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Association of dietary fat type with abdominal bloating: a case control study

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Abstract

Background Abdominal bloating is a common gastrointestinal complaint, especially among women, and can significantly affect quality of life. Despite its high prevalence, the role of dietary fat intake in the development or exacerbation of bloating remains poorly characterized. This study aimed to examine the association between abdominal bloating and the consumption of various types of dietary fats among Iranian women.

Methods A case-control study was performed on 229 women with bloating and 224 women without bloating in Tehran, Iran. Dietary intake was assessed using a validated 168-item food frequency questionnaire (FFQ), and nutrient analysis was conducted using Nutritionist IV software. A binary logistic regression method, adjusted for confounders was applied to assess the link between bloating and the intake of fats.

Results An inverse association was found between abdominal bloating and omega-3 PUFA intake (OR = 0.34, 95% CI: 0.13–0.87, $p = 0.024$), which remained significant after multivariable adjustment. Following correction for multiple comparisons using the Benjamini–Hochberg false discovery rate (FDR, $q < 0.10$), statistical significance was retained only in the fully adjusted model ($q = 0.091$). No significant associations were found for total fat, saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs), total PUFA, cholesterol, or omega-6 PUFA.

Conclusion Higher dietary intake of omega-3 PUFAs may be associated with reduced bloating, while other types of fats showed no significant association. This highlights the potential role of omega-3 fatty acids in managing bloating. Further research is needed to explore causal relationships and the therapeutic implications of these findings.

Keywords Abdominal bloating, Dietary fats, Omega-3 fatty acids, Gastrointestinal symptoms

Introduction

Bloating is among the most prevalent and bothersome issues reported by the general population [1]. Based on the Rome IV criteria, functional abdominal bloating/distension (FABD) is diagnosed in individuals who experience recurrent bloating and/or measurable distension at least once per week over the past three months, with symptom onset at least six months prior, and who do not meet criteria for other gut–brain interaction disorders [2]. A national, cross-sectional telephone survey of households in the United States reported that about 16% of adults have symptoms of abdominal bloating [3]. Compared to men, women were more likely to report experiencing bloating (19% vs. 10.5%) and to have severe symptoms (24% vs. 13%) [4]. The prevalence of bloating was 13.1% in Canada [5], 7% in the United States [6], and up to 25% in Iran [7].

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Given the multifactorial nature of abdominal bloating, factors such as lifestyle, gut microbiota composition, intestinal gas production and transit, and visceral hypersensitivity may all contribute to its manifestation and variability among individuals [8]. The quality of consumed food plays a critical role in the etiology of abdominal bloating and can significantly influence patients' perception and reporting of their symptoms [9]. Diet is a modifiable contributor to abdominal bloating, as colonic bacteria generate gas during the fermentation of ingestible food components—particularly dietary fibers [10].

Few studies have examined the effects of dietary fat on bloating. For example, bloating is reported to be caused by a high-fat meal that occurs 30 min after fat ingestion [11, 12]. However, bloating did not differ significantly between diets high in protein and those high in unsaturated fat, or between diets high in carbohydrate and those high in unsaturated fat, according to another study [13]. Furthermore, the intake of extra virgin olive oil was suggested to alleviate bloating [14]. Given the limited understanding of how dietary fats influence abdominal bloating, this study was warranted to explore whether different types of fat—such as saturated, monounsaturated, and polyunsaturated fats—may exert distinct effects on the occurrence of bloating symptoms.

