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# Smoking Cessation and Weight Gain

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Determinants of weight gain after quitting smoking were investigated in two smoking treatment outcome studies. It was hypothesized that (a) abstinence would result in weight gain; (b) postquitting weight gain would be predicted by pretreatment tobacco use, a history of weight problems, and eating patterns; and (c) relapse to smoking would follow weight gain. The first two hypotheses were confirmed. Year-long abstainers gained more weight than relapsers. Most of the weight gain occurred during the first 6 months following quitting. Number of cigarettes smoked at pretreatment and past maximum body weight correlated positively with weight gain. Scores on a modified version of the Eating Inventory Disinhibition scale, a measure of eating control in specific situations, especially emotional ones, explained 27% of the variance in weight gain among abstinent subjects at a 1-year follow-up. Eating Inventory Hunger scale scores also predicted weight gain at a 1-year follow-up, although the proportion of variance predicted was less. Modified Disinhibition scores, number of cigarettes smoked at pretreatment, and maximum body weight were not correlated among subjects abstinent for the year. Disinhibition score and maximum body weight, however, correlated positively in the entire sample of subjects. Contrary to the third hypothesis, greater weight gain during the first 6 months predicted continued abstinence, not relapse.



Quitting smoking results in weight gain (Blitzer, Rimm, & Giefer, 1977; Bosse, Garvey, & Costa, 1980; Coates & Li, 1983; Comstock & Stone, 1972; Gordon, Kannel, Dawber, & McGee, 1975; Wynder, Kaufman, & Lesser, 1967). Intolerance of weight gain may partially explain the high relapse rates in smoking treatment programs and may be a reason to avoid quitting smoking for some (Wynder et al.). In this article, we present data about the course and causes of such gain in exsmokers.

There are not enough data to suggest a comprehensive model of smoking-cessation-induced weight gain. The literature, our previous work (Ginsberg, Hall, & Tunstall, 1984), and clinical experience, however, suggest three predictors of weight gain, which we examined in this study. The first was pretreatment nicotine intake. In a retrospective survey of reducing-club members, Blitzer et al. (1977) found that weight gain was positively correlated with number of cigarettes smoked before quitting, suggesting that some pharmacologic property of cigarettes may suppress weight gain and that removal of this property may be responsible for the gain. Russell, Raw, and Jarvis (1980) noted that smoking treatment clients given nicotine gum gained less weight than did those given placebo gum, suggesting that nicotine may be the agent in cigarettes responsible for the weight changes. Supporting evidence from animal studies indicates that chronic nicotine administration decreases body weight and that termination of administration increases weight. From human studies of acute nicotine action, there is evidence that nicotine affects important components of the systems that regulate body weight (Wack & Rodin, 1982).

Weight problems are usually chronic. They occur in many circumstances in susceptible individuals and are likely to recur. Our second prediction was, therefore, that weight gain would be more likely in subjects with a history of high body weight and of gaining weight during other quitting attempts.

There is evidence that some people are "restrained" eaters, who eat less than is needed to maintain body weight at set points, probably because of strong social pressures against obesity. Such restraint can be destroyed by many factors, including negative emotions, modeling, and the belief that one has already overeaten (Herman & Polivy, 1980). Cigarettes may be useful in controlling weight through anorexic properties, oral substitution, or suppression of emotions correlated with overeating. Thus, a history of restrained eating should predict weight gain when cigarettes are no longer used.

Last, in a society that values slenderness, weight gain is a cost of quitting smoking. Smokers may return to smoking if a gain occurs. Therefore, we predicted that weight gain during the first 26 weeks following treatment would be positively correlated with relapse at Week 52.

In summary, it was hypothesized that (a) abstinence would result in weight gain, (b) high nicotine dependence would predict weight gain, (c) a history of weight problems would predict weight gain, (d) abstinent subjects who were restrained eaters would be more likely to gain weight, and (e) weight gain during abstinence would precede relapse.

