

RESEARCH

Open Access



# The association between ultra-processed food consumption and adiposity indexes in adults living in Tehran: a dose-response analysis within a cross-sectional study

Mahsa Ranjbar<sup>1</sup>, Neda Asgari Avini<sup>2</sup> and Sakineh Shab-Bidar<sup>2,3\*</sup>

## Abstract

**Background** Ultra-process foods (UPF) were suspected to induce many diseases and threaten consumers' health. The aim of this study was to examine the association between the consumption of highly processed foods and adiposity indexes in Tehranian adults.

**Method** In a cross-sectional design, 850 Tehranian adults were included. NOVA classification was used to assess the intake of UPF in participants. The amount of calorie intake through processed foods was assessed. Adiposity indexes outcomes include body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR), waist-to-height ratio (WHtR), visceral adiposity index (VAI), body roundness index (BRI), and body adiposity index (BAI). Lipid accumulation product (LAP) and triglyceride-glucose index (TyG) were also assessed. Binary logistic regression was used to evaluate the association between the intake of UPF and adiposity indexes measurements.

**Result** there was a higher intake of UPF in men than women ( $p < 0.001$ ). The results of logistic regression revealed that there is a significant association between intake of UPF with WHR (odds ratio (OR): 1.09, 95% confidence interval (CI): 0.73–1.61) and BRI (OR: 2.10, 95% CI: 1.38–3.19) in the crude model. Nevertheless, after adjusting for confounders, the results were insignificant (WHR: OR, 0.77, 95% CI, 0.46–1.27, and BRI: OR, 1.70, 95% CI, 1.05–2.73). No significant association was seen for other outcomes ( $p > 0.001$  for all). The results of dose-response analyses revealed a substantial association between UPF intake and BMI, WHtR, WC, VAI, BRI, BAI, LAP, and TyG.

**Conclusion** UPF consumption was significantly related to increased risk of high-risk adiposity indexes in the dose-response analysis. More studies are needed to strengthen the results of this study.

**Keywords** Food, Processed, Anthropometry, Adult, Body mass index, Waist circumference, Waist-hip ratio, Waist-height ratio, Lipid accumulation product

\*Correspondence:  
Sakineh Shab-Bidar  
s\_shabbidar@tums.ac.ir

<sup>1</sup>Department of Clinical Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences (TUMS), Tehran 14167-53955, Iran

<sup>2</sup>Department of Community Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences (TUMS), No 44, Hojjat-dost Alley, Naderi St., Keshavarz Blvd, P. O. Box 14155/6117, Tehran 14167-53955, Iran

<sup>3</sup>Sports Medicine Research Center, Neuroscience Institute, Tehran University of Medical Sciences, Tehran, Iran



© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

## Introduction

Obesity, which is induced by multi-causal processes, led to an epidemic that affects all around the world [1]. The World Health Organization (WHO) reported that in 2014, over 1.9 billion adults were classified as overweight, with 650 million of them being obese [2]. Obesity is a significant risk factor for several other diseases, including coronary heart disease, ischemic stroke, and type 2 diabetes [3]. Moreover, it is considered a metabolic disease itself [4]. The quality of diet is known as a major modifiable risk factor for weight management, and it has considerable evidence for protective factors [5]. Furthermore, the causes of the worldwide epidemic of obesity may lie in changing social behaviors and environmental factors [6]. Changes in the food system are mainly characterized by the increasing supply of energy-efficient and cost-effective food products [7, 8]. This shift in food systems has been accompanied by notable changes in diets over the past few decades, particularly marked by a sharp rise in the consumption of ultra-processed foods (UPFs) [9]. Like the higher incidence and prevalence of obesity, there has been a notable rise in the production and consumption of UPF, and research indicated that currently, about 50 to 60 of the total daily energy in some individuals is allocated to UPFs [10]. These food groups are often high in calories, but they lack nutritional value, contributing to excessive calorie consumption without providing essential nutrients, which can result in obesity [11]. It is worth mentioning that studies suggest that the consumption of UPFs may also disrupt metabolic processes, which is another potential stem of weight gain and obesity-related complications [12].

The NOVA classification is a system that categorizes foods into groups based on the degree and amount of their industrial processing [13]. According to NOVA, UPF is a group that includes foods or beverages formulated mainly or entirely from food-derived materials, with little or no presence of the original unaltered food [14]. UPFs are known for their high content of simple sugars, fats, and salt while being low in fiber and micronutrients. They often contain numerous additives, including flavor enhancers (such as phosphates, sweeteners, emulsifiers, colors, and wetting agents), and generally, they have lower dietary quality than UPFs [15]. These foods undergo multiple processing methods, including high-temperature extrusion, molding, and frying. They often contain various food additives and industrial substances used to replicate or improve the sensory qualities of foods or mask undesirable ingredients [16]. UPF consumption is growing worldwide [17, 18]. UPF consumption in Iran is a crucial problem due to the nutritional transition from traditional to more Western food patterns [19]. Numerous ecological studies have indicated that as the consumption of UPFs increases, so does the prevalence of

overall obesity [18, 20]. In many past studies, it has been shown that UPF consumption was associated with overweight or obesity [20–23]. In a two-week randomized crossover study, Hall KD and colleagues demonstrated that participants on an ultra-processed diet consumed approximately 500 extra calories per day compared to those on an unprocessed diet, which was closely associated with weight gain [24]. More than primary indicators of obesity, including weight and body mass index (BMI), which may not illustrate obesity properly, other anthropometric indexes are drawing attention these days [25]. Visceral adiposity index (VAI), body roundness index (BRI), body adiposity index (BAI), lipid accumulation product (LAP), and triglyceride-glucose index (TyG) are some cases in point [26]. Although some studies have revealed an association between the consumption of UPFs and obesity, this association is unclear for the adiposity indexes. Because of this, this study aimed to investigate the relationship between the consumption of UPF and adiposity indexes in adults living in Tehran.

## Methods

This study utilized data from a previous research project titled ‘The Association Between Lunch Composition and Obesity in Iranian Adults,’ conducted between 2018 and 2019, which aimed to assess the link between meal-based dietary patterns and obesity [27]. Five healthcare centers in Tehran city were selected using the clustered method. Medical Ethics Committee of the Tehran University of Medical Sciences, Tehran, Iran (Ethic number: IR.TUMS.MEDICINE.REC.1401.695) approved this study.

### Study participants

The population of this study included 850 adult participants. Inclusion criteria were: (1) healthy adult dwellers in Tehran city, (2) aged between 20 and 59, and (3) willing to participate. Furthermore, participants were excluded if they were suffering from diseases such as diabetes, cancer and cardiovascular diseases, Alzheimer’s, Parkinson’s, chronic liver and kidney diseases, history of stroke, brain and heart attack, rheumatoid arthritis, and other chronic diseases based on the person’s statement, and did not complete the food recall questionnaires.

### Sampling

According to past reports, the prevalence of obesity in the population of the study is about 65% [28]. As a result, the sample size was calculated using the following formula:  $n = z^2 \cdot p(1-p) / d^2 = 546$ , with a maximum estimation error of 5%, and 0.04 for the  $d$  value. Because two two-stage clustered methods were used, and the effect of the response rate in different health centers on the total results, considering the effective coefficient of the cluster design of 1.5, the total calculated sample size was 816. In

this study, 850 participants were entered equally from different health centers using a two-stage cluster and simple random sampling.

### Dietary assessment

In this study, a 168-item food frequency questionnaire, validated before, was used by a trained researcher to examine participants' dietary intake. Dietary data were changed to grams per day. We used Nutritionist IV (N4) software to estimate the energy and nutrient intakes by the US Department of Agriculture's food composition database modified for Iranian foods [29]. In order to assess the intake of UPF in participants, NOVA classification was used [16]. Due to NOVA, UPF includes creamy cheese, ice creams, chocolate milk, sausages, burgers, hotdogs, pizza, cakes, biscuits, candies, chocolates, mayonnaise, margarine, and hydrogenated oils, carbonated soft drinks, potato chips, and pufak. For the inter-individual and UPF differences in intake, an estimate of normal intake was made by considering the contribution to energy consumption as a percentage of total energy consumption. Moreover, the individual intakes from different food groups were also defined to represent a total energy intake of UPF%.

### Clinical assessment

All participants were instructed to fast for 8–12 h prior to blood sampling. The serum samples were centrifuged and transferred into clean cryotubes for storage at  $-80^{\circ}\text{C}$  until analysis. High-density lipoprotein cholesterol (HDL cholesterol) was measured using the cholesterol oxidase phenol-amino-pyridine method, while triglycerides (TG) were assessed using an enzymatic method based on glycerol-3-phosphate oxidase phenol-amino-pyrene, utilizing an automatic machine (Selectra E, Vitalab, Netherlands). The inter- and intra-assay coefficient variances (CVs) were both below 10%.