Methods

Study design and participants

In this case-control study, 250 women with abdominal bloating and 250 women without abdominal bloating were randomly selected among the people referred to Shohada Tajrish Hospital in Tehran, Iran. Sample size was calculated using OpenEpi (www.openepi.com), based on expected odds ratios [15], exposure prevalence, 95% confidence level, and 80% statistical power, ensuring adequate sensitivity to detect moderate associations. Abdominal bloating was defined as if they experience recurrent bloating (defined as a feeling of abdominal fullness, pressure, or trapped gas) and/or distension (defined as a quantifiable increase in abdominal circumference) "on average at least once per week" for the last 3 months with onset at least 6 months prior to diagnosis. Eligibility criteria for the case group included female sex, age between 35 and 70 years, self-reported abdominal bloating, written informed consent to participate, and a diagnosis of bloating within the past three months [16]. The inclusion criteria of the control group included female sex, not suffering from abdominal bloating or other gastrointestinal symptoms or disorders, age between 35 and 70 years, and consent to participate in the study. The age range of 35–70 years was chosen to include middle-aged and older adults who are more likely to report gastrointestinal symptoms such as bloating, while reducing potential confounding from age-related diseases common in individuals over 70. Following the application of exclusion criteria—including incomplete questionnaires, presence of other gastrointestinal disorders, implausible energy intake values (< 800 or >4,000 kcal/day), and reported use of fat-containing or fatty acid supplements—21 participants from the case group and 26 from the control group were excluded. Ultimately, 229 individuals with abdominal bloating and 224 without bloating were included in the final analysis (Fig. 1).

Baseline data, including anthropometric measurements, medical history, alcohol consumption, tobacco use, education level, and socioeconomic variables, were collected through a face to face interview. Height and weight were measured by trained nutritionists using a standard Seca caliper and scale with an accuracy of 0.5 cm and 100 g, and then the body mass index (BMI) of the individuals was calculated. Information related to physical activity was collected using the International Physical Activity Questionnaire, whose validity and reliability have already been confirmed in Iran [17]. People were evaluated and compared in terms of activity level based on MET (Metabolic Coefficient of Activity).

Dietary fat intake

Dietary intake of the participants in the study was evaluated by completing a valid semi-quantitative 168-item food frequency questionnaire (FFQ), which has already been confirmed in terms of validity and reliability [18]. Consideration was given to household measures for portion sizes, which were subsequently converted to grams using standardized conversion factors. Nutrient intake was then estimated based on the Food Composition Table (FCT) provided by the United States Department of Agriculture (USDA) (USDA, Release 11, 1994, adapted for Iranian foods).

Dietary data were analyzed using Nutritionist IV software (version 4; First Databank Division, The Hearst Corporation, San Bruno, CA, USA). Intake of total fat, saturated fatty acids (SFA), monounsaturated fatty acids (MUFA), and polyunsaturated fatty acids (PUFA) was calculated and examined.

Statistical analysis

Sociodemographic and dietary characteristics of participants in the case and control groups were compared using chi-square tests for qualitative variables and independent t-tests for quantitative variables. Daily intake of fatty acids were compared between the case and control groups using an independent t-test. To investigate the association between abdominal bloating and dietary fatty acids, binary logistic regression analysis was used, and three regression models were formulated: Model (1) Adjusted for dietary intake of other types of fats, Model (2) further adjustments for age, calorie intake, education,