## Method

### *Subjects*

Subjects were 255 smokers (122 men and 133 women) who participated in one of two smoking treatment trials. Details of these trials have been reported elsewhere (Hall, Rugg, Tunstall, & Jones, 1984; Hall, Tunstall, Rugg, Jones, & Benowitz, 1985). The first of these trials crossed two levels of aversive smoking (6 vs. 30 s) and two relapse-prevention strategies

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(skill vs. discussion) in a 2 × 2 factorial design. The second trial compared the following three conditions: (a) an intensive behavioral treatment, (b) nicotine gum available for 6 months in a low-contact group, and (c) intensive behavioral and gum treatment combined. Weight gain following smoking was not addressed as a treatment issue in either program.

For the entire sample, mean age was 37.02 years ($SD$ = 7.56), and mean pretreatment body weight was 147.26 lb, or 66.94 kg ($SD$ = 26.10). Mean number of cigarettes smoked at pretreatment was 30.93 ($SD$ = 15.46), and mean pretreatment cotinine was 243.17 ng/ml ($SD$ = 142.29).

### *Procedure*

Subjects completed a questionnaire packet before treatment began. It included (a) the Tobacco and Drug Use History (Bachman, 1980), a questionnaire tapping subjects' past and current use of tobacco and other common drugs and experiences during previous withdrawal attempts; (b) two items regarding weight history, maximum weight ever reached and weight gained after last quitting attempt; and (c) a brief version of the Eating Inventory (Stunkard & Messick, 1985) for subjects in Trial 2. This questionnaire includes three factor-based scales—Cognitive Restraint, Disinhibition, and Hunger. The Cognitive Restraint subscale taps knowledge about nutrition and use of this knowledge (e.g., "When I have eaten my quota of calories, I am usually good about not eating any more"). The Disinhibition scale taps uncontrolled eating in response to specific emotional and situational cues ("When I feel anxious I find myself eating"). The Hunger scale taps perceived hunger ("I am usually so hungry that I eat more than three times a day"). Our scoring of the Disinhibition scale differed from that of Stunkard and Messick. Items reflecting past weight fluctuation contributed to a high Disinhibition score. Items indicating current eating inhibitions also contributed to a high score because cigarette use was hypothesized to have an inhibitory effect on eating. The remaining two scales did not lend themselves to such modifications. Thirty-three items were selected from the original inventory.

Before beginning treatment, blood samples were obtained for cotinine analyses. Cotinine is a nicotine metabolite that can be detected in blood for 24 to 48 hr after smoking, unlike nicotine itself, which has 2- to 3-hr half-life. Because of its longer half-life, cotinine is probably the best measure of a subject's usual nicotine intake. Subjects in Treatment Trial 1 had blood samples drawn at the first treatment session, with no restrictions on smoking. Due to the requirements of other experimental protocols, subjects in Treatment Trial 2 had blood samples at a morning session after an 8-hr cigarette fast.

Subjects were weighed on a balance beam scale at pretreatment and at 3, 26, and 52 weeks from study start. Subjects wore street clothes but removed shoes or boots.

In both trials, abstinence from cigarettes was determined by self-report, verified by expired air carbon monoxide levels (<10 ppm was coded as abstinent) at all assessments. At Weeks 26 and 52, abstinence was also verified by serum thiocyanate levels (<85 mg/ml was coded as abstinent) and by reports of significant others. These values were chosen because they are within the nonsmoking range in an urban environment.

## Results

### *Preliminary Analyses*

Hypotheses about predictors of weight gain during abstinence were tested on those subjects who maintained their abstinence for 1 year ($n$ = 81). For these subjects, mean age was 37.16 years ($SD$ = 7.36), and pretreatment body weight was 145.33 lb, or 66.06 kg ($SD$ = 25.22) Mean number of cigarettes smoked at pretreatment was 25.89 ($SD$ = 12.44), and pretreatment cotinine was 196.85 mg/ml ($SD$ = 111.58). There were little missing data. Body weight for 7 subjects (8%) was missing at Week 26; weight for 6 subjects (7%) was missing at Week 52.

Differences in weight gain because of abstinence were tested on the entire sample ($N$ = 255). At Week 3, less than 7% of the sample had missing data. At both Weeks 26 and 52, 20% of the sample was lost (abstinence data obtained by telephone or mail or subject not weighed). Lost subjects were more likely to be relapsed than abstinent at both Weeks 26, $\chi^2(1) = 10.48$, $p < .001$ (77% relapsed), and 52, $\chi^2(1) = 9.97$, $p < .002$ (84% relapsed). At Week 26, lost subjects were more likely to be men, $\chi^2(1) = 4.24$, $p < .04$ (62% were men). Lost subjects did not differ from the rest of the sample on other variables. We concluded that it was unlikely that missing data would confound the primary analyses.

