

PAPER

Changes in intake of fruits and vegetables in relation to risk of obesity and weight gain among middle-aged women

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OBJECTIVE: To examine the changes in intake of fruits and vegetables in relation to risk of obesity and weight gain among middle-aged women.

DESIGN: Prospective cohort study with 12 y of follow-up conducted in the Nurses' Health Study.

SUBJECTS: A total of 74 063 female nurses aged 38–63 y, who were free of cardiovascular disease, cancer, and diabetes at baseline in 1984.

MEASUREMENTS: Dietary information was collected using a validated food frequency questionnaire, and body weight and height were self-reported.

RESULTS: During the 12-y follow-up, participants tended to gain weight with aging, but those with the largest increase in fruit and vegetable intake had a 24% of lower risk of becoming obese (BMI ≥ 30 kg/m²) compared with those who had the largest decrease in intake after adjustment for age, physical activity, smoking, total energy intake, and other lifestyle variables (relative risk (RR), 0.76; 95% confidence interval (CI), 0.69–0.86; *P* for trend < 0.0001). For major weight gain (≥ 25 kg), women with the largest increase in intake of fruits and vegetables had a 28% lower risk compared to those in the other extreme group (RR, 0.72; 95% CI, 0.55–0.93; *P* = 0.01). Similar results were observed for changes in intake of fruits and vegetables when analyzed separately.

CONCLUSIONS: Our findings suggest that increasing intake of fruits and vegetables may reduce long-term risk of obesity and weight gain among middle-aged women.

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Introduction

Consumption of five or more servings of fruits and vegetables per day is recommended to reduce the risk of cardiovascular diseases (CVD) through the beneficial combinations of micronutrients, antioxidants, phytochemicals, and fiber in these foods.^{1–4} Also, benefits of fruit and vegetable intake for other chronic diseases⁵ have been suggested. Although a study on food intake patterns indicated that a diet rich in fruits and vegetables was associated with smaller gains in body mass index (BMI),⁶

other studies on dietary pattern and BMI could not consistently predict changes in BMI or obesity development.^{7,8} In addition, data directly relating intake of fruits and vegetables with risk of obesity and long-term weight gain are limited. The present analysis is to examine the direct relation between intake of fruits and vegetables and risk of obesity and long-term weight gain among middle-aged women. We focused on the associations between changes in intake of fruits and vegetables over a 12-y period and risk of obesity and major weight gain.

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Methods

Study population

The NHS was established in 1976 when 121 700 female registered nurses aged 30–55 y from 11 US states responded

to mailed questionnaires regarding their medical history and health practices. In this analysis, we considered 1984 as the baseline since the food frequency questionnaire (FFQ) was expanded in that year. At baseline, we excluded women with history of cardiovascular disease, cancer, or diabetes. We also excluded those who provided incomplete information (ie no data on body weight) or implausible information (ie total daily energy intake <600 or >3500 calories). A total of 74063 women remained in the analysis. The study design, data collection, and analysis were approved by the Brigham and Women's Hospital, and the Harvard School of Public Health Institutional Review Boards.

Assessment of fruit and vegetable intake

Using a semiquantitative FFQ, participants were asked to report the frequencies of 16 fruit items and 28 vegetable items consumed during the previous year. For each fruit or vegetable, a standard unit or portion size was specified. Nine responses were possible, ranging from 'never' to 'six or more times per day'. The response to each food item was converted to average daily intake for each individual participant. The average daily intakes of each fruit and vegetable were summed to compute the total fruit and vegetable intake. Changes in fruit and vegetable intakes were defined by taking the difference between the two measurements in 1984 and in 1994. In this analysis, some specific vegetables were combined into groups.⁹ For example, broccoli, cabbage, cauliflower, and Brussels sprouts were grouped as cruciferous vegetables; carrots, yellow squash, yams, and sweet potatoes were categorized as dark and yellow vegetables; spinach, kale, and lettuce were in the group of green leafy vegetables; and corn, mixed vegetables, celery, eggplant, mushrooms, and beets were categorized into other vegetables. The validity of the FFQ has been evaluated;¹⁰ when comparing FFQ with four 7-day dietary records, the correlation coefficient was 0.80 for apples, 0.79 for bananas, 0.84 for grapefruits, 0.69 for broccoli, and 0.73 for tomatoes.¹¹

