Expert Commentary

Levels of trans fats in the food supply and consumption in Australia

An **Expert Commentary** brokered by the Sax Institute for The National Heart Foundation of Australia. December 2017.

An **Expert Commentary** brokered by the Sax Institute for The National Heart Foundation of Australia
December 2017

This report was prepared by:

Jason HY Wu, Shauna Downs, Elise Catterall, Milan Bloem, Miaobing Zheng, Lennert Veerman, Jan Barendregt, Beth Thomas.

The George Institute and The National Heart Foundation of Australia

Enquiries regarding this report may be directed to the:

Manager
Knowledge Exchange Program
Sax Institute
www.saxinstitute.org.au
knowledge.exchange@saxinstitute.org.au
Phone: +61 2 91889500

Suggested Citation:

Wu J, Downs S, Catterall E, Bloem M, Zheng M, Veerman L, Barendregt J, Thomas, B. Levels of trans fats in the food supply and population consumption in Australia: an Expert Commentary rapid review brokered by the Sax Institute (www.saxinstitute.org.au) for The National Heart Foundation of Australia, 2017.

Levels of trans fats in the food supply and population consumption in Australia

An **Expert Commentary** brokered by the Sax Institute for The National Heart Foundation of Australia, December 2017.

This report was prepared by Jason HY Wu, Shauna Downs, Elise Catterall, Milan Bloem, Miaobing Zheng, Lennert Veerman, Jan Barendregt, Beth Thomas.

Contents

Levels of trans fats in the food supply and population consumption in Australia 3

1 Glossary 6

2 Executive summary 7

Background 7

Summary of findings 7

Conclusions 8

3 Levels of TFA in the food supply and TFA intake in Australia 9

Data sources 9

Ruminant and industrially manufactured TFA in Australian food products 9

Table 1. Maximum level of TFA detected in ruminant products in Australia* 10

Current level of TFA in foods 10

Figure 1. TFA levels in processed and takeaway foods in Australia in 2013 11

Comparison of TFA level in foods to other countries 11

Table 2. TFA level in food: comparison between Australia and other countries 12

Changes in TFA level in food over time in Australia 12

Figure 2. TFA content across 19 comparable food categories assessed by FSANZ in 2005–2007, 2008–2009, and 2013.[3] 13

TFA intake in Australia: NNPAS results 13

Table 3. TFA intake in Australia based on the 2011–2012 NNPAS survey 15

Table 4. TFA intake in Australia compared to other countries 15

Table 5. TFA intake in Australia according to education and income levels based on the 2011–2012 NNPAS survey* 16

Proportional contribution of ruminant and manufactured sources to TFA intake 17

Discussion 17

TFA in foods 17

Relating observed changes in TFA to voluntary reformulation to reduce TFA 17

TFA intake 18

Table 6. Total amount of TFA obtained from a hypothetical 'high TFA menu' 18

4 TFA attributable mortality 19

Methods 19

Exposure and relative risks 19

Table 7. Relative risk (and 95% confidence interval) for heart disease mortality for each 2% point increase in energy intake from trans-fatty acids, by age group[16] 19

Potential impact fraction 20

Calculation methods and uncertainty 20

Results 20

Table 8. Attributable TFA heart disease mortality in 2010, by sex and age group and in total, with 95% uncertainty intervals 21

Discussion 21

5 Impact of policies to reduce TFA consumption 23

Systematic review methods 23

Search terms and databases 23

Inclusion criteria 23

Overview of search 23

Study extraction 24

Study quality assessment 24

Findings of systematic review 24

Overview of included studies 24

Figure 3. Systematic review flow chart 25

Findings of modelling studies (2000–2016) 25

Table 9. An overview of the modelling studies included in the systematic review 27

Quality of modelling studies 30

TFA interventions in the 'real world' (2013–2016) 30

Table 10. Overview of the 'real life' TFA policy studies included in the systematic review 31

The impact of TFA interventions in the 'real world' (2013–2016) 35

Table 11. Changes in the consumption of fatty acids following the implementation of TFA intervention 36

Synthesis of the evidence from 2000–2016 36

Discussion 36

6 References 37

7 Supplementary tables and figures 40

Supplementary Table 1. Summary of FSANZ reports on TFA level in foods 40

Supplementary Table 2. TFA as % of total fat in Australian processed and takeaway foods in 2013 41

Supplementary Table 3. The underlying assumptions of the included modelling studies 43

Supplementary Table 4. Summary of the relevant results of studies from the 2000-2012 review that used a pre/post study design (adapted from [34]) 45

Supplementary Table 5. The percentage of foods classified as TFA free post policy interventions (adapted from[34]) 54

Supplementary Table 6. Changes in the fatty acid composition of foods after the introduction of a TFA policy (adapted from Downs 2013,[34]) 55

Supplementary Figure 1. Products sampled from takeaway shops 56

Supplementary Figure 2. Products sampled from independent retailers and supermarkets 57

1 Glossary

AUSNUT	Australian Food, Supplement and Nutrient Database
CHD	Coronary Heart Disease
CVD	Cardiovascular Disease
EPHPP	Effective Public Health Practice Project
FSANZ	Food Standards Australia and New Zealand
FDA	US Food and Drug Administration
GBD	Global Burden of Disease
GRAS	Generally Recognized as Safe
iTFA	Industrially Manufactured Trans Fatty Acids
NNPAS	National Nutrition and Physical Activity Survey
PAFs	Population Attributable Fractions
PIFs	Potential Impact Fractions
rTFA	Naturally Occuring Trans Fats
SFA	Saturated Fatty Acid
TFA	Trans Fat
WHO	World Health Organisation

2 Executive summary

Background

One of the strategic aims of the National Heart Foundation of Australia (Heart Foundation) is to provide national leadership to advocate for improvements to the Australian food supply. Existing research strongly indicates that industrially manufactured trans fatty acids (iTFA) adversely impact cardiovascular risk factors and increase risk of coronary heart disease (CHD). Currently, the Heart Foundation holds two specific policy and advocacy positions with regards to trans fat (TFA):

- Mandatory labelling of trans fats on nutrition information panel of packaged foods
- Elimination of the use of industrially produced trans fats.

To ensure ongoing effectiveness and relevance, the Heart Foundation engaged an expert group to conduct an expert review that will provide the evidence to allow the Heart Foundation to examine its current positions on TFA. The expert group approached this aim by examining available data and recent policy developments in Australia and globally, focusing on 3 important questions:

1. What are current TFA levels in foods and consumption levels in Australia?
2. What is the burden of CHD attributable to TFA intake?
3. How effective are policy interventions at reducing TFA intake and what are the downstream impacts of such policies on the incidence of CHD?

Summary of findings

The Australian government has so far engaged with the food industry in an effort to lower iTFA in the food supply through voluntary product reformulation. While this approach has yielded substantial reductions in iTFA for edible oil spreads, there has been little meaningful change in iTFA level for most other products in the last 15 years, based on sequential surveys conducted by Food Standards Australia and New Zealand.

TFA levels varied substantially within several food categories, with some products sold from independent retailers and supermarkets continuing to contain high levels of iTFA – including popular snack foods such as meat pies, prepared pastries, and popcorn. Conversely, available data suggest that the levels of iTFA are currently relatively low in takeaway foods in Australia. Based on the latest nationally representative dietary survey conducted in 2011–2012, it was ascertained that Australians on average have intakes of TFA at 0.6% of daily energy, with an estimated 60–75% coming from ruminant (natural) sources, and the rest being iTFA.

