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The association between ultra-processed foods intake and the odds of prostate cancer: a case–control study

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Abstract

Background Ultra-processed foods (UPFs) are characterized by poor nutritional composition and the generating neo-formed carcinogens during high levels of processing. The current study aimed to investigate the association between UPFs consumption and the odds of prostate cancer (PC).

Methods This case–control study recruited 62 PC cases and 63 hospital-based controls from two major referral hospitals of Shiraz, Iran, in 2015. Eligible men, newly diagnosed with PC through histological confirmation, were included as cases. Along with demographic and anthropometric information data, participant's dietary intake was assessed using a semi-quantitative food frequency questionnaire. The NOVA classification was employed to categorize food items based on their level of industrial processing. The association between UPFs consumption (as a percentage of daily calorie intake) and the odds of developing PC was estimated using logistic regression models. BMI, education, physical activity, age, and fiber intake were considered confounders in the adjusted model.

Results The study included 60 cases and 60 controls, with mean UPFs intake of 8.3% and 6.4%, respectively. The crude analysis showed no significant association between UPFs intake and PC odds ratio (OR) (OR = 1.96, confidence interval (CI) 95%: 0.94–4.05, $P = 0.069$). However, after adjusting for potential confounders, the association became significant, with high versus low UPFs intake associated with 2.81 times higher odds of PC (OR = 2.81, CI 95%: 1.18–6.65, $P = 0.019$).

Conclusion Our findings highlight UPFs consumption as a factor associated with higher odds of PC in the Iranian male population. The study emphasizes the importance of monitoring industrial food processing practices and implementing measures to reduce UPFs consumption.

Keywords Diet, Ultra-processed foods, Prostate cancer, Case–control studies

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Introduction

Prostate cancer (PC) accounts for the fifth leading cause of cancer-related death globally [1] and is the second most common cancer among males in Iran [2]. According to a systematic review conducted in 2019, the incidence rate of PC in Iran was estimated at 8.7 per 100,000 population [3]. Several risk factors have been associated with increased PC risk, including age, race, family history, smoking [4], and dietary factors such as total, saturated, and trans fats [5–7], processed meats [8, 9], and adherence to a western-type dietary pattern [10]. Given the high morbidity of PC, its prolonged clinical course, and the potential for recurrence [1, 11], investigation the dietary risk factors and the protective factors like fiber [12] and plant-based diets [13] on PC risk has garnered increasing attention [14].

Ready-to-eat processed foods have become dominant in the global food system, serving as a key driver of industrialization during the nutrition transition from traditional fresh diets to those higher in animal-sourced processed foods, saturated fats, refined carbohydrates, and caloric sweeteners [15, 16]. Ultra-processed foods (UPFs), initially classified under the NOVA food classification system developed by Monteiro et al., account for over half of total dietary energy intake in high-income countries and between one-fifth and one-third in middle-income countries [17]. These foods are characterized by high levels of calories, sodium, added sugar, and saturated fats, with minimal or no fiber or whole food content [18, 19]. Consequently, they contribute to cardiometabolic abnormalities, particularly obesity and type 2 diabetes—two established risk factors for various diseases, including cancer [20, 21]. Beyond issues related to nutritional composition, UPFs pose additional risks due to chemical compounds used in packaging, such as bisphenol A (BPA) and phthalates, which have been linked to impaired reproductive function, endocrine disruption, and cancer [22, 23]. Furthermore, high temperature cooking used in their preparation can generate neo-formed carcinogens, including acrylamide, polycyclic aromatic hydrocarbons (PAH), and furan, which may impair the genetic information, hormone regulation, reproductive health, and ultimately contribute to cancer development [22, 24, 25].

Given the above, the carcinogenic properties of UPFs have been hypothesized and consistently supported by several large-scale studies [26–28]. Although limited studies have examined the impact of UPFs consumption on specific cancer types, results vary depending on tumor sites such as breast [29, 30], colorectal [31, 32], and PC [33].

