SATURATED FATTY ACIDS vs TRANS FATTY ACIDS: DIFFERENTIATED EFFECTS IN NUTRITION AND PUBLIC HEALTH
INTRODUCTION

Over the past years, non-communicable diseases (NCDs) are the leading cause of morbidity and mortality in the world. These diseases include cardiovascular disease (CVD), respiratory disease, cancer and diabetes, among others. According to the World Health Organization (WHO), NCDs affect low and middle income countries, where more than 75% (31 millions) of deaths occurred due to said diseases. Additionally, NCDs are the first cause for disability and its threat continues to grow (http://www.who.int/mediacentre/factsheets/fs355/es).

The risk of NCDs continues growing as the populations’ dietary habits change since the intake of foods that are rich in fat, saturated fat, industrially produced trans fats, sodium and sugars, among others, plus a sedentary lifestyle, increases on a daily basis. The amount of fat and the ratio of the consumed saturated fatty acids (SFA), unsaturated (UFA) and trans-fatty acids (TFA) are related to the risk of NCDs (PAHO, 2008).

Robust evidence indicates that a balanced diet, including a proper intake of SFAs, a reduction in sugar intake, the elimination of TFAs from foods, as well as a healthy lifestyle, contribute to protect health and prevent cardiovascular diseases within the population.

Currently there are different projects aimed at reducing the contribution of TFAs in foods, among them, there are technological alternatives and regulatory measures at a national and global level.

OVERVIEW OF FATTY ACIDS

The majority of lipids make up for the most important energy reserve of the body, providing 9 kilocalories per gram (Kcal/g), carrying fat-soluble vitamins and are found in a great variety of foods and preparations. Moreover, they develop physiological, immune and structural functions.

They are referred to as fats when found in a solid form and as oils when they are in a liquid form, according to room temperature. The most common ones are glycerolipids, which are composed mainly of triglycerides (TG). The latter are usually accompanied by small amounts of phospholipids (PL), monoacylglycerol (MG), diacylglycerol (DAG) and sterols and sterol esters. Fatty acids are the main components of these lipids which are necessary in human nutrition as a source of energy and to meet metabolic and/or structural functions (Fahy, 2005, FAO/WHO Expert Consultation, 2008).

Fatty acids may be classified by taking into account several characteristics such as length of the chain (short, medium, long or very long) or the chain’s saturation (saturated and unsaturated). Even though the metabolic role of fatty acids depends upon the length and saturation, this document will analyze the effect of the latter on human health.

The most common fatty acids (FA) of the diet have been subdivided in two groups according to the degree of unsaturation: Saturated fatty acids (SFA), which do not have double bonds and unsaturated fatty acids (UFA), which have a certain degree of unsaturation. It is important to bear in mind that this last group can be subdivided in monounsaturated fatty acids (MUFAs) having one double bond and polyunsaturated fatty acids (PUFAs) having two or more double bonds. As a general rule, these fatty acids have an even number of carbon atoms and unbranched structures. Double bonds of unsaturated fatty acids that exist in nature are often cis oriented.

From a chemical standpoint, a cis configuration means that the hydrogen atoms, carbon and other compounds bonded to double bonds, are in a single plane. If hydrogen atoms are found in opposite planes, the configuration is called trans (FAO/WHO Expert Consultation, 2008).
**Saturated fatty acids**

Fatty acids that contain only carbon-carbon single bonds, in other words, that do not have double bonds are called saturated. The abbreviation includes the number of carbon atoms and a zero after a colon, for example C18:0, which means that the fatty acid has 18 carbon atoms and that it has no double bonds in it. Saturated fatty acids (SFA) have an endogenous synthesis, necessary for some physiological and structural functions and they are also found in foods that we eat on a daily basis.

According to expert consultation of FAO/WHO, the FA including the saturated ones, are classified in the following subgroups:

- Short chain saturated fatty acids: 3 to 7 carbon atoms.
- Medium chain saturated fatty acids: 8 to 13 carbon atoms.
- Long chain saturated fatty acids: 14 to 20 carbon atoms.
- Very long chain saturated fatty acids: 21 or more carbon atoms.

SFAs are mainly found in animal and dairy fats, but considerable levels have also been observed in tropical oils such as the palm oil and coconut oil (FAO/WHO Expert Consultation, 2008).

The main saturated fatty acids are described in Chart No. 1. SFAs are the least chemically reactive and therefore are the most stable and have a longer shelf life than unsaturated fatty acids. If the chain is longer then the fusion point increases. They are solid at normal room temperature.

Among the short chain acids, butyric and caproic acids are the most important ones of this group. They are present mainly in dairy fats and are not a key feature of the most common vegetable oils. Regarding medium chain acids, caprylic, capric and lauric belong to this group, the first two are present in milk fat and the third one is present in coconut oil and palm oil. Among long chain saturated fatty acids, the most important ones are palmitic and stearic. The first one is present in palm oil and cocoa butter, in animal fat (terrestrial and marine) and cottonseeds. Stearic acid is present in the majority of vegetable oils (FAO/WHO Expert Consultation, 2008).

### Chart No. 1. Main saturated fatty acids and their food source.

<table>
<thead>
<tr>
<th>Common name</th>
<th>Abbreviation</th>
<th>Main sources</th>
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<tbody>
<tr>
<td>Butyric</td>
<td>C4:0</td>
<td>Dairy fat</td>
</tr>
<tr>
<td>Caproic</td>
<td>C6:0</td>
<td>Dairy fat</td>
</tr>
<tr>
<td>Caprylic</td>
<td>C8:0</td>
<td>Dairy fat, palm and coconut oils</td>
</tr>
<tr>
<td>Capric</td>
<td>C10:0</td>
<td>Dairy fat, palm and coconut oils</td>
</tr>
<tr>
<td>Lauric</td>
<td>C12:0</td>
<td>Palm and coconut oils</td>
</tr>
<tr>
<td>Myristic</td>
<td>C14:0</td>
<td>Dairy fat, palm and coconut oils</td>
</tr>
<tr>
<td>Palmitic</td>
<td>C16:0</td>
<td>The majority of fats and oils</td>
</tr>
<tr>
<td>Stearic</td>
<td>C18:0</td>
<td>The majority of fats and oils</td>
</tr>
</tbody>
</table>

*Taken and modified from: Fats and Fatty Acids in Human Nutrition Expert Consultation. Study of FAOs Diet and Nutrition. FAO and FINUT, 2012*
**Trans fatty acids**

Unsaturated fats have carbon-carbon double bonds (-CH = CH-) and depending upon space orientation there can be two configurations: *Cis* and *Trans*.

Unsaturated fats having a *cis* configuration and are found in a less packed form, tend to be liquid at room temperature and are subject to rancidification. On the other hand, *trans* double bonds have a more rigid configuration. Their double bonds require less space, in other words they are more packed and rigid, having a melting point close to room temperature (intermediate point between saturated and unsaturated fats in *cis* configuration.) This melting point gives it favorable organoleptic characteristics such as texture and flavor (Giacopini, 2008).

There are two sources of *trans* fat generators: Natural and industrial. Natural *trans* fats are formed in the rumen of polygastric animals such as cows, sheep and goats through a partial bio-hydrogenation process of unsaturated fatty acids. This bio-hydrogenation occurs by the action of gastric isomerases bacteria in the rumen, which change the *cis* double bonds of unsaturated fats to the *trans* position. It is estimated that approximately 5% of total intake of TFAs in the world’s diet comes from fats of ruminants.

TFAs that are generated by means of an industrial process, are made through partial hydrogenation of oils. This process is carried out under pressure and temperature conditions were hydrogen gas is added to the oil in the presence of a metal catalyst (nickel.) In this process, double bonds provide structural modifications: The double bond can be hydrogenated transformed into a single bond (saturated), the location of the double bond changes (formation of positional isomers) and the other bond can change its spatial configuration giving rise to *trans* isomers. When hydrogenation is complete, the result is a product that contains 100% of saturated fatty acids. On the contrary, a blend of saturated, monounsaturated and polyunsaturated *cis* and *trans* fatty acids is produced when the hydrogenation process is partial. *Trans* isomers that are largely formed in the partial hydrogenation of oils are the linoleic fatty acids (Castro-Martinez, 2010).