While TFA intake is relatively low in Australia compared to other countries, about one in ten Australians continue to exceed the current World Health Organization's (WHO) recommended limit of 1% daily energy from TFA. Analyses based on the national survey further identified a social-economic gradient for TFA consumption. Among Australians in the lowest fifth of income and education brackets, about 1/7 exceeded the WHO recommended TFA limit. Epidemiologic modeling was conducted using these up-to-date estimates of TFA consumption in Australia and the known association between TFA and CHD risk. It was estimated that around 1.5% of all heart disease mortality in Australia in 2010 was attributable to TFA intake. In absolute terms, this equates to almost 500 deaths because heart disease is a leading cause of death in Australia.

A systematic review of published literature was subsequently conducted: it suggested that currently more than 10 countries globally have implemented at least one policy targeting the reduction of iTFA from the food supply, including mandatory labelling and bans at both the local and national level. Appraisal of the identified evidence suggests that although all policy approaches led to reductions in TFA levels in foods and subsequent intakes, stronger policies were likely to have more pronounced effect. The systematic review also identified several recent cost-effectiveness analyses studies in other countries. These studies indicated that policies including mandatory labelling and imposing limits or bans on iTFA in foods not only could reduce CHD burden, but also bring about substantial economic benefits and cost savings that amount to millions of dollars over 5–10 years. Modelling studies also suggested that compared to mandatory labelling, limits or bans on iTFA have the greatest potential to reduce CHD risk for the most disadvantaged groups and thereby reduce health inequalities.

Conclusions

- The available data demonstrate that some products made available by independent retailers and supermarkets contained high levels of iTFA, while levels in takeaway foods were relatively low. Further, there has been no consistent trend of reduction in surveyed product categories over the previous decade. The continued existence of food products with high TFA levels in Australia means that sub-groups of the Australian population are still potentially exposed to high levels of TFA due to regular consumption of high TFA products. Further development and implementation of policies to reduce iTFA in Australia may therefore be warranted, and such efforts should prioritize packaged foods.
- Further progress to reduce iTFA in the Australian food supply is possible, as the available data demonstrates:
    1. Broad variability of TFA level within food categories suggesting that further reformulation of packaged food products could be feasible
    2. iTFA is estimated to contribute to approximately one-third and ruminant TFA approximately two-thirds of total TFA intake. Since there are no plausible ways to remove ruminant TFA from the food supply, complete removal of iTFA would result in 100% of TFA coming from ruminant sources. Labelling schemes and bans/limits should target and apply to iTFA.
- The observed socio-economic gradient in TFA consumption in Australia should be taken into consideration when formulating policies to reduce iTFA from the food supply. Modeling studies from overseas suggest compared to mandatory labelling, placing limits on the level of iTFA in foods or banning the use of iTFA are likely to reduce health inequality attributable to CHD. Our review highlights the need for additional investigations to model other population health metrics impacted by reduction in iTFA (e.g. health adjusted life years gained) and the economic impact of such approaches in Australia.
- Additional studies examining the relative merit of removing iTFA vs. other policy interventions aimed at improving the absolute and proportional burden of CHD mortality, such as the reformulation of foods to lower sodium levels. Such studies will help to inform and prioritize policy and advocacy efforts.
- In the last decade or so, there is a growing momentum at both the local and national level around the world to implement policies aimed at the removal of iTFA to improve public health.
- Comprehensive, high quality and periodic surveys need to continue to monitor levels of TFA in the food supply. Such data will be required to inform policy makers regarding the efficacy of policies to reduce iTFA.

3 Levels of TFA in the food supply and TFA intake in Australia

In the first section of the report we identified and reviewed data for current level of TFA in processed and takeaway foods, as well as conducted analyses to assess the current TFA intake level of Australians based on the 2011–2012 National Nutrition and Physical Activity Survey (NNPAS). Throughout the report, we aimed to place Australian TFA data in context by assessing historical trends and comparisons against findings from other countries where possible.

Data sources

Food Standards Australia and New Zealand (FSANZ) conducted a series of surveys (Supplementary Table 1) over the past decade to assess the levels of trans fatty acids (TFA) in the Australian food supply.[1-3] The investigations were conducted between 2001–2005, 2008–2009 and 2013. The 2001–2005 survey was not centrally coordinated but instead pooled data from five separate sources, and had a relatively small sample size (n=330). In contrast, the subsequent surveys were nationally coordinated, with the 2013 survey having the broadest geographical coverage in terms of location (including all states in Australia) and products selected (by deliberately including products with different quality and prices within each product category). In addition to the FSANZ reports, we also conducted a literature search to identify published academic articles. Electronic searches were searched via Medline, Embase, and AMED. Search terms used included trans fatty acids, reformulation, diet, snack and takeaway foods. We searched for papers in Australia that met the following inclusion criteria: 1) reported TFA levels in foods, 2) assessed TFA intake in any location and by any population, 3) were conducted between 2007 and 2016. For TFA in food, two articles were initially identified, and hand searches of these papers identified one more relevant paper. These three studies were conducted in 1993 and between 2006 and 2008, and reported the level of TFA in selected food products.[4-6] For TFA intake, there were no relevant studies identified via literature search. Based on the data available and considering our inclusion criteria, we focused on the FSANZ surveys from 2007, 2009, and 2013 since these are the most comprehensive data available, and used results from the academic publications to supplement the FSANZ findings. To ascertain TFA intake in Australia, we utilized the National Nutrition and Physical Activity Survey (NNPAS) data, which was conducted between 2011–2012 and is the latest nationally representative dietary survey available for Australians.[7]

Ruminant and industrially manufactured TFA in Australian food products

Naturally occurring TFAs (rTFA) are obtained from the meat and milk of ruminant animals such as cattle and sheep. TFAs are also manufactured via industrial processes (referred to as iTFA in this report) including partial hydrogenation and deodorization of vegetable oils, and heating oils at very high temperatures. It should be noted that the methods used by FSANZ to assess TFA do not reliably distinguish between rTFA and iTFA.[3] Although the amount of rTFA can vary depending on feeding practices, season, and location,[8] rTFAs generally occur at relatively low levels in ruminant products. In analyses conducted by the New South Wales Food Authority and FSANZ, the average levels of rTFA for ruminant products were generally ≤ 2%, except for butter (~5%, Table 1),[1, 9] which is consistent with data from other countries that have found rTFA to contribute on average between 2–5% of the total fatty acids in ruminant derived foods.[10] In comparison, partially hydrogenated vegetable oils (PHVO), the major source of iTFA, can consist of up to 60% of total fatty acids as TFA. These findings are particularly relevant when considering products that could contain both rTFA and iTFA (e.g. meat pies and sausage rolls), as it would be expected that if all the TFA in these products came from ruminant sources, then TFA should not exceed 2-5% of total fat.

Table 1. Maximum level of TFA detected in ruminant products in Australia*

Product type	Number of samples analysed (n)	Mean TFA content (range) (g/100g fat)
Beef, raw	5	1.4 (0-2.4)
Beef sausages, raw or cooked	5	1.4 (0.4-1.9)
Butter	3	5.1 (4.8-5.5)
Cheese, cheddar, natural & processed	5	2.1 (2-2.2)
Cream	5	2.2 (2.1-2.3)
Ice cream	5	1.7 (1.1-1.9)
Lamb, raw	5	2.5 (1.2-4.4)
Milk, full fat, plain and flavoured	10	1.8 (0-3.2)
Yoghurt	5	1.7 (0.9-3.6)

*Based on report by New South Wales Food Authority and FSANZ survey between 2005-2007.[1, 9]

Current level of TFA in foods

The distribution of TFA (as a percentage of total fat) in processed and takeaway foods as analysed by FSANZ in 2013 is presented in Supplementary Table 2 and Figure 1. To aid interpretation of the data and to put these findings into context, TFA levels in foods were compared with existing regulations pertaining to TFA content in Denmark and several other European countries — these regulations stipulate that iTFAs cannot exceed two grams per 100 grams of oil or fat in food products (that is, iTFA should be equal or less than 2% of total fat).[11] The 2% iTFA limit is presented as the vertical red line in Figure 1.

