Diet Quality, Physical Activity, Smoking Status, and Weight Fluctuation Are Associated with Weight Change in Women and Men[1–3]

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Abstract

The effect of diet quality on weight change, relative to other body weight determinants, is insufficiently understood. Furthermore, research on long-term weight change in U.S. adults is limited. We evaluated prospectively patterns and predictors of weight change in Framingham Offspring/Spouse (FOS) women and men (n = 1515) aged ≥30 y with BMI ≥ 18.5 kg/m^2 and without cardiovascular disease, diabetes, and cancer at baseline over a 16-y period. Diet quality was assessed using the validated Framingham Nutritional Risk Score. In women, older age ($P < 0.0001$) and physical activity ($P < 0.05$) were associated with lower weight gain. Diet quality interacted with former smoking status (P-interaction = 0.02); former smokers with lower diet quality gained an additional 5.2 kg compared with those with higher diet quality (multivariable-adjusted P-trend = 0.06). Among men, older age ($P < 0.0001$) and current smoking ($P < 0.01$) were associated with lower weight gain, and weight fluctuation ($P < 0.01$) and former smoking status ($P < 0.0001$) were associated with greater weight gain. Age was the strongest predictor of weight change in both women (partial R^2 = 11%) and men (partial R^2 = 8.6%). Normal- and overweight women gained more than obese women ($P < 0.05$) and younger adults gained more weight than older adults ($P < 0.0001$). Patterns and predictors of weight change differ by sex. Age in both sexes and physical activity among women as well as weight fluctuation and smoking status in men were stronger predictors of weight change than diet quality among FOS adults. Women who stopped smoking over follow-up and had poor diet quality gained the most weight. Preventive interventions need to be sex-specific and consider lifestyle factors. J. Nutr. 140: 1287–1293, 2010.

Introduction

Overweight and obesity have reached epidemic proportions, putting two-thirds of American adults and one-third of adults globally (1,2) at risk for many chronic diseases, including cardiovascular disease (CVD),[13] type 2 diabetes mellitus, and certain forms of cancer (3). Although diet is recognized as a key determinant of overweight and obesity, the role of specific nutrients, particularly fats (4,5) and carbohydrates (6), is controversial, in part due to collinearity and interactions of nutrients and inability to detect small nutrient effects. Dietary patterns, which consider total diet and address the confounding inherent in single nutrient analyses, are increasingly used in nutritional epidemiology to assess associations between diet and disease (7–12). Theoretical (a priori) patterns are based on expert dietary guidelines or composite evidence-based nutrient scoring systems; empirical (a posteriori) patterns, by contrast, are derived statistically by cluster and factor analysis or reduced rank regression and define food and nutrient intake as actually

consumed. Overall diet, assessed by both types of patterns, has been shown to be associated with body weight and to predict short- and medium-term weight change (12–16). However, its effect on long-term weight change, relative to other factors influencing body weight, is insufficiently understood. The relationship between empirical patterns and weight change was modified by BMI status in the Swedish Mammography Cohort (12); smoking, likewise, modified the association between empirical patterns and subclinical heart disease in the Framingham cohort (7). The effect of other body weight determinants on the relationship between theoretical dietary patterns and weight change has not been examined.

Moreover, few studies have examined prospectively long-term weight change patterns in U.S. adults (17). This information, as well as that on the relative impact of diet quality on weight change, is vital for formulation of targeted interventions to reduce overweight and obesity, which is a national health priority (18).

We have previously examined associations with theoretical (11,19,20) and empirical (8) dietary patterns of the Framingham Offspring/Spouse Nutrition Studies cohort and adiposity measures including the long-term development of overweight, total obesity, and abdominal obesity. Our objectives in this study were to: 1) examine the patterns of long-term weight change among Framingham men and women over 16 y of follow-up; 2) evaluate how diet quality compares with demographic, anthropometric, biological, clinical, and other lifestyle factors in predicting weight change in our participants; and 3) assess the effect of these factors, in particular smoking status, on the association between diet quality and weight change.

Materials and Methods

Study population. For over 50 y, the Framingham Heart Study has investigated the risk factors for, and the natural progression of, CVD among residents of Framingham, Massachusetts (21). In 1971, a second-generation cohort was recruited and 5124 Framingham Study offspring and their spouses (2483 men and 2641 women) were invited to participate in the Framingham Offspring/Spouse Study (FOS) (22).

