Coffee consumption and risk of localized, advanced and fatal prostate cancer: a population-based prospective study

A. Discacciati¹, N. Orsini¹, S.-O. Andersson², ³, O. Andrén², ³, J.-E. Johansson², ³, C.S. Mantzoros⁴, ⁵ & A. Wolk¹

¹Unit of Nutritional Epidemiology, Division of Epidemiology, Institute of Environmental Medicine, Karolinska Institutet, Stockholm; ²School of Health and Medical Sciences, Örebro University, Örebro; ³Department of Urology, Örebro University Hospital, Örebro, Sweden; ⁴Division of Endocrinology, Diabetes and Metabolism, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston; ⁵Section of Endocrinology, Boston VA Healthcare System, Harvard Medical School, Boston, USA

Received 10 September 2012; revised 23 January 2013; accepted 4 February 2013

Background: The epidemiological evidence on possible relationships between coffee consumption and prostate cancer (PCa) risk by subtype of the disease (localized, advanced) and fatal PCa risk is limited.

Materials and methods: A population-based cohort of 44 613 Swedish men aged 45–79 years was followed up from January 1998 through December 2010 for incidence of localized (n = 2368), advanced (n = 918) and fatal (n = 515) PCa. We assessed the associations between coffee consumption and localized, advanced and fatal PCa risk using competing-risk regressions. We examined possible effect modification by body mass index (BMI).

Results: For localized PCa, each one cup increase in daily coffee consumption was associated with a 3% reduced risk [sub-hazard ratio (SHR) = 0.97, 95% confidence interval (CI) = 0.95–0.99]. For advanced and fatal PCa, we found a non-significant inverse association; each one cup increase was associated with a 2% reduced risk of advanced [SHR (95% CI) = 0.98 (0.95–1.02)] and fatal PCa [SHR (95% CI) = 0.98 (0.93–1.03)]. We observed evidence of effect modification by BMI for localized PCa (Pinteraction = 0.03); the inverse association was stronger among overweight and obese men (BMI ≥ 25 kg/m²) compared with normal-weight men (BMI < 25 kg/m²).

Conclusions: We observed a clear inverse association between coffee consumption and risk of localized PCa, especially among overweight and obese men.

Key words: coffee, epidemiology, prospective cohort study, prostate cancer

introduction

Coffee, the most common beverage in the Western world excluding water [1], has been observed to reduce the concentrations of both plasma insulin and insulin-like growth factor 1 levels (IGF-1) through increased levels of adiponectin [2–6], an endogenous insulin sensitiser which is inversely associated with obesity [7–9]. Coffee has also been observed to be associated with increased sex hormone-binding globulin (SHBG) concentrations [10, 11] and reduced oxidative stress [12–14]. These effects on insulin, IGF-1, SHBG and oxidative stress may also be relevant to development and progression of prostate cancer (PCa). Therefore, an association between coffee consumption and PCa risk, possibly modified by body size, is biologically plausible.

The association of coffee consumption with total PCa incidence was examined in five small cohort studies, all of which observed non-statistically significant associations [15–19]. Incidence of PCa by subtype of the disease, namely localized/low-grade and advanced/high-grade, was examined in only two cohorts and an inverse relationship with the risk of advanced [20] and high-grade PCa [21] was observed. PCa mortality was statistically significantly inversely associated with coffee consumption in one cohort study [20], but not in two others [22, 23]. None of the previous studies examined the possibility of effect modification by body size.

As the available evidence is limited, the aim of our population-based cohort study is to examine the relationships between coffee consumption and risk of localized, advanced and fatal PCa. Since Sweden is one of the countries with the highest coffee consumption per capita worldwide [24], our cohort provides a particularly wide range of coffee consumption. In addition, our study is the first to examine whether the association between coffee consumption and risk
of localized, advanced and fatal PCa may be modified by body size, as measured by body mass index (BMI).

materials and methods
The population-based cohort of Swedish men was established in 1997–1998, when all the men (n = 100 303) aged 45–79 years residing in Västmanland and Örebro counties in central Sweden received an invitation to participate in the study, along with a self-administered questionnaire. A total of 48 645 men returned the questionnaire, which included questions about coffee consumption, as well as information about diet, smoking habits, weight, height and physical activity and other lifestyle factors.

