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## **Invited commentary**

## Olive oil, high-oleic acid sunflower oil and CHD

The low levels of CHD morbidity and mortality in Mediterranean regions is partly attributed to dietary factors. Olive oil is a key component of the traditional Mediterranean diet, therefore there has been intensive investigation to determine how olive oil mediates this effect. CHD has a very complex pathophysiology characterised by atherosclerosis, which is the result of aberrant lipoprotein metabolism and a local inflammatory response in the vascular endothelium, and thrombosis. Olive oil is a rich source of monounsaturated fatty acids (MUFA) and antioxidant phenolic compounds. Both of these components mediate the cardio-protective effects associated with olive oil in a number of ways. Traditionally the cardio-protective effect of the Mediterranean diet was ascribed to the hypocholesterolaemic effect of dietary MUFA. Numerous intervention studies have shown that the substitution of dietary saturated fatty acids (SFA) for MUFA significantly reduces total LDL cholesterol concentrations (Mensink & Katan, 1989). More recently, studies have shown that diets rich in MUFA also have favourable effects on triacylglycerol (TAG)-rich lipoprotein metabolism, coagulation and inflammation, all of which have the potential to contribute to the beneficial effects ascribed to the Mediterranean diet. A cross-cultural study and a dietary intervention trial has shown that young men consuming greater amounts of MUFA rather than SFA metabolise postprandial TAG more efficiently (Roche et al. 1998; Zampelas et al. 1998). This was also associated with lower levels of coagulation factor VII activity, which would confer an anti-thrombotic effect. Yaqoob et al. (1998) also demonstrated that a dietary MUFA significantly reduced the concentration of intercellular adhesion molecule 1 (ICAM-1). The adhesion molecule ICAM-1 is involved in the inflammatory response of the vascular endothelium that leads to the formation of the atherosclerotic plaque.

The nutritional benefits of MUFA-rich diets are well accepted, but olive oil is an expensive commodity. High-oleic acid sunflower oil (HOSO) represents a cheaper source of dietary MUFA and it could be particularly valuable in enriching the diets of Northern Europeans with MUFA at the expense of SFA. Nevertheless, olive oil is an important source of potent anti-oxidant phenolic compounds, and the health benefits of these non-nutritive dietary components should not be overlooked. The phenols in olive oil are potent inhibitors of reactive oxygen species and they protect against lipid and lipoprotein oxidation (Fito *et al.* 2000), which would otherwise contribute to the progression and pathogenesis of atherosclerosis. In this issue of the *British Journal of Nutrition*, Oubiña *et al.* (2001) report a study comparing the effect of extra virgin

olive oil (EVOO) and HOSO on serum and LDL-peroxides, eicosanoid production and thrombosis in postmenopausal women. The study demonstrated that serum peroxides, plasma \alpha-tocopherol and thromboxane B2 levels were higher after the HOSO diet compared with the EVOO diet. Generally olive oil contains lower amounts of  $\alpha$ -tocopherol compared with vegetable oils (Owen et al. 2000). Accordingly, the HOSO oil provided more  $\alpha$ -tocopherol, which explains the greater concentrations of plasma αtocopherol after the HOSO diet. The EVOO oil supplied significantly greater amounts of polyphenols, sterols and squalene, and this dietary treatment was associated with lower serum peroxide levels after the EVOO diet. However, there was no significant difference between dietary treatments on LDL peroxides. The authors measured several indices of the thrombogenic ratio, including platelet-rich plasma and urinary thromboxane B2 and urinary 6-keto prostaglandin  $F_{1\alpha}$ , the stable metabolite of prostaglandin G<sub>2</sub>, which is anti-thrombogenic. The olive oil diet significantly lowered platelet rich plasma thromboxane B<sub>2</sub> concentrations. The authors ascribed this effect to a combination of the greater levels of linoleic acid in the HOSO and the greater amount of phenols in the EVOO.

To conclude, the paper by Oubiña et al. (2001) shows that the minor non-nutritive components of EVOO may have potential beneficial effects on serum peroxide production and thrombogenesis. The study design did not allow for a control or low-MUFA dietary treatment, which would have allowed investigation of the relative efficacy of the two MUFA-rich diets, with and without the phenolic compound that are exclusive to EVOO. Nevertheless, the paper presents very interesting data. Furthermore, it is important to realise the heterogenous nature of the phenolic compounds present in olive oil. Therefore the relative efficacy of the individual phenolic compounds should be defined in vivo.

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