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## ORIGINAL CONTRIBUTIONS

# Anthropometric Measurements and Vertebral Deformities

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To investigate the association between anthropometric indices and morphometrically determined vertebral deformity, the authors carried out a cross-sectional study using data from the European Vertebral Osteoporosis Study (EVOS), a population-based study of vertebral osteoporosis in 36 European centers from 19 countries. A total of 16,047 EVOS subjects were included in this analysis, of whom 1,973 subjects (915 males, 1,058 females) (12.3%) aged 50 years or over had one or more vertebral deformities ("cases"). The cases were compared with the 14,074 subjects (6,539 males, 7,535 females) with morphometrically normal spines ("controls"). Data were collected on self-reported height at age 25 years and minimum weight after age 25 years, as well as on current measured height and weight. Body mass index (BMI) and height and weight change were calculated from these data. The relations between these variables and vertebral deformity were examined separately by sex with logistic regression adjusting for age, smoking, and physical activity. In females, there was a significant trend of decreasing risk with increasing quintile of current weight, current BMI, and weight gain since age 25 years. In males, subjects in the lightest quintile for these measures were at increased risk but there was no evidence of a trend. An ecologic analysis by country revealed a negative correlation between mean BMI and the prevalence of deformity in females but not in males. The authors conclude that low body weight is associated with presence of vertebral deformity. *Am J Epidemiol* 1997;146:287–93.

anthropometry; osteoporosis; spinal diseases

A number of observations have linked body size and weight to osteoporosis and resultant fracture. Low body weight is an established risk factor for hip fractures in women (1, 2). Based on anthropometric measurements, Cummings et al. (2) found that weight-gain after age 25 years was an important protective factor whereas height at age 25 years was a risk factor. The Mediterranean Osteoporosis Study (MEDOS) of hip fracture incidence (1) in southern Europe showed that low body weight and low body mass index were risk factors for fractures. Low body weight has also been found to be a risk factor for hip fractures in the majority of other studies (3–6). However, Hemenway et al. (7) did not find any relation between body mass

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Abbreviations: CI, confidence interval; EVOS, European Vertebral Osteoporosis Study; MEDOS, Mediterranean Osteoporosis Study; OR, odds ratio.

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index and hip fractures but found that increasing height was a risk factor for hip fractures. In contrast to the data available on the relation between body weight or body mass index and the risk of hip fractures, there are very few data on the impact of body weight or body mass index on vertebral fracture risk. Although hip fracture risk might be related to the fact that subjects with a lower body mass are more vulnerable to the effects of falling, there are an abundance of studies that show that bone mineral density is correlated with body composition, mainly body weight (8–13). Thus, other sites of osteoporotic fracture, specifically the spine, might be associated with low body mass.

The purpose of the present study, therefore, was to investigate the relation between anthropometric measurements in subjects recruited from various regions in Europe and the occurrence of vertebral deformities as defined using a standardized morphometric technique. The data were gathered as part of a large multicenter, multinational population survey, the European Vertebral Osteoporosis Study (EVOS).

## MATERIALS AND METHODS

Details of the methods used in EVOS are provided elsewhere (14–18). In this analysis, those individuals with a vertebral deformity ascertained from the survey were compared with those without deformity, with respect to key anthropometric variables under study.

In brief, men and women from 36 centers from 19 countries took part in this study. Each center was invited to recruit an age- and sex-stratified random sample of 600 subjects (300 women and 300 men) aged 50 years and over from a population-based sampling frame, with the aim of recruiting 50 individuals of both sexes in each 5-year age group from 50–54 to 75–79 years.

The nature of the sampling frames varied between countries and is described elsewhere (16). For most of the centers, the sampling frames comprised a listing of the general population normally drawn up for administrative or health care purposes. The median center response rate was 54 percent (16). Studies of samples of non-responders showed no important bias with respect to osteoporosis risk (17). Subjects were invited to take part by letter of invitation for an interviewer-administered life-style questionnaire (15), which included questions on recalled minimum body weight after age 25 years and height at age 25 years. Data were also obtained on current and past cigarette smoking and on physical activity. The latter graded the most strenuous level of regular activity either at work or at home on a 1–4 scale. Current height and weight were recorded using routine clinical instruments available in each center. Height was measured without shoes and weight measured with the participants wearing light clothing prior to being x-rayed. Body mass index was calculated as weight (kg)/height $(m)^2$ for both current and recalled measures.

