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ORIGINAL RESEARCH

Correlation Between Metabolic Dysfunction-Associated Steatotic Liver Disease and the Risk of Urinary Incontinence in Adult Women

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Objective: This study examined the relationship between metabolic dysfunction-associated steatotic liver disease (MASLD) and urinary incontinence in adult women and evaluated the potential contribution of the fatty liver index (FLI) in this context.

Methods: The study utilized data from the National Health and Nutrition Examination Survey (NHANES) database, spanning from 2001 to 2018. The study included 17,221 adult female participants. Individuals exhibiting FLI values of 60 or greater were diagnosed with hepatic steatosis. Incontinence type and MASLD status were evaluated by analyzing questionnaire data and calculating the FLI. Logistic regression models were employed to examine the correlation between FLI, MASLD, and urinary incontinence, with potential confounding variables controlled through multivariate adjustment models. Furthermore, restricted cubic spline curve (RCS) modeling and subgroup analysis were employed to elucidate the relationship between variables further.

Results: The median age of participants in the MASLD group was higher than that of the non-MASLD group (53 vs 46 years, $P < 0.001$). The findings indicated a positive association between FLI and MASLD and the risk of urinary incontinence. Specifically, the risk of stress urinary incontinence (SUI), urgency urinary incontinence (UUI), and mixed urinary incontinence (MUI) all increased significantly with increasing quartiles of FLI (OR 2.44, 1.91, 2.30, respectively, $P < 0.001$). In the multivariate-adjusted model, SUI, UUI, and MUI risk was 76%, 50%, and 69% higher in patients with MASLD than those without MASLD. RCS analysis demonstrated a significant nonlinear positive correlation between FLI and the risk of SUI, UUI, and MUI, respectively.

Conclusion: This study's findings indicate a significant association between MASLD and the risk of developing urinary incontinence. Additionally, the results suggest that FLI and MASLD may act as independent risk factors for urinary incontinence.

Keywords: metabolic dysfunction-associated steatotic liver disease, urinary incontinence, fatty liver index, adult women, correlation study

Introduction

Metabolic dysfunction-associated steatosis liver disease (MASLD) is a chronic liver disease with a solid correlation to obesity, insulin resistance, and metabolic syndrome.[1] In recent years, with the prevalence of obesity and metabolic syndrome on a global scale, the incidence of MASLD has risen dramatically, becoming a significant challenge in international public health.[2,3] MASLD is characterized by the abnormal accumulation of fat in the liver and is often accompanied by systemic metabolic abnormalities, including insulin resistance, dyslipidemia, and systemic inflammation. These pathological processes significantly increase the risk of complications, including cardiovascular disease, diabetes, and chronic kidney disease.[1,4,5]

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Urinary incontinence is a prevalent urological disorder among adult women, significantly impacting their quality of life and social participation.[6,7] The pathogenesis of this condition is complex, involving multiple aspects of pelvic floor muscle function, nervous system regulation, and hormone levels.[8,9] In particular, the age-related decline in muscle mass, strength, and function is typical among older women. These physiological changes are frequently accompanied by sarcopenia, which significantly elevates the risk of urinary incontinence.[10,11] Although MASLD and urinary incontinence are often considered distinct entities in clinical practice, there is mounting evidence that they may be associated.[12] MASLD primarily affects the liver, yet its metabolic disorders and systemic inflammation may impact urinary function via many mechanisms, including neuroendocrine, vascular, or immune, which may elevate the risk of urinary disorders such as urinary incontinence. The relationship between MASLD and urinary incontinence is currently understudied, and the evidence needs to be more conclusive. It is, therefore, essential to explore the association between MASLD and urinary incontinence to gain insight into its pathogenesis and inform the development of effective prevention and treatment strategies.

This study aims to examine the relationship between MASLD and urinary incontinence in adult women, utilizing the National Health and Nutrition Examination Survey (NHANES), a vast and representative cross-sectional database. This study will address the following research questions: Does MASLD increase the risk of urinary incontinence in adult women? Furthermore, is there a differential association between MASLD and different types of urinary incontinence (stress urinary incontinence, urgency urinary incontinence, and mixed urinary incontinence)? What is the role of metabolic indicators (fatty liver index) in the association between MASLD and urinary incontinence?

This study contributes to understanding the relationship between MASLD and urinary incontinence. Furthermore, it may provide new ideas and intervention targets for clinical practice. By identifying MASLD as an independent risk factor for urinary incontinence, we can implement early screening and intervention strategies for high-risk populations. This will reduce the incidence of urinary incontinence and the risk of complications.

Materials and Methods

Study Population

The data utilized in this study were obtained from the NHANES database from 2001 to 2018. This database encompasses the findings of a cross-sectional survey conducted biennially by the Centers for Disease Control and Prevention (CDC). It is noteworthy that the study protocol for the NHANES database was approved by the National Center for Health Statistics (NCHS) Ethics Review Board, and all participants signed an informed consent form. By relevant NIH policies, the data in the NHANES database, which was not obtained through direct contact with participants, could be used directly for data analysis without further review by the institutional ethics committee. The Ethics Committee of the Changzhou Third People's Hospital has determined that no further ethical review approval is necessary for this study. The study used the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for report writing.

A total of 91,351 participants from nine consecutive cycles of the NHANES survey were included in this study. The exclusion criteria included the following: (1) participants under the age of 20, (2) male participants, (3) pregnant participants, (4) participants with missing data on urinary incontinence diagnosis, and (5) participants with missing data on demographic characteristics, missing data on chronic diseases, missing indicators required for calculation of fatty liver index (FLI), and missing some important biochemical indicators. Following a rigorous screening process, 17,221 eligible participants were ultimately included in the data analysis phase of this study (Figure 1).

MASLD Assessment

In the initial stages of MASLD, the disease is typically identified by an abnormal fat accumulation within the liver tissue. In the absence of data from ultrasound assessment of hepatic steatosis and transient elastography of the liver over multiple follow-up cycles, the evaluation of MASLD status is primarily based on the calculation of the FLI,[13] which is calculated using the following formula:

$$\mathrm{FLI} = \frac{\left(e^{0.953 \times \ln(\text{triglyceride}) + 0.139 \times \mathrm{BMI} + 0.718 \times \ln(\text{glutamyl transpeptidase}) + 0.053 \times \mathrm{WC} - 15.745}\right)}{\left(1 + e^{0.953 \times \ln(\text{triglyceride}) + 0.139 \times \mathrm{BMI} + 0.718 \times \ln(\text{glutamyl transpeptidase}) + 0.053 \times \mathrm{WC} - 15.745}\right)} \times 100$$

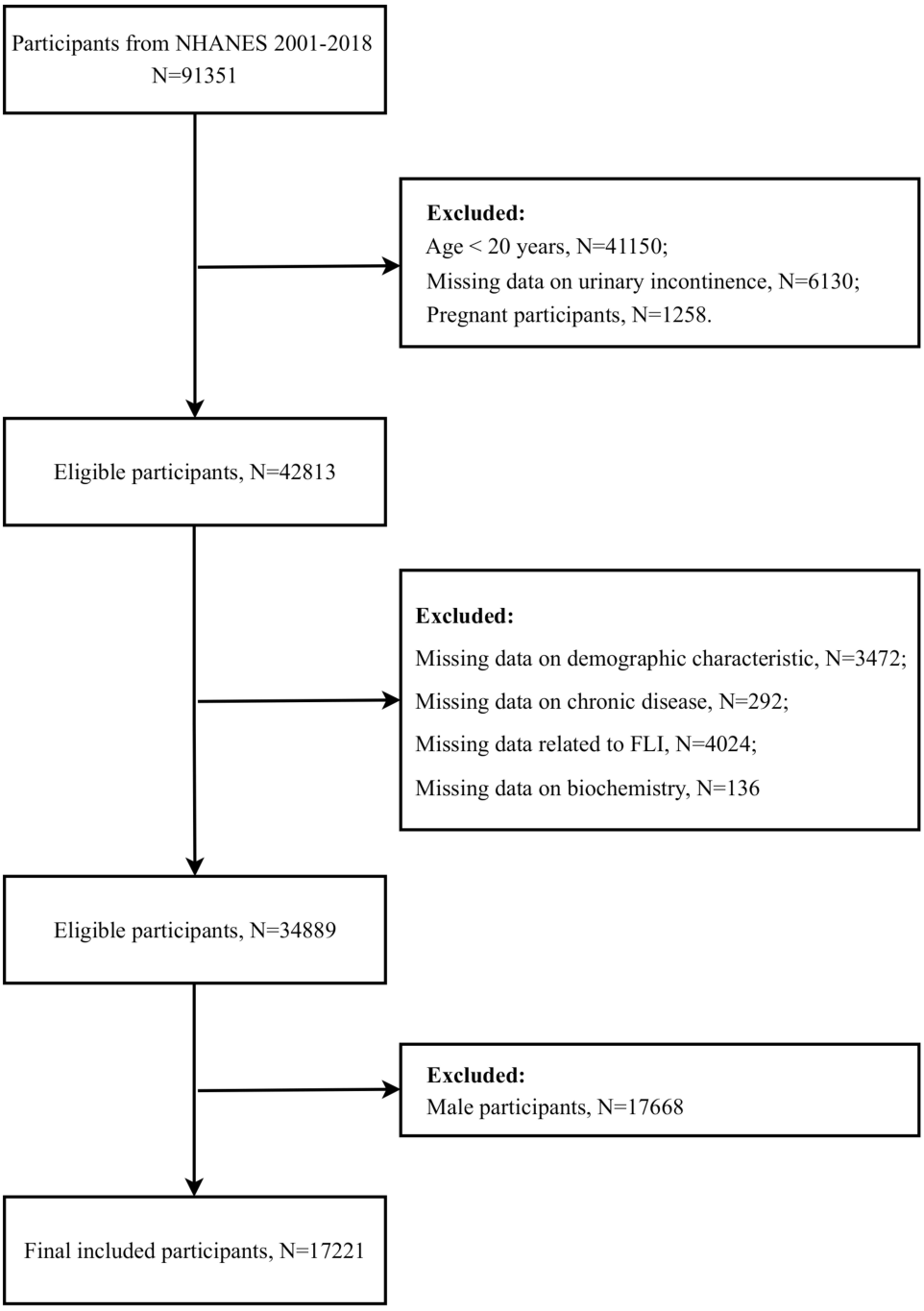

Figure 1 Participant screening flowchart.
Abbreviations: FLI, Fatty liver index.

