Body-mass index and cancer mortality in the Asia-Pacific Cohort Studies Collaboration: pooled analyses of 424 519 participants

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Summary

Background **Excess bodyweight is an established risk factor for several types of cancer, but there are sparse data from Asian populations, where the proportion of overweight and obese individuals is increasing rapidly and adiposity can be substantially greater for the same body-mass index (BMI) compared with people from Western populations.**

Methods **We examined associations of adult BMI with cancer mortality (overall and for 20 cancer sites) in geographic populations from Asia and from Australia and New Zealand (ANZ), within the Asia-Pacific Cohort Studies Collaboration, by use of Cox regression analysis. Pooled data from 39 cohorts (recruitment 1961–99, median follow-up 4 years) were analysed for 424 519 participants (77% Asian; 41% female; mean recruitment age 48 years) with individual data on BMI.**

Findings **After excluding those with follow-up of less than 3 years, 4872 cancer deaths occurred in 401 215 participants. Hazard ratios for cancer sites with increased mortality risk in obese (BMI ≥30 kg/m²) compared with normal weight participants (BMI 18·5–24·9 kg/m²) were: 1·21 (95% CI 1·09–1·36) for all-cause cancer (excluding lung and upper aerodigestive tract), 1·50 (1·13–1·99) for colon, 1·68 (1·06–2·67) for rectum, 1·63 (1·13–2·35) for breast in women 60 years or older, 2·62 (1·57–4·37) for ovary, 4·21 (1·89–9·39) for cervix, 1·45 (0·97–2·19) for prostate, and 1·66 (1·03–2·68) for leukaemia (all after left censoring at 3 years). The increased risk associated with a 5-unit increase in BMI for those with BMI of 18·5 kg/m² or higher was 1·09 (95% CI 1·04–1·14) for all cancers (excluding lung and upper aerodigestive tract). There was little evidence of regional differences in relative risk of cancer with higher BMI, apart from cancers of the oropharynx and larynx, where the association was inverse in ANZ and absent in Asia.**

Interpretation **Overweight and obese individuals in populations across the Asia-Pacific region have a significantly increased risk of mortality from cancer. Strategies to prevent individuals from becoming overweight and obese in Asia are needed to reduce the burden of cancer that is expected if the obesity epidemic continues.**

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Introduction

More than 1 billion people worldwide are overweight or obese, conditions that are a main risk factor for chronic diseases, including some cancers.[1] Because of its widespread prevalence and effect on many health conditions, excess weight is ranked as the seventh most important contributor to mortality worldwide.[2] As in the west, many Asian countries such as China, South Korea, Thailand, and Singapore are experiencing a steep rise in the prevalence of obesity in their populations,[3] although compared with the west, the prevalence remains low.[3,4]

Excess weight and obesity are associated with increased risk of several site-specific cancers, with a 5 kg/m² higher body-mass index (BMI) typically associated with risk ratios in the range of 1·10–1·60.[5] The evidence linking excess weight and obesity to cancer has mainly come from Western populations, with few data from Asians,[6–8] in whom adiposity can be substantially greater for the same BMI.[9,10] Furthermore, the background prevalence of other dietary and lifestyle risk factors for cancer might also differ between Western and Asian populations,[11] which could affect the association between excess weight and subsequent cancer risk. Recent meta-analyses of Asian data, mainly from Japan or Hawaii, show that higher BMI is associated with increased risks of cancer of the female breast[5] and colorectum[12] in populations from Asia or the Asia-Pacific region, compared with North America, Europe, and Australia.

The present study, which uses data from the Asia-Pacific Cohort Studies Collaboration (APCSC), has two aims: to examine associations between BMI and site-specific cancer mortality in populations of the Asia-Pacific region, and to assess whether the magnitude and direction of the associations are consistent between geographic populations in Asia and outside Asia (Australia and New Zealand) which, to the best of our knowledge, has not been previously systematically examined using individual participant data.

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†Listed in webappendix

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Left censoring
Exclusion of study participants with follow-up time less than a certain duration

Methods

Procedures

The APCSC is a large, collaborative, data-pooling project involving data from more than 600 000 participants in the Asia-Pacific region. Details of the collaboration have been described elsewhere.[13,14] Briefly, studies were eligible for inclusion if they had the following criteria: a study population from the Asia-Pacific region; a prospective cohort study design; at least 5000 person-years of follow-up; date of birth (or age), sex, and blood pressure recorded at baseline; date of death or age at death recorded during follow-up. Studies were excluded if participant entry was dependent on a particular condition or risk factor. Studies were classified as Asian if participants were recruited from mainland China, Hong Kong, Taiwan, Japan, South Korea, Singapore, or Thailand, and as ANZ if participants were from Australia or New Zealand. We did not have individual-level data on ethnicity, but since immigration from Asia to ANZ is a recent occurrence and unlikely to have predated cohorts that were established from the mid-1960s to mid-1980s, it is unlikely that the ANZ cohorts would have included a substantial proportion of Asians. Since the APCSC is based on existing data, no ethics approval was needed for the present study.

BMI was calculated as weight in kilograms divided by height in metres squared (kg/m²). Baseline data on height and weight (or BMI) and at least one cancer event were available from 39 cohorts in the APCSC database. Of the 575 458 participants 20 years or older in these cohorts, 26% were excluded because of missing follow-up for cancer mortality (n=1062), missing BMI values (n=149 861), or a reported BMI of less than 12 kg/m² (n=9) or greater than 60 kg/m² (n=7). Thus, 424 519 participants with baseline data on age, sex, BMI, and cancer mortality were included in the present analysis.

Cancer mortality was classified according to the ninth or tenth revision of the International Classification of Diseases (ICD-9 or ICD-10). Older ICD-7 codes reported by some studies were recoded into version 9 or 10 by the project secretariat. Five small studies did not use ICD codes: Capitol Iron, Beijing Aging, EGAT, Tanno Sobetsu, and Aito Town. These studies contributed less than 5% of all cancer deaths (343 of 7211), most of which (>80%) were grouped into a category for other specified or unspecified sites and included in the analysis of all cancer, or included in specified categories by the project secretariat using all available information. Summary reports were referred back to principal investigators of each collaborating study for review and confirmation. Histological subtypes are not available in the APCSC.

