

Boone-Heinonen Janne (Orcid ID: 0000-0002-0368-0545)

Weeks Heidi (Orcid ID: 0000-0003-3467-7987)

Bauer Katherine (Orcid ID: 0000-0003-3512-3994)

## ABSTRACT

**Background:** Prenatal nutrition impacts offspring appetite regulation in animal models.

However, evidence from humans is scarce.

**Objective:** To determine associations between indicators of prenatal nutrition and appetite regulation among young children.

**Methods:** Participants included 454 low-income mother/child dyads (mean child age = 45.2 months (SD=9.7)). Children's appetite regulation was ascertained with the maternal-reported Child Eating Behavior Questionnaire and objectively assessed using the eating in the absence of hunger protocol. Using hierarchical linear regression, we modeled child appetite regulation measures as a function of prenatal nutrition indicators (child birth weight z-scores [BWz, BWz<sup>2</sup>]; maternal pre-pregnancy body mass index [BMI], gestational weight gain [GWG]), adjusted for sociodemographic characteristics.

**Results:** Among girls, higher and lower birth weight were associated with greater energy consumed in the absence of hunger, primarily sweet foods [coeff (95% CI): BWz 0.17 (0.05, 0.28), BWz<sup>2</sup> 0.15 (0.04, 0.26)], but not food responsiveness or food enjoyment. Higher birth weight was also associated with greater satiety responsiveness among girls. Among boys, birth weight was unrelated to measures of appetite regulation. Associations between maternal BMI and GWG and child appetite regulation were inconsistent.

**Conclusions:** Among low-income girls, but not boys, indicators of adverse prenatal conditions were associated with poor objectively-measured appetite regulation during early childhood.

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## INTRODUCTION

In the United States, the prevalence of childhood obesity remains high with no signs of decline: 26% of children have obesity and 7.9% have severe obesity.<sup>1</sup> In the current food environment, individuals with a poorer ability to self-regulate energy intake are vulnerable to overconsumption, particularly of highly-palatable, energy dense foods, resulting in excess weight gain.<sup>2</sup> Therefore, identifying modifiable factors that disrupt appetite regulation is a public health priority. Growing evidence suggests that exposures during the prenatal period, such as maternal diet, may influence the development of neuronal and hormonal factors involved in offspring appetite regulation.<sup>3,4</sup> Poor appetite regulation therefore may be a critical mechanism underlying developmentally-induced predisposition to obesity.<sup>5</sup> However, human evidence is limited.

In animal models, both fetal undernutrition and overnutrition induces elevated appetite, preferences for foods high in sugar and fat, and subsequent obesity.<sup>6</sup> Mechanisms underlying effects on appetite regulation include alterations to the hypothalamic appetite-regulating network, nutrient sensing, and neuronal development; and leptin and insulin signaling.<sup>7</sup> Human evidence that prenatal exposures impact appetite regulation is critical for understanding these processes in the context of environmental and genetic variability that exists in human populations,<sup>8,9</sup> but is, to date, limited and focused on fetal undernutrition. Measures of appetite regulation can be broadly classified as *food avoidant* behaviors such as picky eating or food refusal, and *food approach* behaviors such as low satiety response, which are associated with later obesity.<sup>10</sup> In studies using parent-reported child eating behaviors, low birth weight [LBW]

or intrauterine growth restriction [IUGR] is associated with the greater eating in response to external food cues, strength of attempts to restrain eating,<sup>11</sup> and food avoidant behaviors.<sup>12,13</sup> Among the even smaller number of studies to examine objectively-measured appetite regulation, young children born with IUGR (compared to normal birth size) have demonstrated greater impulsive eating ascertained with a snack delay test.<sup>14</sup> Similarly, adolescents born with lower birth weight experience elevated neural activation in response to viewing palatable foods.<sup>11</sup> In summary, human evidence is consistent with mechanistic understanding of prenatal influences on neurocircuitry that drive appetite regulation, but remains small, focused on children born with low birth weight, based outside of the U.S., and largely based on parent-reported eating behaviors.