Assessment of obesity and weight gain

Body weight was self-reported through the questionnaire every other year with high validity (correlation between self-reported weights and measured weights, $r=0.96$; mean difference, 1.5 kg).¹² Participants were also asked to report their height, and BMI was calculated as weight in kilograms divided by the square of height in meters. Obesity was defined as a BMI of 30 kg/m² or greater. Major weight gain was defined as weight gain 25 kg or greater during the 12-y follow-up.

Statistical analysis

Details of the analytic methods used in the present analysis were described previously.¹³ Briefly, we ranked changes in intake of fruits and vegetables from the largest decrease to

the largest increase during the follow-up period and used quintiles of this variable in the analyses. For example, the first quintile included women who had a large decrease in fruit and vegetable intake (median change = -2.36 servings/day), and the fifth quintile included those with a large increase in intake (median change = +3.99 servings/day). Odds ratios (ORs) were computed by using logistic regression models. In addition to age (5-y category)-adjusted ORs, we estimated multivariate ORs by simultaneously adjusting for age, year of follow-up, change in physical activity (differences in metabolic equivalent (MET) score), change in cigarette-smoking status (increase, decrease, no change), baseline BMI or weight, changes in alcohol consumption and caffeine intake, change in use of hormone replacement therapy (from current use to no use, same use pattern, from no use to current use), and changes in energy-adjusted intakes of saturated fat, polyunsaturated fat, monounsaturated fat, *trans*-unsaturated fatty acid, protein, and total energy. We also examined the multivariate ORs by controlling for baseline covariates rather than changes in covariate status. The median values of quintiles of changes in fruit and vegetable intake were used as a continuous variable for the tests for linear trend. To estimate the mean difference of changes in body weight or BMI from 1984 to 1996 by category of fruit and vegetable intake, we used general linear models with least-square means. We also stratified data according to whether participants' baseline BMIs were greater or less than 25 kg/m² or whether they had incident chronic diseases including diabetes, CVD or cancer. All *P*-values were two tailed and <0.05 was the level of significance. Data were analyzed with SAS (version 8; SAS Institute Inc, Cary, NC, USA).

Results

At baseline, the median daily intake of fruits was 1.9 servings and that of vegetables was 3.2 servings. Participants had similar mean BMIs and body weights across the quintiles of intake of fruits and vegetables. Participants with high fruit and vegetable intakes exercised more, smoked less, and were more likely to use postmenopausal hormones (Table 1).

During the 12 y of follow-up, 10% (6530) of nonobese participants (65 294) at baseline became obese. We observed an inverse association between increased intake of fruits or vegetables and risk of obesity (Table 2). Compared with women who were in the quintile with the largest decrease in intake of fruits (median change in intake of fruits = -1.27 servings/day), women with the largest increase in intake of fruits (median change = +1.86 servings/day) had a 25% lower risk of becoming obese after adjustment for age (OR, 0.75; 95% confidence interval (CI), 0.69-0.81; *P* for trend <0.0001). Further adjustment for physical activity, total energy intake and other covariates did not appreciably alter the OR (0.76; 95% CI, 0.68-0.84; *P* for trend = 0.0007). Also, women with the largest increase in vegetable intake (median

Table 1 Age-adjusted characteristics of study population (N=74 063 women) by quintiles of fruits and vegetables intake in 1984^a