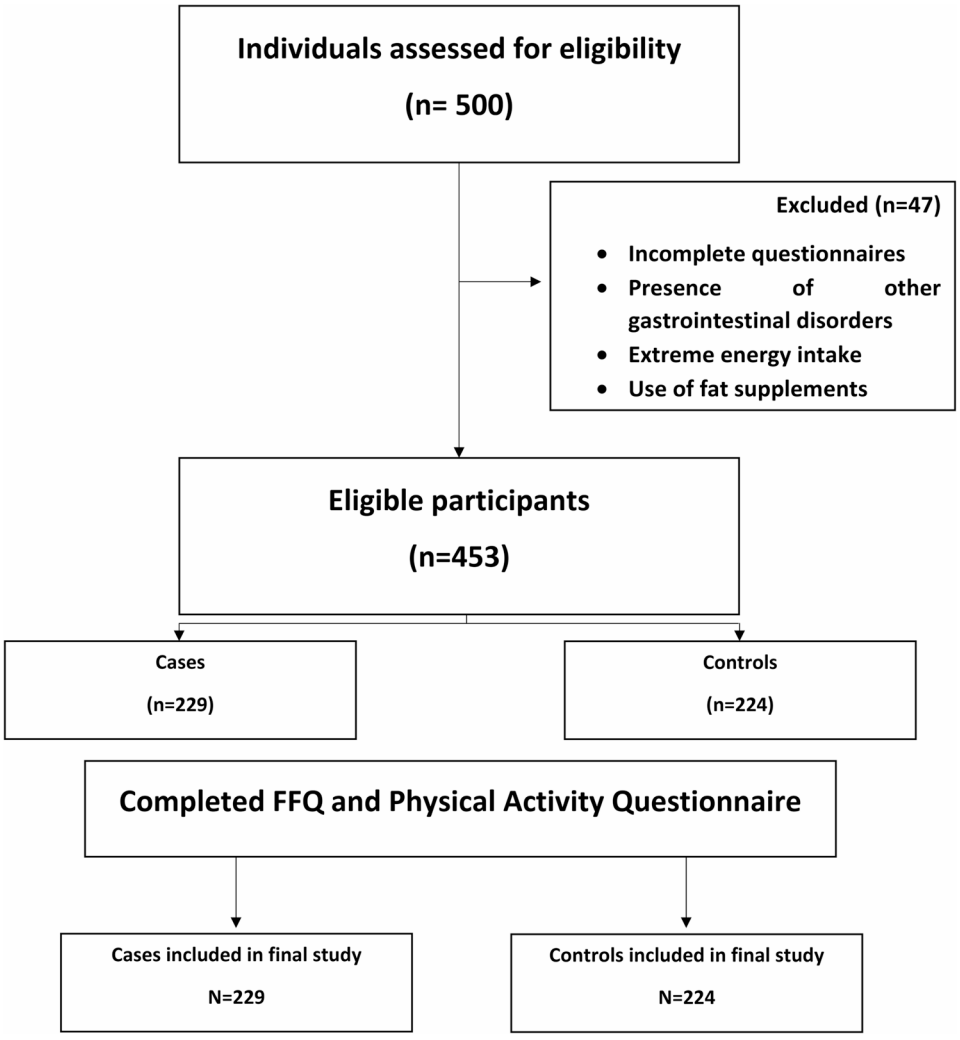

Fig. 1 STROBE-compliant participant flow diagram

and marital status. Model (3) additional adjustment for physical activity. Because seven dietary fat variables were examined in each model, we applied the Benjamini–Hochberg false discovery rate (FDR) procedure to control for multiple comparisons. Raw p-values were first obtained for each predictor, then ranked in ascending order. Adjusted q-values were calculated using the Benjamini–Hochberg algorithm, which rescales p-values according to their rank within the family of tests. We prespecified an FDR threshold of $q = 0.10$, meaning that up to 10% of findings declared significant may represent false positives. Both raw p-values and FDR-adjusted q-values are reported in the tables, and statistical significance was considered at $q < 0.10$. All nutrient intakes were energy-adjusted using the residual method before inclusion in regression models to control for confounding by total energy intake. Linearity of the relationship between continuous nutrient intakes and the log odds of bloating was assessed using fractional polynomial modeling and LOWESS plots. Fatty acids intake demonstrated an approximately linear association and was therefore modeled as a continuous variable. SPSS software version 20 (SPSS Inc., Chicago, USA) was used for all statistical analyses.

Results

The general characteristics of the participants are presented in Table 1; Fig. 1. Among blood pressure measurements, only right arm systolic blood pressure (SBP) showed a statistically significant difference between the case and control groups, with higher values observed in the control group (mean ± SD: 111.35 ± 15.66 mmHg vs. 107.63 ± 13.06 mmHg; $P = 0.02$).