Meanwhile, while maternal obesity is consistently associated with up to a three-fold greater risk of obesity in the next generation,<sup>15</sup> evidence for the influence of fetal overnutrition on appetite regulation among human offspring is scant. The few studies that examine higher birth weight in relation to child appetite regulation focus on food avoidant behaviors,<sup>12,13</sup> as opposed to food approach behaviors, despite the strong linkage between food approach behaviors and high risk for obesity. Children born large for gestational age tend to be less likely to be perceived by parents as poor eaters (eating small or insufficient quantity)<sup>12</sup> and less likely to exhibit persistent picky eating,<sup>13</sup> but otherwise show no clear patterns in differences in food avoidant behaviors.<sup>12</sup> In one recent U.S. study, girls, but not boys, exposed prenatally to impaired maternal glucose tolerance exhibited elevated eating in the absence of hunger in early

adolescence,<sup>16</sup> the manifestation of strong food approach tendencies, but when such changes emerge in childhood have not been studied.

Knowledge of the impact of both fetal under- and over-nutrition on appetite regulation offers the potential to identify intervention targets for the primary and secondary prevention of childhood obesity and later development of cardiometabolic disease.<sup>17</sup> Therefore, the objective of this study was to test the hypothesis that indicators of fetal under- and over-nutrition (higher or lower birth weight, higher maternal pre-pregnancy body mass index [BMI], excessive gestational weight gain [GWG]) are associated with objectively-measured and parent-reported food approach behaviors among a sample of low-income young children.

## **METHODS**

Data were obtained from two cohorts of low-income parent/child dyads recruited from south-central Michigan: ABC Preschool and ABC Toddler. In ABC Preschool, parents and their 3 to 5-year-old children were recruited between 2009 and 2011 based on their participation in Head Start, the federally-supported program that provides preschool to children with household incomes at or below the federal poverty line. To be eligible for ABC Preschool, parents had to be fluent in English and have less than a four-year college degree. Children who were <35 weeks gestational age at birth; had significant perinatal or neonatal complications, serious medical problems or food allergies; or in foster care were excluded from recruitment into the ABC studies.

ABC Toddler aimed to enroll a demographically similar cohort of low-income parent/child dyads as ABC Preschool. ABC Toddler recruited biological mothers and their toddler-aged (21-27 months) children between 2011 and 2014 through flyers posted in community agencies serving low-income families. With the exception of only recruiting children's biological mothers, inclusion and exclusion criteria for ABC Toddler and ABC Preschool were identical. Data from the toddlers' 33 month visit were used in the current study.

For the current study, dyads in the ABC studies were excluded if the parent participating was not the child's biological mother (n=27) or were missing data on parental relationship (N=22), the child's gestational age at birth was  $\leq 37$  weeks or missing (N=55), or the dyad was missing all measures of eating behavior (N=61) or all measures of perinatal exposures (N=5). Preterm births ( $\leq 37$  weeks) were excluded from this analysis to minimize the influence of feeding difficulties resulting from prematurity.<sup>18,19</sup> The final study sample included 454 mother/child dyads, 307 from ABC Preschool and 147 from ABC Toddler. This represents 72.8% of the total 624 dyads from these studies. All mothers provided written informed consent and the University of Michigan Institutional Review Board approved the studies.

## **Measures**

Measures were conducted with mother/child dyads during study visits conducted by trained research staff either in community settings or the dyads' homes.

**Prenatal characteristics.** Mothers reported their child's due date, birthdate, and birthweight.

The child's gestational age at birth was calculated by comparing their due date to birthdate. Child birthweight z-scores (BWz) accounting for gestational age were calculated using nationally-representative reference values identified by Oken, et al;<sup>20</sup> then, for descriptive purposes, classified as small (SGA, <10<sup>th</sup> percentile), appropriate (AGA), and large (LGA; >90<sup>th</sup> percentile). Mothers self-reported their pre-pregnancy weight, shown in other studies to be reliable 2-5 years after birth.<sup>21</sup> Mothers' height was either measured using standardized protocols by trained research assistants during the study visit (N=487) or self-reported (N=4). Pre-pregnancy BMI was then calculated from these measures. Mothers also reported their GWG, shown in prior work to correlate ( $r=0.63$ ) with documented GWG.<sup>22</sup> GWG was categorized as insufficient, adequate, and excessive based on the 2009 Institute of Medicine's pre-pregnancy weight status-specific guidelines: underweight (28-40 pounds), normal weight (25-35 pounds), overweight (15-25), obesity (11-20).<sup>23</sup>

