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Dieting, restraint, and disinhibition predict women's weight change over 6 y^{1–3}

Jennifer S Savage, Lesa Hoffman, and Leann L Birch

ABSTRACT

Background: Although disinhibited eating is positively associated with higher weight in women, it is not known whether restrained eating and dieting moderate the influence of disinhibited eating on weight change.

Objective: The objective was to investigate over 6 y the interactive effects of restrained and disinhibited eating and self-reported dieting to lose weight as predictors of weight gain in women.

Design: Data were collected from non-Hispanic white women ($n = 163$) every 2 y. Height and weight were measured in triplicate. Dietary restraint and disinhibition were assessed by using the Eating Inventory. Participants were also asked if they were “currently dieting to lose weight.” Multilevel modeling was used to examine change in weight as a function of time-invariant and time-varying predictors, including dietary restraint, dietary disinhibition, and self-reported dieting.

Results: After covariates were adjusted for, growth curve models showed that within-person increases in restraint over time were associated with concurrent decreases in weight and that higher levels of restraint moderated the positive association between dietary disinhibition and weight. Women who reported dieting at study entry were heavier at study entry and gained more weight over time than did nondieters. Finally, a significant interaction between restraint, disinhibition, and dieting showed that restraint moderated the effect of disinhibition on weight differently in nondieters than in dieters.

Conclusions: Increasing levels of dietary restraint may be beneficial in moderating weight by attenuating the positive association between disinhibition and weight in dieting women. An understanding of weight and weight change requires examination of the interactive effects of restraint, disinhibition, and dieting. *Am J Clin Nutr* 2009;90:33–40.

INTRODUCTION

Dieting to lose weight is common among women of all ages and body weights. Although 1 in 3 American women currently report dieting to lose weight (1), weight gain and obesity in the general population continues to be a major public health issue. Longitudinal studies estimate that, on average, adults gain 0.5–1.0 kg/y (2, 3). However, we have few longitudinal studies to provide information on factors associated with different patterns of weight change within individuals during adulthood.

Dietary restraint and disinhibition, as measured by the Eating Inventory (EI) (4), are psychological constructs that assess behavioral control and attitudes toward food and eating. Dietary

restraint is defined as a tendency to consciously restrict or control food intake, whereas dietary disinhibition is defined as a tendency to overeat in the presence of palatable foods or other disinhibiting stimuli, such as emotional stress (5). Dietary disinhibition is positively associated with weight (6–9), but the association between dietary restraint and weight remains unclear (7–12). Recently, Hill et al (13) argued that, within the current environment, cognitive controls of eating are necessary to moderate weight gain. However, cross-sectional studies have shown that it is not the independent effects of restraint and disinhibition, but their interaction that predicts body weight, with restraint moderating the effect of disinhibition on body weight (8, 9, 14). Specifically, individuals who are more disinhibited and also more restrained (ie, who generally show restraint but are prone to context-induced disinhibition) have lower body mass indexes (BMIs) than do individuals who are less restrained (14).

Several weight-reduction treatment programs showed that greater increases in restraint are associated with greater weight loss (10, 15, 16), whereas others report no association (17, 18). Two prospective longitudinal studies have examined associations between dietary restraint or disinhibition and weight change in free-living individuals (11, 19). However, these studies did not examine the conjoint effects of dietary restraint and disinhibition (8, 9, 14). Therefore, little is known about the long-term effects of this interaction on weight change, and, in particular, on unintentional weight gain among free-living premenopausal women.

Research assessing the effect of self-reported dieting on weight change has produced inconsistent results, which may be due to the fact that dieting has no agreed-on definition and is often an ambiguous term that is rarely clearly defined. Some findings provide evidence that dieting may contribute to the current

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obesity epidemic by triggering reactive or compensatory overeating and weight gain, which then may result in cycles of dieting and weight gain (20–24). Therefore, dieting may promote weight gain (12, 25, 26). For example, Lowe et al (12) found that when participants were asked to self-identify as currently dieting or not dieting, dieters gained 3 times as much weight as nondieters. However, it is also plausible that people who are heavier and gain more weight over time tend to diet in their attempts to reverse the weight gain. Dieting may be a mere marker of individuals most prone toward weight gain (27–30). Thus, it is unclear whether dieting is ineffective, resulting in weight gain, or whether dieting is a proxy for an obesigenic lifestyle that may be incompatible with dieting success.

Research designed to differentiate self-reported dieting and dietary restraint has shown that the effect of dietary restraint on weight differs depending on current dieting status, which suggests that dieting and dietary restraint are related but separate constructs. Taken together, these findings suggest that weight change may be best understood by examining the interactive effects of restraint, disinhibition, and current dieting status. On the basis of previous research (7–9, 23, 31), we expect that 1) dietary restraint alone will not be a significant independent predictor of weight gain, 2) higher levels of disinhibition will predict greater weight gain over time, 3) dietary restraint will moderate the positive relation between disinhibition and weight gain, 4) this association will vary across dieters and nondieters, and 5) self-reported dieting will be associated with greater weight gain.

