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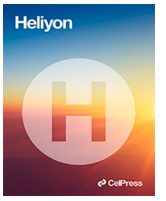

Research article

Association between dietary fatty acids and urinary incontinence

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ABSTRACT

Background: Dietary nutrient intake contributes to urination; however, the association between dietary nutrient intake, especially that of fat, and urinary incontinence (UI) is not well understood. The most common types of UI include stress UI (SUI) and urgency UI (UUI).
Objective: To investigate the potential effect(s) of dietary fat intake on UI and explore its mechanism of action in relation to body mass index (BMI).
Methods: A cross-sectional survey of data from 15,121 individuals (20–85 years of age) from the National Health and Nutrition Examination Survey (2001–2008), a random population-based sample, was performed. Data regarding dietary nutrient intake were collected through 24 h dietary recall interviews. UI and covariate data were collected through in-person interviews. UI was assessed according to the American Urological Association Symptom Index. The odds ratio (OR) for SUI and UUI were calculated using multivariate logistic regression analysis. The mediation effect was estimated using observational mediation analysis.
Results: Higher total fat intake was positively associated with increased odds for developing UI (OR 1.44 [95% confidence interval (CI) 1.08–1.93]). Females who consumed more saturated fatty acids (SFA), monounsaturated fatty acids (MUFA), and polyunsaturated fatty acids (PUFA) were more likely to develop SUI. BMI partially explained the association between total fat, SFA, MUFA, and PUFA and SUI; the proportions of the mediation effect of BMI were 14.7%, 13.0%, 18.7%, and 16.3%, respectively.
Conclusions: Results of this study emphasize the key role of dietary fat intake in the prevalence of UI. Higher fat intake was positively associated with UI and BMI partially mediated the effect of fat intake on SUI.

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1. Introduction

Urinary incontinence (UI) is a source of daily concern among those affected by the condition. It lowers the quality of life of millions of adults worldwide, affecting nearly 50% females and 39% of males [1]. UI is a type of lower urinary tract symptom (LUTS) defined as involuntary urine loss [2], and its frequency increases with age. Possible causes of UI include abnormal autonomic nervous system activity, chronic bladder inflammation, sleep disorders, and depression [3,4]. Nutrition contributes to whole-body homeostasis, including systemic inflammation, autonomic nervous system function, and bladder function [5,6]. Although dietary nutrition is associated with prostatic inflammation [7], the role of dietary nutrients in UI remains unclear.

Several studies have shown that nutrient intake can affect UI [8,9]. Results have revealed positive associations between UI and total energy, sodium, zinc, calcium, and vitamin C. Lower fat or higher protein intake reduces the risk for symptomatic benign prostate hyperplasia [10]. Our previous studies also indicated a critical role of fatty acid metabolism in regulating prostate development, which may lead to urinary retention [11,12]. Therefore, fatty acids may play a crucial role in the development of UI. However, fatty acid subtypes, such as saturated (SFA), polyunsaturated (PUFA), and monounsaturated (MUFA), have specific effects on UI, and the role of diet-sourced fatty acids in urinary control remain unclear.

Fat intake directly regulates systemic energy metabolism and affects body weight [13]. According to previous studies, obesity is linked to UI through increased abdominal pressure, weak pelvic muscles, and pelvic innervation disorder [14–18]. The effect of fat intake on UI may be mediated by body mass index (BMI). As such, we examined the associations between fat intake, BMI, and UI using data from the National Health and Nutrition Examination Survey (NHANES), a cross-sectional survey, and focused on urological symptoms and dietary nutrient intake.

2. Materials and methods

2.1. Data source

NHANES is a cross-sectional study designed to evaluate the health and nutritional status of adults and children in the United States. Written informed consent was obtained from all participants and the study was approved by the National Center for Health Statistics (NCHS). Datasets from four NHANES cycles (2001–2002, 2003–2004, 2005–2006, and 2007–2008) were used in this study because they included questionnaires related to dietary fat intake and urinary symptoms.