In the case of PC, Diets rich in UPFs often align with higher consumption of dietary risk factors and lower intakes of protective nutrients [34–36]. Despite this, limited evidence exists on the relationship between UPFs consumption and PC. Two prospective large-scale studies conducted in UK [27] and Europe [28] found positive associations between UPFs consumption and the incidence of overall cancer and certain site-specific cancers, but not for PC. In contrast, research by Trudeau et al. in Canada [33] reported an increased odds of PC associated with higher processed foods consumption but not UPFs.

To the best of our knowledge, no studies have examined this relationship in Iranians or other Asian populations. Given the scarcity of research on this topic, which predominantly focuses on Western population, and considering the similarity between nutritional profile of UPFs and established dietary risk factors for PC, our study aimed to investigate the association between UPFs consumption and PC in the Iranian male population.

Method

Participants

The current case–control study was conducted in two major referral hospitals for urological disorders—Namazi and Shahid Faghihi—in Shiraz, Iran, between April and September 2015. Patients with histologically confirmed, newly diagnosed PC and candidates for radical or open prostatectomy were recruited as cases. Controls were randomly selected from patients admitted to the same hospitals for non-neoplastic and non-diabetes conditions, including eye (n = 21), ENT (ear, nose, and throat) ($n = 20$), kidney ($n = 8$), nervous system ($n = 5$) and gastrointestinal ($n = 9$) disorders.

All controls underwent a detailed clinical evaluation, including medical history, current symptoms, physical signs, and laboratory tests, by their specialist to confirm the absence of PC. Exclusion criteria included a history of metabolic disorders, cancer at other sites, or adherence to special dietary regimens for chronic diseases within the last year. Ultimately, 62 PC cases and 63 controls were included in the study. Additional exclusion criteria were total energy intake of < 800 or > 4,200 kcal/day and poor response to the food frequency questionnaire (FFQ), defined as not responding to > 70 items [37].

The study sample size was calculated based on a prior study by Askari et al. [38]. Ethical approve for this research was obtained from the Ethics Committee of Shiraz University of Medical Sciences (IR.SUMS.REC.1394.S438). Some details of the present study have been published previously [39, 40].

Data collection

Patient medical records were accessed through the cancer registry database of the hospitals, which are the largest and frequently referred medical centers in southern

Iran, serving patients from various provinces for all types of diseases, including cancer [41]. In addition to medical records, trained research staff collected demographic information and dietary intakes via face-to-face interviews and conducted anthropometric measurements.

General information collected included ethnicity, education level, physical activity level, and medication use. Weight was measured to the nearest 0.1 kg using a digital scale (Glamor BS-801, Hitachi, China) and height was measured to the nearest 0.1 cm using a non-stretchable tape. Measurements were taken from upright standing participants wearing light clothing and no shoes. Body mass index (BMI) was calculated using the formula: weight (kg)/height(m)^2.

To evaluate the dietary consumption of participants, a valid and reliable semi-quantitative FFQ was used [42, 43]. The serving sizes of FFQ items were presented to participants using a validated food album and a set of household measuring tools (e.g., teaspoon, tablespoon, spatula, glass, cup, bowl, and plate) [44]. Participants then recorded their consumption frequency for each item in terms of daily, weekly, monthly, or annual intake. The FFQ was previously described in detail by Jalilpiran et al. along with its analyze approach [45].

To estimate food quantities in gram weights, two software applications, Borland Delphi Professional version 7.0 and Visual Basic 2008 (VB 9.0), were used. Total daily energy intake (TDEI) and the dietary content of macronutrients and various micronutrients were subsequently extracted using Nutritionist IV software.

