RISK ASSESSMENT REPORT: TRANS FATTY ACIDS IN THE NEW ZEALAND AND AUSTRALIAN FOOD SUPPLY

UPDATE 2009

In 2006, Food Standards Australia New Zealand (FSANZ) conducted a risk assessment in relation to *trans* fatty acids (TFA) in the New Zealand and Australian food supply (see Attachment 1 for the 2006 report). This report is an update of the evidence on TFA that has emerged since the release of the 2006 report.

Findings of the 2009 Risk Assessment Update

This 2009 updated risk assessment addresses the following three questions:

1. *What is the relationship between TFA intake, biomarkers of disease, and outcomes of public health significance?*
2. *Are there differences in health effects according to ruminant or manufactured TFA?*
3. *Compared to the health impact of saturated fatty acids, what is the impact of TFA on biomarkers and outcomes of coronary heart disease?*

A literature search identified 30 new studies for inclusion in the update. The majority of these studies used a case-control,[1] prospective cohort[2], or cross-sectional design[3]. Cross-sectional studies do not allow cause and effect to be established, and the other two designs can be problematic in the dietary area owing to the small relative risks generally being examined and the difficulties in controlling adequately for inter-related nutrients. The 2006 report included consideration of the bulk of randomised controlled trials[4] examining the relationship between TFA intake, disease outcome and biomarkers of disease. This study design is suited to establishing the cause and effect, and estimating the magnitude of the effect. Therefore, most studies identified as part of this update do not have a strong enough design to alter the conclusions of the original 2006 report.

In relation to question 1, the findings from eight new studies predominantly support an association between increasing TFA intake and increased risk of coronary heart disease biomarkers. This is consistent with the findings of the large cohort studies and randomised controlled trials considered in the 2006 report.

1 Case-control studies compare history of food intakes in people with a specific condition with those that do not have the condition
2 Prospective cohort studies follow a group of people over time, measure food intake, and look at who does and who does not develop a particular condition
3 Cross-sectional studies measures current food intake and current disease status in a group of people at one point in time
4 Randomised controlled trials randomly assigns a group of people into those who undergo an intervention and those that do not or that undergo a different intervention (eg different dietary intakes) then follows up to determine who develops the condition and who does not

The research examining the postulated association between TFA intake and cancer risk included two studies on colorectal cancer, two on prostate cancer, and one on breast cancer. These were predominantly nested case-control studies. The findings in this area are still too limited to draw any inferences beyond noting that further investigation is warranted.

Two new studies indicated that maternal TFA intake is reflected in the TFA content of breast milk, which is consistent with previous findings.

One recent small cohort study investigated the association between TFA intake and gestational diabetes, gestational hypertension, and preeclampsia. Another very small cohort study investigated the association of TFA intake and birth outcome. These two studies represent very limited new data. Further research is required before any conclusions about an association between TFA and maternal health or pregnancy outcomes can be made.

In relation to the second question, three new studies comparing the effects of manufactured and ruminant TFA serve to highlight the ongoing uncertainty in this area. The findings of two randomised controlled trials were inconsistent with one another, and the one prospective cohort study had important limitations. Further randomised controlled trials with appropriate comparisons and sufficiently large sample sizes are needed before any firm conclusions can be reached.

No new data has come forth in relation to the third question in terms of the relative size of the effect of saturated fatty acids (SFA) and TFA on blood lipids. However, two studies have assessed the relative effects of different options for replacing fatty acid mixes rich in TFA with SFA rich alternatives. These studies were considered relevant in the broader context of the desire to replace TFA in food manufacture with viable alternatives. The results suggest care is warranted when encouraging substitutions for TFA, as these may not necessarily have more favourable effects on coronary heart disease (CHD) risk factors than what they are replacing. It is a good reminder that all fatty acids, whether the favourable *cis*-monounsaturates or *cis*-polyunsaturates or the adverse SFA and TFA, need to be considered when trying to predict the effect of a food or ingredient on blood lipids. This is especially important given that the contribution of different fatty acids to CHD risk factors is constantly being updated; e.g. predictive equations that describe the effects of different fatty acids on blood lipids have recently been extended to ApoA1, ApoB and Lipoprotein(a) lipid fractions.

