

Low-fat dietary pattern reduces urinary incontinence in postmenopausal women: post hoc analysis of the Women's Health Initiative Diet Modification Trial



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BACKGROUND: Urinary incontinence affects >40% of women in the United States, with an annual societal cost of >\$12 billion and demonstrated associations with depressive symptoms, social isolation, and loss of work productivity. Weight has been established as an exposure that increases urinary incontinence risk and certain dietary components have been associated with urinary incontinence symptoms. We hypothesized that diet plays a key role in the association between weight and urinary incontinence in US women.

OBJECTIVE: This study aimed to examine the effect of a low-fat diet on urinary incontinence in postmenopausal women as a post hoc analysis of a randomized controlled trial of diet modification.

STUDY DESIGN: This was a post hoc analysis of the Women's Health Initiative Dietary Modification randomized controlled trial of 48,835 postmenopausal women from 40 US centers assigned to a dietary intervention (20% energy from fat, 5 fruits or vegetable servings, and 6 whole grain servings daily and an intensive behavioral modification program) or to the usual diet comparison group. The outcome was urinary incontinence at 1 year.

RESULTS: Of the participants, 60% were randomized to the usual diet comparison group and 40% to the dietary modification intervention. After adjusting for weight change, women assigned to the dietary modification intervention were less likely to report urinary incontinence (odds ratio, 0.94; 95% confidence interval, 0.90–0.98; $P=.003$), more likely to report urinary incontinence resolution (odds ratio, 1.11; 95% confidence interval, 1.03–1.19; $P=.01$), and less likely to develop urinary incontinence (odds ratio, 0.92; 95% confidence interval, 0.87–0.98; $P=.01$) in adjusted models.

CONCLUSION: Dietary modification may be a reasonable treatment for postmenopausal women with incontinence and also a urinary incontinence prevention strategy for continent women. Our results provide evidence to support a randomized clinical trial to determine whether a reduced fat-intake dietary modification is an effective intervention for the prevention and treatment of urinary incontinence. In addition to providing further insights into mechanisms of lower urinary tract symptoms, these findings may have a substantial impact on public health based on the evidence that diet seems to be a modifiable risk factor for urinary incontinence.

Keywords: diet, menopause, postmenopause, urinary incontinence, weight, Women's Health Initiative dietary modification, Women's Health Initiative

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The authors report no conflict of interest.

The findings of this study were presented at the annual scientific meeting of the American Urogynecologic Society, Providence, RI, October 3–7, 2017. This trial was registered with ClinicalTrials.gov (ClinicalTrials.gov Identifier: NCT00000611).

The Women's Health Initiative is funded by the National Heart, Lung, and Blood Institute, National Institutes of Health, and the US Department of Health and Human Services, and the study utilized resources obtained through the Veteran Affairs Puget Sound Health Care System, Seattle, WA.

Cite this article as: Rogo-Gupta LJ, Yang L, Stefanick ML, et al. Low-fat dietary pattern reduces urinary incontinence in postmenopausal women: post hoc analysis of the Women's Health Initiative Diet Modification Trial. *Am J Obstet Gynecol Glob Rep* 2022;2:100044.

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2666-5778/\$36.00

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AJOG Global Reports at a Glance

Why was this study conducted?

This study aimed to examine the effect of a low-fat dietary intervention on urinary incontinence (UI) symptoms in postmenopausal women. We performed an intent-to-treat post hoc analysis of the Women's Health Initiative Dietary Modification randomized controlled trial after 1 year of following a diet intervention designed to reduce fat and increase fruit, vegetable, and whole grain intake.

Key findings

After adjusting for weight change, women in the intervention group reported a lower incidence of incontinence and were more likely to report incontinence resolution than women assigned to the usual diet comparison group.

What does this add to what is known?

Our results provide evidence to support a randomized clinical trial to determine whether a reduced fat-intake dietary modification is an effective intervention for prevention and treatment of UI, suggesting that diet is a modifiable risk factor for UI.