Members of the FOS cohort participate in standardized clinical assessments approximately every 4 y, including a complete physical examination (exam) and laboratory tests, noninvasive diagnostic testing, and updating of medical histories and other pertinent clinical information. At FOS exam 3 (1984–1988), dietary intake of the FOS cohort was comprehensively examined and characterized as the Framingham Nutrition Studies (FNS). These participants completed a single 24-h recall and the Framingham FFQ; 70% also completed the 3-d dietary records (23–25). A total of 3686 women and men (72% of the original offspring cohort) aged 30 y and older with BMI ≥18.5 kg/m^2 attended exam 3; 2966 (80%) of these additionally attended exam 7 (1998–2001). Of these, 2563 (86%) had complete covariate data and 2269 (89%) were free of CVD, diabetes mellitus, and cancer. Of those healthy FNS participants who attended exams 3 and 7 and had complete covariate data, 1515 participants (67%; 825 women and 690 men) had complete 3-d dietary record nutrient intake data; this is the sample used in this study. FNS women and men with complete 3-d dietary data had lower BMI and were less likely to be obese or smoke cigarettes than those without complete dietary data. Women with complete dietary data, in addition, had lower weight (kg), lower prevalence of weight fluctuation, and were more likely to be of normal weight (BMI) than those without complete dietary data (data not shown).

Boston University Medical Center's Human Subjects Institutional Review Board approved the study protocol and all participants provided written informed consent.

Nutrient intake and the Framingham Nutritional Risk Score. Nutrient intake was estimated from 3-d dietary records at exam 3 (1984–1988) using the Minnesota Nutrition Data System software

[version 2.6; Food Database 6A; Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN (26)] as described elsewhere (27,28). Participants were instructed by a registered dietitian in the clinic to record their intake over 2 weekdays and 1 weekend day while adhering to their usual eating practices. Participants were trained to estimate portion sizes using a validated 2-dimensional food portion visual aid (28). Dietary records were processed by trained coders who adhered to standardized protocols.

The Framingham Nutritional Risk Score (FNRS) is a validated 19-nutrient index for assessing diet quality (23,24) (**Supplementa1 Table 1**). The nutrients, which include total energy, protein, total fat, monounsaturated fat (MUFA), SFA, alcohol, cholesterol, sodium, carbohydrate, PUFA, fiber, calcium, selenium, vitamins C, B-6, B-12, and E, folate, and β-carotene, are evidence-based and were originally selected for their relation to CVD risk. Nutrient intake levels among all FOS women (*n* = 1265) and men (*n* = 1200) with 3-d dietary records were ranked from lowest to highest. Ranks were assigned so that an individual with a relatively more desirable intake level (e.g. lower fat or higher vitamin or mineral intake) received a lower rank, whereas a person with a less desirable intake level (e.g. higher total fat or lower micronutrient intake) received a higher rank. An overall composite nutritional risk rank was computed using the mean of the ranks of 19 individual nutrients (23–25). A higher intake of MUFA received a higher rating, because it is derived mainly from animal sources (e.g. beef and fat) rather than vegetable sources (e.g. olive oil) among FNS participants. The FNRS has been shown to predict overweight and obesity (19), abdominal obesity (11), and metabolic syndrome (11).

Weight status and BMI. BMI [weight (kg)/height (m^2)] was calculated from measured height and weight at exam 3 (1984–1988); participants were weighed using a calibrated scale while their height was determined according to standardized procedures using a stadiometer (29). BMI categories (normal weight: BMI 18.5–<25.0 kg/m^2; overweight: BMI 25–<30 BMI kg/m^2; obese: BMI ≥30 kg/m^2) are based on the NIH criteria that were adopted from the WHO classification (30).

Covariates. Sociodemographic, behavioral, anthropometric, and metabolic factors are routinely measured at Framingham exams according to extensively published methods (31). Age, parity, menopausal status, smoking status, physical activity, usual weight pattern, hypertension and lipid medications, and use of hormone replacement therapy were self-reported (8,31).

