Saturated fatty acid intake and cardiovascular risk

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This editorial refers to ‘Dietary intake of saturated fatty acids and incident stroke and coronary heart disease in Japanese communities: the JPHC Study’, by K. Yamagishi et al., on page 1225

Since the 1960s, clinical and animal studies have shown that reduction of dietary saturated fatty acid (SFA) consumption is associated with reduced risk of cardiovascular disease (CVD).1,2 Thus, reduction of SFA intake is now one of the central strategies of dietary recommendations to reduce CVD worldwide.1,2 For example, the World Health Organization and the US Dietary Guidelines recommend dietary consumption of < 10% of total energy intake from SFAs,3 while the American Heart Association guidelines recommend even more strict dietary SFA consumption of < 7%.2 However, it has always been a matter of debate whether SFA consumption is truly associated with CVD risk, particularly with risk of stroke, because of insufficient evidence regarding the disorder. Recently, Siri-Tarino et al. performed a meta-analysis to summarize the evidence related to the association between dietary SFA intake and risk of coronary artery disease (CAD), stroke, and CVD (including stroke) in 21 prospective epidemiological studies, where 11 006 out of 347 747 subjects developed CAD or stroke during 5–23 years of follow-up.4 Because the pooled relative risk (RR) estimate comparing extreme quintiles of SFA intake for CAD, stroke, and CVD was 1.07 [95% confidence interval (CI) 0.96–1.19; P = 0.22], 0.81 [95% CI 0.62–1.05; P = 0.11], and 1.00 [95% CI 0.89–1.11; P = 0.95], respectively, they concluded that SFA intake was not significantly associated with increased risk of CAD, stroke, or CVD.5

However, there seems to be an insignificant but mild trend for the relationship between dietary SFA intake and stroke (RR 0.81, 95% CI 0.62–1.05; P = 0.11), which may warrant further investigations.5

Yamagishi et al. have reported the results from the Japan Public Health Center-based prospective (JPHC) Study that examined whether dietary SFA intake is associated with risk of stroke and its subtypes as well as that of CAD amongst Japanese, whose average dietary SFA intake is lower than in Western populations.6

The JPHC Study comprised a total of 38 004 men and 43 847 women from two subcohorts: Cohort I, aged 45–64 in 1990, and followed-up through 2009; and Cohort II, aged 45–74 in 1998, and followed-up through 2007. The major strengths of the JPHC Study include the large sample size, detailed evaluation of endpoints, and accurate diagnosis of stroke subtypes with computed tomography (CT)/magnetic resonance imaging (MRI). Consequently, the JPHC Study examined a larger number of cardiovascular events with more detailed analysis, giving a strengthened power in statistics and more useful clinical messages as compared with previous studies. After adjustment with multiple factors, including age, sex, energy intake, cohort, cigarette smoking status, alcohol intake, body mass index, sports at leisure time, walking and standing time, perceived mental stress, energy-adjusted dietary intakes of carbohydrate, protein, cholesterol, vegetables, fruit, and calcium, the JPHC Study revealed inverse associations of dietary SFA intake with total stroke [n = 546; adjusted hazard ratio (HR) (95% CI) for the highest vs. lowest quintiles = 0.77 (0.65–0.93), P for trend = 0.002], intraparenchymal haemorrhage [n = 150; 0.61 (0.43–0.86), P for trend = 0.005], and ischaemic stroke [n = 319; 0.84 (0.67–1.06), P for trend = 0.08], providing additional epidemiological evidence that dietary SFA intake is inversely associated with total stroke in a Japanese population.7 This inverse relationship between SFA intake and stroke observed in the JPHC Study is consistent with the previous observation obtained in the Japan Collaborative Cohort (JACC) Study for Evaluation of Cancer Risk.8 The JACC study comprised 58 453 Japanese men and women aged 40–79 years at baseline (1988–1990) with a 14.1-year follow-up and revealed inverse associations of dietary SFA intake with mortality from total stroke [n = 976; multivariable HR (95% CI) for highest compared with lowest quintiles: 0.69 (0.53–0.89), P for trend = 0.004], intraparenchymal haemorrhage [n = 224; 0.48 (0.27–0.85), P for trend = 0.03], and ischaemic stroke [n = 321; 0.58 (0.37–0.90), P for trend = 0.01]. Thus, it may be concluded that there exists an inverse association between dietary SFA intake and both incidence and mortality from stroke at least in a Japanese population, where SFA levels are lower than in Western populations.

