



Advancing gestation does not attenuate biobehavioural coherence between psychological distress and cortisol



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ABSTRACT

Background: Despite little evidence to suggest that HPA axis responses to psychological provocation are attenuated during pregnancy, it is widely held that dampening of the HPA axis response to psychological distress serves a protective function for the mother and fetus. The current study was designed to assess changes in biobehavioral coherence between psychological distress and cortisol over the course of pregnancy.

Methods: Ambulatory assessment of ecologically relevant psychological distress and salivary cortisol were repeated in all three trimesters for 82 pregnant women. Samples were collected 5 times per day over the course of 2 days in each trimester.

Results: Psychological distress and cortisol were positively associated, $\beta = .024$, $p < .01$, indicating that increases in psychological distress were associated with increases in cortisol. Gestational age did not moderate this association, $\beta = .0009$, $p = .13$, suggesting that negative psychological experiences remain potent stimuli for the HPA axis during pregnancy.

Conclusion: Biobehavioral coherence between ecologically relevant experiences of psychological distress and cortisol is not attenuated with advancing gestation.

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1. Introduction

Psychological distress during pregnancy is associated with adverse obstetric (e.g., intra-uterine growth restriction, preterm birth) and developmental outcomes (e.g., exaggerated behavioral and hypothalamic–adrenal–pituitary [HPA] axis response to stress; Kapoor et al., 2006). Animal studies suggest that psychological distress generally has a larger effect on fetal development when the exposure occurs earlier rather than later in gestation (Mueller and Bale, 2007; Schneider et al., 1999). The effects of exposure timing on human development and the underlying mechanisms, however, are not as well understood. Moreover, in light of evidence for progressive adjustment within maternal physiological systems involved in stress responses (one of the pathways by which psychological distress may impact fetal development; de Weerth and Buitelaar, 2005) and data indicating that the prevalence of

self-reported affective disorders may be as high as 51% in some groups (Halbreich, 2004) with approximately 14% of pregnant women meeting diagnostic criteria for an affective disorder (Alder et al., 2007), understanding how and when psychological distress ‘gets under the skin’ of the fetus is an important conduit to improving infant health and development.

It is well-known that biological systems are more vulnerable to exposure during periods of rapid development (Davis and Sandman, 2010; Moore and Persaud, 1998), which may partially explain why the effects of psychological distress do not appear to be uniform across gestation or across developmental domains. For example, the effects of psychological distress during pregnancy on infant cognitive development may be greatest when the exposure occurs early (15 weeks) in pregnancy (Davis and Sandman, 2010; Laplante et al., 2004) whereas adverse birth outcomes have been associated with exposure during the 5th and 6th months of gestation (Class et al., 2011). Findings from the Western Australian Pregnancy Cohort (Raine) Study, one of the largest prospective cohorts of pregnancy and childhood (Robinson et al., 2011), suggest that behavioral morbidity is higher in children who were exposed to maternal distress at 18 versus 32 weeks gestational age (GA). In contrast, reports from another large cohort study,

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the Avon Longitudinal Study of Parents and Children (ALSPAC; O'Connor et al., 2002), suggest that maternal distress at 32 but not 18 weeks GA was associated with child behavioral problems. The inconsistent results may in part reflect differences in the measurement of maternal distress, infant behavior and parenting. The overall pattern of results, however, reinforces the notion that diverse developmental outcomes may have differing periods of vulnerability to psychological distress that are influenced by the type, intensity and duration of the exposure (Wadhwa et al., 2001).

In addition to the effects of developmental changes in biological vulnerability, the timing of exposure may affect development because the physiological “dose” associated with psychological distress may decrease as a function of gestational age. Changes in maternal physiological reactivity to negative psychological experiences during pregnancy are supported by research with animals suggesting that in most mammals, the major stress systems mount dampened responses (Slattery and Neumann, 2008), including marked attenuation of the HPA axis response to both physical and mixed physical and psychological stressors (Neumann et al., 1998; Russell et al., 2008). The prevailing interpretation of these findings is that physiological changes during pregnancy may serve to protect the fetus and mother from the potentially negative effects of noxious psychological experiences (de Weerth and Buitelaar, 2005; DiPietro et al., 2012; Petraglia et al., 2001; Slattery and Neumann, 2008).

As a consequence of significant species differences in the structure and function of the placenta and in the nature of stress responses, there is a need to exercise caution in extrapolating animal findings suggesting attenuation of reactivity to humans (Michael and Papageorgiou, 2008). Nevertheless, reports from human studies have typically supported the proposal that responses to negative psychological stimulation are progressively dampened throughout pregnancy (de Weerth and Buitelaar, 2005; DiPietro et al., 2005; Entringer et al., 2010; Klinkenberg et al., 2009; Matthews and Rodin, 1992; Slattery and Neumann, 2008; Wadhwa, 2005). Overall, the evidence in these studies is most consistent for the autonomic nervous system (ANS) relative to HPA reactivity. The lack of consistent results from studies of the HPA axis may be a product of differences in the nature of the challenge presented. For example, chemical and physical stimulation of the HPA axis in late gestation, including a standard corticotrophin releasing hormone (CRH) test (Schulte et al., 1990) and a painful cold pressor task (Kammerer et al., 2002), failed to produce a robust cortisol response in pregnant women, while the dexamethasone test (Odagiri et al., 1988) produced less suppression of cortisol production compared to non-pregnant women. These studies support the notion of attenuated HPA axis reactivity. Studies involving what are arguably more psychological forms of stimulation, in contrast, have been less consistent. For example, fetal blood transfusion (a potentially potent psychological stressor) did not produce a maternal cortisol response in the 2nd or 3rd trimesters (Gitau et al., 2001) whereas public speaking and mental arithmetic did (de Weerth et al., 2007a,b; Nierop et al., 2006). Considering the evidence to date, there may be a selective dampening of the HPA axis during pregnancy such that tasks designed to elicit social and mental stress remain potent activators.