Physical activity was evaluated by a physical activity index (scores ranged from 24 to 120) based on the number of hours in a typical 24-h day that participants spent doing specific activities that are categorized as sedentary, slight, moderate, or heavy (32). Self-reported dietary behavior was evaluated using the Framingham Food Habit Questionnaire and included usual weight pattern in a year described as stable (± 2.3 kg) or fluctuating (± 4.5 kg) body weight. Response categories were: weight stable and underweight; weight stable and weight just right; weight stable and overweight; weight fluctuates and underweight; weight fluctuates and weight just right; weight fluctuates and overweight (33). BMI was used to establish underweight (BMI <18.5), weight just right BMI (BMI 18.5–<25.0 kg/m^2), and overweight (BMI ≥25 kg/m^2). Blood pressure was determined by duplicate measurements on the participant's left arm using a mercury sphygmomanometer with the participant in a sitting position (34). CVD was defined as coronary artery disease (myocardial infarction, coronary insufficiency, angina pectoris, coronary death), cerebrovascular disease (stroke, transient ischemic attack), peripheral artery disease (intermittent claudication), and heart failure; diabetes mellitus was defined as either fasting blood glucose level of ≥7 mmol/L or treatment with insulin or an oral hypoglycemic agent (31); cancer classification was based on the 1976 WHO International Classification of Disease for Oncology code 185 and includes all cancers except melanoma (38). Diagnoses of CVD and cancer were confirmed with medical records (31). All covariates were measured at exam 3 except for physical activity, which was available at exam 2.

Statistical analysis. The primary objective of our study was to determine patterns of long-term weight change as well as predictors of

weight change in FNS participants over 16 y of follow-up. Weight change (absolute) was defined as weight at exam 7 minus weight at exam 3. Analyses were restricted to women and men without prevalent CVD, type 2 diabetes mellitus, and cancer at baseline who attended exams 3 and 7 and had complete dietary and covariate data. We conducted sex-specific analyses given the gender differences in weight experiences and dietary exposures (8,11,25,36). Baseline characteristics analyzed include age, weight, BMI, parity (women), physical activity index, and the FNRS. Postmenopause (yes/no) and hormone replacement therapy (yes/no) in women, weight fluctuation (yes/no), BMI category (normal weight, overweight, and obese), hypertension treatment (yes/no), lipid treatment (yes/no), and smoking status were also examined at baseline, and development of disease (yes/no) during follow-up. Cigarette smoking status was coded in 3 categories: current, former, and nonsmokers. Current smokers were defined as participants who reported smoking 1 or more cigarettes per day at exam 3 or exam 7 or at both exams; former smokers were defined as participants who stopped smoking between exams 3 and 7; nonsmokers were participants who reported not having smoked at both exams. Socioeconomic and marital status were not evaluated because data were not collected. Baseline characteristics were summarized for each sex using means ± SD for continuous measures or as numbers and percentages for categorical variables.

Patterns of weight change. The study sample was classified into 3 baseline BMI categories (normal weight: BMI 18.5–<25.0 kg/m²; overweight: BMI 25–<30 BMI kg/m²; obese: BMI ≥ 30 kg/m²) and evaluated for mean weight change over 16 y of follow-up. Weight change was also analyzed using 10-y baseline age groups (30–<40, 40–<50, 50–<60, and 60–<70 y of age). The 70–<80 age decile was combined with the 60–< 70 age group due to its small sample size.

We assessed if weight change varied according to BMI category and age group using both age-adjusted and multivariable-adjusted ANCOVA models. Factors selected in backward elimination in the primary analysis (predictors of weight change) were used in the final multivariable-adjusted models and least-squares means ± SE of weight change were calculated for each BMI and age category. Post hoc pair-wise differences were assessed using Tukey's honestly significant difference test where indicated. The SAS PROC GLM was used to fit ANCOVA models (37).

Predictors of weight change. We used the Wilks-Shapiro test to assess that weight change variable was normally distributed. To determine the predictors of weight change, we fitted sex-specific linear regression models with weight change as the outcome variable. To account for collinearity among covariates and select the final set of covariates, we conducted regression analyses using backward elimination with $P <$ 0.05 for retention in the model. The final model contained the following covariates: baseline age, weight, weight fluctuation and physical activity index, smoking category, and the FNRS. Because weight change can depend on initial weight status (12,38), baseline weight was forced in the final model. Results were summarized using regression coefficients ± SE and partial coefficient of determination (R^2), which depicts the proportion of total variation accounted for by each predictor of weight change.