2.1.1. Study population

The present study involved data from 15,121 participants who responded to questionnaires during 24 h dietary recall interviews and kidney/urological/prostate conditions. Participants with incomplete UI or baseline data were excluded from analysis. A flow-diagram illustrating participant selection is presented in Fig. 1.

2.1.2. Questionnaire data assessment

In the NHANES, UI was assessed using three questions: does your urine leak during physical activities? (question KIQ042); do you urinate before reaching the toilet? (question KIQ044); and does your urine leak during non-physical activities? (question KIQ046). According to the American Urological Association Symptom Index (AUASI), UI is defined in patients experiencing at least weekly or monthly leakage of volumes of more than a few drops in the past 12 months. A positive response to question KIQ042 was defined as stress UI (SUI), while a positive response to question KIQ044 was defined as urgency UI (UUI). Positive responses to question KIQ046

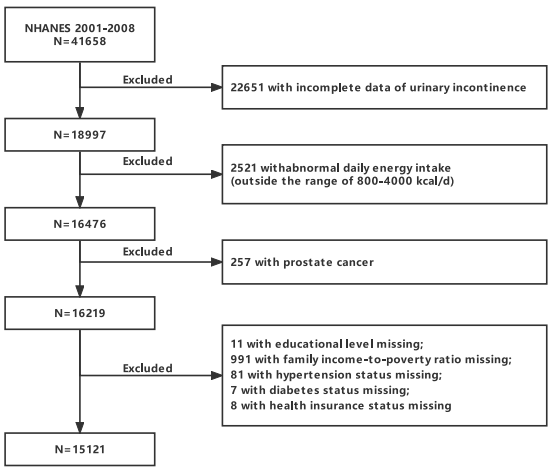

Fig. 1. Flowchart the sample from NHANES.

indicated other types of UI(s). These definitions are consistent with those of the International Continence Society.

2.2. Statistical analysis

Information regarding daily dietary nutrient intake was obtained through a 24 h dietary recall interview conducted during the NHANES. Intake of the most specific nutrients was correlated with total energy intake; thus, dietary nutrient intake may be non-causally associated with UI owing to confounding by total energy intake. Fatty acid intake was estimated based on these two recalls. To eliminate the mixed effects of individual food consumption, nutrient density (nutrients divided by energy [g/1000 kcal]) was calculated, and data regarding dietary nutrient intake were log-transformed to improve normality in the regression analyses.

A mediating-effect model was used to determine whether obesity mediated the association between fat intake and SUI. Only intermediates that fulfilled the following criteria as potential mediators were analyzed: significant associations between exposure and outcome variables; and significant association between the exposure variables and mediators [19]. The direct and indirect effects (DE and IE, respectively) were estimated using the PROCESS macro of SPSS version 16.0 (IBM Corporation, Armonk, NY, USA) [20], in which DE represents the estimated effect of fat intake on SUI after controlling for BMI, and IE is the estimated effect of fat intake on SUI that operates through BMI. Statistical analyses were performed using SPSS version 16.0.

Odds ratio (OR) and corresponding 95% confidence interval (CI) for UI status and its association with dietary fat intake were calculated using logistic regression. The reference group in the logistic regression was participants without UI or those with a BMI <25.0 kg/m^2. Multivariate logistic regression analyses were performed to determine whether fat intake was associated with UI, while controlling for race, sex, age, education level, BMI, health insurance, family income-to-poverty ratio, diabetes, and hypertension (Table 2). Furthermore, the association between specific fatty acid intake and UI was evaluated using multivariate logistic regression analysis. The sampling design of the NHANES requires weighted observations that are inversely proportional to the probability of selection. Appropriate sample weights, strata, and cluster design variables were considered under NCHS guidance. All statistical tests were two-sided and performed at an alpha value of 0.05. Statistical analyses were performed using SPSS version 16.0 and R version 4.2.0 (R Foundation for Statistical Computing, Vienna, Austria <http://www.r-project.org/>).