Introduction

Urinary incontinence (UI) affects >40% of US women. The number of affected women is projected to increase by 55% from 18.3 million in 2010 to 28.4 million by the year 2050.^{1,2} UI has a profound societal impact with an annual cost of more than \$12 billion and demonstrated associations with depressive symptoms, social isolation, and loss of work productivity.^{3–5}

Being overweight is one established risk factor for UI but a precise pathophysiological relationship is unknown. Based on a randomized controlled trial that demonstrated improvement in UI among women with overweight and obesity following weight reduction, weight loss has been recommended as an effective treatment for UI.^{6,7} The relationship between weight loss and UI improvement is, however, challenging to study, because of the many confounders including lifestyle factors, such as diet composition, physical activity, hormonal status, socioeconomic status, aging, and medical treatments.⁸ Only a few longitudinal studies have examined the possible association between diet, an important confounder in the relationship between weight and UI, and UI. One study found that increased vegetable, bread, and chicken consumption led to a reduced risk for overactive bladder, a condition associated with UI, among women⁹ and that

higher fat and lower bread intake led to a higher risk for activity-related UI.¹⁰ Incident UI was twice as likely to occur among participants with the highest dietary fat intake than those with the lowest dietary fat intake.¹⁰ In addition, specific micronutrients have been associated with risk of onset of lower urinary tract symptoms.^{9,11} Considering these data, we hypothesized that, apart from the association with weight, a low-fat diet reduces UI in postmenopausal women.

Our primary objective was to examine the impact of a low-fat dietary intervention on UI symptoms in postmenopausal women. To understand this impact, we performed an intent-to-treat analysis of the intervention overall. We also sought to examine whether the dietary intervention differentially impacted women with and without incontinence to determine whether the intervention reduced the development of new UI symptoms or improved the resolution of UI symptoms. Our secondary objective was to analyze the association between UI symptoms and specific dietary intervention components such as fat, fruits and vegetables, whole grains, and total energy individually.

Materials and Methods
Study cohort

This is a post hoc analysis of the randomized controlled Women's Health

Initiative Dietary Modification (WHI DM) trial that enrolled a group of 48,835 postmenopausal women between 1993 and 1998 at 40 US clinical centers with the primary outcomes of breast and colorectal cancer. Detailed descriptions of the intervention and methodology of the WHI DM trial have been published previously.^{12–14} Eligibility criteria included an age of 50 to 79 years, postmenopausal status, willingness to provide informed consent, and at least a 3-year life expectancy. Women consuming <32% of their calories from fat as estimated by a food frequency questionnaire (FFQ) were excluded from enrollment in the WHI DM trial.¹⁵ Women were excluded from our analysis if their UI status was missing at baseline or at year 1. All other WHI DM trial participants were included in the analysis. Women were randomly assigned to a dietary modification intervention (DM-I) or to the usual diet comparison group (DM-C). The DM intervention was aimed at reducing total fat intake and increasing fruit, vegetable, and whole grain intake.

Exposure

Specifically, intervention (DM-I) participants were asked to change their daily diet so that 20% of their energy intake was from fat and to include 5 servings of fruits and vegetables and 6 servings of whole grains. Groups of 8 to 15 DM-I participants entered an intensive behavioral modification program, which included 18 group sessions in year 1 led by specially trained and certified nutritionists and registered dietitians with focus on both nutritional and behavioral topics each session. In contrast, DM-C participants received health education materials at the time of randomization and no contact with nutrition interventionists. Women were encouraged to adjust their diet to maintain their baseline weight; however, 21% of intervention (DM-I) participants and 7% of control (DM-C) participants lost weight.¹⁶ Although weight loss was not a goal, intervention participants lost 2 kg more on average between baseline and year 1 than controls.

Participants reported their dietary intake by completing an FFQ intended to detail intake of selected nutrients at the time of, and the 3 months before, completion of the questionnaire (www.whi.org). Outcomes were self-reported and not otherwise evaluated or confirmed.

Outcomes

Self-reported questionnaire responses (Form 37—Thoughts and feelings) were used to define UI. Women who answered yes to the question, “Have you ever leaked even a very small amount of urine involuntarily and you couldn’t control it?” (Form 37—Question 121) were considered to have UI; whereas those who responded no to the question were considered not to have UI. Those who responded yes were then asked, “How often does this leaking urine occur?” (Form 37—Question 122) and those who answered “Not once during the past year” were also considered to have no UI, whereas those who responded “less than once a month,” “greater than once a month but less than once a week,” “one or more times a week but less than every day,” or “daily” were considered to have UI. The outcomes are self-reported and not adjudicated.