We excluded participants who returned an incomplete questionnaire (n = 92), died before 1 January 1998 (n = 55) or had a previous cancer diagnosis (n = 2592). We also excluded men with missing (n = 1121) or implausible (>12 cups/day) coffee consumption (n = 172), leaving thus 44 613 men available for the analyses. This population-based cohort is representative of Swedish males aged between 45 and 79 years in terms of age distribution, prevalence of overweight and educational level [25]. PCa incident rates are also comparable: for example, in 1998, the incident rates in Sweden and in the cohort, among men aged 45–79 years, were 314 and 339 cases per 100 000 men, respectively [26].

assessment of coffee consumption and covariates
Data on coffee consumption were collected at baseline through a self-administered food-frequency questionnaire (FFQ) that included 96 food items commonly consumed in the study population. Participants reported their daily or weekly average coffee consumption (in cups) during the previous year. We calculated coffee consumption in grams per day by multiplying the number of cups by age-specific coffee servings. The cup size was standardized to 200 ml (200 g) of coffee beverage. The FFQ-based information on coffee has been validated against data from 14 24-h recall interviews over 1 year in a group of 248 men (Spearman’s coefficient = 0.71) (Wolk, unpublished).

Data on tea and alcohol consumption were collected in a similar way as that for coffee. Details on the assessment of the other covariates are available elsewhere [27].

case ascertainment and follow-up of the cohort
Study participants were followed from 1 January 1998 to 31 December 2010. Incident cases of PCa were ascertained by computerized record linkage with the Swedish National Cancer Register which has been estimated to be nearly 100% complete [28]. Information on tumor-node-metastasis (TNM) stage, Gleason grade and prostate-specific antigen (PSA) values was available from the Swedish Prostate Cancer Quality Registry. We classified incident cases by subtype as localized (T1-2 and NX-0 and (MX-0 or PSA < 20 ng/ml or Gleason grade ≤ 7)) or advanced (T3-4 and NX-1 and (MX-1 or PSA > 100 ng/ml or Gleason grade ≥ 8]).

Information on fatal PCa cases was ascertained through linkage to the Swedish Register of Death Causes at the National Board of Health and Welfare. Classification of deaths was based on International Classification of Diseases (ICD-10, code 61).

statistical analysis
Descriptive statistics, age-standardized to the distribution of the entire cohort, were used to present the baseline characteristics of the study participants according to categories of coffee consumption: none, <1, 1–3, 4–5 or ≥6 cups/day.

Fine and Gray’s competing-risk regressions were used to estimate PCa SHRs and 95% CIs for each category of coffee consumption compared with the most frequent category (1–3 cups/day) [29]. For the localized PCa incidence analysis, we considered death and non-localized PCa cases as competing events. We defined competing events for the advanced PCa incidence analysis in an analogous fashion. For the analysis of fatal PCa, deaths from other causes than PCa were considered as competing events.

For incident PCa analyses, each subject contributed person-time from 1 January 1998 until the date of PCa diagnosis, death from any cause or study end (31 December 2010), whichever came first. For fatal PCa analysis, each participant accrued follow-up time from 1 January 1998 until the date of PCa death, death from other causes or study end (31 December 2010), whichever came first. Age was used as the time scale [30].

Multivariable models were adjusted for potential confounders (Table 2). We checked whether the assumption of proportionality of the sub-hazards was reasonable by regressing Schoenfeld’s residuals against the ranks of analysis time. There was no evidence of departure from this assumption.

We calculated the SHR associated with each one cup/day increase in coffee consumption. To examine a potential nonlinear dose-response relationship, we modeled coffee consumption using restricted cubic splines with three knots at fixed quantiles of coffee distribution (0.10, 0.50 and 0.90) [31]. A P-value for nonlinearity was calculated by testing that the coefficient of the second RCS transformation was different from zero. Furthermore, possible effect modification by BMI (<25 versus ≥25 kg/m²) was tested by adding an interaction term in the competing-risk regressions.

Undiagnosed PCa at baseline might have produced lower urinary tract symptoms (LUTSs), which in turn could have led to a reduced coffee consumption. Therefore, we examined whether LUTSs were associated with lower coffee consumption at baseline, leading thus to possible reverse causation. Using information from the baseline FFQ, we calculated the International Prostate Symptom Score (IPSS), ranging from 0 (no symptoms) to 35 (severe LUTS). Details on the calculation of this score are available elsewhere [32]. We utilized logistic quantile regression to examine a possible association between IPSS and median coffee consumption, controlling for age [33].

