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Parents' genetic attributions for children's eating behaviors: Relationships with beliefs, emotions, and food choice behavior

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Abstract

Considering genetic influences on children's eating behavior could result in reduced self-efficacy for healthy child feeding and less healthy feeding behavior among parents. Indeed, one's eating behaviors are typically thought of as the volitional aspects of weight management that one can directly control. The current study assessed parental genetic attributions for their child's eating behavior, and relationships between these attributions and self-efficacy, guilt, and feeding behaviors. Participants included 190 parents of a child between 4–7 years old. Parents' genetic attributions for child eating behaviors were lower than genetic attributions related to child weight. Higher genetic attributions for child eating behaviors were related to lower self-efficacy for feeding the right amounts of food, higher-calorie food choices for the child in a virtual reality-based buffet simulation, and higher levels of guilt. The current findings suggest that heightened beliefs about role of genetics in children's EB is associated with maladaptive affect and behavior among parents. This should be kept in mind when considering whether, when, and how to provide information to parents highlighting the role of genetics in children's eating behavior.

Keywords

eating behavior; feeding behavior; genetics; parent; child

1. Introduction

Evidence continues to accrue that behavioral tendencies related to eating and diet are, in part, inherited. Genetic factors underlie specific eating behaviors (EB) that contribute to food

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Author contributions:

SP conceived of and planned the analysis. SP and HY conducted the analysis. SP and HY wrote the manuscript. Both authors have approved the final article.

Ethics statement

The study reported here was reviewed by the IRB of the National Human Genome Research Institute. Participants gave informed consent before taking part.

The authors have no conflicts to report.

intake, such as one's food preferences and tendency to eat when not hungry [1–4]. While the concept that genes influence one's weight or obesity risk is increasingly well-known in clinical and lay community settings [5–7], genetic influences on EB are less commonly considered [8]. Given future visions of employing genomic approaches for weight management [9], communication about the influence of genetics on EB may one day become part and parcel of weight interventions. As such, it will be important to understand the potential cognitive, affective and behavioral ramifications of making genetic attributions for EB.

Past work suggests that there may be cause for concern that communicating about the connection between genetics and EB could result in fatalistic attitudes wherein children's unhealthful eating feels inevitable and uncontrollable. To the extent that individuals believe that genes underlie their own EB, they are also more likely to report reduced self-efficacy for weight management [8]. This is consistent with the notion that one's EB are typically thought of as the volitional aspects of weight management that one can directly control [10]. Thus, considering potential genetic (and therefore unalterable) influences on EB could disrupt a primary route of intended weight control. If these notions do indeed lead to fatalism, this would signal the need for special care in crafting communication approaches to accompany future genomics-based efforts to address weight management and rising rates of obesity.

Thus far, there is very little literature addressing the influence of genetic causal beliefs with respect to EB, and all centers on self-oriented beliefs and attitudes among adults. In contrast, it is often recommended that dietary interventions start in childhood, especially for children who are at risk of obesity later in life [11, 12]. As such, intervention strategies to improve parental feeding behavior for their young children have become a focus of attention as new interventions are developed [13]. For this reason, it will be crucial to understand how parents interpret the concept of genetic influence on children's EB and whether these notions are subject to fatalistic interpretations.

While there are no known studies that address parents' interpretations of genetic factors that influence their children's EB, there is limited research regarding parents' reactions to weight-related genomic information about their children, and findings are mixed. One study found that provision of genomic obesity risk information to parents of young children may be a promising strategy for improving parental feeding behavior; here, parents who received genomics-based information about their child's risk for obesity in adulthood chose to feed fewer calories to their child than control [14]. However, other work has suggested that mass media-style information about genetic influences on children's weight has little influence on parents' obesity risk perception for their child. In fact, a previous study found that providing information about the interaction between genes and the family home environment to parents of children with overweight was associated with disengagement from and rejection of an obesity risk message [15].

