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REVISIÓN

Stearic acid: a possible substitute for *trans* fatty acids from industrial origin

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RESUMEN

El acido esteárico: un posible sustituto para los ácidos grasos *trans* de origen industrial.

Los isómeros *trans* que contienen los aceites parcialmente hidrogenados de origen industrial, han sido cuestionados y la recomendación es reducir su consumo. La industria de alimentos se enfrenta a un dilema, ya que para disminuir los isómeros *trans* debe reducir los aceites parcialmente hidrogenados y reemplazarlos por grasas ricas en ácidos grasos saturados. La investigación ha demostrado que los ácidos grasos saturados tienen efecto negativo en los lípidos plasmáticos y su consumo se asocia con un alto riesgo cardiovascular, por lo cual la recomendación es reducir el consumo de estos ácidos grasos. Sin embargo, no todos los ácidos grasos saturados se comportan de la misma forma, siendo el ácido esteárico (18:0) la excepción. El ácido esteárico presenta bajo nivel de absorción intestinal y no modifica negativamente los lípidos plasmáticos, por lo cual se considera como "neutro" para la salud cardiovascular. Los niveles plasmáticos de la apoproteína B-100, que determina las concentraciones de las VLDL y LDL (transportadoras de triglicéridos y colesterol, respectivamente) no son modificados por dietas que aportan hasta 7% de la energía como ácido esteárico. Marcadores de riesgo cardiovascular, como la activación de factores de agregación plaquetaria o los niveles de la proteína C reactiva, no son modificados por dietas que aportan ácido esteárico, como ocurre con otros ácidos grasos saturados. La confirmación del efecto "neutro" del ácido esteárico es una perspectiva para el desarrollo de grasas con alto contenido de este ácido graso para reemplazar las grasas hidrogenadas que contienen isómeros *trans*. Esta revisión discute estos aspectos.

PALABRAS-CLAVE: Ácido esteárico – Alternativa a las grasas hidrogenadas – Efecto metabólico neutro – Isómeros trans – Salud cardiovascular.

SUMMARY

Stearic acid: a possible substitute for *trans* fatty acids from industrial origin.

Trans isomers, contained in partially hydrogenated oils, which are used in the food industry, have been questioned and nowadays trends are heading towards reducing their consumption. The food industry is facing a dilemma, since in order to remove *trans* fatty acids, hydrogenated fats should be eliminated and replaced by fats rich in saturated fatty acids. Scientific research has shown that saturated fatty acids have negative effects on the lipid profile and its consumption is associated with a higher cardiovascular risk. Therefore it is recommended to avoid their consumption. Nevertheless, not all fatty acids behave in the same way, with stearic acid (18:0) the exception. Stearic acid has a low level of intestinal absorption and its intake does not negatively modify the lipid profile. For this reason, it is considered a "neutral" fatty acid with regard to cardiovascular health. B-100 apolipoprotein, whose levels determine plasma VLDL and LDL concentration (triglycerides and cholesterol carriers, respectively), is not modified by diets which provide up to 7% of the energy as stearic acid. Markers of cardiovascular risk, such as activation of platelet aggregation factors or C-reactive protein levels, are not modified by diets providing stearic acid, as occurs with other saturated fatty acids. The confirmation of the "neutral" effect of stearic acid represents a perspective for the development of fats with high contents of this fatty acid to replace hydrogenated fats containing *trans* isomers. The present review discusses these aspects.

KEY-WORDS: *Alternatives to hydrogenated fats – Cardiovascular health – "Neutral" metabolic effect – Stearic acid –* trans *Isomers.*

1. INTRODUCTION

The dietary consumption of fatty acids with *trans* isomerism (TFA) has been called into question by health and food authorities due to their adverse effects on overall health and mainly on cardiovascular health derived from their consumption (Ascherio *et al.*, 1996; Hu *et al.*, 1997; Hunter, 2006; Mozaffarian *et al.*, 2009). Several epidemiological and clinical studies have unmistakably demonstrated that TFA acids produce an increase in plasmatic total cholesterol levels, in LDL-cholesterol levels (bad cholesterol) and in total triglycerides (Kris-Etherton *et al.*, 2005; Mozaffarian *et al.*, 2006; Hunter, 2006).