Statistical analysis

Person-years were calculated for each eligible participant as the time from study recruitment until death from cancer, death from another cause, the last date they were known to be alive, or end of follow-up—whichever came first. Associations between BMI and cancer mortality were estimated with Cox proportional-hazards regression models stratified by study and, where appropriate, also by sex, with adjustment for age at baseline and using elapsed time since baseline measurement as the time scale. To investigate possible confounding by pre-existing disease and weight loss at baseline (ie, reverse causality), age-adjusted and smoking-adjusted hazard ratios (HRs) were estimated before and after 3 years **left censoring** of the data. Unless otherwise stated, results describe the left-censored data. We estimated HRs for four categories of BMI using WHO classification: BMI less than 18·5 kg/m² (underweight), 18·5–24·9 kg/m² (normal range), 25·0–29·9 kg/m² (overweight), and 30 kg/m² or higher (obese), using the normal range as the reference category. Since most participants were within the normal range, this category was further subdivided using the intermediate WHO cut points[15] of 18·5–22·9 kg/m² and 23·0–24·9 kg/m², to investigate changes in the shape of the risk pattern for five versus four categories of BMI. The 95% CI for categories of BMI were obtained by the method of floating absolute risks.[16]

In separate analyses, further adjustments were made for smoking status (not current *vs* current smoking) and drinking status (not current *vs* current alcohol consumption). Participants with missing values were assigned to a separate category for those variables. The main Cox model was adjusted for age at baseline and smoking status, with missing smoking data as a separate category. Attained age of less than 60 years, or 60 or more years, was taken as a proxy for menopausal status (no direct data were available) when estimating the risk of fatal breast cancer (adjustment for attained age and smoking status). This age cut-point is similar to that used in other studies of breast-cancer mortality[17] and was chosen to account for the relatively high survival[18,19] and the fact that few events occurred before age 50 years. After excluding the underweight category, we tested for a log-linear trend in HR across BMI categories, by fitting the main Cox model with BMI as a continuous variable, using three levels on an ordinal scale. To ease comparability with other studies, we calculated the overall effect of a 5 kg/m² increase in BMI, for BMI higher than 18·5 kg/m², for all cancer sites. We tested for regional differences in risk by adding interaction terms between region (Asia *vs* ANZ) and BMI to the Cox models for categorical (18·5–24·9 *vs* ≥25·0 kg/m²) and continuous (>18·5 kg/m²) effects after left censoring. Further interaction testing was limited to testing for sex differences to reduce the potential for type I errors. We also examined the effect of restricting analyses to participants with known values for all covariates; 94% of participants (n=400 277) had data on smoking status and 91% (n=388 109) had data on both smoking and drinking status. Since the effect was negligible (webtable 1), analyses were done for the entire sample. The main Cox model was adjusted for age at baseline and smoking status, with missing smoking data as a separate category, without additional adjustment for

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drinking status. All statistical analyses were done using SAS version 9.1.3 for Windows. Plots were created with R software. All tests were two-sided with statistical significance set at $p<0·05$.

Role of the funding source

The sponsors had no role in the conduct of the study or the writing of this report. CLP and FB had full access to the raw data as analysts. The corresponding author (CLP) had final responsibility to submit for publication.

Results

Individual participant data were included from 39 cohorts (webtable 2), consisting of 424 519 people, of whom 326 387 (77%) were from Asian cohorts and 175 364 (41%) were female; mean age was 48 years; and median year of recruitment was 1992 in cohorts from Asia and ANZ. Comparing baseline characteristics with the Asian cohorts, individuals from ANZ had higher BMI (mean 26·6 kg/m² [SD 3·7] in men and 26·0 kg/m² [4·9] in women *vs* 23·0 kg/m² [2·7] in men and 22·6 kg/m² [3·1] in women from Asia); were generally older; and had a lower proportion of current smokers in men (20% ANZ *vs* 60% Asia), but higher in women (14% ANZ *vs* 6% Asia). Selected baseline characteristics and cancer follow-up of study participants (before left censoring) by sex and categories of BMI are presented separately for Asia (table 1) and ANZ (table 2).

During the 2 738 020 person-years of follow-up (median 4 years), there were 7211 cancer deaths, of which 4130 (57%) occurred in Asian cohorts. The most common cancer site overall was lung (20%, n=1478), followed by stomach (12%, n=855), liver (11%, n=774), and large intestine (9%, n=668), but the distribution varied by sex and region (table 3). After excluding individuals with follow-up of less than 3 years, there were 401 215 study

	BMI missing	12·0–18·4 kg/m²	18·5–24·9 kg/m²	25·0–29·9 kg/m²	30·0–60·0 kg/m²
Males					
Number of participants	118 246	7209	143 292	42 947	1996
Number of cancer deaths	1008	287	2150	461	42
Person-years, follow-up	871 441	47 860	801 239	216 162	10 884
Age (years) at baseline, mean (SD)	41·9 (6·9)	52·3 (13·7)	46·5 (9·2)	46·7 (8·7)	49·4 (11·3)
BMI (kg/m²), mean (SD)	..	17·5 (0·9)	22·1 (1·7)	26·5 (1·2)	31·8 (2·6)
Weight (kg), mean (SD)	60·0 (9·0)*	48·3 (4·7)	62·3 (6·6)	75·1 (6·1)	88·8 (9·6)
Height (m), mean (SD)	1·69 (0·05)*	1·66 (0·07)	1·67 (0·06)	1·68 (0·06)	1·67 (0·07)
Smoking status (%)					
Not current smoker	40·7%	31·3%	36·7%	44·1%	47·0%
Current smoker	58·8%	66·1%	58·5%	50·5%	49·3%
Unknown	0·5%	2·6%	4·7%	5·4%	3·7%
Drinking status (%)					
Not current drinker	75·1%	44·5%	32·5%	30·3%	41·3%
Current drinker	24·2%	49·6%	61·6%	64·1%	54·2%
Unknown	0·7%	5·8%	6·0%	5·7%	4·5%
Females					
Number of participants	31058	8355	96814	22861	2913
Number of cancer deaths	149	156	780	224	30
Person-years, follow-up	239 421	54 565	550 748	131 004	17 027
Age (years) at baseline, mean (SD)	41·1 (7·3)	48·3 (14·6)	45·1 (10·1)	49·2 (10·5)	52·1 (11·6)
BMI (kg/m²), mean (SD)	..	17·4 (0·9)	21·8 (1·7)	26·6 (1·3)	32·3 (2·8)
Weight (kg), mean (SD)	54·0 (8·5)*	42·1 (4·3)	53·2 (5·6)	64·3 (5·6)	76·5 (8·8)
Height (m), mean (SD)	1·59 (0·05)*	1·55 (0·06)	1·56 (0·06)	1·55 (0·06)	1·54 (0·07)
Smoking status (%)					
Not current smoker	97·4%	80·6%	83·6%	83·4%	84·3%
Current smoker	1·5%	11·9%	4·4%	5·8%	10·6%
Unknown	1·1%	7·5%	11·9%	10·8%	5·0%
Drinking status (%)					
Not current drinker	95·0%	82·6%	80·1%	81·1%	86·0%
Current drinker	3·8%	7·6%	9·2%	8·6%	7·3%
Unknown	1·2%	9·8%	10·7%	10·3%	6·7%

BMI=Body-mass index. *Subsample of participants with missing BMI, but available data on either weight or height.

