HHS Public Access

Author manuscript

Cancer Causes Control. Author manuscript; available in PMC 2015 August 01.

Published in final edited form as:

Cancer Causes Control. 2015 August ; 26(8): 1173–1180. doi:10.1007/s10552-015-0611-7.

Association of Hypertension and Obesity with Renal Cell Carcinoma Risk: A Report from the Shanghai Men's and Women's Health Studies

Tian Shen,
Division of Epidemiology, Vanderbilt University School of Medicine, Nashville, TN, USA

Visiting from Fudan University School of Public Health and Shanghai Jiao Tong University School of Public Health, Shanghai, China

Xiao-Ou Shu,
Division of Epidemiology, Vanderbilt University School of Medicine, Nashville, TN, USA

Yong-Bing Xiang,
Shanghai Cancer Institute, Shanghai, China

Hong-Lan Li,
Shanghai Cancer Institute, Shanghai, China

Hui Cai,
Division of Epidemiology, Vanderbilt University School of Medicine, Nashville, TN, USA

Yu-Tang Gao,
Shanghai Cancer Institute, Shanghai, China

Wei Zheng, and
Division of Epidemiology, Vanderbilt University School of Medicine, Nashville, TN, USA

Loren Lipworth*
Division of Epidemiology, Vanderbilt University School of Medicine, Nashville, TN, USA

Abstract

Purpose—Consistently reported associations between hypertension and obesity and renal cell carcinoma (RCC) risk have largely come from studies in Western populations. These associations were examined in a case-control study nested in the Shanghai Women's Health Study (SWHS, 1996–2000) and Shanghai Men's Health Study (SMHS, 2001–2006).

Methods—Overall, 271 incident RCC cases (124 women, 147 men) were identified through December 31, 2011, and 2,693 controls were individually matched by sex, age and calendar time at cohort enrollment, and menopausal status (for women). Participants completed a structured questionnaire by in-person interview at baseline, and conditional logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs).

*Correspondence to Dr. Loren Lipworth, Division of Epidemiology, Vanderbilt University School of Medicine, 2525 West End Avenue, Suite 800, Nashville, TN, 37203-1738 (loren.lipworth@vanderbilt.edu).

Conflict of interest The authors declare that they have no conflict of interests.

Results—Self-reported hypertension was associated with a significant 40% increased risk of RCC among women and men (95% CI: 1.1, 1.9). Body mass index (BMI), modeled continuously, was associated with an elevated risk of RCC among men, with an OR of 1.5 (95% CI: 1.1, 2.0) per 5 kg/m^2 increase in BMI, but not among women.

Conclusions—Hypertension is independently associated with risk of RCC among both women and men in Shanghai, while overweight and obesity appear to be associated with risk of RCC in Chinese men only.

Keywords

Body mass index; Carcinoma, renal cell; Case-control; Hypertension; Obesity

Introduction

In the United States, kidney cancer is the 6th and 8th most commonly diagnosed primary cancer among men and women, respectively. Approximately 85% of kidney cancers are renal parenchyma (renal cell) cancers, while the remainder are mainly urothelial cancers of the renal pelvis. Epidemiologic studies, primarily conducted in Western populations, have consistently shown that hypertension and obesity are among the strongest risk factors for renal cell carcinoma (RCC). [1–5]

The incidence of RCC, as well as the prevalence of obesity and hypertension, is increasing rapidly in Asian populations. According to incidence data from 32 cancer registries in China from 2003 to 2007, the incidence of kidney cancer and unspecified urinary organs cancer was ranked 12th among all cancers[6], and the age-standardized incidence in China (3.84/10^5 in 2008) was still higher than the average level of developing countries. [7] In urban Shanghai, China, from 1972–1994, the rates for all cancers combined decreased approximately 0.5% per year, but incidence rates for kidney cancer have increased rapidly since the mid-1980s, with an annual percent increase of 4.4% for men and 2.8% for women over the 23 year period. [8] To our knowledge, only one prior case-control study has examined the associations between hypertension and obesity and RCC in a Chinese population, and data on the association in other Asian populations are sparse.

Methods

Study Population

This study is based on two ongoing prospective cohorts, the Shanghai Women's Health Study (SWHS) and the Shanghai Men's Health Study (SMHS). Briefly, the SWHS recruited 74,942 adult Chinese women aged 40–70 years from 7 urban Shanghai communities between 1996 and 2000, with a response rate of 92%; the SMHS recruited 61,480 adult Chinese men aged 40–74 years from 8 communities in urban Shanghai between 2002 and 2006, with a response rate of 74.1%. Incident cases of RCC were ascertained by the Shanghai Cancer Institute through biennial follow-up and linkage of the cohorts to the population-based Shanghai Cancer Registry.

