Practice of Epidemiology

Scaling of Weight for Height in Relation to Risk of Cancer at Different Sites in a Cohort of Canadian Women

Geoffrey C. Kabat®, Moonseong Heo, Anthony B. Miller, and Thomas E. Rohan

* Correspondence to Dr. G. C. Kabat, Department of Epidemiology and Population Health, Albert Einstein College of Medicine, 1300 Morris Park Avenue, Bronx, NY 10461 (e-mail: Geoffrey.Kabat@einstein.yu.edu).

Initially submitted February 24, 2012; accepted for publication May 15, 2012.

Many studies have examined the associations of body mass index (weight (kg)/height (m)²) with risk of various cancers. However, optimal scaling of weight for height may depend on the population studied. The authors used data from a large cohort study of women (Canadian National Breast Cancer Screening Study, 1980–2000; n = 89,835) to examine how the scaling of weight for height (W/Hx) influenced the association with risk of 19 different cancers. Cox proportional hazards models were used to estimate the hazard ratio for each cancer site with W/Hx, with x increasing from 0 to 3.0 by increments of 0.1. The correlation between weight and W/Hx decreased monotonically with increasing x, whereas W/Hx was minimally correlated with height when x = 1.4. W/Hx showed significant positive associations with postmenopausal breast cancer, endometrial cancer, kidney cancer, and lung cancer in never smokers. W/Hx was inversely associated with lung cancer in ever smokers. The value of x for which W/Hx produced the largest statistically significant hazard ratio ranged from 0.8 (endometrial cancer) to 1.7 (postmenopausal breast cancer). For lung cancer in ever smokers, the inverse association was statistically significant for all values of x. These findings suggest that the scaling of weight for height may vary depending on the cancer site and that optimal scaling may be considerably different from W/H² or, alternatively, that a range of scaling should be considered when examining the association of body weight with risk of disease.

adiposity, body mass index, cancer, cohort studies, scaling

Abbreviations: BMI, body mass index; CNBSS, Canadian National Breast Screening Study; W/Hx, weight/heightx.

Body mass index (BMI, determined as weight in kilograms divided by height in meters squared), which was originally referred to as the Quetelet Index after the Belgian mathematician Adolphe Quetelet who first proposed it in 1835 (1), has become the most widely used weight-for-height index of adiposity in large population studies of children and adults (2–4). The fact that many large epidemiologic studies only have information on weight and height and the ease of computing BMI appear to have contributed to its popularity. BMI is positively associated with increased risk of a number of cancers (including endometrial, postmenopausal breast, colorectal, esophageal adenocarcinoma, and renal cancers) (5–7) and with increased cancer incidence and mortality overall (6, 8), as well as with increased risk of diabetes and cardiovascular disease (9).

Inverse associations of BMI with a number of cancers have also been observed, specifically for premenopausal breast, lung, and oropharyngeal cancers and squamous cell carcinoma of the esophagus (6, 7).

Although BMI has been adopted as a proxy for adiposity and has been widely used to assess the impact of obesity on health, the limitations of BMI as a marker of adiposity have long been recognized (10–14). BMI does not distinguish between body fat and lean mass, and individuals who are physically fit will tend to have a higher BMI because of their increased muscle mass (10). On the other hand, postmenopausal women are more likely to have BMIs that reflect reduced lean mass, and so they may have a relatively low BMI but more fat than do younger women. BMI was adopted because in the original populations...
studied (predominantly middle-aged men) (2, 13, 15), it was more strongly correlated with direct measures of adiposity (e.g., skinfold thickness and hydrostatic weighing) than was weight alone and because it was strongly correlated with weight and minimally correlated with height. However, other studies suggest that the optimal weight-for-height index is dependent on the population and that weight (kg)/height (m)^2 (W/H^2), where the scaling factor x takes values other than 2, performs better than BMI because it is highly correlated with weight and virtually independent of height (14, 16–18).

The fact that a number of cancers are associated with height (19, 20) (and also with BMI) further underscores the need for a weight-for-height measure that is minimally correlated with height so as to allow independent assessment of the associations of indices of obesity and of height with cancer. Renehan (21) recently pointed out that the association of BMI with cancer at different sites may be confounded by weight and height (19, 20) (and also with BMI) further underscores the need for a weight-for-height measure that is minimally correlated with height (14, 16–18).