Table 1: **Baseline characteristics and cancer follow-up for Asian cohorts, by sex and categories of BMI**

	BMI missing	12·0–18·4 kg/m²	18·5–24·9 kg/m²	25·0–29·9 kg/m²	30·0–60·0 kg/m²
Males					
Number of participants	284	248	17 888	26 692	8883
Number of cancer deaths	38	15	671	965	300
Person-years, follow-up	3939	2019	167 895	214 069	63 895
Age (years) at baseline, mean (SD)	53·0 (20·6)	58·0 (19·0)	52·3 (17·0)	56·0 (14·4)	56·8 (13·5)
BMI (kg/m²), mean (SD)	..	17·4 (1·0)	23·0 (1·5)	27·2 (1·4)	32·6 (2·7)
Weight (kg), mean (SD)	76·3 (10·4)*	52·0 (5·9)	69·7 (7·2)	81·4 (7·6)	96·5 (11·5)
Height (m), mean (SD)	1·75 (0·08)*	1·72 (0·08)	1·74 (0·07)	1·73 (0·07)	1·72 (0·07)
Smoking status (%)					
Not current smoker	73·9%	62·1%	74·9%	82·0%	82·0%
Current smoker	23·9%	37·9%	25·0%	17·9%	17·9%
Unknown	2·1%	0·0%	0·1%	0·1%	0·1%
Drinking status (%)					
Not current drinker	37·0%	42·3%	37·4%	44·2%	47·0%
Current drinker	62·3%	54·0%	56·0%	51·2%	50·7%
Unknown	0·7%	3·6%	6·5%	4·6%	2·4%
Females					
Number of participants	273	661	20 967	14 698	8095
Number of cancer deaths	17	18	496	381	235
Person-years, follow-up	4173	7859	230 986	146 164	75 643
Age (years) at baseline, mean (SD)	51·8 (21·2)	45·7 (16·3)	48·1 (13·5)	53·8 (11·6)	54·4 (10·5)
BMI (kg/m²), mean (SD)	..	17·6 (0·8)	22·4 (1·6)	27·1 (1·4)	33·9 (3·7)
Weight (kg), mean (SD)	63·1 (11·9)*	46·6 (4·5)	58·8 (6·0)	69·4 (6·4)	85·1 (11·4)
Height (m), mean (SD)	1·62 (0·06)*	1·62 (0·07)	1·62 (0·06)	1·60 (0·06)	1·58 (0·07)
Smoking status (%)					
Not current smoker	74·0%	72·2%	83·2%	87·8%	89·7%
Current smoker	23·8%	27·5%	16·6%	12·1%	10·2%
Unknown	2·2%	0·3%	0·2%	0·1%	0·1%
Drinking status (%)					
Not current drinker	52·7%	51·3%	60·5%	73·5%	80·7%
Current drinker	45·8%	41·3%	34·3%	23·4%	17·9%
Unknown	1·5%	7·4%	5·2%	3·2%	1·4%

BMI=Body-mass index. *Subsample of participants with missing BMI, but available data on either weight or height.

Table 2: **Baseline characteristics and cancer follow-up for Australia and New Zealand cohorts, by sex and categories of BMI**

participants (95% of study sample) and 4872 cancer deaths, of which 309 263 (77%) participants and 2627 (54%) deaths were from Asian cohorts. Of the excluded cancer deaths (n=2339), 588 (25%) were from lung cancer.

Assumptions were met for the Cox proportional-hazards models when examining the association between BMI and cancer mortality in participants with BMI greater than 18·5 kg/m², for all cancers and for all site-specific cancers. In age and smoking-adjusted analyses, there was a U-shaped relation between BMI and mortality from all cancers combined; there was an increased risk of mortality in both the underweight (<18·5 kg/m²; HR 1·13, 95% CI 1·00–1·28) and obese categories (≥30 kg/m²; HR 1·11, 95% CI 1·00–1·23), compared with the normal weight reference group (18·5–24·9 kg/m²; table 4). However, the overall trend between BMI and all cancers in individuals with BMI 18·5 kg/m² or higher was weak and non-significant (HR 1·03, 95% CI 0·99–1·08; p_{trend}=0·32; table 4). This was due to the opposing effects of BMI with cancers of the lung and upper aerodigestive tract (UADT), which after excluding 1866 cases, resulted in a positive and significant association between BMI and all cancers in individuals with BMI of 18·5 kg/m² or higher: the HR per 5 kg/m² increase in BMI was 1·09 (95% CI 1·04–1·14; p_{trend}=0·003; table 4). In this analysis, overweight and obese individuals had a higher risk of mortality from cancer than did normal-weight participants: HR 1·06 (95% CI 1·00–1·12) and HR 1·21 (95% CI 1·09–1·36), respectively (table 4). We did a sensitivity analysis to examine the effect of the large Korean study (KMIC) on all cancers, and the results were not substantially changed by the exclusion of this study: HR per 5-unit increment in BMI for all cancers was 1·03 (95% CI 0·99–1·08) versus 1·05 (95% CI 1·00–1·10) after exclusion of KMIC.

In six of the 20 cancer sites examined—colon, rectum, breast in women aged 60 years or older, ovary, cervix, and

	All	Asia		Australia and New Zealand	
		Male	Female	Male	Female
All cancers (ICD code)*	7211 (100%)	2940 (100%)	1190 (100%)	1951 (100%)	1130 (100%)
UADT†	388 (5%)	207 (7%)	59 (5%)	99 (5%)	23 (2%)
Oesophagus (150/C15)	229 (3%)	117 (4%)	42 (4%)	52 (3%)	18 (2%)
Oropharynx (140-149/C00-C14) and larynx (161/C32)	159 (2%)	90 (3%)	17 (1%)	47 (2%)	5 (0.4%)
Stomach (151/C16)	855 (12%)	503 (17%)	222 (19%)	100 (5%)	30 (3%)
Large intestine (153-154/C18-21)†	668 (9%)	128 (4%)	85 (7%)	263 (13%)	192 (17%)
Colon (153/C18)	429 (6%)	52 (2%)	44 (4%)	183 (9%)	150 (13%)
Rectum (154/C18-20)	233 (3%)	73 (2%)	39 (3%)	79 (4%)	42 (4%)
Liver (155/C22)	774 (11%)	610 (21%)	108 (9%)	38 (2%)	18 (2%)
Pancreas (157/C25)	301 (4%)	110 (4%)	48 (4%)	84 (4%)	59 (5%)
Lung (162/C33-34)	1478 (20%)	706 (24%)	196 (16%)	442 (23%)	134 (12%)
Skin, malignant melanoma (172/C43)	88 (1%)	5 (0.2%)	0 (0%)	58 (3%)	25 (2%)
Breast, female (174/C50)	324 (4%)	(-)	98 (8%)	(-)	226 (20%)
Female genital organs (179-183/C53-56)‡	198 (3%)	(-)	98 (8%)	(-)	100 (9%)
Uterus (179,182/C55)	37 (1%)	(-)	17 (1%)	(-)	20 (2%)
Cervix (180/C53)	60 (1%)	(-)	45 (4%)	(-)	15 (1%)
Ovaries (183/C56)	99 (1%)	(-)	34 (3%)	(-)	65 (6%)
Prostate (185/C61)	278 (4%)	37 (1%)	(-)	241 (12%)	(-)
Bladder (188/C67)	120 (2%)	43 (1%)	6 (1%)	55 (3%)	16 (1%)
Kidney (189/C64-65)	93 (1%)	26 (1%)	6 (1%)	43 (2%)	18 (2%)
Brain and nervous system (191-192/C70-72)	191 (3%)	56 (2%)	22 (2%)	77 (4%)	36 (3%)
Haematological cancers	454 (6%)	113 (4%)	46 (4%)	186 (10%)	109 (10%)
Lymphoma (200-202/C81-85)	201 (3%)	50 (2%)	24 (2%)	81 (4%)	46 (4%)
Myeloma (203/C90)	69 (1%)	11 (0.4%)	6 (1%)	33 (2%)	19 (2%)
Leukaemia (204-208/C91-95)	184 (3%)	52 (2%)	16 (1%)	72 (4%)	44 (4%)
Other and unspecified sites	1001 (14%)	396 (13%)	196 (16%)	265 (14%)	144 (13%)

ICD codes refer to versions 9 and 10. ICD=International Classification of Diseases. UADT=upper aerodigestive tract. *Includes all specified sites and other and unspecified sites. †Includes the subcategories colon, rectum, four cases of anal cancer, and two cases with missing ICD code. ‡Includes the subcategories uterus, cervix, ovaries, one case of placenta cancer, and one case with missing ICD code.

