# Parental obesity moderates the relationship between childhood appetitive traits and weight

**Bernard F. Fuemmeler**[1], **Cheryl A. Lovelady**[2], **Nancy L. Zucker**[1], and **Truls Østbye**[1]

[1]Duke University Medical Center, Durham NC

[2]University of North Carolina at Greensboro, Greensboro NC

## Abstract

This study examined the independent and combined associations between childhood appetitive traits and parental obesity on weight gain from 0 to 24 months and body mass index (BMI) z score at 24 months in a diverse community-based sample of dual parent families (n = 213). Participants were mothers who had recently completed a randomized trial of weight loss for overweight/obese post-partum women. As measures of childhood appetitive traits, mothers completed subscales of the Child Eating Behavior Questionnaire, including Desire to Drink (DD), Enjoyment of Food (EF), and Satiety Responsiveness (SR), and a 24-hour dietary recall for their child. Heights and weights were measured for all children and mothers and self-reported for mothers' partners. The relationship between children's appetitive traits and parental obesity on toddler weight gain and BMI z score were evaluated using multivariate linear regression models, controlling for a number of potential confounders. Having two obese parents was related to greater weight gain from birth to 24 months independent of childhood appetitive traits, and while significant associations were found between appetitive traits (DD and SR) and child BMI z score at 24 months, these associations were observed only among children who had two obese parents. When both parents were obese, increasing DD and decreasing SR was associated with a higher BMI z-score. The results highlight the importance of considering familial risk factors when examining the relationship between childhood appetitive traits on childhood obesity.

## Keywords

Childhood eating behaviors; parental obesity; childhood obesity; prevention

## INTRODUCTION

Obesity is a multifactorial condition reflecting a complex interaction between individual predisposition, social, and environmental factors.(1) The rise in obesity prevalence among

Corresponding Author: Bernard F. Fuemmeler, Ph.D., M.P.H., Community and Family Medicine, Duke University Medical Center, Box 104006 DUMC, Durham NC 27710; Phone: (919) 681-7171; Fax: (919) 684-5108; bernard.fuemmeler@duke.edu.

children is particularly alarming given that early childhood obesity not only results in a number of adverse health consequences during childhood, but also tracks into adolescence and adulthood.(2) Recent data suggests that for many children who are overweight, the onset occurs early in development before the age of 2 years; however, the reasons for this are not well understood.(3) A better understanding of the factors associated with excess weight gain during early development is fundamental to developing effective childhood prevention and treatment strategies.

Socio-economic factors along with gestational age, birth weight, and length of breast feeding are factors related to early childhood weight gain.(4) However, other factors such as individual differences in disposition related to eating and food are also relevant. For instance, early theoretical models of obesity (e.g. Stunkard and Schachter's externality model) posited that obese individuals may be less sensitive to internal physiological cues of satiety and more responsive to the presence of food, as well as environmental stimuli associated with food consumption (e.g. food commercials, images of food, etc.).(5, 6) A number of recent studies have examined the tenets of these models by investigating childhood appetite regulation as a potential behavioral marker of obesity susceptibility.(7–9) Lab-based studies have found that observations of "eating in the absence of hunger" (EAH) and "bite frequency" predict weight status and weight gain.(10, 11) Such observations may represent a child's dispositional responsiveness to satiety or heightened enjoyment of food. Studies of community samples have also provided evidence that these dispositional differences or appetitive traits measured using psychometric approaches may be relevant to childhood BMI and risk for obesity. Specifically, psychometric constructs such as parent-reported child "Satiety Responsiveness" is associated with lower BMI, and both greater "Enjoyment of Food" and "Food Responsiveness" are associated with higher BMI and weight gain.(9, 12, 13) These psychometric constructs have also been shown to be convergent with behavioral measures, such as EAH and eating rate and higher caloric intake. Notably, definitively establishing the direction of influence is not possible in cross-sectional studies such as these. However, recent longitudinal studies provide further support that appetitive traits contribute, in part, to weight gain rather than the other way around. (14) These studies suggest that children differ in their appetitive traits and that these differences could explain why some children may be more sensitive to external food cues or less sensitive to internal satiety cues. These factors could contribute to an increased food intake and ultimately higher risk for obesity.

Another important risk factor for childhood obesity is having parents who are obese. Children with 2 obese parents are 10 to 12 times more likely to be obese.(15, 16) Weight gain in early childhood (3 to 5 years of age) is also significantly greater among children with overweight or obese parents or among those born of overweight or obese mothers.(17) children of heavier parents have been found to exhibit lower levels of physical activity and have greater preference for high fat foods and lower preference for healthier foods.(18, 19) This familial influence may be through genetic mechanisms or through the environment.

Childhood appetitive traits and familial risk factors, like parental obesity, may be independently associated with child obesity, and if these factors are independent, it would be informative to know which (appetitive traits or parental obesity) is more important in

relation to children's food intake or risk for obesity. However, circumstantial evidence suggests children's appetitive traits may vary depending on whether or not they have other familial risk factors, such as obese parents.(19) For instance, higher levels of EAH have been observed among children born to mothers who were obese prior to pregnancy compared to their counterparts born to mothers who were lean. (8) The link between appetitive traits and obesity may therefore depend on other factors like parental obesity. Understanding the conditions under which appetitive traits relate to children's food intake and risk for obesity would allow for more precise conclusions about these associations.

The purpose of this study was therefore to examine the extent to which children's appetitive traits (food responsiveness, enjoyment of food, desire for drinks, and satiety responsiveness), and parental obesity status are associated with food intake, weight gain from birth, and BMI z score at 24 months. This study extends previous research by 1) examining these associations among very young children during a critical time when eating patterns and preferences for certain foods are established and 2) evaluating whether these associations between appetitive traits and risk for obesity were moderated by parental obesity.