### Radiology

Lateral thoracic and lumbar spine radiographs were taken according to a standard protocol which included details concerning positioning of subjects and radiographic technique (19, 20). The thoracic film was centered at T7 and the lumbar film at L2. Prior to the study, each center forwarded sample radiographs to the radiology coordinating center in Berlin for quality assessment and to check compliance with the protocol.

There is no gold standard for defining the presence of vertebral deformity. For epidemiologic studies, the presence or absence of deformity is ascertained by quantitative assessment of vertebral shape (21).

All study radiographs were evaluated morphometrically using a translucent digitizer and cursor, and six points were marked on each vertebral body from T4 to L4 to describe vertebral shape. Using these six points, anterior (Ha), middle (Hm), and posterior heights (Hp) were determined for each vertebral body. A number of algorithms have been proposed for defining deformity based on these heights, and the algorithm proposed by McCloskey et al. (22) was used in this study. This algorithm has been shown (23) to have acceptable construct validity against low bone mass when compared with other available methods.

### Statistical methods

Logistic regression modeling was used to examine the associations between vertebral deformity and anthropometric indices. As the relation between these variables and deformity risk may be nonlinear, the former were entered into the model as categorical measurements after division by quintiles, as well as continuous variables. All analyses were adjusted for age, current cigarette smoking, and current level of physical activity, and were undertaken separately by sex. The analyses were also repeated after adjustment for "center" to allow for any residual confounding from that source. The results were identical to the non-center-adjusted odds ratios, and it is these odds ratios that are presented here. Adjustment for smoking and physical activity also made virtually no difference to the results from the age-only adjusted model. The data presented are those after adjustment for age. SAS software (24) was used throughout.

**TABLE 1. Summary data on cases and controls: European Vertebral Osteoporosis Study, 1990–1993**

| | Males, by vertebral deformity | | Females, by vertebral deformity | |
|---|---|---|---|---|
| | Present ($n$ = 915) | Absent ($n$ = 6,539) | Present ($n$ = 1,058) | Absent ($n$ = 7,535) |
| | Mean (SD*) | Mean (SD) | Mean (SD) | Mean (SD) |
| Age (years) | 66.3 (8.9) | 64.2 (8.5) | 68.4 (8.7) | 63.0 (8.4) |
| Current height (m) | 1.70 (0.07) | 1.71 (0.07) | 1.57 (0.07) | 1.59 (0.06) |
| Height at age 25 years (m) | 1.73 (0.07) | 1.73 (0.07) | 1.61 (0.07) | 1.62 (0.06) |
| Height loss (m) | −0.025 (0.030) | −0.019 (0.027) | −0.041 (0.035) | −0.027 (0.032) |
| Current weight (kg) | 77.2 (12.6) | 79.0 (11.7) | 65.8 (11.8) | 68.6 (11.9) |
| Weight at age 25 years (kg) | 66.5 (9.5) | 66.9 (9.4) | 54.6 (8.1) | 55.5 (8.3) |
| Weight gain (kg) | 10.7 (10.0) | 12.1 (10.2) | 11.0 (9.7) | 13.1 (10.5) |
| Current BMI* (kg/m$^2$) | 26.6 (3.6) | 26.9 (3.6) | 26.6 (4.5) | 27.2 (4.6) |

* SD, standard deviation; BMI, body mass index.

## RESULTS

There were 1,973 subjects (915 males, 1,058 females) (12.3 percent) with one or more deformities ("cases") and 14,074 subjects (6,539 males, 7,535 females) without deformity ("controls") for whom complete anthropometric data were available. The cases, as expected, were older than the controls. The descriptive data on height and weight are shown in table 1. The cases, both males and females, were slightly shorter and lighter than the controls. The recalled weight at age 25 years was also lower in the cases. Further weight gain since age 25 years was also lower in the cases.

Data on the relation with height is shown in table 2 for both males and females. We observed no evidence from the data of recalled height at age 25 years that

