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Body Mass Index and Renal Cell Cancer: The Influence of Race and Sex

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Abstract

Background—Obesity is a risk factor for renal cell (or renal) cancer. The increasing prevalence of obesity may be contributing to the rising incidence of this cancer over the past several decades. The effects of early-age obesity and change in body mass index (BMI) on renal cancer have been studied less thoroughly, and the influence of race has never been formally investigated.

Methods—Using data gathered as part of a large case-control study of renal cancer (1,214 cases and 1,234 controls), we investigated associations with BMI at several time points, as well as with height. Adjusted odds ratios (ORs) and 95% confidence intervals (CIs) were computed using logistic regression modeling. Race- and sex-stratified analyses were conducted to evaluate subgroup differences.

Results—Obesity (BMI ≥ 30 kg/m²) early in adulthood (OR=1.6 [95% CI=1.1 to 2.4]) and 5 years before diagnosis (1.6 [1.1 to 2.2]) was associated with renal cancer. The association with early-adult obesity was stronger among whites than blacks (Test for interaction, $P=0.006$), while the association with obesity near diagnosis was marginally stronger in women than men (Test for interaction, $P=0.08$). The strongest association with renal cancer was observed for obese whites both in early adulthood and prior to interview (2.6 [1.5 to 4.4]); this association was not present among blacks. Estimates of the annual excess rate of renal cancer (per 100,000 persons) attributed to both overweight and obesity (BMI > 25 kg/m²) ranged from 9.9 among black men to 5.6 among white women.

Conclusion—Obesity, both early and later in life, is associated with an increased risk of renal cancer. The association with early obesity appears to be stronger among whites than blacks.

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Cancers of the kidney and renal pelvis account for 3 to 4 percent of all new cancers diagnosed in the United States, with an estimated 58,000 cases diagnosed in 2010.¹ Renal cell cancer is the predominant form, accounting for approximately 90% of kidney cancers diagnosed, mostly adenocarcinomas originating in the renal parenchyma.² The incidence of renal cell (or renal) cancer has increased over the past several decades in the U.S.^{2,3} and continues to rise, although the increasing incidence appears confined to patients diagnosed with early-stage disease.³

The epidemic increases in the prevalence of obesity in the U.S. may contribute to the observed trends in renal cancer incidence, as obesity is an established risk factor.⁴ The risk of renal cancer associated with body mass index (BMI) increases in a dose-response manner, with a 2-fold increase in risk observed among the obese (BMI ≥ 30 kg/m²) and a nearly 3-fold increase among the severely obese (BMI ≥ 35 kg/m²).⁵ It has been estimated that approximately 25% of renal cancer diagnosed in the U.S. is attributed to excess body weight.⁶ Body size in early adulthood, abdominal obesity and weight fluctuation have all been linked to renal cancer; however, these measures have been less thoroughly investigated and the totality of reported findings is inconclusive.^{5,7-9} Height has also been shown to be positively related to renal cancer in some studies^{5,10-13} but not all.^{14,15} Other established risk factors for renal cancer include hypertension and cigarette smoking.²

According to Surveillance, Epidemiology, and End Results (SEER) data, both the incidence of renal cancer and subsequent mortality are approximately two times higher among men than women.¹ The racial differences in incidence and mortality are less pronounced, with Asians experiencing the lowest rates of renal cancer. The incidence is higher among blacks than whites with comparable mortality rates.⁷ The prevalence of known risk factors, most notably obesity and hypertension, vary by both race and sex and may have a differential influence on risk of renal cancer in these groups. The Kidney Cancer Study, sponsored by the National Cancer Institute (NCI), is one of the largest studies of epidemiologic risk factors for renal cancer in the United States. The aims of the current analysis are to investigate the race- and sex-specific associations of renal cancer with BMI (both in early adulthood and closer to time of diagnosis) change in BMI, and adult height.

Methods

Study Population

The Kidney Cancer Study is a population-based case-control study conducted in the midwest U.S. with cases and controls recruited in Detroit, Michigan (Wayne, Macomb and Oakland Counties) and Chicago, Illinois (Cook County). Patients aged 20 to 79 years, newly diagnosed with histologically confirmed adenocarcinoma of the renal parenchyma (ICD-O3; topography C64.9) were eligible for participation. In Detroit, renal cancer cases were identified through the Metropolitan Detroit Cancer Surveillance System, one of NCI's Surveillance, Epidemiology and End Results (SEER) registries, between 1 February 2002 and 31 January 2007. In Chicago, renal cancer cases diagnosed in 2003 were identified through review of the pathology reports at Cook County and nearby hospitals.