Prospective studies with repeated assessment throughout pregnancy are needed to determine if biobehavioural coherence between psychological distress and the HPA axis is progressively attenuated over the course of pregnancy. At present, there are only two such published reports. Entringer et al. (2010) used a standard Trier Social Stress Task (TSST) to assess cortisol reactivity among pregnant and non-pregnant women. Modification of the TSST protocol to reduce physical discomfort during the procedures (women were reclined in a comfortable chair), however, led to a failure to produce a cortisol increase in either group. In a field

study involving 603 pregnant women, Obel et al. (2005) reported no association between life events/worry and diurnal cortisol among women in the 2nd trimester. In contrast, women in the 3rd trimester with higher self-reported stress had elevated levels of evening cortisol, suggesting that basal cortisol response to stress may increase rather than decrease over the course of gestation.

In the present study we not only assessed the biobehavioural coherence between psychological distress and cortisol but also we tested the specific claim that this coherence is progressively attenuated with advancing gestation (Glynn et al., 2001, 2004). The study was designed to build upon previous cross-sectional research in which basal levels of cortisol were positively associated with changes in self-reported psychological distress (Giesbrecht et al., 2012). The goal was to prospectively assess subjective experiences of psychological distress and cortisol within the day-to-day experiences of pregnant women over the course of gestation. Characterizing the biobehavioural coherence between psychological distress and cortisol during gestation has important implications for understanding the potentially differential effects of psychological distress on developmental outcomes arising from different phases of pregnancy.

2. Method

2.1. Participants

Eighty five pregnant women who were enrolled in an ongoing longitudinal study of nutrition during pregnancy (see <http://www.apronstudy.ca> for further details) participated. Women were excluded if they were >14 weeks gestation or if they reported any of the following: (a) taking a steroid medication, (b) smoking, (c) consuming alcohol or ‘street’ drugs, (d) recent dental work or tendency for oral bleeding (leading to falsely elevated cortisol values (Kivlighan et al., 2004)), (e) known pregnancy or fetal complications (e.g., preeclampsia, fetal genetic anomalies), or illness during data collection (e.g., fever). GA at each assessment was determined based on last reported menstrual period and confirmed by at least one ultrasound. Prior to data collection, participants provided informed consent to the procedures. The study procedures were approved by the University of Calgary Conjoint Health Research Ethics Board.

2.2. Procedures

Participants completed the sampling procedures three times during pregnancy: Time 1 (T1) prior to 14 weeks GA, Time 2 (T2) at 21 weeks GA, and Time 3 (T3) at 32 weeks GA. At T1, participants attended an individualized session where they were instructed on the use of the personal digital assistant (PDA) data collection device and Salimetrics Oral Swab (Salimetrics, Pennsylvania, USA) for saliva collection. On each assessment occasion, women self-collected saliva at home over 2 consecutive days (excluding weekends in order to rule out potential weekend-weekday differences in stress and diurnal cortisol (Schlotz et al., 2004) for a total of 6 days across pregnancy. Samples were obtained on the following schedule: upon waking (allowing for individualized wake times), 30 min after waking, and semi-randomly after the anchor times of 1100 h, 1530 h, and 2000 h. The semi-random signals occurred on the PDA once within 15 min following the anchor times to reduce the possibility of changes in mood associated with anticipation of the signal. To facilitate adherence to the study protocol, the PDA was programmed to allow a 20 min response window following the signal, after which data were considered missing.

Each time the PDA rang, it first provided a unique code corresponding to a pre-labeled saliva tube and instructed participants to place the saliva roll under her tongue. Each response to the PDA was marked by a time stamp permitting precise modeling of the diurnal patterns. The PDA administered the psychological distress questionnaire during the saliva collection.

2.3. Measures

2.3.1. Cortisol

Participants were asked to refrain from consuming food, caffeine, citric drinks and dairy, and to avoid vigorous exercise or brushing teeth in the 30 min prior to saliva collection and to report adherence to these guidelines. Whole saliva was obtained from under the tongue. Saliva samples were stored at -20°C until they were shipped frozen to Salimetrics, State College, PA. All samples were assayed for salivary cortisol using a highly sensitive enzyme immunoassay. The test has a lower limit of sensitivity of $0.003\text{ }\mu\text{g/dl}$, standard curve range from 0.012 to $3.0\text{ }\mu\text{g/dl}$, and average intra- and inter-assay coefficients of variation 3.5% and 5.1%, respectively. Method accuracy, determined by spike and recovery, and linearity, determined by serial dilution are 100.8% and 91.7%. A random 10% of samples were assayed in duplicate to confirm reliability; the intra-assay coefficient of variation

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