NAME OF PRESENTING AUTHOR: Olivia C. Robertson

EMAIL ADDRESS OF PRESENTING AUTHOR: rober511@purdue.edu

# Pubertal timing, child body mass index and the obesogenic environment

Olivia C. Robertson[1], Kristine Marceau[1], Misaki N. Natsuaki[2], Daniel S. Shaw[3], David Reiss[4], Leslie D. Leve[5], Jenae M. Neiderhiser[6], Jody M. Ganiban[7]

[1]Department of Human Development and Family Studies, Purdue University

[2]Department of Psychology, University of California, Riverside

[3]Department of Psychology, University of Pittsburgh

[4]Child Study Center, Yale University

[5]Prevention Science Institute, University of Oregon

[6]Department of Psychology, Penn State University

[7]Department of Psychology, George Washington University

KEYWORDS: Pubertal timing, Gene by Environment Interaction, Childhood Obesity, Obesogenic environment

ABSTRACT:

The obesogenic family environment is considered a key contextual factor for child body mass index (BMI), and elevated child BMI is a strong predictor for early pubertal timing. As shared genetic influences and metabolic processes underlie the development of obesity and pubertal timing, and may be influenced by obesogenic environmental factors, we expect that family obesogenic environments may exacerbate heritable risk on pubertal timing. Using cohort I (*n* = 361) of the Early Growth and Development Study, a US-based sample of children adopted into non-relative families at birth, we examined whether the obesogenic family environment moderated the effect of heritable risk (birth parent BMI) for earlier puberty in 11-year-old boys and girls in a two-step linear regression framework – first adjusting for covariates and second, including childhood BMI as an additional predictor. Obesogenic family environment was quantified by latent profiles derived from indicators of adoptive parent characteristics, feeding behavior, and parenting: average (*n*=276), weight concerned (*n*=52), and low control (*n*=31). Greater genetic risk and child BMI were associated with earlier pubertal timing in boys, but there were no interactions. For girls, there was an interaction (*p* = .045) such that among girls in the weight concerned (distinguished by above average parent concern for child's weight) profile, heritable risk predicted later pubertal timing. Whereas for girls in the average profile, there was no association. The interaction effect was attenuated by childhood BMI. Results suggested that the limited role of G x obesogenic Environment on pubertal timing operates via childhood BMI.

GRANT SUPPORT: The funding for this study was provided by grant R01 HD042608 from the Eunice Kennedy Shriver National Institute of Child Health & Human Development and the National Institute on Drug Abuse, NIH, U.S. PHS (PI Years 1–5: David Reiss, MD; PI Years 6–10: Leslie Leve, Ph.D.), R01 DA020585 from the National Institute on Drug Abuse, the National Institute of Mental Health and OBSSR, NIH, U.S. PHS (PI: Jenae Neiderhiser, Ph.D.), R01 MH092118 from the National Institute of Mental Health, NIH, U.S. PHS (PIs: Jenae Neiderhiser, Ph.D. and Leslie Leve, Ph.D.), R01 DK090264 from the National Institute of Diabetes and Digestive and Kidney Disease, NIH, U.S. PHS (PI: Jody Ganiban, Ph.D.), R56 HD042608 from the Eunice Kennedy Shriver National Institute of Child Health & Human Development, NIH, U.S. PHS (PI: Leslie Leve, Ph.D.), R01 DA045108 (PI: Jenae Neiderhiser, Ph.D.), UH3OD023389 (PIs: Leslie Leve, Ph.D., Jenae Neiderhiser Ph.D., Jody Ganiban Ph.D.) and K01 DA039288 (PI: Kristine Marceau, Ph.D.).