

PostScript

LETTERS TO THE EDITOR

Treating sleep apnoea is cost effective

The paper by George¹ showing a decrease in road accidents with continuous positive airway pressure (CPAP) therapy in the July issue of *Thorax* was accompanied by an editorial quantifying the cost savings based on USA accident costs.² As most sleep services in the UK are having difficulty funding CPAP treatment, there is a need to put the cost benefits into a UK context, which we have done using George's data.

According to the UK Department of Transport official figures for 1999,³ fatal accidents cost £1 253 140 each, accidents with personal injury cost £49 800, and property damage only accidents cost £1300 each. George's study was performed in Ontario and examined accidents involving either personal injury, more than \$500 of damage, or a traffic violation. In 1999 Ontario had 221 962 such accidents, of which 763 were fatal, 55 764 were associated with personal injury, and 165 435 with property damage only.⁴

Thus, using George's data,¹ treating 500 patients with CPAP for 5 years would prevent 1.03 fatal accidents at a saving of £1.292 million, 75.4 personal injury accidents at a saving of £3.753 million, and 224 property damage accidents at a cost saving of £0.290 million. The total accident related cost saving would therefore be £5.335 million compared with a treatment cost of £0.4 million (500 CPAP units at £300 and £100 per annum for consumables and follow up costs), giving a saving of £4.935 million at a 12.3-fold return on pounds spent.

These savings do not take into account those from the marked and now well documented improvements in work performance, quality of life,⁵ and blood pressure,⁶ and decrease in hospitalisation costs⁷ resulting from CPAP treatment. It is time health service planners recognised these benefits and this cost efficacy, and that sleep apnoea services were accorded the appropriate priority in the health budget.

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The editors will decide as before whether to also publish it in a future paper issue.

- Bahammam A, Delaive K, Ronald J, et al. Health care utilization in males with obstructive sleep apnea syndrome two years after diagnosis and treatment. *Sleep* 1999;**22**:740-7.

Polyunsaturated fats and asthma

The paper by Haby *et al* on the prevalence and risk factors for asthma in preschool children¹ appeared in Australian newspapers under the headline "Fatty diet may double risk of childhood asthma". But the authors have not logically established their assertion that 17% of the cases of recent asthma in Australian rural children 3-5 years old can be attributed to a high intake of polyunsaturated fats. Five reasons make this conclusion improbable.

(1) The questionnaire was sent by post and patients were not helped professionally to complete it. Only two questions were asked about diet and it seems from table 1 in the paper that there were three possible answers for bread spread and five for frying oil. Predominantly polyunsaturated fats/oils would have been in the last "other" group, presumably used by parents who weren't sure of the answer. No questions were asked about the rest of the diet, either possibly allergenic foods or about fish, the major source of omega-3 polyunsaturated fatty acids. In earlier papers some of the same authors reported an inverse relationship between total fish consumption and bronchial reactivity² or between fresh (not canned) oily fish and asthma.³

(2) At least five randomised controlled intervention trials (some in this journal) have found no benefit in patients with asthma given high intakes of omega-3 polyunsaturated fish oils⁴⁻⁸ which Haby *et al* think of as the good oil against which omega-6 polyunsaturates such as linoleic acid could compete.

(3) Morris *et al*⁹ have given high intakes of omega-6 polyunsaturated fatty acids to patients with asthma for 8 weeks with alternating control diets containing the same amount of saturated and monounsaturated fats. There was no deterioration in symptoms, bronchodilator usage, or lung function tests.

(4) In 77 866 US nurses no relation could be found between doctor diagnosed asthma and dietary intake of fatty acids by a group of epidemiologists experienced in quantifying intake of different fatty acids.¹⁰

(5) In vitro work indicates that leukotrienes from omega-6 or omega-3 fatty acids do not have different effects on bronchial constriction,¹¹ and those derived from eicosapentaenoic acid, an omega-3 fatty acid, do not have a dampening effect on eosinophils, the predominant effector in asthma.¹²

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Authors' reply

We thank Dr Truswell for his comments on our paper which investigated a wide range of risk factors for asthma in children of preschool age. We certainly agree with the caution he advises in translating findings from an observational study to clinical or public health practice. We advocate that evidence from randomised controlled trials is an essential prerequisite to such advice. However, we do not believe that the data he cites invalidate the observation we have made on the association between the use of polyunsaturated oils and spreads and the presence of asthma.

