Lin *et al. Lipids in Health and Disease* (2025) 24:164
https://doi.org/10.1186/s12944-025-02570-1

Lipids in Health and Disease

RESEARCH Open Access

# The association between the intake of polyunsaturated fatty acids and chronic constipation and diarrhea: NHANES 2005–2010

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## Abstract

**Background** Polyunsaturated fatty acids (PUFAs) have shown notable protective effects in various diseases. This study aims to investigate the associations between PUFAs intake and both chronic constipation (CC) and chronic diarrhea (CD).

**Methods** Data from three survey cycles (2005–2006, 2007–2008, and 2009–2010) of the National Health and Nutrition Examination Survey (NHANES) were used for analysis. 24-hour dietary recall interviews were employed to evaluate PUFAs intake. The associations between PUFAs intake and both CC and CD were analyzed via multivariable weighted logistic regression (WLR). Furthermore, stratified analysis and restricted cubic splines analysis were carried out.

**Results** 7723 participants were included, among whom 545 (8.35%) were CC patients and 579 (7.50%) were CD patients. According to the results of multivariable MLR, a nonlinear association between PUFAs intake and CC was found (P for nonlinear < 0.05), where the increased daily intake was related to a declined prevalence of CC [OR = 0.976 (0.959, 0.993), $P = 0.007$] in the fully adjusted model. However, there was no significant evidence of an association between PUFAs intake and CD ($P > 0.05$).

**Conclusion** PUFAs intake was negatively associated with CC and was not strongly associated with CD. It suggested that adjusting daily PUFAs intake may alleviate CC symptoms.

**Trial registration** Not applicable.

**Keywords** Polyunsaturated fatty acids, Chronic constipation, Chronic diarrhea, NHANES

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## Background

Chronic constipation (CC) and chronic diarrhea (CD) stand as two common gastrointestinal disorders, with prevalence rates up to 17% and 20% [1, 2], respectively. These conditions greatly impact the patient's life and cause higher healthcare expenses. CD is marked by more than 25% watery or loose stools, while CC features less than three bowel movements within a week, with stools being lumpy, hard, or dry. CC and CD are influenced by such factors as gender, age, lifestyle, physical condition, and diet. Although their etiologies are complicated, diet is considered to be related to the development and progression of these gastrointestinal disorders.

Numerous studies have highlighted the notable impact of dietary fat on gut function. Most importantly, different types of fatty acids (FAs) may affect the development and progression of CC and CD through various mechanisms [3]. Saturated FAs are closely linked to inflammation, insulin resistance, and metabolic disorders, potentially exacerbating gut symptoms. In contrast, PUFAs have anti-inflammatory effects and act as protective factors in gut health [4]. Although previous studies investigated the association between dietary fat intake and gut health, the specific effect of PUFAs intake on CC and CD remains unestablished.

This study aimed to assess the associations between PUFAs intake and both CC and CD using data from the National Health and Nutrition Examination Survey (NHANES) database, providing new insights into the effect of PUFAs on gut health. The findings may also offer a theoretical basis for future dietary interventions.

## Methods

### Data source and sample selection

Data from three survey cycles (2005–2006, 2007–2008, and 2009–2010) of NHANES were used for analysis. The NHANES database serves as a cross-sectional survey carried out annually by the National Center for Health Statistics at the Centers for Disease Control and Prevention in the United States, with data released biennially. Participants were randomly screened via a stratified, multistage cluster sampling design, and information was collected through interviews. The objective of NHANES is to help develop comprehensive public health policies and promote health education across a broad population.

Before becoming involved in the survey, participants had received detailed explanations about the nature and purpose of this survey and signed informed consent. The Institutional Review Board of the National Center for Health Statistics approved this survey. After anonymization, the data were made public to optimize resource utilization. This study required no additional approval, as it solely utilized publicly available data.

This cross-sectional study investigated participants in NHANES from 2005 to 2010. Firstly, as the study aimed to investigate adults, individuals younger than 20 years were excluded. Additionally, participants with inflammatory bowel disease were excluded, as this disease itself can cause diarrhea and constipation symptoms. Furthermore, participants with insufficient data on PUFAs intake or those lacking critical variables, such as missing information on gut health questionnaires or bowel movement data, were excluded. Lastly, variables with missing values were also excluded. Figure 1 illustrates the participant screening process.

### Study variables

#### *Measurement of PUFAs*

24-hour dietary recall interviews were carried out to collect dietary data. The method modified by Lagerstedt et al. was employed to determine the content of dietary FAs [5]. This study selected seven different types of PUFAs for each participant: linoleic acid (LA, g), docosahexaenoic acid (DHA, g), eicosapentaenoic acid (EPA, g), arachidonic acid (ARA, g), stearidonic acid (SDA, g), alpha-linolenic acid (ALA, g), and docosapentaenoic acid (DPA, g). The intake of omega-3 and omega-6 was calculated respectively. Omega-3 included ALA, SDA, EPA, DHA, and DPA, while omega-6 included LA and ARA. The total polyunsaturated fatty acid (TPFA) intake was then calculated by summing the omega-3 and omega-6 intakes [5].