UPFs intake

The purpose and extent of industrial food processing serve as the basis for categorizing food into four classes in NOVA classification: (1) unprocessed or minimally processed foods, (2) processed culinary ingredients, (3) processed foods, and (4) UPFs [17]. As outlined in the Food and Agriculture Organization of the United Nations Guideline [46] for using the NOVA classification, the distinction between UPFs and the first three groups lies in the use of more intensive processes methods, such as chemical modification, extrusion, moulding, and prefrying, compared to milder processes like drying, squeezing, grinding, fractioning, steaming, boiling, roasting, pasteurization, freezing, vacuum packaging, non-alcoholic fermentation, pressing, refining, extracting, canning and bottling.

Also, certain ingredients can help distinguish UPFs from the other three NOVA groups, including high-fructose corn syrup, invert sugar, maltodextrin, dextrose, hydrogenated or interesterified oils, flavours, colours, emulsifiers, artificial sweeteners, thickeners, bulking, carbonating, and glazing agents.

In line with the above, a team of experienced nutritionists selected UPFs items from the administered FFQ and categorized them into six subgroups: (1) non-dairy beverages (artificial fruit drinks and carbonated soft drinks) (2) cookies and cakes (mass-produced industrial breads, confectionary, biscuits, creamy and non-creamy pastries, muffins, pies, pancakes, cookies, cakes) (3) dairy beverages (sugar-sweetened milk-based drinks, ice cream, cream cheese) (4) fast foods and processed meats (French fries, pizza, burger, sausages and other reconstituted meat products) (5) sweets (chocolate, candies, jam, jelly, Sohan, Gaz, sweet tahini halva) (6) others (packaged salty snacks, sauces, margarine). The daily energy contribution of UPFs was estimated as a percentage of TDEI, and for the subgroups, as a percentage of total UPFs consumption.

Statistical analysis

Data analysis was performed using SPSS software, version 22 (SPSS Inc., Chicago, IL, USA). The Kolmogorov–Smirnov test was applied to assess the normality of data distribution. For continuous variables with normal distribution, the Independent Samples *T*-test was used, and the mean ± standard deviation (SD) was reported. The Mann–Whitney test was applied to variables without a normal distribution, with the median (interquartile range (IQR)) reported. Categorical variables were analyzed using the Chi-square test. Regression models were used to examine the association between UPFs and PC, adjusting for age, BMI, fiber intake, education, and physical activity in the final model. A two-tailed *P*-value of less than 0.05 was considered statistically significant.

Results

Due to inadequate responses to the FFQ, two cases and three controls were excluded from the study, resulting in a final sample of 60 cases and 60 controls, with a response rate of 96%.

Table 1 presents a comparison of the general characteristics between cases and controls. Cases were significantly older, with a median (IQR) age of 65.5 (13.0) years compared to 60.0 (11.5) years for controls (p-value = 0.003). Additionally, cases exhibited significantly lower physical activity levels: only 20% of cases engaged in high levels of physical activity compared to 40% of controls, and 38.3% of cases were never or less active compared to 20% of controls (p-value = 0.024). The median (IQR) UPFs contribution to TDEI was 8.3% (6.7) among cases and 6.4% (6.6) among controls (p-value = 0.334), while the median (IQR) fiber intake was 20.1 (8.8) g/day for cases and 23.3 (11.6) g/day for controls (p-value = 0.187). However, the mean total fat intake was significantly higher in the case group (69.3 g) compared to the control group (54.4 g,

Table 1 The characteristic of the study participants between case and control groups