Thermal treatments within the deodorization processes, in the refining of vegetable or fish oils, or extended heating and frying at high temperatures of oils at high temperatures, might also generate TFA. Thermal treatments mainly produce geometric isomers and few positional isomers. TFAs start to form at 150°C and it significantly increases at temperatures higher than 220°C. Some European countries have established that the frying temperature must not exceed 180°C (Suaterna, 2009).

Contents of TFA in food is highly variable. They can be present in foods made with partially hydrogenated fats (margarines, shortenings, pastry products, precooked dishes, hamburgers, snacks, dehydrated soups, among others) and the lowest diet contributions are present in meats and dairy products. TFAs of animal origin from ruminant fat are found in concentrations of 4% to 6%, while industrialized foods might have a higher concentration if they do not meet established standards provided by the different regulatory entities. However, during past years, especially in the early ’90s, the food industry has improved its technological processes and since then products with very low TFAs contents, are being produced (Chardigny, 2008).

Current nutrition recommendations for the population include a reduction of SFAs intake and the elimination of TFAs, since there is enough evidence to proof its connection with the development of NCDs. The World Health Organization (WHO) recommends to reduce the total consumption of fat to less than 30% of the total caloric value (TCV), saturated fats to less than 10% and *trans* fats to less than 1% of the daily caloric value and to substitute these fats for non-saturated fats.

**HEALTH EFFECTS OF FATTY ACIDS INTAKE**

Since last century, diverse investigations have demonstrated that there is a connection between the intake of fatty acids (FA) in the diet and NCDs. Excessive calorie intake and sedentary life styles promote weight gain and an excessive storage of fat tissue, which affects overall health condition. An increase in the amount
and the change in the fat quality intake of the diet is an important characteristic of nutritional transition reflected in the diet regimes of the countries.

There has been a great emphasis on the importance of total fat intake, even though the importance of taking specific fat acids has been overlooked for many years. Studies over the past decades have demonstrated that it is not only important to reach the goal of total fat intake (20-35% total caloric value (TCV)), but also to check the quality of fat intake. Being able to accept that each fat acid has a different impact on health, even though they belong to the same category, has led its study towards new frontiers and highlights the fact that the study of a nutrient should not only be done globally but each of its components and the interaction between them should be seen as well. (Vannice, 2014)

A varying proportion of daily calorie intake of fats comes from saturated fatty acids. Only in the two most prosperous regions (parts of North America and Europe) saturated fats account for 10% or more of the energy contribution. In other less developed regions, the proportion of dietary energy by means of saturated fatty acids is lower, between 5% and 8%, and for the most part, it does not vary in time.

The first evidence of TFAs adverse effects on health were published in the ‘90s, when diverse studies applied on humans showed that its intake increased the risk of suffering cardiac alterations as much or even more than saturated FA (Mensink, 1990).

In different parts of the world, 80% of dietary trans-fat comes from processed foods such as pastry products, margarines and snacks among others, and 20% comes from foods of animal origin (Vandana, 2011).

It is well known that an excessive intake of TFAs has a significant connection with CVD, as well as inflammatory conditions, insulin resistance and obesity. These FA lead to an endothelial dysfunction and an unfavorable alteration of blood lipids, including an increase in low-density lipoprotein (LDL) and a decrease of high-density lipoprotein (HDL).

However, recent systematic review articles about randomized studies and prospective cohort, have requested a re-evaluation of the current intake guidelines and SFA effects on health.

Furthermore, the need to eliminate TFAs at a global level has intensified due to countless research that has shown its adverse effects on health.

**Saturated Fatty Acids**

During the early ‘50s, Keys and col, with the study titled “Atherosclerosis, a Problem in Newer Public Health” better known as the seven countries study (United States, Canada, Australia, England, Italy and Japan) stated the hypothesis that a high-fat diet produced a greater amount of cardiovascular disease (CVD) and vice versa, better known as the “diet-heart hypothesis” (Keys, 1957). However, simultaneously, the scientific community questioned this hypothesis due to some flaws. One of those critics was Dr. Yerushalmy, from Berkeley University in California, who stated in his document, published in 1957, that whilst Keys’ results from the seven analyzed countries seemed to support the hypothesis, at that moment there were statistics not only of those countries but also of 22 countries around the world. When data from all countries was included, the apparent link between fat intake and the onset of cardiovascular diseases disappeared. For example, in Finland the mortality rate caused by cardiovascular diseases was 24 times higher than in Mexico, even when the fat intake rate in both countries was similar.

The other critique to this study was based on the fact that Keys only observed the connection between two phenomena and did not assess the cause-effect as such, which opened the possibility for the existence of other confounding factors that were producing the effect. For example, even though Americans consumed more fat than Japanese did, quite likely they also consumed more sugar, white bread and watched more television making them more sedentary.

In spite of the flaws identified in the Keys and col study, the diet-heart hypothesis he stated was much promoted by the American Heart Association (AHA) and by the media.
Furthermore, Keys published another study carried out in seven countries (United States, Japan, Italy, Greece, Yugoslavia, Finland and Netherlands) and considered as his greatest contribution to the study of this issue, where he stated that the fat intake of animal origin was a strong indicator of the onset of heart attacks in a 5-year period. Moreover, he observed a connection between dietary cholesterol and mortality due to cardiovascular diseases. These observations led him to conclude that saturated fats of animal-based sources, and no other type of fats, increased plasmatic cholesterol and ended up producing cardiovascular disease (Keys, 1984).

Even though this study also had methodological problems, such as, for example, the fact that this association was not observed in 3 out of 7 countries (Finland, Greece and Yugoslavia) and that the eastern part of Finland reported 5 times more cardiovascular problems than the western part of the same country, and although the difference in animal fat intake was almost identical, this hypothesis was very popular during several years. It governed both what was presented in the management guidelines of cardiovascular diseases and what was considered to be a healthy diet.

Subsequently, Kromhout et al found a highly positive correlation between CVD with the intake of the 4 long chain SFAs (lauric, myristic, palmitic and stearic) and TFAs.

However, an analysis of the study about the risk factors of developing CVD in 80.082, carried out during 14 years by Harvard’s School of Public Health and published by Frank Hu in 1999, did not find a correlation between the consumption of foods rich in saturated fats and a higher risk of developing CVD. Even though cholesterol was higher, it managed to estimate that the substitute of 5% of the energy came from SFA by means of unsaturated fat, and thus might reduce the risk of CVD in 42%. From these results, we must recognize that it is more important to know about the type of fat consumed and its correlation with CVD, than the percentage of total consumed fat.

Mensik in 1992 and 2003 carried out a study that was later resumed by the WHO in 2016, which demonstrated that the lauric (12:0), myristic (14:0) and palmitic (16:0) fat acids have a similar effect on the plasmatic levels of lipids, generating an increase of LDL and HDL and a reduction of triglycerides when carbohydrates are replaced in the diet. On the other hand, stearic fat acid does not show this effect when the intake of carbohydrates is replaced (Mensink, 1992; Mensink, 2003; Mensink, 2016). Broadly speaking, they demonstrated that each of the saturated fat acids might have a slightly different behavior regarding cardiovascular disease, where:

In the case of lauric acid, the proven net effect has been the reduction of Cholesterol:HDL (CT:HDL) ratio in a similar level to PUFA or MUFA; therefore, coconut oil, high in lauric acid, has been suggested. It is beneficial for cardiovascular health; however, diverse studies have demonstrated that this oil, rich in caproic (8:0), caprylic (10:0), lauric and myristic acids, increase plasmatic levels of LDL and the CT:HDL ratio compared with olive and palm oils where saturated fatty acids are mainly provided by palmitic acid (Sundram, 1994; Voon, 2011). Furthermore, studies in animal models have demonstrated that coconut oil is atherogenic, especially when they are fed with diets that provide cholesterol (Fless, 1982).

