

## RESEARCH ARTICLE

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# Lifestyle-Related Factors and Dietary Components that Affect the Risk of Prostate Cancer in a Brazilian Study Group

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### Abstract

**Background:** The incidence of prostate cancer (PCa) is increasing among Brazilian men. There are only a few studies about risk factors for PCa among Brazilian men, and to date, there are no studies about dietary components that affect PCa risk. **Methods:** A case-control study was performed on data from 125 PCa patients from a reference center for cancer treatment in Northeast Brazil. Data were obtained from medical records and interviews and compared with 250 age-matched controls. Binary logistic regression analysis was applied to obtain odds ratios (ORs) and confidence intervals (95% CIs) of the variables. **Results:** According to the regression analysis model, hypertension, family history and ever smoking increased the risk of PCa 2.940 (95% CI: 1.81-4.78), 2.268 (95% CI: 1.31-3.92) and 2.715 (95% CI= 1.67-4.41) times ( $p < 0.001$ ;  $p = 0.003$ ;  $p < 0.001$ ), respectively. The consumption of red and processed meat increased the risk of disease by 0.6% (OR= 1.006; 95% CI: 1.00-1.01;  $p = 0.048$ ) and 7.2% (OR= 1.072; 95% CI: 1.02-1.13;  $p = 0.012$ ), respectively, for each gram of intake per day. The consumption of cruciferous vegetables reduced the risk by 22.0% for each g intake per day (OR= 0.978; 95% CI: 0.96-1.00;  $p = 0.052$ ). **Conclusion:** Hypertension, family history and smoking increased PCa risk among men in the present study. Additionally, red and processed meat increased the risk of disease, and cruciferous vegetables had a protective effect. The present results indicate that lifestyle-related factors and dietary components affect PCa risk among Brazilian men. Health authorities should include information about these risk and protective factors in their PCa prevention campaigns.

**Keywords:** Prostate cancer- Lifestyle-related risk factors- Dietary components

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### Introduction

According to the International Agency for Research on Cancer (IARC), PCa accounts for approximately 1.4 million cases and is the third most prevalent cancer worldwide [1]. In 2020, with over 375,000 deaths, PCa was the fifth leading cause of death among men globally [1, 2]. In developed Western countries, the incidence rates of PCa have already plateaued or even declined, whereas in developing countries, they are expected to increase [3]. Compared with developed countries, developing countries are also characterized by higher mortality rates [4].

In Brazil, it is estimated that there will be 71,730 new cases of PCa during the triennial between 2023 and 2025 [5]. In the years from 2010 to 2023, the incidence of PCa increased from 52.35 to 67.86 new cases per 100,000 men, with strong differences among distinct regions [5]. In the northern region, the incidence of PCa increased only slightly between 2010 and 2023, from 24.00 to 28.40 cases per 100,000 men. In the southern region of the country, the incidence of PCa declined between 2010 and 2023, from 69.00 to 57.23 cases per 100,000 men [5, 6]. This is in sharp contrast to the Northeast Region of Brazil, where the

incidence increased from 44.00 to 73.28 cases per 100,000 men within the same time period [5, 6]. The incidence of PCa has not drastically increased in any other region of the country, such as in the Northeast Region of Brazil [5]. As the Northeast and North Regions include populations with similar age structures, differences in PCa incidence are difficult to explain by demographic factors alone.

The annual campaign in November (“Novembro Azul”), is dedicated to prostate cancer awareness and seeks to encourage men to take care of their health and undergo preventive exams [4]. However, as in most other countries, Brazil also does not have an organized PCa screening program. On one hand, different frequencies of early detection of PCa among Brazilian regions may contribute to observed differences of PCa incidence. On the other hand, biological and life-style related risk factors may also contribute to the observed differences: Men of African ancestry are at greater risk of PCa, and they potentially develop more aggressive forms of disease at a younger age than men of European ancestry [7]. Northeast Brazil has a trihybrid population of Indigenous, European and African ancestry [8]. As the African contribution to admixture in the Northeast is greater than that in other

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Brazilian regions, genetic factors may contribute to different incidence rates across distinct regions.

It is well established in literature that diet and nutrients can modify PCa risk. In British prospective studies, vegetarian and pescarian diets lowered the risk of PCa [9-11]. Similarly, in a recent American prospective study, a healthy plant-based diet decreased the risk of PCa [12]. Previous studies associated tomato products and lycopene with a lower risk of PCa [13]. Red meat and dairies, in contrast, were associated with increased PCa risk [9]. Despite the use of some well-established dietary components as protective and risk factors for PCa, conflicting results concerning other components, especially nutrients such as selenium, vitamin E and D, have been reported [14].

The Brazilian literature on PCa has focused mainly on the epidemiology of the disease and descriptive clinical and socioeconomic data of patient groups [15-21]. In an ecological database study, the consumption of alcohol and the application of pesticides were indirectly associated with the risk of PCa [22]. A recent review of the Brazilian literature concerning PCa has shown that only three case-control studies exist that addressed the identification of only a few risk factors [23]: the application of molecular markers in a study group including 104 cases and 109 controls focused on ancestry as a risk factor in the state of Bahia [24]. A study of 125 cases and 251 controls in the southern state of Santa Catharina exclusively addressed family history as a risk factor [25]. Finally, in a study of 91 cases and 91 controls performed in the state of Paraíba, anthropometric measures, ancestry, family history and smoking status were analyzed [26].

To the best of our knowledge, there are no Brazilian studies about the risk of PCa that include dietary components of patients and controls. The increasing rates of PCa in Northeast Brazil and the low number of studies that have focused on few risk factors underscore the need to better understand the possible causes of disease in this population. The present study was performed in Northeast Brazil. Family history, ancestry, and potential lifestyle-related risk factors in combination with the dietary characteristics of individuals were compared between 125 patients and 250 controls.