### Assessment of adiposity indexes

Weight was assessed using a digital scale with a precision of 0.1 kg (808Seca), with participants not wearing shoes or heavy clothing. Height was measured in standing without shoes using a wall stadiometer, accurate to 0.1 cm (Seca, Germany). BMI was determined by dividing weight in kilograms by the square of height in meters. Waist circumference (WC) was measured at the midpoint between the lowest rib and the iliac crest while the participant was exhaling, and hip circumference was taken with a tape measure. The waist-to-hip ratio (WHR) and waist-to-height ratio (WHtR) were calculated for each individual. The VAI was computed using a specific formula that incorporates WC (cm), BMI ( $\text{kg}/\text{m}^2$ ), TG (mg/dl), and high-density lipoprotein (HDL) (mg/dl) [30].

$$\text{Men VAI} : \left( \frac{WC}{39.68 + (1.88 \times BMI)} \right) \times \left( \frac{TG}{1.03} \right) \times \left( \frac{1.31}{HDL} \right)$$

$$\text{Woman VAI} : \left( \frac{WC}{39.58 + (1.89 \times BMI)} \right) \times \left( \frac{TG}{0.81} \right) \times \left( \frac{1.52}{HDL} \right)$$

Other indicators that have been introduced for body fat are BRI and BAI. BRI depends on WC and height (both in cm), and BAI on hip circumference (cm) and height (m) [30, 31].

$$\text{BRI} : 364.2 - 365.5 \sqrt{1 - \frac{\left( \frac{WC}{2\pi} \right)^2}{(0.5 \text{ height})^2}}$$

$$\text{BAI} : \frac{\text{Hip circumference}}{\text{Height}^{1.5}}$$

LAP is known as an indicator for lipid accumulation, depending on WC (cm) and TG (mmol/L) [32].

$$\text{Men LAP} : (WC - 65) \times TG$$

$$\text{Woman LAP} : (WC - 58) \times TG$$

TyG, introduced as a new indicator of metabolic diseases, is calculated using TG and fasting blood glucose (FBS) (mg/dL for both) [33].

$$\text{TyG} : \ln(TG \times FBS) / 2$$

### Covariates

A trained interviewer collected pertinent information regarding age (continuous), gender (categorical), education (categorical), marital status (categorical), tobacco use (categorical), and occupation (categorical). The International Physical Activity Questionnaire (IPAQ) was utilized to evaluate participants' physical activity levels, which includes seven questions concerning the frequency and duration of activities. The findings are expressed in terms of the metabolic equivalent of task minutes (MET-minutes) [34]. Participants were then classified into three activity levels as categorical variable: high ( $>3000$  MET-minutes/week), moderate (600–3000 MET-minutes/week), and low ( $<600$  MET-minutes/week). In our analysis, we utilized adjusted effect sizes within a dose-response framework, employing restricted cubic splines to model the relationship between the covariates and the outcome variable. This approach allowed us to flexibly capture potential non-linear associations without imposing strict parametric assumptions. Regarding the

covariates, we treated them as continuous variables to preserve the granularity of the data and to avoid potential information loss that might arise from categorization. For instance, age was modeled as a continuous variable, enabling us to explore its nuanced effects across the entire spectrum rather than grouping it into predefined categories. This decision aligns with best practices in epidemiological studies, where continuous modeling is often preferred to maintain statistical power and to accurately reflect the underlying biological or behavioral processes.

### Statistical analysis

Statistical analyses in this study were run by SPSS version 26 (IBM). UPF intake, obtained from NOVA classification, was categorized into 4 quartiles by the software. In order to compare the demographic and clinical variables of included participants according to the quartiles of UPF intake, we used the independent sample t-test for continuous and Chi-square test for categorical variables. For showing the UPF consumption and relative contribution of food groups in different quartiles, the one-way analysis of variance (ANOVA) was used. Anthropometric measurements were categorized into two groups with the following cut-offs: BMI:  $25 \text{ kg/m}^2 < \text{vs.} \geq 25 \text{ kg/m}^2$  [35], WC:  $<90 \text{ vs.} \geq 90 \text{ cm}$  in men and  $<80 \text{ vs.} \geq 80 \text{ cm}$  in women [36]. WHR: including  $<0.95 \text{ vs.} \geq 0.95$  in men and  $<0.80 \text{ vs.} \geq 0.80$  in women [37]. WHtR:  $<0.5 \text{ vs.} \geq 0.5$  for both genders [38]. VAI:  $<4.11 \text{ vs.} \geq 4.11$  in men and  $<4.28 \text{ vs.} \geq 4.28$  in women [30]. BRI:  $<4.75 \text{ vs.} \geq 4.75$  in men and  $<6.17 \text{ vs.} \geq 6.17$  in women [30]. BAI:  $<25.6 \text{ vs.} \geq 25.6$  in men and  $<37.7 \text{ vs.} \geq 37.7$  in women [39]. LAP: 45.65 in men and 46.91 in women [40]. TyG: 8.63 in men and 8.54 in women [40]. Except for BMI, WC, and LAP, other outcomes were indexes that did not have units [41–43]. Logistic regression was done in order to assess the UPF intake and odds of adiposity measurements, and results were reported as odds ratio (OR) and 95% confidence intervals (95% CI). The results were further adjusted for age, sex, education level, occupation, marital status, smoking, supplement intake, physical activity, and energy intake. For all analyses, the statistical significance level has been set at 0.05. Moreover, we examined the potential non-linear relation between % UPF intake from total calorie intake (continuous) and adiposity outcomes with restricted cubic splines (RCS) [44] using STATA software version 17. The results were adjusted for age, sex, education level, occupation, marital status, smoking, supplement intake, physical activity, and energy intake.

### Results

850 eligible adults who fulfilled the inclusion criteria entered the study. Table 1 shows the detailed demographic and clinical variables according to the quartiles of UPF intake. There were significant differences regarding

sex ( $p < 0.001$ ), occupation ( $p = 0.002$ ), and smoking status ( $p = 0.005$ ). Moreover, weight, VAI, and BIA were significantly increased in the higher quartile compared to the lower one ( $p < 0.05$  for all). There was no significant difference considering other outcomes ( $p > 0.05$  for all). Table 2 displays the relative contribution of food groups in UPF consumption. Regarding the results, all food groups revealed significant differences between quartiles of UPF consumption ( $p < 0.05$  for all), except for soft drinks ( $p = 0.268$ ). The results of the association between adiposity indexes and the UPF categories are presented in Table 3. Logistic regression revealed a significant difference between higher quartiles compared to lower quartiles in WHR, WC, and BAI in the crude model ( $p < 0.05$  for both). However, after adjusting for potential confounders, including age, sex, education level, occupation, marital status, smoking, supplement intake, and physical activity, the results were insignificant. The results for other adiposity measurements were insignificant ( $p > 0.05$  for all). Further, the results of dose-response analyses using restricted cubic splines analysis show a significant association between BMI, WHtR, WC, VAI, BRI, BAI, ALP, TyG, and the proportion of UPF intake (Table 4). Figure 1 illustrates the results of dose-response analysis for BMI. BMI experienced a marginal decline to 10% of UPF intake, followed by a substantial increase for higher proportions of UPF intake ( $P_{\text{nonlinearity}} < 0.001$ ,  $P_{\text{dose-response}} < 0.001$ ). WHtR saw a moderate increase for higher proportions of UPF intake ( $P_{\text{nonlinearity}} < 0.001$ ,  $P_{\text{dose-response}} < 0.001$ ) (Fig. 2). The figure for WC indicates a sharp increase to approximately 10% of the percentage of UPF intake, which continued by a moderate rise ( $P_{\text{nonlinearity}} < 0.001$ ,  $P_{\text{dose-response}} < 0.001$ ) (Fig. 3). VAI remained unchanged at 0 to UPF intake of 10%, after which it dropped ( $P_{\text{nonlinearity}} < 0.001$ ,  $P_{\text{dose-response}} = 0.007$ ) (Fig. 4). BRI increased markedly over the whole proportion of intakes ( $P_{\text{nonlinearity}} < 0.001$ ,  $P_{\text{dose-response}} = 0.085$ ) (Fig. 5). BAI and LAP rose steadily to 10% and then plateaued with a slight upward curve ( $P_{\text{nonlinearity}} < 0.001$ ,  $P_{\text{dose-response}} < 0.001$ , for both) (Figs. 6 and 7). Also, the trend for TyG was the same until 10%; however, it decreased moderately in the higher percentages of UPF intake ( $P_{\text{nonlinearity}} < 0.001$ ,  $P_{\text{dose-response}} < 0.001$ ) (Fig. 8). The results for WHR were insignificant ( $P_{\text{nonlinearity}} = 0.399$ ,  $P_{\text{dose-response}} = 0.926$ ) (Fig. 9).

### Discussion

The findings of this study indicated that greater consumption of UPFs did not have a significant association with WHR, BRI, after adjusting for confounders and BMI, WHtR, WC, VAI, BRI, BAI, LAP, and TyG in the crude model and after adjusting for confounders through logistic regression. The dose-response analysis showed UPF intake was associated with an elevated risk of higher