Covariates

Baseline and 1-year demographics, health evaluations, and symptom questionnaires were reviewed. Dietary intake was collected using the FFQ responses. We included nutrients previously associated with urinary symptoms in the analysis^{9,10} and collected the following information: (1) percentage of daily energy from fat (kcal), (2) daily fat intake (grams), (3) daily energy intake (kcal), (4) daily servings of vegetables and fruits (number of daily servings), and (5) daily servings of grains (number of daily servings). Measured weight and height were used to calculate body mass index (BMI) and analyzed as a covariate. Additional covariates included demographic and medical characteristics and physical activity measured in metabolic equivalent of task (MET) hours per week. Participant age, weight,

physical activity level, alcohol intake, number of term pregnancies, years since menopause, and waist circumference were analyzed as continuous variables, whereas the remainder were evaluated as categorical variables. Specific dietary components were based on calculated data from self-reported FFQs and analyzed as effect modifiers of the overall association between diet and UI. The FFQs were self-reported and not adjudicated.

Statistical analyses

Baseline characteristics were summarized using number and percentage for categorical variables, whereas mean and standard deviations are presented for continuous variables. The absolute standardized difference between the intervention and control arms was calculated.¹⁷

To address our primary objective, we explored whether the dietary intervention was associated with self-reported UI at year 1 by performing a logistic regression model adjusted for weight change between baseline and year 1. Because we included all women who were randomized in the DM trial in the primary analyses, we did not adjust for other baseline characteristics. To allow the impact of the dietary intervention on incontinence symptoms to vary by baseline continence status, we analyzed the women with and those without incontinence at baseline as 2 separate subcohorts. The first (subcohort 1) included women without UI at baseline who reported UI at year 1. Clinically, this subcohort represented women who developed de novo UI during the year of the intervention. The second (subcohort 2) included women with UI at baseline who recovered from UI by the end of year 1. Clinically, this subcohort represented women who experienced incontinence resolution during the year of the intervention. Because we did not include all randomized women when performing this subanalysis, we adjusted for the additional covariates age, race and ethnicity, dietary water intake, relevant medications, and hormone replacement trial (HT) treatment.¹⁸

In a secondary analysis, we evaluated how each of 5 specific dietary components would modify the intervention effect on the risk of developing incontinence at year 1. We fitted 5 separate logistic models for each of the 3 outcomes, including the interaction term between dietary components and the intervention arm. We tested the interaction term’s significance using a likelihood ratio test to determine whether dietary intake was an effect modifier. In the sensitivity analyses, we repeated all analyses while adjusting for an additional covariate in the model, namely weight change between baseline and year 1, because of the known association between weight change and incontinence.^{6,7}

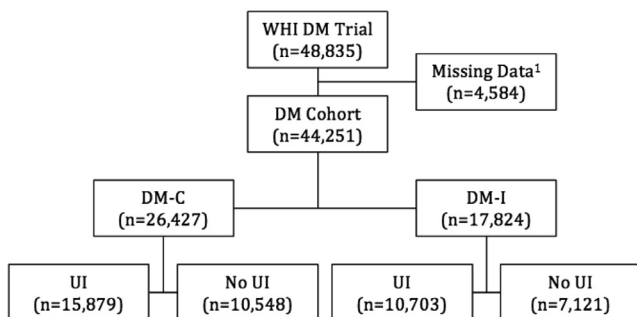
All tests were 2-sided and *P* values of <.05 were considered to be statistically significant. Absolute standardized differences ≥ 0.1 were considered to be large. To address missing data, imputations were applied to all models using multivariate imputation by chained equations.¹⁹ The results from each imputation data set were combined using Rubin’s rule and an overall multivariate imputation estimate and associated standard error were summarized.²⁰ All analyses are performed in SAS 9.3 and R 3.3.3.²¹ The Stanford University institutional review board determined that the research protocol was exempt (# 41284).

