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Anxious women do not show the expected decrease in cardiovascular stress responsiveness as pregnancy advances

M.A.K.A. Braeken[a,b,c], A. Jones[d], R.A. Otte[a], D. Widjaja[e], S. Van Huffel[e], G.J.Y.J. Monsieur[f], C.M. van Oirschot[g], B.R.H. Van den Bergh[a,c,h,*]

[a] Department of Psychology, Tilburg University, The Netherlands
[b] REVAL Rehabilitation Research Center, Biomedical Research Institute, Faculty of Medicine and Life Sciences, Hasselt University, Belgium
[c] Department of Psychology, KU Leuven, Belgium
[d] Institute of Cardiovascular Science, University College London, UK
[e] Department of Electrical Engineering, ESAT-STADIUS Center for Dynamical Systems, Signal Processing and Data Analytics, KU Leuven, Medical IT, iMinds, Belgium
[f] Fontys University of Applied Sciences, The Netherlands
[g] Sint Elisabeth Hospital Tilburg, The Netherlands
[h] Department of Welfare, Public Health and Family, Flemish Government, Brussels, Belgium

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ABSTRACT

Altered stress responsiveness is a risk factor for mental and physical illness. In non-pregnant populations, it is well-known that anxiety can alter the physiological regulation of stress reactivity. Characterization of corresponding risks for pregnant women and their offspring requires greater understanding of how stress reactivity and recovery are influenced by pregnancy and women's anxiety feelings. In the current study, women were presented repeatedly with mental arithmetic stress tasks in the first and third pregnancy trimester and reported their trait anxiety using the state trait anxiety inventory. Cardiovascular stress reactivity in late pregnancy was lower than reactivity in the first pregnancy trimester (heart rate (HR): $t(197) = 4.98$, $p < .001$; high frequency heart rate variability (HF HRV): $t(196) = -2.09$, $p = .04$). Less attenuation of stress reactivity occurred in more anxious women (HR: $b = 0.15$, SE = 0.06, $p = .008$; HF HRV: $b = -10.97$, SE = 4.79, $p = .02$). The study design did not allow the influence of habituation to repeated stress task exposure to be assessed separately from the influence of pregnancy progression. Although this is a limitation, the clear differences between anxious and non-anxious pregnant women are important, regardless of the extent to which differing habituation between the groups is responsible. Less dampened stress reactivity through pregnancy may pose long-term risks for anxious women and their offspring. Follow-up studies are required to determine these risks.

1. Introduction

Pregnant women undergo marked changes in maternal cardiovascular function during pregnancy, such as increased basal stroke volume (SV) and heart rate (HR) (Abbas, Lester, & Connolly, 2005; Silversides & Colman, 2007). The autonomic nervous system (ANS) plays a central role in these changes. Basal ANS activity is shifted towards higher sympathetic (e.g., shorter pre-ejection period (PEP) and increased skin conductance level (SCL)) and lower vagal modulation (e.g., reduced HR variability (HRV)) over the course of pregnancy (DiPietro, Costigan, & Gurewitsch, 2005; Ekholm & Erkkola, 1996; Kuo, Chen, Yang, Lo, & Tsai, 2000).

These changes go along with attenuated cardiovascular responses to stress, as HR and blood pressure (BP) reactivity are typically attenuated as pregnancy progresses (DiPietro, Costigan, & Gurewitsch, 2003; Entringer et al., 2010; Matthews & Rodin, 1992). Studies of ANS stress responsiveness during pregnancy are rare. Evidence of declining SCL stress reactivity between 24 and 36 weeks gestation is an important finding (DiPietro et al., 2003) and a report of decreased HRV responsiveness with advancing pregnancy is valuable but this finding had marginal statistical significance (Klinkenberg et al., 2009). No studies exist examining PEP reactivity to laboratory stressors during pregnancy. Moreover, studies of stress reactivity in pregnancy have generally examined the magni-

* Corresponding author at: Faculty of Social and Behavioural Sciences, Tilburg University, Warandelaan 2, PO Box 90153, 5000 LE Tilburg, The Netherlands. Fax: +31 13 466 2067.
E-mail address: Bea.vdnBergh@uvt.nl (B.R.H. Van den Bergh).

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tude of physiological change following an acute stressor and have generally not reported on how pregnant women recovered from stress (Christian, 2012; de Weerth & Buitelaar, 2005).

If stress reactivity attenuates during pregnancy, as suggested by these early studies, it raises the question of why this might occur. One possibility is that high stress reactivity in pregnancy could be detrimental to the mother, the child, or both. Therefore, a protective mechanism may be operating to limit stress-induced changes in the hormonal, cardiovascular and metabolic environments of the pregnancy that could be harmful. For example, previously it has been demonstrated that stress is associated with vasoconstriction, which can alter uteroplacental blood flow, reducing oxygen and nutrition delivery with potentially negative effects on fetal growth (Alder, Fink, Bitzer, Hösli, & Holzgreve, 2007; Copper et al., 1996; McCubbin et al., 1996) and nervous system development (Sjöström, Valentin, Thelin, & Marsál, 1997). However, more recent studies could not replicate the association between maternal anxiety and reduced uterine blood flow (Mendelson, DiPietro, Costigan, Chen, & Henderson, 2011; Monk et al., 2012).