SUBJECTS AND METHODS

Participants

The study participants were part of a larger longitudinal study designed to examine parental influences on girls' growth and development (32–34). The larger study included 197 non-Hispanic white married couples and their daughters who were examined on 4 occasions, with 2-y intervals between assessments. Families with age-eligible female children within a 5-county radius were identified by using available marketing information (Metromail Inc, Chicago, IL). These families received mailings providing information about the study and were recruited by using follow-up phone calls. Families were not recruited on the basis of the child's or parent's weight or concern about weight. Only data for mothers are considered in this study. Only women with complete weight, dieting, dietary restraint, and disinhibition data at all times of measurement were included in this study. Attrition was primarily due to family relocation outside of the study area. No significant difference was found between the initial weight, weight change, dietary restraint or disinhibition, and dieting status of participants lost to follow-up ($n = 34$) and of participants remaining in the study through year 6 ($n = 163$). Moreover, pregnant and lactating women ($n = 22$) were not removed from analyses because there were no significant differences on the measures listed above between these women and women who were not pregnant or lactating.

Design and procedures

Data were collected on 4 occasions across a 6-y period, with 2-y intervals between each time of measurement. At each time of

assessment, women completed a series of self-report questionnaires during a scheduled visit to the laboratory. The Pennsylvania State University Institutional Review Board approved all study procedures.

Measures

Participants completed a background questionnaire that assessed years of education, combined family income, weekly work hours, general health, and dieting at study entry. In addition, participants reported dieting status at the 2-, 4-, and 6-y follow-up. Specifically, participants were asked "Are you currently dieting to lose weight?" Dieting was defined as eating less or exercising more to lose weight. A sum score of dieting at each occasion was also created to assess the persistence or frequency of dieting ranging from 0 occasions to dieting at all 4 occasions.

Weight and body mass index

Height and weight measurements were assessed in triplicate at each occasion by a trained staff member following the procedure outlined by Lohman et al (35). Participants were dressed in light clothing and were measured while shoeless. Height was measured in triplicate to the nearest 10th of a centimeter with a stadiometer (Shorr Productions Stadiometer; Irwin Shorr, Olney, MD). Weight was measured in triplicate to the nearest 10th of a kilogram with an electronic scale (Seca Electronic Scale; Seca Corp, Birmingham, United Kingdom). Average height and weight were used to calculate BMI [weight (kg)/height squared (m)]. Recommendations made by the World Health Organization (36) were used to classify women as overweight (BMI ≥ 25) and obese (BMI ≥ 30).

Restraint and disinhibition

The EI developed by Stunkard and Messick (4) consists of 51 true-false items designed to tap 3 subscales: 1) dietary restraint (21 items), 2) dietary disinhibition (16 items), and 3) susceptibility to hunger (14 items). For the purpose of this study, only restraint and disinhibition subscales were used. The restraint scale measures cognitive control of eating (eg, "I consciously hold back at meals in order not to gain weight"). The dietary disinhibition scale measures loss of cognitive control of eating (eg "Sometimes when I start eating, I just can't seem to stop"). Scores for each subscale are calculated by summing respective items. Internal consistency coefficients (Cronbach's α) for restraint were 0.87, 0.86, 0.83, and 0.86 and for disinhibition were 0.83, 0.84, 0.83, and 0.82 at times 1, 2, 3, and 4, respectively.

Statistical analyses

Multilevel models (ie, growth curve models) were estimated to examine over 6 y the overall pattern of and individual differences in weight change (kg) among women (37, 38). Weight at each occasion was examined as the primary outcome for 2 reasons: 1) height did not change among our sample of premenopausal women, and 2) long-term weight maintenance has been defined as a weight change of $<3\%$ of body weight (39). Because women differed in age at baseline, 2 options for modeling change over time were possible: a time-in-study model with age at baseline as a covariate or an age-as-time model. A time-in-

study model was selected because preliminary analyses suggested that the cross-sectional effect of age at baseline was not significant. Thus, change was specified as a function of time-in-study, and age at baseline was also included as a control variable.

Restricted maximum likelihood (REML) was used to report model parameters and to assess the significance of random effects; df values were estimated by using the Satterthwaite method. The 95% CI for sample random variation around each fixed effect was calculated as ± 1.96 SD of its accompanying random variance term. Time-in-study was centered at the first occasion such that the intercept represented initial status in all models. The interclass correlation from the unconditional means model (ie, empty model; intercept only) was calculated as 0.92, which indicated that 92% of the variance for weight across 6 y occurred between persons. The interclass correlation for restraint was 0.65 and for disinhibition was 0.77, which indicated that most of the variance for restraint and disinhibition was also between persons. All analyses were conducted using SAS PROC MIXED software (version 9.1, 2007; SAS Institute, Cary NC).

RESULTS

Descriptive characteristics

Descriptive statistics are provided in **Table 1**. The mean (\pm SD) age of women at study entry was 35.7 ± 4.7 y (range: 24.1–46.6 y). All women were married at study entry; however, 19 couples divorced over the 6-y study period. In addition, $\approx 15\%$, 49%, 28%, and 8% of women had 1, 2, 3, or ≥ 4 children, respectively, at study entry. Women were, on average, well educated, and approximately equal numbers of families reported incomes at entry into the study of $< \$35,000$, between $\$35,000$ and $\$50,000$, and $> \$50,000$. Moreover, 64% reported being employed at baseline, working a mean of 19 h/wk. More than 50% of the sample was overweight at baseline. Specifically, 54% of participants were classified as overweight ($\text{BMI} \geq 25$) at baseline; this proportion increased to 60% at the 6-y follow. Approximately 30% of women reported currently dieting at each occasion. Examining the persistence or frequency of dieting showed that 46% of women indicated that they never dieted, whereas 17%, 16%, 18%, and 3% of women reported dieting on 1, 2, 3, or 4 occasions, respectively.

