his total cortisol output. Thus, as our results suggest, mothers may be more physiologically attuned to their own psychological stress as well as their partner's physiological stress.

Alternatively, the association between higher father cortisol and infant executive functions may be partially mediated by postnatal caregiving quality. Persistently, high levels of prenatal paternal cortisol may continue into the postnatal period and negatively affect a father's responsiveness and sensitivity during interactions with his infant, thereby impacting the infant's executive function abilities. This interpretation is consistent with studies that have found evidence that perinatal paternal cortisol is related to postnatal caregiving quality (Bos et al., 2018; Kuo et al., 2018), and the hypothesis that hormonal changes in fathers during pregnancy prepare the father for the transition to parenthood and caregiving (Saltzman & Ziegler, 2014). However, stronger linkage with one's partner may buffer the negative effects of high cortisol on paternal caregiving interactions, thereby supporting infant executive functions. Thus, partners who are better able to co-regulate each other's stress physiology may be better able to co-regulate their infant and scaffold self-regulation, even when fathers have high levels of physiological stress.

4.3 | Strengths, limitations, and future directions

One of the strengths of our analysis was the inclusion of several psychosocial and behavioral variables. Our results were observed while controlling for other potential confounding factors, such as mother and father reports of stress, relationship quality, parental involvement, social support, infant social-emotional behavior, and observations of caregiving sensitivity. Although we cannot rule out the possibility that third variables at intervening time points might explain our findings, we

suggest that the inclusion of these covariates strengthens our results and supports the idea that cortisol linkage is a robust and unique interpersonal process that is important for infant development.

More generally, our results suggest that physiological markers do not necessarily function in congruence with self-reports of psychosocial functioning or global observations of behavior and may operate independently of these. Our results support the hypothesis that physiological linkage may operate as a “hidden” regulator of infant development (Hofer, 1994). Accordingly, physiological processes can provide a “window” into hidden, or subtle, developmental processes that are not easily captured by self-report measures or global scales of behavioral observation. In support of this, several other studies have shown evidence of dissociations between physiological and psychological measures of perinatal stress and psychosocial functioning in mothers (e.g., Graignic-Philippe et al., 2014; E. P. Davis & Sandman, 2010; Voegtline et al., 2013). Furthermore, similar to our results, others have found that prenatal maternal stress physiology is uniquely associated with infant outcomes, over and above either self-report measures of psychosocial stress and/or observational measures of caregiving (e.g., E. P. Davis & Sandman, 2010; Nazzari et al., 2020).

Despite the strengths of our analysis, there are several limitations that we should point out. First, we operationalized linkage as a concurrent, bi-directional process between mothers and fathers, which assumes that both partners are influencing each other. However, as our cortisol data are correlational, we cannot determine directionality in who is influencing whom. Future studies using cross-lagged analyses with repeated cortisol samples across multiple days could further explore directionality of mother–father linkage processes. Relatedly, although our data are longitudinal, they are also correlational, and as such, we cannot infer causality in the relations between mother–father cortisol linkage and infant executive functions. An important question that our analysis raises and that we cannot evaluate in our data is what is driving differences in linkage. That is, why do some couples display lower versus higher levels of cortisol linkage? Our moderation result suggests that the father's physiological self-regulation (total cortisol output of fathers) may play a causal role in linkage. However, our data do not permit us to assess whether father cortisol influences cortisol linkage or linkage influences father cortisol. Notably, we were not able to control for one particular factor likely to influence hormonal linkage—the amount of time partners spent together (Papp et al., 2013). However, we did include as covariates mother's and father's employment statuses, which may serve as a loose proxy measure of the amount of time parents spend together. Understanding the causes of parental linkage will be important for providing insight into targeted interventions for improving relationship functioning, especially for expectant parents. Follow-up analyses with the present sample and others are needed to investigate potential mediating mechanisms involved in mother–father linkage and its influence on infant executive function development.

Third, it is important to note that our sample was low-risk and comprised predominantly white and middle to high socioeconomic status families. Thus, the generalizability of our results is constrained to a narrow demographic. More research is needed to understand

how linkage functions in other populations, particularly non-white and higher-risk populations. Additionally, we only measured cortisol over two days during late pregnancy in mothers and fathers. However, cortisol levels change dramatically across pregnancy (Morsi et al., 2018). Thus, follow-up studies assessing parent cortisol linkage at various points during pregnancy will be important to understanding how prenatal linkage might impact fetal development. Relatedly, we did not have any postnatal measures of cortisol. Future studies could include cortisol linkage measures during the postnatal period to better differentiate the prenatal versus postnatal contributions of parent linkage to infant development.

Last, we did not consider the role of genetic factors, which likely play an important role in fetal programming and executive function development (Bridgett et al., 2015; Deater-Deckard, 2014). Thus, future research is needed to further examine the roles of genes and gene-by-environment interactions in these relations.

5 | CONCLUSION

The vast majority of human studies on perinatal factors predicting infant outcomes have not integrated physiological measures with psychosocial and behavioral ones (Yim et al., 2015). Furthermore, human perinatal studies that have included physiology have focused only on the mother and not the father. Integrating physiological measures of both mothers and fathers with psychosocial and behavioral measures will be critical in advancing our understanding of the transition to parenthood and how it impacts child development. We believe that, although it also leaves many questions unanswered, this study makes an important contribution by taking a first step toward understanding the role that parental physiological linkage plays in child development.