It has been also demonstrated that TFA reduce HDL-cholesterol levels (good cholesterol) (Hu *et al*., 1997). There is also evidence that TFA can increase type 2 diabetes complications (Salmeron *et al*., 2001; Tanasescu *et al*., 2004; Saravanan *et al*., 2005). These effects have a negative incidence on health, for they increase in the risk of cardiovascular diseases, which are the main reason for death in the population of the western world. Nowadays, it is considered that the TFA negative effect on health can be at least compared with the effect of saturated fatty acids (SAFA) (Sundran *et al*., 1997; Hu *et al*., 1997; Valenzuela & Morgado., 1999; Judd *et al*., 2002; Hunter, 2006). According to epidemiological studies and in order to avoid a possible incidence of cardiovascular risk, health authorities have proposed to limit TFA consumption to 2% of the total energy supply (US FDA, 2003; Uauy *et al*., 2009).

Different countries have adopted policies aiming at a drastic reduction in TFA consumption in their populations (Stender *et al*., 2006). Since June 1st 2003, Denmark has forbidden any content higher than 2% of TFA in both locally produced and imported fats hence the use of partially hydrogenated fats has been essentially eliminated from this country (Leth *et al*., 2005). Countries such as Norway, Finland, and the Netherlands have similar initiatives (Aro, 2005). Since 2003, Canada has become the first country in America to regulate the compulsory labelling of TFA and in 2006 it was proposed that TFA should not exceed 2% of the total fat content in vegetable oils and soft margarines for spread, and 5% of the total fat content in other foods. The United States introduced the compulsory labelling of TFA in 2006 (Eckel *et al*., 2007), an initiative that was followed by many other countries in Latin America, mainly MERCOSUR countries (Argentine, Brazil, Paraguay and Uruguay), and Chile (Valenzuela, 2008). Important fast food chains have withdrawn TFA from their products in different Latin American countries (Argentina, Brazil, Chile, Uruguay) (Valenzuela, 2008a). Most recently, the state of New York, in the United States, banned the use of hydrogenated products containing TFA in fast food restaurants. In response to these requirements, different industrial and fast food companies have announced the removal or future elimination of fats containing TFA in their products (Korver & Katan, 2006; Mozaffarian & Clarke, 2009).

In recent years, The World Health Organization (WHO) has held three meetings of Scientific Update on the elimination of TFA: PAHO (Pan American Health Organization) meeting, Washington DC, USA, August 2007; PAHO meeting, Washington, DC, USA, November 2007; and WHO meeting, in Geneva, Italy, February 2008. Their findings indicate that the replacement of TFA in partially hydrogenated vegetable oil with alternative fats and oils would substantially lower cardiovascular disease risk through multiple mechanisms beyond those on cholesterol-lipoprotein fractions (Kris-Etherton *et al*., 2005), thus explaining in part the difference derived from estimates based on controlled dietary interventions focusing mainly on serum cholesterol fractions versus prospective cohort studies having cardiovascular disease events as their main outcome (Hunter *et al*., 2010). It is important to highlight that in Europe, the starting point was in the early 1990s, when the TFA reduction in certain margarines was implemented by means of suppressing partially hydrogenated fats (Morin, 2007). This situation has been envied by Americans and in 1994 Harvard researchers, lead by epidemiologist Walter Willett, encouraged people to join the European initiative (Ascherio *et al*., 1996). Willett´s group urged Americans to stop eating TFA and encouraged the Food and Drug Administration (FDA) to add them to food labels, a step the agency considered (Ascherio *et al*., 1996). The same researchers also urged companies to follow Europe's lead in improving hydrogenation and producing margarine without TFA (Hu *et al*., 1997). Most recent food surveys pointed out that TFA consumption had effectively decreased in several European countries mainly due to the reformulation of several food products, for example spreads (Korver & Katan, 2006).