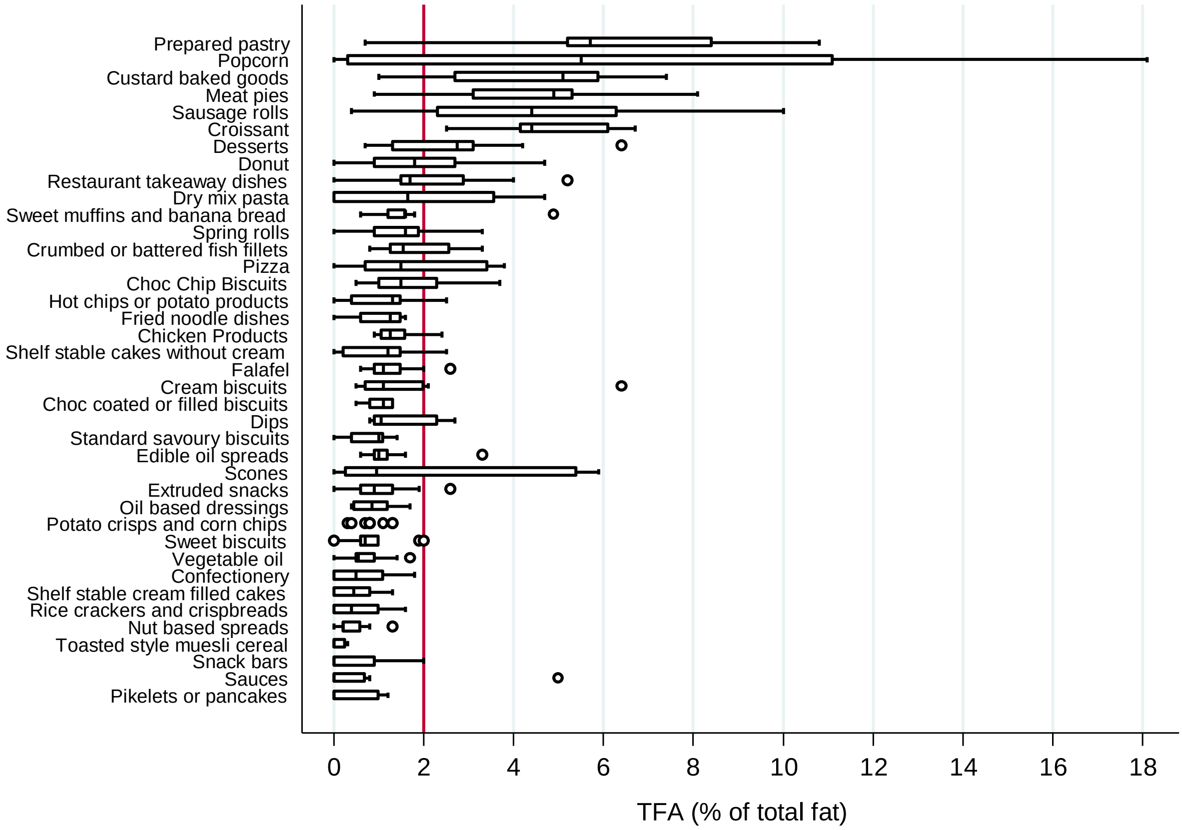


Figure 1. TFA levels in processed and takeaway foods in Australia in 2013

As can be seen in Figure 1, categories with the highest median TFA levels included prepared pastry, popcorn, custard baked goods, meat pies and sausage rolls, croissants, and desserts. These products were sampled from independent retailers and supermarkets, not takeaway shops (results for the different types of stores sampled are presented in Supplementary Figures 1 and 2). About 75% of the products tested in Australia met the Danish regulations, while about 25% exceeded the limit, although some products in this latter category likely contained both rTFA and iTFA. When products that likely contain both rTFA and iTFA were excluded (including pizza, meat pies, sausage rolls, croissants, desserts, and custard baked goods), 14% of the remaining products surveyed exceeded the Danish upper limit for TFA. In some product categories, the range of TFA was very broad and in some of the products the level of TFA exceeded the 2% limit several fold – for example, up to 18% in one of the popcorn products tested, and 10% or over in prepared pastry and sausage rolls.

Comparison of TFA level in foods to other countries

To further interpret current levels of TFA in Australian foods, product categories with the highest TFA (those with median TFA levels over 2% of total fats) were compared to similar products found in other countries (Table 2). Overseas data was selected from the most recent available data (less than 10 years) identified from our literature search, and where similar product categories were available for comparison.

Table 2. TFA level in food: comparison between Australia and other countries

Product type	Australia, 2013	Canada, 2010[10]	UK, 2013[12]	Netherlands, 2011[13]	Malaysia, 2014[14]
			TFA level as % of total fat Mean (±SD)*		
Prepared pastry	6.3 ±3.2	2.1 ±8.1	-	-	-
Popcorn	6.8 ±7.5	4.4 ±11.8	-	-	-
Custard baked goods	4.4 ±2.4	1.3 ±1.4	-	-	-
Meat pies	4.4 ±2.2	-	2.4	-	-
Sausage rolls	4.8 ±3.0	2.5 ±4.6	-	-	-
Croissants	4.8 ±1.4	2.0 ±1.2	-	-	-
Desserts	2.7 ±1.7	1.8±3.7	4.0	4.7	2.1
Margarine/spread	1.9 ±0.7	2.7 ±7.2	0.7	1.4	0.2

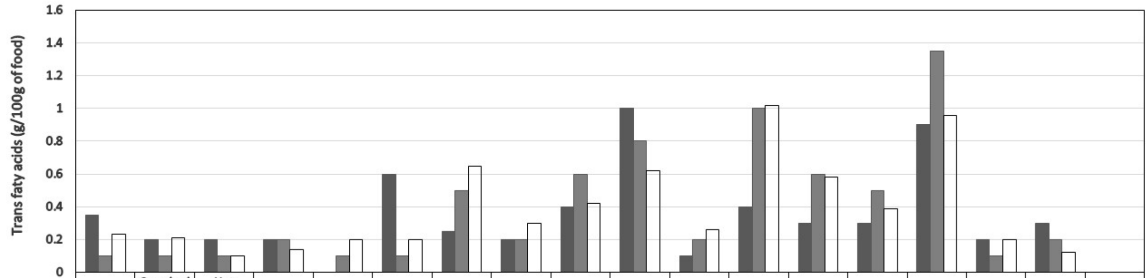
*SD was presented where available.

As can be seen from the table, for several product categories such as prepared pastry, popcorn, custard baked goods, croissants and meat pies, the mean TFA level of Australian products was from 54 to over 200% higher than comparable products from Canada, UK, and the Netherlands. Conversely, mean TFA levels for desserts and margarine/spreads were comparable or lower in Australian products when compared to those from other countries.

