Genetic causal attributions for weight status and weight loss during a behavioral weight gain prevention intervention

Megan A. McVay, PhD[a,b], **Dori M. Steinberg, PhD**[c], **Sandy Askew, MPH**[c], **Kimberly A. Kaphingst, ScD**[d,e], and **Gary G. Bennett, PhD**[c,f]

[a]Center for Health Services Research, Durham VA Medical Center, Durham NC

[b]Department of Psychiatry and Behavioral Sciences, Duke University Medical Center, Durham NC

[c]Duke Global Digital Health Science Center, Duke Global Health Institute, Duke University, Durham NC

[d]Department of Communication, The University of Utah, Salt Lake City, UT

[e]Huntsman Cancer Institute, The University of Utah, Salt Lake City, UT

[f]Department of Psychology & Neuroscience, Duke University, Durham, NC

Abstract

Purpose—Emerging evidence suggests that attributing one's weight to genetics may contribute to the adoption of obesogenic behaviors. We examined if weight-related genetic attributions were associated with weight change during a weight gain prevention intervention.

Methods—Participants (n=185) were from a randomized clinical trial of a digital health weight gain prevention intervention for Black women age 25–44 with BMI 25.0–34.9kg/m^2. Weight-related genetic attributions (weight status attribution and weight loss attributions) were measured at baseline and 12 months.

Results—Among intervention participants, high genetic attribution for weight loss was associated with greater weight loss at 12 months (−2.7 kg vs 0.5 kg) and 18 months (−3.0 kg vs 0.9 kg). Among usual care participants, high genetic attribution for weight status was associated with greater 18-month weight gain (2.9 kg vs 0.3 kg). The intervention reduced likelihood of high genetic attribution for weight loss at 12 months (p=0.05). Change in likelihood of genetic attribution was not associated with weight change over 12 months.

Conclusion—Impact of genetic attributions on weight differs for those enrolled and not enrolled in an intervention. However, weight gain prevention intervention may reduce genetic attribution for weight loss.

Corresponding Author: Megan McVay, PhD, 508 Fulton St. (152), Durham NC 27705. Phone: 919-286-0411 x5691, Fax: 919-416-8025, ; Email: megan.mcvay@duke.edu

Keywords

Obesity; Prevention; Weight loss; Genetic determinism

INTRODUCTION

The causes of excess adiposity are multifactorial and include genetic, psychosocial, environmental, and physiological factors. The relative importance of these factors is unknown, leaving individuals to develop their own attributions for the causes of excess weight. Theoretical models suggest that individuals' causal attributions for weight may in turn exert an influence on their weight-related behaviors, and that causal attributions to genetics may be especially influential. For example, Attribution Theory posits that causal attributions have three dimensions: locus of control (internal vs. external), stability (changeable vs not changeable), and controllability (controllable vs. uncontrollable) [1]. Causal attributions that are perceived to be more internal, stable, and uncontrollable are associated with reduced engagement in goal-directed behavior [1]. Because genetic causal attributions tend to be internal, stable, and uncontrollable [2–3], they may contribute to suboptimal weight management behaviors.

Several studies have found deleterious effects of genetic attributions on health behaviors. For example, genetic causal attributions for lung cancer, hypertension, colon cancer, and skin cancer were associated with engaging in a greater number of risky health behaviors, such as cigarette smoking, consuming a poor quality diet, and not using sunscreen [4]. Specifically with regard to weight-related attributions and behaviors, a few observational studies have looked at associations between genetic attributions and weight-related behaviors, with one study not finding an association [5] and another finding a positive association [6]. In a study using an experimental design, researchers found that participants who were provided a genetics-based explanation for obesity consumed more food in a lab-based eating episode than those provided a psychosocial explanation [7]. On the whole, this existing literature suggests that greater genetic attributions for weight may contribute to lower likelihood to enact weight management behaviors, possibly contributing to less weight loss or to weight gain during a weight management intervention. However, the impact of genetic attributions on weight change during a weight management intervention has not yet been tested, to our knowledge.