2. TFA ISOMERS ORIGIN AND CONSUMPTION

TFA isomers have two main origins: biological and technological (Valenzuela & Morgado, 1999). Biological ones come from products derived from ruminant animals (beef, beef tallow, milk and its derived products), and they do not involve more than 5-10% of the total consumption of TFA isomers in European and American countries (Larqué *et al*., 2001). Therefore, in these countries TFA intake comes essentially from technological sources, (90-95%) mainly hydrogenated fats, frying processes and to a lesser degree from edible oils which are treated with a deodorization process (Larqué *et al*., 2001; Craig-Schmidt, 2006). Current guidelines recommend an intake lower than 1% of the energy as TFA (Eckel *et al*., 2007). In the United States current consumption is around 2.5-3% of the total energy, meaning 5.8-6.0g/day (Allison *et al*., 1999), although it might reach 10g/day or more in some segments of the population. Consumption in Latin America varies remarkably from country to country, but the average is around 4.5-5.0 g/day (Valenzuela, 2008a). Consumption in European countries varies depending on the country as well, but it is generally lower than in American countries, ranging from 1.4 to 5.0g/day in a remarkable decreasing rate from North to South (higher in the North than in the South, eg: Netherlands vs Spain) (Kromhout *et al*., 1995; Hutshof *et al*., 1999). The TFA source is completely different in Europe. A great deal of TFA fat has animal origin (40 to 60% vs. 5 to 10% in the US), also with a significant decreasing rate from North to South. Animal products are the main

source with 60%; dairy products around 50% (butter 35% and cheese 17%) and ruminants beef 10%. Biscuits, pastry, industrial bakery and cooked dishes come next with 30-40% (Husthof *et al*., 1999). An important issue to highlight is that the information gathered from countries that recently joined the European Union shows that Eastern European countries seem to have a much higher consumption than in Western Europe.

From the food industry point of view, it is very difficult to reduce the use of hydrogenated fats, the main source of TFA isomers, since these fats are essential for the manufacturing of several food products. Hydrogenated fats work as a base for adding other nutrients, they have thermal stability, they provide palatability and crispy characteristics to products, etc (Valenzuela & Morgado, 1999; Korver & Katan, 2006). In this way, it is currently a challenge for the food industry to substitute TFA present in their products without altering organoleptic characteristics such as appearance and stability. Within the few available alternatives to this possible replacement, the ones most commonly found are mainly oils with low polyunsaturated fatty acid contents, or with high SAFA contents (palm oil, high oleic sunflower oil, low linoleic soybean oil, etc.) (Tarragó-Trani *et al*., 2006). However, nowadays, stearic acid (C18:0) (STA) turns out to be a recent alternative with great significance (Hunter *et al*., 2010). How could an SAFA replace TFA isomers? The purpose of this review is to analyze the evidence of the neutral effects of STA in lipid and vascular parameters which constitute the markers of cardiovascular disease risk, and to state the reason why this fatty acid contained in fats can constitute a reasonable alternative for the substitution of TFA in our diet.

3. STA, A "DIFFERENT" FATTY ACID

STA is a saturated fatty acid present in fats of both animal and vegetal origin. Following palmitic acid (C16:0), it is the most widely consumed fatty acid in the United States as well as in the Western population in general (Ervin *et al*., 2004). Palmitic acid (16:0) amounts to 56% of the total consumption and stearic acid 26%, approximately. The rest of the SFA consumption consists of myristic (C14:0), and lauric acid (C12:0), and in lower quantities butyric (C4:0), capric (C10:0), caproic (C6:0), and caprylic acid (C8:0). STA is taken from animal fat (bovine, porcine, ovine and marine fish), and in lower amounts from vegetable fats (coconut oil, soybean oil, corn oil, cocoa butter, etc). Figure 1 shows the STA contents in different fats of regular consumption.

