PostScript

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Food allergy in childhood

We thank Colver, MacDougall, and Cant for their response to our paper.¹ The message underpinning our paper was that severe allergic reactions to foods are not as uncommon as MacDougall *et al* suggested.² However, we are not comparing like with like and the problem lies in the definition of a severe food reaction. MacDougall *et al* only identified the extreme end of the clinical spectrum (children who have suffered cardio-respiratory arrest, received at least two doses of epinephrine (adrenaline), a fluid bolus, inotropic support, or at least two doses of nebulised bronchodilator—all unvalidated and unreferenced outcome measures). Having set the threshold for inclusion so high, it is hardly surprising that the incidence of this extreme outcome is so low. What was the authors' purpose in identifying this extreme population? If it was reassurance about the low incidence of life threatening reactions, then it should be appreciated that in a recent American series,³ 8/10 children who died of food allergy had previously reacted to the food that caused the fatality. Also, in a UK survey, 21/92 (23%) of peanut allergic patients with a history of a mild reaction subsequently had a severe reaction to peanut.⁴ MacDougall's data should not be used to dilute care given to children with less severe food allergy.

MacDougall *et al* have failed to identify the much larger population, of the most interest to paediatricians, allergists, and parents: children who have suffered a less extreme but still severe food allergy reaction (or anaphylaxis) characterised by airway narrowing.⁵ It is important to identify and study this large group as there is no consensus on how they should be managed. We have shown that intervention with a structured management plan, incorporating assessment of severity, avoidance advice and tailored medication for self treatment, together with a patient held treatment plan, reduces the risk and severity of further reactions, in both adults and children.⁶

It is clear that the British Paediatric Surveillance Unit (BPSU) survey missed children referred to allergy centres, not run by paediatricians. MacDougall *et al* studied

only inpatient admissions and not outpatient referrals. It is erroneous to assume that children referred to an allergy clinic would have been seen by a paediatrician. We look after over 790 outpatient referrals of children with nut allergy; 218 (29%) have had reactions involving respiratory symptoms and 58 (8%) severe dyspnoea and collapse. This number would be much larger if all foods were considered, and this effect is repeated for other allergy centres around the country. These children with severe reactions are derived from approximately 1/30th of the UK population. The majority were referred directly from their general practitioner and have had no contact with a paediatrician, and therefore would not have been notified to the BPSU. The fact that the number of inpatient admissions in MacDougall's paper matched that from a previous survey is irrelevant, as neither considers the large number of children who present to allergy outpatient clinics via their general practitioner.

We present data to estimate the incidence of those severe reactions that were missed by MacDougall's paper, using a different and more appropriate definition of severity. We compare them to MacDougall's estimate of 0.19 severe food reactions per 100 000 children per year. We have data from 791 children with nut allergy, 58 of whom had at least one severe nut induced reaction; the data were collected over six years, giving an incidence of at least 1.93 severe reactions per 100 000 children per year for nut allergy alone: a figure 10-fold higher than MacDougall's data for all food allergy. This estimate is a minimum, as each child has had at least one severe reaction (possibly more) and is for nut allergy alone, rather than all food allergies.

The estimate of the incidence of deaths due to food allergy produced by MacDougall *et al* is an underestimate, as the authors did not consider asthmatic deaths, where no allergic cause was suspected and we thank Colver for acknowledging this. There are 60-fold more deaths due to asthma, than those identified as due to a severe food reaction by MacDougall *et al*. If only a few of these were due to food allergy, it would alter the figures dramatically.

Colver has cited Bock's important series of fatal food reactions,³ which showed 80% (8/10) of deaths in under 16 year olds were due to peanuts or nuts, the youngest being 2 years old. It is not clear, however, why we should be reassured by MacDougall's finding of no peanut allergy induced deaths in children under 13 years. Do the authors not consider tree nut allergy induced deaths in 2 year olds, or peanut allergy induced deaths in 14–16 year olds relevant?

Finally, we agree that the issues surrounding provision of adrenaline autoinjectors require good data on the clinical features and natural history of nut allergy. However, to consider adrenaline autoinjectors in isolation is missing the point. The provision of adrenaline autoinjectors should be seen in the broader context of a comprehensive management plan for all children with food allergy. Children with mild food allergy reactions can go on to have more severe

reactions.⁴ We have shown that enrolment of children with nut allergy of all severities in such a management plan, consisting of repeated specialist advice on allergen avoidance, together with tailored medication including oral antihistamine with or without inhaled adrenaline or injectable adrenaline, results in a reduced number and severity of further reactions.⁶ Colver has emphasised the efficacy of this treatment plan in children, by highlighting that only three adults (aged 27–41 years) and no children had further severe reactions.