Results

Patients with missing UI data were excluded ($n=4584$), leaving 44,251 of the original DM cohort of 48,835 women for this post hoc analysis (Figure). Of the 44,251 WHI DM participants included in this analysis, 26,427 (60%) had been randomized to the DM-C and 17,824 (40%) to the DM-I (Figure). Most of the cohort was non-Hispanic White (83%) and had a college level education or higher (78%). There were no large differences between the control and intervention participants at baseline (Table 1) (absolute standardized differences ≥ 0.1 were considered to be large). However, there were large differences between arms in diet at year 1 and weight change

FIGURE

Cohort selection of postmenopausal women included from the WHI DM Trial



WHI DM Trial = Women's Health Initiative Dietary Modification Trial
 DM-I = Dietary Modification-Intervention
 DM-C = Dietary Modification-Control

¹ Excluded women who did not answer the question, "Have you ever leaked even a very small amount of urine involuntarily and you couldn't control it?"

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TABLE 1

Demographic, dietary, and exercise characteristics of postmenopausal women in the Women's Health Initiative Dietary Modification trial cohort by dietary modification study arm at screening visit

Characteristic	DM study arm		ASD
	Control (n=26,427)	Intervention (n=17,824)	
Demographic			
Age ^a (y)	62.37 (6.84)	62.34 (6.83)	0.005
Term pregnancies ^a	3.20 (1.56)	3.18 (1.54)	0.013
Years since menopause ^a	14.37 (9.05)	14.25 (8.95)	0.013
Ethnicity			0.015
Asian or Pacific Islander	628 (2.4)	409 (2.3)	
American Indian or Alaskan Native	97 (0.4)	70 (0.4)	
Black or African-American	2599 (9.8)	1821 (10.2)	
Hispanic or Latino	884 (3.3)	594 (3.3)	
White (non-Hispanic)	21,874 (82.8)	14,696 (82.5)	
Other	294 (1.1)	196 (1.1)	
No data	27 (0.2)	62 (0.2)	
Education			0.017
Above college	7247 (27.4)	5016 (28.1)	
College	13,229 (50.1)	8863 (49.7)	
High school	5801 (22.0)	3842 (21.6)	
No data	150 (0.6)	103 (0.6)	
Smoking			0.021
Current smoker	1718 (6.5)	1126 (6.3)	
Never smoked	13,683 (51.8)	9118 (51.2)	
Past smoker	10,785 (40.8)	7391 (41.5)	
No data	241 (0.9)	189 (1.1)	

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between baseline enrollment and year 1 (Table 2).

Primary analysis

In the DM-I subgroup, 10,703 women (60.0%) reported UI at baseline, whereas 11,779 (66%) reported UI at 1 year. In the DM-C subgroup, 15,879 (60.1%) reported incontinence at baseline, whereas 17,856 (68%) reported incontinence at 1-year. After adjusting for weight change, women in the intervention group were less likely to report any incontinence at 1 year (odds ratio [OR], 0.94; 95% confidence interval [CI], 0.90–0.98; $P=.001$) (Table 3).

Secondary analysis

In the analysis of the 19,805 women without baseline UI (subcohort 1), 5536 (28%) developed incontinence by year 1. When adjusted for weight change, HT, age, and ethnicity, those enrolled in the low-fat dietary intervention were less likely to develop de novo incontinence (OR, 0.92; 95% CI, 0.87–0.98; $P=.013$) at year 1 than the controls. In the analysis of women with baseline UI (subcohort 2), those who participated in the low-fat dietary intervention were more likely to report clinical resolution of UI than controls (OR, 1.11; 95% CI, 1.02–1.19; $P=.011$) in the model adjusted for weight change, HT, age, and ethnicity (Table 3). The dietary modification was beneficial for both the prevention of de novo UI and for the resolution of existing UI.

Analysis of specific dietary components previously associated with UI revealed that individual components did not modify the association between the dietary intervention and incontinence in any group (Table 4). The results of the sensitivity analysis evaluating weight change over the study period were unchanged from the main findings.

