

A case-control study of vitamin D status and asthma in adults

K. Allan¹, G. Devereux¹, G. McNeill¹, A. Wilson², A. Avenell¹ and W. Fraser³

¹*Institute of Applied Health Sciences, University of Aberdeen, Aberdeen AB25 2ZD, UK*, ²*Biomedicine Group, School of Medicine, University of East Anglia, Norwich, NR4 7TJ, UK* and ³*Metabolic Bone Diseases Unit, Department of Clinical Chemistry, University of Liverpool, Duncan Building, Daulby St, Liverpool L69 3GA, UK*

It has been suggested that the rapid increase in the prevalence of asthma in developed countries in recent decades may be the result of changes in diet and lifestyle⁽¹⁾. Asthma is associated with an alteration in the balance of T-helper lymphocytes with an increase in pro-inflammatory Th2 cells. As vitamin D may directly suppress Th2 differentiation^(2,3), it has been hypothesised that low vitamin D status, as a consequence of sun avoidance behaviours and an increasingly indoor lifestyle, could contribute to the rising prevalence of asthma⁽⁴⁾.

The present study was designed to compare the vitamin D status of age and sex-matched adults with and without physician-confirmed asthma. The study was conducted in the Chest Clinic, Aberdeen Royal Infirmary and the Department of Respiratory Medicine; Norfolk and Norwich University Hospital, Norfolk. One hundred and sixty participants aged between 18 and 50 years were recruited, 80 with physician-confirmed mild/moderate asthma and 80 age and gender-matched controls. Cases and controls were assessed within a month of each other to control for seasonal variation of sunlight exposure. Controls were individuals without asthma who had a smoking history of <10 pack-years. The majority of controls (70%) were recruited from local daycase surgery units, the remainder being recruited after advertising in local press. Ninety-four participants were recruited in Aberdeen between June 2007 and April 2008, and 66 in Norwich between October 2007 and September 2008. Vitamin D status was assessed by serum 25-hydroxyvitamin D₃ measured by HPLC-tandem mass spectrometry.

Mean serum 25-hydroxyvitamin D₃ concentration was 8.68 ng/ml (95% CI 7.60, 9.75), being lower in Aberdeen 6.78 ng/ml (95% CI 5.32, 8.25) than Norwich 11.5 ng/ml (95% CI 10.2, 12.8). In Aberdeen, 76% of the participants had serum levels below the generally accepted cut-off for a deficiency of 10 ng/ml⁽⁵⁾. In Norwich, this figure was 42%. In winter (December–February), these proportions rose to 92.3% and 46.4%, respectively. There was no significant difference in the serum 25-hydroxyvitamin D₃ concentrations between cases and controls: 8.50 ng/ml (95% CI 7.06, 9.95) v. 8.86 (95% CI 7.22, 10.5). Conditional logistic regression adjusting serum 25-hydroxyvitamin D₃ levels for age, gender, smoking status, BMI and season of assessment revealed no difference in serum 25-hydroxyvitamin D₃ levels between cases and controls (OR asthma v. control 0.98 (95% CI 0.91, 1.04), *P* = 0.50). Similar multivariable analysis demonstrated association neither between 25-hydroxyvitamin D₃ levels and asthma severity nor lung function (FEV₁ % predicted).

This study does not find evidence to support the use of vitamin D as an adjunct to conventional therapy in asthma in adults.

This study was funded by NHS Grampian Endowment Funds.

1. Devereux G (2006) The increase in the prevalence of asthma and allergy: food for thought. *Nat Rev Immunol* **6**, 869–874.
2. Jirapongsananuruk O, Melamed I & Leung DY (2000) Additive immunosuppressive effects of 1,25-dihydroxyvitamin D₃ and corticosteroids on TH1, but not TH2, responses. *J Allergy Clin Immunol* **106**, 981–985.
3. Urry Z, Xystrakis E, Richards DF *et al.* (2009) Ligation of TLR9 induced on human IL-10-secreting Tregs by 1α,25-dihydroxyvitamin D₃ abrogates regulatory function. *J Clin Invest* **119**, 387–398.
4. Litonjua AA & Weiss ST (2007) Is vitamin D deficiency to blame for the asthma epidemic? *J Allergy Clin Immunol* **120**, 1031–1035.
5. Devereux G, MacDonald H & Hawrylowicz C (2009) Vitamin D and asthma: time for intervention? *Am J Respir Crit Care Med* **179**, 739–742.