All reported P-values are two-sided. All statistical analyses were carried out with Stata release 12.1 (StataCorp, College Station, TX).

results
During 13 years of follow-up, we documented 2 368 localized and 918 advanced cases of PCa, while 315 cases were unclassified (508 401 person-years). During the same period, we documented 515 cases of fatal PCa (525 550 person-years). The median (interquartile range) coffee consumption in the cohort was 3 (2–4) cups per day. Age-standardized baseline characteristics by category of coffee consumption are presented in Table 1. Men who consumed the most coffee (≥6 cups/day) were younger and more likely to be less-educated and current smokers. Higher levels of coffee consumption were associated with lower levels of tea consumption (Spearman’s coefficient = −0.3). Mean BMI, physical activity and alcohol consumption did not appreciably vary across categories of coffee consumption.

For localized PCa, we observed an inverse dose–response relationship with coffee consumption (Table 2). From the multivariable competing-risk regression, we observed that men who consumed six or more cups per day had a 19% reduced
risk compared with men in the reference category (1–3 cups/day) \([\text{SHR (95% CI)} = 0.81 (0.69–0.96)]\). Cases characterized by a Gleason grade equal to seven might be a combination of more benign and malignant tumors. Therefore, we carried out a sensitivity analysis where, in the classification criteria for localized PCa cases, we replaced the condition ‘Gleason grade \(\leq 7\)’ with the more stringent condition ‘Gleason grade < 7’.

The results from this analysis did not virtually change \([\text{SHR (95% CI)} = 0.81 (0.69–0.96)]\).

By modeling coffee consumption as a continuous variable, we observed a 3% decreased risk of localized PCa for each one cup increase in daily coffee consumption \([\text{SHR (95% CI)} = 0.97 (0.95–0.99)]\). In a restricted cubic spline model, we found no evidence of a nonlinear dose–response relationship.

### Table 1. Age-adjusted baseline characteristics by coffee consumption in the cohort of Swedish men aged 45–79 years

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Category of coffee consumption</th>
<th>None</th>
<th>Less than one cup per day</th>
<th>One to three cups per day</th>
<th>Four to five cups per day</th>
<th>Six or more cups per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of subjects</td>
<td></td>
<td>1993</td>
<td>3436</td>
<td>24 571</td>
<td>9744</td>
<td>4869</td>
</tr>
<tr>
<td>Age at baseline (mean, years)</td>
<td></td>
<td>61</td>
<td>65</td>
<td>62</td>
<td>38</td>
<td>55</td>
</tr>
<tr>
<td>Tea consumption (mean, g/day)</td>
<td></td>
<td>597</td>
<td>321</td>
<td>167</td>
<td>94</td>
<td>70</td>
</tr>
<tr>
<td>Body Mass Index (BMI, mean, kg/m²)</td>
<td></td>
<td>26</td>
<td>26</td>
<td>26</td>
<td>26</td>
<td>26</td>
</tr>
<tr>
<td>Physical activity (mean, MET-h/day)</td>
<td></td>
<td>41</td>
<td>41</td>
<td>41</td>
<td>42</td>
<td>42</td>
</tr>
<tr>
<td>Alcohol consumption (mean, g/day)</td>
<td></td>
<td>9</td>
<td>10</td>
<td>11</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Total energy intake (mean, kcal/day)</td>
<td></td>
<td>2624</td>
<td>2475</td>
<td>2589</td>
<td>2805</td>
<td>2962</td>
</tr>
<tr>
<td>Education (university, %)</td>
<td></td>
<td>22</td>
<td>20</td>
<td>18</td>
<td>14</td>
<td>11</td>
</tr>
<tr>
<td>History of diabetes (yes, %)</td>
<td></td>
<td>9</td>
<td>11</td>
<td>9</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>Family history of prostate cancer (PCa, yes, %)</td>
<td></td>
<td>7</td>
<td>8</td>
<td>7</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Smoking status (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current smoker</td>
<td></td>
<td>17</td>
<td>18</td>
<td>21</td>
<td>29</td>
<td>43</td>
</tr>
<tr>
<td>Former smoker</td>
<td></td>
<td>32</td>
<td>38</td>
<td>40</td>
<td>40</td>
<td>36</td>
</tr>
<tr>
<td>Never smoker</td>
<td></td>
<td>51</td>
<td>43</td>
<td>39</td>
<td>31</td>
<td>21</td>
</tr>
</tbody>
</table>

\(^a\)All factors (except age) were adjusted to the age distribution of the study participants at baseline.