Characteristics	Fruits			Vegetables			Fruits and vegetables		
	1st	3rd	5th	1st	3rd	5th	1st	3rd	5th
Quintile									
# of participants	14 879	14 754	14 764	14 834	14 814	14 812	14 810	14 816	14 813
Median intake (servings/day)	0.61	1.91	3.85	1.54	3.20	5.98	2.63	5.24	9.30
Mean (s.d.) age, y	49 (7)	51 (7)	52 (7)	50 (7)	51 (7)	52 (7)	49 (7)	51 (7)	52 (7)
Body mass index, mean (s.d.), kg/m ²	24.9 (5)	24.9 (5)	24.8 (5)	24.8 (5)	24.9 (5)	25.0 (5)	24.8 (5)	24.9 (5)	25.0 (5)
Weight, mean (s.d.), kg	67 (14)	67 (13)	67 (13)	67 (13)	67 (13)	67 (13)	66 (14)	67 (13)	67 (13)
Weight at age 18, mean (s.d.), kg	57 (9)	57 (8)	57 (9)	57 (9)	57 (9)	58 (9)	57 (9)	57 (9)	58 (9)
Physical activity, mean (s.d.), METs/wk	11 (20)	14 (19)	18 (25)	11 (18)	14 (21)	18 (25)	10 (18)	14 (20)	19 (26)
Vigorous activity, ≥1/week, %	37	46	55	37	46	56	36	46	57
Current smoker, %	39	21	17	29	24	21	34	23	18
Hypertension, % ^b	20	21	22	21	20	22	20	21	22
Hypercholesterolemia, % ^b	8	8	9	7	8	9	7	8	9
Pre-menopausal, %	45	46	45	45	46	45	45	46	45
Current use of hormone replacement therapy, % ^b	13	14	15	13	14	14	12	14	14
Alcohol, mean (s.d.), g	8 (13)	7 (11)	6 (10)	6 (11)	7 (11)	8 (12)	7 (12)	7 (11)	7 (11)
Dietary daily intake, mean (s.d.) ^c									
Total, kcal	1509 (496)	1715 (482)	2056 (530)	1464 (466)	1745 (482)	2026 (545)	1438 (460)	1733 (472)	2086 (530)
Carbohydrate, g	169 (34)	184 (27)	204 (29)	186 (34)	184 (30)	188 (32)	177 (34)	184 (29)	197 (31)
Glycemic load, g	131 (31)	141 (26)	153 (26)	146 (31)	141 (27)	139 (27)	139 (31)	141 (27)	146 (28)
Glycemic index	77 (6)	76 (5)	74 (5)	75 (5)	76 (5)	73 (5)	78 (6)	76 (5)	74 (5)
Protein, g	70 (14)	72 (13)	72 (13)	66 (13)	71 (12)	76 (13)	67 (13)	72 (12)	75 (13)
Polyunsaturated fat, g	12 (3)	12 (3)	20 (3)	11 (3)	12 (3)	12 (3)	12 (3)	12 (3)	11 (3)
Monounsaturated fat, g	25 (5)	23 (4)	23 (4)	24 (5)	23 (4)	21 (4)	24 (5)	23 (4)	20 (4)
Saturated fat, g	24 (5)	22 (4)	20 (4)	24 (5)	22 (4)	20 (4)	24 (5)	22 (4)	20 (4)
Trans-unsaturated fat, g	4 (1)	3 (1)	3 (1)	4 (1)	3 (1)	3 (1)	4 (1)	3 (1)	3 (1)
Cholesterol, mg	294 (112)	289 (92)	269 (88)	279 (105)	288 (92)	286 (97)	286 (109)	288 (91)	277 (92)
Fiber, g	13 (4)	16 (4)	19 (5)	13 (4)	16 (4)	20 (5)	12 (3)	16 (4)	21 (5)
Caffeine, mg	380 (264)	312 (223)	265 (205)	338 (256)	306 (245)	302 (219)	342 (245)	316 (228)	281 (211)

^aFruits: including apple, pear, orange, grapefruit, peach, banana, strawberry, blueberry, cantaloupe, raisin, prune, and fruit juices. Vegetables: including cruciferous vegetable, dark yellow vegetable, tomatoes, green leafy vegetable, legumes, and other vegetable such as corn, mixed vegetable, celery, eggplant, mushroom, and beet. MET indicates metabolic equivalent. Owing to the larger number of subjects, all *P*-values for age-adjusted differences in covariates across quintiles were <0.05, except for the means of body mass index and weight. ^bHypertension, hypercholesterolemia and current use of hormone replacement therapy were defined by self-report. ^cAdjusted for total energy intake.

change = +2.80 servings/day) had a significantly lower risk of obesity after adjustment for potential confounders (OR, 0.84; 95% CI, 0.75–0.93; *P* for trend = 0.0002). Similar results were observed when we combined intakes of fruits and vegetables (Table 2).

