Review Article

Role of trans fatty acids in health and challenges to their reduction in Indian foods

Ghafoorunissa MSc PhD

Former Deputy Director, Department of Biochemistry, National Institute of Nutrition, Hyderabad, India

Evidence indicates that dietary trans fatty acids (TFA) obtained from partially hydrogenated vegetable oils (PHVO) increase the risk of coronary heart disease (CHD). Studies have implicated TFA in increasing the risk and incidence of diabetes. Furthermore, TFA may compromise fetal and early infant growth and development. In rats, partial substitution of either linoleic acid (18:2 n–6) with saturated fatty acids (SFA, 6 en %) or SFA with TFA (3 en % from vanaspati) decreased peripheral insulin sensitivity, but these effects were greater in TFA group. Since a large proportion of Indian population is insulin resistant, the TFA content in Indian edible fats/oils and foods should be reduced. Vanaspati (PHVO) provides up to 40% TFA, is used in Indian cooking and in the preparation of commercially fried, processed, bakery, ready-to-eat and street foods. TFA in biscuits and sweets range 30-40 and 6-26% of total fatty acids respectively. There is no regulation on TFA content in vanaspati, bakery fats and shortenings. Reduction in Indian edible fats/ oils and foods can be achieved by: a) specifying limits of TFA in vanaspati, bakery fats and shortenings by upgrading technology; b) advocating the substitution of natural plant oils containing lower percent of polyunsaturated fatty acids for PHVO. Indian edible oil industry needs to develop and adopt alternative technologies to produce zero TFA. Consumer education about negative health effects of TFA and providing food based guidelines to reduce TFA consumption in the entire population need to be actively pursued.

Key Words: Indian edible oils/foods, hydrogenation, transfatty acids, diabetes, coronary heart disease

INTRODUCTION

Trans fatty acids (TFA) are formed during industrial PHVO, a process widely commercialized in the mid 19th century to produce solid fats as healthier substitutes for animal fats. The TFA content of PHVO depends on the variables of the hydrogenation process (time, catalyst, temperature, hydrogen pressure), the types and proportions of oils and therefore variation in the composition of monounsaturated fatty acids (MUFA) and PUFA. The total TFA content can be upto 50% of total fatty acids, 18:1 9t (elaidic acid) is the major TFA, the other isomers are, 18:110t and 18:111t. PHVO also contain small amounts of trans positional isomers of 18:2n–6 and α-linolenic acids(18:3n–3).[1] Partial hydrogenation of oils increases the melting point and provides the required texture and consistency to prepare margarines, shortenings, bakery and frying fats and processed foods and increases the shelf life of the food products.

During refining of vegetable oils, deodorization step contributes to formation of TFA, 18:3n–3 is more sensitive to isomerisation. It is therefore recommended that the severity of deodorization conditions should be reduced to limit TFA content in refined oils to < 1 % of total fatty acids.

In ruminants, TFA are formed by bio-hydrogenation of MUFA & PUFA in fore-stomach. In consequence, milk, meat and other body fats of ruminants contain 2-9 % TFA. However, the contents of individual isomers differ, 18:1 11t (vaccenic acid) is the major TFA and the other 18:1 isomers are 9*t*, 10*t*, 6/8*t*, 12*t*, 13/14*t*. Conjugated linoleic acids (9c,11t-18:2 and 10*t*,12*c*-18:2) constitute < 1 % of TFA.[1]

Early studies which concluded TFA from PHVO to be nutritionally safe

Since 1950s several human studies have documented that serum total cholesterol levels predict CHD risk. Studies on effects of PHVO in humans showed either moderate or no cholesterolemic effects and animal studies showed inhibition in biosynthesis of long chain polyunsaturated fatty acid (LCPUFA) from 18:2n–6. Since adequate dietary18:2n–6 prevented this inhibition and its levels are high in most human diets, TFA from PHVO have been considered to be safe[2]. In 1985 FASEB consultation commissioned by US Food and Drug Administration (FDA) concluded that 'TFA from PHVO appear to be the equivalent of oleic acid (cis MUFA) in their cholesterolemic properties in humans'. Subsequently PHVO have been advocated as healthier substitutes for animal fats.

