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# Overweight and obesity in adults and risk of renal cell carcinoma in Canada

**Summary**

**Objectives:** To assess the role of overweight and obesity in renal cell carcinoma (RCC) risk in Canada.

**Methods:** Mailed questionnaires were used to obtain data on 1279 (691 male and 588 female) newly diagnosed, histologically confirmed RCC cases and 5370 population controls, between 1994 and 1997, in eight Canadian provinces. Data were collected on socio-economic status, height, weight, smoking habits, alcohol use, diet, and residential and occupational histories. Weight was expressed as body mass index (BMI). Odds ratios (ORs) and 95% confidence intervals (CIs) were derived using unconditional logistic regression.

**Results:** The study found an increased risk of RCC associated with overweight and obesity among both male and female adults; the test for trend was statistically significant ($p < 0.0001$ for both sexes). Compared with normal BMI, the adjusted ORs for obese class III (BMI $\geq 40.00$) were 3.7 (95% CI = 1.5–9.4) and 3.8 (95% CI = 2.3–6.4) among males and females, respectively.

**Conclusions:** These findings indicate that overweight and obesity play an important role in the etiology of renal cell among both males and females.

**Keywords:** **Renal cell carcinoma – Body mass index – Odds ratio – Logistic regression.**

Evidence is now emerging to suggest that the prevalence of overweight and obesity are increasing worldwide at an alarming rate; many chronic diseases are associated with obesity (WHO 2000). For instance, the risk of death from cardiovascular disease, cancer, and other diseases increased across the range of moderate and severe overweight for both men and women in a large American cohort study (Calle et al. 1999). The increasing prevalence of obesity could thus explain part of the increased incidence of renal cell carcinoma (RCC).

RCC, although still relatively uncommon, has been increasing in incidence in Canada and other countries around the world (Liu et al. 1997; Tavani & La Vecchia 1997). RCC, which represents 80–85% of all tumors of the kidney, is responsible for 2% of all cancer deaths in developed countries. Although epidemiological studies have indicated that cigarette smoking, overweight or obesity and medical history of diseases are related to RCC, the aetiology of RCC is still not well understood (Tavani & La Vecchia 1997). A consistently increased risk of RCC with increasing BMI has been observed for both men and women in many studies, and in some, relationships were stronger among women than men (IARC 2002). This study uses body mass index (BMI) to assess the role of overweight and obesity in RCC risk for Canadian men and women.

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## Methods

This study is part of the National Enhanced Cancer Surveillance System (NECSS) which collected individual data from a population-based sample, including 18 types of cancer, and 5380 population controls between 1994 and 1997, in the Canadian provinces of British Columbia, Alberta, Saskatchewan, Manitoba, Ontario, Prince Edward Island, Nova Scotia and Newfoundland.

*Cases*

A total of 2199 (995 female and 1204 male) histologically confirmed incident cases of kidney cancer were ascertained by participating provincial cancer registries between 1994 and 1997. Of these, 174 patients (7.9%) with kidney cancer (70 female and 104 male) had died at the time of the physician request; 152 patients (6.9%) of those diagnosed with kidney cancer (79 female and 73 male) were not contacted because the physician did not provide consent (generally because the patient was too ill). Of 1873 questionnaires sent (85.2% of kidney cancer patients ascertained), 1497 questionnaires were returned, a response rate for patients contacted of 79.9%. Of these, 1279 (691 male and 588 female) cases of RCC as defined by ICDO-2 (Percy et al. 1990) were included in this study.

*Controls*

The NECSS used frequency matching in the selection of population controls to obtain an age/sex distribution similar to that of all cancer cases in the NECSS (i.e., 18 cancer types: liver, testis, pancreas, brain, stomach, bladder, kidney, colon, rectum, prostate, breast, lung, bone, salivary, leukaemia, multiple myeloma, non-Hodgkin's lymphoma, and mesothelioma). Provincial cancer registries collected information from controls using the same protocol as for the cases. The strategies for identifying population controls varied by province depending on data availability and accessibility. In Prince Edward Island, Nova Scotia, Manitoba, Saskatchewan and British Columbia, an age group- and sex-stratified random sample of the province's population was obtained through the Provincial Health Insurance Plans. In Ontario, Ministry of Finance data were used to obtain a stratified random sample. Newfoundland and Alberta used random digit dialling to obtain a population sample.