For other countries with different culinary preferences to Australia it was not possible to find similar foods in order to make a comparison, despite various reports of high TFA levels in foods common to their country. For example, TFA levels for savoury snacks in India have been reported to contain between 25 and 54% of TFA as total fat.[15]

Changes in TFA level in food over time in Australia

In the 2013 FSANZ report, levels of TFA in 19 food product categories were compared from 2005 to 2013, as shown in Figure 2. This analysis includes only those product categories that were comparable over the different surveys, but does include many of the key product categories with high TFA content including bakery goods, edible oils, and meat products. The figure shows the median concentration of TFA (expressed as TFA in grams/100 grams of the food products). Overall, median TFA concentration was lower in 8 of the 19, higher in 7 of the 19, and remained comparable in the other food categories. Results were largely consistent when the analysis was restricted to a set of branded products (n=51) that were sampled in both 2008–09 and 2013.[3]



	Chicken Nuggets products	Crumbed /Battered Fish Fillets	Hot Chips/Potato Poducts	Pizza	Extruded Snacks	Potato Crisps&Corn Chips	Edible Oil Spreads	Oil Based Dressings	Meat Pies	Sausage Rolls	Chocolate Biscuits	Croissant	Custard Baked Goods	Donut	Prepared Pastry	Savoury Biscuits-Standard	Shelf Stable Cakes	Snack Bars
	Takeaway foods				Snack foods		Fats&Oils		Meat products		Bakery products							Other
■ 2005-2007	0.35	0.2	0.2	0.2	<LOR	0.6	0.25	0.2	0.4	1	0.1	0.4	0.3	0.3	0.9	0.2	0.3	<LOR
■ 2008/2009	0.1	0.1	0.1	0.2	0.1	0.1	0.5	0.2	0.6	0.8	0.2	1	0.6	0.5	1.35	0.1	0.2	<LOR
□ 2013	0.23	0.21	0.1	0.14	0.2	0.2	0.65	0.3	0.42	0.62	0.26	1.02	0.58	0.39	0.96	0.2	0.12	<LOR

*<LOR, lower than the limits of reporting

Figure 2. TFA content across 19 comparable food categories assessed by FSANZ in 2005–2007, 2008–2009, and 2013.[3]

Evidence of how TFA levels may have changed in foods over time was also sought from published literature. In a study by Mansour et al in 1993 in Australia, TFA levels in 13 margarine spreads were assessed and found to be very high. With 13 products sampled, TFA (as percentage of total fatty acids) ranged between 8 and 14.5%.[4] In comparison, the FSANZ 2013 reported the level of TFA in edible oil spreads to be less than or equal to 3.3% (n=13 products analysed). FSANZ data was not compared to the study by McCarthy et al, as a different analytical method was used to assess TFA levels and the study also only examined a small numbers of products per category (five or less).[5] The study by Wijesundera et al in 2006 also only had a few food categories comparable with FSANZ data, and the results were mostly similar (not shown).

TFA intake in Australia: NNPAS results

As part of the 2011–13 Australian Health Survey [16], the 2011–12 NNPAS was conducted throughout Australia from May 2011 to June 2012 in approximately 9,500 private dwellings (77% of participating dwellings) across Australia to collect dietary intake and physical activity information of the Australian population aged two years and over. A stratified multistage area sampling was used for sample selection to ensure the selected sample was representative of the Australian population. Day one face-to-face 24-hour dietary recall data from adults aged 19 years and over were collected using the five-phase automated multiple-pass method. The dietary intake data were coded according to Australian Food, Supplement and Nutrient Database (AUSNUT) food codes. Individual food items were categorised into food classification groups based on the food and measures database developed by FSANZ.

TFA intake (mg/day) and percentage energy from TFA were calculated by age groups, and separately by sex. Information regarding other social-demographic variables including education level (Bachelor and higher, diploma and certificate, no school qualification, not determined) and income quintiles was also obtained from the NNPAS survey. Linear regression analyses were conducted to assess the associations between age, gender, education level, and income, and TFA intake (expressed as percent of total energy). Personal weighting factors were applied to the dataset to ensure that the survey estimates conformed to the population estimates by sex, age, area of usual residence and seasonal effects. All statistical analyses were performed using SPSS 20.0 (SPSS Inc, Chicago, IL, USA) with statistical significance set as $P < 0.05$ (two-sided).

The average TFA intake among adults aged 19 years and over was 1.39 ± 1.14 g/day (mean ± SD), which equates to 0.59 ± 0.38% of daily energy intake. The 95th percentile was ~3.6g/day. Table 3 illustrates TFA intake by age and gender-specific groups. TFA intake was generally similar between men and women across the age groups. The 90th percentile for TFA intake was around 1% of daily energy intake both men and women, that is, the usual intake of TFA is over 1% of total daily energy for about 10% of Australian adults. Mean TFA intake level in Australia is relatively low in comparison to other countries (Table 4).

TFA intake differed according to the level of education and income (Table 5). Participants with higher education and more income had significantly lower levels of TFA intake. For example, average TFA intake was approximately 10% lower among participants with a Bachelor or higher degree compared to those with no school qualification. In a multivariate model adjusting for age, gender, education and income, both education and income were significantly associated with TFA intake ($P\text{-value}_{trend} \leq 0.001$ for both). The percentage of participants who exceeded the 1% WHO TFA intake cut off in the lowest level of income and lowest level of education was 14.2% (253 out of 1776) and 14.1% (515 out of 36)

Table 3. TFA intake in Australia based on the 2011–2012 NNPAS survey

Age (years)	19-24		25-34		35-44		45-54		55-64		65-74		75+	
Gender	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
n	560	526	878	879	873	882	830	855	705	725	459	478	307	384
	TFA intake as percent of total energy													
Mean±SD	0.59±0.35	0.57±0.39	0.56±0.36	0.59±0.39	0.58±0.33	0.55±0.33	0.56±0.34	0.56±0.37	0.61±0.41	0.56±0.40	0.59±0.38	0.58±0.39	0.65±0.41	0.64±0.42
90th percentile	0.97	1.02	1.04	1.09	1.02	1.04	1.00	0.97	1.21	1.12	1.05	1.00	1.21	1.11
95th percentile	1.18	1.46	1.29	1.29	1.19	1.24	1.21	1.20	1.45	1.30	1.23	1.38	1.42	1.36

Table 4. TFA intake in Australia compared to other countries

Countries and year of dietary survey	TFA intake as % of energy		Survey methods and population
	Mean	SD	
Australia, 2013[3]	0.6	0.4	1-day 24-hour diet recall, nationally representative population survey
Brazil, 2008-2009[17]	1.4	-	2-day food diary, nationally representative population survey
Canada, 2004[18]	1.4	-	24-hour diet recall, nationally representative population survey
Iran, 2001-2003[19]	4.2	-	3 consecutive 24-hour dietary recalls, nationally representative population survey
Netherlands, 2003[13]	0.8	-	24-hour diet recall, sample aged 19-30
UK, 2008-2011[12, 20]	0.8	0.4	4-day food diary, nationally representative population survey
US, 2007-2009, Men[21]	1.9	-	1-day 24-hour diet recall, cross-sectional survey of adults in Minneapolis
US, 2007-2009, Women[21]	1.7	-	1-day 24-hour diet recall, cross-sectional survey of adults in Minneapolis

Table 5. TFA intake in Australia according to education and income levels based on the 2011–2012 NNPAS survey*

						P-trend†
Education level	No school qualification (n=3636)	Diploma and certificate (n=3177)	Bachelor and higher (n=2385)			
Mean±SD	0.60±0.39	0.59±0.38	0.54±0.32			<0.0001
90th percentile	1.12	1.05	0.95			
95th percentile	1.30	1.32	1.15			
Income quintiles	Q1	Q2	Q3	Q4	Q5	
(Q1 lowest, Q5 highest)	(n=1776)	(n=1531)	(n=1530)	(n=1812)	(n=1776)	
Mean±SD	0.62±0.42	0.60±0.39	0.57±0.36	0.56±0.34	0.56±0.34	0.001
90th percentile	1.16	1.08	1.06	1.00	0.99	
95th percentile	1.45	1.32	1.29	1.21	1.21	

*n=143 and 916 participants did not have data on education and income, respectively and were excluded from these analyses

†Based on multivariate regression adjusting for age, gender, education, and income. Education and income groups were coded sequentially as 1, 2, 3 etc. and analysed as continuous variables to obtain the P-values for trend.