Table 1
Weighted Characteristics of Study Participants in NHANES (2001–2008), overall and by Urinary Incontinence Status (n = 15121).

	Total (n = 15121)	No Urinary incontinence (n = 9629)	Urinary incontinence (n = 5492)	*P* Value
Age in years, mean (SD)	46.7 (16.8)	43.6 (16.2)	52.6 (16.2)	<0.001
Age group, %				
20-29	18.6	23.9	8.6	
30-39	19.1	21.8	14.1	
40-49	20.6	20.2	21.4	
50-59	18.0	15.8	22.1	
60-69	12.3	10.0	16.6	
≥70	11.3	8.2	17.2	
Gender, %				<0.001
Male	46.3	61.4	17.9	
Female	53.7	38.6	82.1	
Race/ethnicity, %				<0.001
Mexican American	7.5	8.0	6.6	
Other Hispanic	3.8	4.1	3.2	
Non-Hispanic White	73.5	71.8	76.6	
Non-Hispanic Black	10.5	11.0	9.7	
Other races	4.7	5.1	3.9	
Education level, %				<0.001
Less than high school	17.3	16.4	19.0	
High school/GED	25.0	24.3	26.3	
Above high school	57.8	59.4	54.8	
Family income-to-poverty ratio, %				0.86
≤1	12.4	12.4	12.5	
>1	87.6	87.6	87.5	
Body mass index, kg/m^2, %				<0.001
<25.0	32.6	34.4	29.3	
25.0–29.9	34.9	36.6	31.6	
≥30.0	32.5	29.0	39.1	
Covered by health insurance, %				<0.001
Yes	83.0	81.5	85.8	
No	17.0	18.5	14.2	
Diabetes, %	7.6	6.0	10.5	<0.001
Hypertension, %	30.2	25.4	39.2	<0.001
Total fat intake, gm/day, mean (SD)	37.6 (10.0)	37.3 (10.0)	38.4 (9.9)	<0.001
Normalized fat intake, gm/1000 Kcal (SD)	79.56 (37.01)	81.99 (37.96)	74.97 (34.68)	<0.001

Table 2
Odds ratios for urinary incontinence by dietary fat intakes among participants with or without UI (n = 15121).

	Samples size	OR (95% CI)	*P* value
Total Fat, median g/1000 Kcal			
UI	5492	1.44 (1.08–1.93) *	0.014
Stress UI	3692	1.77 (1.27–2.49) ***	<0.001
Urgency UI	3244	1.15 (0.83–1.59)	0.397
Mixed UI	1624	1.53 (0.99–2.40)	0.060
Other UI	1191	1.11 (0.69–1.82)	0.671
SFA, median g/1000 Kcal			
UI	5492	1.40 (1.11–1.75) **	0.004
Stress UI	3692	1.42 (1.10–1.85) **	0.008
Urgency UI	3244	1.12 (0.87–1.45)	0.365
Mixed UI	1624	1.18 (0.85–1.66)	0.326
Other UI	1191	1.18 (0.81–1.72)	0.400
MUFA, median g/1000 Kcal			
UI	5492	1.26 (0.98–1.62)	0.067
Stress UI	3692	1.46 (1.09–1.95) *	0.011
Urgency UI	3244	1.13 (0.86–1.49)	0.394
Mixed UI	1624	1.36 (0.94–1.99)	0.108
Other UI	1191	1.05 (0.70–1.60)	0.814
PUFA, median g/1000 Kcal			
UI	5492	1.19 (0.99–1.44)	0.070
Stress UI	3692	1.36 (1.09–1.69) **	0.006
Urgency UI	3244	1.10 (0.89–1.36)	0.385
Mixed UI	1624	1.30 (0.97–1.73)	0.072
Other UI	1191	0.92 (0.67–1.26)	0.598

UI = urinary incontinence; SFA = saturated fat; MUFA = monounsaturated fat; PUFA = polyunsaturated fat.
OR = odds ratio; CI = confidence interval.
*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$.
The distributions of nutrients intakes were standardized with individual energy intake and log-transformed to improve the normality in the regression analyses.
All models controlled for age, gender, race/ethnicity, education level, body mass index, health insurance, family income-to-poverty ratio, diabetes, and hypertension.
The reference group in logistic regression was participants without UI.

