POSITION PAPER ON INDUSTRIAL TRANS FATTY ACIDS (TFA)

The European Society of Cardiology (ESC) and the European Association for Cardiovascular Prevention & Rehabilitation (EACPR) look forward to the publication of the European Commission Reporting on Trans Fatty Acids due in December this year.

Trans fatty acids (TFA) are widely recognized as the most harmful fat with regard to causing cardiovascular disease and the detrimental effects of TFA on heart health and mortality are now beyond dispute. Cardiovascular disease (CVD) is the main cause of death in Europe with very significant differences in mortality rates between countries. Each year CVD causes over 4 million deaths in Europe and over 1.9 million deaths in the European Union (EU). CVD causes 47% of all deaths in Europe and 40% in the EU.

Based on the effects on cardiovascular health of TFA intake, the experiences from different interventions to limit TFA intake and the accumulating knowledge on differential consumption of TFA across Europe, the European Society of Cardiology believes that a regulatory intervention is necessary to ensure that all EU citizens can effectively minimise their TFA intake.

Legislative approaches to reduce industrially-produced TFA have proven effective in many countries. In the EU, although current policies and practices have reduced average TFA consumptions, policy should be guided by actual intake data of vulnerable groups and not just population averages. A key aspect is inequality in limitation of TFA intake: while mean intake declines, intake may remain substantial in subgroups. Without a regulatory intervention, inequality in CVD mortality linked to TFA intake is likely to increase in the EU.

The European Society of Cardiology and the European Association for Cardiovascular Prevention & Rehabilitation therefore recommend that the European Commission brings forward a proposal for an EU-wide regulation to eliminate industrially produced TFA in foodstuffs marketed in the EU. Action at the EU level will support the functioning of the internal market, while at the same time ensuring a high level of health protection for all.

The European Society of Cardiology (ESC) represents over 80,000 cardiology professionals across Europe and the Mediterranean. Its mission is “to reduce the burden of cardiovascular disease in Europe”. The ESC provides an array of scientific and educational activities, such as the production and continuous updating of Clinical Practice Guidelines, the organisation of educational courses and initiatives, pan-European surveys on specific disease areas. It also organises the ESC Congress, the largest medical meeting in Europe, as well as subspecialty congresses, in conjunction with its constituent bodies. The ESC edits and publishes 9 of the world’s leading journals on cardiology.

The European Association for Cardiovascular Prevention and Rehabilitation (EACPR) is a registered branch of the European Society of Cardiology. Its mission is “to prevent the incidence of cardiovascular (CV) disease, by implementing strategies to reduce the burden of CV risk factors and incidence of CV disease, and develop appropriate models for healthy CV lifestyle”.

To reduce the burden of cardiovascular disease in Europe

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TFA Effects on Cardiovascular Health

There is no known safe level of consumption of TFA. European Guidelines on Cardiovascular Disease (CVD) Prevention in Clinical Practice\(^6\) published jointly by the European Society of Cardiology and a number of key scientific communities in Europe, recommend that less than 1% of total energy intake should come from TFA and that industrially produced TFA should be eliminated.

Industrial TFA have no demonstrable health benefits, but considerable potential to harm. They are created when fish or vegetable oils are partially hydrogenated to convert large numbers of naturally occurring cis unsaturated double bonds into trans unsaturated double bonds. A high industrial TFA content provides physical and chemical properties that are attractive to food manufacturers, including the creation of relatively inexpensive solid or semi-solid fat. The process also destroys labile omega-3 acids and this reduces the propensity for fat to become rancid, increases shelf life and optimizes deep frying applications\(^7\).

The primary health concerns relating to TFA are an association between increasing TFA intakes, raised serum cholesterol levels and CVD deaths. Meta-analyses of controlled dietary trials evaluated the effect on serum lipids of isocaloric replacement of saturated fat (SFA) or cis unsaturated fatty acids (CFA) with TFA. As compared to isocaloric intakes of SFA or CFA, consumption of TFA raises levels of low-density lipoprotein cholesterol (LDL), reduces levels of high-density lipoprotein cholesterol (HDL) and increases the ratio of LDL to HDL as well as the ratio of total cholesterol to HDL cholesterol\(^8\). Epidemiological studies suggest that a change of one unit in the ratio of total and HDL-cholesterol is associated with a 53% increase in the risk of myocardial infarction\(^9\). Pooled intervention trials of isocaloric replacement of 1 E% CFA with 1 E% of industrial TFA resulted in a 0.055 increase in the LDL to HDL ratio (95% CI 0.044-0.066), which would correspond to a 3 to 12% increase in the risk of CVD\(^10\). Meta-analysis of prospective cohort studies of habitual TFA consumption and coronary heart disease risk, found that a 2% higher energy intake from TFA, as an isocaloric replacement of carbohydrate, was associated with 23% higher Coronary Heart Disease (CHD) risk (RR 1.23; 95% confidence interval 1.11-1.37)\(^9\).

