Dietary Saturated Fat Intake Is Negatively Associated With Weight Maintenance Among the PREMIER Participants

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Research finding on the composition of macronutrient intakes on body weight has not been consistent. Furthermore, little research has examined the impact of subcomponents of macronutrients such as saturated fat or plant protein on body weight. The purpose of this report was to examine the impact of saturated fat, animal and plant protein, and other macronutrient intakes at the end of an intensive intervention on subsequent follow-up body weight. This is a secondary, observational data analysis using data from PREMIER, an 18-month randomized clinical trial that enrolled a total of 810 participants. Participants completed group and individual sessions designed to help them improve blood pressure (BP) control by making lifestyle changes. Dietary intakes were assessed by two 24-h diet recalls at baseline, 6, and 18 months. Body weight and physical fitness were monitored regularly. Regression models were used to examine the impact of animal or plant protein and other macronutrient intakes on subsequent body weight. After controlling for potential confounders, none of the calorie-contributing nutrient intakes at baseline was associated with subsequent weight at 6 or 18 months. However, a greater intake of saturated fat at 6 months was associated with higher weight at 18 months ($P = 0.002$). A greater intake of plant protein at 6 month was marginally associated with lower absolute weight at 18 month ($P = 0.069$). We conclude that macronutrient intakes before the intervention were not associated with subsequent body weight at 6 or 18 months. However, a lower saturated fat intake achieved after 6-month intervention predicts a lower body weight at 18 months and thus greater weight-loss maintenance.

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INTRODUCTION

Dietary protein has been suggested to influence body weight and weight control. A higher protein intake, or a lower carbohydrate to protein ratio, has been shown to improve weight loss better than isoenergetic diets with lower protein intake (1), to increase satiety and thermogenesis, which, in turn, decreases energy intake (2) and energy expenditure (3), respectively. However, randomized trials have not consistently supported an impact of protein on body weight (4–6). The role of protein intake on the magnitude or efficiency of weight loss is still unclear. Furthermore, very little research has been conducted to examine the impact of protein source on body weight.

Using data from the PREMIER clinical trial, we explored the potential impact of animal and plant protein intakes on body weight over the course of 18 months while simultaneously including all other energy-contributing nutrients in the analysis. Specifically, we examined the following two research questions: (i) What is the impact of baseline dietary intakes on subsequent weight at 6 and 18 months; (ii) What is the impact of 6-month dietary intakes on 18-month weight? Results of this study will expand our understanding of the role of animal and plant protein and other macronutrient intakes on weight status and may assist future intervention programs in designing behavioral guidelines for weight control.

METHODS AND PROCEDURES

Study design

PREMIER was a randomized clinical trial designed to determine the effects of two multicomponent lifestyle interventions on blood pressure (BP) (7). Detailed descriptions of the study design, the intervention programs, and the main results have been published elsewhere (7,8). Participating institutions included the NHLBI Project Office (Bethesda, MD), the Coordinating Center (Kaiser Permanente Center for Health Research in Portland, OR) and four clinical centers (Duke University Medical Center, Durham, NC; Johns Hopkins University, Baltimore, MD; Pennington Biomedical Research Center, Baton Rouge, LA; and Kaiser Permanente Center for Health Research, Portland, OR). Institutional review boards at each center and an external protocol review committee approved the protocol. Each participant provided written consent.

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Study participants

A total of 810 participants were recruited and randomized into the study. Individuals were eligible if they were not taking antihypertensive medication and had a systolic BP of 120–159 mm Hg and diastolic BP of 80–95 mm Hg, based on the mean BP over three screening visits. Other inclusion criteria were age 25 or older and BMI 18.5–45.0 kg/m^2. Major exclusion criteria were regular use of drugs that affect BP, risk category C (target organ damage and/or diabetes) according to the sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC-VI), use of weight-loss medications, prior cardiovascular event, heart failure, angina, cancer diagnosis or treatment in the past 2 years, consumption of >21 alcoholic drinks/week, and pregnancy, planned pregnancy, or lactation.

