

### Title:

Relative fat mass and constipation in US adults – A cross-sectional study of 11,380 participants from NHANES 2005-2010

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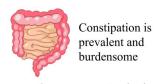
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# Background



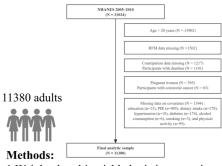
RFM is a novel, more accurate obesity measure

RFM Constipation

The relationship between RFM and Constipation remains unclear

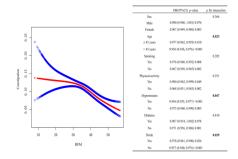
$$RFM = 64 - (\frac{20 \times Height(cm)}{WC(cm)}) + (12 \times Gender)$$

## Study design



- 1. Weighted multivariable logistic regression
- 2.Smooth curve fitting and threshold effect analyses
- 3. Subgroup analyses and interaction tests
- 4.propensity score matching

# Findings





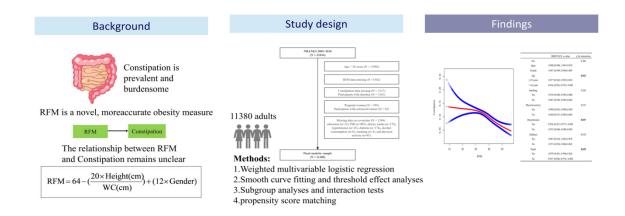
Relative fat mass and constipation in US adults – A cross-sectional study of 11,380 participants from NHANES 2005-2010

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### **Abstract**

**Background:** Constipation is a common gastrointestinal disorder closely associated with obesity. Relative Fat Mass(RFM) is a newer anthropometric index that offers a more precise reflection of body fat distribution than traditional methods. Despite its advantages, the potential link between RFM and the likelihood of experiencing



constipation has not been thoroughly examined. This study was therefore designed to explore the association between RFM and constipation

Methods: Data were obtained from the 2005-2010 cycles of the National Health and Nutrition Examination Survey (NHANES). Constipation was defined using the Bristol Stool Form Scale and questionnaire responses. Weighted multivariable logistic regression models were employed to evaluate the association between RFM and constipation. Propensity score matching (PSM) was used to balance baseline covariates between groups. Analyses were conducted both before and after PSM to test the robustness of the findings. Smooth curve fitting and threshold effect analyses were conducted to explore potential nonlinear relationships. Subgroup analyses and interaction tests were used to assess possible heterogeneity across different population strata.

**Results:** A total of 11,380 participants were included in the final analysis, among whom 1,206 were classified as having constipation. Logistic regression revealed that in the fully adjusted model, each one-unit increase in RFM was associated with a 2.9% reduction in the odds of constipation (OR = 0.971, 95%CI: 0.956-0.986, p = 0.0011). Furthermore, PSM analyses confirmed the robustness of the results. The inverse association between RFM and constipation was more pronounced among individuals aged > 45 years, those with hypertension, and those who did not consume alcohol (all p for interaction < 0.05). Smooth curve fitting and threshold effect analysis indicated a nonlinear relationship, with an inflection point at an RFM of 36.06.

**Conclusion:** Our study suggests a significant inverse association between RFM and constipation. Further prospective studies are warranted to validate this relationship.

**Keywords:** Relative fat mass. Constipation. Cross-sectional study.

### 1. Introduction

Constipation is a common gastrointestinal functional disorder with global prevalence, and its epidemiological characteristics and pathophysiology have increasingly become a focus of research. Epidemiological studies have shown that



the median prevalence of constipation in the United States is approximately 16% [1], while the global average prevalence is around 14% [2]. In the U.S. alone, constipation accounts for approximately 2.5 million outpatient visits annually, placing a substantial burden on the healthcare system[1]. Chronic constipation is not only associated with psychological comorbidities such as anxiety and depression [3], but is also closely linked to reduced quality of life. Moreover, emergency department visits and hospital admissions related to constipation are on the rise [4].

