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Review

Quantitative meta-analysis of maternal prenatal salivary cortisol and newborn birthweight does not identify effect of fetal sex

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ABSTRACT

Background: Heightened concentration of maternal cortisol is a frequently proposed mechanism linking adverse maternal environments with poor birth outcomes, including birth weight. It is commonly hypothesized that prenatal exposures have sexually dimorphic effects on fetal development, however few studies have assessed the effects of fetal sex on the relationship between maternal cortisol and birth outcomes.

Methods: In a previous systematic review and meta-analysis we obtained data from authors of included studies to calculate trimester-specific correlations between maternal prenatal salivary cortisol and newborn birth weight. Given that this data was well-poised to address the unknown effects of fetal sex on the relationship between maternal cortisol and birth outcomes, we contacted authors a second time with request to unblind sex into the correlations. An updated database search was conducted to identify potentially relevant articles published within 2018 and two additional articles were included.

Results and discussion: Eleven studies with a total of 2236 maternal-fetal dyads demonstrated negative correlations for both males, -0.15 (95% CI -0.24 to -0.06, $I^2 = 98.5\%$, $p < 0.001$) and females -0.21 (95% CI -0.25 to -0.17, $I^2 = 93.3\%$, $p < 0.001$). Sex difference were not statistically significant, $p = 0.62$. Despite greater exposure to cortisol and lower birth weight among females, the association did not differ by sex.

1. Introduction

Fetal sex alters pregnancy-related adaptations in the maternal hypothalamic-pituitary-adrenal (HPA) axis (DiPietro et al., 2011; Giesbrecht et al., 2015). Specifically, women pregnant with a female fetus exhibit higher mean levels of salivary cortisol during the later weeks of pregnancy (DiPietro et al., 2011) and demonstrate flatter daytime salivary cortisol slopes compared to women pregnant with a male (Giesbrecht et al., 2015). The reason for these differences is not clear, however we speculate that sex differences in maternal HPA axis function may be associated with sex differences in fetal growth. This speculation is supported by two lines of reasoning.

First, fetal synthesis of cortisol does not differ by sex and yet female fetuses have increased concentration of cortisol in both arterial and venous umbilical cord blood circulation (Giesbrecht et al., 2016). Importantly, evidence supporting this claim comes from scheduled caesarean delivery without antecedent labour. When labour occurred, cortisol concentrations were elevated, and sex differences were not found (Wynne-Edwards et al., 2013). These data suggest that females are exposed to higher concentrations of cortisol at the end of gestation and that the probable source of this increase is the maternal or placental milieu.

Second, in a previous systematic review and meta-analysis, we reported that higher maternal cortisol during pregnancy was associated with newborn birth weight (Cherak et al., 2018). That review combined data from both fetal sexes because most studies did not separately

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report associations by sex. Given that exposure to higher concentrations of cortisol during gestation is associated with reduced birthweight, we speculated that greater overall exposure in females may contribute to lower overall birthweight among females, and that this association would be stronger in females relative to males. Furthermore, our previously reported association between maternal cortisol and neonatal birth weight may have been confounded by sex because full-term females tend to have the higher cortisol exposure and the lower birthweights compared to males. Therefore, disambiguating the effects of sex are crucial to understanding the association between maternal cortisol and neonatal birth weight.

2. Methods

Our initial review was pre-registered in the International Prospective Register of Systematic Review (PROSPERO, [ID: CRD42017079183]) and followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines for systematic reviews. The databases MEDLINE, EMBASE, PsycINFO, and CINAHL were used for a literature search from November 1947 until November 2nd, 2017 and updated on January 1st, 2019 (Supplementary Table 1). Two additional studies were identified and included, for a total of 11 studies. We included data from observational studies that measured maternal diurnal salivary cortisol and newborn birth weight from human studies of participants with healthy, uncomplicated, full-term (37–40 weeks, postdates excluded), singleton births. From each included paper, a Pearson's correlation coefficient (r) between the maternal diurnal cortisol area under the curve with respect to ground (AUCg [μg/dL per day]) and the newborn birth weight (grams) was extracted from the published statistical analyses for each gestational trimester. If a Pearson's correlation coefficient was not explicitly stated or able to be calculated from the published study, we contacted the authors to specifically request this information. The coefficient for each included study was then transformed to the Fisher's z scale and random-effects meta-analysis was performed with transformed values. Out of the eleven included studies, only four reported separate findings for males and females, therefore we contacted authors with requests to unblind sex into the data. Therefore, the data reported here make a unique contribution beyond our original review and the original papers themselves. Here we report sex-specific correlations between an AUCg for maternal prenatal salivary cortisol with continuous measures of newborn birth weight.

