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- **Trans Fatty Acids and Their Health Implications**
- **Methylmalonic Acidemia**

Nutrition Foundation of India Building, C-13 Qutab Institutional Area, New-Delhi-110016
Tel: 91-11-26857814,26962615 Fax: 91-11-26857814
E-mail: nfi@nutritionfoundationofindia.res.in; crnssindia@gmail.com Website: www.crnssindia.res.in

To Our Readers

Dear Readers,

The current issue (52) of the CRNSS Update Series "Nutrition in Disease Management" consists of a lead article about a widely discussed subject in the field of nutrition, trans fatty acids. The article describes details of research on trans fatty acids carrier out both nationally and internationally and also discusses the relevance of trans fatty acids in specific clinical situations.

The lead article is followed by a very brief description of a practical approach to dietary management in a patient with methylmalonic acidemia, a disorder of amino acid metabolism, which is a nutritional challenge.

Dr. Sarath Gopalan
Executive Director, CRNSS
and Editor

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Trans Fatty Acids and Their Health Implications

Dr. Ahmed Ibrahim
Scientist D
National Institute of Nutrition
Hyderabad, Andhra Pradesh, India

Introduction

Fats and oils are the major components of our diet. In addition to providing a concentrated source of energy, dietary fat has several physiological functions such as providing essential fatty acids, facilitating the delivery of fat soluble vitamins, improving texture and palatability and providing satiety. The nutritional and health benefits of dietary fats depend on the type of fatty acids and the minor components such as tocopherols, tocotrienols, lignans, phytosterols etc present in the non-glyceride fraction of the vegetable oils. Therefore, current recommendations on dietary fat are now laying emphasis on the type of fat rather than the quantity. The pathogenesis of several diet-related chronic diseases such as cardiovascular disease, type 2 diabetes mellitus , hypertension, inflammatory bowel disease, certain type of cancers, neurological and neuropsychiatric disorders are directly or indirectly related to dietary fats.

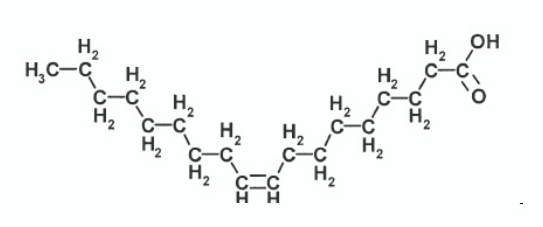
Trans fatty acids are unsaturated fatty acids that contain one or more double bonds in trans configuration (1). They are formed predominantly during partial hydrogenation of vegetable oils. The hydrogenation process was developed in the early part of the twentieth century to meet the increasing demand of solid fats and to replace animal fats which contain high levels of saturated fatty acids and cholesterol. Partial hydrogenation converts liquid vegetable oils into solid fats which are less expensive and have longer shelf-life as compared to animal fats. This process not only alters the physical properties of vegetable oil but also substantially alters the biological and health effects of fatty acids. There is now strong evidence that trans fatty acids are deleterious to human health and are more harmful from this standpoint than saturated fatty acids. Since trans fatty acids pose a serious threat to human health, there is a need for their reduction in the diet.

Chemistry of trans fatty acids

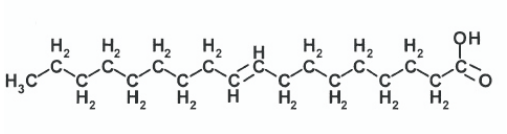
The double bonds present in the acyl chain of the fatty acids occur either in cis configuration (hydrogen on same side of the plane) or trans configuration (hydrogen on opposite side of the plane). The unsaturated fatty acids present in nature (vegetable oils and endogenously synthesized fatty acids) are in cis configuration. Trans fatty acids are formed in large amounts during artificial processing of vegetable oils while some amount of trans fatty acids exist naturally in dairy products and meats. Depending on the position of the double bond, several positional isomers are possible. During partial hydrogenation of vegetable oils, the cis double bonds present in the fatty acids are converted

into trans configuration. This change in configuration completely alters the physical property of the vegetable oils. The cis configuration induces a characteristic "U" shaped bend in the acyl chain; therefore they are less tightly packed and exist

as liquid at room temperature due to lower melting point. Compared to cis, trans configuration has more rigid structure similar to saturated fatty acids and tightly packed. Therefore the fatty acids in trans configuration exists as solid at room temperature due to higher melting point (Figure 1).