\(^b\)Cup size standardized to 200 g.

MET, metabolic equivalent of task.

### Table 2. Sub-hazard ratios (SHRs) with 95% confidence interval (95% CI) of prostate cancer (PCa) by category of coffee consumption in the cohort of Swedish men, 1998–2010

<table>
<thead>
<tr>
<th>Median coffee consumption (g/day)</th>
<th>Category of coffee consumption (cups/day)</th>
<th>$P_{\text{trend}}$</th>
<th>For every one cup/day increase</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>None</td>
<td>&lt;1</td>
<td>1–3</td>
</tr>
<tr>
<td>Localized PCa</td>
<td>129/22 622</td>
<td>212/36 379</td>
<td>1397/275 029</td>
</tr>
<tr>
<td>Age-adjusted SHR (95% CI)</td>
<td>1.21 (1.01–1.45)</td>
<td>1.01 (0.88–1.17)</td>
<td>1.00 (0.86–1.16)</td>
</tr>
<tr>
<td>Fully adjusted SHR (95% CI)</td>
<td>1.13 (0.93–1.37)</td>
<td>1.00 (0.86–1.16)</td>
<td>0.93 (0.83–1.03)</td>
</tr>
<tr>
<td>Advanced PCa</td>
<td>37/22 622</td>
<td>93/36 379</td>
<td>582/275 029</td>
</tr>
<tr>
<td>Age-adjusted SHR (95% CI)</td>
<td>0.87 (0.62–1.21)</td>
<td>0.95 (0.76–1.18)</td>
<td>0.94 (0.79–1.13)</td>
</tr>
<tr>
<td>Fully adjusted SHR (95% CI)</td>
<td>0.96 (0.68–1.35)</td>
<td>0.97 (0.78–1.21)</td>
<td>0.95 (0.79–1.14)</td>
</tr>
<tr>
<td>Fatal PCa</td>
<td>28/23 490</td>
<td>63/37 952</td>
<td>316/285 611</td>
</tr>
<tr>
<td>Age-adjusted SHR (95% CI)</td>
<td>1.24 (0.84–1.82)</td>
<td>1.15 (0.88–1.50)</td>
<td>1.04 (0.82–1.33)</td>
</tr>
<tr>
<td>Fully adjusted SHR (95% CI)</td>
<td>1.24 (0.83–1.97)</td>
<td>1.19 (0.90–1.56)</td>
<td>1.01 (0.79–1.30)</td>
</tr>
</tbody>
</table>

\(^a\)All SHRs are from models with age as the time scale. Multivariable models were additionally adjusted for tea (g/day, continuous), alcohol (g/day, continuous), BMI (kg/m², continuous), personal history of diabetes (yes or no), family history of PCa (yes, no or don’t know), smoking status (current, former, never smoker or missing), physical activity (MET-h/day, continuous), education (less than high school, high school, university or missing) and total energy intake (kcal/day, continuous). SHRs and 95% CIs were calculated using Fine and Gray’s competing-risk regressions.

\(^b\)Cup size standardized to 200 g.

Linear trends across categories were tested using the median coffee consumption within categories as a continuous variable.

SHR, sub-hazard ratio; CI, confidence interval; BMI, body mass index.
incident rates of localized PCa, directly standardized to the age distribution of the cohort, were 601 and 412 cases per 100,000 person-years for the lowest (nondrinkers) and highest (≥6 cups/day) categories of coffee consumption, respectively.

For advanced and fatal PCa, we observed a non-significant inverse linear association with coffee consumption. Each one cup increase in daily coffee consumption was associated with a 2% reduced risk of advanced [SHR (95% CI) = 0.98 (0.95–1.02)] and fatal PCa [SHR (95% CI) = 0.98 (0.93–1.03)]. No evidence of nonlinearity was observed for advanced (P_{nonlinearity} = 0.15) or fatal PCa (P_{nonlinearity} = 0.62). Age-standardized incident rates of advanced PCa for the two extreme categories were 182 (nondrinkers) and 168 (≥6 cups/day) cases, respectively, per 100,000 person-years. Age-standardized PCA mortality rates were 125 (nondrinkers) and 88 (≥6 cups/day) PCa deaths per 100,000 person-years.

The relationships between coffee consumption, modeled as a continuous variable, and the risk of localized, advanced and fatal PCa are presented graphically in Figure 1.