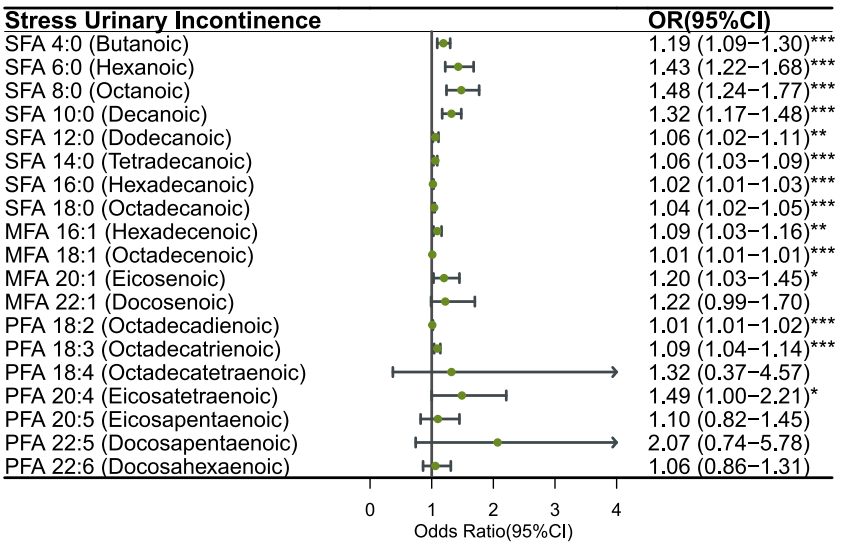

Fig. 2. Odds Ratios for Stress Urinary Incontinence across Fat Acids Groups among Participants. SFA = saturated fat; MUFA = monounsaturated fat; PUFA = polyunsaturated fat. OR = odds ratio; CI = confidence interval. *$P < 0.05$. **$P < 0.01$. ***$P < 0.001$.

3. Results

3.1. Overall

Of the 15,121 survey participants, 5492 (36.3%) reported UI. The mean (±SD) of the 15,121 participants was 49.6 ± 18.5 years. Among the participants with UI, 67.2% (n = 3692) experienced SUI and 59.1% (n = 3244) experienced UUI. From 2001 to 2008, females were more commonly affected by UI than males (52.7% versus [vs.] 17.6%, respectively).

3.2. Fat intake and UI

The weighted differences in demographic and dietary characteristics of participants with and without UI were compared (Table 1). Participants who reported UI were significantly more likely to be older (52.6 ± 16.2 vs. 43.6 ± 16.2 years; P < 0.001) and non-Hispanic white (76.6% vs. 71.8%; P < 0.001), with a higher BMI (BMI ≥30.0 kg/m^2 [39.1%] vs. 29.0%; P < 0.001), and total fat intake (38.4 ± 9.9 vs. 37.3 ± 10.0; P < 0.001), and developed diabetes (10.5% vs. 6.0%; P < 0.001) and hypertension (39.2% vs. 25.4%; P < 0.001).

The results of multivariate analyses of dietary fat intake and UI are summarized in Table 2. Total fat intake was positively associated with UI (OR 1.44 [95% CI 1.08–1.93]). Compared with UUI, SUI was more significantly associated with total fat intake (UUI, OR 1.15 [95% CI 0.83–1.59] vs. SUI, OR 1.77 [95% CI 1.27–2.49]). Due to the crucial role of SFAs and unsaturated fats in systemic inflammation and oxidative stress, the relationship between SFAs, unsaturated fatty acids, and UI were analyzed. Regression results revealed that SFA, MUFA, and PUFA significantly increased the risk for SUI (SFA, OR 1.42 [95% CI 1.10–1.85] vs. MUFA, OR 1.46 [95% CI 1.09–1.95] vs. PUFA, OR 1.36 [95% CI 1.09–1.69]). However, no significant association was observed between the risk for UUI and intake of different types of fat. The ORs for developing SUI with different fatty acid types, including 8 types of SFAs (4:0–18:0), 4 types of MUFA (16:1–22:1), and 7 types of PUFA **(omega-6 [n-6] PUFA**, 18:2–20:4; **omega-3 [n-3] PUFA**, 20:5–22:6) are presented in Fig. 2. Among these specific fatty acids, SFA (4:0–18:0), MUFA (16:1–20:1), and n-6 PUFA (18:2–18:3, 20:4) were significantly and positively associated with SUI.

