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# ARTIFICIAL TRANS FATTY ACIDS IN FOOD SUPPLY: A GLOBAL HEALTH THREAT

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**Abstract:** *Purpose:* Evidence of adverse health effects of artificial *trans* fatty acids (TFAs) have accumulated since 1990s, yet TFAs are widely used by several food manufacturers around the world. This review aimed to: ascertain the available evidence of the known unfavourable biochemical properties of artificial TFAs, their metabolic functions and health consequences; estimate their average intake levels and trends in different countries in order to critically evaluate whether more action is required to eliminate them from the diet.

*Methodology:* The published evidence was searched by employing: *Medline, Pubmed, InterScience, BioMed Central and Annual Reviews.*

*Findings:* With reference to human health, evidences from epidemiological, retrospective and observational studies revealed that the consumption of TFAs could outweigh the health risks posed by saturated fat consumption. The main health concerns included unfavourably altered blood cholesterol concentrations, insulin resistance, foetal brain and neural disturbances, proinflammatory and carcinogenic responses. Great variation exists in the global trends of industrial TFAs intake, being low in Mediterranean region, Japan and Scandinavia and high in parts of United States of America and Iceland. Besides the intense use of TFAs by food manufacturers and in eateries, the use of TFAs in food products is often poorly regulated and ill-informed to consumers.

*Value:* Since competitive alternatives to TFAs have made them non-mandatory a broad public health intervention at government level to regulate or completely eliminate them from the national diet is warranted.

**Keywords:** *Artificial Trans Fatty Acids; Coronary Heart Disease; Trans Fatty Acid Intake Levels; Regulation and Labelling*

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Dietary fats and oils are the cornerstone of a human diet, providing essential unsaturated fatty acids, fat-soluble vitamins, and energy (Bal *et al.*, 2003). Dietary fats consist from saturated, mono- and polyunsaturated fatty acids, which are involved in anabolism of various tissues and biologically active substances (Bal *et al.*, 2003). Despite the research and wide knowledge about dietary fats, in nutrition science, the role of dietary lipids is one of the most complexes, controversial and debated areas (Nishida and Uauy, 2009). The very first expert consultation was already held in Rome in year 1977, where the Food and Agriculture Organization (FAO) and World Health Organization (WHO) gathered together under a topic: *Role of Dietary Fats and Oils in Human Nutrition*. Since then, this event has taken place in year 1993 and 2002. The first meeting was held to determine nutritional values and to discuss the current evidence of the various physiological effects of different type of fats, whereas the later events updated the recommended intake levels and predicted their health consequences (Nishida and Uauy, 2009). The focus in last decade has been dedicated mainly in preventing and controlling the morbidity and premature mortality caused by non-communicable diseases. Thus the importance in nutrition research has been to gain better understanding of dietary fats and their biological properties (Nishida and Uauy, 2009).

*Trans* fat regulations and bans are one of the current heated topics among nutrition science in western countries, whereas also in rapid rate modernising developing countries (American Heart Association, 2008). The purpose of this review was to critically evaluate and summarise whether the current evidence of the health risks of TFA consumption is well evidence based. Additionally, this review aimed to gather the current data of the industrial use of TFAs, whereas also of

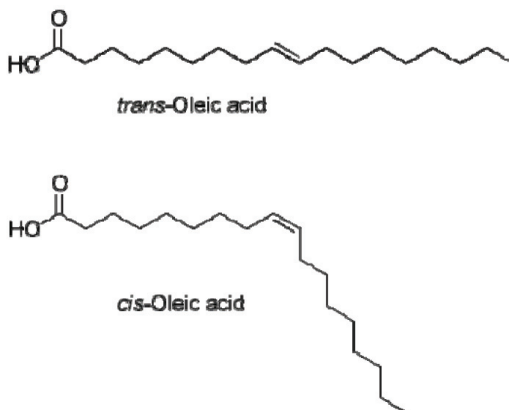
the intake of TFAs around the globe. This was also made in light of studying the governmental regulation and legislation approaches towards the use of TFAs. The aim was to come with well-reasoned conclusion whether more action from the behalf of the nutrition science, government, media and food industry is required to limit the existence of TFAs in food supply, by giving suggestions for improvements.

### **HISTORICAL BACKGROUND**

While the scientific research investigating the health consequences of different dietary fats verges close to understanding the currently used fats in food supply, the food manufacturing technology has been keen to make new fat innovations for better business. Before the 19<sup>th</sup> century, the food supply only contained naturally occurring oils and fats, such as beef tallow, butter, lard and various vegetable oils, including *trans* fats (TFAs) from ruminant (Enig, 2008; Hunter and Applewhite, 1991; Sommerfield, 1983) and from certain plants sources (Valenzuela and Morgado, 1999). However, the constant pressure to improve products and sales has forced the food industry to develop fats that are simultaneously cheap to produce and use, has an excellent taste and texture, but also which have a long shelf-life. This is how *trans* fatty acids evolved and found their way to food supply. In ruminants, bacteria living in the stomach make small amounts of natural *trans* fats (Heckers and Melcher, 1978), whereas the hydrogenation process yielding man-made *trans* fats (TFAs) was first discovered back in 1890s by a French chemist. In the beginning of the 19<sup>th</sup> century another scientist, Wilhelm Normann found that liquid oils can be hydrogenated to form TFAs. Normann patented the hydrogenation process and introduced TFAs (also known as partially hydrogenated oils) as the first man-made fats to join our diets (American Heart Association, 2008).