Proportional contribution of ruminant and manufactured sources to TFA intake

In 2009, FSANZ conducted modelling of the relative contribution of ruminant compared with manufactured TFA to total TFA intake in Australians.[2] It should be noted that FSANZ concluded that this part of the dietary modelling is indicative only and should be interpreted with caution, as the methods used to assess TFA in foods do not distinguish between rTFA and iTFA (section 1.2), and a number of assumptions had to be made that further contributed to uncertainty in the data. For example, in mixed food, assumptions had to be made regarding the recipe and the proportion of different foods, which directly influence the estimated proportions of each source of TFA (ruminant as against manufactured). Based on this assessment, FSANZ concluded that rTFA contributes about 60–75% of TFA intake. Further removal of iTFA from the food supply in Australia will mean that eventually rTFA should contribute to about 100% of TFA intake. For comparison, in the previously published TRANSFAIR study that assessed TFA intake in 14 western European countries, rTFA ranged between 28 and 79% of total TFA intake, with countries in the Mediterranean typically having over 50% of total TFA from rTFA.[22]

Discussion

TFA in foods

Levels of TFA in some food products sold from independent retailers and supermarkets continue to be very high, substantially exceeding the 2% iTFA limit currently enforced in Denmark. Many of these food products are popular snack foods such as meat pies, croissants and popcorn. While, in these types of foods, the exact contribution of rTFA to the total TFA can vary, overall it is likely to be low, because 1) rTFA accounts for 2–5% of total fat in ruminant products, and 2) substantial variability is observed in total TFA level, suggesting that differences are likely accounted for by the addition of iTFA. The majority of takeaway foods surveyed in Australia had TFA levels of less than 2% of total fat, and exhibited substantially less variability in TFA content compared to processed foods sold in independent retailers and supermarkets.

While limited data are available from FSANZ tracking changes in TFA level over time, these results need to be interpreted with caution, due to the differences in sampling methodology and analytical methods used to determine TFA (see Supplementary Table 1).[3] Similar limitations apply to comparisons of TFA levels in foods between FSANZ surveys and academic publications. Nevertheless, existing data suggests:

1. The levels of TFA in edible oil spreads have decreased over time, suggesting that the majority of the spreads are now made from non-hydrogenated vegetable oils. Most of this reduction likely occurred prior to 2000, when leading manufacturers voluntarily removed iTFA from edible oil spreads,[23]
2. Between 2005 and 2013 there was no clear pattern of consistent reduction in TFA level. In some categories median TFA was reduced by 50–60% in 2013 compared to 2005–2007 (for example, hot chips and potato crisps), in others median TFA increased by 150% or more (for example, chocolate biscuits and croissants), while in yet other categories, median levels of TFA seemed to randomly fluctuate between the years (for example, meat pies and prepared pastry),
3. There was a lack of data available to allow assessment of whether the variability (that is, the range of TFA in foods) has reduced over time, which would be another potential outcome of the regulatory reduction in commitment of the food industry.

Relating observed changes in TFA to voluntary reformulation to reduce TFA

The apparent lack of appreciable change in TFA level in foods over time calls into question the effectiveness of the non-regulatory approach that has been in place since 2007, which required voluntary reduction of TFA by the food industry. It should be noted that an independent review on food labelling, commissioned by the Australian Government in 2011, also specifically recommended that mandatory labelling of TFA should be implemented if TFA has not been phased out of the food supply by 2013.[24] While the FSANZ monitoring data suggest this goal was not met in 2013, there has been no further action on the part of the government to enact mandatory labelling. Based on the average TFA intake level of approximately 0.6% of energy, the Ministerial Forum questioned whether phasing out TFA was actually required and called for additional research.[25]

Further reductions in TFA from foods are likely to be feasible, especially in product categories that are currently high in TFA. This is based on the observed high variability of TFA within product categories, and the higher mean TFA level in Australian products compared to similar products found overseas.

TFA intake

The majority of Australians have a TFA intake of less than 1% of total energy, the WHO recommended upper level of intake.[26] However, around 10% of Australians continue to have intake exceeding this level. Differences in socio-demographic characteristics including education and income are associated with variability in TFA intake. Although it has been hypothesized that socioeconomically disadvantaged groups likely disproportionately represent those with above average TFA intake,[27] our analyses quantify the magnitude of this gradient and represent the most recent, nationally representative results in Australia. Finally, relative to other countries (including both developed countries with similar food supply and developing countries) the population average TFA intake level in Australia appears to be low.

Our modelling of TFA intake utilizes NNPAS data which uses the 'average' TFA level for each product category. While this is accepted methodology for estimating average TFA intake at the population level, it will not identify sub-groups who are exposed to high levels of TFA due to their preference/regular consumption of high TFA products. The extent to which a high TFA intake is possible in such subgroups in Australia is demonstrated in Table 6, using a hypothetical 'high-TFA menu'.[28] The continued existence of specific products with high TFA levels suggests that daily consumption of ~7 g/day (~3% of daily energy intake) of TFA is possible. Such high levels of TFA intake have also been demonstrated to be possible in other countries, with the notable exception in Denmark due to its regulatory limit on iTFA level in foods.[28]

Table 6. Total amount of TFA obtained from a hypothetical 'high TFA menu'

Product	Weight of product consumed per day (g)	TFA (g/100g product)*	TFA consumed (g)
Meat Pie	175	0.96	1.68
Custard baked goods	100	0.52	0.52
Popcorn	100	4.83	4.83
Total			7.03

*Based on FSANZ 2013 analyses[3]. Products with the highest TFA level in each of the product category were selected for this hypothetical menu.

4 TFA attributable mortality

In order to assess the urgency of interventions to reduce TFA consumption, it is useful to quantify the impact of the exposure on public health. For TFA, consistent evidence from metabolic trials and observational cohort studies suggest increased consumption of TFA adversely impacts lipid risk factors and relates to elevated risk of CHD death.[29] This section of the commentary details modelling we conducted to estimate the absolute number and proportional burden of CHD mortality attributable to TFA intake in Australia.

Methods

Exposure and relative risks

We followed methods previously employed by the Global Burden of Disease (GBD) study.[16] In essence, potential impact fractions (PIF, see below) are calculated by comparing two scenarios: 1) the current level of TFA intake in Australia estimated using the 2011–2012 NNPAS (see section 1.6), and 2) a counterfactual (hypothetical) setting where TFA consumption in Australia was lowered to a 'theoretical minimum distribution'. The interpretation of the PIF is the proportion of heart disease deaths avoided if the Australian population would always have been exposed to TFA at the level of the theoretical minimum instead of the current observed level.

