

Original Article

Environmental and Psycho-social Factors Related to Prostate Cancer Risk in the Chinese Population: a Case-control Study*



LI Mei Ling¹, LIN Ji¹, HOU Jian Guo², XU Lei³, CUI Xin Gang⁴, XU Xing Xing^{1,5},
YU Yong Wei⁶, HAN Xue⁷, WANG Guo Min³, GUO Jian Ming³, XU Dan Feng⁴,
THOMPSON Timothy C⁸, CAO Guang Wen¹, and ZHANG Hong Wei^{1, #}

1. Department of Epidemiology, Secondary Military Medical University, Shanghai 200433, China; 2. Department of Urology, Changhai Hospital, Second Military Medical University, Shanghai 200433, China; 3. Department of Urology, Zhongshan Hospital, Fudan University, Shanghai 200032, China; 4. Department of Urology, Changzheng Hospital, Second Military Medical University, Shanghai 200003, China; 5. Center for Disease Control and Prevention of Zhabei District, Shanghai 200072, China; 6. Department of pathology, Changhai Hospital, Second Military Medical University, Shanghai 200032, China; 7. Center for Disease Control and Prevention of Yangpu District, Shanghai 200090, China; 8. Department of Genitourinary Medical Oncology-Research, University of Texas MD Anderson Cancer Center Houston, 77030, USA

Abstract

Objective To study the risk environmental and psycho-social factors associated to prostate cancer (PCa) in Chinese population.

Methods 250 PCa patients and 500 controls were enrolled in this case-control study. Information was collected and logistic regression analysis was used to estimate the odds ratios (OR) and 95% confidence intervals (95% CI) for relationship between lifestyle, eating habits and psycho-social factors with PCa risk.

Results Green vegetables and green tea were associated with a decreased risk of PCa (OR=0.39, 95% CI: 0.28-0.53; OR=0.59, 95% CI: 0.40-0.87, respectively). Family history of PCa (OR=7.16, 95% CI: 2.01-25.49), history of prostate diseases (OR=2.28, 95% CI: 1.53-3.41), alcohol consumption (OR=1.97, 95% CI: 1.33-2.90), red meat consumption (OR=1.74, 95% CI: 1.20-2.52), barbecued (OR=2.29, 95% CI: 1.11-4.73) or fried (OR=2.35, 95% CI: 1.24-4.43) foods were related with increased PCa risk. Negative psycho-social factors including occupational setbacks (OR=1.61, 95% CI: 1.00-2.59), marital separation (OR=1.94, 95% CI: 1.29-2.91), self-contained suffering (OR=2.37, 95% CI: 1.58-3.55), and high sensitivity to the personal comments (OR=1.73, 95% CI: 1.18-2.54) were related to PCa.

Conclusion Regular consumption of green vegetables and green tea may suggest protective effects on PCa. Alcohol consumption, red meat consumption and barbecued or fried foods were associated with PCa. Negative psycho-social factors may also play a role in the incidence of PCa in Chinese population.

Key words: Case-control study; Lifestyle; Dietary factors; Psycho-social factors; Prostate cancer

Biomed Environ Sci, 2014; 27(9): 707-717

doi: 10.3967/bes2014.089

ISSN: 0895-3988

www.besjournal.com (full text)

CN: 11-2816/Q

Copyright ©2014 by China CDC

*This work was supported by grants from the National Natural Science Foundation of China (No.81072377).

#Correspondence should be addressed to ZHANG Hong Wei, Tel: 86-21-81871061, E-mail: hwzhang@smmu.edu.cn

Biographical note of the first author: LI Mei Ling, female, born in 1988, Master Degree, occupying in molecular epidemiology of cancer.

INTRODUCTION

Prostate cancer (PCa) is the second common male malignant tumor and the sixth leading cause of cancer deaths worldwide, and it has remarkable geographical and ethnic difference in incidence and mortality^[1]. The age-standardized incidence rate of PCa (per 100,000 persons) was 104.2 in Australia/New Zealand, 94.1 in Western Europe, 85.6 in Northern America, but only 8.2 in Eastern Asia. As for age-standardized mortality rates of these countries, the highest mortality rate occurs in Australia/New Zealand (15.4/100,000) compared with that in Western Europe (12.4), Northern America (9.9) and Eastern Asia (2.5)^[1]. In 2008, the age-standardized incidence of PCa by world population was 6.73/100,000, which was accounted for 3.33% of men's malignant tumors incidence in China, and the average annual growth rate was 12.07% during 1998-2008^[2]. Although the incidence rate of PCa in China has remained relatively low, it has rapidly increased in recent years. Apart from aging population that contributes to an increased incidence of PCa, environmental factors can also play a role in increasing PCa risk^[3]. Currently, the well-established risk factors for PCa are ethnicity, age and family history of PCa^[4]. In addition, some modifiable lifestyle factors such as smoking and obesity have been found to be associated with the incidence of PCa^[5]. A recent study also found that heavy smoking was related to increased risk of PCa and higher Gleason grade in African American men, but not in European American men^[6]. Several studies indicated that consumption of vegetables, green tea and physical activity may be protective factors for PCa^[7-9]. Although some studies have shown associations between PCa and environmental factors that are specific to different ethnic backgrounds, overall the results of these studies are inconclusive. Psycho-social factors have also been recognized as important factors in the development of cancer and in cancer treatment^[10-11], but few of these studies were focused on PCa. To address this issue, we conducted a case-control study to investigate the associations between PCa and lifestyle, eating habits, and psycho-social factors in the Chinese population.