Variables	Cases (60)	Controls (60)	*P*-value
Age (year) ^	65.5 (13.0)	60.0 (11.5)	**0.003**
BMI (kg/m^2) *	24.8 ± 3.6	25.8 ± 3.4	0.121
UPFs (energy %)	8.3 (6.7)	6.4 (6.6)	0.311
Energy (kcal/day) *	2712.2 ± 593.5	2596.1 ± 712.7	0.334
Fiber (g/day) ^	20.1 (8.8)	23.3 (11.6)	0.187
Total fat (g/day) *	69.3 ± 37.3	54.4 ± 22.2	**0.009**
SFA (g/day) *	21.7 ± 8.2	18.5 ± 8.8	**0.045**
MUFA (g/day) ^	15.2 (8.0)	13.7 (9.3)	0.098
PUFA (g/day) ^	7.1 (3.9)	7.2 (5.5)	0.845
Ethnicity, Fars, % &	80.0	76.7	0.825
Education, % &			0.134
Illiterate and Primary	68.3	53.3	
Diploma and Academic	31.7	46.7	
Physical activity, % &			**0.024**
Never or Less	38.3	20.0	
Moderate	41.7	40.0	
High	20.0	40.0	
Lipid medication, yes, % &	10.0	10.0	1.000
HTN medication, yes, % &	31.7	21.7	0.302
Aspirin use, yes, % &	16.7	25.0	0.369

Significant values are shown in bold

BMI body mass index, *UPFs* ultra-processed foods, *SFA* saturated fatty acids, *MUFA* monounsaturated fatty acids, *PUFA* poly unsaturated fatty acids

^ Using Mann–Whitney for abnormal continuous variables and values are median (IQR)

* Using independent samples T-test for normal continuous variables and values are mean ± SD

& Using chi-square test for categorical and values are numbers and percentage

p-value = 0.009). Additionally, saturated fatty acid (SFA) intake differed significantly between the two groups, with cases consuming 21.7 g compared to 18.5 g in controls (*p*-value = 0.045).

The characteristic of the study participants with lower and higher than mean intake of UPFs between case and control groups are presented in Table 2. Among cases, individuals with higher than mean UPFs intake had higher energy intake (*p*-value = 0.005), UPFs consumption (*p*-value = < 0.001), total fat intake (*p*-value = 0.041), SFA intake (p-value = 0.001), and MUFA intake (*p*-value = 0.001) compared to those with lower than mean of UPFs intake. Among controls, higher intake of energy (*p*-value = < 0.001), UPFs (*p*-value = < 0.001), fiber (*p*-value = 0.032), total fat (*p*-value = < 0.001), SFA (*p*-value = < 0.001), MUFA (*p*-value = < 0.001), and PUFA (*p*-value = 0.001) were observed in the high UPFs intake group compared to the low UPFs intake group.

Both the crude and multivariable model 2, which adjusted for potential confounders including BMI, education, physical activity, age, and fiber intake, didn't show any significant association between UPFs consumption (as a continuous variables) and odds ratio (OR) of PC (crude; OR = 1.026, confidence interval (CI) 95%: 0.95–1.09, *P* value = 0.455, model 2; OR = 1.037, CI 95%: 0.95–1.12, *p*-value = 0.372). In the comparison of low versus high UPFs intake, no significant association with the odds of PC was observed in the crude model (OR = 1.96, CI 95%: 0.94–4.05, *P* value = 0.069). However, after adjusting for factors in model 2, a high UPFs intake emerged as a significant risk factor for PC, with 2.81 times greater odds of PC compared to low UPFs intake (OR = 2.81, CI 95%: 1.18–6.65, *p*-value = 0.019) (Table 3).

Discussion

The results of our case–control study showed a positive association between UPFs intake and the odds of PC, but this association became significant only after adjusting for the effects of age, BMI, education, physical activity, and fiber intake.

Contrary to our findings, the NutriNet-Santé cohort study [26], conducted on French population with an average follow-up of five years, found a positive association between UPFs intake and the risk of overall cancer, but not PC. Similarly, the UK Biobank cohort study [27] by Chang et al. did not able identify any association between UPFs intake and PC risk during a median follow-up of 9.8 years. Also, the EPIC study [28] which involved substituting 10% of UPFs with 10% of minimally processed foods, showed in no effect on PC risk, but an increase in the risk of overall cancer. Emerging evidence from a case–control study conducted by Trudeau et al. done in Canada, with 1919 prostate cancer patients and 1991 controls [33], confirmed a 29% greater odds of PC related to consuming higher amounts of processed foods, especially among patients with high grade PC compared to those with low grade PC, but not specifically for UPFs. Results from the cross-sectional study by Sciacca et al. [47], conducted on Italian patients with clinically localized prostate adenocarcinoma, indicated a borderline significant association between UPFs consumption and higher odds of PC in the energy-adjusted model, though association was no longer significant after adjustment for other confounders such as age, BMI, education, smoking status, and physical activity. Another study using data from the multi case–control study (MCC)-Spain [48] similarly confirmed the lack of any association between UPFs intake and PC.

