POLISH JOURNAL OF FOOD AND NUTRITION SCIENCES

http://journal.pan.olsztyn.pl
e-mail: pjfns@pan.olsztyn.pl

Pol. J. Food Nutr. Sci.
2010, Vol. 60, No. 2, pp. 107-114

FORMATION AND PROPERTIES OF *TRANS* FATTY ACIDS – A REVIEW

Anna Żbikowska

Faculty of Food Technology, Warsaw University of Life Science, Warsaw, Poland

Key words: *trans* fatty acids, hydrogenation, sources of TFAs

The structure of the carbon chain of *trans* fatty acids (TFAs) is similar to that of the saturated fatty acids (SFA) molecules, which takes effect in higher melting points. There are two major sources of TFAs, those that come from ruminant animals and those that are produced during technological process. Results of numerous studies show that the intake of either saturated or TFAs raises blood levels of LDL cholesterol. This fraction of cholesterol is a risk factor of heart diseases. In addition to raising "bad" cholesterol, TFAs reduce blood levels of HDL cholesterol which protects against heart diseases. In contrast to hydrogenated TFAs, those originating from ruminants are healthy components of diet.

Although a negative influence of TFAs on health has been scientifically proven, the UE has not yet adopted any regulations concerning reduction of TFAs levels in food (except Denmark) or labelling TFAs content. Nevertheless, on the basis of many experiments, a conspicuous decrease of TFA level in foods is observed. Food and nutritional organisations recommend that the intake of TFAs by all population groups should be kept as low as possible, which is about 1% of energy intake or less.

INTRODUCTION

Over the past decades, research dealing with *trans* fatty acids (TFAs) has intensified and now encompasses a wide variety of multidisciplinary fields that affect food science and technology, nutrition, physiology, toxicology, analytical chemistry. As of 31 March 2010, SCOUPS and MedLine provide comprehensive list of 6926 and 3690 articles, respectively, directly concerning TFAs. The authors emphasize health risks and benefits of *trans* fatty acids including conjugated fatty acids in food. The scientists and technologists discuss on the revision of the nutritional-labeling regulation. The influence of processing on the production of TFAs in food and level of consumption are the subject of numerous research groups in many countries. The link between cardiovascular risk and consumption of TFAs and conjugated linoleic acid (CLA) has been long time studied. Also the public independent organizations (*e.g.*, French Food Safety Agency in France) took a part in discussion on TFAs.

This review will focus on the formation of *trans* and conjugated fatty acids, their biological properties, the main sources in human diet, their daily intakes, and regulatory approach to control the content of TFAs in foods.

PHYSICOCHEMICAL CHARACTERISTICS OF *TRANS* FATTY ACIDS

Both nutritional and technological properties of fats depend on their fatty acids composition and position in molecules of triacylglycerols (TAG), (Figure 1). Unsaturated fatty acids show two types of isomerism: geometric and positional (changes in the position of the double bond).

Steric configuration (geometric isomerism) is characteristic of the chemical compounds which contain at least one double bond. It also refers to fatty acids. Double bonds provide rigidity to the molecule and result in specific molecular configurations. Naturally occurring fatty acids in foods usually have *cis* configuration, in the molecule having a "V" shape. In *trans* fatty acids, the hydrogen atoms are on the opposite sides of the molecule, and the molecule assumes a nearly linear configuration similar to that for saturated fatty acids (Figure 2) [Hunter, 2005]. The term *trans* refers to all unsaturated fatty acids in which at least one double bond exists in *trans* configuration.

The consequence of the different tertiary structures of *cis* and *trans* isomers is various crystalline packing that results in different melting points. TFAs have considerably higher melting points than the corresponding *cis*- isomers [Feldman *et al.,* 1996]: melting point of oleic acid is 14°C, and respectively that of the *trans* form – elaidic acid – is 45°C [Koletzko & Desci, 1997].

The European Food Safety Authority (EFSA) stated that "TFAs are unsaturated fatty acids that have at least one

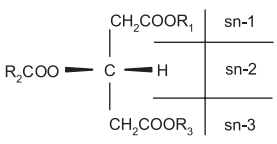

FIGURE 1. Fatty acids position in molecule of TAG.

Author's address for correspondence: Anna Żbikowska, Department of Food Technology, Warsaw Agricultural University (SGGW), ul. Nowoursynowska 159C, 02-776 Warsaw, Poland; tel.: (48 22)59 375 25; (48 22) 59 375 27; e-mail: anna_zbikowska@sggw.pl

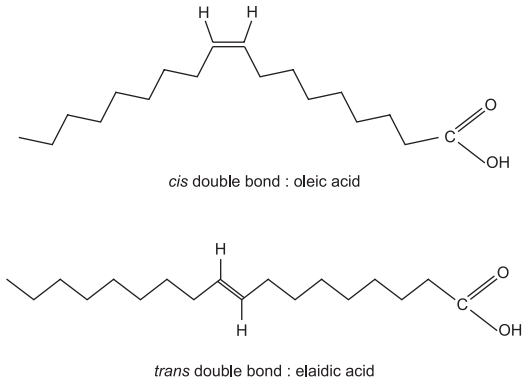

FIGURE 2. Structural formulas for the *cis*-fatty acid – oleic acid, and its *trans* isomer – elaidic acid.

double bond in the *trans* configuration. Some polyunsaturated TFAs have a conjugated structure (*e.g.* CLA in milk fat), *i.e.* have double bonds which are not separated by a methylene group, but most have isolated (non-conjugated) double bonds" [EFSA, 2004].

SOURCES OF TFAs

There are two major sources of TFAs, those that come from ruminant animals and those that are produced during technological process (Table 1). *Trans* isomers are not found in natural vegetable oils, whereas they occur in small quantities in animal fats (in milk fat from 2% to 8%, and from 2% to 5% in beef).

