Saturated Fats and Health: Current Thinking

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INTRODUCTION

The saturated fats issue is not a new challenge to palm oil, as it was some three decades ago that the matter was first raised. The anti-tropical oil campaign, which labelled palm oil as saturated artery-clogging fat was first raised in the 1980s in the United States. As a response to this allegation, the then Palm Oil Research Institute of Malaysia (PORIM) had embarked on several nutrition studies which had revealed that palm oil did not elicit elevated coronary heart disease (CHD)-risk responses, but rather it was the *trans* fatty acids resulting from partial hydrogenation of soft oils that were deleterious to health. Nevertheless, the current dietary recommendations for reducing dietary saturated fats have had some implications on palm oil applications in some sectors, and as such on palm oil trade. As food manufacturers continue to look for lower saturated fat alternatives to palm oil, the share of palm oil in food products continues to decline. For example, total imports of oils and fats into the United Kingdom had decreased about 16.8%, from 1.67 million tonnes in 2005 to 1.39 million tonnes in 2010, while imports of palm oil had declined even further by 37.6%, from 0.939 million tonnes to 0.602 million tonnes. Besides, the percentage share of palm oil in the total oils and fats imports basket also declined, from 56.2% in 2005 to 43.3% in 2010. During this period, a significant increase was seen in the imports of sunflower oil, which had increased from 0.126 million tonnes to 0.275 million tonnes. The share of sunflower oil in the imports basket had also increased from 7.5% to 14.8%.

Several epidemiological studies has established strong positive correlations between dietary factors, particularly intake of total fat and saturated fat with blood cholesterol levels and CHD. Intake of dietary fat, particularly that of saturated fats has become the focus of attention, due to their supposed links to CHD risks. A generalisation that all saturated fatty acids (SFA) are deleterious to health, and as such, oils and fats containing a relative predominance of SFA are nutritionally undesirable has therefore evolved. Current dietary recommendations (*Report of the Joint FAO/WHO Expert Consultation on Fats and Fatty Acids in Human Nutrition*, 2010) advise reducing the intake of dietary saturated fats to <10% energy. The recommendation is based on evidence that SFA, C12:0-C16:0 increase LDL cholesterol and total:HDL cholesterol ratio in comparison to *cis* monounsaturated fatty acids (MUFA) or polyunsaturated fatty acids (PUFA). Based on evidence from epidemiologic studies and controlled clinical trials, it was reported that replacing SFA with PUFA decreases the risk of CHD.

A Saturated Fats Symposium held at the University of Copenhagen, Denmark on 28-29 May 2010 attended by experts in the field has reviewed the evidence on dietary fatty acids, CHD risk and consensus on the following had been reached: (1) replacing SFA with PUFA consistently reduces the risk of CHD based on evidence from epidemiologic, clinical and mechanistic studies; (2) there is insufficient evidence to judge the effects of replacing SFA with MUFA on CHD risk; (3) no clear association by replacing SFA with carbohydrate on CHD risk and insulin resistance (Astrup

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et al., 2011). Based on the current recommendations and evidence on SFA, this review attempts to look at emerging thoughts on saturated fats and health.

RECENT QUESTIONS ON VALIDITY OF SATURATED FATTY ACID (SFA) REDUCTION AS STRATEGY TO REDUCE CORONARY HEART DISEASE (CHD) RISKS

Some 40 years ago, Keys and Hegsted (Keys *et al.*, 1959; Hegsted *et al.*, 1965), working independently, had developed regression equations that predicted the quantitative effects of dietary fatty acid classes, SFA, MUFA and PUFA on plasma total cholesterol. Their conclusions were that dietary SFA raised plasma total cholesterol, while PUFA had the exact opposite effect, and that the effects of the former were considerably more potent than that of the latter. It was also concluded that MUFA had a generally neutral effects on plasma total cholesterol. Subsequent regression analyses were developed by other investigators (Mensink and Katan, 1992; Hegsted *et al.*, 1993; Yu *et al.*, 1995) to describe the effects of dietary fatty acids on plasma total cholesterol, HDL and LDL cholesterol levels. The regression equations developed provided useful tools for predicting changes in plasma cholesterol and lipoprotein concentrations in response to the different fatty acid classes, and the general consensus was therefore for reduction of the intake of dietary fats and particularly SFA towards reducing CHD risks.