MATERIALS AND METHODS

Study Design

One 1:2 case-control study was conducted

based on hospital and community populations. 250 cases and one group of 250 controls were from Changhai and Changzheng Hospitals of the Second Military Medical University, and Zhongshan Hospital of the Fudan University, between 1 January 2007 and 1 July 2013. These three hospitals were separately located in Yangpu, Huangpu, and Xuhui District in Shanghai city. Another group of 250 controls were from communities. A questionnaire was designed to collect the basic demographic and environmental information (including lifestyle, history of diseases, and eating habits) for both of cases and controls. We also explored psycho-social factors including negative life events and personality which might be related to the incidence of PCa. The investigators had been trained to interview the subjects by face-to-face method with the designed questionnaires.

Cases and Controls Selection

250 cases were newly diagnosed patients with PCa by histopathological verification after radical prostatectomy. 250 controls were non-tumor patients from pneumology or Cardiology departments from the same hospitals as cases, and the other 250 controls were non-tumor community people with normal value of PSA (<4.0 ng/mL) from different District in Shanghai. All controls were matched by race and age (within 5 years old), location and free of PCa. All of the controls provided blood samples for the examination of PSA. Those with PSA value >4.0 ng/mL were excluded from the study.

Data Collection

All participants signed informed consents and provided data with regard to basic demographic information, lifestyle and habits, history of diseases, family history of tumor, and eating habits applicable to the most recent one-year period. The demographic information included primarily nationality, age, educational level, occupation, weight, and height. Information collected with regard to eating habits included the intake frequency and quantity of meat (red meat: pork, mutton, and beef) consumption, vegetable consumption, and fruit consumption per week; and the cooking methods. For the lifestyle and behaviors, the frequencies of alcohol consumption, smoking and tea consumption were investigated. We also collected information about urinary system diseases, hypertension, and diabetes mellitus. To explore the

relationships between psycho-social factors and PCa, information regarding specific adverse life events and personality traits was included in our study. The definition for the main exposure variables were as follows; smoking: at least 1 cigarette a day for more than six months; alcohol consumption: at least once a week, lasting 6 months or more; tea consumption: at least a cup of tea a day for more than six months; physical activity: at least three times a week and more than 30 min each time; marital separation: separated from partner more than three consecutive months; self-contained suffering: a sorrowful experiences that are usually not shared with others or openly addressed, but instead, addressed in solitary fashion; high sensitivity to the personal comments from other people: be oversensitive to other peoples' comments on him; occupational setbacks: underwent serious or much setbacks during his career.

Statistical Analysis

Data were entered (double entry) and SPSS (Statistical Package for Social Sciences; Version 16.0, Chicago, IL, USA) was used for all statistical analyses. For descriptive statistics, percentages or mean±SD were given for the two groups of cases and controls. Student's *t*-test and Chi-square test was adopted to compare differences for enumerable data and category data, respectively. To study the association between exposure of environmental factors and PCa risk, logistic regression method was used for the calculation of odds ratio (OR) as well as its 95% confident interval (95% CI). Except red meat consumption (<4 times/week and ≥4 times/week), fruits (<7 times/week and ≥7 times/week) and cooking methods (No/Yes), the other dietary variables were grouped into three categories of <1 time/week, 1-3 times/week, and ≥4 times/week. All lifestyle such as green tea consumption, history of diseases and psycho-social factors were binary variables. Multivariate logistic regression model was adopted to obtain relatively independent risk factors by adjusting for confounding factors. *P*<0.05 was taken to indicate statistical significance.

RESULTS

The basic demographic characteristics of the study subjects are shown in Table 1. There were no statistically significant differences in the distribution of age, ethnic group, educational level, occupation, and BMI between the cases and the controls. The

ages of the cases and controls were ranged from 33 to 90 years (median, 70.3 years). Of them, 179 cases (71.6%) were over 65 years; 248 cases (99.2%) were self-reported Han nationality as much as that of the controls. BMI was determined at the diagnoses of PCa.

Table 2 presents the associations between lifestyle, history of disease and PCa. Green tea consumption and physical activity were significantly associated with declined risks of PCa (OR=0.66, 95% CI: 0.48-0.90; OR=0.70, 95% CI: 0.51-0.95, respectively). Family history of PCa (OR=7.92, 95% CI: 2.60-24.11), urinary system disease (OR=2.33, 95% CI: 1.47-3.68), family history of breast cancer (OR=2.75, 95% CI: 1.14-6.62), and history of prostate disease (OR=2.11, 95% CI: 1.52-2.93) were related to the increased risk of PCa. Smoking was not a dose-response relationship risk factor of PCa (OR=1.38, 95% CI: 1.02-1.88). Alcohol consumption was also correlated with an increased risk of PCa (OR=2.10, 95% CI: 1.54-2.86), and there was a significant difference with regard to the types of alcohol (i.e., liquor, rice wine, grape wine, and beer) between the alcohol consumers of case group and control group (*P*<0.001). But there was no dose-response relationship between risk of PCa and the frequency of alcohol consumption or the number of years of alcohol consumption (data not shown).