There is little research done around caproic and caprylic fatty acids, which is why the atherogenic effect of coconut oil is in part due to the low content of linoleic and oleic acids compared with other oils such as palm oil that provides 10% and 40%, respectively.

As far as the palmitic fatty acid and considering the current commercial importance that palm growing has around the world, several studies have compared the effect of palm olein (fraction of palm oil that contains approximately 45% of palmitic acid, 42% of oleic acid and 10-12% of linoleic acid) with refined olive oil on the levels of plasmatic lipids. Both palm and olive oils provide the same amounts of linoleic and stearic acids and traces of lauric and myristic, which is why they allow for a reliable comparison of the oleic acid vs. palmitic acid effect. Two cross over studies conducted in men showed the difference in TC and LDL levels (Ng, 1992; Choudhury, 1995). On the other hand, the Danish study conducted by Tholstrup and col found out that the TC and LDL levels slightly increased in those subjects who consumed palm oil (0.15 mmol) but that there were no significant differences in the CT:HDL ratio (Tholstrup, 2011).
Similarly, there are studies comparing the effect of palm oil with other oils, as is the case of the study conducted by Khosla in 1992 on primates. This study demonstrated that palm oil, having a high fat acid content, did not show any differences regarding the effect of LDL when compared to safflower oil, having a high oleic acid content (Khosla, 1992).

Siri-Tarino, et al., performed a meta-analysis of prospective epidemiological studies that assessed the correlation between the intake of saturated fat with coronary disease (CD), cardiovascular disease (CVD) and cerebrovascular stroke (CS). It identified 21 studies, with the monitoring of 347,747 patients ages 5 to 23, 11,006 of whom developed the coronary or cerebrovascular disease. Estimates of combined relative risk that compared extreme quartiles of the saturated fat intake were: 1.07 (IC of 95%: 0.96; 1.19; P = 0.22) for CD, 0.81 (IC of 95%: 0.62; 1.05; P = 0.11) for (CVA), and 1.00 (IC of 95%: 0.89; 1.11; P = 0.95) para las CVD. The meta-analysis concluded that there is no significant evidence to reach this correlation.

Fretts, et al., examined the correlation of SFAs of plasmatic phospholipids, with total and cause-specific mortality in 3,941 seniors from the cardiovascular health study which is a prospective study based on the adult population older than 65 years, monitored since 1992 until 2011. The COX Proportional Hazard Model was used to approach this study and the total and cause-specific mortality ratio with the plasmatic levels of palmitic acid phospholipids (16:0), stearic acid (18:0), arachidic acid (20:0), behenic acid (22:0) and lignoceric acid (24:0). During this monitoring, 3,134 deaths occurred and the highest concentrations of stearic, behenic and lignoceric SFAs in plasmatic phospholipids were associated with a lower risk of mortality. In contrast, the highest concentrations of palmitic FA in plasmatic phospholipids, was associated with a higher risk of mortality. The study also found that there is no association between the concentration in plasma of arachidic FA with total mortality. Authors conclude that the association of SFA levels in plasmatic phospholipids differ according to the SFA chain and suggest that future studies should analyze the circulating SFA levels in a more detailed way (Fretts, 2016).

Pauaschitz, et al., studied the correlation between self-reported dietary intake of fatty acids and the risk of subsequent coronary accidents and mortality, in patients with coronary disease. This study included those patients who participated in “the Western Norway B-Vitamin Intervention” and filled out a semi-quantitative questionnaire containing 169 points about food frequency after undergoing a coronary angiography. Estimated quartiles of daily intakes of fatty acids and coronary events were analyzed (unstable angina pectoris, non-fatal heart attack and coronary death) and as secondary points: Acute myocardial infarction, fatal coronary accidents and deaths for all causes, using the COX Regression Model. This study included 2,412 patients (81% men with and average age of 61.7 years). After a median follow-up of 4.8 years, a total of 292 patients (12%) suffered at least one mayor event during this follow-up. High intakes of saturated fatty acids were associated with other risk factors such as high blood pressure, tobacco smoking, and diets with a high caloric intake, among others.) However, there were no significant associations between the SFA intake and the risk of coronary events or secondary factors. The authors concluded that there was no association between the SFA intake and the incidence of coronary events or mortality in patients with CVD (Puaaschitz, 2015).

Lin et al., have shown that SFAs (capric-stearic) enhance the activity of the protein family that bind to the transcription factor of the regulating element of sterols (SREBP) and as a result, it increases the transcription of lipogenic genes (FAS, SDC-1). Besides, they stimulate the activation of the liver X nuclear co-receptor/retinoic acid promoting VLDL secretion. At the same time, SFAs reduce non-proteasome intracellular degradation of the apolipoprotein B-100. By doing so, the rise of lipoproteins enriched with SFAs would enable said particle to escape and degrade through that way. This translates in the increase of total cholesterol and plasmatic triglycerides (Lin, 2005; Osterud, 2003).

The intake of diets that are rich in long chain SFAs (C14 - C18 carbons) raise the levels of postprandial lipids, while diets that are rich in short and medium chain SFAs (C2 - C14) do not produce significant changes in it (Sanders, 2003).
Souza, et al., carried out a systematic review of the correlation between saturated fat and industrialized or ruminant trans-fat intake. It included all death causes such as cardiovascular disease, coronary heart disease (CHD), ischemic cerebrovascular disease and type 2 diabetes, and reveals that the intake of saturated fats is not associated with mortality for all causes, nor with any of the abovementioned diseases, but the evidence is heterogeneous and with methodological constraints. With respect to trans fats, the intake originating from industrialized foods is associated with mortality for all causes. The authors also recommend considering proper alternatives in order to substitute saturated fats and trans fats, when guidelines prevail about the intake of macronutrients (Souza, 2015).

The differences of specific SFAs effects, particularly with lauric, myristic, palmitic and stearic acids, on circulating lipids and lipoproteins, are difficult to interpret. Having the data about final points of CVD, evidence is insufficient to favor a concrete SFA. Basically, almost none of the recommendations from international organizations make a distinction between different SFAs because its predominant sources are the same foods. (Ros, 2015).

Finally, it is important to bear in mind that when referring to fat acids and their effect on cardiovascular health, let us not forget that by consuming foods with a high fat source including SFA, other components are also being consumed which can mitigate or boost the effect that fat acids can have in the human body. This is the case when palm oil is being consumed, because even though nearly 50% of its fat acids are saturated, it is a source of tocotrienols (vitamin E component). Through diverse global studies in both animals and human beings, these proof to have a hypocholesterolemic, antithrombotic effect favoring the regression of atheromatous plaque (Theriault, 1999; Theriault 2000; Aggarwal, 2010; McNamara, 2010).

Current research has shown us that, even though the type of fat in the diet might affect the concentration of serum cholesterol, there is not always a link between the formation of atheromatous plaque and its high values. Reported studies during the past decade, seem to demonstrate that atherogenesis can start with the formation and phagocytosis of oxidized LDL from macrophages.

The atherogenic process is divided in several steps that include peroxidation of UFAs present in LDL and the phagocytosis of these LDLox from macrophages. The latter is a process that requires the endothelial expression of adhesins. (Sylvester y Theriault 2003). LDL’s consists of subclasses. The smallest and more permanent in the blood seem to be more prone to peroxidation, whereby it is possible to say that a high UFA diet, although reducing Cols levels; might increase the risk of forming plaque, therefore, developing CVD (Sylvester, 2003).