Oleic acid (18:1, cis isomer)
Melting point: - 5°C (liquid)



Elaidic acid (18:1, trans isomer)
Melting point : 42° C (solid)

Figure 1. Structure and melting point of cis and trans fatty acids

Sources of trans fatty acids

Trans fatty acids in the diet originate from three sources (1).

Industrial hydrogenation

Trans fatty acids are formed mainly during partial hydrogenation of vegetable oils and the trans fatty acid content of the hydrogenated fat varies from 10-40%. Several factors such as polyunsaturated fatty acid (PUFA) composition of the native oil, type of catalyst and hydrogenation conditions such as temperature and pressure determine the trans fatty acid levels and the type of trans isomer. The major trans isomer present in the partially hydrogenated vegetable oil is 18:1 t (80-90%) isomer. Among the 18:1t isomer, elaidic acid (18:1 Δ9 t) is the major trans isomer (85-95%). Other trans isomers include 16:1 t, 18:2 t and 18:3 t.

Ruminal biohydrogenation

Small amount of trans fatty acids are also formed by bacterial hydrogenation of PUFA in the stomach of ruminants. Therefore meat and dairy products of ruminants (cattle, sheep, goat etc) contain trans fatty acids in the range of 1-5%. In contrast to elaidic acid which is the major trans fatty acid present in hydrogenated fats, the ruminant fat contains vaccenic acid (18:1 Δ11 t) as the

major trans isomer. In addition to vaccenic acid, ruminant fat also contain conjugated linoleic acid in which the double bond is separated by single bond.

Thermal treatments

Vegetable oil processing such as refining and deodorization and other thermal treatments such as cooking and frying also generate trace amounts (1-3%) of trans fatty acids. Unlike partial hydrogenation, thermal treatments of vegetable oils induce mainly 18:2 and 18:3 trans isomers. Trans fatty acid formation during thermal treatment of vegetable oils depends on temperature and duration of the treatment.

Trans fatty acids in Indian diets

In developed countries such as Europe and North America, the average intake of trans fatty acids vary between 2 – 4 energy % or between 5 - 10g/person/d in an average 2000 kcal/d diet (2). In India, partial hydrogenation of vegetable oil was introduced in 1960s and marketed under the brand name "Vanaspati". It is mainly used as a substitute for ghee. Vanaspati account for 10% of total production of vegetable oils. Approximately 55% of vanaspati manufactured in India is consumed mainly in Haryana, Punjab, Uttar Pradesh and Himachal Pradesh where vanaspati is used as a cooking medium (3). In these states, the maximum consumption can be 20g/person/d. Trans fatty acid content of vanaspati is widely variable. Analysis of trans fatty acid content of various brands of vanaspati collected from markets across the country in our laboratory showed considerable variability in fatty acid content (5-38% of total fatty acids). The low trans fatty acid content in some brands of vanaspati could be due to use of high proportion of palm oil or its fraction for the hydrogenation process. Vanaspati is widely used in the preparation of deep fried fast foods, sweets and savoury items and bakery products. In recent years, the intake of trans fatty acids is increased due to increased consumption of fast foods, ready to eat foods and bakery products which are usually prepared using vanaspati. The limited data obtained in our laboratory on various types of biscuits and sweets purchased from local bakeries showed that trans fatty acid range between 30-40% and 6-25% of total fatty acids respectively (3). Ready to eat foods rich in fat such as samosa, paratha, bhatura, pakoras, pulao and halwa are very popular in India. High levels of trans fatty acids has been reported in some of the commonly consumed ready to eat food items such as halwa, fried potato chaat, bhatura and pulao (4). Intake of trans fatty acid is also dependent on income. A recent study conducted in New Delhi, India, showed high intake of trans fatty acids (> 1 energy %) in urban adult slum dwellers belonging to the low socio economic stratum of society (4).

