Height and cancer incidence in the Million Women Study: prospective cohort, and meta-analysis of prospective studies of height and total cancer risk

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Summary
Background Epidemiological studies have shown that taller people are at increased risk of cancer, but it is unclear if height-associated risks vary by cancer site, or by other factors such as smoking and socioeconomic status. Our aim was to investigate these associations in a large UK prospective cohort with sufficient information on incident cancer to allow direct comparison of height-associated risk across cancer sites and in relation to major potential confounding and modifying factors.

Methods Information on height and other factors relevant for cancer was obtained in 1996–2001 for middle-aged women without previous cancer who were followed up for cancer incidence. We used Cox regression models to calculate adjusted relative risks (RRs) per 10 cm increase in measured height for total incident cancer and for 17 specific cancer sites, taking attained age as the underlying time variable. We also did a meta-analysis of published results from prospective studies of total cancer risk in relation to height.

Findings 1297 124 women included in our analysis were followed up for a total of 11·7 million person-years (median 9·4 years per woman, IQR 8·4–10·2), during which time 97 376 incident cancers occurred. The RR for total cancer was of 1·16 (95% CI 1·14–1·17; p<0·0001) for every 10 cm increase in height. Risk increased for 15 of the 17 cancer sites we assessed, and was statistically significant for ten sites: colon (RR per 10 cm increase in height 1·25, 95% CI 1·19–1·30), rectum (1·14, 1·07–1·22), malignant melanoma (1·32, 1·24–1·40), breast (1·17, 1·15–1·19), endometrium (1·19, 1·13–1·24), ovary (1·17, 1·11–1·23), kidney (1·29, 1·19–1·41), CNS (1·20, 1·12–1·29), non-Hodgkin lymphoma (1·21, 1·14–1·29), and leukaemia (1·26, 1·15–1·38). The increase in total cancer RR per 10 cm increase in height did not vary significantly by socioeconomic status or by ten other personal characteristics we assessed, but was significantly lower in current than in never smokers (p<0·0001). In current smokers, smoking-related cancers were not as strongly related to height as were other cancers (RR per 10 cm increase in height 1·05, 95% CI 1·01–1·09, and 1·17, 1·13–1·22, respectively; p=0·0004). In a meta-analysis of our study and ten other prospective studies, height-associated RRs for total cancer showed little variation across Europe, North America, Australasia, and Asia.

Interpretation Cancer incidence increases with increasing adult height for most cancer sites. The relation between height and total cancer RR is similar in different populations.

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Introduction Tall people are at increased risk of cancer. Increasing cancer risk with increasing adult height has been reported for all cancers combined and for several common cancers, such as those of the breast, ovary, prostate, and large bowel. Evidence is limited, however, for incident, rather than fatal, disease and for less common cancer sites. Moreover, it is not clear to what extent height-associated risks vary by cancer site, or how other factors, such as smoking and socioeconomic status, affect these associations. Because the range of height in a given population is usually narrow, large numbers of events are needed for reliable estimation of risk. Therefore we report here on the relation between height and cancer incidence in a prospective cohort study of more than 1 million middle-aged women in the UK. We also did a meta-analysis of published results from prospective studies on the relation between height and total cancer incidence or mortality.

Methods
Participants Between 1996 and 2001, 1·3 million middle-aged women invited to attend the UK’s National Health Service (NHS) Breast Screening Programme completed a Million Women Study recruitment questionnaire, which asked, among other things, about social, demographic, and lifestyle factors, including current height and weight. Of women who answered a study questionnaire in 2006–07, a sample selected at random (on the basis of day of birth) were asked in 2006–09 to have their height measured by their family doctor: 3762 women did so. In this validation sample, the correlation between measured and reported heights was excellent (Pearson correlation coefficient 0·88).
All participants gave written consent to take part in our study, and approval was obtained from the Oxford and Anglia Multi-Centre Research Ethics Committee. All study participants have a unique NHS number and are automatically followed up for death, emigration, and cancer registration through the NHS central registers with that number and other identifying details. The registers regularly provide study investigators with information on the date of any such event in participants, and code the underlying cause of death and cancer site with the International Classification of Diseases, 10th revision (ICD-10). Follow-up is complete for over 99% of study participants.