Where BMI denotes body mass index, and WC stands for waist circumference. According to the findings of previous studies, individuals with FLI values below 60 are deemed to have a low risk of hepatic steatosis. In contrast, individuals with FLI values at or above 60 are considered to have a high risk of hepatic steatosis and are diagnosed with hepatic steatosis accordingly.[14] Furthermore, a diagnosis of MASLD is confirmed by meeting any of the following five cardiometabolic criteria:

1. A body mass index (BMI) of 25 kg/m² or greater or a waist circumference (WC) of 94 cm or greater for males and 80 cm or greater for females;

2. A fasting plasma glucose (FPG) level of 100 mg/dL or greater, or a 2-hour postprandial blood glucose level of 140 mg/dL or greater, or a glycosylated hemoglobin (HbA1c) level of 5.7% or greater, or a diagnosis of diabetes mellitus (DM) or receipt of glucose-lowering therapy for DM;
3. Blood pressure at or above 130/85 mmHg or antihypertensive medication;
4. Fasting plasma triglyceride (TG) levels of 150 mg/dL or greater, or lipid-lowering therapy;
5. Men with high-density lipoprotein cholesterol (HDL-c) levels below 40 mg/dL and women with HDL-c levels below 50 mg/dL or lipid-lowering therapy.[1]

Assessment of Urinary Incontinence

The history of urinary incontinence was evaluated by extracting pertinent data from the Kidney Conditions - Urology file under the heading of Questionnaire Data. In response to the question, "During the past 12 months, {have you/has SP} leaked or lost control of even a small amount of urine with an activity like coughing, lifting or exercise?" Individuals who responded affirmatively were classified as having a history of stress urinary incontinence (SUI), whereas those who responded negatively were classified as having no history of SUI. The question "During the past 12 months, {have you/has SP} leaked or lost control of even a small amount of urine with an urge or pressure to urinate and {you/he/she} could not get to the toilet fast enough?" was posed. Individuals who responded affirmatively were classified as having a history of urgency urinary incontinence (UUI), whereas those who responded negatively were classified as having no history of UUI. Mixed urinary incontinence (MUI) was defined as a combination of SUI and UUI.

Covariate Assessment

Multivariate adjustment models were constructed to examine the influence of confounding variables on the relationship between FLI, MASLD, and urinary incontinence. In this study, the covariates included a multitude of demographic characteristics, lifestyle habits, and chronic disease history, including age (in years), race, level of education, marital status, family income, alcohol consumption habits (yes/no), smoking status (yes/no), level of physical activity (vigorous/moderate/inactive), menopause status, and history of chronic diseases such as metabolic syndrome, DM, hypertension, coronary artery disease, stroke, and cancer.

Regarding race categorization, the participants were subdivided into four major groups: Mexican American, non-Hispanic White, non-Hispanic Black, and Other Race. Regarding educational attainment, participants were categorized into one of three groups based on their level of education: less than 9th grade, 9th-12th grade, and more than 12th grade. Marital status was dichotomized into two categories: cohabitation and solitude. Family income was classified according to the Poverty-to-Income Ratio (PIR) criterion, as defined in the US government report, into the following categories: low (PIR $\leq$ 1.3), medium (1.3 < PIR $\leq$ 3.5), and high (PIR > 3.5). About lifestyle habits, smoking status was determined based on whether the participant had smoked at least 100 cigarettes in their lifetime and whether they were currently still smoking. In contrast, alcohol consumption was defined as the consumption of at least 12 alcoholic beverages of any type in any given year. The physical activity assessment was based on two criteria: the engagement in vigorous exercise that resulted in a significant increase in respiration or heart rate, and the engagement in moderate-intensity exercise that resulted in a slight increase in respiration or heart rate. The status of menopause was determined by administering a reproductive health questionnaire. An individual is classified as postmenopausal if they respond in the negative to the question "Have you had at least one menstrual period in the past 12 months?" and then select "hysterectomy" or "menopause" as the reason for their amenorrhea. The diagnostic criteria set forth by the National Cholesterol Education Program Adult Treatment Program III were employed to define metabolic syndrome. Subjects who met three or more of the following criteria were classified as having metabolic syndrome: (1) TG $\geq$ 150 mg/dL; (2) HDL-C < 40 mg/dL in men and < 50 mg/dL in women; (3) FPG level $\geq$ 110 mg/dL; (4) WC > 102 cm in men and > 88 cm in women; and (5) a systolic blood pressure of greater than 130 mmHg and/or a diastolic blood pressure of greater than 85 mmHg. About the medical history variables, the history of diabetes was based on whether the participant had been diagnosed with diabetes by a physician, had a FPG level of 126 mg/dL or greater, or a HbA1c level of 6.5% or greater, and was using diabetes medication or insulin. A history of hypertension was established based on whether the participant had been diagnosed with hypertension by a physician or was currently taking medication prescribed for hypertension. A history of

coronary heart disease was identified through reliance on the participant's self-report of a diagnosis of coronary heart disease, angina, or a heart attack by a physician. Similarly, a history of stroke was determined based on whether the participant had been diagnosed with these conditions by a physician. The scope of cancer included in this study covered any type of cancer or malignancy diagnosed by a healthcare professional.

Statistical Analysis

The Kolmogorov–Smirnov test was employed to ascertain whether the continuous variables exhibited a normal distribution. For variables that exhibited a normal distribution, we expressed them in the form of mean ± standard deviation. Conversely, we expressed variables that did not conform to a normal distribution using the median (and the 25th and 75th percentiles). To compare the differences between these variables, we employed either a one-way ANOVA or a Kruskal–Wallis test, depending on the distributional characteristics of the data. Frequencies and percentages were calculated for categorical variables, and the chi-square test was employed to assess differences between groups.

To investigate the relationships between FLI, quartiles of FLI, MASLD, and urinary incontinence in greater depth, we constructed logistic regression models. We calculated the ratio of ratios (OR) and their 95% confidence intervals (CI). Three multivariable-adjusted models were built to assess these relationships more precisely and address potential confounding variables. Of these, Model 1 was unadjusted; Model 2 built on Model 1 by incorporating age and race as adjusting factors; and Model 3 further built on Model 2 by expanding the adjustment for the variables of educational level, marital status, family PIR, smoking, alcohol consumption, physical activity, DM, hypertension, coronary heart disease, stroke, and cancer. Furthermore, a restricted cubic spline curve (RCS) model was employed to investigate the potential dose-response relationship between FLI and urinary incontinence. To gain further insight into the relationship between MASLD and urinary incontinence risk in different subgroups, we conducted a stratification analysis based on the variables of age, race, education, marital status, family PIR, smoking, alcohol consumption, physical activity, menopause status, metabolic syndrome, diabetes, and hypertension, and performed interaction analyses.

All statistical analyses employed a two-sided test, and a P-value of less than 0.05 was used to determine statistical significance. All statistical analyses were conducted using R 4.4.0 (provided by the R Foundation at http://www.R-project.org) and SPSS version 23.0 (provided by IBM, Armonk, NY, USA). The graphs were plotted using GraphPad Prism version 9.0 (provided by GraphPad Software, Inc., USA).

Results

Baseline Characteristics of Participants Based on FLI Quartiles

In this study, participants were classified into four groups based on their FLI quartiles, and the baseline characteristics of participants in each group were compared. The results demonstrated a statistically significant correlation between increasing FLI quartiles and median age (39 years in Quartile 1 to 52 years in Quartile 4, $P < 0.001$). The racial distribution demonstrated a notable decline in the proportion of non-Hispanic white individuals with increasing FLI. In contrast, the proportion of non-Hispanic black individuals and Mexican Americans exhibited an upward trend. The proportion of individuals with low educational attainment (less than 9th grade) increased significantly with increasing FLI, from 4.85% in Quartile 1 to 9.89% in Quartile 4 ($P < 0.001$). The proportion of individuals living alone, families with low PIR, and those who smoke and drink increased with higher FLI. The proportion of postmenopausal women in the group with a higher FLI was higher. Furthermore, the prevalence of DM, hypertension, metabolic syndrome, coronary heart disease, stroke, and cancer demonstrated a significant correlation with elevated FLI. Furthermore, there were significant changes in BMI, WC, FPG, HbA1c, total cholesterol (TC), TG, alanine aminotransferase (AST), glutamate aminotransferase (ALT), glutamyl transpeptidase (GGT), and total bilirubin with increasing quartiles of FLI ($P < 0.001$). Notably, significant associations were observed between FLI quartiles and the three types of urinary incontinence (SUI, UUI, and MUI). As the FLI quartiles increased, the percentage of participants who reported SUI rose from 29.25% to 52.48%, UUI from 18.47% to 38.50%, and MUI from 9.76% to 25.59%. These findings demonstrate a positive correlation between FLI quartiles and the prevalence of SUI, UUI, and MUI ($P < 0.001$) (Table 1). This suggests that FLI is associated with metabolic diseases and urinary health problems.