In the microflora of ruminants' alimentary tract an enzyme called isomerase can be found which causes isomerisation of steric configuration of unsaturated acids around the double bond – the *cis* form develops into *trans* [Corl *et al.,* 2002, 2003]. *Trans* fatty acids generated by enzyme hydrogenation of polyunsaturated fatty acids in the rumen of ruminants are transported to milk and deposited fat. Biohydrogenation of linoleic (LA) and α-linolenic (ALA) acids yields predominantly in *trans* isomer of vaccenic acid (C18:1 t11). The rate of its production largely depends on the availability of LA and ALA in the roughage, e.g. TFAs level in cow's milk is about four times higher during the summer than during wintertime (about 4 and 1%, respectively). In Europe, the contribution of milk and dairy products as well as meats to dietary intake of TFAs are about 30% and 10%, respectively [Koletzko & Desci, 1997]. *Trans*-vaccenic acid (C18:1 t11) accounts for 67-86% of the total TFA [Larqué *et al.,* 2001; EFSA, 2004; Kraft *et al*., 2006, Stender *et al*., 2006].

TABLE 1. Major *trans* fatty acids in foods [Gebauer *et al*., 2007].

Common name	Chemical name of common isomers	Major source
Elaidic acid	C18:1 t9	Partially hydrogenated oils
Vaccenic acid	C18:1 t11	Ruminant meat and milk
Linolelaidic acid	C18:2 t9t12	Partially hydrogenated oils
Conjugated linoleic acid (CLA)	C18:2 c9t11 C18:2 t10c12	Ruminant meat and milk

One of the very important problems of the fat industry is a constant deficit of natural vegetable solid fats. The exceptions are palm and coconut oils which due to high levels of saturated fatty acids are solid. Solid fats can be obtained from liquid oils by using different technological processes. The most commonly used is partial hydrogenation, in which the produced solid fats have the melting point around 37°C. The hydrogenated fats also exhibit higher resistance to oxidative changes.

Partial hydrogenation affects mostly geometrical and positional *trans*-18:1 isomers while reduced the amount of PUFA in the original oil (Table 2) [Ledoux *et al.,* 2007].

Elaidic acid (C18:1t9) typically is the major isomer in industrial sources of TFAs. The C18:1t isomers constitute approximately 80-90% of the total TFA in foods. Other isomers including C16:1t, C18:2t, C18:3t, and long chain polyunsaturated TFA can also be an important component of the total TFAs [Weggemans *et al.,* 2004; Kraft *et al.,* 2006; Stender *et al*., 2006].

However, the hydrogenation is strongly criticized, mainly for its „unnatural" and purely chemical character, but especially for generating TFAs [Liu *et al.,* 2007]. Partially hydrogenated fats can even contain up to 50% of *trans* isomers [Guillén & Cabo, 1997].

The formation of TFAs during hardening process can be explained by the mechanism in which the impermanent half-hydrogenated molecules occur before the hydrogenation is completed (Figure 3). Under the partial hydrogenation conditions, the weaker access to hydrogen atoms causes tha, in respect of thermodynamics, the molecule of unsaturated fatty acid finds it easier to lose one hydrogen and transform double bond into *trans* configuration than accepting another and changing into the saturated form [Baryłko-Pikielna & Osucha, 1990; Niewiadomski, 1993].

TFAs level in fats during hydrogenation stays in equilibrium between *trans* and *cis* form, *trans* to *cis* ratio is 2:1 [Drozdowski, 2002]. *Trans* bonds prevail, because such a structure is energetically more stable [Baryłko-Pikielna & Osucha, 1990]. For instance, in hardened rape oils there may be found even 20 types of isomers with *trans* bond situated between 6 and 16 carbon in acid C18:1.

TFAs produced during heat treatments depend on process variables such as temperature and length of treatment [Devinat *et al.,* 1980; Liu *et al.,* 2007]. Thermal treatments of fats and oils such as deodorization, cooking, frying *etc*., generate TFA isomers with limited double bond migration along the carbon chain. Unlike partial hydrogenation, heating induces the formation of mainly *trans* 18:2 and *trans*-18:3 [Wolff, 1993; Ledoux *et al.,* 2007].

TABLE 2. TFAs in partially hydrogenated vegetable oil.

TFA	Level (% total TFA)
Trans C18:1	85-95
Trans C18:2	8-22
Trans C18:3	<1 – 7
Trans C16:1	~0.04

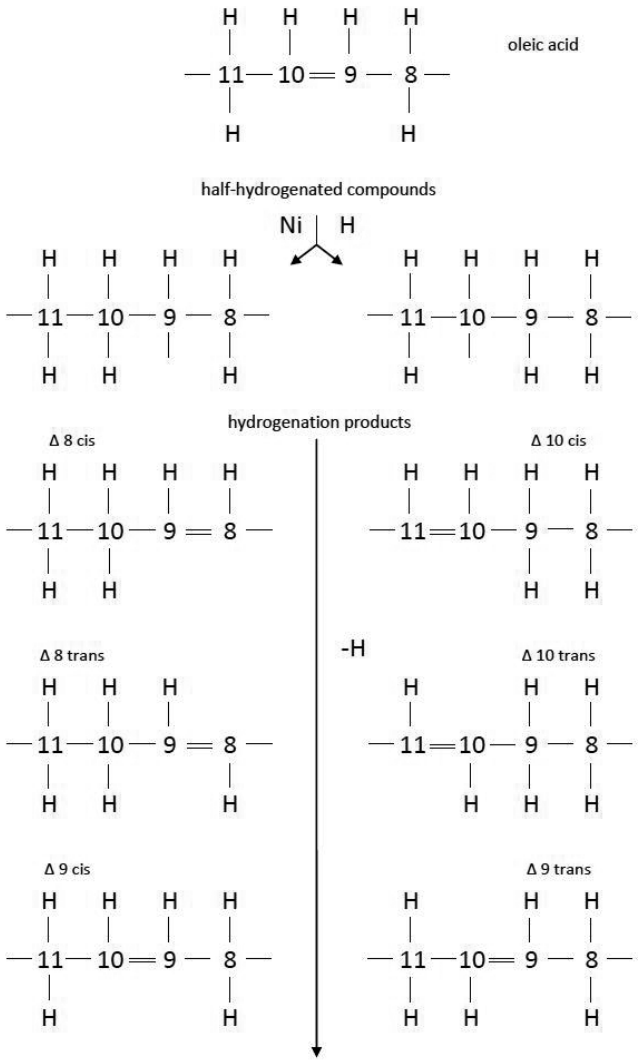

FIGURE 3. Mechanism of generation of *trans* isomers during hydrogenation.