Similarly, the SFA effect on the vascular function has been reviewed in studies such as the 24 week RISCK trial in which no difference in arterial stiffness was found nor in the flow-mediated dilation (FMD) when SFAs in the diet were replaced by carbohydrates or MUFAs (Sanders, 2013). Likewise, the CRESSIDA study, which compared the current dietary guidelines in the United Kingdom with the traditional diet, demonstrated that there was no effect on FMD when there was an intake of SFAs (Reidlinger, 2013). It seems like the effect reported by Keogh and col in Australia, as far as the deleterious impact of SFAs on the endothelium, relates to the impact that fatty acids have on the plasmatic levels of triglycerides in the postprandial period. This effect seems to be higher in MUFAs than in SFAs. Therefore, current scientific evidence seems to support the hypothesis that SFAs do not have an adverse effect on FMD (Sanders, 2013).

Furthermore, thrombosis and inflammation are other factors to take into account when studying fatty acids and cardiovascular health. Concerning this topic, Kaptoge and col demonstrated that moderately elevated levels of C-reactive protein (CRP) and high fibrinogen concentrations are associated with inflammatory and coagulation processes and therefore they increase cardiovascular risk (Kaptoge, 2012). However, although the ratio between body fat percentage and high CRP and fibrogen levels has been described, the direct ratio between SFAs vs MUFA and the increase of these markers has not been described (Jebb, 2010). Initial studies suggested that stearic acid was thrombogenic, however,
a prospective cohort study found that the coagulation factor VII (FVIIc) activity is the one associated with a fatal risk of cardiovascular disease and that its levels are associated with fat intake. On the other hand, the study states that the SFA effect on lipemia and FVII depends upon the chain’s length and the physical properties of the oil (Sanders, 2000; Oakley, 1998; Sanders 2001). Medium and short chain SFAs do not have effect on postprandial lipemia nor FVII. Nevertheless, it is important to bear in mind that the effect of fats on FVII is transitory and reflects the recent fat intake, and that the difference between SFAs and MUFAs is only relevant in a 24-hour period after the consumption of a high fat diet. (Sanders, 2006). Moreover, it seems that SFAs do not to have an effect on the fibrinolysis indexes. The RISCK study could not demonstrate the long-term effect of SFAs intake on the FVII activity, however, it is important to consider that the subjects in this study knew they could not to eat foods high in fat on the previous day of the measurements (Jebb, 2010).

Lastly, it is important to consider the effect that SFAs have on insulin resistance since it has been seen that it is associated with the metabolic syndrome and, thus, with cardiovascular risk. The review performed by Willet and Hu in 2009, suggests that SFAs might have an adverse effect on insulin release and glucose homeostasis (Riserus, 2009). However, these conclusions were based only on a series of small trials and a single robust study: KANWU, which validated the hypothesis and showed that when substituting the intake of SFAs of animal fat origin and shortening for MUFA, then, insulin sensitivity improved but that said improvement was statistically at the border of significance (Vessby, 2001).

Furthermore, the RISCK and LIPGENE studies or the one carried out by Bos and Col in 2010 did not find evidence of the adverse effects on insulin sensitivity when having an SFA based diet. Consequently, we might say, that current evidence suggests there is no difference between the effect that SFAs and MUFAs have on insulin resistance in humans (Jebb, 2010; Tierney, 2005; Bos, 2010).

It is also worth mentioning the effect of dairy fat, containing more than 400 different fat acids (from 4 to 26 carbon atoms), among which more than half of the fat acids present in milk are SFA, which is why its consumption has been discouraged indiscriminately. However, the specific SFA effects on health have been studied broadly, highlighting the biological activities of short chain fatty acids, whose presence is dairy fat specific and represent 10-12% of the total SFAs. Among these, of particular relevance, is butyric acid (C4:0) since it acts as a gene modulator and it is well known for playing an important role in cancer prevention [Parodi, 2006]. Caprylic and capric acids (C8:0 y C10:0) promote antiviral activities and caproic acid (C6:0) has been described as an antitumor agent for inhibiting the growth of cancer cell lines [German, 1999]. Additionally, these fatty acids have been described as a quick energy source having the potential to reduce fat deposit and with antiviral and antibacterial functions, which are most responsible for reducing gastrointestinal disorders [Sun, 2002]. Another SFA present in dairy fat having a content higher than 10% and that does not have a negative effect on health either is stearic acid (C18:0). Its intake does not affect the increase of serum cholesterol; thus it cannot be considered atherogenic (Mensink, 2003; Alhazmi, 2012).

Trans fatty acids
A 2% energy increase from TFA significantly raises the risk of suffering from cardiovascular diseases up to 23% (Mozaffarian, 2006).

Bendsen, et al., published a systematic review and meta-analysis, which revealed that a TFA intake between 2.8 and 10 g/day could increase the risk of suffering from coronary events in 22% and fatal coronary events in 24%. Negative effects of TFAs on cardiovascular health are because they produce alterations of lipoprotein metabolism. Its increased intake relates to the rise in the concentration of total cholesterol and LDL cholesterol in the blood and with the reduction of HDL cholesterol. Moreover, they can promote the inflammatory status affecting markers such as C-reactive protein, among others (Bendsen, 2011).

A meta-analysis of 60 studies, published in 2003 by Mensink et al., indicates that monounsaturated TFAs increase the ratio between total cholesterol content and HDL cholesterol and its intake increases cardiovascular
risk more than the SFAs. The mechanisms that would explain these effects are unknown and the protein involvement that bond to cholesterol esters and certain liver receptors has been suggested (Mensink, 2003).

The Nurse’s Health Study carried out in the United States, followed a cohort of more than 121,000 women during 20 years since 1976. The results of this research established that there is a relationship between a high TFA intake and an increase in the risk of developing cardiovascular diseases. Oomen et al., obtained similar data in a 10-year cohort study conducted in 667 adults, finding out that a high TFA intake contributes to the risk of developing a coronary disease (Hu, 2005; Oomen, 2001).

Various clinical studies demonstrate that TFAs have an adverse effect on the lipid profile, similar to some saturated fatty acids. They raise the serum concentrations of triglycerides having low-density lipoproteins (LDL) and of lipoprotein (a) (Lp (a)). Furthermore, they lower serum concentration of high-density lipoproteins (HDL). These are in charge of reducing the unfavorable effects of consuming large amounts of fats while carrying them from the peripheral tissue towards the liver for its subsequent oxidation, which is why its decrease contributes to the development of the allergenic process (Lopez-Garcia, 2005; Matthan, 2004). Aro et al., describe this same effect when the intake of TFAs were high (8,7% of the energy) and that this effect was higher, compared with same amounts of stearic FA and dairy fat (Aro, 1997).

TFAs have revealed that they can also affect inflammatory markers including C-reactive protein (CRP), interleukin-6 (IL-6) and the tumor necrosis factor alpha (TNF-α). In turn, inflammatory factors can play an important role in the development of diabetes, arteriosclerosis, plaque rupture and cardiac arrest (Mozaffarian, 2006).