## Materials and Methods

### *Study population*

Cancer patients and healthy controls were recruited at the oncological treatment reference center, “Fundação Assistencial da Paraíba” (FAP) hospital, in Campina Grande, State of Paraíba, Northeast Brazil. The public FAP hospital receives mainly low-income patients and treats approximately 50% of all PC cases in the state. Some patients receive PC treatment from towns and villages that can be as far as 400 km away from the FAP hospital. Campina Grande has approximately 0.4 million inhabitants and is the second largest urban center in the state of Paraíba. It is located inland of the state, approximately 120 km away from the capital João Pessoa at the Atlantic coast. Like other states of Northeast Brazil, Paraíba has a mixed-ethnicity population comprising

individuals of Native American, African, and European ancestry.

The present study was performed between July and September 2024. Patients with PCa who were undergoing treatment during this time interval at the FAP hospital were eligible for the study. Patients with in situ tumors and those whose PC diagnosis was more than 24 months prior were excluded from the study. Among the 218 patients, 78 with disease recurrence were excluded from the study. Among the 140 remaining eligible PC patients, ten (7.1%) refused to participate and five (3.6%) had incomplete medical records. The remaining 125 patients were included in the study. Data from 250 age-matched ( $\pm 5$  years) controls were obtained from the FAP hospital. Controls with any type of cancer, or other chronic disease were excluded from the study. The controls at the FAP hospital were all healthy companions of patients with other types of disease than cancer. Controls and cases were residing in the same state and municipalities.

### *Sampling of data*

The clinical and histopathological data of patients were obtained from medical records of the medical archive of the FAP hospital. High-risk PCa was defined according to the National Comprehensive Cancer Network (NCCN) as T3a–T4, Gleason score  $\geq 8$ , or PSA level  $\geq 20$ , and very high-risk PCa was defined as T3b or T4 disease [27]. Data concerning age and weight, which were registered during hospital admission, were also obtained from medical records.

Patients and healthy controls were interviewed face-to-face via a structured questionnaire. A similar questionnaire was used in previous studies Ribeiro et al. [32]. All the interviews were conducted by one of the authors via a questionnaire, to which the participants responded verbally. Patients were interviewed in the chemotherapy and radiotherapy units at the FAP hospital. Controls were randomly selected from the waiting rooms of the health centers, namely, the hospital lounge, and invited directly by one of the authors to participate as volunteers. Among several family members, only one person was recruited as a control.

Body mass index (BMI) was defined according to the World Health Organization (WHO) as follows: underweight  $< 18.5$  kg/m<sup>2</sup>; normal weight = 18.5–24.99 kg/m<sup>2</sup>; overweight = 25.0–29.99 kg/m<sup>2</sup>; and obesity  $\geq 30.0$  kg/m<sup>2</sup> [28]. Minimum wage and multiple values were used to characterize income. This is a popular and well-known method for defining economic levels among low- and middle-class subjects. The minimum wage was R\$1,420.00 (US\$ 272.54; January 1, 2024). Basic income was defined as  $\leq 1$  minimum wage; middle income was defined as  $> 1$  and  $\leq 2$  minimum wages; and high income was defined as  $> 2$  minimum wages. Ethnic origin information was obtained from the self-reports of the participants.

The level of education was defined as follows: incomplete basic schooling and not knowing how to read or write was defined as “Analphabetic”; complete basic schooling lasting nine years was considered “basic”; complete medium schooling lasting 12 years was defined

as “medium”; and higher educational levels were defined as “high”. Overall physical activity was defined as low, moderate or strong regular bodily movement according to a previous study [29].

Dietary intake was measured, using a validated Quantitative Food Frequency Questionnaire (QFFQ), applied in previous cancer studies of populations in the Brazilian Northeast region [30-32]. These previous studies served as a methodological basis for applying the instrument, ensuring greater consistency and comparability of the data obtained. The questionnaire was administered individually, in a private environment, by one of the authors and included a list of foods with questions about the frequency of consumption and the size of portions eaten during the period prior to the diagnosis of PCa. To reduce memory bias, participants were also asked about possible changes in diet before and after the diagnosis of the disease.

In order to standardize the estimation of food portions, a photographic food quantification manual was used, consisting of 96 foods represented in different sizes and homemade measures [33]. The portion sizes were self-reported by the participants, based on a visual comparison with the images in the manual. The information obtained was recorded in Microsoft Excel version 2013 spreadsheets, which allowed for its organization and subsequent statistical analysis. In the case of fish consumption, the questionnaire did not distinguish between freshwater and marine fish. However, as the patients did not live in coastal regions, it is assumed that consumption refers predominantly to freshwater fish. With regard to cruciferous vegetables, cabbage and broccoli were specifically considered.

#### Statistical analyses

All the statistical analyses were performed via SPSS Statistics™ software (SPSS; IBM Company; version 29). The t test was used to compare continuous variables, whereas Fisher's exact test and Pearson's chi-square ( $\chi^2$ ) test were used for categorical variables. Binomial logistic regression analysis was used to quantify the associations between variables and the risk of PCa. The results are presented as adjusted odds ratios (ORs) and 95% confidence intervals (CIs). The p values were obtained via likelihood ratio tests. Initially, all variables with a significance of  $<0.2$  in the univariate analysis were used for modeling. The backward method was then used to select significant variables ( $p \leq 0.05$ ), and the final model was assessed for adequacy via the likelihood ratio test.

## Results

Among the 125 PCa patients, 86 (68.8%) had grade 3 and 4 disease and no patient had grade 5 disease (Table 1). Of all patients 35 (28.0%) presented disease at stages III and IV (Table 1). The most commonly used therapy, radiotherapy, was applied in 116 (92.8%) out of 125 patients (Table 1). Among all 125 patients, 57 (45.6%) had high and very high risk PCa. Bone metastases were detected in eight (14.0%) of these 57 patients.

