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Exposure to Maternal Diabetes *in utero* and Offspring Eating Behavior: The EPOCH Study

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Abstract

The risk of becoming overweight among offspring exposed to gestational diabetes (GDM) *in utero* is two-fold higher than in the general population. The responsible mechanisms are likely multifactorial, with some evidence that GDM exposure alters brain satiety signaling, which may impact eating behavior. To better understand these effects, we investigated the relationship between GDM exposure, eating behavior, and total energy intake in 268 adolescents from the Exploring Perinatal Outcomes among Children cohort, who were exposed ($n=50$) or not exposed ($n=217$) to GDM *in utero*. Eating behavior was measured by the Eating in the Absence of Hunger in Children and Adolescents (EAH-C) questionnaire, which included subscale scores for Negative Affect, External Stimuli, and Fatigue/Boredom. Total energy intake (kcal/day) was derived from the Block Kid's Food Questionnaire. The associations between GDM exposure and the outcomes of total score and each EAH-C subscale were evaluated in separate multivariable models. In addition to the main predictor, GDM, the models included a GDM-by-sex interaction term and were adjusted for important covariates. The associations between EAH-C total and subscale scores and the outcome of total energy intake were also tested in separate multivariable models. Female offspring exposed to GDM *in utero* (vs unexposed males and females) were more likely to continue eating beyond satiation due to feelings of boredom and fatigue ($\beta=0.47$, 95% CI: 0.11, 0.83), and in general (EAH-C total score; $\beta=4.20$, 95% CI: 0.56, 7.86) compared to unexposed males. All EAH-C subscale and total scores were significantly, positively associated with higher

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energy intake ($p < 0.05$ for all, respectively). Our findings highlight the need for further investigation into the possible early life programming of eating behaviors by GDM exposure *in utero*.

Keywords

eating in the absence of hunger; gestational diabetes; adolescents

INTRODUCTION

In the United States, gestational diabetes (GDM) occurs in approximately 9% of all pregnancies [1]. Offspring of mothers who experienced GDM in pregnancy have an increased lifetime risk of metabolic disease and obesity, with a two-fold greater risk of being overweight or obese compared to their unexposed counterparts [2, 3]. The mechanism by which GDM exposure imparts increased risk of obesity in offspring remains unclear, however. Recent evidence from human studies and animal models suggest that one contributing factor may be altered eating behavior, specifically, eating in the absence of hunger, which may result from dysfunctional hunger and satiety signaling.

Feelings of hunger and satiety arise from physiological cues in the gut and the brain, and involve hormones that signal energy homeostasis, such as leptin and insulin, which are produced in proportion to body fat. Specifically, both leptin and insulin act directly on the hypothalamus to reduce food intake in murine models [4]. Both leptin and insulin resistance can affect the brain's response to satiety signaling. Rat offspring of diabetic pregnancies have significantly elevated fasting leptin, leptin resistance of the hypothalamus and subsequent increased food intake in adulthood [4]. Therefore, it may be postulated that while elevated levels of these hormones signal the appropriate energy status to the brain, resistance to these signals may blunt long-term regulation of food intake through disruption of brain satiety signaling, possibly resulting in eating in the absence of hunger.

In humans, eating in the absence of hunger (EAH) is related to excess body weight, and has been observed in children as young as preschool age [5, 6]. In multiple studies, obese children and young adults demonstrated significantly greater EAH compared to normal weight individuals [7–9], suggesting that individuals who eat in the absence of hunger are more likely to have excess adiposity. Further, in young girls, baseline EAH was found to be associated with weight gain measured over a follow-up period of 4 years [9].

While much of the research has focused on family environment and parental feeding practices as contributors to a child's EAH [10, 11], developmental origins of EAH have also been observed [12]. Male offspring born to mothers who were obese in pregnancy had significantly greater EAH compared to those born to non-obese mothers [13], even after controlling for parental feeding practices and snacking behaviors. This helps to support the hypothesis that *in utero* exposures to abnormal maternal metabolic milieu may contribute to an altered eating behavior phenotype in the offspring, despite the home environment. Specifically, maternal conditions in which the developing fetus is substantially overnourished, such as in diabetic pregnancies (CITE), could have However, no studies to

date have investigated the relationship between exposure to gestational diabetes (GDM) *in utero* and offspring EAH.