Controls were frequency-matched to cases on age at diagnosis (within 5-year intervals), sex and race. Eligible controls between the ages of 20 and 64 years were identified through the Department of Motor Vehicles (DMV) and controls aged 65 to 79 through Medicare eligibility files. A sampling strategy was devised to increase black participation. We attempted to recruit all eligible black renal cancer patients and a sample of eligible white renal cancer patients. The goal was to recruit 2 controls per case (2:1 ratio) among blacks and one control per case (1:1 ratio) among whites. Because prior information on race for controls under age 65 was unavailable, we oversampled from DMV addresses from census

block groups identified as having a high proportion of black residents based on U.S. 2000 Census data.¹⁶

Of the 1,918 eligible cases, 171 died before contact or interview, 92 could not be located, and 21 moved from the area. In addition, physicians refused permission to contact 63 cases. Of the 1,571 cases contacted for enrollment, 221 declined participation, and 133 could not be interviewed due to poor health, impairment or non-response after multiple attempts to contact; the remaining 1,217 cases (78% of those attempted) were enrolled.

Of the 2,718 controls identified who were presumed eligible to participate, 41 died before contact or interview, 345 could not be located, and 63 moved from the area. Of the 2269 contacted for enrollment, 677 declined and 357 could not be interviewed because of poor health, impairment or non-response after multiple attempts to contact; the remaining 1,235 (54% of those attempted) were enrolled.

Response rates among cases were similar between men (77%) and women (78%) and similar among whites (78%) and blacks (79%). Response rates among controls were higher among women (57%) than men (53%) and higher among blacks (56%) than whites (53%). The study was approved by the human subjects review board at all participating institutions, and written informed consent was obtained from all participants before interview. Each participant received some financial compensation for their time.

Anthropometric Survey Questions

Trained interviewers conducted computer-assisted personal interviews in participants' homes to elicit information on demographic variables, height and weight, medical history, smoking history, family history of cancer, and other potential risk factors. Participants were asked to provide blood and mouth-rinse samples, and cases were asked for consent to access medical records and tumor-tissue samples.

Participants were asked to provide their height at the time of interview and at age 21, as well as their weight at the time of interview, 5 years prior to interview, and at age 21. They also reported their maximum and minimum weights (excluding periods of pregnancy and the year after pregnancy for women) between the age of 21 and 2 years prior to interview, and the ages at which they were at their maximum and minimum weights. Of the 1,217 cases and 1,235 controls enrolled into the study, 3 cases and 1 control were excluded due to missing data on either reported height or weight, leaving 1,214 cases and 1,234 controls available for analysis.

Statistical Methods

All statistical analyses were performed using SAS statistical software, version 9.2 (SAS Institute Inc., Cary, NC). We calculated the frequencies and proportion of cases and controls according to important characteristics. Early-adult BMI (in kg/m²) was calculated based on self-reported weight and height at age 21. BMI 5 years prior to interview and maximum BMI were calculated based on self-reported height at time of interview, weight 5 years prior to interview, and maximum weight, respectively.

We developed sample weights to reduce the potential for bias arising from differential sampling rates for controls and cases, survey non-response, and deficiencies in coverage of the population at risk in the DMV and Medicare files. Sample weights for controls also include a post-stratification adjustment, so that the weighted distribution of controls across the matching variables matches exactly the weighted distribution of cases. In addition to being consistent with the objectives of the frequency matching, the post-stratification

adjustment reduces the variability of the weights.¹⁷ Full detail of the evaluation for response bias and the development of the sample weights has been described previously.¹⁶

We estimated the risk of developing renal cancer by deriving odds ratios (ORs) and 95% confidence intervals (CIs) from unconditional logistic regression models, using the post-stratification weights and controlling for potential confounders. Jackknife replicate weights were created to estimate standard errors for the calculation of 95% confidence intervals and computation of test statistics and p-values in the weighted risk analyses.¹⁸ All analyses were conducted using the sample weights.