Patients and controls were, on average, 71.53 (SD=

9.3) and 72.56 (SD= 8.4) years old ( $p= 0.279$ ; Table 2). Among the 125 cases and 250 controls, 69 (55.2%) and 79 (31.6%) had hypertension, respectively ( $p< 0.001$ ; Table 2). Family history was reported by 41 (32.8%) and 46 (18.4%) patients and controls, respectively ( $p= 0.002$ ; Table 2). Anthropometric measures, alcohol consumption, socioeconomic characteristics and the frequency of diabetes were not significantly different between the two groups (Table 2). Ever smoking was a characteristic of 83 (66.4%) and 103 (41.2%) patients and controls, respectively ( $p< 0.001$ ; Table 2). All the 83 cases and 100 controls reported having smoked, on average, 14.24 (SD= 11.6) and 9.18 (SD= 5.6) cigarettes per day ( $p< 0.001$ ), respectively. All the 83 cases and 89 controls reported having smoked on average for 32.61 (SD= 15.3) and 29.30 (SD= 11.7) years ( $p= 0.114$ ), respectively. The mean time interval between the end of smoking and the end of the study was 22.02 (SD= 12.2) and 18.71 (SD= 8.3) years for 74 (89.61%) cases and 56 (37.33%) controls, respectively ( $p= 0.068$ ).

The intake of dietary components is summarized in Table 3. The cases and controls consumed, on average, 63.19 g (SD= 48.6) and 51.53 g (SD= 28.5), respectively, of red meat per day ( $p= 0.004$ ; Table 3). Patients and controls consumed, on average, 3.81 (SD= 6.0) and 2.1 (SD= 3.6) g of processed meat per day, respectively ( $p< 0.001$ ; Table 3). The consumption of cruciferous vegetables was 3.63 g (SD= 11.4) and 6.43 g (SD= 13.25) per day for the cases and controls, respectively ( $p= 0.045$ ; Table 3).

Logistic regression analysis was applied to identify

Table 1. Clinical Characteristics of 125 PC Patients

Mean PSA	10.70 (SD= 19.18)
N (%)	
Gleason	
6	13 (10.4%)
7	59 (47.2%)
8	42 (33.6%)
9	11 (8.8%)
Grade	
1	13 (10.4%)
2	26 (20.8%)
3	33 (26.4%)
4	53 (42.4%)
Stage	
I	32 (25.6%)
II	58 (46.4%)
III	24 (19.2%)
IV	11 (8.8%)
Treatment	
R	16 (12.8%)
C + H	9 (7.2%)
C + R	74 (59.2%)
S + C + R	26 (20.8%)

C, Chemotherapy; S, Surgery; H, Hormone therapy; R, Radiotherapy.

Table 2. Baseline Characteristics of PC Patients and Controls of the Study Group

	Case (N= 125)	Control (N= 250)	p value
Mean weight (kg)	74.15 (SD= 14.0)	74.25 (SD= 13.2)	0.946
Mean height (cm)	121.93 (SD= 70.0)	130.81 (SD= 65.2)	0.226
Mean Age	71.53 (SD= 8.2)	72.56 (SD= 8.4)	0.279
	N (%)	N (%)	
Age			
50- 59 years	14 (11.2%)	17 (6.8%)	0.471
60- 69 years	35 (28.0%)	72 (28.8%)	
70- 79 years	49 (39.2%)	97 (38.8%)	
≥80 years	27 (21.6%)	64 (25.6%)	
BMI			
Normal weight	52 (41.6%)	99 (39.6%)	0.741
Overweight	52 (41.6%)	114 (45.6%)	
Obesity	21 (16.8%)	37 (14.8%)	
Hypertension			
Yes	69 (55.2%)	79 (31.6%)	<0.001
No	56 (44.8%)	171 (68.4%)	
Diabetes			
Yes	16 (12.8%)	36 (14.4%)	0.401
No	109 (87.2%)	214 (85.6%)	
Ancestry			
European	45 (36.0%)	36 (14.4%)	0.638
Mixed background	64 (51.2%)	136 (54.4%)	
African	16 (12.8%)	78 (31.2%)	
Family history of first degree relatives			
Yes	41 (32.8%)	46 (18.4%)	0.002
No	84 (67.2%)	204 (81.6%)	
Ever smoked			
Yes	83 (66.4%)	103 (41.2%)	<0.001
No	42 (33.6%)	147 (58.8%)	
Alcohol consumption before diagnosis			
Yes	100 (80.6%)	194 (77.9%)	0.32
No	24 (19.4%)	55 (22.1%)	
Missing	1	1	
Working in agriculture			
Yes	73 (58.4%)	155 (62.0%)	0.287
No	52 (41.6%)	95 (38.0%)	
Education level			
Analphabetic	40 (32.0%)	63 (25.2%)	0.545
Basic	71 (56.8%)	156 (62.4%)	
Middle	10 (8.0%)	20 (8.0%)	
High	4 (3.2%)	11 (4.4%)	
Income			
Basic	5 (4.0%)	6 (2.4%)	0.366
Middle	94 (75.2%)	203 (81.2%)	
High	26 (20.8%)	41 (16.4%)	
Civil state			
Living in a stable union	97 (77.6%)	179 (71.6%)	0.131
Not living in a stable union	28 (22.4%)	71 (28.4%)	

Table 3. Intake of Dietary Components are Shown as Mean and Median Values for All Individuals, Cases and Controls. Mean values were compared between cases and controls