We acknowledge that our questionnaire was a crude tool for assessing dietary fatty acid intake and that the observed association with polyunsaturated oils and spreads may not be due to the balance of omega-3 and omega-6 fatty acids. Nevertheless, there was an observed association and this needs to be

explained. It is possible that the effects are attributable to differences in other related dietary constituents or to unmeasured confounding. Our view was that the relative intake of omega-3 and omega-6 fatty acids was the most plausible explanation. Our previous observation in children of primary school age that those with asthma reported a reduced intake of fish lends some support to this explanation.¹

The lack of effect of short term omega-3 or omega-6 supplementation in subjects with established asthma does not preclude the potential for benefit arising from long term modification of fatty acid intake. Similarly, the lack of effect in the adults studied in the Nurses Health Study² does not preclude the potential for adverse effects in young children.

Our findings, together with other observational data demonstrating higher consumption of polyunsaturated fats among children with atopic disease,³ should alert the scientific community to the need for further research, particularly long term randomised controlled trials, to elucidate the role of fatty acid consumption in the expression of asthma in children.

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Polyunsaturated fats and asthma

Haby *et al*¹ found that the consumption of polyunsaturated fats was a risk factor for asthma in Australian preschool children and inferred from this that modification of polyunsaturated fat consumption may reduce the incidence of asthma. However, being a cross sectional epidemiological study, a causal relationship cannot be established and the results need to be interpreted with great caution. Other data not discussed by the authors also need to be highlighted.

Firstly, the hypothesis that there may be a relationship between polyunsaturated fatty acid intake and asthma was initially based on the "observation" that the prevalence of asthma in Australia had increased concurrently with an increasing intake of polyunsaturated fats in the 1980s. However, this has not been the case since the 1990s. Current levels of polyunsaturated fats are low, with only 3.7–4.0% of energy coming from polyunsaturated fats in 2–7 years olds who

participated in the 1995 national dietary survey.² These levels are below the National Health and Medical Council of Australia new draft recommendations for adults (6–8% of energy from n-6 polyunsaturated fats).³ Factors involved in the fall in polyunsaturated fat intake include the introduction of margarines with a lower fat content, an increase in the monounsaturated fat content of margarines, and a reduction in the overall consumption of margarine.

Secondly, the questions asked by Haby *et al* with regard to dietary intake do not adequately assess polyunsaturated intake. While reliability of these questions was shown in the paper, their validity was not. The intake of fats and oils on bread or toast and the type used in food preparations is not a good predictor of polyunsaturated fat intake. Indeed, fats and oils account for only approximately 23% of the total polyunsaturated fat intake of 2–11 year olds in Australia.⁴ Furthermore, it is interesting that the paper did not report any findings relating to fish intake which, due to their high n-3 content, may offset any effect of n-6 polyunsaturated fats.

Thirdly, interventional studies are more likely to establish or refute a causal relationship between dietary fat intake and asthma. The results from randomised controlled studies in adults and children with asthma, including one conducted by the Institute of Respiratory Medicine in Sydney, have repeatedly failed to show any change with modification of dietary polyunsaturated fat intake.^{5,6}

Fourthly, the biological mechanism proposed by the authors focuses on the effects of dietary polyunsaturated fats in increasing prostaglandin E₂ production and its possible role in promoting airway inflammation. However, data from clinical studies suggest, on the contrary, that prostaglandin E₂ has a protective effect on asthmatic bronchoconstriction.⁷ Other possible mechanisms, such as the modification of leukotriene synthesis, have also not been shown to have a significant overall anti-inflammatory effect.⁸ Hence, from our current knowledge, a significant biologically plausible mechanism to interpret this association as a causal relationship is difficult to find.

So, while the association between dietary polyunsaturated fats and asthma in children is interesting, the overall evidence argues against a causal relationship. This should be taken into account in interpreting and publicising these results to avoid drawing premature conclusions about dietary modification and contributing to a negative impact on other public health outcomes.

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NOTICE

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