3. Results

Eleven articles were included (Bolten et al., 2011; Braithwaite et al., 2018; Bublitz et al., 2016a, b; D'Anna-Hernandez et al., 2012; Giesbrecht et al., 2015; Gilles et al., 2018; Guardino et al., 2016; Hompes et al., 2012; Kivlighan et al., 2008; Spicer et al., 2013) (Supplementary Fig. 1). Two of the articles (Braithwaite et al., 2018; Gilles et al., 2018) were not included in our previous review. Characteristics and demographics of the eleven included studies are presented in Table 1 and Supplementary Table 2, respectively. Descriptive results of study quality assessment are presented in Supplementary Figure 2.

Of the eleven included studies, all were set in high-income countries (6, USA; 1, Canada; 1, Belgium; 2, Germany; 1, England). Measurement of salivary cortisol varied in regard to number and timing of cortisol sampling per day. The median number of samples per day was three and the maximum number of samples per day was six.

From 2236 maternal-infant dyads, 52.6% (n = 1177) were males. The mean newborn birth weight was 3345 g (SD 143 g). There was no significant sex difference in mean newborn birth weight between males and females; males weighed on average 3369 g (SD 167 g) with females at 3320 g (SD 119 g). Pearson's correlation coefficients calculated for each study were subdivided by fetal sex (Fig. 1). All studies reported negative associations between maternal cortisol and infant birthweight for both males and females, with the exception of one study (Braithwaite) that reported a significant positive association for males. Overall, associations for male fetuses (-0.15, 95% CI -0.24 to -0.06, $p < 0.001$) was not significantly different from females (-0.21, 95% CI -0.25 to -0.17, $p < 0.001$) (Q $p = 0.62$), despite that eight of eleven studies reported a less negative albeit non-significantly different association compared to females. Whereas high heterogeneity was present for between-study associations for male fetuses (Q = 98.5%), due to a single study that reported a positive correlation, the correlation for female fetuses show lower heterogeneity (Q = 93.3%). The

Table 1
Characteristics and results of included studies on sex differences in the association between maternal prenatal salivary cortisol and birth weight as published.

Author	Year	Country	Weeks of pregnancy (SD)	Number of consecutive days	Number of times per day	Fetal characteristics
Bolten et al.	2011	Germany	13–18 35–37	Not stated	4	N = 94 n_{male} = 43
Braithwaite et al.	2018	England	32	2	3	N = 241 n_{male} = 115
Bublitz et al.	2016a	USA	24 (3) 36 (1)	3	3	N = 184 n_{male} = 95
Bublitz et al.	2016b	USA	24 (3) 36 (1)	3	3	N = 217 n_{male} = 113
D'Anna-Hernandez et al.	2012	USA	17.3 (1.8) 28.1 (1.5) 34.3 (1.4)	3	3	N = 55 n_{male} = 28
Giesbrecht et al.	2015	Canada	14.9 (3.7) 32.4 (1.0)	2	4	N = 294 n_{male} = 153
Gilles et al.	2018	Germany	36.8 (1.9)	1	3	N = 405 n_{male} = 229
Guardino et al.	2016	USA	2nd trimester 3rd trimester	1	3	N = 343 n_{male} = 178
Hompes et al.	2012	Belgium	10.8 (2.0) 23.6 (2.2) 34.9 (1.9)	1	4	N = 100 n_{male} = 48
Kivlighan et al.	2008	USA	36	2	3	N = 98 n_{male} = 49
Spicer et al.	2013	USA	13–16 34–37	1	6	N = 205 n_{male} = 126

Footnotes: Non-overlapping of participants was ensured between Bublitz et al., 2016a and Bublitz et al., 2016b (i.e., each maternal-fetal dyad was represented only once).