Table 3
Odds ratios for urinary incontinence by dietary fat intakes among women in NHANES (2001–2008) (n = 8075).

	Samples size	OR (95% CI)	*P* value
Total Fat, median g/1000 Kcal			
UI	4255	1.64 (1.13–2.36) **	0.009
Stress UI	3345	1.83 (1.27–2.66) **	0.001
Urgency UI	2228	1.19 (0.79–1.81)	0.398
Mixed UI	1408	1.63 (1.00–2.68)	0.050
Other UI	859	1.37 (0.81–2.30)	0.240
SFA, median g/1000 Kcal			
UI	4255	1.46 (1.10–1.93) **	0.009
Stress UI	3345	1.42 (1.07–1.88) *	0.015
Urgency UI	2228	1.11 (0.81–1.53)	0.506
Mixed UI	1408	1.21 (0.84–1.76)	0.302
Other UI	859	1.15 (0.73–1.81)	0.555
MUFA, median g/1000 Kcal			
UI	4255	1.41 (1.03–1.93) *	0.030
Stress UI	3345	1.50 (1.10–2.06) *	0.011
Urgency UI	2228	1.20 (0.85–1.71)	0.309
Mixed UI	1408	1.43 (0.95–2.18)	0.088
Other UI	859	1.04 (0.64–1.73)	0.867
PUFA, median g/1000 Kcal			
UI	4255	1.28 (1.01–1.62) *	0.042
Stress UI	3345	1.47 (1.16–1.86) **	0.002
Urgency UI	2228	1.09 (0.84–1.43)	0.516
Mixed UI	1408	1.42 (1.04–1.94) *	0.029
Other UI	859	0.90 (0.62–1.32)	0.596

UI = urinary incontinence; SFA = saturated fat; MUFA = monounsaturated fat; PUFA = polyunsaturated fat.
OR = odds ratio; CI = confidence interval.
*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$.
The distributions of nutrients intakes were standardized with individual energy intake and log-transformed to improve the normality in the regression analyses.
All models controlled for age, gender, race/ethnicity, education level, body mass index, health insurance, family income-to-poverty ratio, diabetes, and hypertension.
The reference group in logistic regression was participants without UI.

3.3. UI in females

Due to the different causes and morbidities of UI in both sexes, the impact of fat intake on UI in males and females was analyzed. Results of multivariate analyses revealed that the association between UI and fat intake in adult males was not significant (Table S1). Thus, the focus was turned to UI among female participants in this analysis. Total fat intake was significantly and positively associated with SUI in female participants (OR 1.83 [95% CI 1.27–2.66]) (Table 3). The ORs for SFA, MUFA, and PUFA for SUI were 1.42 (95% CI 1.07–1.88), 1.50 (95% CI 1.10–2.06), and 1.47 (95% CI 1.16–1.86), respectively (Table 3). Further analysis of specific fatty acids revealed that female participants who consumed more SFA (4:0–18:0), MUFA (16:1–20:1), and n-6 PUFA (18:2, 18:3, and 20:4) were more likely to report SUI (Fig. 3).