BIOLOGICAL PROPERTIES OF TFAs

Cis isomers of fatty acids play important biological roles, whereas *trans* isomers do not show such an activity. TFAs enter all the metabolic pathways like other fatty acids do [Chardigny *et al.,* 2007]. Dietary *trans* isomers are incorporated into membranes and other cellular structures. Most of TFAs occur in abdomen tissue, blood plasma and women's milk fat [Stender & Dyerberg, 2004]. *Trans* fatty acids are transported across the placenta and secreted in human milk in amounts that depended on the maternal dietary intake [Innis, 2006].

There is strong supporting evidence that TFAs promote negative influence on the cholesterol level in blood. Moreover, their interaction is at least as harmful as saturated fatty acids (SFAs). TFAs not only increase the levels of low density lipoprotein cholesterol (LDL-C or "bad") as do SFAs, but also decrease the levels of high density lipoprotein cholesterol (HDL-C or "good") in the blood. The LDL fraction may locate in blood vessels and lead to atherogenic changes, namely to arteriosclerosis [Müller *et al.,* 1998; Van Duijn, 2000; Aro, 2001; De Roos *et al.,* 2001; Sundram *et al.,* 2003; Brooker & Mann, 2008]. According to Juttelstad [2004], the intake of fats containing reasonable levels of TFAs does not contribute to the changes in cholesterol concentration in blood.

In the experiments conducted in Holland on a group of people over 64 years, it was stated that TFAs levels in diet might have an influence on the development of coronary heart diseases [Oomen *et al*., 2001]. Those whose diet was rich in the *trans* form, not only industrially produced but also occurring in nature, more often suffered from ischemic heart diseases and consequently many of them had a heart attack. During this experiment, in the diet of the surveyed the levels of TFAs became lower due to the decrease in TFAs' levels in commercially available products, which as a result minimized the risk of the incidence of ischemic heart disease. According to this observation, Oomen *et al.* [2001] stated that reducing the amount of energy coming from TFAs by about 2-4% results in a 23% decrease in mortality rate due to a heart attack.

Also Czech scientists reported that people who have more TFAs in their fat tissue more frequently suffer from heart diseases and arteriosclerosis. However, they claimed that the total level of the consumed TFAs is unimportant, only the level of elaidic acid plays a vital role, because its presence evidently affects the occurrence of diseases of the circulatory system [Dlouhy *et al.,* 2003].

TFAs AND CANCER

Present data suggest that the association between TFAs in adipose tissue and the incidence of cancers of the breast, prostate and colon is still equivocal [Bakker *et al.,* 1997; Kohlmeister *et al.,* 1997; McKelvey *et al.,* 1999; Slattery *et al.,* 2001]. However, there are suggestions of increased risk caused by TFAs.

Nevertheless, it cannot be ruled out that dietary TFAs may contribute to changes in the functioning of cell membranes, which as a result may lead to the development of some cancer tissue [Kohlemaister *et al.,* 1997; Holmes *et al.,* 1999; McKelvey *et al.,* 1999; Slattery *et al.,* 2001; Larqué *et al.,* 2001].

EXPOSURE OF FOETUS AND NEWBORNS TO *TRANS* FATTY ACIDS

Dietary *trans* fatty acids are transformed by the placenta to the foetus and incorporate into foetal tissues. According to Koletzko & Desci [1997] TFAs may contribute to infantile birth weight in preterm and health term babies as well as reduce the duration of pregnancy.

According to McDonald & Min [1996] as well as Verschuren & Zevenbergen [1990], TFAs which make less than 5% of the energy supplied every day with food do not possess negative properties, on condition that the level of dietary linoleic acid is reasonable, that is to say – it does not make less than 2% of the energy.

TFAs AND DIABETES

The relation – if any – between the intake of TFAs and the appearance of type-2 diabetes has not yet been settled.

There are, however, some indications that TFAs may rise the insulin level [Meyer *et al.,* 2001; Larqué *et al.,* 2001; Van Dam *et al.,* 2001; Bray *et al.,* 2002].

Conjugated dienes of linoleic acid (CLA)

The *trans* isomer of vaccenic acid (C18:1 t11), a precursor of conjugated linoleic acid (CLA), is the TFA isomer in ruminant fat. CLAs are a group of positional geometric conjugated isomers of linoleic acid. The two predominant isomers, with known bioactive properties, are *cis*-9, *trans*-11 (c9 t11), and *trans* -10, *cis* -12 (t10 c12) [Rainer, 2004]. Contrary to industrially produced TFAs, CLAs have a positive influence on human's health [Fritsche *et al.,* 2001; Stanley, 2004]. They were first discovered by Pariza and his group when investigating the carcinogenic components of grilled beef [Pariza, 1985]. Studies have established that isomer C 18:1 11t is an anticarcinogenic factor [Wolff, 1995; Corl *et al.,* 2003; Wahle *et al*., 2004; Gerber, 2007]. It was observed among animals that only a high daily intake of CLAs may prevent cancer development. For rats the intake was at least 0.1 g of CLAs/100 g of fats, which corresponds to 1.5–3.0 g/person/day [Bartnikowska *et al.,* 1999; Turini, 1999; Larqué *et al.,* 2001; Yu, 2001; Belury, 2002].