Corresponding Author: Dr Ghafoorunissa, Flat # 402 Maphar Comfortek Apartments, Shanthinagar Colony Masab Tank, Hyderabad – 500028, Andhra Pradesh, India
Tel: + 91 040 23324138
Email: ghafoorunissanin@yahoo.com
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Recent data which justify concerns about safety of TFA for human health

Coronary heart disease. The current concepts on the patho- physiological processes involved in the causation and complications of CHD are beyond increase in serum total cholesterol; and comprise disturbances in lipoprotein metabolism (high LDL and low HDL cholesterol, high triglycerides and ox-LDL), endothelial dysfunction, inflammation, prothrombotic shift in arterial homeosatasis (high lipoprotein (a), and thromboxane/ prostacyclin ratio), and insulin resistance.[3] Evaluation of the effects of dietary TFA on these processes has changed our perception of PHVO as 'healthy alternatives' to animal fats. Since 1990 several controlled studies in humans on the effects of substitution of TFA for oleic acid have documented increase in the level of total and LDL cholesterol, - similar to effects of SFA (atherogenic). However, unlike substitution of SFA for oleic acid, TFA resulted in decrease in the levels of HDL, (anti-atherogenic). Further, a dose response relationship has been reported in the range of TFA intakes between 3-11 % of dietary energy. A meta analysis of 12 randomized controlled trials on the effects of isocaloric replacement of SFA or *cis* MUFA/PUFA with TFA on serum lipids showed that compared with the consumption of equal calories from SFA or cis MUFA/ PUFA, TFA raised LDL cholesterol (atherogenic), reduced HDL cholesterol (anti-atherogenic), increased ratio of total cholesterol: HDL cholesterol, a powerful predictor of CHD risk. The adverse effects on serum LDL and HDL explain just part of CHD risk.[4] TFA also increase other intermediate risk end points of cardiovascular disease, namely, increase in serum triglycerides, lipoprotein (a), serum markers of chronic inflammation and endothelial dysfunction and decrease in particle size of LDL. Epidemiological studies have shown a strong positive association between TFA intake and CHD risk. A meta analysis of four prospective cohort studies showed that increase of ~2 % energy TFA was associated with increase in incidence of CHD by 25% Since the adverse effects of dietary TFA have been observed at intakes as low as 1-3 %energy (2-7g/ person /day), Mozaffarain et al. advocate <0.5 %energy TFA to minimize health risks.[4]

Insulin resistance and diabetes. Insulin resistance (IR) of peripheral tissues is central to the pathogenesis of type 2 diabetes. It precedes the development of diabetes and is associated with a cluster of cardio specific risk factors (metabolic syndrome). Evidence on the effects of TFA on IR and in increasing risk of diabetes is conflicting and limited in scope.[5] A review of randomized cross-over feeding studies indicates that in overweight or diabetic individuals high intakes of TFA increased postprandial insulin .Two prospective studies - The Nurses' Health study, and The Health Professionals Follow- up study have shown that TFA were positively associated with increased risk of diabetes. However, in The Iowa Women's Health Study no association was found between TFA intake and diabetes. Recent studies show that IR initiates in adipocytes, and affects insulin sensitivity in skeletal muscle and liver. Further, nuclear transcription factors (PPARs, TNFα), and adipocytokines exert profound effects on insulin sensitivity.[6] Dietary fatty acids modify insulin sensitivity through their incorporation into structural lipids in skeletal muscle and adipose tissue that affects fluidity and responses of insulin receptors.[7]

Table. Summary of results on effect of dietary SFA/ TFA on insulin sensitivity in rats #

Group	SFA	TFA
	Plasma	
Insulin	↑	↑
Triglycerides	↑	↑
	Functional parameters	
Adipose tissue		
Lipolysis	↑	↑
Ins stim. antilipolysis	↓	↓↓
Glucose transport	↓	↓↓
Diaphragm		
Glucose transport	±	↓
	Structural parameters	
Adipocyte		
LCPUFA*	↓	↓
Fluidity	±	↓
Skeletal muscle **		
Triglycerides	±	↑
Linoleic acid (18:2n-6)	↓	↑***
LCPUFA	↓	↓↓***

Ref 13 &14

Arrows indicate changes by one way ANOVA ($p < 0.05$); *plasma membrane phospholipids; **diaphragm, soleus, extensor digitalis longus; ***decreased desaturase activity

TFA and early human development. An adequate supply of LCPUFA is critical during fetal and infant early development. Recent evidences indicate that nutritional imbalances in *utero*, in addition to affecting birth weight and infant early growth and development may also influence the risk of diet-related chronic diseases in late adult life.[3] Hence, during pregnancy, lactation and infancy, it is important to ensure adequate intake of 18:2n–6 and 18:3n–3 and minimize factors which interfere with their availability and inhibit biosynthesis of LC PUFA.

