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Adipokines measured prenatally and at birth are associated with infant negative affect

Kelly R. Molloy, Hanna C. Gustafsson, Anna S. Young, Jennifer C. Ablow, Joel T. Nigg, Elinor L. Sullivan

OHSU Department of Psychiatry

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Abstract

Increasing evidence suggests that greater maternal adiposity during pregnancy is related to offspring risk for mental health disorders (Rivera, Christiansen, & Sullivan, 2015), although the mechanisms through which these effects are conveyed remain poorly understood. One hypothesis is that alterations in maternal concentrations of adipokines such as leptin and adiponectin may explain this association (Valleau & Sullivan, 2014). Leptin and adiponectin influence the *in-utero* environment and glucoregulation in the parent and child, which has implications for fetal development and infant psychopathology.

The goal of the current study was to explore the mechanisms through which prenatal adipokines (leptin and adiponectin) influence the relationship between prenatal adiposity and infant negative affect.

Data (N=310) were collected from a cohort of pregnant individuals and their children, followed from the 2nd trimester of pregnancy through 6 months postpartum. Second trimester adiposity (percent body fat) was assessed using air displacement plethysmography (BOD POD). Concentrations of adiponectin and leptin were assessed in maternal plasma at 24-26 weeks (Yalinbas, Binay, Simsek, & Aksit, 2019) gestation and in umbilical cord plasma. Infant temperament was assessed through behavioral coding of the still face paradigm (Adamson & Frick, 2003) and arm restraint task (Planalp, Van Hulle, Gagne, & Goldsmith, 2017) at 6 months postpartum.

As expected, maternal 2nd trimester adiposity was associated with greater leptin ($\beta=.61, p<.001$) and with lower adiponectin ($\beta=-.40, p<.001$) during the 2nd trimester. Decreased 2T adiponectin predicted greater infant NA during both behavioral tasks (β range=-19-.27, $ps<.05$). Also, cord

blood leptin was associated with increased infant NA during the still face paradigm only. Prenatal leptin and cord blood adiponectin were not significantly associated with infant NA.