

REVIEW ARTICLE

De-Mystifying Saturated Fats – A Perspective

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Citation

Mani I, Kurpad AV. De-Mystifying Saturated Fats – A Perspective. Indian J Comm Health. 2014;26, Suppl S1:31-36

Source of Funding : Nil **Conflict of Interest:** None declared

Abstract

Since the 1980s the world has been repeatedly informed about the harmful effects of saturated fatty acids. The USDA recommends that SFA consumption should be < 10 en%, while the American Heart Association goes a step further and suggests that the intakes should be reduced to < 7 en%. However, recent findings are increasingly questioning this advice, showing evidence that consumption of SFA may actually be better than increasing intake of either carbohydrates or polyunsaturated fatty acids. This article aims to summarize some of this information, with emphasis on its relevance to Indian diets.

Key Words

Saturated Fatty Acids; PUFA; Cardiovascular Diseases; Under-nutrition

Introduction

The relationship between dietary consumption of fats and fatty acids, and serum cholesterol was first suggested by Ancel Keys in the 1950s [1]. But it was in 1970 that he published his seminal work as the Seven Countries Study [2], where he claimed that the incidence of CHD was directly related to serum cholesterol values, which in turn was related to the average consumption of saturated fatty acids (SFA) [3].

The Seven Countries Study was the basis on which the USDA and the American Heart Association began their dietary recommendations to reduce consumption of SFA and increase that of polyunsaturated fats (PUFA) [4]. This was also the beginning of the increasing bad press on the use of oils containing higher levels of SFA such as coconut oil and palm oil.

Questions about the Seven Countries Study

Reports in the 1990s started raising doubts about this association. Grundy [5] stated that there was no epidemiological support for increasing intakes of n-6 PUFAs, and claimed that it lowered HDL, increased LDL oxidation, and even promoted cancer in laboratory animals. A meta-analysis of all

population, cohort and case-control studies at that time questioned the 'harmful' effects of dietary SFA and the 'protective' effects of PUFA [6]. Furthermore, the validity of the seven countries data was under scrutiny since Keys had only presented data that suited his theory and conveniently left out the remaining data [7]. Figure 1 shows both the selective as well as the total data compiled by Keys. As can be seen from the two graphs, the inclusion of the total data set considerably reduced the strength of the association between fat intake and cardiovascular deaths, but it did not completely negate the existence of association. This only highlighted the fact that regional / ethnic differences are likely to play a role in the responses of a given population to dietary fatty acids.

Current intakes of dietary fat and incidence of cardiovascular diseases

Intakes of fatty acids vary around the world, and so do the intakes of SFA. A recent review of dietary intakes in various parts of the world [8] showed that total fat intake ranged from 11 – 46 en% while SFA intake ranged from 3 – 21 en% (mean intake 11.6 ± 3.3 en%). A more comprehensive review of SFA intakes across the world [9] with a larger dataset of

184 countries showed a similar mean intake of SFA (11.7 ± 4.7 en%), but a wider range (2.3 – 27.5 en%). The current recommendations of fat intakes by FAO/WHO are as follows: Fat: 20 – 35 en%, SFA: < 10 en%, PUFA: 6 – 11 en% (2.5 - 9 en% LA and 0.5 – 2 en% ALNA), and EPA+DHA: 0.25 - 2 g/day. The general conclusion is that in many countries adults continue to have higher SFA and lower PUFA intakes than recommended, and that public health should focus on improving these ratios.

A comparison of dietary SFA intakes with the WHO data [10] on mortality due to cardiovascular disease (CVD) and type 2 diabetes (T2D) showed a non-significant, but positive correlation between the two ($r = 0.11$, $p = 0.13$). A scatterplot of this data (Figure 2) shows that the intakes of SFA in majority of the world ranges between 5 - 15 en%, with a small number of countries ($n=25$) showing higher intakes. A scatterplot without these 25 countries shows a complete lack of correlation between SFA intakes and mortality (Figure 3). In order to get a better understanding of these results, the data was then split into 5 different groups based on economic status, as defined by the World Bank – low income, lower middle-income, upper-middle income, high income-non OECD, and upper income-OECD countries.

[Table 1](#) shows the overall SFA intakes in these different regions of the world and mortality due to CVD and T2D. SFA intakes are the highest in the high income-OECD countries, but the mortality due to CVD and T2D in this group is significantly lower than in any of the other groups. Furthermore, the correlation between SFA intake and mortality is significant only for one region – the upper-middle income countries.

This implies that various other factors, apart from SFA intake, probably play a role in incidence of CVD. It has been shown that the lower incidence of CVD mortality in developed / high-income countries is driven mainly by preventive interventions that prolong survival even after CVD is manifest [11]. On the other hand, the high burden of CVD in developing countries is due to an increase in prevalence of risk factors along with poor access to quality health care. Nutrition transition in these countries is also accompanied by increase in other risk factors such as smoking and consumption of tobacco, and decreased physical activity [12]. A detailed

discussion of various confounding factors that could play a role is beyond the scope of this review.