As shown in Table 2, the control group had a significantly higher intake of omega-3 polyunsaturated fatty acids compared to the case group (mean ± SD: 1.32 ± 0.739 g/day vs. 1.16 ± 0.61 g/day; $P = 0.04$). The groups did not differ in their consumption of calories,

Table 1 Participants' general attributes

	Controls	Cases	*P*
Age (years)	60.72 ± 8.79	48.82 ± 8.03	0.05
Height (cm)	155.597 ± 5.4447	156.480 ± 5.5275	0.15
Weight (kg)	70.319 ± 11.3618	71.828 ± 11.6213	0.24
BMI (kg/m^2)	29.0159 ± 4.32407	29.2939 ± 4.24418	0.56
Physical activity (MET)	1.6833 ± 1.60348	1.3881 ± 1.42393	0.09
Right arm DBP	71.05 ± 9.564	70.05 ± 8.652	0.34
Right arm SBP	111.35 ± 15.662	107.63 ± 13.057	0.02
Diabetic	35.8%	64.2%	0.99
Hypertensive	39.5%	60.5	0.36

Chi-square tests were used for qualitative variables, while independent t-tests were applied to quantitative variables

Abbreviations: MET Metabolic Coefficient of Activity, *DBP* Diastolic Blood Pressure, *SBP* Systolic Blood Pressure

Table 2 Participants' dietary intake

	Controls	Cases	*P*
Energy (Kcal)	2554.81 ± 348.95	2607.31 ± 518.91	0.26
Protein (g)	82.49 ± 18.14	84.93 ± 26.85	0.31
Carbohydrate (g)	366.18 ± 57.06	373.59 ± 69.31	0.28
Total fat (g)	92.62 ± 17.28	93.95 ± 23.92	0.55
Cholesterol (mg/d)	277.70 ± 134.93	263.79 ± 141.69	0.37
SFA (g/d)	28.52 ± 9.85	30.10 ± 13.53	0.21
MUFA (g/d)	33.64 ± 13.19	33.56 ± 12.92	0.96
PUFA (g/d)	19.10 ± 7.62	19.26 ± 7.82	0.86
PUFA n-3 (g/d)	1.32 ± 0.739	1.16 ± 0.61	0.04
PUFA n-6 (g/d)	6.50 ± 5.74	6.06 ± 5.17	0.54

Independent t-tests were applied to quantitative variables

Abbreviations: SFA Saturated Fatty Acids, *MUFA* Mono Unsaturated Fatty Acids, *PUFA* Poly Unsaturated Fatty Acids, *PUFA n-3* ω3 Poly Unsaturated Fatty Acids, *PUFA n-6* ω6 Poly Unsaturated Fatty Acids

protein, carbohydrates, total fat, saturated fatty acids, cholesterol, monounsaturated fatty acids, and other polyunsaturated fatty acids.

Table 3 shows the associations between different types of dietary fats and abdominal bloating across three logistic regression models. In Model 1, adjusted only for intake of other fat types, no significant associations were observed except for omega-3 PUFA, which demonstrated an inverse relationship with bloating (OR = 0.34, 95% CI: 0.13–0.87, $p = 0.024$). After additional adjustment for age, energy intake, education, and marital status (Model 2), this association persisted (OR = 0.28, 95% CI: 0.10–0.80, $p = 0.017$). In the fully adjusted model including physical activity (Model 3), omega-3 PUFA continued to show a protective effect (OR = 0.27, 95% CI: 0.09–0.76, $p = 0.013$; Fig. 2).

Following multiple-comparison adjustment using the Benjamini–Hochberg false discovery rate (FDR, applied separately within each model with $q < 0.10$ as the significance threshold), only the association between omega-3 PUFA and abdominal bloating remained statistically significant in the fully adjusted model ($q = 0.091$). All other fat types (total fat, SFA, MUFA, total PUFA, cholesterol, omega-6 PUFA) did not retain statistical significance after FDR correction.