**Child appetite regulation measures.** Appetite regulation was ascertained via mother-report on the Children's Eating Behavior Questionnaire (CEBQ) and objectively assessed by children's measured intake during the Eating in the Absence of Hunger (EAH) protocol, a measure of appetite regulation that reflects children's consumption of highly-palatable snack foods after a satiating meal. The CEBQ is a 35-item instrument consisting of eight subscales of children's eating behaviors; for all subscales, mothers rate their child's eating behaviors on five-point

Likert scales ranging from “Never” (1) to “Always” (5); higher scores indicate greater endorsement of the eating behavior.<sup>24</sup> Three CEBQ subscales reflecting food approach behaviors were included in the current study based on their previous consistent associations with child obesity risk: enjoyment of food, which assesses a mother’s perception of her child’s desire to eat and enjoyment of food (4 items, Cronbach’s  $\alpha = 0.80$ ); food responsiveness (eating in response to environmental food cues; 5 items, Cronbach’s  $\alpha = 0.78$ ); and satiety responsiveness (attention to internal cues of fullness; 5 items, Cronbach’s  $\alpha = 0.73$ ).<sup>25,26</sup>

The EAH protocols used with children in ABC Preschool and Toddler cohorts have been described in detail previously.<sup>27,28</sup> In ABC Preschool, children’s EAH was measured immediately after breakfast at Head Start. Following breakfast, the research assistant determined the child’s satiation using cartoon figures, and proceeded with engaging the child in the EAH task only if the child reported that they were not hungry. The research assistant then invited the child to a separate room and for 10 minutes gave them free access to toys and pre-measured bowls of Trix® cereal (32 grams, 120 calories), mini Chips Ahoy® cookies (90g, 435 kcal), fruit snacks (164g, 631 kcal), Cheez-Its® (60g, 300 kcal), pretzel sticks (36g, 129 kcal) and Chicken in a Biskit® crackers (60g, 310 kcal). Children were told, “You can play with any of the toys and eat any of the foods on this table. I’m going to do some work.” After 10 minutes, the food remaining was weighed to determine the amount of each food children consumed and the corresponding energy intake. Higher energy consumption represents a higher degree of EAH.



Minor modifications to the EAH protocol was used during ABC Toddler given the developmental capacity of toddlers. Briefly, the protocol was conducted in dyads' homes and mothers were asked to have their children fast for one hour and then serve a typical lunch. After lunch, the research assistant presented a standardized plate of highly-palatable, developmentally-appropriate sweet and salty snack foods (Nabisco Original Chips Ahoy chocolate chip cookies: 22.0g, 106.4 kcal; Nabisco Original Oreo cookies: 23.2g, 109.3 kcal; Keebler Animal Cookies, Frosted: 19.0g, 97.9 kcal; Nabisco Rainbow Candy Blast Chips Ahoy cookies: 33.4g, 176.8 kcal; Kellogg's Keebler Fudge Stripe chocolate-coated cookies: 23.6g, 122.1 kcal; Pringles potato chips: 18.2g, 97.6 kcal; Frito-Lay Cheetos cheese puffs: 20.3g; 108.8 kcal). Research assistants then told the child, "Here are some special treats you can eat." and to signal to the toddler that they could eat the foods offered, the research assistant ate one cookie off the plate and said, "I'm going to have one, too. Mmm this is really good. You can eat as much as you want." The child was then given free access to the food. The mother was asked not to interact with the child during the protocol to minimize external prompts to eat. After 10 minutes, the plate of food was removed. Remaining food was weighed and kcal of each food consumed were calculated.