#### *Gut health questionnaire*

Participants were classified as having CD or CC according to their answers to the gut health questionnaire. A color-coded card with the Bristol Stool Form Scale (BSFS) (types 1–7) was shown to participants to help them identify their stool type [6]. The results were aligned with previous studies. Participants whose most common stool type was BSFS type 1 (separate hard lumps, like nuts) or type 2 (sausage-shaped but lumpy) were classified as having CC. Participants with stool types BSFS 6 (fluffy, with ragged edges, mushy) or BSFS 7 (watery, no solid pieces) were defined as having CD. Other participants were classified as having normal bowel function.

#### *Covariables*

Confounding factors related to CD and CC were adjusted. The covariables encompassed gender, race, age, ethnicity (non-Hispanic White, non-Hispanic Black, other Hispanic, Mexican American, other race including multi-racial), marital status (currently married, previously married, single), educational background (less than high school, high school graduate, post-high school education), alcohol consumption (never drank, currently drinks, former drinker), family poverty-to-income

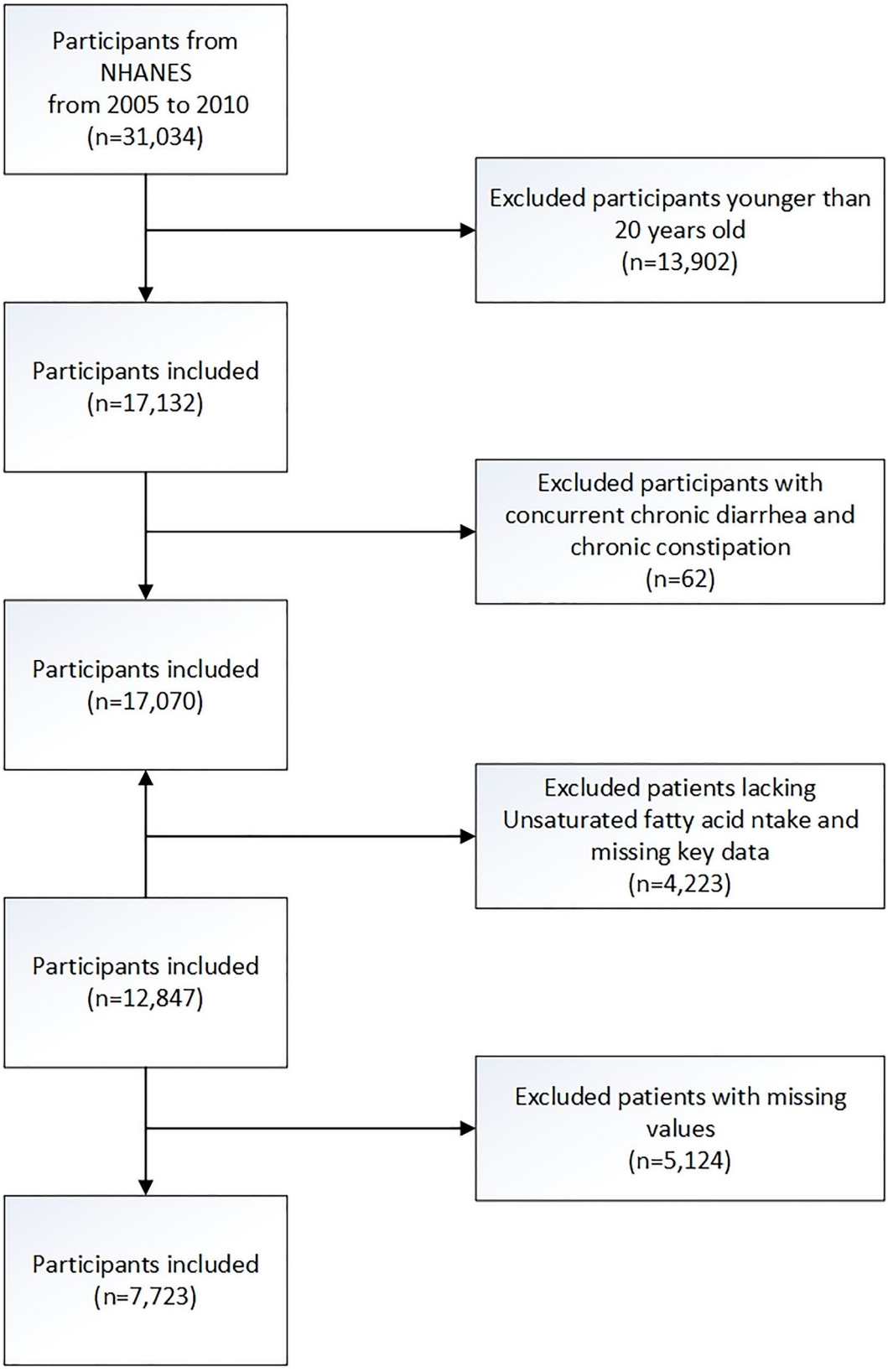


**Fig. 1** Flowchart of participant inclusion

ratio (PIR ≤ 1.3, 1.31–3.5, ≥ 3.5), smoking status (never smoked, currently smokes, former smoker), body mass index (BMI < 25, 25–30, > 30), and vigorous physical activity (yes, no). Comorbidities included hypertension, hyperlipidemia, diabetes, and depression. Hypertension, hyperlipidemia, and diabetes were diagnosed based on self-reported diagnoses. Depression was diagnosed using the PHQ-9 scale, one of the most commonly used tools for screening depression [7]. This scale contained nine items scored from 0 to 3, and the total score was used for assessment.