Previous FNS research showed an interaction between empirical dietary patterns and current smoking (7); thus, we tested for interaction between diet quality as measured by the FNRS and smoking status. We also tested for an interaction between diet quality and the other predictor variables (i.e. age, weight, BMI, BMI category, physical activity index, weight fluctuation, hypertension treatment, lipid treatment, disease development, and, among women, parity and menopausal status). Stratified regression analyses were conducted when an interaction term was significant ($P < 0.05$). In secondary analyses, we conducted analyses in FNRS tertiles among all women and men as well as in smoking categories of women. Results were presented as least-squares means and 95% CI. In secondary analyses, we also evaluated weight change in relation to individual component nutrients of the FNRS. We additionally analyzed weight change in a subsample of participants who never smoked before, at baseline, and during follow-up (never smokers) to control for potential residual confounding by smoking status (12,39–41).

All analyses were performed using SAS (version 9.1, SAS Institute) (42). $P < 0.05$ was considered significant. All statistical tests were 2 sided.

Results

At baseline, FNS women had greater prevalence of normal weight, weight fluctuation, and smoking, whereas men had higher weights, higher BMI, and greater prevalence of overweight and obesity. Men were also more likely to develop chronic disease during follow-up (**Table 1**).

Patterns of weight change in women and men. Over one-half of women (62%) and men (55%) gained weight during the 16 y, about one-fifth (women: 18%; men: 21%) lost weight, and nearly one-quarter (women: 20%; men: 24%) maintained stable weight (−2 to +2 kg). Mean weight change was 4.6 ± 0.3 kg among women and 3.6 kg ± 0.3 in men.

Among men, there were no significant differences in weight change across baseline BMI categories, but normal weight and overweight women gained more weight than obese women (4.9 ± 0.4 and 5.0 ± 0.6 vs. 2.0 ± 1.2 kg, respectively; $P < 0.05$) (**Fig. 1***A*).

In both men and women, a uniform decreasing gradient in weight change was observed ranging from 8.0 kg (women) to 7.4 kg (men) in the youngest baseline age group (30–<40 y) to −0.02 kg (women) to 0.4 kg (men) in the oldest baseline age category (60–<70 y) ($P < 0.0001$) (Fig. 1*B*).

Predictors of weight change in women. In multivariable linear regression analysis in women, older age ($P < 0.0001$) and higher levels of physical activity ($P < 0.05$) were associated with less weight gain (**Table 2**). The effect of diet quality on weight change was modified by former smoking status (P-interaction = 0.02). In stratified analyses, former smokers with lower diet quality (tertile 3 of the FNRS) gained on average an additional 5.2 kg compared with those with higher diet quality (P-trend =

TABLE 1 Characteristics of women and men[1]

Characteristic	Women	Men
n	825	690
Age, *y*	48. 7 ± 9.2	48.5 ± 9.0
Weight, *kg*	65. 2 ± 11.7	83.1 ± 11.8
BMI, *kg/m²*	24.7 ± 4.3	26.8 ± 3.4
Parity, *number of births*	2.6 ± 1.7	–
Postmenopause, *%*	48.8	–
Hormone replacement therapy, *%*	6.1	–
Nonsmokers, exams 3 and 7, *%*	75.8	79.3
Former smokers, exams 3–7, *%*	13.8	12.6
Smokers, exam 3 or 7, *%*	10.4	8.1
Weight fluctuation, *%*	22.5	18.7
Physical activity index[2]	33.7 ± 4.6	35.3 ± 5.9
Normal weight (BMI 18.5–<25 kg/m²), *%*	63.0	29.4
Overweight (BMI 25.0–<30 kg/m²), *%*	25.5	54.8
Obese (BMI ≥30 kg/m²), *%*	11.5	15.8
Hypertension treatment, *%*	12.4	14.1
Lipid treatment, *%*	0.7	0.7
Developed disease during follow-up,[3] *%*	17.9	29.3
FNRS [4]	667.1 ± 138.5	584.3 ± 116.8

[1] Values are mean ± SD or percent and are from baseline unless otherwise noted. Unadjusted means for the continuous variables and proportions for categorical variables were calculated.

[2] Physical activity index was measured at exam 2.