In the previous cohort studies [26–28], UPFs contributed between 18.71% and 48.6% of TDEI. Interestingly, in our study, this contribution was found to be much lower at 7.15%, highlighting the pivotal role of geographic variations and cultural differences in shaping dietary habits, and consequently, the consumption of UPFs within each

Table 2 The characteristic of the study participants between lower and higher mean intake of UPFs between case and control groups

Variables	Cases (60)			Controls (60)		
	Lower than the mean of UPFs ($n=25$)	Higher than the mean of UPFs ($n=35$)	*P*-value	Lower than the mean of UPFs ($n=35$)	Higher than the mean of UPFs ($n=25$)	*P*-value
Age (year) ^	66 (15.5)	65 (11)	0.400	64 (13)	59 (11.5)	0.118
BMI (kg/m^2) *	25.40 ± 3.97	24.44 ± 3.38	0.331	25.92 ± 3.39	25.76 ± 3.62	0.866
UPFs (energy %)	3.78 (2.96)	10.78 (5.41)	< 0.001	4.80 (3.23)	11.40 (8.71)	< 0.001
Energy (kcal/day) *	2455 ± 618.02	2895.99 ± 508.11	0.005	2327.25 ± 640.68	2972.50 ± 643.78	< 0.001
Fiber (g/day) ^	19.18 (7.95)	22.05 (7.77)	0.136	20.70 (13.09)	26.69 (9.51)	0.032
Total fat (g/day) *	57.16 ± 41.69	78.09 ± 31.77	0.041	42.35 ± 14.82	71.25 ± 20.02	< 0.001
SFA (g/day) *	17.59 ± 8.12	24.62 ± 7.02	0.001	13.94 ± 5.69	24.98 ± 8.41	< 0.001
MUFA (g/day) ^	12.4 (7.23)	17.49 (9.30)	0.001	10.09 (5.19)	19.65 (9.17)	< 0.001
PUFA (g/day) ^	6.35 (3.93)	7.39 (4.36)	0.110	5.82 (4.11)	9.16 (5.24)	0.001
Ethnicity, Fars, % &	20 (80%)	28 (80%)	1	26 (74.3%)	20 (80%)	0.606
Education, % &			0.963			0.861
Illiterate and Primary Diploma and Academic	17 (68%) 7 (32%)	24 (68.6%) 11 (31.4%)		19 (54.3%) 16 (45.7%)	13 (52%) 12 (48%)	
Physical activity, % &			0.694			0.549
Never or Less Moderate High	11 (44%) 10 (40%) 4 (16%)	12 (34.3%) 15 (42.8%) 8 (22.9%)		6 (17.1%) 16 (45.8%) 13 (37.1%)	6 (24%) 8 (32%) 11 (44%)	
Lipid medication, yes, % &	4 (16%)	2 (5.7%)	0.223	1 (2.9%)	5 (20%)	0.073
HTN medication, yes, % &	11 (44%)	8 (22.9%)	0.083	8 (22.9%)	5 (20%)	0.791
Aspirin use, yes, % &	6 (24%)	4 (11.4%)	0.294	9 (25.7%)	6 (24%)	0.880

Significant values are shown in bold

BMI body mass index, *UPFs* ultra-processed foods, *SFA* saturated fatty acids, *MUFA* monounsaturated fatty acids, *PUFA* poly unsaturated fatty acids

^ Using Mann–Whitney for abnormal continuous variables and values are median (IQR)

* Using independent samples T-test for normal continuous variables and values are mean ± SD

& Using chi-square test for categorical and values are numbers and percentage

specific populations. The discrepancies in UPFs intake levels can also be attributed to differences in food processing practices between countries with higher levels of food industrialization and regions like Iran. For example, in Iran, the traditional method of bread-making, which involves simple ingredients such as wheat flour, water, salt, and yeast may not classify bread as an UPFs. However, in some other countries, commercially produced breads containing additives are more commonly consumed and are classified as UPFs.