### Statistical analysis

Means ± standard deviations (SD) were used to represent normally distributed continuous variables, while those not following a normal distribution were expressed as medians and interquartile ranges [M(Q1, Q3)]. T-tests or Kruskal-Wallis H tests were used for group comparisons. Frequencies and percentages [n (%)] were used to express categorical variables, and chi-square ($\chi 2$) or Fisher's exact tests were employed for group comparisons. The variance inflation factor was calculated to assess multicollinearity among variables and those with high multicollinearity were excluded. Additionally, the association between PUFAs intake and gut health (CD and CC) was evaluated via multivariable weighted logistic regression (WLR) models. Three models were constructed: an unadjusted model (Model 1), a model adjusted for basic information (family PIR, race, gender, age) (Model 2), and a fully adjusted model (age, gender, race, family PIR, alcohol consumption, smoking status, BMI, vigorous physical activity) (Model 3). The non-linear association between exposure and outcome was analyzed via Restricted Cubic Splines (RCS). To assess the robustness of the association between PUFAs intake and CC, and to explore potential interactions between variables, stratified analyses were carried out based on age, gender, BMI, hypertension, and diabetes. R software (version 4.4.1) was used to perform statistical analyses, and a *p*-value below 0.05 indicated statistical significance.

## Results

### Baseline characteristics of the study population

A total of 7723 individuals were selected. The characteristics of the study population are illustrated in Table 1. Among individuals with CC, 25% were male and 75% were female, with a median age of 49 years. Among those with CD, 39% were male and 61% were female, with a median age of 50 years. The overall prevalence of CD and CC among U.S. adults was 7.49% and 7.06%, respectively. Individuals with CC were more likely to be female, non-Hispanic White, and physically inactive ($P < 0.05$). There were no significant differences in age, omega-3 to omega-6 ratio, hypertension, or hyperlipidemia ($P > 0.05$). Individuals with CC were more likely to be female, non-Hispanic White, and physically inactive ($P < 0.05$). There was no evidence of significant differences in age, omega-3 to omega-6 ratio, hypertension, or hyperlipidemia ($P > 0.05$).

### Association between dietary PUFAs intake and chronic constipation and diarrhea

#### *Association between dietary PUFAs intake and chronic constipation*

WLR models were used to analyze the association between dietary PUFAs intake and CC, with confounding factors adjusted. Model 1, Model 2, and Model 3 were used for analysis. The association between dietary PUFAs intake and CC in the three models is illustrated in Table 2. Model 1 (OR = 0.961, 95% CI: 0.945, 0.977, $P < 0.001$), Model 2 (OR = 0.975, 95% CI: 0.959, 0.992, $P = 0.004$), and Model 3 (OR = 0.976, 95% CI: 0.959, 0.993, $P = 0.007$) suggested a significant association between PUFAs intake and CC. These results indicated that higher PUFAs intake was associated with a lower prevalence of CC, suggesting PUFAs potentially play a protective role. Significant associations were also found between CC and omega-3 (Model 1: OR = 0.716, 95% CI: 0.596, 0.861, $P < 0.001$; Model 2: OR = 0.815, 95% CI: 0.684, 0.971, $P = 0.023$; Model 3: OR = 0.817, 95% CI: 0.686, 0.973, $P = 0.007$) and omega-6 (Model 1: OR = 0.957, 95% CI: 0.940, 0.975, $P < 0.001$; Model 2: OR = 0.973, 95% CI: 0.956, 0.991, $P = 0.004$; Model 3: OR = 0.974, 95% CI: 0.956, 0.992, $P = 0.007$). These results revealed that higher omega-3 and omega-6 intake was associated with a lower prevalence of CC, suggesting their protective roles in reducing CC risk. However, there was no evidence of a significant association between the omega-3 to omega-6 ratio and CC ($P > 0.05$).

RCS analysis was carried out to investigate the non-linear relationship between exposure and outcome, with the number of knots set to five. As shown in Fig. 2, there was no significant evidence of a non-linear association between total PUFAs intake and CC (non-linear $P > 0.05$). Increased PUFAs intake can reduce the prevalence of CC, suggesting PUFAs potentially play a protective role. Similarly, there was no significant evidence of a non-linear association between omega-6 intake and CC (non-linear $P > 0.05$), confirming it as a potential protective factor. However, there was no evidence of a non-linear association between omega-3 intake and CC (omega-3 non-linear $P = 0.035$). The results indicated that the prevalence of CC significantly decreased when omega-3 intake exceeded 1.4 g ($P = 0.035$), suggesting a potential threshold effect.