3.4. Mediation effect of obesity on the relationship between fat intake and SUI in females

After adjusting for age, race/ethnicity, education level, BMI, health insurance, family income-to-poverty ratio, diabetes, and hypertension, the total effects of fat, SFA, MUFA, and PUFA on BMI were examined, with results summarized in Table 4. Based on previous studies investigating obesity and UI [21], a mediation analysis was performed to evaluate the mediating effect of BMI on the association between fat intake and SUI. The average mediation effects of total fat, SFA, MUFA, and PUFA on SUI were 0.0244 (95% CI 0.0152–0.03; $P < 0.001$), 0.0149 (95% CI 0.0067–0.02; $P < 0.001$), 0.0195 (95% CI 0.0114–0.03; $P < 0.001$), and 0.0132 (95% CI 0.0080–0.02; $P < 0.001$), respectively (Table 5, Fig. 4). The mediating effects of BMI, which accounted for the total effects of total fat, SFA, MUFA, and PUFA on SUI, were 14.7%, 13.0%, 18.7%, and 16.3%, respectively (Fig. 4). Therefore, the model in this study revealed a partial mediating effect of BMI on the effect of fat intake on SUI among females.

4. Discussion

This cross-sectional analysis of a community-based sample of citizens in the United States revealed that individuals with higher fat intake exhibited a higher risk for UI. Furthermore, a positive relationship was found between different fatty acid types (i.e., SFA, MUFA, and PUFA) and SUI among female participants. These results were significant when adjusted for age, race, education level, health insurance, family income-to-poverty ratio, BMI, diabetes, and hypertension in the multivariate models. Although the association between fat intake and UI was not significant in males, several SFA types (SFA 8:0, SFA10:0) were found to be positively associated with UUI in male participants in further regression analysis, which is consistent with the results of previous studies investigating LUTS in males [22–25]. Moreover, the mediation effect of BMI was analyzed, which indicated that obesity exhibited a partial mediating effect on the effect of fat intake on SUI in females.

In the past few decades, there has been an enormous change in the quality of dietary fat owing to the industrial revolution. A Western diet, with near n-3 PUFA deficiency and elevated levels of SFA, n-6 PUFA, and trans fatty acids, has gained a huge market share [26]. Inflammation plays a crucial role in the development of LUTS [27], whereas SFA, MUFA, n-3 PUFA, and n-6 PUFA play different roles in inflammatory processes [28]. n-6 PUFA- and SFA-rich diets are believed to increase neuroinflammation, whereas MUFA- and n-3 PUFA-rich diets are believed to have the opposite effect [29]. The exact mechanism of UI remains unclear, although some studies have reported that an abnormal sympathetic nervous system and inflammatory disorders may play roles in this process

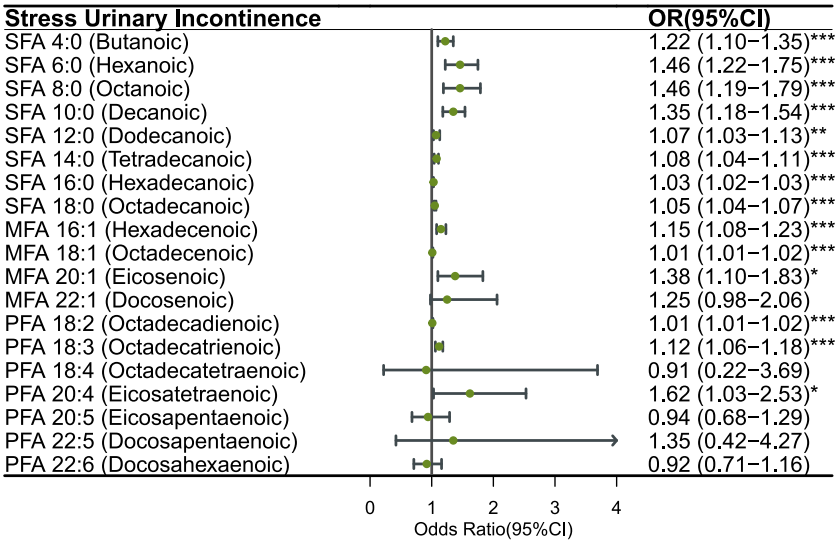

Fig. 3. Odds Ratios for Stress Urinary Incontinence across Fat Acids Groups among Women. SFA = saturated fat; MUFA = monounsaturated fat; PUFA = polyunsaturated fat. OR = odds ratio; CI = confidence interval. *$P < 0.05$. **$P < 0.01$. ***$P < 0.001$.