Of 8117 questionnaires sent to selected potential controls, 573 questionnaires were returned because of a wrong address. A total of 5380 questionnaires (2704 for males and 2676 for females) were completed, representing 66.3% of controls ascertained and 71.3% (5380/7544) of controls contacted. For the present analyses, 10 controls (eight male and two female) were excluded because age was missing.

*Data collection*

The cancer registries identified most cases within one to three months of diagnosis through pathology reports. After obtaining physician consent for cases, questionnaires were mailed to cancer cases and controls by cancer registries. If the questionnaire was not completed and returned, a reminder postcard was sent out after 14 days and a second copy of the questionnaire at four weeks; after six weeks telephone follow-up was used, if required, to complete the questionnaire. Information was collected on socio-economic status, employment history, residential history, height, weight, smoking history, alcohol use, physical activity, dietary history, and use of vitamin or mineral supplements.

For weight, we collected information on how much subject weighed "about two years ago" (in pounds or kilograms) and the most subject had ever weighted. The body mass index (BMI), calculated as weight (kg) / height squared ($m^2$), was used to assess overweight and obesity. The WHO Technical Report Series 894 (2000) was used to classify BMI in adults as follows: underweight (BMI < 18.50), normal weight (BMI 18.50–24.99), overweight (BMI ≥ 25.00): pre-obesity (BMI 25.00–29.99); obese class I (BMI 30.00–34.99); obese class II (BMI 35.00–39.99); and obese class III (BMI ≥ 40.00).

Physical activity two years previous to the survey was assessed based on session frequency, seasons of participation and average time per session for each of 12 categories of the most common types of moderate and strenuous leisure-time physical activity in Canada: walking for exercise; jogging or running; gardening or yard work; home exercise or exercise class; golf or racquet sports; bowling or curling; swimming or water exercise; skiing or skating; bicycling; social dancing or other strenuous exercise.

The dietary portion of the questionnaire, which examined eating habits two years previous, was based on the Block-NCI Health Habits and History Questionnaire (Block et al. 1986) and modified somewhat to reflect the Canadian diet in collaboration with the Bureau of Biostatistics and Computer Applications, Food Directorate, Health Canada. A 70 item food frequency questionnaire provided data on Canadian eating patterns and the major source of nutrients in the Canadian diet, as well as the general changes in the individual's diet compared with 20 years ago. For each food item, cases and controls were asked to describe how often (per day, per week, per month) on average they ate the serving size specified of each item. The food groups were categorized based on quartile cut-off points for consumption reported by controls.

Information on alcohol consumption for two years previous was also collected, specifically beer, wine, and liquor.

Smoker was defined as having smoked at least 100 cigarettes in one's entire life; the questionnaire collected information on age when the person first started cigarette smoking, how many years they smoked in total, cigarettes (on average) per day, and age when they stopped smoking (if not currently a smoker).

Odds ratios and 95% confidence intervals (CIs) were computed as a measure of the relative risk. Unconditional logistic regression analysis was used for multivariate analyses using SAS software (SAS Institute Inc. 1996).

## Results

The demographic characteristics of cases and controls for RCC by sex are presented in Table 1. A total of 1279 newly diagnosed RCC cases (691 male and 588 female) and 5370 controls (2696 male and 2674 female) were included in this study. About 95% of the cases were older than 40 years old. For both males and females, cases of RCC reported a lower education level. For females, cases were more likely to report a low family income. Income adequacy was not reported for 20.3% and 23.3% of male and 29.6% and 28.6% of female cases and controls, respectively.

Table 2 shows the odds ratios for RCC according to selected factors among males and females. An inverse association between alcohol use and RCC was observed in both genders. Total consumption of fruit showed a statistically significant inverse association with RCC risk among females (test for trend $p < 0.01$). For both genders, total consumption of vegetables was also related to decreased risk; high total consumption of meat was associated with increased risk. No association between physical activity and risk of RCC was observed in this study.