In sum, we found that prenatal mother–father cortisol linkage was, on average, positively associated with infant executive functions at 24 months. Additionally, this association was moderated by average levels of prenatal father cortisol such that higher father cortisol was associated with lower infant executive functions in the presence of lower cortisol linkage. Broadly, this suggests that paternal physiology plays a potentially important role in shaping the prenatal environment with consequences for infant self-regulation development. This has important implications for the prenatal programming hypothesis by expanding its scope beyond just mothers to also include fathers. Altogether, in line with conceptual principles of families as dynamic systems, our results emphasize the possibility that the interdependent and co-regulated functioning between mother and father stress physiology may be an important factor impacting child development (Butler, 2011; Byrd-Craven & Clauss, 2019; Cox & Paley, 1997; Sbarra & Hazan, 2008). In short, relational and interpersonal factors are fundamental organizers of human development (Field, 2012; Fogel, 1993; Sameroff, 2009; Vygotsky, 1978). With this principle in mind, continued research incorporating physiological, psychosocial, and behavioral measures of the mother, father, and child is needed to advance our understanding of the complex interpersonal dynamics at work in the mother–father

relationship, the transition to parenthood, and how these associations impact child development.

ACKNOWLEDGMENTS

This work was supported by grants from the ESRC (ES/L016648/1); NWO (464-13-141); and NSF (1429152). The roles of the first and fourth authors were also supported by the NSF Graduate Research Fellowship Program under Grant No. DGE1342536. Any opinions, findings, and conclusions, or recommendations expressed in this material are those of the author(s) and do not necessarily reflect the views of the National Science Foundation. No authors have any conflicts of interest with regard to the work presented in this manuscript.

CONFLICT OF INTEREST

No authors have any conflicts of interest with regard to the work presented in this manuscript.

DATA AVAILABILITY STATEMENT

Data are available from the UK Data Service repository: Hughes, C., Devine, R.T., Mesman, J., & Blair, C. (2018). The New Fathers and Mothers Study: Well-being, Parenting and Children's Self-Regulation. [Data Collection]. Colchester, Essex: UK Data Service, <https://doi.org/10.5255/UKDA-SN-853278>.

AUTHOR CONTRIBUTIONS

Stephen H. Braren wrote the manuscript and ran the analysis. Stephen H. Braren and Rosemarie E. Perry interpreted the results with help from Annie Brandes-Aitken, Andrew Ribner, Natalie Brito, and Clancy Blair. All authors critically reviewed the manuscript and provided feedback. Andrew Ribner, Clancy Blair, and the NewFAMS Team collected the data. Clancy Blair and the NewFAMS Team designed and executed the study.

ORCID

Stephen H. Braren  <https://orcid.org/0000-0003-0891-2648>

Natalie Brito  <https://orcid.org/0000-0003-1016-0094>

REFERENCES

- Abraham, E., & Feldman, R. (2018). The neurobiology of human allomaternal care; implications for fathering, coparenting, and children's social development. *Physiology & Behavior*, 193(Part A), 25–34. <https://doi.org/10.1016/j.physbeh.2017.12.034>
- Aiken, L. S., & West, S. G., 1991. *Multiple regression: Testing and interpreting interactions*. Sage Publications, Inc.
- Ainsworth, M. D. S., Bell, S. M., & Stayton, D. J. (1974). Infant-mother attachment and social development: Socialization as a product of reciprocal responsiveness to signals. In M. P. M. Richards (Ed.), *The integration of a child into a social world* (pp. 99–135). Cambridge University Press.
- Allison, P. D. (2012). Handling missing data by maximum likelihood. SAS Global Forum 2012, 22–25 April, Orlando, FL, USA.
- Almanza-Sepulveda, M. L., Fleming, A. S., & Jonas, W. (2020). Mothering revisited: A role for cortisol? *Hormones and Behavior*, 121, 104679. <https://doi.org/10.1016/j.yhbeh.2020.104679>
- Arnsten, A. F. (2009). Stress signaling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, 10(6), 410–422. <https://doi.org/10.1038/nrn2648>

