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Van den Bergh, B.R.H.

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ABSTRACTS

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STRESS IN PREGNANCY: ANXIETY SYMPTOMS IN CHILDHOOD AND HPA FUNCTION AND DEPRESSIVE SYMPTOMS IN ADOLESCENCE

Van den Bergh B.R.H.
University of Leuven, Dept. of Psychology, Leuven, Belgium; Tilburg University, Dept. of Developmental, Clinical and Cross-cultural Psychology, Tilburg, The Netherlands

In a prospective-longitudinal study maternal anxiety was measured at 12–22, 23–32 and 32–40 weeks of pregnancy, with the State Trait Anxiety Inventory. 72 firstborns of 86 mothers, recruited at the Obstetrical and Gynecological Board consultations of a University Hospital were followed up to age 8–9, and 58 up to age 14–15. At the age of 8–9 hierarchical multiple regression analyses showed that maternal state anxiety during pregnancy explained 9% of the variance in self-reported anxiety, as measured with the State Trait Anxiety Inventory for Children. Effects remained when controlling for child's gender, parents' educational level, smoking during pregnancy, birth weight and postnatal maternal anxiety. At the age of 14–15, HPA-axis function was measured through establishing a saliva day-time cortisol profile (shortened version); during a week-end day 3 saliva samples were provided, namely immediately after awakening, at noon and in the evening. Severity of depressive symptoms was measured with the Children's Depression Inventory. We tested the following two hypotheses: (a) that maternal anxiety during pregnancy predicts altered HPA axis function (e.g., a flattened cortisol profile) in the adolescent offspring; (b) that altered HPA axis function mediates the association between prenatal maternal anxiety and depressive symptoms in these adolescents. Repeated measurements regression analysis and ordinary least-squares regression analyses indicated that maternal anxiety at 12–22 weeks of pregnancy was in female and male offspring associated with a diurnal cortisol profile that was attenuated due to elevated cortisol secretion in the evening. Moreover, in female adolescents this flattened cortisol curve was associated with depressive symptoms. Effects remained when controlling for covariates such as smoking during pregnancy, birth weight, obstetric risk, postnatal maternal anxiety and Tanner puberty phase. Our results indicate that maternal anxiety during pregnancy enhances neurobiological vulnerability to depressive symptoms, possibly by altering (or 'programming') foetal physiology. They also demonstrate the mediating role of HPA axis dysregulation in linking an adverse foetal environment to depressed mood.

If our results can be replicated in future research they may lead to a re-orientation of the target of primary prevention and treatment of anxiety symptoms in childhood and depressive symptoms in adolescence.