Overall, dietary fatty acids seem to influence the

<table>
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<tr>
<th>Action sites</th>
<th>Effects</th>
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<tbody>
<tr>
<td>Liver tissue</td>
<td>Increase c-LDL, c-VLDL and triglycerides.</td>
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<tr>
<td></td>
<td>Decrease HDL</td>
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<tr>
<td></td>
<td>Decrease size of LDL</td>
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<tr>
<td></td>
<td>Increase gluconeogenesis</td>
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<tr>
<td>Fat tissue</td>
<td>Decrease the uptake of triglycerides</td>
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<td></td>
<td>Decrease cholesterol esterification</td>
</tr>
<tr>
<td></td>
<td>Increase free fatty acids</td>
</tr>
<tr>
<td></td>
<td>Increase inflammatory response mediated by the activation of JNK</td>
</tr>
<tr>
<td>Muscular tissue</td>
<td>Increase insulin resistance</td>
</tr>
<tr>
<td></td>
<td>Decrease glucose uptake</td>
</tr>
<tr>
<td></td>
<td>Promotes triglyceride deposits</td>
</tr>
<tr>
<td>Endothelium</td>
<td>Decrease nitric oxide</td>
</tr>
<tr>
<td></td>
<td>Activate NADPH oxidase</td>
</tr>
<tr>
<td></td>
<td>Increase reactive oxygen species (ROS)</td>
</tr>
<tr>
<td></td>
<td>Increase ICAM-1, VCAM-1 and E-selectins</td>
</tr>
<tr>
<td>Monocytes and/or macrophages</td>
<td>Activate NF-KB</td>
</tr>
<tr>
<td></td>
<td>Increase FNT-a e IL-6</td>
</tr>
</tbody>
</table>

*Chart No. 2. TFAs effects in the function and properties of some tissues and cells.*

*Taken and adapted: Consumption of trans fatty acids and cardiovascular risk Fernando Manzur J., MD., FACC. Ciro Alvear S., QF., MSc. Alicia Alayón, Bact. MSc. Cartagena, Colombia. Rev Colomb Cardiol 2009; 16: 103-111*
physiopathology of diabetes mellitus, through its incorporation in the structure of lipids in the skeletal muscle and fatty tissue, which can produce alterations in the function of insulin receptors. The hypothesis of its action has been that TFAs reduce insulin sensitiveness through its effects in the increase of interleukin-6 (IL6) concentrations, the tumor necrosis factor alpha (TNF-α) and prostaglandins. The mechanisms by which trans fats act over systemic inflammation or the endothelial function are not well established. It seems that, by means of in vitro studies, TFAs are incorporated in endothelial membrane cells and in the membranes of monocytes/macrophages and adipocytes, where they could directly affect the signaling pathway related with inflammation. However, current available studies suggest that TFAs are pro-inflammatory and alter the endothelial function (Estadella, 2013).

There were differences found in the response to postprandial insulin between TFA and monounsaturated fatty acid based diets when comparing 3 diets with different types of fatty acids (trans, saturated fats and monounsaturated fats) in patients having overweight and diabetes mellitus type 2.; however, there were no significant differences between TFA based diets and saturated fatty acids (Castro-Martinez, 2010). Overall, fatty acids might directly or indirectly, modulate metabolic and inflammatory responses from the endoplasmic reticulum. By means of these types of effects, TFAs would affect the function and responses of many types of cells. For example they would alter the inflammatory responses of adipocytes, as the c-Jun N-terminal kinases (JNKs) activation, that regulates the expression of genes that mediate inflammation processes in response to cellular stress (Manzur, 2009).

As of this date, there have been epidemiological investigations as well as clinical studies in order to determine if the harmful effect of TFA coming from an industrial origin, can also be produced when consuming large amounts of animal origin TFAs. Two published works demonstrate that the intake of biological or natural origin TFAs have a minor impact on cardiovascular health than those having a technological origin, particularly in HDL cholesterol. TFAs of natural origin would not modify the levels of this lipoprotein and those of technological origin, would. In any case, the background information reveals that the TFA intake is harmful for human health, which is why all possible efforts should be made to reduce its consumption. (Chardigny, 2008; Motard-Belander, 2008).

Regarding dairy fat, TFA’s content varies mainly depending on the livestock feed (between 2-6% of the total fatty acids), being the trans isomer 11, C18:1 or vaccenic acid (VA), the most important quantitatively isomer representing 30 to 50% of the total FTAs. In this respect, there is a long series of studies that point towards the fact that the consumption of moderate amounts of FTA through the intake of dairy products, might not contribute to increase cardiovascular risks [Gebauer, 2011], and that VA particularly might have beneficial and protective effects with regard to atherosclerosis in animal models [Gayet-Boyer, 2011]. These possible contradictions may be because of the double role that VA plays in metabolism, since it is at the same time an FTA and a precursor for rumenic acid (RA) (isomer cis-9, trans-11-CLA). It is currently recognized that about 90% of RA of milk is produced endogenously involving the activity of Delta9-desaturase enzyme from VA. This conversion of VA into CLA has also been determined in rodents, swines and humans [Turpeinen y col. 2002]. Another recently described trans fatty acid, the Trans-Palmiteoleic, found in whole milk and its by-products, would be related with the risk reduction of diabetes and metabolic syndrome [Mozaffarian and col. 2010; Mozaffarian and col. 2013]. (FELAPE, 2017)

Replacement of saturated and trans fatty acids for other nutrients
The effects of reducing SFAs from the diet depend upon the type of nutrient by which they are replaced. Compared with carbohydrates (CHO), SFAs increase the concentrations of total cholesterol and LDL cholesterol, and moderately, those of HDL cholesterol. On the other hand, the substitution of SFA for monounsaturated fatty acids (MUFA) present in olive oil, avocado, dried fruits or polyunsaturated fatty acids (PUFA), present in canola oil, sunflower, soy, decrease total cholesterol and LDL and
slightly increase HDL cholesterol.

Well controlled metabolic studies can predict that substituting SFAs in a 5% proportion of the diet’s energy, for PUFAs, reduces LDL cholesterol in 0,3mmol/l (5-7 %), without a significant reduction of HDL cholesterol and therefore it reduces total cholesterol ratio: HDL cholesterol in 0,18 (Mensink, 2003).

In this sense, the expert committee of FAO/WHO has stated that there is strong evidence that substituting SFA (C12:0 - C16:0) for PUFA, decreases LDL cholesterol concentration and total cholesterol ratio: HDL cholesterol. A similar but smaller effect is obtained by substituting SFAs for MUFAs. The substitution of SFAs (C12:0 - C16:0) for CHO decreases cholesterol concentration, both LDL and HDL, but the total cholesterol ratio does not change: HDL cholesterol (FAO, 2010).

The nutrition panel of the European Food Safety Authority (EFSA) has also issued a favorable opinion about health or property claims of foods with a smaller SFA content. Particularly, it has revealed the existence of a cause-effect relationship between the intake of SFA diet blends and an increase in cholesterolemia and that the intake of foods with reduced SFA amounts might help to maintain normal LDL cholesterol concentrations in blood. Similarly, EFSA issued a favorable opinion about substituting a blend of TFA for cis MUFA and/or cis PUFA in foods or diets (in a gram per gram ratio) and the maintenance of normal LDL cholesterol concentrations (EFSA, 2011; EFSA 2011a).

Summary of scientific evidences about the intake of SFAs and TFAs and its relationship with NCD risk

Effects on the lipid profile: The intake of SFAs compared with CHO increases total cholesterol and LDL cholesterol significantly, and HDL cholesterol, moderately. Substituting SFAs in the diet for PUFAs or MUFAs decreases total cholesterol and LDL and, in a slightly manner, HDL cholesterol. Level 1++.
Recommendation: To substitute SFAs for PUFAs or MUFAs to improve the lipid profile. Grade A.

Effects on cardiovascular risk: The substitution of SFAs in the diet for PUFAs decrease CHD risks. Level 1++
Recommendation: Substitute SFA for PUFA to decrease CHD. Grade A.

Effects of specific SFAs on the lipid profile and CHD risk: The intake of stearic acid, in relation with other SFAs, it is associated with a discrete improvement of the lipid profile, even though there are no evidences, which demonstrate an improvement of cardiovascular risk and that support the specific recommendations for different types of SFAs. Level 3.
Recommendation: There is not enough evidence about cardiovascular risk indicators in order to support different recommendations about specific SFAs. Grade D.

Based on an intake of 2% of the energy, TFAs relate with diverse cardiovascular risk factors and they contribute to increase CHD risk. Level 1+.
Recommendation: Intake of TFAs must be as low as possible, not exceeding 1% of total energy. As a result, when establishing objectives and recommendations of nutrients, the intake of TFAs must be limited. Grade B.