	All (N= 375)		Cases (N= 125)		Controls (N= 250)		P- value*
	Mean (SD)	Median	Mean (SD)	Median	Mean (SD)	Median	
Fish (g/day)	13.64 (13.6)	13.33	14.20 (18.2)	11.19	13.36 (10.7)	13.33	0.571
Red meat (g/day)	55.41 (36.8)	42.85	63.19 (48.6)	48.57	51.53 (28.5)	42.85	0.004
Processed meat (g/day)	2.67 (4.6)	0	3.81 (6.0)	0	2.1 (3.6)	0	<0.001
Chicken (g/day)	40.46 (24.4)	38.57	40.00 (29.1)	36	40.69 (21.7)	38.57	0.797
Milk (ml/day)	86.00 (81.6)	93.33	94.37 (77.3)	93.33	81.82 (83.6)	89.52	0.15
Cheese (g/day)	15.95 (17.2)	12	17.58 (23.5)	11	15.13 (13.0)	12	0.197
Green salad (g/day)	10.71 (11.4)	4.28	10.05 (11.8)	4.28	11.03 (11.2)	4.28	0.44
Tomato (g/day)	29.84 (25.9)	28	28.87 (26.6)	20	30.33 (25.56)	28	0.612
Cruciferous (g/day)	5.49 (12.7)	0	3.63 (11.4)	0	6.43 (13.25)	0	0.045
Beetroot (g/day)	5.24 (9.9)	0	4.80 (9.6)	0	5.46 (10.02)	0	0.539
Carrot (g/day)	9.59 (10.0)	8	9.29 (10.2)	4.28	9.74 (9.9)	8	0.685
Paprika (g/day)	0.72 (2.1)	0	0.66 (2.2)	0	0.74 (2.07)	0	0.715

Table 4. Odds ratios (OR) and confidence intervals (95%CI) are shown for single variables (ORcrude) and in an adjusted model (ORadjusted) as the chance to have PCa (N= 125). The control served as reference group

	ORcrude (95% CI)	P	ORadjusted (95% CI) <sup>1</sup>	P
Hypertension	2.667* (1.71-4.15)	<0.001	2.940 (1.81-4.78)	<0.001
Family history	2.165* (1.32-3.54)	0.003	2.268 (1.31-3.92)	0.003
Smoking	2.820* (1.80-4.42)	<0.001	2.715 (1.67-4.41)	<0.001
Dietary components				
Fish (g/week)	1.004 (0.99-1.02)	0.571		
Chicken (g/day)	0.999 (0.99-1.01)	0.797		
Red meat (g/day)	1.009 (1.00-1.02)	0.005	1.006 (1.00-1.01)	0.048
Processed meat (g/day)	1.083 (1.03-1.14)	0.01	1.074 (1.02-1.13)	0.012
Milk (g/day)	1.002 (1.00-1.01)	0.177		
Cheese (g/day)	1.008 (1.00-1.02)	0.212		
Green salad (g/day)	0.992 (0.97-1.01)	0.43		
Tomatoe (g/day)	0.998 (0.99-1.01)	0.607		
Cruciferous (g/day)	0.979 (0.96-1.00)	0.052	0.978 (0.96-1.00)	0.052
Beetroot (g/day)	0.993 (0.97-1.02)	0.993		
Carrot (g/day)	0.995 (0.97-1.02)	0.681		
Peppers (g/day)	0.980 (0.88- 1.09)	0.707		

<sup>1</sup>Variables were adjusted among each other and for age.

single variables that modulated the risk of PCa (Table 4). Hypertension, family history and smoking increased the risk of PCa as single variables ( $p < 0.001$ ;  $p = 0.002$ ;  $p < 0.001$ ; Table 4). Furthermore, the intake of red and processed meat increased the risk ( $p = 0.005$ ;  $p = 0.001$ ; Table 4). The consumption of cruciferous vegetables instead reduced the risk of PCa by 21.0% for each gram of intake per day (OR= 0.979; 95% CI: 0.959-1.000;  $p = 0.052$ ; Table 4).

A stepwise binary logistic regression model was constructed to identify independent variables that increased the risk of PCa (Table 4). In this model, hypertension, family history and ever smoking increased the risk 2.940 (95% CI: 1.81-4.78), 2.268 (95% CI: 1.31-3.92) and 2.715 (95% CI= 1.67-4.41) times ( $p < 0.001$ ;  $p = 0.003$ ;  $p < 0.001$ ; Table 4), respectively. The consumption of red meat

increased the risk of PCa by 0.6% for each gram of intake per day (OR= 1.006; 95% CI: 1.00-1.01;  $p = 0.048$ ; Table 4). The consumption of processed meat increased the risk of disease by 7.2% for each gram of intake per day (OR= 1.072; 95% CI: 1.02-1.13;  $p = 0.012$ ; Table 4). Finally, the consumption of cruciferous vegetables reduced the risk of PCa by 22.0% for each g of intake per day (OR= 0.978; 95% CI: 0.96-1.00;  $p = 0.052$ ; Table 4).

## Discussion

The Brazilian campaign “Novembro azul” (Blue November) aims to disseminate information about men’s health and strengthen the Ministry of Health’s recommendations for cancer prevention, early diagnosis and screening [5]. However, the underlying modifiable



risk factors for disease among Brazilian men are poorly understood. To the best of our knowledge, this is the first Brazilian study that attributed dietary components to an increased risk of PCa. The consumption of red and processed meat increased the risk of PCa, whereas the consumption of cruciferous vegetables reduced the risk among Brazilian men in the study group. Additionally, a family history of PCa, smoking and hypertension were associated with an increased risk of disease.

In the present study, hypertension increased the risk of PCa approximately threefold. A previous meta-analysis including 21 and 14 studies indicated an association of hypertension with an increased risk of PCa [34, 35]. A cohort study performed in Iceland also indicated that hypertension increased the risk [36]. However, in a recent Chinese study hypertension did not increase PCa risk [37]. The etiology of high blood pressure and PCa is not fully understood, but animal studies have indicated that increased PCa risk and hypertension are common androgen-mediated mechanisms [38].