**Procedures**

Our main endpoints were incident invasive cancer at 17 individual sites with at least 1000 incident cases: mouth and pharynx (ICD-10 C00-C14), oesophagus (C15), stomach (C16), colon (C18), rectum (C19-20), pancreas (C25), lung (C34), malignant melanoma (C43), breast (C50), endometrium (C54), ovary (C56), kidney (C64), bladder (C67), central nervous system (C70-72, D32, 33, 42, and 43), non-Hodgkin lymphoma (C82-85), multiple myeloma (C90), and leukaemia (C91-95). We included all other invasive cancers (the remaining ICD-10 C codes, except non-melanoma skin cancer [C44]) as “other and unspecified” cancers.

We defined smoking-related cancers as those for which we defied smoking-related cancers as those for which the International Agency for Research on Cancer (IARC) has concluded there is sufficient evidence of carcinogenicity in human beings in relation to active tobacco smoking, of the sites listed above, mouth and pharynx, oesophagus, stomach, colorectum, pancreas, lung, mucinous tumours of the ovary, kidney, and myeloid leukaemia (C92), and, additionally, liver (C22), larynx, nasal cavity and nasal sinus (C30-32), cervix (C53), and urinary tract, including renal pelvis and ureter (C65, 66, 68). When comparing smoking-related and other cancers, we excluded from our analysis cancers of ill-defined and unspecified sites, which might include some smoking-related cancers (ICD-10 C26, C39, C57, C76-80 and C95-96), and cancers of the ovary (for a substantial proportion of which histological subtype was not known, and which might have included mucinous tumours).

Height was reported by participants at recruitment in feet and inches, and converted to centimetres for our analysis. For the analyses, women were divided into six categories of reported height (<155 cm [reference group], 155–159·9 cm, 160–164·9 cm, 165–169·9 cm, 170–174·9 cm, and 175 cm and taller); we took the average mean measured height in that category to be the mean measured height in that category in the sample whose height was measured in 2006–09. Where appropriate, mean measured heights are reported standardised to the distribution of self-reported heights within the whole population, or relevant subgroup.

We excluded women from our analyses if they had any type of cancer other than non-melanoma skin cancer. Table 1 presents characteristics at recruitment and follow-up for cancer incidence in the Million Women Study.

| Height in cm | All women |
|-------------|-----------|
| <155        | 52·8 (4·1) |
| 155         | 56·5 (2·3) |
| 160         | 160·4 (2·9) |
| 165         | 164·9 (2·9) |
| 170         | 169·0 (2·9) |
| ≥175        | 173·8 (4·3) |

The categories of height are those reported at recruitment, and mean values are those measured in a randomly selected sample. Standardised to the distribution of categories of self-reported height in our whole analysis population.

**Table 1: Baseline characteristics by height and follow-up for incident cancer in the Million Women Study**

![Table 1](https://www.thelancet.com/oncology/Vol12/figure3.png)
cancer (ICD10 C44) registered before recruitment and if they did not have valid information on height at recruitment (including a small proportion, about 0.05% whose reported height was <120 cm or >200 cm). For analyses including endometrial and/or cervical cancers, we excluded women if they reported a hysterectomy at recruitment, or if their hysterectomy status was unknown; similarly, for analyses of ovarian cancer, we excluded women if they reported a bilateral oophorectomy at recruitment, or if their oophorectomy status was unknown.

We calculated woman-years from the date of recruitment to the date of first cancer registration (at any site), death, or the last date of follow-up, whichever was first. For analyses of cancer incidence, the last date of follow-up was Dec 31, 2008, for the UK regions of East Anglia and South West; June 30, 2008, for Oxford, Thames, West Midlands, and North West (Mersey); and Dec 31, 2007, for Northern and Yorkshire, Trent, North West (Manchester and Lancashire), and Scotland.

