Vitamin D deficiency and the asthma epidemic

The enthusiastic editorial by Weiss and Litonjua (Thorax 2007;62:745–6) may have left some readers with the impression that a single cause of the rise in asthma, multiple sclerosis, inflammatory bowel disease and type 1 diabetes in the West has now been discovered, namely vitamin D deficiency, and that primary prevention of asthma and autoimmunity is just around the corner. Indeed, their bold conclusion portrays vitamin D supplementation as a likely panacea for many ills worldwide.

Weiss and Litonjua argue that the epidemiological evidence to implicate prenatal vitamin D and vitamin E deficiency in the aetiology of asthma and atopy is sufficiently compelling to justify urgent prenatal supplementation trials. They cite their own observational data linking a higher maternal intake of vitamin E and vitamin D to a lower risk of early wheezing, but dismiss contrary evidence which suggests that a higher vitamin D status in utero and infancy may increase the risk of atopic conditions in later life.1,2 However, these latter findings, and recent data in adults,3 are in keeping with the original “vitamin D hypothesis” of Wjst and Dold, not cited in the editorial, which proposed that increasing intake of vitamin D, as a result of fortification of foods such as margarine, may have contributed to the rise in atopy in the West.4 I would argue therefore that the vitamin D story is, at present, rather more confused than Weiss and Litonjua suggest, and that before rushing into prenatal nutrient supplementation trials, we need more convincing data to support their hypothesis, and greater confidence that such an intervention would be safe.

Given the failure to translate observational associations between antioxidant deficiency and asthma into beneficial interventions in adults, we need to be more sure that observational links with prenatal nutrition are not confounded, and that longer term follow-up of birth cohorts does not reveal a positive relation between prenatal vitamin D status and atopy. Demonstration of interactions between prenatal vitamin D status and vitamin D receptor polymorphisms on asthma and atopic outcomes would strengthen causal inference. As the authors themselves point out, we know very little about the effects of vitamin D and E on fetal and immune development, and it would be prudent to heed a recent lesson from a trial of prenatal vitamin C and E supplementation in which, compared with placebo, vitamin supplementation increased the risk of low birth weight.5

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Author’s reply

We would like to thank Dr Shaheen for his interest in our recent editorial (Thorax 2007;62:745–6). Shaheen brings up several important points, which we were unable to discuss because of the space limitations. Many of these points have been discussed in our recently published commentary in another journal.1 We agree with one of the points that Shaheen makes—namely, we certainly acknowledge that there is some evidence for a contrary hypothesis that Wjst and Dold put forth, as we have discussed.1 However, there are other points where we disagree.

Firstly, Shaheen states that the Wjst–Dold vitamin D hypothesis was a “result of fortification of foods such as margarine, which may have contributed to the rise in atopy in the West”.1 This is incorrect; many observational associations between vitamin D and asthma are in keeping with the original vitamin D hypothesis.2–4 We agree that this is of some relevance. However, genetic polymorphisms in VDR and its binding protein will only explain a small percentage of phenotypic variation in vitamin D levels and will be much less important than environmental factors such as dietary supplements and sun exposure behaviours in determining vitamin D levels, which are primarily a measure of recent rather than chronic exposure.

We regret that the limits of the editorial did not allow a complete review of this interesting and important topic.

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