Because the protocol, including foods offered to children, varied between the cohorts, children's total kcal intake and intake of sweet and salty foods were standardized within each cohort to a mean of 0 and SD of 1, and then combined across the cohorts.

**Sociodemographic and other characteristics.** Mothers reported sociodemographic characteristics for herself and her child including the child's sex and race and ethnicity; and her own educational attainment. Child race/ethnicity included non-Hispanic white, black, biracial or other; and Hispanic; due to small cell sizes, race/ethnicity was analyzed as non-Hispanic white versus other in multivariable analysis. Children's age at the time of data collection was calculated from their birthdate. Children's height and weight were measured using standardized protocols by trained research assistants during the study visit. Using the 2000 CDC growth charts,<sup>29</sup> sex and age-specific BMI z-scores and percentiles were calculated. Based on the BMI-for-age percentiles, child BMI was classified as underweight (<5<sup>th</sup>), normal weight ( $\geq$ 5<sup>th</sup> to <85<sup>th</sup>), overweight ( $\geq$ 85<sup>th</sup> to <95<sup>th</sup>), or obesity ( $\geq$ 95<sup>th</sup>).

### **Statistical analysis**

All analyses were conducted using SAS 9.4. All analyses were stratified by sex, recognizing previously-described sex differences in prenatal development indicators and obesity-related outcomes.<sup>7,30</sup> Linear regression was used to model each eating behavior as a function of one of three prenatal development indicators (BWz, maternal pre-pregnancy BMI, or gestational weight gain) and a priori confounders (BWz: maternal education and race/ethnicity; maternal BMI: maternal education, race/ethnicity, age; GWG: maternal education, race/ethnicity, age, pre-pregnancy BMI). Non-linear associations with continuous independent variables (BWz, maternal BMI) were assessed by testing for quadratic and cubic terms (Wald significance of the quadratic

or cubic term  $p < .10$ ). BWz quadratic terms were significant in several models and retained, for comparability, for all models. The BWz cubic term was significant and retained for satiety responsiveness in girls. Statistical significance was assessed at the 95% confidence level.

## RESULTS

Data from 222 girls and 232 boys were included in the current study. Over 40% of mothers had a high school or less education and 45-46% were from minority racial/ethnic groups (Table 1). Mean BWz was -0.17 for girls and -0.25 for boys, with variation across the birth weight spectrum: SGA prevalence was 14.2% in girls and 13.3% in boys, LGA prevalence was 9.5% in girls and 4.6% in boys. Mean maternal pre-pregnancy BMI was in the overweight range (approximately 28 kg/m<sup>2</sup>), and over half of mothers gained more weight than recommended during pregnancy. Among girls, 38.5% had overweight or obesity and 32.0% of boys had overweight or obesity. Girls and boys consumed a mean of 77.4 and 101.0 kcal in the absence of hunger, respectively, approximately 80% from sweet foods. Sweet and total foods eaten the absence of hunger were weakly correlated with food responsiveness (Spearman: 0.17, 0.18, respectively) and uncorrelated with enjoyment of food (Spearman: 0.03, 0.04) and satiety responsiveness (Spearman: -0.06, -0.04) (data not shown).

In adjusted analysis, girls born with lower or higher BWz consumed more kcal from sweet foods and, correspondingly, total kcals, in the absence of hunger than girls with moderate BWz (Table 2; Figure 1). Also among girls, BWz exhibited U-shaped associations with parent-

reported food responsiveness and enjoyment of food, although these associations were not statistically significant; the cubic association with satiety responsiveness exhibited lower satiety responsiveness for lower BWz, moderate satiety responsiveness in the normal range of BWz, with higher satiety responsiveness in the higher range of BWz. Higher maternal pre-pregnancy BMI was associated with significantly higher food responsiveness and enjoyment of food (Table 2). GWG was not associated with appetite regulation in girls.

In boys, prenatal predictors were, in general, not related to measures of appetite regulation (Table 3; Figure 1). In two exceptions, higher maternal pre-pregnancy BMI was associated with *higher* satiety responsiveness and less than recommended GWG was associated with lower satiety responsiveness in the child.