Table 3: **Site-specific number of cancer deaths and percent distribution, by sex and within region (n=424 519)**

prostate—there was evidence of a significant log-linear trend in HRs with BMI after controlling for age and smoking status (table 4; all p_{trend} values <0·05). There was also a borderline significant trend for leukaemia (p_{trend}=0·09). Since the total number of leukaemia cases (n=129, left censored) was too small to do separate analyses, myeloid (71 cases, 55%) and lymphoid leukaemias (27 cases, 21%) were combined with other specified or unspecified cell types in this analysis. An increased risk was also evident in the overweight category (≥25–29·9 kg/m²) for rectum and prostate cancer (table 4), but not in the upper (23·0–24·9 kg/m²) compared with lower (18·5–22·9 kg/m²) end of the normal BMI range (figure 1).

For cancer sites where there was an inverse association between BMI and cancer (table 4), the reduced risk was significant for UADT cancers, including oropharynx and larynx (analysed together), and lung cancer, in the overweight compared with normal weight category. Since these cancers are mainly caused by smoking, and thereby to avoid the possibility of confounding (smokers tend to have lower BMI compared with never smokers), we also examined the association in never smokers (n=181 920, 68% women) compared with ever, ex, or current smokers (n=178 943, 11% women). Among those who had never smoked, the inverse association in the overweight category persisted for lung cancer (HR 0·42 [95% CI 0·27–0·65], p_{trend}=0·01 *vs* 0·67 [0·57–0·79], p_{trend}=0·004 in smokers; figure 2), but not for UADT overall (HR 1·17 [0·75–1·83], p_{trend}=0·91 *vs* 0·54 [0·37–0·79], p_{trend}=0·01) or for oropharynx and larynx cancers (HR 1·62 [0·91–2·89], p_{trend}=0·76 *vs* 0·36 [0·20–0·65], p_{trend}=0·005). All p values for trend are for BMI greater than 18·5 kg/m² (results by smoking status presented in webtable 1).

No clear associations were found between BMI and other cancer sites—ie, oesophagus, stomach, liver, pancreas, melanoma (skin), breast in all women or women younger than 60 years, bladder, kidney, uterus, brain and nervous system, lymphoma, myeloma, or all haematological cancers combined.

There was little evidence of a regional difference in the associations between cancer and BMI higher than 18·5 kg/m², except for oropharynx and larynx, where the

	BMI (kg/m²)				Trend ≥18·5 (kg/m²)	
	12·0–18·4 (HR [95% CI])	18·5–24·9 (HR [95% CI])	25·0–29·9 (HR [95% CI])	30·0–60·0 (HR [95% CI])	Per 5 units (HR [95% CI])	p_{trend}
All cancer	1·13 (1·00–1·28)	1·00 (0·96–1·04)	0·97 (0·91–1·02)	1·11 (1·00–1·23)	1·03 (0·99–1·08)	0·32
All cancer*	1·12 (0·96–1·31)	1·00 (0·96–1·04)	1·06 (1·00–1·12)	1·21 (1·09–1·36)	1·09 (1·04–1·14)	0·003
UADT	1·12 (0·70–1·81)	1·00 (0·85–1·17)	0·72 (0·55–0·96)	0·69 (0·38–1·24)	0·78 (0·62–0·98)	0·07
Oesophagus	1·26 (0·72–2·19)	1·00 (0·81–1·23)	0·79 (0·55–1·14)	0·80 (0·37–1·74)	0·87 (0·65–1·16)	0·36
Oropharynx–larynx	0·86 (0·34–2·19)	1·00 (0·78–1·28)	0·65 (0·43–0·98)	0·56 (0·23–1·41)	0·66 (0·46–0·95)	0·09
Stomach	1·19 (0·87–1·62)	1·00 (0·89–1·12)	1·05 (0·88–1·25)	1·04 (0·67–1·63)	1·10 (0·95–1·28)	0·66
Large intestine	0·73 (0·39–1·35)	1·00 (0·88–1·13)	1·25 (1·07–1·45)	1·57 (1·23–2·00)	1·15 (1·02–1·29)	0·001
Colon	0·63 (0·26–1·56)	1·00 (0·86–1·17)	1·13 (0·94–1·36)	1·50 (1·13–1·99)	1·14 (0·99–1·31)	0·02
Rectum	0·86 (0·37–2·02)	1·00 (0·80–1·25)	1·44 (1·11–1·86)	1·68 (1·06–2·67)	1·13 (0·91–1·40)	0·03
Liver	1·13 (0·78–1·64)	1·00 (0·89–1·13)	1·06 (0·87–1·30)	1·10 (0·63–1·91)	1·11 (0·94–1·31)	0·58
Pancreas	0·82 (0·41–1·63)	1·00 (0·84–1·19)	1·06 (0·83–1·36)	0·90 (0·54–1·49)	1·02 (0·83–1·25)	0·88
Lung	1·11 (0·86–1·44)	1·00 (0·92–1·09)	0·68 (0·59–0·79)	0·83 (0·64–1·08)	0·86 (0·77–0·96)	0·003
Skin, melanoma	3·21 (0·76–13·5)	1·00 (0·70–1·43)	0·68 (0·44–1·05)	1·03 (0·57–1·86)	1·13 (0·82–1·54)	0·77
Breast, female <60 years	0·27 (0·04–1·95)	1·00 (0·80–1·25)	1·13 (0·72–1·76)	0·93 (0·42–2·09)	1·13 (0·97–1·33)	0·84
Breast, female ≥60 years	0·71 (0·22–2·24)	1·00 (0·81–1·23)	1·13 (0·85–1·50)	1·63 (1·13–2·35)	1·19 (1·03–1·38)	0·03
Female genital organs	1·25 (0·62–2·49)	1·00 (0·79–1·26)	1·41 (1·05–1·90)	2·65 (1·77–3·98)	1·37 (1·15–1·64)	0·0002
Ovaries	..	1·00 (0·72–1·39)	1·46 (0·98–2·16)	2·62 (1·57–4·37)	1·38 (1·09–1·73)	0·003
Uterus	1·76 (0·40–7·65)	1·00 (0·58–1·72)	1·45 (0·75–2·80)	1·41 (0·42–4·67)	1·31 (0·83–2·06)	0·44
Cervix	2·11 (0·93–4·77)	1·00 (0·65–1·55)	1·29 (0·68–2·46)	4·21 (1·89–9·39)	1·45 (1·00–2·11)	0·02
Prostate	1·13 (0·44–2·90)	1·00 (0·82–1·23)	1·41 (1·14–1·74)	1·45 (0·97–2·19)	1·18 (0·96–1·44)	0·046
Bladder	1·39 (0·51–3·76)	1·00 (0·75–1·34)	1·11 (0·75–1·65)	0·72 (0·29–1·79)	1·09 (0·78–1·54)	0·87
Kidney	1·17 (0·28–4·97)	1·00 (0·70–1·43)	1·42 (0·96–2·12)	1·59 (0·78–3·24)	1·20 (0·86–1·66)	0·19
Brain and nervous system	1·32 (0·56–3·07)	1·00 (0·80–1·25)	0·95 (0·70–1·28)	0·99 (0·57–1·73)	0·97 (0·75–1·24)	0·89
Haematological cancers	1·21 (0·64–2·27)	1·00 (0·85–1·17)	1·18 (0·97–1·43)	1·28 (0·93–1·78)	1·15 (0·99–1·34)	0·12
Lymphoma	1·29 (0·51–3·30)	1·00 (0·78–1·29)	1·35 (1·00–1·81)	0·97 (0·54–1·73)	1·07 (0·84–1·36)	0·57
Myeloma	1·94 (0·57–6·68)	1·00 (0·70–1·43)	0·87 (0·54–1·41)	1·20 (0·59–2·43)	1·05 (0·73–1·50)	0·78
Leukaemia	0·82 (0·25–2·67)	1·00 (0·78–1·28)	1·18 (0·87–1·60)	1·66 (1·03–2·68)	1·29 (1·03–1·61)	0·09