CLAs have been proven to favorably modify the body composition in the mouse model (leading to a reduced body fat and increased lean body mass), which has been assumed to be an anti-obesity effect. Thus, the mechanisms of oxidation and storage after CLAs intake have been studied in more detail. In humans, current data have shown that there was no consensus on the reduction of body fat induced by CLAs. Evidence for decreased body fat has been provided for overweight or obese subjects; this anti-obesity effect has been attributed to the 10*t*,12*c*-18:2 CLA isomer. The required doses have been found to range from 1.7 to 6.8 g/day for the CLAs and to be 2.6 g/day for 10*t*,12*c*-18:2. However, it should be noticed that CLAs exert a weak effect, even after 1 year of treatment. The weaker effect in humans than in animal models may be due not only to inter-specific differences, but also to the level of lipid intake, which is much higher in humans than in animals [Léger *et al*., 2007].

CLAs have been recently receiving growing attention because they have exhibited the ability not only to decrease the cholesterol level in blood but also the ability to prevent the arteriosclerosis. They have also been reported to exert beneficial regulatory effects on immune function, lipid and eicosanoid metabolism, cytokine and immuno-globulin production and to modulate the expression of a number genes [Wahle *et al.,* 2004; Pariza, 1985].

Worthy of noticing is, however, that the literature suggests that CLA has no significant effect on insulin sensitivity in lean, healthy subjects. However, an elevated insulin response after high TFA intake *versus* monounsaturated fatty acids in subjects with type 2 diabetes could indicate increased insulin resistance. Furthermore, CLA isomer t10 c12 clearly impairs insulin sensitivity in insulin-resistant and diabetic subjects. More data are, however, needed before we can conclude more firmly on the effect of TFAs on insulin sensitivity [Risérus *et al.*, 2002; Risérus, 2007].

Technological aspect of TFAs

Trans fatty acids, mainly due to their physical properties (melting point), are eagerly used in baking and confectionary industry. During The World Fat Congress in Brighton in 1999, it was even stated that fats with *trans* isomers are essential for baking cakes of good value [Żbikowskka & Krygier, 2003]. The positive effects of TFAs on the quality (volume, sensory values) of sponge cakes and puff- pastry cakes were observed [Krygier & Żbikowska 2004, 2005; Żbikowskka & Krygier, 2005; Żbikowska *et al.,* 2005].

Sources of TFAs in human diet and their daily intake

The sources of TFAs can be divided, in respect of their origin, into two major groups:

- Natural fats, which occur in dairy and other animal fats (milk, butter and meat of ruminants) as well as products made of them.
- Modified vegetable oils (mainly by hydrogenation), which are used for the production of hardened margarine spreads and industrial fats as well as products made of them. Such products are sweet snacks (baked products, doughnuts, chocolates), salt snacks (French-fried potatoes, chips) instant soups and souses and take-away foods. The TFAs level in such products is dependent on the sources of fats and technology of their production. TFAs (produced by commercial modification) occurred in human diet after 1902, when Norman used hydrogenation for the first time.

According to Canadian scientists only 11% of TFAs come from margarine products. The majority of them come from fats "hidden" for example in pastries, take-away foods and animal fats [Pelletier *et al.,* 1998]. The studies on the level of TFAs in commercial products in Poland have been carried out for many years. Daniewski *et al*. [1998] found from 0.1 to 72.6% of TFAs in baking and confectionary fats and from 0.1% to 45.5% in fats extracted from baked and confectionary goods. Later studies showed an insignificant decrease in TFAs in baking and confectionary fats to 70%, and in confectionary goods to 40% [Balas, 2004]. But according to Mojska [2004], the level of TFAs in baked and confectionary goods was up to 10%. A considerable decrease of TFA level has been observed especially in soft margarines (tub margarines), in which the TFA level in most cases does not exceed 1% [Żbikowska *et al*., 2007]. The study of Wagner *et al.* [2008] confirmed the general trend to reduced TFAs content in food group marketed in the UE.

The daily intake of TFAs varies and depends on the country, the intake of TFAs in different countries is shown in Table 3. Due to the public and scientific discussion, TFAs content of foods has been reduced worldwide thereby also reducing the intake of TFAs [Craig-Schmidt, 2006; Wagner *et al*. 2008]. In 1975, the average intake in Europe was 6 g/day [Stender *et al.,* 2006], in the TRANSFAIR study the intake varied between 1.2 and 6.7 g/day with a lower intake in the south [Hulshof *et al.,* 1999]. A constant decrease in TFAs intake is observed. For example, in the USA between years 1980-1997 a 23.8% fall of TFA energy intake was observed among men and 13% among women [Harnack *et al*., 2003].

Some of food and nutrition organisations, for example WHO/FAO, American Heart Association (AHA), recommend

TABLE 3. Daily intake of TFAs in different countries.

Daily intake (g/person/day)	Sources of TFAs	Country	Reference
1.1	margarines and shortenings	Denmark	Ovesen *et al*. [1998]
4.0	different sources	Norway	Van Poppel [1998]
5.4	different sources	Iceland	Van Poppel [1998]
0.2	margarines	Spain	Alonso *et al*. [2000]
2.4	different sources	Spain	Parcerisa *et al*. [1999]
1.1	margarines	Belgium	Greyt *et al*. [1996]
3.8	different sources	Germany	Steinhart & Pfalzgraf [1992]
8.4	different sources	Canada	Ratnayake *et al*. [1998]
8,1	different sources	USA	Hunter & Applewhite [1991]
8,0	different sources	USA	Kritchevsky [1996]
4,4	different sources	Dutch	Oomen *et al*. [2001]
4-6	different sources	Great Britain	Kritchevsky [1996]
2.8	different sources	Great Britain	Hulshof *et al*. [1999]
0.6	different sources	South Korea	Craig-Schmidt [1999]
0.1-0.3	different sources	Japan	Craig-Schmidt [1999]
3.3 – 6.9	different sources	Poland	Baryłko-Pikielna & Osucha [1998]
1.6	different sources	Italy	Hulshof *et al*. [1999]
2.7 (men) 2.1 (women)	different sources different sources	France	Laloux *et al*. [2007]

that TFAs be no more than 1% of total energy intake [Gebauer *et al.,* 2007].