Discussion

This case–control study examined the relationship between abdominal bloating and the consumption of various dietary fats. Among the seven fat types assessed, only omega-3 PUFA intake showed evidence of a protective association. Mean intake was slightly higher in controls (1.32 ± 0.74 g/day) compared with cases (1.16 ± 0.61 g/day), suggesting that habitual consumption within this achievable dietary range may be linked to reduced odds of bloating. Although the difference in intake was modest, the association remained consistent across progressively adjusted models. Importantly, after correction for multiple comparisons using the Benjamini–Hochberg false discovery rate, statistical significance was retained only in the fully adjusted model (Model 3, $q = 0.091$). This finding highlights omega-3 PUFA as a potential dietary factor associated with lower symptom burden, whereas other fat types (total fat, SFA, MUFA, total PUFA, cholesterol, omega-6 PUFA) did not show significant associations after FDR adjustment.

Previous studies have demonstrated a link between dietary intake and dyspeptic symptoms, with several reports indicating that sensations of fullness and bloating are commonly associated with higher fat consumption. Pilichiewicz et al. conducted a study involving twenty patients diagnosed with functional dyspepsia (FD), of which 17 were women, alongside 21 healthy participants,

Table 3 Association between dietary fat types and abdominal bloating

Predictor	Model 1 OR (95% CI)	*p*	q	Model 2 OR (95% CI)	*p*	q	Model 3 OR (95% CI)	*p*	q
Total fat	1.017 (0.984–1.051)	0.312	0.488	1.025 (0.971–1.081)	0.372	0.372	1.026 (0.972–1.082)	0.359	0.388
PUFA	1.035 (0.934–1.141)	0.488	0.488	1.068 (0.957–1.190)	0.240	0.372	1.073 (0.963–1.197)	0.203	0.355
Cholesterol	0.997 (0.988–1.006)	0.470	0.488	0.993 (0.983–1.004)	0.207	0.372	0.993 (0.982–1.003)	0.172	0.355
SFA	0.967 (0.916–1.021)	0.220	0.488	0.975 (0.922–1.030)	0.365	0.372	0.976 (0.922–1.032)	0.388	0.388
MUFA	1.015 (0.979–1.053)	0.410	0.488	1.021 (0.983–1.060)	0.282	0.372	1.021 (0.983–1.061)	0.286	0.388
PUFA n-3	0.340 (0.134–0.866)	0.024	0.168	0.281 (0.099–0.795)	0.017	0.119	0.265 (0.093–0.758)	0.013	0.091
PUFA n-6	0.908 (0.800–1.030)	0.134	0.469	0.896 (0.782–1.025)	0.109	0.372	0.887 (0.773–1.018)	0.088	0.308

Binary logistic regression models were fitted. Multiple comparisons were controlled using the Benjamini–Hochberg False Discovery Rate (FDR) procedure, performed separately within each model ($m = 7$). *Q*-values < 0.10 were considered statistically significant