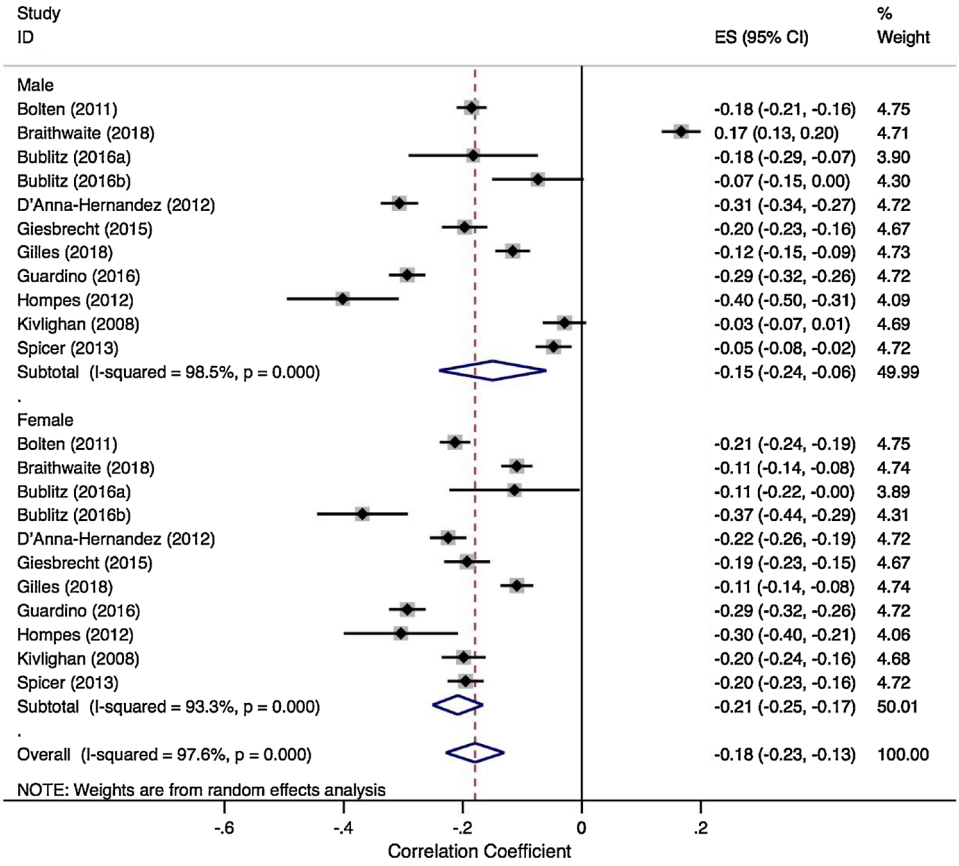

Fig. 1. Forest plot of effect size (ES) and corresponding 95% confidence interval (CI) for included studies on sex differences in the association between maternal prenatal salivary cortisol and birth weight. The solid vertical line represents no sex difference, and the dashed line shows the meta-analytic mean.

difference between the effects of maternal salivary cortisol on newborn birth weight in males, as opposed to females, was 0.06 (95% CI -0.23 to 0.64). For every unit increase in AUCg (μg/dL per day) newborn birth weight decreased by 0.6 ($R^2 = 0.05$) and 1.3 ($R^2 = 0.16$) grams for males and females, respectively.

4. Discussion and conclusion

In this review, we evaluated eleven studies that examined the association between maternal prenatal salivary cortisol during pregnancy and newborn birth weight sub-divided by fetal sex. Fetal sex did not affect the association between maternal prenatal salivary cortisol and full-term newborn birth weight. The significant negative association for male and female fetuses suggests that excess maternal salivary cortisol has similar implications for fetal growth in both males and females. Accordingly, factors that increase cortisol among pregnant women (e.g., stress) are likely to result in similar decreases in birthweight among both males and females.

As shown in Fig. 1, all studies, with the exception of (Braithwaite et al., 2018), observed negative associations between maternal prenatal salivary cortisol and birth weight for both males and females. Braithwaite and colleagues conducted a large prospective cohort study from which they determined a significant positive association in males and a negative association that did not reach statistical significance in females. The authors offer no explanation for their unique findings compared to the rest of the literature for which negative associations for both males and females are reported. Furthermore, we could not identify any notable difference in methodology that would have contributed to the discrepant results, however we note that this was the only study that did not adjust for potential confounders. Since the developmental ontogeny of fetal adrenal steroidogenic function also begins to resemble the adult at approximately 30 weeks of gestational age (Ishimoto and Jaffe, 2011), we speculate that the sex difference seen in the Braithwaite cohort starting at 32 weeks could arise from a developmental asynchrony between males and females in adrenal steroidogenic capability.

Cortisol is an important factor in fetal growth (Liggins, 1994) and normal fetal development and maturation are dependent upon optimal increase in maternal cortisol with advancing gestation (Davis et al., 2010). Increases in maternal cortisol during pregnancy are normative (Allolio et al., 1990; Jung et al., 2011; Soma-Pillay et al., 2016) and appear to be important for proper fetal maturation especially later in pregnancy, which is facilitated by greater proportion of maternal cortisol crossing the placental barrier (Benediktsson et al., 1997). Because cortisol is a critical factor involved in fetal growth, and because there are known sex differences in fetal exposure to cortisol and in neonatal birthweight, we expected to observe sex differences in the association between maternal cortisol and neonatal birthweight.