The ORs for height and BMI among males and females are shown in Table 3. A total of 16 cases (four males and 12 females) and 119 controls (41 males and 78 females) in this study reported being underweight (BMI < 18.50); these subjects were combined with the normal BMI group in the analyses. After adjusting for 10-year age group, province, education, pack-years of smoking, alcohol use and total consumption of meat and of vegetables or fruit, significantly elevated ORs were observed among males and females for increased BMI, with a dose-response relationship (test for trend $p < 0.0001$ for both). Compared with normal BMI, the highest risk was found for obese class III: the adjusted ORs were 3.7 (95% CI = 1.5–9.4) for males and 3.8 (95% CI = 2.3–6.4) for females. No association was observed between height alone and RCC risk (using quartiles determined by responses of controls).

## Discussion

Our findings from this large population-based case-control study add evidence that overweight and obesity in adults are associated with an increased risk of RCC in both men and women, with a dose-response relationship. High BMI,

**Table 1** Demographic characteristics of cases and controls for renal cell carcinoma, NECSS Study, Canada, 1994–1997

| | Men | | | | Women | | | |
|---|---|---|---|---|---|---|---|---|
| | cases N | controls N | OR[a] (95% CI) | p value for trend | cases N | controls N | OR[a] (95% CI) | p value for trend |
| **Age** | | | | | | | | |
| 20–29 | 2 | 164 | | | 3 | 64 | | |
| 30–39 | 16 | 276 | | | 30 | 196 | | |
| 40–49 | 113 | 231 | | | 92 | 259 | | |
| 50–59 | 185 | 400 | | | 149 | 585 | | |
| 60–69 | 253 | 969 | | | 209 | 813 | | |
| ≥ 70 | 122 | 656 | | | 105 | 419 | | |
| total | 691 | 2696 | | | 588 | 2674 | | |
| **Family income** | | | | | | | | |
| low | 78 | 412 | 1.0 ref. | 0.57 | 122 | 439 | 1.0 ref. | 0.02 |
| lower middle | 129 | 459 | 1.4 (1.0–1.9) | | 107 | 449 | 0.8 (0.6–1.1) | |
| upper middle | 204 | 718 | 1.3 (1.0–1.7) | | 124 | 636 | 0.7 (0.5–0.9) | |
| high | 140 | 478 | 1.2 (0.9–1.7) | | 61 | 386 | 0.5 (0.4–0.7) | |
| not reported | 140 | 629 | 1.1 (0.8–1.4) | | 174 | 764 | 0.8 (0.6–1.0) | |
| **Education** | | | | | | | | |
| 1–8 yrs | 136 | 511 | 1.0 ref. | 0.004 | 124 | 352 | 1.0 ref. | 0.0001 |
| 9–13 yrs | 349 | 1246 | 0.9 (0.7–1.2) | | 317 | 1455 | 0.6 (0.5–0.8) | |
| ≥ 14 yrs | 196 | 898 | 0.7 (0.5–0.9) | | 138 | 831 | 0.5 (0.4–0.7) | |
| not reported | 10 | 41 | 0.9 (0.4–1.8) | | 9 | 36 | 0.7 (0.3–1.6) | |

[a] Adjusted for age and province.