On average, women gained 3.9 kg or 5.6% of their initial body weight across the 6-y period. Descriptive statistics for all predictor and outcome variables at each occasion are shown in **Table 2**. A multilevel model with a random linear slope for time showed that both weight [weight (kg) = $70.28 + 1.26 \times \text{years}$; $P < 0.001$] and BMI ($\text{BMI} = 26.01 + 0.47 \times \text{years}$; $P < 0.001$) increased significantly over time. From baseline to year 6, 69%

of women gained weight (≥ 1 kg). Similar analyses showed that restrained eating (restraint = $9.38 - 0.41 \times \text{years}$; $P < 0.001$) and disinhibited eating (disinhibition = $7.50 - 0.25 \times \text{years}$; $P < 0.001$) decreased significantly over time. The percentage of women who reported dieting remained relatively stable over time.

Unconditional polynomial models for weight change

Polynomial models were first specified with a random intercept only. A fixed linear effect of time was significant ($P < 0.001$), such that average weight increased across time. The addition of a random linear effect (including covariance between the random intercept and random linear effect) resulted in a significant improvement to the model, REML deviance difference (2) = 61, $P < 0.001$. The mean weight at baseline was 71.5 kg (random-effects 95% CI: 39, 105 kg). The mean linear rate of change was 0.63 kg/y (random-effects 95% CI: $-0.40 - 1.66$), which indicated that not all women gained weight over time. The addition of fixed and random quadratic effects did not significantly improve the fit of the model, which indicated no acceleration or deceleration of change over time.

Conditional polynomial models for weight change

We then estimated conditional (predictive) models to examine the interactive effects of restraint, disinhibition, and dieting on weight change over time. To facilitate interpretation of main effects and interactions across levels of analysis, the effects were separated into between-person effects (ie, whether or not a woman was higher or lower on a predictor at baseline, relative to other women in the sample) and within-person effects (ie, whether a woman increased or decreased in a predictor relative to baseline). The initial effects of dietary restraint and disinhibition were represented by the baseline value of each predictor, which was then centered at 8.5 for restraint and at 7.0 for disinhibition to facilitate interpretation of the model intercept and main effects. Change in restraint and disinhibition were represented as each person's deviation (change) from their own baseline value at each subsequent occasion. The baseline effect of dieting was represented with a dummy code for dieting at study entry, and time-varying dieting was represented as whether an individual was currently dieting or not at each occasion. The results are presented in **Table 3**, such that within-person and between-person effects were discussed separately for models 1 and 3. Lower-order effects in models 2 and 4 were used to decompose the higher-order interactions.

Do baseline restraint and disinhibition predict weight change?

As shown in Table 3 for model 1, there was a significant positive effect of dietary disinhibition at baseline, such that greater levels of disinhibition at baseline were related to higher levels of weight at baseline. This relation was strengthened over time, as indicated by a significant positive interaction of baseline disinhibition and time, such that higher levels of disinhibition at baseline predicted greater weight gain over time. However, there were no significant effects of dietary restraint at baseline on weight at baseline (main effect of restraint) or change in weight over time (interaction of baseline restraint with time).

TABLE 1

Sample characteristics of subjects at baseline

	Mean \pm SD	Range
BMI (kg/m^2)	26.5 ± 6.2	17.7–56.2
Height (cm)	164.3 ± 3.1	150.5–186.1
Weight (kg)	71.4 ± 16.7	45.5–149.1
Age (y)	35.7 ± 4.7	24.1–46.6
Education (y)	14.7 ± 2.3	12–20

TABLE 2Descriptive statistics for all predictor and outcome variables at each occasion¹

	Baseline	Year 2	Year 4	Year 6	P
Weight (kg)	71.4 ± 16.7	72.8 ± 16.9	74.1 ± 16.9	75.4 ± 18.0	<0.001
Dietary restraint score	9.1 ± 5.8	8.5 ± 4.8	8.1 ± 4.8	7.8 ± 4.8	<0.001
Dietary disinhibition score	7.1 ± 4.0	7.1 ± 4.1	7.0 ± 3.9	6.3 ± 3.7	<0.001
Currently dieting to lose weight [n (%)]					0.19
No	112 (69)	111 (68)	118 (72)	113 (69)	
Yes	51 (31)	52 (32)	45 (28)	50 (31)	

¹ Values are means ± SDs. Results are from a multilevel model analysis using random coefficients.*Do within-person changes in restraint and disinhibition predict weight change?*

Change from baseline in dietary restraint was significantly negatively associated with weight; within-person increases in restraint were associated with decreases in weight over the same time interval. The within-person effect of change in dietary restraint varied significantly over individuals, as indicated by a significant model improvement on adding a random slope, REML deviance difference (3) = 27, $P < 0.001$, such that 95% of the sample was expected to show within-person effects of change in restraint from -1.28 to 0.50, which indicated that while the effect of restraint on weight was negative on average, the effect was not negative for all participants. Change from baseline in dietary disinhibition was positively related to weight; within-person increases in disinhibition were related to concurrently higher weight. A random slope for disinhibition was also tested, but was not significant.

Does dietary restraint moderate the positive association between dietary disinhibition and weight?