In exploring risk factors for constipation, an increasing number of studies have investigated the potential association between obesity and constipation. However, the findings remain inconsistent. Some studies, including one conducted among Italian residents, have reported a higher prevalence of constipation in obese individuals, potentially attributable to dietary habits and levels of physical activity [5]. Other studies have suggested that obesity may influence the development of constipation through mechanisms such as alterations in the gut microbiota and impaired gastrointestinal motility [6, 7]. Additionally, obesity has been identified as a risk factor for various gastrointestinal symptoms. For instance, a significant positive correlation has been observed between higher obesity prevalence and diarrhea [8], and the prevalence of constipation is reportedly higher among obese adults [9]. However, some studies have found no significant association between obesity and constipation [10].

These conflicting findings highlight the limitations of using Body Mass Index (BMI) as a measure of obesity. BMI does not differentiate between fat and lean body mass, making it an imprecise indicator for assessing the relationship between adiposity and constipation. To overcome the shortcomings of BMI, Woolcott OO et al. [11] introduced the RFM index, which was developed using data from bioelectrical impedance analysis and dual-energy X-ray absorptiometry. The calculation of RFM utilizes an individual's height, waist circumference (WC), and sex, allowing for a more accurate estimation of total body fat. Previous research has demonstrated strong associations between RFM and metabolic or chronic diseases such as diabetes [12], cardiovascular disease [13], and kidney disease [14].



However, to date, no systematic studies have assessed the epidemiological relationship between RFM and constipation. Therefore, this study aims to use RFM as a novel adiposity index to further explore the potential link between obesity and constipation.

#### 2. Materials and Methods

### 2.1 Data Source and Participants

Data for this research were sourced from the NHANES, a continuous program that evaluates the health and diet of the non-institutionalized civilian populace in the U.S. To achieve a nationally representative sample, NHANES utilizes a sophisticated, multistage probability sampling methodology. Detailed information about NHANES is available at: <a href="http://www.cdc.gov/nchs/nhanes/index.htm">http://www.cdc.gov/nchs/nhanes/index.htm</a>.

This study utilized cross-sectional data from the 2005-2010 NHANES cycles, with an initial sample size of 31,034 participants. The following exclusion criteria were applied sequentially:

- (1) Individuals younger than 20 years of age (n = 13,902);
- (2) Participants with missing data on height or waist circumference (n = 1,562), those without gastrointestinal health questionnaire data (n = 1,217), or those reporting diarrhea symptoms (n = 1,101);
- (3) Pregnant women (n = 395) and individuals with colorectal cancer (n = 83);
- (4) Participants with missing data for covariates (n = 1,394).

After applying these rigorous exclusion criteria, a total of 11,380 participants were included in the final analysis (Figure 1).

### 2.2 Exposure Variable

RFM was calculated using the following formula [15]:



RFM = 
$$64 - (\frac{20 \times \text{Height(cm)}}{\text{WC(cm)}}) + (12 \times \text{Gender})$$

Where sex is coded as 1 for females and 0 for males. Height and waist circumference were measured by trained professionals at the NHANES Mobile Examination Center . Height was obtained using a standardized stadiometer, while waist circumference was measured at the intersection of the midaxillary line and the uppermost lateral border of the iliac crest at the end of normal expiration. Measurements were recorded to the nearest 0.1 cm.

## 2.3 Constipation

Constipation was operationally defined through two complementary standardized criteria derived from gastrointestinal health questionnaires, leveraging the established physiological correlation between stool consistency and colonic transit time wherein fecal morphology serves as a proxy for intestinal passage duration. First, according to the Bristol Stool Form Scale (BSFS), individuals reporting stool types 1 (separate hard lumps, like nuts) or 2 (sausage-shaped but lumpy) were classified as having constipation. Types 3 (like a sausage but with cracks on the surface), 4 (like a smooth, soft sausage or snake), and 5 (soft blobs with clear-cut edges) were considered normal. Participants reporting type 6 (fluffy pieces with ragged edges) or type 7 (watery, no solid pieces) were excluded [16]. Secondly, constipation was identified when participants self-reported a bowel movement frequency of two times per week or fewer [3].Participants meeting either criterion were considered constipated ensuring comprehensive phenotyping of both slow-transit and dyssynergic constipation subtypes.