Time for objective reassessment of data

There have been a few large-scale community dietary intervention studies conducted in developing countries such as China and Mauritius, where a reduction in risk factors has not resulted in a reduction in mortality. The Mauritius project, which was a government led program to change cooking oil from Palm oil to Soybean oil, showed a decrease in cholesterol levels in 5 years. However, this was accompanied by an increase in obesity and diabetes, which was unrelated to cholesterol levels [13]. On the other hand, in Poland, a switch from animal fats to vegetable fats (mainly rapeseed and soybean) resulted in a >25% decrease in CHD mortality [14].

On the whole, this data emphasizes the need for a re-investigation of the role of fatty acids in CVD, specifically with reference to their dietary source. Studies in the last decade have provided new information on the functions of SFA, especially with respect to myristic acid [15, 16]. A meta-analysis on the effects of Palm oil on cardiovascular health suggests that while there is a wide variation in the results obtained depending on factors such as type of populations, basal diets consumed, age of volunteers, funding agencies etc [17], the overall results do suggest that palm oil based diets are better than diets containing trans-fatty acids, at least with respect to their effects on blood lipid profiles.

A recent review of the effects of dairy products [18] on cardiovascular health has clearly indicated that consumption of milk, cheese and yoghurt are inversely associated with CVD risk. The data also suggest a potential protective role of full-fat milk, cheese and yoghurt on the risk of CVD disease. Similarly, studies with coconut oil, which is rich in medium chain SFA, have shown beneficial effects on blood lipids and have also been implicated as a potential route to weight management / control [19, 20].

Fats in Indian diets: Indian diets are predominantly cereal-pulse based diets in which the main source of fats is in the form of vegetable oils. Consumption of animal fat is low, with the exception of milk and other dairy products. The data from NNMB suggests that over the past three decades there has been a slight increase in percent dietary energy from fats. Although there are vast differences in fat intakes

between rural and urban populations, the overall fat intake is < 15 en% [21]. However, in spite of this, the incidence of CVD and T2D are among the highest in the world. Although there are a number of factors that contribute to this, scientists and nutritionists continue to place much of the blame on increasing fat and SFA intakes [22, 23].

Reports show that although the total per capita caloric intake has increased over the last 30 years, the contribution of animal sources continues to be minimal [24]. The increase in calories appears to be mainly due to increase in intake of vegetable oils (Table 2). On the face of it, it appears that this increased oil consumption could be a major contributor to increasing CHD mortality. However, surveys by the Indian Agricultural Research Institute (IARI) [25] clearly suggest that the trends in consumption of different edible oils in India have changed over the last 30 years. There has been a significant increase in the consumption of oils like sunflower, soybean and other unsaturated vegetable oils, reflecting a greater concern for consumption of healthier edible oils, both in urban and rural areas. This indicates that the increased consumption of fat is more of PUFA rather than SFA. (Table 3)

The fact that there has been an increase in incidence of CVD and T2D in spite of an increase in PUFA consumption again reinforces the idea that the major contributing factors to CVD may not necessarily be dietary fats, at least, not necessarily SFA. Other factors such as sedentary lifestyle, smoking, increased consumption of processed foods, pollution, stress at workplace, availability of healthcare facilities, etc, probably play a much bigger role.

Moreover, at the other end of the spectrum, India continues to face the problem of low birth weight and stunting in children, which is a net result of under-nutrition [26]. Under-nutrition, especially in pregnant women, results in low birth weight babies, and evidence points to the association of low birth weight with increased risk of adulthood chronic disease [27]. According to the NFHS data [28], the incidence of under-nutrition in Indian women (36%) appears to be far more than the incidence of overweight, and this is a problem that requires an increased rather than a decreased intake of fats.

Studies on pregnant Indian women [29] suggest that intakes of fat (~23 en%), and especially SFA (~8 en%), is significantly lower than intakes in developed countries, which range between 34-38 en% and 12-16 en% respectively [30]. It is possible that this may influence the absorption of fat-soluble vitamins, resulting in additional deficiencies during pregnancy. It has also been observed, in a pregnancy cohort dataset from St John's Hospital, Bangalore, that higher intake of SFA during pregnancy result in significantly better birth weights, and lower incidence of low birth weight babies (data not shown). Analysis of food group intake in this dataset showed that the increase in SFA intake was directly related to milk consumption, which is mainly in the form of short and medium chain SFA. This suggests that increasing SFA intake, at least in the form of milk and milk products, may actually be beneficial in pregnant women (manuscript under preparation). It is interesting to note that most reported studies on fat intakes during pregnancy focus either on the adverse effects of very high fat/SFA consumption, or on the benefits of n-3PUFA intake [31,32]. The essential role of SFA in Indian diets containing low to moderate levels of fats has not been investigated so far, and this is a critical area that needs to be studied.