The present results indicated a positive association between family history and PCa risk. Family history is a well-established strong risk factor for PCa [39-42]. The overall increase in PCa risk among men depends on the degree and number of relatives who had PCa [40-43]. A recent cohort study revealed that men with indications for biopsy and a family history of PCa also have a moderately increased risk of high-grade PCa [43]. Furthermore, a family history of high-grade or metastatic disease increased the chance of similar high-risk PCa among men [41-44]. Several previous Brazilian studies also associated family history with increased risk of disease [11, 23].

Smoking increased the risk of PCa among men in the present study. Smoking is associated with advanced-stage disease, high-risk metastatic disease, obesity, and poor survival in PCa patients [45-48]. Previous studies, including data on the global burden of disease between 1990 and 2019, associated smoking with increased PCa risk [49, 50]. However, other studies did not indicate this positive association: In two meta-analyses, including 17 and 24 cohort studies, smoking was not associated with increased risk but was associated with an increased chance of death among PCa patients [49, 51]. A Japanese study did not indicate an increased PCa risk in smokers [46]. Recent studies have even associated smoking with a reduced PCa risk [47, 50]. Five Swedish cohort studies and a meta-analysis of 44 cohort studies indicated that this risk reduction among smokers was found in studies performed during the prostate-specific antigen (PSA) screening era, whereas in studies before this era, smoking increased the risk of disease [47, 52]. The authors attributed the lower risk of smokers to poor PCa screening adherence [47, 52]. In agreement with the present findings, previous Brazilian studies also indicated an increased risk of PCa in men who smoke [26, 53]. To the best of our knowledge, there are no comparative data available concerning the adherence of Brazilian smokers and nonsmokers to PCa screening.

In the present study, red and processed meat increased the risk of PCa. The consumption of red and even stronger processed meat is a well-established risk factor for PCa. A recent umbrella review of 72 meta-analyses associated

processed meat intake with increased PCa risk [54]. A recent meta-analysis including 25 studies indicated that an increase in processed meat intake increased the risk of PCa [55]. An umbrella review of meta-analyses of cohort studies associated processed and red meat intake with increased PCa risk [56]. These findings are in agreement with those of large studies based on UK Biobank data, which revealed that vegetarians and pescetarians had a reduced PCa risk [9-11]. The consumption of meat-associated saturated and trans fatty acids contributes to the etiology of PCa through increased oxidative stress, inflammation, alterations in lipid metabolism, growth factor signaling and the disruption of prostate hormonal regulation [57].

Data from the 2017–2018 Brazilian Household Budget Surveys revealed that Brazilians consume an average of 84 g/day of red and processed meat [58]. A recent survey concluded that 47.3% of Brazilian men consumed more than the recommended 70.0 g/day of red meat [58]. Additionally, 31.7% consumed  $\geq 50.0$  g/day to  $<150$  g/day of processed meat, and 15.4% consumed  $\geq 150$  g/day of processed meat [59]. The authors attributed the high consumption of red and processed meat to increasing future costs of the public Brazilian health system due to colorectal cancer [59]. High consumption of red and processed meat may also contribute to increasing rates of PCa among Brazilian men. The present study is the first to indicate a positive association between PCa risk and the consumption of processed and red meat in a Brazilian population.

The present data indicate that cruciferous vegetables may be a protective factor against PCa among Brazilian men. A previous meta-analysis of seven cohort and six population-based case-control studies indicated that cruciferous vegetables decreased the risk of PCa [60]. Furthermore, cruciferous vegetables reduced the progression of disease among PCa patients [61]. Cruciferous vegetables are rich in glucosinolates, and their protective activity has been attributed to the degradation products of these substances [62]. To date, no previous Brazilian studies have investigated the consumption of cruciferous vegetables and their potential protective effects on PCa. The protective effect in the present study had borderline significance. Larger case-control studies should confirm if cruciferous vegetables really have a protective function against PCa among Brazilian men.

The present study had several important limitations. The study was based on a small number of patients and controls. This may have obscured associations among risk factors and PCs. It was therefore not possible to compare risk groups, as for example smoking controls and patients. Furthermore, due to the small sample size, it was also not possible to detect differences of the impact of risk factors, depending on the aggressiveness of PCa. Furthermore, the results cannot be extrapolated to other Brazilian populations with different lifestyle habits and dietary patterns. The recruitment of controls was stochastic, but the possibility of selection bias cannot be eliminated. In the present study group, BMI was not significant different among cases and controls. Furthermore, data about metabolic syndrome was not assessed. However,

hypertension could also be the consequence of obesity, or metabolic syndrome among patients. This could generate a bias. Smoking and hypertension may increase risk of PCa and other chronic diseases. As controls had neither PCa nor any other chronic disease, these risk factors may have biased. A recall bias among men in the present study group also cannot be excluded. Information about ancestry was based on subjective information about participants in a population with an extremely high degree of admixture. Subjective information was a source of uncertainty that may have obscured the possible association between the risk of PCa and ancestry.

In conclusion, hypertension, a family history of PCa and smoking increased the risk of disease in the present Brazilian study group. The consumption of red and processed meat was also associated with a greater risk of developing the disease. In contrast, the consumption of cruciferous vegetables had a protective effect. The present results indicate that modifiable protective and risk factors may play important roles in the etiology of PCa among Brazilian men. Lifestyle and dietary choices may play important roles in modulating the risk of PCa. This emphasizes the need for prevention strategies by health authorities. Prevention should focus on information transfer regarding hypertension, smoking and diet. Prospective Brazilian cohort studies are needed to confirm the present findings and identify other protective and risk factors for PCa.

## Author Contribution Statement

IAS conducted the interviews and tabulated the data. MW performed the data analysis. MW designed the study and drafted the manuscript. IAS and MW contributed to the conception and critical review of the manuscript. Both authors read and approved the final manuscript.