In addition to a possible influence of genetic attributions on response to a weight management intervention, it is also possible that a weight management intervention could lead to changes in genetic causal attributions for weight. The Common Sense Model suggests that causal attributions influence perceptions of appropriateness of a particular treatment [8]. Specifically, a "symmetry heuristic" has been described, such that causal attributions that are biological in nature (including genetic) are associated with inferences that biologically-based treatments are appropriate, whereas non-biological causal attributions are associated with belief in the appropriateness of behavioral interventions [4, 8–9]. Although the existing data on the symmetry heuristic has focused on the possible effect of causal attributions on treatment preference and choice, the reverse relationship can

also be posited: that experience with particular treatments may alter causal attributions. However, this has not yet been examined in weight management.

In the current study, we sought to determine if baseline genetic causal attributions for weight status and weight loss were associated with weight change among participants enrolled in a weight gain prevention intervention or usual care. We hypothesized that greater genetic attributions would be associated with weight gain or less weight loss. We also examined whether a weight gain prevention intervention affected genetic attributions for weight status and weight loss. We hypothesized that involvement in a behavioral intervention might reduce genetic attributions for weight status and weight loss. These aims were examined as part of secondary data analyses from the Shape Program, a randomized controlled trial comparing a 12-month weight gain prevention intervention to usual care among premenopausal Black women attending community health center clinics ([10]). In that trial, the intervention successfully prevented weight gain at 12 months [mean difference (95% CI= −1.4 kg (2.8 to 0.1)] and at 18 months [mean difference (95% CI= −1.7 kg (3.3 to 0.2)].

METHODS

Participants

The Shape program trial is described in detail elsewhere ([10]–[11]). Participants were eligible if they were Black women aged 25 to 44 years, had a body mass index between 25 and 34.9 m/kg^2, and had the ability to read and write in English. Participants were excluded if they were pregnant or within 12 months postpartum, had a myocardial infarction or stroke in the previous 2 years, or had profound cognitive, developmental or psychiatric disorders. Patients were recruited from 6 community health centers in central North Carolina operated by Piedmont Health. Participants were required to have had at least 1 visit to the community health center in the past 24 months. Participants who appeared eligible via medical record abstraction were recruited with invitational brochures followed by a screening call for eligibility. Potentially eligible patients then attended an in-person baseline study visit. A total of 194 participants were recruited and randomized (see Consort in primary outcomes manuscript, 10). Patients were randomization via a computer program with equal allocation (1:1) across treatment arms. Study design precluded blinding of patients or study staff ([12]). After randomization, 3 participants in usual care and 6 in the intervention become ineligible (due to pregnancy, relocating, or cancer diagnosis) and were not included in these analyses. This trial was registered with clinicaltrials.gov and has the clinical trial registry identifier NCT00938535. The Duke University Institutional Review Board and the Piedmont Health Board of Advisors approved all study procedures. Informed consent was obtained from all subjects.

Usual Care

In addition to the current standard of care delivered by their providers, usual care participants received semi-annual newsletters covering topics unrelated to weight, nutrition or exercise, e.g., finances, health and beauty.

Intervention

The Shape intervention used the interactive obesity treatment approach (iOTA), which aims to produce energy deficits through modification of routine obesogenic behaviors (e.g., no sugary drinks, no fast food, walk 10,000 steps per day). Participants were assigned 3 tailored behavior change goals at study onset and these goals were updated every 2 months. Behavior change goals were selected by an algorithm based on participants' need for a specific behavior change, self-efficacy, readiness, and intended caloric deficit. Participants received weekly interactive voice response (IVR) calls during which they were asked to report their progress towards their goals and received tailored feedback (e.g., description of trends in progress, reinforcement, short skills training tips). Participants also received monthly counseling calls, lasting 20 minutes with registered dietitians trained in motivational interviewing principles. These calls focused on identification of barriers to behavior change, resolving ambivalence about behavior change, providing skills training, and goal setting. Calls were based on a script guided by a web-based application. Participants also received regular skills training materials such as tracking logs and were given free access to local YMCAs. Additional details on the Shape Program intervention are provided in Foley et al. 2012 ([11]).

Measures

Clinical measures—Weight and height were measured on calibrated wall-mounted stadiometer (Seca 214) and electronic scales (Seca Model 876), respectively. Measures were taken by trained research assistants with participants wearing hospital gowns.