**Table 1** Baseline information table

| Characteristic | Overall | Chronic constipation | Chronic diarrhea | None | *P*-value |
|---|---|---|---|---|---|
| | *N* = 7723 | *N* = 545 | *N* = 579 | *N* = 6599 | |
| Gender | | | | | < 0.001 |
| Female | 4093(53%) | 404(75%) | 333(61%) | 3356(51%) | |
| Male | 3630(47%) | 141(25%) | 246(39%) | 3243(49%) | |
| Age | 49(39,60) | 49(37,61) | 50(39,62) | 49(39,60) | 0.381 |
| Race | | | | | 0.013 |
| Mexican American | 1057(5.4%) | 61(4.8%) | 91(5.6%) | 905(5.4%) | |
| Other Hispanic | 569(3.4%) | 51(4.0%) | 47(4.6%) | 471(3.2%) | |
| Non-Hispanic White | 4262(77%) | 281(71%) | 295(75%) | 3686(77%) | |
| Non-Hispanic Black | 1552(10.0%) | 139(16%) | 125(11%) | 1288(9.5%) | |
| Other Race - Including multi-racial | 283(4.8%) | 13(3.9%) | 21(3.7%) | 249(4.9%) | |
| Income | | | | | 0.001 |
| Not poor | 3037(52%) | 165(42%) | 178(46%) | 2694(53%) | |
| Near poor | 1810(15%) | 149(18%) | 184(20%) | 1477(14%) | |
| Poor | 2876(33%) | 231(40%) | 217(34%) | 2428(33%) | |
| Octadecadienoic | 14(10,20) | 12(8,17) | 13(9,20) | 14(10,20) | < 0.001 |
| Octadecatrienoic | 1.34(0.91,1.96) | 1.12(0.81,1.63) | 1.22(0.86,1.99) | 1.36(0.94,1.97) | < 0.001 |
| Octadecatetraenoic | 0.001(0.000,0.013) | 0.001(0.000,0.011) | 0.001(0.000,0.013) | 0.001(0.000,0.013) | 0.618 |
| Eicosatetraenoic | 0.12(0.07,0.19) | 0.10(0.07,0.15) | 0.12(0.07,0.20) | 0.12(0.07,0.19) | < 0.001 |
| Eicosapentaenoic | 0.01(0.00,0.03) | 0.01(0.00,0.02) | 0.01(0.00,0.03) | 0.01(0.00,0.03) | < 0.001 |
| Docosapentaenoic | 0.012(0.004,0.025) | 0.009(0.002,0.019) | 0.013(0.003,0.028) | 0.013(0.004,0.025) | 0.004 |
| Docosahexaenoic | 0.03(0.01,0.08) | 0.02(0.01,0.06) | 0.04(0.01,0.10) | 0.03(0.01,0.08) | 0.002 |
| Omega 3 | 1.47(1.00,2.17) | 1.25(0.85,1.79) | 1.36(0.93,2.20) | 1.50(1.02,2.19) | < 0.001 |
| Omega 6 | 14(10,20) | 12(8,17) | 13(9,20) | 14(10,20) | < 0.001 |
| Total PUFAs | 15(11,22) | 13(9,19) | 15(10,23) | 16(11,22) | < 0.001 |
| Omega3/omega6 | 0.11(0.09,0.13) | 0.11(0.09,0.13) | 0.11(0.09,0.13) | 0.11(0.09,0.13) | 0.795 |
| Frequency of defecation | 7(7,14) | 7(4,7) | 14(7,14) | 7(7,14) | < 0.001 |
| BMI | | | | | < 0.001 |
| Obese | 3183(39%) | 203(31%) | 295(50%) | 2685(39%) | |
| Overweight | 2609(33%) | 169(33%) | 175(27%) | 2265(34%) | |
| Under or normal weight | 1931(28%) | 173(37%) | 109(22%) | 1649(27%) | |
| Smoke | | | | | 0.018 |
| Former | 2217(27%) | 129(23%) | 187(31%) | 1901(27%) | |
| Never | 4100(54%) | 318(59%) | 269(46%) | 3513(55%) | |
| Now | 1406(18%) | 98(17%) | 123(23%) | 1185(18%) | |
| Drink | | | | | 0.002 |
| Former | 1246(14%) | 108(18%) | 102(18%) | 1036(13%) | |
| Never | 965(9.8%) | 94(14%) | 89(9.7%) | 782(9.5%) | |
| Now | 5512(76%) | 343(68%) | 388(72%) | 4781(77%) | |
| Diabetes | | | | | < 0.001 |
| No | 6655(91%) | 466(91%) | 453(84%) | 5736(91%) | |
| Yes | 1068(9.3%) | 79(9.5%) | 126(16%) | 863(8.8%) | |
| Hypertension | | | | | 0.071 |
| No | 4561(65%) | 338(67%) | 280(58%) | 3943(65%) | |
| Yes | 3162(35%) | 207(33%) | 299(42%) | 2656(35%) | |
| Hyperlipidemia | | | | | 0.132 |
| No | 4408(59%) | 313(62%) | 296(54%) | 3799(59%) | |
| Yes | 3315(41%) | 232(38%) | 283(46%) | 2800(41%) | |
| Physical activity | | | | | 0.017 |
| No | 6030(75%) | 450(82%) | 469(78%) | 5111(74%) | |
| Yes | 1693(25%) | 95(18%) | 110(22%) | 1488(26%) | |
| Depression | | | | | < 0.001 |
| No | 7176(94%) | 494(90%) | 491(88%) | 6191(95%) | |
| Yes | 547(6.0%) | 51(9.9%) | 88(12%) | 408(5.2%) | |