Table 1 Baseline Characteristics of Participants Based on FLI Quartiles

Variables	FLI				*P*
	Quartile 1 (n = 4305)	Quartile 2 (n = 4305)	Quartile 3 (n = 4305)	Quartile 4 (n = 4306)	
Age (years)	39.00 (28.00,53.00)	51.00 (36.00,66.00)	55.00 (41.00,67.00)	52.00 (40.00,63.00)	<0.001
Race, n (%)					<0.001
Mexican American	461 (10.71)	655 (15.21)	884 (20.53)	786 (18.25)	
Non-Hispanic White	2288 (53.15)	2074 (48.18)	1826 (42.42)	1867 (43.36)	
Non-Hispanic Black	630 (14.63)	819 (19.02)	908 (21.09)	1115 (25.89)	
Other Race	926 (21.51)	757 (17.58)	687 (15.96)	538 (12.49)	
Education Level, n (%)					<0.001
Less than 9th grade	209 (4.85)	392 (9.11)	561 (13.03)	426 (9.89)	
9–12th grade	1222 (28.39)	1538 (35.73)	1682 (39.07)	1759 (40.85)	
More than 12th grade	2874 (66.76)	2375 (55.17)	2062 (47.90)	2121 (49.26)	
Marital Status, n (%)					<0.001
Cohabitation	2438 (56.63)	2415 (56.10)	2327 (54.05)	2269 (52.69)	
Solitude	1867 (43.37)	1890 (43.90)	1978 (45.95)	2037 (47.31)	
Family PIR, n (%)					<0.001
Low (≤1.3)	1073 (24.92)	1218 (28.29)	1429 (33.19)	1661 (38.57)	
Medium (1.3–3.5)	1543 (35.84)	1695 (39.37)	1713 (39.79)	1631 (37.88)	
High (>3.5)	1689 (39.23)	1392 (32.33)	1163 (27.02)	1014 (23.55)	
Smoke, n (%)					<0.001
Yes	1486 (34.52)	1560 (36.24)	1625 (37.75)	1877 (43.59)	
No	2819 (65.48)	2745 (63.76)	2680 (62.25)	2429 (56.41)	
Alcohol, n (%)					<0.001
Yes	2885 (67.02)	2557 (59.40)	2365 (54.94)	2308 (53.60)	
No	1420 (32.98)	1748 (40.60)	1940 (45.06)	1998 (46.40)	
Physical Activity, n (%)					<0.001
Inactive	948 (22.02)	1267 (29.43)	1544 (35.87)	1624 (37.71)	
Moderate	1659 (38.54)	1812 (42.09)	1839 (42.72)	1796 (41.71)	
Vigorous	1698 (39.44)	1226 (28.48)	922 (21.42)	886 (20.58)	
Menopausal, n (%)					<0.001
Yes	1319 (30.64)	2282 (53.01)	2629 (61.07)	2484 (57.69)	
No	2986 (69.36)	2023 (46.99)	1676 (38.93)	1822 (42.31)	
Diabetes mellitus, n (%)					<0.001
Yes	153 (3.55)	435 (10.10)	828 (19.23)	1442 (33.49)	
No	4152 (96.45)	3870 (89.90)	3477 (80.77)	2864 (66.51)	
Hypertension, n (%)					<0.001
Yes	636 (14.77)	1382 (32.10)	1862 (43.25)	2334 (54.20)	
No	3669 (85.23)	2923 (67.90)	2443 (56.75)	1972 (45.80)	
Metabolic syndrome, n (%)					<0.001
Yes	42 (0.98)	694 (16.12)	1889 (43.88)	2809 (65.23)	
No	4263 (99.02)	3611 (83.88)	2416 (56.12)	1497 (34.77)	
Coronary heart disease, n (%)					<0.001
Yes	52 (1.21)	112 (2.60)	130 (3.02)	164 (3.81)	
No	4253 (98.79)	4193 (97.40)	4175 (96.98)	4142 (96.19)	
Stroke, n (%)					<0.001
Yes	85 (1.97)	138 (3.21)	178 (4.13)	214 (4.97)	
No	4220 (98.03)	4167 (96.79)	4127 (95.87)	4092 (95.03)	
Cancer, n (%)					<0.001
Yes	334 (7.76)	433 (10.06)	474 (11.01)	474 (11.01)	
No	3971 (92.24)	3872 (89.94)	3831 (88.99)	3832 (88.99)	

(*Continued*)

Table 1 (Continued).

Variables	FLI				*P*
	Quartile 1 (n = 4305)	Quartile 2 (n = 4305)	Quartile 3 (n = 4305)	Quartile 4 (n = 4306)	
BMI (kg/m^2)	22.00 (20.27,23.70)	26.22 (24.48,28.10)	30.46 (28.30,32.68)	37.63 (34.20,42.01)	<0.001
WC (cm)	78.70 (74.30,83.40)	90.70 (86.70,94.90)	100.90 (96.20,105.60)	115.90 (109.20,124.50)	<0.001
FPG (mg/dL)	86.00 (80.00,92.00)	90.00 (84.00,98.00)	93.00 (86.00,104.00)	98.00 (89.00,118.00)	<0.001
HbA1c (%)	5.20 (5.00,5.50)	5.40 (5.20,5.70)	5.60 (5.30,5.90)	5.80 (5.40,6.30)	<0.001
TC (mg/dL)	184.00 (162.00,209.00)	198.00 (171.00,226.00)	200.00 (174.00,229.00)	198.00 (171.00,227.00)	<0.001
TG (mg/dL)	71.00 (54.00,95.00)	105.00 (77.00,146.00)	135.00 (96.00,190.00)	161.00 (112.25,232.00)	<0.001
HDL-c (mg/dL)	65.00 (55.00,76.00)	58.00 (49.00,69.00)	53.00 (44.00,62.00)	47.00 (40.00,56.00)	<0.001
AST (U/L)	21.00 (18.00,24.00)	21.00 (18.00,25.00)	22.00 (19.00,26.00)	22.00 (18.00,27.00)	<0.001
ALT (U/L)	16.00 (13.00,20.00)	17.00 (14.00,22.00)	19.00 (15.00,25.00)	21.00 (17.00,28.00)	<0.001
GGT (IU/L)	13.00 (10.00,17.00)	16.00 (12.00,21.00)	19.00 (15.00,28.00)	24.00 (18.00,39.00)	<0.001
Total Bilirubin (mg/dL)	0.70 (0.50,0.90)	0.60 (0.50,0.80)	0.60 (0.40,0.80)	0.50 (0.40,0.70)	<0.001
SUI, n (%)					<0.001
Yes	1259 (29.25)	1693 (39.33)	1995 (46.34)	2260 (52.48)	
No	3046 (70.75)	2612 (60.67)	2310 (53.66)	2046 (47.52)	
UUI, n (%)					<0.001
Yes	795 (18.47)	1195 (27.76)	1389 (32.26)	1658 (38.50)	
No	3510 (81.53)	3110 (72.24)	2916 (67.74)	2648 (61.50)	
MUI, n (%)					<0.001
Yes	420 (9.76)	692 (16.07)	871 (20.23)	1102 (25.59)	
No	3885 (90.24)	3613 (83.93)	3434 (79.77)	3204 (74.41)	

Notes: Data are shown as median (25th, 75th percentiles) or percentages, p <0.05 considered statistically significant.

Abbreviations: FLI, Fatty liver index; PIR, Poverty-to-income ratio; BMI, Body mass index; WC, Waist circumference; FPG, Fasting plasma-glucose; HbA1c, Hemoglobin A1c; TC, Total cholesterol; TG, Triglyceride; HDL-c, High density lipoprotein cholesterol; AST, Aspartate aminotransferase; ALT, Alanine transaminase; GGT, Gamma-glutamyl transferase; SUI, Stress urinary incontinence; UUI, Urgency urinary incontinence; MUI, Mixed urinary incontinence.

Comparison of Baseline Characteristics Between MASLD and Non-MASLD Participants

The results demonstrated that participants in the MASLD group exhibited a higher median age (53 vs 46 years, P < 0.001) and notable discrepancies in racial distribution, educational level, marital status, and family PIR when compared to the non-MASLD group (P < 0.001). The MASLD group displayed a higher prevalence of smokers and a lower level of physical activity, with a deficient proportion of individuals engaging in moderate and vigorous physical activity (P < 0.001). The MASLD group exhibited a higher proportion of women in postmenopausal (P < 0.001). About metabolic indicators, the MASLD group exhibited significantly elevated BMI, WC, FPG, HbA1c, and TG. At the same time, HDL-c was markedly reduced, indicating the presence of more severe metabolic disorders in the MASLD group. Furthermore, the prevalence of DM, hypertension, metabolic syndrome, coronary heart disease, stroke, and cancer was significantly higher in the MASLD group than in the non-MASLD group (P < 0.001). Additionally, a significant correlation was observed between MASLD and the type of urinary incontinence. Specifically, the percentage of participants reporting SUI was 50.66% in the MASLD group, which was higher than the 35.36% observed in the non-MASLD group. Similarly, the percentage of participants reporting UUI was 36.53%, which was higher than the 23.88% observed in the non-MASLD group. Additionally, the percentage of participants reporting MUI was 24.01%, higher than the 13.42% observed in the non-MASLD group (Table 2). These findings reinforce the association between MASLD and urinary incontinence, indicating that metabolic dysfunction may harm urinary health.

Correlation Analysis of FLI, MASLD, and Urinary Incontinence

A multi-model analysis of the relationship between FLI, MASLD, and different types of urinary incontinence (SUI, UUI, and MUI) revealed a positive association between FLI and the risk of incontinence. The coefficient of FLI, when used as a continuous variable, was significantly positive in all three models (OR=1.01, 95% CI: 1.01–1.01, P < 0.001), indicating that each unit increase in FLI was associated with a significant increase in the risk of urinary incontinence. In the quartile