**Statistical analysis**

We used Cox regression models to estimate relative risks (RRs) and CIs in relation to height at recruitment, taking attained age as the underlying time variable. We stratified all analyses by age at recruitment (<52, 53–55, 56–58, 59–61, 62–64, ≥65 years) and region (ten regions covered by ten cancer registries), and adjusted, as appropriate, for quintiles of socioeconomic group (based on Townsend deprivation score), body-mass index (<22.5, 22.5–24.9, 25.0–27.4, 27.5–29.9, ≥30 kg/m²), strenuous exercise (less than once a week, once a week or more), alcohol consumption (none, ≤2 units per week, >2 units per week), smoking (never, past, current ≥1 cigarette per day), age at menarche (<13, 13, ≥14 years), parity (0, 1–2, ≥3 full-term pregnancies), and age at first birth (<25, ≥25 years). We assigned missing values of adjustment variables a separate category and did sensitivity analyses restricted to women with available information on all adjustment variables. Information on all variables was available for women with available information on all variables a separate category and did sensitivity analyses.

We calculated the RR per 10 cm increase in height as a trend across the six category means using the measured mean height in each category. We trended across the six category means using the measured mean height at recruitment.

Analysis stratified by age at recruitment and region and adjusted for socioeconomic status, smoking, alcohol intake, body mass index, strenuous exercise, age at menarche, parity, and age at first birth.

**Table 2: Relative risks (RRs) and 95% floated CIs (FCIs) for total cancer incidence, by category of height reported at recruitment (mean measured height)**

| Mean height (cm) | Women | Incident cancers | RR (95% FCI) |
|------------------|-------|------------------|-------------|
| <155 cm (mean 152·8 cm) | 233 516 | 25 792 | 1·00 (0·98–1·02) |
| 155 cm (mean 156·5 cm) | 196 773 | 24 213 | 1·08 (1·07–1·10) |
| 160 cm (mean 160·4 cm) | 388 516 | 28 806 | 1·12 (1·11–1·14) |
| 165 cm (mean 164·9 cm) | 288 893 | 22 571 | 1·20 (1·18–1·22) |
| 170 cm (mean 169·0 cm) | 142 289 | 11 902 | 1·28 (1·25–1·30) |
| ≥175 cm (mean 173·8 cm) | 46 138 | 4 092 | 1·37 (1·33–1·42) |

We excluded women if they did not have valid information on height at recruitment (including a small proportion, about 0.05% whose reported height was <120 cm or >200 cm). For analyses including endometrial and/or cervical cancers, we excluded women if they reported a hysterectomy at recruitment, or if their hysterectomy status was unknown; similarly, for analyses of ovarian cancer, we excluded women if they reported a bilateral oophorectomy at recruitment, or if their oophorectomy status was unknown.

Where two categories of exposure are compared (as in the text) conventional CIs are given. For analyses of total cancer, more than two categories are compared (as in the figures), floated CIs (FCIs) were estimated by treating the RRs as floating absolute risks (FARs). Use of floated methods allows valid comparisons to be made between any two exposure groups, even if neither is the baseline group. All results are presented in the text with 95% CIs, but for analyses by cancer site, when multiple RRs were estimated, 99% CIs are given in the figures.

Where we present results in the form of plots, the RRs and their corresponding FCIs or CIs are represented by squares and lines with the area of each square proportional to the variance of the logarithm of the corresponding RR. This shows the amount of statistical information involved.

**Meta-analysis**

We identified published prospective studies of adult height and risk of total cancer (incidence or mortality) through electronic searches of published work (Medline and Embase, up to April, 2011) with combinations of the search terms “height”, “body size”, “anthropometry”, “neoplasms”, “mortality”, and “risk factors”, and
Relative risks (RRs) and 99% CIs per 10 cm increase in height for incident cancer at 17 specific sites

Table 3: Relative risks (RRs) and 95% CIs per 10 cm increase in height, for total incident cancer: effect of adjustment by various factors

| Number of incident cancers | RR* & 99% CI |
|---------------------------|--------------|
| Mouth and pharynx         | 0.94 (0.82–1.08) |
| Oesophagus                | 1.04 (0.91–1.19) |
| Stomach                   | 1.03 (0.90–1.18) |
| Colon                     | 1.25 (1.17–1.33) |
| Rectum                    | 1.14 (1.05–1.24) |
| Pancreas                  | 1.05 (0.95–1.17) |
| Lung                      | 1.03 (0.98–1.08) |
| Melanoma                  | 1.32 (1.22–1.42) |
| Breast                    | 1.17 (1.14–1.20) |
| Endometrium               | 1.19 (1.12–1.26) |
| Ovary                     | 1.17 (1.09–1.25) |
| Kidney                    | 1.29 (1.15–1.45) |
| Bladder                   | 1.00 (0.88–1.14) |
| CNS                       | 1.20 (1.09–1.32) |
| Non-Hodgkin lymphoma      | 1.21 (1.12–1.31) |
| Multiple myeloma          | 1.13 (0.99–1.29) |
| Leukaemias                | 1.26 (1.11–1.42) |
| Other and unspecified     | 1.15 (1.10–1.21) |
| Total cancer              | 1.16 (1.14–1.17) |