**Table 2** Relationship between polyunsaturated fatty acids and chronic constipation and diarrhea

| Variables | Model 1 | | Model 2 | | Model 3 | |
|---|---|---|---|---|---|---|
| | OR(95%CI) | *P* value | OR(95%CI) | *P* value | OR(95%CI) | *P* value |
| Chronic constipation | | | | | | |
| Total PUFAs | | | | | | |
| Continuous | 0.961( 0.945, 0.977) | < 0.001 | 0.975( 0.959, 0.992) | 0.004 | 0.976( 0.959, 0.993) | 0.007 |
| T1 | - | | - | | - | |
| T2 | 0.634(0.466, 0.863) | 0.005 | 0.718 (0.524, 0.982) | 0.039 | 0.700(0.511, 0.958) | 0.027 |
| T3 | 0.513( 0.392, 0.672) | < 0.001 | 0.686 (0.523, 0.899) | 0.008 | 0.684(0.515, 0.909) | 0.011 |
| Omega 3 | | | | | | |
| Continuous | 0.716(0.596, 0.861) | < 0.001 | 0.815(0.684, 0.971) | 0.023 | 0.817(0.686, 0.973) | 0.025 |
| T1 | - | | - | | - | |
| T2 | 0.638(0.466, 0.872) | 0.006 | 0.720(0.529, 0.981) | 0.038 | 0.711(0.525, 0.962) | 0.029 |
| T3 | 0.461(0.334, 0.637) | < 0.001 | 0.596(0.430, 0.827) | 0.003 | 0.594(0.427, 0.826) | 0.003 |
| Omega 6 | | | | | | |
| Continuous | 0.957(0.940, 0.975) | < 0.001 | 0.973(0.956, 0.991) | 0.004 | 0.974(0.956, 0.992) | 0.007 |
| T1 | - | | - | | - | |
| T2 | 0.656(0.487, 0.883) | 0.006 | 0.740(0.546, 1.003) | 0.052 | 0.726(0.533, 0.988) | 0.042 |
| T3 | 0.514(0.392, 0.674) | < 0.001 | 0.682(0.518, 0.898) | 0.008 | 0.682(0.511, 0.911) | 0.011 |
| Omega3/omega6 | | | | | | |
| Continuous | 1.056(0.119, 9.382) | 0.96 | 0.813(0.089, 7.424) | 0.851 | 0.676(0.073, 6.267) | 0.721 |
| T1 | - | | - | | - | |
| T2 | 0.951(0.699, 1.294) | 0.744 | 0.941(0.700, 1.265) | 0.68 | 0.970(0.721, 1.305) | 0.834 |
| T3 | 1.069(0.800, 1.428) | 0.646 | 1.029(0.783, 1.352) | 0.835 | 1.016(0.772, 1.338) | 0.905 |
| Chronic diarrhea | | | | | | |
| Total PUFAs | | | | | | |
| Continuous | 0.999(0.986, 1.012) | 0.867 | 1.007(0.995, 1.020) | 0.229 | 1.008(0.996, 1.021) | 0.187 |
| T1 | - | | - | | - | |
| T2 | 0.774(0.566, 1.059) | 0.106 | 0.845(0.615, 1.162) | 0.291 | 0.888(0.636, 1.238) | 0.469 |
| T3 | 0.884(0.655, 1.193) | 0.412 | 1.063(0.795, 1.422) | 0.673 | 1.114(0.814, 1.525) | 0.486 |
| Omega 3 | | | | | | |
| Continuous | 0.967(0.844, 1.109) | 0.626 | 1.023(0.901, 1.162) | 0.715 | 1.043(0.912, 1.192) | 0.523 |
| T1 | - | | - | | - | |
| T2 | 0.746(0.568, 0.979) | 0.035 | 0.803(0.610, 1.057) | 0.115 | 0.830(0.621, 1.110) | 0.199 |
| T3 | 0.900(0.663, 1.222) | 0.49 | 1.046(0.769, 1.421) | 0.77 | 1.088(0.783, 1.512) | 0.603 |
| Omega 6 | | | | | | |
| Continuous | 0.999(0.985, 1.014) | 0.906 | 1.009(0.995, 1.022) | 0.203 | 1.010(0.996, 1.024) | 0.173 |
| T1 | - | | - | | - | |
| T2 | 0.733(0.543, 0.989) | 0.042 | 0.803(0.593, 1.087) | 0.151 | 0.840(0.609, 1.157) | 0.273 |
| T3 | 0.926(0.678, 1.265) | 0.624 | 1.122(0.826, 1.526) | 0.451 | 1.171(0.841, 1.630) | 0.335 |
| Omega3/omega6 | | | | | | |
| Continuous | 0.981(0.040, 23.79) | 0.991 | 0.711(0.024, 20.75) | 0.839 | 1.005(0.032, 31.14) | 0.998 |
| T1 | - | | - | | - | |
| T2 | 1.033(0.756, 1.410) | 0.835 | 1.014(0.742, 1.385) | 0.931 | 1.015(0.730, 1.412) | 0.927 |
| T3 | 0.970(0.700, 1.345) | 0.853 | 0.921(0.662, 1.282) | 0.618 | 0.952(0.677, 1.338) | 0.767 |

Model 1: unadjusted

Model 2: adjusted for gender, age, race, income

Model 3: adjusted for gender, age, race, income, drink, smoke, BMI, physical activity

#### *Association between dietary PUFA intake and chronic diarrhea*

WLR models were employed to explore the association between dietary PUFAs intake and CD, with confounding factors adjusted. Model 1, Model 2, and Model 3 were used for analysis. Table 2 illustrates the association between dietary PUFAs intake and CD in the three models. However, there was no evidence of significant associations between CD and PUFAs, omega-3, or omega-6 intake in any model ($P > 0.05$).