As the latest round of follow-up for both cohorts is ongoing, and to maximize our sample size, we conducted a nested case-control study among those with complete outcome information. Through December 31, 2011, a total of 271 incident RCC cases, defined as having an International Classification of Disease, Ninth Revision (ICD-9), code of 189.0, were identified, 147 among men and 124 among women. Ten controls were randomly selected for each case by incidence density sampling from the cohorts and matched on sex, age at cohort enrollment (±2 years), calendar time of recruitment (±30 days) and menopausal status (for women). Overall, 269 cases were successfully matched with 10 controls each, while 1 female case was matched with only 2 controls and 1 male case was matched with only 1 control, yielding a total of 2,693 controls.

Data collection

After obtaining informed consent, a trained interviewer administered an in-person interview at cohort enrollment using a structured questionnaire which included information on demographics, lifestyle, dietary habits, medical history including hypertension status and other characteristics. Hypertension status was reported by the participant via two questions: “Have you ever been diagnosed with high blood pressure?” and “How old were you when you had it?” Hypertension duration was calculated as age at enrollment minus age at diagnosed hypertension. Anthropometric measurements including current weight, height, and circumferences of waist and hips were also collected during the baseline interview. Detailed methodology for SWHS and SMHS has been published elsewhere. [9, 10]

Statistical Methods

Measured current weight and height were used to calculate body mass index (BMI, kg/m^2) and measured waist and hip circumference were used to calculate waist-hip ratio (WHR) at enrollment. World Health Organization-recommended BMI cut-off points[11] were used to classify BMI into underweight (<18.5 kg/m^2), normal (18.5–24.9 kg/m^2), overweight (25–29.9 kg/m^2) and obese (≥30 kg/m^2). Secondary analyses were performed using lower cut-offs for overweight (23–24.9 kg/m^2) and obesity (≥25 kg/m^2), as has been proposed for Asian populations. WHR was categorized into quartiles of WHR, based on the distribution among cases and controls combined, separately for women and men.

Baseline characteristics were compared between cases and controls using t-tests for continuous variables and chi-square tests for categorical variables. Conditional logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for the association between RCC and various factors. Self-reported hypertension and continuous BMI (or categorical BMI, continuous WHR, categorical WHR) were included as primary exposures of interest. Additional covariates included in the models were education (elementary and below/middle and high school/high professional, college and above), smoking history (never smoker/former smoker/current smoker), current alcohol drinking status (yes/no), and family history of cancer (yes/no). Additional sensitivity analyses were conducted by excluding cases diagnosed in the first two years of follow-up and their matched controls.

All *P* values presented were 2-tailed and *P* values less than 0.05 were considered statistically significant. *P* values for trend were calculated by entering the categorical variables as a continuous variable in the model. Statistical analyses were performed using SAS 9.3 (SAS Institute, Cary, NC).

Results

Selected baseline demographic, anthropometric, lifestyle and other characteristics of the 271 RCC cases and 2,693 matched controls are presented in Table 1, overall and separately for men and women. Overall, 45.8% of the cases and controls were women. The average age at enrollment of cases and controls was 58 years, slightly younger among women than men (57 vs. 59 years). Average BMI was somewhat higher for cases (24.6 kg/m^2) than controls (24.1 kg/m^2; *P*=0.01), while mean WHR was 0.9 for both cases and controls. Although the distribution of BMI by categories did not differ significantly among cases compared with controls, either overall or stratified by sex, the prevalence of overweight or obesity (BMI≥25.0) was higher (42.0%) for cases than controls (36.7%). The frequency of self-reported hypertension among cases overall was 41.7%, which was significantly higher than that among controls (31.9%; *P*=0.001). A similar significant difference between cases and controls was observed among women and men when considered separately, although the prevalence of hypertension was higher among men. Compared with controls, cases were significantly more likely to have a family history of cancer (34.0 vs. 27.1%; *P*=0.02) and less likely to drink alcohol (11.1 vs. 16.0%; *P*=0.03); however, the prevalence of alcohol drinking was much lower among women (2.4% of cases and 3.0% of controls) than men (18.4% of cases and 26.9% of controls) and the inverse association with alcohol was significant only among men (*P*=0.03). No statistically significant differences were observed in education or smoking history between cases and controls, either overall or by sex.