The dearth of research regarding the correlates of causal attributions for children's EB is problematic, as these correlates will be important to understand. First, parents typically oversee feeding and weight management efforts directed at their children, and therefore will

be the primary recipients of any genomic information concerning their child's propensities and risks for later life. Second, previous findings related to genetic causal attributions for obesity indicate that parents' beliefs about the genetic underpinnings of children's EB may be associated with parental behaviors aimed at weight management for the child. Indeed, as posited by Weiner's attribution theory [16, 17], perceived control over conditions or traits – including beliefs about the extent of their genetic underpinnings – are important predictors of the extent to which individuals may be willing to exert effort in this domain in the future. Finally, attributions are also linked with harmful versus helpful attitudes and behavior associated with those conditions and traits [18, 19]. Therefore, it will be essential to understand whether the causal attributions under study will be related to fatalistic attitudes and/or a reduction in self-efficacy for engaging in healthy child feeding behavior. A reduction in self-efficacy would be expected to lead to decrements in healthful child feeding.

Second, parents pass down genetic propensities related to EB to their children. Therefore, receipt of information about these propensities could influence parents' emotional states in addition to their child-focused beliefs and behavior. Previous work has shown that providing weight-related genomic information to parents about their children may result in altered feelings of guilt [20], and parents frequently feel guilt associated with transmission of genetic risk for overweight and obesity specifically [21–23]. It follows that beliefs among parents that they have passed down eating-related behavioral tendencies to their child may be associated with guilt, particularly to the extent that parents are dissatisfied with their own EB. Guilt itself is experienced as negative emotion, and it is yet unsettled as to whether and how guilt operates downstream to influence parent feeding behavior [21, 22]; however, given its salience in prior work, it will be an important emotion to explore in this context.

The current report elucidates the nature of parental genetic attributions for their children's EB, as well as the potential affective, cognitive, and behavioral correlates of those attributions. We do so by exploring data collected within a larger trial. Here, we assess parents' genetic and structural environment attributions for children's EB, as well as their food choice behavior and cognitive and affective outcomes (i.e., self-efficacy and guilt). Structural environment causal attributions (hereafter referred to as simply "environmental" attributions) serve as a primary point of comparison with genetic attributions, as environmental causes are typically more commonly top-of-mind with respect to EB and have been used as a comparison in previous work [8].

This study is exploratory and hypothesis-generating, and as such we did not enter the analysis with specific hypotheses. Rather, we proposed three research questions: 1) How do levels of genetic attributions for children's EB compare with other attributions, specifically, environmental attributions for EB, and environmental and genetic attributions for weight in general? 2) What parental characteristics (e.g., demographic factors, beliefs) are associated with higher genetic attributions for children's EB? 3) How are parents' genetic attributions for their child's EB associated with behavioral (feeding-related), cognitive, and affective variables?

2. Method

2.1 Participants

Participants included 190 parents (66% mothers) of all weight statuses, and with a biological "index child" that they were instructed to consider throughout the study. The index child was a child within the defined age range of 4 and 7 years old with no major food allergies or dietary-related health conditions. If participants had more than one child who fit this description, the index child was the child with the closest birthday date (in the case of twins, the child the parent named first was selected). Participants were recruited by online and newspaper advertisements, flyers, from databases of individuals interested in research, and by word of mouth. All participants gave informed consent for the study and were compensated $60 for their participation. All study activities were approved by the IRB of the National Human Genome Research Institute. See the larger study for more information on additional exclusion criteria and other details [24].

2.2 Procedure

The purpose of the larger trial was to assess the interactive influences of emotion and framing of messages about fruit and vegetable feeding for children. Messages were framed to emphasize either potential gains or losses associated with feeding fruits and vegetables to children, and parents were induced to either an angry or fearful emotional state. Thus, every participant received the same substantive information, though the framing differed by experimental condition. For more detail about these materials, please see [24]. The primary behavioral variable of interest in the original study was servings of fruits and vegetables chosen for one's child, which was assessed using a validated virtual reality (VR)-based feeding measure called the VR Buffet [25].

A pre-test questionnaire was administered online. Participants then attended a lab visit where they learned how to use the VR Buffet and received experimental manipulations (emotion induction and framed message), each of which were followed by short manipulation-check questionnaires. Participants then selected food for their index child in the VR Buffet and filled out a final questionnaire. Data for the current analysis were drawn from the pre-test questionnaire, food choice behavior in the VR Buffet, and the final questionnaire that followed the VR Buffet. For all variables that followed experimental manipulations, we controlled for experimental condition in analyses.