A total of 669 (0.9%) women had weight gain of 25 kg or greater during the 12y of follow-up. We found that a considerable decrease in the intake of fruits or vegetables was associated with higher risk for long-term major weight gain (Table 2).

We stratified data according to whether or not participants were overweight (BMI ≥ 25 kg/m²) at baseline. Women who were overweight at baseline gained less weight during the follow-up than did women who were not overweight (Figure 1). Among those who were overweight (38.5%), women in the highest quintile of change in fruit and vegetable intake gained 0.76 kg less weight on average compared with women in the lowest quintile. For women who were not overweight at baseline, the difference in weight gain was 0.52 kg between the fifth quintile and the first quintile of change in fruit and vegetable intake. In addition, women who developed chronic diseases, including cancer, CVD and diabetes during the follow-up period,

gained less weight than did women without incident chronic disease (Figure 2). Among those with incident chronic disease, women with the largest increase in intake of fruits and vegetables gained 0.77 kg less weight than did women in the lowest quintile. The weight gain difference was 0.57 kg among those who did not have chronic diseases.

Discussion

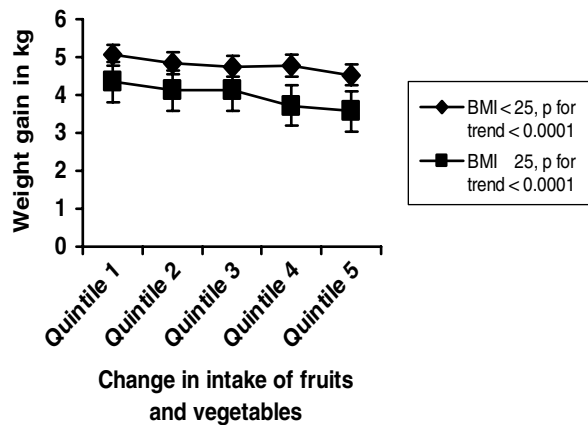
In this 12-y prospective cohort study among middle-aged women, we found an inverse association between the increase of intake of fruits and vegetables over time and risk of obesity or weight gain. These associations were not materially modified by major chronic disease status or baseline body weight. Our findings suggest a link between fruit and vegetable intake and weight gain and support a beneficial effect of increasing intake of these foods on the risk of obesity.

One concern is the accuracy of fruit and vegetable intake measured by FFQ. The FFQ that we used in the dietary assessment has been previously evaluated and reasonably reflects long-term diet including fruit and vegetable

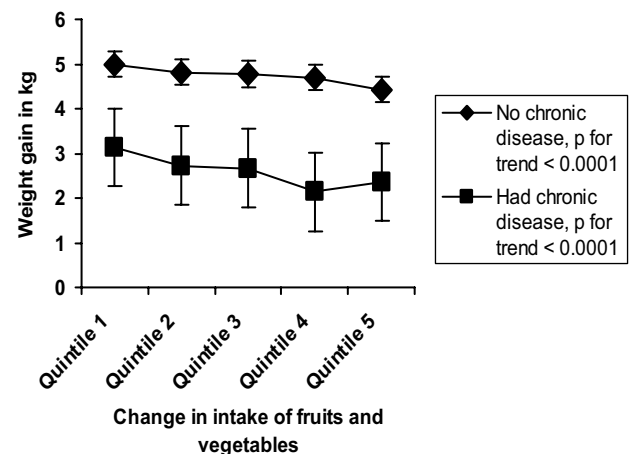
Table 2 Odds ratios (95% confidence intervals) of obesity or major weight gain according to changes in fruit and vegetable intake from 1984 to 1994