Few studies have used weight-for-height indices other than BMI or compared the performances of these indices in estimating of the independent associations of obesity indices with the risk of specific cancers. It is possible that the optimum weight-for-height index differs for different cancer sites both because weight and height may each have different associations with specific cancers and because body weight may increase (or decrease) the risk of cancer at different sites via different mechanisms (22, 23).

We therefore used measured weight and height data from 89,835 women enrolled in the Canadian National Breast Screening Study (CNBSS) prospective cohort to compare the performance of a range of weight-for-height measures as predictors of the risk of different cancers, both with and without adjustment for potential confounding factors.

**MATERIALS AND METHODS**

**Study population and questionnaire**

The CNBSS is a randomized controlled trial of screening for breast cancer, the details of which have been described elsewhere (24, 25). The Human Experimentation Committee at the University of Toronto and Human Experimentation Committees at 15 CNBSS collaborating centers approved the study. In brief, 89,835 women aged 40–59 years were recruited from the general Canadian population between 1980 and 1985. On enrollment into the study, information was obtained from participants on demographic, hormonal, reproductive, and lifestyle characteristics using a self-administered questionnaire. Participating women had their weight and height measured at the initial examination.

Starting in 1982, a self-administered food frequency questionnaire developed for the CNBSS (26) was distributed to all new attendees at all screening centers and to women returning to the centers for rescreening. The food frequency questionnaire elicited information on usual portion size and consumption of 86 food and beverage items, including alcoholic beverages, as well as 2 questions about usual level of moderate and vigorous physical activity during the past month (hours per day). The questionnaire included photographs of portion sizes to assist respondents in quantifying intake. Responses were used to estimate the daily intake of calories, alcohol, and selected nutrients using a nutrient database developed by modifying the US Department of Agriculture’s food composition tables to include typically Canadian foods (27). A total of 49,654 dietary questionnaires were returned and were available for analysis. Use of the self-administered questionnaire was validated by comparison with an interviewer-administered version of the same questionnaire (26).

**Ascertainment of index cancers and deaths**

Incident cases of cancer and deaths from all causes were ascertained by means of computerized record linkages to the Canadian Cancer Database and the National Mortality Database, respectively. The linkages yielded data on cancer incidence and death to December 31, 2000, for women in Ontario, December 31, 1998, for women in Quebec, and December 31, 1999, for women in other provinces. For the present analyses, study participants were considered at risk from their date of enrollment until the date of diagnosis of 1 of 19 different types of cancer, termination of follow-up (the date until which cancer incidence data were available for women in the corresponding province), or death, whichever occurred first. We excluded women who were missing height or weight measurements at baseline (n = 868) and women who were diagnosed with cancer before their date of enrollment (n = 711). After exclusions, during an average of 16.4 years (1,429,734 person-years) of follow-up, a total of 5,679 incident invasive cancers were ascertained among 88,256 women. Where an individual had more than 1 cancer diagnosis, we selected the first diagnosis for inclusion, and follow-up of that individual stopped at that point. We selected types of cancer for which there were more than 90 cases for inclusion in the analysis. The number of cases by site and person-years of follow-up are given in Table 1. Analyses of endometrial cancer were restricted to women with intact uteri at baseline, and cases of ovarian cancer were restricted to women with at least 1 intact ovary at baseline.

**Statistical analysis**

We examined the correlation of W/H^2 with weight and height for increments of x of 0.1, ranging from x = 0 to x = 3.0. Cox proportional hazards models were used to estimate hazard ratios and 95% confidence intervals for the association of W/H^2 with each cancer site, where x ranged from 0 to 3.0 in increments of 0.1. Specifically, for each cancer site, we fitted 31 age-adjusted models (model 1) and another 31 multivariable-adjusted models (model 2), resulting in a total of 1,178 model fittings and the same number of hazard ratios.