A meta-analysis of case–control and cohort studies by Fabiani et al. [10] aligns with a meta-analysis of case–control studies by Grosso et al.[49] in investigating the carcinogenic properties of the western dietary pattern. Adherence to a western dietary, characterized by an abundance of fast food, processed meats, sweets, and salty snacks, has been established as a risk factor for PC according to both aforementioned meta-analyses [10, 49], as well as case–control studies in the Iranian men population [38, 45]. Given the similarities between the dietary profile of a diet high in UPFs and the western dietary pattern, the results of studies by Grosso et al. and Fabiani et al. further support the findings of the present study.

Several mechanisms have been established to explain the relationship between UPFs and PC, which can be divided into three domains: (1) the inferior nutritional content of UPFs, (2) the alteration of food matrices, and (3) the presence of existing or neo-formed carcinogens attributed to ultra-processing. Higher UPFs consumption is associated with an increased intake of calorie, salt, added sugar, and saturated fats, but limited intake of fiber and anti-oxidants [17, 19]. Insulin resistance and obesity, both strong cancer risk factors, result from the unbalanced composition of diet high in UPFs. This dietary imbalance also disrupts glucose-insulin regulation and increase insulin and insulin-like growth factor-1 (IGF-1) in the blood [20, 50]. Additionally, a pro- inflammatory adipokine profile (e.g., higher leptin levels, which may act as an inflammatory, proliferative, and anti-apoptotic agent, compared to lower adiponectin levels, which have anti-proliferative properties) following diets rich in saturated fats and sugars and low in fiber can contribute to

Table 3 Associations between ultra-processed foods with prostate cancer

Median of UPFs	Case/Control	Model 1		Model 2	
		OR	95% CI	OR	95% CI
Ultra-processed foods					
UPFs (% energy)	60/60	1.026	0.959-1.099	1.037	0.958-1.122
P-value		0.455		0.372	
Ultra-processed foods category					
Lower than the mean intake of UPFs (≤ 7.15 % energy)	25/35	Ref.	Ref.	1.000	Ref.
Higher than the mean intake of UPFs (> 7.15 % energy)	35/25	1.960	0.948-4.050	2.812	1.188-6.650
P-value		0.069		0.019	

UPFs ultra-processed food

Obtained from logistic regression

These values are odd ratio (95% CIs)

Significant values are shown in bold

Model 1: Crude Model

Model 2: adjusted for BMI, education, physical activity, age, and fiber intake

tumor formation and various cancers, including PC and breast cancer [50, 51].

The deconstructing of food during ultra-processing—a concept that explains the different behavior of a component or nutrient in food form as compared to its isolated form—alter nutrient bioavailability and bio-accessibility, which further contributes to insulin resistance [52].