Fatty acid absorption into the human digestive system essentially depends on the position that fatty acids have in dietary triglycerides (Mu & Høy, 2004), which amount to 90-95% of our fat consumption. The rest of our intake consists of phospholipids (3.5-4%), and different sterols (1.5-2%), such as cholesterol, phytosterols, phytostanols, etc. (Carroll, 1958). In animal fats, SAFAs usually occupy *sn-1* and *sn-3* positions of triglycerides, and in a lower proportion they are found in the *sn-2* position (Bracco, 1994). In bovine, porcine and ovine fats, STA is much more frequently found in the *sn-1* and *sn-3*, than in the *sn-2* position. In vegetable fats, SAFAs, especially STAs, mainly occupy the *sn-1* and *sn-3* positions of triglycerides as well (Mattson & Volpenhein, 1964). The *sn-2* Position of these fats and oils is frequently occupied by unsaturated fatty acids, such as oleic acid (C18:1) and less frequently by linoleic (C18:2),

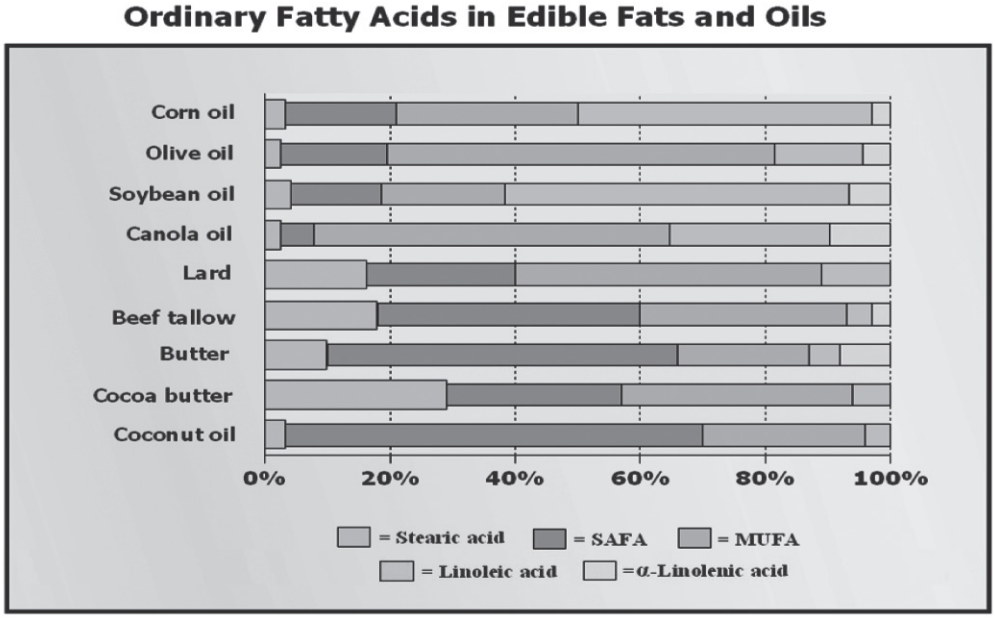

Figure 1
Common fatty acids in edible fats and oils. MUFA: monounsaturated fatty acids.
Modified from Kris-Etherton et al., 2005.

and alpha linolenic (C18:3) acids (Christie & Moore, 1972). This special stereoisomerism has an important impact on the absorption degree of each fatty acid in the human small intestine (Kubow, 1996; Hunter, 2001).

Human digestive lipases, both lingual, gastric, as well as pancreatic, have special specificity to hydrolyze the *sn-1* and *sn-3* positions of dietary triglycerides (Bracco, 1994; Hunter, 2001), in such a way that less than 20% of the triglycerides are totally hydrolyzed into free fatty acids and glycerol (Mu & Høy, 2004). In this way, the main products of triglyceride intestinal hydrolysis are monoglycerides with *sn-2* sterified fatty acids, and fatty acids released from the *sn-1* and *sn-3* positions. This is how STA, released from both animal and vegetal fats, will be present in the small intestine lumen, after the digestive process, primarily in a free form and secondarily as part of *sn-2* monoglycerides (Kritchevsky, 1994; Hunter, 2001). When STA is located in the *sn-2* position in the triglycerides it is well absorbed, but when found in the *sn-1* or *sn-3* positions it is only partially absorbed, ranging from 37 to 55% (Mattson *et al*., 1979).

