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Letter to the Editor

Curcumin and diabetes: a role for the vitamin D receptor?

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A recent paper published in the *British Journal Nutrition*[1] provides interesting data supporting the idea that curcumin can reduce metabolic and inflammatory problems induced by diabetes, including hyperglycaemia itself. Turmeric, the natural source of curcumin, is widely used as a colouring agent for sauces and manufactured foods as well as for cooking curries. The beneficial effects of curcumin on the liver, reported by the authors, will be especially important to pursue now that obesity and type 2 diabetes mellitus are increasing in prevalence and obesity comes to match malnutrition as a major health problem in many countries[2]. In discussion of the possible reasons for the beneficial effects of curcumin, including increased insulin secretory responsiveness to glucose, increases in hepatic glucokinase activity and increased antioxidant-related protection of pancreatic islets[1], the authors do not consider whether any of the non-insulin-related effects of curcumin could be accounted for by the vitamin D ligand-like actions of curcumin. Hormonal vitamin D (calcitriol) activates vitamin D receptors (VDR) and has mechanistic effects including increasing insulin secretory responsiveness to glucose, reducing abnormal islet renin–angiotensin activity, reducing inflammation and reducing abnormalities in several of the disorders used to define the metabolic syndrome[3,4]. Thus, alternative activating ligands for the VDR such as curcumin can be expected to have similar effects[5]. The possibility that curcumin has some VDR-activating effect in the study of Gutierres *et al.*[1] could be tested by determining whether or not serum parathyroid hormone (PTH) concentrations decrease with curcumin supplementation. A decrease in circulating PTH with curcumin treatment would support the suggestion that both insulin-related and non-insulin-related beneficial effects of curcumin could be mediated, at least in part, through VDR activation. This is because vitamin D supplementation in human hypovitaminosis D increases insulin secretory responses to glucose (in the absence of established type 2 diabetes mellitus) and may also reduce abnormally raised insulin resistance and suppress inflammation[6].

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References

1. Gutierres VO, Pinhiero CM, Assis RP, *et al.* (2012) Curcumin-supplemented yoghurt improves physiological and biochemical markers of experimental diabetes. *Br J Nutr* **108**, 440–448.
2. Wang Y, Chen HJ, Shaikh S, *et al.* (2009) Is obesity becoming a public health problem in India? Examine the shift from under- to over-nutrition problems over time. *Obes Rev* **10**, 456–474.
3. Boucher BJ (2012) Is vitamin D status relevant to metabolic syndrome? *Dermatoendocrinol* **4**, 212–224.
4. Cheng Q, Li YC, Boucher BJ, *et al.* (2011) A novel role for vitamin D: modulation of expression and function of the local renin–angiotensin system in mouse pancreatic islets. *Diabetologia* **54**, 2077–2081.
5. Haussler MR, Whitfield GK, Kaneko I, *et al.* (2012) Molecular mechanisms of vitamin D action. *Calcif Tissue Int* (Epublication ahead of print version 11 July 2012).
6. Hewison M (2012) Vitamin D and immune function: autocrine, paracrine or endocrine? *Scand J Clin Invest Suppl* **243**, 92–102.