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risk of fractures and falls. Some think that the use of these high intermittent doses are not physiological and therefore the results from these studies are claimed not to be compelling. The debate is likely to continue and trials with high daily doses are ongoing.

Nevertheless, existing evidence does not lend support to the commonly held belief that vitamin D supplementation in general prevents osteoporosis, fractures, and non-skeletal diseases. Consequently, the impression that vitamin D is a sunshine vitamin and that increasing doses lead to improved health is far from clear. Without stringent indications—ie supplementing those without true insufficiency—there is a legitimate fear that vitamin D supplementation might actually cause net harm. A report from the Institute of Medicine6 also emphasized that there might be risks from both low and high concentrations of vitamin D and that there might be a U-shaped curve of risk, as has been noted with other vitamin supplements. Until more information is available, it would be prudent to choose a cautious approach to vitamin D supplementation and to put more emphasis on the development of evidence-based cut-off points for vitamin D inadequacy.

Karl Michaelsson
Department of Surgical Sciences, Uppsala University, 751 85 Uppsala, Sweden
karl.michaelsson@surprci.uu.se
I declare that I have no conflicts of interest.

1 Holick MF. Vitamin D deficiency. NEJM 2007; 357: 266-81.

TANDEM: understanding diabetes and tuberculosis

The alarming rise in cases of type 2 diabetes poses a serious threat to global tuberculosis control. The number of people with diabetes is expected to rise to at least 592 million by 2035. Currently, more than 80% of adults with diabetes live in low-income or middle-income countries, and diabetes generally presents at a younger age in these countries. People with diabetes are three times more likely to develop active tuberculosis than are people without diabetes, and there are now more tuberculosis patients with concomitant diabetes than with HIV coinfection.

Basic knowledge to help understand and control the intertwined epidemics of tuberculosis and diabetes is urgently needed. Awareness of the potential public health and clinical importance of the association between the two diseases is increasing, thanks partly to a collaborative framework for care and control of tuberculosis and diabetes issued by the International Union Against Tuberculosis and Lung Disease and WHO in 2011. Likewise, the International Diabetes Federation recognises the link with tuberculosis and supports efforts to simultaneously address this double burden of disease. However, many questions remain unanswered.

First, screening of patients with tuberculosis for diabetes could improve detection, early treatment, and secondary prevention of diabetes. Likewise, screening of patients with diabetes for tuberculosis could increase detection and control of tuberculosis. However, such screening is not routinely done in most settings, and the optimum and cost-effective approach has yet to be defined. Second, diabetes is associated with increased tuberculosis treatment failure, relapse, and death, but whether optimum glucose control reduces these effects is unclear. Third, how to achieve optimum glucose control is unknown because both tuberculosis itself and antituberculosis drugs hamper glucose control in diabetes (eg, rifampicin increases the metabolism of most oral antidiabetic drugs). Fourth, no study has assessed the treatment needs of patients with newly diagnosed diabetes after tuberculosis.