[3] Diseases developed during follow-up include CVD, diabetes mellitus, or cancer.

[4] Overall nutrient risk score based on the consumption of 19 nutrients, computed from the sum of the mean scores of the 19 nutrients for each man and woman in the cohort.
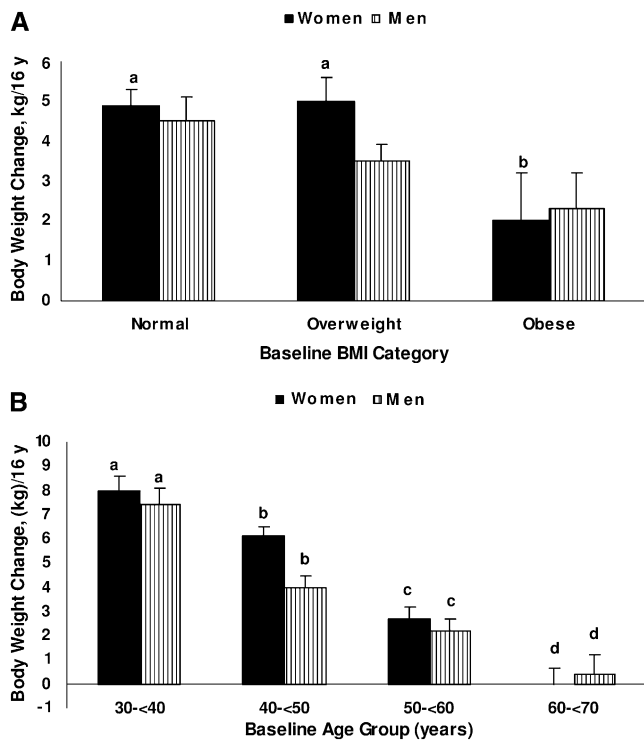
FIGURE 1 Multivariable-adjusted mean weight change (kg) over 16 y by baseline BMI category (*A*) and baseline age group (y) (*B*) in women and men. All values are least-squares mean ± SE, $n = 825$ women (normal weight: $n = 520$; overweight: $n = 210$; obese: $n = 95$) and $n = 690$ men (normal weight: $n = 203$; overweight: $n = 378$; obese: $n = 109$); $n = 825$ women (30–<40 y: $n = 153$; 40–<50 y: $n = 296$); 50–<60 y: $n = 255$; 60–<70 y: $n = 121$) and $n = 690$ men (30–<40 y: $n = 122$; 40–<50 y: $n = 252$); 50–<60 y: $n = 233$; 60–<70 y: $n = 80$). The 60–<70 y and 70–<80 y age categories were combined, because only 3 men and 4 women were in the 70–<80 y age category. Analyses using ANCOVA models were adjusted for baseline age, baseline weight, baseline physical activity index, and FNRS × smoking category interaction in women; baseline age, baseline weight, baseline weight fluctuation, and baseline smoking category in men. For each sex, means without a common letter differ, $P < 0.05$ (Tukey's honestly significant difference test).

TABLE 2 Predictors of weight change in women[1]

	β	SE	P	Partial R_r^2 %
Age, y	−0.3086	0.0291	<0.0001	11.0
Weight, kg	−0.0002	0.0226	0.9917	0.001
Physical activity index	−0.1174	0.0576	0.0418	0.6
Smokers, exam 3 or 7	0.4062	4.5949	0.9296	0.02
Former smokers, exams 3–7	−8.8098	4.6646	0.0593	
Nonsmokers, exams 3 and 7 (referent category)	0	—	—	
FNRS[2]	0.0012	0.0023	0.6071	0.2
FNRS × smokers	−0.0020	0.0061	0.7456	0.02
FNRS × former smokers	0.0154	0.0064	0.0172	
FNRS × nonsmokers (referent category)	0	—	—	

[1] Values are from baseline unless otherwise noted, $n = 825$ women. Multivariable linear regression model was adjusted for baseline age, baseline weight, baseline physical activity index, and FNRS × smoking category interaction.
[2] Overall nutrient risk score based on the consumption of 19 nutrients, computed from the sum of the mean scores of the 19 nutrients for each woman in the cohort.

drate, fiber, calcium, selenium, vitamin C, vitamin B-6, vitamin B-12, folate, vitamin E, and β-carotene were significantly lower in these women than in those with higher diet quality (data not shown).