Models stratified by study and sex. Uncensored data presented in webtable 3. Number of events in each BMI category see webtable 4. BMI=body-mass index. UADT=upper aerodigestive tract. ..Too few or no events. *All cancers after excluding lung and UADT.

***Table 4*: Age- and smoking-adjusted HRs for cancer mortality according to BMI after left censoring at 3 years (n = 401 215)**

association was inverse in ANZ but absent in Asia (table 5): HR for a 5 kg/m² increase in BMI was 0·46 (95% CI 0·27–0·77) in ANZ, compared with 0·99 (0·60–1·62) in Asia (p_{region}=0·04). The association with lung cancer was similar: HR 0·86 (0·75–0·99) in ANZ compared with 0·85 (0·71–1·02) in Asia (p_{region}=0·95) per 5 kg/m².

For all cancers and subsites positively associated with BMI in the main analysis, the region-specific HRs for ANZ (table 5) for categorical and continuous effects were similar (large intestine, prostate), or slightly strengthened (female genital organs, leukaemia), compared with the overall, left-censored HRs (table 4). The HRs for Asia were generally lower and non-significant, with wider CIs compared with ANZ. After subdividing the normal range, there was little evidence of increased cancer risk in Asia compared with ANZ in the upper BMI range (23·0–24·9 kg/m²), but there were few subsites with a sufficient number of events in the reference category (18·5–22·9 kg/m²) for both regions (webtable 5). Results are only shown for cancer of the large intestine (figure 3). Breast cancer by age group was not further stratified by region, because of small numbers in Asia.

In the left-censored data there was little evidence of a sex difference in the observed BMI-cancer associations (webtable 6). A sex difference was found for cancer of the large intestine, but only in the categorical analysis (p_{sex}=0·06): HR for mortality among the obese compared with normal-weight participants was 1·99 (95% CI 1·45–2·73) in men, compared with 1·28 (0·91–1·78) in women.

Discussion

In the present study, based on data from more than 400 000 individuals from 39 prospective cohort studies in the Asia-Pacific region, we found that among individuals with a BMI higher than 18·5 kg/m², there was a positive and continuous association between BMI and all-cancer mortality, such that a 5 kg/m² increase in BMI was associated with a relative risk (HR) of 1·09 (95% CI 1·04–1·14), after excluding cases of lung and UADT

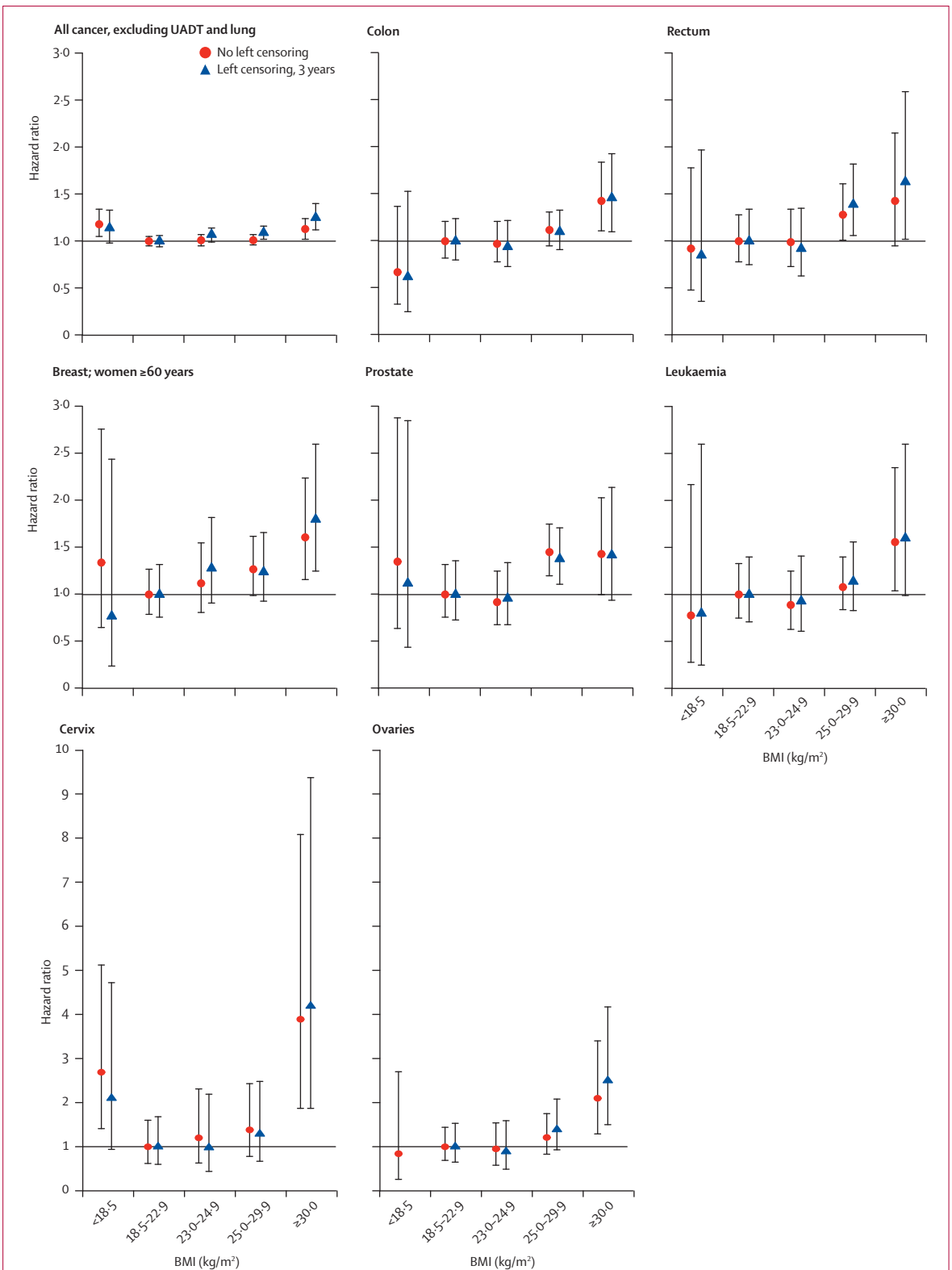

Figure 1: **Age- and smoking-adjusted hazard ratios with 95% CI for cancer mortality before and after left censoring at 3 years according to categories of body-mass index**
Models stratified by study and sex. UADT=upper aerodigestive tract.