Early-adult BMI was categorized as normal ($\text{BMI} < 25.0 \text{ kg/m}^2$), overweight ($\text{BMI} = 25.0\text{--}29.9 \text{ kg/m}^2$), or obese ($\text{BMI} \geq 30 \text{ kg/m}^2$). BMI prior to interview and maximum BMI were categorized similarly, with one additional category to estimate the odds of renal cancer among more severely obese participants ($\text{BMI} \geq 35 \text{ kg/m}^2$). In all logistic models, normal BMI was used as the referent for all three BMI measures. The final models simultaneously adjusted for BMI measures, age, education, smoking history, hypertension, study center and family history of kidney cancer. The characterization of confounding variables included in the final models is presented in Table 1. Height was treated as a continuous variable in the logistic models, producing an odds ratio per one inch increase, as well as dichotomized at the median among controls (which varied depending upon the chosen subgroup for analysis). We also examined change in BMI between age 21 and 5 years prior to interview. To accomplish this, we created four indicator variables, with the referent group composed of participants who were considered not to be obese ($< 30 \text{ kg/m}^2$) at both points in time (low / low). We compared this group to participants who were (1) not obese at 21 years of age, but obese closer to time of interview (low / high), (2) obese at 21 years, but not obese near interview (high / low), and (3) obese at both points (high / high). Stratified analysis was also performed based on race and sex for all models.

Lastly, we calculated the population attributable risk (PAR percent) to estimate the proportion of cases attributed to both overweight and obesity ($\text{BMI} > 25.0 \text{ kg/m}^2$) for adults aged 45–79 years by race and sex. We computed PAR% and associated 95% CIs using a variation of the Bruzzi method,^{19;20} with the same covariates and sample weights as in the principal analyses. The excess incidence attributed to overweight and obesity was calculated using PAR% and race- and sex-specific renal cancer incidence rates derived from the Detroit SEER database for cases diagnosed 2002–2006, using U.S. 2000 Census data for population denominators.

Results

The majority of cases and controls participating in the study were white (70% of cases and 58% of controls) and male (59% of cases and 56% of controls) (Table 1). The mean age at diagnosis among cases was 59 years (standard deviation = 11.3 years). Cases were more likely than controls to report a history of hypertension and a positive family history of kidney cancer, and less likely to report any education beyond high school.

The associations between measures of body size and renal cancer are summarized for all subjects combined in Table 2, with the results stratified by race and sex in Table 3. Early-adult obesity ($\geq 30 \text{ kg/m}^2$) was associated with a 60% increase in odds of renal cancer after adjustment for covariates ($\text{OR} = 1.6 [1.1 \text{ to } 2.4]$). Modeled continuously, the odds of renal cancer were approximately 4% greater with each additional unit in kg/m^2 for BMI at age 21. These associations were similar among men and women (Test for interaction, $P = 0.68$). However, there were differences by race (Test for interaction, $P = 0.006$); a positive association between early-adult BMI and renal cancer was observed among whites (1.07

[1.03 to 1.12] per unit increase in BMI), while no association was observed among blacks (0.99 [0.96 to 1.03]).

Excess body mass 5 years prior to interview was also associated with an increase in the odds of renal cancer, with cases 50% more likely than controls to be classified as obese (1.5 [1.2 to 2.1]). Again, there was a 2% increase in the odds of renal cancer for each unit increase (in kg/m^2) in BMI 5 years prior to interview, with BMI modeled continuously (Table 2). The observed associations with BMI measured close to the time of interview were not materially different between whites and blacks (Test for interaction, $P=0.76$) and marginally different between men and women (Test for interaction, $P=0.08$). Severely obese (BMI $\geq 35 \text{ kg/m}^2$) white and black women both had a two-fold increase in the odds of renal cancer (Table 3). Associations between maximum BMI and renal cancer (both overall and stratified) were similar to those observed for BMI 5 years prior to interview. Adult height was unrelated to renal cancer overall (1.00 [0.98 to 1.02] per one-inch increase in height) and within race- and sex-specific subgroups.