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### Ethical consideration

The data sampling protocol was reviewed and approved by the National Ethics Research Committee (CAAE Brazil platform: 18518819.4.0000.5187 and 78651524.1.0000.5187). Written informed consent was obtained from all participants. Consent to publish data anonymously was obtained from each participant.

### Data availability statement

Data are available upon request.

### Conflict of interest

The authors declare no conflict of interest.

## References

1. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020: Globocan estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin*. 2021;71(3):209-49. <https://doi.org/10.3322/caac.21660>.
2. Leslie Iw, soon-sutton tl, sajjad ria, siref le. Prostate cancer, 2023 may 30. In: Statpearls [internet]. Treasure island (fl): Statpearls publishing; 2023.
3. Teoh JYC, Hirai HW, Ho JMW, Chan FCH, Tsoi KKF, Ng CF. Global incidence of prostate cancer in developing and developed countries with changing age structures. *PLoS One*. 2019;14(10):e0221775. <https://doi.org/10.1371/journal.pone.0221775>.
4. Sharma R. The burden of prostate cancer is associated with human development index: Evidence from 87 countries, 1990-2016. *Epma j*. 2019;10(2):137-52. <https://doi.org/10.1007/s13167-019-00169-y>.
5. National cancer institute (INCA). 2023 estimate: Cancer incidence in brazil. José alencar gomes da silva national cancer institute. Rio de janeiro: Inca; 2023.
6. National cancer institute (INCA). 2010 estimate: Cancer incidence in brazil. José alencar gomes da silva national cancer institute. Rio de janeiro: Inca; 2010.
7. McHugh J, Saunders EJ, Dadaev T, McGrowder E, Bancroft E, Kote-Jarai Z, et al. Prostate cancer risk in men of differing genetic ancestry and approaches to disease screening and management in these groups. *Br J Cancer*. 2022;126(10):1366-73. <https://doi.org/10.1038/s41416-021-01669-3>.
8. Salzano FM, Sans M. Interethnic admixture and the evolution of latin american populations. *Genet Mol Biol*. 2014;37(1 Suppl):151-70. <https://doi.org/10.1590/s1415-47572014000200003>.
9. Parra-Soto S, Ahumada D, Petermann-Rocha F, Boonpoor J, Gallegos JL, Anderson J, et al. Association of meat, vegetarian, pescatarian and fish-poultry diets with risk of 19 cancer sites and all cancer: Findings from the uk biobank prospective cohort study and meta-analysis. *BMC Med*. 2022;20(1):79. <https://doi.org/10.1186/s12916-022-02257-9>.
10. Watling CZ, Schmidt JA, Dunneram Y, Tong TYN, Kelly RK, Knuppel A, et al. Risk of cancer in regular and low meat-eaters, fish-eaters, and vegetarians: A prospective analysis of uk biobank participants. *BMC Med*. 2022;20(1):73. <https://doi.org/10.1186/s12916-022-02256-w>.
11. Weller M. Vegetarian diets and cancer risk. *BMC Medicine*. 2022;20:81. <https://doi.org/10.1186/s12916-022-02282-8>.
12. Loeb S, Fu BC, Bauer SR, Pernar CH, Chan JM, Van Blarigan EL, et al. Association of plant-based diet index with prostate cancer risk. *Am J Clin Nutr*. 2022;115(3):662-70. <https://doi.org/10.1093/ajcn/nqab365>.
13. Zu K, Mucci L, Rosner BA, Clinton SK, Loda M, Stampfer MJ, et al. Dietary lycopene, angiogenesis, and prostate cancer: A prospective study in the prostate-specific antigen era. *J Natl Cancer Inst*. 2014;106(2):djt430. <https://doi.org/10.1093/jnci/djt430>.
14. Wilson KM, Mucci LA. Diet and lifestyle in prostate cancer. *Adv Exp Med Biol*. 2019;1210:1-27. [https://doi.org/10.1007/978-3-030-32656-2\\_1](https://doi.org/10.1007/978-3-030-32656-2_1).
15. Gonçalves I, Padovani C, Popim R. Demographic and epidemiological characterization of men with prostate cancer. *Ciência & saúde coletiva*. 2008;13:1337-42.
16. Ribeiro P, Silva R, Santos K, Loureiro F, Costa P, Uruçu L, et al. Clinical and epidemiological analysis of 348 cases of prostate adenocarcinoma attended in a cancer care center in Maranhão, Brazil. *Rev Bras Canc*. 2013;59:513-21. <https://doi.org/10.32635/2176-9745.RBC.2004v59n1.968>.