Genetic attributions—Attributions for the role of genes in weight status, diabetes, and heart disease were assessed using a measure that has been used in a previous study ([12]). In the current study, to assess attributions for weight status, participants were asked "How much do you think a person's weight is caused by genes?" with the response options of *completely, mostly, somewhat, a little,* and *not at all.* Similar questions were asked with regard to perceived causes of heart disease and diabetes. Participants who endorsed *completely* or *mostly* for the role of genes were characterized as having "high genetic attribution" for weight status and those endorsing other response options were considered to have "low genetic attributions" for weight status.

An item was also developed for this study to assess participants' perceptions about the role of genes in their own weight loss. The item stated: "My genes play a role in whether or not I lose weight." Response options were *strongly agree, somewhat agree, neither agree nor disagree, somewhat disagree*, and *strongly disagree*. Those who endorsed *strongly agree* and *somewhat agree* were considered to have high genetic attribution for weight loss, and those who endorsed the other options were considered to have low genetic attribution for weight loss. Responses were dichotomized for both genetic attribution variables to reflect a conceptual distinction between perceptions of high and low attributions for the role of genetics in weight. ([12]).

Other psychosocial and behavioral measures—Psychosocial variables were measured at baseline. Body image was measured with the 14-item Figure Rating Scale, a

validated measure of body image that is designed to assess current and past body size as well as perceived attractiveness of body figure drawings ([13]). Eating characteristics were measured with the Three Factor Eating Questionnaire, which has three subscales: dietary restraint, uncontrolled eating (sometimes called disinhibited eating), and emotional eating ([14]). Physical activity self-efficacy was measured with five items assessing confidence in ability to exercise under various challenging circumstances ([15]). Moderate and vigorous physical activity was measured with a 6-item scale derived from the Behavioral Risk Factor Surveillance System ([16]). Participants also were asked to report if they had attempted weight loss in the past year (yes or no) and how much weight gain they expected in the next 2 years (0–5 lbs, 5–10 lbs, > 10 lbs).

Statistical Analyses

Analyses included 185 participants who remained eligible at 12 months. We initially conducted chi-squared or two-sided t-tests of means to compare those low and high in genetic attribution for weight and weight loss on the baseline psychosocial and behavioral variables of interest. We also conducted a spearman's rank correlation on uncollapsed genetic attribution variables to obtain an association between genetic attribution for weight status and weight loss, given the importance of these two variables. To test our first aim related to association between genetic attributions and weight change, we conducted a longitudinal mixed-effects model analysis with the dependent variable of weight and the independent variables of genetic attribution, time, and the interaction of time and genetic attribution. Separate analyses were conducted in the intervention and the usual care arms. Baseline age, education, and clinic site were included in the model as covariates. We used a restricted maximum likelihood estimation approach and specified a random intercept and unstructured covariance matrix. Next, we conducted that same analyses but with genetic attribution for weight loss as the variable of interest.

To achieve our second aim of determining if the treatment changed genetic attributions, we conducted a repeated measures analysis using generalized estimating equation (GEE) models with a binary distribution, logit link function, and an unstructured covariance matrix. The independent variables in these models were treatment arm (usual care or intervention), time (baseline and 12 months), and the interaction of time and treatment group. The dependent variable was an indicator of genetic attribution level (high versus low), with attributions for weight and weight loss tested separately. Intervention site was included in these models as a covariate. In post-hoc analyses to further probe our findings, we modeled the association between the change in weight and genetic attribution for weight loss over time, adjusting for site, and using a similarly specified GEE model. Because we were primarily interested in probing observations from the intervention session for this post-hoc analysis, we present findings only from that group, although results were similar in the usual care group and when results were pooled.

RESULTS

Descriptive data

Study participants had a mean age of 35.4 (SD=5.5). Mean weight at baseline was 81.1 (SD=8.8) kg and mean BMI was 30.2 kg/m^2 (SD=2.5). A majority of participants were employed (71.4%) and 26.5% were married or living with a partner. Most participants had an education level of less than a college degree (79.7%) and an annual income of less than $30,000 (74.3%). Metabolic syndrome criteria were met by 30.8% of participants. The correlation between genetic attribution for weight status and genetic attribution for weight loss was ρ=.26 (p=.0004).