## METHODS

### Participants

Participants in the current study were recruited from three large obstetrics clinics in Southeastern, US for a larger behavioral randomized controlled trial, Active Mother's Postpartum (AMP).(20, 21) Eligibility for AMP was based upon BMI 25 measured by study staff at the 6 week postpartum obstetrics appointment. Women who did not speak English, were aged < 18 years, or had health conditions that prevented them from walking a mile unassisted were excluded from participation. The AMP intervention was designed to enhance weight loss in postpartum women who were overweight or obese prior to pregnancy. Participants were randomized at 6–8 weeks postpartum to either the AMP intervention (n=225) or the attention control group (n=225). The AMP intervention, which lasted 9 months with post-intervention and follow-up assessments, focused primarily on improving lifestyle behaviors in the mother. The intervention did not encourage the adoption of certain parenting styles or efforts to improve the health of their newborn infant. The mean weight loss was 0.90 kg (±5.1 kg) in the intervention group and 0.36 kg (±4.9 kg) in the control group, which was not a statistically significant difference. There were also no significant group differences in improvement of diet or increased physical activity.(20, 21) At their final follow-up (24 months postpartum) mothers and their 2 year old children were recruited for the current observational study, *AMP Too for Twos!*

Of the 450 participants who were enrolled in the main AMP trial, 309 agreed to participate in the current study. After excluding 43 single parent families, 266 mothers of dual-parent families were asked if they would be willing to deliver a survey packet to their partner, which included a letter describing the study, a consent form, a brief survey, and a pre-paid return envelope. From these 266 dual-parent families, 213 partners returned surveys (80% of the eligible 266). In nearly all cases, parents in this sample were biological (93%) or the partner was living in the home with the mother and target child (97%). Compared to the

participants who originally enrolled in the AMP trial (n=450), participants in the sample for this study (n=213) were more likely to be White (Cramer's V = −.18), have a college degree (Cramer's V = .19), have a family income greater than $60,000 (Cramer's V = .20), and were slightly older (Cohen's d = .27). However, there were no significant differences between the two groups as related to percent calories from fat calculated from dietary recalls (Cohen's d = .01), number of television viewing hours per day (Cohen's d = .03), number of reported minutes per week of walking for physical activity (Cohen's d = .18), and BMI (Cohen's d = .14). Thus, although there were some socio-demographic differences between the two groups, the differences were small (i.e., effect sizes < .3;(22)) and there were no differences between the groups with respect to key health behaviors. All procedures were approved by the collaborating University's Institutional Review Boards.

### Measures

**Child eating behaviors—**Eating behaviors were assessed using the Children's Eating Behavior Questionnaire (CEBQ).(23) Items for the CEBQ were developed from focus groups and interviews with parents of children 2–6 years of age and the mean age of the sample for testing psychometrics of the items was 4.2 (± 1.4) years.(23) The scale is being used in studies with samples of children ranging in age from 2 to 11 years of age.(13, 24, 25) Items have Likert scale response options ranging from 1 (never) to 5 (always). For this study, we restricted our analysis to the following CEBQ subscales: Enjoyment of Food (EF), Food Responsiveness (FR), Desire to Drink (DD), and Satiety Responsiveness + Slowness in Eating (SR). Satiety Responsiveness and Slowness in Eating were combined as they have been shown to load onto the same factor.(23) Domains of EF, FR and DD reflect a general avidity toward eating and food (e.g., "my child loves food (EF);" "if allowed, my child would eat too much (FR);" and "if given the chance, my child would always be having a drink (DD)." SR reflects how easily a child reaches satiety (e.g., my child cannot eat a meal if he/she has had a snack just before"). The CEBQ has been shown to have high internal consistency, good test-retest reliability, stability over time (23, 26), and these particular subscales have been correlated with weight.(9) Cronbach's alphas for subscales in this sample were acceptable (SR = .70; EF = .86; FR = .71; DD = .84).

**Child anthropometrics—**At two years of age children's weights (to the nearest tenth of a pound) and standing heights (to the nearest quarter inch) were measured by study staff during a visit to the laboratory using a Seca portable stadiometer and Tanita BWB-800 scale. Measurements were completed with children wearing casual attire with belts and shoes removed. BMI z score was calculated using the Centers for Disease Control and Prevention SAS macro which computes age and gender adjusted standardized scores.(27)

**Parental weight status—**At the same study visit in which children's weights and heights were measured, mother's weights and heights were also measured. The mothers' partners self-reported height and weight as part of their surveys. Since the purpose of this study was to examine how parental obesity related to children's BMI, we quantified parental weight status into three groups: 0 = neither parent was obese (BMI < 30); 1 = one parent was obese (BMI ≥ 30), but the other was not (BMI < 30); and 2 = both parents were obese (BMI of mother ≥ 30 and BMI of partner ≥ 30).

**Dietary Intake**—Dietary intake of children was assessed similarly to the Feeding Infants and Toddlers Study.(28) The primary caregiver (in most all cases the mother) reported on their child's diet. Dietary recalls were collected on 2 randomly selected days over a 2 week period. Mothers had been given a packet with 2-dimensional visuals to assist in determining food portion sizes. The visuals included various examples of toddler food portions and eating implements (e.g., "sippy-cups" and small bowls). If children attended daycare, mothers were given a form for the daycare provider to record the child's dietary intake (type of food eaten and amount). Mothers used this list to complete the 24-hour recall. The dietary intake of the children was assessed by telephone, using the Nutrition Data System for Research (NDS-R, University of MN), a valid and established method for assessing energy intake.(28, 29) These data included estimated energy [kilocalorie (kcal)] intake available from 183 of the 208 (88%) children. There were no differences in primary demographic characteristics (maternal education and child's race) between mothers who provided dietary information vs. those who did not.