This study investigated the relationship between GDM exposure *in utero* and EAH in a sample of 268 adolescents participating in the Exploring Perinatal Outcomes in Children (EPOCH) study. EAH was measured by adolescent self-reports, utilizing the Eating in the Absence of Hunger in Children and Adolescence (EAH-C) questionnaire. We also investigated the association between EAH and daily total energy intake (kcal/day) in GDM-exposed and unexposed offspring. In these analyses, we hypothesized that GDM-exposed adolescents would demonstrate greater EAH, and that EAH would be positively associated with total energy intake. We further hypothesized that adolescent sex would significantly modify the relationship between GDM exposure and EAH, and that GDM-exposed female adolescents would have significantly different EAH compared to their contemporaries in the other groups.

METHODS

Participants

EPOCH is a historical prospective cohort study of children and adolescents born between January 1, 1992 and December 31, 2002 to mothers enrolled in the Kaiser Permanente of Colorado Health Plan at the time of the participants' birth. The perinatal database at Kaiser Permanente of Colorado was used to identify children who were exposed or not exposed to gestational diabetes (GDM) *in utero* [14, 15]. Participants and their biological mothers were invited to two research visits: the first occurred between July 1, 2006 and June 30, 2009 (ages 6–13, T1), and the second visit occurred between July 1, 2012 and June 30, 2015 (ages 12–19, T2). The eligible cohort for the current analysis includes all participants who attended the T2 research visit (N=417), and who completed the Eating in the Absence of Hunger for Children and Adolescents (EAH-C) questionnaire at T2 (N=268, 50 exposed, 217 unexposed).

Maternal Diabetes Status and Other Factors

Exposure to GDM *in utero* was defined by physician-diagnosed GDM, as reported in the perinatal database at Kaiser Permanente of Colorado, as described previously [14, 15]. Maternal pre-pregnancy weight, participant (offspring) birth weight, and gestational age at birth were also abstracted from the medical record. Maternal height (cm) was collected in-person at the T1 research visit. Maternal pre-pregnancy body mass index (BMI) was calculated as kg/m^2 from the medical record-reported pre-pregnancy weight and the in-person height taken at T1. Maternal education, race/ethnicity, and household income were self-reported at the T1 visit.

Offspring Measures of EAH-C and Other Factors

Participant eating in the absence of hunger (EAH) assessments were conducted at the T2 visit (adolescence) with the EAH-C questionnaire. The EAH-C is a 14-item, self-report questionnaire that assesses the frequency by which an individual continues to eat when already full in response to a stimulus (e.g. emotional or external) [7]. Participant responses

are reported on a 5-point Likert scale with a score of 1 meaning “Never”, and a score of 5 meaning “Always”. Three subscale scores were derived from the 14 items by averaging responses across items corresponding to each subscale. The three subscales represent EAH due to feelings of anger or frustration (negative affect), or feelings of boredom or tiredness (fatigue/boredom), or external cues such as other people continuing to eat (external stimuli) (subscale score range: 1 – 5). A total EAH-C score was generated by summing individual item scores across all questionnaire items, representing EAH, overall (total score range: 14 – 70).

At each research visit, participant pubertal development was assessed by self-report with an adapted diagrammatic representation of Tanner staging [16]. Age and participant sex were self-reported by the biological mother. Participant weight (kg) and height (cm) were measured by trained research personnel at the T1 and T2 research visits, and BMI calculated as kg/m². At both research visits, participants completed a Block Kid’s Food Questionnaire [17] to estimate total energy intake (kcal/day), and a 3-day physical activity recall questionnaire [18]. Physical activity results are reported as the participants meeting or not meeting the standard of at least 1 hour of moderate-to-vigorous physical activity per day within the 3-day recall period [19].