As shown in model 2, the interaction of baseline restraint and baseline disinhibition was significantly negative, meaning that higher levels of restraint at baseline were related to a less positive effect of disinhibition at baseline on weight. Thus, being highly restrained moderated the positive association between disinhibition and weight. In addition, the interaction of baseline disinhibition and change in restraint was also significantly negative: although greater levels of disinhibition at baseline were associated with higher levels of weight at baseline, the positive relation of baseline disinhibition to weight was reduced at times when women had higher levels of dietary restraint than that observed at baseline (increases in restraint from baseline). Alternatively, the within-person negative relation between changes in restraint on concurrent weight was greater in persons with higher levels of disinhibition at baseline. Thus, our third

TABLE 3Parameter estimates (Est) predicting weight change in women¹

Term	Model 1: restraint + disinhibition			Model 2: restraint × disinhibition			Model 3: dieting			Model 4: 3-factor interaction		
	Est	SE	P	Est	SE	P	Est	SE	P	Est	SE	P
Intercept	71.71	1.08	<0.0001	72.27	10.09	<0.0001	65.68	2.24	<0.0001	69.37	2.37	<0.0001
Age at baseline	0.10	0.23	0.66	0.10	0.22	0.67	-0.00	0.27	0.99	0.08	0.22	0.70
Linear time in study	0.65	0.08	<0.0001	0.64	0.08	<0.0001	0.39	0.13	0.001	0.33	0.15	0.02
Baseline restraint	-0.32	0.25	0.21	-0.37	0.25	0.14				-0.14	0.58	0.81
Baseline restraint by time	-0.02	0.02	0.32	-0.01	0.08	0.41				-0.03	0.02	0.18
Change in restraint	-0.39	0.07	<0.0001	-0.35	0.07	<0.0001				-0.36	0.07	<0.0001
Baseline disinhibition	2.46	0.30	<0.0001	2.45	0.30	<0.0001				3.20	0.64	<0.0001
Baseline disinhibition by time	0.06	0.02	0.01	0.04	0.2	0.05				0.03	0.02	0.14
Change in disinhibition	0.24	0.10	0.01	0.21	0.10	0.02				0.20	0.09	0.03
Baseline disinhibition by baseline restraint				-0.15	0.08	0.05				0.20	0.16	0.21
Baseline disinhibition by change in restraint				-0.08	0.02	0.0001				-0.08	0.02	<0.0001
Dieting at baseline							9.03	2.69	0.001	3.73	2.69	0.17
Dieting at baseline by time							0.36	0.15	0.02	0.42	0.18	0.02
Dieting status at each occasion							-1.25	0.44	0.001	0.33	0.51	0.52
Baseline restraint by baseline dieting										-0.23	0.66	0.72
Baseline disinhibition by baseline dieting										-0.74	0.76	0.33
Baseline restraint by baseline disinhibition by baseline dieting										-0.48	0.19	0.01

¹ A multilevel model analysis was used. Model 1, predictors include main effects of restraint and disinhibition; model 2, includes interactions between restraint and disinhibition; model 3, includes only the main and interactive effects of dieting; and model 4, includes a 3-factor interaction among restraint, disinhibition, and dieting.

hypothesis that dietary restraint would moderate the positive relation between disinhibition and weight was supported both between-persons and within-persons. The interactions of change in disinhibition and baseline restraint and between change in restraint and disinhibition were also tested but were not significant. Model 2 was also tested, including higher-order interactions with time; however, none of these additional terms were significant, which indicated that the moderation effects did not vary over time.

Does dieting status independently predict weight change?

The independent effects of self-reported dieting on weight were examined in model 3. There was a significant effect for baseline dieting status, such that women who reported dieting at baseline had greater weights at baseline (main effect of dieting) and significantly greater weight gains over time (interaction of dieting with time) than did nondieters. For example, women who reported dieting at study entry on average gained 4.5 kg over 6 y, whereas women who did not report dieting only gained 2.34 kg. However, time-varying dieting (dieting at each occasion) was significantly predictive of lower current weight. Similar results emerged after adjustment for initial body weight (data not shown). We also tested a 3-factor interaction among baseline dieting and time and the persistence or frequency of dieting during the course of this study after including all lower-order 2-factor interactions and main effects, but this association was not significant. In other words, the finding that dieters at study entry gained significantly more weight than did nondieters did not vary by the persistence of dieting (data not shown.) Last, post hoc analyses showed that women who dieted at one or more time points gained significantly more weight than did women who never dieted after adjustment for mother's age, initial weight, and dieting status at study entry. Thus, our hypothesis that dieting would be related to weight gain was partially supported, in that baseline dieters did gain more weight over time, but not supported in that weight was lower in dieters when actually dieting.

Does the moderation of restraint on the effect of disinhibition on weight vary by dieting status?