	Quintiles of changes in intake					P for trend
	Q1	Q2	Q3	Q4	Q5	
Fruits						
Median change	-1.27	-0.29	0.22	0.80	1.86	
Obesity (BMI ≥ 30)						
Model 1	1.00	0.95 (0.88–1.03)	0.85 (0.78–0.92)	0.82 (0.76–0.89)	0.75 (0.69–0.81)	< 0.0001
Model 2	1.00	0.95 (0.85–1.05)	0.86 (0.77–0.96)	0.84 (0.76–0.94)	0.76 (0.68–0.84)	0.0007
Major weight gain (≥ 25 kg)						
Model 1	1.00	0.70 (0.56–0.89)	0.75 (0.59–0.94)	0.71 (0.56–0.90)	0.72 (0.57–0.91)	0.008
Model 2	1.00	0.68 (0.53–0.89)	0.78 (0.60–1.01)	0.72 (0.55–0.94)	0.73 (0.56–0.95)	0.03
Vegetables						
Median change	-1.72	-0.40	0.36	1.21	2.80	
Obesity						
Model 1	1.00	0.89 (0.82–0.96)	0.89 (0.83–0.97)	0.82 (0.76–0.89)	0.88 (0.81–0.95)	0.0005
Model 2	1.00	0.92 (0.83–1.03)	0.99 (0.89–1.10)	0.85 (0.76–0.94)	0.84 (0.75–0.93)	0.0002
Major weight gain						
Model 1	1.00	0.72 (0.57–0.91)	0.68 (0.54–0.86)	0.72 (0.57–0.91)	0.70 (0.56–0.89)	0.006
Model 2	1.00	0.89 (0.69–1.14)	0.86 (0.66–1.12)	0.88 (0.68–1.15)	0.76 (0.59–0.99)	0.05
Fruits and vegetables						
Median change	-2.36	-0.49	0.64	1.83	3.99	
Obesity						
Model 1	1.00	0.84 (0.78–0.91)	0.86 (0.79–0.93)	0.79 (0.73–0.86)	0.79 (0.73–0.86)	< 0.0001
Model 2	1.00	0.86 (0.77–0.95)	0.87 (0.78–0.96)	0.81 (0.72–0.90)	0.76 (0.69–0.86)	< 0.0001
Major weight gain						
Model 1	1.00	0.71 (0.56–0.89)	0.70 (0.56–0.88)	0.62 (0.49–0.79)	0.72 (0.57–0.91)	0.002
Model 2	1.00	0.80 (0.62–1.03)	0.82 (0.63–1.05)	0.73 (0.56–0.95)	0.72 (0.55–0.93)	0.01

Analyses of obesity conducted among 65 294 women who were not obese in 1984; analyses of major weight gain included all participants. Model 1: adjusted for age (5-y categories). Model 2: adjusting for age, year of follow-up, change in physical activity (differences in metabolic equivalent (MET) score), change in cigarette smoking status (increase, decrease, no change), changes in alcohol consumption and caffeine intake, change in use of hormone replacement therapy (from current use to no use, same use pattern, from no use to current use), and changes in energy-adjusted intakes of saturated fat, polyunsaturated fat, monounsaturated fat, *trans*-unsaturated fatty acid, protein, and total energy and baseline BMI.

**Figure 1** Multivariate adjusted weight gain by quintiles of change in intake of fruits and vegetables according to BMI in 1984.

intake.^{11,14} The FFQ's validity for assessing fruit and vegetable intake was further supported by the fact that it has predicted the risk of cardiovascular diseases in this cohort.¹⁵ In the main analyses, we defined the changes in fruit and vegetable intakes as well as other nutrient intakes as the differences between the two measurements in 1984 and

**Figure 2** Multivariate adjusted weight gain by quintiles of change in intake of fruits and vegetables according to incident chronic disease status.

in 1994. Similar associations were observed when we used the updated dietary intakes in every 2- or 4-y interval, albeit with less magnitude (data not shown). Participants might change their dietary practices if they perceived themselves to be overweight during the follow-up. However, they were

likely to increase the intake of fruits and vegetables according to the dietary recommendation in the past decades. These dietary changes would lead to an underestimation of the inverse association between changes in intake of fruits and vegetables and weight gain.