RR (95% CI)

- Adjusted by age and region only: 1.14 (1.13–1.15)
- Additionally adjusted separately by:
  - Socioeconomic status: 1.15 (1.13–1.16)
  - Alcohol: 1.14 (1.13–1.15)
  - Smoking: 1.15 (1.13–1.16)
  - Body-mass index: 1.15 (1.14–1.17)
  - Strenuous exercise: 1.14 (1.13–1.16)
  - Age at menarche: 1.14 (1.13–1.16)
  - Parity: 1.13 (1.12–1.15)
  - Age at first birth: 1.14 (1.13–1.15)
- Adjusted simultaneously for all of the above: 1.16 (1.15–1.18)

Analysis restricted to 1087489 women (81797 with cancer) with information on all adjustment variables.

Figure 2: Relative risks (RRs) and 99% CIs per 10 cm increase in height for incident cancer at 17 specific sites and for total cancer

The dotted line represents the RR per 10 cm increase in height for total cancer. *RRs are adjusted for age, region, socioeconomic status, smoking, alcohol intake, body-mass index, strenuous exercise, age at menarche, parity, and age at first birth.

Role of the funding source

The funding sources did not influence the design of the study, the collection, interpretation and analysis of the data, the preparation of this report, or the decision to publish. BJC, DC, and JG had access to the raw data for the study. The corresponding author had full access to all data and the final responsibility for the decision to submit for publication.

Results

The 1297124 women included in our analysis had a mean age at recruitment of 56.1 years (SD 4.9) and an average year of birth of 1942. The median length of follow-up was 9.4 years per woman (IQR 8.4–10.2 years), for a total of 11.7 million person-years, during which 97376 incident cancers were notified.

Table 1 shows characteristics of the study population, including measured height, by six categories of height reported at recruitment. Taller women tended to be of higher socioeconomic status, to drink more alcohol, to be more active, to have a later age at menarche, to have fewer children, and to have their first child later in life than shorter women. Taller women were less likely to be obese or to be current smokers. Based on heights measured in the validation sample, the mean height in the study population was 160.9 cm (SD 6.4).

Total cancer incidence rose with increasing height (table 2). Comparing women in the tallest group with
those in the shortest group (a difference of 21 cm: mean measured heights 174 cm and 153 cm), the adjusted RR for total incident cancer was 1·37 (95% CI 1·33–1·42; p<0·0001). The RR for total cancer was 1·16 (1·14–1·17; p<0·0001) per 10 cm increase in height (figure 1).

Figure 2 shows the RRs per 10 cm increase in height for the 17 separate cancer sites we assessed, for all other cancers and for total cancer. The height-associated RRs are greater than 1·0 for 15 of the 17 specific sites, and are significantly increased for ten specific sites and for the group of other and unspecified cancers: colon (RR per 10 cm increase in height 1·25, 95% CI 1·19–1·30), rectum (1·14, 1·07–1·22), malignant melanoma (1·32, 1·24–1·40), breast (1·17, 1·15–1·19), endometrium (1·19, 1·13–1·24), ovary (1·17, 1·11–1·23), kidney (1·29, 1·19–1·41), central nervous system (1·20, 1·12–1·29), non-Hodgkin lymphoma (1·21, 1·14–1·29), leukaemia (1·26, 1·15–1·38), and other cancers (1·15, 1·11–1·20). For no cancer site was there a significant decrease in risk with increasing height. There is heterogeneity across cancer sites (contrast test χ² [17 degrees of freedom] = 115·2; p<0·0001) mostly because of the greater than average increase in risk with increasing height for colon cancer and for malignant melanoma, and the lower than average risk for lung cancer. Breast cancer accounts for half of incident cancers in our study and the results for breast cancer therefore dominate the overall results. However, the overall RR of incident cancer in relation to height was not materially altered when we excluded breast cancer cases from our analysis (RR per 10 cm increase in height 1·19, 95% CI 1·17–1·21).