REGULATORY APPROACH TO CONTROL TFAs LEVELS IN FOODS

Nowadays the UE has not yet introduced regulations concerning TFAs levels in foods. Denmark as the first country recommended restriction on and phasing out of the use of industrially produced TFAs in food. Since 2003 in Denmark the levels of *trans* isomers in food have been regulated by appropriate regulations, according to which industrially produced TFAs, those from partially hydrogenated oils, should be limited to 2% of the total amount of fat or oil in food. On the other hand, the products which do not contain more than 1% of TFAs should be labelled as "TFAs free". According to this directive, CLAs are not included to the total TFAs content [Stender & Dyerberg, 2003].

Another method of reducing TFAs intake is labeling foods with information about the *trans* isomers content, which as a result will enable consumers to choose products with low TFAs content. The first country which applied such a solution was Canada [Morin, 2005]. The obligation of labelling processed and packaged food has come into force on the 1st January 2003 and it applies to small portions of products in which TFAs level is not higher than 0.2 g. Following the Canadian example, the USA have introduced similar obligation, according to which TFAs level should be no higher than 0.5 g per serving. TFAs must be declared in the nutrition label of conventional foods and dietary supplements on a separate line immediately under the line for the declaration of SFA. Food and Drug Administration (FDA) has decided not to separate between industrially produced TFAs and TFAs of ruminant origin. Consequently, dairy products will be labeled with content of TFAs [Yurawecz, 2004; FDA, 2003; Satchithanandam *et al*., 2004].

On the 20th of June 2007, the Minister of Health announced that Health Canada adopted the recommendations of the *Trans* Fat Task Force with respect to the amount of *trans* fat in foods. These recommendations from the *Trans* Fat Task Force included two major objectives:

1. to limit the *trans* fat content of vegetable oils and soft spreadable margarines to 2% of the total fat content; and

2. to limit the *trans* fat content for all other foods to 5% of the total fat content, including ingredients sold to restaurants.

The Minister called on the food industry to achieve these limits within two years. The Minister also announced that if significant progress has not been made in the next two years, Health Canada will develop regulations to ensure that the recommended levels are met. In doing so, companies and food manufacturers are encouraged to replace *trans* fats with healthier alternatives such as monounsaturated and polyunsaturated fats and to not replace *trans* fats with SFA.

To ensure that the industry is making progress in meeting the 2% and 5% limit of the total fat, Health Canada will closely monitor the actions of the industry *via* the *Trans* Fat Monitoring Program. Canada is the first country to publish this type of monitoring data.

In contrast to Denmark and Canada, some countries, such as the Netherlands, have opted against government regulations, yet have made significant progress in reducing TFAs in the food supply [Health Canada, 2008].

In Australia the government is currently not regulating TFAs in the food supply, although it has issued a statement

of support for the recently established National Collaboration on *Trans* Fats which will propose initiatives aimed at reducing the amount of TFAs in food sold in Australia.

In January 2007 the European Parliament issued Regulations no 1924/2006 "On nutrition and health claims made on food", which have come into force in July 2007.

According to these Regulations, manufactures may declare in the nutrition label that the products are low in saturated fatty acids on condition that the total SFA and TFAs content does not exceed 1.5 g/100 g of solid product or 0.75 g/100 mL of liquid, and in both cases TFAs and SFA do not make more than 10% of energy intake.

A claim that a food does not contain SFA, and any claim likely to have the same meaning for the consumer, may only be made where the sum of SFA and *trans*-fatty acids does not exceed 0.1 g of SFA per 100 g or 100 mL [Regulation…, 2006].

CONCLUSIONS

The presented characteristic shows that TFAs are still a problematic matter. TFAs have been constantly criticized since the sixties. A constant discussion on the nutritional role of TFA has contributed to the fact that several countries have introduced labelling of the content of TFAs in food products (USA) or issued regulations on reducing TFAs content (Denmark). Countries which have not adopted any recommendations, yet have made significant progress in reducing TFAs in the food supply. As a result, a decrease in TFAs intake has been observed. Food and nutrition organisations recommend that TFAs consumption by all population groups should be kept as low as possible, which is about 1% of energy intake or less.

ACKNOWLEDGEMENTS

This work was carried out with the financial support from the Polish Ministry of Science and Higher Education under research grant No. N N 312 200035.