Also, the data were analyzed to insure comparability of experimental conditions and experiments. We compared (a) all subjects and (b) abstinent subjects only across treatment conditions within each trial and between trials with conditions collapsed to determine if the conditions differed in weight changes at each assessment. No significant differences were found.

### *Data Analysis Strategy*

The primary data analysis strategy for testing hypotheses was hierarchical multiple regression for sets (Cohen & Cohen, 1975). In each analysis, independent variables were entered as a single set. Tests of individual variables were protected for inflation in alpha levels because of multiple tests in that individual tests were completed only if the sets predicted significant proportions of variance. Hierarchical multiple regression was preferred to traditional analysis of variance (ANOVA) because regression methods more readily yield estimates of variance explained.

Dose–response relations are of interest when the relation of a drug to behavior is studied. Therefore, the hypothesis about the relation of nicotine dependence to weight gain was tested by a repeated measures ANOVA, rather than hierarchical regression, because a repeated measures analysis allows a quantitative description of the relation between dose and weight over time. Simple correlations were used to explore the relations between predictor variables.[1]

### *Abstinence and Weight Gain*

Hierarchial regressions indicated that abstinence did result in significant weight gain at Weeks 3, $F(1, 235) = 4.43$, $p < .034$; 26, $F(1, 199) = 25.25$, $p < .001$; and 52, $F(1, 196) = 55.12$, $p < .001$. Despite the level of significance reached, smoking status did not explain a substantial proportion of the variance at Week 3 ($sr^2$ = .018, smoking $M$ = 1.86, $SD$ = 2.56; abstinent $M$ = 2.99, $SD$ = 2.77). Proportions of variance explained by abstinence at Weeks 26 $sr^2$ = .1111, smoking $M$ = 3.23, $SD$ = 5.81; abstinent $M$ = 7.58, $SD$ = 6.43) and 52 ($sr^2$ = .2205, smoking $M$ = 1.71, $SD$ = 6.74; abstinent $M$ = 8.87, $SD$ = 6.51) were of a great

[1] These data did satisfy the requirements for use of change scores (Cohen & Cohen, 1975). Measurement of body weight by a balance beam scale is highly reliable. The correlations of weight between the assessment periods approached unity (range = .996 to .980). The standard deviations at each time point showed minimal variability, range = 25.22 (Week 26) to 28.20 (Week 3).

enough magnitude to be of both practical and theoretical interest. Tests used were three hierarchical regressions. In each, sex and abstinence status, entered as a single set, were the independent variables. Weight change from pretreatment to the assessment period was the dependent variable. Sex of subject was not significant at any assessment.

### *Tobacco Dependence and Weight Gain*

As described here, a repeated measures ANOVA indicated a positive dose–response relation between pretreatment cigarette intake and weight gain. Pretreatment cotinine levels were not related to weight gain. Cigarette intake was partitioned into four levels (<10, 10 to 19, 20 to 29, and ≥30). Cotinine was dichotomized at the median score for the treatment trial. Week-3 data were retained in the analysis despite the low proportion of variance predicted by abstinence so to better describe the shape of the dose-response curve. The interaction between time and pretreatment cigarette intake was significant, $F(9, 204) = 1.95$, $p < .047$, but the interaction between time and cotinine was not.

Figure 1 shows the mean weight gain as a function of pretreatment number of cigarettes smoked. There was not a significant increase in body weight over time for subjects smoking less than 10 cigarettes per day. Subjects smoking 10 to 19 cigarettes per day showed a significant increase, $F(3, 33) = 11.30$, $p < .006$, with significant increases (protected $t$ test, $p < .001$) between all weeks except pretreatment and Week 3 and Weeks 26 and 52; for 20 to 29 cigarettes per day, $F(3, 87) = 44.28$, $p < .001$; and for 30 or more cigarettes per day, $F(3, 75) = 54.91$, $p < .001$. For these last two categories, differences between all assessments, except those between Weeks 26 and 52, were significant at $p < .0001$.