Here we discuss potential explanations for this result. First, perhaps the underlying mechanisms for maternal cortisol production operate differently for male and female fetuses. There is considerable evidence that the placenta is sexually differentiated, and that males continue to grow during adverse maternal stress situations whereas females reduce growth rate in response to maternal stress (Clifton, 2010).

Cortisol is known to exert a wide array of metabolic, endocrine, and immune effects on most if not all cells, and is known to play a key role in the body's response to psychological and physiological stress and maintenance of homeostasis. The obligatory role of cortisol underlying the development of the brain and other organ systems is highlighted by the expression of glucocorticoid receptors in most fetal tissue, and the necessity of these receptors for survival of the fetus (Sapolsky, 2000). In the fetal brain, specifically, cortisol plays a role in multiple aspects of

development, including neurogenesis, gliogenesis, synaptogenesis, and growth of axons and dendrite (Moisiadis and Matthews, 2014). Indeed, head circumference at birth (a marker of brain size in humans) is strongly correlated with newborn birth weight in a manner that is not sex-dependent (Epstein and Epstein, 1978). Although there are several examples in the animal and human literature to suggest that prenatal exposures produce sexually dimorphic developmental consequences, no study to date has investigated whether the maternal cortisol biosynthesis during pregnancy is responsive to sex hormones. Our knowledge on whether the pathways underlying cortisol production with the ability to reduce newborn birth weight in a sex-specific manner, is therefore incomplete.

A second potential explanation is that sex differences in newborn birthweight may only be revealed when a prenatal challenge or adverse environment exists *in utero*. It has long been reported that male fetuses tend to be more vulnerable to prenatal and perinatal adversity (Gualtieri et al., 1985). Sex-specific fetal growth strategies are thought to be responsible, specifically that the female growth strategy results in greater adaptive flexibility in short-term, and especially so in conditions of maternal stress, but increased long-term risk for developmental psychopathology. This is seen most clearly in work on pregnancies complicated by maternal asthma where female fetuses reduce their growth whereas males do not (Murphy et al., 2003), but males have greatly increased incidence of intrauterine growth restriction if the mother experiences an acute exacerbation of asthma (Murphy et al., 2005). Since only healthy, uncomplicated, full-term singleton pregnancies are included in the present review, it is reasonable that no sex differences were observed (Clifton, 2010).

One limitation of this work is that all studies were conducted in high-income countries. The findings of this review may not represent a diverse ethnic and sociodemographic of women. Another limitation to this analysis is that the relationship between maternal free cortisol and fetal cortisol exposure is complex. This is because saliva does not contain cortisol bound to cortisol binding globulin (CBG) or to albumin. Furthermore, CBG binds progesterone in addition to cortisol that is abundant in intervillous serum (Benassayag et al., 2001), and mutations that decrease CBG levels result in female-biased secondary sex ratios (Lei et al., 2015). Undoubtedly, this further adds to the challenges of using maternal salivary cortisol to predict fetal cortisol exposure.

There are several potential implications for this work. First this work contributes to clinical practice by providing evidence that although maternal prenatal stress may adversely impact newborn birth weight, that there is no difference in impact on birth weight between males and females provides clinicians with assurance that the impact remains unrelated to fetal sex. Second, the authors suggest that fetal sex should continue to be assessed in future research to understand why sex differences in fetal cortisol exposure and birth weight are unrelated to the association between these variables. Ongoing efforts to gather this data will allow for more robust meta-analyses that includes fetal sex. Third, while these findings are of general interest, it is also possible that these findings offer more direct implication for pregnancies in which low birthweight is a pre-existing risk factor.

To conclude, we report the absence of a sex difference in the negative association between maternal prenatal cortisol and newborn birth weight. These findings add to an emerging body of literature investigating possible sex-specific fetal programming mechanisms aimed to better understand the early origins of sex differences in developmental psychopathology.

Contributors

All authors have materially participated in the research and/or article preparation, and have approved the final article. The contribution of each individual author is as follows:

SJC; conception and design of the study, acquisition of data, analysis and interpretation of data, drafting the article, revising the article critically, and final approval of the version to be submitted.

MEM; conception and design of the study, acquisition of data, analysis and interpretation of data, revising the article critically, and final approval of the version to be submitted.

KEWE; analysis and interpretation of data, drafting the article, revising the article critically, and final approval of the version to be submitted.

TW; revising the article critically and final approval of the version to be submitted.

GFG; conception and design of the study, revising the article critically, and final approval of the version to be submitted.

Conflict of interest statement

The authors declare that they have no conflict(s) of interest.

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Appendix A. Supplementary data

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