Compared with subjects who were never classified as obese, those who were obese both early in adulthood and near the time of interview were more likely to be cases (2.0 [1.3 to 3.0]); this association was similar among men and women but confined to whites (Table 4). Subjects who were not considered obese earlier in life but were obese near the time of interview were 50% more likely to be diagnosed with renal cancer (1.5 [1.2 to 1.9]), with no differences by race or sex. Lastly, BMI loss during the same time frame was unrelated to renal cancer overall, with the small number of subjects precluding an estimation of race- and sex-specific associations. Results were similar when the BMI cutpoint for early adulthood body size was dropped to 25 kg/m^2 (thus including both the overweight and obese categories) (data not shown).

The annual incidence of renal cancer among black men aged 45 to 79 years is 49.1 per 100,000, which is approximately 15% higher than white men (42.9 per 100,000) (Table 5). Among black women aged 45–79, the annual incidence of renal cancer is 24.6 per 100,000, similar to that of white women (23.4 per 100,000). Point estimates for PAR% for BMI $\geq 25 \text{ kg/m}^2$ among the four sex-race subgroups ranged from 16% in white men to 25% in black women. However, because of the higher incidence of renal cancer among men overall, the annual incidence attributable to overweight and obesity was highest among black men (9.9 per 100,000) and lowest among white women (5.6 per 100,000).

Discussion

The results from this large case-control study indicate that excess body weight, both early and later in life, is associated with an increased risk of renal cancer. Our results suggest that early-adult obesity is associated with renal cancer among whites but not blacks. Excess weight closer to the time of diagnosis is associated with renal cancer among both blacks and whites, and the association is generally higher among women than men. Whites who were consistently obese throughout life were the most likely to be diagnosed with renal cancer; this association was not observed among blacks.

Our population-based design allows us, with certain assumptions, to calculate the possible reduction in rate of renal cancer accomplished by reducing the prevalence of overweight and obesity in the population. Our calculations of incidence rate attributable to BMI, just like those for PAR%, require (1) that the observed odds ratios are measures of the causal effects of BMI; (2) that the renal cancer rate upon reducing BMI below 25 kg/m^2 will be the same as in those whose BMI has remained below 25 kg/m^2 ; and (3) despite the fact that 3 of 4 race-sex subgroup PAR% estimates were not considered statistically significant (at an

$\alpha=0.05$), our point estimate of PAR% represents the best estimate of the proportion of incidence attributed to BMI > 25 kg/m². Under these assumptions, the rate of renal cancer caused by overweight and obesity and preventable by lowering BMI to <25 kg/m² would be 9.9, 6.8, 6.2 and 5.6 per 100,000 per year in black men, white men, black women, and white women, respectively. Our estimate of the absolute difference in incidence between blacks and whites before and after removing the effect of overweight and obesity suggests that BMI explains a substantial portion (50%) of the racial disparity in both men and women. The racial gap in incidence (per 100,000 per year) narrowed from 6.2 to 3.1 among men and from 1.2 to 0.6 among women.

Our investigation is the first to evaluate the relative importance of increased body mass by race and sex, and our findings were consistent with most studies conducted almost exclusively among whites linking obesity to renal cancer.^{5;8;9;13;15;21–23} Some studies,^{9;21;15;24;25} but not all,^{5;23;26;27} suggest that this relationship may be more important for women than men. Studies examining the influence of weight change on risk of renal cancer^{5;8;24;25} also suggest an elevation in risk with adult weight gain after adjustment for baseline BMI. An investigation within a large prospective cohort reported risk of renal cancer to be positively associated with weight gain; however, the increases in risk appeared to be confined to men and women with low BMI (<22.5 kg/m²) at age 18 years.⁵

Most investigations report an independent effect of obesity and hypertension on renal cancer risk,^{21;22} although one study reported evidence of interaction between these two risk factors.²³ We did not find evidence for interaction in this investigation ($P=0.62$). Our race-specific evaluation for effect modification of hypertension on obesity and renal cancer was approximately additive among both blacks and whites. As has been previously reported,¹⁶ hypertension appeared to be more strongly associated with renal cancer among blacks than among whites (eTable,<http://links.lww.com/EDE/A605>), signaling that the renal cancer risk-factor profiles may be different in blacks and whites.