4. STA DIGESTION AND ABSORPTION

Studies have demonstrated that, comparatively, STA is absorbed in a lower proportion than other SAFAs such as lauric, myristic and palmitic, and even MUFA, as oleic, or type C18:1 TFA isomers (Baer, 2003). The general conclusion is that STA is less absorbed than other dietary SAFAs, therefore its plasmatic concentration would be lower when compared with the ones obtained with similar amounts of other SAFAs (Kris-Etherton *et al*., 1997; Kris-Etherton *et al*., 2005). The reason for the reduction in the presence of STA at plasmatic level, compared with equivalent amounts of other SAFAs, is not yet fully known (German & Dillard, 2004). It has been postulated that due to its relatively high melting point (70°C), when released into the intestinal lumen under the effect of enzymatic hydrolysis, it would form calcium and/or magnesium insoluble salts, which would be eliminated through depositions (Mattson *et al*., 1979). Other researchers have also postulated that a certain percentage of absorbed STA would be turned into oleic acid through intestinal cell desaturation (Garg, 1992). This transformation has been estimated to be around 9% and 14% (Rhee *et al*., 1997). Whatever the reason might be, the point is that STA absorption is lower than that of SAFA with fewer carbon atoms (Kris-Etherton *et al*., 2005), particularly when found in *sn-1* and *sn-3* positions. Nevertheless, the lower absorption is not exclusive when STA takes up these triglyceride positions. SALATRIM is a structured lipid which contains long chain SAFAs, mainly STA, in *sn-1* and *sn-3* positions, and short chain fatty acids (acetic, propionic and/or butyric) in the *sn-2* position. For this reason, SALATRIM is a proved low digestibility fat and its consumption produces the elimination of high quantities of STA through depositions (Finley *et al*., 1994). This characteristic has made it a popular low calorie fat. However, when SALATRIM is previously randomized through interesterification, the molecule loses symmetry and thus leaves a significant proportion of STA in the *s-2* position. When the resulting products effect is tested, the fatty acid also shows low absorption (Berry & Sanders, 2005). Hence, it is suggested that regardless of the position STA occupies in triglycerides (*sn-1*, *sn-2* or *sn-3*), its intestinal absorption is always low (Berry & Sanders, 2005; Berry *et al*., 2007).

It has been proposed that STA would be a bad substrate for acyl-cholesterol-acyl-transferase enzyme (ACAT) which is responsible for cholesterol re-esterification in the intestine (Daumerie et al., 1992). If cholesterol is not re-esterified into intestine cells, it is again transported to the intestinal lumen and eliminated with faeces (Rodríguez-Meléndez *et al*., 2006). It has also been proposed that STA would enhance cholesterol hepatic excretion through bile (Imaizumi *et al*., 1993), although this increase does not occur experimentally in hamsters (Hassel *et al*., 1997). Moreover, it has been demonstrated that STA inhibits the expression of the Nieman-Pick C1 L1 transporter (NPC1L1) in the FH 74 cell line of intestinal cells (Hunter 2001). This transporter is responsible for carrying cholesterol from intestinal lumen to the enterocyte (Rodríguez-Meléndez *et al*., 2006). As a result of both mechanisms, lower cholesterol absorption at the intestinal level would be produced due to the effect of STA.

4.1. Effects on plasmatic lipids

Predictive equations for the differential effects of fatty acids on plasmatic lipids developed independently by Keys *et al*., (1965) and Hegsted *et al*., (1965) several decades ago, demonstrated the hypercholesterolemic effect of SAFA and its consequent increase in cardiovascular risk. However, when these equations were applied to STA, the fatty acid appeared with a “neutral” effect on plasmatic cholesterol levels. More complex mathematical equations, like the one developed by Mensink y Katan (1992), demonstrated the neutral effect of the fatty acid, not only on total cholesterol, but also on LDL- and HDL- cholesterol levels. Similar results were obtained by Yu *et al*. (1995), who developed new predictive formulae starting from regression studies, concluding that STA has a neutral effect on the lipid profile in men as well as in women. Moreover, when comparing its effect with lauric, palmitic or myristic acid, STA shows a neutral or slightly positive effect on plasmatic lipids (Hunter, 2001). A meta-analysis carried out by Mensink *et al*. (2003) on 60 controlled clinical trials compared the effect of SAFA on the modification of plasmatic lipids with respect to their substitution by carbohydrates in the diet. It was demonstrated that STA reduces cholesterol plasmatic levels and