**Table 1** Demographic and clinical variables according to the quartiles of ultra-processed foods intake

Variables	Quartiles of ultra-processed food intake				P-value
	Q1 (0–24.9%)	Q2 (25–49.9%)	Q3 (50–74.9%)	Q4 (75–100%)	
	(n = 212)	(n = 213)	(n = 213)	(n = 212)	
<b>Age (years)</b>	45.27 (10.56)	43.72 (10.41)	44.56 (11.13)	45.49 (10.61)	0.313 <sup>a</sup>
<b>Sex, n (%)</b>					
Male	41 (19.3)	43 (20.2)	79 (37.1)	103 (48.6)	0.001 > <sup>b</sup>
Female	171 (80.7)	170 (79.8)	134 (62.9)	109 (51.4)	
<b>Physical activity status, n (%)</b>					
Low	135 (63.7)	138 (64.8)	129 (60.6)	137 (64.6)	0.787 <sup>b</sup>
Moderate	77 (36.3)	75 (35.2)	84 (39.4)	75 (35.4)	
<b>Education status, n (%)</b>					
illiterate	20 (9.4)	12 (5.6)	20 (9.4)	21 (9.9)	0.013 <sup>b</sup>
Under diploma	47 (22.2)	46 (21.6)	62 (29.1)	69 (32.5)	
Diploma	79 (37.3)	46 (30.0)	65 (30.5)	53 (25.0)	
University educated	66 (31.1)	91 (42.7)	66 (31.0)	69 (32.5)	
<b>Occupation, n (%)</b>					
Employee	42 (19.8)	58 (27.2)	59 (27.7)	61 (28.8)	0.048 <sup>b</sup>
Housekeeper	131 (61.8)	119 (55.9)	117 (54.9)	109 (51.4)	
Retired	28 (13.2)	27 (12.7)	34 (16.0)	39 (18.4)	
Unemployed	11 (5.2)	9 (4.2)	3 (1.4)	3 (1.4)	
<b>Marital status, n (%)</b>					
Single	15 (7.1)	32 (15.0)	25 (11.7)	20 (9.4)	0.219 <sup>b</sup>
Married	179 (84.4)	164 (77.0)	173 (81.2)	172 (81.1)	
Divorced	18 (8.5)	17 (8.0)	15 (7.0)	20 (9.4)	
<b>Smoking status, n (%)</b>					
Not smoking	197 (92.9)	187 (87.8)	189 (88.7)	197 (92.9)	0.373 <sup>b</sup>
Quit smoking	5 (2.4)	12 (5.6)	12 (5.6)	7 (3.3)	
Low smoking	10 (4.7)	14 (6.6)	12 (5.6)	8 (3.8)	
<b>Supplement intake, n (%)</b>					
Yes	30 (14.2)	29 (13.6)	34 (16.0)	38 (17.9)	0.601 <sup>b</sup>
No	182 (85.8)	184 (86.4)	179 (84.0)	174 (82.1)	
<b>BMI (kg/m<sup>2</sup>)</b>	27.94 (4.31)	28.81 (4.25)	27.97 (4.76)	28.05 (4.65)	0.013 <sup>a</sup>
<b>WC (cm)</b>	91.90 (11.49)	90.12 (12.59)	92.45 (12.79)	93.82 (12.62)	0.021 <sup>a</sup>
<b>WHR</b>	0.88 (0.08)	0.86 (0.08)	0.89 (0.08)	0.90 (0.08)	0.001 > <sup>a</sup>
<b>WHtR</b>	0.57 (0.07)	0.56 (0.08)	0.56 (0.08)	0.57 (0.07)	0.466 <sup>a</sup>
<b>VAI</b>	5.69 (3.74)	5.47 (3.81)	5.27 (3.90)	5.01 (3.37)	0.269 <sup>a</sup>
<b>BRI</b>	4.89 (1.70)	4.75 (1.89)	4.78 (1.71)	4.95 (1.68)	0.602 <sup>a</sup>
<b>BAI</b>	33.20 (6.74)	33.08 (7.48)	31.88 (6.60)	31.47 (6.60)	0.018 <sup>a</sup>
<b>LAP</b>	51.20 (33.35)	52.12 (32.40)	51.99 (32.53)	51.09 (33.07)	0.992 <sup>a</sup>
(cm.mmol/L)					
<b>TyG</b>	8.74 (0.59)	8.85 (0.62)	8.82 (0.63)	8.81 (0.53)	0.266 <sup>a</sup>

Abbreviations: Q, quartiles; BMI, body mass index; kg/m<sup>2</sup>, Kilogram per square meter; WC, waist circumference; cm, centimeter; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio; VAI, visceral adiposity index; BRI, Body roundness index BAI, body adiposity index; LAP, lipid accumulation product; mmol, milli mole; L, liter; TyG, triglyceride–glucose index

The data are presented as mean ± standard deviation (SD) or numbers (percent)

a ANOVA, b Pearson Chi–Square

BMI, WHtR, WC, BRI, BAI, and LAP. For TyG, the risk increased by 10%, but after that, it declined. A higher intake of UPF was related to the lower amount of VAI. WHR showed insignificant results.

Similarly to the results of our study, the study by Sukyoung Jung et al. on Korean adults concluded there was an association between higher intake of UPFs, higher

adiposity, and lower skeletal muscle mass [45]. A cohort study by Mengxi et al. concluded that increased consumption of UPFs was linked to a slight but significant rise in BMI [46]. Marie Beslay et al., in a French cohort, demonstrated that Increased intake of UPF was linked to a rise in BMI and a greater risk of being overweight and obese [47]. Junxiu Liu et al. reported that increased



**Table 2** Ultra-processed food consumption and relative contribution of food groups

Quartiles of ultra-processed food intake					
Variables	Q1 (0-24.9%)	Q2 (25-49.9%)	Q3 (50-74.9%)	Q4 (75-100%)	P-value
	(n = 212)	(n = 213)	(n = 213)	(n = 212)	
Food groups (Kcal/d)					
Hydroge- nated fat, mayon- naise, and margarine	9.76 (16.11)	31.90 (40.05)	88.88 (90.65)	243.27 (243.60)	0.001>
Sweets snacks <sup>a</sup>	20.04 (21.40)	43.41 (35.30)	73.77 (80.66)	134.25 (138.47)	0.001>
Salty snacks <sup>b</sup>	4.64 (9.04)	23.00 (30.09)	42.07 (61.38)	91.33 (155.25)	0.001>
Dairy prod- ucts <sup>c</sup>	5.76 (9.04)	15.10 (17.52)	27.37 (37.59)	49.48 (65.11)	0.001>
Processed meat <sup>d</sup>	3.44 (6.80)	6.31 (11.95)	8.01 (14.13)	22.63 (41.99)	0.001>
Soft drink	2.34 (6.19)	5.44 (9.23)	12.70 (29.10)	24.12 (43.09)	0.001>
Creamy cheese	3.66 (10.10)	15.26 (27.42)	25.67 (49.44)	45.18 (73.55)	0.001>
Energy from UPF (Kcal/d)	49.67 (29.81)	140.55 (39.72)	278.49 (175.27)	610.28 (286.89)	0.001>
% of total energy from UPF	2.16 (1.20)	6.15 (1.21)	12.20 (2.45)	32.13 (15.91)	0.001>

Abbreviations: Q, quartiles; <sup>a</sup> includes cakes, biscuits, candies, and chocolate; <sup>b</sup> potato chips and pufak; <sup>c</sup> includes ice creams and chocolate milk; <sup>d</sup> includes sausages, burgers, and cut colds

The data are presented as mean ± standard deviation (SD)

intake of UPFs was linked to more body fat levels in the abdominal area of U.S. adults [48]. Another study on Brazilian women showed that a higher intake of UPF is related to higher fat mass [49]. Camila Zancheta et al., in a study that assessed UPF intake and adiposity indicators, reported a significant association between UPF intake and an increase in the risk of higher BM, WCP, and percentage body fat [50]. However, the study results by Crisóstomo et al. showed that BMI and WC had no significant association with UPF consumption in older people and adults [51]. As this study expected a considerable relation, they considered the reverse causality observed could be related to potential changes in dietary habits and lifestyle, as well as the underreporting or exclusion of certain food items by participants. A different cross-sectional study in the United Kingdom yielded similar findings, revealing no positive correlation between UPF consumption and BMI. The authors attribute this outcome to the combined analysis of processed and ultra-processed food groups [52]. Based on our research, studies on other indicators that we assessed in this study were scarce. Gholami et al., in a cross-sectional study of

overweight and obese women, assessed the NOVA score and genetic risk score and its association with BAI and lipid profile and reported significant results regarding higher BAI and NOVA scores [53]. Mirmiran et al. conducted a restricted cubic spline analysis, similar to our study, which indicated that the risk of being metabolically unhealthy while maintaining a normal weight increases progressively when UPFs account for at least 20% of total energy intake. Additionally, their findings showed a positive correlation between energy intake from UPFs and the risk of being metabolically unhealthy in both normal-weight and overweight/obese individuals [54]. For TyG, the study by Hosseininassab et al., which aimed to assess UPF consumption and cardiometabolic risk factors in overweight and obese women, showed TyG has no significant association to their UPF intake [55]. It is worth noting that participants in higher tertiles of NOVA had significantly higher TyG-WC. This study only included women who were overweight or obese, so it may have impacted the results compared to the general population. Our study revealed that the TyG increases until 10% and then decreases, so its relation is unclear and should be examined in future studies. An unpredicted result was obtained for VAI, significantly reducing higher amounts of UPF intake. This index is calculated by BMI, WC, HDL, and TG levels. Since BMI and WC revealed a significant dose-response association, we hypothesized that HDL and TG levels may be responsible for this result. As mentioned before, in our study, TyG also decreased after a 10% intake of UPF, and it seems the results related to lipid profile were insignificant. Few studies assess the association of WHR and UPF intakes. Lane et al. reported there were no significant differences in WHR between quartiles of UPF intakes among adolescent girls in northeastern Iran [56]. In another study by Cristina et al. in Brazilian adolescents, the results were the same and no significant association was found with higher intakes of UPF [57]. A possible reason why our study found no association between waist-to-hip ratio WHR and UPF consumption could be due to the complex nature of body fat distribution and the multiple factors influencing WHR beyond diet alone [58, 59]. For example, WHR is strongly influenced by genetics and hormones, particularly sex hormones like estrogen and testosterone [60]. Even with high UPF intake, genetic predisposition may play a larger role in fat distribution. It seems there is no association between WHR and UPF intakes; however, because of a small number of studies, future studies should assess this association.