Age, weight, physical activity, smoking category, diet quality, and the FNRS-smoking category interaction explained 12% of the variation in weight change. Age accounted for the greatest variability (partial $R^2 = 11\%$) in weight change. Physical activity
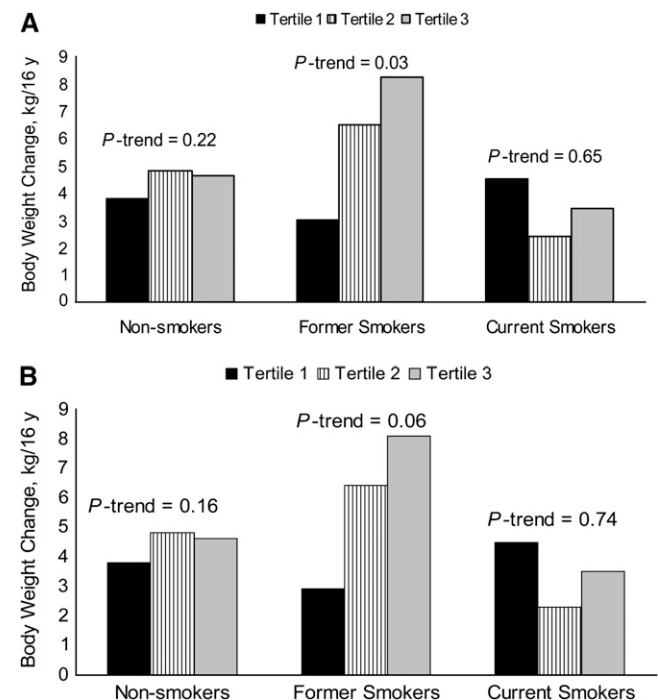
FIGURE 2 Age-adjusted (*A*) and multivariable-adjusted (*B*) mean weight change (kg) in smoking categories by FNRS tertiles among women. All values are least-squares mean (95% CI), $n = 825$ women (nonsmokers: $n = 625$; former smokers: $n = 114$; current smokers: $n = 86$). Multivariable-adjusted analyses, using ANCOVA models, were adjusted for baseline age, baseline weight, baseline physical activity index, baseline FNRS tertile, baseline smoking category, and FNRS tertile × smoking category interaction.

0.03) in age-adjusted analyses (**Fig. 2***A*). However, significance of this increase was attenuated (*P*-trend = 0.06) in the multivariable-adjusted model (Fig. 2*B*). In secondary analyses, the multivariable-adjusted mean weight gain for combined FNRS tertiles 2 and 3 was significant compared with that of tertile 1 [7.5 (6.0, 9.1) vs. 2.9 (−0.5, 6.3) kg; $P < 0.05$]. Mean weight change in nonsmokers, former smokers, and current smokers was 4.4 ± 0.3, 5.8 ± 0.8, and 3.5 ± 0.9 kg, respectively ($P < 0.05$). In age-adjusted analyses, former smokers had a higher mean FNRS compared with nonsmokers ($P < 0.0001$). They also had significantly greater intakes of MUFA and alcohol as well as lower intakes of carbohydrate, fiber, calcium, vitamin C, vitamin B-6, folate, vitamin E, and β-carotene relative to nonsmokers (data not shown).

In single-nutrient analyses of FNRS components, higher total fat consumption was associated with greater weight gain (β = 0.10; $P < 0.05$) and alcohol intake (β = −0.47; $P < 0.05$) was associated with lower weight gain. In age-adjusted analyses, women with lower diet quality had a higher mean FNRS ($P < 0.0001$) compared with those with higher diet quality. They similarly had significantly higher consumption of total fat, SFA, MUFA, cholesterol, and alcohol. Intakes of energy, carbohy-

contributed a smaller proportion (partial R^2 = 0.6%) of total variation and the interaction between diet quality and smoking category accounted for only 0.02% of the overall variation in weight change.

Predictors of weight change in men. In multivariable linear regression analysis in men, older age ($P < 0.0001$) and current smoking ($P < 0.01$) were associated with lower weight gain, whereas weight fluctuation ($P < 0.01$) and former smoking status ($P < 0.0001$) were associated with greater weight gain (**Table 3**). Diet quality was not associated with weight change in men. Age, weight, weight fluctuation, and smoking category jointly accounted for 10.6% of the variation in weight change. Age accounted for the largest variability (partial R^2 = 8.6%) in weight change followed by weight fluctuation (partial R^2 = 1.4%). Diet quality contributed only 0.5% of the variance.