17. Zacchi sr, amorim mhc, souza mac, miotto mhmb, zandonade E. Association of sociodemographic and clinical variables with initial staging in men with prostate cancer. *Cad saúde colet.* 2014;22:93–100. <https://doi.org/10.1590/1414-462x201400010014>
18. Araujo J, Conceição V, Azevedo R, Oliveira R, Maria M, Zago F. Social and clinical characterization of men with prostate cancer treated at a university hospital. *Rev Min Enferm.* 2015;19. <https://doi.org/10.5935/1415-2762.20150035>.
19. Löbler r. Epidemiologic profile of patients with prostate cancer submitted to a public hospital in southern brazil. *Rev bras onc clin.* 2015;11:25–28.
20. Quijada pds, fernandes pa, oliveira ds, santos bmo. Prostate cancer: Picture of a reality of patients in treatment. *Rev enferm ufpe online.* 2017;11:2490-99.
21. Mota tr, barros dp. Profile of prostate cancer patients at a referral hospital in the state of pernambuco, pernambuco cancer hospital, recife-pe, brazil, 2019. *Rev bras anal clin.* 2019. <http://doi.org/10.21877/2448-3877.201900766>.
22. da Silva JF, da Silva AM, Lima-Luz L, Aydos RD, Mattos IE. Correlation between agricultural production, clinical and demographic variables and prostate cancer: An ecological study. *Cien Saude Colet.* 2015;20(9):2805-12. <https://doi.org/10.1590/1413-81232015209.00582015>.
23. Silva IAd, Messias MCA, Weller M. Fatores de risco associados ao desenvolvimento do câncer de próstata: Uma revisão da literatura brasileira. *Arch Health Invest.* 2024;13(1):83-91. <https://doi.org/10.21270/archi.v13i1.6316>.
24. Oliveira JS, Ferreira RS, Santos LM, Marin LJ, Corrêa RX, Luizon MR, et al. Self-declared ethnicity and genomic ancestry in prostate cancer patients from brazil. *Genet Mol Res.* 2016;15(4). <https://doi.org/10.4238/gmr15048769>.
25. Koseki I, Rosso M, Sá G, Conti R, Bernardi R, Silva A, et al. Prostate cancer profiles and associated factors in criciúma – santa catarina, brazil. *Medicina (Ribeirao Preto Online).* 2019;52:104-9. <https://doi.org/10.11606/issn.2176-7262.v52i2p104-109>.
26. Benedito E, Brito N, Weller M. Risk factors of prostate cancer: A case-control study in northeast brazil fatores de risco do câncer de próstata: Estudo caso-controle no nordeste do brasil. *Saúde e Pesquisa.* 2022;10072. <https://doi.org/10.17765/2176-9206.2022v15n1.e10072>.
27. Chang AJ, Autio KA, Roach M, 3rd, Scher HI. High-risk prostate cancer-classification and therapy. *Nat Rev Clin Oncol.* 2014;11(6):308-23. <https://doi.org/10.1038/nrclinonc.2014.68>.
28. World health organization (WHO). Global report on obesity: A comprehensive overview of the state of the global obesity epidemic. 2022. WHO, GENEVA, switzerland.
29. Facin D, Gomes M, Domingues M. Atividade física e câncer colorretal: Estudo de caso-controle no município de pelotas. *Revista Brasileira de Cancerologia.* 2021;67. <https://doi.org/10.32635/2176-9745.RBC.2021v67n4.1457>.
30. Lima FE, Fisberg RM, Slater B. Development of a Quantitative Food Frequency Questionnaire (QFFQ) for a case-control study of diet and breast cancer in João Pessoa-PB. *Brazilian Journal of Epidemiology.* 2003;6:373-9.
31. Lima FE, Latorre Mdo R, Costa MJ, Fisberg RM. Diet and cancer in northeast brazil: Evaluation of eating habits and food group consumption in relation to breast cancer. *Cad Saude Publica.* 2008;24(4):820-8. <https://doi.org/10.1590/s0102-311x2008000400012>.
32. Alves Ribeiro RR, Rolim de Brito I, Andrade Souza K, de Castro Souza L, Almeida de Oliveira T, Weller M. Risk of colorectal cancer in a brazilian population is differentially associated with the intake of processed meat and vitamin e. *Nutr Cancer.* 2022;74(3):820-9. <https://doi.org/10.1080/01635581.2021.1926519>.
33. Crispim SP, Fisberg RM, Almeida CCB, Nicolas G, Knaze V, Pereira RA, et al. Photographic Manual of Food Quantification, 1st ed., 2017, Federal University of Paraná, Curitiba, Paraná, Brazil.
34. Esposito K, Chiodini P, Capuano A, Bellastella G, Maiorino MI, Parretta E, et al. Effect of metabolic syndrome and its components on prostate cancer risk: Meta-analysis. *J Endocrinol Invest.* 2013;36(2):132-9. <https://doi.org/10.1007/bf03346748>.
35. Liang Z, Xie B, Li J, Wang X, Wang S, Meng S, et al. Hypertension and risk of prostate cancer: A systematic review and meta-analysis. *Sci Rep.* 2016;6:31358. <https://doi.org/10.1038/srep31358>.
36. Dickerman BA, Torfadottir JE, Valdimarsdottir UA, Wilson KM, Steingrimsdottir L, Aspelund T, et al. Midlife metabolic factors and prostate cancer risk in later life. *Int J Cancer.* 2018;142(6):1166-73. <https://doi.org/10.1002/ijc.31142>.
37. Gao X, Li R, Jin T, Tang H. The association between metabolic syndrome and prostate cancer risk: A large-scale investigation and study of chinese. *Front Endocrinol (Lausanne).* 2022;13:787268. <https://doi.org/10.3389/fendo.2022.787268>.
38. Reckelhoff JF. Gender differences in the regulation of blood pressure. *Hypertension.* 2001;37(5):1199-208. <https://doi.org/10.1161/01.hyp.37.5.1199>.
39. Pernar CH, Ebot EM, Wilson KM, Mucci LA. The epidemiology of prostate cancer. *Cold Spring Harb Perspect Med.* 2018;8(3):353-61. <https://doi.org/10.1101/cshperspect.a030361>.
40. Johns LE, Houlston RS. A systematic review and meta-analysis of familial prostate cancer risk. *BJU Int.* 2003;91(9):789-94. <https://doi.org/10.1046/j.1464-410x.2003.04232.x>.
41. Ren ZJ, Cao DH, Zhang Q, Ren PW, Liu LR, Wei Q, et al. First-degree family history of breast cancer is associated with prostate cancer risk: A systematic review and meta-analysis. *BMC Cancer.* 2019;19(1):871. <https://doi.org/10.1186/s12885-019-6055-9>.
42. Bergengren O, Pekala KR, Matsoukas K, Fainberg J, Mungovan SF, Bratt O, et al. 2022 update on prostate cancer epidemiology and risk factors-a systematic review. *Eur Urol.* 2023;84(2):191-206. <https://doi.org/10.1016/j.eururo.2023.04.021>.
43. Clements MB, Vertosick EA, Guerrios-Rivera L, De Hoedt AM, Hernandez J, Liss MA, et al. Defining the impact of family history on detection of high-grade prostate cancer in a large multi-institutional cohort. *Eur Urol.* 2022;82(2):163-9. <https://doi.org/10.1016/j.eururo.2021.12.011>.
44. Jansson KF, Akre O, Garmo H, Bill-Axelsson A, Adolfsson J, Stattin P, et al. Concordance of tumor differentiation among brothers with prostate cancer. *Eur Urol.* 2012;62(4):656-61. <https://doi.org/10.1016/j.eururo.2012.02.032>.
45. Giovannucci E, Rimm EB, Ascherio A, Colditz GA, Spiegelman D, Stampfer MJ, et al. Smoking and risk of total and fatal prostate cancer in united states health professionals. *Cancer Epidemiol Biomarkers Prev.* 1999;8(4 Pt 1):277-82.
46. Sawada N, Inoue M, Iwasaki M, Sasazuki S, Yamaji T, Shimazu T, et al. Alcohol and smoking and subsequent risk of prostate cancer in japanese men: The japan public health center-based prospective study. *Int J Cancer.* 2014;134(4):971-8. <https://doi.org/10.1002/ijc.28423>.
47. Jochems SHJ, Fritz J, Häggström C, Järholm B, Stattin P, Stocks T. Smoking and risk of prostate cancer and prostate cancer death: A pooled study. *Eur Urol.* 2023;83(5):422-31.



