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Prenatal Programming of Temperament Elysia Poggi Davis[1], Dawn A. Korsen[1], Laura M. Glynn[1], Chris Dunkel Schetter[2], Calvin Hobel[3], Aleksandra Chicz-Demet[1] & Curt A. Sandman[1]; *[1]Department of Psychiatry & Human Behavior, University of California, Irvine, 92868 [2]Department of Psychology, University of California, Los Angeles, [3]Maternal Fetal Medicine, Cedars Sinai, Los Angeles, USA*

A significant proportion of variation in infant and adult health outcomes and disease risk is attributable to developmental processes during fetal life in response to a variety of social, psychological, physiological influences. Although a large and impressive body of literature supports this notion of fetal or developmental origins of health and disease, the major limitations in this field are that an overwhelming majority of these studies have (a) employed a retrospective design, and (b) used measures of birth phenotype (e.g. birth weight/size, length of gestation) as predictors of subsequent health outcomes. Thus, these studies are unable to ascertain the nature of the intrauterine milieu during fetal development, and it is unlikely that birth phenotype, *by itself,* plays a causal role in this relation. Intrauterine exposure to glucocorticoids (GCs) is one mechanism that may mediate these effects on the fetus. We examined the consequences of prenatal exposure to GCs for the development of behaviorally inhibited temperament, a risk factor for the development of social anxiety disorders. Although there is abundant evidence from animal models that increased prenatal GC exposure results in amplified behavioral inhibition and reduced coping in aversive situations later in life, there are few human studies examining the consequences of prenatal GC exposure. There is, however, evidence that prenatal maternal anxiety and prenatal exposure to synthetic GCs has consequences for fearful or behaviorally inhibited temperament. **Study 1:** Maternal salivary cortisol levels were measured longitudinally at four time points during pregnancy (14, 19, 25 and 33 weeks of gestation). Infant behavior was assessed at two months of age in 121 full term infants (58 male, 63 female) with subscales of the Infant Behavior Questionnaire (Gartstein & Rothbart, 2003). As expected, maternal cortisol levels increased from early to late pregnancy, $F(3,118) = 39.7$, $p < .05$, ? = .38. Interestingly, women with a steeper rise in cortisol had infants who displayed more fear behaviors at two months of age, after controlling for postnatal maternal psychological state (partial $r(121) = .22$, $p < .05$). **Study 2** Using the Children's Behavior Questionnaire (Ahadi, Rothbart, & Ye, 1993), we additionally assessed 28 of the children (12 male, 16 female), from the sample described above, when they were five years of age. Higher maternal cortisol during the early second trimester, but not later in pregnancy, was significantly related to fearful/behaviorally inhibited temperament (partial $r(27) = .52$, $p < .05$) after controlling for maternal psychological state at the time of child assessment. **Discussion** Elevated maternal cortisol during pregnancy has consequences for behavioral inhibition in infancy and childhood, with earlier exposures exerting a stronger influence. These data suggest that prenatal GC exposure has persisting consequences for development.