

## Plasma lipid fractions as indicators of dietary $\alpha$ -linolenic and linoleic acid

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Many health benefits of an increased intake of n-3 fatty acids have been claimed. To study the effects of these fatty acids on diseases such thrombosis, inflammation, blood pressure and mental and visual development, one needs an accurate measure of consumption. The most common dietary assessment methods (weighed food records and dietary recall questionnaires) have widely known inherent errors. Therefore, it was hypothesised that one or more plasma lipid fractions could reflect dietary intake of  $\alpha$ -linolenic (ALA, C18:3 n-3) and linoleic acid (LA, C18:2 n-6).

The effects of dietary intake of ALA-rich and LA-rich diets on the fatty acid profiles of plasma triglycerides, cholesteryl esters, and phospholipids were investigated. Twelve healthy male subjects aged 19 to 35 years commenced a 14 day stabilisation period in which they consumed a diet typical of the Australian diet (37% energy (en) from fat; 16% en from saturated, and 5.8% en from n-6 polyunsaturated). The subjects then commenced a 42 day experimental period in which they were randomly allocated to an ALA-rich diet (n = 6) ( $10.2 \pm 0.6$  g/d) or a LA-rich diet (n = 6) ( $22.0 \pm 0.0$  g/d). Both diets were identical in nutritional composition except for the ALA and LA content.

An increase in consumption of ALA from the stabilisation period to the experimental period of  $1.4 \pm 0.5$  grams per day (g/d) to  $10.2 \pm 0.6$  g/d resulted in a significant increase ( $P < 0.05$ ) in ALA in all plasma lipid fractions. It was expected that the ALA rich diet would increase the percentage of plasma eicosapentaenoic acid (EPA) and docosapentaenoic acid (DPA) by desaturation and elongation of ALA (1). A significant increase ( $P < 0.05$ ) occurred in all fractions for EPA. For DPA however, a significant increase was found only in the phospholipid fraction. As a linear relationship was found between dietary ALA intake and the concentration of ALA in the plasma lipid fractions, these fractions are indicative of dietary ALA.

In contrast, an increase in consumption of LA from  $15.7 \pm 2.1$  g/d to  $22.0 \pm 1.0$  g/d did not result in a significant increase in LA or its longer chain metabolite arachidonic acid (AA) in any plasma lipid fraction. Our results therefore suggest that plasma lipid fractions cannot be used as indicators of dietary LA. A possible explanation comes from a study by Emken et al (2) who found that a two-fold increase in LA-intake caused a 40% decrease in incorporation of LA into the plasma fractions. Increased fatty acid oxidation and storage stimulated by increased dietary LA appears to be responsible.

1. Dumm GD, Brenner RR. Oxidation and desaturation of  $\alpha$ -linolenic, linoleic, and stearic acids by human liver microsomes. *Lipids* 1975;10:315-7.
2. Emken EA, Adolf RO. Dietary linoleic acid influences desaturation and acylation of deuterium-labelled linoleic and  $\alpha$ -linolenic acid in young adult males. *Biochim Biophys Acta* 1994;1213:277-88.