Discussion

Principal findings

This post hoc analysis of a year-long dietary intervention designed to reduce fat intake and increase intake of fruit, vegetables, and whole grains in postmenopausal women shows that the

TABLE 1

Demographic, dietary, and exercise characteristics of postmenopausal women in the Women's Health Initiative Dietary Modification trial cohort by dietary modification study arm at screening visit (continued)

Characteristic	DM study arm		ASD
	Control (n=26,427)	Intervention (n=17,824)	
Region			0.005
Midwest	5484 (20.8)	3679 (20.6)	
Northeast	6272 (23.7)	4228 (23.7)	
South	6586 (24.9)	4479 (25.1)	
West	8085 (30.6)	5438 (30.5)	
Hormone status			
Hysterectomy	11,437 (43.3)	7673 (43.0)	0.005
Bilateral oophorectomy	5369 (20.3)	3542 (19.9)	0.012
Female hormone exposure	17,317 (65.5)	11,607 (65.1)	0.01
WHI HT enrollment	4380 (16.5)	2889 (16.2)	0.03
Estrogen alone control	957 (3.6)	601 (3.4)	
Estrogen alone intervention	931 (3.5)	557 (3.1)	
Estrogen and progesterone control	1172 (4.4)	845 (4.7)	
Estrogen and progesterone intervention	1320 (5.0)	886 (5.0)	
Weight			
Weight ^a (kg)	76.34 (16.35)	76.65 (16.61)	0.019
Waist circumference, ^a cm	88.78 (13.65)	88.90 (13.79)	0.008
BMI category			0.022
Underweight (<18.5)	83 (0.3)	59 (0.3)	
Normal (18.5–24.9)	6927 (26.2)	4632 (26.0)	
Overweight (25.0–29.9)	9492 (35.9)	6366 (35.7)	
Obesity I (30.0–34.9)	6015 (22.8)	4010 (22.5)	
Obesity II (35.0–39.9)	2629 (9.9)	1868 (10.5)	
Extreme obesity III (≥40)	1164 (4.4)	819 (4.6)	
No data	117 (0.4)	70 (0.4)	
Diet and exercise			
Multivitamin use	9500 (35.9)	6423 (36.0)	0.011
Alcohol, weekly servings	2.06 (3.99)	2.04 (3.89)	0.005
Dietary water intake ^a	1549.65 (621.36)	1544.89 (623.59)	0.008
Recreational physical activity, MET h per wk ^a	10.22 (12.06)	10.14 (11.71)	0.006
Medication use			
Diuretic	3547 (13.4)	2379 (13.3)	0.002
Anticholinergic	0 (0.0)	0 (0.0)	<0.001
α -blocker (antagonist)	64 (0.2)	60 (0.3)	0.018
B-blocker (antagonist)	2105 (8.0)	1472 (8.3)	0.011
B agonist	661 (2.5)	448 (2.5)	0.001

Data are presented as number (percentage), unless otherwise specified.

ASD, absolute standardized difference; BMI, body mass index; MET, metabolic equivalent of task; DM, Dietary Modification; WHI HT, Women's Health Initiative Hormone Replacement Trial.

^a Continuous variable presented as mean (standard deviation).

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intervention is associated with a significant reduction in UI symptoms, including resolution of incontinence, and a reduction in the risk of developing de novo incontinence.

Results

Only a few longitudinal studies have examined the possible association between the total diet and UI after accounting for weight. One such study among United Kingdom women from the Leicestershire *Medical Research Council Incontinence Study* of women >40 years old²² described a lower risk for overactive bladder with increased intake of vitamin D, protein, and potassium⁹ and a higher risk of activity-related UI with higher fat, zinc, and vitamin B12 intake.^{10,22} US epidemiologic data are derived mostly from the Boston Area Community Health (BACH) Survey, a community-based study of urologic symptoms and risk factors among adults aged 30 to 79 years in Boston, Massachusetts, from 2002 to 2005. These data also suggested that for US women, decreasing saturated fat relative to polyunsaturated fat and decreasing total calories had a beneficial effect on UI.²³ Other published data for US women has been primarily focused on the relationship between weight and UI. Epidemiologic evidence suggests that obesity is a risk factor for the development of UI²⁴ and randomized control trials have shown an improvement in UI with weight reduction among women with overweight and obesity^{6,7} with the strongest impact on stress UI.²⁵ This body of work is relevant because the increase in weight among American women continues. Although this and other research establishes weight as a factor that increases UI risk, the exact mechanism by which weight loss decreases UI has not yet been defined.