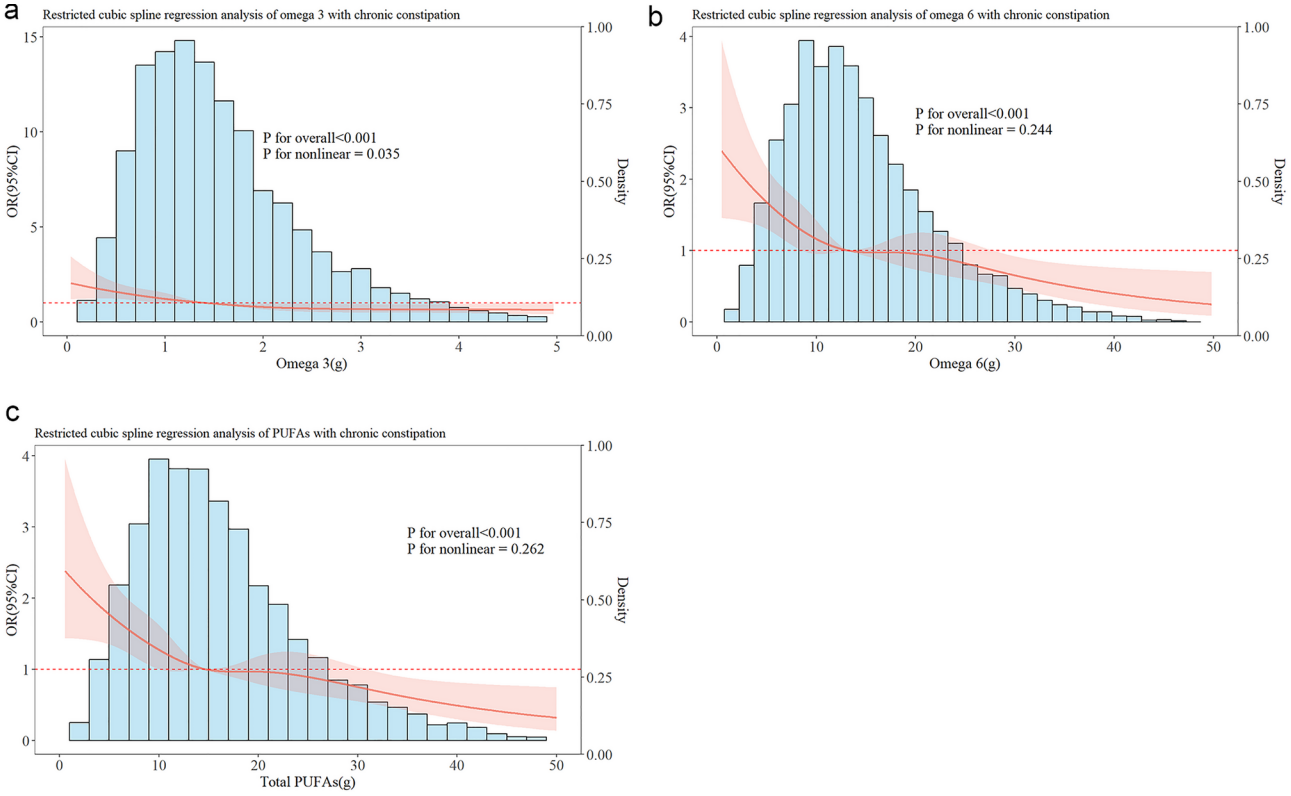


**Fig. 2** Results of RCS analysis of PUFAs, Omega 6, Omega 3 and chronic constipation; (**a**) Restricted Cubic Splines regression analysis of Omega 3 with chronic constipation; (**b**) Restricted Cubic Splines regression analysis of Omega 6 with chronic constipation; (**c**) Restricted Cubic Splines regression analysis of PUFAs with chronic constipation

### *Association between specific PUFAs and chronic constipation or diarrhea*

The associations between specific dietary FA (LA, ALA, SDA, ARA, EPA, DPA, and DHA) and both CD and CC across the three adjusted models are illustrated in Table S1. The results revealed that, in all adjusted models, only ALA and LA were related to a reduced prevalence of CC (OR < 1, $P < 0.05$). However, there was no evidence of significant associations between CD and any of the seven FAs ($P > 0.05$).

### Stratified analysis

As shown in Fig. 3, stratified analysis was carried out based on hypertension, gender, age, diabetes, and BMI. The results revealed that the association between exposure and outcome in different subgroups was consistent with that of the total population, with higher PUFAs intake being related to a lower prevalence of CC. This association was more pronounced in individuals with obesity, those under 50 years old, patients with diabetes, and those with hypertension. However, this association was not significant among males, females, overweight individuals, those with underweight or normal weight, individuals aged 50 and above, as well as those without hypertension or diabetes. No significant interaction effects were found (P for interaction > 0.05).