Statistical Analysis

All analyses were performed in SAS, version 9.4 (SAS Institute Inc., Cary, NC). Statistical significance was defined as $p < 0.05$ for all hypothesis tests. Participants’ characteristics by GDM exposure status were compared using univariate descriptive statistics. T-tests and Wilcoxon rank-sum tests were conducted to compare continuous variables. Frequency differences among categorical variables were tested with chi-square or Cochran Mantel-Haenszel tests.

The primary outcomes of interest were the subscale scores of the EAH-C, negative affect, fatigue/boredom, and external stimuli, as well as the total EAH-C score. Sex was included in all models as a potential effect modifier of GDM exposure (dichotomous), the primary exposure of interest. An *a priori* list of covariates was chosen for the model, and included participant age at T2 and participant race. Tanner stage at T2, maternal pre-pregnancy BMI, and participant BMI at T2 were all considered as additional covariates. However, in all models with EAH-C subscale and total scores as the outcome, maternal pre-pregnancy BMI, Tanner stage, and participant BMI at the T2 research visit did not affect the main effects of GDM exposure and participant sex, nor were they, themselves significant in the models. Therefore, participant age at T2 and participant race were the only covariates included in the final models. Four group contrasts were generated to test our *a priori* hypothesis that GDM-exposed females are significantly different from all other groups. A Bonferroni p-value adjustment for multiple comparisons was applied with significance defined at a p-value of less than 0.0125 ($0.05/4 = 0.0125$).

Normality of the model residuals was assessed for each outcome variable. The distribution of the residuals for the negative affect subscale score was zero-inflated. Therefore, the negative affect score was dichotomized into subscale scores < 1 and ≥ 1 . For analyses including the continuous outcomes of fatigue/boredom and external stimuli subscale scores,

and the EAH-C total score, general univariate linear models were employed. For the outcome of negative affect EAH, a logistic regression model was used.

The association between EAH and energy intake at T2 was also examined. The primary outcome of interest in all models was total energy intake (kcal/day). As primary predictors, each of the EAH-C subscale scores, and the total EAH-C score were used in four independent models. Normality of the residuals for total energy was confirmed; therefore, we employed general univariate linear models to test the association between EAH and energy intake in each of the four models. An *a priori* list of covariates was selected, and included GDM exposure status, participant age at T2, sex, race, BMI at T2, and whether the participant met the physical activity recommendations (yes/no) at T2. This group of covariates was included in all models with total energy intake as the outcome.

RESULTS

Table 1 includes the descriptive comparisons between the exposed and unexposed groups. On average, GDM-exposed participants were younger ($p < 0.01$) and more likely to be non-Hispanic white ($p < 0.01$). Despite the younger age of the exposed group, they did not differ significantly by pubertal stage, as measured by self-reported Tanner staging ($p = 0.34$). The two groups also did not differ significantly by current BMI ($p = 0.78$), energy intake ($p = 0.55$), or physical activity ($p = 0.97$) at the T2 research visit.

In modeling the influence of GDM exposure *in utero* on EAH, we found that the interaction term between GDM exposure and participant sex was significant for the outcomes of EAH-C total score ($F_{1,258} = 4.83$, $p = 0.03$) and the fatigue/boredom subscale score ($F_{1,258} = 7.67$, $p < 0.01$). Table 2 gives the model effect estimates for the contrasts between the sex by exposure groups. On average, female participants exposed to GDM *in utero* had significantly higher EAH-C total scores compared to the average of all other groups ($\beta = 4.52$, 95% CI: 1.06, 7.98, $p = 0.01$). Specifically, GDM-exposed females had significantly higher EAH-C total scores compared to exposed males ($\beta = 5.94$, 95% CI: 1.76, 10.11, $p < 0.01$). Exposed females also had significantly higher EAH-C scores due to fatigue and/or boredom compared to the average of all other groups ($\beta = 0.52$, 95% CI: 0.18, 0.86, $p < 0.01$), and specifically, compared to exposed and unexposed males ($\beta = 0.67$, 95% CI: 0.26, 1.08, $p < 0.1$; $\beta = 0.47$, 95% CI: 0.11, 0.83, $p = 0.01$). For reference, Table 3 presents unadjusted and model-adjusted mean EAH-C scores for fatigue and/or boredom and total score for the four groups (Exp-F, Exp-M, Un-F, and Un-M) tested by the GDM-by-sex interaction.

