conclusion is the author's "impression that the increased incidence of peanut or nut allergy is real" and the statement that "there has been a considerable increase in the rate of referrals for food allergy." Even more disturbingly, Hugh A Sampson cites this study in his editorial in support of his conclusion that "the prevalence of peanut and nut allergy is increasing."<sup>2</sup>

While the incidence of nut allergy may indeed be rising, we believe that authors have a responsibility not to overstate their case, particularly on issues that are likely to be of interest to the media. Ewan should provide us with the evidence that led her to conclude that nut allergy is becoming common so that we can decide on this important issue, for ourselves and for our patients.

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- 1 Ewan PW. Clinical study of peanut and nut allergy in 62 consecutive patients: new features and associations. BMJ 1996;312:1074-8. (27 April.)
- 2 Sampson HA. Managing peanut allergy. *BMJ* 1996;**312**:1050-1. (27 April.)

## Reduced exposure might increase allergic sensitisation

EDITOR,—Pamela W Ewan makes the important statement that the incidence of peanut and nut allergy is rising and that sensitisation seems to occur early in life.1 Regrettably, she does not provide evidence to anv back her recommendation that "young allergic children should avoid peanuts and nuts to prevent the development of this allergy" and her extraordinary suggestion that avoidance should be practised until the age of 7. Hugh A Sampson, in the accompanying editorial, makes similar recommendations and further suggests that mothers who are breast feeding should eliminate peanuts from their diet.2

Firstly, there is no evidence that avoiding foods during lactation or early childhood prevents allergic sensitisation to these foods. Indeed, in certain cultures that consume large quantities of peanuts, peanut allergy seems to be less of a problem than it is in Britain. Secondly, allergic sensitisation may occur in utero, but no advice is given on maternal diet during gestation. Thirdly, exposure to peanuts and other food allergens during lactation and childhood may be important in the development of immunological tolerance and may prevent allergic sensitisation to these foods. Finally, avoidance measures would serve only to reduce exposure to peanuts to low levels, and this could paradoxically increase allergic sensitisation to peanuts: low dose exposure to allergens (rather than high dose exposure) favours production of IgE,3 and as little as 1 µg of inhaled allergen a year may be sufficient to induce allergic sensitisation via the airways.4

Prospective data comparing consumption of peanuts by children who are allergic to them and by atopic controls are required before broad policy recommendations are made. History contains far too many examples of uninformed health policies that were based on insufficient data and achieved unintended effects.

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- 1 Ewan PW. Clinical study of peanut and nut allergy in 62 consecutive patients: new features and associations. BMJ 1996;312:1074-8. (27 April.)
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## Author's reply

EDITOR,—I am aware that various creams (for eczema, cracked nipples, and massage) contain arachis (peanut) oil. While these are possible sources of sensitisation, it has not yet been established whether this oil is allergenic. One study showed no effect of giving arachis oil orally to patients who were allergic to peanuts,¹ whereas another showed that it exacerbated eczema.² Such products have exacerbated eczema in some of my patients. More data are needed, and research is in progress. The makers of chamomile ointment are reformulating their product without arachis oil.

John A Wilson and Sheila Jones and Ian Jones question my suggestion that the incidence of peanut allergy has increased. This is based on 16 years' experience in major allergy centres. The rise in referrals began in the early 1990s. Studies are under way to measure prevalence, but one difficulty will be that no previous data exist. If Wilson has population based data on prevalence then he should publish them. Some of the rise will be due to increased public awareness, but I believe that a real change has also occurred. I have data showing that the age at sensitisation is falling, and most of the 62 patients on whom I reported had become allergic by the age of 2—that is, the cases were of recent onset.

Wilson questions the value of diagnosis and management. At the allergy clinic our approach is two pronged. Avoidance is the key, and expert advice is essential since peanuts and nuts are now often hidden in foods. Many of the children who died knew that they were allergic (exactly as Wilson describes), practised avoidance, but had not had professional advice. Secondly, we provide drugs for self treatment of reactions after inadvertent ingestion. This does not always mean adrenaline for injection (unpublished data).

My advice was that young atopic children (not all children) should avoid peanuts and nuts because of the strong association (96%) with other atopic disease. I agree with Gideon Lack and Jean Golding, however, that more studies are needed. I postulate that factors that are important in the increase in peanut allergy are the increase in atopic disease and early and increased exposure to peanuts. Avoidance can reduce sensitisation to food allergens,<sup>3</sup> but the effects of the dose of antigen on the production of cytokines are complex (S M Hugh et al, unpublished findings).<sup>4</sup> Genetic and other factors are clearly important in induction of the Th2 phenotype.

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## Sesame allergy is also a problem

EDITOR,-Hugh Sampson's editorial on managing peanut allergy omits one important point1: medical identification bracelets should be worn at all times. Unsurprisingly, attention focuses on peanuts,2 but sesame allergy, although less common than peanut allergy, can be every bit as severe. Sesame is used extensively in the food industry, and the seeds present a danger because of their versatility.3 I report here my most recent allergic reaction to sesame. I was looking forward to an evening out with my daughter in law: a meal in a restaurant and then a visit to a theatre. I telephoned the restaurant to advise it of my serious allergy and then packed my "survival kit" (injectable adrenaline, an adrenaline inhaler, and a note that backs up my Medic-Alert bracelet). After my first anaphylactic shock in 1981 I was issued with an American kit containing a pre-filled adrenaline syringe and tablets of chlorpheniramine maleate. Eventually, this was replaced with the standard injectable adrenaline that is issued by the NHS. I had never felt comfortable with this: it had to be assembled before use, and I wondered how I would cope in an emergency.

I reminded the restaurant staff about my allergy; I always feel a bit uneasy when eating out. A glass of champagne calmed my nerves, and then the soup arrived. I tend to avoid soup when eating out, but this was made in house and I was assured that it did not contain sesame. It did. Within seconds my mouth started to tingle, my ears burnt, my neck flushed, and my hands started to itch—characteristic signs of an allergic reaction. I rinsed out my mouth and tried to assemble the syringe. Impossible! Could anyone, in such a stressful situation? I cursed the syringe, abandoned it, and used my inhaler instead. The restaurateur was frantic: "Is there a doctor in the house?" There wasn't.

We sat outside and waited for an ambulance. I was gasping for breath and wanted to be sick. I was. A warm glow came over me, and everything just faded away. In the ambulance the paramedics clamped an oxygen mask on my face. My son, who must have driven like Fangio, arrived at the hospital just as the casualty officer was preparing an injection. I smiled when I heard his voice: "Excuse me, my mother is allergic to sesame; it's used in some drugs." We had done our homework.

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## Value of ECGs in identifying heart failure due to left ventricular systolic dysfunction

EDITOR,—We wish to reply to the letters<sup>1</sup> about our short report.<sup>2</sup>

We are pleased to learn that Suresh Khandekar and colleagues are following our example in using electrocardiography to identify heart failure due to left ventricular systolic dysfunction, but we do not understand why they use an automated report for interpreting electrocardiograms. While we appreciate Kamlesh Khunti and Robert McKinley's concerns about the interpretation of electrocardiograms in general

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