We adjusted our results in figures 1 and 2 and in table 2 by age, region, socioeconomic status, smoking, alcohol, body-mass index, physical activity, age at menarche, parity, and age at first birth. Table 3 shows the effect of adjustment by potential confounding variables on the RR for total cancer per 10 cm increase in height in an analysis restricted to the 1087489 women with full information on all adjustment variables. Compared with the risk with adjustment for age and region only (RR 1·14, 95% CI 1·13–1·15), additional adjustment by the remaining factors decreases the RR slightly to 1·16 (1·15–1·18).

Figure 3 shows the RR for total cancer per 10 cm increase in height, and the mean measured height, in subgroups of women defined by their year of birth, socioeconomic status, smoking, alcohol consumption, body-mass index, physical activity, age at menarche, parity, age at first birth, menopausal status, and use of oral contraceptives and hormone replacement therapy. As we expected, women born before 1939 were shorter than women born in 1946 or later (mean measured height 159·9 vs 161·5 cm), as were women from the lowest compared to the highest socioeconomic tertile (160·1 vs 161·4 cm). However, the height-associated RR for total cancer did not vary significantly by these or by most other characteristics. Figure 4 shows this lack of variation by socioeconomic status. Although the risk for total cancer is somewhat higher in women in the lowest tertile of socioeconomic status, the pattern of risk by height is similar in all three tertiles. Of the 12 personal characteristics we assessed, only smoking status substantially modified the size of the height-related RRs (figure 3). The RR per 10 cm greater height was 1·19 (95% CI 1·17–1·21) in never smokers, but only 1·11 (1·08–1·14) in current smokers (p<0·0001 for heterogeneity).

![Figure 3: Relative risks (RRs) and 99% CIs per 10 cm increase in height for all incident cancer, by various characteristics at recruitment](https://www.thelancet.com/oncology/Vol_12_August_2011/789)

**Figure 3:** Relative risks (RRs) and 99% CIs per 10 cm increase in height for all incident cancer, by various characteristics at recruitment

The dotted line represents the RR per 10 cm increase in height for all women. *Standardised to the distribution of self-reported heights within each subgroup of the whole study population. †RRs are adjusted as appropriate for age, region, socioeconomic status, smoking, alcohol intake, body-mass index, strenuous exercise, age at menarche, parity, and age at first birth.
Figure 5 shows the RRs per 10 cm increase in height by cancer site in never smokers and in current smokers (results in past smokers are uninterpretable, because they are a heterogeneous group with a wide range of times since last smoking). The mix of cancers differs in the two groups with, as expected, a higher proportion of women with lung and other smoking-related cancers in current smokers than in never smokers. In never-smokers, heterogeneity across cancer sites was substantially weaker (p=0.004) than in current smokers (p<0.0001).

For smoking-related cancers, the RR per 10 cm greater height was substantially smaller in current smokers than in never smokers (1.05 vs 1.17, p for difference=0.0004; figure 6). By contrast, for other specified cancers height-associated RRs were similar in current smokers and in never smokers, and close to our estimate for smoking-related cancers in never smokers (figure 6).

Published evidence suggests that current smoking is not a strong risk factor for colorectal cancer and the number of these cancers is large, so we undertook a sensitivity analysis with colorectal cancer classed as not related to smoking (ie, as in the latest full report on...
smoking and cancer available from 1ARC). The overall pattern of RRs remained similar, with lower risk for smoking-related cancers than for other cancers in current smokers, although the difference between these risks was reduced (RR per 10 cm height 1·02, 95% CI 0·97–1·06, in current smokers and 1·10, 1·03–1·17, in never smokers; p for difference=0·05); for other specified cancers, risks remained similar to those in our main analysis (RRs 1·18, 1·14–1·22, in current smokers and 1·19, 1·16–1·21, in never smokers).

Because breast cancer dominates our findings, we repeated our analyses shown in figure 3 separately for the five most common cancers in our study: breast, lung, colon, endometrium, and ovary, and for the remaining cancers. Overall, we did not identify significant heterogeneity, by the 12 factors we show in figure 3, for these cancer sites (χ² test for heterogeneity aggregated across all characteristics: colon p=0·7, lung p=0·2, breast p=0·3, endometrium p=0·5, ovary p=0·2, remaining cancers p=0·2).