Trans fatty acids in health and disease

Cardiovascular disease

Among the non communicable diseases, cardiovascular disease is one of the leading causes of death in developing and developed countries. The type of dietary fat is an important and modifiable risk factor for cardiovascular disease

(5). Several epidemiological studies have shown that high intake of trans fatty acids increases the risk of coronary heart disease (CHD). The strongest epidemiological evidence relating trans fatty acid intake to the risk of CHD comes from four major prospective studies namely: Health professionals follow up study, USA 2005; Nurses' health study, USA 2005; Zutphen elderly study, Netherlands 2005 and the Alpha tocopherol beta carotene cancer prevention study, Finland 1997 (6).

These studies unequivocally demonstrated the association of habitual trans fatty acid intake with the incidence of CHD events (myocardial infarction or CHD death). Based on the meta analysis of these prospective studies (around 140,000 subjects) it has been estimated that, a 2 energy % (approximately 4 g /day on a 2000 kcal diet) increase in trans fatty acid intake was associated with 23 % increase in incidence of CHD (pooled relative risk 1.23, 95% CI 1.11-1.37, $p<0.001$). In addition, retrospective case control studies in which adipose tissue trans fatty acid levels (a biomarker of trans fatty acid intake) was related to risk of non fetal myocardial infarction showed increased risk for each 2 energy % trans fatty acid intake (pooled relative risk 1.29, 95% CI 1.11-1.49, $p<0.001$). Moreover, compared to saturated fatty acids, trans fatty acid consumption increases the CHD risk to much greater extent. A case control study conducted in two major Indian cities (New Delhi and Bangalore) showed increased risk of ischemic heart disease with vanaspati consumption (7). There seems to be also an association between trans fatty acid intake and sudden cardiac death.

A case control study demonstrated significant association between trans fatty acid levels in erythrocyte membrane and risk of sudden cardiac death after adjusting other risk factors (8). Similarly a nested case control study also showed that high levels plasma phospholipid trans fatty acid was associated with increased risk of sudden cardiac death (9). In all these studies the trans isomer of linoleic acid (18:2 trans) was associated with risk rather than the 18:1 trans . Thus in addition to the effects on CHD, TFA consumption may also increase the risk of sudden cardiac death.

The CHD risk associated with trans fatty acid consumption could be partly due to its effect on lipoprotein metabolism. Until 1990 it was believed that both saturated and trans fatty acids have similar effects on lipoprotein metabolism because of structural similarities. However after 1990, a series of metabolic studies demonstrated that trans fatty acids are more atherogenic than saturated fatty acids due to the adverse effects of trans fatty acids on lipoprotein metabolism (6,10). Saturated fatty acids increase LDL cholesterol whereas trans fatty acids not only increase LDL cholesterol but also decrease HDL cholesterol. Because of combined effects of trans fatty acids on LDL and HDL, trans fatty acids increase LDL:HDL ratio which is the powerful predictor of CHD risk to a greater extent. The adverse effect of trans fatty acids on lipoprotein metabolism has been suggested to be due to increasing the activity of cholesterol ester transfer protein (11). Trans fatty acids also alter other intermediate risk CHD end points namely increased serum triglycerides and lipoprotein (a) and reduce LDL particle size (6).

The relation between trans fatty acid intake and incidence of CHD reported in prospective studies has been greater than that predicted by changes in plasma lipid levels suggesting that trans fatty acids may also influence the other risk factors of CHD in addition to its impact on lipoprotein metabolism (6). Subclinical inflammation is an important risk factor for CHD. Evidence from several epidemiological studies documented that high intake of trans fatty acid was associated with increased levels of proinflammatory markers such as TNFa, IL-6 and C-reactive protein (12). In patients with established CHD, membrane trans fatty acid levels were independently associated with activation of systemic inflammation. These findings were supported by the randomized trials. A recent intervention study further confirmed that high intake of trans fatty acids induce a low grade systemic inflammation (13). Thus in addition to the impact on lipoprotein metabolism, the proinflammatory effects of trans fatty acids are also likely to account for their adverse effects on CHD risk.