2009 Conclusions

In relation to intake of TFA and risk of disease, the most consistent and robust evidence is for its adverse effect on blood lipids. TFA appear to raise LDL- and lower HDL-cholesterol concentrations; a change associated with an increased risk of cardiovascular disease. Several cohort studies also show a direct association with TFA intake and risk of cardiovascular disease. The evidence examining the link between TFA intake and some forms of cancer, type 2 diabetes, age-related macular degeneration, early development problems, and pregnancy outcome is sparse. Studies of an appropriate design to determine if there is a causal relationship, and the size of the effect, are limited or have simply not been done for these conditions.

A difference in the effects of TFA produced by ruminants such as cows and that produced by partial hydrogenation of vegetable oil has been suggested. There are some differences in the fatty acid isomers that comprise ruminant TFA and those that comprise manufactured TFA, but there are also considerable overlaps. Further, the specific proportions of different TFA isomers varies with feed in the case of ruminant TFA, and with the choice of oil and

processing conditions in the case of manufactured TFA. These variations across countries and over time make this a difficult area of study. The evidence to date does not allow for a clear distinction to be made between the effects of ruminant and manufactured TFA. In the absence of any definitive evidence, the recommendation to reduce saturated fat intake, and hence animal fats (which include ruminant TFA) is still relevant. However, trying to eliminate TFA from diets by avoiding meat and dairy foods entirely could also reduce the intake of desirable dietary components for which these foods are good sources.

Evidence for TFA having a more adverse effect on blood lipids compared with SFA on an equal energy basis is compelling. However, in the context of replacing manufactured TFA in foods where they are currently used for technical or other purposes requires consideration. It is important to investigate the effects of suggested alternatives, as TFA are only one component that effect blood lipid levels. A low TFA ingredient may have a more adverse effect overall than a high TFA ingredient, depending on the rest of its composition.

Replacing carbohydrate in the diet with an isoenergetic amount of TFA adversely raises total and LDL-C concentrations. The evidence from dietary intervention trials, summarised in a systematic review, is consistent and compelling. Replacement of *cis* fatty acids with isoenergetic amounts of TFA adversely raises the LDL:HDL cholesterol ratio. In a systematic review, a linear dose-response between percentage energy intake from TFA and change in LDL:HDL cholesterol was found with no evidence of a diminishing or threshold effect . The evidence for a dose-response effect is consistent, at least for TFA intakes in excess of 3% energy intake.

A joint review by FAO and WHO of dietary factors associated with cardiovascular disease has recommended that population TFA intakes should contribute less than 1% of population daily energy intakes. Based on the findings of a recent 2008-09 survey on TFA content in foods, mean dietary intakes of TFA (both ruminant and industrial sources) are estimated at approximately 0.6% of dietary energy intake in Australia and New Zealand. These values have decreased from a mean of 0.7% of energy intake previously estimated using pre-2007 TFA concentration data. The decrease reflects a 20-45% reduction in the population intakes of manufactured TFAs that have occurred from changes in manufacturing practice to reduce TFA content in foods.

The full potential for reducing Australian and New Zealand TFA intakes from manufactured sources is approximately one-quarter to one-third of total TFA intakes. Complete removal of TFA of manufactured origin cannot be achieved because some TFA formation occurs during the industrial process of oil deodorization. Some TFA formation is also likely to occur during high temperature cooking with vegetable oils containing polyunsaturated fatty acids. Reductions in intakes of ruminant sources of TFA could be achieved if people chose low fat dairy products and lean meats.

Because of the uncertainty as to whether the blood lipid dose-response effect occurs at low levels of TFA intake, and because associations with CHD incidence are unknown at low intakes, it is not possible to estimate the true extent of disease risk reduction that would occur in Australia and New Zealand if the TFA ingestion in the populations was reduced below already low intakes. Nevertheless, there may be a health benefit if TFA intakes in Australia and New Zealand continue to decrease.