- Arnsten, A. F., & Li, B. M. (2005). Neurobiology of executive functions: Catecholamine influences on pre-frontal cortical functions. *Biological Psychiatry*, 57(11), 1377–1384. <https://doi.org/10.1016/j.biopsych.2004.08.019>
- Asparouhov, T., & Muthén, B. (2010). *Plausible values for latent variables using Mplus*. <https://www.statmodel.com/download/Plausible.pdf>
- Bargh, J. A., Schwader, K. L., Hailey, S. E., Dyer, R. L., & Boothby, E. J. (2012). Automaticity in social-cognitive processes. *Trends in Cognitive Sciences*, 16(12), 593–605. <https://doi.org/10.1016/j.tics.2012.10.002>
- Barrett, J., & Fleming, A. S. (2011). Annual Research Review: All mothers are not created equal: neural and psychobiological perspectives on mothering and the importance of individual differences. *Journal of Child Psychology and Psychiatry*, 52(4), 368–397. <https://doi.org/10.1111/j.1469-7610.2010.02306.x>
- Beckes, L., & Coan, J. A. (2011). Social baseline theory: The role of social proximity in emotion and economy of action. *Social and Personality Psychology Compass* 5(12), 976–988. <https://doi.org/10.1111/j.1751-9004.2011.00400.x>
- Beebe, B., & Steele, M. (2013). How does microanalysis of mother-infant communication inform maternal sensitivity and infant attachment? *Attachment & Human Development*, 15(5–6), 583–602. <https://doi.org/10.1080/14616734.2013.841050>
- Beijers, R., Buitelaar, J. K., & de Weerth, C. (2014). Mechanisms underlying the effects of prenatal psychosocial stress on child outcomes: beyond the HPA axis. *European Child & Adolescent Psychiatry*, 23(10), 943–956. <https://doi.org/10.1007/s00787-014-0566-3>
- Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate: A practical and powerful approach to multiple testing. *Journal of the Royal Statistical Society. Series B (Methodological)*, 57(1), 289–300. <http://www.jstor.org/stable/2346101>
- Berg, S. J., & Wynne-Edwards, K. E. (2002). Salivary hormone concentrations in mothers and fathers becoming parents are not correlated. *Hormones and Behavior*, 42(4), 424–436. <https://doi.org/10.1006/hbeh.2002.1841>
- Blair, C., & Raver, C. C. (2015). School readiness and self-regulation: A developmental psychobiological approach. *Annual Review of Psychology*, 66, 711–731. <https://doi.org/10.1146/annurev-psych-010814-015221>
- Blair, C., & Ursache, A. (2011). A bidirectional model of executive functions and self-regulation. In K. D. Vohs & Baumeister, R. F. (Eds.), *Handbook of self-regulation: Research, theory, and applications*, (2nd ed., pp. 300–320). Guilford Press.
- Bobin-Bègue, A. (2019). Rhythms in early development. In G. Apter, E. Devouche & M. Gratièr. (Eds.), *Early interaction and developmental psychopathology* (pp. 55–86). Springer. https://doi.org/10.1007/978-3-030-04769-6_4
- Bos, P. A. (2017). The endocrinology of human caregiving and its intergenerational transmission. *Development and Psychopathology*, 29(3), 971–999. <https://doi.org/10.1017/S0954579416000973>
- Bos, P. A., Hechler, C., Beijers, R., Shinohara, K., Esposito, G., & de Weerth, C. (2018). Prenatal and postnatal cortisol and testosterone are related to parental caregiving quality in fathers, but not in mothers. *Psychoneuroendocrinology*, 97, 94–103. <https://doi.org/10.1016/j.psyneuen.2018.07.013>
- Bowers, M. E., & Yehuda, R. (2016). Intergenerational transmission of stress in humans. *Neuropsychopharmacology*, 41(1), 232–244. <https://doi.org/10.1038/npp.2015.247>
- Braren, S. H., Brandes-Aitken, A., Ribner, A., Perry, R. E., & Blair, C. (2020). Maternal psychological stress moderates diurnal cortisol linkage in expectant fathers and mothers during late pregnancy. *Psychoneuroendocrinology*, 111, 104474. <https://doi.org/10.1016/j.psyneuen.2019.104474>
- Bridgett, D. J., Burt, N. M., Edwards, E. S., & Deater-Deckard, K. (2015). Intergenerational transmission of self-regulation: A multidisciplinary review and integrative conceptual framework. *Psychological Bulletin*, 141(3), 602–654. <https://doi.org/10.1037/a0038662>
- Briggs-Gowan, M. J., Carter, A. S., Irwin, J. R., Wachtel, K., & Cicchetti, D. V. (2004). The brief infant-toddler social and emotional assessment: Screening for social-emotional problems and delays in competence. *Journal of Pediatric Psychology*, 29(2), 143–155. <https://doi.org/10.1093/jpepsy/jsh017>
- Brumberg, H. L., & Shah, S. I. (2020). Got daddy issues? Fathers impact on perinatal outcomes. *Seminars in Perinatology*, 44(4), 151238. <https://doi.org/10.1016/j.semperi.2020.151238>
- Brunton, P. J., & Russell, J. A. (2011). Neuroendocrine control of maternal stress responses and fetal programming by stress in pregnancy. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 35(5), 1178–1191. <http://doi.org/10.1016/j.pnpbp.2010.12.023>
- Busada, J. T., & Cidlowski, J. A. (2017). Mechanisms of glucocorticoid action during development. *Current Topics in Developmental Biology*, 125, 147–170. <https://doi.org/10.1016/bs.ctdb.2016.12.004>
- Buss, C., Davis, E. P., Hobel, C. J., & Sandman, C. A. (2011). Maternal pregnancy-specific anxiety is associated with child executive function at 6–9 years age. *Stress (Amsterdam, the Netherlands)*, 14(6), 665–676. <https://doi.org/10.3109/10253890.2011.623250>
- Buss, C., Davis, E. P., Shahbaba, B., Pruessner, J. C., Head, K., & Sandman, C. A. (2012). Maternal cortisol over the course of pregnancy and subsequent child amygdala and hippocampus volumes and affective problems. *Proceedings of the National Academy of Sciences of the United States of America*, 109(20), E1312–E1319. <https://doi.org/10.1073/pnas.1201295109>
- Butler, E. A. (2011). Temporal Interpersonal emotion systems: The “TIES” that form relationships. *Personality and Social Psychology Review*, 15(4), 367–393. <https://doi.org/10.1177/1088868311411164>
- Butler, E. A., & Randall, A. K. (2013). Emotional coregulation in close relationships. *Emotion Review*, 5(2), 202–210. <https://doi.org/10.1177/1754073912451630>
- Byrd-Craven, J., & Claus, N. (2019). The psychobiology of family dynamics: Bidirectional relationships with adrenocortical attunement. In A. W. Harrist & B. C. Gardner (Eds.), *Biobehavioral markers in risk and resilience research* (pp. 13–30). https://doi.org/10.1007/978-3-030-05952-1_2
- Calkins, S. D., Perry, N. B., & Dollar, J. M. (2016). A biopsychosocial model of self-regulation in infancy. In L. Balter & C. S. Tamis-LeMonda (Eds.), *Child psychology: A handbook of contemporary issues* (3rd ed., pp. 3–20). Psychology Press. <https://doi.org/10.4324/9781315764931>
- Camerota, M., & Willoughby, M. T. (2020). Prenatal risk predicts preschooler executive function: A cascade model. *Child Development*, 91(3), e682–e700. <https://doi.org/10.1111/cdev.13271>
- Cowan, P., & Cowan, C., 1990. Who does what? In J. Touliatos, B. F. Perlmutter & M. Straus (Eds.), *Handbook of family measurement techniques*. (pp. 447–448). Sage.
- Cox, M. J., & Paley, B. (1997). Families as Systems. *Annual Review of Psychology*, 48(1), 243–267. <https://doi.org/10.1146/annurev.psych.48.1.243>
- Davis, E. P., Glynn, L. M., Waffarn, F., & Sandman, C. A. (2011). Prenatal maternal stress programs infant stress regulation. *Journal of Child Psychology and Psychiatry*, 52(2), 119–129. <https://doi.org/10.1111/j.1469-7610.2010.02314.x>
- Davis, E. P., Head, K., Buss, C., & Sandman, C. A. (2017). Prenatal maternal cortisol concentrations predict neurodevelopment in middle childhood. *Psychoneuroendocrinology*, 75, 56–63. <https://doi.org/10.1016/j.psyneuen.2016.10.005>
- Davis, E. P., & Sandman, C. A. (2010). The timing of prenatal exposure to maternal cortisol and psychosocial stress is associated with human infant cognitive development. *Child Development*, 81(1), 131–148. <https://doi.org/10.1111/j.1467-8624.2009.01385.x>
- 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. <https://doi.org/10.1002/dev.21743>
- De Carli, P., Costantini, I., Sessa, P., Visentin, S., Pearson, R. M., & Simonelli, A. (2019). The expectant social mind: A systematic review of face processing during pregnancy and the effect of depression and anxiety.

