PROCEEDINGS
RAPESEED OIL SYMPOSIUM

Scandinavian Journal of Nutrition/Näringsforskning Vol 37:52-54, 1993

Dietary fat quality and serum lipoproteins – an update

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Summary

When various fats are fed to humans, differences in the chain lengths and the number and geometry of the double bonds in fatty acids will induce marked differences in the concentrations of lipids and lipoproteins in fasting blood plasma. Relative to dietary carbohydrates, the saturated fatty acids lauric, myristic and palmitic acid raise both LDL and HDL cholesterol and lower VLDL and cholesterol triglycerides. Stearic acid has little effect on LDL but may lower HDL somewhat relative to most other fatty acids. Oleic and linoleic acid raise HDL and lower LDL somewhat when substituted for dietary carbohydrates, both effects being more pronounced for linoleic acid. Both unsaturates lower triglycerides. *Trans* isomers of oleic acid lower HDL and raise LDL and lipoprotein (a). The fatty acids in unhydrogenated fish oil very potently lower triglycerides but may somewhat raise LDL.

Thus under metabolic-ward conditions, when body weight is forcibly kept constant, replacement of hard fats rich in saturated or *trans* fatty acids by unsaturated oils will produce a more favourable lipoprotein profile than replacement by carbohydrates. However, under conditions of unlimited food intake high-oil diets could lead to obesity, which would undo their favourable effects.

Introduction

The major differences between different dietary fats are the chain lengths of their constituent fatty acids and the number and geometry of the double bonds contained in these. When various fats are fed to humans these differences induce marked differences in the concentrations of plasma lipids and lipoproteins in the fasting state (1). These effects set off dietary lipids from other nutrients. Thus the nature and concentration of fasting plasma proteins is not affected to any marked extent by the amount and composition of protein in the diet as long as minimal protein and amino acid needs are met. The same holds for glucose: populations eating high- or low-carbohydrate diets have roughly similar fasting blood sugar levels as long as body mass index and physical activity are comparable. In contrast, differences in amount and type of fat in the diet can induce differences of 50% or more in the serum cholesterol level.

Despite intense research efforts, the mechanisms that link dietary fat quality with plasma lipoprotein concentrations have not yet been clarified (2). However, there are plenty of empirical data to allow a reliable prediction of how plasma lipid and lipoproteins in man will change when the amount and quality of fat in the diet are changed.

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Saturates, oleic acid, and linoleic acid

The fatty acids that occur most commonly in the human diet are listed in Table 1. It is for these fatty acids that most of the information regarding lipids and lipoproteins is available. We have summarized the results of 27 trials on dietary fatty acids and plasma lipoproteins that met certain criteria in a meta-analysis (1); the major results of that analysis are given in Figure 1. Figure 1 indicates the changes in plasma lipids and lipoproteins that take place when 1% of dietary energy in the form of carbohydrate is replaced by a particular class of fatty acids. Carbohydrates are used as a starting point because there does not exist such a thing as "the effect of polyunsaturated fatty acids"; fat provides energy (calories), and if one adds a particular fatty acid to the diet one must at the same time remove an equivalent amount of some other energy-yielding food component, otherwise the subject will gain weight. The choice of the "exchange medium" is arbitrary; in Figure 1 the effects of fatty acids are calculated in the case where they replace carbohydrates.

As shown in Figure 1, replacement of carbohydrates by fat raises HDL, the effect being more marked the more saturated the fat. Increasing the dietary fat intake also paradoxically decreases the fasting level of neutral fat (triglycerides) in blood plasma; dietary saturates, monounsaturates and (n-6) polyunsaturates all produce this effect to about the same extent.

In contrast, effects on LDL are markedly different, with saturates raising LDL, and (n-6) polyunsaturates – i.e. linoleic acid – slightly lowering it. As a result, oils high in linoleic acid produce the highest HDL/LDL ratio, but the difference with monounsaturated oils is rather slight.

Thus diets high in oils produce the most favourable plasmalipoprotein profiles in metabolic-ward studies where body weight is forcibly kept constant. In clinical practice, however, allowing patients the unrestrained consumption of dietary oils entails a serious risk of caloric overconsumption and weight gain, which would reverse the beneficial effects of high-oil diets on HDL and triglycerides. In addition obesity will increase blood pressure and diabetes risk. Therefore oils should be used with caution and moderation.

Stearic acid

The major saturated fatty acid in human diets is palmitic acid (C16:0), followed by stearic, myristic and lauric acid (Table 1). In mixed diets, lauric, myristic and palmitic acid together usually make up same 60-70% of all saturates, and they are the fatty acids responsible for the cholesterol-raising effect of saturates. The classical studies of *Ahrens et al* (3), *Keys et al* (4), and *Hegsted et al* (5), as well as more recent studies (6,7) showed that the effect of stearic acid on total cholesterol is much less than that of lauric, myristic and palmitic, and more closely approximates the effect of oleic acid. However, only the two most recent trials measured the effect of stearic acid on HDL cholesterol, and both observed a fall relative to unsaturates – non-significant in the study of *Bonanome*

(6), but significant in that of *Zock* (7). Thus stearic and oleic acid might not be entirely equivalent as far as HDL is concerned.

Trans fatty acids

Trans isomers of oleic acid (Figure 2) are produced during the hydrogenation of linoleic and α-linolenic acid, either in the rumen of the cow or in oil hardening factories. Such *trans* fatty acids have effects on serum lipoproteins markedly different from those of the *cis*-isomer, oleic acid: *trans* fatty acids lower HDL and raise LDL (7,8). In addition they raise the plasma levels of lipoprotein(a) (9), a complex of LDL with apoprotein(a) which is a mutant of the pro-enzyme plasminogen that is involved in the dissolution of blood clots (10). Lipoprotein(a) is a particularly atherogenic lipoprotein (11) that was hitherto thought impervious to dietary effects. Thus the overall effect of *trans* C18:1 fatty acids on plasma lipoproteins is unfavourable. However, many people eat no more than a few grams of *trans* fatty acids per day, and such quantities have only negligible effects on lipoprotein levels.