The following covariates were included in the multivariable model: age at enrollment (continuous), oral contraceptive use (ever vs. never), hormone therapy (ever vs. never), menopausal status (premenopausal, perimenopausal, or...
postmenopausal), years of education (<12, 12, or >12), and pack-years of smoking (never smoked, 1–9, 10–19, 20–29, 30–39, or ≥40). The model for colorectal cancer also included terms for parity (0, 1, 2, or ≥3 children). For breast cancer, we performed analyses stratified by menopausal status (premenopausal vs. postmenopausal), and for lung cancer, we performed analyses after stratification by smoking status (ever vs. never). For breast cancer, the models stratified by menopausal status also included age at menarche (<12, 12, 13, or ≥14 years), parity (0, 1, 2, or ≥3 children), family history of breast cancer in a first-degree relative (yes vs. no), and history of benign breast disease (yes vs. no). In addition to the analysis of the entire cohort, we carried out analyses for 6 sites in the dietary subcohort to include covariates that were available only for women who completed the food frequency questionnaire (n = 49,654). For the 6 cancer sites, we compared the results of the multivariable model used in the entire cohort but restricted to the dietary subcohort with the results of the models including additional variables, as follows: premenopausal and postmenopausal breast cancer (alcohol intake in g/day (continuous) and physical activity level in hours/day (continuous)); colorectal cancer, colon cancer, and rectal cancer (alcohol intake, physical activity level, dietary folate, and red meat intake); and endometrial cancer (physical activity).

All statistical significance tests were 2-sided. All analyses were performed using SAS, version 9 (SAS Institute, Inc., Cary, North Carolina).

**RESULTS**

Table 2 presents the distribution of personal characteristics of study participants by quintiles of weight and BMI. Mean age was 49.0 years. Twenty-seven percent of women had more than 12 years of education and 53.4% were postmenopausal. The percentages of postmenopausal women and women who experienced menarche before 12 years of age were strongly and positively associated with BMI, whereas postmenopausal), years of education (<12, 12, or >12), and pack-years of smoking (never smoked, 1–9, 10–19, 20–29, 30–39, or ≥40). The model for colorectal cancer also included terms for parity (0, 1, 2, or ≥3 children). For breast cancer, we performed analyses stratified by menopausal status (premenopausal vs. postmenopausal), and for lung cancer, we performed analyses after stratification by smoking status (ever vs. never). For breast cancer, the models stratified by menopausal status also included age at menarche (<12, 12, 13, or ≥14 years), parity (0, 1, 2, or ≥3 children), family history of breast cancer in a first-degree relative (yes vs. no), and history of benign breast disease (yes vs. no). In addition to the analysis of the entire cohort, we carried out analyses for 6 sites in the dietary subcohort to include covariates that were available only for women who completed the food frequency questionnaire (n = 49,654). For the 6 cancer sites, we compared the results of the multivariable model used in the entire cohort but restricted to the dietary subcohort with the results of the models including additional variables, as follows: premenopausal and postmenopausal breast cancer (alcohol intake in g/day (continuous) and physical activity level in hours/day (continuous)); colorectal cancer, colon cancer, and rectal cancer (alcohol intake, physical activity level, dietary folate, and red meat intake); and endometrial cancer (physical activity).

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the percentages of women who had more than 12 years of education, who were nulliparous, or who were ever contraceptive users were strongly and inversely associated with BMI.

In the total population, W/H was (necessarily) maximally correlated with weight when x = 0 (i.e., W/H$^0$ = W), and the correlation declined monotonically, yet very slowly, with increasing x (Figure 1). On the other hand, W/H was minimally correlated with height when x = 1.4. Thus, it appears that the ideal scaling in this population is x = 1.4, where the correlation with height is 0 and that with weight is still very high (0.95).

In Figure 2, age- and multivariable-adjusted hazard ratios are presented for the association of W/H with 19 different cancers as x increased from 0 to 3.0 in increments of 0.1. In multivariable-adjusted models, W/H showed significant positive associations with postmenopausal breast cancer, endometrial cancer, kidney cancer, and lung cancer in never smokers and was inversely associated with lung cancer in ever smokers. The values of x that produced the largest statistically significant hazard ratio were 1.7 for postmenopausal breast cancer, 1.1 for kidney cancer, 0.8 for endometrial cancer, and 1.3 for lung cancer in never smokers. For lung cancer in ever smokers, the hazard ratio decreased monotonically with increasing x and was highly statistically significant for all values of x. For the 5 sites that showed statistically significant results, the estimates from the age- and multivariable-adjusted models were generally similar. For pancreatic and cervical cancers, W/H showed positive associations in age-adjusted models but not in multivariable-adjusted models. The following sites/types showed no association with W/H: premenopausal breast, colorectum, colon, rectum, thyroid, bladder, ovary, brain, melanoma, leukemia, non-Hodgkin’s lymphoma, and multiple myeloma.