Since the relation between the intake of TFA and the incidence of CHD reported in prospective studies has been greater than that predicted by changes in serum lipid levels alone, it has been suggested that TFA may also influence other risk factors of CHD. TFA may promote CVD through the following pathways: Systemic inflammation, endothelial dysfunction, insulin resistance, visceral adiposity, arrhythmias and the development of diabetes\(^7\). TFA intake may have yet more deleterious effects: inverse associations have been observed between TFA and essential long-chain polyunsaturated fatty acids in the blood lipids of pregnant women and their offspring at birth and in human milk. This may have implications for neurodevelopment in infancy, as the long-chain PUFA are crucial to neurodevelopment\(^11\).
Legislation to remove industrial TFA has been successfully implemented in Denmark, Norway, Iceland, Austria, Hungary, Switzerland, Seattle, New York and then across the USA. Experiences from Denmark show that industrial TFA can be replaced with healthier substitutes without increasing the cost or reducing the quality of foods. Analyses of foods before and after implementation of legislative restrictions on the content of industrially produced TFA in foods have demonstrated widespread compliance with little evidence for adverse effects on food availability, price or quality. Replacement of industrial TFA with other fats is therefore feasible and would result in benefits especially in foods previously having higher levels of TFA (35-45%), such as the bakery goods found in Eastern Europe.

Because of health concerns, the food industry has eliminated industrial produced TFA acids (industrial TFA) from foods in most of Western Europe, so the average consumption of TFA has declined to 1-2 energy percent over the last twenty years. However, TFA values in foods bought in Eastern Europe are still high and may contribute to the much higher death rates from CHD in Central and Eastern Europe than in Northern, Southern and Western Europe. Similarly, the reduction in TFA through legislative means in Denmark implemented in 2003 is likely to help explain the 70% drop in cardiac mortality seen in Denmark between 1980 and 2009.

Furthermore, food items with a high content of TFA are found in ethnic shops across Europe: a survey sampling biscuits in 20 mainly central and eastern European countries in 2012-13 analysed for TFA content found that in seven predominantly western countries no TFA was found whereas in nine predominantly Eastern European countries, products with intermediate or very high TFA content were common. Additionally, in ethnic shops in western European countries, high TFA content was also common. This finding supports that in subgroups under current EU legislation, sale of products containing TFA is disparately distributed within and between EU countries resulting in higher intake of TFA in Eastern Europe and among the socioeconomically deprived and ethnic minorities in Western European Countries.

Voluntary reductions in the amount of industrial TFA in foods by food producers appear to work well in some countries but not in others. Food producers in a given country or in neighboring countries may remove industrial TFA from their products but importing products with high TFA content from more distant countries may counteract the initiatives, as demonstrated by high availability of TFA-containing food products in ethnic supermarkets in Sweden. This is worrisome, since mortality from CVD among ethnic minorities exceeds that of the indigenous population in several European Countries.

At the moment there are different regulations in the EU countries, from a regulatory maximum content of 2g/100 g in Denmark to mandatory labeling, voluntary reformulation or voluntary labeling. A recent systematic review comparing effectiveness of policies for reducing dietary TFA concluded that policies aimed at restricting the TFA content of food
were associated with significant reductions in TFA levels without increasing total fat content. Such policies are feasible, achievable and likely to have an effect on public health. The review compared voluntary self-regulation, labeling alone, labeling and voluntary limits, local and national regulatory interventions. It concluded that although all interventions resulted in some decrease in TFA content, national and local regulatory interventions were the most effective and powerful.

3. European Cardiovascular Disease Statistics 2012
5. Stender et al, BMJ Open 2014
6. Perk et al, European Heart Journal 2012
7. Mozaffarian & Stampfer, 2010
15. Craig-Schmidt MC, Atheroscler Suppl 2006
17. Kravic et al, Hem Ind 2011