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HPA-AXIS FUNCTION AND DEPRESSION IN ADOLESCENCE: ARE THEY RELATED TO PRENATAL EARLY LIFE STRESS?

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Early-life exposure to adverse environmental cues during critical windows of time in the prenatal and/or early postnatal life period could predispose the individual for somatic andmental diseases. This especially holds for stress-related disorders such as depression in which HPA-axis dysregulation plays a pathophysiological role. This is in line with the 'fetal (or developmental) programming-hypothesis' which has been tested in numerous preclinical experimental. We tested this hypothesis in humans in a prospective longitudinal study in which maternal emotional state was measured during each pregnancy trimester and after pregnancy. When the offspring was 14-15 years old, HPA-axis function was measured through establishing a saliva day-time cortisol profile. Severity of depressive symptoms was measured with the Children's Depression Inventory. 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. Our results indicate that maternal anxiety during pregnancy enhances neurobiological vulnerability to depressive symptoms, possibly by altering (or 'programming') foetal physiology. 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 depressive symptoms. Preliminary results of a study on the association between prenatal exposure to maternal anxiety and cortisol stress responsivity during inoculation in the four month old will be presented.

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