### *Weight History and Weight Gain*

The hypothesis that a history of weight gain would predict current weight gain for abstinent subjects was supported by the hierarchical multiple regression analyses described here. At both time points, greater past maximum body weight predicted greater weight gain during abstinence. Independent variables were entered in the following three steps: (a) sex of subject, (b) maximum body weight and amount of weight gained after last quitting attempt, and (c) the interaction of sex and the second set. Because the proportion of variance explained by abstinence at Week 3 was so small, only data from Weeks 26 and 52 were analyzed: at Week 26, $F(2, 53) = 7.44$, $p < .01$, $sr^2 = .2099$, for the maximum body weight and amount of weight gained, due to the contribution of maximum weight, $t(1) = 3.80$, $p < .01$, $sr^2 = .2083$; at Week 52, $F(2, 53) = 4.00$, $p < 0.2$, $sr^2 = .1301$, for the maximum body weight and amount of weight gained, due to the contribution of past maximum weight, $t(1) = 2.87$, $p < .01$, $sr^2 = .1294$. Sex and interaction terms did not contribute significantly to variance explained at any assessment.

### *Eating Inventory Scores and Weight Gain*

As shown here, regression analyses indicated that high scores on the modified Disinhibition scale predicted weight gain for abstinent subjects. High Hunger scale scores also predicted greater weight gain at Week 52, but the proportion of variance was not so great. Again, Week-3 analyses were omitted because of the small proportion of variance explained by abstinence at that time. Independent variables were entered in a hierarchical multiple regression in the following order: (a) sex of subject, (b) Eating Inventory scores, and (c) the interaction of sex with the Eating Inventory scores. At Week 52, the set of main effects explained a significant proportion of variance, $F(3, 29) = 10.57$, $p < .001$, $sr^2 = .3944$. This was mostly due to the contribution of Disinhibition score, $t(1) = 3.94$, $p < .001$, $sr^2 = .2734$. The relation of weight change at Week 52 (residualized for sex) to Disinhibition score is shown in Figure 2. The main effect for Hunger also explained a significant proportion of the variance at Week 52, $t(1) = 2.33$, $p < .02$, $sr^2 = .0960$. Neither sex nor the interaction set predicted significant proportions of variance.

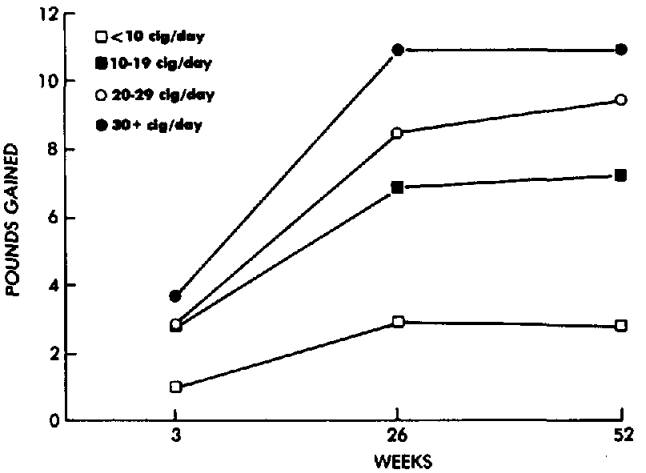


*Figure 1.* Pretreatment number of cigarettes smoked and gain in body weight at Weeks 3, 26, and 52.

### *Correlations Among Tobacco Dependence, Weight History, and Eating Inventory Scores*

For subjects who were abstinent the entire 52-week study period, maximum weight, pretreatment cigarette intake, and eating situations scores did not correlate significantly. For the entire sample, maximum weight and Disinhibition score correlated significantly ($r = .325$, $p < .001$). Inspection of the data indicated truncated upper distributions for the abstinent sample on these two variables, which may have contributed to the lack of correlation.[2]

### *Prediction of Relapse*

Weight change at Week 26 did predict relapse but in the opposite direction from that hypothesized; the greater the weight gain, the greater the probability of continued abstinence, $F(1, 94) = 9.26$, $p < .003$, $sr^2 = .087$. The mean gain during the first

[2] Some might wonder whether these are predictors of weight gain in adults in general rather than unique for abstinent exsmokers. We did attempt to replicate the significant relations observed in the sample abstinent for the entire year with subjects who were smoking at 1 year. Significant proportions of variance explained were not found for number of cigarettes smoked at pretreatment, maximum body weight, or the eating situations scales.