Table 2 shows the adjusted ORs and 95% CIs for the associations of RCC risk with hypertension, BMI and WHR, in the total study population and separately for women and men. Self-reported hypertension was associated with a significantly increased risk of RCC overall, with an OR of 1.4 (95% CI: 1.1, 1.9) in the multivariable model. This increased risk of RCC was observed among both women and men, with ORs of 1.5 (95% CI: 0.996, 2.3) and 1.3 (95% CI: 0.9, 2.0), respectively, although neither reached statistical significance.

Among men, but not among women or overall, BMI was positively associated with risk of RCC when modeled as a continuous variable. The *P* value for the interaction between BMI and sex was 0.08. For every 5 kg/m^2 increase in BMI among men, the risk of RCC increased 50% after multivariable adjustment (95% CI: 1.1, 2.0). When BMI was modeled as a categorical variable with WHO recommended cut-offs, there was a tendency towards increased RCC risk among men in the highest category of BMI (≥30.0; OR, 1.5; 95% CI: 0.6, 4.1). Among women, increased risk of RCC was observed among those who were underweight (BMI<18.5), with an OR of 2.6 (95% CI: 1.02, 6.7), compared to normal weight women; however, the number of women in the underweight group was small. Using cut-offs proposed for an Asian population increased the numbers of participants in the top category of BMI (BMI≥25.0); however, while the ORs for both obese women and men were slightly elevated, no statistically significant association was observed between categorical

BMI and RCC risk. No significant association was observed between WHR and RCC, either overall or in sex-stratified analyses.

Similarly, neither education level nor smoking status or family history of cancer was significantly associated with RCC risk in our study population. Current alcohol drinking was modestly inversely associated with RCC risk (OR, 0.7; 95% CI: 0.5, 1.05), and similarly among women and men, albeit not statistically significant so.

Longer duration of hypertension was associated with an increased risk of RCC. Compared with the non-hypertensive group, both women and men with duration of hypertension <10 years had an increased risk of RCC, with an OR of 1.3 (95% CI: 0.9, 1.9) overall, and 1.4 (95% CI: 0.8, 2.4) for women, and 1.3 (95% CI: 0.8, 2.1) for men. For those who had hypertension for more than 10 years, the RCC OR increased somewhat to 1.5 (95% CI: 1.1, 2.1) overall, 1.6 (95% CI: 0.99, 2.7) and 1.4 (95% CI: 0.9, 2.9) for women and men, respectively. The trend was statistically significant overall (*P* value for trend=0.0118) and among women (*P* value for trend=0.0435).

When we repeated the analyses with exclusion of 12 women and 27 men whose diagnosis of RCC occurred during the first 2 years of follow-up, and their matched controls, the results were similar to the overall results presented in Tables 1 and 2. The multivariable OR comparing hypertension vs. no hypertension was 1.4 (95% CI: 1.03, 1.9) overall, 1.4 (95% CI: 0.9, 2.3) among women, and 1.3 (95% CI: 0.9, 2.1) among men; and the OR for continuous BMI per 1 kg/m^2 increase was 1.0 (95% CI: 1.004, 1.1) overall, 1.0 (95% CI: 0.9, 1.1) among women, 1.1 (95% CI: 1.03, 1.2) among men. An increased risk for RCC persisted in this analysis among underweight women (BMI<18.5), with an OR of 2.6 (95% CI: 1.02, 6.8), compared to normal weight women.

Discussion

In this case-control study nested within two large prospective cohorts, we found that self-reported hypertension is an independent risk factor for RCC, with a significant 40% increased risk among men and women in Shanghai, China, after adjusting for possible confounders. Participants who were diagnosed with hypertension 10 or more years before cancer diagnosis experienced an even greater 50% increase in risk of RCC, thereby minimizing the possibility of reverse causation and suggesting that the association between hypertension and RCC is unlikely to be a consequence of RCC. The results of our sensitivity analysis, excluding cases diagnosed within two years of cohort enrollment, lend further support to this conclusion. The finding of increased RCC risk associated with hypertension is consistent with numerous case-control and cohort studies that have been conducted in Western populations[12–18], as well as the former population-based case-control study conducted in Shanghai, China, 1992 [19].

RCC is one of the malignancies most consistently and strongly associated with overweight and obesity among both men and women, regardless of study design or population [2–4, 6, 12, 13]. A recent quantitative summary analysis of the epidemiologic evidence reported risk ratios for RCC of 1.24 (95% CI: 1.15, 1.34) among men and 1.34 (95% CI: 1.25, 1.43)

among women per 5 kg/m^2 increase in BMI [6]. Proposed mechanisms for an effect of obesity on the risk of RCC include renal hemodynamics caused by obesity; lipid peroxidation; obesity-induced chronic inflammatory state; and a special endocrine and metabolic milieu, named “obesogenic” environment, which accelerates the development of RCC [4].