Impaired endothelial function is the primary event in the atherosclerotic disease. Habitual consumption of trans fatty acids has been shown to positively associated with circulating markers of endothelial function such as E.selectin and cell adhesion molecules (12). In a randomized trial, replacement of saturated fatty acids with trans fatty acids was associated with increase in E-selectin levels (12). In another study, the brachial artery flow mediated vasodilation which directly assesses the functional endothelial health was decreased by dietary trans fatty acids to greater extent than saturated fatty acids (12). Moreover, a recent prospective cohort study demonstrated that high intake of trans fatty acids was associated with increased risk of hypertension (14). Based on epidemiological and controlled trails, it is well established that trans fatty acids increase the CHD risk more than saturated fatty acids. The comparative effects of saturated and trans fatty acids on various CHD risk factors are given in Table – 1.

Ruminant trans fatty acids and CHD risk

In addition to hydrogenated fat, small amount of trans fatty acids which are present in ruminant fat such as dairy products and meats are consumed by humans. Although the deleterious effects of industrially produced trans fatty acids on CHD risk are well established, the role of ruminant trans fatty acids on CHD risk are debatable. This is because, the type of trans isomer present in ruminant fat is different from that in hydrogenated fat and may exert different metabolic effects. Results from few epidemiological studies have suggested that ruminant trans fatty acids may be less detrimental to heart health than industrially produced trans fatty acids (15). Other epidemiological studies observed that both ruminant and industrially produced trans fatty acids have similar negative effects on CHD risk (15). Interventional studies have demonstrated that high intake of trans fatty acids (4en%) from ruminant fat adversely affects cardiovascular risk factors whereas moderate intake (1.5 en%) which is well above the upper limit of present human consumption does not have any effect (16). A recent meta analysis of several observational studies showed that ruminant trans fatty acids have no association with CHD risk possibly due to low intake levels (17).

Table -1 : Comparative effect of saturated and trans fatty acids on various CHD risk factors

CHD risk factors	Saturated fatty acids	Trans fatty acids[a]
Lipoprotein metabolism		
Triglycerides	↑	↑
Total Cholesterol	↑	↑
LDL	↑	↑
HDL	±	↓
LDL:HDL ratio	↑	↑↑
Lipoprotein (a)	±	↑
Systemic inflammation	↑	↑
Endothelial function	±	↓
Blood pressure	?	↑

Insulin resistance and type 2 diabetes

Insulin resistance is an important risk factor for type 2 diabetes. It precedes the development of type 2 diabetes and is associated with multiple cardiovascular risk factors (obesity, dyslipidemia, low HDL cholesterol, hypertension and impaired glucose tolerance) known as metabolic syndrome. Recent studies have shown that insulin resistance is initiated in adipose tissue which in turn affects insulin sensitivity of skeletal muscle and liver. Several animal and human studies investigated the impact of dietary trans fatty acids on glucose-insulin homeostasis. Animal studies conducted in our laboratory demonstrated that both saturated and trans fatty acids decrease insulin sensitivity as evidenced by increase in plasma insulin levels (marker of insulin resistance) and decrease in peripheral insulin sensitivity (decreased adipose tissue and skeletal muscle glucose transport) (18,19). However compared to saturated fatty acids, trans fatty acids decrease the insulin sensitivity to a greater extent. Increasing linoleic acid (n-6 PUFA) in the diet did not prevent the adverse effects of trans fatty acids on insulin sensitivity suggesting that it is necessary to reduce the absolute levels of trans fatty acids. In addition, saturated and trans fatty acids differentially alter the expression of genes associated with insulin sensitivity (resistin, adiponectin, GLUT-4, LPL and PPARγ) in adipose

tissue (20). These animal studies documented that trans fatty acids are more deleterious than saturated fatty acids on insulin sensitivity.