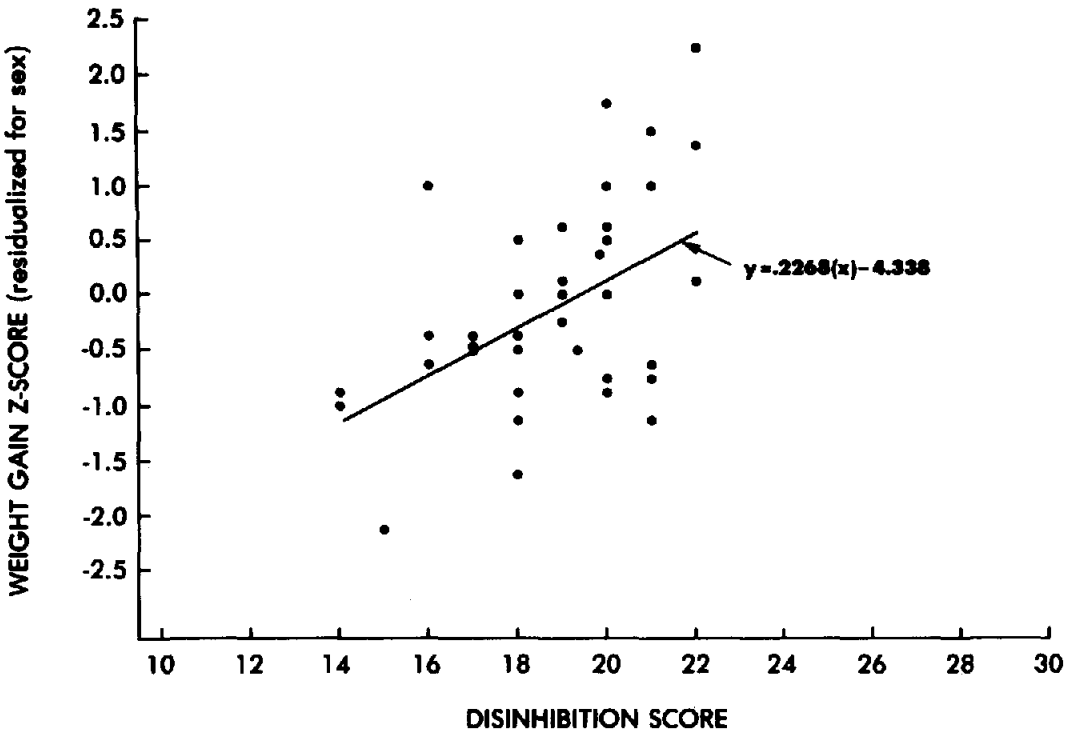


*Figure 2.* Scatterplot for weight change (residualized for sex) and Disinhibition scores at Week 52.

26 weeks was 8.726 lb, or 3.97 kg ($SD$ = 6.497) for subjects abstinent at Week 52 ($n$ = 84). For subjects smoking ($n$ = 24) at Week 52, the mean gain in the first 26 weeks was 4.32 lb, or 1.98 kg ($SD$ = 4.983). The test used was a hierarchial multiple regression including only those subjects abstinent at all assessments to Week 26. Abstinence status at Week 52 was the dependent variable. Sex of subject, weight change to Week 26, and the interaction of these two variables were the independent variables. Effects for sex and the interaction were not significant.

To determine whether the relation of abstinence to weight gain was due to confounding variables, we completed three exploratory hierarchical regressions, with age, pretreatment cigarette intake, and pretreatment cotinine each replacing sex in the equations. Weight change to Week 26 remained significant with the effect of each of these variables removed from the equation.

## Discussion

The weight gain following quitting smoking was rapid. Weight change at 3 weeks after quitting smoking was greater for abstinent subjects than for subjects who continued to smoke. Weights at 6 months were not significantly different from those at 1 year.