In the present Chinese population-based nested case-control study, BMI was found to be associated with a significantly elevated risk of RCC among men, but not among women; the ORs corresponding to a 5 kg/m^2 increase in BMI, for comparability with the above summary analysis, were 1.5 (95% CI: 1.1, 2.0) among men and 1.0 (95% CI: 0.8, 1.3) among women. This finding is quite similar to a Korean prospective cohort study published in 2008, which reported a weaker association between BMI and kidney cancer among women than men. In that study, compared with normal BMI between 23.0 to 24.9, the age-adjusted hazard ratio for kidney cancer among men was 1.11 (95%CI: 0.93, 1.31) for overweight (BMI 25.0–29.9) and 1.38 (95%CI: 0.76, 2.52) for obesity (BMI≥30), as compared to 0.92 (95%CI: 0.64, 1.31) and 1.21 (95%CI: 0.58, 2.53), respectively, among women [20].

One possible explanation for the weaker observed association between BMI and RCC among Asians than among Europeans or Americans may be the lower prevalence of obesity among Asians, which limits the power to assess the association between BMI and RCC risk using standard BMI cut-offs. In the Shanghai Men’s and Women’s Health Studies, only 2.6% men and 5.1% women were obese (BMI≥30) [21]; in the Korean study the corresponding percentages were 0.8% and 2.4%, respectively [20]; in contrast, in most European or American population studies, this percentage was much higher, e.g. 13.8% of men and 18.0% of women in the Multiethnic Cohort [12]. Using Asian-specific cut-offs for BMI can increase the case numbers in the top category. In analyses using these modified cut-offs, we observed similar but not significant weak positive associations between obesity and RCC among men and women. However, according to a 2004 consensus statement from WHO, evidence remains insufficient to support the lower cutoff points for Asian populations to define overweight and obesity. [11] Thus, to enhance the comparability of our results with those of other studies, our primary analyses were based on the existing WHO cutoff points for overweight (BMI≥25) and obesity (BMI≥30). A significantly increased risk for RCC was observed among underweight Chinese women in our study, and it persisted in sensitivity analyses in which reverse causation was minimized, warranting additional investigation.

Although BMI is the standard index for assessing general obesity in epidemiologic studies, it does not capture body fat distribution [22–24], which varies widely across multiethnic populations for a given BMI value [25]. Waist-to-hip ratio (WHR), a surrogate measure of visceral obesity less influenced by variation in lean mass, is also reported to be positively associated with RCC in the Women’s Health Initiative Study [26] and the Iowa Women’s Health Study [26–28], and a shared genetic locus for WHR and RCC risk has recently been identified [29]. However, in the present study, no statistically significant association was observed between WHR and RCC risk among women or men, which is similar to the finding in the European Prospective Investigation into Cancer and Nutrition Study [30].

There are several strengths of our study. To our knowledge, only one epidemiologic study of RCC has been conducted among a Chinese population, a retrospective case-control study in Shanghai, China published in 1992. Thus, our study is the first based on two well-designed cohorts to prospectively examine RCC risk factors in China. All study participants were residents of urban Shanghai and ethnic Chinese, which helped to minimize potential confounding by ethnicity. We randomly selected 10 matched controls for each cases, which enable us to include all available cases in the study and at the same time to achieve a comparable statistical power as a cohort analysis were employed.

There are some limitations of the present study. First, although our study was based on two large cohort studies, the number of RCC cases was low which resulted in overall low statistical power. With additional cohort follow-up, more RCC cases will accrue which will increase the sample size and improve precision. Second, the low proportion of obese subjects (BMI≥30.0) in this study population (4.4% of cases and 4.2% of controls) limited our power to assess RCC risk in relation to categorical BMI using standard WHO cut-offs. Although the proportion of women who smoked or drank alcohol was low, which limited our ability to evaluate associations between these exposures and RCC risks in women, hypertension and BMI were the focus of our analysis and these factors were considered primarily as potential confounders. Third, we did not have measured blood pressure at baseline which could have resulted in an under estimation of the hypertension-RCC association. We also did not have specific information on antihypertensive medications use. However, most previous epidemiologic studies suggest that no particular type of commonly used antihypertensive medications was consistently associated with RCC risk, and that it is hypertension itself that plays a role in the etiology of this malignancy. [1, 4, 13, 15, 16, 31] Fourth, Shanghai is one of the most industrialized cities in China, and our study was based on two cohorts conducted in urban Shanghai. Thus, the results may not be generalized to the whole population of China, especially rural China. Finally, we cannot completely rule out potential residual confounding from unknown or imperfectly measured covariates.