Compared to animal studies, human studies provided variable results on trans fatty acid intake and insulin resistance (21). Short term human studies showed that among lean and healthy subjects trans fatty acid intake do not have significant effect on insulin resistance. However among individuals predisposed to insulin resistance, trans fatty acid intake resulted in higher levels of plasma insulin levels. The observation from prospective cohort studies of trans fatty acid intake and type 2 diabetes risk have been mixed (21). Two studies showed no relation between trans fatty acid consumption and diabetes risk, whereas another larger study showed significant positive association between trans fatty acid intake and diabetes risk particularly when the individuals were obese and were less physically active . Further studies are required to confirm the effects of trans fatty acid on insulin resistance and diabetes.

Recent studies have shown that dietary factors which affect insulin sensitivity may have an impact on some form of infertility. Factors known to increase insulin resistance such as obesity and less physical activity may increase the risk of infertility due to ovulatory dysfunction. Polycystic ovary syndrome is a condition that involves excess production of androgen and can lead to infertility. It is also associated with CHD, diabetes and metabolic syndrome. Recent prospective cohort study documented that high intake of trans fatty acids may increase the risk of ovulatory infertility possibly by increasing insulin resistance (22). Each 2en% increase in intake of trans fatty acids was associated with 73% increased risk of ovulatory infertility. Hence women with polycystic ovary syndrome should restrict the intake of trans fatty acids to reduce the risk of infertility.

Animal studies have shown that maternal trans fatty acid consumption may have long term adverse effects on glucose-insulin homeostasis in the offspring. Several studies have shown that high intake of trans fatty acids during pregnancy and lactation predispose the offspring to insulin resistance in the adult life (23,24). Our study also demonstrated that maternal intake of trans fatty acids even at low levels (1 en%) irreversibly alter the glucose metabolism of the offspring which may predispose the offspring to the development of insulin resistance and type 2 diabetes in adult life (25). These findings strengthen the importance of restricting the intake of trans fatty acids during pregnancy and lactation.

Weight gain and obesity

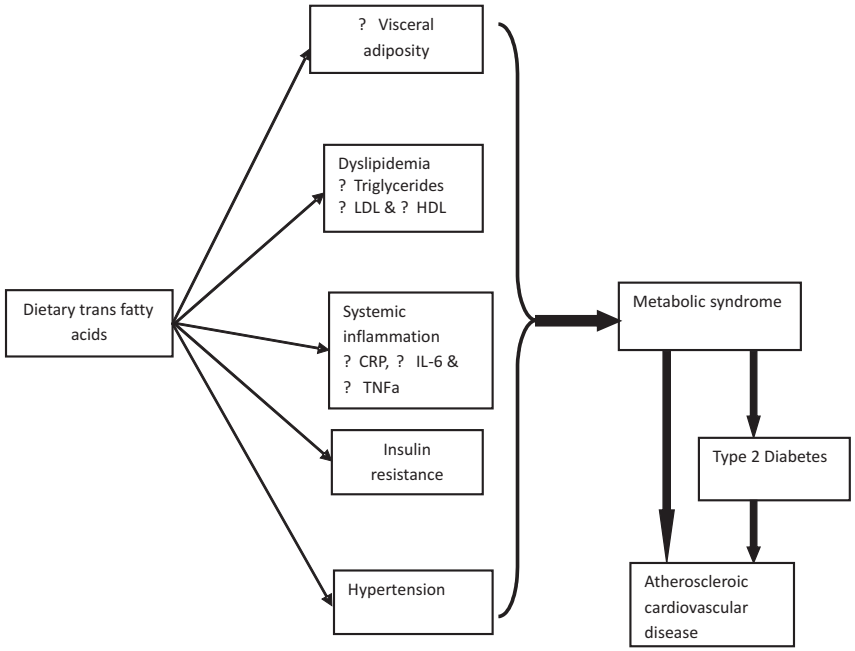
It is well established that high intake of saturated fatty acids induce adiposity thereby increase the incidence of obesity. Similarly high intake of trans fatty acids appears to increase the adiposity particularly the visceral fat accumulation. A long term study in monkeys showed that high intake of trans fatty acids (8 en%) significantly increases weight gain with increased intra-abdominal fat deposition and associated with insulin resistance (26). Further,