Intervention

After eligibility was established, study participants were randomly assigned to one of three intervention groups: (i) a behavioral lifestyle intervention that implemented established recommendations (EST) ($n = 268$), (ii) a behavioral lifestyle intervention that implemented established recommendations plus the DASH dietary pattern (EST+DASH) ($n = 269$), or (iii) an advice only control group ($n = 273$). The intervention lasted a total of 18 months with an intensive schedule for the first 6 months (weekly group sessions for 3 months and then biweekly for another 3 months) and then a maintenance schedule of monthly meeting for the last 12 months. Both EST and EST+DASH participants received intensive sessions for weight loss, increasing moderate-intensity physical activity, sodium reduction, and alcohol moderation. In addition, individuals in the EST+DASH intervention were counseled to implement the DASH dietary pattern with the goals of increasing daily intake of fruits and vegetables (9–12 servings) and low-fat dairy products (2–3 servings), and reducing intake of saturated fat (≤7% kcal) and total fat (≤25% kcal). Neither active intervention group was counseled specifically to increase plant protein. In contrast, the control group received a single 30-min individual advice session only at the time of randomization. All intervention sessions were provided by centrally trained interventionists, mainly registered dietitians, in a non-didactive fashion. Throughout the trial, participants in both active interventions kept food diaries, monitored dietary calories and sodium intake and recorded minutes of physical activity. Participants in the EST+DASH group also monitored servings of fruits, vegetables, and dairy products and grams of fat. Participants received a program manual and other printed materials specific to their assigned intervention.

Measurements

All measurements were obtained at clinic visits at baseline (before randomization and before the initiation of any group sessions), and 6 and 18 months (postrandomization). Nutrient intake data was collected via unannounced 24-h dietary recalls conducted by telephone interviews. Two dietary recall interviews (one weekday interview and one weekend day interview) were administered within a period of 3 weeks after each clinic visit by the Diet Assessment Center of the Pennsylvania State University. Nutrient and food group intakes were then calculated using the Nutrition Data System Version NDS-R 1998 (NCC, University of Minnesota, Minneapolis, MN). Across the four clinical sites, the completion rate of the diet recall averaged 98%. The average of the two recalls for each time point was used for the analysis in this report.

Fitness was assessed using a two-stage, 10-min submaximal treadmill exercise test (9) developed for PREMIER (7). A 7-day interviewer-administered physical activity recall was used to assess energy expenditure from physical activity (10).

Temporal ordering

In the PREMIER study, dietary intake based on self-report was assessed within 3 weeks after each study visit (baseline, 6 months, and 18 months). This means that body weight measurements were collected up to 3 weeks prior to the assessment of macronutrient intakes for each visit. That is, logistical reasons prevented the study team from measuring dietary intake in the days leading up to clinical visits, which would have facilitated a more proper analysis of the impact of nutrient intake on body weight by providing possibly higher resolution of the effects. We do not, therefore, examine questions such as "What is the impact of baseline to 6-month changes in nutrient intake on baseline to 6-month changes in body weight?" Carrying out this type of analysis confuses the impact of nutrient intake on body weight with the impact that weight may have on nutrient intake since we cannot exclude the possibility that prior levels of body weight may affect the level and type of subsequent dietary intake. Thus, our scientific questions and data analysis strategy (see below) respect the real temporal ordering of macronutrient intake and weight measurements found in the data. For example, rather than examining the "concurrent" association between macronutrient intake and weight at each time point, our scientific questions examined the impact of macronutrient intake on subsequent (future) weight.

Statistical analysis

Differences across time in nutrient and food group intakes were examined by *F*-test. Multivariate full-information maximum likelihood longitudinal regression analyses were used to examine research questions 1 and 2 (listed in Introduction section) (11). Two separate models were fit to the data. For question 1, the outcome variable used was the longitudinal (6 and 18 month) measure of weight. The longitudinal model for question 1 included a dummy variable for time (6 months vs. 18 months), continuous baseline nutrient measures including energy, saturated fat, other fat, fiber, nonfiber carbohydrate, animal protein and plant protein, alcohol, and time-by-nutrient interaction terms. For question 2, the outcome variable used was the 18-month measure of weight. The model for question 2 also included the same continuous baseline nutrient measures.