In men and non-pregnant women, stress reactivity differs according to level of anxiety. High anxiety is associated with exaggerated cardiovascular (HR and BP) stress responses (Gramer & Saria, 2007; Pointer et al., 2012). There is some evidence that depression and anxiety affect stress responsiveness during pregnancy but this has not been studied extensively (Christian, 2012; de Weerth & Buitelaar, 2005). Findings have also been inconsistent, suggesting that anxious pregnant women may have lower (Monk et al., 2000; Saisto, Kaaja, Helske, Ylikorkala, & Halmesmäki, 2004) or unaltered (Monk, Myers, Sloan, Ellman, & Fifer, 2003) HR and BP reactivity to psychological stress. Our study was designed to address this lack of evidence and to assess both reactivity to and recovery from stress.

Our aims were: (a) to characterize typical *autonomic* stress responsiveness (i.e., reactivity and recovery) through different pregnancy trimesters and (b) to test the hypothesis that anxiety may alter stress responsiveness during pregnancy.

2. Methods

2.1. Participants

The prenatal early life stress (PELS) study is a longitudinal study and focuses on associations between prenatal stress risk factors, birth outcomes and altered pregnant women's physiology and child's psychophysiology and neurodevelopment. The national ethics committee and the ethics committee of the Sint Elisabeth hospital, Tilburg, The Netherlands both approved the study protocol. Pregnant women were recruited from a hospital and midwiferies around Tilburg. They filled out questionnaires about their emotions and their ECG was recorded during each pregnancy trimester. After birth, psychophysiological measurements of the children and mothers took place. All participants and their partners provided written informed consent. None of the participants smoked, drank alcohol, were under treatment for a current mental disorder, or used cardiovascular medications or antidepressants.

One hundred and seventy women completed stress tasks during the first (8–14th week of gestation, $N = 133$) and/or third pregnancy trimester (31–37th pregnancy week, $N = 138$) and 157 of these 170 participants filled out a standardized anxiety self-report questionnaire at 15–22 weeks of pregnancy.

2.2. Material

2.2.1. Relaxation and stress tasks

In the first and third pregnancy trimesters, each pregnant woman undertook a 25-min task consisting of five testing phases, lasting for 5 min each. Stress was induced in the second and fourth phase, with the remainder being relaxation phases. Participants viewed peaceful pictures and listened to restful music during the relaxation phases (Taelman, Vandeput, Vlemincx, Spaepen, & Van Huffel, 2011). During the stress phases, participants were asked to solve complex mental arithmetic problems, involving five mathematical operations on 2–3 digit numbers without verbalization (e.g., 361 + 17/24 × 2 + 13). They were asked to choose the correct answer from three possibilities presented by a computer. Feedback on the task was given after completion of the last phase (Vlemincx, Taelman, De Peuter, Van Diest, & Van den Bergh, 2011).

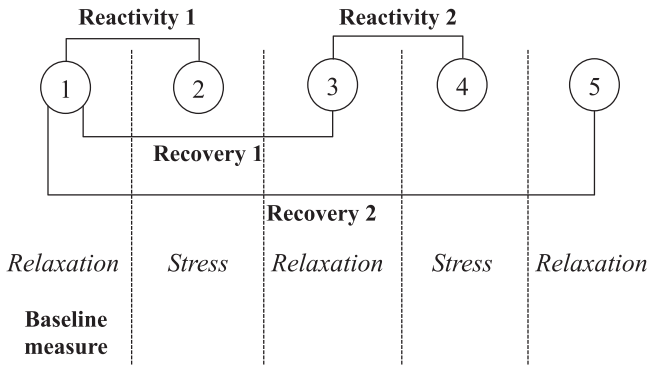

Fig. 1. Diagram showing how the reactivity and recovery measures were calculated from the HR/HRV data collected during the mental arithmetic stress task. As indicated in the figure, the measurement in the first phase was use as a baseline measure.

2.2.2. ECG and ICG recording

Maternal electrocardiography (ECG) and impedance cardiography (ICG) was recorded with the Vrije Universiteit Ambulatory Monitoring system (VU-AMS) during the stress task using seven Ag/AgCl electrodes placed according to the VU-AMS configuration guidelines (Goedhart, Sluis, Houtveen, Willemsen, & Geus, 2007). The skin was cleaned with alcohol to keep electrode resistance low. Cardiovascular measurements were determined for the three relaxation phases and two stress phases, each lasting 5 min. The first relaxation phase was used as baseline measure. Cardiovascular *reactivity* to the stress phase was calculated by subtracting the cardiovascular measurements of each of the stress phases with the level of the previous relaxation phase. Hence, we explicitly choose to work with two reactivity measures, one derived from the difference between phase 1 and 2 (i.e., reactivity to first stressor presentation), and one between phase 3 and 4 (i.e., reactivity to second stressor presentation). Cardiovascular *recovery* from a stressor presentation was derived from the subtraction between cardiovascular measurements in the relaxation phase that follows the stressor presentation and baseline cardiovascular measurements (i.e., in the first relaxation phase) (Stewart & France, 2001). Fig. 1 shows a graphical representation of the calculation of the reactivity and recovery measures.

2.2.3. The state trait anxiety inventory (STAI)

In the second pregnancy trimester the participants filled out the questions constituting the trait anxiety subscale of the Dutch, psychometrically validated version of the state trait anxiety inventory (STAI) (Van der Ploeg, Defares, & Spielberger, 1980). The trait subscale has been identified recently as the best instrument to assess general maternal anxiety during pregnancy (Nast, Bolten, Meinlschmidt, & Hellhammer, 2013). Trait anxiety refers to differences in anxiety proneness and is seen as a personality trait. This subscale contains 20 items scored from 1 to 4 and has a reliability coefficient (Cronbach's alpha) of 0.75 in our sample.