A T Clark, P W Ewan

Addenbrooke's Hospital, Cambridge, UK;
andrew.clark@addenbrookes.nhs.uk

References

- 1 Clark AT, Ewan PW. Food allergy in childhood: have the dangers been underestimated? *Arch Dis Child* 2003;88:79–81.
- 2 MacDougall CF, Cant AJ, Colver AF. How dangerous is food allergy in childhood? The incidence of severe and fatal allergic reactions across the UK and Ireland. *Arch Dis Child* 2002;86:236–9.
- 3 Bock SA, Munoz-Furlong A, Sampson HA. Fatalities due to anaphylactic reactions to foods. *J Allergy Clin Immunol* 2001;107:191–3.
- 4 Hourihane JO'B, Kilburn SA, Dean P, *et al*. Clinical characteristics of peanut allergy. *Clin Exp Allergy* 1997;27:634–9.
- 5 Project team of the Resuscitation Council, UK. Emergency medical treatment of anaphylactic reactions. *J Accid Emerg Med* 1999;16:243–7.
- 6 Ewan PW, Clark AT. Long-term prospective observational study of patients with peanut and nut allergy after participation in a management plan. *Lancet* 2001;357:111–15.

Symptomatic vitamin D deficiency among non-Caucasian adolescents living in the United Kingdom

We have previously drawn attention to a resurgence of vitamin D deficiency rickets in young children of South Asian (Indian, Pakistani, and Bangladeshi) and Middle Eastern origin, living in the UK.^{1,2} We now describe nine (one male) non-Caucasian, adolescents (age 11–17 years) who presented with symptomatic vitamin D deficiency between 1997 and 2002. Table 1 summarises their ages, ethnic origins, clinical symptoms, signs, and relevant biochemical findings at presentation. All presented with symptoms of vitamin D deficiency, which included lower limb pains, difficulty in walking or climbing stairs, carpal/pedal spasms, and hypocalcaemic convulsions. Clinical signs included positive Chvostek sign, inability to stand up unaided from a squatting position due to proximal myopathy, bowed legs (*genu varum*), and knock-knees (*genu valgum*). Three patients (cases 1, 6, 9) had radiological changes of vitamin D deficiency with widening and fraying of metaphyses, but these changes were not as severe as those seen in toddlers with vitamin D deficiency rickets. As shown in table 1, biochemical features of the disease included low serum concentrations of 25-hydroxycholecalciferol (25(OH)D; a measure of an individual's vitamin D status), increase

Table 1 Demographic and clinical data of the patients

Case	Age (y) and gender	Ethnic origin	Symptoms	Signs	Corrected Ca (mmol/l) (2.15–2.65)	P (mmol/l) (0.7–1.4)	ALP* (IU/l)	PTH (pg/ml) (10–60)	25OHD† (ng/ml) (5–30)	1,25(OH) ₂ D (pg/ml) (20–50)
1	13 ♀	Iranian	Pain in ankles and knees	+ve Chvostek's sign¶	1.41	1.31	1685	418	<2.0	21
2	17 ♀	Indian	Pain in back, thighs, knees, and calves. Difficulty walking, climbing stairs and running	Proximal myopathy‡	1.89	0.61	519	518	<2.0	47
3	14 ♀	Indian	Pain in hips, knees, and calves	Proximal myopathy‡, knock-knee, with 7 cm separation of medial malleoli	1.98	0.94	1871	146	10	127
4	11 ♀	Afghanistani	Pain in knees, difficulty walking	Proximal myopathy‡, bowed legs, with 5 cm separation of upper medial tibial condyles	2.25	0.7	2484	278	3.6	35
5	16 ♂	Pakistani	Muscle cramps, facial twitches, stridor, carpopedal spasms, and convulsions	+ve Chvostek's sign§	2.00	0.72	310	180	<2.0	16
6	12 ♀	Pakistani	Non-specific limb pains	Proximal myopathy‡, +ve Chvostek's sign§	2.00	0.72	1107	222	3.0	72
7	15 ♀	Pakistani	Severe carpopedal spasms	Proximal myopathy‡, +ve Chvostek's sign§	1.48	1.43	723	390	2.2	83
8	14 ♀	Pakistani	Carpopedal spasms, pain in knees, difficulty climbing stairs	Severe knock-knee deformity with 20 cm separation of medial malleoli, +ve Chvostek's sign§	1.78	1.27	3688	1173	<2.0	Lost
9	14 ♀	Pakistani	Pain in knees, difficulty climbing stairs	Severe knock-knee deformity with 22 cm separation of medial malleoli, +ve Chvostek's sign§	1.76	0.80	10202	703	<2.0	28

ALP, alkaline phosphatase; PTH, parathyroid hormone.

*Serum ALP concentrations vary with age and stage of pubertal development.

†Serum 25(OH)D concentrations <5 ng/ml are associated with rickets or osteomalacia and those >5 ng/ml but <15 ng/ml are considered to be insufficient.

‡Inability or difficulty in standing up unaided from a squatting position.

