

Trans Fatty Acids, does Exist Safety Dosage?

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Abstract

Recent data regarding Trans Fatty Acids (TFAs) have implicated this lipid as being particularly deleterious to human health. TFAs are unsaturated fatty acids that contain at least one non conjugated double bond in the trans configuration, resulting in a more linear shape. The presence of a trans double bond in a fatty acid chain results in a smaller bond angle, or kink, than in a cis double bond, resulting in a fatty acid chain conformation that is more similar to a saturated fatty acid than to an unsaturated fatty acid. Several studies have identified an association between trans-fat intake and a risk of neurodegenerative diseases, cardiometabolic disease and pro-inflammatory effects. It focuses on a series of recent studies that have supporting the pathogenic role through human or animal experiments *in vitro* or *in vivo*. Overall, the findings of this short communication suggest that fat composition is more important than the quantity of fat consumed in terms of dietary *cis* and *trans* fatty acids.

Introduction

In the 1960s, following public health campaigns aimed at decreasing the use of animal fats, food industry began using substantial amounts of partially hydrogenated vegetable oils in processed food, in this way, initiated the *trans* fatty acid (TFA) consumption [1].

In course of the time, evidences has showed a rise consumption of partially hydrogenated lipids (enriched with TFA) which were relation to diverse comorbidities, cardiovascular diseases, systemic inflammation, dyslipidemias, endothelial dysfunction and more recently neurodegenerative diseases [2-4].

Independent important health concerns above, companies keep inserting TFA in manufactured products, due the following commercial advantages: (i) enhance their structure, lubrication, and texture (that is, consistency/hardness, springiness, brittleness, and chewiness); (ii) increase their shelf life; (iii) increase their flavor stability; (iv) decrease their food sensitivity to oxidation; (v) increase their stability against liquefaction; (vi) increase their stability during storage at room temperature; (vii) increase their stability during frying; and (viii) enhance their emulsion stability [5].

It is important to comment research from Otite et al. [6], which evaluated more than 230 brand-name food products (2007 to 2011) in US supermarkets, that contained TFA in the composition but were cited as “free TFA”, due the quantity, for serving, allowed by food legislation.

Chemical structure and hydrogenation

TFA are unsaturated fatty acids, with at least one double bond in trans conformation, generating in a fatty acyl chain conformation more linear, in comparison to the cis conformation, more similar to a saturated fatty acid than an unsaturated fatty acid [7].

During soybean oil hydrogenation (the mot used in this process), the oleic acid presents in this soybean oil, goes through geometrical

isomerisation during hydrogenation, becoming elaidic acid (EA), thus the “natural” oleic acid is *transformed* into elaidic acid during the hydrogenation process, and becomes an “unnatural” fatty acid [8]. EA is a monounsaturated fatty acid (MUFA) containing 18 carbons, with a double bond at position 9 in the trans configuration (9-*trans*-C18:1, 9-*trans*-octadecenoic acid) [9]. The overwhelming majority of double bonds in dietary fat occur in the cis conformation.

It is important to comment that, beyond the industrial hydrogenation process, that TFA are produced when the oil, liquid at room temperature, is converted in a solid lipid, thermal treatments as refining of vegetable oils (edible oils are refined to remove certain impurities/naturally present attributes) or during the process of domestic frying of vegetable oils (1500C–1900C or more, during this process studies have shown a loss of cis double bonds along with an increase in trans unsaturation) are capable to produce TFA [10].

Trans fatty acids: health risk

Nowadays there is a global consensus to decrease TFA consumption produced by partial hydrogenation of vegetable oils, these recommendations are based on numerous studies demonstrating many adverse health effects of TFA, regardless of the amount consumed [11].

A previous study by our group showed that dietary supplementation with Partially Hydrogenated Soybean Oil (PHSO) in animal models impaired inflammatory parameters in Cerebrospinal Fluid (CSF) and blood, induced insulin resistance, altered lipid profiles and caused hepatic damage In many analyses, there were no significant differences between the Low Partially Hydrogenated Soybean Oil (LPHSO) and High Partially Hydrogenated Soybean Oil (HPSO) diets [2].