the relationship between total cholesterol and HDL-cholesterol. The modification of both parameters is a positive marker for reduced cardiovascular risk. Similar studies have demonstrated that dietary STA, contributing from 9% to 40% of the total energy, does not modify total plasmatic cholesterol, and even in some cases it is reduced (Aro *et al*., 1997; Nestel *et al*., 1998; Snook *et al*., 1999). A more recent study by Thijssen & Mensink (2005), showed that diets with 7% of STA produce changes in the lipid profile similar to those yielded by a diet having an equal amount of oleic or linoleic acid. In this work, a detailed evaluation on the lipoprotein profile was carried out by Nuclear Magnetic Resonance (NMR), showing that the three fatty acids have similar effects on total and LDL-cholesterol reduction. Surprisingly for researchers, STA also increased HDL-cholesterol, an effect which has been previously observed only with oleic and linoleic acids. Another remarkable observation on Thijssen & Mensink's work (2005) is that STA does not modify LDL size, that is, it does not increase smaller sized LDLs, which are considered more atherogenic than larger sized LDLs (Gardner *et al*., 1996). The experimental protocol used by Thijssen & Mensink involved fats only from natural origin, where STA mainly occupies sn-1 and sn-3 positions. A more recent work by Sundran *et al* (2007), demonstrates that an interesterified fat, with a high proportion of STA (40%), negatively modifies the lipid profile, also increasing the plasmatic glucose levels. However, an analysis on the fatty acid triglyceride sterochemistry, shows that 15% of STA is in the *sn-2* position. This result emphasizes the concept that the beneficial effects derived from STA are obtained when it takes up *sn-1* and *sn-3* of triglyceride positions. An equal effect to other saturated fatty acids is observed when it is located in the *sn-2* position (Sundran *et al*., 2007).

Apolipoproteins make up the protein part of lipoproteins and enable the selective recognition of these proteins by different tissues. B-100 apolipoprotein (ApoB-100) is only present in VLDL and LDL which derive from VLDL. In this way, high levels of ApoB-100 are indicative of an active triglyceride (by VLDL) and cholesterol (by LDL) transport. On the contrary, low ApoB-100 plasmatic levels mean low VLDL and LDL concentrations. Two studies demonstrated that when STA provides either 9.3% or 36% of the energy, a reduction in ApoB-100 from 10% to 18% is produced (Tholstrup *et al*., 1994; Aro *et al*., 1997). However, the latter study also demonstrated that STA increases lipoprotein (a) plasmatic levels. This type of lipoprotein has similar characteristics to LDL, which is related to a higher atherogenicity and considered an emerging marker of cardiovascular disease (Clevidence *et al*., 1997). This effect has also been shown by a study on fasting (Tholstrup *et al*., 1995), and in post-prandial conditions (Tholstrup & Samman, 2004). The available data concerning the changes in lipoprotein (a) concentration with dietary TFA are limited, and further studies concerning a relationship between TFA and lipoprotein (a) are needed.

4.2. STA, activation of thrombogenic factors and blood pressure

The activation of thrombogenic factors due to post-prandial lipemia effect has been associated with an increase in cardiovascular risk. A meal rich in STA increases lipemia, but to lower levels than lipemia resulting from taking oleic and elaidic acids (Sanders *et al*., 2000), or palmitic acid (Mennen *et al*., 1998), probably due to the low absorption effect of STA already discussed. The increase in post-prandial lipemia raised the concentration of Factor VIIc activated form, a coagulation factor dependent on vitamin K. Fatty acids in general increase the activation of this Factor (Mitropoulos *et al*., 1994; Mennen *et al*., 1998). However, comparatively, STA produces a lower activation of this factor than oleic acid, as observed by Tholstrup *et al*. (1994) and later confirmed by Sanders *et al*. (2000); also lower than palmitic acid (Mennen *et al*., 1998).