The results of analyses carried out in the dietary subcohort for 5 cancer sites were similar to the results reported for the entire cohort in Figure 2; however, for postmenopausal breast cancer in the expanded model, the range of statistically significant hazard ratios was limited to x = 0 to 0.2 (results not shown).

To further examine the effect of different scaling, we compared the association of 4 indices (W, W/H, W/H$^{1.4}$, and W/H$^{2}$) with the risk of cancer at the 5 sites that showed significant associations in the primary analysis (Table 3). For 3 types of cancer (endometrial cancer, kidney cancer, and lung cancer in ever smokers), quintiles of weight showed a steeper trend compared with the other indices. For kidney cancer and lung cancer in never smokers, there was a trend toward higher P values with increasing scaling. Kidney cancer showed the greatest range in P values, which decreased with increasing values of the scaling factor. However, all indices showed significant associations with all 5 sites. Particularly for endometrial cancer and lung cancer in ever smokers, all 4 indices gave comparably strong associations. Addition of height as a covariate in models for all 4 indices did not materially affect the results that are presented in Table 3.

**DISCUSSION**

We examined the scaling of W/H, where x ranged from 0 to 3, in relation to the risk of cancer at 19 anatomic sites. For the 4 sites that showed a statistically significant positive association with W/H, the highest value of x (that was statistically significant) ranged from 0.8 (endometrial cancer) to 1.7 (postmenopausal breast cancer). For lung cancer in ever smokers, all values of x between 0 and 3.0 yielded statistically significant inverse associations. Two sites (cervix uteri and pancreas) showed positive associations in the age-adjusted model but not in the multivariable-adjusted model. The 12 remaining sites were not associated with
W/H^x for any value of x between 0 and 3. There was a high degree of consistency when scaling was between 0 and 2 as to whether a significant association with a cancer outcome was observed. Furthermore, inclusion of height as a covariate did not materially affect the associations of weight-for-height indices with cancer outcomes. To the best of our knowledge, no previous study has examined the performance of a range of different weight-for-height indices in association with different cancer sites.

BMI is by far the most widely used index of obesity/adiposity in epidemiologic studies. However, it has long been recognized that BMI is an imperfect indicator of adiposity because it does not distinguish between fat mass and lean mass (10, 13, 14). Three issues in particular are relevant to determining the optimal weight-for-height measure to assess the association of adiposity/obesity with disease risk in epidemiologic studies: 1) the ideal weight-for-height index should be maximally correlated with weight (itself a proxy for adiposity) and minimally correlated with height; 2) the index should be maximally correlated with adiposity in validation studies using more accurate techniques to measure body fat, such as hydrostatic weighing, bioelectrical impedance, and dual energy x-ray absorptiometry; and 3) the index should show consistent associations with obesity-related outcomes. We discuss each of these issues below.

First, numerous studies have addressed the question of what the optimal weight-for-height index to serve as an indicator of adiposity/obesity in large epidemiologic studies is (2, 13–18, 28–31). Although there is general agreement that the optimal index is that which is maximally correlated with weight and least correlated with height, there is disagreement about which index performs best. Several studies (mainly limited to men) (2, 13, 15) that compared W/H, W/H^2, and W/H^3 concluded that W/H^2 was the best of the 3 indices. However, other studies suggested that the best index may differ between men and women (14, 17, 18) and may depend on the population studied (16–18). Benn (16) argued that the exponent should be derived from the population under study and proposed use of W/H^p, where p could be a noninteger. In studies by Florey (14), Lee et al. (17), and Micozzi et al. (18), x was consistently lower in females than in males. The work of Benn (16), Florey (14), and Lee et al. (17) suggests that the best weight-for-height index will depend on the population studied.