## DISCUSSION

Among low-income toddler and preschool-aged girls, but not boys, both higher and lower BWz were associated with greater energy consumed in the absence of hunger, particularly from sweet foods. Eating in the absence of hunger among children is indicative of poor appetite regulation and predictive of excess weight gain and obesity.<sup>27,31,32</sup> Further, also among girls, lower BWz was associated with lower satiety responsiveness, another eating behavior that places children at increased risk for obesity,<sup>33,34</sup> while higher BWz was associated with higher satiety responsiveness. In contrast, associations between maternal BMI and GWG with child appetite regulation were inconsistent. Together, these findings partially support the hypothesis that

adverse prenatal conditions are associated with poor appetite regulation during early childhood, but suggest variation across food approach behaviors. Further, some exposures (e.g., factors that lead to higher infant birthweight), may be more important than others (e.g., maternal pre-pregnancy weight status) in altering children's appetite regulation. Finally, the lack of similar associations observed among boys also suggest sex differences in the potential impacts of the prenatal environment factors that influence child growth, as has been observed in previous research.<sup>7,30</sup>

The observed associations between lower BWz and greater eating in the absence of hunger among girls are consistent with prior evidence that intrauterine growth restriction and low birth weight are associated with greater external and restrained eating, neuronal response to viewing of palatable foods in children,<sup>11</sup> objectively-assessed impulsive eating in girls,<sup>14</sup> and sweet taste preference.<sup>35</sup> This study contributes to this growing but still small body of evidence by demonstrating associations between lower birth weight and an objective measure of appetite regulation. These eating behaviors may serve to explain associations between low birthweight and risk for cardiometabolic disease<sup>4</sup> and, in some study populations, obesity.<sup>36</sup>

With regard to the observed sex differences, prior work suggests that developmental programming occurs in a sex-dependent manner,<sup>37-39</sup> yet sex differences in birth weight-eating behavior associations are mixed. Silveria and colleagues found associations between IUGR and impulsive eating in girls but not boys,<sup>14</sup> while Van Deutokem found similar associations between birth weight and satiety responsiveness in boys and girls;<sup>40</sup> yet most studies lacked sufficient

sample size to examine sex differences. Further research on mechanisms underlying sex-specific associations between birth weight and appetite regulation is needed.

Our study adds to the extant literature regarding birthweight and child appetite regulation by providing some of the first evidence for associations across the full spectrum of birth weight and child eating behavior, including large birth size (LGA or high birth weight), which is strongly associated with later obesity.<sup>41</sup> A small number of studies examine birth weight as a continuous variable, finding *less* picky eating and *less* neuronal response to food with increasing birth weight.<sup>11,13</sup> Yet consideration of only linear associations may distort associations by precluding identification of adverse outcomes at both the lower and higher ends of the spectrum of birth size. Oliveria and colleagues examined LGA births, finding mixed associations suggestive of lower risk of maternal-reported food avoidant eating behaviors.<sup>12</sup> However, the study did not examine food approach behaviors. In girls, our findings that higher birth weight is associated with greater satiety responsiveness are counter to expectation, and may reflect differences between objectively-measured and parent-reported appetite regulation, or differences in specific dimensions of food approach behavior (e.g., food responsiveness, satiety responsiveness) that may drive eating in the absence of hunger. Influences of specific prenatal exposures with multiple dimensions of food approach behavior in both girls and boys offer an important area for future research.

The current study's largely null associations between pre-pregnancy BMI or GWG and children's food approach behavior are inconsistent with extensive animal evidence that diet-

induced maternal obesity alters neurocircuitry in the offspring that increases appetite and food intake.<sup>6,7</sup> These effects are observed during the both the prenatal and postnatal period, reflecting effects that occur through intrauterine mechanisms and lactation. Our findings likely reflect greater complexity of appetite regulation in humans, involving environmental, psychosocial, and genetic drivers that are an active area of research.<sup>8,10</sup> Additionally, we were not able to examine associations between postnatal factors (e.g., lactation) and eating behaviors. It is also possible that prenatal influences on appetite regulation manifest later in childhood.