Table 4
Association of fat intake with BMI in women (n = 8075).

	OR (95% CI)	*P* value
Total Fat	1.97 (1.34–2.88) ***	<0.001
SFA	1.58 (1.18–2.13) **	0.002
MUFA	1.59 (1.15–2.21) **	0.004
PUFA	1.35 (1.06–1.73) *	0.016

SFA = saturated fat; MUFA = monounsaturated fat; PUFA = polyunsaturated fat.
OR = odds ratio; CI = confidence interval.
*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$.
The distributions of nutrients intakes were standardized with individual energy intake and log-transformed to improve the normality in the regression analyses.
All models controlled for age, race/ethnicity, education level, health insurance, family income-to-poverty ratio, diabetes, hypertension.
The reference group in logistic regression was female participants (BMI <25.0 kg/m^2).

Table 5
The results of mediation analysis among stress urinary incontinence in women in NHANES (2001–2008).

	Estimation	95% CI lower	95% CI upper	*P* value
FAT				
ACME	0.0244	0.0152	0.03	<0.001
ADE	0.1372	0.0688	0.23	<0.001
Total effect	0.1615	0.0962	0.25	<0.001
Prop.Mediated	0.1473	0.0920	0.30	<0.001
SFA				
ACME	0.0149	0.0067	0.02	<0.001
ADE	0.0982	0.0481	0.15	<0.001
Total effect	0.1130	0.0581	0.17	<0.001
Prop.Mediated	0.1299	0.0710	0.24	<0.001
MUFA				
ACME	0.0195	0.0114	0.03	<0.001
ADE	0.0871	0.0102	0.15	<0.001
Total effect	0.1066	0.0356	0.17	<0.001
Prop.Mediated	0.1874	0.0751	0.61	<0.001
PUFA				
ACME	0.0132	0.0080	0.02	<0.001
ADE	0.0677	0.0216	0.11	<0.001
Total effect	0.0809	0.0323	0.13	<0.001
Prop.Mediated	0.1634	0.0882	0.35	<0.001

SFA = saturated fat; MUFA = monounsaturated fat; PUFA = polyunsaturated fat. ACME, average causal mediation effect; ADE, average direct effect.

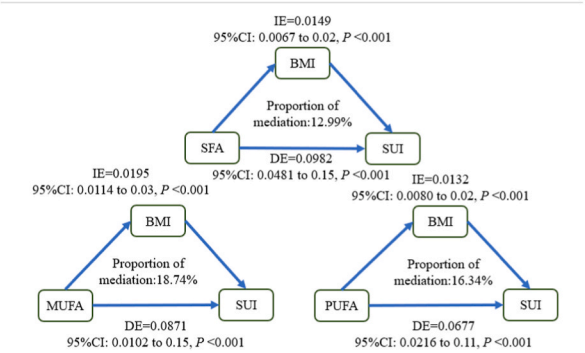

Fig. 4. Mediation Effect of BMI Between Fat Intake and Stress Urinary Incontinence in Women. SFA = saturated fat; MUFA = monounsaturated fat; PUFA = polyunsaturated fat. *$P < 0.05$. **$P < 0.01$. ***$P < 0.001$.

[30]. SFA, MUFA, and PUFA have been reported to regulate bladder function-related systematic inflammation and oxidative stress according to previous studies [31,32]. Thus, we analyzed individual SFA, MUFA, and PUFA to identify their potential connections with UI. Among the fatty acids included in this analysis, all SFA and 3 n-6 PUFA types were positively associated with SUI in female

participants, which is consistent with the proinflammatory effects of SFA and n-6 PUFA [33]. MUFA and n-3 PUFA, which are anti-inflammatory nutrients, did not exert a protective effect against UI in our study. In fact, MUFA intake increased the risk for SUI in female participants. A plausible explanation for the increased odds of UI is that high MUFA intake increases oxidative damage [34], which may theoretically increase the risk for UI [35]. To date, no study has investigated the relationship between UI and the intake of specific fatty acids, and the present study–at least, in part–fills this knowledge gap. Further studies are required to elucidate the biological mechanisms underlying the relationship between fat intake and UI subtype.