A 3-factor interaction was tested (after including all relevant main effects and 2-factor interactions) to examine whether the degree of moderation of the disinhibition effect by restraint at baseline on weight would vary across dieters and nondieters at study entry. As shown in model 4 and **Figure 1**, the 3-factor interaction was significant and can be interpreted as follows. In nondieters at baseline (left side of figure), higher disinhibition predicts higher weight to a greater extent when restraint is higher, but in dieters at baseline (right side of figure), higher disinhibition predicts higher weight to a greater extent when restraint is lower. Alternatively, in persons with lower restraint (solid lines), dieters at baseline are predicted to weigh more than nondieters at baseline when disinhibition is higher but not when disinhibition is lower, but in persons with higher restraint (dashed lines), dieters at baseline are predicted to weigh more than nondieters at baseline when disinhibition is lower. Finally, we note that in model 4, the effect of baseline restraint on change in weight over time remained nonsignificant, and the effect of baseline disinhibition on change in weight over time became nonsignificant, but dieters at baseline were still pre-

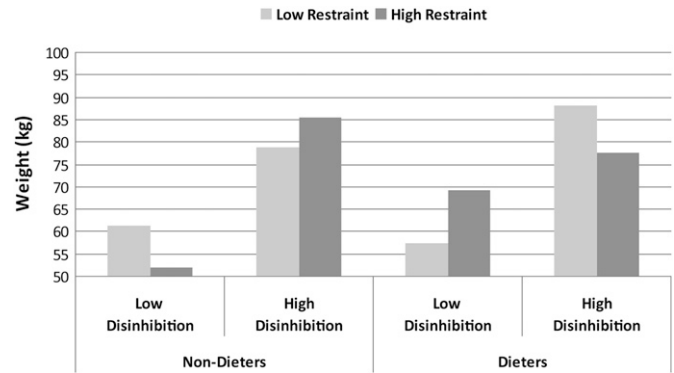


FIGURE 1. Moderation effects of restraint on disinhibition and weight at study entry. The effect differed between nondieters and dieters ($n = 163$). Weight values were predicted by model 4 (3-factor interaction model) based on ± 1 SD of the mean for each predictor: high and low restraint (± 5) and high and low disinhibition (± 4). Light bars represent low restrained eaters; dark bars represent highly restrained eaters. Weights were estimated by using multilevel modeling (SAS PROC MIXED; SAS Institute, Cary, NC).

dicted to gain more weight over time than nondieters at baseline (interaction between dieting and time.) No 2-factor or 3-factor interactions of baseline restraint, disinhibition, and dieting on change over time were found.

The additive effects of the 3 predictors on weight change over time (interaction between dieting and time as well as the interaction among baseline restraint, disinhibition, and dieting) are shown in **Figure 2**. Dieters (right panel) were predicted to gain more weight over time than nondieters (left panel), and persons with higher disinhibition (lines 1 and 2) also were predicted to be heavier at study entry and to gain more weight over time than persons with lower disinhibition (lines 3 and 4), although the effect of disinhibition on change over time was nonsignificant once controlled for the effect of dieting on change over time. Finally, those who reported lower restraint and higher disinhibition (line 1) were predicted to weigh the heaviest over time,

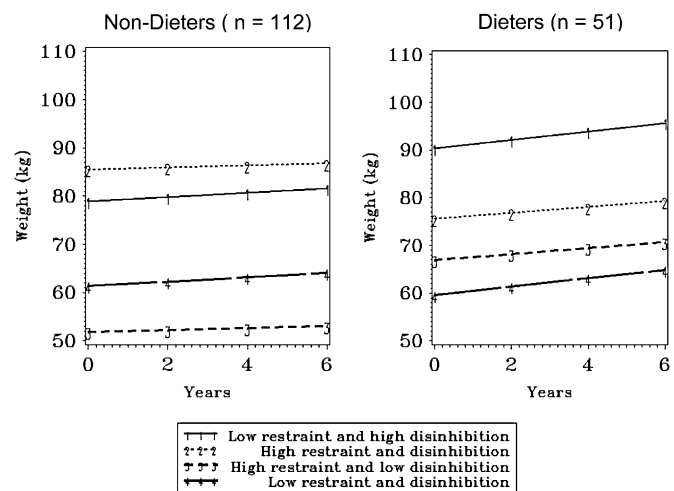


FIGURE 2. Effect of dieting status, dietary restraint, and dietary disinhibition on weight change over time ($n = 163$). Weight values were predicted by model 4 (3-factor interaction model) based on ± 1 SD of the mean for each predictor: high and low restraint (± 5) and high and low disinhibition (± 4). Solid lines represent low restrained eaters; dashed lines represent highly restrained eaters. Weights were estimated by using multilevel modeling (SAS PROC MIXED; SAS Institute, Cary, NC).

particularly dieters, whereas those who reported higher restraint but lower disinhibition (left panel, line 3) were predicted to weigh the lightest, but only nondieters; dieters were predicted to weigh the lightest when reporting both lower restraint and lower disinhibition (right panel, line 4). Model 4 was also examined using BMI as the outcome, which gave similar results (data not shown).

DISCUSSION

This study examined the independent and combined effects over 6 y of dieting, dietary restraint, and disinhibition on weight and weight gain in women. On average, women gained 3.9 kg (8.5 lb) over 6 y. Large individual differences in actual weight change were noted, ranging from an observed weight loss of 20.9 kg to an observed weight gain of 30.1 kg. The average weight gain noted among our sample is consistent with other longitudinal studies, which have shown that adults tend to gain 0.5–1.0 kg/y (2, 3). Results from the final multilevel (growth curve) model can be summarized as follows. Higher baseline levels and current levels of dietary disinhibition were associated with higher baseline and current levels of weight, respectively. Women who were dieting at baseline gained more weight over time, but weight gain over time did not vary by baseline restraint or disinhibition. Also consistent with previous literature (9, 10), individual differences in baseline restraint did not predict baseline weight, but within-person decreases in restraint were related to concurrently higher weight, particularly for persons with higher levels of baseline disinhibition. These findings indicate that practicing higher overall levels of dietary restraint does not appear to be an effective weight-maintenance strategy per se, but that practicing more restraint than usual may moderate weight gain, particularly among persons with higher levels of baseline disinhibition. Finally, we observed differential relations between baseline restraint and disinhibition among baseline dieters and nondieters. These interactions indicate the importance of examining the conjoint effects of restraint, disinhibition, and dieting on weight.