Table 2 Baseline Characteristics of Participants with and without MASLD

Variables	Total (n = 17221)	NON-MASLD (n = 9915)	MASLD (n = 7306)	*P*
Age (years)	49.00 (35.00, 64.00)	46.00 (32.00, 63.00)	53.00 (40.00, 64.00)	<0.001
Race, n (%)				<0.001
Mexican American	2786 (16.18)	1368 (13.80)	1418 (19.41)	
Non-Hispanic White	8055 (46.77)	4924 (49.66)	3131 (42.86)	
Non-Hispanic Black	3472 (20.16)	1730 (17.45)	1742 (23.84)	
Other Race	2908 (16.89)	1893 (19.09)	1015 (13.89)	
Education Level, n (%)				<0.001
Less than 9th grade	1588 (9.22)	768 (7.75)	820 (11.22)	
9–12th grade	6201 (36.01)	3253 (32.81)	2948 (40.35)	
More than 12th grade	9432 (54.77)	5894 (59.45)	3538 (48.43)	
Marital Status, n (%)				<0.001
Cohabitation	9449 (54.87)	5576 (56.24)	3873 (53.01)	
Solitude	7772 (45.13)	4339 (43.76)	3433 (46.99)	
Family PIR, n (%)				<0.001
Low (≤1.3)	5381 (31.25)	2712 (27.35)	2669 (36.53)	
Medium (1.3–3.5)	6582 (38.22)	3754 (37.86)	2828 (38.71)	
High (>3.5)	5258 (30.53)	3449 (34.79)	1809 (24.76)	
Smoke, n (%)				<0.001
Yes	6548 (38.02)	3538 (35.68)	3010 (41.20)	
No	10673 (61.98)	6377 (64.32)	4296 (58.80)	
Alcohol, n (%)				<0.001
Yes	10115 (58.74)	6180 (62.33)	3935 (53.86)	
No	7106 (41.26)	3735 (37.67)	3371 (46.14)	
Physical Activity, n (%)				<0.001
Inactive	5383 (31.26)	2670 (26.93)	2713 (37.13)	
Moderate	7106 (41.26)	4041 (40.76)	3065 (41.95)	
Vigorous	4732 (27.48)	3204 (32.31)	1528 (20.91)	
Menopausal, n (%)				<0.001
Yes	8714 (50.60)	4374 (44.11)	4340 (59.40)	
No	8507 (49.40)	5541 (55.89)	2966 (40.60)	
Diabetes mellitus, n (%)				<0.001
Yes	2858 (16.60)	795 (8.02)	2063 (28.24)	
No	14363 (83.40)	9120 (91.98)	5243 (71.76)	
Hypertension, n (%)				<0.001
Yes	6214 (36.08)	2523 (25.45)	3691 (50.52)	
No	11007 (63.92)	7392 (74.55)	3615 (49.48)	
Metabolic syndrome, n (%)				<0.001
Yes	5434 (31.55)	1173 (11.83)	4261 (58.32)	
No	11787 (68.45)	8742 (88.17)	3045 (41.68)	
Coronary heart disease, n (%)				<0.001
Yes	458 (2.66)	198 (2.00)	260 (3.56)	
No	16763 (97.34)	9717 (98.00)	7046 (96.44)	
Stroke, n (%)				<0.001
Yes	615 (3.57)	271 (2.73)	344 (4.71)	
No	16606 (96.43)	9644 (97.27)	6962 (95.29)	
Cancer, n (%)				<0.001
Yes	1715 (9.96)	918 (9.26)	797 (10.91)	
No	15506 (90.04)	8997 (90.74)	6509 (89.09)	

(*Continued*)

Table 2 (Continued).

Variables	Total (n = 17221)	NON-MASLD (n = 9915)	MASLD (n = 7306)	*P*
BMI (kg/m^2)	28.15 (23.98, 33.41)	24.69 (22.10, 27.34)	34.40 (31.12, 38.83)	<0.001
WC (cm)	95.50 (84.90, 107.20)	86.70 (79.70, 93.40)	109.35 (102.70, 118.50)	<0.001
FPG (mg/dL)	91.00 (84.00, 101.00)	88.00 (82.00, 96.00)	96.00 (88.00, 111.00)	<0.001
HbA1c (%)	5.50 (5.20, 5.80)	5.30 (5.10, 5.60)	5.70 (5.40, 6.20)	<0.001
TC (mg/dL)	195.00 (169.00, 223.00)	192.00 (167.00, 220.00)	199.00 (173.00, 227.00)	<0.001
TG (mg/dL)	111.00 (75.00, 167.00)	90.00 (65.00, 128.00)	152.00 (106.00, 219.00)	<0.001
HDL-c (mg/dL)	55.00 (46.00, 67.00)	61.00 (51.00, 72.00)	49.00 (42.00, 58.00)	<0.001
AST (U/L)	21.00 (18.00, 25.00)	21.00 (18.00, 25.00)	22.00 (18.00, 26.00)	<0.001
ALT (U/L)	18.00 (15.00, 23.00)	17.00 (14.00, 21.00)	20.00 (16.00, 27.00)	<0.001
GGT (IU/L)	17.00 (13.00, 25.00)	15.00 (11.00, 20.00)	22.00 (16.00, 35.00)	<0.001
Total Bilirubin (mg/dL)	0.60 (0.50, 0.80)	0.60 (0.50, 0.90)	0.60 (0.40, 0.70)	<0.001
SUI, n (%)				<0.001
Yes	7207 (41.85)	3506 (35.36)	3701 (50.66)	
No	10014 (58.15)	6409 (64.64)	3605 (49.34)	
UUI, n (%)				<0.001
Yes	5037 (29.25)	2368 (23.88)	2669 (36.53)	
No	12184 (70.75)	7547 (76.12)	4637 (63.47)	
MUI, n (%)				<0.001
Yes	3085 (17.91)	1331 (13.42)	1754 (24.01)	
No	14136 (82.09)	8584 (86.58)	5552 (75.99)	

Notes: Data are shown as median (25th, 75th percentiles) or percentages, p <0.05 considered statistically significant.
Abbreviations: MASLD, Metabolic dysfunction-associated steatotic liver disease; PIR, Poverty-to-income ratio; BMI, Body mass index; WC, Waist circumference; FPG, Fasting plasma-glucose; HbA1c, Hemoglobin A1c; TC, Total cholesterol; TG, Triglyceride; HDL-c, High density lipoprotein cholesterol; AST, Aspartate aminotransferase; ALT, Alanine transaminase; GGT, Gamma-glutamyl transferase; SUI, Stress urinary incontinence; UUI, Urgency urinary incontinence; MUI, Mixed urinary incontinence.

analysis of FLI, a significant increase in the risk of SUI, UUI, and MUI was observed with increasing quartiles of FLI. To illustrate, the risk of SUI increased from the reference value of quartile 1 to 2.67 in quartile 4 (Model 1), and this risk decreased slightly to 2.44 in the adjusted Model 3, though it remained significant. Similarly, both UUI and MUI demonstrated a comparable trend, exhibiting a notable elevation in risk with rising FLI quartiles. Furthermore, the presence of MASLD was observed to elevate the risk of urinary incontinence significantly. In all models, the MASLD group exhibited a significantly elevated OR for urinary incontinence compared to the non-MASLD group. Specifically, the OR value for SUI decreased from 1.88 in Model 1 to 1.76 in Model 3, the OR value for UUI decreased from 1.83 to 1.50, and the OR value for MUI decreased from 2.04 to 1.69. However, the P values for these values were less than 0.001 in all models (Table 3). In conclusion, FLI and MASLD may be considered independent risk factors for urinary incontinence, and this association remained significant even after adjusting for multiple potential confounding factors.

RCS Analysis

In this study, we employed an RCS analysis to assess the association between FLI and the three types of urinary incontinence (SUI, UUI, and MUI). The results demonstrated a statistically significant correlation between FLI and the risk of SUI, UUI, and MUI after adjusting for age, race, education, marital status, family PIR, smoking, alcohol consumption, physical activity, DM, hypertension, coronary heart disease, stroke, and cancer (P for overall < 0.001). Furthermore, all these variables exhibited a significant nonlinear relationship (P for nonlinear < 0.001). These findings indicate a notable nonlinear correlation between FLI and urinary incontinence. Figure 2 illustrates a noteworthy increase in SUI, UUI, and MUI risk with elevated FLI levels.

Table 3 Relationship Between FLI, MASLD, and Urinary Incontinence in Different Models

Variables	Model 1		Model 2		Model 3	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Stress urinary incontinence						
FLI	1.01 (1.01 ~ 1.01)	<0.001	1.01 (1.01 ~ 1.01)	<0.001	1.01 (1.01 ~ 1.01)	<0.001
Categories						
Quartile 1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Quartile 2	1.57 (1.43 ~ 1.72)	<0.001	1.41 (1.28 ~ 1.55)	<0.001	1.40 (1.27 ~ 1.54)	<0.001
Quartile 3	2.09 (1.91 ~ 2.28)	<0.001	1.85 (1.69 ~ 2.03)	<0.001	1.82 (1.66 ~ 2.00)	<0.001
Quartile 4	2.67 (2.45 ~ 2.92)	<0.001	2.56 (2.33 ~ 2.81)	<0.001	2.44 (2.21 ~ 2.69)	<0.001
MASLD						
No	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Yes	1.88 (1.76 ~ 2.00)	<0.001	1.84 (1.73 ~ 1.96)	<0.001	1.76 (1.65 ~ 1.89)	<0.001
Urgency urinary incontinence						
FLI	1.01 (1.01 ~ 1.01)	<0.001	1.01 (1.01 ~ 1.01)	<0.001	1.01 (1.01 ~ 1.01)	<0.001
Categories						
Quartile 1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Quartile 2	1.70 (1.53 ~ 1.88)	<0.001	1.27 (1.14 ~ 1.41)	<0.001	1.25 (1.12 ~ 1.39)	<0.001
Quartile 3	2.10 (1.90 ~ 2.32)	<0.001	1.47 (1.32 ~ 1.64)	<0.001	1.40 (1.25 ~ 1.56)	<0.001
Quartile 4	2.76 (2.51 ~ 3.05)	<0.001	2.13 (1.92 ~ 2.36)	<0.001	1.91 (1.71 ~ 2.13)	<0.001
MASLD						
No	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Yes	1.83 (1.72 ~ 1.96)	<0.001	1.63 (1.52 ~ 1.75)	<0.001	1.50 (1.39 ~ 1.61)	<0.001
Mixed urinary incontinence						
FLI	1.01 (1.01 ~ 1.01)	<0.001	1.01 (1.01 ~ 1.01)	<0.001	1.01 (1.01 ~ 1.01)	<0.001
Categories						
Quartile 1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Quartile 2	1.77 (1.56 ~ 2.02)	<0.001	1.41 (1.24 ~ 1.61)	<0.001	1.38 (1.21 ~ 1.58)	<0.001
Quartile 3	2.35 (2.07 ~ 2.66)	<0.001	1.79 (1.57 ~ 2.04)	<0.001	1.66 (1.45 ~ 1.90)	<0.001
Quartile 4	3.18 (2.82 ~ 3.59)	<0.001	2.68 (2.36 ~ 3.04)	<0.001	2.30 (2.01 ~ 2.62)	<0.001
MASLD						
No	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
Yes	2.04 (1.88 ~ 2.20)	<0.001	1.90 (1.75 ~ 2.06)	<0.001	1.69 (1.55 ~ 1.84)	<0.001

Notes: Model 1: crude. Model 2: adjusted for Age, Race. Model 3: adjusted for Age, Race, Education Level, Marital Status, Family PIR, Smoke, Alcohol, Physical Activity, Diabetes mellitus, Hypertension, Coronary heart disease, Stroke, Cancer.
Abbreviations: FLI, Fatty liver index; MASLD, Metabolic dysfunction-associated steatotic liver disease; PIR, Poverty-to-income ratio; OR, Odds ratio; CI, Confidence interval.