In animal models Chen et al. [12] showed that a hyperlipidic diet with TFA exhibit atherosclerotic lesions and suppressed transforming growth factor- β (TGF- β), according to the author, a risk factor to atherosclerosis.

In humans Han et al. [13], in a double blind crossover study, 19 subjects consumed partially hydrogenated lipids for 32 days, the authors concluded that partially hydrogenated lipids increases production of inflammatory cytokines (TNF- α e IL-6), that have been associated with the pathophysiology of atherosclerosis. According to Spielman et al. [14], an excess in peripheral pro-inflammatory mediators, some of which can cross the blood brain barrier (BBB), may trigger neuroinflammation, subsequently exacerbating neurodegeneration.

In relation to hepatic tissue, Shao and Ford [15], showed that elaidic acid potently induced SRE, a family of transcription factors that regulate cholesterol and fatty acid biosynthesis, interesting to comment that oleic acid, *cis* analogous did not alter it. Our previous results indicated effects of the partially hydrogenated soybean oil diet on hepatic tissue: alterations in triglyceride levels, superoxide dismutase (SOD) activities and the ratio of SOD/Catalase (CAT)+Glutathione Peroxidase (GPx) in the low partially hydrogenated soybean oil diet [2].

Chàjes et al.[16] showed a trend of increased risk of weight gain during the 5-year follow-up with TFA consumption, their data suggest that a high intake of industrial *trans* fatty acids may decrease the risk of weight loss, particularly in women.

Manco et al. [17] mentions a relation to the high levels of circulating fatty acids with impaired in glucose transport and insulin sensibility, this theory is based in the accumulation of triglycerides in many non-hepatic tissues, inducing disorders to pancreatic activity. Corroborating to these findings, our data demonstrated that treating rats with the PHSO diet led to worse fasting glycaemia and serum insulin in both low and high concentrations, suggesting a possible inflammatory response to this lipid [2].

Trans fatty acids: actions in the central nervous system

There is growing evidence that lipid metabolism, oxidative stress and inflammatory mechanisms may also participate in the pathogenesis of AD [18]. Results have shown that CSF IgG antibodies to OxLDL are significantly increased in AD patients and in patients with frontotemporal lobar degeneration compared with controls [19]. Our group demonstrated that OxLDL results, specifically for the LPHSO diet, this concentration of lipids resulted in the same harmful effects in comparison to the hyperlipidic interventions (no significant difference) and showed the highest LDL antibody values with PHSO in both the low and high lipid concentrations, suggesting that the type of lipid is more important than its concentration and supporting a possible inflammatory component to the PHSO diet that is independent of concentration [2].

Research with approximately 3 years from the baseline interview, evaluated the effects of dietary fats on the development of Alzheimer disease (AD), the author concluded that the consumption of hydrogenated fats may increase risk to AD [20]. Corroborating to this data, Banard et al. [4], indicate relationships between trans fat intake with dementia and risk of cognitive disorders. *In vitro*, Grimm et al. [21], clearly show that TFA compared to *cis* fatty acids increase oligomerization and aggregation of A β , resulting in an increased production of amyloid beta (A β) peptides, main components of senile plaques, which are a characteristic neuropathological hallmark for Alzheimer's disease.

In a review about the relation to TGA and stroke during a median follow-up of 7 years, the authors identified association between TFA

intake and stroke, for every 2 g/d increase in TFA intake, there was a 14% increase in the risk of stroke in men [22].

Lastly, TFA are associated with all cause mortality, probably because of higher levels of intake of industrial trans fats, according to a refined review with 50 papers to de Souza et al. [23].

Conclusion

Although there are still doubts about TFA toxicity in the organism, the current references demonstrate that the TFA consumption impairs inflammatory and oxidative markers in both cerebrospinal fluid and plasma, which are risk factor for the most non-transmissible chronic diseases. Overall, these findings suggest that fat composition is more important than the quantity of fat consumed in terms of *cis* and *trans* fatty acid diets.

Conflict of Interest

The authors declare no conflict of interest.

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