- <https://doi.org/10.1016/j.eururo.2022.03.033>.
48. Ellis ET, Fairman BJ, Stahr SD, Bensen JT, Mohler JL, Song L, et al. Cigarette smoking and prostate cancer aggressiveness among african and european american men. *Cancer Causes Control*. 2024;35(9):1259-69. <https://doi.org/10.1007/s10552-024-01883-3>.
  49. Huncharek M, Haddock KS, Reid R, Kupelnick B. Smoking as a risk factor for prostate cancer: A meta-analysis of 24 prospective cohort studies. *Am J Public Health*. 2010;100(4):693-701. <https://doi.org/10.2105/ajph.2008.150508>.
  50. Cui H, Zhang W, Zhang L, Qu Y, Xu Z, Tan Z, et al. Risk factors for prostate cancer: An umbrella review of prospective observational studies and mendelian randomization analyses. *PLoS Med*. 2024;21(3):e1004362. <https://doi.org/10.1371/journal.pmed.1004362>.
  51. Al-Fayez S, El-Metwally A. Cigarette smoking and prostate cancer: A systematic review and meta-analysis of prospective cohort studies. *Tob Induc Dis*. 2023;21:19. <https://doi.org/10.18332/tid/157231>.
  52. Yang X, Chen H, Zhang S, Chen X, Sheng Y, Pang J. Association of cigarette smoking habits with the risk of prostate cancer: A systematic review and meta-analysis. *BMC Public Health*. 2023;23(1):1150. <https://doi.org/10.1186/s12889-023-16085-w>.
  53. Souza C, Tomazi L, Da D, Oliveira S. Smoking as a risk factor for prostate cancer in the population of southwestern bahia, 2019. *Revista Saúdecom*. 2019;15. <https://doi.org/10.22481/rsc.v15i4.4994>.
  54. Huang Y, Cao D, Chen Z, Chen B, Li J, Guo J, et al. Red and processed meat consumption and cancer outcomes: Umbrella review. *Food Chem*. 2021;356:129697. <https://doi.org/10.1016/j.foodchem.2021.129697>.
  55. Nouri-Majd S, Salari-Moghaddam A, Aminianfar A, Larijani B, Esmailzadeh A. Association between red and processed meat consumption and risk of prostate cancer: A systematic review and meta-analysis. *Front Nutr*. 2022;9:801722. <https://doi.org/10.3389/fnut.2022.801722>.
  56. Grosso G, La Vignera S, Condorelli RA, Godos J, Marventano S, Tieri M, et al. Total, red and processed meat consumption and human health: An umbrella review of observational studies. *Int J Food Sci Nutr*. 2022;73(6):726-37. <https://doi.org/10.1080/09637486.2022.2050996>.
  57. Oczkowski M, Dziendzikowska K, Pasternak-Winiarska A, Włodarek D, Gromadzka-Ostrowska J. Dietary factors and prostate cancer development, progression, and reduction. *Nutrients*. 2021;13(2):496. <https://doi.org/10.3390/nu13020496>.
  58. Brazilian institute of geography and statistics (ibge). Household budget survey 2017–2018: First results. Brazilian institute of geography and statistics; 2019.
  59. Rezende LFM, Malhão TA, da Silva Barbosa R, Schilithz AOC, da Silva RCF, Moreira LGM, et al. The current and future costs of colorectal cancer attributable to red and processed meat consumption in brazil. *BMC Health Serv Res*. 2023;23(1):1182. <https://doi.org/10.1186/s12913-023-10169-4>.
  60. Liu B, Mao Q, Cao M, Xie L. Cruciferous vegetables intake and risk of prostate cancer: A meta-analysis. *Int J Urol*. 2012;19(2):134-41. <https://doi.org/10.1111/j.1442-2042.2011.02906.x>.
  61. Richman EL, Carroll PR, Chan JM. Vegetable and fruit intake after diagnosis and risk of prostate cancer progression. *Int J Cancer*. 2012;131(1):201-10. <https://doi.org/10.1002/ijc.26348>.
  62. Rutz J, Thaler S, Maxeiner S, Chun FK, Blaheta RA. Sulforaphane reduces prostate cancer cell growth and

proliferation in vitro by modulating the cdk-cyclin axis and expression of the cd44 variants 4, 5, and 7. *Int J Mol Sci*. 2020;21(22):8724. <https://doi.org/10.3390/ijms21228724>.



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