- Neuroscience & Biobehavioral Reviews*, 102, 153–171. <https://doi.org/10.1016/j.neubiorev.2019.04.013>
- Deater-Deckard, K. (2014). Family matters: Intergenerational and inter-personal processes of executive function and attentive behavior. *Current Directions in Psychological Science*, 23(3), 230–236. <https://doi.org/10.1177/0963721414531597>
- Devine, R. T., Ribner, A., & Hughes, C. (2019). Measuring and predicting individual differences in executive functions at 14 months: A longitudinal study. *Child Development*, 90(5), e618–e636. <https://doi.org/10.1111/cdev.13217>
- Dietz, L. J., Jennings, K. D., Kelley, S. A., & Marshal, M. (2009). Maternal depression, paternal psychopathology, and toddlers' behavior problems. *Journal of Clinical Child & Adolescent Psychology*, 38(1), 48–61.
- Dipietro, J. A., Irizarry, R. A., Costigan, K. A., & Gurewitsch, E. D. (2004). The psychophysiology of the maternal-fetal relationship. *Psychophysiology*, 41(4), 510–520. <https://doi.org/10.1111/j.1469-8986.2004.00187.x>
- Edelstein, R. S., Wardecker, B. M., Chopik, W. J., Moors, A. C., Shipman, E. L., & Lin, N. J. (2015). Prenatal hormones in first-time expectant parents: Longitudinal changes and within-couple correlations. *American Journal of Human Biology*, 27(3), 317–325. <https://doi.org/10.1002/ajhb.22670>
- El Marroun, H., White, T. J., Fernandez, G., Jaddoe, V. W., Verhulst, F. C., Stricker, B. H., & Tiemeier, H. (2017). Prenatal exposure to selective serotonin reuptake inhibitors and non-verbal cognitive functioning in childhood. *Journal of Psychopharmacology*, 31(3), 346–355. <https://doi.org/10.1177/0269881116665335>
- Fekedulegn, D. B., Andrew, M. E., Burchfiel, C. M., Violanti, J. M., Hartley, T. A., Charles, L. E., & Miller, D. B. (2007). Area under the curve and other summary indicators of repeated waking cortisol measurements. *Psychosomatic Medicine*, 69(7), 651–659. <https://doi.org/10.1097/PSY.0b013e31814c405c>
- Feldman, R., Greenbaum, C. W., & Yirmiya, N. (1999). Mother–infant affect synchrony as an antecedent of the emergence of self-control. *Developmental Psychology*, 35(1), 223–231. <https://doi.org/10.1037/0012-1649.35.1.223>
- Feldman, R. (2009). The development of regulatory functions from birth to 5 Years: Insights from premature infants. *Child Development*, 80(2), 544–561. <https://doi.org/10.1111/j.1467-8624.2009.01278.x>
- Field, T. (2012). Relationships as regulators. *Psychology*, 3(6), 467–479.
- Finegood, E. D., & Blair, C. (2017). Poverty, parent stress, and emerging executive functions in young children. In K. Deater-Deckard & R. Panneton (Eds.), *Parental stress and early child development: Adaptive and maladaptive outcomes* (pp. 181–207). Springer. https://doi.org/10.1007/978-3-319-55376-4_8
- Fogel, A. (1993). *Developing through relationships*. University of Chicago Press.
- Funk, J. L., & Rogge, R. D. (2007). Testing the ruler with item response theory: Increasing precision of measurement for relationship satisfaction with the Couples Satisfaction Index. *Journal of Family Psychology*, 21(4), 572–583. <http://doi.org/10.1037/0893-3200.21.4.572>
- Gettler, L.T., 2014. Applying socioendocrinology to evolutionary models: Fatherhood and physiology. *Evolutionary Anthropology*, 23(4), 146–160. <https://doi.org/10.1002/evan.21412>
- Gitau, R., Cameron, A., Fisk, N. M., & Glover, V. (1998). Fetal exposure to maternal cortisol. *Lancet*, 352(9129), 707–708. [https://doi.org/10.1016/S0140-6736\(05\)60824-0](https://doi.org/10.1016/S0140-6736(05)60824-0)
- Glover, V., & Capron, L. (2017). Prenatal parenting. *Current Opinion in Psychology*, 15, 66–70. <https://doi.org/10.1016/j.copsyc.2017.02.007>
- Glynn, L. M., Howland, M. A., & Fox, M. (2018). Maternal programming: Application of a developmental psychopathology perspective. *Development Psychopathology*, 30(3), 905–919. <https://doi.org/10.1017/S0954579418000524>
- Glynn, L. M., & Sandman, C. A. (2011). Prenatal origins of neurological development. *Current Directions in Psychological Science*, 20(6), 384–389. <https://doi.org/10.1177/0963721411422056>