**TABLE 2. Influence of height on vertebral deformity: European Vertebral Osteoporosis Study, 1990–1993**

| Variable and quintile | Males ($n$ = 7,454) | | | Females ($n$ = 8,593) | | |
|---|---|---|---|---|---|---|
| | Range (m) | Odds ratio* | 95% CI† | Range (m) | Odds ratio* | 95% CI |
| Height now (m) | | | | | | |
| 1st (shortest) | <1.66 | 1.00‡ | | <1.54 | 1.00‡ | |
| 2nd | 1.66–1.69 | 0.80 | 0.63–0.99 | 1.54–1.56 | 0.65 | 0.52–0.81 |
| 3rd | 1.70–1.72 | 0.98 | 0.79–1.22 | 1.57–1.60 | 0.65 | 0.53–0.79 |
| 4th | 1.73–1.76 | 0.87 | 0.69–1.09 | 1.61–1.64 | 0.84 | 0.67–1.04 |
| 5th (tallest) | >1.76 | 0.85 | 0.68–1.08 | >1.64 | 0.74 | 0.58–0.93 |
| $p$ value ($\chi^2$ trend) | | | 0.002 | | | 0.000 |
| Height at age 25 years (m) | | | | | | |
| 1st (shortest) | <1.67 | 1.00‡ | | <1.57 | 1.00‡ | |
| 2nd | 1.68–1.71 | 0.93 | 0.74–1.16 | 1.57–1.59 | 1.11 | 0.88–1.40 |
| 3rd | 1.72–1.74 | 1.01 | 0.80–1.27 | 1.60–1.63 | 0.84 | 0.68–1.03 |
| 4th | 1.75–1.78 | 0.99 | 0.78–1.24 | 1.64–1.67 | 1.22 | 0.99–1.51 |
| 5th (tallest) | >1.78 | 1.1 | 0.90–1.43 | >1.67 | 1.12 | 0.90–1.40 |
| $p$ value ($\chi^2$ trend) | | | 0.430 | | | 0.160 |
| Height loss since age 25 years (cm) | | | | | | |
| 1st (greatest) | >4 | 1.00‡ | | >5 | 1.00‡ | |
| 2nd | 3–4 | 0.72 | 0.57–0.92 | 3–5 | 0.73 | 0.60–0.88 |
| 3rd | 2 | 0.46 | 0.36–0.58 | 2 | 0.69 | 0.55–0.85 |
| 4th | 1 | 0.49 | 0.38–0.63 | 1 | 0.55 | 0.42–0.71 |
| 5th (least) | 0 or height gain | 0.75 | 0.61–0.92 | 0 or height gain | 0.61 | 0.48–0.78 |
| $p$ value ($\chi^2$ trend) | | | 0.000 | | | 0.000 |

* Adjusted for age.
† CI, confidence interval.
‡ Referent group.

either tall or short stature was a risk factor for the disease, on the assumption that at that age individuals were free of vertebral deformity. However, we did find a reduced occurrence of vertebral deformity, in both sexes, for those whose current height was above the lowest quintile, with no obvious evidence of a linear trend in either sex. The reduction in height as a consequence of deformity might, in part, explain this observation. We therefore examined height loss—calculated as recalled height at age 25 years minus current height. For both males and females, as shown (table 2), those above the highest quintile of loss had the strongest association with vertebral deformity.

The data on weight and body mass index are shown in table 3. For current weight in both males and females, there was evidence of a threshold phenomenon; subjects in the lightest quintile had the greatest prevalence of deformity, and a statistically significant reduction in deformity was seen at all heavier levels. We observed similar relations when current body mass index was examined. However, although there was evidence of a trend of decreasing prevalence of deformity with increasing body mass index in females, no such trend was found in males (table 3). Finally, in both males and females, subjects with the greatest gain in weight had a statistically significant reduction in prevalence of vertebral deformity.

The data were also analyzed treating height and weight as continuous variables. Using this approach, we observed in males a 4 percent reduction in deformity prevalence (odds ratio (OR) = 0.96, 95 percent CI 0.93–0.99) for each 5 kg increase in weight along with a 2 percent reduction in prevalence (OR = 0.98, 95 percent CI 0.96–1.0) for each unit increase in body mass index. Similar findings were observed in females with a 6 percent reduction in prevalence for each 5 kg increase in body weight (OR = 0.94, 95 percent CI 0.91–0.96) and a 2.5 percent reduction in prevalence for each unit increase in body mass index (OR = 0.97, 95 percent CI 0.95–1.0).

Ecologic analysis was undertaken by grouping centers into countries and correlating vertebral deformity prevalence with mean body mass index after age adjustment. These data showed a significant negative correlation in females ($r = -0.66$, $p < 0.01$) but no relation in males ($r = 0.17$, not significant) (figure 1).

## DISCUSSION

The main finding from this large multicenter, population-based study is that low body weight was associated with the occurrence of vertebral deformity in both males and females, both absolutely and adjusted for height when considered as body mass index.