**Other measures**—Parents reported on level of educational attainment, age, and their child's race/ethnicity. Child's birth weight and gestational weeks were reported by mothers when they first enrolled in AMP study, which was shortly after the birth of their child (6–8 weeks). Breast feeding amount was summarized by a lactation score, a measure of breastfeeding "intensity" combining the duration and exclusivity of breastfeeding.(30) This score was derived from the detailed monthly feeding data collected at the 12-month follow-up. A value was assigned for each month—0 if formula fed, 1 if mixed, and 2 if fully breastfed. The resulting score has a possible range of 0–24, and more explanatory power than a simple measure of duration.(31)

## Analysis

The outcomes for these analyses included weight gain from birth (measured by the change in kilograms from birth to 24 months of age), BMI z score at 24 months, and energy intake at 24 months. The initial analyses involved 1) bivariate Pearson r correlations between children's eating behaviors (EF, FR, DD, SR), BMI z-score, weight gain from birth, energy intake at 24 months, and parental BMI; and 2) mean comparisons (general linear model) for BMI z score and weight gain from birth for parental obesity categorical variable (0,1,2) controlling for intervention arm and birth weight. We also calculated the odds of being overweight (BMI z score $\geq 85^{th}$ percentile and $< 95^{th}$ percentile) and obesity (BMI z score $\geq 95^{th}$ percentile) using a multinomial logistic regression for children with one or both parents obese relative to neither obese controlling for intervention arm and birth weight. Separate multivariate linear regression models were performed to examine associations between children's eating behaviors (EF, FR, DD, or SR), the parental obesity variable, and their interaction on each of the anthropometric outcomes (BMI z score and weight gain from birth) and energy intake. All multivariate models included treatment arm, children's age, gender, race, gestational age at birth, birth weight, lactation score, and age and educational level of mothers and their partners. Only main effects models (i.e., models not including interaction terms) are reported when interaction terms were not significant. Post-hoc probes were conducted for all significant interaction effects.(32)

## RESULTS

In the overall sample (Table 1), the prevalence of children with BMI z scores exceeding the 85th percentile was 25% (n = 54) which is slightly higher than the national average of 21% for children ages 2 to 5 years.(33) On average children were born full-term (mean gestational age = 39 weeks).

### Children's appetitive traits and anthropometrics

Table 2 displays the bivariate correlations between CEBQ subscales, BMI z-score, and weight gain from birth. Significant correlation coefficients were in the expected direction with subscales assessing Enjoyment of Food (EF), Food Responsiveness (FR), and Desire to Drink (DD) being positively correlated with BMI z score and weight gain from birth and Satiety Responsiveness (SR) being negatively correlated. All correlations were statistically significant except for the association between EF and weight gain from birth. Mother's BMI and partner's BMI were not related to childhood appetitive traits. Energy intake was significantly associated with FR (r = .18, p < .05) and SR (r = −.17, p < .05), but was not related to child's BMI z score.

### Parental obesity and childhood anthropometrics

Table 3 displays the mean weight gain from birth and BMI z score for children with neither parent obese, one parent obese, and both parents obese. Statistical comparison of means controlled for intervention arm and birth weight. Mean weight gain from birth and BMI z score both increased with increasing parental obesity. The greatest mean difference was observed among children who have two obese parents relative to children with neither parent obese (mean weight gain 9.9 kg vs 9.2 kg and BMI z score 0.7 vs. 0.3). The means for each CEBQ subscale were also evaluated in relation to parental obesity status (neither, one or both obese) controlling for intervention status, child's age in months, gender and race. There were no statistically significant mean differences for EF, FR, DD, or SR by category of obesity status in these models (all p values > .05; data not shown).

### Multivariate linear regression analyses and interaction effects

**Main effects models without interaction—**Appetitive traits were not statistically significantly related to weight gain from 0 to 24 months in models that included the parental obesity variable (one or two parents who were obese) as well as the other covariates (child's age, gender, race, gestational age, birth weight, lactation score, mother's and partner's educational level, mother's and partner's age, and intervention status). Having two obese parents was significantly associated with child weight gain, controlling for appetitive traits and the other covariates (child's age, gender, race, gestational age, birth weight, lactation score, mother's and partner's educational level, mother's and partner's age, and intervention status). In a reduced model, which retained the covariates but removed the appetitive traits, having one parent who was obese was not statistically associated with greater weight gain from birth (β = 0.26, s.e. = 0.23, p = 0.25), but having two parents who were obese was associated greater weight gain from birth (β = 0.60, s.e = 0.26, p < .05) (data not shown in table).

Controlling for parental obesity status and the other covariates (child's age, gender, race, gestational age, birth weight, lactation score, mother's and partner's educational level, mother's and partner's age, and intervention status), there were no significant associations observed between appetitive traits and BMI z score, with one exception: FR was significantly associated with BMI z score. Specifically, a one unit increase in FR was associated with about a .25 unit increase above the BMI z score intercept (see Table 4).

Neither the appetitive traits nor the parental obesity variable was associated with energy intake in multivariate models. In a reduced model, excluding parental obesity but retaining the covariates, both FR and SR were significantly associated to average energy intake in same direction as the bivariate correlations (above). Specifically, controlling for covariates (child's age, gender, race, gestational age, birth weight, lactation score, mother's and partner's educational level, mother's and partner's age, and intervention status), a one unit increase in FR was associated with an increase of 82.2 kilocalories above the intercept ($\beta$ = 82.2, s.e.=38.7, p <.05). A one unit increase in SR was associated with a decrease of 95.2 kilocalories below the intercept ($\beta$ = −95.2, s.e.=44.2, p <.05) (data not shown in table).

**Models with main effects and interaction effect**—In the models examining weight gain from 0 to 24 months and BMI z-score with parental obesity status and child appetitive traits, there were significant interactions between parental obesity status and SR (Table 4 and Figures 1a, 1b). A post-hoc probe of the significant interaction for weight gain indicated that SR was statistically significantly associated with weight gain for children who had two obese parents ($\beta$ = −1.25, p <.01), but not associated when one parent was obese ($\beta$ = −.21 p = .64), or when neither parent was obese ($\beta$ = −.20, p = .48). Likewise, the post-hoc probe of the interaction examining BMI z score indicated that SR was statistically significantly associated with BMI z score for children with two obese parents ($\beta$ = −.81, p < .01), but was not associated when one ($\beta$ = −.16, p = .55), or when neither parent was obese ($\beta$ = −.12, p = .49). A significant interaction between parental obesity status and DD on BMI z score was also found (Table 4 and Figures 1c). DD was significantly associated with BMI z score for children who had two obese parents ($\beta$ = .28, p <.05), but not associated when one parent was obese ($\beta$ = .15, p = .12), or when neither parent was obese ($\beta$ = .07, p = .37).