**Table 2** Odds ratios for renal cell carcinoma by selected risk factors, NECSS Study, Canada, 1994–1997

| Factors | Males | | | | | Females | | | |
|---|---|---|---|---|---|---|---|---|---|
| | cases | controls | | p value for trend | | cases | controls | | p value for trend |
| | N | N | OR[a] (95% CI) | | | N | N | OR[a] (95% CI) | |
| **Current smoking status** | | | | | | | | | |
| never smoker | 157 | 704 | 1.0 ref. | 0.79 | | 283 | 1333 | 1.0 ref. | 0.75 |
| ex-smoker | 386 | 1354 | 1.2 (1.0–1.5) | | | 195 | 805 | 1.3 (1.0–1.6) | |
| current smoker | 113 | 558 | 0.9 (0.7–1.2) | | | 83 | 479 | 0.9 (0.7–1.2) | |
| not reported | 35 | 80 | | | | 27 | 57 | | |
| **Total alcohol (servings/week)[b]** | | | | | | | | | |
| None | 217 | 751 | 1.0 ref. | 0.0003 | None | 342 | 1245 | 1.0 ref. | 0.0003 |
| 1–3 | 160 | 653 | 0.8 (0.6–1.0) | | <3 | 137 | 727 | 0.8 (0.6–1.0) | |
| 3.1–8.5 | 161 | 632 | 0.7 (0.6–0.9) | | 3–7 | 66 | 430 | 0.6 (0.5–0.9) | |
| ≥ 8.6 | 152 | 658 | 0.6 (0.5–0.8) | | ≥ 7.1 | 43 | 270 | 0.6 (0.4–0.9) | |
| not reported | 1 | 2 | | | | 0 | 2 | | |
| **Moderate activity (hr/month)[c]** | | | | | | | | | |
| < = 4.2 | 138 | 572 | 1.0 ref. | 0.96 | ≤ 5.2 | 115 | 499 | 1.0 ref. | 0.58 |
| 4.3–11.6 | 76 | 354 | 0.9 (0.6–1.2) | | 5.3–12.0 | 69 | 321 | 1.0 (0.7–1.4) | |
| 11.7–24.4 | 74 | 355 | 0.9 (0.6–1.2) | | 12.1–22.8 | 57 | 322 | 0.9 (0.6–1.3) | |
| ≥ 24.5 | 88 | 354 | 1.0 (0.7–1.4) | | ≥ 22.9 | 63 | 320 | 0.9 (0.6–1.3) | |
| **Strenuous activity (hr/month)[c]** | | | | | | | | | |
| None | 132 | 575 | 1.0 ref. | 0.89 | None | 150 | 571 | 1.0 ref. | 0.19 |
| < = 1.7 | 62 | 277 | 0.9 (0.6–1.3) | | ≤ 0.8 | 41 | 217 | 0.9 (0.6–1.3) | |
| 1.8–9.0 | 60 | 249 | 1.0 (0.7–1.5) | | 0.9–3.7 | 33 | 208 | 0.7 (0.4–1.1) | |
| ≥ 9.1 | 61 | 265 | 1.0 (0.7–1.5) | | ≥ 3.8 | 46 | 237 | 0.8 (0.6–1.3) | |
| not reported | 61 | 269 | | | | 34 | 229 | | |
| **Total consumption of fruit (servings/week)[c]** | | | | | | | | | |
| <3.9 | 164 | 686 | 1.0 ref. | 0.10 | < 5.9 | 188 | 679 | 1.0 ref. | 0.01 |
| 3.9–8.4 | 193 | 664 | 1.2 (0.9–1.5) | | 5.9–10.0 | 124 | 658 | 0.6 (0.5–0.8) | |
| 8.5–14.0 | 187 | 704 | 1.1 (0.8–1.4) | | 10.1–16.0 | 135 | 685 | 0.7 (0.5–0.9) | |
| >14.0 | 146 | 640 | 0.8 (0.6–1.1) | | >16.0 | 141 | 650 | 0.7 (0.5–0.9) | |
| not reported | 1 | 2 | | | | 0 | 2 | | |
| **Total consumption of vegetables (servings/week)[c]** | | | | | | | | | |
| <11.9 | 168 | 657 | 1.0 ref. | 0.08 | <13.4 | 166 | 664 | 1.0 ref. | 0.03 |
| 11.9–17.4 | 186 | 673 | 0.9 (0.7–1.2) | | 13.4–18.8 | 150 | 670 | 0.8 (0.6–1.1) | |
| 17.5–24.4 | 184 | 693 | 0.9 (0.7–1.2) | | 18.9–25.5 | 136 | 663 | 0.7 (0.5–1.0) | |
| >24.4 | 152 | 671 | 0.8 (0.6–1.0) | | >25.5 | 136 | 675 | 0.7 (0.5–1.0) | |
| not reported | 1 | 2 | | | | 0 | 2 | | |
| **Total consumption of meat (servings/week)[c]** | | | | | | | | | |
| <4.9 | 149 | 704 | 1.0 ref. | 0.12 | <3.5 | 136 | 691 | 1.0 ref. | 0.07 |
| 4.9–8.0 | 167 | 658 | 1.2 (0.9–1.6) | | 3.5–6.0 | 136 | 655 | 1.1 (0.8–1.5) | |
| 8.1–12.4 | 176 | 664 | 1.1 (0.8–1.5) | | 6.1–9.4 | 138 | 661 | 1.1 (0.8–1.5) | |
| >12.4 | 198 | 668 | 1.3 (1.0–1.8) | | >9.4 | 178 | 665 | 1.3 (1.0–1.8) | |
| not reported | 1 | 2 | | | | 0 | 2 | | |