Our findings suggest that poor appetite regulation may be a mechanism through which intrauterine programming promotes obesity risk, while guiding areas in need of additional research. First, greater understanding of the independent and joint roles of prenatal and infant growth on appetite regulation is needed.<sup>40</sup> Second, the role of prenatal determinants on distinct aspects of appetite regulation – for example, enjoyment of food or satiety responsiveness – requires further study. Third, the contributions of genetic and ongoing environmental factors that influence both prenatal growth and postnatal development of appetite regulation is needed. For example, shared genetic variants are associated with maternal weight status and prenatal and postnatal growth,<sup>42</sup> potentially through appetite regulation. Further research with in depth characterization of maternal pre-pregnancy, prenatal, and child characteristics can help inform our causal understanding of the mechanisms by which the prenatal environment impacts children's eating and growth.

## Limitations and strengths

Findings from this study should be interpreted in the context of several limitations. First, our exposures of interest – BWz, maternal pre-pregnancy BMI, and GWG – were calculated using data retrospectively reported by mothers. While maternal report of birth weight has been shown to be valid in prior research, there is variation in body composition at any given BMI.<sup>43</sup> Self-reported pre-pregnancy weight and GWG are also known to have error, including misclassification error of GWG, potentially attenuating the observed associations.<sup>22,44</sup> Second, we lacked measures of eating behaviors during infancy, precluding investigation of the emergence of poor appetite regulation during early childhood or the extent to which adiposity during infancy played a role in toddler and preschoolers' appetite regulation. These limitations are balanced by the fact that this study is one of the first investigations of prenatal determinants of objectively-measured appetite regulation in humans, including maternal weight status prior to and during pregnancy, in a U.S. study population spanning two critical periods of early childhood: toddler and preschool age.

## CONCLUSION

Study findings partially support the hypothesis that exposures during the prenatal period may influence appetite regulation in early childhood. Among girls, higher BWz and lower BWz were associated with worse objectively-measured food approach behavior. Because appetite regulation may be an important mechanism underlying prenatally-induced predispositions to



obesity, environmental or behavioral strategies to counteract altered appetite regulation thus may be particularly needed to prevent obesity among high-risk children such as these. However, the current study's sex-specific findings and lack of associations between maternal characteristics and children's food approach behavior, which are counter to many findings from the animal literature, suggest further study is needed to understand the early life etiology of appetite regulation among humans.

#### **CONFLICTS OF INTEREST STATEMENT**

The authors declared no conflict of interest

#### **ACKNOWLEDGMENTS**

We thank participants in the ABC toddler and preschool studies. JBH and KWB conceived and designed the study and drafted the manuscript. HMW and JS conducted statistical analysis. ALM and JCL conceptualized and designed the parent study and provided critical review of the manuscript. All authors reviewed and approved the final manuscript as submitted. This research was supported by NICHD R01HD069179 (PI: Lumeng and Miller) and NIDDK K01DK102857 (Boone-Heinonen).

## CAPTIONS

Table 1: Characteristics of mother/child dyads in the Appetite, Behavior, and Cortisol (ABC)

Study, by child sex

Table 2: Association of indicators of adverse prenatal conditions and eating behaviors in girls

Table 3: Association of indicators of adverse prenatal conditions and eating behaviors in boys

Figure 1: Association of birth weight z-score and eating behaviors in girls and boys<sup>a</sup>

<sup>a</sup> Toddler and preschool-age girls in the Appetite, Behavior, and Cortisol (ABC) Study. Estimates obtained from sex-stratified linear regression modeling a single appetitive trait as a function of birth weight z-score and control variables (maternal education and race/ethnicity).

