TREATING SLEEP APNOEA IS COST EFFECTIVE

The paper by George1 showing a decrease in road traffic crashes associated 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. 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, fatal accident cost £1 253 140 each, accidents with personal injury cost £49 800, and property damage only accidents cost £1 300 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.2

Thus, using George’s data, 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 £1.292 million, 75.4 personal injury accidents at a saving of £1.292 million, 224 property damage accidents at a saving of £1.292 million, and 163 455 property damage accidents at a saving of £1.292 million, 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, and blood pressure, and increase in hospitalisation costs 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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1 George CP. Reduction in motor vehicle collisions following treatment of sleep apnoea with nasal CPAP. Thorax 2001;56:508–12.

POLYSATURATED FATS AND ASTHMA

The paper by Haby et al on the prevalence and risk factors for asthma in preschool children3 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 cases of recent asthma in Australian rural children 3–5 years old can be attributed to high intake of polysaturated fats. Five reasons make this conclusion improbable.

(1) The questionnaire was sent by post and parents 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 polysaturated 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 polysaturated fatty acids. In earlier papers some of the same authors reported an inverse relationship between total fish consumption and bronchial reactivity, or between fresh (not canned) oily fish and asthma.4

(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 polysaturated fish oils,5 which Haby et al think of as the good oil against saturated oils and spreads and the presence of cysteine-containing leukotrienes in the guinea-pig lung strip. Eur J Pharmacol 1986;120:157–162.

(3) Morris et al have given high intakes of omega-6 polysaturated 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.6

(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.7

(5) In vitro work indicates that leukotrienes from omega-6 or omega-3 fatty acids do not have different effects on bronchial constriction,8 and those derived from eicosapentaenoic acid, an omega-3 fatty acid, do not have a diminishing effect on eosinophils, the predominant effector in asthma.9

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References

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 authors’ emphasis on the need for further research into the effects of dietary fatty acids on asthma. We certainly agree with the authors’ emphasis on the need for further research into the effects of dietary fatty acids on asthma. We certainly agree with the authors’ emphasis on the need for further research into the effects of dietary fatty acids on asthma. We certainly agree with the authors’ emphasis on the need for further research into the effects of dietary fatty acids on asthma. We certainly agree with the authors’ emphasis on the need for further research into the effects of dietary fatty acids on asthma.

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Log on to our website (www.thoraxjnl.com) 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, and blood pressure, and decrease in hospitalisation costs 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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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. 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 Study 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, 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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References

Polysaturated fats and asthma

Haby et al 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. 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). Factors involved in the fall in polyunsaturated fat intake include the introduction of margarines with a lower fat content, an increase in the mono and polyunsaturated 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. 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.

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, the data from clinical studies suggest, on the contrary, that prostaglandin E2 has a protective effect on asthmatic bronchoconstriction. Other possible mechanisms, such as the modification of leukotriene synthesis, have also been shown to have a significant overall anti-inflammatory effect. 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 publishing 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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References

NOTICE

Scadding-Morrison Davies Joint Fellowship in Respiratory Medicine 2002

This fellowship is available to support visits to medical centres in the UK or abroad for the purpose of undertaking studies related to respiratory medicine. Applications are invited from medical graduates practising in the UK, including consultants and irrespective of the number of years in that grade. There is no application form, but a curriculum vitae should be submitted together with a detailed account of the duration and nature of the work and the centres to be visited, confirming that these have agreed to provide the facilities required. Please state the sum of money needed for travel and subsistence. A sum of up to £15 000 can be awarded to the successful candidate, or the sum may be divided to support two or more applications. Applications should be sent to Dr I A Campbell, Secretary to the Scadding-Morrison Davies Fellowship, Llandough Hospital, Penarth, Vale of Glamorgan CF64 2XX, UK by 31 January 2002.