Despite our observation that height was unrelated to renal cancer, height has been shown to be positively associated with renal cancer in a number of studies.^{5;10–13} Height is driven by both genetic and environmental factors, and while the mechanism has not been fully elucidated, it has been postulated that nutrition, particularly early in life, plays a role in carcinogenic effects related to height.¹⁰ Hormones, including insulin-like growth factors (IGFs), are important in growth and may influence cancer risk by increasing mutagenic potential with enhanced cell division.²⁸

Our finding of a racial difference in the influence of early-adult obesity and renal cancer deserves further exploration. There are clear racial differences in body composition at the same level of BMI. Black men and women have been shown to have smaller waist circumferences and waist-to-hip ratios, and decreased visceral fat; these differences are accompanied by lower fasting insulin levels, and are observed in both young and older adults.^{29–31}

Obesity has been hypothesized to influence renal carcinogenesis through mechanisms tied to insulin resistance and resulting hyperinsulinemia, and altered concentrations of adipocytokines, producing a chronic inflammatory response.^{32;33} Others have posited that lipid peroxidation is an important mediator of renal carcinogenesis suggesting that the byproducts of lipid peroxidation can form DNA adducts leading to mutation.³⁴ This pathway may explain the enhanced renal cancer risk among obese persons, as well as those with a history of hypertension.³⁴

A limitation of the current study is that the information gathered on weight and height was based entirely on self-report. The accuracy of self-reported weight and height has been well

studied and there are a number of factors shown to influence the level of bias in recall of these measures. Men and women typically overestimate their height. However, while men tend to overestimate their weight, women tend to underestimate their weight, producing an underestimation in corresponding BMI for women.³⁵ Other factors that reduce the accuracy of self-reported measures of body composition include older age and excess weight.^{36;37} However, studies do not suggest any difference in reporting bias between blacks and whites.^{35;36} Assuming that the error in self-reported measures does not systematically differ between cases and controls, the net effect of these errors would produce a bias toward the null. And while BMI is considered a reasonable measure of obesity, it is not an optimal measure because of its inability to distinguish lean body mass from adipose tissue. Furthermore, our finding that BMI loss between early and later life was positively associated with renal cancer might be explained by the fact that for cases, weight loss closer to the time of interview may be influenced by the disease process itself. This possibility exists despite the strategy of selecting newly diagnosed cases and requesting information on weight 5 years prior to interview and presumably prior to cancer diagnosis. Despite our observation of an independent association of obesity in both early and later life with renal cancer (i.e. simultaneous adjustment for BMI at both time periods), one must be cautious in attributing the effect of obesity to any specific point in time over the lifecourse.

Another limitation of this study was the low response rate among controls, which is typical of recent population-based case-control studies. Our use of sample weights helped to reduce the potential for bias arising from non-response, as the weights account for differential non-response across subgroups defined by factors such as age, race (or relative concentration of blacks in the residential area), sex, and county of residence, for which data were available for both respondents and non-respondents. However, we cannot entirely rule out the possibility that selection bias influenced our results.

This study has several strengths, perhaps the most important of which was our ability to recruit a large number of blacks, allowing us to evaluate racial differences in the relationship between body size and renal cancer. All cases were histopathologically confirmed. Detailed information was collected on weight and height at multiple points in time over the lifecourse allowing for the examination of both early and later life BMI on risk, as well as the effect of change in BMI. Likewise, information on hypertension history, smoking behavior and family history of renal cancer allowed for their adjustment as potential confounders. An additional strength of this study is the presentation of estimates of the excess rate of renal cancer attributable to overweight and obesity – and therefore potentially preventable. These estimates address the public health impact of overweight and obesity on renal cancer risk across several demographic groups more directly than do estimates of relative risk.

Overall, our findings suggest that obesity is associated with renal cancer in both races, but were generally stronger among whites than blacks, particularly in early adulthood. Additional investigation is needed to assess whether observed differences across population subgroups are replicable. To elucidate causal pathways, future investigations should focus on differences by race and sex in molecular markers tied to the metabolic consequences of obesity as they relate to renal cancer.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Characteristics of cases and controls participating in the Kidney Cancer Study