REFERENCES

1. Alonso L., Fraga M.J., Juarez M., Determination of *trans* fatty acids and profiles in margarines marketed in Spain. J. Am. Oil Chem. Soc., 2000, 77, 131–136.
2. Aro A., Complexity of issue of dietary *trans* fatty acids. Lancet, 2001, 357, 732–733.
3. Bakker N., van't Veer P., Zock P.L., The study group: Adipose fatty acids and cancers of the breast, prostate and colon: An ecological study. Int. J. Cancer, 1997, 72, 587–597.
4. Balas J., Fatty acids in food products – oils, margarines, butter, confectionary products, fast food and another. Żyw. Czł. Metab., 2004, 31, 2, 181–192 (in Polish; English abstract).
5. Bartnikowska E., Obiedziński M., Grześkiewicz S., The role of CLA. Przem. Spoż., 1999, 53, 7, 16–18 (in Polish).
6. Baryłko-Pikielna N., Osucha A., Nutritional role of *trans* fatty acids. Przem. Spoż., 1990, 44, 7, 162–163 (in Polish).
7. Belury M., Non all *trans*-fatty acids are alike: What consumers may lose we oversimplify nutrition facts. J. Am. Diet. Assoc., 2002, 102, 1606–1607.
8. Bray G.A., Lovejoy J.C., Smith Sr., DeLany J.P., Lefevre M., Hwang D., Ryan D.H., York D.A., The influence of different fats and fatty acids on obesity, insulin resistance and inflammation. J. Nutr., 2002, 132, 2488–2491.
9. Brooker C.S., Mann J.I., *Trans* fatty acids and cardiovascular health: translation of the evidence base. Nutr. Metab. Card. Dis., 2008, 18, 448–456.
10. Chardigny J., M., Clouet P., Combe N., Quignard-Boulangé A., Schmidt B., Lagarde M., Léger C.L., Metabolism of trans and conjugated fatty acids. Eur. J. Lipid Sci. Technol., 2007, 109, 930–934.
11. Corl B.A., Baumgard J.M., Griinari P., Delmonte K.M., Morehouse, M.P. Yurawecz D.E. Bauman: *Trans*-7, cis -9 CLA is synthesized endogenously by 9-desaturase in daily cows. Lipids, 2002, 37, 681–688.
12. Corl B.A., Babano D.E., Bauman C., *Cis*-9, trans-11 CLA derived endogenously from trans-11 18:1 reduces cancer risk in rats. J. Nutr., 2003, 33, 2893–2900.
13. Craig-Schmidt M.C., Word-wide consumption of *trans* fatty acids. Atherosclerosis Suppl., 2006, 7, 1–4.
14. Craig-Schmidt M.C., Worldwide consumption of *trans*-fatty acids. 1999, Abstracts of 23rd World Congress and Exhibition of the International Society for Fat Research (ISF), Brighton 3–7 October 1999, p. 50.
15. Daniewski M., Jacórzyński B., Mielniczuk E., Pawlicka M., Balas J., Fatty acids in Polish margarines in produced in years 1996 and 1997. Przem. Spoż., 1998, 52, 57 (in Polish; English abstract).
16. De Roos N.M., Schouten E.G., Katan M.B., Consumption of a solid fat rich in lauric acid results in a more favourable serum lipid profile in healthy men and women than consumption of a solid fat rich in *trans*-fatty acids. J. Nutr. Hum. Nutr. Metabol., 2001, 242–245.
17. Devinat G., Scamaroni L., Naudet M., Isomérisation de l'acide linolénique durant la désodorisaton des huiles de colza et de soja. Rev. Franc Corps Gras., 1980, 27, 283–287.
18. Dlouhy P., Tvrzická E., Staòková B., Vecka M., Žak A., Straka Z., Fanta J., Pachl J., Kubisová D., Rambousková J., Bilková D., Andel M., Higher content of 18:1 *trans* fatty acids in subcutaneous fat of persons with coronorographically documented atherosclerosis of the coronary arteries. Ann. Nutr. Metab., 2003, 47, 302–305.
19. Drozdowski B., Lipidy. Food Chemistry (red. Sikorski Z.E.). 2002, WNT, Warszawa (in Polish).
20. FDA, Food labeling: *trans* fatty acids in nutrition labeling, nutrient content claims, and health claims. Federal Register, 2003, 68, 41434–41506.
21. EFSA, opinion of the scientific panel on dietetic products, nutrition and allergies on a request from the commission related the presence of trans fatty acids in foods and the effect on human health of the consumption of trans fatty acids. EFSA J., 2004, 81, 1–49.
22. Feldman E.B., Kris–Etherton P.M., Kritchevsky D., Lichtenstein A.H., Position paper on *trans* fatty acids. Am. J. Clin. Nutr., 1996, 63, 663–670.
23. Fritsche S., Rumsey T. S., Yurawecz M. P., Ku Y., Fritsche J., Influence of growth promoting implants on fatty composition including conjugated linoleic acid isomers in beef fat. Eur. Food Res. Technol., 2001, 212, 621–629.