The indexes examined in this study were anthropometric (including BMI, BRI, WC, VAI, and BRI), a combination of anthropometric and biochemical (including BAI, LAP), and just biochemical (TyG). As the physiology of complications in obesity is a combination of

**Table 3** The association between anthropometric indexes and the UPF categories

Variables	Quartiles of ultra-processed food intake				P trend
	Q1	Q2	Q3	Q4	
	(0–24.9%)	(25–49.9%)	(50–74.9%)	(75–100%)	
	(n = 212)	(n = 213)	(n = 213)	(n = 212)	
<b>BMI (kg/m2)</b>					
Crude	1.00	0.54 (0.36–0.83)	0.93 (0.60–1.45)	0.95 (0.61–1.79)	0.537
adjusted	1.00	0.57 (0.37–0.90)	0.95 (0.59–1.53)	0.84 (0.52–1.36)	0.983
<b>WC (cm)</b>					
Crude	1.00	1.83 (1.13–2.95)	0.91 (0.59–1.40)	1.03 (0.66–1.59)	<b>0.037</b>
adjusted	1.00	0.56 (0.33–0.94)	0.68 (0.40–1.15)	0.65 (0.381–1.06)	0.959
<b>WHR</b>					
Crude	1.00	2.11 (1.38–3.23)	0.98 (0.66–1.46)	1.09 (0.73–1.61)	<b>0.002</b>
adjusted	1.00	0.45 (0.27–0.73)	0.68 (0.42–1.12)	0.77 (0.46–1.27)	0.666
<b>WHrT</b>					
Crude		1.07 (0.63–1.08)	0.80 (0.49–1.32)	0.73 (0.45–1.20)	0.720
adjusted	1.00	0.92 (0.52–1.62)	0.76 (0.43–1.34)	0.95 (0.52–1.73)	0.720
<b>VAI</b>					
Crude	1.00	1.21 (0.82–1.77)	1.12 (0.76–1.64)	1.00 (0.68–1.46)	0.267
adjusted	1.00	0.96 (0.64–1.42)	0.99 (0.66–1.48)	1.095 (0.72–1.65)	0.659
<b>BRI</b>					
Crude	1.00	1.02 (0.65–1.59)	1.45 (0.94–2.23)	2.10 (1.38–3.19)	<b>0.001&gt;</b>
adjusted	1.00	1.24 (0.76–2.03)	1.33 (0.82–2.16)	1.70 (1.05–2.73)	0.030
<b>BAI</b>					
Crude	1.00	0.95 (0.64–1.41)	1.11 (0.75–1.65)	1.84 (1.25–2.72)	0.001
adjusted	1.00	1.09 (0.70–1.71)	0.79 (0.49–1.25)	1.10 (0.69–1.74)	0.999
<b>LAP (cm.mmol/L)</b>					
Crude	1.00	1.22 (0.83–1.79)	1.24 (0.84–1.82)	0.96 (0.65–1.41)	0.878
adjusted	1.00	1.33 (0.90–1.97)	1.33 (0.89–1.98)	0.97 (0.64–1.45)	0.939
<b>TyG</b>					
Crude	1.00	1.33 (0.90–1.97)	1.22 (0.83–1.81)	1.04 (0.70–1.53)	0.950
adjusted	1.00	1.32 (0.88–1.96)	1.24 (0.83–1.85)	1.05 (0.70–1.58)	0.824

Abbreviations: Q, quartiles; OR, odds ratio; CI, confidence interval; UPF, ultra-processed food; BMI, body mass index; kg/m<sup>2</sup>, Kilogram per square meter; WC, waist circumference; cm, centimeter; WHR, waist-to-hip ratio; WHrT, waist-to-height ratio; VAI, visceral adiposity index; BRI, Body roundness index BAI, body adiposity index; LAP, lipid accumulation product; mmol, milli mole; L, liter; TyG, triglyceride-glucose index

The data are presented as OR (95% CI)

Adjusted for age, sex, education level, occupation, marital status, smoking, supplement intake, physical activity, and energy intake

anthropometric and biochemical processes [61], LAP and BAI could cover both sides and help to better examine the effects of UPFs intakes. These two indexes, significantly increased by higher intakes of UPFs.

The mechanism that induces the association between UPF intake and higher anthropometric adiposity indexes lies in the high caloric density of these food items. UPFs are often high in calories, added sugar, and saturated fat (SFA) but low in nutrients such as fiber, and this leads to consuming more calories without feeling full and promoting weight gain [62, 63]. Diets high in UPF offer insufficient dietary fiber that prevents physiological satiety and feeling of fullness, leading to excessive eating and gradual weight gain over time [24]. High levels of sugar, unhealthy fats, and salt in UPFs can trigger reward pathways in the brain, resulting in cravings and overconsumption in individuals [64]. Other ingredients, such as artificial flavors and sweeteners, can make UPFs more

palatable and increase their appeal and consumption frequency [65, 66]. Another worthy note that we should consider is that these food items are often marketed in more significant portions, so they encourage overeating as a result [67]. Furthermore, the imbalance of macronutrients can disrupt hunger and satiety hormonal signals such as leptin and ghrelin [68]. Leptin and ghrelin are crucial hormones regulating appetite and energy balance [69, 70]. Leptin, produced by adipose tissue, signals satiety and hinders hunger, while ghrelin, secreted by the stomach, stimulates appetite [71]. A rapid blood glucose spike occurs after ingesting high levels of simple sugars, leading to hyperglycemia. After that, a sharp insulin response can change leptin sensitivity [72, 73]. Leptin signaling may be diminished by chronic high sugar intake, leading to leptin resistance where the body fails to recognize satiety cues [74]. On the other hand, high-calorie, low-nutrient UPFs may not adequately suppress ghrelin

**Table 4** The parameters of the restricted cubic spines model for outcomes of the study

Variables	Dose	Mean Difference (MD)	Standard Error (SE)	95% CI (Lower, Upper)	P-value
BMI (kg/m <sup>2</sup> )	1	-0.01	0.01	(-0.03, -0.00)	0.043
	2	0.04	0.01	(0.02, 0.06)	< 0.001
WC (cm)	1	0.12	0.02	(0.08, 0.16)	< 0.001
	2	-0.08	0.02	(-0.12, -0.04)	< 0.001
WHR	1	0.00	0.00	(-0.00, 0.00)	0.842
	2	0.00	0.00	(-0.00, 0.00)	0.926
WHtR	1	0.00	0.00	(0.00, 0.00)	< 0.001
	2	-0.00	0.00	(-0.00, -0.00)	< 0.001
VAI	1	0.00	0.01	(-0.10, 0.01)	0.733
	2	-0.02	0.01	(-0.03, -0.00)	0.007
BRI	1	0.01	0.00	(0.01, 0.02)	< 0.001
	2	-0.00	0.00	(-0.01, 0.00)	0.082
BAI	1	0.05	0.01	(0.03, 0.07)	< 0.001
	2	-0.04	0.01	(-0.06, -0.02)	< 0.001
LAP (cm.mmol/L)	1	0.35	0.06	(0.23, 0.46)	< 0.001
	2	-0.35	0.06	(-0.50, -0.23)	< 0.001
TyG	1	0.00	0.00	(0.00, 0.01)	< 0.001
	2	-0.00	0.00	(-0.01, -0.00)	< 0.001

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; WHtR, waist-to-height ratio; VAI, visceral adiposity index; BRI, Body roundness index BAI, body adiposity index; LAP, lipid accumulation product; TyG, triglyceride-glucose index

The data are presented as mean difference (MD) (95% confidence intervals (CI))

Adjusted for age, sex, education level, occupation, marital status, smoking, supplement intake, physical activity, and energy intake

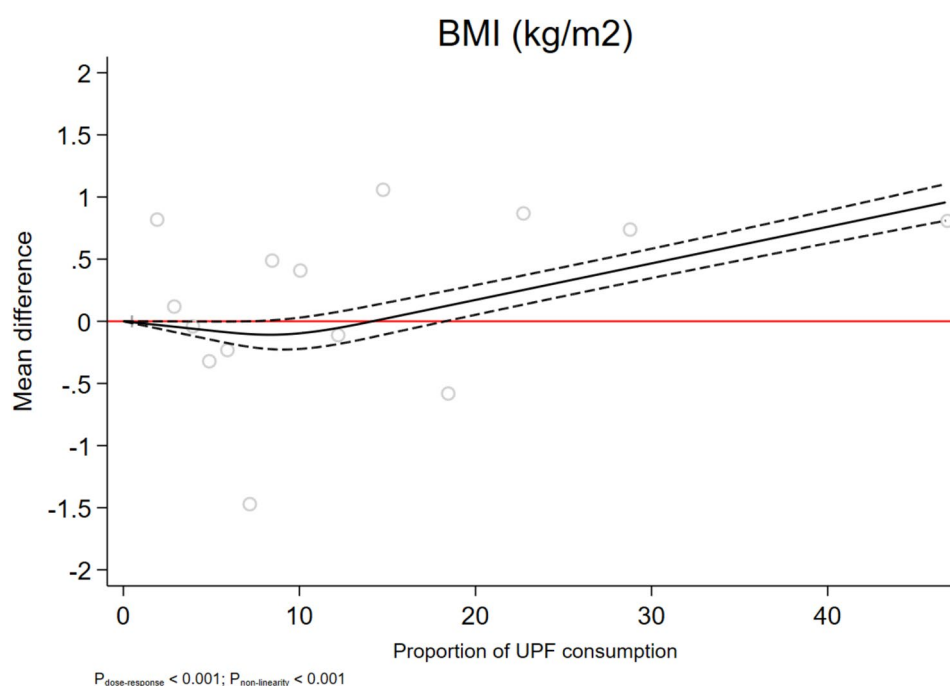
secretion, resulting in prolonged hunger signals that promote weight gain [75, 76]. Moreover, UPFs may have a disruptive effect on the standard rhythmic patterns of ghrelin secretion. Studies indicate that consuming highly palatable, energy-dense foods can alter the timing and promote ghrelin release, resulting in unhealthy eating behaviors [77, 78].

The strength of this study lies in the assessment of various adiposity-related anthropometric and biochemical indexes. Additionally, applying both linear and nonlinear methods, such as logistic regression and dose-response analysis, to all outcomes adds robustness to our findings.