- Goldberg, D.P., Gater, R., Sartorius, N., Ustun, T.B., Piccinelli, M., Gureje, O., Rutter, C., 1997. The validity of two versions of the GHQ in the WHO study of mental illness in general health care. *Psychological Medicine*, 27(1), 191–197.
- Goldstein, L. H., Diener, M. L., & Mangelsdorf, S. C. (1996). Maternal characteristics and social support across the transition to motherhood: Associations with maternal behavior. *Journal of Family Psychology*, 10, 60–71. <https://doi.org/10.1037/0893-3200.10.1.60>
- Graignic-Philippe, R., Dayan, J., Chokron, S., Jacquet, A. Y., & Tordjman, S. (2014). Effects of prenatal stress on fetal and child development: A critical literature review. *Neuroscience and Biobehavioral Reviews*, 43, 137–162. <https://doi.org/10.1016/j.neubiorev.2014.03.022>
- Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*, 27(1–2), 199–220. [http://doi.org/10.1016/S0306-4530\(01\)00045-2](http://doi.org/10.1016/S0306-4530(01)00045-2)
- Gunnar, M. R., Talge, N. M., 2007. Neuroendocrine measures in developmental research. In L. A. Schmidt & S. J. Segalowitz (Eds.), *Developmental Psychophysiology: Theory, systems, and methods* (pp. 343–364). Cambridge University Press.
- Ha, T., Yeung, E. W., Rogers, A. A., Poulsen, F. O., Kornienko, O., & Granger, D. A. (2016). Supportive behaviors in adolescent romantic relationships moderate adrenocortical attenuation. *Psychoneuroendocrinology*, 74, 189–196. <https://doi.org/10.1016/j.psyneuen.2016.09.007>
- Hanington, L., Heron, J., Stein, A., & Ramchandani, P. (2012). Parental depression and child outcomes—is marital conflict the missing link?. *Child: care, health and development*, 38(4), 520–529. <https://doi.org/10.1111/j.1365-2214.2011.01270.x>
- Harrist, A. W., & Waugh, R. M. (2002). Dyadic synchrony: Its structure and function in children's development. *Developmental Review*, 22(4), 555–592. [http://doi.org/10.1016/S0273-2297\(02\)00500-2](http://doi.org/10.1016/S0273-2297(02)00500-2)
- Hofer, M. A. (1994). Hidden regulators in attachment, separation, and loss. *Monographs of the Society for Research in Child Development*, 59(2/3), 192–207. <https://doi.org/10.2307/1166146>
- Hughes, C., Devine, R. T., Foley, S., Ribner, A. D., Mesman, J., & Blair, C. (2020). Couples becoming parents: Trajectories for psychological distress and buffering effects of social support. *Journal of Affective Disorders*, 265, 372–380. <https://doi.org/10.1016/j.jad.2020.01.133>
- Hughes, C., Devine, R. T., Mesman, J., & Blair, C. (2018). *The new fathers and mothers study: Well-being, parenting and children's self-regulation 2014–2018*. [Data Collection]. Colchester, Essex: UK Data Service. <https://doi.org/10.5255/UKDA-SN-853278>
- Hughes, C., Devine, R. T., Mesman, J., & Blair, C. (2020). Understanding the terrible twos: A longitudinal investigation of the impact of early executive function and parent–child interactions. *Developmental Science*, 23(6), e12979. <https://doi.org/10.1111/desc.12979>
- Hughes, C., & Ensor, R. (2005). Executive function and theory of mind in 2 year olds: A family affair? *Developmental Neuropsychology*, 28(2), 645–668. https://doi.org/10.1207/s15326942dn2802_5
- Hughes, C. H., & Ensor, R. A. (2009). How do families help or hinder the emergence of early executive function? *New Directions for Child and Adolescent Development*, 2009(123), 35–50. <https://doi.org/10.1002/cd.234>
- Kalomiris, A. E., & Kiel, E. J. (2018). Mother–toddler cortisol synchrony moderates risk of early internalizing symptoms. *Infancy*, 23(2), 232–251. <https://doi.org/10.1111/inf.12216>
- Kaltsas, G. A., Chrousos, G. P., & Sternberg, E. M. (2007). The neuroendocrinology of stress. In J. T. Cacioppo, L. G. Tassinary & G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 303–318). Cambridge University Press.
- Khouri, J. E., Gonzalez, A., Levitan, R. D., Pruessner, J. C., Chopra, K., Basile, V. S., Masellis, M., Goodwill, A., & Atkinson, L. (2015). Summary cortisol reactivity indicators: Interrelations and meaning. *Neurobiology of Stress*, 2, 34–43. <https://doi.org/10.1016/j.ynstr.2015.04.002>
- Kiecolt-Glaser, J. K., & Wilson, S. J. (2017). Lovesick: How couples' relationships influence health. *Annual Review of Clinical Psychology*, 13, 421–443. <https://doi.org/10.1146/annurev-clinpsy-032816-045111>

