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PRENATAL ORIGINS OF OBESITY RISK

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Obesity affects nearly 18% of children and adolescents in the United States. There is increasing evidence that prenatal maternal stress signals influence fetal growth, child obesity, and metabolic risk. Catch-up growth, a rapid and dramatic increase in body size within the first two years of life, is an early predictor of poor health outcomes, including obesity. In the studies presented here, we evaluate the role of prenatal stress hormones in the programming of infant growth and obesity risk. The current prospective, longitudinal cohort provides a unique opportunity to evaluate the span from fetal to infant developmental periods and will provide new insight into the origins of obesity. Healthy term-born individuals (n=246; 120 girls, 126 boys) and their mothers were followed from early gestation through 2 years. Maternal hypothalamic-pituitary-adrenal (HPA) and placental axis hormones, including cortisol and placental corticotropin-releasing hormone (CRH), were evaluated at 5 gestational intervals. Child body size was evaluated at birth, 3, 6, 12 and 24 months. Associations between prenatal stress hormones and postnatal growth patterns were examines. In the first study, infants with the highest placental CRH exposure during the third trimester exhibited low birth weight followed by a rapid increase in BMI (catch-up growth) across infancy. For the second study, three distinct prenatal trajectories of plasma cortisol were identified. Women exhibiting an atypical plasma cortisol profile characterized by chronically elevated cortisol levels with a minimal increase across pregnancy, had infants with an exaggerated increase in BMI across the first 6 months. These findings provide evidence that dysregulated patterns of prenatal maternal cortisol and elevated placental CRH exposure predict growth patterns in infancy through childhood, independent of postnatal factors. Thus, we propose that placental CRH and maternal cortisol exposure during fetal development are involved in prenatal programming of obesity risk.