latter were prepared using defatted BSA, according to Mahoney et al. [11]. After 48 h, [3H]thymidine (0.2 μCi/well) was added to each well and the cells incubated for a further 18 h. The cells were harvested on to glass-fibre discs, which were washed and dried before measurement of radioactivity.

The maximal [3H]thymidine incorporation obtained in the presence of LPS was 14.9 ± 2.5% (mean ± S.E.M. from four separate cell preparations) of that obtained in the presence of Con A. The results of the effect of fatty acids upon LPS-stimulated lymphocyte proliferation are shown in Table 1. The effect of fatty acids on Con A-stimulated lymphocyte proliferation are shown for comparison. B lymphocyte proliferation was inhibited by all fatty acids except 14:0; the greatest inhibition was caused by 20:4, n-6, 18:2, n-6, 20:5, n-3 and 22:6, n-3, and the least inhibition was caused by 14:0 and 16:0. The two fish-oil-derived fatty acids tested caused similar degrees of inhibition and both were significantly less inhibitory than 20:4, n-6. These results indicate that a wide range of fatty acids are capable of inhibiting B lymphocyte proliferation in vitro. It must be noted, however, that all fatty acids, except 14:0, caused significantly less inhibition of B lymphocyte proliferation than that of T lymphocyte proliferation (Table 1), indicating that B lymphocytes are less susceptible to the inhibitory effects of fatty acids. This may result from differing abilities of the two cell types to incorporate or metabolize exogenously supplied fatty acids. Whatever the basis of the different susceptibilities of T and B cells, it is clear that fatty acids in general have an immunosuppressive effect but that the effect is greatest with polyunsaturated fatty acids.

It is possible that the fatty acid-induced inhibition of lymphocyte proliferation is due to modification of the fatty acid composition of membrane phospholipids, which could alter fluidity of the membrane. Such changes could effect many properties such as membrane potential, affinity of receptors or signal transduction. Whatever the mechanism involved, these results suggest that proliferation of both T and B lymphocytes, and hence an immune response, could be altered by dietary lipid manipulation.

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A longitudinal study of plasma n-3 fatty acid levels in a family with X-linked retinitis pigmentosa

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A number of years ago we identified a family affected by the X-linked form of retinitis pigmentosa (RP), a hereditary eye disease which primarily appears to involve the rod photoreceptor cells. This large kindred, the 'G family', with five affected males in two generations, was analysed for plasma lipoproteins and fatty acids. It was found that the affected men had lower levels of a particular fatty acid, docosahexaenoic acid (DHA) ([C22,6n-3], than did their non-affected relatives [1, 2].

DHA is interesting in that it comprises about 50% of the fatty acids in the rod outer segment membranes, the highest concentration in any tissue in the body [3]. It has been shown that animals deprived of DHA or its precursors in the n-3 synthetic pathway show reduced visual function [4-6]. Thus it is possible that the low levels of DHA in affected men in our X-linked family have an adverse effect on the retina, and contribute to the pathogenesis of the disease. Following on from these observations, we have now shown that a number of other X-linked families and one autosomal dominant family also show low levels of DHA [2]. These results have been confirmed in an American study [7], and low DHA has also been described in a large Usher syndrome family (RP plus congenital deafness) [8].

It is possible, however, that the low levels of plasma DHA are due not to a biochemical defect but to some extrinsic factor, such as diet. For this reason we analysed the diet of our patients and their relatives over a period of 1 week; no significant dietary differences were found between affected and non-affected family members which could explain the differences in DHA levels. A related study, on a cross-section of RP patients, also showed no correlation between habitual exercise, employment, and plasma lipid levels [9]. If low DHA is the result of an intrinsic biochemical defect in our patients, it might be expected to persist over a period of time. In this paper we report on the determination of plasma fatty acid levels in the G family four times over a period of 7 years, and a comparison of the analyses.

Fasting blood samples were obtained from family members, plasma prepared, lipids extracted using chloroform-methanol, and fatty acids methylated as previously described [2]. In the first three analyses, samples were analysed by packed column gas chromatography on a Silar 10C column, using a temperature programme of 200°C for 4

Abbreviations used: RP, retinitis pigmentosa; DHA, docosahexaenoic acid.
Polyunsaturated fatty acid metabolism in miniature poodles with an inherited retinal degeneration

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Retinitis pigmentosa (RP), a hereditary disease of the retina characterized by progressive night blindness and tunnel vision, has been the subject of many studies, but its aetiology is still unknown. We have been particularly interested in investigating the abnormal plasma lipid levels found in many types of RP [1, 2] as it is known that certain lipids are required for optimal retinal function. In particular, the retina has a high concentration of docosahexaenoic acid (DHA) \( (\text{C}_{22:6, n-3}) \) [3] and animals deprived of this fatty acid or its precursors in the \( n-3 \) family have decreased visual function [4–6]. In two types of RP, X-linked and autosomal dominant, we found significantly decreased levels of DHA in affected


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