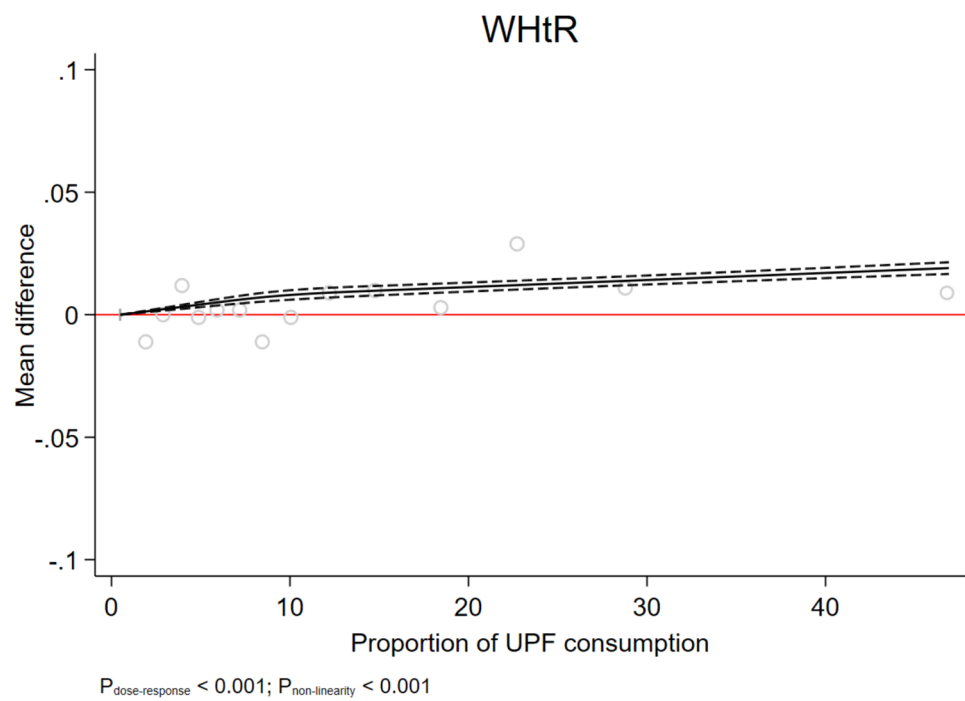
Nevertheless, some limitations should be acknowledged. First, the FFQ used in this study might be subject to reporting bias from participants. Second, we did not categorize the age groups of participants, which limited our ability to investigate the associations within specific categories (e.g., adults or the elderly). Third, the cross-sectional design of this study precludes the establishment of causal relationships. Finally, while the sample size of 850 Tehranian adults provides valuable insights, the findings may not be generalizable to broader populations.

## Conclusion

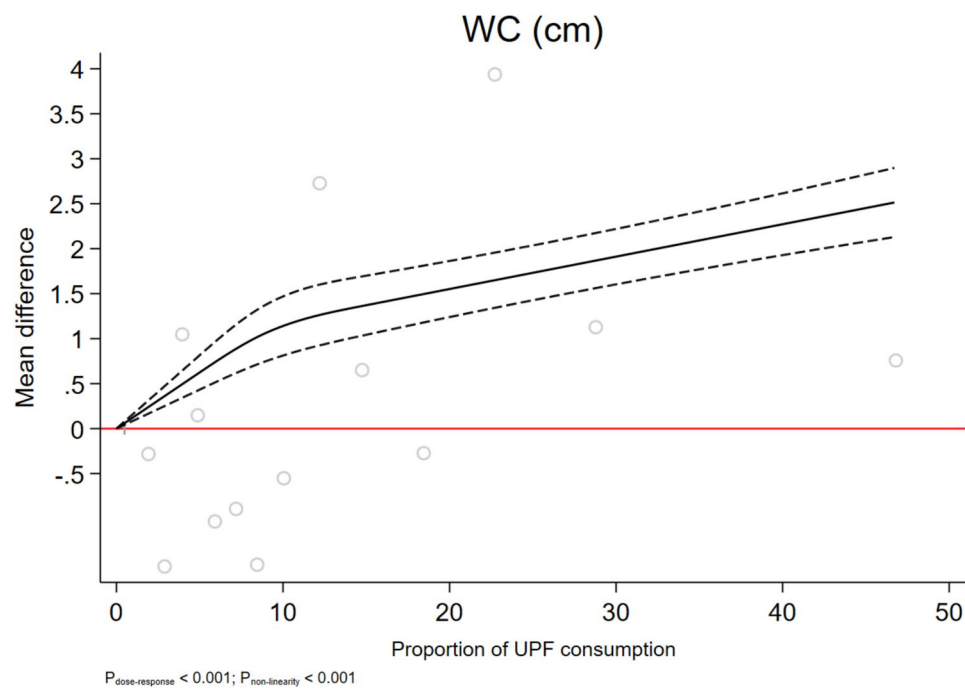
Based on this study's findings, individuals may consider moderating their intake of UPFs as part of a balanced diet. While our study observed an association between higher UPF consumption and unfavorable adiposity-related measures such as BMI, WHtR, and LAP, it is

**Fig. 1** Body mass index (BMI)

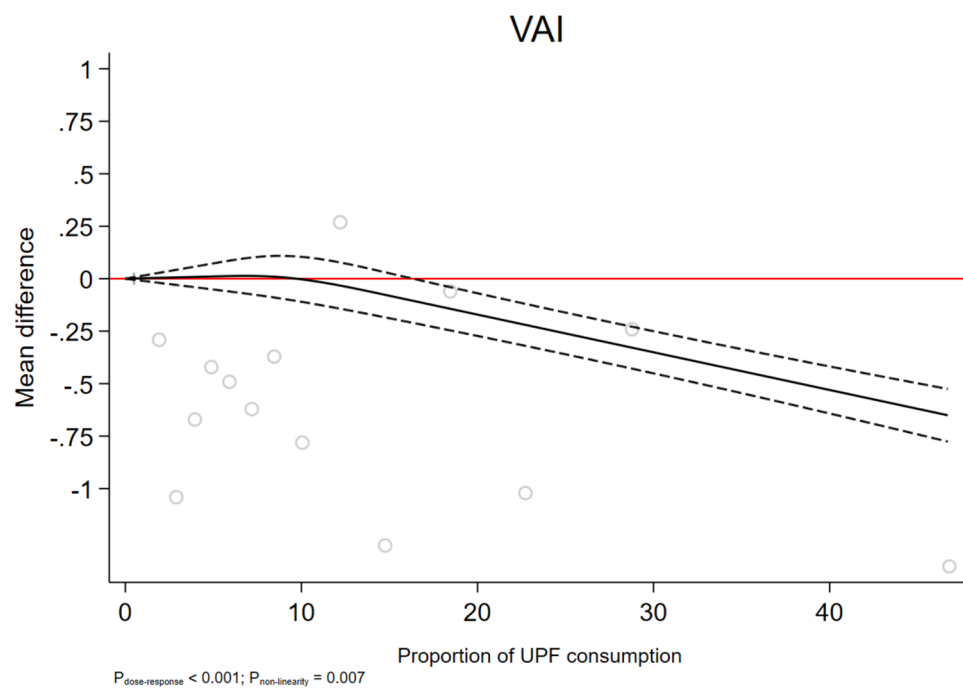




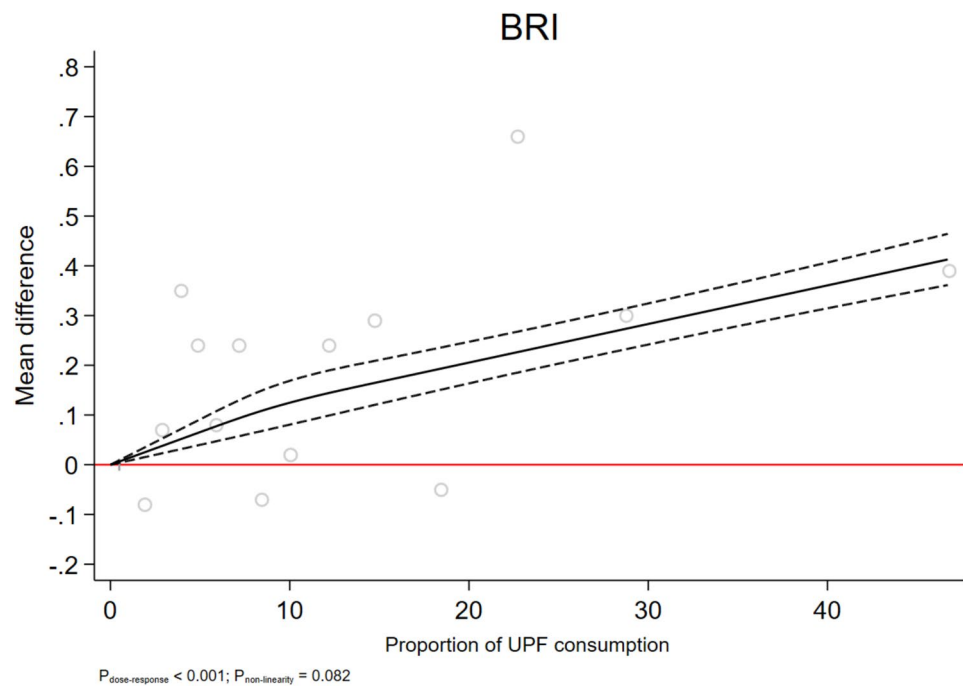
**Fig. 2** Waist-to-height ratio (WHtR)