- Kivlighan, K. T., DiPietro, J. A., Costigan, K. A., & Laudenslager, M. L. (2008). Diurnal rhythm of cortisol during late pregnancy: associations with maternal psychological well-being and fetal growth. *Psychoneuroendocrinology*, 33(9), 1225–1235. <https://doi.org/10.1016/j.psychoneu.2008.06.008>
- Kopp, C. B. (1982). Antecedents of self-regulation: A developmental perspective. *Developmental Psychology*, 18(2), 199–214. <https://doi.org/10.1037/0012-1649.18.2.199>
- Kuo, P. X., Braungart-Rieker, J. M., Burke Lefever, J. E., Sarma, M. S., O'Neill, M., & Gettler, L. T. (2018). Fathers' cortisol and testosterone in the days around infants' births predict later paternal involvement. *Hormones and Behavior*, 106, 28–34. <https://doi.org/10.1016/j.yhbeh.2018.08.011>
- Laurent, H. K., Sbrilli, M., Dawson, D., Finnegan, M., & Ramdas-Neal, D. (2020). Disentangling levels of mother–infant neuroendocrine attunement and longitudinal relations with maternal risk and protective factors. *Developmental Psychobiology*, 63, 88–97. <https://doi.org/10.1002/dev.21997>
- Laws, H. B., Sayer, A. G., Pietromonaco, P. R., & Powers, S. I. (2015). Longitudinal changes in spouses' HPA responses: Convergence in cortisol patterns during the early years of marriage. *Health Psychology*, 34(11), 1076–1089. <https://doi.org/10.1037/hea0000235>
- Leclere, C., Viaux, S., Avril, M., Achard, C., Chetouani, M., Missonnier, S., & Cohen, D. (2014). Why synchrony matters during mother–child interactions: a systematic review. *PLoS One*, 9(12), e113571. <https://doi.org/10.1371/journal.pone.0113571>
- Letourneau, N., Leung, B., Ntanda, H., Dewey, D., Deane, A. J., Giesbrecht, G. F., & APRON Team (2019). Maternal and paternal perinatal depressive symptoms associate with 2- and 3-year-old children's behaviour: findings from the APRON longitudinal study. *BMC Pediatrics*, 19(1), 435. <https://doi.org/10.1186/s12887-019-1775-1>
- Liu, S., Rovine, M. J., Klein, L. C., & Almeida, D. M. (2013). Synchrony of diurnal cortisol pattern in couples. *Journal of Family Psychology*, 27(4), 579–588. <https://doi.org/10.1037/a0033735>
- McHarg, G., Ribner, A. D., Devine, R. T., & Hughes, C. (2020). Screen time and executive function in toddlerhood: A longitudinal study. *Frontiers in Psychology*, 11, 570392. <https://doi.org/10.3389/fpsyg.2020.570392>
- Meyer, D., & Sledge, R. (2020). The potential role of cortisol as a biomarker of physiological interdependence in romantic couples: A systematic review. *Psychoneuroendocrinology*, 121, 104834. <https://doi.org/10.1016/j.psychoneu.2020.104834>
- Meyler, D., Stimpson, J. P., & Peek, M. K. (2007). Health concordance within couples: A systematic review. *Social Science & Medicine*, 64(11), 2297–2310. <https://doi.org/10.1016/j.socscimed.2007.02.007>
- Miller, S. E., & Marcovitch, S. (2015). Examining executive function in the second year of life: Coherence, stability, and relations to joint attention and language. *Developmental Psychology*, 51, 101–114. <https://doi.org/10.1037/a0038359>
- Moisiadis, V. G., & Matthews, S. G. (2014). Glucocorticoids and fetal programming part 1: Outcomes. *Nature Reviews Endocrinology*, 10(7), 391–402. <https://doi.org/10.1038/nrendo.2014.73>
- Monk, C., & Hane, A. A. (2014). Fetal and infant neurobehavioral development: basic processes and environmental influences. In A. Wenzel (Ed.), *The Oxford handbook of perinatal psychology* (pp. 53–86). Oxford University Press. <https://doi.org/10.1093/oxfordhb/9780199778072.013.20>
- Monk, C., Lugo-Candelas, C., & Trumpff, C. (2019). Prenatal developmental origins of future psychopathology: Mechanisms and pathways. *Annual Review of Clinical Psychology*, 15, 317–344. <https://doi.org/10.1146/annurev-clinpsy-050718-095539>
- Morsi, A., DeFranco, D., & Witchel, S. F. (2018). The hypothalamic-pituitary-adrenal axis and the fetus. *Hormone Research in Paediatrics*, 89(5), 380–387. <https://doi.org/10.1159/000488106>
- Muthén, B., & Asparouhov, T. (2012). Bayesian structural equation modeling: A more flexible representation of substantive theory. *Psychological Methods*, 17(3), 313–335. <https://doi.org/10.1037/a0026802>
- Nazzari, S., Fearon, P., Rice, F., Ciceri, F., Molteni, M., & Frigerio, A. (2020). Neuroendocrine and immune markers of maternal stress during pregnancy and infant cognitive development. *Developmental Psychobiology*, 62(8), 1100–1110. <https://doi.org/10.1002/dev.21967>
- Neuenschwander, R., Hookenson, K., Brain, U., Grunau, R. E., Devlin, A. M., Weinberg, J., Diamond, A., & Oberlander, T. F. (2018). Children's stress regulation mediates the association between prenatal maternal mood and child executive functions for boys, but not girls. *Development and Psychopathology*, 30(3), 953–969. <https://doi.org/10.1017/S095457941800041X>
- O'Connor, T. G., Bergman, K., Sarkar, P., & Glover, V. (2013). Prenatal cortisol exposure predicts infant cortisol response to acute stress. *Developmental Psychobiology*, 55(2), 145–155. <https://doi.org/10.1002/dev.21007>
- Palumbo, R. V., Marraccini, M. E., Weyandt, L. L., Wilder-Smith, O., McGee, H. A., Liu, S., & Goodwin, M. S. (2017). Interpersonal autonomic physiology: A systematic review of the literature. *Personality and Social Psychology Review*, 21(2), 99–141. <https://doi.org/10.1177/1088868316628405>
- Papoušek, H., & Papoušek, M. (2002). Intuitive parenting. In M. H. Bornstein (Ed.), *Handbook of parenting: Biology and ecology of parenting* (Vol. 2, 2nd ed., pp. 183–203). Lawrence Erlbaum Associates Publishers.
- Papp, L. M., Pendry, P., Simon, C. D., & Adam, E. K. (2013). Spouses' cortisol associations and moderators: testing physiological synchrony and connectedness in everyday life. *Family Process*, 52(2), 284–298. <https://doi.org/10.1111/j.1545-5300.2012.01413.x>
- Pearson, R. M., Lightman, S. L., Evans, J., 2009. Emotional sensitivity for motherhood: late pregnancy is associated with enhanced accuracy to encode emotional faces. *Hormones and Behavior*, 56(5), 557–563. <https://doi.org/10.1016/j.yhbeh.2009.09.013>
- Perry, R. E., Braren, S. H., Blair, C., & the Family Life Project Key Investigators. (2018). Socioeconomic risk and school readiness: Longitudinal mediation through children's social competence and executive function. *Frontiers in Psychology*, 9, 1544. <https://doi.org/10.3389/fpsyg.2018.01544>
- Perry, R. E., Finegood, E. D., Braren, S. H., & Blair, C. (2019). The social neuroendocrinology and development of executive functions. In P. H. M. Oliver C. Schultheiss (Ed.), *Routledge international handbook of social neuroendocrinology* (pp. 530–543). Routledge.
- Philpott, L. F., Leahy-Warren, P., FitzGerald, S., Savage, E., 2017. Stress in fathers in the perinatal period: A systematic review. *Midwifery*, 55, 113–127. <https://doi.org/10.1016/j.midw.2017.09.016>
- Pietromonaco, P. R., Uchino, B., & Dunkel Schetter, C. (2013). Close relationship processes and health: implications of attachment theory for health and disease. *Health Psychology*, 32(5), 499–513. <https://doi.org/10.1037/a0029349>
- Plantin, L., Olukoya, A. A., & Ny, P. (2011). Positive health outcomes of fathers' involvement in pregnancy and childbirth paternal support: A scope study literature review. *Fathering: A Journal of Theory, Research, and Practice about Men as Fathers*, 9(1), 87–102. <https://doi.org/10.3149/ft.0901.87>
- Pruessner, J. C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (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. <https://www.ncbi.nlm.nih.gov/pubmed/12892658>
- Radloff, L. S., 1977. The CES-D scale: a self report depression scale for research in the general population. *Applied Psychological Measurement*, 1(3), 385–401.
- Rash, J. A., Thomas, J. C., Campbell, T. S., Letourneau, N., Granger, D. A., & Giesbrecht, G. F. (2016). Developmental origins of infant stress reactivity profiles: A multi-system approach. *Developmental Psychobiology*, 58(5), 578–599. <https://doi.org/10.1002/dev.21403>

- Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (Vol. 1). Sage.
- Raz, S. (2014). Behavioral and neural correlates of cognitive-affective function during late pregnancy: an Event-Related Potentials Study. *Behavioral Brain Research*, 267, 17–25. <https://doi.org/10.1016/j.bbr.2014.03.021>
- Robles, T. F., Slatcher, R. B., Trombello, J. M., & McGinn, M. M. (2014). Marital quality and health: a meta-analytic review. *Psychological Bulletin*, 140(1), 140–187. <https://doi.org/10.1037/a0031859>
- Saltzman, W., & Ziegler, T. E. (2014). Functional significance of hormonal changes in mammalian fathers. *Journal of Neuroendocrinology*, 26(10), 685–696. <https://doi.org/10.1111/jne.12176>
- Samdan, G., Kiel, N., Petermann, F., Rothenfuß, S., Zierul, C., & Reinelt, T. (2020). The relationship between parental behavior and infant regulation: A systematic review. *Developmental Review*, 57, 100923. <https://doi.org/10.1016/j.dr.2020.100923>
- Sameroff, A. J. (2009). Conceptual issues in studying the development of self-regulation. In S. L. Olson & A. J. Sameroff (Eds.), *Biopsychosocial regulatory processes in the development of childhood behavioral problems* (pp. 1–18). Cambridge University Press. <https://doi.org/10.1017/cbo9780511575877.002>
- Saxbe, D. E., Adam, E. K., Schetter, C. D., Guardino, C. M., Simon, C., McKinney, C. O., Shalowitz, M. U., & Eunice Kennedy Shriver National Institute for Child Health and Human Development (NICHD) Community Child Health Network (CCHN). (2015). Cortisol covariation within parents of young children: Moderation by relationship aggression. *Psychoneuroendocrinology*, 62, 121–128. <https://doi.org/10.1016/j.psyneuen.2015.08.006>
- Saxbe, D. E., Rossin-Slater, M., & Goldenberg, D. (2018). The transition to parenthood as a critical window for adult health. *American Psychologist*, 73(9), 1190–1200. <https://doi.org/10.1037/amp0000376>
- Saxbe, D. E., Edelstein, R. S., Lyden, H. M., Wardecker, B. M., Chopik, W. J., & Moors, A. C. (2017). Fathers' decline in testosterone and synchrony with partner testosterone during pregnancy predicts greater postpartum relationship investment. *Hormones and Behavior*, 90, 39–47. <https://doi.org/10.1016/j.yhbeh.2016.07.005>
- Saxbe, D., Khoddam, H., Corner, G. W., Stoycos, S. A., & Khaled, M. (2018). Physiological correlates associated with interpersonal emotion dynamics. In A. K. Randall (Ed.), *Interpersonal emotion dynamics in close relationships* (pp. 110–128). Cambridge University Press. <https://doi.org/10.1017/9781316822944.008>
- Saxbe, D., & Repetti, R. L. (2010). For better or worse? Coregulation of couples' cortisol levels and mood states. *Journal of Personality and Social Psychology*, 98(1), 92–103. <https://doi.org/10.1037/a0016959>
- Sbarra, D. A., & Hazan, C. (2008). Coregulation, dysregulation, self-regulation: an integrative analysis and empirical agenda for understanding adult attachment, separation, loss, and recovery. *Personality and Social Psychology Review*, 12(2), 141–167. <https://doi.org/10.1177/1088868308315702>
- Schneiderman, I., Kanat-Maymon, Y., Zagoory-Sharon, O., & Feldman, R. (2014). Mutual influences between partners' hormones shape conflict dialog and relationship duration at the initiation of romantic love. *Social Neuroscience*, 9(4), 337–351. <https://doi.org/10.1080/17470919.2014.893925>
- Seckl, J. R., & Meaney, M. J. (2004). Glucocorticoid programming. *Annals of the New York Academy of Sciences*, 1032, 63–84. <https://doi.org/10.1196/annals.1314.006>
- Senese, V. P., Miranda, M. C., De Falco, S., Venuti, P., Bornstein, M. H., 2018. How becoming a mother shapes implicit and explicit responses to infant cues. *Developmental Psychobiology*, 60(8), 950–962. <https://doi.org/10.1002/dev.21772>
- Shin, H., Park, Y. J., & Mi, J. K. (2006). Predictors of maternal sensitivity during the early postpartum period. *Journal of Advanced Nursing*, 55(4), 425–434. <https://doi.org/10.1111/j.1365-2648.2006.03943.x>
- Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., Jacobs, G. A., 1983. *Manual for the State-Trait Anxiety Inventory*. Consulting Psychologists Press.
- Sroufe, L. A. (1996). *Emotional development: The organization of emotional life in the early years*. Cambridge University Press. <https://doi.org/10.1017/cbo9780511527661>