In addition to partially hydrogenated vegetable oils, partially hydrogenated fish oils are an important source of trans fatty acids in the Netherlands, Norway and the United Kingdom. Such hardened fish oils contain mainly *trans* isomers with a chain length of 20 or 22 rather than of 18 carbon atoms; their effects on lipoproteins in man have not been studied in depth, and could conceivably be quite different from those of elaidic acid (*trans* C18:1n-9/Δ9) and its isomers.

Eicosapentaenoic and docosahexaenoic acid

Eicosapentaenoic acid (EPA; C:20n-3,n-6, n-9,n-12,n-15) and docosahexaenoic acid (DHAC22:6n-3,n-6,n-9,n-12,n-15,n-18) typically occur in fatty fish and fish oil. These very-long-chain (n-3) polyunsaturates do not share the LDL-lowering effect of linoleic acid (C18:2n-6,n-9). On the contrary: several studies have shown that fish oils raise LDL and apoprotein B (12-16). They do, however, have a remarkably potent effect on triglycerides and VLDL, which are already lowered by intakes of a few grams of fish oil per day. Whether this effect is responsible for the lower incidence of coronary heart disease that has been seen in fish-eating people in epidemiological studies (17) remains unclear; fish oils can also modulate many other physiological processes, including blood coagulation.

Table 1. The most important fatty acids in foods and their daily intake in middle-aged men in the early 1960's in Finland and the Netherlands.

Name	Formula[a]	Intake (gram/day)[b] Finland	the Netherlands
Saturated fatty acids			
Lauric acid	C12:0	4	2
Myristic acid	C14:0	12	8
Palmitic acid	C16:0	38	30
Stearic acid	C18:0	20	15
Monounsaturated fatty acids			
Oleic acid	C18:1(n-9)	40	29
Polyunsaturated fatty acids			
Linoleic acid	C18:2(n-6	8	12
α-Linolenic acid	C18:3(n-3)	2	2
Eicosapentaenoic acid	C20:5(n-3)	0.4	0.3
Docosahexaenoic acid	C22:6(n-3)	0.3	0.1

[a] *C18:2(n-6) refers to a fatty acid with a chain length of 18 carbon atoms and 2 double bords, located at the 6th and 9th carbon atom from the methyl end of the molecule. Enzymes in the human body can introduce double bonds into fatty acids, but not closer to the methyl end than n-7. Therefore all fatty acids with n-6 (ω6) or n-3 (ω3) double bonds must be provided by the diet.*

[b] *Kromhout et al, unpublished measurements. Data on Finland are means of duplicate diets collected in East- and in West-Finland.*

Figure 1. Predicted changes in serum lipids and lipoproteins when 1 percent of dietary carbohydrates is replaced by fatty acids of a particular class under isocaloric metabolic-ward or equivalent conditions.

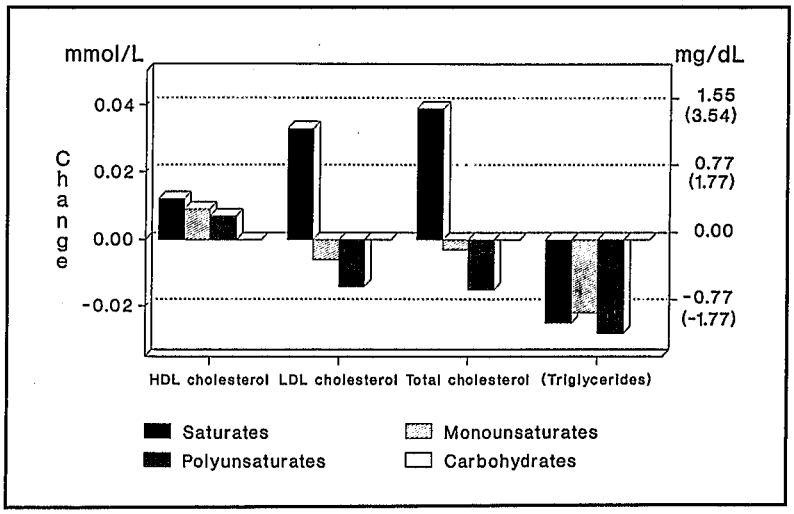
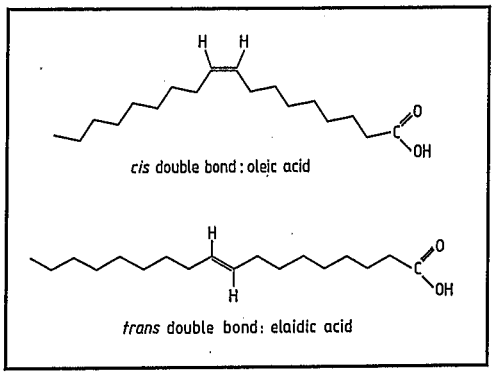


Figure 2. Structure of *cis*- and *trans* C18:1.



Conclusions, and perspective for rapeseed oil

Scandinavian people have historically carried a heavier burden of coronary heart disease than countries located more southernly. A number of factors, both genetic and environmental, are probably responsible for this. There is extensive evidence that the high intake of saturated fatty acids traditional to Scandinavia has played a role in causing this high rate of heart disease through effects on plasma LDL concentrations. Replacement of part of these saturates by unsaturated fatty acids from unhydrogenated rapeseed and other vegetable oils would probably help to reduce the rate of coronary heart disease, or at the very least shift the incidence to a later age.

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