### 2.4 Covariates

Covariates were selected based on previous studies [17-19], and included: sex, age, race, education level, family poverty income ratio (PIR), dietary intake (phosphorus, selenium, niacin, protein, fat, carbohydrates, dietary fiber, energy, cholesterol), physical activity level, diabetes, smoking status, alcohol consumption, and hypertension. Hypertension was defined based on responses to the questions: "Have



you ever been told by a doctor that you have high blood pressure?" and "Are you currently taking medication for high blood pressure?". Diabetes was defined by self-report of a physician diagnosis, insulin use, or use of oral hypoglycemic medications. Alcohol consumption was categorized as drinking (≥12 alcoholic beverages in the past year) or non-drinking. Smoking status was classified as never smokers (fewer than 100 cigarettes in lifetime) and ever smokers (≥100 cigarettes). Physical activity was divided into vigorous (e.g., heavy lifting or construction work that significantly increases breathing or heart rate) and non-vigorous activity based on questionnaire responses.

### 2.5 Statistical Analysis

NHANES employs a complex survey design incorporating clustered and stratified sampling. Therefore, all descriptive and inferential analyses incorporated sample weights, cluster, and strata to account for the survey design and ensure nationally representative estimates. Descriptive statistics were calculated for participant characteristics. Continuous variables were presented as mean ± standard deviation (SD), and group differences were tested using survey-weighted t-tests. Categorical variables were expressed as number (percentage), with group comparisons performed using survey-weighted Chi-square tests. Multivariable logistic regression was used to estimate the odds ratios (ORs) and 95% CIs for the association between RFM and constipation. Three progressively adjusted models were constructed: Model 1: adjusted for age, sex, race/ethnicity, education level, and PIR; Model 2: further adjusted for dietary factors including phosphorus, selenium, niacin, protein, fat, carbohydrates, dietary fiber, energy; Model 3: additionally adjusted for smoking, alcohol use, hypertension, diabetes, and physical activity. To examine potential nonlinear relationships, smooth curve fitting and threshold effect analysis were conducted to identify inflection points. Subgroup analyses and interaction tests were performed to evaluate effect modification across different population strata.

To verify the robustness of the main findings and reduce potential confounding, we performed a 1:2 nearest-neighbor PSM analysis. A caliper width of 0.02 was applied



to restrict the maximum allowable difference in propensity scores between matched pairs. All covariates included in the multivariable model were used as balancing variables during the matching process. Standardized mean differences (SMDs) were calculated to assess matching quality. All statistical analyses were performed using EmpowerStats and R software, with a two-sided p value < 0.05 considered statistically significant.

#### 3. Results

### 3.1 Baseline Characteristics of the Study Population

A total of 11,380 participants were included in the final analysis, among whom 1,206 were classified as having constipation. Participants in the constipation group were younger on average and had significantly higher RFM and a higher proportion of females compared to the non-constipation group. In terms of socioeconomic status, individuals with constipation had a lower family poverty income ratio and a lower level of education. Additionally, the constipation group had a lower proportion of alcohol consumers and those engaging in vigorous physical activity, but a higher proportion of never smokers. With regard to nutritional intake, the constipation group exhibited significantly lower intakes of total energy, protein, dietary fiber, and other key nutrients (all p < 0.05), except for total sugar intake, which did not differ significantly between the two groups. There were no statistically significant differences in the prevalence of hypertension or diabetes between the constipation and non-constipation groups (p > 0.05) (Table 1) .

**4.** To control for confounding factors and improve comparability between groups, PSM was performed at a 1:2 ratio between the constipation and non-constipation groups. After matching, covariates were balanced between the two groups, providing a foundation for subsequent sensitivity analyses (Table 1).