Subgroup Analysis of the Relationship Between MASLD and Urinary Incontinence

A subgroup analysis was conducted to investigate the influence of diverse demographic and clinical characteristics on the strength of the association between MASLD and urinary incontinence. The results demonstrated that MASLD was associated with a markedly elevated risk of diverse forms of urinary incontinence across all patients (OR = 1.76 for SUI, OR = 1.50 for UUI, OR = 1.69 for MUI, all P < 0.001), after adjusting for a multitude of confounding variables, including demographic characteristics, lifestyle habits, and multiple chronic diseases. Subgroup analyses demonstrated that the risk of SUI, UUI, and MUI was significantly elevated in patients with MASLD compared to patients without MASLD across all ages, races, education levels, marital status, family PIR, smoking habits, drinking habits, physical activity level, menopausal status, DM, hypertension, and metabolic syndrome status subgroups (all P values < 0.01). Notably, the subgroup analyses of age, education level, marital status, and menopausal status revealed a significant interaction between MASLD and the risk of SUI (interaction P value < 0.05). Specifically, MASLD had a particularly

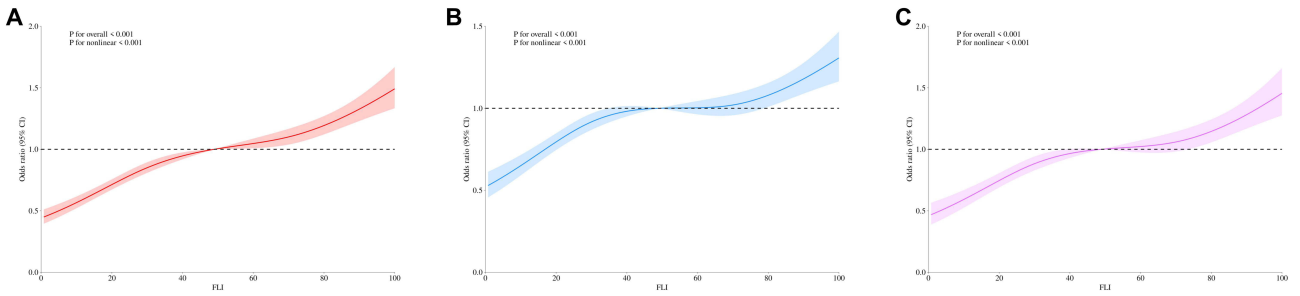

Figure 2 Non-linear relationship between FLI and SUI (**A**), UUI (**B**), and MUI (**C**). The solid line displays the odds ratio, with the 95% CI represented by shading. They were adjusted for age, race, education level, marital status, family PIR, smoke, alcohol, physical activity, diabetes mellitus, hypertension, coronary heart disease, stroke, and cancer.
Abbreviations: FLI, Fatty liver index; CI, Confidence interval; PIR, Poverty-to-income ratio; SUI, Stress urinary incontinence; UUI, Urgency urinary incontinence; MUI, Mixed urinary incontinence.

pronounced impact on SUI risk in the age 20–39, high education level, solitude, and premenopause subgroups (Figure 3). In contrast, the association between MASLD and UUI risk remained consistent across subgroups, with no significant interaction observed (interaction P value > 0.05) (Figure 4). Regarding MUI risk, the associations between MASLD and all other subgroups, except the age and menopausal status subgroups, remained consistent, with no significant interactions observed (interaction P value > 0.05). Similarly, the impact of MASLD on MUI risk was more pronounced in the 20–39 age and premenopause groups (Figure 5).

Discussion

This study investigated the potential correlation between MASLD and urinary incontinence, utilizing a large sample of adult women from the NHANES database. The findings indicated that both FLI and MASLD were independent risk factors for urinary incontinence. The risk of SUI, UUI, and MUI all increased significantly with increasing quartiles of FLI, and this relationship remained significant even after adjustment for demographic characteristics, lifestyle habits, and multiple chronic diseases. Similarly, the risk of SUI, UUI, and MUI was significantly higher in MASLD patients than in non-MASLD patients, and this relationship remained robust after adjustment for multiple covariates. This indicates that FLI and MASLD are not merely markers of metabolic disorders; they may also be strongly associated with the development of urinary incontinence.

MASLD is characterized by a constellation of metabolic abnormalities, exerting a complex influence on the pathogenesis of urinary incontinence. In this study, we observed that the risk of urinary incontinence was markedly elevated in patients with MASLD compared to non-MASLD patients, particularly after adjusting for a range of confounding variables. This association remained statistically significant. This indicates that MASLD is not merely a liver disease but has a broader impact on systemic metabolism and hormone levels, affecting urinary health. While this study was not designed to directly elucidate the precise mechanisms through which MASLD leads to urinary incontinence, integrating existing literature and an understanding of physiopathology allows for formulating several potential hypotheses.

Patients with MASLD are frequently associated with a range of metabolic disorders, including insulin resistance, glucose metabolism disorders, and dyslipidemia.[15] Such metabolic irregularities may exert a direct influence on the urinary system. Insulin resistance affects systemic glucose metabolism and may result in bladder detrusor dysfunction by influencing the metabolism and function of bladder smooth muscle, increasing the risk of urinary incontinence.[16] Insulin resistance may reduce neurotrophic factors, which can impair the health and functionality of nerve cells. This, in turn, can affect the normal contraction and relaxation of the bladder.[17] Furthermore, hyperglycemia may affect bladder nerve conduction and muscle contraction, promoting incontinence.[16,18] Moreover, it has been demonstrated that hypertriglyceridemia can result in urinary incontinence by downregulating the expression of Anoctamin 1 in urethral smooth muscle and reducing the effect of urethral spontaneous tone on urethral movement.[19]

Patients with MASLD exhibit a pronounced chronic inflammatory response and oxidative stress state, accompanied by elevated levels of inflammatory mediators such as C-reactive protein (CRP) and tumor necrosis factor-alpha (TNF-α).[20]

Variables	n (%)	NON-MASLD	MASLD	OR (95% CI)	P	P for interaction
All patients	17221 (100.00)	3506/9915	3701/7306	1.76 (1.65 ~ 1.89)	<0.001	
Age						0.002
20-39	5584 (32.43)	884/3842	679/1742	1.86 (1.63 ~ 2.13)	<0.001	
40-59	5875 (34.12)	1366/3069	1581/2806	1.57 (1.40 ~ 1.76)	<0.001	
≥60	5762 (33.46)	1256/3004	1441/2758	1.50 (1.34 ~ 1.68)	<0.001	
Race						0.082
Mexican American	2786 (16.18)	537/1368	754/1418	1.50 (1.28 ~ 1.76)	<0.001	
Non-Hispanic White	8055 (46.77)	1965/4924	1769/3131	1.77 (1.60 ~ 1.95)	<0.001	
Non-Hispanic Black	3472 (20.16)	404/1730	654/1742	1.76 (1.50 ~ 2.05)	<0.001	
Other Race	2908 (16.89)	600/1893	524/1015	2.13 (1.79 ~ 2.52)	<0.001	
Education Level						0.039
Less than 9th grade	1588 (9.22)	307/768	432/820	1.56 (1.26 ~ 1.92)	<0.001	
9–12th grade	6201 (36.01)	1203/3253	1488/2948	1.66 (1.49 ~ 1.86)	<0.001	
More than 12th grade	9432 (54.77)	1996/5894	1781/3538	1.87 (1.70 ~ 2.06)	<0.001	
Marital Status						0.034
Cohabitation	9449 (54.87)	2168/5576	2066/3873	1.70 (1.55 ~ 1.86)	<0.001	
Solitude	7772 (45.13)	1338/4339	1635/3433	1.85 (1.67 ~ 2.05)	<0.001	
Family PIR						0.967
Low	5381 (31.25)	924/2712	1338/2669	1.76 (1.56 ~ 1.98)	<0.001	
Medium	6582 (38.22)	1302/3754	1420/2828	1.78 (1.60 ~ 1.99)	<0.001	
High	5258 (30.53)	1280/3449	943/1809	1.73 (1.52 ~ 1.97)	<0.001	
Smoke						0.704
No	10673 (61.98)	2109/6377	2058/4296	1.75 (1.61 ~ 1.91)	<0.001	
Yes	6548 (38.02)	1397/3538	1643/3010	1.78 (1.60 ~ 1.98)	<0.001	
Alcohol						0.156
No	7106 (41.26)	1293/3735	1640/3371	1.68 (1.51 ~ 1.86)	<0.001	
Yes	10115 (58.74)	2213/6180	2061/3935	1.82 (1.66 ~ 1.99)	<0.001	
Physical Activity						0.693
Inactive	5383 (31.26)	981/2670	1393/2713	1.69 (1.50 ~ 1.90)	<0.001	
Moderate	7106 (41.26)	1486/4041	1577/3065	1.82 (1.64 ~ 2.02)	<0.001	
Vigorous	4732 (27.48)	1039/3204	731/1528	1.70 (1.48 ~ 1.96)	<0.001	
Menopausal						0.010
No	8507 (49.40)	1644/5541	1369/2966	1.80 (1.63 ~ 2.00)	<0.001	
Yes	8714 (50.60)	1862/4374	2332/4340	1.54 (1.41 ~ 1.69)	<0.001	
Diabetes mellitus						0.693
No	14363 (83.40)	3162/9120	2564/5243	1.77 (1.65 ~ 1.91)	<0.001	
Yes	2858 (16.60)	344/795	1137/2063	1.56 (1.31 ~ 1.86)	<0.001	
Hypertension						0.175
No	11007 (63.92)	2430/7392	1735/3615	1.81 (1.66 ~ 1.98)	<0.001	
Yes	6214 (36.08)	1076/2523	1966/3691	1.55 (1.39 ~ 1.73)	<0.001	
Metabolic syndrome						0.236
No	11787 (68.45)	2981/8742	1400/3045	1.72 (1.57 ~ 1.88)	<0.001	
Yes	5434 (31.55)	525/1173	2301/4261	1.51 (1.32 ~ 1.72)	<0.001	

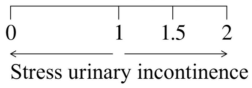

Figure 3 Subgroup analysis of the relationship between MASLD and stress urinary incontinence. Adjusted variables: age, race, education level, marital status, family PIR, smoke, alcohol, physical activity, diabetes mellitus, hypertension, coronary heart disease, stroke, and cancer. The model was not adjusted for the stratification variables themselves in the corresponding stratification analysis.