[a] Adjusted for 10-year age group, province, education, body mass index (<25.00, 25.00–29.99, ≥30.00); ORs for smoking status and alcohol use are also adjusted for total consumption of meat and vegetables and fruit; ORs for food variables are adjusted for smoking status and alcohol consumption.
[b] Includes liquor, beer and wine.
[c] Cutpoints by quartile determined by consumption reported by controls; information on physical activity did not include province of Ontario. Total vegetables: Tomatoes, carrots, broccoli, cabbage, cauliflower, brussel sprouts, spinach or other greens, yellow squash, green beans, corn, peas or any other vegetable; soups with vegetables; baked, boiled or mashed potatoes; french fries or fried potatoes; sweet potatoes; tofu or soybeans; baked beans or lentils. Total fruit: Apples, pears, oranges, bananas, cantaloupe or other fruit fresh or canned: Total consumption of meat: Beef, pork, lamb, hamburger, hotdogs, bacon, sausage, smoked meat, luncheon meat, liver.

particularly BMI ≥ 40.00 (obese class III), was strongly associated with an increased risk of developing RCC in both sexes.

In some studies, obesity has been associated with an increased risk of RCC among women, although evidence of the association among men is considered weaker (McLaughlin et al. 1984; Mellemgaard et al. 1994b; Mellemgaard et al. 1995; Chow et al. 1996). A record-linkage study of 43965 obese Danes found an increased incidence for cancer of the kidney in women only (Moller et al. 1994). Other research, however, indicates that obesity increases the risk of RCC among both men and women (Yu et al. 1986; Asal et al. 1988; Yuan et al. 1998; Shapiro et al. 1999b), consistent with our results. A large cohort study, which followed 363992

**Table 3** Odds ratios for renal cell carcinoma by height and body mass index (BMI), NECSS Study, Canada, 1994–1997

| Factors | Males | | | | | Females | | | |
|---|---|---|---|---|---|---|---|---|---|
| | cases | controls | | p value for trend | | cases | controls | | p value for trend |
| | N | N | OR[a] (95% CI) | | | N | N | OR[a] (95% CI) | |
| Total subjects | 691 | 2696 | | | | 588 | 2674 | | |
| **Height** (cm)[b] | | | | | **Height** (cm)[b] | | | | |
| < 172 | 133 | 673 | 1.0 ref. | 0.84 | < 158 | 181 | 853 | 1.0 ref. | 0.76 |
| 172-177 | 251 | 1081 | 0.9 (0.7–1.2) | | 158-162 | 148 | 773 | 0.9 (0.7–1.1) | |
| 178-180 | 131 | 396 | 1.1 (0.8–1.5) | | 163-167 | 149 | 621 | 1.0 (0.8–1.4) | |
| >180 | 174 | 541 | 1.0 (0.7–1.3) | | >167 | 109 | 422 | 1.0 (0.7–1.3) | |
| not reported | 2 | 5 | | | | 1 | 5 | | |
| **BMI** | | | | | | | | | |
| normal weight (18.50–24.99)[c] | 147 | 1044 | 1.0 ref. | 0.0001 | | 221 | 1517 | 1.0 ref. | 0.0001 |
| overweight (≥ 25.00) | | | | | | | | | |
| pre-obesity (25.00–29.99) | 369 | 1225 | 2.2 (1.7–2.7) | | | 200 | 795 | 1.5 (1.2–1.9) | |
| obese class I (30.00-34.99) | 144 | 331 | 2.8 (2.2–3.8) | | | 100 | 232 | 2.5 (1.9–3.4) | |
| obese class II (35.00-39.99) | 21 | 67 | 1.9 (1.1–3.3) | | | 31 | 76 | 2.7 (1.7–4.4) | |
| obese class III (≥ 40.00) | 8 | 16 | 3.7 (1.5–9.4) | | | 33 | 43 | 3.8 (2.3–6.4) | |
| not reported | 2 | 13 | | | | 3 | 11 | | |