The exposure to GDM by sex interaction term was non-significant in the models with negative affect ($\chi^2 = 0.42$, $p = 0.43$) and external stimuli ($F_{1,258} = 0.70$, $p = 0.70$) subscale scores as the outcome. In a reduced model of the external stimuli subscale score, without the interaction term, neither the main effects of sex ($\beta = 0.03$, 95% CI: -0.13 , 0.20 , $p = 0.68$), nor GDM exposure ($\beta = -0.12$, 95% CI: -0.35 , 0.11 , $p = 0.31$) were significant. However, sex had a significant main effect (OR = 2.66, 95% CI: 1.59, 4.46, $p < 0.001$) in the model with the negative affect subscale score. Here, female participants had nearly 3 times greater odds of having a negative affect score ≥ 1 compared to male participants (OR = 2.67, 95% CI: 1.59, 4.46, $p < 0.01$), regardless of GDM exposure.

Table 4 gives the model effect estimates generated from the four models assessing the associations between the EAH-C total and subscale scores, and total energy intake of the participants at the T2 research visit. All EAH-C subscale scores and the EAH-C total score were significantly and positively associated with higher total energy intake of participants ($p < 0.05$ in all models, respectively), and these associations were independent of the participants' current BMI and physical activity level. Sex was also a significant, independent predictor of total energy intake, with females having significantly lower intake, on average, compared to males ($p < 0.01$ in all models, respectively).

DISCUSSION

These results provide evidence that GDM exposure *in utero* is significantly associated with eating in the absence of hunger (EAH), but only in female adolescents. We further found that higher scores on the overall EAH-C questionnaire, as well as on any individual subscale were significantly associated with higher total energy intake, independent of adolescent BMI and physical activity. Together, these findings suggest that the effect of GDM exposure *in utero* on increased risk of obesity later in life may be working, in part, through EAH and increased caloric intake.

This is the first study to investigate the relationship between GDM exposure *in utero* and EAH, and while the mechanism linking GDM to EAH remains unknown, a possibility may be through disrupted satiety signaling. EAH as a behavioral construct of hyperphagic eating may indirectly measure an altered response to satiety. In a study of Hispanic children, Fisher and colleagues found a significant, positive association between EAH and fasting serum levels of leptin and insulin [12], two important long-term satiety signaling hormones. Unfortunately, these associations were not adjusted by child weight, and given the cross-sectional design of the study, the relationship between EAH and leptin and insulin may be attributable to the adiposity of the child. In offspring of diabetic dams, leptin resistance in the hypothalamus is associated with increased caloric intake and higher weight [4], suggesting dysregulation of long-term satiety signaling due to diabetes exposure *in utero*. This may be a potential mechanism by which GDM exposure *in utero* leads to greater eating in the absence of hunger. Our group has reported elevated serum leptin levels at birth in EPOCH participants exposed to GDM *in utero* [20]. Therefore, while leptin secretion may be higher, leptin resistance as a result of GDM exposure may impede the brain's response and result in EAH. However, the impact of GDM exposure on leptin and subsequent EAH and child weight gain or obesity status in humans must be studied longitudinally to draw more substantial conclusions regarding this hypothesized mechanism.

We found that eating in the absence of hunger in response to fatigue and boredom was higher among exposed females compared to the other three groups. GDM-exposed females also had higher overall EAH-C scores compared to these groups. This trend among female offspring in the EPOCH cohort is contrary to the EAH literature where EAH studies have found that males are more likely to eat beyond satiation, compared to females [5, 12]. However, our findings may be explained by exposure to GDM *in utero*, which previous studies had not investigated. Importantly, there is some evidence that exposure to GDM *in utero* affects offspring differently, depending on sex [21–23]. Given the possibility that

GDM exposure could have sexually dimorphic consequences, our findings require further investigation and replication in other cohorts.