<sup>b</sup> Objectively measured appetitive traits ascertained from the Eating in the Absence of Hunger protocol (n=195)

<sup>c</sup> Parent-reported appetitive traits ascertained from the Children's Eating Behavior Questionnaire (n=201)

\* Quadratic birth weight z-score term is significant (Wald  $p < 0.1$ )

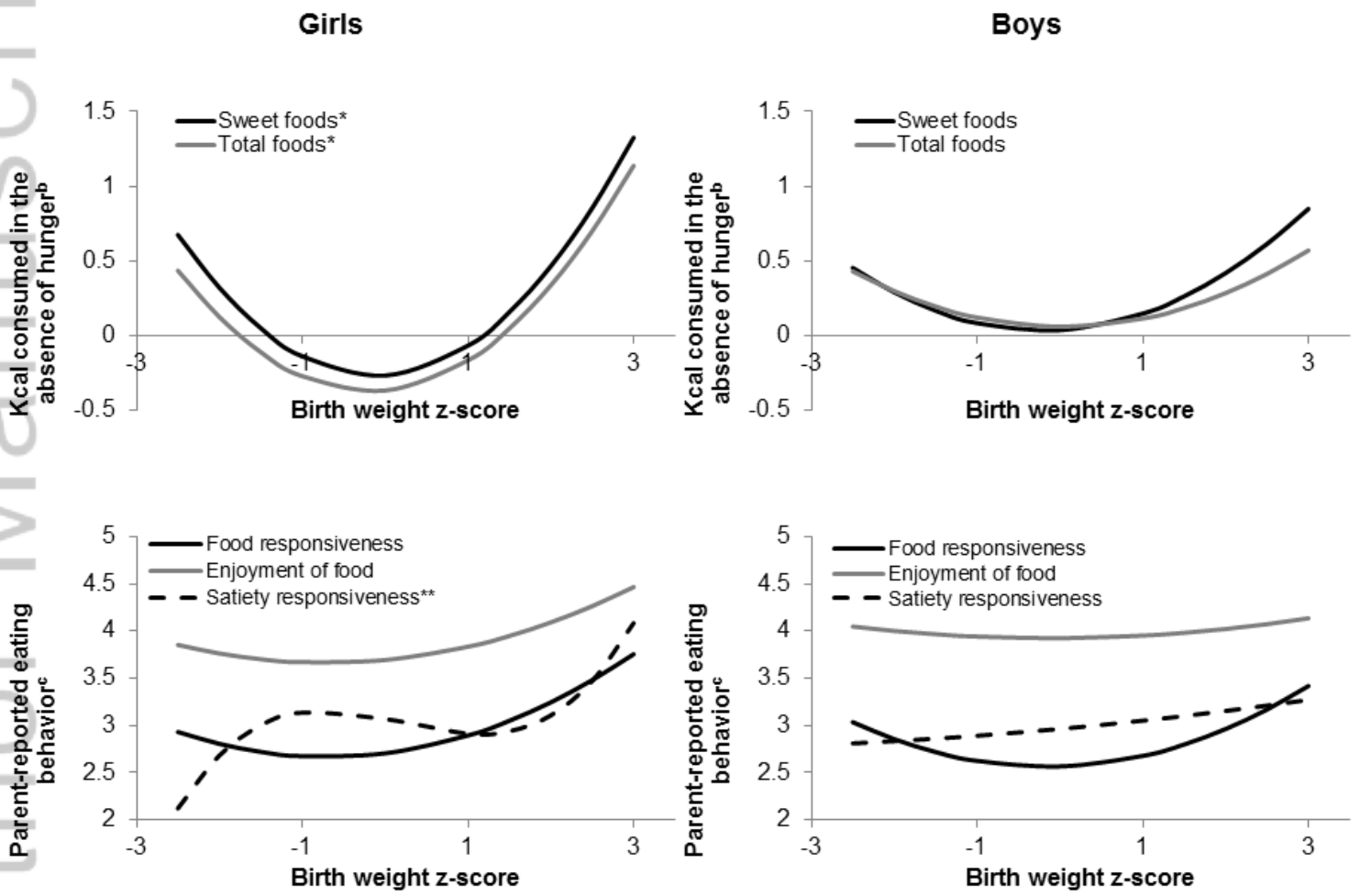
\*\* Cubic birth weight z-score term is significant (Wald  $p < 0.1$ )

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