### Sensitivity analysis

Based on the fully adjusted model, dietary fiber intake was further included to verify the robustness of the association between PUFAs intake and CC. The results indicated that total PUFAs intake remained significantly associated with CC (Table S2). Specifically, higher intake of PUFAs was associated with a lower prevalence of CC (OR = 0.978, 95% CI: 0.961, 0.995, $P = 0.012$), suggesting the protective role of PUFAs. The results also indicated significant associations between omega-3 and CC, as well as between omega-6 and CC. In model 4, omega-3 was associated with a reduced prevalence of CC (OR = 0.833, 95% CI: 0.706, 0.933, $P = 0.032$), as was omega-6 (OR = 0.976, 95% CI: 0.958, 0.995, $P = 0.014$). However, there was no evidence of a significant association between the ratio of omega-3 to omega-6 intake and CC ($P > 0.05$).

## Discussion

Based on a nationally representative sample cohort of the U.S. population, this cross-sectional study involving 7723 adults revealed a significant negative association

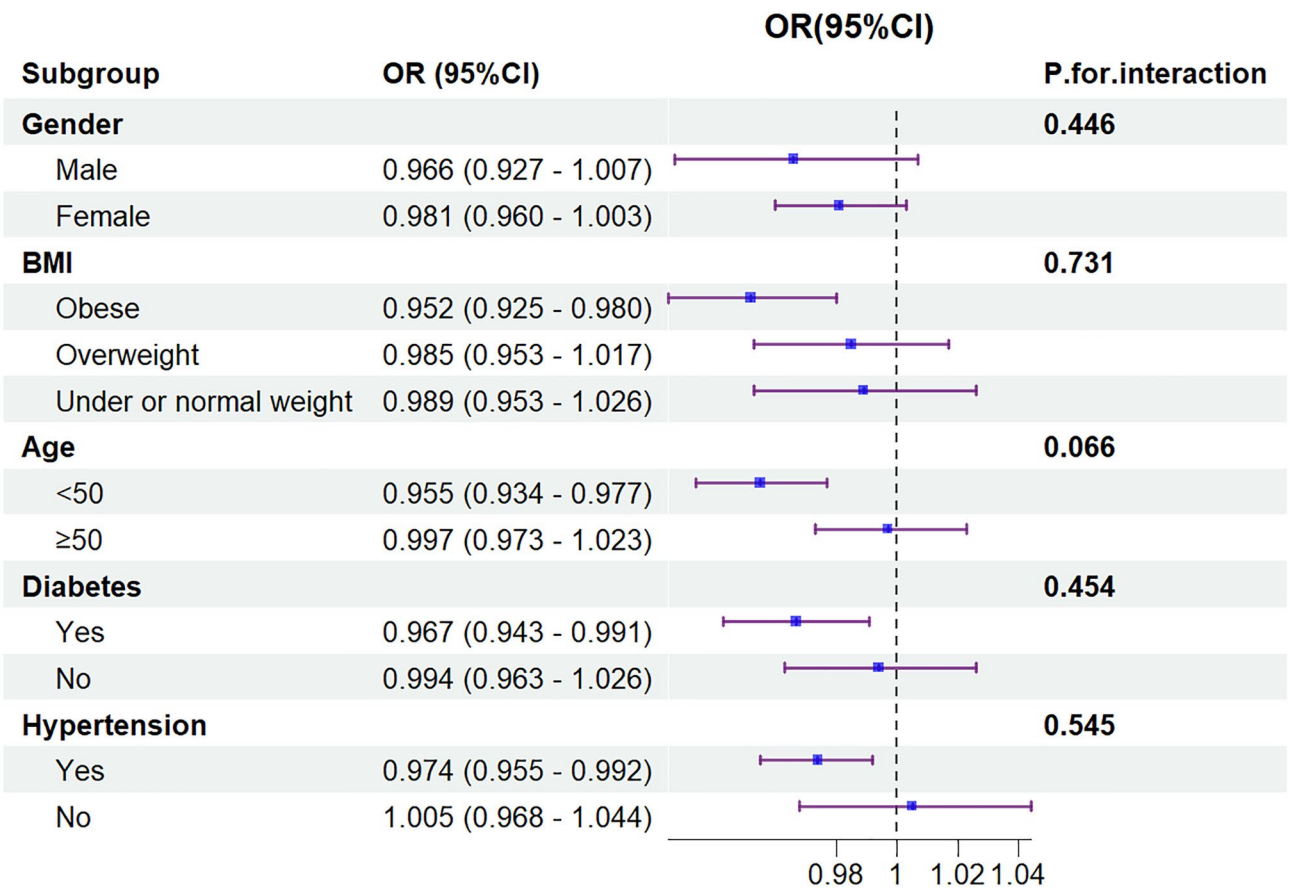


**Fig. 3** Subgroup analysis of PUFAs intake and chronic constipation (forest plot)

between dietary PUFAs intake and CC. Both omega-3 and omega-6 intake were protective factors for CC. After adjusting for all confounding variables (race, sex, age, educational background, BMI, income, hypertension, smoking status, diabetes, and alcohol consumption), this association remained statistically significant. Stratified analysis based on different variables indicated that this negative association was consistent across various groups, suggesting that this association can apply to diverse populations. However, there was no evidence of significant associations between PUFAs, omega-3 and omega-6 intake, and CD.