The primary aim of the present study was to examine a 3-factor interaction among dietary restraint, dietary disinhibition, and dieting. On the basis of previous research suggesting that dietary restraint is a multidimensional construct encompassing both past and current dieting (31), we hypothesized that restraint would not independently predict weight or weight change, but that higher levels of disinhibition would be associated with higher weight, with restraint moderating this effect differently for dieters and nondieters. In support of our hypothesis, restraint moderated the positive association between disinhibition and weight (model 2), the pattern of which was different for women who reported currently dieting or not dieting at study entry (model 4). Among dieters, restraint attenuates the positive association between disinhibition and weight, whereas among nondieters, restraint exacerbates the relation between disinhibition and weight. In other words, nondieters showed a greater effect of disinhibition when restraint was higher, whereas dieters showed a greater effect of disinhibition when restraint was lower. Therefore, baseline restraint itself was not an independent predictor of weight or weight change, but rather together with dieting moderated the association between disinhibition and weight—a finding consistent with previous cross-sectional research (8, 9, 14). For example, a study that assessed this interaction in pre-

menopausal women using a 2×2 factorial design (high/low restraint \times high/low disinhibition) found that women reporting low restraint but high disinhibition were significantly heavier than the other 3 groups, whereas the low-restraint and low-disinhibition groups had the lowest BMI (14).

These findings are also consistent with previous research findings that restrained nondieters may be more likely to overeat and gain weight than restrained dieters, which thereby motivates them to start a new weight-loss diet (31, 40–41). Specifically, research designed to differentiate how restraint and dieting relate to weight change showed that, in terms of weight loss, dieters who were also restrained lost significantly more weight than restrained and unrestrained nondieters (41). Therefore, being highly restrained while currently dieting was an effective strategy to promote short-term weight loss; however, a history of repeated dieting may be a proxy risk factor for unsuccessful eating control, weight cycling, and weight gain over time. These findings are consistent with those of Hill et al (13), who argue that chronic cognitive control over eating is needed to moderate weight gain in our current environment; dietary restraint was intended to measure such cognitive control (5). In combination, these findings indicate that relative increases in dietary restraint may be useful in moderating weight gain and weight maintenance among disinhibited women who report dieting to lose weight. However, further longitudinal analyses are needed to replicate these findings in other samples of free-living adults while also examining potential sex differences.

As predicted, and consistent with previous research (6–11, 42), interpretation of the main effects in model 1 showed that baseline dietary restraint scores were not associated with weight or weight change over time, whereas women reporting higher baseline dietary disinhibition scores were heavier at baseline and gained more weight over time, before dieting status was controlled for (model 2). Our findings are consistent with those of Lauzon-Guillain et al (11), who assessed relations between eating behavior and adiposity over a 2-y period and found that baseline restraint was not associated with subsequent adiposity change. Similarly, a cross-sectional study conducted by Hays et al (9) show that disinhibition was a significant predictor of weight gain and BMI based on recalled weight history data, whereas restraint was not an independent predictor of weight change. One potential explanation for these findings is that restrained eaters may be eating less than they want but apparently not less than they need (43). In combination, these findings suggest that disinhibition may be a stronger predictor than restraint on weight and weight gain over time.

Much of the existing literature noted above was either cross-sectional or only assessed eating behavior on 2 occasions and thereby used a change score to predict weight gain. For example, a prospective study assessing changes in weight from baseline to 6-y follow-up showed that higher restraint behavior and decreases in restraint promoted weight gain over time (19). To date, the present study was the first to examine how changes in eating behavior and dieting (within-person effects) across 4 occasions of measurement predict changes in weight over time in free-living individuals. It is important to note that, even though our effects primarily involved baseline predictors, the reliability with which these effects can be estimated is improved given the inclusion of 4 occasions of data. Although differences in dietary restraint between individuals were not predictive of weight gain,

within-person increases in restraint over time were associated with concurrent decreases in weight over time. These results are consistent with those of a weight-loss intervention conducted by Foster et al (10), who found that greater increases in restraint during a weight-loss treatment program were associated with significantly greater weight loss. Moreover, a study evaluating predictors of long-term weight maintenance showed that an increase in dietary restraint during weight loss was a significant predictor of weight maintenance over 2 y (44). Findings from the present study (model 2) indicate that higher usual levels of restraint does not make a difference, but increasing dietary restraint might be effective in promoting weight maintenance by moderating weight gain. Whereas causality cannot be determined in the present study, findings suggest that the effects of interventions to encourage restraining “more than usual,” especially among disinhibited eaters and self-reported dieters, should be explored.