To implement this modelling, TFA was treated as a continuous exposure with a lognormal distribution because TFA consumption cannot be negative and the distribution is skewed to the right. We defined the theoretical minimum distribution for TFA with a mean of 0.5% of energy intake, with a standard deviation of 0.05, which was the theoretical distribution assumed by the GBD nutrition and chronic disease expert group.[16, 30] The 0.5% energy from TFA was chosen because iTFA is linearly associated with CHD risk and therefore its consumption should be minimized, and if iTFA was eliminated completely from the food supply, then all of the TFA would come from ruminant sources.[16, 30] In Australia, 0.5% of energy from TFA appears to be a reasonable level to set for the theoretical minimum distribution: taking the mean of 0.6% of energy from total TFA currently based on the NNPAS, and the estimates by FSANZ that rTFA contribute up to 75% of total TFA intake in Australia, then elimination of iTFA would leave mean rTFA intake of 0.6% x 75%, or approximately 0.5% of energy. Globally, this value for the theoretical minimum distribution is also consistent with the three lowest TFA intakes observed in the GBD study (Barbados, Finland, and Italy all presented intakes of less than or equal to 0.5% energy from TFA).

The relation between TFA intake and CHD mortality was modelled as a 'per unit' relative risk, which was based on meta-analyses of the observed association between TFA intake and CHD mortality in prospective cohort studies. The relative risks for heart disease mortality for each 2% point increase in energy intake from TFA are shown in Table 7.

Table 7. Relative risk (and 95% confidence interval) for heart disease mortality for each 2% point increase in energy intake from trans-fatty acids, by age group[16]

	25-34	35-44	45-54	55-64	65-74	75 and over
RR	1.42	1.40	1.33	1.27	1.22	1.16
LCI	1.28	1.27	1.22	1.18	1.15	1.11
UCI	1.57	1.54	1.45	1.36	1.29	1.21

LCI: lower confidence interval. UCI: upper confidence interval.

We used CHD mortality as assessed by the GBD 2010 study.[31] These numbers are higher than what is reported by the Australian Bureau of Statistics in 2010 because the GBD reassigns deaths that have been coded from so-called 'garbage codes' to what is assumed to be the most likely real cause.[31]

Potential impact fraction

The potential impact fraction (PIF) calculates the proportional change in disease incidence (or mortality) after a change in risk factor exposure in the population as a whole. It takes risk factor exposure and the relative risk of the disease as its inputs. For a continuous risk factor distribution the equation is[32]:

$$PIF = \frac{\int_a^b RR(x)P(x)dx - \int_a^b RR(x)P^*(x)dx}{\int_a^b RR(x)P(x)dx}$$

Where *x* is the risk factor level, *P(x)* is the original risk factor distribution, $P^*(x)$ is the risk factor distribution after the change, *RR(x)* is the relative risk as a function of risk factor exposure level, *dx* denotes that the integration is done over *x*, and *a* and *b* are the integration boundaries. For the present study, we calculate PIFs by age group and sex with the observed distributions of TFA exposure and the theoretical minimum distribution. The *RR(x)* function is implemented using the "per unit" relative risks of Table 1, which implies that the relative risk increases exponentially with increasing exposure.

Calculation methods and uncertainty

Calculations are done in MS Excel, with the help of two add-in programs. The EpigearXL add-in provides functions to calculate the integrals of the multiplication of a lognormal distribution with a "per unit" relative risk function. The Ersatz add-in allows doing Monte Carlo simulation in Excel. Both add-in programs are available from www.epigear.com. Two of the inputs, the relative risks and the exposure have sampling uncertainty. We assume, by virtue of the central limit theorem, that the mean exposure has a normal distribution with the standard error of the mean as its standard deviation. For the relative risks we use Ersatz's ErRelativeRisk function.[33] The 2010 heart disease mortality numbers have uncertainty as well,[31] but since these are population numbers the uncertainty is not sampling-related (it pertains to coding practices and such) and it is therefore not included in the uncertainty analysis. We did a Monte Carlo simulation with 2000 iterations to quantify the effect of the sampling uncertainty on the attributable mortality.

Results

Table 8 shows the results of the TFA attributable heart disease mortality for the base year of 2010. The attributable deaths were overwhelmingly in the over 75 age group, but more so for women than for men. Overall, Australia had 487 heart disease deaths due to TFA exposure in 2010. This is about 1.52% (95% uncertainty limits: 1.15% – 1.92%) of all heart disease mortality.

Table 8. Attributable TFA heart disease mortality in 2010, by sex and age group and in total, with 95% uncertainty intervals

	Males	95% UI		Females	95% CI		Total	95% CI	
Age	**Mean**	**LUI**	**UUI**	**Mean**	**LUI**	**UUI**	**Mean**	**LUI**	**UUI**
25-34	1	1	1	0	0	1	1	1	2
35-44	5	4	7	1	1	2	7	5	9
45-54	14	10	19	3	2	4	18	12	23
55-64	34	23	44	8	5	11	42	28	54
65-74	45	31	59	20	14	26	65	45	85
75+	161	108	221	193	128	263	355	239	480
Total	260	202	326	226	162	297	487	367	615

LUI: lower uncertainty interval. UUI: upper uncertainty interval.

Discussion

Our results show that about 1.5% of all heart disease mortality in 2010 was attributable to TFA exposure. In absolute terms, this equates to almost 500 deaths because heart disease is a major cause of death in Australia.

Our current assessment represents an updated analysis of the Global Burden of Disease study by Wang et al.[16] They found a much higher percentage of heart disease mortality attributable to TFA: 5.9% (95% CI, 5.5% – 6.4%). The difference seems entirely due to a different estimate of exposure: they estimated that on average 1.3% of energy intake was due to TFA in 2010; when we put this exposure into our calculation it returns a comparable attributable percentage of 6.0% of heart disease mortality. The large differences in the estimated mean TFA consumption are likely due to major methodological differences in dietary assessment methods. In the current analysis, we utilized the most up-to-date individual level, nationally representative NNPAS dietary survey data in 2011–2012, which also took into account the level of TFA in different foods using the relatively recent 2009 FSANZ TFA in foods survey. In comparison, The Global Burden of Disease study estimated TFA intake in Australia using statistical modelling, which relied on inputs that included the 1995 Australian Dietary survey, country and region level covariates such as lagged distributed income, and food disappearance data.[16, 30] Authors of the GBD paper noted that a limitation of their study was that TFA consumption data was relatively limited in most nations compared with other major dietary factors.[16, 30] While the NNPAS survey has its own methodological challenges, it is likely to more accurately quantify current mean TFA consumption in Australia.

The estimate in this report calculates attributable deaths using population attributable fractions (PAFs). A number of limitations should be noted. Due to its underlying assumptions, the PIF methodology does not take into account competing risk factors and overestimates the proportion of death attributable to a single risk factor. As a consequence, our attributable deaths will be overestimated, although this effect is rather small for small risk factors like TFA. A second reason why PAFs cause overestimation is that it is a static calculation, that is, it assumes that the counterfactual population has always been exposed to the theoretical minimum level of TFA. In a real population, a one-off change from the current level to the theoretical minimum would imply time lags in the decrease in heart incidence disease risk, and longer ones (because of the existing pool of prevalent cases) in heart disease mortality. It should also be noted that the attributable deaths calculation assumes that TFA intake can be reduced to the theoretical minimum, while in practice policy interventions may not be able to achieve this. Finally, attributable deaths only captures one dimension of the health impact of TFA consumption, but does not inform other important public health parameters including age at death, life years lost, morbidity, health adjusted years of life lost, which are outside the scope of the current commentary but should be the focus of future analyses.

In conclusion, the relative impact of TFA exposure on heart disease mortality in Australia is limited, but in absolute terms still substantial. Policy making to reduce exposure may therefore be desirable, especially as currently TFA levels continue to be high in some food products in Australia (see section 1). Our determination of the attributable deaths due to TFA is an important first step, but in order to further inform policy priorities additional analyses with richer outcomes are needed such as health adjusted life years gained for specific interventions, and the cost and cost-effectiveness of such interventions.

