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

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References

Authors’ reply
We thank Dr Trusswell for his comments on our paper which investigated a wide range of risk factors for asthma in children of pre-school 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 about 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
Polyunsaturated fats and asthma

Haby et al. demonstrated 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 intake might 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's current dietary guidelines 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 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. 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, intervention 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 the n-6 content, thus 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.

Other possible mechanisms, such as the modification of leukotriene synthesis, have also not 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.

References


NOTICE

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