In contrast, however, the results regarding CAD are inconsistent between the JPHC Study and the JACC Study; the JPHC Study

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revealed a positive association of dietary SFA intake with myocardial infarction \( n = 124; \, 1.39 \, (0.93–2.08), \, P \, \text{for trend} = 0.046 \), particularly among men,4 whereas the JACC Study found no association of dietary SFA intake with mortality from ischaemic heart disease \( n = 836; \, 0.89 \, (0.68–1.15), \, P \, \text{for trend} = 0.59 \).5 Possible explanations for the discrepancy include the facts that a relatively younger population was examined in the JPHC Study and that the incidence and mortality of myocardial infarction as an endpoint were different between the two studies. In other words, the discrepancy could be due, at least in part, to the increase in the incidence of, but not that in mortality from, myocardial infarction in a relatively younger generation in Japan, particularly in males. In addition, there might have been a difference in food sources of SFAs between the two studies, such as meat, plants, and dairy products, because individual SFAs may affect CAD risk differently and most SFA-rich foods contain other constituents related to CAD risks.6

Another strength of the JPHC Study is an approach searching for a cut-off level of dietary SFA intake. To explore the optimal level of dietary SFA intake to prevent CAD or stroke, the authors included the results obtained from previous studies by plotting dietary SFA intake and crude incidence of and mortality from haemorrhagic stroke, ischaemic stroke, and CAD. By this approach, they suggested a threshold of \( \sim 20 \, \text{g/day} \) of dietary SFA intake for the inverse relationship of SFA intake with stroke, especially with haemorrhagic stroke. Although the present results should be confirmed by future studies including meta-analyses, dietary SFA intake \( >20 \, \text{g/day} \) could be an optimal cut-off level to prevent stroke. However, it should be underlined that dietary SFA intake \( >20 \, \text{g/day} \) could also be a risk for development of CAD, particularly of myocardial infarction, despite controversies even in the Japanese population, where dietary SFA intake is lower than in Western populations. It remains to be examined whether reduction of dietary SFA to \( \sim 20 \, \text{g/day} \) could actually reduce CAD in Western populations as well as in Asian populations.

As an effort to reduce SFA consumption, previous observational and randomized clinical trials have tried to show benefits of replacement of SFAs with other substitutes to reduce CAD risk.7–9 Among such substitutes examined, polyunsaturated fatty acids (PUFAs) are the most promising candidates to reduce CAD risk despite some controversies (Figure 1).7 In populations consuming a Western diet, the replacement of 1% of energy from SFAs with PUFAs is associated with a reduction in CAD incidence of 2–3%.7 In contrast, there is little evidence to support the effect on CAD risk of replacing SFAs with monounsaturated fatty acids (MUFA),10 although MUFAs have been reported to reduce LDL-cholesterol levels.11 The benefit of substituting carbohydrates for SFAs is also unclear or limited, even though unrefined carbohydrate with a low glycaemic index might be beneficial.11 Thus, to date, the best candidate substitutions of SFAs could be PUFAs, particularly \( n-3 \) PUFAs, because a series of studies have shown that a high intake of fish oil and \( n-3 \) PUFAs could reduce the incidence of myocardial infarction, ischaemia–reperfusion injury, ventricular arrhythmias, and death, including sudden cardiac death, despite some controversies.12–16 Further studies are warranted to determine whether specific foods could be appropriate alternatives for SFAs and also whether \( n-3 \) PUFAs could be appropriate supplement to reduce cardiovascular risks.

Conflict of interest: none declared.

References