The chemical structure of unsaturated (marine or vegetable) oil normally has *cis*-double bonds (Fig. 1.), which inhibit crystallization (Groff *et al.*, 2000; Katan, 1998; Nishida and Uauy, 2009). Within the partial hydrogenation process (catalytic hydrogenation process, the oil is exposed to heat with the presence of metal catalysts (e.g. nickel) and hydrogen gas turning the oil into a semi-solid fat (Nishida and Uauy, 2009). Within this process the double bonds of the oil are either converted to saturated bonds, to *trans*-configuration (Fig. 1.) or are shifted to a new position in the aliphatic chain (Ascherio and Willett, 1997). Codex Committee on Nutrition and Foods for Special Dietary Uses in 2004 agreed to use the chemical structure and announced that *trans* fatty acid would be defined to be all the mono- and polyunsaturated fatty acids having non-conjugated interruption at least in one methylene group carbon-carbon double bonds in the *trans* configurations, including all the geometrical isomers of these fatty acids (Moss, 2006; Nishida and Uauy, 2009).

Partial hydrogenation process and *trans* fats seemed to be an



**Figure 1:**  
Chemical structure of  
unsaturated oil  
(Oleic acid) with  
*cis*-double bond and  
*trans*-configuration.

ideal invention for the food industry (Sgoutas and Kummerow, 1970). TFAs resist spoiling, can withstand repeated heating without breaking down (Food Standards Agency, 2007), are semi-solid in room temperature and provide desired consistency. In addition, TFAs are cheap which allows their wide use among the food industry and easier transportation of the products (eHow, 2009; Sgoutas and Kummerow, 1970).

While TFAs were making their revolution among the food industry, scientists were toiling to investigate their possible health effects (Hayakawa *et al.*, 2000). During the 1990s multiple research studies indicated negative metabolic effects of TFAs, whereas of their relation with coronary heart - and other diseases (Nishida and Uauy, 2009). These findings were gathered, analysed and presented by one major comprehensive study published in year 2006 in the *New England Journal of Medicine*. This triggered several campaigns against the use of *trans* fat. Around this time the debates initiated between food manufacturers, nutrition scientists and government whether to label *trans* fat content on food packages (American Heart Association, 2008).

Despite, the evidence from the health risks of having TFAs in food supply, it has been a great challenge to transfer the knowledge from the scientific world to general public.

### **INDUSTRIAL TRANS FATTY ACIDS IN THE DIET**

*Trans* fats, natural and artificial, have been and still are present in a wide range of food products around the globe (Ascherio *et al.*, 1994; Fritsche and Steinhart, 2006; Wagner *et al.*, 2000). It has been estimated that 40% of products in the typical supermarket in USA contains *trans* fat, and United Kingdom (UK) among other European countries is not far behind (Heckers and Melcher, 1978; Tsanev *et al.*, 1998). *Trans*

fat oils are commonly found in margarine (Demmelmair *et al.*, 1996; Fritsche and Steinhart, 2006; Heckers and Melcher, 1978; Tsanev *et al.*, 1998; Wagner *et al.*, 2000) with TFA content ranging from 10% to 30%, however even values as high than 60% have been found (Ascherio *et al.*, 1994). Other typical products include processed foods such as cookies, crackers, biscuits, confectionery products, cakes, wide range of cereal bars, chips, ice-cream, pizza (Hunter and Applewhite, 1991; Innis *et al.*, 1999; Wagner *et al.*, 2000), pastries, french fries and even bread (Ascherio *et al.*, 1994; Enig *et al.*, 1983). The FDA once estimated when the *trans* fat boom was in its peak, that approximately 95% of prepared cookies, 100% crackers and 80% of frozen breakfast products contained *trans* fat (Harvard School of Public Health, 2009). They are also used in many restaurants as cooking oils for frying, called “liquid shortening” (Ban Trans Fats, 2007). One of the biggest concerns however is the TFA content in food products consumed markedly by pregnant mothers (Aitchison *et al.*, 1977), infants (infant formulas) (Chardigny *et al.*, 1996; Koletzko and Bremer, 2008) and children (Demmelmair *et al.*, 1996).

Beside commercial foods, it is also worth noting that ordinary vegetable oils (e.g. Canola oil) convert to *trans* fat at high temperature over time, especially when re-using the same oil over a time (Ban Trans Fats, 2007). Additionally, some studies have suggested that microwave heating has an effect on fat oxidation and fatty acid isomer formation. This could significantly increase the TFA content in food supply, since microwave ovens are widely used in western households (Herzallah *et al.*, 2005). However, more research is required how this electromagnetic field affects in formation of *trans* fatty acids (Herzallah *et al.*, 2005). Another interesting area of a research is food irradiation and how TFAs naturally present in food products will react when exposed to gamma rays. Food irradiation has long been an issue for scientist to assure its safety and approval among

general public. This being said, TFAs has shown a good resistance to the oxidising process (Brito *et al.*, 2002).