Eating in the absence of hunger has been associated with higher caloric intake [24], which we confirmed in our analysis of the EPOCH cohort. Since increased energy intake is an established risk factor for the development of obesity [25], our findings suggest that adolescents in EPOCH with higher EAH and higher caloric intake may be at higher risk of obesity later in life. However, cross-sectional studies, which predominate in the literature, have produced inconsistent findings for the relationship between EAH and overweight and obesity in children and adolescence, depending on how EAH was measured (e.g. observed versus self-report questionnaire) [8, 12, 26, 27]. Prospective studies investigating the association between EAH and subsequent weight gain in childhood and adolescence are also inconsistent [9, 28]. Kelly and colleagues found no association between adolescent EAH and weight gain within one year of follow-up [28], while Francis et al. reported a significant association between EAH and weight change from 5 to 9 years old in daughters of overweight mothers [9]. The lack of an association found by Kelly et al. could be due to the short follow-up period of just one year, over which long-term risk of obesity cannot be assessed. EAH and the resulting increased energy intake may have a cumulative effect on obesity risk. Therefore, studies with longer follow-up periods are needed to accurately assess the relationship between EAH and obesity development.

Our analysis adds novel evidence of a potential pathway linking exposure to GDM *in utero* and increased risk of later life obesity in the offspring. Our findings should be interpreted cautiously, however, as there are several notable limitations that should be considered. One limitation is our use of the self-report EAH-C questionnaire to assess EAH, rather than directly observing EAH in a controlled laboratory setting or at home. Therefore, we must consider the possibility of random error in our EAH measure, given the self-report nature of the instrument, which may be prone to misreporting. However, we do not believe that this is a source of bias in our analysis because it is unlikely that the GDM exposed adolescents were more prone to misreport compared to their unexposed counterparts. Further, the EAH-C, while used in this analysis as a measure of eating beyond satiety, was designed to measure emotional eating, making it difficult to parse out the influence of disrupted physiologic satiety signaling versus eating in response to high-affect and low-affect emotional cues without quantification of satiety hormones. Despite this limitation, our analysis found significant differences in EAH between the exposed and unexposed offspring, suggesting that GDM exposure *in utero* is acting on this system, either physiologically or behaviorally.

An additional limitation to consider was our use of a self-report food frequency questionnaire to estimate energy intake, which may also be prone to misreporting or underreporting. Again, however, we do not expect this to have introduced bias or systematic error given that it is unlikely that exposed adolescents underreported their diet more than unexposed adolescents. Finally, we must also consider potential, unmeasured confounders such as the household environment, offspring depressive symptoms, and parental feeding practices and EAH behavior.

In conclusion, our study provides preliminary evidence that exposure to GDM *in utero* may alter eating behavior in the offspring. Altered eating behavior could mediate the risk of obesity later in life in GDM exposed individuals. Further work is needed to establish the longitudinal relationship between EAH and obesity, as well as the potential mediating role of altered eating behaviors.

Acknowledgments

Shapiro conceived the analysis idea, analyzed the data, and wrote the full manuscript. Dabelea conceived the EPOCH study. All co-authors contributed to review and revisions of the manuscript drafts and final product.

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Table 1

Descriptive characteristics of EPOCH participants, by GDM exposure status.