As shown in Table 3, some eating habits were related to the incidence of PCa. Among these behaviors, consumption of red meat (OR=1.51, 95% CI: 1.11-2.05), consumption of barbecued (OR=4.74, 95% CI: 2.74-8.19) or fried (OR=3.63, 95% CI: 2.23-5.90) foods increased the risk of PCa. In contrast, consumption of green vegetables (1-3 times/week: OR=0.40, *P*=0.005; ≥4 times/week: OR=0.17, *P*<0.001) and steamed food (OR=0.61, *P*=0.001) were associated with decreased risks of PCa.

The associations between psycho-social factors and PCa are shown in Table 4. The variable of negative event frequency (NEF) was the sum of different adverse life events which may significantly influence a person during his life. The analysis demonstrated that this index was associated with an increased risk of PCa (OR=2.04, 95% CI: 1.47-2.83). Some factors such as death of spouse (OR=1.85, 95% CI: 1.08-3.14), occupational setbacks (OR=2.84, 95% CI: 1.97-4.10), serious diseases (OR=1.54, 95% CI: 1.06-2.25), marital separation (OR=2.08, 95% CI: 1.49-2.89) and disharmonious relationship with spouse (OR=3.66, 95% CI: 2.10-6.36) had statistically

significant correlation with an increased risk of PCa. As for personality, self-contained suffering and high sensitivity to the personal comments from other people were risk factors of PCa, with ORs of 2.38 (95% CI: 1.73-3.27) and 2.27 (95% CI: 1.65-3.13), respectively.

Multivariate logistic regression analysis was conducted to evaluate independent factors related to the development of PCa. According to our univariate analysis, sixteen risk factors and four protective factors were selected for analysis in logistic regression model. As shown in Table 5, consumption of green vegetables and green tea had statistically significant associations with decreased risks of PCa (OR=0.39, 95% CI: 0.28-0.53; OR=0.59, 95% CI: 0.40-0.87, respectively). Occupational

setbacks (OR=1.61, 95% CI: 1.00-2.59) and two types of personality including self-contained suffering (OR=2.37, 95% CI: 1.58-3.55) and high sensitivity to the personal comments from other people (OR=1.73, 95% CI: 1.18-2.54) were psycho-social risk factors of PCa. Factors of alcohol consumption, marital separation, family history of PCa, and history of prostate diseases were associated with the development of PCa, of which family history of PCa was the most significant risk factor related to PCa (OR=7.16, 95% CI: 2.01-25.49). Some eating habits such as consumption of barbecued or fried foods and relatively high red meat consumption, were also statistically significant risk factors for PCa (OR=2.29, 95% CI: 1.11-4.73; OR=2.35, 95% CI: 1.24-4.43; OR=1.74, 95% CI: 1.20-2.52, respectively).

Table 1. The Demographic Characteristics of Cases and Controls.

Variables	Cases (n=250)		Controls (n=500)		P value
	No.	%	No.	%	
Age (years)					
<65	63	25.2	159	31.8	0.144
65-69	51	20.4	108	21.6	
70-74	60	24.0	100	20.0	
75-79	56	22.4	96	19.2	
80-84	13	5.2	32	6.4	
≥85	7	2.8	5	1.0	
Ethnic group					
Han	248	99.2	496	99.2	0.681
Others	2	0.8	4	0.8	
Educational level*					
Elementary school	34	13.6	63	12.6	0.121
Junior high school	65	26.0	164	32.8	
Senior high school	71	28.4	154	30.8	
Junior college	43	17.2	61	12.2	
University degree	37	14.8	58	11.6	
Profession/Occupation					
Worker	65	26.0	181	36.2	0.090
Farmer	29	11.6	53	10.6	
Cadres and staff	89	35.6	136	27.2	
Medical professionals	6	2.4	12	2.4	
Teacher	18	7.2	32	6.4	
Others	43	17.2	86	17.2	
Body mass index (kg/m ²)					
<24	161	64.4	357	71.4	0.104
24.0-27.9	77	30.8	118	23.6	
≥28	12	4.8	25	5.0	

Note. * Elementary school: education in 1-6 years; Junior high school: education in 7-9 years; Senior high school: education in 10-12 years; Junior college: education in 13-15 years; University degree: education in 13-17 years.

Table 2. The Association between Lifestyle, History of Diseases, and Prostate Cancer