24. Gebauer S.K, Psota T.L., Kris-Etherton P.M., The diversity of health effects of individual *trans* fatty acid isomers. Lipids, 2007, 42, 787–799.
25. Gerber M., Razanamaheta L., Bougnoux P., *Trans* fatty acids and cancers: AFSSA recommendations. Eur. J. Lipid Sci. Technol., 2007, 109, 954–959.
26. Greyt W., Radanyi O., Kellens M., Huyghebaert A., Contribution of *trans*-fatty acids from vegetable oils and margarines to the Belgian diet. Fat, 1996, 98, 30–33.
27. Guillén M.D., Cabo N., Infrared spectroscopy in the study of edible oils and fats. J. Sci. Food Agric., 1997, 75, 1–11.
28. Harnack L., Lee S., Schakel S.F., Trends in the *trans* – fatty acid composition of the diet in a metropolitan area: The Minnesota Heart Survey. J. Am. Diet Assoc., 2003, 103, 1160–1166.
29. Health Canada, 2008, [www.hc-sc.gc.ca./fn-an/nutrition/gras-trans-fats/tfa-age_question-eng.php].
30. Holmes M.D., Hunter D.J., Colditz G.A., Stampfer M.J., Hankonson S.E., Speizer F.E., Rosner B., Willet W.C., Association of dietary intake of fat and fatty acids with risk of breast cancer. J. Am. Med. Assoc., 1999, 281, 914–920.
31. Hulshof K.F., Van Erp-Baart M.A., Anttolainen M., Becker W., Church S.M., Court C., Intake of fatty acids in Western Europe with emphasis on trans fatty acids: The TRANSFAIR study. Eur. J Clin. Nutr., 1999, 53, 143–157.
32. Hunter J.E., Applewhite T.H., Reassessment of trans fatty acids availability in the US diet. Am. J. Clin. Nutr., 1991, 54, 363–369.
33. Hunter J.E., Dietary levels of *trans*-fatty acids: basis for health concerns and industry efforts to limit use. Nutr. Res., 2005, 25, 499–513.
34. Innis S.M., Trans fatty intakes during pregnancy, infancy and early childhood. Atherosclerosis. Suppl., 2006, 7, 17–20.
35. Juttelstad A., The marketing of *trans* fat-free foods. Food Technol., 2004, 58, 1, 20–22.
36. Kohlemaister L., Simonsen N., Van't V.P., Strain J.J., Martin-Moreno J.M., Margolin B., Hutterunen J.K., Fernandez-Crehuet Navajas J., Martin B.C., Thamm M., Kardinaal A.F.M., Kok F.J., Adipose tissue *trans* fatty acids and breas cancer in European Community Multicenter Study on Antioxidants, Myocardial Infraction, and Breast cancer, Cancer Epid, Biom. Prev., 1997, 6, 705–710
37. Koletzko B., Desci T., Metabolic aspects of *trans* fatty acids. Clin. Nutr., 1997, 16, 229–237.
38. Kraft J., Hanske L., Mockel P., Zimmermann S., Hartl A., Kramer J.K, Jahreis G., The conversion efficiency of trans-11 and trans-12 18:1 by Delta-9 – desaturation differ in rats. J. Nutr., 2006, 136, 1209–1214.
39. Kritchevsky D., The effects of dietary *trans* fatty acids. Chem. Industry, 1996, 15, 565–567.
40. Krygier K, Żbikowska A., Rutkowska J., The influence of trans fatty acids content on the quality of puff-pastry cakes. 2005, in: Conference Materials of the 3rd World Congress and Exhibition of the International Society for Fat Research (ISF): Modern Aspects of Fats and Oils. 25–28 September 2005 Prague, Czech Republic. p. 75.
41. Krygier K., Żbikowska A., The influence of trans fatty acids content on sponge – cake quality. 2004, *in*: Conference Materials of the 3rd World Congress and Exhibition of the International Society for Fat Research (ISF): Modern Aspects of Fats and Oils. 25–28 September 2004 Edinburgh, England, p. 75.
42. Ledoux M., Juanèda P., Sèbedio J.L, Trans fatty acids: definition and occurrence in foods. Eur. J. Lipid. Sci. Technol., 2007, 109, 891–900.
43. Laloux L., du Chaffaut L., Rayanamahefa L., Lafaz L., Trans fatty acids content of foods intake levels in France. Eur. J. Lipid Sci. Technol., 2007, 109, 918–929.
44. Larqué E., Salvador Z., Gilb A., Dietary *trans* fatty acids in early life: a review. Early Hum. Devel., 2001, Suppl., 31–41.
45. Léger C.L., Razanamahefa L., Margaritis I., Health risks and benefits of *trans* fatty acids including conjugated fatty acids in food – Synopsis of the AFSSA report and recommendations. Eur. J. Lipid Sci. Technol., 2007, 109, 887–890.
46. Liu W.H., Inbaraj B.S., Chen B.H., Analysis and formation of *trans* fatty acids in hydrogenated soybean oil during heating. Food Chem., 207, 104, 1740–1749.
47. McDonald R.E., Min D.B., Food Lipids and Health. 1996, Marcel Dekker, New York, p. 164.
48. McKelvey W., Greenland S., Chen M-J., Longnecker MP., Frankl HD., Lee ER., Haile RW., A case-control study of colorectal adenomatous polyps and consumption of foods containing partially hydrogenated oils. Cancer Epid. Biom. Prev., 1999, 8, 519–524.
49. Meyer K.A., Hushi L.H., Jacobs D.R., Folsom A.R., Dietary fat and influence of type 2 diabetes in older women. Diab. Care, 2001, 24, 1528–1535.
50. Mojska H., Is there a need for labelling food products with the content of *trans* isomers of fatty acids? Przem. Spoż., 2006, 11, 38–41 (in Polish; English abstract).
51. Morin O., Trans fatty acids: New developments. OCL. Oléagineux, Corps Gras, Lipides, 2005, 12, 414–421.
52. Müller H., Jordal O., Kierulf P., Kirkhus B., Pedersen J.I., Replacement of partially hydrogenated soybean oil by palm oil in margarine without unfavorable effects on serum lipoproteins. Lipids, 1998, 33, 879–887.
53. Niewiadomski H., Technologia tłuszczów jadalnych. 1993, WNT Warszawa (in Polish).
54. Oomen M.C., Ockë M.C., Feskens E.J.M., Erp-Baart M.A., Kok F.J., Kromhout D., Association between *trans* fatty acids intake and 10-year risk of coronary heart disease in the Zutphen Elderly Study: a prospective population-based study. The Lancet, 2001, 357, 746–751.
55. Ovesen L., Leth T., Hansen K., Fatty acid composition and contents of *trans* monounsaturated fatty acids in frying fat and margarines and shortenings marketed in Denmark. J. Am. Oil. Chem. Soc., 1998, 75, 1079–1083.
56. Parcerisa J., Codony R., Boatella J., Rafecas M., Fatty acids including *trans* content of commercial bakery products manufactured in Spain. J. Agric. Food Chem., 1999, 47, 2040–2043.
57. Pariza M.W., Hargreaves W.A., A beef-derived mutagenesis modulator inhibits initiation of mouse epidermal tumours by 7,12-dimethylbenz[*a*]anthracene. Carcinogenesis, 1985, 6, 591–593.
58. Pelletier G., Hollywood R., Bacler S., Leyte D., *Trans* fatty acids in Canadian margarine: recent trends. J. Am. Oil Chem. Soc., 1998, 75, 1587–1594.
59. Rainer L., Heiss C.J., Conjugated linoleic acid: health implications and effected on body composition. J. Am. Diet Assoc., 2004, 104, 963–968.
60. Ratnayake W.M.N., Pelletier G., Hollywood R., Bacler S., Leyte D., *Trans* fatty acids in Canadian margarines: recent trends. J. Am. Oil Chem. Soc., 1998, 75, 1587–1594.