	GDM-Exposed (N = 50)	Unexposed (N = 217)	P
Age (years) ¹ , mean (SD)	15.4 (0.8)	16.3 (1.1)	<0.01
Sex (female), n (%) ^{1,2}	21 (42.0)	111 (51.1)	0.24
Race, n (%) ^{1,2} :			<0.01
NHW	31 (62.0)	79 (36.4)	
NHB	2 (4.0)	25 (11.5)	
Hispanic	15 (30.0)	98 (45.2)	
Other	2 (4.0)	15 (6.9)	
Household income ^{1,2} , n (%):			0.39
< \$50,000	7 (14.0)	45 (20.7)	
\$50,000 – \$75,000	12 (24.0)	38 (17.5)	
> \$75,000	29 (58.0)	121 (55.8)	
Don't know or refused	2 (4.0)	13 (6.0)	
Tanner stage ¹ , median (IQR)	4.5 (1.0)	5.0 (1.0)	0.34 ³
BMI ¹ , mean (SD)	23.6 (5.6)	23.8 (4.4)	0.78
BMI z-score ¹ , mean (SD)	0.59 (1.14)	0.48 (1.13)	0.56
Total energy intake (kcal/day) ¹ , mean (SD)	1690 (802)	1621 (688)	0.55
PA recommendations met ¹ , n (%)	37 (75.5)	161 (75.2)	0.97
EAH-C total ¹ , mean (SD)	26.0 (8.1)	26.6 (7.3)	0.65
EAH-C negative affect ¹ , median (IQR)	1.2 (1)	1.2 (0.6)	0.88 ³
EAH-C external stimuli ¹ , mean (SD)	2.30 (0.69)	2.51 (0.68)	0.05
EAH-C fatigue/boredom ¹ , mean (SD)	1.91 (0.89)	1.93 (0.70)	0.85

NHW = Non-Hispanic White; NHB = Non-Hispanic Black; PA = physical activity

¹: Characteristic of child at the EPOCH-II visit (T2), when EAH was assessed.²: Column percent values are reported.³: Wilcoxon Rank-sum test for non-parametric data used due to non-normal distribution.

Table 2

Effect estimates for the association between GDM exposure in utero by offspring sex and EAH-C subscales and total score.

	EAH-C Total Score ² β (95% CI)	EAH-C Subscales		
		Negative Affect ¹ OR (95% CI)	External Stimuli ² β (95% CI)	Fatigue/Boredom ² β (95% CI)
GDM-Exposure*Sex ³ :				
Exp-F vs All Others	4.52 (1.05, 7.98)[†]	–	–	0.52 (0.18, 0.86)[†]
Exp-F vs Un-M	4.20 (0.56, 7.86)	–	–	0.47 (0.11, 0.83)[†]
Exp-F vs Un-F	3.43 (–0.21, 7.06)	–	–	0.44 (0.08, 0.80)
Exp-F vs Exp-M	5.94 (1.76, 10.11)[†]	–	–	0.67 (0.26, 1.08)[†]
GDM-exposure (yes) ⁴	–	1.24 (0.61, 2.51)	–0.12 (–0.35, 0.11)	–
Sex (female) ⁴	–	2.67 (1.59, 4.46)^{††}	0.03 (–0.13, 0.20)	–
Race:				
NHW	Ref.	Ref.	Ref.	Ref.
Hispanic	0.73 (–1.27, 2.72)	1.06 (0.60, 1.87)	0.10 (–0.09, 0.28)	0.04 (–0.16, 0.24)
NHB	–0.84 (–4.08, 2.40)	0.73 (0.30, 1.78)	–0.07 (–0.37, 0.22)	–0.05 (–0.37, 0.27)
Other	0.27 (–3.54, 4.08)	1.63 (0.52, 5.14)	–0.02 (–0.37, 0.33)	–0.02 (–0.40, 0.35)
Age @ T2	0.87 (0.03, 1.72)^{††}	1.23 (0.96, 1.56)	0.08 (0.004, 0.16)^{††}	0.09 (0.002, 0.17)^{††}

[†] p < 0.0125; Bonferroni p-value adjustment applied for all contrasts shown (0.05/4 = 0.0125).

^{††} p < 0.05; no p-value correction needed.

NHW = Non-Hispanic White; NHB = Non-Hispanic Black

¹: Due to non-normality of the scores, the negative affect subscale score was dichotomized into a score <1 or ≥ 1 and modeled using multivariable logistic regression. Values in table reflect odds ratios and 95% confidence intervals.

²: EAH-C subscale scores for external eating and fatigue/boredom eating, and the EAH-C total score were modeled using univariate general linear models.

³: Exp-F = GDM-exposed female offspring; Exp-M = GDM-exposed male offspring; Un-F = unexposed female offspring; Un-M = unexposed male offspring.