5 Impact of policies to reduce TFA consumption

This section of the report provides an overview of the existing evidence of the impact of TFA policy approaches to reducing TFA intakes worldwide. A systematic review of the literature from 2013 onwards was conducted. This review builds on a previously published review that examined the evidence of the impact of trans fat policies worldwide from 2000 to2012[34]. This section updates that review by examining the literature from 2013 to 2016. A brief synthesis of the evidence compiled from both reviews is reported in the Study quality assessment section.

Systematic review methods

Search terms and databases

A systematic literature search was conducted using the Medline, Embase and Cinahl databases to identify peer-reviewed articles that examined the impact of TFA policy in March 2016. The main search terms were 'trans fat' and 'policy'. Additional search terms to represent TFA were: 'trans fatty acids', 'hydrogenation', 'vanaspati', 'elaidic acid' and 'margarine'. Policy terms included 'regulation', 'nutrition policy', 'health policy', 'legislation', 'ban', 'intervention', 'labelling', 'law', 'standards' and 'restriction'. Terms were included both as key words as well as free text searches in titles and abstracts, depending on the database being searched. The search terms and databases used were the same as the previously published systematic review. Although the grey literature was examined in that review, given time constraints only the peer-reviewed literature was included in this review.

Inclusion criteria

The inclusion criteria for the review were that the studies were: 1) empirical, 2) examined a TFA policy (labelling, voluntary limits, bans, etc.), and 3) examined the effect of the policy on TFA levels/availability (in food, the blood, dietary consumption, breast milk, etc.). Unlike the previous review, modelling studies of TFA policies were included to enable the inclusion of studies that examined: 1) differences in policy impacts on specific segments of the population (for example, lower socioeconomic groups), and 2) cost-effectiveness studies. Studies that modelled the potential impact of front-of-pack labelling on TFA intakes were not included. Given that the previous review did not include modelling studies, the modelling studies published in 2000–2012 were added as part of the updated review.

In addition to the aforementioned inclusion criteria, only studies that had pre and post data were included in the review. This differed from the previous review, which included studies even if they solely presented findings post policy intervention.

Overview of search

The records identified through our searches were exported into EndNote, version X5 (Thomson Reuters, Philadelphia, PA, 2011) and duplicates were subsequently removed. The records were then imported into Covidence (Veritas Health Innovation, Deerfield, IL) — an online software product to facilitate the systematic review process — to screen the records. Two researchers (SD and MB) independently reviewed the titles and abstracts of all records identified in our search. After removing records that did not meet the inclusion criteria, the full texts of the remaining articles were reviewed by both researchers to determine whether the studies met our inclusion criteria. In the event that the two researchers disagreed, a third researcher (JW) was consulted and a decision reached by consensus. The reference lists of the full articles were also examined in order to identify additional studies that met the inclusion criteria.

Study extraction

One researcher (MB) extracted data from the full texts of the articles included in the review. The data extractions were then reviewed by a second researcher (SD). The following data were extracted from each study: aim, location, design, type and year of intervention, data collection period, population, sample size, outcomes measured and their assessment method, results, summary of findings, conclusions, limitations, assumptions (for modelling studies) and source of funding.

Study quality assessment

In order to assess the overall quality of the studies that collected primary data (that is, non-modelling studies) included in the review, the Effective Public Health Practice Project (EPHPP) Quality Assessment Tool [35] was used. We adapted the tool to reflect the study designs included in the review. The tool is designed to assess study quality based on six domains: selection bias, study design, confounders, blinding, data collection method, and withdrawals/dropouts. Given the types of studies included in the review, blinding and withdrawals/dropouts were not included in our quality assessment. Each study was given a ranking of weak (score=1), moderate (score=2) or strong (score=3) for each of the remaining four domains based on the EPHPP Quality Assessment Tool's dictionary[35, 36]. An overall score was then generated by compiling the scores for each domain and assigning an overall study quality rating of weak, moderate or strong. If a given study had two or more weak ratings it was considered weak, if it had one weak rating it was considered moderate, and if it had no weak ratings it was considered strong. The quality assessment tool used in this study was more extensive than that used in the previous review. For the 2000–2012 review, study evidence was assessed based on previously published dimensions of evidence, largely focussed on study design ranked from I (strongest) to IV (weakest)[37].

Findings of systematic review

Overview of included studies

Figure 3 depicts the search flow diagram of this review. We searched the full text of 37 articles (35 identified in our search and 2 modelling studies from 2000—2012), of which 12 were included in the review. The main reason for excluding articles based on the full text review was because they were: duplicates (n=3), included in previous review (n=5), had no TFA policy (n=3), were a commentary (n=3) or did not have a pre-post study design (n=11).

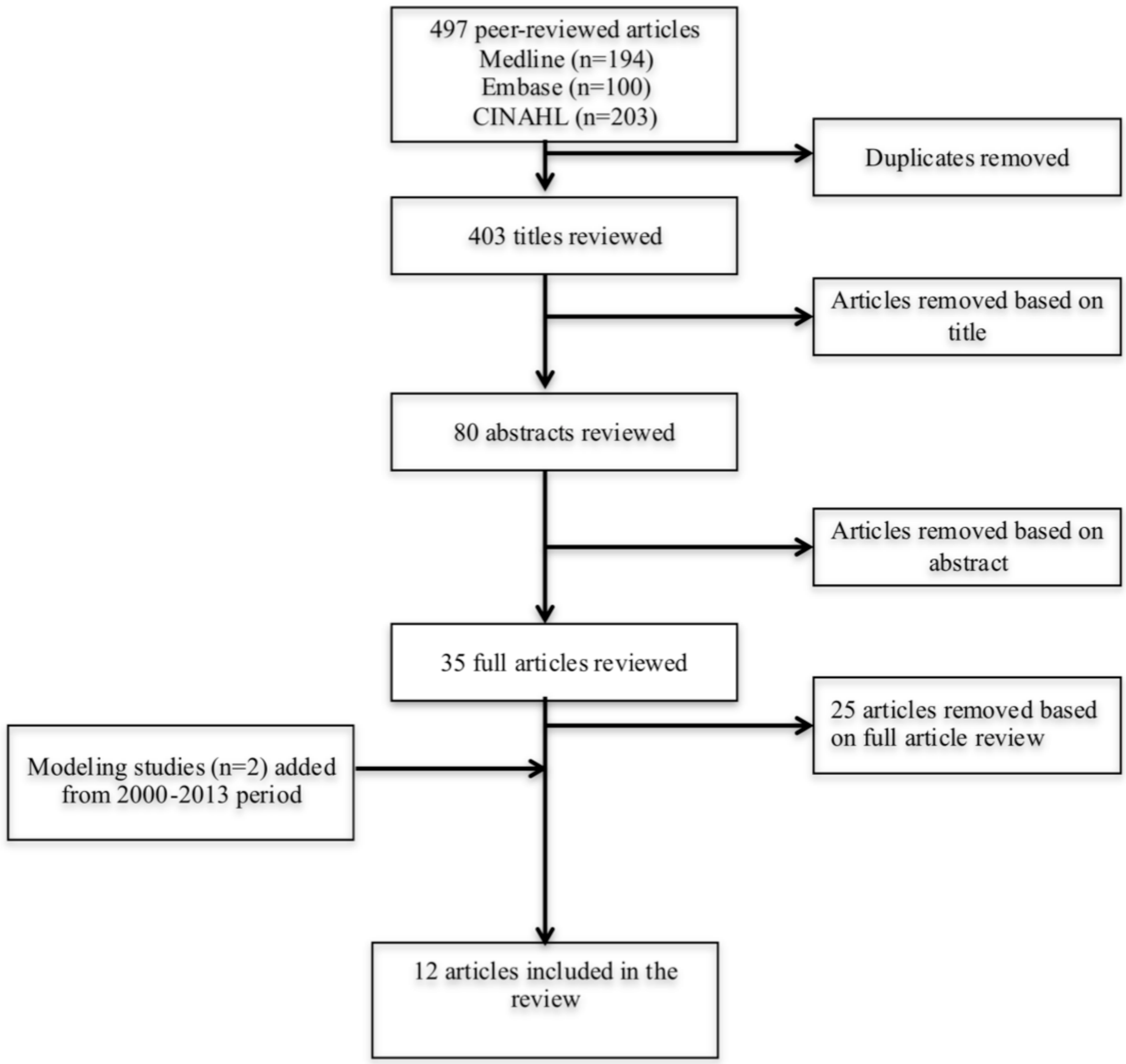


Figure 3. Systematic review flow chart

Findings of modelling studies (2000–2016)