In addition to fat intake, we analyzed the intake of other nutrients, such as energy and protein. Participants with higher energy intake were more likely to report SUI and UUI (OR 2.85 [95% CI 2.12–3.84]; OR 1.84 [95% CI 1.38–2.46], respectively) in this analysis. For male participants, the OR for energy intake for UUI was 2.56 (95% CI 1.56–4.20). This result is consistent with previous analyses of LUTS in the BACH (2002–2005) and Health Professionals Follow-up Study [22,25]. Protein intake was inversely associated with UUI in females (OR 0.53 [95% CI 0.36–0.78]). However, Bauer et al. revealed that higher protein intake was associated with an increased risk for UUI in postmenopausal women [36].

Previous studies have demonstrated that obesity is associated with a high prevalence of UI, and fatty acid intake is associated with obesity [17,21,37]. However, our mediation analysis revealed that BMI explained only 14.7% of the mediating effect of total fat intake on SUI. This indicates the presence of other factors that could explain the effect of fatty acid intake on SUI, and these factors may be the subject of follow-up research.

Our study had unavoidable limitations due to its cross-sectional design. First, dietary nutrient data were obtained through interviews, which may have inevitably introduced information errors and recall bias. Many participants with abnormal energy intake were excluded to reduce bias. Second, we revealed the potential association between dietary fat intake and UI but could not confirm causality. Most participants were interviewed twice to collect data regarding their dietary nutrient intake. To some extent, dietary data may reflect long-term eating habits. Similarly, measurement errors in self-reported macronutrient intake can result in imprecise effect estimates; accordingly, we performed a subgroup analysis according to sex and UI subtype to reduce this error. The lower number of males with UI included in this study may have led to failure to find an association. Despite these limitations, this study provides high-quality observational data supporting the hypothesis that higher fat intake (especially SFA, MUFA, and n-6 PUFA) is associated with SUI.

5. Conclusions

In conclusion, the intake of total fat, SFA, MUFA, and n-6 PUFA were positively associated with UI—particularly SUI—in females. To some extent, BMI mediated the effect of fat intake on SUI, and the risk for SUI may be reduced by controlling body weight and total fat intake.

Ethics statement

The NCHS Ethics Review Board approved NHANES. (Approval No: NHANES 2001–2004 Protocol #98-12; NHANES 2005–2008 Protocol #2005-06).

Data availability statement

The data used in this study are available at https://www.cdc.gov/nchs/nhanes/. The dataset associated with this study was uploaded to the Figshare database (accession number: 10.6084/m9.figshare.24270856).

CRediT authorship contribution statement

Dajun Gao: Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Caoxu Zhang:** Writing – original draft, Methodology. **Qi chen:** Funding acquisition. **Zhi Cao:** Software. **Peizhang Li:** Writing – original draft. **Guangdong Zhou:** Conceptualization. **Huan Xu:** Writing – review & editing, Writing – original draft, Funding acquisition, Conceptualization. **Bin Xu:** Methodology. **Zhong Wang:** Funding acquisition.

Declaration of competing interest

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Abbreviations

UI	urinary incontinence
SFA	saturated fat

MUFA	monounsaturated fat
PUFA	polyunsaturated fat
AUASI	American Urological Association Symptom Index
LUTS	lower urinary tract symptoms
NHANES	National Health and Nutrition Examination Survey
SUI	stress urinary incontinence
UUI	urgency urinary incontinence
OR	odds ratio
CI	confidence interval
DE	Direct effect
IE	Indirect effect

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e28595.

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