Associations of baseline genetic causal attributions with other baseline characteristics

Across both study groups, high genetic causal attribution for *weight status* was associated with high genetic causal attribution for diabetes, heart disease, and weight loss (Table 1a). It was also associated with higher self-reported uncontrolled eating. High genetic causal attribution for *weight loss* was associated with lower physical activity self-efficacy and higher self-reported uncontrolled eating (Table 1b).

Associations between baseline genetic attributions and weight change

In the intervention arm, genetic causal attribution for *weight status* was not associated with weight change at any measurement time point (Table 2). However, high genetic causal attribution for *weight loss* was associated with greater weight loss at 12 and 18 months (Table 2).

In the usual care arm, high genetic causal attribution for *weight status* was associated with greater weight gain at 18 months (Table 2). However, genetic causal attribution for *weight loss* was not associated with weight change.

In order to determine if observed associations between baseline genetic attributions and weight change were independent of other related variables, we conducted sensitivity analyses in which we added as covariates to the model those variables that were significantly related to genetic attributions in the bivariate associations. Thus, we repeated the regression analyses described above; in weight status models, we adjusted for uncontrolled eating and in weight loss models, we adjusted for uncontrolled eating and physical activity self-efficacy. The addition of these covariates did not change the direction or significance of any of the relationships examined, nor were the covariates significant predictors of weight change in the model.

Effects of intervention on genetic attributions over time

For genetic attribution for *weight loss*, we observed a significant time by treatment group interaction, indicating differential change over time between treatment arms in genetic causal attributions for weight loss (chi-squared= 6.08, p=0.05). In particular, we found that, at baseline, the intervention and usual care groups did not differ on likelihood of high or low genetic causal attributions for weight loss, whereas at 12 months the intervention group participants were more likely to have low genetic causal attribution for weight loss

compared to usual care participants (Table 3 and Figure 1). No significant effects were observed for the likelihood of genetic attribution for *weight status*.

In post-hoc analyses we tested whether change in genetic causal attribution observed in the intervention group was associated with change in weight in that arm. We found that the odds of having low genetic causal attribution for weight loss did not change significantly with weight over time (p = .75). For every 5 kilograms of weight lost between baseline and 12 months, the odds of having a low genetic causal attribution for weight loss increased by 5% (OR: 1.05, 95% CI: 0.77, 1.45).

DISCUSSION

Contrary to our hypothesis, greater weight loss was observed during a weight gain prevention intervention among participants who believed that genes play a greater role in weight loss compared to participants who attributed a lesser role to genes. In contrast, among participants who did not receive a weight gain prevention intervention (usual care arm participants), believing that genes play an important role in weight status was associated with weight gain over an 18 month period. We also found that participation in the weight gain prevention intervention led to a decrease in attribution of weight loss to genes over time.

Previous research has provided initial evidence that genetic causal attributions for weight may contribute to obesogenic behaviors ([6]–[7]). However, few studies have attempted to establish if there is a relation between genetic causal attributions and outcomes in a behavioral weight management intervention. The current results suggest that higher genetic attributions for weight loss at baseline may contribute to greater weight loss for individuals enrolled in a weight gain prevention intervention. Notably, these effects were observed even after controlling for related concepts of uncontrolled eating and self-efficacy. One potential explanation we considered for this relation was that the intervention produced a reduction in genetic attributions, which in turn contributed to weight loss. Such an effect might be expected to have more benefit for those who start with higher genetic causal attributions, and thus could account for our observation. While this hypothesis was supported by our finding that the intervention reduced genetic causal attributions for weight loss, in further analyses, we found that changes in attributions were not associated with weight change in the intervention participants.

This study is the first, to our knowledge, to show that participation in a weight management intervention can change perceptions about genetic causality for weight. This finding is consistent with past research highlighting the symmetrical relationship between causal attributions and treatment approach choice ([8]). To the extent that the current intervention increased participants' sense of personal control over their weight, it may have led to a concurrent reduction in beliefs of the role of genes. While this finding suggests that change in genetic attribution may be an active ingredient in the intervention, this is not supported by our follow-up analyses, which showed no association between change in genetic attribution and change in weight. Notably, the present intervention had a weight gain prevention focus, and achieved small weight losses; it is unclear if results would differ in an intervention

focused on weight loss where larger changes in weight would be expected. Further research is needed to confirm these findings and to extend them to weight loss interventions.