### 3.2 Association Between RFM and Constipation

As shown in Table 2, when RFM was treated as a continuous variable, a significant inverse association with constipation risk was observed across all regression models:



Model 1: OR=0.976, 95%CI: 0.961-0.991, Model 2: OR=0.973, 95%CI: 0.958-0.988, Model 3: OR= 0.971, 95%CI: 0.956-0.986. When RFM was categorized into quartiles, the inverse association remained statistically significant in all models (all p for trend < 0.05). In the Model 3, participants in the Q4 had a 45.3% lower risk of constipation compared to those in the Q1 (OR = 0.547, 95% CI: 0.385-0.777).

In the propensity score matched sample, the inverse association remained significant (Model 3: OR = 0.975, 95% CI: 0.961-0.988, p < 0.001), with a p for trend < 0.001, further confirming the robustness of the main findings (Table 2).

### 3.3 Nonlinear Association Between RFM and Constipation

A nonlinear relationship between RFM and constipation was observed based on the smooth curve fitting analysis, with an inflection point identified at 36.06 (Figure 2 and Table 3). When RFM exceeded 36.06, each one-unit increase in RFM was associated with a 5.1% reduction in the risk of constipation (OR = 0.949, 95% CI: 0.933-0.966, p < 0.001). The log-likelihood ratio test indicated that the nonlinear model fit the data significantly better ( p < 0.001).

## 3.4 Subgroup analysis

Subgroup analyses and interaction tests were conducted to evaluate whether the association between RFM and constipation differed across population subgroups. As shown in Table 4, significant interactions were observed for age, hypertension status, and alcohol consumption (all p for interaction < 0.05). Specifically, the inverse association between RFM and constipation was stronger among individuals aged > 45 years (OR = 0.956, 95% CI: 0.936-0.976), those with hypertension (OR = 0.956, 95% CI: 0.935-0.977), and non-drinkers (OR = 0.957, 95% CI: 0.940-0.974), compared to their respective counterparts (age  $\leq$  45 years, without hypertension, and alcohol consumers).

### 5. Discussion

This study is the first to systematically evaluate the association between RFM and constipation. Regression analyses demonstrated a significant inverse association



between RFM and the risk of constipation. Furthermore, smooth curve fitting and threshold effect analyses revealed a nonlinear relationship between RFM and constipation. Subgroup analyses suggested that the protective effect of higher RFM was more pronounced among individuals aged over 45 years, those with hypertension, and non-drinkers. Sensitivity analyses further confirmed the robustness of this association.

Our findings are consistent with recent studies on body fat distribution and constipation. For example, both the body roundness index and visceral adiposity index have been reported to be inversely associated with constipation, suggesting that an appropriate level of visceral fat may play a protective role in bowel function [3, 20]. The connection between obesity and constipation is multifaceted, with several interconnected mechanisms at play. For instance, current research suggests that excess adipose tissue may release pro-inflammatory factors that activate intestinal immune responses and impair the integrity of the epithelial barrier. This chain of events can interfere with the regulation of gut motility by the enteric nervous system, potentially leading to delayed transit [7]. Additionally, obesityrelated changes in gut microbiota composition may lower the production of shortchain fatty acids (SCFAs), key microbial metabolites known to influence enteric neural signaling and gut hormone secretion, both of which are essential for maintaining normal peristalsis [6]. The low-fiber diets commonly observed among individuals with obesity may further reduce microbial diversity and SCFA output, weakening the neural stimulation of the bowel and contributing to prolonged transit time. In contrast, populations that consume high-fiber diets consistently exhibit faster gut transit than those following typical low-fiber Western diets, underscoring the established relationship between inadequate fiber intake and constipation [21, 22]. Importantly, these mechanisms are not isolated; for instance, dysbiosis can exacerbate intestinal inflammation, which in turn alters the microbiota, creating a vicious cycle that worsens constipation symptoms.