Variables	Cases (n=250)		Controls (n=500)		OR (95% CI)	P value
	No.	%	No.	%		
Smoking status						
No	103	41.2	246	49.2	1.00	0.038
Yes	147	58.8	254	50.8	1.38 (1.02-1.88)	
Alcohol consumption						
No	118	47.2	326	65.2	1.00	<0.0001
Yes	132	52.8	174	34.8	2.10 (1.54-2.86)	
The frequency of alcohol consumption						
<1 time /W	118	47.2	326	65.2	1.00	
1-4 times/W	74	29.6	96	19.2	2.13 (1.47-3.08)	
>4 times/W	58	23.2	78	15.6	2.05 (1.38-3.06)	<0.0001
The number of years of alcohol consumption						
<1 year	118	47.2	326	65.2	1.00	0.001
1-25 years	50	20.0	69	13.8	2.00 (1.32-3.05)	
>25 years	82	32.8	105	21.0	2.16 (1.51-3.09)	
Tea consumption						
No	90	36.0	130	26.0	1.00	0.005
Yes	160	64.0	370	74.0	0.63 (0.45-0.87)	
Green tea consumption						
No	106	42.4	163	32.6	1.00	0.008
Yes	144	57.6	337	67.4	0.66 (0.48-0.90)	
Black tea consumption						
No	242	96.8	479	95.8	1.00	0.503
Yes	8	3.2	21	4.2	0.75 (0.33-1.73)	
Physical activity						
No	110	44.0	177	35.4	1.00	0.022
Yes	140	56.0	323	64.6	0.70 (0.51-0.95)	
History of prostate diseases						
No	152	60.8	383	76.6	1.00	<0.0001
Yes	98	39.2	117	23.4	2.11 (1.52-2.93)	
Urinary system diseases						
No	207	82.8	459	91.8	1.00	<0.0001
Yes	43	17.2	41	8.2	2.33 (1.47-3.68)	
Family history of prostate cancer						
No	235	94.0	496	99.2	1.00	<0.0001
Yes	15	6.0	4	0.8	7.92 (2.60-24.11)	
Family history of breast cancer						
No	238	95.2	491	98.2	1.00	0.019
Yes	12	4.8	9	1.8	2.75 (1.14-6.62)	

Table 3. The Association between Eating Habits and Prostate Cancer

Variables/Frequency	Cases (n=250)		Controls (n=500)		OR (95% CI)	P value
	No.	%	No.	%		
Red meat consumption						
<4 times/W	122	48.8	295	59.0	1.00	0.008
≥4 times/W	128	51.2	205	41.0	1.51 (1.11-2.05)	
Animal offal						
<1 time/W	130	52.0	299	59.8	1.00	0.061
1-3 times/W	115	46.0	197	39.4	1.34 (0.99-1.83)	
≥4 times/W	5	2.0	4	0.8	2.88 (0.76-10.88)	
Poultry						
<1 time/W	121	48.4	248	49.6	1.00	0.728
1-3 times/W	100	40.0	217	43.4	0.95 (0.69-1.30)	
≥4 times/W	29	11.6	35	7.0	1.70 (0.99-2.91)	
Fish						
<1 time/W	50	20.0	112	22.4	1.00	0.939
1-3 times/W	112	44.8	247	49.4	1.02 (0.68-1.52)	
≥4 times/W	88	35.2	141	28.2	1.40 (0.91-2.14)	
Soy products						
<1 time/W	57	22.8	94	18.8	1.00	0.147
1-3 times/W	114	45.6	252	50.4	0.75 (0.50-1.11)	
≥4 times/W	79	31.6	154	30.8	0.85 (0.55-1.30)	
Green vegetables						
<1 time/W	36	14.4	17	3.4	1.00	0.005
1-3 times/W	72	28.8	86	17.2	0.40 (0.21-0.76)	
≥4 times/W	142	56.8	397	79.4	0.17 (0.09-0.31)	
Fruits						
<7 times/W	144	57.6	279	55.8	1.00	0.639
≥7 times/W	106	42.4	221	44.2	0.93 (0.68-1.26)	
Cooking methods						
Steam/stew						
No	124	49.6	187	37.4	1.00	0.001
Yes	126	50.4	313	62.6	0.61 (0.45-0.83)	
Boil						
No	60	24.0	111	22.2	1.00	0.580
Yes	190	76.0	389	77.8	0.90 (0.63-1.29)	
Barbecue						
No	207	82.8	479	95.8	1.00	<0.001
Yes	43	17.2	21	4.2	4.74 (2.74-8.19)	
Fry						
No	203	81.2	470	94.0	1.00	<0.0001
Yes	47	18.8	30	6.0	3.63 (2.23-5.90)	

Table 4. The Association between Psycho-social Factors and Prostate Cancer

Risk Factors	Cases (n=250)		Controls (n=500)		OR (95% CI)	P value
	No.	%	No.	%		
Introverted personality						
No	157	62.8	341	68.2	1.00	0.140
Yes	93	37.2	159	31.8	1.27 (0.92-1.75)	
Self-endure suffering ^a						
No	80	32.0	264	52.8	1.00	<0.0001
Yes	170	68.0	236	47.2	2.38 (1.73-3.27)	
High sensitivity to the personal comments from other people ^b						
No	141	56.4	373	74.6	1.00	<0.0001
Yes	109	43.6	127	25.4	2.27 (1.65-3.13)	
Negative event frequency						
≤2 times	152	60.8	380	76.0	1.00	<0.0001
>2 times	98	39.2	120	24.0	2.04 (1.47-2.83)	
Death of spouse						
No	222	88.8	468	93.6	1.00	0.022
Yes	28	11.2	32	6.4	1.85 (1.08-3.14)	
Occupational setbacks ^c						
No	170	68.0	429	85.8	1.00	<0.0001
Yes	80	32.0	71	14.2	2.84 (1.97-4.10)	
Serious disease						
No	192	76.8	418	83.6	1.00	0.024
Yes	58	23.2	82	16.4	1.54 (1.06-2.25)	
Life difficulty						
No	201	80.4	424	84.8	1.00	0.127
Yes	49	19.6	76	15.2	1.36 (0.92-2.02)	
Marital separation						
No	157	62.8	389	77.8	2.08 (1.49-2.89)	<0.0001
Yes	93	37.2	111	22.2		
Disharmonious relationship with spouse						
No	214	85.6	478	95.6	1.00	<0.0001
Yes	36	14.4	22	4.4	3.66 (2.10-6.36)	
Death of parent						
No	80	32.0	192	38.4	1.00	0.086
Yes	170	68.0	308	61.6	1.33 (0.96-1.83)	
Unfortunate of relatives ^d						
No	202	80.8	427	85.4	1.00	0.106
Yes	48	19.2	73	14.6	1.39 (0.93-2.08)	