[a] Adjusted for 10 year age group, province, education, pack-years of smoking, alcohol use and total consumption of meat and of vegetables and fruit.
[b] Cutpoints by quartile determined by values reported by controls; ORs for height are also adjusted for weight.
[c] Normal weight group also includes those with BMI < 18.50: 16 cases (four male and 12 female) and 119 controls (41 male and 78 female).

Swedish men for as long as 25 years, found that obesity increased the risk of RCC, with a dose-response relationship, and another Swedish cohort study with one to 29 years of follow-up found significant risk elevation for RCC cancer among obese patients, both men and women (Chow et al. 2000; Wolk et al. 2001). Bergstrom et al. (2001a) reviewed 24 studies, 22 of which included both men and women. In their quantitative analysis which included 14 studies of men and 14 of women, the association of increased risk of RCC with increased BMI was equally strong among men and women. Another recent meta-analysis indicated that 25% of cancer of the kidney in both sexes was attributable to excess weight (Bergstrom et al. 2001b).

One consideration in discussing risks associated with obesity is the role of estrogen. Estrogens induce renal tumours in animal models (Kirkman 1959; Hodgson et al. 1998), and sex hormone receptors have been demonstrated in normal and malignant renal tissue in hamsters and in humans (Jakse & Muller-Holzner 1988; Cortes-Vizcaino & Llombart-Bosch 1993). Obesity increases the serum concentrations of free estrogens (Hsiech et al. 1998; Cohen 1999); these may increase the risk of RCC. Obese women, especially those who are post-menopausal, have a higher estrogen level than non-obese women (Kalin & Zumoff 1990). Associations with some reproductive factors and the use of menopausal estrogens and RCC risk have been observed by Lindblad et al. (1995) & Chow et al. (1995). However, other epidemiologic studies of RCC among women have found no clear association between the risk of this cancer and the use of exogenous estrogens (Gago-Dominguez et al. 1999; McLaughlin & Lipworth 2000). In obese men, the estrogen level is also increased and the testosterone level is normal or decreased (Riva et al. 1992). Estradiol concentration increased with increasing BMI in healthy, elderly Greek men (Hsiech et al. 1998).

The NECSS study was conducted with a large population-based sample from eight Canadian provinces. However, our data on weight and height were self-reported, and we did not carry out a validation study in a sample of participants. In general, self-reported weights are considered by some to be sufficiently accurate in epidemiological studies (Stunkard & Albaum 1981; Stewart 1982; Le Marchand et al. 1988). Others have indicated that self-reported weight and height are highly correlated with measured values, but obese subjects in general under-report their weight more than non-obese subjects while underweight subjects overestimate their body size (Kuskowska et al. 1989; Stevens et al. 1990).

In our study, we asked for weight two years previous to the survey to avoid measuring weight loss due to disease. Recalling weight two years previous could give misleading results, for example, cases might report a weight much higher or lower than their actual weight two years earlier. The possibility of differential misclassification (recall bias) cannot be excluded in retrospective case-control studies. However, the consistency of our findings with other case-control and prospective cohort studies argues against recall bias. Specifically, a case-control study which collected data for height and weight from medical records also indicates that increased BMI was associated with increases in risk of RCC for both men and women (Shapiro et al. 1999a).

Because 14.8% of the kidney cancer cases were too ill or had died, and our data did not include information from proxies, assessments for nonparticipating individuals were not available. Thus we could not determine the extent to which selection bias was introduced.