The identified RFM threshold of 36.06, approximately corresponding to the 75th



percentile of the study population, indicates that the risk of constipation becomes more apparent among individuals with relatively high body fat. This threshold may serve as an exploratory reference point beyond which excessive adiposity, particularly central or visceral fat, could adversely affect gastrointestinal motility. For context, RFM values are generally higher in females due to sex-based differences in body fat distribution; in our dataset, a value of 36.06 was more commonly observed in older adults or individuals with obesity. At lower RFM levels, insufficient fat stores may contribute to pelvic floor weakness, impaired intestinal lubrication, or nutrient deficiencies, potentially affecting bowel function [23]. Conversely, moderate fat levels may support colonic motility through anti-inflammatory or neuroendocrine mechanisms involving adipokines such as leptin and adiponectin. However, when RFM exceeds this potential inflection point, excessive visceral fat may induce chronic low-grade inflammation, insulin resistance, or autonomic dysregulation, thereby impairing gut motility [3]. These interpretations remain hypothetical and require further validation in mechanistic and longitudinal studies. Nevertheless, the observed threshold may offer a useful anthropometric marker for identifying individuals at higher risk of constipation.

Subgroup analyses revealed heterogeneity in the RFM and constipation association across different populations. In the subgroup analysis, the inverse link between RFM and constipation was more evident among participants older than 45. This could be attributable to age-associated physiological changes, such as decreased muscle mass and higher levels of visceral fat, which are known to influence the motility of the gastrointestinal system[24]. The more pronounced association among non-drinkers may be explained by studies showing that alcohol intake positively correlates with fecal microbiota diversity [25], indicating that alcohol may weaken the protective role of RFM via gut microbiota disruption. Hypertension also modulates this association; sympathetic overactivation in hypertensive patients suppresses gut motility [26, 27], and antihypertensive medications may impair intestinal smooth muscle contraction, thereby aggravating constipation [28]. From a microbiota perspective, dysbiosis in hypertension reduces SCFA production, which not only



weakens the blood pressure-lowering effect of SCFAs via G-protein-coupled receptors but also impairs serotonin-mediated colonic motility reflexes[29]. Moreover, the stronger protective effect of high RFM in females may be related to estrogen-induced visceral sensitivity [30], and elevated levels of progesterone and estradiol during the luteal phase, which slow gastrointestinal transit and harden stool consistency [31]. It should be noted that the subgroup analyses were exploratory in nature. Although several interaction terms reached nominal statistical significance (p for interaction < 0.05), no correction for multiple comparisons was applied, and therefore these findings should be interpreted with caution and validated in future studies.

This study innovatively employed RFM, a novel body composition index, and utilized nationally representative NHANES data. Through multiple regression models and threshold effect analysis, we identified a significant inverse association between RFM and constipation risk. However, several limitations should be acknowledged. First, the cross-sectional design precludes causal inference. Constipation was selfreported, potentially introducing recall bias, and objective measures such as transit time or stool consistency were not available, limiting the evaluation of underlying pathophysiological mechanisms. Although we adjusted for a wide range of covariates, the possibility of residual confounding remains. In particular, we were unable to account for unmeasured confounders such as the use of medications affecting gastrointestinal motility (e.g., opioids, laxatives), menopausal status, and the presence of functional bowel disorders such as irritable bowel syndrome, all of which may influence constipation risk. These factors could introduce bias into our findings and should be considered in future research. Lastly, as our data were derived from the U.S. NHANES population, the generalizability of the results to other populations may be limited. Future studies should incorporate imaging-based assessments of body composition and adopt prospective designs to better elucidate the mechanisms linking RFM to constipation.

### 5. Conclusion

This study observed a nonlinear inverse association between RFM and constipation risk, suggesting that maintaining an appropriate level of visceral fat may be



associated with a reduced risk of constipation. These findings provide etiological clues for future research. However, due to the cross-sectional nature of the study, causal relationships cannot be inferred, and the underlying mechanisms remain unclear. Future large-scale, multicenter, prospective studies are needed to clarify the causal relationship between RFM and constipation.