Note. ^aSelf-endure suffering: a sorrowful experiences that are usually not shared with others or openly addressed, but instead, addressed in solitary fashion. ^bHigh sensitivity to the personal comments from other people: be oversensitive to other peoples’ comments on him. ^cOccupational setbacks: underwent serious or much setbacks during his career. ^dUnfortunate of relatives: his relatives got serious diseases or died which can bring a serious impact on him.

Table 5. Multivariate Logistic Regression Analysis for Risk Factors of Prostate Cancer

Factors	β	SE	OR (95% CI)	P value
Green vegetables ^a	-0.946	0.159	0.39 (0.28-0.53)	<0.0001
Green tea consumption	-0.528	0.197	0.59 (0.40-0.87)	0.0070
Red meat consumption ^b	0.555	0.189	1.74 (1.20-2.52)	0.0030
Barbecue cooking	0.830	0.370	2.29 (1.11-4.73)	0.0250
Fry cooking	0.853	0.324	2.35 (1.24-4.43)	0.0080
Alcohol consumption	0.676	0.198	1.97 (1.33-2.90)	0.0010
Family history of prostate cancer	1.968	0.648	7.16 (2.01-25.49)	0.0020
History of prostate diseases	0.825	0.205	2.28 (1.53-3.41)	<0.0001
High sensitivity to the personal Comments from other people	0.548	0.195	1.73 (1.18-2.54)	0.0050
Self-endure suffering	0.862	0.207	2.37 (1.58-3.55)	<0.0001
Occupational setbacks	0.477	0.242	1.61 (1.00-2.59)	0.0490
Marital separation	0.662	0.206	1.94 (1.29-2.91)	0.0010

Note. All of the variables in this table are binary except ‘green vegetables’ and ‘red meat consumption’.
^a It is classified to three groups of ‘<1 time/W’, ‘1-3 times/W’, and ‘≥4 times/W’. ^b It is classified to two groups of ‘<4 times/W’ and ‘≥4 times/W’.

DISCUSSION

The present study investigated the risk factors related to PCa in Chinese men. The results suggested that some environmental factors were associated with the risk of PCa, such as consumption of green tea and alcohol, family history of PCa, red meat consumption, green vegetables and barbecued or fried foods. The results from this study also showed that some psycho-social factors, such as self-contained suffering, more sensitive to other personal comments, occupational setbacks and marital separation were related to the risk of PCa. In addition to environmental factors, psycho-social factors may also associate with the development of PCa in Chinese population.

Green tea consumption was an independent protective factor for PCa in our study. A case-control study in southeast China also found that the consumption of green tea was a statistically significantly protective factor for PCa, and there was an association between the decreased risk of PCa and the duration and quantity of green tea consumption^[12]. One prospective cohort study indicated that there was an inverse association between green tea consumption and advanced PCa^[13]. However, results from another prospective cohort study did not show green tea consumption as a protective factor for PCa^[14]. To date, epidemiological studies have not resulted in conclusive and consistent evidence for an anticancer

effect of green tea with regard to PCa. This may be due to the differences in study design, tea consumption patterns and confounding factors among different populations. With regard to the mechanism of the chemoprevention functions of green tea, some studies from cell culture experiments, animal studies, preclinical and clinical trials showed that specific components of green tea, especially catechins, were able to influence vital signal transduction pathways and alter enzyme activities, resulting in suppression of cell proliferation, induction of apoptosis, suppression of angiogenesis, and inhibition of invasion and metastasis of PCa^[15-19].

Family history of PCa was an important risk factor of PCa for Chinese males in our study. It has also been confirmed that family history of PCa was the most important risk factor in the development of PCa within other ethnic groups^[20-21]. The risk of PCa in U.K. males with a first-degree family history of PCa was approximately 2.5-fold higher than that in men without a family history^[22]. In our study, we also found that the condition of non-malignant prostate disease was a risk factor for PCa. This may be related to an association between prostate diseases and inflammation, which has been shown to contribute to the risk of prostate carcinogenesis^[23-25].

Alcohol consumption may alter the levels of estrogen and androgen by influencing hormone metabolism^[26-27], which in turn can affect the hormone dependent status of malignancies such as breast cancer or PCa. Therefore, the role of alcohol

consumption in PCa has become an important aspect of investigation in the field. Our results showed that alcohol consumption was associated with increased risk of PCa. However, a dose-dependent relationship was not found. With regard to alcohol types, cases of PCa preferred Chinese liquor, compared to controls (results were not showed). Epidemiologic studies conducted in Western countries demonstrated that alcohol consumption was not associated with the incidence of PCa^[28-29], but may be related to advanced PCa^[30]. A recent prospective study in Japan demonstrated a positive association between alcohol consumption and PCa. The results of that study showed that alcohol consumption was associated with advanced PCa in a dose-dependent fashion^[31]. One case-control study from southern China showed that alcohol consumers carrying the CYP1A1 Val allele or the CYP2E1C1/C1 genotype were at increased risk of PCa^[32], which indicated that alcohol may increase the risk of PCa through the interaction with specific SNPs. Furthermore, alcohol consumption could influence sex steroid hormone levels and result in higher testosterone and lower SHBG of alcohol consumers^[33]. This may be another potential mechanism for the relationship between alcohol consumption and PCa incidence. Further studies are needed to address the mechanisms that underlie the association between alcohol consumption and PCa.