Because there was no strong variation by cancer site in our study except in smokers, we did a meta-analysis of published studies of all-cancer risk, noting for each study our study except in smokers, we did a meta-analysis of cancers p=0·2). Because breast cancer dominates our findings, we repeated our analyses shown in figure 3 separately for the five most common cancers in our study: breast, lung, colon, endometrium, and ovary, and for the remaining cancers. Overall, we did not identify significant heterogeneity, by the 12 factors we show in figure 3, for these cancer sites (χ² test for heterogeneity aggregated across all characteristics: colon p=0·7, lung p=0·2, breast p=0·3, endometrium p=0·5, ovary p=0·2, remaining cancers p=0·2).

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Articles

Because of the large size of this study we were also able to undertake subgroup analyses by 12 potential confounding factors, in particular by socioeconomic status. Women in higher socioeconomic groups are on average taller (table 1), and socioeconomic status is related to total cancer incidence (figure 4), yet the association between height and risk of cancer was similar for women of low, medium, and high socioeconomic status. As in other studies that could adjust for a range of potential confounding factors, our results suggest that the relation between height and cancer risk is not due to other known risk factors for cancer.

Our findings show that the height-related RR of cancer was lower for smoking-related cancers than for other cancers, but only in current smokers. In accordance with our findings Kabat and colleagues have reported that lung cancer incidence in the Women’s Health Initiative study showed a stronger association with height in never smokers than in current or past smokers. Our test for potential modification of height-related cancer risks by smoking status used a multiplicative model. However, on an absolute scale there is little difference between current and never smokers in the excess cancer incidence rates. Smoking-related cancers are more common in current smokers than in never smokers, with age-standardised incidence rates of 599 and 176 per 100 000 women per year, in this cohort. The estimated excess age-standardised incidence rate for every 10 cm increase in height for smoking-related cancers is about 30 per 100 000 women per year, both in current and in never smokers (599×0·05 for current smokers and 176×0·17 for never smokers).

We found no other modification of height-associated RR by the 11 other factors we assessed, either for total cancer, or separately for the five most common cancers (breast, lung, colon, endometrium, and ovary). However, even in our large study we had limited power to assess modification of height-related risk by these factors.

There was little variation in height-associated RRs at specific cancer sites in never smokers, in whom the effect of height on cancer risk is free from modification by smoking. Most other studies have not made direct