### Declaration

### Ethics approval and consent to participate

Not applicable.

### **Consent for publication**

Not applicable.

### Availability of data and materials

The data and materials in the current study are available from the corresponding author on reasonable request.

## **Competing interests**

The authors declare that they have no potential conflicts of interest.

## Acknowledgements

Not applicable.

## **Funding**

Not applicable.

### **Authors' contributions**

LM, WXX and WW contributed to the study design. LM conducted the literature search. WXX and WW acquired the data. LM wrote the article and performed data analysis. WXX, WW revised the article and gave the final approval of the version to be submitted. All authors read and approved the final manuscript.

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Table 1 Baseline characteristics before and after propensity score matching

	Before PSM			After PSM			
Variables	Non- constipation (n=10174)	Constipation (n=1206)	p- value	Non- constipation (n=2360)	Constipation	p- value	SMD
Age (years)	49.20±17.63	46.53±18.14	<0.001	46.99±17.43	46.67±18.12	0.620	0.017
RFM	34.69±8.45	36.86±8.08	<0.001	37.68±8.35	36.81±8.08	0.003	0.106
PIR	2.67 ±1.62	2.26±1.56	<0.001	2.36±1.56	2.27±1.57	0.095	0.059
Sex, %			<0.001			0.525	0.024
Male	5460(53.7)	373(30.9)		710(30.1)	373 (31.2)		
Female	4714 (46.3)	833 (69.1)		1650 (69.9)	823 (68.8)		
Race, %			<0.001			0.592	0.059
Mexican American	1782 (17.5)	193 (16.0)		417 (17.7)	193 (16.1)		
Other Hispanic	770 (7.6)	115 (9.5)		196 (8.3)	115 (9.6)		
Non-Hispanic White	5270(51.8)	537 (44.5)		1045 (44.3)	536 (44.8)		

Non-Hispanic Black	1945(19.1)	319 (26.5)		614(26.0)	310 (25.9)		
Other Race	407 (4.0)	42 (3.5)		88 (3.7)	42 (3.5)		
Education level,			<0.001			0.228	0.061
Under high school	2562 (25.2)	374 (31.0)		672(28.5)	371 (31.0)		
High school	2408 (23.7)	339 (28.1)		657 (27.8)	333 (27.8)		
College graduate or above	5204 (51.1)	493 (40.9)		1031 (43.7)	492 (41.1)		
Alcohol consumption, %			<0.001			0.992	0.005
Yes	7538(74.1)	771(63.9)		1512(64.1)	769 (64.3)		
No	2636 (25.9)	435 (36.1)		848(35.9)	427 (35.7)		
Hypertension, %			0.007			0.256	0.042

Yes	3434 (33.8)	360 (29.9)		756 (32.0)	360(30.1)		
No	6740 (66.2)	846 (70.1)		1604(68.0)	836 (69.9)		
Diabetes, %			0.473			1.000	0.001
Yes	1148 (11.3)	145 (12.0)		285 (12.1)	144 (12.0)		
No	9026 (88.7)	1061 (88.0)		2075 (87.9)	1052 (88.0)		
Physical activity, %			<0.001			1.000	0.001
Yes	2512 (24.7)	242 (20.1)		475 (20.1)	240(20.1)		
No	7662 (75.3)	964 (79.9)		1885(79.9)	956 (79.9)		
Smoking, %			<0.001			0.531	0.023
Yes	5276 (51.9)	693 (57.5)		1381 (58.5)	686 (57.4)		
No	4898 (48.1)	513 (42.5)		979(41.5)	510 (42.6)		
Diet-related indi	cators						
Enorgy (kcal)	2161.78±1034.1	1898.16±89	<0.001	1921.94±901.49	1898.94±89	0.472	0.026
Energy (kcal)	4	8.64	\U.UU1	1521.541501.49	8.29	0.472	0.026
Protein (gm)	83.20±43.74	70.86±38.31	<0.001	72.04±36.75	71.01±38.33	0.436	0.027