Distinction between TFAs produced by industrially partial hydrogenation and naturally by rumen bacteria: Available evidence is insufficient to determine if there exists any difference between TFA from different sources (coming from ruminants or industrially produced) related to CD risk. Level 1.
Recommendations: The intake of TFAs coming from rumen expecting healthy effects cannot be advised due to lack of sufficient evidence. Grade D (Ros, 2015).

SATURATED FATS AND TRANS FATS, PUBLIC HEALTH AND REGULATION

At a global level and according to the World Health Organization WHO, the first mortality and morbidity causes are CVD, respiratory diseases, cancer and diabetes, which have claimed the lives of close to 388 million people. Furthermore, its effects on productivity and economy are startling and represent a high cost for countries and families. There is much evidence that eliminating TFAs from foods
is a strategy to protect health and prevent cardiovascular diseases. It is also a feasible procedure from the industrial standpoint. Currently, there are regulatory projects to suppress gradually said fatty acids and the Pan American Health Organization/World Health Organization PAHO/WHO lead this initiative, aimed at improving the diet and encouraging a healthier lifestyle in the Americas.

Generally speaking, the possible approaches to limit TFA levels in foods and among the population can be broken down in legislative measures, on one side, and voluntary measures on the other. Legislative measures may consist of establishing TFA limits in food products (in either the ingredients or the final product) or make TFA health claims mandatory in the nutritional information. By having a voluntary reformulation or when possible a voluntary mention of TFA content in the nutritional information, operators of food companies will be able to decide if they reformulate the products or not and if they inform consumers or not about TFAs (European Commission, 2015).

Through the Non Communicable Disease Unit, PAHO/WHO convened the creation of a task force called “Trans Fats Free America”. These are the conclusions and recommendations of said task force:

1. Industrially produced TFAs present in food supply must be eliminated in the Americas, and the preferred option must be unsaturated fats, including polyunsaturated fatty acids of the omega-3 family, given their cardiovascular protective effect. Saturated fats must be used only as TFA substitutes when they become unpredictable for specific applications and, given technological advances of foods, this must occur infrequently.

2. Even though industry’s voluntary measures are welcome, there is a need for regulatory measures to protect in a more quickly and effective way, the population’s health in the Region. Moreover, the regulatory framework helps to level the “playing field” for all the industry (local and international, small and large) besides, it also ensures the provision of the same benefits for all sectors of society (particularly for rural and poor populations.) Based on reliable data about costs, latest technologies and supply matters, the elimination of industrially produced TFAs is feasible and achievable, the speed of progress in fulfilling this goal over time must take into account the diverse local realities of each country.

3. The key policy measure recommended is to adopt, by means of legislative measures, a <2% limit of the total fat amount as TFAs in vegetable oils and soft margarines (spreadables) and of <5% for the remaining foods, as proposed by the Canadian workgroup about TFAs. Other possible regulatory measures are: a) nutritional labelling to inform TFA content in foods throughout the region; b) setting standards to regulate claims about healthy properties of foods and c) claims about types of fats and oils, especially the TFAs, present in foods that are served in restaurants, in food aid programs and school feeding programs; and other food service suppliers.

4. The Task Force is committed to work with industry leaders in order to identify confluence points for action, as well as to expedite the progressive reduction process of TFAs and promote the adoption of healthier oils and fats in foods throughout America. For this purpose, the Task Force will propose an action plan to the direct stakeholders, governments and the industry, which must be applied after consulting with these diverse sectors.

5. National governments are particularly encouraged to support the efforts of industries and small food services in their effort to eliminate TFAs and adopt healthier oils and fats.

6. The Task Force recommends PAHO/WHO to: a) lead the effort of progressive TFA reduction; b) help Member States to make policies and build public health capacity regarding nutrition, as well as lab and human resources to adequately measure progress and impact; c) make “Trans Fats Free America”, at a medium-term, a policy and strategy priority in your Region’s health program; d) provide technical assistance to governments so they prepare the
legislation and the required regulations to eliminate TFAs, and e) encourage Member States to indicate in the Joint Commission FAO/WHO of the Codex Alimentarius, the need to take into account relevant recommendations stated by this Task Force.

7. It is necessary to investigate several topics to improve some of the suggested measures, but this should not delay the adoption of measures to eliminate TFAs from industrial production. Among the research topics proposed it is worth mentioning: a) the definition of the optimal combination of polyunsaturated fatty acids n-3 y n-6 and polyunsaturated and monounsaturated fatty acids to replace TFAs, depending on the sources available of fats and oils; b) the improvement of the source characterization and amounts of TFA that different populations consume in the Americas and; c) the attainment of appropriate sampling methods and specific biological markers to carry out studies about TFA exposure and its biological effects.

The development of regulatory measures aim at protecting the consumer's health regarding the content of nutrients in industrially based foods. These measures inform and promote the selection of healthy foods by decreasing the intake of SFA and TFA rich foods, and in this way the risk of NCDs. Following there is a description of the content regulation of SFAs and TFAs in different countries. Chart No. 3.

ALTERNATIVES TO DECREASE THE CONTENT OF TFAs IN FOODS

During the past years, global nutritional recommendations suggest the reduction of TFA consumption, mainly of industrial origin. Currently, the food industry has worked to produce margarines and fat-free trans fats, categorized as virtually trans-free fats (VTF) products and are presently commercialized in several countries in Latin America (PAHO, 2008).

To reduce TFAs in hydrogenated products, the methodology that is currently being developed, is based on the use of oils with a high degree of saturation (palm oil or palm kernel) or of vegetable oils (soy, sunflower) previously hydrogenated, until reaching a high degree of saturation, being the TFA level minimum or non-existent in both cases. These oils are blended with refined vegetable oils relatively unsaturated (sunflower, soy, corn) which also have minimum TFAs in their origin (List, 2004).

One of the most common modern techniques to reduce TFA content in fats and oils, used in food manufacturing, is the interesterification process which consists of rearranging fatty acids in the triglycerides that form both types of oils (highly hydrogenated and non-hydrogenated). This process can be done chemically or enzymatically by using a blend of different oils. The chemical process produces a blend of variable triglycerides having a variable composition since it consists of randomly exchanging fat acids. The enzymatic process is more expensive, but it enables you to obtain products with a composition and specific properties according to the producer's requirements. (Jutteland, 2004).

There are also oils obtained from seeds that are produced through plant breeding techniques. Another option used by some producers is the use of genetically modified plants to obtain oils with low linoleic acid content or a moderate or high oleic acid content. These oils have enhanced features or characteristics, which contain non-significant proportions of TFAs and high proportions of cis monounsaturated, when compared with partially hydrogenated oils besides they have great thermal stability. These oils can be reused in products such as margarines, in frying processes or in industrial or home baking. (Eckel, 2007; Tarrago-Trani, 2006; PAHO, 2008)

As an example of these types of oils, we have: Soy oil with a high concentration of oleic acid and a low concentration of linolenic acid, soy oil with a low content of linolenic acid, soy oil with a minimum content of linolenic acid. There is also sunflower oil with a higher content of oleic acid, sunflower oil with a high content of oleic acid, fractions of palm oil and palm kernel with a low content of saturated fatty acids, etc. The fats industry has incorporated new procedures and a wide variety of products to develop fats with low TFA content as well as to produce foods with a minimum TFA content (PAHO, 2008).
### Saturated Fatty Acids