TFA can be incorporated in placenta, breast milk, maternal and fetal tissues. Studies have shown negative association between TFA and LC PUFA content in cord blood and breast milk lipids. Furthermore, negative associations have been shown between TFA intake/exposure and indices of fetal and infant early development (birth weight, length, gestational age). Although a causal relationship between TFA exposure and adverse effects on fetal and infant early development has not been established, the available data justifies concerns regarding safety of TFA in diets of pregnant and lactating women and in infants.[8]

Today, there is scientific consensus that TFA increase CHD risk. WHO 2003 recommends that TFA should be limited to < 1% total energy in human diets.[3] As a consequence, in several developed countries reducing the intake of TFA has become part of nutrition policy.

Indian Scenario

Edible oils and Vanaspati. In India the annual total consumption of edible oils/fats is ~12 million metric tons; ~40% of demands are met from imports. Palm oil constitutes ~75% of the imports.[9]

Edible oil/fat intake in India is income-dependent and highly skewed; intake in rural and urban poor (including pregnant and lactating mothers) is much lower than the desirable levels and there are strong regional preferences for the type of edible oil(s).[10,11] Our earlier work documented that large segments of Indian population have inadequate n–3 PUFA nutritional status.[11]

Vanaspati (PHVO) entered India in 1960s as a solid cooking fat that was promoted as vegetable ghee. It accounts for ~10 % of total production of edible oils. Indian vanaspati is prepared by partial hydrogenation of mixture of vegetable oils. The choice of oil used for vanaspati varies with domestic availability and imports. In order to increase the oilseed utilization and production, the government of India subsidizes the price of minor and unconventional oils used for manufacture of vanaspati. The 'Vegetable oil products regulation' order 1998 provides list of 20 oils including imported edible oils which are permitted for hydrogenation of vegetable oils. Data from Directorate of Vanaspati shows that ~25% indigenous oils and ~75% imported oils are used in manufacture of vanasapati. Palm oil and its fractions constitute a major proportion of imported oils. In India, there is no regulation on TFA content in vanaspati, bakery fats and shortenings. Analysis of fatty acid composition in our laboratory of currently available brands/batches of vanaspati sold in market (n=24) across the country showed wide variation in total TFA content, elaidic acid is the major trans isomer. The number of brands/batches having different range of TFA is as follows: 11, 5 - 15%, 5, >15-20 and 8, >20 -38 % of total fatty acids. The lower TFA levels is some brands/batches is due to use of higher proportion of palm oil or its fractions in the mixture of feed oils used for hydrogenation.

In north India, vanaspati is used as a cooking medium, maximum consumption can be ~20g/person /day.[10] In Delhi which is multicentric market, 37% of market is for vanaspati.[12] Vanaspati is widely used in the preparation of commercially fried, processed, bakery, ready to eat and street foods. In bakery industry, vanaspati, butter and speciality fats (margarines, shortenings, gel) account for 60, 20 and 10 % respectively of total usage. The limited data obtained in our laboratory on various type of biscuits (n=14) and Indian sweets (n=8) purchased from local bakeries show that TFA range between 30-40 and 6-26% of total fatty acids respectively. In recent years consumption of foods containing vanaspati has increased. Therefore, in the current urban Indian diets (particularly those of middle and upper income groups) high intake of TFA may be part of several changes in dietary and other lifestyle patterns (high energy, fat calories and SFA, low dietary fibre, micronutrients and n–3 PUFA) which contribute to the present day high prevalence of diet – related chronic diseases.

EFFECTS OF TFA FROM VANASPATI ON INSULIN SENSITIVITY IN RATS

To evaluate the effects of TFA from vanaspati on insulin sensitivity,[13,14] WNIN male weanling rats were divided into 4 groups and fed casein based diet containing 10% groundnut oil (control group; SFA–4 %en and 18:2n–6 - 8% en) or palmolein (SFA group; SFA - 10 %en and 18:2n–6 - 2% en) or blends of vanaspati and safflower oil in ratios of 9.3:0.7 (TFA1 group ; TFA – 3% en and 18:2n–6 - 2% en), or 8:2 (TFA2 group ; TFA – 3% en and 18:2n–6 - 4% en) for 12 weeks. At the end of 12 weeks of feeding, basal serum glucose and insulin, total triglycerides and total cholesterol and after oral glucose load serum glucose and insulin were measured. To assess the effects on membrane lipid composition, adipocyte plasma membrane and skeletal muscle phospholipid fatty acid composition and intra-myocellular triglyceride content were determined. To assess insulin action in adipocytes, insulin stimulated glucose uptake and anti-lipolysis, and lipolysis were measured. Insulin stimulated glucose uptake was determined in diaphragm. The results summarized in table show that compared with control group, SFA as well as TFA fed groups had high levels of fasting plasma insulin and triglycerides, In both SFA and TFA fed groups in adipocyte, LCPUFA in plasma membrane phospholipids, antilipolytic effect of insulin and insulin–stimulated glucose transport were decreased as compared to control group.