More than 40 years after the development of Keys and Hegsted's equation, extensive research have been conducted to examine the role of SFA on CHD risk. Current evidence from several large scale epidemiologic studies and meta-analysis does not support the previously perceived relationship between SFA and CHD risk. Siri-Tarino *et al.* (Siri-Tarino *et al.*, 2010) have pointed out that an independent association of SFA intake with CHD risk had not been consistently established in prospective epidemiologic and clinical studies. The replacement of SFA with PUFA or MUFA could lower both LDL and HDL cholesterol, but the replacement of SFA with carbohydrate, particularly refined carbohydrate, also could exacerbate the atherogenic dyslipidemia associated with insulin resistance and obesity. The finding by Siri-Tarino *et al.* (2010) was further supported by Micha and Mozaffarian (2010), with conclusion drawn on the insufficient evidence for different chain-length-specific effects on other risk pathways or, more importantly, disease endpoints. The replacement of SFA with PUFA was found to modestly lower CHD risk, with approximately 10% risk reduction for a 5% energy substitution. A positive aspect of SFA was reported by Yamagishi *et al.* (2010), who concluded that SFA intake was inversely correlated with mortality from total stroke, including intraparenchymal aemorrhage and ischemic stroke subtypes on cohort of 58 453 Japanese men and women from the Japan Collaborative Cohort Study for Evaluation of Cancer Risk (JACC Study). However, the findings were not consistent with other meta-analysis (Skeaff and Miller, 2009; Mozaffarian *et al.*, 2010) suggesting a favourable effect of replacing SFA with PUFA on CHD risk reduction. The total SFA may not reflect the effects of individual SFA on cardiovascular health and major food sources while other constituents in foods may influence CHD risk.

SPECIFIC SFA HAVE DIFFERENT EFFECTS ON LIPID PROFILES IN RELATION TO CORONARY HEART DISEASE (CHD) RISK

The different fatty acids of various chain lengths and degree of unsaturation exhibit different biological properties. The most ubiquitous of fatty acids in nature, palmitic acid (C16:0) which is also the predominant fatty acid in palm oil needs some special consideration, as there remains unsolved, the question of the different impacts on plasma cholesterol, of the different fatty acids. Hence, the subsequent parts of this review will focus on dietary feeding trials that specifically examine the cholesterolemic response of C16:0 *vis-à-vis* individual SFA (C12:0-C14:0) and MUFA (C18:1). A number of clinical trials has been conducted to show the neutrality of palmitic acid on lipid profiles relative to other fatty acids. Palmitic acid is less cholesterolemic than C12:0-C14:0 and comparable with C18:0 and C18:1. Meta-analysis conducted by Mensink *et al.* (2003) demonstrated that C12-C16:0 are more cholesterolemic than C18:0 and C18:1. The clinical studies suggest that the cholesterolemic response to C16:0 is dependent on factors such as age, baseline cholesterol levels, dietary fat load, and the presence of adequate levels of C18:2 in the diets. Studies that have elicited a hypercholesterolemic effect from C16:0 have been conducted on elderly (age ≥ 59 years) hypercholesterolemic subjects consuming high-fat diets (~40%en) (Mattson and Grundy, 1985; Bonanome and Grundy, 1988;

Denke and Grundy, 1991; Vega-Lopez *et al.*, 2006), subjects fed high levels of dietary cholesterol (Zock and Katan, 1992) or dietary fats (37%-40%en) (Nestel *et al.*, 1992; 1994; Temme *et al.*, 1996; Zock *et al.*, 1994); or subjects consuming low levels of C18:2 (Vega-Lopez *et al.*, 2006; Montoya *et al.*, 2002). On the other hand, in studies conducted in normal healthy volunteers, consuming moderate dietary fat levels (~30% en) with moderate cholesterol intake (<300 mg per day) (Choudhury *et al.*, 1995; Ng *et al.*, 1992; Sundram *et al.*, 1995), C16:0 did not adversely affect plasma lipids and evidence suggests that it is comparable to oleic acid. In addition, it has been shown that C16:0 is not hypercholesterolemic if intake of dietary linoleic acid remained above 4.5%en (Clandinin *et al.*, 1999; 2000; French *et al.*, 2002). Furthermore, there is an emerging body of evidence which suggests that it is not only the fatty acid composition of dietary fats that is important in influencing cholesterol response, but equally important, is the positional distribution of the fatty acids on the triacylglycerols. The available evidence indicates that C16:0 is atherogenic when placed at the *sn*-2 position of the triacylglycerol moiety. Hence, vegetable oils such as palm oil and palm olein, though containing relatively high amounts of C16:0 (~46%), do not exert hypercholesterolemic responses as the C16:0 is predominantly in the *sn*-1 and –3 positions.

CONCLUSION

Despite the existing recommendations of various national and international health authorities to limit dietary intakes of saturated fats as a means to modulate CHD risks, controversy still remains as to the validity of these recommendations. The wealth of confounding factors demonstrates that saturated fat is not an overwhelming input variable for any population studied to date, and is not the only variable associated with heart disease. Research is needed to clarify the role of dietary saturated fats in comparison with specific forms of carbohydrates on CHD risk markers. The content of total saturated fat and individual SFA may have different effects on CHD risk. In addition, future research is also needed to focus on the effects of different sources of dietary saturated fat (animal fat *vs.* vegetable oil) on CHD risk markers. There is also a need to take into account individual variability, age- and sex-specific factors as well as the multiple actions and functions of each of the different saturated fats and a more individual view to assessment of diet and risk. The recommendation for absolute reduction strategies without considering the replacement nutrient may also be brought into question.

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