Differences in other risk factors such as smoking, alcohol use and diet have been accounted for in our analysis, but we could not consider the potential confounding effects of medication and medical history; in particular, we did not have data on hypertension or use of anti-hypertensive medication, for which associations are reported in some studies (McCredie & Stewart 1992; McLaughlin et al. 1995; Yuan et al. 1998; Gago-Dominguez et al. 1999; Chow et al. 2000); but not others (Mellemgaard et al. 1994a; Heath et al. 1997; Shapiro et al. 1999b). However, obesity and hypertension independently increase the risk of RCC, suggesting that these factors act to increase this risk through different mechanisms (Chow et al. 2000).

In summary, our findings support previous studies: overweight and obesity play an important role in the development of RCC for both genders. Maintaining a healthy body weight over the lifetime may be an effective strategy to control RCC.

**Zusammenfassung**

**Risiko eines Nierenzellkarzinoms für übergewichtige und adipöse Erwachsene in Kanada**

**Zielsetzung:** Untersuchen, welche Rolle Übergewicht und Adipositas beim Auftreten von Nierenzellkarzinomen (NZK) in Kanada spielen.

**Methoden:** Schriftliche Befragung in acht kanadischen Provinzen von 1994 bis 1997 zur Datenerfassung von 1279 neu diagnostizierten, histologisch bestätigten NZK-Fällen (691 Männer, 588 Frauen) sowie 5370 Kontrollfällen in der Bevölkerung. Erfasst wurden sozioökonomischer Status, Körpergrösse und -gewicht, Rauchgewohnheiten, Alkoholkonsum, Ernährung sowie Angaben zu Wohnsitz- und Berufsgeschichte. Die Gewichtsangaben wurden als Körpermasseindex (Body Mass Index, BMI) präsentiert. Odds Ratios (ORs) und 95-Prozent-Konfidenzintervalle (KI) wurden mittels unbedingter logistischer Regression ermittelt.

**Ergebnisse:** Es wurde ein erhöhtes NZK-Risiko bei männlichen und weiblichen Erwachsenen mit Übergewicht und Adipositas festgestellt; die Trendanalyse war statistisch signifikant ($p < 0{,}0001$ für beide Geschlechter). Im Vergleich zu Personen mit einem normalen BMI betrugen die adjustierten/bereinigten ORs für Männer und Frauen in der Gewichtsklasse III (BMI $\geq 40$) 3,7 (95% KI = 1,5–9,3) bzw. 3,8 (95% KI = 2,3–6,4).

**Schlussfolgerung:** Diese Ergebnisse zeigen, dass Übergewicht und Adipositas eine bedeutende Rolle in der Ätiologie von Nierenzellkarzinomen bei männlichen und weiblichen Erwachsenen spielen.

**Résumé**

**Surpoids et obèsité chez l'adulte et risque de carcinome de la cellule rénale au Canada**

**Objectifs:** Évaluer la contribution du surpoids et de l'obésité au risque d'hypernéphrome au Canada.

**Méthodes:** Des questionnaires ont été envoyés par la poste pour recueillir des données sur 1279 nouveaux cas diagnostiqués d'hypernéphrome qui ont été confirmés par un examen histologique (691 hommes et 588 femmes) et sur 5370 témoins, entre 1994 et 1997, dans huit provinces canadiennes. Des données ont été recueillies sur le niveau socio-économique, la taille, le poids, les habitudes de consommation de tabac et d'alcool, l'alimentation et les antécédents résidentiels et professionnels. Le poids a été exprimé au moyen de l'indice de masse corporelle (IMC). Les odds ratios (OR) et les intervalles de confiance (IC) à 95% ont été calculés à l'aide d'un modèle de régression logistique non conditionnelle.

**Résultats:** L'étude a mis en évidence un risque accru d'hypernéphrome associé à l'excès de poids et à l'obésité autant chez les hommes que chez les femmes; le test visant à évaluer la tendance était statistiquement significatif. Comparativement à l'IMC normal, les OR ajustés étaient respectivement de 3,7 (IC à 95% = 1,5–9,4) et 3,8 (IC à 95% = 2,3–6,4) pour les obèses de classe III (IMC $\geq 40$ kg/m$^2$) de sexe masculin et féminin.

**Conclusions:** Ces résultats indiquent que l'excès de poids et l'obésité jouent un rôle important dans l'étiologie de l'hypernéphrome chez les adultes des deux sexes.

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