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Legislation</th>
<th>Reference values for labelling and nutritional property claims</th>
</tr>
</thead>
<tbody>
<tr>
<td>European Union</td>
<td>2006</td>
<td>Regulation (EC) No 924/2006 of the European Parliament and of the Council of December 20, 2006 concerning nutrition and health claims made on foods.</td>
<td>Without saturated fats: A food can only be claimed as saturated free if the sum of its saturated fats and trans fatty acids is no greater than 0.1g per 100g for solids or 100ml for liquids. Low in saturated fats: A food can be claimed as having a low content of saturated fats if the sum of saturated fats and trans fatty acids is no greater than 1.5g per 100g for solids or 0.75g per 100ml for liquids.</td>
</tr>
<tr>
<td>United Kingdom</td>
<td>2013</td>
<td>Guide to creating a front of pack (FoP) nutrition label for prepakced products sold through retail outlets.</td>
<td>The front of pack nutrition label must consider the reference values so as to &quot;label&quot; a product as having low, medium and high SFA.</td>
</tr>
<tr>
<td>USA</td>
<td>2013</td>
<td>Code of Federal Regulations Title 21 [CFR 101.9(c)] Guidance for industry; a food labeling guide on specific requirements for nutrient content claims.</td>
<td>Saturated fat free: Foods containing less than 0.5g of saturated fat and less than 0.5g of TFAs per amount of consumed reference and per labeled portion. Low in saturated fat: Foods that contain 25% less SFAs of the reference food.</td>
</tr>
<tr>
<td>Colombia</td>
<td>2011</td>
<td>Resolution 333 of 2011 Technical resolution about nutritional marking and labeling requirements that canned foods must comply with for human consumption.</td>
<td>Saturated fat free: Contains less than 0.5g of saturated fat. Low in saturated fat: Contains a maximum of 1g saturated fat and the calories coming from SFAs must not exceed 15% of total calories.</td>
</tr>
</tbody>
</table>

### Trans Fatty Acids

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>Legislation</th>
<th>Reference values for labelling and nutritional property claims</th>
</tr>
</thead>
<tbody>
<tr>
<td>Denmark</td>
<td>2003</td>
<td>Executive Order No. 160 of March 11, 2003 on the Content of Trans Fatty Acids in Oils and Fats.</td>
<td>It prohibits the sale of oils and fats that have a higher TFA content than what it is defined in Article 3.</td>
</tr>
<tr>
<td>United States</td>
<td>2003</td>
<td>Food and Drug Administration Guidance for Industry; Trans Fatty Acids in Nutrition Labeling, Nutrient Content Claims, Health Claims; Small Entity Compliance Guide.</td>
<td>TFAs must be listed as “trans fats” or “Trans” in a separate line of saturated fats, in the nutrition labeling. The content of trans fats must be expressed in grams per portion.</td>
</tr>
<tr>
<td>Canada</td>
<td>2005</td>
<td>Health Canada’s &quot;Fact Sheet on Trans Fats&quot; Food and Drug Regulations (FDR).</td>
<td>It is mandatory to claim the product’s trans fats’ content in the nutritional labelling. Claims such as “free of trans fatty acids”, or “reduction of trans fatty acids” or “low in trans fatty acids” can be expressed on the label with respect to TFA content.</td>
</tr>
<tr>
<td>United States - New York</td>
<td>2006</td>
<td>Section 81.08 of the New York City Health Code. Prohibits serving foods that have more than 0.5g TFAs per portion, in all of the city’s restaurants. The elimination of trans fats will be carried out in two stages in all of the food service establishments.</td>
<td></td>
</tr>
<tr>
<td>Argentina, Brazil, Paraguay and Uruguay</td>
<td>2006</td>
<td>MERCOSUR/GMC/RES. Nº 46/03 Technical regulation of Mercosur about nutritional marking of canned foods.</td>
<td>It will be mandatory to claim the content of trans fats.</td>
</tr>
<tr>
<td>United States - Boston</td>
<td>2008 - 2009</td>
<td>Guidelines for the implementation and compliance of artificial trans fats regulation of the Boston Public Health Commission.</td>
<td>No food service establishment must store, distribute, prepare, keep for service or serve food or beverages that contain artificial trans fats.</td>
</tr>
</tbody>
</table>
| Austria               | 2009  | Austrian Ministerial Decree No. 267 of August 20, 2009 regarding trans-fat content in food. | Prohibits the sale of products that contain more than 2g of trans fat per 100g of total fat; this prohibition is not applicable if: 
- The total fat content of the food product is less than 20% (4g for each 100g of total fat) 
- The total fat content of the food product is less than 3% (19g for each 100g of total fat) |
| Colombia              | 2011  | Resolution 2508 of 2012 Technical regulation about the requirements that canned foods, containing trans fats and/or saturated fats, must comply with for human consumption. | TFA content in fats, vegetable oils and spreadable margarines that are sold directly to the consumer will not exceed 2g per 100g of fat. The content of TFAs in fats and oils used as raw materials in the food industry, or as inputs in bakeries, restaurants or food services (catering), can contain up to 5g of trans fatty acids per 100 g of fat. |
| Denmark               | 2012  | Institute of Food and Resource Economics, University of Copenhagen, Denmark. | Since October 1, 2012 there is a tax for foods that contain more than 2.3g of saturated fats per 100g of total fat. |
| Norway                | 2012 - 2013 | Draft Regulations related to trans fatty acids in foods 2013/9013/N - C50A. | Prohibits the sale of products that contain more than 2g of TFA per 100g of fat to the final consumer. |
| Hungary               | 2014  | Decree 71/2013 The Ministry of Human Resources on the highest permitted amount of trans fats in food products, the conditions of, and inspections by, the authorities on the distribution of food products containing trans fats and the rules for tracking the population’s consumption of trans fats No. 2013/0371/HU - C50A. | The decree includes the maximum content of TFA in food products that are available for final consumers. General provisions establish a 2g limit of trans fats per 100g of total fat. |

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**Chart No. 3 Content regulation of SFAs and TFAs in different countries.**

**Saturated Fatty Acids**

**Trans Fatty Acids**

BENEFITS OF REPLACING TRANS FATS

The Commission’s report to the European Parliament and the Council regarding trans fats in foods and in the diet in general of the European Union population (2015), shows that modelling studies have calculated the effects in the reduction of TFAs from the diet in the morbidity-mortality of coronary heart disease (CHD), in spite of adopted measures. A study in the United Kingdom estimated that reductions of 0.5 and 0.8 % of daily energy in TFA intake might lower approximately in 3.500 and 4.700 the number of deaths in this country related to coronary heart diseases (O’Flaherty, 2012). In the United States, according to a cost estimate and possible sanitary effects, a reduction of 0.64 % of the daily energy intake of TFAs, would avoid each year, according to two alternative scenarios, a mean of 15.000 and 58.000 episodes of coronary heart disease. This represents around 1.2 % and 4.5 % of the overall CHD episodes in the United States, and 5.000 and 15.000 deaths related with CHD, which approximately represent 1.5 % and 4.4% of all the deaths related with CHD in the United States.

Taking into account the effects on the total cholesterol level, HDL cholesterol and numbers based on prospective studies, an energy reduction of 2% in trans-fat intake (~4.5 g/d for a person who consumes 2.000 kcal/day) would approximately avoid between 30.000 to 130.000 cases of ischemic heart disease episodes per year in South America. Similarly, an energy reduction of 4% would avoid approximately a double number of cases, in other words, between 60.000 and 260.000 episodes of ischemic heart disease per year (Note: Potential beneficial effects are not included for the case of other ischemic heart-angina episodes, coronary revascularization, sudden death having a cardiac or diabetes origin). (Work Commission of Argentina, 2011).

Certainly, absolute numbers of avoided cases are higher in countries or regions having larger populations (for example, Brazil), but a significant number of cases could also be avoided in countries and regions having smaller populations, due in many cases, to high rates of ischemic heart disease in the population (for example, Central America) (PAHO, 2008).

These estimates are based upon the substitution of trans fats for polyunsaturated or cis monounsaturated fats. The exact and balanced amounts of different cis unsaturated fats will depend on the replacement oils used, as mentioned before. Some trans fats can also be replaced by saturated fats, for example, those which contain tropical oils (palm oil, palm kernel, coconut) and animal fats. Work Commission in Argentina, 2011.)