Our data confirm those from the retrospective data of Blitzer et al. (1977). Number of cigarettes smoked per day predicted weight gain. That cotinines were not correlated with weight gain is puzzling if nicotine is the primary agent in suppressing weight. Lack of correlation between cotinine and weight gain was probably not due to the difference in cotinine collection methods. Data from both Trials 1 and 2 were examined separately. Significant correlations between weight gain and cotinine were not found with either sample. Cotinines have been predictors of treatment outcome in both trials (Hall, Benowitz, Jones, Herning, & Jacob, 1984; Hall et al., 1985), suggesting that they may, in fact, tap levels of pretreatment nicotine dependence. Support for the role of nicotine in preventing weight gain is further weakened by the failure of nicotine gum to prevent gain in those subjects who received it in the second treatment trial. Of all subjects in the trial, 88% reported using the gum 3 weeks from the beginning of treatment, 59% reported using it at 12 weeks, and 32% used it at 26 weeks. Timing or method of delivery of nicotine, rather than dose, may be critical. Frequent dosing by cigarette puffs may be an optimal nicotine delivery system to prevent weight gain. Eating may also replace the oral component of smoking.

Previous high body weight predicted weight gain for abstinent subjects. The correlation of past maximum weight with weight gain represents more than the tendency of heavier people to gain more weight. Similar correlations were observed between past maximum weight and percentage of body weight gain (not reported by us because of the difficulties that arise when percentages are used in parametric statistical tests). Weight gained after the last quitting attempt did not predict weight gain during the present attempt, perhaps because of recall difficulties. Amount of weight gain may be difficult to remember if the gain is small or if the individual is subject to frequent weight fluctuations.

The robust correlation of Disinhibition scale score and weight gain during abstinence suggests that disinhibited eating plays a role in individual differences in cessation-induced weight gain. It suggests that some smokers use cigarettes to control overeating and to moderate body weight, either by suppression of emotions or by using cigarettes to suppress appetite in high-risk situations. As these speculations imply, cessation-induced weight gain may be related to the broader problem of the regulation of body weight.

Subjects who gained more weight during the first 26 weeks were more likely to be abstinent at 52 weeks. We predicted the opposite, assuming that weight gain would serve as a stimulus for relapse to cigarettes. In retrospect, these data are congruent with the findings of Carroll and Meisch (1984) in a program of laboratory studies with rats and nonhuman primates. These investigators demonstrated that food deprivation increases the reinforcing properties of many drugs. Nicotine or other potentially reinforcing components of cigarettes have not been tested. If the effect does occur with nicotine, subjects who deprive themselves of food to prevent weight gain associated with stopping smoking may inadvertantly increase the reinforcing properties of smoking and increase the probability of relapse. These speculations are compatible with earlier data indicating that subjects who are more overweight at pretreatment are less likely to relapse than are those who are less overweight (Ginsberg et al., 1984). Alternate explanations are that subjects who are unconcerned about weight gain are less likely to relapse to smoking when small gains are noticed or to use other means to control their gain and that the combined psychological cost of weight control and smoking abstinence efforts outweighs the benefit of abstinence (Hall, 1980).

Several of the hypotheses in this paper were tested at more than one time point. Also, both the data set and the hypothesis tested did not lend themselves to strategies that minimize the number of tests performed. Some would say that an experimentwise error term should have been used. Had we done so, the probability level for rejecting the null hypotheses would have been .006 (Fisher's method; Lindman, 1974). We chose to use the traditional .05 alpha level. In part, the traditional alpha was used because these data were unusually consistent. That is, most effects were significant either at both time periods or not at all. The problem of interpreting a single significant result in the midst of a number of nonsignificant results did not arise. Also, we reasoned that in evaluating a health risk, the consequences of a Type II error would be more serious than those of a Type I error. That is, it would be more serious to ignore a potential health risk than to believe there is no risk when the risk factor actually does exist.

We observed similar patterns of relations for tobacco dependence and weight history in both samples. Thus, although a formal cross-replication was not completed, we are at least confident that the relations reported do not reflect the aberrant behavior of a single sample. This is not the case for the Eating Inventory, which was administered only to the subjects in the second trial. Due to the magnitude of the relation between Disinhibition scale score and weight change, we have a moderate degree of confidence in the replicability of this finding. The correlation between Disinhibition score and maximum body weight provides additional construct validity, as does a positive but nonsignificant relation at Week 26 between weight gain and Disinhibition scores. We are less convinced that the relationship between Hunger scale score and weight gain at Week 52 will be replicated. There is no evidence of this relation at other assessments, and the proportion of variance explained is smaller.

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