To investigate the trends in TFA consumption, dietary recalls have been used as a common method (Ballison *et al.*, 1999; Harnack *et al.*, 2003). These are all based on rough estimations with relatively high risk of human errors. Hence, despite numerous estimations, it has been a difficult task to collect and obtain accurate data of the intake of TFAs (Enig *et al.*, 1990; Innis *et al.*, 1999). Challenges in data collection has been in lack of adequate information of the TFA content in food products, the possible conversion of chemical configuration during food procession (Clandinin *et al.*, 2001; Craig-Schmidt, 2006), whereas also due to a lack of official universal food databases and -tables (Hayakawa *et al.*, 2000; Skeaff and Gowens, 2006; Weggemans *et al.*, 2004). Thus, the data on consumption of TFAs around the globe is sparse. Alternative and more accurate methods have been invented to detect and quantify the *trans* fatty acid content in food products (Chardigny *et al.*, 1996; Enig *et al.*, 1983; Priego-Capote *et al.*, 2004). This being said work has also been conducted to find suitable biomarkers to analyse the TFA intake directly from the human body (Baylin *et al.*, 2002; London *et al.*, 1991). To give examples of the methods to detect and quantify the *trans* fatty acid content in food products, one is to examine the fatty acid profiles by using methyl and isopropyl ester derivates and applying high-performance liquid chromatography, and silver nitrate thin-layer chromatography (Chardigny *et al.*, 1996), whereas one other method is to use a combination of silver ion thin-layer chromatography (Ag-TLC) and compare it with gas chromatography, specifically gas-liquid chromatography (GC) (Precht and Molkentin, 1995). One markedly applied TFA content analysing method has also been gas liquid chromatography on a 15-metre capillary column coated with SP-2340 (Enig *et al.*, 1983), whereas one recent innovation has

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been microwave-assisted *Soxhlet extraction* (FMASE)-method. This is a method where modified *American Oil Chemists' Society Practice Cd 14d-96* is used for determination of *trans* fatty acid content of an food product (Priego-Capote *et al.*, 2004). All of the above methods give a detailed fatty acid composition, but the latter has been proposed as a standard TFA detection method (Priego-Capote *et al.*, 2004). In turn, to give examples of the methods to analyse the TFA intake from the human body, adipose-tissue sampling (Baylin *et al.*, 2002) and blood samples has been used (Baylin *et al.*, 2002; Mensink and Hornstra, 1995). Assessing the fatty acid content by analysing adipose tissue biopsy has become the predominant method (Baylin *et al.*, 2002; London *et al.*, 1991). The justification of its excellence has been its accuracy and simplicity. The method does not rely on human memory and yields objective information of the TFA intake (Baylin *et al.*, 2002). The half-life of the fat is relatively long (slow turnover, Baylin *et al.*, 2002), which is an average of two years, giving long-term information of the dietary intake of various fatty acids (London *et al.*, 1991). The criticism of the adipose tissue biopsies is the variations in TFA concentrations between individuals due to differences in genetic, body compositional factors and metabolism, whereas also habitual factors, such as the level of physical activity and smoking (Baylin *et al.*, 2002). Thus, it has been proposed that for the best accuracy, adipose tissue biopsy analysing method should be accompanied with other frequently used methods, such as validated food-frequency questionnaires (Baylin *et al.*, 2002). In addition, the combination of the method is most usable in epidemiological and group or population studies (Baylin *et al.*, 2002; London *et al.*, 1991). These introduced inventions have significantly reduced human errors and potentially could provide uniform nationwide food databases in future, giving detailed information of the TFA content in various industrial food products. Additionally, uniform detecting methods to analyse the intake of TFAs could also be practiced.

Furthermore, these inventions could promote the investigations to follow the trends in intake of TFA around the world.

In lack of previously introduced improvements in uniformity in investigation standards, the current estimations are unfortunately mainly based on food intake questionnaires, whereas also spread and inaccurate data available of the TFA content in various food products available. However, referring to these estimations the intake of industrial *trans* fatty acids in Western Europe was reported to be mainly 16.5% from bakery products and 35.5% from oils and fats, whereas the naturally occurring *trans* fatty acids from milk and cheese accounted 18.8%. In Japan, unlike in Western countries, the main intake of *trans* fatty acids is from household margarines and oils instead of industrial products (Hayakawa *et al.*, 2000). In 1980s the daily intake of TFAs ranged from 2g to 15g in Western Europe and USA (Katan, 1998), whereas another estimation was that the average daily intake (per capita) of TFAs in industrialized countries was from 1g to 2g in Korea and Japan to about 5g to 8g in the USA and alarming 13g in the UK (Hayakawa *et al.*, 2000). Similar figures in industrialized countries were also found earlier by a study conducted in 1995 by *International Life Sciences Institute* and also by Allison *et al* in year 1999. However, the most comprehensive study with the total intake of TFAs data was revealed by Hulshof *et al* in year 1999 in *TransFair* study (Table 1.) (Fernández-San, 2009; Hulshof *et al.*, 1999). This study collected the information by using dietary surveys, focusing on the quantity and consumption frequency of *trans* fats, whereas also analysed various food samples (Hayakawa *et al.*, 2000). The data was gathered from 14 European countries using the post 1995 data on food composition (Weggemans *et al.*, 2004). According to *TransFair* study, Mediterranean area, Finland and Germany were found to have a low intake of TFAs (below 1 E% per capita, Hayakawa *et al.*, 2000), whereas Great Britain had

moderate intake (1.3 E%) and the highest intake was found to be in Iceland (2.0 E%) (Table 1). Referring to the *Finravinto* study made in 2002 on Finnish nutrition, *trans* fatty acids intake is the lowest when compared to other Scandinavian countries. *Trans* fat intake was only ca 1g-1.5g, which is 0.5 E%. This is thought to be because Finnish margarine industry adopted interestification method to make vegetable oils hard already in 1990s (Evira – Finnish Food Safety Authority, 2009). Since the *TransFair* study, the amount of TFAs in the food products have been steadily declining (Weggemans *et al.*, 2004), whereas the consumption in developing countries in contrast is believed to be increasing together with industrialisation (Hayakawa *et al.*, 2000).