In post hoc analyses, individual FNRS nutrients were not associated with weight change (data not shown). In age-adjusted analyses, men with lower diet quality had a higher mean FNRS ($P < 0.0001$) relative to those with higher diet quality. They also had significantly higher intakes of protein, total fat, SFA, MUFA, cholesterol, and alcohol as well as lower intakes of energy, carbohydrate, fiber, calcium, selenium, vitamin C, vitamin B-6, vitamin B-12, folate, vitamin E, and β-carotene compared with those with higher diet quality (data not shown).

In secondary analyses among never smokers only, similar findings in women and men were observed. Adjusting for energy intake did not qualitatively alter the main results.

Discussion

In the FNS cohort, women who stopped smoking and had poor diet quality gained the most weight during the 16-y period. The small sample size of former smokers may have somewhat attenuated the main findings obtained, although secondary analyses confirmed the significance of diet quality-smoking cessation interaction. Both men who stopped smoking and who had weight fluctuation during follow-up were also at risk for larger weight gain. Older adults, men who continued smoking, and women who exercised more had lower weight gain. Age was the strongest predictor of weight change in both men and women.

Similar to our findings, older age was consistently predictive of lower weight gain among both men and women in other

TABLE 3 Predictors of weight change in men[1]

	β	SE	P	Partial R_r^2 %
Age, *y*	−0.2603	0.0310	<0.0001	8.6
Weight, *kg*	−0.011	0.0248	0.6546	0.07
Weight fluctuation	2.4238	0.7469	0.0012	1.4
Smokers, exam 3 or 7	−2.7360	1.0215	0.0076	0.01
Former smokers, exams 3–7	3.750	0.8449	<0.0001	
Nonsmokers, exams 3 and 7 (referent category)	0	—	—	
FNRS[2]	0.0034	0.0024	0.1607	0.5

[1] Values are from baseline unless otherwise noted, n = 690 men. Multivariable linear regression model was adjusted for baseline age, baseline weight, baseline weight fluctuation, and baseline smoking category.

[2] Overall nutrient risk score based on the consumption of 19 nutrients, computed from the sum of the mean scores of the 19 nutrients for each man in the cohort.

studies (39,43,44). Physical activity was also associated with lower weight gain among women only in previous research in the FOS cohort (13). However, weight fluctuation was predictive of greater weight gain in both sexes in other research (45). Current smoking modified the association between empirical dietary patterns (derived by cluster analysis) and carotid atherosclerosis in previous FNS research (7). We know of no other study, however, that has found an interaction between theoretical patterns and former smoking status, specifically among former smokers only or that has evaluated the relative influences of body weight determinants, including diet quality, on weight change. Predictors of obesity, but not weight change, were examined in the ATTICA study; in relation to sociodemographic, clinical, and other behavioral factors, diet quality as assessed by the Mediterranean Diet Score was not associated with incident obesity (46).

Our findings are also consistent with limited but emerging prospective studies that have examined patterns of weight change in adult populations. Younger participants in the US (17,47), Canada, Scotland, Norway, and Australia (43,44,48–50) experienced the greatest weight gain. Normal-weight and overweight American (17) and Norwegian (49,50) women as well as overweight Australian women (44) likewise had greater weight gains. Only the First National Health and Nutrition Examination Survey (17) had a long follow-up period (20 y) similar to our study; the other studies were of short and medium duration (5–11 y of follow-up).

Smoking status and body weight are generally more strongly inter-related in women than in men. Women are also more vulnerable to weight gain upon ceasing smoking (40,41,51). Evidence suggests that weight gain secondary to smoking cessation is due to higher intakes of energy, total fat, carbohydrates, in particular sucrose and to a lesser extent alcohol, biological parameters such as fat oxidation imbalance and higher lipoprotein lipase activity, as well as decreased energy expenditure, resting metabolic rate, and physical activity (40,41,51). These factors may alter the levels of monoamines and neuropeptides involved in the regulation of food intake and energy expenditure (40,41,51). The higher consumption of MUFA (derived mainly from animal sources in FNS subjects) and alcohol among FNS women who stopped smoking supports these findings. They also had a higher FNRS, which reflects poor overall diet quality and is characterized by higher intakes of dietary lipids (cholesterol as well as percentage of energy from total and saturated fats, and MUFA) and alcohol and lower levels of micronutrients (11,19). Smoking is hypothesized to lower an individual's specific "set-point" (normal body weight level), which is reverted to on ceasing smoking; hence, individuals would gain weight (40,41,51).