|       | Mean year of birth | Mean height (cm) | Proportion of current smokers | Cancer outcome | Number of cancers | RR (95% CI) per 10 cm increase |
|-------|-------------------|-----------------|-------------------------------|---------------|------------------|-------------------------------|
| Men   |                   |                 |                               |               |                  |                               |
| Batty (UK, 2006)³ | 1917             | 177             | 42%                           | Mortality     | 3101             | 1·10 (1·05–1·16)              |
| Davey-Smith (UK, 2000)² | 1919             | 170             | 57%                           | Mortality     | 997              | 1·07 (1·08–1·18)              |
| Tulinius (Iceland, 1997)² | 1924             | 177             | 54%                           | Incidence     | 1275             | 1·10 (1·03–1·20)              |
| Giannouli (USA, 2004)² | 1928             | 176             | 20%                           | Incidence     | 3270             | 1·11 (1·04–1·17)              |
| Hebert (USA, 1997)² | 1929             | 178             | 10%                           | Incidence     | 2566             | 1·10 (1·04–1·17)              |
| Albarde (USA, 1988)² | 1931             | 174             | 40%                           | Incidence     | 341              | 1·12 (1·06–1·56)              |
| Jousilahti (Finland, 2000)² | 1935             | 172             | 47%                           | Mortality     | 659              | 1·04 (0·97–1·11)              |
| Batty (Australasia, 2010)² | 1936             | 179             | NA                            | Mortality     | 1951             | 1·10 (1·03–1·17)              |
| Okasha (UK, 2000)² | 1938             | 175             | 34%                           | Mortality     | 311              | 0·98 (0·79–1·21)              |
| Sung (South Korea, 2009)² | 1943             | 168             | 53%                           | Incidence     | 23725            | 1·10 (1·06–1·12)              |
| Batty (Asia, 2010)² | 1946             | 168             | NA                            | Mortality     | 3281             | 1·07 (1·00–1·14)              |
| All men |                   |                 |                               |               |                  |                               |
| Women  |                   |                 |                               |               |                  |                               |
| Davey-Smith (UK, 2000)² | 1919             | 158             | 42%                           | Mortality     | 848              | 1·08 (1·06–1·12)              |
| Tulinius (Iceland, 1997)² | 1925             | 164             | 41%                           | Incidence     | 1490             | 1·10 (1·01–1·25)              |
| Albarde (USA, 1988)² | 1935             | 161             | 30%                           | Incidence     | 302              | 1·08 (0·88–1·32)              |
| Jousilahti (Finland, 2000)² | 1935             | 158             | 14%                           | Mortality     | 441              | 0·96 (0·84–1·10)              |
| Batty (Australasia, 2010)² | 1938             | 161             | NA                            | Mortality     | 1131             | 1·19 (1·08–1·30)              |
| Okasha (UK, 2000)² | 1939             | 163             | 20%                           | Mortality     | 97376            | 0·89 (0·57–1·40)              |
| Million Women Study (UK, 2011) | 1942             | 161             | 20%                           | Incidence     | 97376            | 1·16 (1·14–1·17)              |
| Batty (Asia, 2010)²² | 1943             | 156             | NA                            | Mortality     | 1134             | 1·14 (1·02–1·24)              |
| Sung (South Korea, 2009)² | 1943             | 155             | 3%                            | Incidence     | 9443             | 1·14 (1·10–1·19)              |
| All women |                   |                 |                               |               |                  |                               |
| Total  |                   |                 |                               |               |                  |                               |

Figure 7: Meta-analysis of results from prospective studies: study-specific and summary relative risks (RRs) and 95% CIs for all cancer per 10 cm increase in height

The dotted lines represent the summary RRs. NA=not available. *Mean years of birth estimated as necessary. †Includes 24% (men) and 2% (women) pipe or cigar smokers. ‡Category midpoints used to estimate mean heights in height categories. §Method of Chêne and Thompson used to estimate mean heights in height categories.
comparisons across cancer sites or between smokers and non-smokers. In general, studies have found taller people to be at increased risk of a range of cancers with varying causes, with no individual cancer site consistently identified as showing no association. Our finding of differences in height-related RR between smokers and never smokers might provide an explanation for some reported inconsistencies in height-associated risk for smoking-related cancers.

Our meta-analysis of height and total cancer risk shows that findings are very consistent for incidence and for mortality, and in populations from Europe, North America, Asia, and Australasia with mean years of birth ranging over 30 years, and with mean heights ranging from 155 cm to 179 cm. Women in these studies were less likely than men to be current smokers (figure 7) and this might partly explain the slightly higher height-associated RR in women than in men in our meta-analysis. The overall result in women is also strongly weighted by the results from the Million Women Study, in which there has been allowance for measurement error, and more extensive adjustment than in the other studies, both of which tended to increase the estimated RR. As in any meta-analysis of published data, our findings need to be interpreted in the knowledge that other studies with relevant data might not have published their results.

The similarity of the height-associated RR for different cancers and in different populations suggests that a basic common mechanism, possibly acting in early life, might be involved. Adult height reaches its maximum between the ages of 20 and 30 years. Variation in height relates to genetic and environmental influences acting mostly in the first 20 years, or so, of life; environmental factors, including childhood nutrition and infections, are believed to predominate.

Hormone levels, especially of growth factors such as insulin-like growth factors (IGFs), both in childhood and in adult life, might be relevant. Circulating levels of IGFs in adulthood and childhood affect cancer risk; IGF-I levels in childhood and adolescence are related to cancer incidence some 10–15% above that expected if population height had remained constant. This assumes, of course, that the effect of height is independent of changes in other risk factors.

Contributors
VB, BC, JG, and GR contributed to the conception and design of the study and ISW to collection of validation data. DC, BC, JG, GR, and VB contributed to the analysis and interpretation of the data. JG drafted the report, which was critically revised for important intellectual content by BC, GR, and VB. All authors approved the report.

Conflicts of interest
We declare that we have no conflicts of interest.

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