Second, studies have attempted to validate different weight- and height-based indices of obesity against more direct measurements of body fat, including skinfold thickness, hydrostatic weighing, and dual-energy x-ray absorptiometry (2, 13, 14, 18, 28, 29, 31). Benn's index and W/H^2 have generally shown the strongest correlations with direct measures of adiposity (28, 29). In an analysis data from the National Health and Nutrition Examination Survey, Micozzi et al. (18) found that in men, W/H^2 showed a high correlation with weight, the highest correlation with measured body fat, and no significant correlation with height, whereas in women W/H and W/H_1.5 showed the highest correlations with weight and subscapular skinfold thickness and no significant correlation with height.

Larsson et al. (31) examined optimized predictions of absolute and relative amounts of body fat, measured using dual-energy x-ray absorptiometry, by different weight-for-height indices in which the power for height ranged from 0 to 3.0. Their study population consisted of over 1,100 randomly selected subjects from the general populations of 2 Swedish cities and 149 obese subjects from the same 2 cities. Correlations between W/H and W/H^2 and absolute body fat measured using dual-energy x-ray absorptiometry in men and women were high (0.91 and 0.88, respectively, in men and 0.97 and 0.96, respectively, in women). The optimal value of x in W/H^x in predicting absolute body fat was close to 1.0 in both men and women, whereas W/H^2 was the optimal predictor of percent body fat, although BMI was not proportional to either measure of body fat in severely obese subjects. The authors concluded that W/H might be a more optimal weight-for-height index than BMI, particularly at high body weights.

Third, we are unaware of previous studies in which investigators have examined the performance of a range of weight-for-height indices in relation to the risk of cancer at different sites. Lee and Kolonel (32) compared the performance of 4 weight-for-height indices (W/H, W/H^2, W/H^3, and W/H^p (Benn’s index)) in relation to the risk of lung cancer in a small, nested case-control study. Although all 4 indices showed an inverse association with risk, only W/H and W/H^p showed monotonic decreasing trends in the odds ratio by BMI quintiles. The authors concluded that different mass indices were not interchangeable and that W/H^p was the “body mass index of choice in epidemiologic analysis” (32, p. 377).

Our results suggest that the optimal scaling of W/H^x in measuring adiposity may differ by cancer site and may be below the value of 2.0, which is used for BMI. In the present study, the value of x that was least correlated with height and maximally correlated with weight was 1.4. However, the optimal value of x differed by cancer site/type. All 5 sites that showed significant associations with W/H^x also showed significant associations when x = 2 (i.e., for BMI); however, for 4 of the 5 sites, the magnitude of the hazard ratios (absolute value) was larger for values of x < 2 than for x = 2. This suggests that the statistical power to detect a significant association with W/H^x may be greater for the optimal value of x compared with using BMI. Nevertheless, the fact that for specific cancers W/H^x showed the largest hazard ratio for specific values of x does not necessarily imply that that value of x is maximally associated with reliably measured body fat or that similar correlations would be obtained using different sample populations. At the very least, our results indicate that it may be useful to consider a range of weight-for-height indices when examining the association of body weight with disease.

There has been inconsistency among different studies as to the association of BMI with certain cancer sites (e.g., thyroid cancer (33)), and there is inconsistency within some studies by sex (e.g., studies of colorectal cancer (34)). Some of these inconsistencies may be due to confounding, which in some studies was positively associated with thyroid and colorectal cancers (19, 20, 33). The need for a weight-for-height index of adiposity that is uncorrelated with height takes on added importance in light of the
fact that a number of cancers are positively associated with height (19, 20). Renehan (21) recently pointed out that the association of BMI with a given outcome may be confounded by height. However, our analyses of specific cancers indicated that confounding of the association of a range of weight-for-height indices by height was minimal. Further studies are needed that examine the independent associations of indices of adiposity and height with the risk of specific cancers. Furthermore, although obesity and leaness are associated with a number of cancers, the association of obesity with different cancers may operate through different pathways (22, 23, 35). For example, the associations of obesity with postmenopausal breast cancer and endometrial cancer may involve estrogen, whereas that of obesity with colorectal cancer may operate through insulin and related growth factors and that of obesity with renal cancer may operate through effects on blood pressure (35).