The relationship between dietary factors and the etiology of PCa has also been an area intensive investigation during recent years. Epidemiological studies indicated that the occurrence of PCa was closely associated with dietary structure and components, especially a diet rich in saturated fat, meat and milk^[34]. Our results showed that three dietary factors were related to PCa (i.e., red meat consumption, green vegetables and barbecued or fried foods). Epidemiological studies, although inconsistent, have generally been suggestive of a significant association between high meat intake and risk of PCa. A case-cohort study showed a positive association between PCa and the red meat intake^[35]. However, a prospective study demonstrated that the total meat intake was not related to risk of PCa, but that well-cooked meat was a risk factor of PCa^[36]. Saturated fatty acids contained in red meat might increase testosterone levels, which is an important hormone related to the incidence and progression of PCa^[37]. The traditional Chinese diet contains a higher content of vegetables and soybean products, and is lower in red meat content than other diets; this may

be related to the lower incidence of PCa than that in Western countries.

Our study showed that consumption of green vegetables was an independent protective factor for the development of PCa. Vegetables and fruits contain a variety of vitamins, minerals, and other nutrients such as vitamin C, vitamin E, selenium, lycopene, which have been associated with reduced incidence, progression and mortality of PCa^[38-40]. With regard to fruit consumption, we did not demonstrate a positive association with PCa; this result is consistent with another study^[41]. Recent evidence revealed that soy products contain relatively large amounts of isoflavonoids which could influence estrogen and testosterone metabolism, and soy products are important protective factors for the development and mortality of PCa^[42-43]. In our study, there was no statistically significant difference in soy products consumption between the case group and control group. A plant-based diet with low fat intake may contribute to the prevention of PCa and inhibition of PCa progression. Further studies are warranted to ascertain the role of diet in PCa.

The results of our research also indicated that different methods of food preparation were highly associated with the incidence of PCa. Consumption of barbecued food and fried food were both independently correlated with increased risk of PCa. The results of previous studies have shown that barbecued and fried foods contain large quantities of carcinogenic chemicals such as polycyclic aromatic hydrocarbons, heterocyclic amines, acrylamide and acrolein^[44]. These are consistent with our study. A recent study showed that intake ≥ 1 /week of French fries, fried chicken and fried fish were positively associated with PCa^[45]; intake of well-done grilled or barbecued red meat was shown to increase the risk of aggressive PCa^[46]. Based on the positive relationship between specific methods of food preparation and PCa risk, increased attention should be paid to this diet-related factor.

In our study, we found that personality such as self-contained suffering; high sensitivity to the personal comments from other people had a positive association with the development of PCa. It was also found that occupational setbacks and marital separation were associated with the risk of PCa. In addition to environmental factors, psycho-social factors may also play an important role in the process of carcinogenesis. Psychological trauma can produce negative emotions in individuals,

which may lead to a decline in immune function, and contribute to cancer incidence and progression^[47]. Chronic stress or a high incidence of traumatic life events may reduce host resistance to carcinogenesis^[48]. A study on the relationship between adverse life events and breast cancer risk showed that breast cancer patients had significantly more adverse life events and difficult life situations than controls did^[49]. Our results suggest that chronic negative psycho-social factors, including the suffering from adverse life events may decrease immune function, disturb the balance of metabolism and hormone secretion, and ultimately contribute to carcinogenesis. Psychological counseling should be regarded as an essential and primary component for PCa prevention and treatment.

In this study, effective methods were used to control biases which may influence the quality of case-control studies. During this study, maximum effort was paid to study design, information collection and statistical analysis. For controlling selection bias, all cases and controls were strictly selected in accordance with the inclusive criteria. Furthermore, we designed two types of control for making a more representative control group. For decreasing information bias, all investigators were trained strictly and comprehensively to ensure the consistency and accuracy for understanding the questionnaires. In addition, all cases and controls were volunteered to participate this study and the participants(>80 years old) were interviewed by twice, even the effective methods were taken in this study, the recall bias can not be avoided by the inclusion of very elderly subjects (more than 80 years old). Finally, we excluded the patients who can not answer the questionnaires by himself or spouse. As for confounding bias, all controls were matched by race and age. However, there are some limitations in our study. First, the sample sizes including 250 cases and 500 controls may be not sufficient to detect weak associations between environmental factors and PCa. Second, with regard to the interview that addressed eating habits, the recall bias cannot be fully avoided; this is a common bias in case-control studies. Third, the interview design for psycho-social factors was not comprehensive; even though we recruited newly diagnosed case of PCa and conducted our interview within short time after the diagnoses, there is also a possibility that if the patient knows the information of his illness, he would have a more negative perception of the world around him, which may

affect the data quality of psychosocial factors; our results with regard to psycho-social factors need to be validated in further studies. In summary, with above effective measures used to control and reduce possible bias, the results from this study were reliable, and provided new information to understand the risk or protective factors of PCa in Chinese population.