The observation that higher baseline genetic attributions were associated with greater weight loss during the intervention might suggest that it would be effective to discuss with weight management intervention participants the influence of genes in weight loss, while still emphasizing the importance of behavior change. However, the observed lack of association between change in genetic attributions and change in weight does not lend support to the value of intervening on this variable, and additional study is needed before recommendations are warranted. Future studies should test the effects of messages focused on the role of genes in weight loss during weight management interventions.

In contrast to intervention participants, among usual care participants we observed that higher genetic causal attributions were associated with greater weight gain. In this study arm, it was genetic causal attributions for weight *status,* not weight *loss*, that were significantly associated with weight change. These results indicate that, in the absence of a weight management intervention, genetic causal attributions about weight may indeed promote weight gain. Limited past research has examined the impact of genetic attributions on longitudinal weight outcomes in any population. A study by Hilbert et al. ([5]) found that a measure of physiological/genetic attributions for obesity were not associated with weight change over a 6 month period; however, this study was limited by the confounding of genetic and non-genetic physiological factors and the short follow-up period. Results of the current study are consistent with previous experimental ([7]) and cross-sectional research ([6]) linking genetic attributions to weight gain promoting health behaviors. These results suggest that public health messages, and clinicians discussing weight with patients, should strive to focus on the importance of behavior change in weight control, especially for those who perceive genetic susceptibility.

In this study, we separately considered genetic attributions for weight status and for weight loss. Analyses showed that, although these variables were significantly related, they appear to be distinct constructs with differential relationships to weight change. Further study of how these two types of weight-related genetic attribution are related to one another and to other variables is warranted. Qualitative evaluations, in particular, might provide additional information on differences in how individuals perceive the role of genetics in their current weight status compared to the role of genetics in weight loss. Additionally, future studies focusing on genetic attributions related to weight may benefit from measuring these constructs separately.

At baseline, genetic attributions were associated with greater disinhibitions around eating. The reason for this association is unclear. It is possible that genetic attributions contribute to lower perceived control over eating. It is also possible that experiencing more difficulty controlling one's eating could contribute to the development of stronger genetic causal attributions. Additional longitudinal data may assist with clarifying the direction and causal nature of this relationship.

This study has several limitations. It is not known if results will generalize to individuals who differ from our study participants, who were premenopausal Black women in the primary care setting. However, this study population is an important one, as Black premenopausal women are at particularly high risk for weight gain. These findings also add to our understanding of causal attributions for weight status and weight loss in a study population with varying levels of educational attainment. Our analyses looking at the effect of genetic attributions on weight change are observational, and there may be unobserved confounding variables that are contributing to the association. We used single item measures of causal attributions. Additionally, this study focused specifically on perceptions about causality. A broader perspective that incorporates other constructs from theories that include causality (e.g., Common Sense Model, Attribution Theory) may provide additional predictive value for weight change.

In summary, we found that high genetic causal attributions for weight loss at baseline were associated with greater weight loss during a weight gain prevention intervention. However, among participants not enrolled in an intervention (usual care participants), high genetic attributions for weight status were associated with weight gain over an 18 month period. We also found that participation in a weight gain prevention intervention contributed to a reduction in likelihood of attributing weight loss to genes.

Acknowledgments

References

1. Weiner B. An attributional theory of achievement motivation and emotion. Psychol Rev. 1985; 92:548–573. [PubMed: 3903815]

2. Jeong S. Effects of news about genetics and obesity on controllability attribution and helping behavior. Health Commun. 2007; 22:221–228. [PubMed: 17967144]

3. Dar-Nimrod I, Heine SJ. Genetic essentialism: On the deceptive determinism of DNA. Psychol Bull. 2011; 137:800. [PubMed: 21142350]

4. O'Neill SC, McBride CM, Alford SH, Kaphingst KA. Preferences for genetic and behavioral health information: The impact of risk factors and disease attributions. Ann Beh Med. 2010; 40:127–137.