⁴: Main effects are reported for models where the interaction between GDM-exposure and offspring sex was non-significant (p > 0.05).

Table 3

Unadjusted and model-adjusted group mean EAH-C score for fatigue and/or boredom and EAH-C total score.

	Unadjusted		Adjusted ^I	
	EAH-C Total Score Mean (SD)	Fatigue/Boredom Mean (SD)	EAH-C Total Score Mean (95% CI)	Fatigue/Boredom Mean (95% CI)
Females:				
GDM-Exposed	29.33 (8.98)	2.29 (0.99)	30.00 (26.54, 33.47)	2.35 (2.01, 2.69)
Unexposed	26.92 (6.86)	1.94 (0.65)	26.58 (24.98, 28.17)	1.91 (1.75, 2.07)
Males:				
GDM-Exposed	23.65 (6.62)	1.64 (0.70)	24.07 (21.07, 27.06)	1.68 (1.38, 1.97)
Unexposed	26.21 (7.82)	1.92 (0.76)	25.80 (24.14, 27.47)	1.88 (1.72, 2.05)

^I: Adjusted EAH-C subscale scores for fatigue/boredom eating, and the EAH-C total score were modeled using univariate general linear models, adjusted by race/ethnicity and age (Table 2).

Table 4

Effect estimates for the association between EAH-C subscale and total scores, and self-reported total energy intake at T2.

	Total Energy¹ β (95% CI)	Total Energy² β (95% CI)	Total Energy³ β (95% CI)	Total Energy⁴ β (95% CI)
EAH-C Total Score ¹	19.2 (7.6, 30.8) ^{††}	–	–	–
EAH-C Subscales:				
Negative Affect ²	–	163.6 (25.8, 301.4) [†]	–	–
External Stimuli ³	–	–	146.0 (18.5, 273.5) [†]	–
Fatigue/Boredom ⁴	–	–	–	192.5 (75.4, 309.7) ^{††}
GDM-exposure (yes)	63.7 (–182.1, 309.4)	102.0 (–143.3, 347.3)	65.4 (–177.3, 308.1)	68.7 (–173.8, 311.2)
Sex (female)	–285.3 (–461.0, –109.6) ^{††}	–264.1 (–438.6, –89.7) ^{††}	–295.2 (–468.9, –121.4) ^{††}	–297.8 (–471.6, –123.9) ^{††}
Race:				
NHW	Ref.	Ref.	Ref.	Ref.
Hispanic	–96.0 (–292.4, 100.4)	–109.5 (–306.6, 52.4)	–99.4 (–293.8, 95.0)	–107.4 (–301.8, 87.0)
NHB	39.1 (–288.2, 366.4)	39.5 (–288.1, 367.1)	–9.5 (–340.2, 321.2)	4.7 (–325.8, 335.2)
Other	–186.2 (–554.0, 181.6)	–173.0 (–541.1, 195.0)	–163.1 (–527.1, 201.0)	–175.9 (–539.7, 187.8)
Age @ T2	64.3 (–20.3, 149.0)	55.9 (–29.5, 141.3)	52.8 (–31.5, 137.0)	51.8 (–32.5, 136.0)
PA recommendations met (yes) @ T2	178.8 (–27.9, 385.5)	198.4 (–8.3, 405.1)	152.1 (–53.6, 357.8)	166.7 (–38.3, 371.7)
BMI @ T2	–15.2 (–31.2, 0.8)	–13.3 (–29.3, 2.7)	–11.5 (–27.4, 4.4)	–13.3 (–29.2, 2.3)

[†] p < 0.05;

^{††} p < 0.01

NHW = Non-Hispanic White; NHB = Non-Hispanic Black; PA = physical activity

¹: Model 4 where the main predictor is EAH-C *total score*.

²: Model 1 where the main predictor is EAH-C *negative affect* subscale score.

³: Model 2 where the main predictor is EAH-C *external stimuli* subscale score.

⁴: Model 3 where the main predictor is EAH-C *fatigue/boredom* subscale score.