## DISCUSSION

We found significant associations between parental obesity, children's appetitive traits and BMI z score among a sample of 24 month old children, controlling for a number of variables. Specifically, a positive relationship between Food Responsiveness (FR) and BMI z score was significant and independent of parental obesity status. Lower Satiety Responsiveness (SR) and greater Desire to Drink (DD) were also found to be associated with a higher BMI z score at 24 months, but this association was observed only among children with two obese parents. Appetitive traits were associated with energy intake at 24 months (greater FR and lower SR was associated with greater energy intake), but controlling for parental obesity status reduced these associations to non-significance, indicating potential confounding of parental obesity status in these associations.

The present findings are consistent with previous reports from the U.K. linking EF, FR, DD, and SR with standardized BMI score in older children (8 – 9 years) (9) and SR and EF with BMI standardized score in younger children (3–5 years).(13) In these studies, the associations were maintained even after controlling for child's age, sex, and socio-economic factors. However, previous studies did not account for parental weight status or examine how parental weight status might modify these associations. Our findings support and extend these previous reports by taking into account parental weight status in the relationship between appetitive traits and childhood BMI. It is reasonable to suspect that parent's weight status is a relevant modifying variable in these associations. Using the same measure of childhood appetitive traits, at least one study has found higher scores on the FR and DD constructs among children who have overweight parents (19). Likewise, behavioral measures of satiety are greater among children born of mothers who were overweight.(7, 8) The findings presented here extend previous literature to support the notion that the association between children's appetitive traits and their risk for obesity may be modified by other relevant familial risk factors, like having parents who are obese. The study is also unique in that the associations between appetitive traits and weight and diet outcomes were evaluated in young children. Continued studies are needed that address these associations in young children as this age may represent a sensitive period of development in the pathway to weight regulation throughout childhood.

The observation of a significant interaction between SR and parental obesity in association with weight gain mirrors the associations observed with BMI z score in that SR was related to weight gain only among children who had two obese parents. Overall, the average weight gain from 0 to 24 months (9.51 kg) was fairly high for this cohort of children of primarily overweight mothers and fathers. Weight gain between 8.15kg and 9.76kg during this developmental period has been considered "risky" growth.(34, 35) Among children with two parents who were obese, a one unit decrease in SR was associated with a 1.3 kg increase in weight gain from 0 to 24 months and an increase in BMI z score of 0.81 relative to the average case. Thus, children lower in satiety responsiveness appear to have a higher BMI z score at 24 months and greater early weight gain particularly in familial contexts where there are two obese parents. A moderating effect of parental obesity on the relationship between DD and BMI z score was also observed. It is notable that significant associations were present for DD and SR in relation to BMI z score among children with two obese parents, but FR or EF were not. One possibility is that certain types of appetitive traits are more easily discernible when children are younger. DD and SR may be more noticeable during earlier development when caregivers are providing most of the feeding opportunities as opposed to later circumstance when children beginning to independently access the types of food they enjoy eating.

Children with certain appetitive traits who have obese parents may have a higher BMI z score at 24 moths for a number of reasons. In general, parental obesity may represent parenting and environmental qualities as well as genetic risk factors. The early work of Stunkard and Schachter's externality model suggested that obese adults have difficulty recognizing internal satiety signals and are over responsive to external food cues.(5, 6, 36) Obese parents may be inadvertently modeling these eating behaviors during sensitive developmental periods when children are forming their general orientation toward food and

eating. Parents may be modeling maladaptive behaviors all along the food consumption sequence: from attentiveness to food cues, capacities to inhibit food responsiveness when making food selections, to demonstrating sensitivity to somatic signals in terminating a meal. The feeding practices (e.g., offering food in response to distress) of obese parents might also differ from those of non-obese parents and parent feeding practices could shape or encourage the expression of specific eating tendencies. Notably, it is not clear whether these appetitive traits are completely learned behaviors and influenced by nurturing or if they are influenced by biologically mediated mechanisms, such as genetic differences. SR and EF have been associated with specific gene variants suggesting a biologically mediated component.(37) Appetitive traits observed in children could share common underlying neurological substrates that are modulated in part by genotype differences, which are inherited via positive assortative mating.(38) While it is unclear why the associations we observed were significant when both parents were obese rather than when only one parent was obese, it is possible that two obese parents constitute a more unambiguous model. Perhaps, having one non-obese parent attenuates or even reverses the negative modeling by the obese parent. Having two obese parents might also increase the propensity for biologically mediated eating behavior traits, or influence the expression of these traits through nurturing patterns that may be more prevalent when both parents are obese. Yet another possibility is that obese parents are more vigilant to external food cues or recognize their own insensitivity to satiety and more likely to notice and report these similar traits in their children. It will be important in future studies to begin to deconstruct what exactly is being measured by accounting for parental obesity, since this is such a strong risk factor for childhood obesity and may modify other childhood factors and traits that are related to risk for obesity.

Mechanistic explanations that underlie the associations between parental obesity, children's appetitive traits and childhood BMI are difficult to discern in the context of this study. In this study, parental BMI was not strongly correlated with children's appetitive traits, so it is unlikely that the effect of parental obesity on childhood obesity is mediated by childhood appetitive traits. Also, in our data, strong associations between energy intake and BMI z score were not present, which would need to be established to support a mediation hypothesis that appetitive traits influence BMI via increased energy intake. We did explore whether this association between energy intake and BMI z score varied when parents were obese, but there were no significant effects of parental obesity on these associations (data not shown), suggesting that this pathway is not supported even in contexts where both parents are obese. To our knowledge, extant studies have not established different types of mediating pathway with statistical certainty. As for the influence of childhood appetitive traits on BMI z score via increased caloric intake, it may be difficult to elucidate this pathway using a cross-sectional design. In this study, dietary recalls were conducted over the phone and shortly after the participants visited the lab when their heights and weights were measured. Longitudinal studies would allow a better examination of these associations. It would be helpful to know whether early childhood appetitive traits increase caloric intake (or dietary patterns) and ultimately obesity as children grow and develop, and whether the trajectories of increased caloric intake are steeper among those with obese parents.