5. Hilbert A, Dierk JM, Conradt M, et al. Causal attributions of obese men and women in genetic testing: implications of genetic/biological attributions. Psychol Health. 2009; 24:749–761. [PubMed: 20205024]

6. Wang C, Coups E. Causal beliefs about obesity and associated health Behaviors: Results from a population-based survey. Int J Behav Nutr Phys Act. 2010; 7:19. [PubMed: 20199677]

7. Dar-nimrod I, Cheung BY, Ruby MB, Heine SJ. Can merely learning about obesity genes affect eating behavior? Appetite. 2014; 81:269–276. [PubMed: 24997408]

8. Marteau TM, Weinman J. Self-regulation and the behavioural response to DNA risk information: A theoretical analysis and framework for future research. Soc Sci Med. 2006:1360–1368. [PubMed: 16162383]

9. Wright AJ, Sutton SR, Hankins M, Witwell SCL, Macfarlane A, Marteau TM. Why does genetic causal information alter perceived treatment effectiveness? An analogue study. Br J Health Psychol. 2011; 17:294–313. [PubMed: 22107338]

10. Bennett GG, Foley P, Levine E. Behavioral treatment for weight gain prevention among black women in primary care practice: A randomized clinical trial. JAMA Intern Med. 2013; 173:1770–1777. [PubMed: 23979005]

11. Foley P, Levine E, Askew S, et al. Weight gain prevention among black women in the rural community health center setting: The Shape Program. BMC Public Health. 2012; 12:305. [PubMed: 22537222]

12. Ashida S, Goodman M, Pandya C, et al. Age differences in genetic knowledge, health literacy and causal beliefs for health conditions. Public Health Genomics. 2011; 14:307–316. [PubMed: 20829577]

13. Stunkard AJ, Sørensen T, Schulsinger F. Use of the Danish Adoption Register for the study of obesity and thinness. Res Publ Assoc Res Nerv Ment Dis. 1983; 60:115–120. [PubMed: 6823524]

14. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. J Psychosom Res. 1985; 29:71–83. [PubMed: 3981480]

15. Marcus BH, Selby VC, Niaura RS, Rossi JS. Self-efficacy and the stages of exercise behavior change. Res Q Exerc Sport. 1992; 63:60–66. [PubMed: 1574662]

16. Centers for Disease Control and Prevention. Behavioral Risk Factor Surveillance System user's guide. Atlanta: 1998.
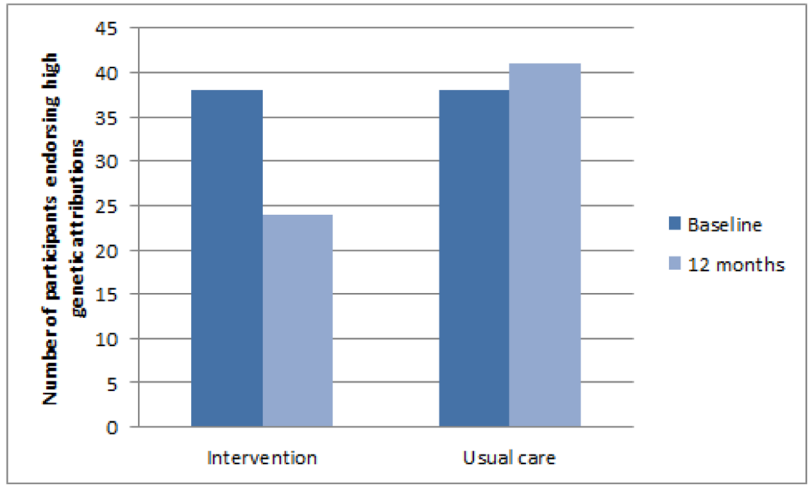
Figure 1.
Endorsement of high genetic attribution for weight loss by study arm at baseline and 12 months.