trans fatty acid intake was associated with hyperphagia, increased fat accumulation in liver and visceral adipose tissue and impaired glucose tolerance, all of which are important features of the metabolic syndrome (27). Prospective observational studies showed that, compared to other fatty acids, trans fatty acid consumption increased body weight and waist circumference to a greater extent (28). Moreover, high intake of trans fatty acids during pregnancy and lactation increases adiposity in breastfed infants and their mothers. These adverse effects of trans fatty acids on adiposity are of major concern since the recent increase in incidence of obesity, especially childhood obesity, in India, could be attributed to an increase in consumption of fast foods (29).

The effects of trans fatty acids on several cardiovascular risk factors including dyslipidemia, insulin resistance, visceral adiposity, systemic inflammation and hypertension, suggest that trans fatty acid consumption increases cardiovascular risk by contributing to the metabolic syndrome (Figure – 2).

Fetal and infant development

Figure -2 : Potential role of dietary trans fatty acids on metabolic syndrome and atherosclerotic cardiovascular diseases.



Long chain PUFA namely arachidonic acid and docosahexaenoic acid are critically important during pre and post natal growth and development (30). Arachidonic acid and docosahexaenoic acid are synthesized from the essential fatty acids, linoleic acid (n-6) and α-linolenic acid (n-3) respectively through series of desaturation and chain elongation pathways. Dietary factors which impair the metabolism of n-6 and n-3 PUFA or their incorporation into developing tissues may have adverse effects on fetal and infant development. Trans fatty acids are transferred from mother to the fetus across the placenta and incorporated into fetal tissues. Trans fatty acids are also transferred from the lactating mother to the infant during breat feeding.

As trans fatty acids are known to impair the metabolism of n-6 and n-3 PUFA to long chain PUFA, they may have adverse effects on fetal growth and development (31). In premature infants, plasma trans fatty acid level was inversely associated with birth weight. Another study showed an inverse relationship between long chain PUFA and trans fatty acids in cord blood lipids of full term infants. Similarly, high maternal intake of trans fatty acids was inversely related to length of gestation and birth weight. A recent cohort study demonstrated that maternal trans fatty acid intake is inversely related to long chain PUFA content in milk (32). Trans fatty acid consumption has been reported to be associated with the development of preeclampsia during pregnancy.

A retrospective case control study showed that pregnant women with high levels of erythrocyte trans fatty acid were at a much higher risk of preeclampsia than pregnant women with low levels of the same (33). Similarly, another case control study also showed that erythrocyte trans fatty acid levels, particularly the 18:2 trans, were positively associated with risk of preeclampsia (34). Further, increased fetal loss has been attributed due to high intake of trans fatty acids possibly by down regulating the nuclear transcription factor PPARΔ which plays a pivotal role in placental function (35). In India, the prevalence of low birth weight is high due to maternal undernutrition and poor antenatal care. Moreover, PUFA intake is also low. Since trans fatty acids have a negative impact on PUFA metabolism, it is important to minimize the consumption of trans fatty acids during pregnancy and lactation to prevent the adverse effects of trans fatty acids on fetal growth and development.

Cancer

Few studies investigated the relation between trans fatty acid consumption and risk of some cancers (36). A recent cross-sectional study documented that individuals with high intake of trans fatty acids had higher prevalence of colorectal cancer. The EURAMIC study which investigated the association between trans fatty acid content in adipose tissue and the incidence of breast, prostate and colon, demonstrated a positive association between trans fatty acids and incidence of breast and colon but not prostate cancer. In a case control study, serum trans fatty acid levels were positively associated with incidence of breast cancer. Overall the role of trans fatty acids in causation of cancer remains inconclusive and further studies are needed to establish an

association.