Clinical implications

The statistically significant difference associated with the dietary intervention investigated in this post hoc analysis translates to a reduction in the overall UI risk of 8% to 11%. Our results demonstrate the potential of a low-fat

TABLE 2
Weight and dietary intake of postmenopausal women in the Women's Health Initiative Dietary Modification trial cohort by Dietary Modification study arm at year 1

Characteristic	DM study arm Control (n=26,427)	Intervention (n=17,824)	ASD
Weight at year 1, kg ^a	76.26 (16.68)	74.35 (16.69)	0.114
Weight change between baseline and year 1, kg ^a	-0.01 (8.36)	-2.27 (8.25)	0.271
Fat-percent of energy, Kcal			1.506
0–27.9	3683 (13.9)	12,606 (70.7)	
27.9–32.2	4971 (18.8)	2350 (13.2)	
32.2–36.8	7064 (26.7)	1417 (7.9)	
≥36.8	10,246 (38.8)	1148 (6.4)	
No data	463 (1.8)	303 (1.7)	
Fat-total intake, g			0.905
0–46.2	8172 (30.9)	12,349 (69.3)	
46.2–59.8	5630 (21.3)	2758 (15.5)	
59.8–76	5212 (19.7)	1392 (7.8)	
≥76	6950 (26.3)	1022 (5.7)	
No data	463 (1.8)	303 (1.7)	
Energy intake, Kcal			0.157
0–1382.11	10,527 (39.8)	7905 (44.4)	
1382.11–1663.6	5184 (19.6)	3800 (21.3)	
1663.6–1909.48	3612 (13.7)	2466 (13.8)	
≥1909.48	6641 (25.1)	3350 (18.8)	
No data	463 (1.8)	303 (1.7)	
Vegetables and fruits, daily servings			0.519
0–2.3	5989 (22.7)	1898 (10.6)	
2.3–3.3	5818 (22.0)	2466 (13.8)	
3.3–4.6	6120 (23.2)	3661 (20.5)	
≥4.6	8037 (30.4)	9496 (53.3)	
No data	463 (1.8)	303 (1.7)	
Grains, daily servings			0.336
0–3	8310 (31.4)	3724 (20.9)	
3–4.3	6970 (26.4)	4093 (23.0)	
4.3–5.9	5838 (22.1)	4259 (23.9)	
≥5.9	4846 (18.3)	5445 (30.5)	
No data	463 (1.8)	303 (1.7)	

Data are presented as number (percentage) unless otherwise specified.

ASD, absolute standardized difference; DM, Dietary Modification.

^a Continuous variable are presented as mean (standard deviation).

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dietary intervention to reduce the prevalence and incidence of incontinence among postmenopausal women in the United States.

There are possible biologic explanations for an association between nutrients and UI.

One explanation is an inflammatory process. Epidemiologic evidence from both the Leicestershire MRC Incontinence Study and the BACH survey reported positive associations among total fat, saturated fat, and total energy intake and UI (both adjusted for weight).^{10,23} Serum C-reactive proteins have been positively associated with lower urinary tract symptoms.²⁶ It is plausible that a higher intake of dietary fat induces up-regulation of inflammatory mechanisms, such as serum C-reactive proteins and proinflammatory cytokines, leading to increased urinary symptoms.²⁶ The dietary intervention in the WHI included a reduction of fat, and therefore our results revealed a reduction in urinary symptoms plausibly because of an attenuation of the inflammatory response seen with high-fat diets. A second possible explanation relates to bowel function. The association between bowel and urinary function is generally accepted, specifically that constipation is associated with worsening urinary symptoms²⁷ and that fiber intake is inversely associated with constipation. Therefore, it is possible that the increase in fiber intake in the DM-I trial also contributed to improvement in UI through an improvement in bowel function. Although our secondary analysis did not reveal statistically significant associations with individual nutrients such as fiber (Table 4), it is worth mentioning that the OR suggested that moderate grain intake may be associated with a decrease in UI. Lack of specific data on bowel function precluded further analysis.