## THE PHYSIOLOGICAL EFFECTS OF TRANS FATTY ACIDS

The adverse health effects of *trans* fatty acids have been under intense investigation in multiple different study settings, including epidemiological and clinical investigations. These have

| Country        | TFAs (% energy) | TFAs (g/day) |
|----------------|-----------------|--------------|
| Iceland        | 2.0             | 5.4          |
| Netherlands    | 1.6             | 4.3          |
| Belgium        | 1.4             | 4.1          |
| Norway         | 1.5             | 4.0          |
| United Kingdom | 1.3             | 2.8          |
| France         | 1.2             | 2.3          |
| Denmark        | 1.0             | 2.6          |
| Germany        | 0.8             | 2.2          |
| Spain          | 0.7             | 2.1          |
| Portugal       | 0.6             | 1.6          |
| Italy          | 0.5             | 1.6          |

**Table 1:**  
Average intake of  
trans fatty acids  
(TFAs) in the diet  
of various European  
countries (1995-1999)

yielded strong and continual evidence of TFAs threatening health in various ways which are summarised below. Despite the current evidence, *The Task Force on Trans Fatty Acids of the American Society for Clinical Nutrition* and *American Society for Nutritional Sciences* encourages continuing the research on these matters (Kummerow *et al.*, 1999). The different factors to be taken into consideration when assessing the link between *trans* fatty acids and health are the possible inherent errors that take place especially in retrospective studies. Other factors, as mentioned in challenges when assessing TFA content in food supply are the amount, variability and specificity of *trans* fatty acids in the diet or food item, the possible interaction with other nutrients, whereas also the accuracy of the food databases or – tables (Clandinin and Wilke, 2001).

### **Membrane fluidity**

The biological effects of TFAs and their influence on cell metabolism have been under continuous investigation since 1980s to 2009 (Fernández-San, 2009). The cell membrane, which is composed from lipids and fatty acids determines the physiological functions of a cell (Fernández-San, 2009). *Trans* fatty acids as a part of the make up of the cell membranes are found to alter the membrane fluidity. The *trans*-configuration and degree of saturation makes the cell membranes more rigid disturbing the various functions of human cells and tissues, including the activity of various enzymes of the cells (Diefenbach *et al.*, 1992; Fernández-San, 2009). This argument was also confirmed in a study where the physical properties of cell membranes between *cis*- and *trans*-containing phosphatidylcholines (PCs) were compared. The principal finding in differences was that the cell membrane consisting more *trans* fatty acids allows less membrane perturbation (Roach *et al.*, 2004).

Already in 1977 the evidence from different experiments indicated that maternal milk is markedly influenced by the dietary factors, such as caloric intake and by the ratio between carbohydrates and lipids (Aitchison *et al.*, 1977). Later studies have found that *trans* isomeric fatty acids in food supply interact and negatively affect the metabolism of essential polyunsaturated fatty acids, especially in premature children (Desci and Koletzko, 1995; Koletzko, 1991). Furthermore, the evidence suggests that tissue growth and development can be significantly disturbed also in later life of a child due to an inhibition of arachidonic acid biosynthesis by TFAs (Desci and Koletzko, 1995). Carlson *et al* (1997) has summarised different studies made before 1997 investigating the role of TFAs in infant and foetal development. Despite the challenge in research caused by the complexity of nutritional, genetic, whereas also by environmental and ethical factors, the review concluded that TFAs do inversely correlate with measures of growth and development (Carlson *et al.*, 1997).

Currently, there is strong data explaining the general health effects of the TFAs on humans, including growing and developing foetus and children (Fernández-San, 2009; Hayakawa *et al.*, 2000). The more recent studies have supported the earlier studies, concluding that *trans* fats alter the metabolic pathways of essential fatty acids by disturbing their incorporation to membrane phospholipids, but also their conversion to eicosanoid causing deficiencies of these fatty acids. The inhibition of *trans* fats in metabolism of gamma-linolenic- and arachidonic acid can alter the platelet aggregation and vascular function through the effect of *trans* fats to prostaglandins and other eicosanoid metabolism (Fernández-San, 2009; Hayakawa *et al.*, 2000). Furthermore, the disturbed

formation of arachidonic and docosahexaenoic acids from their precursors, the brain and nervous tissue of the foetus (requiring high amounts of these acids) could be then interrupted (Hayakawa *et al.*, 2000; Katan, 1998).