However, adipocyte plasma membrane fluidity, decreased only in TFA groups and the magnitude of decrease in the anti-lipolytic effect of insulin and insulin stimulated glucose transport were greater in TFA groups. The data on skeletal muscle (soleus, diaphragm and extensor digitorum longus) lipid composition showed that dietary TFA increased intra myocellular triglycerides, increased 18:2n–6 and decreased LCPUFA levels. The changes in diaphragm lipids in TFA fed groups were associated with decrease in insulin stimulated glucose transport. These findings suggest that diets providing 10% en SFA decreased adipocyte insulin sensitivity. However, substitution of 2% en SFA and 1% en cis MUFA with TFA decreased insulin sensitivity to a greater extent. The data on expression of genes associated with insulin sensitivity in adipose tissue (resistin, adiponectin, GLUT4, LPL and PPAR) suggest that dietary SFA and TFA differently affect the genes associated with insulin sensitivity.[15] The magnitude of increase in TFA and decrease in insulin sensitivity in adipocytes and skeletal muscle was similar in TFA1 and TFA2 groups. Since increase in dietary 18:2n–6 from 2 to 4% en did not prevent TFA induced decrease in insulin sensitivity, it is necessary to reduce the absolute intake of TFA. Further, maternal diets providing 1%en TFA from vanaspati during fetal and postnatal early growth and development of pups decreased the relative mRNA expression of adiponectin in late adult life (Ghafoorunissa etal, unpublished). These findings suggest that maternal TFA intake may program F1 generation to IR. The above findings in rats, have provided key insights in support of the effects of TFA in leading to IR and therefore possibly increasing the risk of developing type 2 diabetes.

In India, a large proportion of population is insulin resistant and the prevalence of diabetes and CHD is high. Furthermore, the incidence of low birth weight is high

due to maternal under-nutrition and poor antenatal care. To combat these nutritional and health problems, reduction of TFA intake along with other dietary and life style changes need to be actively pursued.

Options for reducing TFA in edible oils and foods consumed in India

Government of India under Prevention of Food and Adulteration Act (PFA**)** needs to specify limits on the content of TFA in PHVO (vanaspati, bakery fats and, shortenings), refined fats and oils and processed foods and the regulatory framework would have to achieve effective enforcement of the above specifications.

The edible oil and food industry in India should be proactive in responding to options which can be implemented immediately and also invest in alternative technologies that are being developed to reduce / eliminate TFA.

Edible oils. To achieve the lower TFA limits in Indian vanaspati, bakery fats and shortenings, the industry would have to improve existing hydrogenation technology, use blends of vegetable oils of lower unsaturation, use fractions high in solids derived from natural oils (palm, palm kernel, coconut). For refined vegetable oils quality parameters should include TFA. It is perhaps necessary to generate data–base on TFA content in refined oils, and if the data–base shows high TFA, modifications in deodorization step have to be considered so as to ensure that TFA are within the specified limits. The edible oil industry needs support and investment to develop, set up and operate alternative technologies (enzymatic interesterification). Alongside, it is necessary to evaluate the long term nutritional and health effects of fats and oils obtained through these new technologies.

Food industry. The food processing industry should select zero / low TFA oils and substitute natural vegetable fats soft at room temperature for hard fats. Furthermore, even the food processing technology should minimize formation of TFA.

Nutrient label. To give consumers a choice the nutrient label on fats/oils and processed foods should give contents of TFA and SFA separately. Nutrient labeling will also encourage manufacturers to lower TFA content in their products.

Increase public and political awareness. Substitution of natural vegetable oils and fats soft at room temperature for hard fats should be emphasized. In **t**he Indian food based dietary guidelines (FBDG), those for reducing TFA have to be included. Alongside, key messages on the negative health effects of TFA and information on products which contain TFA should be prepared.

Restaurants/fast food joints. Steps to be initiated to insist that restaurants and fast food joints disclose use of 'PHVO' in served preparations.

In brief combination of different approaches outlined for industry, their effective enforcement by regulatory framework and consumer education to cut down intake of foods / fats and oils containing high levels of TFA would contribute to reducing TFA in Indian diets.

AUTHOR DISCLOSURES

Ghafoorunissa, no conflicts of interest.

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