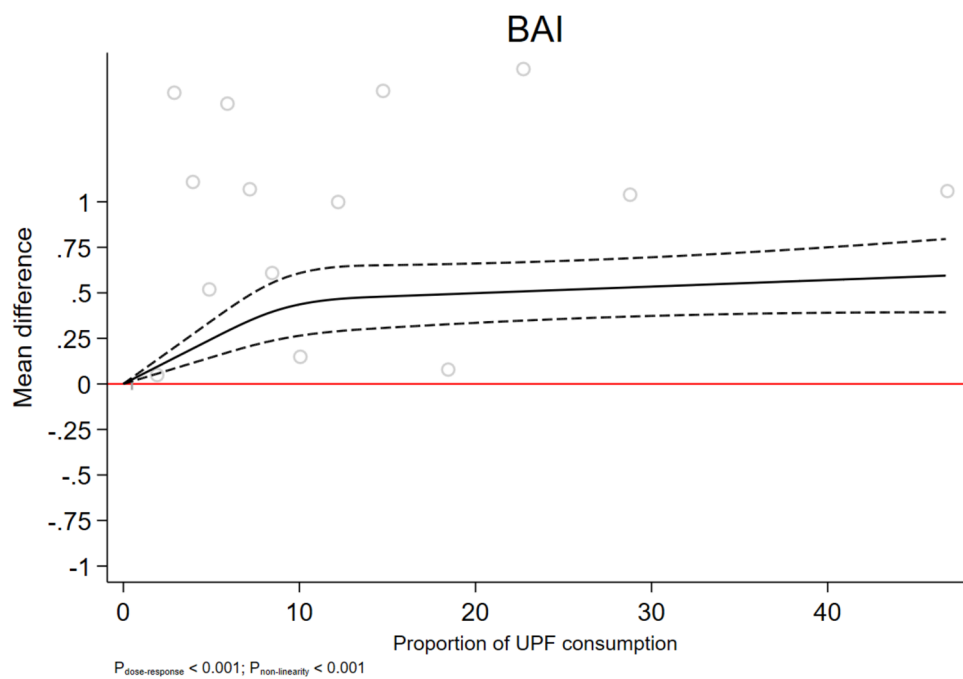
**Fig. 3** Waist circumference (WC)



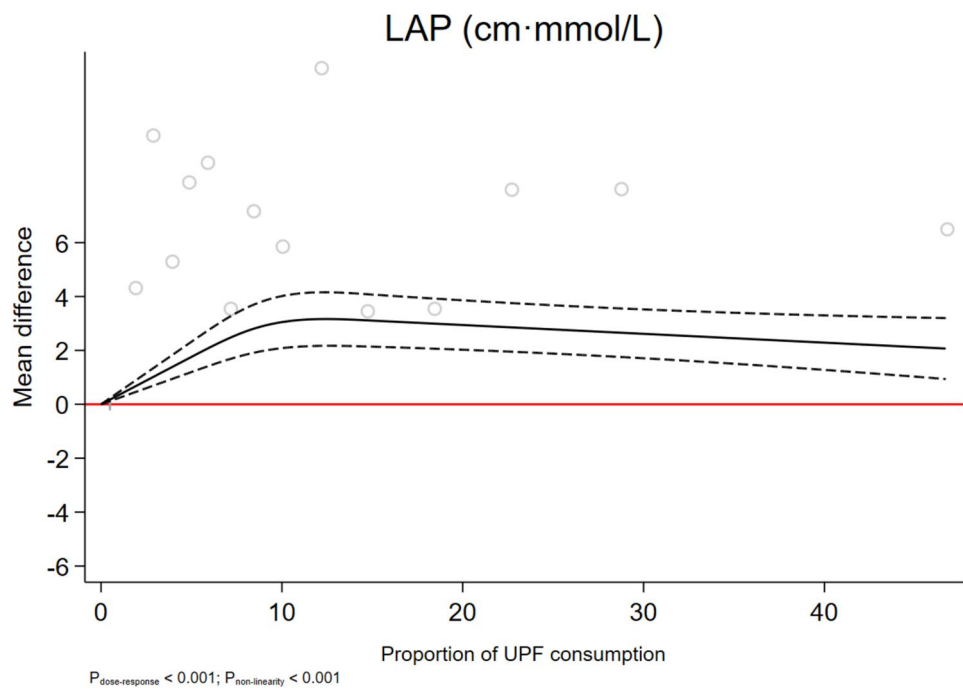
**Fig. 4** Visceral adiposity index (VAI)



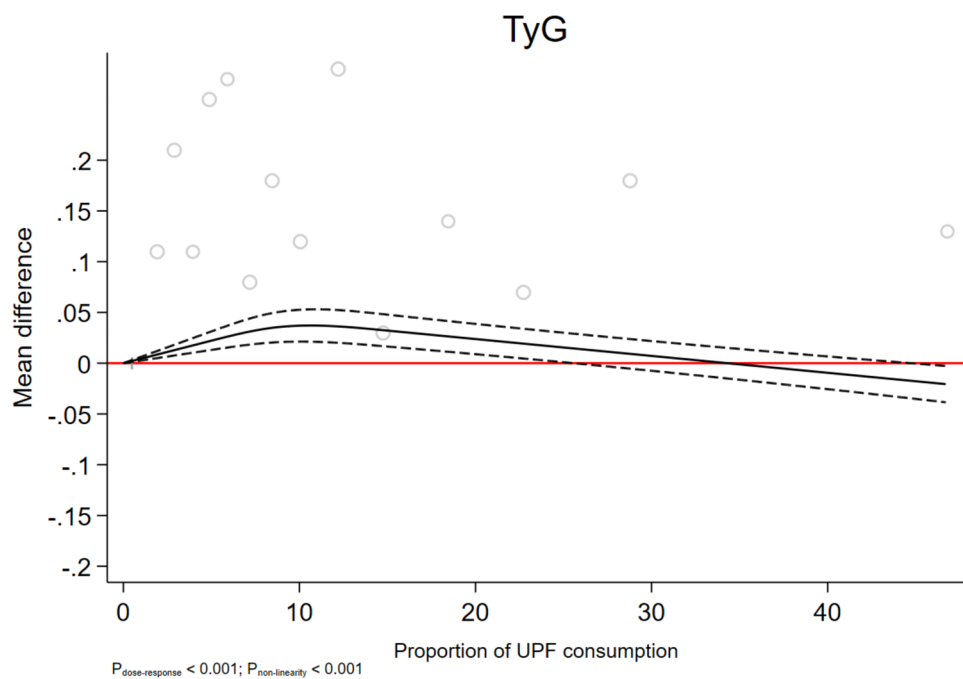
**Fig. 5** Body roundness index (BRI)



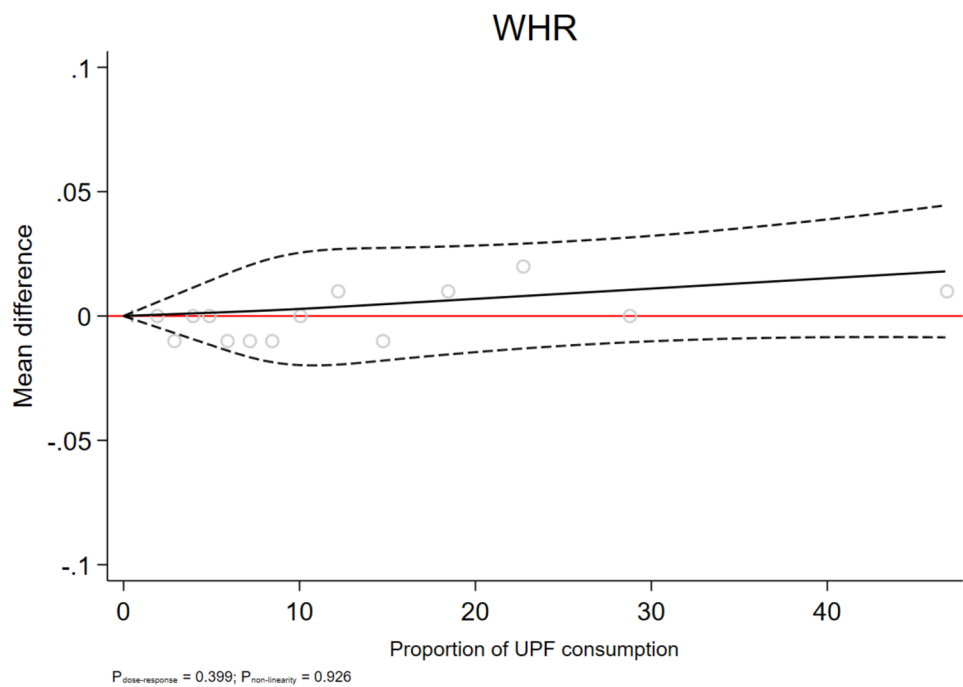
**Fig. 6** Body adiposity index (BAI)



**Fig. 7** Lipid accumulation product (LAP)



**Fig. 8** Triglyceride-glucose index (TyG)



**Fig. 9** Waist-to-hip ratio (WHR). The solid black lines indicate the estimated values, while the red dashed lines highlight the knot points, and the black dashed lines illustrate the 95% confidence intervals (CI)

important to note that this is a cross-sectional study, and causation cannot be inferred.

Nevertheless, limiting UPF intake, when feasible, may contribute to healthier dietary patterns and better overall health. Future research is needed to validate these

findings further and provide stronger evidence for public health recommendations.

**Abbreviations**

UPF	Ultra-process foods
BMI	Body mass index
WC	Waist circumference

WHR	Waist-to-hip ratio
WhtR	Waist-to-height ratio
VAI	Visceral adiposity index
BRI	Body roundness index
BAI	Body adiposity index
LAP	Lipid accumulation product
TyG	Triglyceride-glucose index
OR	Odds ratio
CI	Confidence interval
WHO	World Health Organization
UPF	Ultra-processed food
BMI	Body mass index
VAI	Visceral adiposity index
BRI	Body roundness index
BAI	Body adiposity index
LAP	Lipid accumulation product
TyG	Triglyceride-glucose index
TG	Triglycerides
CVs	Coefficient variances
WC	Waist circumference
WHR	Waist-to-hip ratio
WhtR	Waist-to-height ratio
HDL	High-density lipoprotein
IPAQ	International Physical Activity Questionnaire
RCS	Restricted cubic splines

## Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12889-025-22812-2>.