We examined whether the association between coffee consumption and localized, advanced and fatal PCa differed according to the BMI (<25 versus ≥25 kg/m²). We observed evidence of effect modification for localized PCa (P_{interaction} = 0.03). Among overweight and obese men (BMI ≥ 25 kg/m²), we observed a threshold effect with a decreased risk of localized PCa among coffee drinkers compared with nondrinkers. Among normal-weight men (BMI < 25 kg/m²), this threshold effect was not observed (Table 3). We observed no evidence of effect modification for advanced (P_{interaction} = 0.83) or fatal PCa (P_{interaction} = 0.40). A similar threshold effect for localized PCa was observed among obese men, when we stratified by waist circumference (<102 versus ≥102 cm) (data not shown).

To examine a potential effect of reverse causation on our results, we assessed whether LUTSs were associated with lower coffee consumption at baseline. Using a logistic quantile regression, we observed no statistically significant association between the IPSS score and median coffee consumption, controlling for age (P = 0.82). Furthermore, we excluded the first 4 years of follow-up from the analyses to examine whether preclinical PCa symptoms could have affected coffee consumption. The multivariable results did not appreciably change; SHRs for each one cup increase in daily coffee consumption were 0.96 (0.94–0.98), 0.99 (0.96–1.03) and 0.97 (0.92–1.02) for localized, advanced and fatal PCa, respectively.

discussion

In this population-based prospective cohort study, we observed a statistically significant inverse linear relationship between coffee consumption and the risk of localized PCa, where each one cup increase in daily coffee consumption was associated with a 3% reduced risk. Coffee consumption was also observed to be nonsignificantly associated with a 2% lower risk of advanced and of fatal PCa for each one cup increase. For localized PCa, we observed evidence of effect modification by BMI.
Only two studies on coffee consumption and incidence of PCa by subtype of the disease have been carried out so far [20, 21]. The authors of the Health Professionals Follow-Up study, based on 896 advanced and 3221 non-advanced PCa cases, observed a significant 53% decreased risk of advanced PCa among high coffee consumers (≥ 6 cups/day) compared with nondrinkers [relative risk (RR) (95% CI) = 0.47 (0.28–0.77)]. In a small prospective Scottish study, including 70 high-grade (Gleason > 7) and 41 low-grade PCa cases (Gleason < 7), a 53% decreased risk of high-grade PCa was observed among men who consumed three or more cups per day compared with nondrinkers [RR (95% CI) = 0.47 (0.22–1.01)]. A non-statistically significant 46% reduction in low-grade PCa risk was observed for high coffee consumers (≥ 3 cups/day) [21].

Two prospective cohort studies examined the association between coffee consumption and fatal PCa [22, 23], while a third used a comparable composite outcome [20]. The Seventh-day Adventists cohort, based on 93 PCa deaths, found a non-statistically significant 30% reduced risk of fatal PCa in men drinking two or more cups per day compared with nondrinkers. However, this study adjusted only for age and the range of the exposure was particularly narrow [22]. In the study by Hsing et al., including 149 PCa deaths, no association between coffee consumption and fatal PCa was observed [23]. The authors of the Health Professionals Follow-Up study, based on 642 lethal PCa cases, observed a RR of 0.40 (95% CI = 0.22–0.75) comparing high drinkers (≥ 6 cups/day) with nondrinkers [20].

Intrinsic differences among the study populations as well as differences in coffee preparation which can influence its composition, such as coffee brewing methods, brewing strength or coffee bean roasting, could explain the different observed findings. Different biological mechanisms may explain an inverse association between coffee consumption and risk of fatal PCa [22, 23], while a direct association between lower consumption and fatal PCa [24].

Adiponectin concentration is inversely associated with obesity [7, 9]. Thus, increased adiponectin levels due to coffee consumption may be more beneficial to overweight and obese men. This would explain the threshold effect that we observed among overweight and obese men, where already a limited
amount of daily coffee consumption was sufficient to reduce the risk of localized PCAs compared with nondrinkers. Reverse causation could in principle explain our findings, since men with an undiagnosed PCAs might reduce their coffee consumption as a consequence of urinary symptoms. However, no association between urinary symptoms and median coffee consumption was observed in our data. Furthermore, the observed associations did not change after excluding the first 4 years of follow-up.