### **Inflammation and endothelial cell dysfunction**

One of the first controlled dietary intervention studies investigating the effect of TFAs on different markers of inflammation was conducted by Baer *et al* (2004). These markers are *fibrinogen*, *C-reactive protein* (CRP), *interleukin 6*, *E-selectin*, from which *interleukin 6* is known as a pro-inflammatory cytokines, whereas *E-selectin* instead is categorised as an adhesion molecule and CRP can be read as a acute phase protein (Baer *et al.*, 2004). Baer *et al* (2004) found that these markers are significantly negatively altered by consuming diet rich in TFAs. The data has increased ever since by numerous studies (Willett, 2006), such as study conducted by Lopez-Garcia *et al* in year 2005 and Sun *et al* in year 2007. Mozaffarian *et al* (2009) have gathered a good number of these studies, including controlled feeding studies and long-term observational studies made on humans, and written a comprehensive review published in *Medline* (2008). Furthermore, the very first *in vitro* evidence instead was gained from the study by Harvey *et al* (2008) who demonstrated the incorporation of *trans* fatty acids into endothelial tissues causing pro-inflammatory responses and malfunction.

### **Cholesterolemic effects**

The evidence from regression analyses of the influence of individual *trans* fatty acid on blood parameters has accumulated with the lead of Keys *et al* and Hegsted *et al* since the year 1965 (Kris-Etherton and Yu, 1997). This evidence has been further defined later by Hegsted *et al*, Mensink and Katana

and Yu *et al.*, explaining the effect of TFAs on total cholesterol, whereas also to lipoprotein cholesterol concentrations (Kris-Etherton and Yu, 1997). Numerous metabolic studies have concluded their investigations on *trans* fat consumption on blood parameters that they increase low density lipoprotein (LDL) cholesterol and decrease the high density lipoprotein (HDL) cholesterol, and negatively affects the ratio between HDL cholesterol and total cholesterol concentration (Clifton *et al.*, 2004; Katan *et al.*, 1994; Koletzko and Desci *et al.*, 1997; Mensink *et al.*, 1994; Troisi *et al.*, 1992; Wood *et al.*, 1993). In addition TFAs have found to increase lipoprotein[a] (Lp[a]) and plasma triglycerides (Lopez-Garcia *et al.*, 2005; Mensink *et al.*, 1992). Lp[a], a macromolecular complex consisting from apolipoprotein B, cholesterol, and other lipids, and apo[a] protein, is genetically driven. Additionally, age has found to have a little impact in Lp[a] concentrations (Mensink *et al.*, 1992). The effect of *trans* fat on plasma lipoproteins is interesting finding, because very few nutrients have been found to alter plasma lipoprotein concentrations (Fernández-San, 2009; Katan, 1998). However, according to some early critics about the link between TFAs and Lp[a], the experiments have been using higher quantities of TFAs than what the average intake of TFAs from the food supply is (Clevidence *et al.*, 1997).

### **Cardiovascular system**

Cardiovascular diseases are the number one causes of death in developed countries and nutrition scientist has been keen to find causal associations with dietary factors, one strong suspect being *trans* fatty acid consumption (Baylin *et al.*, 2003). Until 1997, the data gained from association studies between the coronary heart disease (CHD) and fatty acids in the diet has yielded somewhat inconsistent and controversial data (Pietinen *et al.*, 1997). However, currently there is strong evidence gained from several large-scale epidemiological,

case-control and feeding studies indicating that *trans* fatty acid consumption is a powerful determinant in development of CHD (Harvey *et al.*, 2008; Lemaitre *et al.*, 2006; Mann, 1994; Nelson, 2009; Oh *et al.*, 2005; Roach *et al.*, 2004; Sun *et al.*, 2007; Willett, 2006; Willett and Ascherio, 1994). For example Finnish men with intake of 6.2g of *trans* fats per day increased their risk for CHD by 1.4 when compared to men with lower intake of *trans* fats (Katan, 1998). In addition, the risk between consumption of TFAs and CHD is greatly linked with the low concentration of linoleic acid, either due by disturbed metabolism of linoleic acids by TFAs or by low intake from the diet (Bolton-Smith *et al.*, 1996). One of the leading investigations yielding evidence from the association between chronic intake of TFAs and coronary artery disease (CAD) has been the *St Thomas' Atherosclerosis Regression Study* by Watts *et al* (1996). An example from more recent study expanding and clarifying this evidence is a two year long case-control study with over a 150 completed participants by Clifton *et al* (2004). Other associations with TFA consumption and cardiovascular health concerns has been reported by Aro *et al* (1995) who concluded their *International Multicenter Study* in eight European countries and Israel (EURAMIC) that isomeric TFAs concentration in adipose tissue potentially correlates with myocardial infarction (MI). Furthermore, Stender and Dyerberg (2004) concluded their article by claiming that high TFA consumption is linked with ischemic heart disease (IHD).

Whether the results from the clinical and epidemiological studies of the relationship between TFAs intake and cardiovascular disease are not convincing enough (Hayakawa *et al.*, 2000), there is enough evidence (several controlled trials, observational studies, retrospective case-control studies, prospective cohort studies and epidemiological studies, Mozaffarian *et al.*, 2009) to report that high consumption of TFAs LDL

cholesterol, lowers HDL and thus increase the ratio of total to HDL cholesterol, which is agreed to be a high risk factor for CHD (Hunter and Applewhite, 1991; Katan, 1998; Pe' rez-Ferrer *et al.*, 2009).