In addition, the Commission to the European Parliament (2015) in the abovementioned document recommends that the final effect in terms of TFA intake (and sanitary results) depend on a series of underlying factors, among which we find:

• Level of the population’s nutritional knowledge;
• Diet habits of different groups of the population at a global level (different traditions, different sensitiveness towards price differences, etc.);
• Level of TFA intake of ruminants (dairy products and other ruminant by products that are part of a balanced diet);
• How can foods be reformulated to reduce industrial TFA content? The complete profile of the reformulated product must be considered to ensure, after reformulation, the provision of healthier diet options. For example, there is a concern about the fact that reformulation to reduce TFAs, can lead to an increase of saturated fatty acid content. Even though it is better, from the public health standpoint, substituting TFAs for cis unsaturated fats (which leads to a risk reduction of coronary heart disease between 21% and 24% when 2% of the daily energy does not come from TFAs but from unsaturated and polyunsaturated fatty acids). Moreover, the most unfavorable substitution with saturated fatty acids continues providing significant health benefits (with a reduction of up to 17% of coronary heart disease risk; risk reductions are estimates) (Mozaffarian, 2009).

Some result supervision studies of EU countries indicate that, although there have been substitutions of TFAs for saturated fatty acids in some products, in most cases the important differences have not been seen regarding the content of saturated fatty acids. In addition, the sum of TFA contents and saturated fatty acids has been reduced in most cases, and reformulated products have increased the content of cis unsaturated fats and they have a healthier profile overall. (Correspondence, 2010)
**RECOMMENDED ALTERNATIVES IN FOOD APPLICATIONS TO REPLACE TRANS FATS**

Based on the initiative several replacement alternatives are proposed, in applications were trans fats are normally used, for oils having a high content of MUFAS and PUFAS. Said alternatives are described as follows in Chart 4.

**Chart No. 4. Different alternatives recommended to replace trans fats**

<table>
<thead>
<tr>
<th>Type of application</th>
<th>Suggested Alternative</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>FRYING FATS: FOOD AND INDUSTRIAL SERVICES</td>
<td>Vegetable oils having medium and high oxidative stability</td>
<td>Oils with a high oleic acid content and low linolenic acid content such as high oleic sunflower, high oleic safflower, olive.</td>
</tr>
<tr>
<td>SPREADABLE MARGARINES: HOME USE AND FOOD SERVICE</td>
<td>Margarines made of interesterified vegetable oils, fractionated vegetable oils and vegetable oils</td>
<td>Elaborated from oils with a high content of unsaturated fatty acids (omega 6, 3 and/or 9) such as soy, sunflower, cotton and totally hydrogenated vegetable fats (in other words, trans fat free) and/or solid fractions of vegetable oils. Resulting fats will have a high mono and polyunsaturated content, and a moderate saturated content.</td>
</tr>
<tr>
<td>CONFECTIONERY MARGARINES: HOME USE, FOOD AND INDUSTRIAL SERVICES</td>
<td>Margarines made of interesterified vegetable oils, fractionated vegetable oils and vegetable oils</td>
<td>Elaborated from oils with a high unsaturated fatty acids content (omega 6, 3 and/or 9) such as soy, sunflower, cotton and totally hydrogenated vegetable fats (in other words, trans fat free) and/or solid fractions of vegetable oils. Resulting fats will have a moderate mono and polyunsaturated content, and a moderate saturated content.</td>
</tr>
<tr>
<td>MARGARINES FOR BAKED PRODUCTS AND PUFF PASTRY: INDUSTRIAL USE</td>
<td>Margarines made of interesterified vegetable oils, fractionated vegetable oils, fractionated animal fats and vegetable oils</td>
<td>Elaborated from oils with a high unsaturated fatty acids content (omega 6, 3 and/or 9) such as soy, sunflower, cotton and totally hydrogenated vegetable fats (in other words, trans fat free) and/or solid fractions of vegetable oils. Resulting fats will have a moderate mono and polyunsaturated content, and a moderate saturated content.</td>
</tr>
<tr>
<td>MARGARINES FOR DOUGH AND SHORTENINGS: INDUSTRIAL USE</td>
<td>Margarines made of interesterified vegetable oils, fractionated vegetable oils, fractionated animal fats and vegetable oils</td>
<td>Elaborated from oils with a high unsaturated fatty acids content (omega 6, 3 and/or 9) such as soy, sunflower, cotton and totally hydrogenated vegetable fats (in other words, trans fat free) and/or solid fractions of vegetable oils. Resulting fats will have a moderate mono and polyunsaturated content, and a moderate saturated content.</td>
</tr>
<tr>
<td>INDUSTRIAL USE FATS (SHORTENINGS FOR DOUGHS, CREAMS, FILLINGS, OTHERS)</td>
<td>Interesterified vegetable oils, fractionated vegetable oils, fractionated animal fats and vegetable oils</td>
<td>Elaborated from oils with a high unsaturated fatty acids content (omega 6, 3 and/or 9) such as soy, sunflower, cotton and totally hydrogenated vegetable fats (in other words, trans fat free) and/or solid fractions of vegetable oils. Resulting fats will have a moderate mono and polyunsaturated content, and a moderate saturated content.</td>
</tr>
<tr>
<td>FATS FOR CONFECTIONERY GLAZE</td>
<td>Vegetable fats, fractionated vegetable fats and/or interesterified and/or totally hydrogenated</td>
<td>Elaborated from oils with a high content of saturated fatty acids such as palm kernel, palm and coconut.</td>
</tr>
</tbody>
</table>

CONCLUSIONS

When we refer to fatty acids in general and their health effects, it is important to bear in mind that food provide blends of fatty acids in different proportions and there are no exclusive sources of a fatty acid type. Furthermore, besides fatty acids other components ordinarily consumed can mitigate, maintain or boost the effect that a certain acid might have on the human body.

In the case of SFAs and considering all the reviews and the new knowledge about their effect on health, the conclusion is that they should not be taken into account as a single nutrient or in a “block”, in the diet recommendations for the population. In this regard, although WHO/FAO Expert Committee recommends not exceeding the daily energy that comes from saturated fatty acids in 10% (FAO/WHO Expert Consultation, 2008). It might be possible to provide recommendations of the consumption of each of the SFAs in the future, since scientific evidence indicates that they have differentiated effects on health. Experts suggest to consume up to 10% of TCV that come from SFAs, include MUFAs and guarantee, not only a proper ratio of omega 3 and 6, but a sufficient intake of the first, because in this way a reduction of LDL plasmatic levels and the total cholesterol/HDL would be achieved, thus decreasing CVD risk.

On the other hand, there are sufficient review and intervention studies that support the recommendation of avoiding the consumption of TFAs derived from partial oil hydrogenation, in order to prevent non-communicable diseases, especially CVD. Several health committees and organizations propose the decrease of TFA intake to at least 1% of the total energy intake to achieve a real risk reduction of these diseases.

The methodology current developed to reduce TFAs in partially hydrogenated foods, is based upon the use of highly saturated oils (palm oil or palm kernel) or vegetable oils (soy, sunflower), previously hydrogenated, until reaching a high saturation degree, being the minimum or non-existent TFA level in both cases. These oils are blended with refined and relatively unsaturated vegetable oils (sunflower, soy, corn) which also contain minimum TFA amounts in their origin (List, 2004).

Even though this document provides scientific evidence related with the effect of different types of fatty acids on health, it is key to consider this effect within the context of the different nutrients present in the diet and that NCDs have a multifactorial nature.

Lastly, it is necessary to create public policies that promote a sufficient consumption of saturated fatty acids and the elimination of TFAs from foods, and that additionally generate positive environments for the population. Based upon the use and generation of local data regarding the composition and consumption of fats and oils, it is necessary to generate technologies, policies and information, education and communication strategies that approach different links of the chain, from producers and handlers up to consumers. This will guarantee that TCV coming from SFAs will not exceed 10% and that TFAs are eliminated from processed foods.

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