This study has certain limitations. This was a sample of women who were participating in a postpartum obesity prevention study. Thus, the parents in this study are more likely to be overweight. It is worthwhile to replicate these findings in samples that also include normal weight parents. The overall effects may be underestimated, since the reference group is heavier than one including normal weight parents. While this sample selection may limit generalizability, the oversampling of overweight mothers allowed us to examine childhood appetitive traits in a high-risk sample – a notable addition to the literature. Related to this is that, by design, only two-parent families who were living with the child were included in these analyses. Thus, the findings here may not generalize to situations where only one parent is in the home. Notable in this study also was the use of dietary recalls, which may have precluded our ability to detect associations between appetitive traits and energy intake. Other methods such as the use of doubly labeled water or daily food diaries could be used in future validations studies that aim to determine the association between appetitive traits and energy intake. Such approaches would have been quite burdensome for participants in this study and are typically not feasible in epidemiologic studies. Another limitation is that although trained study staff measured heights and weights for mothers and their children at two years of age, birth weight and the partner's measurements were not. Measurement of birth weight and length on calibrated scales and measured heights and weights of partners is preferable for future studies. Parents were also the source of reporting for their children's appetitive traits in this study. Ratings from parents are often used to collect data on childhood behavior and temperament, and thus, it is standard practice to ask primary caregivers to rate their children's traits.(23) Future studies could include other methods of measurement, such as direct observation. However, it is important to keep in mind that observations, although correlated, may not necessarily be the same as appetitive traits that are reported by caregivers (39). Caution is also warranted in drawing conclusions about causal associations from the findings in this study. Although we did examine changes from birth, this is essentially a cross-sectional design since eating behavior and 24 month heights and weights were measured at the same time. We did include a number of covariates that might influence weight gain from birth, such as gestational age and a measure of the length of lactation, however, unmeasured potential confounders cannot be ruled out. Also, it could be that heavier toddlers are viewed by their mothers as being more responsive to food or being insatiable. We are currently following this cohort of children and parents as the children turn 6 years of age. Future studies will examine whether these associations between childhood traits at 24 months predict subsequent weight gain and if this is modified by other familial risk factors. Studies extending these analyses could also be informed by assessing how parent behaviors or feeding practices contribute to these associations. In general, since randomization to appetitive traits is not possible, longitudinal studies with multiple follow-up assessments will allow for a clearer understanding of the extent to which such traits influence childhood weight gain and the potential mediating and moderating factors that contribute to this association.

## CONCLUSIONS

Our findings indicate that certain factors, like parental obesity, can interact with childhood appetitive traits to heighten the risk of childhood obesity. The extent to which these traits are

modifiable is largely unknown. These characteristics or temperamental dispositional behaviors toward food reflect a range of responding, which potentially increases children's vulnerability to factors in the family environment (e.g., accessibility/availability of energy dense foods) that place children at risk for obesity.(37) However, it may be that, with targeted interventions, select deficits may be strengthened. The standard approach for childhood obesity treatments and interventions has been to address dietary quality and physical activity with attention to modifying the parent's lifestyle behavior or their feeding practices. In pre-school age children, these multi-component, family-based programs have shown modest effectiveness in reducing weight in children who are already obese. (40) Less research has focused on methodologies for directly modifying children's appetitive traits. Strategies for increasing satiety awareness or reducing food responsiveness (e.g., teaching children to more accurately recognize hunger and fullness or reducing their reactivity to food cues) are warranted. Such strategies could complement traditional interventions focused on improving dietary quality and may be especially useful for children whose parents are themselves obese. In short, assessing family risk factors in addition to the individual childhood characteristics may be a particularly useful for further clarifying the associations that appetitive traits have with childhood risk for obesity, and continued research in this area could be useful in informing prevention strategies.

## Acknowledgments

## References

1. Esposito L, Fisher JO, Mennella JA, Hoelscher DM, Huang TT. Developmental perspectives on nutrition and obesity from gestation to adolescence. Prev Chronic Dis. 2009; 6(3):A94. Epub 2009/06/17. [PubMed: 19527595]

2. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. The American Journal of Clinical Nutrition. 2002; 76(3):653–8. [PubMed: 12198014]

3. Harrington JW, Nguyen VQ, Paulson JF, Garland R, Pasquinelli L, Lewis D. Identifying the "tipping point" age for overweight pediatric patients. Clin Pediatr (Phila). 2010; 49(7):638–43. Epub 2010/02/13. [PubMed: 20150210]

4. Gillman MW, Rifas-Shiman SL, Kleinman K, Oken E, Rich-Edwards JW, Taveras EM. Developmental origins of childhood overweight: potential public health impact. Obesity (Silver Spring). 2008; 16(7):1651–6. Epub 2008/05/03. [PubMed: 18451768]

5. Stunkard A. Obesity and the denial of hunger. Psychosomatic Medicine. 1959; 21:281–90. Epub 1959/07/01. [PubMed: 13835450]

6. Schachter S. Obesity and Eating. Science. 1968; 161(3843):751–6. [PubMed: 5663800]

7. Berkowitz RI, Moore RH, Faith MS, Stallings VA, Kral TV, Stunkard AJ. Identification of an obese eating style in 4-year-old children born at high and low risk for obesity. Obesity (Silver Spring). 2009; 18(3):505–12. Epub 2009/09/26. [PubMed: 19779474]

8. Faith MS, Berkowitz RI, Stallings VA, Kerns J, Storey M, Stunkard AJ. Eating in the absence of hunger: a genetic marker for childhood obesity in prepubertal boys? Obesity (Silver Spring). 2006; 14(1):131–8. Epub 2006/02/24. [PubMed: 16493131]

