of diabetic patients' environmental exposures during intrauterine and early life, together with differences in the pattern of seasonality of birth with place and time, may identify environmental exposures specific to the initiation of the diabetogenic process.

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Rate of bone loss from lumbar spine in women with distal forearm fracture

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Fig 1—Annual rate of change of lumbar spine bone mineral density in 86 cases (women with distal forearm fracture) and 297 controls. Results shown as box and whisker plots in which height of the boxes represents interquartile range, horizontal line shows the median, cross shows mean, and vertical lines show the range of the data apart from outliers ($>1.5 \times$ interquartile range above or below interquartile range), which are shown as individual data points

Women who sustain a distal forearm fracture are at increased risk of vertebral osteoporosis, particularly if it occurs before the age of 60.¹ The aim of this study was to determine the mechanism for low bone mineral density in the lumbar spine of these women by prospectively evaluating the rate of bone loss from the spine.

Subjects, methods, and results

From our previous study population,¹ we studied 86 (69%) of the women (aged 50-81) who had sustained a distal forearm fracture between six months and three years before baseline and 297 women from an age matched, population based cohort. These women did not differ from those lost to follow up in their age, weight, height, years after menopause, or baseline bone mass of the lumbar spine. We measured bone mineral density by dual energy x ray absorptiometry (Lunar DPX, Lunar Corporation, Madison, WI, USA) at baseline and after two years. We examined the effect of age on bone mineral density by linear regression analysis and compared regression lines by a multiple linear regression technique. We compared normally distributed variables by unpaired *t* tests.

The cases did not differ from the population based controls with respect to age, weight, height, or years after menopause. Of the controls, 35 (12%) took hormone replacement therapy during the study, compared with 13 (15%) of the cases (95% confidence interval of difference -5% to 12%).

The baseline mean Z score for lumbar spine bone mineral density (the standard deviation from the mean bone mineral density of the controls after normalisation for age and weight) was decreased in the cases (-0.37 (-0.58 to -0.16)). Baseline lumbar spine bone mineral density decreased with age in the controls ($r = -0.29$, $P < 0.0001$) but not in the cases ($r = 0.08$, $P > 0.05$). The slopes and intercepts of this relation differed significantly between the two groups ($P = 0.009$ and $P = 0.003$ respectively). The controls lost bone from the lumbar spine at a rate of -0.59% a year ($P < 0.0001$), but the cases showed no bone loss (fig 1), and this difference was significant (0.59% (0.08% to 1.1%) a year, $P = 0.02$). In the controls the rate of change of lumbar spine bone mineral density was related to age ($r = 0.14$, $P = 0.02$) and years after menopause ($r = 0.12$, $P = 0.05$). There was no such relation in the cases, but the rate of change in these women was related to the time from distal forearm fracture ($r = 0.24$, $P = 0.03$). Exclusion of women who had

taken hormone replacement therapy during the study did not alter the results.

Comment

Distal forearm fracture is a typical manifestation of postmenopausal osteoporosis.² Propensity to fracture is thought to relate to decreased bone mass resulting from increased bone turnover after the menopause.³ We found that women with distal forearm fracture were at increased risk of vertebral osteoporosis: their decrease in lumbar spine bone mass was consistent with a doubling of the risk of vertebral fracture.⁴ Furthermore, these women had decreased rates of bone loss from the lumbar spine. The differences between the groups did not seem to be related to the use of hormone replacement therapy.

We suggest that the mechanism of decreased lumbar spine bone mass in women with distal forearm fracture may differ from that in other fracture syndromes. The decreased lumbar spine bone mass may result from low peak bone mass or a phase of accelerated bone loss in the early postmenopausal years, with subsequent decreased rates of loss. Both explanations would be consistent with our finding that the younger women had the lowest Z scores of lumbar spine bone mineral density. The explanations would also be consistent with an increased incidence of distal forearm fractures in women at the time of the menopause followed by a plateau in later years, as has been demonstrated in many epidemiological studies.^{2,5} We postulate that the excess fractures occur at the time of increased bone turnover, when reversible bone loss in the form of increased resorption is maximal. The finding of greater bone loss in those women with more recent fracture favours the concept of an earlier phase of accelerated bone loss as the cause of low lumbar spine bone mass.

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