CONCLUSION

The results of our study demonstrate that the consumption of green tea and green vegetables had protective effects on the incidence of PCa in the Chinese population. Family history of prostate or breast cancer, smoking, alcohol consumption, red meat consumption, barbecued or fried foods and history of non-malignant prostate diseases can be regarded as primary potential risk factors for the onset of PCa in Chinese men. Adverse life events such as occupational setbacks, marital separation and other psycho-social factors such as self-contained suffering, high sensitivity to the personal comments from other people may be risk factors in the development of PCa. Further studies should focus on the mechanisms of these risk factors.

Received: February 18, 2014;

Accepted: July 29, 2014

REFERENCES

1. Jemal A, Bray F, Center MM, et al. Global cancer statistics. *CA Cancer J Clin*, 2011; 61, 69-90.
2. Han SJ, Zhang SW, Chen WQ, et al. Analysis of the status and trends of prostate cancer incidence in China. *Chinese Clinical Oncology*, 2013; 18, 330-4. (In Chinese)
3. Hassler S, Sjolander P, Gronberg H, et al. Cancer in the Sami population of Sweden in relation to lifestyle and genetic factors. *Eur J Epidemiol*, 2008; 23, 273-80.
4. Gallagher RP, Fleshner N. Prostate cancer: 3 Individual risk factors. *CMAJ*, 1998; 159, 807-13.
5. Leitzmann MF, Rohrmann S. Risk factors for the onset of prostatic cancer: age, location, and behavioral correlates. *Clin Epidemiol*, 2012; 4, 1-11.
6. Murphy AB, Akereyeni F, Nyame YA, et al. Smoking and prostate cancer in a multi-ethnic cohort. *Prostate*, 2013; 73, 1518-28.
7. Chan R, Lok K, Woo J. Prostate cancer and vegetable consumption. *Mol Nutr Food Res*, 2009; 53, 201-16.
8. Thakur VS, Gupta K, Gupta S. Green tea polyphenols causes cell cycle arrest and apoptosis in prostate cancer cells by suppressing class I histone deacetylases. *Carcinogenesis*, 2012; 33, 377-84.
9. Marie-élise Parent, Marie-Claude Rousseau, Mariam El-Zein, et al. Occupational and recreational physical activity during adult life and the risk of cancer among men. *Cancer Epidemiology*,