- Storey, A. E., Walsh, C. J., Quinton, R. L., & Wynne-Edwards, K. E. (2000). Hormonal correlates of paternal responsiveness in new and expectant fathers. *Evolution and Human Behavior*, 21(2), 79–95. [https://doi.org/10.1016/S1090-5138\(99\)00042-2](https://doi.org/10.1016/S1090-5138(99)00042-2)
- Storey, A. E., & Ziegler, T. E. (2016). Primate paternal care: Interactions between biology and social experience. *Hormones and Behavior*, 77, 260–271. <https://doi.org/10.1016/j.yhbeh.2015.07.024>
- Straus, M. A. (1979). Measuring intrafamily conflict and violence: The Conflict Tactics (CT) Scales. *Journal of Marriage and the Family*, 41, 75–88. <http://doi.org/10.2307/351733>
- Talge, N. M., Neal, C., Glover, V., & Early Stress, Translational Research and Prevention Science Network: Fetal and Neonatal Experience on Child and Adolescent Mental Health (2007). Antenatal maternal stress and long-term effects on child neurodevelopment: how and why?. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 48(3-4), 245–261. <https://doi.org/10.1111/j.1469-7610.2006.01714.x>
- Stapleton, L. R. T., Schetter, C. D., Westling, E., Rini, C., Glynn, L. M., Hobel, C. J., & Sandman, C. A. (2012). Perceived partner support in pregnancy predicts lower maternal and infant distress. *Journal of Family Psychology*, 26(3), 453–463. <https://doi.org/10.1037/a0028332>
- Thomas, J. C., Letourneau, N., Campbell, T. S., Giesbrecht, G. F., & Apron Study Team. (2018). Social buffering of the maternal and infant HPA axes: Mediation and moderation in the intergenerational transmission of adverse childhood experiences. *Development and Psychopathology*, 30(3), 921–939. <https://doi.org/10.1017/S0954579418000512>
- Thorson, K. R., West, T. V., & Mendes, W. B. (2018). Measuring physiological influence in dyads: A guide to designing, implementing, and analyzing dyadic physiological studies. *Psychological Methods*, 23(4), 595–616. <https://doi.org/10.1037/met0000166>
- Timmons, A. C., Margolin, G., & Saxbe, D. E. (2015). Physiological linkage in couples and its implications for individual and interpersonal functioning: A literature review. *Journal of Family Psychology*, 29(5), 720–731. <https://doi.org/10.1037/fam0000115>
- Ulrich-Lai, Y. M., & Herman, J. P. (2009). Neural regulation of endocrine and autonomic stress responses. *Nature Reviews Neuroscience*, 10(6), 397–409.
- Velders, F. P., Dieleman, G., Henrichs, J., Jaddoe, V. W. V., Hofman, A., Verhulst, F. C., Hudziak, J. J., & Tiemeier, H. (2011). Prenatal and postnatal psychological symptoms of parents and family functioning: The impact on child emotional and behavioural problems. *European Child and Adolescent Psychiatry*, 20(7), 341–350. <https://doi.org/10.1007/s00787-011-0178-0>
- Voegtline, K. M., Costigan, K. A., Kivlighan, K. T., Laudenslager, M. L., Henderson, J. L., & Dipietro, J. A. (2013). Concurrent levels of maternal salivary cortisol are unrelated to self-reported psychological measures in low-risk pregnant women. *Archives of Women's Mental Health*, 16(2), 101–108. <https://doi.org/10.1007/s00737-012-0321-z>
- Vygotsky, L. S. (1978). *Mind in society: The development of higher psychological processes*. Harvard University Press.
- Wynne-Edwards, K. E. (2001). Hormonal changes in mammalian fathers. *Hormones and Behavior*, 40(2), 139–145. <https://doi.org/10.1006/hbeh.2001.1699>
- Yim, I., Tanner Stapleton, L., Guardino, C., Hahn-Holbrook, J., & Dunkel Schetter, C. (2015). Biological and psychosocial predictors of postpartum depression: systematic review and call for integration. *Annual*

Review of Clinical Psychology 11, 99–137. <https://doi.org/10.1146/annurev-clinpsy-101414-020426>

Zijlmans, M. A., Riksen-Walraven, J. M., & de Weerth, C. (2015). Associations between maternal prenatal cortisol concentrations and child outcomes: A systematic review. *Neuroscience & Biobehavioral Reviews*, 53, 1–24. <https://doi.org/10.1016/j.neubiorev.2015.02.015>

Zimet, G., Dahlem, N., Zimet, S., Farley, G., 1988. The multidimensional scale of perceived social support. *Journal of Personality Assessment*, 52, 30–41.

How to cite this article: Braren, S. H., Perry, R. E., Ribner, A., Brandes-Aitken, A., Brito, N., Blair, C., the New Fathers and Mothers Study (NewFAMS) Team (2021). Prenatal mother–father cortisol linkage predicts infant executive functions at 24 months. *Dev Psychobiol*, 63: e22151. <https://doi.org/10.1002/dev.22151>