In previous FNS research, theoretical and empirical patterns were related to incident overweight and obesity (8,19) and abdominal obesity (11,20) among women. As such, although diet quality may predict transition to excessive weight categories, we conclude from the present study that diet quality as measured by the FNRS is less powerful than other predictors of absolute weight change but exerts its effect on weight gain in sex-specific ways. Our earlier research (25) shows little variation in FNS men's diet, which may also explain the insignificant relationship between diet quality and weight change.

In our analyses of FNRS individual nutrients, higher total fat intake was associated with greater weight gain and higher consumption of alcohol was associated with lesser weight gain among women. None of the nutrients were related to weight change in men in single-nutrient analyses. Alcohol appeared to

be beneficial when evaluated independently but detrimental when assessed jointly with other FNRS components. The low alcohol intake levels in all FNRS tertiles of women (<5% of energy intake) may account for this beneficial effect, consistent with other studies (52,53) that show that light to moderate alcohol consumption is associated with less weight gain. Other studies have also shown that total fat confers risk for weight gain in women (54–56) consistent with our research, as well as in men (54,55,57) unlike our study. Conversely, weight change has not been related to total fat in other studies (39,58). Results for fiber were, however, unexpected, because it is generally associated with less weight gain (6). These inconsistencies may partly be due to the fact that we did not explore interactions between the individual nutrients and smoking status. Also, these findings may highlight confounding in single-nutrient analyses that do not take into account the entire pattern of dietary intakes, as well as biological interactions among nutrients and other metabolic factors.

Among the limitations of this research is the inability to study participants younger than 30 y of age, which could have demonstrated better weight change patterns in younger adults. Similarly, we were limited to women and men who came back for the follow-up exam, which might constitute a somewhat healthier subgroup and thus limit the generalizability of our findings. Only two-thirds of FNS participants had 3-d dietary records; this may have attenuated the diet-weight change association. Dietary records are considered a gold standard for diet assessment and we elected to collect only 3 d, because they have a relatively higher respondent burden. Further, errors associated with any dietary self-report may also have attenuated the findings (59). The FNRS was also not developed specifically to assess weight change. Although it is composed of many of the nutrients that are reported to be relevant to weight change such as total fat, MUFA, PUFA, SFA, protein, carbohydrate, fiber, and calcium (20), other nutrients, including sodium, selenium, vitamin C, vitamin B-6, vitamin B-12, folate, vitamin E, and β-carotene, have no known direct relevance to weight change except as micronutrients that vary overall dietary quality. The FNRS, likewise, does not differentiate between simple and complex carbohydrates, which may differentially affect weight change (6). Finally, although the FOS cohort is exclusively White, our findings may be generalizable to other racial/ethnic populations; biologic mechanisms of diet and weight change are similar in humans, although there may be some genetic variations within- and between-populations. Strengths include a well-characterized population, long follow-up, and evaluation of a broad range of covariates.

These findings showed the differing patterns and predictors of weight change among FOS adults: baseline age in men and women, physical activity in women as well as weight fluctuation and smoking status in men were stronger predictors of weight change than the FNRS, an indicator of diet quality; women who stopped smoking during follow-up and had poor diet quality gained the most weight. When developing preventive behavioral interventions to promote weight loss and prevent weight gain, overweight, and obesity, these results underscore the need for targeting physical inactivity, smoking, and poor diet quality, particularly in women who cease smoking, as well as increased exercise, reduced weight fluctuation, and earlier onset of excess weight especially among men. They also emphasize the need for preventive interventions in normal-weight younger adults as well as in children and adolescents. More research is needed on the effect of diet quality on weight change in younger adults and on weight change in the context of other body weight determi-

nants in other populations. Findings of effect modification are often unable to be replicated (59), so future research is needed to reproduce the interaction effects that we observed in our study.

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