Regulatory aspects of trans fatty acids

It is now clear from the nutritional standpoint that consumption of trans fatty acids is potential harmful and has no health benefit. Substantial epidemiological data demonstrated the adverse effects of trans fatty acids on CHD risk as well as several diet related chronic diseases. Further, trans fatty acids are more harmful than saturated fatty acids and adverse effects are seen even at a low level of intake (1-3 en% or approximately 2-7g/d for a person consuming 2000 calories per day). Therefore, complete or near complete avoidance of trans fatty acids is necessary to minimize the adverse effects on CHD risk and towards meeting this goal, several expert committees have made evidence based statements that recommend limiting intake of trans fatty acids. Most European countries have set an upper limit for trans fatty acid intake at 1-2 en%. The joint FAO/WHO (2003) recommends that trans fatty acid intake in the diet should be kept below 1en%. The current dietary guidelines for the prevention of various diet related chronic diseases in Indians also recommends limiting trans fatty acid intake below 1en% (37). Food labeling has become mandatory in several countries, including India.

Following a long review process in USA, the FDA introduced a legislation that from January 1, 2006, all nutrition labels for conventional foods and supplements must list trans fatty acid content. Canada also passed a similar legislation in 2005. In Denmark, the legislation passed in 2004 mandated that all vegetable oils and fats used for the preparation of foods or imported food items must contain less than 2% trans fatty acids. This essentially eliminated the use of partially hydrogenated vegetable oils in the preparation of food items and reduced CHD related deaths.

In India, there are no clear guidelines for limiting trans fatty acid content of the vegetable oils. Most of the oil industries use unconventional oils and oils which are available in surplus. Moreover, the Indian government also encourages the vegetable oil industry by providing vegetable oils at a subsidized price and even allowing unrefined vegetable oils for the manufacture of vanaspati. The Prevention of Food Adulteration Act (PFA) of the Government of India is yet to specify the limit of trans fatty acid content in vanaspati and processed foods. Recently on the basis of recommendations given by National Institute of Nutrition, Hyderabad, the Food Safety and Standards Authority of India (FSSAI) has proposed to fix the limit for trans fatty acid levels in vanaspati. As per the recommendation, the vegetable oil industry requires to reduce the trans fatty acid content in vanaspati to 10% and should reduce it further to 5% in next three years.

Trans fatty acid alternatives

Several technologies were developed to reduce or eliminate trans fatty acids in food products (38). One such technology is interesterification, in which the fatty acids within the triglycerides are rearranged to yield customized melting characteristics and the resultant product is free from trans fatty acids.

However the health implications of interestified fats are yet to be known. Another method for reducing trans fatty content in food products is modification of the hydrogenation process such as using metal catalysts that prevent the formation of trans isomers and by changing the condition of the hydrogenation process to yield partially hydrogenated fat lower in trans fatty acids. Alternatively, the food industry can use tropical oils (palm oil, palm kernel oil and coconut oil) as the replacement for partially hydrogenated oils in backed goods due to their high saturated fat content and higher melting point. Use of trait enhanced oils such as high oleic sunflower and canola oils, low linolenic canola and soybean oils can reduce the trans fatty acid considerably. These oils produced through plant breeding and genetic engineering technologies have high oxidative stability due to low PUFA content and can be used for deep fat frying.

Conclusions

The major source of trans fatty acids in the diet is partially hydrogenated vegetable oils. Although partially hydrogenated vegetable oils have excellent culinary properties they are detrimental to human health. Evidences from epidemiological, observational and interventional studies have established that trans fatty acids particularly the industrially produced trans fatty acids increase the CHD risk. Ruminant trans fatty acids may not have any impact on CHD risk possibly due to low levels in the diet. It is possible to reduce or even eliminate trans fatty acids through appropriate dietary guidance, consumer education and use of alternative fats and oils such as tropical oils and trait enhanced oils in food preparation. In India the prevalence of CHD, diabetes and obesity are increasing rapidly and reducing the trans fatty acid intake might bring down the risk of these diet related chronic diseases.