### Insulin resistance

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The initial suggestions that there is a positive correlation between TFA intake and insulin resistance and type 2 diabetes mellitus (T2DM\*\*) were already made by Simopoulos in year 1994, but firmer suggestions were based on the findings from the *Nurses' Health Study*, where replacing 2% of TFAs with polyunsaturated fatty acids (PUFAs) were found to decrease the risk of T2DM by 40% in women (Salmeron *et al.*, 2001: Simopoulos, 1994). This finding in year 2001 by Salmeron *et al.* needed further investigation thus there was no epidemiological data available on the relationship between diabetes and TFA consumption. However, there was evidence that some PUFAs will improve insulin sensitivity and reduce plasma triacylglycerol concentrations (Clandinin *et al.*, 2001). Ever since, the evidence has built up from multiple clinical studies that TFAs are found to promote insulin resistance, this was highlighted in a review by Bray *et al.* (2002). Similarly support to this argument has given studies by Lovejoy *et al.* (2002) who measured the changes in insulin secretion, whereas by Mozaffarian *et al.* (2004) and by Lopez-Garcia *et al.* (2005) who studied the relations between TFA intake and inflammatory markers. Mozaffarian *et al.* (2009) also suggested that TFAs could worsen insulin resistance and metabolic syndrome, based on few clinical trials, where insulin resistance got worse in overweight, especially in centrally obese -, inactive - or diabetic individuals. However, the effect on healthy, lean and active subjects remained unknown. It has yet been challenging to differentiate excess adiposity and high intake of TFAs with-in the issue, whereas also the small number in participants

and short study lengths are arguments for requirements for future studies (Larque *et al.*, 2006; Mozaffarian *et al.*, 2009).

To unravel the evidence behind the correlation between TFAs and insulin resistance, it has been suggested that TFAs might modify and cause polymorphism in some genes causing changes in gene-nutrient interaction (Lefevre *et al* 2005; Pisabarro *et al.*, 2004). Pisabarro *et al* (2004) concluded their investigation that chronic high intake of TFAs can cause polymorphism in peroxisome proliferator-activated receptor (PPAR) $\gamma$ 2 Ala allele. This polymorphism has been linked with obesity, insulin resistance, and T2DM. It has been proposed that the polymorphisms in this allele cause increased storing of ectopic triglycerides, especially in  $\beta$ -cells and myocytes (Pisabarro *et al.*, 2004). Another TFAs induced gene polymorphism has been suggested by Lefevre *et al* (2005) in fatty acid-binding protein 2 (FABP2). The potential TFA induced polymorphism takes place at codon 54 (Ala54Thr) causing disturbances between the interaction of dietary fats and insulin sensitivity (Lefevre *et al* 2005).

## Cancer

It is known that more than one third of all the cancers are potentially caused by the diet, dietary fatty acids, including *trans* fatty acids having a major causative role (Slattery *et al.*, 2001). The recent evidence indicates from positive correlation between high TFA intake and breast-, colon- and prostate cancers (Bartsch *et al.*, 1999; King *et al.*, 2005)

Already in 1997, there has been strong evidence indicating a positive association between *trans* fat intake and breast cancer among European populations (Kohlmeier *et al.*, 1997). The evidence have been gained from migrant, case-control, and ecological studies, from which at least case-control studies

has been using adipose biopsies to determine *trans* fatty acids intake from the food supply (Kohlmeier *et al.*, 1997).

### Other health risks

Allergic diseases have also been suggested to be linked with children consuming a diet high in TFAs (Stender and Dyerberg, 2004).

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### RECOMMENDED INTAKE OF TRANS FATTY ACIDS

According to the *Scientific Advisory Committee on Nutrition* (SACN) the maximum intake of *trans* fats should not exceed more than 2% of the total energy, whereas WHO recommended in 2003 that *trans* fat should be limited to below 1% of overall energy intake (Food Standards Agency, 2008; Scientific Advisory Committee on Nutrition, 2007). The WHO recommendation is not a figure based on any scientific studies, which clearly indicate the safe amount of TFAs consumption to be 0g (Food Standards Agency, 2008). The current figure (1% of total energy intake) was chosen in relation to the current consumption figures of TFAs in the modern food chain. However, it can be argued whether this figure is representative for a safe or healthy level of *trans* fat intake (Food Standards Agency, 2008). In 2008, FAO and WHO updated the information based on *Expert Consultation on Fats and Oils in Human Nutrition* meeting (Nishida and Uauy, 2009), resulting recommendations for consumers to reduce the intake of both saturated fat and TFAs, whereas food manufacturers should reduce the level of *trans* fats in their products (Hayakawa *et al.*, 2000).

In USA, based on recommendations made by *American Heart Association* in 2002 and *Dietary Guidelines for Americans* set in 2005, TFAs should be kept as low as possible. In 2006,

the *American Heart Association* updated recommendation for TFAs intake was to be less than 1% of total energy (Marcason, 2006). In 2002, *The National Academy of Sciences* (NAS), supported by a 2006 *New England Journal of Medicine* (NEJM) scientific review, announced that there is no safe intake level. NAS based their recommendations on the facts that TFAs are non-essential with no known benefits but considerable potential harm to health. This being said, NAS did not suggest the elimination of TFAs from the food supply, based on the fact that *trans* fats does occur naturally in some products (Marcason, 2006).

Nutrient recommendations are also extremely important to fix to support the critical time for foetal but also infant development. The developing infant is markedly dependent on fat from the food supply; hence it is vital to take the fatty acid composition of this fat into consideration. To provide steady and sustainable grounding for healthy future development of an child, *trans* fatty acids should not be used in standard infant formulas or at least not exceed 6% of total fatty acids or 3% of total energy (Carroll, 1989).