	All			White				African American			
				Men		Women		Men		Women	
	Cases (n=1214) %	Controls (n=1234) ^a %		Cases (n=439) %	Controls (n=439) %	Cases (n=359) %	Controls (n=272) %	Cases (n=225) %	Controls (n=250) %	Cases (n=135) %	Controls (n=273) %
Age											
20-44	11	11		10	10	11	11	11	12	12	12
45-54	22	22		21	21	17	17	27	26	25	26
55-64	29	29		30	30	28	28	33	32	27	27
65-74	27	27		29	29	27	27	22	22	27	27
75+	11	11		10	10	17	17	7	8	9	8
Education											
Less than HS	17	12		14	11	11	8	28	22	28	15
HS/diploma	35	32		30	26	47	37	27	36	32	30
Some college	26	27		24	22	25	31	31	27	30	40
4 year degree	22	29		32	41	17	24	14	15	10	15
Hypertension											
Never	39	58		42	58	45	64	30	50	24	51
Ever	59	41		55	41	54	35	68	48	76	49
Unknown	2	1		3	1	1	1	2	2	0	0
Study Site											
Chicago	17	17		16	15	13	17	23	21	18	21
Detroit	83	83		84	85	87	83	77	79	82	79
Smoking Status^b											
Never	35	38		30	36	46	46	27	30	44	41
Occasional	5	4		5	4	3	3	9	5	2	6
Former	45	41		53	45	37	38	41	39	32	34
Current	15	17		12	16	14	13	23	26	22	19
Family History of Renal Cancer											

	All		White				African American			
			Men		Women		Men		Women	
	Cases (n=1214) %	Controls (n=1234) ^a %	Cases (n=495) %	Controls (n=439) %	Cases (n=359) %	Controls (n=272) %	Cases (n=225) %	Controls (n=250) %	Cases (n=135) %	Controls (n=273) %
No	96	98	96	98	96	97	95	99	95	98
Yes	4	2	4	2	4	3	5	1	5	2

^a Percentages are weighted

^b Never smokers=smoked less than 100 cigarettes in their lifetime. Occasional smokers=smoked more than 100 cigarettes in their lifetime but had never smoked at least one cigarette a day for six months or longer. Former smokers=smoked more than 100 cigarettes, and smoked at least one cigarette a day for at least six months and quit at least 2 years before referent date (case=date of diagnosis and control=selection). Current smokers=smoked more than 100 cigarettes, and smoked at least one cigarette a day for at least six months and either never quit or quit less than 2 years before referent date.

Table 2

Associations between early to adult, contemporary and maximum adult body mass index (BMI), height and renal cell cancer.

	Cases No. (%) ^a	Controls No. (%) ^a	OR (95% CI) ^b	Test for trend	Test for interaction	
					By sex	By race
BMI (in kg/m ²)						
At age 21						
< 25.0 ^c	757 (65)	890 (77)	1.0			
25.0 to 29.9	317 (27)	230 (19)	1.4 (1.1 to 1.7)			
>= 30.0	103 (8)	61 (4)	1.6 (1.1 to 2.4)			
Per 1 kg/m ² increase			1.04 (1.02 to 1.07)	P=0.0005	P=0.68	P=0.006
5 years prior to interview						
< 25.0 ^c	240 (20)	366 (29)	1.0			
25.0 to 29.9	436 (37)	493 (42)	1.2 (0.9 to 1.5)			
30.0 to 34.9	298 (25)	221 (18)	1.5 (1.2 to 2.1)			
>= 35.0	230 (18)	147 (11)	1.6 (1.1 to 2.2)			
Per 1 kg/m ² increase			1.02 (1.01 to 1.04)	P=0.0013	P=0.08	P=0.76
Maximum						
< 25.0 ^c	133 (11)	201 (17)	1.0			
25.0 to 29.9	355 (30)	460 (41)	1.0 (0.8 to 1.3)			
30.0 to 34.9	356 (31)	310 (25)	1.5 (1.1 to 2.0)			
>= 35.0	356 (28)	236 (18)	1.6 (1.2 to 2.1)			
Per 1 kg/m ² increase			1.02 (1.01 to 1.04)	P=0.0001	P=0.06	P=0.21
Height (in inches)						
< Median ^c	553 (44)	599 (45)	1.0			
Median	661 (56)	635 (55)	1.0 (0.9 to 1.2)			
Per 1 inch increase			1.00 (0.98 to 1.02)	P=0.83	P=0.58	P=0.60

^a Percentages may not equal zero due to rounding

^b Adjusted for age, education, hypertension status (yes/no/unknown), family history of renal cancer (yes/no), smoking history (never/occasional/former/current), study center (Detroit/Chicago). Cases and controls were matched on race and sex.