9. Webber L, Hill C, Saxton J, Van Jaarsveld CH, Wardle J. Eating behaviour and weight in children. Int J Obes (Lond). 2009; 33(1):21–8. Epub 2008/11/13. [PubMed: 19002146]

10. Drabman RS, Cordua GD, Hammer D, Jarvie GJ, Horton W. Developmental trends in eating rates of normal and overweight preschool children. Child Development. 1979; 50(1):211–6. Epub 1979/03/01. [PubMed: 446206]

11. Llewellyn CH, van Jaarsveld CH, Boniface D, Carnell S, Wardle J. Eating rate is a heritable phenotype related to weight in children. American Journal of Clinical Nutrition. 2008; 88(6): 1560–6. Epub 2008/12/10. [PubMed: 19064516]

12. Wardle J, Carnell S. Appetite is a Heritable Phenotype Associated with Adiposity. Ann Behav Med. 2009 Epub 2009/09/05.

13. Carnell S, Wardle J. Appetite and adiposity in children: evidence for a behavioral susceptibility theory of obesity. American Journal of Clinical Nutrition. 2008; 88(1):22–9. Epub 2008/07/11. [PubMed: 18614720]

14. van Jaarsveld CH, Llewellyn CH, Johnson L, Wardle J. Prospective associations between appetitive traits and weight gain in infancy. Am J Clin Nutr. 2011; 94(6):1562–7. Epub 2011/11/11. [PubMed: 22071702]

15. Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, et al. Early life risk factors for obesity in childhood: cohort study. BMJ. 2005; 330(7504):1357. Epub 2005/05/24. [PubMed: 15908441]

16. Whitaker KL, Jarvis MJ, Beeken RJ, Boniface D, Wardle J. Comparing maternal and paternal intergenerational transmission of obesity risk in a large population-based sample. Am J Clin Nutr. 2010 [epub]. Epub 2010/04/09.

17. Griffiths LJ, Hawkins SS, Cole TJ, Dezateux C. Risk factors for rapid weight gain in preschool children: findings from a UK-wide prospective study. Int J Obes. 2010 advance online publication 2 February 2010.

18. Morgan PJ, Okely AD, Cliff DP, Jones RA, Baur LA. Correlates of objectively measured physical activity in obese children. Obesity (Silver Spring). 2008; 16(12):2634–41. Epub 2008/10/18. [PubMed: 18927553]

19. Wardle J, Guthrie C, Sanderson S, Birch L, Plomin R. Food and activity preferences in children of lean and obese parents. Int J Obes Relat Metab Disord. 2001; 25(7):971–7. Epub 2001/07/10. [PubMed: 11443494]

20. Ostbye T, Krause KM, Brouwer RJ, Lovelady CA, Morey MC, Bastian LA, et al. Active Mothers Postpartum (AMP): rationale, design, and baseline characteristics. J Womens Health (Larchmt). 2008; 17(10):1567–75. Epub 2008/12/04. [PubMed: 19049350]

21. Ostbye T, Krause KM, Lovelady CA, Morey MC, Bastian LA, Peterson BL, et al. Active Mothers Postpartum: a randomized controlled weight-loss intervention trial. Am J Prev Med. 2009; 37(3): 173–80. Epub 2009/07/15. [PubMed: 19595557]

22. Cohen, J. Statistical power analysis for the behavioral sciences. L. Erlbaum Associates; 1988.

23. Wardle J, Guthrie CA, Sanderson S, Rapoport L. Development of the Children's Eating Behaviour Questionnaire. J Child Psychol Psychiatry. 2001; 42(7):963–70. Epub 2001/11/06. [PubMed: 11693591]

24. Jaddoe VW, van Duijn CM, van der Heijden AJ, Mackenbach JP, Moll HA, Steegers EA, et al. The Generation R Study: design and cohort update until the age of 4 years. Eur J Epidemiol. 2008; 23(12):801–11. Epub 2008/12/23. [PubMed: 19101808]

25. Jaddoe VW, van Duijn CM, van der Heijden AJ, Mackenbach JP, Moll HA, Steegers EA, et al. The Generation R Study: design and cohort update 2010. Eur J Epidemiol. 2010; 25(11):823–41. Epub 2010/10/23. [PubMed: 20967563]

26. Ashcroft J, Semmler C, Carnell S, van Jaarsveld CH, Wardle J. Continuity and stability of eating behaviour traits in children. Eur J Clin Nutr. 2008; 62(8):985–90. Epub 2007/08/09. [PubMed: 17684526]

27. Centers for Disease Control and Prevention. A SAS Program for the CDC Growth Charts. Apr 7. 2010 Available from: http://www.cdc.gov/nccdphp/dnpao/growthcharts/resources/sas.htm

28. Ziegler P, Briefel R, Clusen N, Devaney B. Feeding Infants and Toddlers Study (FITS): development of the FITS survey in comparison to other dietary survey methods. J Am Diet Assoc. 2006; 106(1 Suppl 1):S12–27. Epub 2005/12/27. [PubMed: 16376627]

29. Moshfegh AJ, Rhodes DG, Baer DJ, Murayi T, Clemens JC, Rumpler WV, et al. The US Department of Agriculture Automated Multiple-Pass Method reduces bias in the collection of energy intakes. Am J Clin Nutr. 2008; 88(2):324–32. Epub 2008/08/12. [PubMed: 18689367]

30. Ohlin A, Rossner S. Maternal body weight development after pregnancy. Int J Obes. 1990; 14(2): 159–73. Epub 1990/02/01. [PubMed: 2341224]

31. Krause KM, Lovelady CA, Ostbye T. Predictors of breastfeeding in overweight and obese women: data from Active Mothers Postpartum (AMP). Matern Child Health J. 2011; 15(3):367–75. Epub 2010/09/08. [PubMed: 20821042]