- 2011; 35, 151-9.
- 10.Zittoun R. Objectives of the study of the quality of life in oncology. *Bull Cancer*, 1986; 73, 601-6.
- 11.Van Andel G, Visser AP, Hulshof MC, et al. Health-related quality of life and psychosocial factors in patients with prostate cancer scheduled for radical prostatectomy or external radiation therapy. *BJU Int*, 2003; 92, 217-22.
- 12.Jian L, Xie LP, Lee AH, et al. Protective effect of green tea against prostate cancer: a case-control study in southeast China. *Int J Cancer*, 2004; 108, 130-5.
- 13.Kurahashi N, Sasazuki S, Iwasaki M, et al. Green tea consumption and prostate cancer risk in Japanese men: a prospective study. *Am J Epidemiol*, 2008; 167, 71-7.
- 14.Montague JA, Butler LM, Wu AH, et al. Green and black tea intake in relation to prostate cancer risk among Singapore Chinese. *Cancer Causes Control*, 2012; 23, 1635-41.
- 15.Johnson JJ, Bailey HH, Mukhtar H. Green tea polyphenols for prostate cancer chemoprevention: a translational perspective. *Phytomedicine*, 2010; 17, 3-13.
- 16.Syed DN, Suh Y, Afaq F, et al. Dietary agents for chemoprevention of prostate cancer. *Cancer Lett*, 2008; 265, 167-76.
- 17.Pandey M, Gupta S. Green tea and prostate cancer: from bench to clinic. *Front Biosci (Elite Ed)*, 2009; 1, 13-25.
- 18.Khan N, Adhami VM, Mukhtar H. Review: green tea polyphenols in chemoprevention of prostate cancer: preclinical and clinical studies. *Nutr Cancer*, 2009; 61, 836-41.
- 19.Henning SM, Wang P, Said J, et al. Polyphenols in brewed green tea inhibit prostate tumor xenograft growth by localizing to the tumor and decreasing oxidative stress and angiogenesis. *J Nutr Biochem*, 2012; 23, 1537-42.
- 20.Chen YC, Page JH, Chen R, et al. Family history of prostate and breast cancer and the risk of prostate cancer in the PSA era. *Prostate*, 2008; 68, 1582-91.
- 21.Madersbacher S, Alcaraz A, Emberton M, et al. The influence of family history on prostate cancer risk: implications for clinical management. *BJU Int*, 2011; 107, 716-21.
- 22.Johns LE, Houlston RS. A systematic review and meta-analysis of familial prostate cancer risk. *BJU Int*, 2003; 91, 789-94.
- 23.Sfanos KS, De Marzo AM. Prostate cancer and inflammation: the evidence. *Histopathology*, 2012; 60, 199-215.
- 24.Narayanan NK, Nargi D, Horton L, et al. Inflammatory processes of prostate tissue microenvironment drive rat prostate carcinogenesis: preventive effects of celecoxib. *Prostate*, 2009; 69, 133-41.
- 25.Nakai Y, Nonomura N. Inflammation and prostate carcinogenesis. *Int J Urol*, 2013; 20, 150-60.
- 26.Gordon GG, Altman K, Southren AL, et al. Effect of alcohol (ethanol) administration on sex-hormone metabolism in normal men. *N Engl J Med*, 1976; 295, 793-7.
- 27.Sierksma A, Sarkola T, Eriksson CJ, et al. Effect of moderate alcohol consumption on plasma dehydroepiandrosterone sulfate, testosterone, and estradiol levels in middle-aged men and postmenopausal women: a diet-controlled intervention study. *Alcohol Clin Exp Res*, 2004; 28, 780-5.
- 28.Baglietto L, Severi G, English DR, et al. Alcohol consumption and prostate cancer risk: results from the Melbourne collaborative cohort study. *Int J Cancer*, 2006; 119, 1501-4.
- 29.Rohrmann S, Linseisen J, Key TJ, et al. Alcohol consumption and the risk for prostate cancer in the European Prospective Investigation into Cancer and Nutrition. *Cancer Epidemiol Biomarkers Prev*, 2008; 17, 1282-7.
- 30.Gong Z, Kristal AR, Schenk JM, et al. Alcohol consumption, finasteride and prostate cancer risk: results from the Prostate Cancer Prevention Trial. *Cancer*, 2009; 115, 3661-9.
- 31.Sawada N, Inoue M, Iwasaki M, 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, 971-8.
- 32.Yang J, Qian LX, Wu HF, et al. Genetic polymorphisms in the cytochrome P4501A1 and 2E1 genes, smoking, drinking and prostate cancer susceptibility: a case-control study in a Han nationality population in Southern China. *Int J Urol*, 2006; 13, 773-80.
- 33.Shiels MS, Rohrmann S, Menke A, et al. Association of cigarette smoking, alcohol consumption, and physical activity with sex steroid hormone levels in US men. *Cancer Causes Control*, 2009; 20, 877-86.
- 34.Raimondi S, Mabrouk JB, Shatenstein B, et al. Diet and prostate cancer risk with specific focus on dairy products and dietary calcium: a case-control study. *Prostate*, 2010; 70, 1054-65.
- 35.Illir Agalliu, Victoria A. Kirsh, Nancy Kreiger, et al. Oxidative balance score and risk of prostate cancer: Results from a case-cohort study. *Cancer Epidemiology*, 2011; 35, 353-61.
- 36.Cross AJ, Peters U, Kirsh VA, et al. A prospective study of meat and meat mutagens and prostate cancer risk. *Cancer Res*, 2005; 65, 11779-84.
- 37.Kolonel LN, Nomura AM, Cooney RV. Dietary fat and prostate cancer: current status. *J Natl Cancer Inst*, 1999; 91, 414-28.
- 38.Aune D, De Stefani E, Ronco A, et al. Fruits, vegetables and the risk of cancer: a multisite case-control study in Uruguay. *Asian Pac J Cancer Prev*, 2009; 10, 419-28.
- 39.Ambrosini GL, de Klerk NH, Fritschi L, et al. Fruit, vegetable, vitamin A intakes, and prostate cancer risk. *Prostate Cancer Prostatic Dis*, 2008; 11, 61-6.
- 40.Konijeti R, Henning S, Moro A, et al. Chemoprevention of prostate cancer with lycopene in the TRAMP model. *Prostate*, 2010; 70, 1547-54.
- 41.Kirsh VA, Peters U, Mayne ST, et al. Prospective study of fruit and vegetable intake and risk of prostate cancer. *J Natl Cancer Inst*, 2007; 99, 1200-9.
- 42.Lee MM, Gomez SL, Chang JS, et al. Soy and isoflavone consumption in relation to prostate cancer risk in China. *Cancer Epidemiol Biomarkers Prev*, 2003; 12, 665-8.
- 43.Gardner CD, Oelrich B, Liu JP, et al. Prostatic soy isoflavone concentrations exceed serum levels after dietary supplementation. *Prostate*, 2009; 69, 719-26.
- 44.Sugimura T. Carcinogenicity of mutagenic heterocyclic amines formed during the cooking process. *Mutat Res*, 1985; 150, 33-41.
- 45.Stott-Miller M, Neuhauser ML, Stanford JL. Consumption of deep-fried foods and risk of prostate cancer. *Prostate*, 2013; 73, 960-9.
- 46.Punnen S, Hardin J, Cheng I, et al. Impact of meat consumption, preparation, and mutagens on aggressive prostate cancer. *PLoS One*, 2011; 6, e27711.
- 47.Schussler G, Schubert C. The influence of psychosocial factors on the immune system (psychoneuroimmunology) and their role for the incidence and progression of cancer. *Z Psychosom Med Psychother*, 2001; 47, 6-41.
- 48.Palesh O, Butler LD, Koopman C, et al. Stress history and breast cancer recurrence. *J Psychosom Res*, 2007; 63, 233-9.
- 49.Forsen A. Psychosocial stress as a risk for breast cancer. *Psychother Psychosom*, 1991; 55, 176-85.