Research implications

Our results support a randomized clinical trial to determine whether a reduced fat-intake dietary modification is an effective intervention for the prevention and treatment of UI. Further studies into the mechanism of action of specific

TABLE 3**Urinary incontinence in postmenopausal women after 1 year of a low-fat dietary intervention compared with controls overall and stratified by baseline urinary incontinence status**

Primary analysis	Baseline status	Year 1 status	Number	OR (95% CI)	P value
DM-I vs DM-C	All women	UI	44,251	0.94 (0.9–0.97)	.001
Subcohort analysis					
Subcohort 1	No UI	UI	17,669	0.92 (0.87–0.98)	.013
Subcohort 2	UI	No UI	26,582	1.11 (1.02–1.19)	.011

CI, confidence interval; DM-C, Dietary Modification-Control; DM-I, Dietary Modification-Intervention; OR, odds ratio; UI, urinary incontinence.

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TABLE 4**Association of individual dietary components with urinary incontinence after 1 year of a low-fat dietary intervention compared with controls overall and stratified by baseline urinary incontinence status**

Variables	Primary analysis		Subcohort 1		Subcohort 2	
	OR (95% CI)	Interaction P value	OR (95% CI)	Interaction P value	OR (95% CI)	Interaction P value
Fat-percent of energy Kcal		.212		.388		.653
0–27.9	1.07 (0.99–1.16)		1.04 (0.92–1.17)		1.09 (0.94–1.26)	
27.9–32.2	0.97 (0.87–1.08)		0.95 (0.80–1.12)		1.07 (0.89–1.29)	
32.2–36.8	0.99 (0.88–1.12)		0.87 (0.71–1.06)		1.02 (0.82–1.27)	
≥36.8	0.92 (0.81–1.05)		0.89 (0.72–1.10)		1.24 (1.00–1.55)	
Total fat intake, g		.492		.843		.186
0–46.2	1.06 (1.00–1.13)		0.96 (0.87–1.05)		0.94 (0.84–1.05)	
46.2–59.8	1.02 (0.93–1.13)		1.01 (0.87–1.18)		1.05 (0.88–1.26)	
59.8–76	1.03 (0.90–1.17)		1.03 (0.85–1.26)		1.12 (0.88–1.42)	
≥76	0.94 (0.81–1.09)		1.03 (0.82–1.29)		1.22 (0.95–1.56)	
Energy intake, Kcal		.144		.112		.925
0–1382.11	1 (0.94–1.06)		0.94 (0.87–1.04)		1.05 (0.94–1.18)	
1382.11–1663.6	0.92 (0.84–1.01)		0.87 (0.76–1.01)		1.07 (0.91–1.27)	
1663.6–1909.48	0.87 (0.78–0.97)		0.81 (0.68–0.97)		1.08 (0.88–1.34)	
≥1909.48	0.95 (0.87–1.05)		1.05 (0.91–1.21)		1.13 (0.96–1.33)	
Vegetables and fruits, daily servings		.631		.824		.177
0–2.3	0.93 (0.83–1.04)		0.94 (0.80–1.12)		1.16 (0.96–1.41)	
2.3–3.3	0.92 (0.84–1.02)		0.97 (0.82–1.13)		1.30 (1.09–1.55)	
3.3–4.6	0.98 (0.89–1.04)		0.89 (0.80–0.98)		1.00 (0.85–1.18)	
≥4.6	0.91 (0.85–0.97)		0.88 (0.79–0.97)		1.09 (0.97–1.22)	
Grains, daily servings		.280		.082		.722
0–3	0.96 (0.89–1.04)		0.97 (0.86–1.1)		1.14 (0.98–1.31)	
3–4.3	0.89 (0.82–0.97)		0.87 (0.76–0.99)		1.19 (1.02–1.39)	
4.3–5.9	0.86 (0.79–0.94)		0.78 (0.68–0.89)		1.10 (0.94–1.29)	
≥5.9	0.94 (0.86–1.02)		0.95 (0.83–1.08)		1.05 (0.90–1.23)	

CI, confidence interval; OR, odds ratio.

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dietary components and UI symptoms are also warranted.

