Childhood trauma and cortisol levels in pregnancy

R.J. Wright (Harvard Medical School and Harvard School of Public Health, Boston, MA), S. Franco Suglia (Harvard School of Public Health, Boston, MA), J.W. Staudenmayer (University of Massachusetts, Amherst, MA), and S. Cohen (Carnegie Mellon University, Pittsburgh, PA), USA

Asthma risk is conceptually linked to neuroendocrine disruption related to stress. Prenatal stress may have long-lasting programming effects on the development of children mediated through altered activity of the maternal hypothalamic-pituitary-adrenocortical (HPA) axis. Adult HPA function may be altered by early trauma. We examined the relationship between retrospective maternal reports on the Childhood Trauma Questionnaire (CTQ) and diurnal salivary cortisol patterns among 187 pregnant mothers enrolled in the Asthma Coalition on Community, Environment, and Social Stress (ACCESS) project, a prospective study designed to examine the effects of early life risk factors on childhood asthma risk. Current psychological symptoms were collected using the PTSD Checklist – Civilian version (PCL-C) and the Edinburgh Depression Scale (EDS). Salivary cortisol was collected five times per day for three days to assess basal awakening response, morning rise, diurnal rhythm, and area under the curve. Repeated measures mixed models were performed controlling for race, income, smoking status, weeks pregnant at time of cortisol sampling, and current psychological symptoms. Higher scores on the overall CTQ ($\beta=-0.007$, $p=0.11$) and emotional abuse CTQ subscales ($\beta=-0.004$, $p<0.01$) and being above the median on this PCL-C ($\beta=-0.01$, $p<0.01$) predicted decreased basal cortisol. The CTQ physical, emotional and sexual abuse subscales were also consistently related to a reduced morning rise and a flatter diurnal slope ($p<0.10$). HPA axis functioning in these pregnant women is altered by early traumatic experiences. These data may have implications for physiologic programming effects and subsequent asthma risk in these children. Funding: R01 HL080674, T32ES007142