

Lifecourse Epidemiology of Adiposity & Diabetes (LEAD) Center

colorado school of public health



Trainee Profile: Christy Hockett, PhD (2017)

Dr. Hockett completed her PhD in May at the Colorado School of Public Health using data from the EPOCH cohort. Her thesis explored the role of maternal diabetes on the timing of offspring puberty. She estimated pubertal timing, defined by age at peak height velocity (APHV), and speed of pubertal growth, defined by peak height velocity (PHV), for each child using multiple height measurements from clinical records. She found that median APHV was reached ~3 months earlier in youth exposed to maternal diabetes compared to unexposed youth.

Exposed females had 10% greater median PHV compared to unexposed females and exposed males had a 3.5% greater median PHV compared to unexposed males (Figure). She also explored whether exposure to maternal diabetes alters hormones of puberty, specifically estradiol (E), testosterone (TT), dehydroepiandrosterone sulfate (DHEA-S), and luteinizing hormone (LH). She found that exposure to maternal diabetes was associated with increased concentrations of luteinizing hormone in exposed females, but not in males, and that exposure to maternal diabetes did not influence estradiol, total testosterone or DHEA-S concentrations.

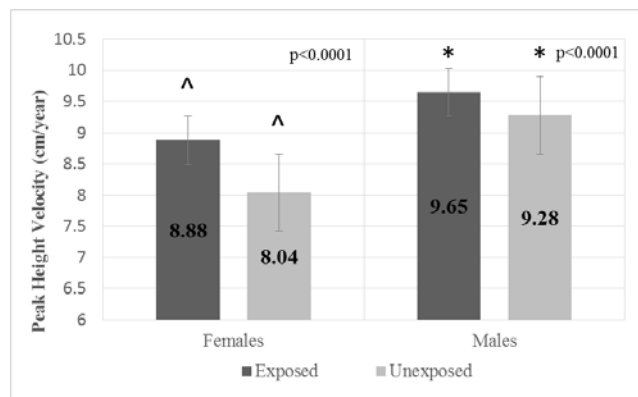


Figure. The sex difference in the association of exposure to diabetes in utero with offspring peak height velocity.

Finally, her findings suggest that the association between exposure to maternal diabetes and offspring adiposity is established early in life, likely before puberty, and tracks throughout puberty, with adiposity being both a possible driver and consequence of earlier pubertal timing.

These findings are important to public health as they suggest a need for obesity and lifestyle interventions among young adults, specifically women of childbearing age, which may help disrupt the intergenerational cycle of obesity.



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