32. Holmbeck GN. Post-hoc probing of significant moderational and mediational effects in studies of pediatric populations. J Pediatr Psychol. 2002; 27(1):87–96. Epub 2001/12/01. [PubMed: 11726683]

33. Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007–2008. JAMA. 2010; 303(3):242–9. Epub 2010/01/15. [PubMed: 20071470]

34. Gungor DE, Paul IM, Birch LL, Bartok CJ. Risky vs rapid growth in infancy: refining pediatric screening for childhood overweight. Arch Pediatr Adolesc Med. 2010; 164(12):1091–7. Epub 2010/12/08. [PubMed: 21135336]

35. Toschke AM, Grote V, Koletzko B, von Kries R. Identifying children at high risk for overweight at school entry by weight gain during the first 2 years. Arch Pediatr Adolesc Med. 2004; 158(5):449–52. Epub 2004/05/05. [PubMed: 15123477]

36. Schachter S, Gross LP. Manipulated time and eating behavior. Journal of Personality and Social Psychology. 1968; 10(2):98–106. [PubMed: 5725908]

37. Wardle J, Carnell S, Haworth CM, Farooqi IS, O'Rahilly S, Plomin R. Obesity associated genetic variation in FTO is associated with diminished satiety. J Clin Endocrinol Metab. 2008; 93(9): 3640–3. Epub 2008/06/28. [PubMed: 18583465]

38. Jacobson P, Torgerson JS, Sjostrom L, Bouchard C. Spouse resemblance in body mass index: effects on adult obesity prevalence in the offspring generation. Am J Epidemiol. 2007; 165(1): 101–8. Epub 2006/10/17. [PubMed: 17041131]

39. Carnell S, Wardle J. Measuring behavioural susceptibility to obesity: validation of the child eating behaviour questionnaire. Appetite. 2007; 48(1):104–13. Epub 2006/09/12. [PubMed: 16962207]

40. Kuhl ES, Clifford LM, Stark LJ. Obesity in Preschoolers: Behavioral Correlates and Directions for Treatment. Obesity (Silver Spring). 2011 Epub 2011/07/16.
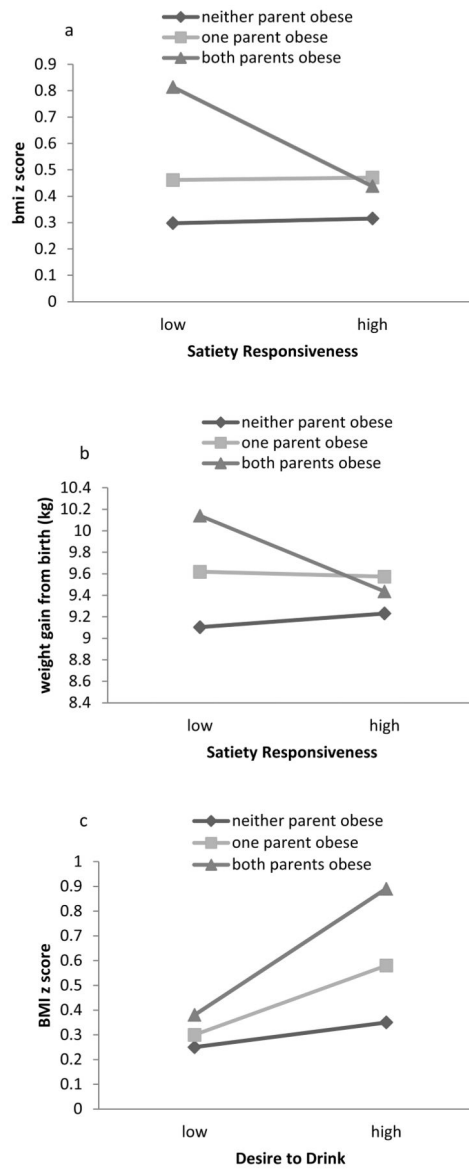
**Figure 1.**
(a, b, c). Weight gain from birth and BMI z score for children high and low on select appetitive traits with neither parent obese, one parent obese, or two parents obese

**Table 1**

Sample characteristics (percentages or means and standard deviations)

| | Overall sample (n = 213) | | |
|---|---|---|---|
| **PARENT CHARACTERISTICS** | | | |
| Mother's age, mean (s.d.) | 33.6 | | 5.1 |
| Partner's age, mean (s.d.) | 35.1 | | 5.7 |
| Mother's Education Level | | | |
| < HS, HS/GED, Voc Degree, Some College | | 30% | |
| Associates Degree, College Graduate or Higher | | 70% | |
| Partner's Education Level | | | |
| < HS, HS/GED, Voc Degree, Some College | | 36% | |
| Associates Degree, College Graduate or Higher | | 64% | |
| Mother's Measured BMI, mean (s.d.) | 32.3 | | 7.3 |
| Mother's Measured BMI (category) | | | |
| Normal weight | | 11% | |
| Overweight | | 33% | |
| Obese | | 56% | |
| Partner's Reported BMI, mean (s.d.) | 28.3 | | 5.0 |
| Partner's Reported BMI (category) | | | |
| Normal weight | | 27% | |
| Overweight | | 42% | |
| Obese | | 31% | |
| **CHILD CHARACTERISTICS** | | | |
| Gender % female | | 44% | |
| Race | | | |
| White | | 64% | |
| Black | | 36% | |
| Age, mean (s.d.) | 24.2 | | 1.3 |
| Gestational age (weeks), mean (s.d.) | 38.5 | | 2.0 |
| Lactation score, mean (s.d.) | 9.7 | | 8.8 |
| Birthweight (kg), mean (s.d.) | 3.4 | | 1.3 |
| 24 month weight (kg), mean (s.d.) | 5.9 | | 0.7 |
| weight gain from birth (kg), mean (s.d.) | 9.5 | | 1.5 |
| 24 month BMI z score, mean (s.d.) | | | |
| BMI Categories | | | |
| < 85th %ile | | 75% | |
| 85th – 95th %ile | | 16% | |
| >95th %ile | | 9% | |
| Average kcal, mean (s.d.) | 1221 | | 296 |
| Average percent calories from fat, mean (s.d.) | 31.0 | | 6.4 |