Abbreviations: MASLD, Metabolic dysfunction-associated steatotic liver disease; PIR, Poverty-to-income ratio; OR, odds ratio; CI, confidence interval.

These pathological processes may increase the risk of urinary incontinence by impairing the functional and structural stability of the pelvic floor muscles and neural tissue, thereby increasing the likelihood of urinary incontinence.[21,22] The presence of inflammatory responses may result in fibrosis, atrophy, and dysfunction of the pelvic floor muscles. Furthermore, oxidative stress has the potential to impair the normal functioning of the bladder and urethra by damaging

Variables	n (%)	NON-MASLD	MASLD	OR (95% CI)	P	P for interaction
All patients	17221 (100.00)	2368/9915	2669/7306	1.50 (1.39 ~ 1.61)	<0.001	
Age						0.050
20-39	5584 (32.43)	469/3842	375/1742	1.59 (1.35 ~ 1.87)	<0.001	
40-59	5875 (34.12)	729/3069	998/2806	1.49 (1.32 ~ 1.69)	<0.001	
≥60	5762 (33.46)	1170/3004	1296/2758	1.35 (1.21 ~ 1.52)	<0.001	
Race						0.188
Mexican American	2786 (16.18)	314/1368	484/1418	1.33 (1.11 ~ 1.60)	0.002	
Non-Hispanic White	8055 (46.77)	1256/4924	1193/3131	1.49 (1.34 ~ 1.66)	<0.001	
Non-Hispanic Black	3472 (20.16)	459/1730	661/1742	1.43 (1.23 ~ 1.67)	<0.001	
Other Race	2908 (16.89)	339/1893	331/1015	1.80 (1.49 ~ 2.19)	<0.001	
Education Level						0.276
Less than 9th grade	1588 (9.22)	220/768	352/820	1.69 (1.35 ~ 2.12)	<0.001	
9–12th grade	6201 (36.01)	878/3253	1109/2948	1.46 (1.30 ~ 1.64)	<0.001	
More than 12th grade	9432 (54.77)	1270/5894	1208/3538	1.48 (1.33 ~ 1.64)	<0.001	
Marital Status						0.941
Cohabitation	9449 (54.87)	1217/5576	1301/3873	1.49 (1.34 ~ 1.65)	<0.001	
Solitude	7772 (45.13)	1151/4339	1368/3433	1.51 (1.36 ~ 1.68)	<0.001	
Family PIR						0.265
Low	5381 (31.25)	675/2712	1043/2669	1.60 (1.41 ~ 1.82)	<0.001	
Medium	6582 (38.22)	951/3754	1019/2828	1.44 (1.28 ~ 1.62)	<0.001	
High	5258 (30.53)	742/3449	607/1809	1.43 (1.24 ~ 1.65)	<0.001	
Smoke						0.302
No	10673 (61.98)	1432/6377	1479/4296	1.43 (1.30 ~ 1.58)	<0.001	
Yes	6548 (38.02)	936/3538	1190/3010	1.59 (1.41 ~ 1.78)	<0.001	
Alcohol						0.367
No	7106 (41.26)	935/3735	1267/3371	1.56 (1.40 ~ 1.75)	<0.001	
Yes	10115 (58.74)	1433/6180	1402/3935	1.44 (1.30 ~ 1.58)	<0.001	
Physical Activity						0.922
Inactive	5383 (31.26)	785/2670	1118/2713	1.47 (1.30 ~ 1.66)	<0.001	
Moderate	7106 (41.26)	1003/4041	1102/3065	1.52 (1.36 ~ 1.71)	<0.001	
Vigorous	4732 (27.48)	580/3204	449/1528	1.45 (1.24 ~ 1.70)	<0.001	
Menopausal						0.051
No	8507 (49.40)	826/5541	755/2966	1.56 (1.38 ~ 1.76)	<0.001	
Yes	8714 (50.60)	1542/4374	1914/4340	1.40 (1.27 ~ 1.54)	<0.001	
Diabetes mellitus						0.352
No	14363 (83.40)	2062/9120	1762/5243	1.53 (1.41 ~ 1.65)	<0.001	
Yes	2858 (16.60)	306/795	907/2063	1.30 (1.09 ~ 1.56)	0.003	
Hypertension						0.471
No	11007 (63.92)	1478/7392	1105/3615	1.53 (1.39 ~ 1.68)	<0.001	
Yes	6214 (36.08)	890/2523	1564/3691	1.43 (1.28 ~ 1.60)	<0.001	
Metabolic syndrome						0.589
No	11787 (68.45)	1994/8742	1050/3045	1.58 (1.43 ~ 1.74)	<0.001	
Yes	5434 (31.55)	374/1173	1619/4261	1.51 (1.30 ~ 1.74)	<0.001	

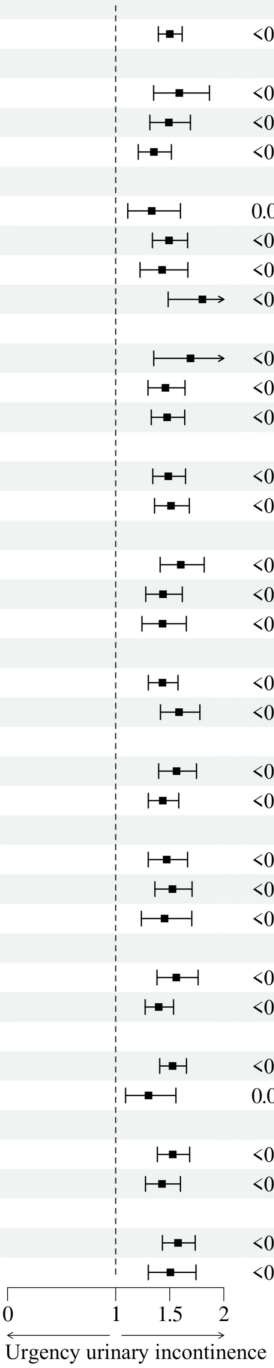

Figure 4 Subgroup analysis of the relationship between MASLD and urgency urinary incontinence. Adjusted variables: age, race, education level, marital status, family PIR, smoke, alcohol, physical activity, diabetes mellitus, hypertension, coronary heart disease, stroke, and cancer. The model was not adjusted for the stratification variables themselves in the corresponding stratification analysis.

Abbreviations: MASLD, Metabolic dysfunction-associated steatotic liver disease; PIR, Poverty-to-income ratio; OR, odds ratio; CI, confidence interval.

nerve endings and conduction pathways.[23,24] Moreover, inflammatory processes may contribute to an elevated risk of incontinence by affecting nerve conduction velocity and sensitivity.[25]

Hormonal levels are a significant factor in the pathogenesis of urinary incontinence. Estrogen plays a pivotal role in the pathogenesis of urinary incontinence in women.[26–28] Patients with MASLD may exhibit disrupted sex hormone levels as a consequence of underlying metabolic abnormalities and obesity. These factors can subsequently impact the function

Variables	n (%)	NON-MASLD	MASLD	OR (95% CI)	P	P for interaction
All patients	17221 (100.00)	1331/9915	1754/7306	1.69 (1.55 ~ 1.84)	<0.001	
Age						0.012
20-39	5584 (32.43)	238/3842	235/1742	1.77 (1.44 ~ 2.18)	<0.001	
40-59	5875 (34.12)	447/3069	705/2806	1.66 (1.44 ~ 1.92)	<0.001	
≥60	5762 (33.46)	646/3004	814/2758	1.44 (1.26 ~ 1.64)	<0.001	
Race						0.478
Mexican American	2786 (16.18)	191/1368	347/1418	1.56 (1.27 ~ 1.93)	<0.001	
Non-Hispanic White	8055 (46.77)	756/4924	826/3131	1.62 (1.44 ~ 1.83)	<0.001	
Non-Hispanic Black	3472 (20.16)	179/1730	347/1742	1.81 (1.48 ~ 2.22)	<0.001	
Other Race	2908 (16.89)	205/1893	234/1015	1.93 (1.54 ~ 2.42)	<0.001	
Education Level						0.554
Less than 9th grade	1588 (9.22)	137/768	252/820	1.81 (1.40 ~ 2.33)	<0.001	
9–12th grade	6201 (36.01)	514/3253	750/2948	1.65 (1.44 ~ 1.88)	<0.001	
More than 12th grade	9432 (54.77)	680/5894	752/3538	1.68 (1.48 ~ 1.90)	<0.001	
Marital Status						0.820
Cohabitation	9449 (54.87)	710/5576	884/3873	1.69 (1.50 ~ 1.90)	<0.001	
Solitude	7772 (45.13)	621/4339	870/3433	1.69 (1.49 ~ 1.92)	<0.001	
Family PIR						0.765
Low	5381 (31.25)	415/2712	717/2669	1.67 (1.45 ~ 1.93)	<0.001	
Medium	6582 (38.22)	504/3754	667/2828	1.80 (1.57 ~ 2.07)	<0.001	
High	5258 (30.53)	412/3449	370/1809	1.52 (1.28 ~ 1.81)	<0.001	
Smoke						0.639
No	10673 (61.98)	779/6377	944/4296	1.64 (1.46 ~ 1.84)	<0.001	
Yes	6548 (38.02)	552/3538	810/3010	1.75 (1.54 ~ 2.00)	<0.001	
Alcohol						0.801
No	7106 (41.26)	534/3735	834/3371	1.72 (1.51 ~ 1.96)	<0.001	
Yes	10115 (58.74)	797/6180	920/3935	1.65 (1.47 ~ 1.85)	<0.001	
Physical Activity						0.968
Inactive	5383 (31.26)	438/2670	745/2713	1.69 (1.47 ~ 1.95)	<0.001	
Moderate	7106 (41.26)	556/4041	712/3065	1.73 (1.52 ~ 1.98)	<0.001	
Vigorous	4732 (27.48)	337/3204	297/1528	1.51 (1.24 ~ 1.83)	<0.001	
Menopausal						0.016
No	8507 (49.40)	459/5541	502/2966	1.74 (1.50 ~ 2.02)	<0.001	
Yes	8714 (50.60)	872/4374	1252/4340	1.52 (1.36 ~ 1.69)	<0.001	
Diabetes mellitus						0.143
No	14363 (83.40)	1142/9120	1132/5243	1.74 (1.58 ~ 1.91)	<0.001	
Yes	2858 (16.60)	189/795	622/2063	1.39 (1.14 ~ 1.69)	0.001	
Hypertension						0.627
No	11007 (63.92)	831/7392	722/3615	1.70 (1.51 ~ 1.91)	<0.001	
Yes	6214 (36.08)	500/2523	1032/3691	1.58 (1.38 ~ 1.79)	<0.001	
Metabolic syndrome						0.989
No	11787 (68.45)	1107/8742	630/3045	1.70 (1.51 ~ 1.90)	<0.001	
Yes	5434 (31.55)	224/1173	1124/4261	1.66 (1.40 ~ 1.96)	<0.001	