2.3 Materials

2.31 Pre-test questionnaire.—Measures from the pre-test questionnaire included demographics as well as parents' beliefs about the role of genetic and environmental factors in children's EB. Items read: "Consider your child's eating behaviors. These include all the ways in which s/he eats, such as what s/he prefers to eat and the reasons s/he makes choices about what foods to eat. Please use the scale below to indicate the extent to which you agree or disagree that [genetic/environmental] factors cause or contribute to your child's eating behaviors" (1=*strongly disagree*, 7=*strongly agree*). Environmental factors were further described as "e.g., availability of healthy foods in our community" while genetic factors were not described further. Parents' attributions for child weight were assessed with single

items which read: “Use the scale below to indicate the extent to which you agree or disagree that [genetic/environmental] factors cause or contribute to your child’s risk for becoming overweight” (1=*strongly disagree*, 7=*strongly agree*). Environmental factors were further described as “e.g., availability of walking paths, availability of healthy foods in our community” while genetic factors were not described further. These items were presented in the context of other causal attributions items (physical activity, family home environment, chance). As such, participants were also considering these other causes, but they were not a focus of the current report.

2.32 Behavioral assessment.—Parents’ food choices for their child were measured using the VR Buffet tool (described in detail elsewhere [14, 25]). Briefly, parents were asked to create a lunch for their child from a VR-based buffet restaurant by selecting from among several foods and drinks and placing servings on a tray. Foods available represented a range of nutrient and calorie densities, and all were palatable to children. The servings of food on the virtual plate were translated into calorie counts based on nutrient databases. The VR Buffet has been validated in previous work relative to real-world food choice behavior among parents [25]. The variable used in the current report was the total calorie count of all food and drink chosen by the parent in the VR Buffet.

2.33 Post-test questionnaire.—Measures administered after the VR Buffet included parents’ self-efficacy regarding ability to serve their child a) healthy food and b) the right amount of food. These were each assessed by a single item on a 1–7 scale (1=*not at all confident*, 7=*very confident*) [26]. Single items were analyzed because other self-efficacy scale items related specifically to fruit and vegetable feeding, and therefore were not relevant to the current research questions. We also assessed parents’ feelings of guilt about a) child feeding and b) passing down genetic risk for obesity. Each was measured with a single item (“I feel guilty about [index child]’s current eating habits in general” and “I feel guilty about the genetic risk for overweight that I may have passed down to [index child]”; 1=*strongly disagree*, 7=*strongly agree*). Finally, we assessed parents’ interest in genetic testing for their child’s obesity risk with a single item (“How interested would you be in learning about a genetic test that could give information about [index child]’s future genetic risk for obesity?”; 1=*not at all interested*, 7=*very interested*). This variable indicates parents’ orientation toward information seeking regarding their child’s potential predispositions.

2.4 Data Analysis

The analytic plan for this work was specified prior to beginning analysis and no data-driven analyses were performed. To assess the differences between genetic and environmental causal attributions for EB and overweight, we conducted paired-samples t-tests. To assess the relationship between demographic variables/parents’ perceptions and EB- and weight-related causal attributions held by parents, we conducted linear regressions. These included all variables of interest, outlined above, in addition to the interaction term between parents’ perceived family history of overweight and their perception of the child’s weight status; we included this particular interaction because it was a predictor of adults’ genetic causal attributions for EB in previous work [8]. To assess relationships between genetic and environmental causal attributions and cognitive, affective, and behavioral correlates, we

conducted linear regressions including both genetic and environmental variables in the model, as well as several covariates. We assessed significance at $p<.05$.

3. Results

3.1 Demographics

Characteristics of the sample and of the index children are reported in Table 1.

3.2 Descriptive statistics and comparisons

See Table 2 for descriptive statistics on all variables. Parents' genetic attributions for their child's EB were lower than environmental attributions for EB, $t(188)=7.19$, $p<.001$. In addition, genetic attributions for EB were lower than genetic attributions for overweight, $t(188)=0.958$, $p<.001$. Environmental attributions for EB and overweight did not significantly differ.

Correlations showed that genetic attributions for EB were significantly positively correlated with environmental attributions for EB ($r=0.232$, $p<.0001$), genetic attributions for overweight ($r=0.37$, $p<.0001$), and environmental attributions for overweight ($r=0.19$, $p=.008$). Environmental attributions for EB were correlated with genetic attributions for overweight ($r=0.36$, $p<.0001$) and environmental attributions for overweight ($r=0.48$, $p<.0001$). Finally, genetic and environmental attributions for overweight were significantly positively correlated ($r=0.43$, $p<.0001$).

