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Editorial

Consumption of saturated fatty acids and coronary heart disease risk

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Coronary heart disease (CHD) remains the leading contributor of overall disease burden globally despite a decline in its mortality in the past decades [1]. Adopting a healthy lifestyle and diet is one of the most cost-efficient strategies for the primary prevention of CHD [2]. Based on the totality of evidence linking fatty acids and cardiovascular risk, the current Dietary Guidelines for Americans recommends lowering saturated fat acid (SFA) intake to <10% of total calories for maintaining an optimal cardiovascular health, through substituting foods rich in polyunsaturated fatty acids (PUFA) for foods high in SFAs [3]. Pooling studies of prospective cohorts and meta-analyses of randomized controlled trials both supported the notion that consuming PUFAs in place of SFAs leads to reduced CHD risk [4,5]. Concordantly, meta-analyses of randomized trials also showed that isocaloric replacement of SFAs by PUFAs lowered low-density lipoprotein cholesterol levels as well as total to high-density lipoprotein (HDL) cholesterol ratio [6]. In contrast to these lines of evidence, systematic reviews and meta-analyses that summarized the evidence of associations between SFAs intake and CHD risk in comparison with various macronutrients (such as PUFAs, refined carbohydrates, or different proteins) observed a significant heterogeneity and an overall null association [7]. This highlights the importance of specifying the macronutrients for comparison when examining SFAs intakes in relation to CHD risk in an isocaloric setting [8].

In this issue of the Journal, Praagman and colleagues [9] conducted a sizeable analysis in the EPIC cohorts and generated novel evidence to the knowledge base pertaining to SFAs and cardiovascular health. In this study, the authors evaluated the association between intake of individual SFAs and CHD risk among 22,050 participants from the EPIC-Norfolk cohort and 53,375 from the Danish Diet, Cancer and Health cohort (EPIC-Denmark). During a median follow-up of 18.8 y in the EPIC-Norfolk and 13.6 y in the EPIC-Denmark, 1204 and 2260 myocardial infarction (MI) were identified and ascertained, respectively, in these two cohorts. Multivariate analyses showed no association between any individual SFAs and MI in the EPIC-Norfolk, nor between C16:0 and C18:0 (constituted ~75% of total SFAs) and MI in the EPIC-Denmark. Moreover, in the EPIC-Denmark, higher consumption of C4:0-C10:0, C12:0, and C14:0 was associated a lower MI risk. In isocaloric replacement analyses, replacement of C16:0 and C18:0 with plant protein was significantly associated with a lower MI risk, whereas replacing SFAs with PUFAs was not associated with a reduced risk of MI in these two EPIC cohort studies.

The study is applaudable for its large size, prospective cohort study design, and detailed assessment of diet and lifestyle factors. In addition, it is among the few studies specifically examined individual SFAs in relation to CHD risk and compared with different macronutrients. While this study certainly generated valuable evidence suggesting SFAs with different chain-length may potentially exert different cardiometabolic health effects, several limitations of the study mandate cautious interpretations of the findings. The most salient limitation probably is the fact that the semi-quantitative food frequency questionnaires (FFQs) used in both cohorts have not been validated for the assessments of individual SFAs in these cohorts. The FFQ used in the EPIC-Norfolk was similar with the 130-item FFQ used in the Nurses' Health Studies (NHS). While the correlation coefficients were 0.71 for total fat and 0.80 for total SFAs in the NHS [10], it was only 0.55 for total fat in the EPIC-Norfolk [9]. In the EPIC-Denmark, the correlation coefficients were 0.48–0.67 for total fat and 0.39–0.49 for total SFA [9]. Many factors, such as reporting errors and measurement errors in nutrient composition database, can come into play to impact the validity of SFA assessments.

Another important limitation is that dietary assessment was conducted at baseline only, and thus consumption of SFAs estimated in the analysis may not reflect actual dietary SFA intake during the long-term follow-up (e.g., 13.6–18.8 y in this study). Zong et al. previously showed that, individual SFA intake changed over time in two US cohorts – the NHS and Health Professionals Follow-up Study, during 1986 to 2010 [10]. In a prospective study setting, in comparison with a single measurement, repeated measurements of dietary intake every few years allow a more accurate assessment of diet and cumulative long-term diets, and take into account of possible dietary changes over time. In addition, repeated measurements reduce random errors, thus providing more precise assessments of diet.

While examining individual SFAs may provide more insight into the roles of specific individual fatty acids in CHD etiology, it is worth noting

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that individual SFAs are highly intercorrelated, and thus it is difficult to unravel their actual independent effects in observational studies. Dietary sources of SFAs usually contain all major SFAs and therefore it is challenging to separate individual SFAs in real life dietary practice. Therefore, current recommendation should continue focusing on reducing consumption of total SFAs, possibly by the substitution of PUFAs [3]. In addition, different SFAs-containing foods have been associated with different cardiovascular effects, possibly owing to the differences in other macro- and micro-nutrients in the foods (e.g., meat vs. nuts). In the current study, the authors also observed that higher intakes of SFAs from meat were associated with a higher MI risk, which is consistent with previous evidence on the association between red meat intake and CHD risk [9]. For CHD prevention, a singular focus on reducing total fat and total SFAs while ignoring the food sources or the overall dietary patterns can be counterproductive. Shifts in fat intake should align with the recommended healthy dietary patterns, which emphasize limited intakes of red and processed meat, and added sugars, lower salt intake, replacement of refined grains with whole grains, and higher consumption of fruits and vegetables [3].

Conflict of interest

We declare no competing interests.

References

[1] G.B.D. DALYs, H. Collaborators, Global, regional, and national disability-adjusted life-years (DALYs) for 333 diseases and injuries and healthy life expectancy (HALE) for 195 countries and territories, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016, Lancet 390 (2017) 1260–1344.
[2] J. Stewart, G. Manmathan, P. Wilkinson, Primary prevention of cardiovascular disease: a review of contemporary guidance and literature, JRSM Cardiovasc. Dis. 6 (2017) 2048004016687211-2048004016687211.
[3] US Department of Health and Human Services & Agriculture, 2015–2020 Dietary Guidelines for Americans, 8th edn. US Dept. of Health and Human Services and US Department of Agriculture, Washington, DC, 2016 https://health.gov/dietaryguidelines/2015/.
[4] M.U. Jakobsen, et al., Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies, Am. J. Clin. Nutr. 89 (2009) 1425–1432.
[5] D. Mozaffarian, R. Micha, S. Wallace, Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials, PLoS Med. 7 (2010), e1000252
[6] R.P. Mensink, Effects of Saturated Fatty Acids on Serum Lipids and Lipoproteins: A Systematic Review and Regression Analysis, World Health Organization, 2016.
[7] R.J. de Souza, et al., Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies, BMJ 351 (2015), h3978
[8] Y. Li, et al., Saturated fats compared with unsaturated fats and sources of carbohydrates in relation to risk of coronary heart disease: a prospective cohort study, J. Am. Coll. Cardiol. 66 (2015) 1538–1548.
[9] Jaike Praagman, et al., Consumption of individual saturated fatty acids and the risk of myocardial infarction in a UK and a Danish cohort, Int. J. Cardiol. (2018) https://doi.org/10.1016/j.ijcard.2018.10.064.
[10] G. Zong, et al., Intake of individual saturated fatty acids and risk of coronary heart disease in US men and women: two prospective longitudinal cohort studies, BMJ 355 (2016), i5796.