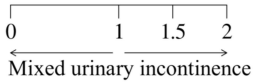

Figure 5 Subgroup analysis of the relationship between MASLD and mixed urinary incontinence. Adjusted variables: age, race, education level, marital status, family PIR, smoke, alcohol, physical activity, diabetes mellitus, hypertension, coronary heart disease, stroke, and cancer. The model was not adjusted for the stratification variables themselves in the corresponding stratification analysis.

Abbreviations: MASLD, Metabolic dysfunction-associated steatotic liver disease; PIR, Poverty-to-income ratio; OR, odds ratio; CI, confidence interval.

of pelvic floor muscles and the urethral sphincter.[29] Reducing estrogen levels may result in the relaxation of pelvic floor muscles and a deficiency in urethral sphincter function, thereby increasing the likelihood of urinary incontinence.[27,30]

Metabolic abnormalities in patients with MASLD may indirectly increase the risk of urinary incontinence by affecting the neurological regulation of the processes above. Metabolic abnormalities may result in autonomic dysfunction, affecting the innervation of the bladder and urethra. This can lead to detrusor overactivity or urethral sphincter

dysfunction.[25,31,32] Furthermore, prolonged metabolic dysfunction may reduce central nervous system bladder control, thereby exacerbating incontinence.[32]

Lifestyle and psychological factors may also be significant in the relationship between MASLD and urinary incontinence. Patients with MASLD are frequently associated with poor lifestyle habits, including a lack of physical activity and high-calorie diets, which may independently increase the risk of urinary incontinence.[33,34] Additionally, MASLD, as a chronic disease, may induce psychological distress and anxiety in patients, which may further contribute to urinary incontinence by affecting the function of the pelvic floor muscles and the neuromodulation of the bladder.[35,36]

The higher prevalence of sarcopenia among older women serves to exacerbate further the risk of urinary incontinence in patients with MASLD. Sarcopenia, a syndrome characterized by reduced skeletal muscle mass and strength that becomes more prevalent with age, not only affects the function of limb muscles but may also spread to the pelvic floor muscles, weakening their function and strength. This, in turn, directly affects the ability of the urethra to close, increasing the risk of urinary incontinence.[37] Some studies have substantiated the assertion that sarcopenia represents an independent risk factor for urinary incontinence, with this correlation being particularly pronounced in older women.[10,38] The presence of sarcopenia may serve to further amplify the risk of urinary incontinence in the context of older women with MASLD. Patients with MASLD often have associated metabolic disorders such as insulin resistance and obesity, all of which may hurt muscle mass and function, thereby serving to exacerbate the symptoms of sarcopenia.[39,40] Furthermore, the treatment of liver disease in patients with MASLD may necessitate a reduction in protein intake, which may also have an additional impact on muscle health.[41] Consequently, for older women with MASLD, the prevention and treatment of sarcopenia is not only crucial for the management of muscle health but may also be an effective strategy for reducing the risk of urinary incontinence.

In this study, we conducted subgroup analyses to evaluate the influence of disparate demographic and clinical characteristics on the relationship between MASLD and urinary incontinence. The results of the subgroup analyses indicated that the relationship between MASLD and the risk of different types of urinary incontinence exhibited some variability across the identified subgroups. The impact of MASLD on the risk of SUI and MUI was particularly pronounced in premenopause and young adult women (20–39 years). The primary factor that may be responsible for these results is the high prevalence of urinary incontinence among postmenopausal women. In this cohort, the supportive function of the pelvic floor muscles tends to deteriorate with age, and the ability of the urethra to close is weakened. This physiologic change constitutes a significant trigger for stress incontinence.[37] Concurrently, estrogen levels decline with age, and estrogen is a vital element in maintaining typical urethral and bladder functionality. A deficiency of estrogen may precipitate atrophy and a reduction in the tone of the urethra and bladder mucosa, which in turn elevates the risk of urinary incontinence.[42] Furthermore, the aging process is frequently accompanied by neurological deterioration and a high incidence of chronic diseases, which have a significant impact on the development of urinary incontinence. In contrast, metabolic disorders have a relatively minor impact in this context. In contrast, in premenopausal (ie, younger) women, pelvic floor muscle function and estrogen levels are typically maintained at normal levels. It is important to note that the age of onset of metabolic disorders such as hyperglycemia, hypertension, and hyperlipidemia is trending younger. This demographic is more susceptible to metabolic disorders due to the stressors associated with life and work, as well as the influence of poor lifestyle habits (eg, irregular diet, lack of physical exercise, etc).[43,44] Concurrently, young adult women exhibit heightened physiological activity and metabolic rate, rendering the impact of metabolic disorders on their urinary systems more pronounced. Consequently, metabolic disorders exert a more pronounced influence on the risk of urinary incontinence in young women than other factors. The correlation between MASLD and SUI risk was more pronounced in the subgroup with a higher level of education. This may be attributed to the observation that individuals with higher educational attainment tend to exhibit heightened concern about their health status and are more likely to recognize and report health issues. Conversely, individuals with a higher level of education may be more susceptible to metabolic disorders and MASLD due to the demands of their profession and the prevalence of elevated work pressure and stress in their lives. These factors collectively may elevate the risk of SUI. Those in a solitary state demonstrated heightened sensitivity to the impact of MASLD on SUI risk. Such differences in social support and lifestyle habits among individuals who live alone may be a contributing factor. Those who live alone may be more prone to adopting unhealthy lifestyles and experiencing psychological distress due to a lack of familial and social support, which in turn increases the risk of metabolic syndrome and urinary incontinence.[45]

The findings of this study have significant clinical implications. First, the study reveals the potential association between MASLD and urinary incontinence, providing clinicians with new perspectives for evaluating and treating patients with urinary incontinence. It is recommended that clinicians pay closer attention to the urinary health of patients with MASLD, particularly those who present with incontinence symptoms, and take appropriate preventive measures. Secondly, this study emphasizes the influence of metabolic diseases on the health of numerous bodily systems, thereby underscoring the necessity for public health authorities to reinforce the prevention and control of metabolic diseases to diminish the prevalence of associated complications. Furthermore, this study offers valuable insights and avenues for future research, including a deeper investigation into the underlying mechanisms of urinary incontinence related to MASLD and the development of targeted prevention and treatment strategies.

Despite the rigor and scientific rigor of the study's design and implementation, some things could be improved. Firstly, the data presented in this study were derived from a cross-sectional survey, which does not allow for the direct inference of causality. Further research should consider prospective cohort studies or intervention trials to validate the causal relationship between MASLD and urinary incontinence. Secondly, this study's assessment of urinary incontinence was based on questionnaire results, which may be susceptible to recall and information bias. Thirdly, the evaluation of MASLD in this study was based on the calculation of FLI. While FLI has been extensively utilized as a surrogate marker for hepatic steatosis, its accuracy and specificity warrant further validation. Fourth, this study focused on adult females. Future studies should be expanded to include males and different age groups better to understand the correlation between MASLD and urinary incontinence. Furthermore, the absence of data about sarcopenia precluded a more detailed examination of the precise impact of this condition on the risk of urinary incontinence in patients with MASLD. This represents a crucial avenue for future investigation. Ultimately, this study did not investigate the precise mechanisms through which MASLD leads to urinary incontinence. Consequently, further basic and clinical research is required to elucidate the underlying physiopathologic processes. Further research may be directed towards elucidating the molecular mechanisms underlying the association between MASLD and urinary incontinence, developing targeted preventive and therapeutic strategies, and evaluating the effects of different interventions.

Conclusion

The collective findings of this study indicate a significant correlation between MASLD and an elevated risk of urinary incontinence. MASLD may elevate the risk of urinary incontinence through many mechanisms, including metabolic disorders, inflammatory responses, oxidative stress, hormone levels, neuromodulation abnormalities, and lifestyle and psychological factors. Nevertheless, further basic and clinical studies are required to elucidate these mechanisms' specific modes of action and interrelationships. Further research should concentrate on these underlying physiopathologic processes to gain a deeper understanding of the relationship between MASLD and urinary incontinence and to provide a basis for developing effective prevention and treatment strategies.

Data Sharing Statement

The National Health and Nutrition Examination Survey dataset is publicly available at the National Center for Health Statistics of the Centers for Disease Control and Prevention (https://www.cdc.gov/nchs/nhanes/index.htm).