In both models, residual error terms were assumed to follow a mean-zero bivariate normal distribution with unstructured covariance matrix. In addition, both models were adjusted for measured confounders of the effects of interest—that is, all observed covariates possibly directly related to both subsequent weight and baseline nutrient intake (for question 1) or nutrient intake at 6 months (for question 2) were included in the regression models. For question 1, these covariates included age, gender, race, income, education, assigned treatment, site, cohort, and baseline weight, fitness level, energy expenditure and energy intake. For question 2, additional covariates included fitness level, energy expenditure, energy intake, and weight at 6 months. Finally, because it was assumed *a priori* that the macronutrients may work together (synergistically) or against each other (antagonistically) to impact weight loss or gain, both models included all two-way interactions between the macronutrients.

In all analyses, pooled data from the three treatment groups were used because the primary aim was to examine the nutrient impact on subsequent weight status regardless of treatment groups. However, treatment was always included in the analysis models. Furthermore, as a secondary aim, we examined whether the impact of nutrient on subsequent weight differed by treatment group, but no impact was observed.

In order to enhance communication of the results in a clinically meaningful way, the following two conventions were observed: First, all variables entered in the regression models were grand mean-centered. This makes it so that the impacts of the macronutrients on subsequent weight represent marginal effects, averaged over the sample. Second, because the different macronutrients have varying s.d., the effects of the macronutrients on weight were reported as standardized effects (i.e., scaled by their s.d.). This allows one to compare the relative impact of one macronutrient vs. the other. Analyses were performed using R (R Foundation for Statistical Computing, version 2.5.0, Vienna, Austria) and SAS software (SAS for Windows version 9.1; SAS Institute, Cary, NC).

RESULTS

A total of 810 participants were recruited and randomized into the PREMIER study. A detailed description of the characteristics of these participants has been published elsewhere (7). In brief, these

Table 1 Body weight and daily nutrient and food group intakes before and after 6 months intervention

Nutrients and food group[a]	Baseline	6 Months	18 Months	*P* value for *F*-test across time
N[b]	807	712	690	
Body weight, kg	95.2 (18.8)	91.2 (18.9)	91.7 (19.3)	<0.001
Protein, % kcal	15.9 (4.0)	17.2 (4.2)	17.6 (4.7)	<0.001
g[c]	77.1 (19.4)	73.7 (18.0)	75.1 (20.0)	0.24
Animal protein, % kcal	10.7 (4.0)	11.7 (4.3)	12.0 (4.7)	<0.001
g	50.8 (22.6)	48.9 (22.6)	49.4 (22.4)	0.24
Plant protein, % kcal	4.9 (1.5)	5.4 (1.5)	5.4 (1.6)	<0.001
g	23.7 (9.9)	22.4 (8.8)	22.6 (9.8)	0.02
Carbohydrate, % kcal	51.3 (9.6)	55.3 (10.6)	53.9 (10.5)	<0.001
g	245.7 (85.4)	233.0 (85.1)	227.7 (83.8)	<0.001
Nonfiber carbohydrate[d], % kcal	47.5 (9.0)	50.5 (9.8)	49.3 (9.7)	<0.001
g	228.7 (80.8)	214.3 (75.9)	209.1 (77.9)	<0.001
Total fat, % kcal	33.1 (7.6)	28.4 (8.8)	29.3 (8.5)	<0.001
g	73.3 (33.0)	56.0 (29.6)	57.2 (28.2)	<0.001
Saturated fat, % kcal	11.0 (3.2)	9.3 (3.6)	9.5 (3.4)	<0.001
g	24.3 (11.8)	18.4 (11.0)	18.3 (9.9)	<0.001
Nonsaturated fat[e], % kcal	22.4 (5.6)	19.4 (6.1)	20.2 (6.2)	<0.001
g	49.1 (22.7)	37.7 (19.7)	38.9 (19.4)	<0.001
Alcohol, % kcal	1.4 (3.1)	1.5 (3.4)	1.5 (3.2)	0.87
g	4.1 (8.9)	4.1 (9.4)	4.03 (9.02)	0.97
Total dietary fiber, g	16.9 (7.8)	18.8 (8.6)	18.6 (9.3)	<0.001
Energy expenditure, kcal/kg/day	33.7 (2.9)	34.2 (2.6)	34.2 (2.7)	0.001
Physical fitness, beats/min	130.5 (14.5)	122.8 (15.8)	121.6 (15.8)	<0.001

[a]All values are presented as mean (s.d.). [b]All data presented from the participants that were included in the analytical models varies among the three time periods due to missing data in either weight, diet recall, or covariate. [c]Gram amount of the macronutrients were included in the analytical models, however, the commonly used % kcal values were also presented. [d]Nonfiber carbohydrate = total carbohydrate – total dietary fiber. [e]Nonsaturated fat = total fat – saturated fat.

participants averaged 50 ± 8.9 years, had a mean body weight of 95.4 ± 18.9 kg, and a mean BMI of 33.1 ± 5.8 kg/m^2. About 62% of them were females and 37.5% had hypertension at baseline. As shown in **Table 1**, body weight and many nutrient intakes significantly changed over the three study time points.