### **PUBLIC HEALTH APPROACH**

The current nutritional and public health advice still aims to lower the intake of total fat, which would ideally decrease the consumption of total energy intake, including also saturated fat and *trans* fats (Lichtenstein *et al.*, 1993). However, it can be strongly argued whether this is effective enough to limit the *trans* fat consumption around the globe (Ascherio *et al.*, 1997). As a result of the currently valid nutritional advice the total fat consumption has declined from 38% to 34% in USA, but only very little reduction was seen in *trans* fat intake (Ascherio *et al.*, 1997). There actually hinders a great worry with the current dietary advice, which might lead the individuals in fear

of saturated fat to choose products containing *trans* fatty acids (Aro *et al.*, 1997). This paradox is exhibited by the commercial advertising, where products that have replaced saturated fats with partially hydrogenated oils (high in *trans* fats) and marketing them as “low in saturated fat” and “low in cholesterol”. To extent the level of complexity even further, the manufacturers are not required to inform the *trans* fatty acid content in the labels in all countries (Ascherio *et al.*, 1997).

Changes are clearly needed in advising public to make better food choices, but effort is also required to increase the pressure towards changing the manufacturing processes (Ascherio *et al.*, 1997). Dietary advice should encourage the public to choose natural vegetable oils in their non-hydrogenated form and to avoid animal fats, but mostly commercial products baked or fried in hydrogenated fats (Ascherio *et al.*, 1997). This would ideally lead public to favour fats low in saturated and *trans* fatty acids (Aro *et al.*, 1997).

### **TRANS FATTY ACIDS AND LABELLING**

In Europe *trans* fat labelling is following the rules of *Directive of the European Communities on Nutrition Labelling*. The use of fats has to be listed on the ingredient list, but *trans* fat content does not have to be mentioned as separate from other type of fats in the Nutrition Fact-panel (Epira – Finnish Food Safety Authority, 2009; New South Wales Government (NSW) Food Authority, 2009). Currently it is legal in many European Union countries to sell food products with maximal obtainable concentration (up to 60%) of the total fat as industrially produced TFAs without any notice. Furthermore, the use of high amounts of TFAs in packaged foods is only mentioned in the list of ingredients as “partially hydrogenated fat/oil”, “hydrogenated fat/oil” or “liquid shortening”. To further confuse consumers, the term “partially hydrogenated fat” has also

been used to characterise a mixture of fully hydrogenated fat and non-hydrogenated unsaturated fat, which does not contain *trans* fat (Stender *et al.*, 2006). There are also numerous products, where TFAs content has been included among the monounsaturated fatty acid content (Fernández-San, 2009). The food labelling terminology has been confusing consumers in Europe, but also in USA. This information can only be understood by devoted and educated consumers (Stender *et al.*, 2006). In countries where TFAs are freely used in the food supply, nutrition education in reading labels is essential (Marcason, 2006).

In USA, in 1999 FDA proposed to include TFAs in Nutrition Facts-panel (Hayakawa *et al.*, 2000) and during 2006, the FDA made this proposal mandatory. However, caution is needed when reading the labels, because manufacturers are allowed to market the product to be *trans* free if the product contains less than 0.5% of TFAs per serving (Marcason, 2006). The FDA has also previously suggested an asterisk in the % Daily Value column, which would state that “intake of *trans* fats should be as low as possible.” This suggestion never materialized because of the huge defence of food industry. Labelling issue cannot be overemphasized. According to FDA estimation, *trans* fat labelling could save between 2000 and 5600 lives per year and 7600 to 17 000 cases of coronary heart disease could be prevented per year (Ban Trans Fats, 2007). This being said, FDA should strictly follow *The Nutrition Labelling and Education Act of 1990*, which clearly states that the FDA “shall” require that the declaration of nutrients “be conveyed to the public in a manner which enables the public to readily observe and comprehend such information and to understand its relative significance in the context of a total daily diet”. Thus, to materialise the above, FDA should be more demanding towards the government and food industry (Moss, 2006). Support to federal regulations to require food

industry to add TFA content in food labels, simultaneously when significantly reducing and eliminating the use of TFAs has been given also by other scientists, such as Koletzko and Desci (1997) and Willet and Ascherio (1994).

## GOVERNMENTAL RESPONSIBILITY

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Government should control the levels of *trans* fats in food supply by better regulation, labelling and misleading claims in food products (Hayakawa *et al* 2000). WHO also has recommended in 2007 that governments around the globe should get rid of partially hydrogenated oils if the *trans* fat labelling is not powerful enough to reduce the intake of TFAs (Wassell and Young, 2007). Mozaffarian *et al* in 2009 also added that TFAs are food additives with no health benefits and there are examples seen that TFA elimination is possible. This can be seen in Denmark (Stender *et al.*, 2006), Switzerland (Ulmer, 2008), Netherlands, California, New York City, Philadelphia and Canada (Food Standards Agency, 2007; Health Canada, 2009; CBC News, 2009). The approach Danish government launched onto the issue in year 2003 functions as an effective model for others working in clearing the food supply from TFAs (Astrup, 2006). In Denmark, labelling was found insufficient to reduce the intake of TFAs (Leth *et al.*, 2006), thus the simple approach prohibited any products containing more than 2% of *trans* fatty acids and offenders are now facing fines and even jail (Food Standard Agency, 2007). The Danish Nutritional Council (established in 1992) convinced the Danish politicians that elimination of industrial TFAs would not effect the availability, taste or price of foods (Astrup, 2006). Since the action taken by the Danish government, in year 2004 Canada became the first country to include TFA details on the nutrition labelling of pre-packaged foods and Tiburon in California became the first TFA free city in USA (Food Standard Agency, 2007). In 2006, New