^c Reference category. Analyses exclude those with missing data

Models mutually adjust for BMI at 21 years and BMI 5 years prior to interview with the exception of maximum BMI and height which are adjusted for BMI at 21 years.

Table 3

Race- and sex-stratified associations of BMI and height with renal cell cancer.

	White			Black		
	All OR (95% CI) ^a	Men OR (95% CI) ^b	Women OR (95% CI) ^b	All OR (95% CI) ^a	Men OR (95% CI) ^b	Women OR (95% CI) ^b
BMI in (kg/m²)						
<i>At age 21</i>						
< 25.0	1.0	1.0	1.0	1.0	1.0	1.0
25.0 – 29.9	1.4 (1.1 to 1.9)	1.4 (1.0 to 1.9)	1.8 (1.0 to 3.2)	1.4 (0.9 to 2.0)	1.4 (0.9 to 2.1)	1.3 (0.6 to 2.7)
>= 30.0	2.1 (1.2 to 3.6)	2.3 (1.2 to 4.4)	1.8 (0.7 to 4.4)	0.9 (0.5 to 1.5)	1.1 (0.5 to 2.7)	0.6 (0.3 to 1.6)
<i>Per 1 kg/m² increase</i>	1.07 (1.03 to 1.12)	1.09 (1.04 to 1.14)	1.06 (0.99 to 1.12)	0.99 (0.96 to 1.03)	1.02 (0.96 to 1.09)	0.97 (0.92 to 1.01)
<i>5 years prior to interview</i>						
< 25.0	1.0	1.0	1.0	1.0	1.0	1.0
25.0 – 29.9	1.2 (0.9 to 1.6)	1.1 (0.8 to 1.7)	1.2 (0.8 to 1.8)	1.3 (0.9 to 1.8)	1.4 (0.8 to 2.4)	1.0 (0.5 to 1.9)
30.0 – 34.9	1.6 (1.1 to 2.2)	1.4 (0.9 to 2.1)	1.7 (1.0 to 2.8)	1.4 (0.9 to 2.3)	1.6 (0.8 to 3.2)	1.1 (0.6 to 2.0)
>= 35.0	1.6 (1.0 to 2.4)	1.2 (0.7 to 2.2)	1.9 (1.0 to 3.6)	1.5 (0.9 to 2.7)	1.1 (0.5 to 2.6)	2.0 (0.9 to 4.1)
<i>Per 1 kg/m² increase</i>	1.02 (1.00 to 1.05)	1.00 (0.98 to 1.03)	1.04 (1.01 to 1.07)	1.03 (1.00 to 1.05)	1.01 (0.96 to 1.06)	1.04 (1.01 to 1.07)
<i>Maximum</i>						
<25.0	1.0	1.0	1.0	1.0	1.0	1.0
25.0 – 29.9	1.2 (0.9 to 1.6)	0.9 (0.5 to 1.3)	1.4 (1.0 to 2.2)	0.7 (0.5 to 1.2)	0.7 (0.4 to 1.4)	0.8 (0.4 to 1.9)
30.0 – 34.9	1.8 (1.2 to 2.5)	1.3 (0.8 to 2.1)	2.0 (1.1 to 3.4)	1.0 (0.6 to 1.6)	1.0 (0.5 to 1.9)	0.9 (0.4 to 2.1)
>= 35.0	1.7 (1.2 to 2.5)	1.1 (0.7 to 1.9)	2.4 (1.4 to 4.1)	1.1 (0.6 to 2.0)	0.9 (0.4 to 2.5)	1.3 (0.6 to 2.7)
<i>Per 1 kg/m² increase</i>	1.03 (1.00 to 1.05)	1.01 (0.98 to 1.04)	1.04 (1.01 to 1.07)	1.01 (0.99 to 1.04)	0.99 (0.93 to 1.04)	1.03 (1.00 to 1.06)
Height (in inches)						
< Median ^c	1.0	1.0	1.0	1.0	1.0	1.0
Median	1.3 (0.9 to 1.7)	1.26 (0.95 to 1.68)	1.1 (0.8 to 1.5)	0.9 (0.6 to 1.4)	1.1 (0.7 to 1.8)	0.8 (0.5 to 1.4)
<i>Per 1 inch increase</i>	1.02 (0.98 to 1.06)	1.03 (0.98 to 1.07)	1.01 (0.96 to 1.07)	1.0 (0.9 to 1.1)	1.0 (0.9 to 1.1)	1.0 (0.9 to 1.1)

Analyses exclude those with missing data. Models mutually adjust for BMI at 21 years and BMI 5 years prior to interview, with the exception of maximum BMI and height at interview which are adjusted for BMI at 21 years.