The principal limitation of this study is that it relied on self-reported coffee consumption, which could have probably led to some non-differential misclassification. However, we observed a good validity of the FFQ-based information on coffee consumption when compared with 14 24-h recall interviews. We have no information on the method of coffee preparation or on whether study participants consumed regular or decaffeinated coffee. However, the majority of men in our cohort likely consumed filtered coffee; the authors of a previous Swedish study observed that 84% of the male coffee drinkers consumed only filtered coffee [41]. Decaffeinated coffee is virtually nonexistent in Sweden and, additionally, similar associations with PCa risk were observed for both regular and decaffeinated coffee in the Health Professionals Follow-up Study [20]. Our study was observational, and thus, we cannot completely rule out the possibility of unmeasured or residual confounding as an explanation of our results. Finally, we cannot completely rule out the possibility of unmeasured or on whether study participants consumed regular or decaffeinated coffee. However, the majority of men in our cohort likely consumed filtered coffee; the authors of a previous Swedish study observed that 84% of the male coffee drinkers consumed only filtered coffee [41]. Decaffeinated coffee is virtually nonexistent in Sweden and, additionally, similar associations with PCa risk were observed for both regular and decaffeinated coffee in the Health Professionals Follow-up Study [20]. Our study was observational, and thus, we cannot completely rule out the possibility of unmeasured or residual confounding as an explanation of our results. Finally, we cannot completely rule out this possibility.

Strengths of this study include the large size of the cohort, its population-based prospective design, the large number of PCa cases and the completeness of case ascertainment. These features reduced the potential risk of selection and recall bias, increased the generalizability of the study findings and allowed us to examine the relationships between coffee consumption and the risk of PCa by subtype of the disease and, at the same time, a possible effect modification by body size. In addition, since Sweden is one of the countries with the highest coffee consumption per capita worldwide [24], the range of coffee consumption in this cohort was particularly wide, with 11% of the men consuming six or more cups per day.

In conclusion, we found that regular coffee consumers had a lower risk of localized PCAs compared with nondrinkers, especially among overweight and obese men. We also observed weak evidence of a decreased risk of advanced and fatal PCAs. Our results are potentially important from a public health point of view, given the lack of established modifiable risk factors for PCAs. Existing epidemiological evidence is, however, still too limited to recommend men to increase their coffee consumption in order to decrease PCa risk. Replication of other prospective epidemiological studies, focusing on PCa subtypes and PCa mortality, as well as further work in understanding the underlying biological mechanisms, is warranted before conclusive public health recommendations could be made.

**funding**

This work was supported by research grants from the Swedish Cancer Foundation (Cancerfonden) and from the Swedish Research Council/Committee for Infrastructure.

**disclosure**

The authors have declared no conflicts of interest.

**references**

Cruciferous vegetables consumption and the risk of female lung cancer: a prospective study and a meta-analysis

Q. J. Wu¹,²,³, L. Xie²,³, W. Zheng⁴, E. Vogtmann⁵, H. L. Li²,³, G. Yang⁴, B. T. Ji⁶, Y. T. Gao³, X. O. Shu⁴ & Y. B. Xiang²,³*

¹Department of Epidemiology, School of Public Health, Fudan University, Shanghai; ²State Key Laboratory of Oncogene and Related Genes, Shanghai Cancer Institute, Shanghai; ³Department of Epidemiology, Shanghai Cancer Institute, Renji Hospital, Shanghai Jiaotong University School of Medicine, Shanghai, China; ⁴Division of Epidemiology, Department of Medicine, Vanderbilt Epidemiology Center, Vanderbilt-Ingram Cancer Center, Vanderbilt University School of Medicine, Nashville; ⁵Department of Epidemiology, University of Alabama at Birmingham, Birmingham; ⁶Division of Cancer Epidemiology and Genetics, National Cancer Institute, Rockville, USA

Received 18 December 2012; revised 9 February 2013; accepted 11 February 2013

Background: Epidemiological studies evaluating the association between cruciferous vegetables (CVs) intake and female lung cancer risk have produced inconsistent results.

Patients and methods: This study followed 74,914 Chinese women aged 40–70 years who participated in the Shanghai Women’s Health Study. CV intake was assessed through a validated food-frequency questionnaire (FFQ) at baseline and reassessed during follow-up. Hazard ratios (HRs) and 95% confidence interval (CIs) were estimated by using Cox proportional hazards models. Furthermore, we carried out a meta-analysis of all observational studies until December 2011.

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