Ethics Statement

The studies involving humans were approved by the National Center for Health Statistics Ethics Review Board. The studies were conducted according to local legislation and institutional requirements. The participants provided their written informed consent to participate in this study. The Ethics Committee of Changzhou Third People's Hospital waived the necessity of ethical approval for this study, as the NHANES is a publicly accessible database.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest in this study.

References

1. Rinella ME, Lazarus JV, Ratziu V, et al. A multisociety Delphi consensus statement on new fatty liver disease nomenclature. *J Hepatol*. 2023;79(6):1542–1556.
2. Huang DQ, El-Serag HB, Loomba R. Global epidemiology of NAFLD-related HCC: trends, predictions, risk factors and prevention. *Nat Rev Gastroenterol Hepatol*. 2021;18(4):223–238.
3. Riazi K, Azhari H, Charette JH, et al. The prevalence and incidence of NAFLD worldwide: a systematic review and meta-analysis. *Lancet Gastroenterol Hepatol*. 2022;7(9):851–861.
4. Fan W, Bradford TM, Török NJ. Metabolic dysfunction-associated liver disease and diabetes: matrix remodeling, fibrosis, and therapeutic implications. *Ann N Y Acad Sci*. 2024;1538(1):21–33.
5. Riley DR, Hydes T, Hernadez G, Zhao SS, Alam U, Cuthbertson DJ. The synergistic impact of type 2 diabetes and MASLD on cardiovascular, liver, diabetes-related and cancer outcomes. *Liver Int*. 2024.
6. Haylen BT, de Ridder D, Freeman RM, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. *Neurourol Urodyn*. 2010;29(1):4–20.
7. Abufaraj M, Xu T, Cao C, et al. Prevalence and trends in urinary incontinence among women in the United States, 2005-2018. *Am J Obstet Gynecol*. 2021;225(2):166.e1–166.e12.
8. Aoki Y, Brown HW, Brubaker L, Cornu JN, Daly JO, Cartwright R. Urinary incontinence in women. *Nat Rev Dis Primers*. 2017;3:17042.
9. Wu JM. Stress Incontinence in Women. *N Engl J Med*. 2021;384(25):2428–2436.
10. Erdogan T, Bahat G, Kilic C, et al. The relationship between sarcopenia and urinary incontinence. *Eur Geriatr Med*. 2019;10(6):923–929.
11. Tarantino G, Sinatti G, Citro V, Santini SJ, Balsano C. Sarcopenia, a condition shared by various diseases: can we alleviate or delay the progression? *Intern Emerg Med*. 2023;18(7):1887–1895.
12. Li X, Zhou W, Hu G. The association between non-alcoholic fatty liver disease and urinary incontinence among adult females in the United States. *BMC Public Health*. 2024;24(1):1373.
13. Bedogni G, Bellentani S, Miglioli L, et al. The Fatty Liver Index: a simple and accurate predictor of hepatic steatosis in the general population. *BMC Gastroenterol*. 2006;6:33.
14. Park J, Kim G, Kim BS, et al. The associations of hepatic steatosis and fibrosis using fatty liver index and BARD score with cardiovascular outcomes and mortality in patients with new-onset type 2 diabetes: a nationwide cohort study. *Cardiovasc Diabetol*. 2022;21(1):53.
15. Fuchs A, Samovski D, Smith GI, et al. Associations Among Adipose Tissue Immunology, Inflammation, Exosomes and Insulin Sensitivity in People With Obesity and Nonalcoholic Fatty Liver Disease. *Gastroenterology*. 2021;161(3):968–981.e912.
16. Li S, Zou J, Wang Z, Wang M, Yuan Y, Lv H. Correlation Between Insulin Resistance and Urinary Incontinence in Female Patients with Type 2 Diabetes Mellitus. *Int Urogynecol J*. 2024;35(2):431–440.
17. Sharma P, Rani N, Gangwar A, Singh R, Kaur R, Upadhyaya K. Diabetic Neuropathy: a Repercussion of Vitamin D Deficiency. *Curr Diabetes Rev*. 2023;19(6):e170822207592.
18. Ebbesen MH, Hannestad YS, Midthjell K, Hunskaar S. Diabetes related risk factors did not explain the increased risk for urinary incontinence among women with diabetes. The Norwegian HUNT/EPINCONT study. *BMC Urol*. 2009;9:11.
19. Chen D, Li H, Wang X, Zhang L, Ji Z, Zhang J. Hypertriglyceridemia Impairs Urethral Spontaneous Tone Through Down-regulation of ANO1 in Mouse Urethral Smooth Muscle Cells. *Urology*. 2022;165:157–163.
20. He Q, Wang Z, Liu G, Daneshgari F, MacLennan GT, Gupta S. Metabolic syndrome, inflammation and lower urinary tract symptoms: possible translational links. *Prostate Cancer Prostatic Dis*. 2016;19(1):7–13.
21. Shinohara M, Sumino Y, Sato F, Kiyono T, Hashimoto N, Mimata H. Tumor necrosis factor-alpha inhibits differentiation of myogenic cells in human urethral rhabdosphincter. *Int J Urol*. 2017;24(6):461–467.
22. Post WM, Widomska J, Grens H, et al. Molecular Processes in Stress Urinary Incontinence: a Systematic Review of Human and Animal Studies. *Int J Mol Sci*. 2022;23(6):3401.
23. Nocchi L, Daly DM, Chapple C, Grundy D. Induction of oxidative stress causes functional alterations in mouse urothelium via a TRPM8-mediated mechanism: implications for aging. *Aging Cell*. 2014;13(3):540–550.
24. Doumouchtsis SK, Loganathan J, Pergialiotis V. The role of obesity on urinary incontinence and anal incontinence in women: a review. *Bjog*. 2022;129(1):162–170.
25. Fuselier A, Hanberry J, Margaret Lovin J, Gomelsky A. Obesity and Stress Urinary Incontinence: impact on Pathophysiology and Treatment. *Curr Urol Rep*. 2018;19(1):10.

26. Gartlehner G, Patel SV, Feltner C, et al. Hormone Therapy for the Primary Prevention of Chronic Conditions in Postmenopausal Women: evidence Report and Systematic Review for the US Preventive Services Task Force. *JAMA*. 2017;318(22):2234–2249.
27. Russo E, Caretto M, Giannini A, et al. Management of urinary incontinence in postmenopausal women: an EMAS clinical guide. *Maturitas*. 2021;143:223–230.
28. Li W, Wang Z, Li X, et al. Associations Between Serum Estrogen Levels and Urinary Incontinence in Women: a Cross-sectional Analysis of NHANES 2013 to 2016. *Urology*. 2024;188:63–69.
29. Opoku AA, Abushama M, Konje JC. Obesity and menopause. *Best Pract Res Clin Obstet Gynaecol*. 2023;88:102348.
30. Huang H, Ding G, Li M, Deng Y, Cheng Y, Jin H. Menopause and stress urinary incontinence: the risk factors of stress urinary incontinence in perimenopausal and postmenopausal women. *J Obstet Gynaecol Res*. 2023;49(10):2509–2518.
31. Hsu LN, Hu JC, Chen PY, Lee WC, Chuang YC. Metabolic Syndrome and Overactive Bladder Syndrome May Share Common Pathophysiologies. *Biomedicines*. 2022;10(8):1957.
32. Kwon J, Kim DY, Cho KJ, et al. Pathophysiology of Overactive Bladder and Pharmacologic Treatments Including β3-Adrenoceptor Agonists - Basic Research Perspectives. *Int Neurourol J*. 2024;28(Suppl 1):12–33.
33. Bauer SR, Kenfield SA, Sorensen M, et al. Physical Activity, Diet, and Incident Urinary Incontinence in Postmenopausal Women: women's Health Initiative Observational Study. *J Gerontol a Biol Sci Med Sci*. 2021;76(9):1600–1607.
34. Gao D, Zhang C, Chen Q, et al. Association between dietary fatty acids and urinary incontinence. *Heliyon*. 2024;10(7):e28595.
35. Zhang X, Ma L, Li J, Zhang W, Xie Y, Wang Y. Mental health and lower urinary tract symptoms: results from the NHANES and Mendelian randomization study. *J Psychosom Res*. 2024;178:111599.
36. Stephan Y, Sutin AR, Terracciano A. Personality traits and the risk of urinary incontinence: evidence from three longitudinal samples. *Int J Geriatr Psychiatry*. 2024;39(4):e6084.
37. Silva RRL, Coutinho JFV, Vasconcelos CTM, et al. Prevalence of sarcopenia in older women with pelvic floor dysfunction. *Eur J Obstet Gynecol Reprod Biol*. 2021;263:159–163.
38. Jerez-Roig J, Farrés-Godayol P, Yildirim M, et al. Prevalence of urinary incontinence and associated factors in nursing homes: a multicentre cross-sectional study. *BMC Geriatr*. 2024;24(1):169.
39. Wong R, Yuan LY. Sarcopenia and metabolic dysfunction associated steatotic liver disease: time to address both. *World J Hepatol*. 2024;16(6):871–877.
40. Li X, He J, Sun Q. The prevalence and effects of sarcopenia in patients with metabolic dysfunction-associated steatotic liver disease (MASLD): a systematic review and meta-analysis. *Clin Nutr*. 2024;43(9):2005–2016.
41. Singh A, Buckholz A, Kumar S, Newberry C. Implications of Protein and Sarcopenia in the Prognosis, Treatment, and Management of Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD). *Nutrients*. 2024;16(5):658.
42. Christmas MM, Iyer S, Daisy C, Maristany S, Letko J, Hickey M. Menopause hormone therapy and urinary symptoms: a systematic review. *Menopause*. 2023;30(6):672–685.
43. Lamerton TJ, Mielke GI, Brown WJ. Urinary incontinence, body mass index, and physical activity in young women. *Am J Obstet Gynecol*. 2021;225(2):164.e1–164.e13.
44. Yuan Y, Huang Y, Huang H, Zeng S, Hu Z. Association Between Added Sugar Intake and Urinary Incontinence in Females: a Cross-sectional Population-based Study. *Int Urogynecol J*. 2024;35(6):1201–1210.
45. Lukacz ES, Santiago-Lastra Y, Albo ME, Brubaker L. Urinary Incontinence in Women: a Review. *JAMA*. 2017;318(16):1592–1604.