Vitamin D deficiency is generally considered to be a major contributor to falls and fractures in older people. Since the landmark study by Chapuy et al,² it has been assumed that treatment with calcium and/or vitamin D reduces the risk of osteoporotic fractures in older people. In this issue of The Lancet, the RECORD study, a large randomised trial of participants with a recent low-trauma fracture, failed to show any benefit of calcium or vitamin D on fracture. Have we come to an erroneous conclusion about vitamin D? If so, in what aspects are the Chapuy data insufficient?

The primary prevention trial of Chapuy et al found that calcium plus vitamin D significantly reduced the risk of hip and non-vertebral fracture compared with calcium alone in elderly women.² However, the vitamin D status of the population was assessed in only 4.3% of 3270 patients. It was assumed that all participants were largely deficient in vitamin D because in this subgroup (n=142), serum 25-hydroxyvitamin D levels were low at baseline (33 nmol/L in the placebo group). A subsequent smaller study by Dawson-Hughes et al³ of 389 people living in the community, whose baseline 25-hydroxyvitamin D levels were much higher at 82.5 nmol/L, also reported benefit from calcium plus vitamin D on bone loss and fracture, although the study was not powered for the latter. It was unclear from these studies whether vitamin D needed to be combined with calcium, and what effect vitamin D had in secondary prevention.

The factorial design of the RECORD trial attempted to determine the relative contributions of calcium versus vitamin D on fractures. In this secondary prevention trial, 5292 ambulatory patients were randomised to receive calcium alone (1000 mg daily), vitamin D3 (800 IU daily), a combination of the two, or placebo. After follow-up of at least 24 months, there were no significant differences in fracture rates between the four groups. Interpretation of the study is limited by two main factors. First, compliance with medication was only moderate. It declined to 63% after 2 years and might have been as low as 45% when non-responders to the questionnaire about compliance were included. On this basis it is difficult to see how the authors can be sure there was "no evidence that true differences may have been obscured by poor compliance". Although the analysis was appropriate by intention-to-treat, it is possible to correct for the effect of non-compliance, which will dilute any physiologically achievable treatment effect in inverse proportion to the degree of compliance and so widen the confidence intervals.⁴ Second, the study perpetuates the limitations of most previous studies by measuring 25-hydroxyvitamin D in only a small sample (n=60—ie, just 1.1% of the study population). Thus the vitamin D status of the trial population at baseline remains largely unknown, although, because the patients were younger than in other studies, ambulatory, and living in the community, they were less likely to have vitamin D deficiency. There have been two subsequent studies of vitamin D in patients living at home. Trivedi et al⁵ reported a significant reduction in fractures after treatment with 100 000 IU cholecalciferol every 4 months compared with placebo in a randomised trial of 2686 patients over 5 years. Serum 25-hydroxyvitamin D was not assessed at baseline but was measured after 4 years of the trial in a subgroup (n=253) and was 53.3 nmol/L in the placebo group. However, a study of 9440 community-dwelling patients aged 75–100 years, randomised to either an annual injection of 300 000 IU.
Smoking cessation, weight gain, and lung function

Lung function normally declines with advancing age, and smoking causes premature onset and acceleration of this age-related decline. In today’s Lancet, Susan Chinn and colleagues report on data from the European Respiratory Health Survey, which involved 6600 men and women from 27 centres. Confirming the findings of others, the investigators report that decline in lung function was greater in current smokers than in those who never smoked. They also describe a significant reduction in lung function associated with weight gain after cessation. This reduction in lung function was greater in men than in women, possibly due to the larger girth accompanying weight gain in men.

Chinn and colleagues’ findings are important. The results draw attention to the fact that acceleration of decline in lung function must be added to the long list of negative health consequences of smoking. The survey shows that slowing of lung decline is an added benefit of quitting. The findings highlight the potential for even greater health benefit if weight gain could be prevented after smoking cessation.

Weight gain is a known risk of smoking cessation, and concern about weight gain has been described as a deterrent to quitting, particularly for women. We have shown in prospective data from the US Nurses’ Health Study that the greater the number of cigarettes smoked before cessation, the greater the weight gain after stopping. In a national sample of US adults, men gained an average of 2.8 kg and women 3.8 kg after quitting.

However, it is important to note that up to 10% of men and 13% of women might gain more than 13 kg after stopping smoking.

Despite the consequences of weight gain, we have also shown that the benefits of smoking cessation, in terms of decreased mortality, greatly outweigh any health risks. Within 2 years of smoking cessation, total mortality rates for former smokers drop by 17% compared with those for continuing smokers. Again, the findings of Chinn and colleagues suggest that these health advantages could potentially be increased by weight control at the time of quitting. Thus avoiding weight gain would not only remove a major barrier to cessation, but also allow for greater health benefits.

What causes this increase in weight, and how might we promote smoking cessation without weight gain? Smoking cessation is followed by change in food preferences, increased caloric intake, decreased metabolic rate, and increased activity of lipoprotein.