Causal attributions' associations with demographics and perceptions—Linear regression results are available in Table 3. In all, genetic attributions for EB were significantly related to index child age and gender, such that genetic attributions were higher for female children and older children. Environmental attributions for EB were related only to parent education, such that more highly-educated parents reported higher environmental attributions.

Genetic attributions for overweight were related to parent education and index child age, such that more highly-educated parents and those with older children reported higher attributions. Finally, environmental attributions for body weight were related to higher parental education alone.

3.3 Causal attributions' associations with intervention-relevant affect and behaviors

Results of all regression analyses are available in Table 4. Linear regressions showed that higher genetic attributions for children's EB were associated with less self-efficacy regarding ability to feed one's child the right amount of food, higher calorie count chosen in the VR Buffet, greater guilt about child eating habits, and greater guilt about passing down genes that increase child risk for overweight. The unstandardized regression coefficient for calories chosen in the buffet was B=32.25, indicating that with each one-unit increase on the genetic attribution for EB scale, parents selected an average of 32 more calories in the VR buffet, holding all covariates constant. Environmental attributions for EB were entered into

the same model and were positively associated with self-efficacy regarding ability to feed one's child healthy food.

Genetic attributions for children's obesity risk were positively associated with calorie count chosen in the VR Buffet and with guilt related to passing down genes that increase one's child's obesity risk. The unstandardized regression coefficient for calories chosen in the buffet was B=44.13, indicating that with each one-unit increase on the genetic attribution for weight scale, parents selected an average of 44 more calories in the VR buffet, holding all covariates constant. Environmental attributions for weight within the same model were not associated with any other variables.

4. Discussion

To more successfully address rising obesity rates and increase health-promoting behavior, new approaches are needed that target weight gain prevention in young children, ostensibly via their parents. Precision medicine offers potential promise in this regard, however, optimizing delivery of these approaches will require understanding the correlates of the genomics-based beliefs that would follow from such interventions among parents. The current findings suggest that heightened beliefs about role of genetics in children's EB is associated with maladaptive affect and behavior among parents, including lower self-efficacy and higher-calorie feeding. This presents important implications for considering whether, when, and how to provide information to parents highlighting the role of genetics in children's eating behavior.

In the current study, parents reported genetic causal attributions for EB at approximately scale midpoint, suggesting that, on average, parents may be unsure or ambivalent about the extent to which EB are influenced by genetic makeup. This is consistent with previous research among adults, wherein participants did not find EB to be a salient target of genetic influence [8]. In addition, parents' genetic attributions for EB were lower than all other types of attributions that were examined here: environmental attributions for EB and weight, as well as genetic attributions for weight. These patterns make sense as genetic influences on EB are infrequently discussed in the media, in healthcare, or in the public sphere. For many individuals, this link may be a new concept.

In this analysis, given our primary interest in genetic attributions, we assessed only one comparison causal factor and chose environment to be that factor, as was the case in existing, related work [8]. Attributions for these two factors were assessed alongside three other causes (physical activity, family environment, and chance), likely prompting participants to consider causality through a wider lens that encompassed more than the two causal factors investigated here. Gaining a fuller sense of these attribution patterns, including their interactions (e.g., gene-environment interactions) will be an important step for future work.

Although parents may have been mixed or unsure about the notion that genetics contribute to their child's EB, to the extent that parents were more supportive of this supposition, this was associated with lower self-efficacy regarding ability to feed one's child the right amount

of food, and choice of greater calorie counts in the VR Buffet. Genetic causal attributions for one's child's general body weight were also related to greater calorie counts chosen in the VR Buffet. These findings stand in contrast to literature demonstrating that linking genetics with weight more generally is *not* associated with fatalism or reduced self-efficacy [27]. That literature is, however, almost entirely self-focused rather than child-focused. Notably, the current study is the first to measure actual behavior in relation to genetic attributions for EB.

It is also notable that beliefs about genetic influences on child EB are related to parents' food choices for the child. Our findings may be explained in several ways. First, parents may have increased genetic attributions for EB when their child has poorer dietary habits or preferences, and parents may have selected foods commensurate with those preferences and propensities. In other words, parents' genetic attributions for EB may be a *consequence* of the child's poor habits. Second, parent choices may be influenced by their own food preferences and eating tendencies, which are often correlated with their child's [28]. Finally, parents may believe that it is not worthwhile to restrict or more carefully curate food choices for their child if the child's genetically-underpinned, and therefore unalterable, EB propensities will win out in the end; this explanation would be consistent with the fatalistic attitudes described above. Future research is needed to disentangle these and other possible mechanisms.