Supplementary Material 1

Supplementary Material 2

## Acknowledgements

The study was supported by the Tehran University of Medical Sciences, Tehran, Iran. The authors would also like to thank all patients who participated in this study.

## Author contributions

M.R., and S.S.B. contributed to the design. S.S.B. contributed to the data collection. M.R. and N.A. contributed to the manuscript drafting and statistical analyses and conducted research, data interpretation, and approval of the final version of the manuscript. S.S.H. has done the critical review, supervised the study, and was responsible for the final content. All authors read and approved the final manuscript.

## Funding

There was no source of funding.

## Data availability

No datasets were generated or analysed during the current study.

## Declarations

### Ethics approval and consent to participate

Medical Ethics Committee of the Tehran University of Medical Sciences, Tehran, Iran (Ethic number: IR.TUMS.MEDICINE.REC.1401.695) approved this study. This study adhered to the Declaration of Helsinki. Informed consent was obtained from all subjects.

### Consent to publish

Not applicable.

### Competing interests

The authors declare no competing interests.

Received: 22 December 2024 / Accepted: 15 April 2025

Published online: 25 April 2025

## References

- Ryan D, Barquera S, Barata Cavalcanti O, Ralston J. The global pandemic of overweight and obesity: addressing a twenty-first century multifactorial disease. *Handbook of global health*: Springer; 2021. pp. 739–73.
- Organization WH. <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight>. 2016.
- Lu Y, Hajifathalian K, Ezzati M, Woodward M, Rimm EB, Danaei G et al. Metabolic mediators of the effects of body-mass index, overweight, and obesity on coronary heart disease and stroke: a pooled analysis of 97 prospective cohorts with 1.8 million participants. 2014.
- Saltiel AR, Olefsky JM. Inflammatory mechanisms linking obesity and metabolic disease. *J Clin Invest*. 2017;127(1):1–4.
- Swinburn BA, Caterson I, Seidell JC, James WPT. Diet, nutrition and the prevention of excess weight gain and obesity. *Public Health Nutr*. 2004;7(1a):123–46.
- Swinburn BA, Kraak VI, Allender S, Atkins VJ, Baker PI, Bogard JR, et al. The global syndemic of obesity, undernutrition, and climate change: the Lancet commission report. *Lancet*. 2019;393(10173):791–846.
- Augustin MA, Riley M, Stockmann R, Bennett L, Kahl A, Lockett T, et al. Role of food processing in food and nutrition security. *Trends Food Sci Technol*. 2016;56:115–25.
- Garnett T. Food sustainability: problems, perspectives and solutions. *Proceedings of the nutrition society*. 2013;72(1):29–39.
- Monteiro CA, Moubarac JC, Cannon G, Ng SW, Popkin B. Ultra-processed products are becoming dominant in the global food system. *Obes Rev*. 2013;14:21–8.
- Forouzanfar MH, Afshin A, Alexander LT, Anderson HR, Bhutta ZA, Biryukov S, et al. Global, regional, and National comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2015: a systematic analysis for the global burden of disease study 2015. *Lancet*. 2016;388(10053):1659–724.
- Monda A, de Stefano MI, Villano I, Allocca S, Casillo M, Messina A, et al. Ultra-Processed food intake and increased risk of obesity: A narrative review. *Foods*. 2024;13(16).
- Lee G-y, Lim JH, Joung H, Yoon D. Association between ultraprocessed food consumption and metabolic disorders in children and adolescents with obesity. *Nutrients*. 2024;16(20):3524.
- Petrus RR, do Amaral Sobral PJ, Tadini CC, Gonçalves CB. The NOVA classification system: a critical perspective in food science. *Trends Food Sci Technol*. 2021;116:603–8.
- Monteiro CA, Cannon G, Moubarac J-C, Levy RB, Louzada MLC, Jaime PC. The UN decade of nutrition, the NOVA food classification and the trouble with ultra-processing. *Public Health Nutr*. 2018;21(1):5–17.
- Adams J, White M. Characterisation of UK diets according to degree of food processing and associations with socio-demographics and obesity: cross-sectional analysis of UK National diet and nutrition survey (2008–12). *Int J Behav Nutr Phys Activity*. 2015;12(1):1–11.
- Monteiro CA, Cannon G, Levy RB, Moubarac J-C, Louzada ML, Rauber F, et al. Ultra-processed foods: what they are and how to identify them. *Public Health Nutr*. 2019;22(5):936–41.
- Slimani N, Deharveng G, Southgate D, Biessy C, Chajes V, Van Bakel M, et al. Contribution of highly industrially processed foods to the nutrient intakes and patterns of middle-aged populations in the European prospective investigation into Cancer and nutrition study. *Eur J Clin Nutr*. 2009;63(4):S206–25.
- Monteiro CA, Moubarac J-C, Levy RB, Canella DS, da Costa Louzada ML, Cannon G. Household availability of ultra-processed foods and obesity in nineteen European countries. *Public Health Nutr*. 2018;21(1):18–26.
- Haghighatdoost F, Hajhashemi P, Mohammadifard N, Najafi F, Farshidi H, Lotfzadeh M, et al. Association between ultra-processed foods consumption and micronutrient intake and diet quality in Iranian adults: a multicentric study. *Public Health Nutr*. 2023;26(2):467–75.
- Nardocci M, Leclerc B-S, Louzada M-L, Monteiro CA, Batal M, Moubarac J-C. Consumption of ultra-processed foods and obesity in Canada. *Can J Public Health*. 2019;110:4–14.
- Mendonça RD, Pimenta AM, Gea A, de la Fuente-Arillaga C, Martinez-Gonzalez MA, Lopes ACS, et al. Ultraprocessed food consumption and risk of overweight and obesity: the university of Navarra Follow-Up (SUN) cohort study. *Am J Clin Nutr*. 2016;104(5):1433–40.
- Silva FM, Giatti L, de Figueiredo RC, Molina MCB, de Oliveira Cardoso L, Duncan BB, et al. Consumption of ultra-processed food and obesity: cross sectional results from the Brazilian longitudinal study of adult health (ELSA-Brasil) cohort (2008–2010). *Public Health Nutr*. 2018;21(12):2271–9.