^a Adjusted for age, sex, education, hypertension status (yes/no/unknown), family history of renal cancer (yes/no), smoking history (never/occasional/former/current), study center (Detroit/Chicago).

^b Adjusted for age, education, hypertension status (yes/no/unknown), family history of renal cancer (yes/no), smoking history (never/occasional/former/current), study center (Detroit/Chicago).

c The median was 68 inches for the total sample and for whites; 67 inches for blacks; 70 inches for both white men and black men; 64 inches for white and black women.

Table 4

Association of change in obesity status^a between age 21 and age 5 years before interview with renal cell cancer by race and sex.

Study Population	Change in Obesity Status ^a					
	Low-Low			High-High		
	Cases No. (%)	Controls No. (%)	OR ^b	Cases No. (%)	Controls No. (%)	OR (95% CI)
<i>All patients^c</i>	643 (56)	810 (70)	1.0	304 (26)	9 (1)	2.3 (0.8 to 6.6)
<i>Whites^d</i>	461 (56)	487 (71)	1.0	176 (26)	4 (1)	3.0 (0.7 to 12.6)
<i>Men^c</i>	272 (56)	296 (69)	1.0	168 (35)	7 (2)	2.2 (0.4 to 11.1)
<i>Women^c</i>	189 (55)	191 (75)	1.0	130 (37)	0 (0)	Undefined
<i>Blacks^d</i>	182 (55)	323 (67)	1.0	128 (36)	5 (5)	1.1 (0.2 to 6.5)
<i>Men^c</i>	125 (60)	167 (70)	1.0	70 (32)	4 (1)	1.1 (0.1 to 8.7)
<i>Women^c</i>	57 (47)	156 (62)	1.0	58 (44)	1 (<1)	1.1 (<0.0 to >99)

Analyses exclude those with missing data.

^aLow =BMI<30kg/m² and High =BMI ≥30 kg/m²

^bReference category.

^cAdjusted for age, education, hypertension status (yes / no / unknown), family history of renal cancer (yes/no), smoking history (never/occasional/former/current), study center (Detroit/Chicago).

^dAdjusted for age, sex, education, hypertension status (yes / no/ unknown), family history of renal cancer (yes/no), smoking history (never/occasional/former/current), study center (Detroit/Chicago).

Table 5

The proportion of renal cell cancer incidence attributable to excess weight (both overweight and obesity [BMI ≥ 25 kg/m²]) by race and sex, U.S. adults aged 45 to 79 years.

Renal Cell Cancer Incidence Ratio ^b (per 100,000 persons per year)					
Sex and Race	Population Attributable Risk % for excess weight ^a	(95% CI)	IR in	IR attributed to excess	IR in the absence of
			SEER ^b	weight (PAR) ^c	excess weight ^d
Men					
Black	20.1	(−13.2 to 53.3)	49.1	9.9	39.2
White	15.9	(−12.9 to 44.7)	42.9	6.8	36.1
Difference ^e			6.2		3.1
Women					
Black	25.1	(−9.2 to 59.5)	24.6	6.2	18.4
White	24.0	(1.8 to 46.1)	23.4	5.6	17.8
Difference ^e			1.2		0.6

^aModels adjusted for age, education, smoking history, hypertension, family history of kidney cancer, BMI at 21 years (weighted).

^bMicroscopically confirmed cases of adenocarcinoma of the kidney (renal parenchyma) or renal pelvis at ages 45–79, Detroit SEER, 2002–2006.

^cIR attributed to excess weight based upon point estimate of PAR%*SEER IR by race and sex.

^dRenal cancer incidence rate in the absence of excess weight = [SEER IR*(1-PAR%)] by race and sex.

^eBlack-white rate difference = The excess RCC incidence in blacks compared to whites (per 100,000 per year) by sex.

PAR indicates population attributable risk; IR, incidence rate.