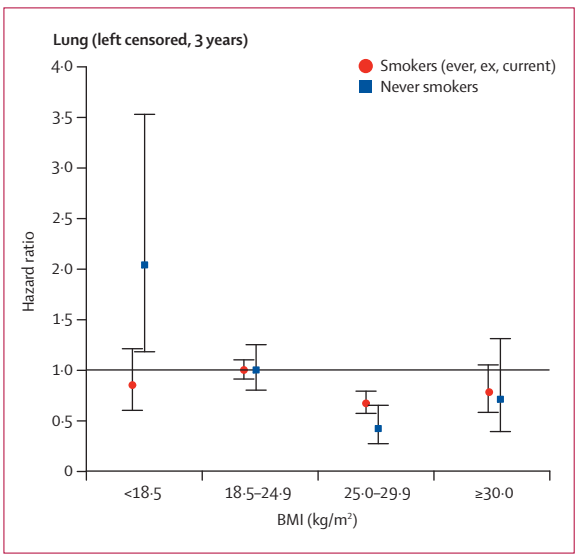

Figure 2: **Age-adjusted hazard ratios with 95% CI for lung cancer mortality in cigarette smokers (ever, ex, or current) compared to never smokers after left censoring at 3 years according to categories of body-mass index**
Models stratified by study and sex.

cancer, which had a negative association with BMI. Compared with individuals of normal weight, the relative risk of dying from cancer was 1·06 (95% CI 1·00–1·12) for those who were overweight and 1·21 (95% CI 1·09–1·36) for those who were obese. The positive association between BMI and all-cancer mortality was mainly driven by cancers of the large intestine, breast (in women older than 60 years), ovary, cervix, prostate, and leukaemia. Of particular note and by contrast with previous findings,[5,12] there was little evidence of regional differences in relative risks between cohorts from Asia and ANZ. This regional consistency in the magnitude of association between excess weight and cancer is consistent with previous APCSC findings, which have shown that associations between many cardiovascular risk factors and cardiovascular disease are broadly similar.[20] The effect sizes (per 5 kg/m² or the obese category) for colon, rectal, breast (in older women), and prostate cancer are largely consistent with previous reports from other large studies of cancer mortality in mainly Western populations, such as the Million Women Study from the UK[21] and the Prospective Studies Collaboration (PSC).[17]

This study substantiates that obesity increases the risk of prostate-cancer mortality, by contrast with studies of incidence, which have reported conflicting findings on the association between BMI and prostate cancer.[22] Evidence suggests that obesity increases the risk of aggressive disease, recurrence, and subsequent death, which might be partly explained by late detection and surgical difficulties during radical prostatectomy.[23] Our study also contributes to the growing evidence that excess weight is associated with increased risk of several haematological cancers.[17,21,24,25] For example, by contrast with the PSC study,[17] but consistent with studies of UK women[21] and US men,[24] we observed an association between BMI and mortality from leukaemia. We did not observe a significant association between BMI and mortality from uterine cancer, which is considered to be strongly related to obesity, possibly because of the small number of events in our study (n=37). However, we did find positive and significant associations between BMI and cancers of the ovaries and cervix, associations that are inconsistent in the literature.[5,7,17,21,24,26,27]

The inverse associations we found between BMI and cancers of the UADT (mainly oropharynx and laynx) and lung are a commonly reported finding.[5,7,8,21] Some reassurance that these associations are unlikely to be due to reverse causality (as a result of only excluding the first 3 years of follow-up) comes from a recent study from the PSC,[17] which reported similar inverse associations after exclusion of the first 10 years of follow-up. By contrast with most studies of BMI and lung cancer in Asian[7,8] and Western populations,[17,21,24] the association remained negative in non-smokers. One possibility is that causes of high background mortality from lung cancer in countries such as China (eg, caused by indoor fuel-use patterns[28]) might be socioeconomically correlated to BMI, but this is speculative.

Our study did not support some widely reported associations between BMI and specific cancer sites, including oesophagus, pancreas, and kidney. Some possible explanations include lack of histological data to distinguish between oesophageal adenocarcinoma and squamous-cell carcinoma, for which BMI might have opposite effects;[5,21] a potentially stronger effect of central obesity than of BMI on pancreatic cancer;[29] and few renal cancer events.

Our study provided many cases of stomach and liver cancer compared with studies from mainly Western populations.[17,30] Our results did not substantiate previous reports of positive associations with excess bodyweight,[7,17,30] possibly indicating that most stomach cancers are of the non-cardia type, for which *Helicobacter pylori* infection and intake of salt or salted foods[18] are more important risk factors than obesity, which might be more strongly related to adenocarcinoma of the gastric cardia (gastro-oesophageal junction).[31] Similarly, infection with the hepatitis B or C viruses, which causes more than 75% of liver cancer cases worldwide,[18] could be the main risk factor in Asia.

The observed BMI-cancer associations were generally consistent across Asia and ANZ, except for oropharynx and laynx. The association was inverse in ANZ, but absent in Asia, which could be a chance finding, or might reflect the early stage of the smoking epidemic in large parts of Asia,[28,32,33] since no association was found in never smokers. However, a regional difference due to smoking was not found for lung cancer, possibly because the inverse association with BMI persisted in never smokers. Although smoking is a common risk factor for cancers of

the oropharynx, larynx, and lung, their aetiologies also differ—eg, for laryngeal cancer there is a multiplicative effect between smoking and alcohol consumption[18]—and cancers of the oropharynx might be related to human papillomavirus infection.[34] Thus, risk factors other than smoking, if associated with BMI, might explain the discrepant regional difference for oropharynx and larynx, and lung cancer.