On the other hand, food processing often involves adding substances to enhance texture, taste, or shelf-life (such as thickening agents, non-caloric artificial sweeteners, and certain industrial additives), which can increase the risk of cancer through mechanisms like gut dysbiosis, metabolic dysfunction, pro-inflammatory cytokines release, systemic inflammation, and impaired immune response [53–55]. Neo-formed contaminants arising from high-temperature cooking (e.g., acrylamide in carbohydrate-rich foods and heterocyclic aromatic amine (HAA) and PAH in meats), as well as synthetic compounds used in UPFs packaging (such as BPA and phthalates), which can know endocrine disruptors, are other potential carcinogens that link UPFs consumption to cancer [23, 24]. The body metabolizes heterocyclic aromatic amines (HAAs) and polycyclic aromatic hydrocarbons (PAHs) to form HAA-DNA adducts and PAH-DNA adducts, which can lead to mutations and drive prostate cells toward a carcinogenic pathway [56, 57]. According to a case–control study conducted in Japan, the strength of the association between HAA and the risk of PC depends on specific genotypes, such as NAT2 and CYP1A1 GA + GG [58]. Notably, a meta-analysis investigating the association between the NAT2 genotype and PC found that this association was significant only in Asian populations [59]. Therefore, the association between HAA and the risk of PC in Asians may be more pronounced than in other populations. In addition, the carcinogenic effects of PAH on prostate cells may occur through the increased expression of vascular endothelial growth factor (VEGF) and hypoxia-inducible factor (HIF), as well as the production of reactive oxygen species (ROS) [60]. Recent molecular assay have demonstrated that BPA-induced epigenetic modifications promote cancer formation and progression in prostate cells by affecting the expression of specific genes involved in regulating angiogenesis, cell proliferation, DNA replication and repair, metabolism, inflammation, and immune response pathways [23].

Strengths and limitations

The strengths of the present study include several key aspects: Firstly, we focused on incident cases of PC before participants were aware of their diagnosis, which minimized the risk of recall and interviewer bias, as they were less likely to alter their habitual diet. Furthermore, by adjusting for relevant covariates, we enhanced the accuracy and reliability of our findings.

Despite these strengths, there are several limitations to consider. First, the study had a small sample size, which may affect the power of the study and increase the margin of error. As with any case–control design, selection and recall biases are inherent. However, we mitigated selection bias by selecting controls from the same locations and during the same time period as the cases. Regarding recall bias, we focused on newly diagnosed cases of PC, and data collection occurred shortly after diagnosis, ensuring that participants were not yet fully aware of the severity of their cancer. Another limitation is the lack of a single diagnostic criterion for both the case and control groups. PC diagnosis in cases was confirmed through histological tests, while the absence of PC in controls was determined by specialist confirmation following detailed clinical evaluation. Despite efforts by nutritionists to categorize food items using the NOVA classification, misclassification remains a possibility due to the complexity of food processing methods. Additionally, some UPFs items may not have been captured in the existing FFQs. To better measure UPFs consumption in future studies, a more tailored FFQ specific to the Iranian population should be developed. Lastly, residual confounders, such as family history of PC and age, were not assessed

or adjusted for, despite their potential association with both UPFs consumption and PC risk.

Conclusion

In conclusion, the findings of our study have demonstrated that a diet high in UPFs, regardless of its low fiber content, is a significant risk factor for PC in the Iranian male population. The escalating trend of UPFs consumption and its potential carcinogenic properties necessitate heightened concern among populations. Thus, advocating for a reduction in UPFs consumption through policy interventions—such as product reformulation, marketing restrictions, front-of-pack UPFs labeling, and raising public awareness about the adverse health implications associated with UPFs consumption—could represent effective measures for preventing specific types of cancer, including PC. Furthermore, the level of food processing and its association with various types of cancer require further attention from scientific researchers in future investigations to better comprehend the consequences of food industrialization and guide populations toward fresh, healthy, and beneficial foods.

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Author contributions
Conceptualization: M.M., M.N., and Y.J.; Methodology: Y.J. and S.F.; Formal analysis and investigation: M.M. and M.N.; Writing – original draft preparation: M.M., Z.M. and M.N.; Writing – review and editing: M.M., Y.J., Z.M., M.N., and S.F.; Supervision: M.N. and S.F.

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Availability of data and materials
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The protocol for this study was approved by the Ethics Committee of Shiraz University of Medical Sciences in Iran, following the guidelines of the Declaration of Helsinki. A consent form was obtained from all participants in the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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