Parents who attributed their child's EB more highly to genetics also exhibited increased guilt, regarding both their child's eating habits and the genetic predisposition for overweight that may have been passed down. Guilt is a negative, aversive experience, and in this sense can be considered an undesirable emotional state. However, guilt is also often considered to be a reparative emotion that can motivate positive behavior [29]. Work exploring the influence of parental guilt is mixed as to whether various types of guilt experiences are associated with positive versus negative versus no changes in child feeding behavior [21, 22, 30]. In the current analysis, guilt was correlated with choosing higher-calorie meals in the VR Buffet (data not shown). This finding, taken together with past work, identifies guilt as an important factor to consider in conjunction with parents' affect, behavior, and beliefs about the genetic underpinnings of children's EB. Given that these pathways are not yet well-understood, it will be essential to further explore the role of guilt in this context, and in parents' subsequent weight management outcomes for their children. Due to our study design, we were unable to disentangle the directional pathways through which genetic causal attributions for EB relate to cognitive, affective, and behavioral outcomes. Future research should employ longitudinal, experimental designs in order to further our understanding of the causal relations at play.

Research has not yet investigated the effect of presenting concepts relating genetics to children's EB to parents, however, the current report identifies groups for whom these notions may be more likely to preexist. Interestingly, characteristics of parents themselves do not relate to the magnitude of these beliefs. This is somewhat consistent with previous work in which the only predictor of holding genetic attributions for EB among adults was perception of weight and family history of overweight, in the absence of any other demographic or social predictors [8]. Here, genetic attributions for children's EB are

stronger among parents of girls and older children. The gender effect can be interpreted in light of findings that parents sometimes pay more attention to and are more thoughtful about the eating habits and behaviors of girls versus boys [30]. Furthermore, as children age, their unique patterns of eating, and how those patterns converge and diverge from those of the parent, may become more visible as direct parental influence begins to wane. We also aimed to assess the relationship between child weight status and parental EB beliefs, but because very few of the index children were perceived by their parents as being overweight, we were not able to effectively examine this. This will be important to pursue in future work, given the identified importance of child weight in processes like parent feeding behavior [31, 32]. Moving forward, when communicating to parents about genetic influences on EB, characteristics of the child under consideration may color response to these messages in that these notions may resonate more strongly when connected with girls and older children.

As with any study, the current report must be considered in light of its limitations. First, the current report considers preexisting beliefs about genetic factors in children's EB rather than assessing response to presentation of informational messages. It also considers genetic factors as separate from environmental ones although in reality these two forces interact to influence eating behavior. Future research should explore the influence of gene-environment-interaction beliefs for eating behaviors. Data were also collected in the context of a larger study on child fruit and vegetable feeding, and as such, these ideas were salient among parents as they completed post-manipulation assessments. Furthermore, this was a convenience sample and while diversity existed on some dimensions, it was lacking on others (e.g., educational attainment). There was also a very small proportion of parents whose index child was already overweight, and as such would be at greatest risk of obesity later in life. Single item measures were used for several measures and were often created de novo where suitable measures did not exist. Finally, as stated above, due to our study design we were unable to make formal claims regarding directionality and causality.

In all, the current report dovetails with previous work in demonstrating that individuals who more highly endorse genetic underpinnings of EB are also more likely to exhibit deleterious dietary beliefs and behaviors. Here, we have specifically shown this to be the case among parents considering genetic factors in their child's EB. Although this may seem disheartening, it also presents an opportunity to craft communication approaches that counter the potential for fatalism. By explicitly pointing out that genetic predispositions are only that – predispositions which can be addressed and potentially overcome, communication efforts could also direct parents toward more positive understandings of these relationships. Future work should consider how such approaches may be a useful adjunct to genomics-based approaches to weight management brought about by scientific and clinical advances.

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Table 1:

Participant demographics (N = 190).