Conclusion

The debate on the putative harmful effects of SFA continues, but in the Indian context it is necessary to consider both ends of the nutrition spectrum. It appears that at least for the lower end of the nutrition spectrum, it may be time for us to consider an overall improvement in intake of SFA by returning to some of the traditional practices of India.

These may include the regular consumption of milk and milk products, as well as the use of traditional cooking oils such as coconut oil, groundnut oil and sesame oil. On the whole, the available data points to the fact that it is time to reassess the role of fats, especially SFA and PUFA in human nutrition, and above all, in Indian diets.

Authors Contribution

Both authors contributed equally to idea generation and data analysis. IM wrote the article with editing by AVK.

Acknowledgement

The discussions with Dr Kiruba Shankar during data analysis are deeply appreciated.

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Tables

TABLE 1 OVERALL SFA INTAKES IN FIVE DIFFERENT ECONOMIC REGIONS OF THE WORLD AND MORTALITY DUE TO CVD AND T2D

Region	n	SFA (en %) Mean (Range)	Mortality (per 100,000 population)	Correlation (r) between mortality and SFA intake
Low income	34	10.6 (2.3 - 16.7)a	854 (283-1343)a	0.1173
Lower middle income	45	12.8 (3.2 - 27.5)	795 (318-1282)a	0.0072
Upper middle income	51	11.4 (6.2 - 23.8)	743 (269-1650)a	0.4212b
High income: non - OECD	27	11.0 (6.8-16.9)a	597 (233-1186)a	0.1882
High income: OECD	24	12.4 (8.0-14.8)	280 (183-571)	0.0890

a – significantly different from High income---OECD group, p<0.05 b – significant correlation p<0.05

TABLE 2 CHANGES IN DIETARY INTAKES OF CALORIES AND FATS IN INDIAN DIETS OVER 20 YEARS

Product (Calories/day/person)	1979 - 81	1982 - 91	1992 - 2001
Total calories	2083	2365	2492
Total animal product	120	163	196
Milk	71	102	111
Total vegetable product	1963	2202	2296
Vegetable oils	127	158	239

Adapted from reference 23

TABLE 3 CHANGES IN DIETARY INTAKES OF CALORIES AND FATS IN INDIAN DIETS OVER 20 YEARS

Item	Rural (1993 - 94)	Urban (1993 - 94)	Rural (2009 - 10)	Urban (2009 - 10)
Groundnut oil	0.120	0.240	0.054	0.126
Mustard oil	0.170	0.150	0.287	0.230
Vanaspati	0.030	0.060	0.036	0.036
Other oils	0.050	0.110	0.243	0.408
All edible oils	0.370	0.560	0.636	0.818

Adapted from reference 24

Figures

FIGURE 1 THE SELECTIVE AND COMPLETE DATA SET OF ANTON KEYS [3, 7]

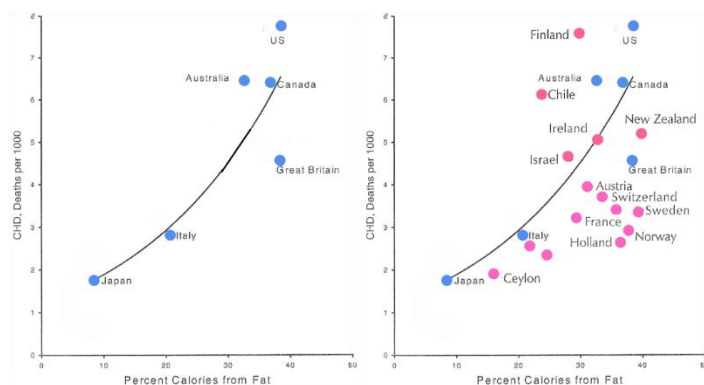


FIGURE 2 SCATTERPLOT OF SFA INTAKE IN 184 COUNTRIES VS MORTALITY DUE TO CVD AND T2D (R = 0.111)

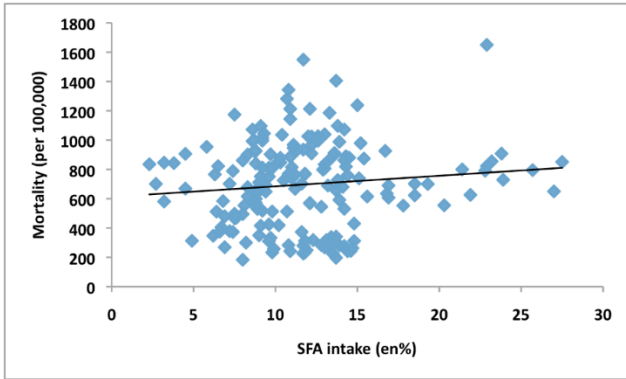


FIGURE 3 SCATTERPLOT OF SFA INTAKE IN 159 COUNTRIES VS MORTALITY DUE TO CVD AND T2D