61. Risérus U., Jovinge S., Casu S., Supplementation with conjugated linoleic acid causes isomer-dependent oxidative stress and elevated C-reactive protein: a potential link to fatty acids induced insulin resistance. Circulation, 2002, 106, 1925–1929.
62. Risérus Regulation (EC) No 1924/2006 of the European Parliament and of the Council, 20 December 2006: "On nutrition and health claims made on foods". *Trans* fatty acids and insulin resistance. Atherosclerosis Suppl., 2006, 7, 37–39.
63. Satchithanandam S., Oles C.J., Spease C.J., Brandt M.M., Yurawecz M.P., Rader J.I., Trans, saturated, and unsaturated fat in foods in US prior to mandatory trans – fat labeling. Lipids, 2004, 39, 11–18.
64. Slattery M.L., Benson J., Ma K.N., Schaffer D., Potter J.D., *Trans* fatty acids and cancer. Nutr. Cancer, 2001, 39, 170–175.
65. Stanley J., Not all *trans* fatty acids have the same physiological effects. Lipid Technol., 2004, 16, 255–257.
66. Steinhart H., Pfalzgraf A., Die Anteile *trans* – Fettsäuren in Lebensmittel. Ernahrungswiss, 1992, 31, 196–204.
67. Stender S., Dyerberg J., Astrup A., High levels of industrially produced trans fat in popular fast foods, New Eng. J. Med., 2006, 354, 1650–1651.
68. Stender S., Dyerberg J., Influence of *trans* fatty acids on health. Ann. Nut. Metab., 2004, 8, 61–66.
69. Stender S., Dyerberg J., The influence of *trans* fatty acids on health. A report of Danish Nutrition Council, 2003, p. 34.
70. Sundram K., French M.A., Clandinin M.T., Exchanging partially hydrogenated fat for palmitic acid in the diet increases LDL-cholesterol and endogenous cholesterol synthesis in normocholesterolemic women. Eur. J. Nutr., 2003, 42, 188–194.
71. Turini M., The impact of conjugated linoleic acid on health. 1999, *in*: Abstracts of 23rd World Congress and Exhibition of the International Society for Fat Research (ISF), Brighton 3–7 October 1999, p. 51.
72. Van Dam R.M, Willett W.C., Rimm E.B., Stampfer M.J., Hu F.B., Dietary fat meat intake in relation to risk of type 2 diabetes in men. Diab. Care, 2001, 25, 417–424.
73. Van Duijn G., Technical aspects of *trans* reduction in margarines. Oléagineux Corps Gras Lipides, 2000, 7, 95–98.
74. Van Poppel G., Intake of trans fatty acids in western Europe; the TRANSFAIR study. Lancet, 1998, 351, 1099.
75. Verschuren P.M., Zevenbergen J.L., Safety evaluation of hydrogenated oils. Food Chem. Toxicol., 1990, 28, 755–757.
76. Wagner K.H, Plasser E., Proell Ch., Kanzler S., Comprehensive studies on the trans fatty acid content of Austrian foods: Convenience products, fast food and fats. Food Chem., 2008, 108, 1054–1060.
77. Wahle Klaus W.J., Heys S.D., Rotondo D., Conjugated linoleic acids: are they beneficial or detrimental to health? Prog. Lipid Res., 2004, 43, 553–587.
78. Weggemans R.M., Rudrum M., Trautwein E.A., Intake of ruminant versus industrial *trans* fatty acids and risk of coronary heart disease-what is the evidence? Eur. J. Lipid Sci. Technol., 2004, 106, 390–397.
79. Wolff R.L., Heat-induced geometrical isomerization of linoleic acid: Effect of temperature and heating time on the appearance of individual isomers. J. Am. Oil Chem. Soc., 1993, 70, 425–430.
80. Wolff R.I., Content and distribution of trans – 18:1 acids in ruminant milk and meat fats, their importance in European diets and their effect on human milk. J. Am. Oil Chem. Soc., 1995, 72, 259–271.
81. Yu L., Free radical scavenging properties of conjugated linoleic acids. J. Agric. Food Chem., 2001, 49, 3452–3456.
82. Yurawecz M.P., FDA requires mandatory labelling of *trans* fat. Inform, 2004, 15, 184.
83. Żbikowska A, Krygier K., The role of *trans* fatty acids in food. 2005, *in:* Materials of the XIII International Scientific Conference for Vegetable Fat Research, Szczyrk, Poland, pp. 48–53 (in Polish; English abstract).
84. Żbikowska A., Krygier K., The influence of *trans* isomers on the quality of cakes. Żywność, Nauka, Technologia, Jakość, 2003, 2, 35 suppl. 207–216 (in Polish; English abstract).
85. Żbikowska A., Rutkowska J., Krygier K., Effects of trans-isomer content in fats on the quality of puff-pastry cakes. 2005, *in*: Culinary Arts and Science V. Global and National Perspectives (eds. J.S.A. Edwards, B. Kowrygo, K. Rejman). Bournemouth University and Warsaw Agricultural University, Wyd. SGGW, pp. 279–291.
86. Żbikowska A., Wirkowska M., Kaźmierczyk M., Quality of margarines marketed in Warsaw. Tłuszcze Jadalne, 2007, 42 (in Polish; English abstract).

Received January 2009. Revision received and accepted March 2010.