Characteristic	***N*(%) or *M(SD)***
Parent gender: Female	126 (66.3%)
Parent weight status: Overweight/Very overweight	103 (54.2%)
Parent education: College graduate	151 (79.5%)
Parent race: White	88 (46.3%)
Parent race: Black	49 (25.8%)
Parent race: Asian	27 (14.2%)
Parent age	37.71 (5.73)
Index child gender: Female	86 (45.3%)
Index child weight status: Overweight/Very overweight	11 (5.8%)
Index child age	5.39 (1.15)
Number of children in family	2.0 (0.88)

Table 2:

Variable descriptive statistics.

Variable	*M(SD)*
Genetic attributions for eating behavior	3.90 (1.76)
Environmental attributions for eating behavior	5.10 (1.94)
Genetic attributions for overweight	5.23 (1.68)
Environmental attributions for overweight	5.05 (1.74)
Calories chosen in VR Buffet	730.76 (303.36)
Self-efficacy, right amount of food	5.76 (1.17)
Self-efficacy, healthy food	6.41 (.96)
Interest in genetic test	5.58 (1.97)
Guilt, child eating habits	2.93 (1.71)
Guilt, passing down genes for overweight	2.40 (1.76)

Note. Response scale for all variables, except calories chosen, is 1–7.

Table 3:

Linear regressions of causal attributions for EB and weight on demographic and perceptual variables.

	EB Attributions		Weight Attributions	
	Genetic	**Environmental**	**Genetic**	**Environmental**
Demographics				
Parent gender (f)	−0.02	−0.21	−0.18	0.13
Parent education (college +)	0.42	**1.31***	**0.74***	**0.73***
Number of biological children	−0.17	−0.04	0.23	−0.05
Index child gender (f)	**0.52***	0.13	0.37	0.43
Index child age	**0.29***	0.12	**0.23***	0.03
Perceptions				
Self-perception overweight (yes)	−0.22	−0.27	−0.25	−0.45
Perceived child overweight (yes)	−0.78	0.22	0.39	0.23
Family history of obesity	−0.25	0.33	0.53	0.37
Perceived child overwt X family history	0.73	−0.82	−0.53	0.04

Note. Unstandardized regression coefficients. Each column represents a separate regression model.

* p<.05

Table 4:

Linear regressions of intervention-relevant variables on genetic and environmental attributions for EB and weight.

	Self-efficacy; amount of food	Self-efficacy; healthy food	Calories in buffet	Interest in genetic test	Guilt; child eating habits	Guilt; genes
EB Attributions						
Parent gender (f)	0.21	0.12	−34.22	−0.35	0.27	0.47
Parent education (college +)	−0.26	−0.008	**−174.03** *	−0.36	−0.18	−0.07
Index child age	−0.04	−0.06	24.35	0.21	0.04	0.08
Index child gender (f)	**0.46** *	0.25	−1.94	**0.62** *	**−0.60** *	0.05
Self-perception overweight (yes)	−0.10	−0.06	34.55	**0.60** *	0.29	**1.13** *
Perceived child overweight (yes)	0.005	0.25	−14.57	0.48	0.83	**1.27** *
Study condition, emotion	0.08	0.02	40.79	0.37	−0.32	−0.31
Study condition, framing	−0.12	−0.17	−13.10	0.35	−0.18	−0.20
EB Genetic attributions	**−0.11** *	−0.05	**32.25** *	0.024	**0.18** *	**0.22** *
EB Env. attributions	0.05	**0.08** *	−11.63	−0.10	−0.11	−0.03
Weight Attributions						
Parent gender (f)	0.19	0.12	−27.49	−0.32	0.31	0.47
Parent education (college +)	−0.22	0.07	**−183.81***	−0.48	−0.27	−0.12
Index child age	0.04	−0.07	21.27	0.21	0.07	0.09
Index child gender (f)	**0.42** *	0.24	5.53	**0.62** *	**−0.54** *	0.07
Self-perception overweight (yes)	−0.07	−0.07	21.14	**0.63** *	0.33	**1.10** *
Perceived child overweight (yes)	−0.01	0.24	−6.28	0.47	0.78	**1.26** *
Study condition, emotion	0.04	0.03	59.28	0.29	−0.32	−0.22
Study condition, framing	−0.11	−0.17	−16.33	0.28	−0.21	−0.25
Weight Genetic attributions	−0.11	0.002	**44.13** *	0.04	0.11	**0.16** *
Weight Env. attributions	0.06	−0.01	−25.78	0.004	0.03	0.02

Note. Unstandardized regression coefficients. Columns within each section represent a separate regression model.

* p <.05