Dietary fats mainly comprise triacylglycerols consisting of 3 individual fatty acids, saturated, monounsaturated, and polyunsaturated, depending on how many double bonds they contain. Specific fatty acids within these categories tend to have varying biologic effects. According to the classic diet-heart hypothesis, high intake of saturated fatty acids (SFAs) and low intake of polyunsaturated fatty acids (PUFAs) increase the concentration of low-density lipoprotein cholesterol, which leads to the deposition of lipid within the subendothelial space after endothelial injury. These plaques narrow the coronary arteries leading to reduced blood flow to the myocardium and are also vulnerable to rupture and thrombosis. On the basis of this understanding, many nutrition guidelines focus on limiting intake of SFAs and replacing them with monounsaturated fatty acids (MUfAs) and PUFAs.1,2 However, the optimal amounts and types of fatty acid consumption have been the subject of considerable debate in the scientific community due to the variation in findings from observational, experimental, and animal studies.

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In this issue of ATVB, Virtanen et al1 add evidence to the observational side of this discussion by reporting on the association of dietary fatty acids with risk of fatal and non-fatal coronary heart disease (CHD) in 1981 men without CHD at baseline from the Kuopio Ischaemic Heart Disease Study, a cohort of men from eastern Finland aged 42 to 60 years in 1984 to 1989. The authors simultaneously report the results of a cross-sectional study on a subgroup of the cohort who completed a carotid ultrasound at baseline to measure the intima-media thickness of the common carotid artery, which is commonly used as a marker of subclinical carotid disease. Information on dietary intake was assessed at baseline with 4 consecutive days of food recording. During the ensuing 21.4 years, there were 183 fatal and 382 non-fatal CHD events. In multivariable models, the authors observed an inverse association of dietary PUFAs with fatal CHD (those with the highest intake of omega-3 and omega-6 PUFAs tended to have the lowest risk), but surprisingly, a nonsignificant trend for a positive association with MUFA. No association was seen with SFA or trans-fat. There was also no association of any fatty acids with nonfatal CHD. In the cross-sectional analyses, PUFa intake was inversely associated with carotid intima media thickness, but there was no association observed with MUfAs or with the other dietary fats.

Several strengths of this research are evident, including its prospective, population-based design, its detailed assessment of the end points, and the adjustment for many covariates. Although the use of food records was cited as an important strength, the extent to which 4 days of food recording provided a more reflective indication of the usual intake of fatty acids beyond a questionnaire or recall was likely to be negligible.

So, what are the implications of these most recent observational findings of dietary fatty acids and CHD? The results are consistent with a recent pooled analysis of 11 observational studies that similarly allowed a direct comparison of different types of fats. In that study, substitution of SFAs with PUFAs was associated with lower risks of fatal and nonfatal CHD.4 Findings are also in agreement with a meta-analysis of observational and experimental studies, which showed a lower risk for CHD among those consuming greater amounts of long-chain omega-3 PUFAs and a lower risk of CHD in randomized trials of omega-6 PUFAs.5 In the current study, Virtanen et al also showed greater intake of PUFAs was associated with less subclinical carotid disease, suggesting that atherosclerosis may be an important intermediary between PUFA intake and CHD (supporting the diet-heart hypothesis).

Surprisingly, however, Virtanen et al1 identified a nonsignificant trend for a higher rate of fatal CHD among those with a higher intake of MUfAs and no association with SFAs. Although unexpected, a positive association of dietary MUfAs with CHD has been observed in the previously mentioned pooled analysis.4 Common to both studies was the inclusion of individuals residing predominantly in northern Europe and the United States, where the main source of MUfAs was likely animal fat and not olive oil or other plant-based sources. Thus, it is possible that the trend for a higher risk for CHD observed with higher intake of MUfAs and the null association with SFAs may be because of the specific foods consumed. In a recent investigation of the SFA composition from different foods and the incidence of cardiovascular disease, de Oliveira et al6 found that the SFA content from meat was associated with a higher risk for cardiovascular disease, whereas SFAs from dairy foods were protective. These opposing results, which may subsequently lead to a null association with total SFA intake, suggest that the different proportions of different SFAs in these foods may differentially influence the risk for cardiovascular disease. In addition, other components of these foods might interact with SFAs by either counterbalancing the adverse
effects in the case of other dairy components (eg, vitamin D, potassium, phosphorus, and calcium) or by increasing its adverse effects in the case of other meat components (eg, cholesterol and sodium). It is likely that this type of analysis of the entire food and not just a single component may have helped to uncover the nature of the positive trend observed with MUFA and the lack of association with SFA. This type of food-based research also offers the opportunity for improved communication and translation with the public and policy makers and may be less susceptible to industry manipulation.

Recently, the American Heart Association reaffirmed their recommendation for replacing SFAs with PUFAs in the diet while also providing specific food-based dietary goals, including recommendations for promoting consumption of fish, nuts, legumes, and seeds, while also limiting intake of processed meats. The results of Virtanen et al seem to support this recommendation.

Disclosures

None.

References


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Dietary Fatty Acids and Coronary Heart Disease
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