York followed Tiburon, whereas the whole USA announced that TFAs are to be listed in nutrition labelling (CBC News, 2009). One year later (in 2007), Philadelphia also banned *trans* fats and *Health Canada* announced that it was adopting the recommendations made by the *Trans Fat Task Force* to limit the content of TFAs to 2% of the total fat content in food industry (Health Canada, 2009). In 2008, California began the nationwide legislation to prohibit the use of TFAs in restaurants and food retailers, which is to be completed by 2010 and 2011 (CBC, 2009). Switzerland instead followed the Denmark's lead to have strict limit of 2% TFA in their food products (Ulmer, 2008).

Countries that does not have any governmental legislation for TFAs, such as Australia, New Zealand and Singapore are arguing that regulations are not necessary because the average TFAs intake in these countries is below WHO's recommendation (Food Standards Australia New Zealand, 2009). Despite it was reported in the *Scotland Sunday newspaper* (2007) that the British government was considering the TFA ban, UK also does not currently have a ban against the use of *trans* fats (Stop Trans Fats, 2009). However, the effective campaigns against *trans* fats since 2004 by a freelance journalist and campaigner on health and environmental issues, Oliver Tickell has maintain the average intake of TFAs relatively low. He has kept substantial website in UK, Europe and around the world called *tfX*, increasing the consumer awareness about the dangers of *trans* fats (Stop Trans Fats, 2009). This being said, the work of Tickell should not lighter the responsibility weight of the governments.

A report published by the *Food Standards Agency* in 2007 investigated two options for regulating *trans* fatty acids, a voluntary and mandatory. A stakeholders meeting for this report involved representatives from *The Nutrition Society*, *The*

*British Heart Foundation, The Heart UK, The National Consumer Council, and tfX* who were all supportive of mandatory regulation of *trans* fats. Despite the declared support at the stakeholders meeting, the lack of support that the *tfX* campaign in UK receives is surprising. Furthermore, the current information provided by *The British Nutrition Foundation, The Food Standards Agency* and *The British Heart Foundation* on *trans* fats should be reassessed. *British Heart Foundation*, which could potentially be one of the leading foundations against the fight against *trans* fats have been found to quote: “*trans* fatty acids may have a small effect in raising cholesterol levels”, which can be misinterpreted by some as the *trans* fats are known to lower the HDL cholesterol and raise the LDL cholesterol (Food Standards Agency, 2007). The approach *tfX* encourages UK to adopt is either the Danish standard, or an updated version of the Danish standard with a 1% TFA maximum. This would eventually lead on to the establishment of an EU wide standard and bring the health benefits the Scandinavia and UK are now enjoying to all Europeans (Tickell Oliver, 2007), whereas provide a simple lead for developing countries.

## CONCLUSION

There is a clear body of evidences for causal association between consumption of industrial TFAs and numerous health risks and metabolic disturbances. The intake of industrial TFAs varies around the globe, still exceeding the WHO’s recommended intake level of 1% of total energy in many European countries and in USA. In addition, the intake of industrial TFAs are increasing rapidly in modernising developing countries. Nutritional and public health advice around the globe has been aiming to lower the intake of total fat, which would ideally decrease the consumption of saturated fat and *trans* fats. However, it has been argued whether this advice is the most effective way to limit the intake of TFAs in

countries where strict TFA reduction is needed. There might even hinder a concern that individuals advised to avoid saturated fat (in order to lower their serum LDL cholesterol) will increase their intake of TFAs.

Beside of giving support for the future association studies between industrial TFAs and health risks, one aim would be to ease the currently faced challenges in collecting and obtaining accurate data of the intake of different nutrients from the food supply, including TFAs. It would be in great importance to generate official universal food databases and -tables. This would minimise the sparseness of the data on consumption of TFAs among other nutrients around the globe. Among health-care professionals, the access to these universal food databases should be made free of charge, whereas also easily found by general public. The governments around the globe in turn should control the levels of TFAs in food supply by better regulation and force improved labelling of TFAs in food products. View of the healthier Europe would include adoption of lower than 1% of TFAs from total energy. This would potentially lead on to the establishment of an EU wide standard.

Public should be advised to use natural vegetable oils and fats and to avoid products baked or fried in hydrogenated fats. Effective planning and action is required in countries where the use and consumption of TFAs still exceeds 1% of total energy intake. The knowledge among the general public about TFAs needs to be forced in multiple ways, in close co-work of public health specialist, government, media and food industry. The clear aim would be to get the message about the health risks of *trans* fats in receptive form and how to correctly read the ingredient lists of food products. In addition, eateries and restaurants going to *trans* fat-free menu should be supported by the government and the food menus promoted as *trans* free.

The changes needed for healthier nation is to provide and encourage societies with healthy environment, whereas also to educate and support in making healthier choices in everyday life. This can be made in light of win-win-win situation between nutrition science, food industry/government, and general public, but the effort is needed from all levels of the society. The knowledge and tools exists, devotion and action is required, whereas also the ability to see the future outcomes.

### BIOGRAPHY

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