In support of our final hypothesis and consistent with previous research (20, 21, 23), when the model accounts for concurrent dieting status, women who dieted at baseline gained significantly more weight over time than did women who did not report dieting at study entry. Moreover, this association did not vary by the persistence of dieting during the course of the study. These findings suggest that dieting may not be an effective long-term weight gain prevention strategy. However, when the independent effects of dieting were explored (model 3), dieters were lighter when actually dieting, which suggests that dieting may be an effective short-term weight-maintenance strategy. One potential explanation is that dieters who go on and off diets may be more prone to weight cycling, resulting in weight gain. In contrast with this theory, evidence also suggests that chronic attempts at eating less than wanted but not less than required can promote overeating and overweight in the current food environment (27, 45). Whereas it is plausible that a person gains weight because they are dieting, it is also possible that a person initiates dieting in response to weight gain. This is congruent with a recent review by Hill (23), who concluded that being overweight makes women more likely to diet rather than dieting causes someone to gain weight over time. Therefore, other influences may be causing them to gain weight and suppress dieting success. Furthermore, it is unclear whether our sample of dieting women would have gained more or less weight over the 6 y had they not dieted. Thus, additional research examining associations between dieting and weight change is warranted to better understand causality.

This study was not without limitations. First, this sample was racially and demographically homogenous and included only women, which prevents us from generalizing to men or to other racial and socioeconomic groups. Moreover, whereas the present study assessed predictors and weight on several occasions (ie, every 2 y) over 6 y, more frequent assessments of weight and dieting may better capture changes in eating behavior and weight cycling; we may have missed short-term fluctuations in dieting, weight gain, and weight loss. Furthermore, individuals who cycle through weight loss and weight gain usually gain significantly more weight over time than do weight maintainers (46). Another potential limitation of this study was that the duration of dieting (ie, months) or type of dieting (ie, healthy compared with unhealthy) and type of restraint (ie, flexible compared with rigid) were not assessed, which may also interact to influence body weight change (25). Finally, the data were self-reported, which is potentially associated with reporting bias.

In conclusion, the present study and other recent reports (14, 23) suggest that being highly restrained may be a protective factor by attenuating the positive association between disinhibition and weight in women, especially among highly disinhibited dieters. However, increasing restraint is not a “one size fits all” solution for weight control. Whether or not restraint attenuates the effect of disinhibition on weight depends on a woman’s current dieting status. Of those currently trying to lose weight, attempts to promote cognitive control of intake may be helpful, whereas, among nondieters, promoting higher levels of restraint may be counterproductive. This pattern of findings suggests that, as in many other areas of prevention research, interventions should be tailored and adapted to individual needs (47).

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REFERENCES

1. Kruger J, Galuska DA, Serdula MK, Jones DA. Attempting to lose weight: specific practices among U.S. adults. *Am J Prev Med* 2004;26:402–6.
2. Burke GL, Bild DE, Hilner JE, Folsom AR, Wagenknecht LE, Sidney S. Differences in weight gain in relation to race, gender, age and education in young adults: the CARDIA study. *Ethn Health* 1996;1:327–35.
3. Brown WJ, Williams L, Ford JH, Ball K, Dobson A. Identifying the energy gap: magnitude and determinants of 5-year weight gain in midage women. *Obes Res* 2005;13:1431–41.
4. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *J Psychosom Res* 1985;29:71–83.
5. Stunkard AJ, Messick S. *Eating inventory manual*. San Antonio, TX: The Psychological Corporation, 1988.
6. Lindroos AK, Lissner L, Mathiassen ME, et al. Dietary intake in relation to restrained eating, disinhibition, and hunger in obese and nonobese Swedish women. *Obes Res* 1997;5:175–85.
7. Hainer V, Kunesova M, Bellisle F, et al. The Eating Inventory, body adiposity and prevalence of disease in a quota sample of Czech adults. *Int J Obes (Lond)* 2006;30:830–6.
8. Williamson DA, Lawson OJ, Brooks ER, et al. Association of body mass with dietary restraint and disinhibition. *Appetite* 1995;25:31–41.
9. Hays NP, Bathalon GP, McCrory MA, Roubenoff R, Lipman R, Roberts SB. Eating behavior correlates of adult weight gain and obesity in healthy women aged 55–65 y. *Am J Clin Nutr* 2002;75:476–83.
10. Foster GD, Wadden TA, Swain RM, Stunkard AJ, Platte P, Vogt RA. The eating inventory in obese women: clinical correlates and relationship to weight loss. *Int J Obes Relat Metab Disord* 1998;22:778–85.
11. Lauzon-Guillain B, Basdevant A, Romon M, Karlsson J, Borys JM, Charles MA. Is restraint eating a risk factor for weight gain in a general population? *Am J Clin Nutr* 2006;83:132–8.
12. Lowe MR, Annunziato RA, Markowitz JT, et al. Multiple types of dieting prospectively predict weight gain during the freshman year of college. *Appetite* 2006;47:83–90.
13. Hill JO, Wyatt HR, Reed GW, Peters JC. Obesity and the environment: where do we go from here? *Science* 2003;299:853–5.
14. Lawson OJ, Williamson DA, Champagne CM, et al. The association of body weight, dietary intake, and energy expenditure with dietary restraint and disinhibition. *Obes Res* 1995;3:153–61.
15. Bjorvell H, Aly A, Langius A, Nordstrom G. Indicators of changes in weight and eating behaviour in severely obese patients treated in a nursing behavioural program. *Int J Obes Relat Metab Disord* 1994;18:521–5.
16. Klesges RC, Isbell TR, Klesges LM. Relationship between dietary restraint, energy intake, physical activity, and body weight: a prospective analysis. *J Abnorm Psychol* 1992;101:668–74.