Strengths of the present study include the availability of measured height and weight from a large population, large numbers of cancers at specific sites, completeness of follow-up due to linkage to national cancer and death registries, and the availability of information on potential confounding variables. However, a major limitation is that we were not able to examine the associations of W/H in men, which would have enabled us to assess consistency between the 2 sexes for cancer sites that occurred in both men and women. Furthermore, we were not able to adjust for several covariates, including alcohol intake and physical activity level, in the full cohort; however, the results were unchanged in analyses in which we controlled for these variables in the dietary sub-cohort. Data on other key covariates, such as family history of colorectal cancer, were unavailable.

In conclusion, in a cohort of nearly 90,000 Canadian women, the scaling of weight for height that was most...
strongly associated with weight and least associated with height was 1.4. W/H was positively associated with the risk of 4 cancers (postmenopausal breast cancer, endometrial cancer, kidney cancer, and lung cancer in never smokers), with x ranging from 0.8 (endometrial cancer) to 1.7 (breast cancer). W/H was inversely associated with lung cancer in ever smokers for all values of x. Our results suggest that the optimal scaling of x may be below 2.0 and may differ for different cancer sites or, alternatively, that a range of scaling of weight for height may be equally informative. Further studies are needed to confirm and extend these findings in other, more diverse populations. Potential implications of our findings are that epidemiologists should consider using different weight-for-height scaling to examine the association of body size with risk of disease in different populations.

Table 3. Hazard Ratios and 95% Confidence Intervals for the Association of Selected Weight-for-Height Indices With Specific Cancer Sites in the Canadian National Breast Screening Study, 1980–2000