Carbohydrate (gm)	260.42±129.78	239.84±115. 78	<0.001	241.35±121.95	239.57±115. 13	0.675	0.015
Total Sugar (gm)	117.92±81.07	117.05±79.2 0	0.726	117.21±79.90	116.55±78.1 7	0.813	0.008
Dietary Fiber (gm)	16.31±9.87	13.56±8.52	<0.001	13.77±8.13	13.61±8.53	0.590	0.019
Fat (gm)	81.16±47.45	70.46±42.95	<0.001	71.62±40.61	70.56±42.98	0.470	0.025
Cholesterol (mg)	300.06±247.88	256.81±214. 78	<0.001	261.81±220.07	257.55±215. 19	0.583	0.020
Niacin (mg)	25.33±15.08	21.38±13.66	<0.001	21.93±13.45	21.43±13.67	0.296	0.037
Phosphorus (mg)	1357.69±687.44	1163.28±602 .32	<0.001	1185.27±610.94	1165.70±60 3.06	0.365	0.032
Selenium (mcg)	112.38±63.87	95.06±53.91	<0.001	95.56±51.89	95.27±53.93	0.878	0.005

Note: For continuous variables: mean ± standard deviation, P-values were calculated using survey-weighted t-tests. For categorical variables: number (percentage), P-values were calculated using survey-weighted Chi-square tests.

PSM: Propensity Score Matching; SMD: Standardized Mean Difference; RFM: Relative Fat Mass; PIR: Poverty Income Ratio.

Table 2 Association between RFM and constipation before and after propensity score matching

	Model 1	Model 2	Model 3	
	OR(95%CI) p-value	OR(95%CI) p-value	OR(95%CI) p-value	
Before PSM				
RFM	0.976 (0.961, 0.991)	0.973 (0.958, 0.988)	0.971 (0.956, 0.986)	
KIIVI	0.005	0.002	0.001	
RFM quartile				
Q1	Reference	Reference	Reference	
7.98 - 28.79	Reference	Reference	Reference	
Q2	0.932 (0.709, 1.224)	0.911 (0.683, 1.215)	0.912 (0.680, 1.224)	
28.8 - 34.2	0.614	0.532	0.548	
Q3	0.928 (0.689, 1.251)	0.893 (0.662, 1.205)	0.878 (0.654, 1.178)	
34.21 - 41.77	0.629	0.467	0.398	
Q4	0.606 (0.425, 0.864)	0.571 (0.402, 0.813)	0.547 (0.385, 0.777)	
41.78 - 56.78	0.009	0.005	0.003	
p for trend	0.006	0.003	0.002	
After PSM				
RFM	0.975 (0.962, 0.987)	0.975 (0.962, 0.988)	0.975 (0.961, 0.988)	
IXIIVI	<0.001	<0.001	<0.001	
RFM quartile				
Q1	Reference	Reference	Reference	
10.85 - 31.38	Reference	Reference	Reference	
Q2	1.033 (0820, 1.302)	1.025 (0.813, 1.293)	1.028 (0.813, 1.300)	
31.39 - 38.26	0.782	0.832	0.815	
Q3	0.884 (0.668, 1.170)	0.878 (0.662, 1.163)	0.881 (0.663,1.170)	
38.27 - 44.12	0.387	0.363	0.380	
Q4	0.619 (0.462, 0.829)	0.615 (0.459, 0.826)	0.616 (0.455, 0.832)	
44.13 - 54.66	0.001	0.001	0.002	
	<0.001	<0.001		

Note: OR: Odds Ratio; 95%CI: 95% Confidence Interval; RFM: Relative Fat Mass;

PSM: Propensity Score Matching.