23. Juul F, Martinez-Steele E, Parekh N, Monteiro CA, Chang VW. Ultra-processed food consumption and excess weight among US adults. *Br J Nutr*. 2018;120(1):90–100.
24. Hall KD, Ayuketah A, Brychta R, Cai H, Cassimatis T, Chen KY, et al. Ultra-processed diets cause excess calorie intake and weight gain: an inpatient randomized controlled trial of ad libitum food intake. *Cell Metabol*. 2019;30(1):67–77. e3.
25. Duan Y, Zhang W, Li Z, Niu Y, Chen Y, Liu X, et al. Predictive ability of obesity- and lipid-related indicators for metabolic syndrome in relatively healthy Chinese adults. *Front Endocrinol*. 2022;13:1016581.
26. Witarto BS, Witarto AP, Visuddho V, Wungu CDK, Maimunah U, Rejeki PS, et al. Gender-specific accuracy of lipid accumulation product index for the screening of metabolic syndrome in general adults: a meta-analysis and comparative analysis with other adiposity indicators. *Lipids Health Dis*. 2024;23(1):198.
27. Akbarzade Z, Djafarian K, Saeidifard NN, Majd SA, Garousi N, Samadi F, et al. The association between lunch composition and obesity in Iranian adults. *Br J Nutr*. 2022;127(10):1517–27.
28. Kiadaliri AA, Jafari M, Mahdavi M-RV, Faghihzadeh S, Kalantari N, Asadi-Lari M. The prevalence of adulthood overweight and obesity in Tehran: findings from urban HEART-2 study. *Med J Islamic Repub Iran*. 2015;29:178.
29. Haytowitz D, Lemar L, Pehrsson P, Exler J, Patterson K, Thomas R, et al. USDA National nutrient database for standard reference, release 24. Washington, DC, USA: US Department of Agriculture; 2011.
30. Baveicy K, Mostafaei S, Darbandi M, Hamzeh B, Najafi F, Pasdar Y. Predicting metabolic syndrome by visceral adiposity index, body roundness index and a body shape index in adults: a cross-sectional study from the Iranian RaNCD cohort data. *Metabolic Syndrome and Obesity: Diabetes*; 2020. pp. 879–87.
31. Freedman DS, Thornton JC, Pi-Sunyer FX, Heymsfield SB, Wang J, Pierson RN Jr, et al. The body adiposity index (hip circumference ÷ height<sup>1.5</sup>) is not a more accurate measure of adiposity than is BMI, waist circumference, or hip circumference. *Obesity*. 2012;20(12):2438–44.
32. Raposo MA, Guimaraes NS, Tupinambás U. Lipid accumulation product index to predict metabolic syndrome in people living with HIV. *Clin Med Res*. 2020;18(4):120–5.
33. Tao L-C, Xu J-n, Wang T-t, Hua F, Li J-J. Triglyceride-glucose index as a marker in cardiovascular diseases: landscape and limitations. *Cardiovasc Diabetol*. 2022;21(1):68.
34. Moghaddam MB, Aghdam FB, Jafarabadi MA, Allahverdipour H, Nikookheslat SD, Safarpour S. The Iranian version of international physical activity questionnaire (IPAQ) in Iran: content and construct validity, factor structure, internal consistency and stability. *World Appl Sci J*. 2012;18(8):1073–80.
35. Shah NR, Braverman ER. Measuring adiposity in patients: the utility of body mass index (BMI), percent body fat, and leptin. *PLoS ONE*. 2012;7(4):e33308.
36. Ahmad N, Adam SIM, Nawi AM, Hassan MR, Ghazi HF. Abdominal obesity indicators: waist circumference or waist-to-hip ratio in Malaysian adults population. *Int J Prev Med*. 2016;7(1):82.
37. Lean M, Han T, Morrison C. Waist circumference as a measure for indicating need for weight management. *BMJ*. 1995;311(6998):158–61.
38. Browning LM, Hsieh SD, Ashwell M. A systematic review of waist-to-height ratio as a screening tool for the prediction of cardiovascular disease and diabetes: 0–5 could be a suitable global boundary value. *Nutr Res Rev*. 2010;23(2):247–69.
39. Gupta S, Kapoor S. Body adiposity index: its relevance and validity in assessing body fatness of adults. *Int Sch Res Notices*. 2014;2014.
40. Rajendran S, Padikkal AKK, Mishra S, Madhavanpillai M. Association of lipid accumulation product and Triglyceride-Glucose index with metabolic syndrome in young adults: A Cross-sectional study. *Int J Endocrinol Metabolism*. 2022;20(2).
41. Lam BCC, Koh GCH, Chen C, Wong MTK, Fallows SJ. Comparison of body mass index (BMI), body adiposity index (BAI), waist circumference (WC), waist-To-Hip ratio (WHR) and waist-To-Height ratio (WHR) as predictors of cardiovascular disease risk factors in an adult population in Singapore. *PLoS ONE*. 2015;10(4):e0122985.
42. Jablonowska-Lietz B, Wrzosek M, Włodarczyk M, Nowicka G. New indexes of body fat distribution, visceral adiposity index, body adiposity index, waist-to-height ratio, and metabolic disturbances in the obese. *Pol Heart J (Kardiologia Polska)*. 2017;75(11):1185–91.
43. Feng J, He S, Chen X. Body adiposity index and body roundness index in identifying insulin resistance among adults without diabetes. *Am J Med Sci*. 2019;357(2):116–23.
44. Durrleman S, Simon R. Flexible regression models with cubic splines. *Stat Med*. 1989;8(5):551–61.
45. Jung S, Seo J, Kim JY, Park S. Associations of Ultra-Processed food intake with body fat and skeletal muscle mass by sociodemographic factors. *Diabetes & Metab J*. 2024.
46. Du M, Wang L, Martín-Calvo N, Dhana K, Khandpur N, Rossato SL et al. Ultraprocessed food intake and body mass index change among youths: a prospective cohort study. *Am J Clin Nutr*. 2024;120(4):836–845.
47. Beslay M, Srour B, Méjean C, Allès B, Fiolet T, Debras C, et al. Ultra-processed food intake in association with BMI change and risk of overweight and obesity: A prospective analysis of the French NutriNet-Santé cohort. *PLoS Med*. 2020;17(8):e1003256.
48. Liu J, Steele EM, Li Y, Yi SS, Monteiro CA, Mozaffarian D. Consumption of ultraprocessed foods and body fat distribution among U.S. Adults. *Am J Prev Med*. 2023;65(3):427–38.
49. Rudakoff LCS, Magalhães EIS, Viola PCAF, de Oliveira BR, da Silva Coelho CCN, Bragança MLBM et al. Ultra-processed food consumption is associated with increase in fat mass and decrease in lean mass in Brazilian women: A cohort study. *Front Nutr*. 2022;9.
50. Shim JS, Ha KH, Kim DJ, Kim HC. Diet quality partially mediates the association between ultraprocessed food consumption and adiposity indicators. *Obesity*. 2023;31(9):2430–9.
51. Crisóstomo JM, Rodrigues LARL, Nascimento LM, Viola PCAF, Frota KdMG. Consumption of ultra-processed foods and anthropometric indicators in adolescents, adults, and the elderly in a capital City in Northeastern Brazil. *Revista De Nutricao*. 2022;35:e210052.
52. Adams J, White M. Characterisation of UK diets according to degree of food processing and associations with socio-demographics and obesity: cross-sectional analysis of UK National diet and nutrition survey (2008–12). *Int J Behav Nutr Phys Activity*. 2015;12:1–11.
53. Gholami F, Lesani A, Soveid N, Rasaei N, Samadi M, Bahrampour N, et al. The interaction between ultra-processed foods and genetic risk score on body adiposity index (BAI), appendicular skeletal muscle mass index (ASM), and lipid profile in overweight and obese women. *Aspects Mol Med*. 2024;3:100044.
54. Mirmiran P, Moslehi N, Golzarand M, Azizi F. Ultra-processed foods consumption and the risk of metabolically unhealthy phenotype in normal-weight and overweight/obese adults: a prospective investigation. *Int J Food Sci Nutr*. 2023;74(4):522–31.
55. Hosseiniinasab D, Shiraseb F, Noori S, Jamili S, Mazaheri-Eftekhari F, Dehghan M, et al. The relationship between ultra-processed food intake and cardio-metabolic risk factors in overweight and obese women: a cross-sectional study. *Front Nutr*. 2022;9:945591.
56. Lane KE, Davies IG, Darabi Z, Ghayour-Mobarhan M, Khayyatizadeh SS, Mazidi M. The association between Ultra-Processed foods, quality of life and insomnia among adolescent girls in Northeastern Iran. *Int J Environ Res Public Health*. 2022;19(10):6338.
57. Enes CC, Camargo CMD, Justino MIC. Ultra-processed food consumption and obesity in adolescents. *Revista De Nutricao*. 2019;32:e180170.
58. Després J-P. Body fat distribution and risk of cardiovascular disease: an update. *Circulation*. 2012;126(10):1301–13.
59. Piché M-E, Poirier P, Lemieux I, Després J-P. Overview of epidemiology and contribution of obesity and body fat distribution to cardiovascular disease: an update. *Prog Cardiovasc Dis*. 2018;61(2):103–13.
60. Bovet J. Evolutionary theories and Men's preferences for women's waist-to-hip ratio: which hypotheses remain? A systematic review. *Front Psychol*. 2019;10:1221.
61. Mafor TT, Rufino R, Costa CH, Lopes AJ. Obesity: systemic and pulmonary complications, biochemical abnormalities, and impairment of lung function. *Multidisciplinary Respiratory Med*. 2016;11:1–11.
62. Marrón-Ponce JA, Flores M, Cediel G, Monteiro CA, Batis C. Associations between consumption of ultra-processed foods and intake of nutrients related to chronic non-communicable diseases in Mexico. *J Acad Nutr Dietetics*. 2019;119(11):1852–65.
63. Rauber F, Louzada MLC, Steele EM, Millett C, Monteiro CA, Levy RB. Ultra-processed food consumption and chronic non-communicable diseases-related dietary nutrient profile in the UK (2008–2014). *Nutrients*. 2018;10(5):587.
64. Oruçoğlu B. Ultra-processed foods içig. and Health. *Current Research in Health Sciences*. 2022.
65. Forde CG. Beyond ultra-processed: considering the future role of food processing in human health. *Proc Nutr Soc*. 2023;82(3):406–18.
66. Teo PS, Tso R, van Dam RM, Forde CG. Taste of modern diets: the impact of food processing on nutrient sensing and dietary energy intake. *J Nutr*. 2022;152(1):200–10.

67. Kraak VI, Davy BM. Multisectoral strategies needed to Establish healthy portion size norms that disincentivize hyperpalatable, energy-dense foods and sugary beverages in food environments linked to obesity and diet-related chronic diseases in the united States. *Curr Developments Nutr*. 2023;7(2):100012.
68. Kelly AL, Baugh ME, Oster ME, DiFeliceantonio AG. The impact of caloric availability on eating behavior and ultra-processed food reward. *Appetite*. 2022;178:106274.
69. Khatib MN, Khatib M, Gaidhane S, Gaidhane A, Zahiruddin QS. Ghrelin for regulating appetite and energy balance: A systematic review. *Natl J Physiol Pharm Pharmacol*. 2014;4(3):101–5.
70. Friedman JM. Leptin and the endocrine control of energy balance. *Nat Metabolism*. 2019;1(8):754–64.
71. Kharbanda C, Bansal S, Aneja PS. Role and significance of Ghrelin and leptin in hunger, satiety, and energy homeostasis. *J Sci Soc*. 2022;49(1):12–6.
72. Moradi S, Ma HK, Bagheri R, Mohammadi H, Jayedi A, Lane MM, et al. Ultra-processed food consumption and adult diabetes risk: a systematic review and dose-response meta-analysis. *Nutrients*. 2021;13(12):4410.
73. Fardet A, Rock E. Ultra-processed foods: a new holistic paradigm? *Trends Food Sci Technol*. 2019;93:174–84.
74. Calcaterra V, Cena H, Rossi V, Santero S, Bianchi A, Zuccotti G. Ultra-processed food, reward system and childhood obesity. *Children*. 2023;10(5):804.
75. Contreras-Rodriguez O, Solanas M, Escorihuela RM. Dissecting ultra-processed foods and drinks: do they have a potential to impact the brain?. *Rev Endocr Metab Disord*. 2022;23(4):697–717.
76. Cummings DE. Ghrelin and the short-and long-term regulation of appetite and body weight. *Physiol Behav*. 2006;89(1):71–84.
77. Sirohi S, Van Cleef A, Davis J. Patterned feeding induces neuroendocrine, behavioral and genetic changes that promote palatable food intake. *Int J Obes*. 2017;41(3):412–9.
78. Stover PJ, Field MS, Andermann ML, Bailey RL, Batterham RL, Cauffman E, et al. Neurobiology of eating behavior, nutrition, and health. *J Intern Med*. 2023;294(5):582–604.

## Publisher's note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.