In conclusion, our study, a Chinese population-based nested case-control study, found a strong association between hypertension and RCC, which is consistent with the findings worldwide. However, it is difficult to speculate why overweight and obesity appears to be associated with risk of RCC in Chinese men only. Further follow up of this cohort is needed for accrual of additional incident RCC cases, as well as additional studies in Asian populations with more thorough documentation of measured blood pressure and treatment of hypertension.

Acknowledgments

This work was supported by the Vanderbilt-Shanghai Chronic Disease Research Training Program (grant D43 TW008213). The Shanghai Women's Health Study (Grant R37 CA070867) and the Shanghai Men's Health Study (Grants R01 CA082729, UM1 CA183021) were supported by the National Cancer Institute at the National Institutes of Health.

References

1. Lipworth L, Tarone RE, McLaughlin JK. Renal cell cancer among African Americans: an epidemiologic review. BMC Cancer. 2011

2. Ildaphonse G, George PS, Mathew A. Obesity and kidney cancer risk in men: a meta-analysis (1992–2008). Asian Pac J Cancer Prev APJCP. 2009; 10:279–286.
3. Mathew A, George PS, Ildaphonse G. Obesity and kidney cancer risk in women: a meta-analysis (1992–2008). Asian Pac J Cancer Prev APJCP. 2009; 10:471–478.
4. Klinghoffer Z, Yang B, Kapoor A, Pinthus JH. Obesity and renal cell carcinoma: epidemiology, underlying mechanisms and management considerations. Expert Rev Anticancer Ther. 2009; 9:975–987. [PubMed: 19589036]
5. McLaughlin JK, Lipworth L, Tarone RE. Epidemiologic aspects of renal cell carcinoma. Semin Oncol. 2006; 33:527–533. [PubMed: 17045081]
6. Renehan AG, Tyson M, Egger M, et al. Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. Lancet. 2008; 371:569–578. [PubMed: 18280327]
7. Zhang Y, Yang G, Wang X. An analysis of incidence of kidney cancer and unspecified urinary organs cancer in China, 2003~2007. China Cancer. 2012; 21:561–565.
8. Jin F, Devesa SS, Chow WH, et al. Cancer incidence trends in urban shanghai, 1972–1994: an update. Int J Cancer J Int Cancer. 1999; 83:435–440.
9. Zheng W, Chow W-H, Yang G, et al. The Shanghai Women's Health Study: rationale, study design, and baseline characteristics. Am J Epidemiol. 2005; 162:1123–1131. [PubMed: 16236996]
10. Cai H, Zheng W, Xiang Y-B, et al. Dietary patterns and their correlates among middle-aged and elderly Chinese men: a report from the Shanghai Men's Health Study. Br J Nutr. 2007; 98:1006–1013. [PubMed: 17524168]
11. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004; 363:157–163. [PubMed: 14726171]
12. Setiawan VW, Stram DO, Nomura AMY, et al. Risk factors for renal cell cancer: the multiethnic cohort. Am J Epidemiol. 2007; 166:932–940. [PubMed: 17656615]
13. Flaherty KT, Fuchs CS, Colditz GA, et al. A prospective study of body mass index, hypertension, and smoking and the risk of renal cell carcinoma (United States). Cancer Causes Control CCC. 2005; 16:1099–1106. [PubMed: 16184476]
14. Yuan JM, Castelao JE, Gago-Dominguez M, et al. Hypertension, obesity and their medications in relation to renal cell carcinoma. Br J Cancer. 1998; 77:1508–1513. [PubMed: 9652770]
15. McLaughlin JK, Chow WH, Mandel JS, et al. International renal-cell cancer study. VIII. Role of diuretics, other anti-hypertensive medications and hypertension. Int J Cancer J Int Cancer. 1995; 63:216–221.
16. Shapiro JA, Williams MA, Weiss NS, et al. Hypertension, antihypertensive medication use, and risk of renal cell carcinoma. Am J Epidemiol. 1999; 149:521–530. [PubMed: 10084241]
17. Choi MY, Jee SH, Sull JW, Nam CM. The effect of hypertension on the risk for kidney cancer in Korean men. Kidney Int. 2005; 67:647–652. [PubMed: 15673312]
18. Weikert S, Boeing H, Pischon T, et al. Blood pressure and risk of renal cell carcinoma in the European prospective investigation into cancer and nutrition. Am J Epidemiol. 2008; 167:438–446. [PubMed: 18048375]
19. McLaughlin JK, Gao Y-T, Gao R-N, et al. Risk factors for renal-cell cancer in Shanghai, China. Int J Cancer. 1992; 52:562–565. [PubMed: 1399137]
20. Jee SH, Yun JE, Park EJ, et al. Body mass index and cancer risk in Korean men and women. Int J Cancer J Int Cancer. 2008; 123:1892–1896.
21. Luu NH, Blot WJ, Xiang YB, et al. Prospective Evaluation of the Association of Nut/Peanut Consumption With Total and Cause-Specific Mortality. JAMA Intern Med. 2015
22. Snijder MB, van Dam RM, Visser M, Seidell JC. What aspects of body fat are particularly hazardous and how do we measure them? Int J Epidemiol. 2006
23. Lovejoy JC, de la Bretonne JA, Klemperer M, Tulley R. Abdominal fat distribution and metabolic risk factors: effects of race. Metabolism. 1996; 45:1119–1124. [PubMed: 8781299]