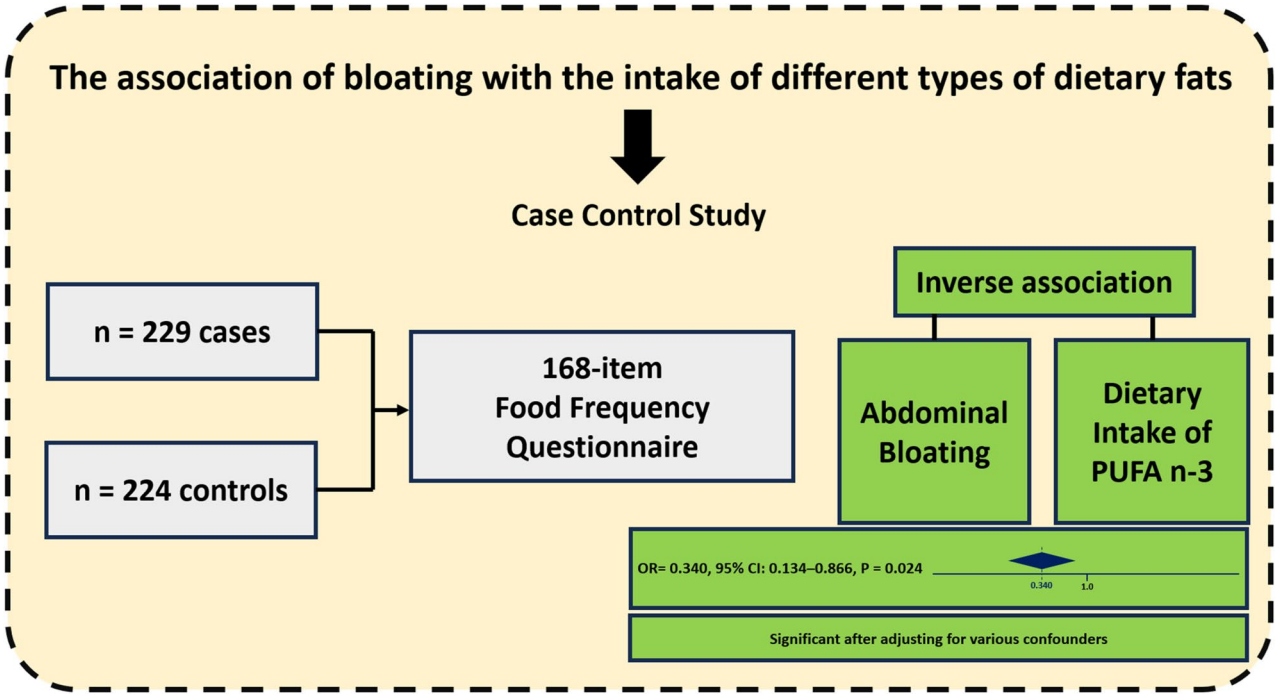

Fig. 2 Association between dietary intake of n-3 Polyunsaturated Fatty Acids (PUFA n-3) and abdominal bloating (Model 1: Adjusted for other dietary fats, Model 2: Additionally adjusted for age, calorie intake, education, marital status, Model 3: Additionally adjusted for physical activity)

including 18 women. These individuals meticulously maintained diet diaries, documenting all food and drink intake, the timing of consumption, and the occurrence, timing, and intensity of dyspeptic symptoms. The findings indicated an association between feelings of fullness and bloating with fat consumption, highlighting a specific role of fatty foods in triggering these symptoms [19]. Another study by Carvalho et al. found that postprandial fullness was the primary concern associated with foods high in fat content. The consumption of fat, which has the potential to delay gastric emptying, may lead to disruptions in gastric motility, resulting in postprandial fullness among dyspeptic patients [20].

Our findings of an inverse association between omega-3 PUFA intake and abdominal bloating differ from those reported in the OmniHeart randomized controlled trial on the ffects of high-fiber diets and macronutrient substitution on bloating, which did not observe a protective effect of unsaturated fats. Several factors may explain this discrepancy. First, OmniHeart was a short-term, controlled feeding study, whereas our case–control design assessed long-term habitual intake, potentially capturing chronic dietary patterns not reflected in short interventions. Second, OmniHeart diets were uniformly high in fiber (~ 30 g/day), a level known to influence bloating and possibly mask or modify the effects of fat intake. Third, the trial examined general unsaturated fats, while our analysis focused specifically on omega-3 PUFA, which may exert distinct physiological effects. Finally, differences in participant characteristics and methods of symptom assessment may also contribute to divergent findings [21].

Previous research has highlighted several biologically plausible pathways through which dietary components may influence gastrointestinal symptoms. Omega-3 fatty acids are well known for their anti-inflammatory properties and may reduce intestinal inflammation, thereby decreasing visceral hypersensitivity and gas-related discomfort [22]. In addition, accumulating evidence suggests that omega-3 intake can modulate gut microbiota composition. For example, Yokota et al. reported that bile acid composition under a high-fat diet can directly shape the gut microbiota [23]. while Costantini et al. demonstrated that omega-3 fatty acids promote short-chain fatty acid (SCFA) production and beneficial microbial shifts [24]. More recently, Zou et al. emphasized that maintaining a balanced omega-6/omega-3 ratio supports gut microbial diversity and anti-inflammatory pathways [25]. Furthermore, Kerman et al. highlighted that the gut microbiome may play a critical role in mediating the effects of omega-3 supplementation, underscoring the importance of microbiota-related endpoints in future trials [26] .