<table>
<thead>
<tr>
<th>Cancer Site and Variable</th>
<th>No. of Cases</th>
<th>Quintiles of Selected Weight-for-height Indices</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th>P for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>HR 95% CI</td>
<td>HR 95% CI</td>
<td>HR 95% CI</td>
<td>HR 95% CI</td>
<td>HR 95% CI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postmenopausal breast</td>
<td>2,356</td>
<td>1.00 1.07</td>
<td>0.92–1.24</td>
<td>1.26 1.10–1.45</td>
<td>1.33 1.16–1.53</td>
<td>1.43 1.24–1.64</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td></td>
<td>1.00 1.13</td>
<td>0.98–1.31</td>
<td>1.18 1.03–1.37</td>
<td>1.43 1.25–1.64</td>
<td>1.45 1.26–1.66</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>W/H</td>
<td></td>
<td>1.00 1.15</td>
<td>1.00–1.34</td>
<td>1.16 1.27–1.67</td>
<td>1.46 1.27–1.67</td>
<td>1.43 1.25–1.65</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>W/H1,4</td>
<td></td>
<td>1.00 1.19</td>
<td>1.04–1.37</td>
<td>1.15 1.00–1.33</td>
<td>1.42 1.24–1.62</td>
<td>1.44 1.26–1.66</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Endometriumb</td>
<td>795</td>
<td>1.00 0.99</td>
<td>0.75–1.30</td>
<td>1.12 0.86–1.46</td>
<td>1.39 1.08–1.78</td>
<td>2.92 2.33–3.67</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td></td>
<td>1.00 0.88</td>
<td>0.67–1.15</td>
<td>1.17 0.91–1.51</td>
<td>1.22 0.95–1.56</td>
<td>2.79 2.24–3.48</td>
<td>&lt;0.0001</td>
<td></td>
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<tr>
<td>W/H</td>
<td></td>
<td>1.00 0.87</td>
<td>0.67–1.14</td>
<td>1.11 0.86–1.43</td>
<td>1.28 1.00–1.63</td>
<td>2.68 2.15–3.33</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>W/H1,4</td>
<td></td>
<td>1.00 1.03</td>
<td>0.79–1.33</td>
<td>1.21 0.94–1.56</td>
<td>1.30 1.01–1.67</td>
<td>2.78 2.24–3.48</td>
<td>&lt;0.0001</td>
<td></td>
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<tr>
<td>Kidney</td>
<td>206</td>
<td>1.00 1.34</td>
<td>0.78–2.29</td>
<td>1.19 0.69–2.06</td>
<td>1.64 0.99–2.72</td>
<td>2.05 1.24–3.37</td>
<td>0.001</td>
<td></td>
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<tr>
<td>Weight</td>
<td></td>
<td>1.00 1.21</td>
<td>0.72–2.04</td>
<td>1.10 0.65–1.87</td>
<td>1.71 1.06–2.78</td>
<td>1.70 1.05–2.77</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>W/H</td>
<td></td>
<td>1.00 1.00</td>
<td>0.59–1.69</td>
<td>1.12 0.67–1.86</td>
<td>1.49 0.93–2.41</td>
<td>1.64 1.02–2.63</td>
<td>0.008</td>
<td></td>
</tr>
<tr>
<td>W/H1,4</td>
<td></td>
<td>1.00 1.03</td>
<td>0.56–1.56</td>
<td>0.92 0.55–1.53</td>
<td>1.54 0.97–2.43</td>
<td>1.43 0.89–2.28</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>Lung (ever smokers)</td>
<td>685</td>
<td>1.00 0.84</td>
<td>0.67–1.05</td>
<td>0.57 0.45–0.73</td>
<td>0.63 0.50–0.79</td>
<td>0.50 0.39–0.64</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Weight</td>
<td></td>
<td>1.00 0.80</td>
<td>0.64–1.00</td>
<td>0.56 0.44–0.72</td>
<td>0.58 0.46–0.74</td>
<td>0.53 0.41–0.67</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>W/H</td>
<td></td>
<td>1.00 0.73</td>
<td>0.59–0.92</td>
<td>0.61 0.48–0.78</td>
<td>0.50 0.39–0.64</td>
<td>0.54 0.42–0.68</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>W/H1,4</td>
<td></td>
<td>1.00 0.84</td>
<td>0.67–1.05</td>
<td>0.65 0.52–0.83</td>
<td>0.53 0.41–0.68</td>
<td>0.57 0.45–0.73</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Lung (never smokers)</td>
<td>104</td>
<td>1.00 0.98</td>
<td>0.46–2.09</td>
<td>1.08 0.52–2.25</td>
<td>1.37 0.69–2.71</td>
<td>1.98 1.03–3.80</td>
<td>0.007</td>
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<tr>
<td>Weight</td>
<td></td>
<td>1.00 1.16</td>
<td>0.54–2.47</td>
<td>1.49 0.73–3.05</td>
<td>1.36 0.66–2.82</td>
<td>2.13 1.09–4.17</td>
<td>0.01</td>
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</tr>
<tr>
<td>W/H</td>
<td></td>
<td>1.00 0.71</td>
<td>0.31–1.62</td>
<td>1.57 0.80–3.12</td>
<td>1.43 0.72–2.85</td>
<td>1.81 0.93–3.51</td>
<td>0.02</td>
<td></td>
</tr>
<tr>
<td>W/H1,4</td>
<td></td>
<td>1.00 1.26</td>
<td>0.56–2.85</td>
<td>2.07 0.99–4.34</td>
<td>1.88 0.89–3.98</td>
<td>2.02 0.96–4.23</td>
<td>0.07</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; HR, hazard ratio; W/H, weight for height.

a Covariates in the main model included age at entry (continuous), oral contraceptive use (ever vs. never), hormone use (ever vs. never), menopausal status (premenopausal, perimenopausal, postmenopausal, or unknown), years of education (<12, 12, or ≥12), and pack-years of smoking (never smoked, 1–9, 10–19, 20–29, 30–39, or ≥40). For postmenopausal breast cancer, the model additionally included age at menarche (<12, 12, 13, or ≥14 years), parity, family history of breast cancer in a first degree relative (yes vs. no), and history of benign breast disease (yes vs. no). The model for lung cancer in never smokers did not include pack-years.

b Analyses of endometrial cancer were restricted to women with an intact uterus.

ACKNOWLEDGMENTS

Author affiliations: Department of Epidemiology and Population Health, Albert Einstein College of Medicine, Bronx, NY (Geoffrey C. Kabat, Moonseong Heo, Thomas E. Rohan); and Dalla Lana School of Public Health, University of Toronto, Toronto, Canada (Anthony B. Miller).

This work was supported by institutional funds to the Albert Einstein College of Medicine. Dr. Rohan is supported by a grant from the Breast Cancer Research Foundation.

Conflict of interest: none declared.

REFERENCES