Strengths and limitations

We acknowledge certain limitations in our analysis. First, we chose to analyze the effect on overall UI without focusing on the UI subtypes (stress urinary incontinence [SUI], urgency urinary incontinence [UUI], and mixed urinary incontinence [MUI]). We felt that the method of self-reported symptom assessment in the WHI cohort did not allow for a true assessment of SUI, UUI, and MUI separately. Trial participants responded to questions on the presence or absence of overall UI and separate symptom and severity questions for SUI and UUI, but not for MUI. We understand that analyses using different methodology might yield different results, however, this approach of overall UI has the advantage of avoiding misclassification. Second, we could not control for certain conditions known for their association with UI such as pelvic organ prolapse. Although this condition was evaluated among the participants in the HT arm of the WHI trial, it was not considered in the randomization of the DM trial and therefore it was not included in the analysis. However, other covariates associated with both UI and prolapse in this study were included such as pregnancy terms, menopausal status, age, weight, hormonal status, and previous hysterectomy. We also did not specifically explore the source of dietary components, such as fat, and thus we are unable to draw conclusions regarding the association of different fat sources with incontinence. Women in the control arm did receive nutritional information, which may have influenced their food choices during the intervention year. Furthermore, we relied on the trial randomization to minimize confounding by other sources such as medications. Although current practice standards include second and third generation medications for urgent urinary incontinence treatment and minimally invasive surgery for SUI,²⁸ practice standards during the years of DM trial enrollment relied on bladder retraining, pelvic exercises, and first generation antimuscarinic medications.^{29,30}

US Food and Drug Administration approval of a first generation antimuscarinic agent for urinary symptoms (Ditropan) occurred in 1975 (oral) and 1979 (liquid) followed by the approval of oxybutynin (generic) in 1989. This first generation of medications was associated with a high discontinuation rate³¹ because of bothersome side effects and frequent dosing schedule. For this reason, we suspected it was unlikely that a significant number of women in this large trial were actively taking antimuscarinics for medical management of incontinence. This was confirmed by our findings that no women in our cohort self-reported taking these medications. This was also a predominantly White, highly educated cohort of women and our findings may not be generalizable to other populations. We also relied on the trial randomization to minimize confounding from fluid intake when appropriate. The DM-I focused on lowering dietary fiber without specific fluid intake instructions. It is possible that DM-I and/or DM-C participants changed the fluid intake choices after enrollment. Although the FFQ did include fluid intake questions, the DM-I intervention did not include fluid intake instructions. Similarly, the nutrition information received by the DM-C group (Dietary Guidelines for Americans) included guidance on daily servings of milk and alcohol but not for other liquids. Although we felt that the trial randomization was sufficient to wash out any differences in fluid intake between the DM-I and DM-C groups, we did adjust for dietary fluid intake as part of the subanalysis as described in the statistical analysis section. Lastly, the DM trial was an intensive short-term intervention and we present 1 year outcome data, therefore, we are unable to draw conclusions regarding the long-term effect of dietary intake on weight and incontinence.

Despite these limitations, our study has many strengths. Our cohort represents a large cohort of postmenopausal US women. This post hoc analysis of a randomized controlled dietary intervention trial is consistent with data from previous large epidemiologic studies^{23,32} and adds valuable insight into the relationship between weight, diet, and UI in

a postmenopausal population. The potential impact of such a low-risk, low-cost intervention to prevent UI in this population with medical comorbidities should be underscored.

The FFQ questionnaire provided extensive self-reported food intake information, for example animal protein type, milk category, types of fat consumed, and types of snacks, deserts, and condiments consumed.³³ Participants were also instructed following FFQ completion, which led to a large data set to use for analysis.³⁴ This information can be used to study the associations between dietary components that were not included in the current study and urinary incontinence symptoms. In addition, future studies on dietary interventions to decrease comorbidities could include more detailed information on urinary incontinence and pelvic organ prolapse. Our data could be used to design a low-risk, low-cost intervention to prevent UI in a more diverse population of continent postmenopausal women or to treat UI among symptomatic women. In designing these prospective randomized trials, we suggest including the most recent evidence-based dietary recommendations (eg, from the Dietary Guidelines for Americans 2020–2025).³⁵ These recommendations include updates compared with that used in the WHI DM trial that may be relevant to a study designed to evaluate the association between intake and urinary symptoms.

Conclusions

Our observations suggest that dietary modification may be both a reasonable treatment option for postmenopausal women with UI and a reasonable prevention option for continent postmenopausal women who desire to stay dry. In our cohort, continent postmenopausal women had an incident UI rate of 30% at 1 year, indicating that there is a need to explore UI prevention regimens in this age group. ■

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