The effect of STA on hemodynamics is still not clear and in some cases is controversial. Multiple intervention studies have demonstrated an inverse correlation between cholesterol plasmatic levels and STA, and the diastolic pressure measured in middle-aged individuals with high cardiovascular disease risk (Simon *et al*., 1996). However, when this measurement is made on healthy individuals, both plasmatic cholesterol as well as STA levels significantly increases left ventricular diastolic pressure (Steer *et al*., 2002). Controlled clinical trials demonstrate that diets with 8 to 13% of the total energy as STA, have no effect on vascular pressure measured in diabetic patients (Storm *et al*., 1997), or in men and women with vascular pressure at normal ranges (Zock *et al*., 1993). This information is important because it corresponds to studies with standard STA intakes.

Finally, there is little evidence on the effect of STA on molecular markers of inflammation, such as cytokines (interleukine-6, for example), adhesion molecules (such as selectins), or acute-phase expression proteins (such as C-reactive protein), which are important to predict cardiovascular disease (Blake & Ridker, 2002). STA does not modify plasmatic levels of these molecular markers if the diet imparts 11% of its energy as STA (Baer *et al*., 2004), although it does increase fibrinogen levels when taking in 11% of the energy as STA (Baer *et al*, 2004). Nevertheless, Kris-Eherton *et al* (2005) do not consider this last point relevant due to the fact that the average consumption STA is lower than 3% of the energy (Ervin *et al*., 2004). Thijssen *et al*. (2005) demonstrated that diets with 7% of the energy as STA, which is significantly higher than average consumption (3%), do not affect blood platelet aggregation. Therefore, researchers conclude that STA would not have thrombogenic effects, making it comparable to oleic or linoleic acid.

5. FINAL CONSIDERATIONS

There is a clear need to alert oil seed producers that there will likely be a requirement for an increased supply of substitute oils in order to replace TFA and that this represents an opportunity to expand or develop new oil seed varieties. The results of scientific updates should provide the evidence and scientific bases to promote discussions between the international scientific community related to nutrition and health as well as to agriculturalists and the food production industry, relevant health professionals, national and international food regulatory agencies, civil society, and the private sector in order to achieve this goal.

Available information, derived from experimental, clinical and epidemiological research, enables us to confirm with reasonable evidence, that when STA is consumed in less than 7% of the total energy, lipid profile, thrombotic factors, hemodynamic and cardiovascular risk molecular markers are not modified (Kris-Etherton *et al*., 2005; Hunter *et al*., 2010). This aspect distinguishes STA from other SAFAs present in diets, such as palmitic, lauric and myristic, while it ranks among MUFAs, such as oleic acid, or polyunsaturated acids, such as linoleic acid. The reason for this "neutral" effect is still unclear, and could be assigned to different factors: a lower absorption and higher excretion in the intestinal lumen when located in the *sn-1* and *sn-3* positions of dietary triglycerides; a partial conversion into oleic acid through intestine cell desaturation; to an inhibitory effect on ApoB-100 synthesis; or to other factors not yet identified. Everything focuses on the fact that fats with a high proportion of STA in the *sn-1* and *sn-3* positions could be very good substitutes for hydrogenated fats with high contents of TFA isomers. It should be pointed out that the neutral effect on cardiovascular diseases attributed to chocolate consumption would be due, in some degree, to a high proportion of STA in the *sn-1* and *sn-3* positions found in cocoa butter, together with its high cytoprotective flavonoids content (Ding *et al*., 2006). Oils with high STA content in the *sn-1* and *sn-3* triglyceride position for frying use, are high temperature resistant due to their saturated characteristics. These could be an adequate substitute for partially hydrogenated fats with high contents of TFA isomers, which are nowadays used in the food industry and mainly for frying processes in fast food chains (DiRienzo *et al*., 2008). In summary, we conclude that STA may be a reasonable substitute for TFA and also for cholesterol-raising SAFAs for solid fat applications, such as baked goods, shortenings, spreads, and margarines.

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