Another concern is that the magnitude of less weight gain (ie 0.52 kg) over time by increasing intake of fruits and vegetables is relatively small. It may not be reliable to detect such a small amount of difference of weight for individuals using self-reported weight information. However, it is likely to reliably detect this level of difference of mean body weight in a large cohort. Although the amount of less weight gain during a long-term period seems small, it is important in terms of public health significance if the benefit is solely from changes in fruit and vegetable intake. Nevertheless, the likelihood of statistically significant results due to the larger sample size cannot be totally excluded.

We also considered the possibility that increasing intake of fruits and vegetables may be simply a marker of healthy lifestyle.¹⁶ However, the inverse associations changed little when we simultaneously adjusted for age, major lifestyle, and dietary factors, including physical activity, smoking, baseline BMI or body weight, alcohol consumption, caffeine intake, use of hormone replacement therapy, and energy-adjusted intakes of saturated fat, polyunsaturated fat, monounsaturated fat, *trans*-unsaturated fatty acid, protein, and total energy intake. Although other unknown risk factors could theoretically confound the results, the inverse relation we observed is unlikely to be completely explained by the residual confounding because the extensive adjustments we made had minimal impact on the age-adjusted ORs. In addition, the relatively homogenous study population with respect to educational level and socioeconomic status reduced the possibility that our findings were seriously biased. Moreover, total energy intake is an important factor in terms of weight change. In this cohort, women with increased intake of fruits and vegetables had higher total energy intake. In secondary analyses without adjustment for total energy intake, the results were not appreciably altered. Thus, the long-term differences in energy intake with increased fruit and vegetable intake may be too subtle to capture by questionnaire, yet still important for weight controls.

Although the best way to adjust for confounding is through a randomized controlled trial, conducting a large, long-term trial of fruit and vegetable intervention would be difficult. In a small, 1-y randomized trial,¹⁷ Epstein and colleagues found that participants in the increased fruit and vegetable group had a significantly reduced probability of being overweight than those who were in the reduced fat and sugar group. In a randomized clinical trial conducted in India among myocardial infarction patients, Singh *et al*¹⁸ found that those who were randomized to low-energy- and fruit- and vegetable-enriched diet for 6 months had greater reduction in central obesity. Our findings are generally consistent with the results from these clinical trials.

The mechanisms for the inverse association between fruit and vegetable intakes and body weight are uncertain. Dietary fiber may be related to body weight regulation through inducing greater satiety among free-living individuals consuming self-selected diets.^{19,20} Also, fruits and vegetables are low energy density food that may be responsible for mediating energy intake rather than dietary composition *per se*.²¹ Nevertheless, further studies are needed to determine other possible biological and physiological mechanisms.

Reducing obesity and overweight is a major public health and clinical challenge. In addition to the development of national and community-based strategies and initiatives, it is important for dietitians and clinicians to counsel individuals on lifestyle and behavior modification.²² Previous studies have established the crucial role of physical activity and energy expenditure in obesity prevention.²³ In addition, a healthy diet is important to further reduce the risk of obesity or overweight. Results from our study support the notion that increased intake of fruits and vegetables may contribute to lower risk of obesity and major weight gain. On the other hand, our findings also suggest that the risk of obesity or overweight would increase if woman were to substantially reduce her fruit and vegetable intake. Middle-aged women on average gain weight with aging. Increasing intake of fruits and vegetables may facilitate slowing down weight gain and reducing the risk of obesity. Moreover, our findings may have additional significance considering the important influence of middle-aged women in their families' eating pattern.

In conclusion, we observed that increased intake of fruits and vegetables was associated with significantly lower risk of obesity among generally healthy middle-aged women. Our study contributes to the growing evidence that intake of fruits and vegetables is inversely related to body weight. Also, our findings support increasing fruit and vegetable intake as part of a dietary strategy to control overweight and obesity.

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