§Twitching of the facial muscles in response to gentle tapping over the skin anterior to the earlobe and below the zygomatic arch.

of serum alkaline phosphatase activity for the age (except in cases 2, 5, and 7), secondary hyperparathyroidism leading to

Figure 1 Severe knock-knee (genu varum) deformity in case 8.

hypophosphataemia (except in cases 7 and 8), and formation of normal, or supranormal serum concentrations of 1,25 dihydroxycholecalciferol (except in case 5). The presenting symptoms in all patients resolved after treatment with oral vitamin D. However, unlike in younger children, genu valgum or genu varum (fig) do not fully correct after treatment with vitamin D after the long bone epiphyses have closed; the residual deformities require surgical correction.

Vitamin D is necessary for adequate bone mineralisation and its deficiency results in rickets in children and osteomalacia in older adolescents and adults. In humans, the main source of vitamin D is that produced by the action of solar ultraviolet light B radiation acting on 7-dehydrocholesterol in skin. Small amounts are also derived from dietary sources: oily fish, eggs, and fortified foods, such as margarine and breakfast cereals. The Committee on Medical Aspects of Food Policy (COMA)³ recommends that all pregnant women, lactating mothers, and children up to the age of 5 years who are at risk of vitamin D deficiency should be encouraged to take supplements. However, there are no

recommendations for vitamin D supplementation in adolescents, although the COMA does recommend provision of vitamin D supplements to vulnerable older children.

Adolescence is a critical period of skeletal mineralisation, when over 35% of the peak bone mass (PBM) of a mature adult is accrued during the four years surrounding the peak pubertal growth spurt.⁴ It is widely accepted that subjects who attain a lower PBM at maturity have a higher risk of sustaining osteoporotic fractures in later life.⁵ Thus, inadequate skeletal mineralisation secondary to vitamin D deficiency during this period might compromise the acquisition of PBM and thus potentially increase the risk of osteoporosis in later life. We therefore recommend that a prospective study is needed to determine the prevalence of vitamin D deficiency among adolescents whose exposure to sunshine is limited for cultural and religious reasons.

S Crocombe, M Z Mughal

Department of Paediatric Medicine, St Mary's Hospital
for Women & Children, Hathersage Road,
Manchester M13 0JH, UK

J L Berry

University Department of Medicine, Manchester Royal
Infirmary, Manchester M13 9WL, UK

Correspondence to: Dr Z Mughal, Consultant
Paediatrician & Honorary Senior Lecturer in Child
Health, Department of Paediatric Medicine, St Mary's
Hospital for Women & Children, Hathersage Road,
Manchester M13 0JH, UK; zulf.mughal@man.ac.uk

References

- 1 **Mughal MZ**, Salama H, Greenaway T, *et al.* Lesson of the week: Florid rickets associated with prolonged breast feeding without vitamin D supplementation. *BMJ* 1999;**318**:39–40.
- 2 **Ashraf S**, Mughal MZ. The prevalence of rickets among non-Caucasian children. *Arch Dis Child* 2002;**87**:263–4.
- 3 **Department of Health**. *Nutrition and one health: with particular reference to calcium and vitamin D* Report on health and social subjects; 49. London: HMSO, 1998.
- 4 **Bailey DA**. The Saskatchewan Pediatric Bone Mineral Accrual Study: bone mineral acquisition during the growing years. *Int J Sports Med* 1997;**18**:191–4.
- 5 **Seeman E**. Reduced bone density in women with fractures: contribution of low peak bone density and rapid bone loss. *Osteoporos Int* 1994;**4**:15–25.

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Milk fats and asthma

Studies performed mainly on adults have suggested that diet may influence asthma. Fruits, vitamin C, vitamin E, and beta-carotene may have a protective effect. Now a birth cohort study in the Netherlands (AH Wijga and colleagues. *Thorax* 2003;**58**:567–72) has provided evidence that in pre-school children the consumption of products containing milk fat might be protective.

The cohort consisted of 2978 children born in three different regions of the country between July 1996 and October 1997. Dietary data were collected when the children were 2 years old and related to data from postal questionnaires on respiratory symptoms at 3 years of age. Recent asthma at age 3 years was less prevalent (3.4% vs 5.6%) in children who at age 2 years had consumed full cream milk every day compared with other children. A similar reduction in recent asthma (1.5% vs 5.1%) was found in children who ate butter every day. The prevalence of recent wheeze was also reduced by daily consumption of milk products (13.7% vs 18.4%) or of butter (7.7% vs 15.4%). Rates of asthma or wheeze were also reduced with daily consumption of brown bread but were not affected by consumption of fruits, vegetables, margarine, or fish.

It is possible that the observed changes in intake of milk fats may be indicators of other aspects of lifestyle but the authors of this paper have been unable to identify such confounding factors. It is also possible, but unproved, that increased intake of saturated fat and reduced intake of polyunsaturated fatty acids might protect against the development of atopy.