Parental Social Disparities in Epigenetic Regulation of Newborns' Imprinted Genes Related to Gestational Growth and Later-Life Health

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Abstract

Children whose parents have lower income and education are at risk for obesity and later-life health risks. Explanations of enduring associations of early-life socioeconomic status (SES) with adult health have focused on stress and behavioral factors, but the biological mechanisms linking these are poorly understood. DNA methylation is the most studied epigenetic mechanism in epidemiologic studies, but social differences in methylation of particular genes are understudied, particularly at birth. We focus on a cluster of particularly well-studied genes in which methylation is linked to gestational growth and increases childhood obesity risk. In adults methylation of these genes predicts multiple health problems. We measured methylation at differentially methylated regions (DMRs) regulating genomically-imprinted genes (*IGF2, H19, DLK1, MEG3, PEG1/MEST, PEG3, PEG10/SGCE, NNAT*, and *PLAGL1*) using umbilical cord blood from 619 infants in Durham, North Carolina in 2010-2011. We examined disparities in DMR methylation levels by race/ethnicity of both parents, and the role that maternal SES may play in explaining race/ethnic epigenetic differences. Unadjusted race/ethnic differences were evident only at the *IGF2, H19, MEG3,* and *NNAT* DMRs, such as. a 0.83SD (p<0.001) difference in *IGF2* methylation between Black and White fathers. Race/ethnicity of mothers vs. fathers acted in opposite directions for *NNAT*. With SES adjustment, race/ethnic differences persisted for the *IGF2* and *NNAT* DMRs. Results suggest social factors may not only influence DNA methylation, but do so in ways that vary by DMR. These findings support the hypothesis

that epigenetics is one path through which prenatal social conditions may explain disparities in phenotypic outcomes in ways which potentially extend across the life course.