When linear models were employed to examine the impact of macronutrient intakes on subsequent weight, none of the baseline macronutrient intakes were associated with body weight at 6 or 18 months (data not shown). **Figure 1** shows the impact of 6-month macronutrient intake on 18-month weight. The point estimates represent standardized effects (i.e., the impact of 1 s.d. change in the macronutrient on subsequent weight on the scale of s.d. in weight). The vertical bars represent 95% confidence intervals for each point estimate. A higher saturated fat intake, but not intake of other fat, at 6 months was associated with a higher weight at 18 months (standardized effect = 0.06, 95% confidence interval = (0.022, 0.098), $P = 0.002$). Specifically, on average a one-unit s.d. increase in saturated fat intake at 6 months (this translates to approximately a 11 g change in our sample, roughly the amount of saturated fat in four pats of butter) was associated with a 0.06 s.d. increase in body weight at 18 months (~1 kg change in body weight). Furthermore, we found that a higher plant protein intake at 6 months was marginally associated with a lower body weight at 18 months (standardized effect = −0.031,

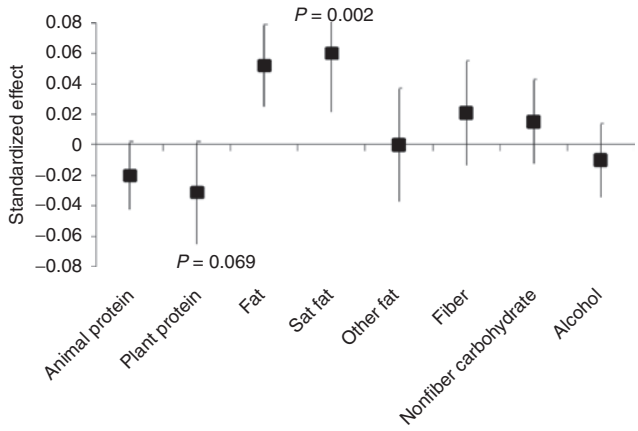

Figure 1 Adjusted effect of the 6-month macronutrients intakes on subsequent body weight at 18 month. A higher saturated fat intake at 6 months was associated with a higher weight at 18 months (saturated fat: standardized effect = 0.06, 95% CI = (0.022, 0.098), $P = 0.002$). On the contrary, plant protein intake was associated in a reverse relationship with weight and the effect approached significance (standardized effect = −0.031, 95% CI = (−0.065, 0.002), $P = 0.069$). The point estimates represent standardized effects (i.e., the impact of 1 s.d. change in the macronutrient on subsequent weight on the scale of s.d. in weight). The vertical bars represent 95% confidence intervals for each point estimate. Total fat effect was estimated from a separate model where total fat was included instead of broken into saturated and other fats. CI, confidence interval.

95% confidence interval = (−0.065, 0.002), $P = 0.069$). Hence, on average a one-unit s.d. increase in plant protein intake at 6 months (~8.8 g) was associated with a 0.031 s.d. decrease in body weight at 18 months (~0.64 kg change in body weight).

DISCUSSION

Our results show that, after the 6 months intervention, dietary saturated fat intake at 6 months, but not the nonsaturated fat, was positively associated with subsequent body weight at 18 months. The higher the saturated fat intake at 6 months, the higher the body weight at 18 months. However, plant protein intake tended to be associated with subsequent body weight inversely. This impact was observed when controlling for potential confounders including demographics, physical activity, and energy expenditure and including all energy-contributing nutrients and fiber in the analysis. Even though a higher fat intake often signifies energy dense diet, this finding suggests that fat source may impact on weight and thus energy balance differently. Findings observed in this study is consistent with that of previous studies showing that adherence to a Mediterranean diet, characterized by a low-saturated fat content, and high plant foods, is associated with lower BMI and obesity (12), reduced adiposity (13), and reduced likelihood of becoming obese among overweight individuals (14).

