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Comment on: Gale EAM (2005) Spring harvest? Reflections on the rise of type 1 diabetes. Diabetologia 48:2245–2250; and Walker M, Mari A, Jayapaul MK et al (2005) Impaired beta cell glucose sensitivity and whole-body insulin sensitivity as predictors of hyperglycaemia in non-diabetic subjects. Diabetologia 48:2470–2476

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To the Editor: Your interesting Editorial makes the point that accelerated emergence of type 1 diabetes in childhood may reflect the loss of some protective factor in the environment, and postulates that this mechanism could also contribute to the rising incidence of type 2 diabetes in childhood and beyond [1]. Walker et al. show that impaired islet beta cell glucose sensitivity and whole-body insulin sensitivity are not only independent predictors of hyperglycaemia in non-diabetic subjects, but together outweigh the predictive effects of the commonly recognised lifestyle and body-build risk factors [2]. As Gale points out [1], an understanding of what it is we have come to lack could lead to the reversal of environmentally mediated increases in the risk of diabetes, in the same way that folic acid supplementation has led to reductions in spinal cord defects in recent years. The urgent question, therefore, is: what environmental factor with all the necessary protective effects have we become short of in recent years? Review of the literature, including 20 papers published in *Diabetologia* (!), suggests that hypovitaminosis D should be considered, since vitamin D has each of the properties required. Hypovitaminosis D developed with reduced exposure to sunlight during the industrial revolution, and although controlled—mainly by supplementation with cod liver oil—after its discovery in the 1920s, has become common again with modern lifestyles [3]. Vitamin D is protective against autoimmune disease, including type 1 diabetes, as is well known in the non-obese diabetic mouse [4], with strong suggestions from epidemiological studies

that early life hypovitaminosis D is a risk factor for type 1 diabetes in childhood and early adult life in humans [5]. Furthermore, adequate supplies of vitamin D are necessary for insulin secretion: vitamin D status is an independent determinant of both insulin sensitivity and secretory capacity in mildly hyperglycaemic and normoglycaemic subjects [6, 7], and reduced vitamin D status is a risk factor associated with type 2 diabetes [8]. Short-term correction of hypovitaminosis D improves insulin secretion in man, as in animals, and can improve hyperglycaemia [6]. Vitamin D and vitamin A interact to influence glycaemia. In addition, too much or too little vitamin A can induce hyperglycaemia in man. Thus, in countries where vitamin A deficiency is common, this factor would require additional consideration. No adequate long-term prospective trials of vitamin D supplementation have as yet examined the effects on the risk of diabetes, though other relevant changes have been reported [9]. People used to grow up with routine exposure to summer sunlight and to take cod liver oil as children and in pregnancy as part of the British World War 2 Welfare Foods Scheme (<http://www.worldwar2exra.co.uk/Online%20Museum/Museum%20Docs/foodration.html>, last accessed in February 2006). Current advice (from Cancer Research UK, available at <http://www.cancerresearchuk.org/sunsmart/staysafe/>, last accessed in February 2006) to avoid more than 15 min of sunshine, to cover up and to use sunscreens (that at $\geq$ factor 8 block 90% of synthesis of vitamin D in skin), together with the consumption of low-fat diets and long working hours spent indoors, mean that many sections of the population currently depend on the few foods containing vitamin D (limited to oily fish, fortified foods [some cereals, British margarine] and egg yolk) in the UK and across the globe [10]. Vitamin D supplement sales have surged following recent widespread media publicity on the meta-analysis by Garland et al. [11] showing ~50% reductions in many common cancers in those taking $\geq 1,000$ IU (25 μ g) of vitamin D per day. Further uptake of

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supplementation may follow the demonstration that childhood bone strength is predicted by maternal vitamin D status in late pregnancy [12]. Adequate prospective trials of vitamin D supplementation, though long overdue for reduction of non-bony diseases, including diabetes, may no longer be possible if—as current press reports suggest—the public are taking the reduction of this risk factor in hand themselves. However, widespread supplementation among the general population could lead to a downturn in the development of both types of diabetes, as was seen for lung cancer and heart disease following reductions in cigarette smoking.

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