24. Szymanska E, Bouwman J, Strassburg K, et al. Gender-dependent associations of metabolite profiles and body fat distribution in a healthy population with central obesity: towards metabolomics diagnostics. Omics J Integr Biol. 2012; 16:652–667.
25. Wells JCK. Ethnic variability in adiposity, thrifty phenotypes and cardiometabolic risk: addressing the full range of ethnicity, including those of mixed ethnicity. Obes Rev Off J Int Assoc Study Obes. 2012
26. Luo J, Margolis KL, Adami H-O, et al. Body size, weight cycling, and risk of renal cell carcinoma among postmenopausal women: the Women's Health Initiative (United States). Am J Epidemiol. 2007; 166:752–759. [PubMed: 17615089]
27. Prineas RJ, Folsom AR, Zhang ZM, et al. Nutrition and Other Risk Factors for Renal Cell Carcinoma in Postmenopausal Women. Epidemiology. 1997; 8:31–36. [PubMed: 9116091]
28. Nicodemus KK, Sweeney C, Folsom AR. Evaluation of dietary, medical and lifestyle risk factors for incident kidney cancer in postmenopausal women. Int J Cancer. 2004; 108:115–121. [PubMed: 14618625]
29. Wu X, Scelo G, Purdue MP, et al. A genome-wide association study identifies a novel susceptibility locus for renal cell carcinoma on 12p11.23. Hum Mol Genet. 2012; 21:456–462. [PubMed: 22010048]
30. Pischon T, Lahmann PH, Boeing H, et al. Body size and risk of renal cell carcinoma in the European Prospective Investigation into Cancer and Nutrition (EPIC). Int J Cancer. 2006; 118:728–738. [PubMed: 16094628]
31. Fryzek JP, Poulsen AH, Johnsen SP, et al. A cohort study of antihypertensive treatments and risk of renal cell cancer. Br J Cancer. 2005; 92:1302–1306. [PubMed: 15812478]

Table 1

Comparison of Demographic, Life Style and Other Characteristics Among Renal Cell Cancer Cases and Matched Controls, Shanghai Women's (1996–2000) and Men's (2001–2006) Health Studies [a]