Dietary fat may also influence gastrointestinal sensitivity through cholecystokinin (CCK) release, which modulates gastric motility and visceral perception; CCK-A receptor antagonists such as dexloxiglumide have been

shown to reduce lipid-induced dyspeptic symptoms [27, 28].

While these mechanisms are biologically plausible and consistent with prior literature, they should be regarded as hypothetical in the context of our study. We did not measure inflammatory biomarkers, gut microbiota composition, or gastrointestinal hormone levels, and therefore cannot provide direct mechanistic evidence. The associations observed in our case–control analysis may reflect downstream effects of omega-3 intake, correlated dietary patterns, or unmeasured confounding factors. Accordingly, mechanistic explanations remain speculative and require confirmation in future studies incorporating physiological, microbiome, or hormone-related endpoints.

Although this study provides valuable insights into the association between dietary fats and abdominal bloating, several limitations should be acknowledged. The observed protective effect of omega-3 PUFA intake may reflect not only the fatty acid itself but also other bioactive compounds present in omega-3–rich foods. The exclusive inclusion of women aged 35–70 years limits generalizability, and reliance on self-reported symptoms and FFQ-based dietary assessment introduces potential recall bias and exposure misclassification. Lack of data on individual omega-3 subtypes (EPA, DHA, ALA) and absence of biochemical validation further constrain interpretation. Moreover, residual confounding from unmeasured variables such as medication use, stress levels, sleep quality, and specific dietary factors (e.g., FODMAP intake, fiber types, meal timing) may partially or entirely explain the observed association between omega-3 PUFA intake and abdominal bloating. Finally, although multiple-comparison adjustment was performed using the Benjamini–Hochberg false discovery rate, statistical significance was retained only for omega-3 PUFA in the fully adjusted model, underscoring the need for cautious interpretation. Future studies incorporating objective biomarkers, validated symptom scales, and broader populations are required to confirm causality and clarify underlying mechanisms.

Conclusion

This case–control study indicates that higher dietary intake of omega-3 polyunsaturated fatty acids may be associated with a reduced risk of abdominal bloating. Although the observed difference in intake between cases and controls was modest, the protective association of omega-3 PUFA persisted across progressively adjusted models and remained statistically significant only in the fully adjusted model after correction for multiple comparisons (FDR q = 0.091). No significant associations were detected for other types of dietary fats. These findings suggest that omega-3 PUFA could represent a potential dietary factor linked to lower symptom burden. To validate this observation and clarify underlying mechanisms, further intervention studies are warranted. If confirmed, such evidence may inform dietary recommendations for individuals with persistent bloating, emphasizing the inclusion of omega-3–rich foods (e.g., fatty fish, walnuts, flaxseed) as part of a targeted nutritional strategy. Future research should also explore the long-term effects of omega-3 supplementation on bloating and related gastrointestinal disorders to determine therapeutic potential.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12876-025-04552-0.

Supplementary Material 1.

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Authors' contributions

MD, KD, AT, MS, AN, MS, SM, and BA designed the study, and were involved in the data collection, analysis, and drafting of the manuscript. SD, AA, MGHH and MGh were involved in the design of the study, analysis of the data, and critically reviewed the manuscript. All authors read and approved the final manuscript.

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Data availability

Data available upon on request of corresponding author.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the principles of the Declaration of Helsinki. The research protocol was approved by the Ethics Committee of Shahid Beheshti University of Medical Sciences, Tehran, Iran (Ethics Code: IR.SBMU.RETECH.REC.1404.414). Written informed consent was obtained from all participants prior to enrollment.
All study participants signed a written informed consent.

Consent for publication

Institutional consent forms were used in this study.

Competing interests

The authors declare that they have no competing interests.

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