**TABLE 3. Influence of weight on risk of vertebral deformity: European Vertebral Osteoporosis Study, 1990–1993**

| Variable and quintile | Males ($n$ = 7,454) | | | Females ($n$ = 8,593) | | |
|---|---|---|---|---|---|---|
| | Range (kg) | Odds ratio* | 95% CI† | Range (kg) | Odds ratio* | 95% CI |
| Weight now (kg) | | | | | | |
| 1st (lightest) | <69.1 | 1.00‡ | | <58.1 | 1.00‡ | |
| 2nd | 69.1–75.0 | 0.72 | 0.57–0.90 | 58.1–64.0 | 0.76 | 0.61–0.93 |
| 3rd | 75.1–81.0 | 0.71 | 0.57–0.89 | 64.1–69.9 | 0.82 | 0.67–1.00 |
| 4th | 81.1–88.3 | 0.63 | 0.50–0.80 | 70.0–77.8 | 0.62 | 0.50–0.77 |
| 5th (heaviest) | >88.3 | 0.75 | 0.60–0.94 | >77.8 | 0.67 | 0.53–0.83 |
| $p$ value ($\chi^2$ trend) | | | 0.217 | | | 0.040 |
| BMI† now (kg/m$^2$) | | | | | | |
| 1st (lightest) | <24.04 | 1.00‡ | | <23.32 | 1.00‡ | |
| 2nd | 24.04–25.86 | 0.87 | 0.70–1.08 | 23.32–25.53 | 0.93 | 0.76–1.15 |
| 3rd | 25.87–27.50 | 0.84 | 0.67–1.05 | 25.54–27.73 | 0.92 | 0.75–1.14 |
| 4th | 27.51–29.74 | 0.71 | 0.56–0.89 | 27.74–30.80 | 0.76 | 0.61–0.95 |
| 5th (heaviest) | >29.74 | 0.80 | 0.64–0.99 | >30.80 | 0.74 | 0.59–0.92 |
| $p$ value ($\chi^2$ trend) | | | 0.029 | | | 0.000 |
| Weight gain since age 25 years (kg) | | | | | | |
| 1st (lowest) | <3.5 | 1.00‡ | | <4.5 | 1.00‡ | |
| 2nd | 3.5–7.9 | 0.91 | 0.72–1.14 | 4.5–8.9 | 1.16 | 0.94–1.44 |
| 3rd | 8.0–13.0 | 0.92 | 0.74–1.15 | 9.0–13.7 | 0.96 | 0.77–1.19 |
| 4th | 13.1–19.7 | 0.69 | 0.54–0.89 | 13.8–20.7 | 0.86 | 0.69–1.07 |
| 5th (highest) | >19.7 | 0.77 | 0.61–0.97 | >20.7 | 0.76 | 0.60–0.95 |
| $p$ value ($\chi^2$ trend) | | | 0.002 | | | 0.000 |

* Adjusted for age.
† CI, confidence interval; BMI, body mass index.
‡ Referent group.

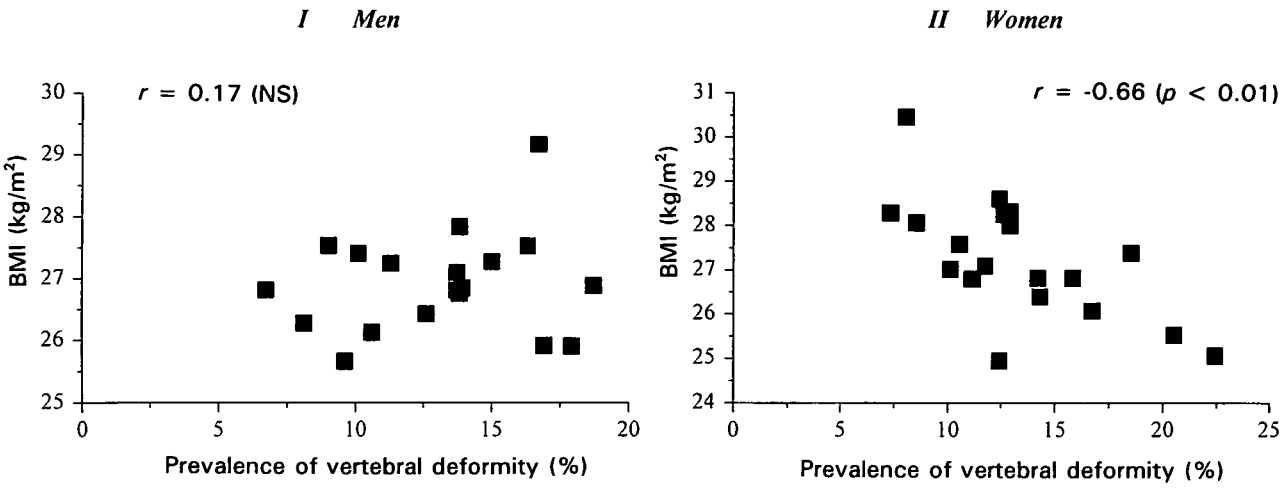


**FIGURE 1.** Correlation between prevalence of vertebral deformity and mean body mass index (BMI), by sex and country: European Vertebral Osteoporosis Study, 1990–1993. NS, not significant.