It is noteworthy that this study suggests the protective effects of PUFAs on CC. Several hypotheses can explain this protective role. First, previous studies have revealed that there were notable differences in gut microbiota composition between CC patients and healthy individuals. Compared with healthy individuals, constipation patients have lower gut microbial diversity, with a marked decrease in Bifidobacteria and Lactobacilli, and an increase in the abundance of Desulfovibrio [8]. Previous studies indicated that PUFAs might regulate immune and inflammatory responses through modulating the gut microbiome [9, 10]. The metabolism and absorption of n-3 PUFAs can be impacted by gut microbiota. Conversely, n-3 PUFAs can influence the diversity and abundance of gut microbiota [11]. N-3 PUFAs are beneficial for the gut microbiome as they can reduce the production of Capitalize Enterobacteriaceae and promote the growth of bifidobacteria. Notably, they help to support normal gut function, regulate gut motility, and increase bowel movement frequency, thereby alleviating constipation symptoms [12, 13]. Based on our findings, the impact of PUFAs (n-3 and n-6 PUFAs) intake on CC was potentially associated with the diversity and composition of gut microbiota. Further prospective studies are required to validate our findings. However, our results revealed that there was no significant association between PUFAs intake and CD, which contradicts previous findings [14]. This may be related to the asymmetrical effects of PUFAs on gastrointestinal motility. PUFAs can improve gut permeability and motility by reducing inflammation or affecting the composition of the gut microbiota, thereby reducing constipation [14, 15]. Nevertheless, the mechanisms underlying CD are more complex, involving infectious, inflammatory diseases, or impaired gut barriers. Hence, the role of PUFAs may not be sufficient to impact

these factors, which could explain the lack of a significant association [16, 17].

Additionally, recent studies revealed changes in the structure and function of the intestinal mucosal barrier in CC patients [18]. On one hand, constipation patients exhibit abnormal gut motility, leading to prolonged retention of harmful microbes in the gut, which directly or indirectly damages the intestinal mucosal barrier [19]. On the other hand, constipation is linked to immune system activation, and low-grade inflammation in the intestinal mucosa. Activated immune cells release inflammatory cytokines and neurotransmitters, causing disrupted mucosal barrier and abnormalities in gut sensation and motility [20]. Recent clinical studies have demonstrated that n-3 PUFAs intake can notably enhance intestinal barrier integrity [4]. Specifically, n-3 PUFAs can impact tight junction proteins, and n-3 long-chain PUFAs can enhance intestinal barrier stability by increasing the expression of occludin and ZO-1 in cell membranes, and reducing cellular degeneration [21]. Moreover, n-3 long-chain PUFAs can act as anti-inflammatory agents and increase tight junction stability by activating G protein-coupled receptor 120 [22]. These findings are aligned with the results of our study, suggesting the protective role of PUFAs against CC. Moreover, increased PUFAs intake may reduce the prevalence of CC, possibly by improving the intestinal mucosal barrier. The underlying mechanisms need to be explored in future studies.

This study utilized a nationally representative sample, enhancing the generalizability of the findings. Moreover, the large sample size ensured greater reliability and broader applicability of the results. Furthermore, the study explored the associations between PUFAs and both CC and CD, as well as provided evidence for the association between daily PUFAs intake and digestive symptoms, offering a basis for potential dietary interventions to manage CC and CD. It also presented new insights into the possible mechanisms underlying these conditions.

However, this study has certain limitations. Firstly, only two 24-hour dietary recalls were included in the NHANES database, rather than three (two working days and one Friday), leading to potential recall bias. To minimize the impact of this bias, sampling weights and multiple imputation methods were applied to ensure the accuracy of dietary intake in the NHANES design, and this study included participants with two valid 24-hour dietary recalls. Secondly, since the NHANES database was a cross-sectional study, causal relationships are not established. Thirdly, other unmeasured confounders, such as related chronic diseases, that could potentially influence the results are not ruled out. Finally, since the database primarily focuses on public nutrition, the data on CD and CC only cover the years 2005–2010 and have not been updated.

## Conclusion

Our findings suggested that PUFAs intake was negatively associated with CC, and increasing daily intake may reduce the prevalence of CC. However, there was no significant association between PUFAs intake and CD. Therefore, adjusting PUFAs intake may relieve CC symptoms. This study highlights the importance of dietary interventions in disease management, and the intake of PUFAs can serve as a new option for front-line clinicians to manage CC.

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12944-025-02570-1.

**Supplementary Material 1**: **Table S1**: Relationship between polyunsaturated fatty acids and chronic constipation and diarrhea (different components).

**Supplementary Material 2**: **Table S2**: Sensitivity analysis.

### Author contributions

Ping Lin: Conceptualization, Methodology, Software, Writing- Original draft preparation. Wei Wang: Data curation. Yun Zhou: Visualization, Investigation. Yong Yang: Supervision. Software, Validation. Ping Liu: Writing- Reviewing and Editing. All authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

### Funding

This research was supported by a Molecular network of metatherian regulating metabolism in hepatocellular carcinoma based on post-translational modification integrated protein technology (AHWJ2023BAa20083).

### Data availability

All data generated or analysed during this study are included in this published article.

## Declarations

### Ethics approval and consent to participate

Not applicable.

### Consent for publication

Not applicable.

### Competing interests

The authors declare no competing interests.

Received: 6 March 2025 / Accepted: 16 April 2025
Published online: 06 May 2025

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