	BMI (kg/m²)				Trend >18·5 (kg/m²)		
	18·5–24·9 (HR [95% CI])	25·0–29·9 (HR [95% CI])	30·0–60·0 (HR [95% CI])	<25 vs ≥25 (p_{region}*)	Per 5 units HR (95% CI)	p_{region}*	p_{trend}
All cancer							
ANZ	1·00 (0·93–1·07)	0·96 (0·90–1·03)	1·12 (1·01–1·24)	0·80	1·05 (1·00–1·11)	0·29	0·20
Asia	1·00 (0·95–1·06)	0·98 (0·89–1·07)	1·07 (0·81–1·43)		1·00 (0·93–1·08)		0·93
All cancer†							
ANZ	1·00 (0·92–1·08)	1·08 (1·01–1·16)	1·23 (1·10–1·37)	0·43	1·10 (1·04–1·17)	0·39	0·004
Asia	1·00 (0·94–1·07)	1·04 (0·94–1·15)	1·20 (0·87–1·65)		1·06 (0·97–1·15)		0·29
UADT							
ANZ	1·00 (0·72–1·38)	0·58 (0·40–0·82)	0·61 (0·33–1·14)	0·16	0·69 (0·50–0·95)	0·28	0·046
Asia	1·00 (0·80–1·26)	0·93 (0·63–1·39)	0·76 (0·19–3·06)‡		0·88 (0·64–1·21)		0·66
Oesophagus							
ANZ	1·00 (0·64–1·57)	0·72 (0·44–1·17)	0·84 (0·37–1·89)	0·75	0·92 (0·61–1·39)	0·69	0·49
Asia	1·00 (0·74–1·35)	0·89 (0·52–1·51)	0·56 (0·08–4·03)‡		0·82 (0·54–1·24)		0·53
Oropharynx–larynx							
ANZ	1·00 (0·63–1·58)	0·45 (0·27–0·76)	0·42 (0·16–1·13)‡	0·08	0·46 (0·27–0·77)	0·04	0·03
Asia	1·00 (0·71–1·42)	1·01 (0·55–1·85)	1·15 (0·16–8·35)‡		0·99 (0·60–1·62)		0·93
Stomach							
ANZ	1·00 (0·71–1·40)	0·89 (0·66–1·20)	0·99 (0·60–1·63)	0·45	1·12 (0·87–1·45)	0·87	0·85
Asia	1·00 (0·88–1·14)	1·11 (0·90–1·39)	1·01 (0·45–2·26)		1·09 (0·91–1·31)		0·49
Large intestine							
ANZ	1·00 (0·83–1·21)	1·27 (1·09–1·48)	1·55 (1·23–1·96)	0·82	1·16 (1·02–1·32)	0·62	0·004
Asia	1·00 (0·80–1·26)	1·18 (0·83–1·67)	2·21 (0·97–5·03)		1·07 (0·80–1·44)		0·11
Liver							
ANZ	1·00 (0·58–1·73)	1·18 (0·76–1·85)	0·97 (0·43–2·19)	0·87	1·04 (0·70–1·53)	0·70	0·93
Asia	1·00 (0·87–1·15)	1·04 (0·84–1·30)	1·27 (0·63–2·56)		1·13 (0·93–1·36)		0·56
Pancreas							
ANZ	1·00 (0·72–1·38)	1·15 (0·88–1·51)	0·88 (0·53–1·47)	0·70	1·06 (0·84–1·34)	0·55	0·89
Asia	1·00 (0·77–1·30)	0·91 (0·57–1·48)	1·35 (0·33–5·51)‡		0·93 (0·63–1·35)		0·95
Lung							
ANZ	1·00 (0·86–1·17)	0·65 (0·54–0·77)	0·84 (0·64–1·09)	0·57	0·86 (0·75–0·99)	0·95	0·02
Asia	1·00 (0·88–1·14)	0·76 (0·60–0·97)	0·78 (0·37–1·65)		0·85 (0·71–1·02)		0·06
Skin, melanoma							
ANZ	1·00 (0·67–1·49)	0·58 (0·38–0·90)	0·97 (0·55–1·73)	..	1·08 (0·78–1·49)	0·10	0·57
Asia	..	..	..		..		..
Breast, female							
ANZ	1·00 (0·79–1·27)	0·99 (0·77–1·28)	1·26 (0·90–1·74)	0·67	1·10 (0·94–1·29)	0·78	0·35
Asia	1·00 (0·71–1·40)	0·88 (0·49–1·57)	1·28 (0·31–5·29)‡		1·03 (0·66–1·60)		0·93
Female genital organs							
ANZ	1·00 (0·67–1·48)	1·56 (1·11–2·21)	2·83 (1·92–4·16)	0·27	1·43 (1·17–1·74)	0·38	0·0003
Asia	1·00 (0·70–1·43)	1·17 (0·67–2·02)	2·09 (0·65–6·70)‡		1·16 (0·76–1·77)		0·30
Prostate							
ANZ	1·00 (0·76–1·32)	1·42 (1·18–1·71)	1·42 (0·96–2·11)	0·99	1·14 (0·92–1·41)	0·31	0·07
Asia	1·00 (0·60–1·66)	1·21 (0·45–3·26)‡	3·89 (0·53–28·3)‡		1·58 (0·87–2·87)		0·34
Bladder							
ANZ	1·00 (0·64–1·56)	0·95 (0·63–1·41)	0·69 (0·29–1·69)‡	0·16	1·08 (0·74–1·57)	0·86	0·53
Asia	1·00 (0·54–1·85)	2·18 (1·01–4·71)	..		1·17 (0·52–2·60)		0·24

(Continues on next page)

	BMI (kg/m²)				Trend >18·5 (kg/m²)		
	18·5–24·9 (HR [95% CI])	25·0–29·9 (HR [95% CI])	30·0–60·0 (HR [95% CI])	<25 vs ≥25 (p_{region}*)	Per 5 units HR (95% CI)	p_{region}*	p_{trend}
(Continued from previous page)							
Kidney							
ANZ	1·00 (0·58–1·71)	1·42 (0·95–2·12)	1·26 (0·59–2·69)	0·80	1·12 (0·77–1·62)	0·38	0·47
Asia	1·00 (0·54–1·85)	1·20 (0·46–3·13)‡	6·38 (1·52–26·9)‡		1·56 (0·81–3·01)		0·11
Brain and nervous system							
ANZ	1·00 (0·70–1·43)	0·86 (0·62–1·21)	1·04 (0·61–1·78)	0·71	1·02 (0·77–1·36)	0·43	0·94
Asia	1·00 (0·70–1·44)	1·15 (0·66–2·01)	..		0·80 (0·47–1·37)		0·88
Haematological cancers							
ANZ	1·00 (0·78–1·28)	1·21 (0·99–1·47)	1·27 (0·93–1·74)	0·94	1·11 (0·94–1·30)	0·20	0·19
Asia	1·00 (0·74–1·35)	1·12 (0·71–1·78)	2·02 (0·63–6·52)‡		1·41 (1·01–1·99)		0·35
Lymphoma							
ANZ	1·00 (0·68–1·48)	1·32 (0·98–1·78)	0·94 (0·53–1·68)	0·63	1·00 (0·76–1·31)	0·26	0·85
Asia	1·00 (0·60–1·65)	1·53 (0·82–2·84)	1·52 (0·20–11·4)‡		1·39 (0·83–2·31)		0·32
Myeloma							
ANZ	1·00 (0·61–1·63)	0·73 (0·45–1·19)	1·00 (0·50–2·03)	0·15	0·92 (0·61–1·37)	0·07	0·80
Asia	1·00 (0·46–2·19)	1·88 (0·64–5·54)‡	5·55 (0·67–46·0)‡		2·13 (0·93–4·90)		0·12
Leukaemia							
ANZ	1·00 (0·66–1·51)	1·43 (1·06–1·94)	1·85 (1·18–2·91)	0·14	1·30 (1·02–1·66)	0·81	0·04
Asia	1·00 (0·67–1·50)	0·65 (0·27–1·54)‡	1·64 (0·23–11·9)‡		1·21 (0·68–2·14)		0·62

Models stratified by study and sex. Number of events in each BMI category see webtable 5. BMI=body-mass index. ANZ=Australia and New Zealand. Asia=mainland China, Hong Kong, Taiwan, Japan, South Korea, Singapore, and Thailand. UADT=upper aerodigestive tract. ..=Too few or no events. *p value of interaction term. †All cancers excluding lung and UADT. ‡HR based on five or fewer events.