Table 1a

Associations of genetic attributions for weight with other clinical and psychosocial variables at baseline.

| | Genetic attribution for weight status | | | |
	Low genetic attribution (n=134)[a]	High genetic attribution (n=49)[a]	Test value[b]	p-value
Age, years, M(SD)	35.2 (5.4)	35.9 (5.7)	−0.80	0.42
Education			3.28	0.19
Less than HS degree, n(%)	11 (8.3)	7 (14.9)		
HS degree through some college, n(%)	30 (22.6)	14 (29.8)		
Associates degree or higher, n(%)	92 (69.2)	27 (55.3)		
Attribution of diabetes to genes, n(%)			11.98	0.0005
Low	77 (57.5)	14 (28.6)		
High	57 (42.5)	35 (71.4)		
Attribution of heart disease to genes, n(%)			28.13	<.0001
Low	91 (68.4)	12 (25.5)		
High	42 (31.6)	37 (75.5)		
Genes role in weight loss, n(%)			6.72	0.01
Low	86 (64.2)	21 (42.9)		
High	48 (35.8)	28 (57.1)		
BMI, kg/m^2, M(SD)	30.0 (2.6)	30.7 (2.4)	−1.56	0.12
Attempted weight loss in past year, n(%)			1.48	0.22
Yes	99 (74.4)	32 (65.3)		
No	34 (25.6)	17 (34.7)		
Expected weight gain in 2 years			1.15	0.56
0–5 lbs, n(%)	49 (37.1)	14 (28.6)		
5–10 lbs, n(%)	59 (44.7)	25 (51.0)		
>10 lbs, n(%)	24 (18.2)	10 (20.4)		
Body image (current-ideal discrepancy), Median (Q1, Q3)	2.0 (1.0, 2.0)	2.0 (1.0, 2.0)	4505.0[c]	0.88[c]
Total min moderate or vigorous activity per day, M(SD)	150.4 (285.4)	235.3 (657.9)	−0.41[d,e]	0.68[d,e]
Physical Activity self-efficacy , M(SD)	2.6 (0.8)	2.5 (0.9)	1.12	0.26
TFEQ Dietary restraint , M(SD)	35.0 (15.4)	30.9 (16.3)	1.59	0.11
TFEQ uncontrolled eating , M(SD)	30.7 (19.4)	38.8 (23.1)	−2.38	0.02
TFEQ emotional eating , M(SD)	37.8 (28.0)	46.8 (29.0)	−1.91	0.06

Notes.

[a] Frequencies may not sum to n due to missing data.

[b] Test value is chi-square value for discrete variables and t-test value for continuous variables.

[c] From Wilcoxon Rank-Sum test.

[d] Test performed on log-transformed values.

[e] Satterthwaite test for unequal variances.

Table 1b

Associations of genetic attributions for weight loss with other clinical and psychosocial variables.

	Genetic attribution for weight loss			
	Low genetic attribution (n=109)[a]	High genetic attribution (n=76)[a]	Test value[b]	p-value
Age, years, M(SD)	35.6 (5.5)	35.1 (5.6)	0.59	0.55
Education			0.51	0.77
Less than HS degree, n(%)	12 (11.2)	7 (9.3)		
HS degree through some college, n(%)	24 (22.4)	20 (26.7)		
Associates degree or higher, n(%)	71 (66.4)	48 (64.0)		
Attribution of diabetes to genes, n(%)			0.43	0.51
Low	57 (52.3)	36 (47.4)		
High	53 (47.7)	40 (52.6)		
Attribution of heart disease to genes, n(%)			2.66	0.10
Low	67 (61.5)	37 (49.3)		
High	42 (38.5)	38 (50.7)		
Genes role in weight, n(%)			6.72	0.01
Low	86 (80.4)	48 (63.2)		
High	21 (19.6)	28 (36.8)		
BMI, kg/m^2, M(SD)	30.3 (2.6)	30.0 (2.5)	0.62	0.54
Attempted weight loss in past year, n(%)			1.99	0.16
Yes	83 (76.1)	50 (66.7)		
No	26 (23.9)	25 (33.3)		
Expected weight gain in 2 years			4.44	0.11
0–5 lbs, n(%)	37 (34.3)	27 (36.0)		
5–10 lbs, n(%)	45 (41.7)	39 (52.0)		
>10 lbs, n(%)	26 (24.1)	9 (12.0)		
Body image (current-ideal discrepancy), Median (Q1, Q3)	2.0 (1.0, 2.0)	2.0 (1.0, 2.0)	6552.5[c]	0.20[c]
Total min moderate or vigorous activity per day, M(SD)	155.9 (158.2)	240.3 (579.1)	−0.67[d,e]	0.50[d,e]
Physical Activity self-efficacy , M(SD)	2.7 (0.8)	2.4 (0.9)	2.13	0.03
TFEQ Dietary restraint , M(SD)	32.8 (16.5)	35.3 (14.3)	−1.08	0.28
TFEQ uncontrolled eating , M(SD)	29.1 (18.5)	38.4 (22.4)	−3.06	0.002
TFEQ emotional eating , M(SD)	38.2 (28.3)	42.8 (28.4)	−1.08	0.28