Variable	Total					Women					Men				
	Cases (n=271)		Controls (n=2693)		*P* Value	Cases (n=124)		Controls (n=1232)		*P* Value	Cases (n=147)		Controls (n=1461)		*P* Value
	%	Mean (SD)	%	Mean (SD)		%	Mean (SD)	%	Mean (SD)		%	Mean (SD)	%	Mean (SD)	
Age at enrollment (years)		58.2 (9.6)		58.3 (9.5)	*a*		57.1 (9.1)		57.1 (9.0)	*a*		59.2 (9.9)		59.2 (9.7)	*a*
Gender (female)	45.8		45.8		*a*	100.0		100.0		*a*	0.0		0.0		*a*
Education															
Elementary and below	17.0		20.3			28.2		33.7			7.5		8.9		
Middle and high school	59.3		58.7		0.35	54.8		51.6		0.45	63.0		64.6		0.67
High professional, college and above	23.7		21.1			16.9		14.7			29.5		26.4		
Smoking history															
Never smoker	67.5		64.7			96.8		95.8			42.9		38.4		
Former smoker	8.9		7.1		0.20	0.8		0.7			15.7		12.5		
Current smoker	23.6		28.2			2.4		3.5			41.5		49.1		
Alcohol drinking (yes)	11.1		16.0		0.03	2.4		3.0		0.93	18.4		26.9		0.02
Weight (kg)		66.5 (10.5)		64.3 (10.2)	<0.001		61.1 (9.0)		60.0 (8.8)	0.19		71.1 (9.4)		68.0 (9.9)	<0.001
Height (cm)		164.2 (8.4)		163.3 (8.4)	0.11		157.6 (5.6)		156.6 (5.4)	0.05		169.7 (5.9)		169.0 (5.9)	0.16
Body mass index (kg/m^2)		24.6 (3.2)		24.1 (3.2)	0.007		24.6 (3.6)		24.5 (3.4)	0.63		24.7 (2.8)		23.8 (3.0)	<0.001
<18.5	2.6		3.1			4.8		2.1			0.7		3.8		
18.5–24.9	55.4		60.3		0.36	50.8		57.1		0.16	59.2		63.0		0.10
25.0–29.9	37.6		32.5			38.7		34.3			36.7		31.0		
≥30.0	4.4		4.2			5.7		6.5			3.4		2.2		
Waist (cm)		84.4 (9.2)		82.7 (9.2)	0.003		80.4 (8.7)		79.6 (8.7)	0.30		87.8 (8.2)		85.3 (8.9)	0.001
Hip (cm)		97.3 (6.8)		95.7 (7.1)	<0.001		98.3 (7.6)		96.8 (7.7)	0.04		96.5 (6.0)		94.8 (6.4)	0.002
Waist-hip ratio[b]		0.9 (0.1)		0.9 (0.1)	0.40		0.8 (0.1)		0.8 (0.1)	0.42		0.9 (0.1)		0.9 (0.1)	0.04
<Q1	20.3		19.2			23.4		19.0			17.7		19.4		
Q1-<Median	24.7		27.7		0.44	26.6		27.8		0.70	23.1		27.5		0.11

Variable	Total					Women					Men				
	Cases (n=271)		Controls (n=2693)		*P* Value	Cases (n=124)		Controls (n=1232)		*P* Value	Cases (n=147)		Controls (n=1461)		*P* Value
	%	Mean (SD)	%	Mean (SD)		%	Mean (SD)	%	Mean (SD)		%	Mean (SD)	%	Mean (SD)	
Median-<Q3	24.4		26.4			22.6		23.4			25.9		29.0		
≥Q3	30.6		26.7			27.4		29.8			33.3		24.2		
Self-reported hypertension (yes)	41.7		31.9		0.001	37.1		28.6		0.05	45.6		34.7		0.01
Hypertension duration (years)		14.4 (10.4)		13.7 (11.3)	0.54		14.0 (10.5)		14.1 (11.6)	0.96		14.6 (10.4)		13.3 (11.1)	0.39
Non-hypertension	58.3		68.1			62.9		71.4			54.4		65.3		
<10 years	18.1		15.0		0.003	15.3		13.2		0.11	20.4		16.6		0.03
≥10 years	23.6		16.9			21.8		15.3			25.2		18.1		
Family history of cancer (yes)[c]	34.0		27.1		0.02	33.1		25.2		0.06	34.7		28.7		0.13
Menopausal status (post)	*a*		*a*		*a*	70.2		70.0		*a*	*a*		*a*		*a*

Abbreviation: SD, standard deviation.

[a] Cases and controls were matched on age, gender, recruitment date and menopausal status (for women only).

[b] For women, Q1=0.78, Median=0.82, Q3=0.85; for men, Q1=0.86, Median=0.90, Q3=0.935.

[c] Family history of cancer includes all kinds of cancer occurred in all immediate family members.

Table 2

Multivariable Odds Ratios and 95% Confidence Intervals for the Association of Hypertension and Anthropometric Variables and Renal Cell Carcinoma, Shanghai Women's (1996–2000) and Men's (2001–2006) Health Studies [a]