***Table 5*: Region-specific age- and smoking-adjusted HRs for cancer mortality according to BMI after left censoring at 3 years (n=401 215)**

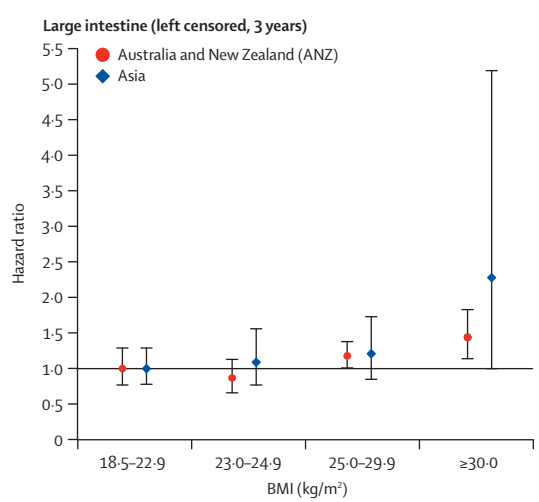

***Figure 3*: Age- and smoking-adjusted hazard ratios with 95% CI for mortality from colorectal cancer by region after left censoring at 3 years according to categories of body-mass index**
Models stratified by study and sex.

In the present study, which included 31 cohorts from Asia, we did not find a higher relative risk of mortality from colorectal cancer in Asians within the normal BMI range (23·0–24·9 kg/m²), as was reported by Ning and colleagues[12] in a recent meta-analysis of prospective (incidence and mortality) and retrospective studies from ten Asian populations. A systematic review and meta-analysis of BMI and cancer incidence by Renehan and co-workers[5] showed similar estimates for colon-cancer risk in the Asia-Pacific region and North America, but possibly higher than in Europe and Australia.[5] However, the association between BMI and breast cancers in women was stronger in the Asia-Pacific region (five studies) than in North America, Europe, and Australia.[5] We did not have enough events to analyse breast cancer by age group and region—only about 25% of breast cancers occurred in Asian populations—but there was no evidence of a difference between regions in the overall analysis of fatal breast cancer.

There is still a paucity of data on BMI and cancer risk from Asian populations, and several challenges for comparing estimates across regions. Current studies of BMI and cancer risk are mainly from Japan,[5] where national screening programmes for colorectal and female breast cancer from age 40 years[35] might inflate incidence rates in younger age groups with lower BMI. In studies of cancer mortality, a higher risk in Asian compared with Western populations for the same BMI range could also reflect poorer survival, at least for cancers where early diagnosis or therapy (or both) can markedly affect outcomes, such as large intestine and female breast.[18]

The main strengths of the present study are its prospective design and large sample size, with more

than three-quarters of participants from Asian populations, for which data on BMI-cancer associations remain very limited. Our results support that the APCSC data on observed regional distribution of cancer sites are in accordance with global cancer statistics, such as a lower proportion of cancers associated with western lifestyles, including large intestine and prostate, but a higher proportion of cancers of the stomach and liver in Asia compared with ANZ.[18] However, the large regional difference in cancer occurrence and BMI level made regional comparisons difficult, with few events and very wide CI in Asia for cancers that are most common in Europe and North America, in particular among the obese.

We were also unable to examine whether associations of cancer mortality with other measures of obesity, such as waist circumference or waist-to-hip ratio, differ across ethnic groups. There has been considerable speculation regarding potential differences in the magnitude of associations between obesity and cardiovascular disease, with some, but not all studies,[36] suggesting that associations are steeper in Asians than in people from Western populations, possibly because Asians have a higher percent body fat per level of BMI and a greater propensity towards central obesity (which is metabolically more unfavourable than global obesity as measured by BMI).[37] In the present study, there was little evidence of regional difference in the magnitude of the associations between BMI and cancer mortality: the test for regional interaction was non-significant for all but one (oropharynx and larynx) of the 20 subsites of cancer examined. However, given that the test for interaction has low power to detect a difference, there is a high probability of type II error.

Other weaknesses of the APCSC relate to non-standardisation of the data collection. For smoking and drinking status, the not-current category was included in several cohorts and therefore used in the analysis to preserve events, although these variables might only provide a crude adjustment. We addressed the issue of residual confounding by smoking by repeating the analyses of cancer sites inversely associated with BMI in never smokers. The APCSC database does not have data on histological subtypes of cancer and there is limited and non-systematic information on socioeconomic status, a parameter that incorporates differences in a wide array of potential confounders, including dietary factors and physical activity. Moreover, we had no information on hormone-replacement therapy or menopausal status in women. Thus, the results should be interpreted with caution. Since a quarter of the cohort were excluded because of missing values for BMI, selection bias might have distorted the observed associations; however, this is unlikely to be a significant source of error since our main findings largely agree with estimates from other meta-analyses and large prospective studies. Finally, many of the cohorts included are occupational or from non-random samples of the population; however, this is unlikely to effect our results or conclusions since the main purpose of the paper was to report on the magnitude (rather than prevalence) of associations between BMI and cancer mortality, which are unlikely to differ between populations from occupational cohorts or nationally representative studies.

Because of the short follow-up, only baseline data on BMI were used. Previous calculations based on APCSC studies with repeated measurements show that BMI has a regression-dilution coefficient close to one, so the current results were not adjusted.[14] Similarly, because of the short follow-up we were only able to left censor the data by 3 years, which might have been an insufficient length of time to exclude all underlying cancers (particularly those with a long latency period) and hence the possibility of reverse causality remains. However, some reassurance is gained by noting comparable results with studies where data were censored by 10 years.[17]

Many countries of the Asia-Pacific region show a steep increase in overweight and obese people in their populations, mainly as a result of a shift from traditional to more western lifestyles, characterised by excess energy consumption, reduced physical activity, and more affluent standards of living.[4] Effective strategies to prevent the increasing proportions of overweight and obese people in Asian populations need to be developed and assessed to reduce the burden of cancer that can be expected if the obesity epidemic continues. However, this study does not show a higher relative risk for cancer mortality in Asian populations compared with Western populations for the same level of BMI, as has been suggested for diabetes and cardiovascular disease.[15]

Contributors

CLP did the statistical analyses, prepared tables and figures, and drafted the manuscript. All coauthors provided comments and suggestions on the manuscript and approved the final version. FB contributed to the statistical analyses and GDB advised on epidemiological aspects. THL, XF, SCH, SHJ, KJ (deceased), and HU are principal collaborators of contributing cohort studies from the region, and provided data for the present study. RRH was responsible for initiation and conduct of the current study and MW was the overall coordinator.

Conflicts of interest

The authors declared no conflicts of interest.

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