Notes.

[a] Frequencies may not sum to n due to missing data.

[b] Test value is chi-square value for discrete variables and t-test value for continuous variables.

[c] From Wilcoxon Rank-Sum test.

[d] Test performed on log-transformed values.

[e] Satterthwaite test for unequal variances.

Table 2

Relationship between baseline genetic attribution for weight and weight loss, and weight change in intervention and control groups adjusting for site, baseline age and education.

	Baseline genetic attribution for *weight status*			Baseline genetic attribution for *weight loss*		
	Low genetic attribution	High genetic attribution	Difference, Mean (95% CI)	Low genetic attribution	High genetic attribution	Difference, Mean (95% CI)
	Mean (SE) Change			Mean (SE) Change		
Intervention group	(n=66)	(n=23)		(n=52)	(n=37)	
Month 6 weight change, kg	−0.8 (0.6)	−0.8 (1.0)	−0.03 (−2.5, 2.5)	0.03 (0.6)	−2.0 (0.8)	−2.1 (−4.1, 0.02)
Month 12 weight change, kg	−0.6 (0.6)	−1.5 (1.0)	−0.8 (−3.1, 1.3)	0.5 (0.6)	−2.7 (0.7)	−3.3 (−5.2, −1.4)
Month 18 weight change, kg	−0.4 (0.7)	−1.5 (1.2)	−1.1 (−3.8, 1.6)	0.9 (0.8)	−3.0 (0.9)	−3.9 (−6.2, −1.5)
Usual care group	(n=67)	(n=24)		(n=55)	(n=38)	
Month 6 weight change, kg	−0.06 (0.5)	1.4 (0.9)	1.4 (−0.6, 3.5)	−0.2 (0.6)	0.8 (0.7)	1.0 (−0.8, 2.8)
Month 12 weight change, kg	0.1 (0.5)	2.1 (0.9)	2.0 (−0.03, 4.0)	0.2 (0.6)	1.1 (0.7)	0.9 (−0.9, 2.7)
Month 18 weight change, kg	0.3 (0.6)	2.9 (1.0)	2.6 (0.4, 4.8)	0.4 (0.7)	1.6 (0.8)	1.1 (−0.9, 3.2)

Table 3

Effects of weight gain prevention intervention on genetic attributions for weight and weight loss, adjusting for site.

	Intervention (n=91)	Usual care (n=94)	Adjusted OR (95% CI)[a]	Test statistic Z value	p-value
Genetic attribution–*weight*					
Baseline			0.9 (0.5, 1.8)	−0.27	0.78
Low genetic attribution, n(%)	66 (72.5)	68 (73.9)			
High genetic attribution, n(%)	25 (27.5)	24 (26.1)			
12 months			1.5 (0.7, 3.1)	1.14	0.26
Low genetic attribution, n(%)	67 (79.8)	66 (72.5)			
High genetic attribution, n(%)	17 (20.2)	25 (27.5)			
Genetic attribution–*weight loss*					
Baseline			0.9 (0.5, 1.7)	−0.20	0.84
Low genetic attribution, n(%)	53 (58.2)	56 (59.6)			
High genetic attribution, n(%)	38 (41.8)	38 (40.4)			
12 months			2.0 (1.1, 3.8)	2.21	0.03
Low genetic attribution, n(%)	61 (71.8)	50 (55.0)			
High genetic attribution, n(%)	24 (28.2)	41 (45.0)			

Notes.

[a] ORs and p-values are for between subjects comparisons at each time point.