	All			Women			Men		
	Number of Cases/Controls	OR	95% CI	Number of Cases/Controls	OR	95% CI	Number of Cases/Controls	OR	95% CI
Hypertension	113/859	1.4	1.1, 1.9	46/352	1.5	0.996, 2.3	67/507	1.3	0.9, 2.0
Hypertension duration									
non-hypertension	158/1834	1.0	Reference	78/880	1.0	Reference	80/954	1.0	Reference
<10 years	49/404	1.3	0.9, 1.9	19/163	1.4	0.8, 2.4	30/243	1.3	0.8, 2.1
≥10 years	64/455	1.5	1.1, 2.1	27/189	1.6	0.99, 2.7	37/264	1.4	0.9, 2.2
P for trend	0.01			0.04			0.14		
BMI (in kg/m^2)									
<18.5	7/82	1.0	0.4, 2.2	6/26	2.6	1.02, 6.8	1/56	0.2	0.03, 1.5
18.5–24.9	150/1624	1.0	Reference	63/704	1.0	Reference	87/920	1.0	Reference
25–29.9	102/875	1.2	0.9, 1.5	48/422	1.2	0.8, 1.9	54/453	1.1	0.7, 1.6
≥30	12/112	1.0	0.5, 2.0	7/80	0.9	0.4, 2.0	5/32	1.5	0.6, 4.1
P for trend	0.44			0.75			0.15		
<18.5	7/82	1.0	0.4, 2.3	6/26	2.5	0.95, 6.6	1/56	0.2	0.03, 1.6
18.5–22.9	83/932	1.0	Reference	37/404	1.0	Reference	46/528	1.0	Reference
23–24.9	67/691	1.0	0.7, 1.5	26/300	0.9	0.5, 1.5	41/391	1.2	0.7, 1.8
25–29.9	102/875	1.2	0.9, 1.6	48/422	1.2	0.7, 1.8	54/453	1.2	0.8, 1.8
≥30	12/112	1.1	0.5, 2.0	7/80	0.8	0.3, 2.0	5/32	1.6	0.6, 4.6
P for trend	0.39			0.74			0.13		
per 5 kg/ m^2 increase in BMI	271/2693	1.2	0.99, 1.5	124/1232	1.0	0.8, 1.3	147/1461	1.5	1.1, 2.0
WHR [b]									
<Q1	55/517	1.0	Reference	29/234	1.0	Reference	26/283	1.0	Reference
Q1-<Median	67/745	0.8	0.6, 1.2	33/343	0.8	0.5, 1.3	34/402	0.9	0.5, 1.5
Median-<Q3	66/711	0.8	0.6, 1.2	28/288	0.7	0.4, 1.3	38/423	0.9	0.5, 1.6
≥Q3	83/720	1.0	0.7, 1.5	34/367	0.7	0.4, 1.3	49/353	1.3	0.8, 2.3
P for trend	0.84			0.27			0.18		

	All			Women			Men		
	Number of Cases/Controls	OR	95% CI	Number of Cases/Controls	OR	95% CI	Number of Cases/Controls	OR	95% CI
per 1 SD increase in WHR	271/2693	1.0	0.9, 1.2	124/1232	0.9	0.7, 1.1	147/1461	1.1	0.97, 1.4
Education									
Elementary and below	46/546	1.0	Reference	35/415	1.0	Reference	11/131	1.0	Reference
Middle and high school	161/1580	1.3	0.8, 2.0	68/636	1.3	0.8, 2.3	93/944	1.1	0.6, 2.2
High professional, college and above	64/567	1.4	0.9, 2.4	21/181	1.4	0.7, 2.8	43/386	1.3	0.6, 2.7
Smoking history									
Never smoker	183/1741	1.0	Reference	120/1180	1.0	Reference	63/561	1.0	Reference
Former smoker	24/192	1.1	0.7, 1.9	1/9	0.9	0.1, 7.9	23/183	1.2	0.7, 1.9
Current smoker	64/760	0.8	0.6, 1.2	3/43	0.7	0.2, 2.2	61/717	0.9	0.6, 1.3
Current alcohol drinking	30/430	0.7	0.5, 1.05	3/37	0.8	0.2, 2.6	27/393	0.7	0.4, 1.05
Family history of cancer [c]	92/729	1.3	0.997, 1.7	41/310	1.5	0.99, 2.2	51/419	1.2	0.8, 1.7

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio; WHR, waist-hip ratio.

[a] Self-reported hypertension (or categorical hypertension duration) and continuous BMI (or categorical BMI), or continuous WHR (or categorical WHR) were included as main effects, adjusted for education, smoking history, current alcohol drinking status (yes/no), family history of cancer (yes/no). Each model includes one anthropometric variable only (either BMI or WHR). The ORs of hypertension, categorical hypertension duration and adjusted covariates were calculated with continuous BMI.

[b] For women, Q1=0.78, Median=0.82, Q3=0.85, SD=0.0535; for men, Q1=0.86, Median=0.90, Q3=0.935, SD=0.0558.

[c] Family history of cancer includes all kinds of cancer occurred in all immediate family members.