There are a number of methodological issues to be considered. First, case definition was based on morphometric analysis and therefore is concerned only with those disorders which lead to an alteration in vertebral shape. Such disorders include trauma, degenerative disease, Scheuermann's disease, as well as osteoporotic fracture. It might be expected that the former disorders could explain proportionately more of the deformities in males. Under our hypothesis that any risk between body mass and vertebral deformity would be predicted by an effect on bone mass, it was of note that the relation with increasing body mass index was stronger in women than in men. Cross-sectional data from EVOS (18) suggest that the vertebral deformities in men compared with women occur at a much earlier stage in life and that their frequency does not increase as markedly with increasing age. This might be related to the fact that some of the vertebral deformities in men are caused by trauma and are not due to osteoporosis.

In this multicenter, multinational study, it was not possible to ensure complete standardization of measurement of current weight and height in all centers, and formal quality control was not used. However, these measurements were undertaken blind to knowledge of the result of the x-ray and within each center identical methods were used for those subjects who were ultimately discovered to be cases and those who were found to be controls. The effect of any misclassification in recording current height and weight would thus be against finding a positive result and would therefore be unlikely to have contributed to our findings. Similarly, recalled height and weight are subject to error (25–27). In four centers, samples of 40 subjects were retested, and the reliability in results was found to be good with no evidence of observer bias (14). In general, height is over- and weight underestimated to a modest degree, leading to an underestimate of body mass index with this error greater in those with the greatest weight. Any such misclassification should be random in relation to deformity risk and again would make it more difficult to find a real result, thereby strengthening the observations of the role of these changes since age 25 years.

The results also showed that subjects in the lowest height quintile and those with the greatest height loss were more likely to have a vertebral deformity. Because this study was cross-sectional, these results may reflect that a decrease in height was a consequence of vertebral deformity, rather than being a risk factor. These data raise an interesting methodological issue in exploring the relation of body mass index to fracture. If deformity leads to height loss, then body mass index will be overestimated in the cases. By adjusting for this, the relation between body mass index and vertebral deformity would be that much stronger.

We found high body weight to be negatively associated with vertebral deformity both in terms of current weight and body mass index and also weight change since the recalled minimum weight after age 25 years. Thus, subjects who gained more weight had fewer deformities. This is consistent with the results for hip fracture from studies by Cummings et al. (2) and also similar to data from the MEDOS case-control study of hip fractures in southern European women (1). In MEDOS, subjects with a body mass index below a threshold level of 25 kg/m$^2$ had the highest risk of fracture. This result is similar to our findings where the threshold level for body mass index was approximately 24 kg/m$^2$ for both males and females.

Other studies have also found a lower body mass index rate in hip fracture cases. Greenspan et al. (4) reported significantly lower body mass index in cases (24 vs. 26 kg/m$^2$). Similar findings were found in Scandinavia by Johnell and Sernbo (5). Furthermore, in an Australian study, Cumming and Klineberg (3) found that subjects above the highest body mass index quintile had a relative risk of 0.3 compared with those in the lowest group. The corresponding value for weight was 0.4.

It is of interest to consider what biologic mechanism might explain the apparent risk from low body mass. There is a direct relation between body mass and bone mineral density in the spine, with subjects who have the greatest weight having the greatest bone mineral density (8–13). This in part might be mediated by a number of factors including 1) greater nutritional status, particularly during peak skeletal growth, 2) greater intake of calcium-containing food, 3) a direct trophic effect of loading on bone, 4) higher levels of estrogens in persons who are obese, or 5) greater spinal muscle mass protecting the spine from external trauma. By contrast, the observation that the risk is concentrated in those in the lowest quintile would suggest that general frailty is a risk factor for spinal bone loss.

In conclusion, in this population-based multicenter study, we found that height and weight were related to vertebral deformity, and that low body mass index and low body weight are both potentially important risk factors. This finding requires confirmation in a prospective study.

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