**Table 2**

Correlations between children's appetitive traits, BMI z score, parent's BMI, and child energy intake

| | EF | FR | DD | SR | BMI z | Weight gain | Mother's BMI | Partner's BMI |
|---|---|---|---|---|---|---|---|---|
| 1 Enjoyment of Food (EF) | -- | | | | | | | |
| 2 Food Responsiveness (FR) | 0.52** | -- | | | | | | |
| 3 Desire to Drink (DD) | −0.02 | 0.21** | -- | | | | | |
| 4 Satiety Responsiveness (SR) | −0.58** | −0.49** | 0.08 | -- | | | | |
| 5 BMI z score | 0.17** | 0.20** | 0.14* | −0.20** | -- | | | |
| 6 Weight gain from birth (kg) | 0.10 | 0.16 | 0.14* | −0.17* | 0.74* | -- | | |
| 7 Mother's BMI | −0.11 | −0.05 | 0.06 | 0.04 | 0.07 | 0.12 | -- | |
| 8 Partner's BMI | −0.01 | 0.01 | 0.13 | 0.00 | 0.14* | 0.17* | 0.19** | -- |
| 9 Average energy intake | 0.14 | 0.18* | 0.13 | −0.17 | 0.10 | 0.13 | 0.02 | 0.08 |

*
p <.05;

**
p < .01

**Table 3**

Association between parental obesity and anthropometrics

| Parent obesity status (n = 204)[1] | Weight gain from birth (kg) | | BMI z score | | 85th %ile vs <85th %ile | | 95th %ile vs <85th %ile | |
| | Mean[2] (s.e.) | 95% CI | Mean[3] (s.e.) | 95% CI | AOR[b] | 95% CI | AOR[b] | 95% CI |
|---|---|---|---|---|---|---|---|---|
| Neither Obese (n=79) | 9.2 (0.16) | 8.86 – 9.49 | 0.3 (0.11) | 0.10 – 0.51 | | | | |
| One Parent Obese (n=73) | 9.6 (0.15) | 9.29 – 9.90 | 0.5 (0.09) | 0.29 – 0.64 | 1.78 | .69 – 4.56 | 1.10 | .30 – 4.01 |
| Both Parents Obese (n=53) | 9.9 (0.23) | 9.46 – 10.34* | 0.7 (0.15) | 0.39 – 0.97* | 2.55 | .97 – 6.74 | 2.40 | .70 – 8.17 |

[1] Sample size is 204 due to missing birthweight data

[2] Unadjusted means shown with significance tests based on mean comparison adjustment for birthweight and intervention status with neither obese as referent

[3] Odds ratio adjusted for birthweight and intervention status.

*
p<.05,

**
p<.01,

**Table 4**

Multivariate linear regression analyses for CEBQ subscales, parental obesity, and interaction predicting weight gain (kg) from birth, BMI z score, and energy intake

| CEBQ subscale | Weight Gain from Birth (kg) | | BMI z score | | Energy Intake | |
|---|---|---|---|---|---|---|
| | Beta | (s.e.) | Beta | (s.e.) | Beta | (s.e.) |
| Intercept | 9.00 | (3.99) | −0.77 | (2.22) | 1601.34 | (737.45) |
| Enjoyment of Food (EF) | 0.13 | (0.16) | 0.20 | (0.11) | 33.66 | (29.73) |
| One Parent Obese | 0.29 | (0.23) | 0.12 | (0.16) | −25.55 | (52.33) |
| Both Parents Obese | 0.61 | (0.26)* | 0.32 | (0.18) | 70.07 | (56.95) |
| One Parent Obese × EF | ... | | ... | | ... | |
| Both Parents Obese × EF | ... | | ... | | ... | |
| Intercept | 8.33 | (3.92) | −1.31 | (2.13) | 1399.15 | (689.67) |
| Food Responsiveness (FR) | 0.23 | (0.19) | 0.25 | (0.10)* | 70.92 | (37.30) |
| One Parent Obese | 0.26 | (0.23) | 0.10 | (0.15) | −46.04 | (49.68) |
| Both Parents Obese | 0.60 | (0.27)* | 0.29 | (0.18) | 70.02 | (56.74) |
| One Parent Obese × FR | ... | | ... | | ... | |
| Both Parents Obese × FR | ... | | ... | | ... | |
| Intercept | 8.34 | (4.12) | −0.60 | (2.39) | 1479.96 | (737.55) |
| Desire to Drink (DD) | 0.12 | (0.11) | −0.15 | (0.06) | 23.79 | (25.27) |
| One Parent Obese | 0.25 | (0.23) | −0.74 | (0.53) | −50.11 | (49.53) |
| Both Parents Obese | 0.57 | (0.27)* | −1.25 | (0.67) | 59.84 | (56.56) |
| One Parent Obese × DD | ... | | 0.26 | (0.15) | ... | |
| Both Parnes Obese × DD | ... | | 0.46 | (0.18)** | ... | |
| Intercept | 9.50 | (3.78) | −0.66 | (2.27) | 1855.46 | (734.94) |
| Satiety Responsiveness (SR) | −0.10 | (0.29) | 0.12 | (0.19) | −83.80 | (43.61) |
| One Parent Obese | −0.04 | (1.53) | 0.88 | (1.00) | −30.84 | (49.94) |
| Both Parents Obese | 3.67 | (1.50)* | 3.16 | (1.02) | 79.21 | (56.29) |
| One Parent Obese × SR | 0.11 | (0.49) | −0.24 | (0.32)** | ... | |
| Both Parents Obese × SR | −1.04 | (0.50)* | −0.96 | (0.33)** | ... | |

Models include child age, gender, race, gestational age, birth weight, lactation score, mother's and partner's education level, mother's and partner's age, and intervention status. Ellipses indicate nonsignificant interaction terms, with only main effects reported

*
p<.05,

**
p<.01