It should be noted that even though total energy intake and energy expenditure were held constant and "adjusted" for in the analysis, these measures were estimates from self-reports and may not have captured the true energy balance. Besides, there may be energy imbalance that the analytical models did not account for. Despite these limitations, it is reasonable to suspect that saturated fat intake may affect energy metabolism beyond its obvious role of energy contribution, or it may reflect other factors that affect energy metabolism. Indeed, it has been shown that replacing saturated fat with unsaturated fats improved insulin sensitivity and abdominal fat without any change in weight or percentage of total body fat (15). Other research also has shown that fat oxidation may be decreased during a high-fat intake due to impaired or insufficient machinery for fat oxidation. A reduction in fat oxidation subsequently may lead to decreased insulin action (16) and may contribute to a positive energy balance (17–20). It is unclear if the improvement in insulin sensitivity resulted from reducing saturated fat intake can also lead to improvement of fat oxidation or not. Nevertheless, the potential influence of saturated fat alone on weight control deserves further research and clarification.

Conversely, we found that plant protein seemed to impact body weight in a positive/beneficial fashion. A higher plant protein intake at 6 months was found to be marginally associated with a lower weight at 18 months. Neither animal protein nor other nutrients (except for saturated fat as described earlier) examined was associated with the follow-up body weight. Many previous studies have examined the association between protein intake and body weight but the findings have not been consistent (21–24). In observational studies, a higher protein intake was found to be associated with a greater adiposity (21) or obesity (22) or inversely associated with energy intake (23) or body weight (25). In addition, randomized trials (4–6,24) have shown no difference between a higher protein and lower protein intakes on body weight or weight loss. Nevertheless, components such as isoflavone from plant proteins have been shown to lower food intake and thus body weight in rats (26,27). It is unclear if examining animal and plant protein separately or specific protein components in the above trials would have produced more consistent findings or not.

According to the NHANES surveys, saturated fat intake in the United States had decreased some in the past 30 years (from about 13 to 11% kcal) but the averaged intake continues to exceed the recently reduced recommendation of <10% kcal (28). In fact, >95% of the US adults continue to consume more than the recommended amount of saturated fat. On the other hand, total protein intake among US adults has decreased slightly for the past 30 years (29,30) and has fluctuated around 15% of daily energy intake. However, the proportion of animal protein intake has increased substantially during this period of time and thus the proportion of plant protein has substantially decreased (31). It is not clear how this increase in animal protein and decrease in plant protein may be associated with the doubling of obesity prevalence also observed during the same time period (32). It is possible that plant protein may affect energy metabolism via mechanisms not directly related to its energy contribution. As suggested by recent studies (33,34), excess of branched chain amino acids, particularly in the context of high-fat diet, may reduce insulin sensitivity or increase insulin resistance (34) and may contribute to weight gain. Because animal protein contributed nearly twice as much branched chain amino acids to the total protein intake than plant protein did among the PREMIER participants (data not shown), it is not clear how this may have affected the potential role of protein on energy metabolism or balance.

There are limitations to this study. First, the original PREMIER trial was not designed to examine the impact of nutrient source on body weight and thus the current findings are not definitive and need to be confirmed and/or clarified with future studies. Second, since the 24-h diet recall is based on self-report and only two recalls were collected at each study time point, it may not have accurately captured the true intakes of the participants. However, all of the existing dietary assessment methodologies carry similar weakness (35). Third, even though the impact of nutrient on subsequent weight did not differ by treatment group and treatment group was included in all the analyses, treatment could potentially contribute additional confounding.

In conclusion, the result of this study suggests that dietary saturated fat intake at the end of intervention positively predicts the weight maintenance outcome at the 1-year follow-up visit, the lower the saturated fat intake the better the weight maintenance. However, plant protein intake seems to predict the weight outcome at 1 year in an inverse direction, the higher the plant protein intake the lower the follow-up weight. The role of saturated fat and plant protein on energy metabolism and body weight needs to be evaluated further.

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DISCLOSURE

The authors declared no conflict of interest.

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