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Methylmalonic Acidemia

Ms. Deepika Aggarwal
Senior Dietitian, Department of Dietetics,
Indraprastha Apollo Hospital, New Delhi, India.

Methylmalonic academia (MMA) is an inherited disorder in which the body is unable to metabolize certain proteins and fats (lipids) appropriately.

The goal of managing MMA is to achieve and maintain optimal nutrient intake and biochemical equilibrium. Maintenance of energy and fluid intake is important to prevent tissue catabolism and dehydration.

MEDICAL NUTRITION THERAPY:

- Maintain normal glucose levels and prevent hypoglycemia.
- **Increased intake of fluids** is recommended to remove abnormal metabolites through urinary excretion and also to normalize blood ammonia levels.
- Dietary protein restriction of 1- 1.5 g/kg/day is recommended. The diet restriction is more on protein-containing foods obtained from animal

DIETARY MANAGEMENT

FOOD GROUP	FOODS ALLOWED	FOODS TO BE AVOIDED
CEREALS	· Wheat and its products like refined flour, semolina etc · Rice and its products like rice flakes, puffed rice etc. · Spaghetti, Macaroni Noodles · Corn (tender)	· Most commercial cakes, cookies etc. **Read the label carefully.** · Avoid Ragi & Bajra
PULSES	All pulses are allowed	Soybean and its products.
FRUITS	All fresh fruits.	Grapes and dried peaches, apricots and dates.
VEGETABLES	All fresh vegetables.	· Read labels carefully and avoid those with unsafe ingredients i.e. those seasoned with margarine, butter, cream etc like in canned products · Spinach, cabbage, lemon and mushroom.
MEAT, POULTRY,FISH, EGGS		· Read labels carefully and avoid those with unsafe ingredients i.e. those seasoned with margarine, butter, cream etc like in canned products · Avoid meat, poultary, fish and eggs.
MILK		· Breast milk · All forms of animal milk · Cream · Cottage cheese · Yogurt · ice-cream · butter milk/lassi · chocolates
FATS	Refined vegetable oils shortenings	· margarine · butter · salad dressing

- and methionine.
- **FOOD LABELS SHOULD BE READ CAREFULLY.**
- People with MMA should have a regular daily supplement of calcium, vitamin B12 and carnitine.

FOOD PLAN :

FOOD GROUP	AMOUNT
CEREALS*	160-180gms
PULSE	60gms(2 katori)
VEGETABLES(root veg)	300-400g
FRUITS	200-250 gms
Oil	30-35gms
Corn starch	50-60 gms
Milk and milk products(diluted)	400-450ml

·20 g cereal = 1 cup rice = 1 cup dalia = 1cup upma/ poha = 4 Marie biscuits

SAMPLE MENU

TIME	SAMPLE MENU	QUANTITY
Bed tea(6 am)	Corn starch feed Marie biscuit	100ml
BREAKFAST (9:00 am)	· vegetable upma/ Suji kheer / nutritive porridge/dalia/iddli · paneer cutlet/aloo tikki	1 cup 1
MID MORNING (12:00 pm)	· corn starch feed · fruit	100ml
LUNCH (2:30 pm)	· rice /chapatti · Dal · Vegetable · Dessert(rice kheer/ custard etc)	1cup/1 in no. 1 cup 1 cup 1 bowl
TEA TIME (4:30 pm)	· Corn starch feed · Snack (any of the breakfast preparation)	
EVENING SOUP (6:30 pm)	· Soup · Partial cooked macroni/pasta	1 bowl 1 bowl
DINNER (9:00pm)	SAME AS LUNCH	
BED TIME (12:00 mn)	· Dessert(rice kheer/ custard etc) With corn starch	1 bowl
At 2:00 am	· Cornstarch feed	100 ml
At 4:00 am	· Cornstarch feed	100 ml

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