Model 1 was adjusted for age, sex, race, education level, and PIR;

Model 2 was further adjusted for dietary intake variables based on Model 1, including phosphorus, selenium, niacin, protein, fat, carbohydrates, dietary fiber, total energy, total sugar, and cholesterol;

Model 3 was adjusted for all covariates.

Table 3 Threshold effect analysis

	OR (95%CI)	p-value
Fitting by the standard linear model	0.971 (0.960, 0.983)	<0.001
Fitting by the two-piecewise linear model		
The inflection point of RFM	36.06	
< 36.06	0.998 (0.978, 1.017)	0.801
> 36.06	0.950 (0.933, 0.966)	<0.001
Log-likelihood ratio		<0.001

Note: OR: Odds Ratio; 95%CI: 95% Confidence Interval; RFM: Relative Fat Mass.

Table 4 Subgroup analysis of the association between RFM and constipation

	OR(95%CI) p-value	p for interaction
Sex		0.366

Male	0.980 (0.960, 1.001) 0.076	
Female	0.967 (0.949, 0.986) 0.003	
Age		0.023
≤ 45 years	0.977 (0.962, 0.993) 0.010	
> 45 years	0.956 (0.936, 0.976) < 0.001	
Smoking		0.285
Yes	0.976 (0.960, 0.992) 0.008	
No	0.967 (0.950, 0.985) 0.002	
Physical activity		0.251
Yes	0.980 (0.962, 0.999) 0.049	
No	0.968 (0.951, 0.985) 0.002	
Hypertension		0.047
Yes	0.956 (0.935, 0.977) < 0.001	
No	0.975 (0.960, 0.990) 0.005	
Diabetes		0.810
Yes	0.967 (0.934, 1.002) 0.078	
No	0.971 (0.956, 0.986) 0.001	
Drink		0.029
Yes	0.978 (0.961, 0.996) 0.026	
No	0.957 (0.940, 0.974) <0.001	

Note:All confounding factors, except for the stratifying factor, were adjusted for all other

variables.

OR: Odds Ratio; 95%CI: 95% Confidence Interval; RFM: Relative Fat Mass.

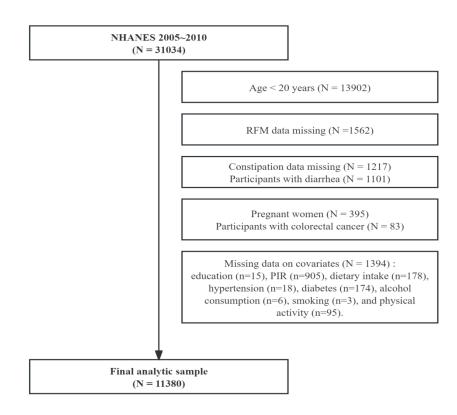


Figure 1. Flowdiagram of participant screening.

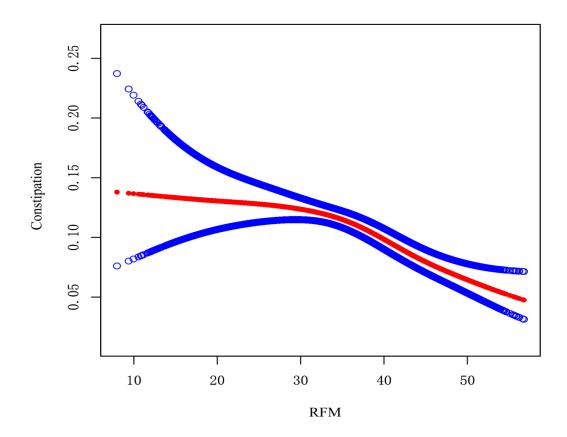


Figure 2. Smoothed curve derived from a multivariable logistic regression model using generalized additive modeling, illustrating the nonlinear association between Relative Fat Mass and the risk of constipation.

Note: All covariates in the fully adjusted model were controlled for. The red line indicates the fitted curve, and the blue-shaded area represents the 95% confidence interval.