17. McGuire MT, Jeffery RW, French SA, Hannan PJ. The relationship between restraint and weight and weight-related behaviors among individuals in a community weight gain prevention trial. *Int J Obes Relat Metab Disord* 2001;25:574–80.
18. van Strien T, van de Laar FA, van Leeuwe JF, et al. The dieting dilemma in patients with newly diagnosed type 2 diabetes: does dietary restraint predict weight gain 4 years after diagnosis? *Health Psychol* 2007;26:105–12.
19. Drapeau V, Provencher V, Lemieux S, Despres JP, Bouchard C, Do Tremblay A. 6-y changes in eating behaviors predict changes in body weight? Results from the Quebec Family Study. *Int J Obes Relat Metab Disord* 2003;27:808–14.
20. Field AE, Austin SB, Taylor CB, et al. Relation between dieting and weight change among preadolescents and adolescents. *Pediatrics* 2003;112:900–6.
21. Stice E, Cameron RP, Killen JD, Hayward C, Taylor CB. Naturalistic weight-reduction efforts prospectively predict growth in relative weight and onset of obesity among female adolescents. *J Consult Clin Psychol* 1999;67:967–74.
22. Gorin AA, Phelan S, Wing RR, Hill JO. Promoting long-term weight control: does dieting consistency matter? *Int J Obes Relat Metab Disord* 2004;28:278–81.
23. Hill AJ. Does dieting make you fat? *Br J Nutr* 2004;92:S15–8.
24. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. *JAMA* 2004;291:2847–50.
25. French SA, Jeffery JW, Murray DM. Is dieting good for you? Prevalence, duration and associated weight and behaviour changes for specific weight loss strategies over four years in US adults. *Int J Obes Relat Metab Disord* 1999;23:320–7.
26. French SA, Jeffery RW, Forster JL, McGovern PG, Kelder SH, Baxter JE. Predictors of weight change over two years among a population of working adults: the Healthy Worker Project. *Int J Obes Relat Metab Disord* 1994;18:145–54.
27. Lowe MR, Levine AS. Eating motives and the controversy over dieting: eating less than needed versus less than wanted. *Obes Res* 2005;13:797–806.
28. Lowe MR, Kral TV. Stress-induced eating in restrained eaters may not be caused by stress or restraint. *Appetite* 2006;46:16–21.
29. Stice E, Presnell K, Groesz L, Shaw H. Effects of a weight maintenance diet on bulimic symptoms in adolescent girls: an experimental test of the dietary restraint theory. *Health Psychol* 2005;24:402–12.
30. Wadden TA, Foster GD, Sarwer DB, et al. Dieting and the development of eating disorders in obese women: results of a randomized controlled trial. *Am J Clin Nutr* 2004;80:560–8.
31. Lowe MR. The effects of dieting on eating behavior: a three factor model. *Psychol Bull* 1993;114:100–21.
32. Fiorito LM, Ventura AK, Mitchell DC, Smiciklas-Wright H, Birch LL. Girls' dairy intake, energy intake, and weight status. *J Am Diet Assoc* 2006;106:1851–5.
33. Birch LL, Fisher JO, Davison KK. Learning to overeat: maternal use of restrictive feeding practices promotes girls' eating in the absence of hunger. *Am J Clin Nutr* 2003;78:215–20.
34. Fisher JO, Mitchell DC, Smiciklas-Wright H, Mannino ML, Birch LL. Meeting calcium recommendations during middle childhood reflects mother-daughter beverage choices and predicts bone mineral status. *Am J Clin Nutr* 2004;79:698–706.
35. Lohman T, Roche A, Martorell R. Anthropometric standardization reference manual. Chicago, IL: Human Kinetics Book, 1991.
36. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO Consultation on Obesity World Health Organization. Geneva, Switzerland: WHO, 1998.
37. Singer JD, Willett JB. Applied longitudinal data analysis: modeling change and event occurrence. New York, NY: Oxford University Press, 2003.
38. Snijders TA, Bosker R. Multilevel analysis. Thousand Oaks, CA: Sage, 1999.
39. Stevens J, Truesdale KP, McClain JE, Cai J. The definition of weight maintenance. *Int J Obes (Lond)* 2006;30:391–9.
40. Lowe MR. Restricted eating and dieting: replication of their divergent effects on eating regulation. *Appetite* 1995;25:115–8.
41. Lowe MR, Timko CA. What a difference a diet makes: towards an understanding of differences between restrained dieters and unrestrained dieters. *Eat Behav* 2004;5:199–208.
42. Hays NP, Bathalon GP, Roubenoff R, McCrory MA, Roberts SB. Eating behavior and weight change in healthy postmenopausal women: results of a 4-year longitudinal study. *J Gerontol A Biol Sci Med Sci* 2006;61:608–15.
43. Stice E, Cooper JA, Schoeller DA, Tappe K, Lowe MR. Are dietary restraint scales valid measures of moderate- to long-term dietary restriction? Objective biological and behavioral data suggest not. *Psychol Assess* 2007;19:449–58.
44. Vogels N, Diepvens K, Westerterp-Plantenga M. Predictors of long-term weight maintenance. *Obes Res* 2005;13:2162–8.
45. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. *N Engl J Med* 1995;332:621–8.
46. St Jeor ST, Brunner RL, Harrington ME, et al. Who are the weight maintainers? *Obes Res* 1995;3(suppl 2):249s–59s.
47. Collins LM, Murphy SA, Nair VN, Strecher VJ. A strategy for optimizing and evaluating behavioral interventions. *Ann Behav Med* 2005;30:65–73.