Seven modelling studies were included in the review (Table 9). The majority of the modelling studies were conducted in the UK (n=5), whilst one was conducted in Argentina and another in Denmark. Four of these studies [38-41] were based on reducing TFA intake by a specific percentage of total energy rather than a specific policy approach. Moreover, three of these studies [38-40] examined several food policies simultaneously. In these cases, only results related to the TFA reduction were reported in this review.

Although the modelling studies were not directly comparable given their varying time horizons, all the studies reported a reduction in CHD/CVD deaths attributed to TFA policies. Of the modelling studies conducted in the UK, annual deaths averted with TFA policies ranged from 112 deaths (with a 0.5% reduction in TFA) [40] averted in 2010 to between 2700 [38] and 3900 [41] deaths averted annually (with a 1% reduction in TFA). For the UK modelling studies that examined CVD deaths averted over a longer time horizon, one study found that between 2006–2015, 3500–4700 CVD deaths would be averted [39] and another found that 7200 deaths would be averted between 2015-2020 [42]. The modelling studies (n=3) that included a cost-effectiveness component all reported that TFA policies would lead to cost savings. Two studies [41, 42] examined the effects of a TFA policy on different socioeconomic groups. Both studies were conducted in the UK and found TFA policies to have a greater effect on lower socioeconomic groups. The first suggested that a TFA ban would reduce inequality in mortality from CHD by 15%, given the higher TFA intakes among lower socioeconomic groups[42]. The second study found that five times as many CHD deaths would be prevented in the lowest quintile of socioeconomic status – they would also gain six times as many life years as compared to the most affluent group[41].

Two modelling studies, one in Argentina [43] and another in Denmark [44], modelled the impact of the countries' actual TFA policies, both showing positive impacts in terms of reducing CHD/CVD rates. Over the course of a ten-year period between 2004 and 2014 Argentina went from having a voluntary TFA labelling policy to having mandatory TFA limits in foods. This study modelled several likely scenarios through linking together different assumptions of the likely effect of TFA on CHD outcomes (for example, based on observed relationship in observational studies as against the effect of TFA on CHD blood lipid risk factors from RCTs) and baseline levels of TFA intake (mean=1.5% of total energy vs. lower bound of 1% and upper bound of 3% of total energy) [43]. Across all the scenarios modelled, TFA regulation had a substantial impact leading to significant reductions in CHD deaths (301 to 1517) and CHD events (1066 to 5373), disability adjusted life years (DALYs) (5237 to 26,394) and health care costs saved (US$17 to US$87 million) each year[43]. In Denmark, a synthetic control group using CVD mortality data from OECD countries without TFA policies was used to examine what the CVD mortality rate would have been if the country did not adopt a TFA 'ban' in 2004[44]. They found that CVD mortality decreased by approximately 14.2 deaths per 100,000 people per year in Denmark with the TFA policy as compared to the counterfactual of not having a TFA policy.

Table 9. An overview of the modelling studies included in the systematic review

Authors, Year and Country	Model type	Study population	Aim of study	TFA policy	Outcomes assessed	Main findings
O'Keeffe, Celine et al.[40] 2013 Republic of Ireland	Validated IMPACT Food Policy Model	Population of Ireland aged 25-84y	To estimate the potential reduction in CVD mortality by decreasing salt, trans fat and saturated fat consumption, and by increasing fruit and vegetable consumption in Irish adults aged 25-84 years in 2010	Conservative policy scenario: reductions in TFA by 0.5% of energy intake More substantial policy scenario: reductions in TFA by 1% of energy intake	CHD and stroke deaths averted	Conservative TFA policy scenario: 112 CVD deaths averted (28% of the total CVD deaths (n= 1070) of combined policy approach) More substantial but feasible TFA policy scenario: 225 fewer CVD deaths (21% of the total CVD deaths (n= 1070) of combined policy approach)
O'Flaherty et al.[39] 2012 United Kingdom	Spreadsheet model	Population of UK aged 25-84y	To estimate how much more CVD mortality could be reduced in the UK through more progressive nutritional targets from 2006 to 2015	Conservative policy scenario: reductions in TFA by 0.5% of energy intake More substantial policy scenario: reductions in TFA by 1% of energy intake	Total CVD deaths averted	Conservative TFA policy scenario: Reduction of 3500 deaths (min 1820, max 6820) More substantial TFA policy scenario: Reduction of 4700 deaths (min 2500, max 8800)
Barton et al.[38] 2011 England and Wales	Spreadsheet model	Population of England and Wales aged 40-79y at time of intervention	To estimate the potential cost effectiveness of a population-wide risk factor reduction program aimed at preventing CVD	Legislation to reduce iTFA intake by approximately 0.5% of total energy content	CVD events avoided, QALYs gained, and savings in healthcare costs for a given effectiveness	2700 deaths annually would be averted. A gain of 570 000 life years, which translates in savings the equivalent of approximately £235m per year. An intervention costing £230m per year would be cost saving.
Pearson-Stuttard et al.[41] 2015 England and Wales	Validated IMPACT-SEC model (named IMPAC-TFA Model)	Population of England and Wales aged ≥25y stratified by age, sex and SEC	To examine the potential effects of reductions of TFA on CHD mortality, CHD related hospital admissions in 2016, 2020 and 2030	Conservative policy scenario: reductions in TFA by 0.5% of energy intake More substantial policy scenario: reductions in TFA	Deaths prevented or postponed, life years gained and hospital	Conservative policy scenario: 1,900 fewer deaths, 4,900 fewer hospital admissions and 19,000 life years gained. More substantial TFA policy scenario: 3,900 fewer deaths, 10,000 fewer hospital admissions and 37,000 life years gained per

				by 1% of energy intake	admissions.	year. Health inequalities would also decrease, five times as many deaths would be prevented and six times as many life years would be gained in the most deprived quintile compared with the most affluent.
Allen, Kirk et al.[42] 2015 England	Validated IMPACT-SEC model	Population of England aged ≥25y stratified by age, sex and SEC	To determine health and equity benefits and cost effectiveness of policies to reduce TFA from processed foods, compared with consumption remaining at the most recent TFA levels in England from 2015 to 2020	Total ban on trans fatty acids in processed foods; improved labelling of trans fatty acids; bans on trans fatty acids in restaurants and takeaways.	Deaths from CHD prevented or postponed; life years gained; QALYs; policy costs to government and industry; policy savings from reductions in direct health care, informal care and productivity loss.	Total ban would prevent or postpone about 7200 deaths from CHD in 2015-2020. This would reduce inequality in mortality from CHD by ~3000 deaths (15%). Improving labelling or removing trans fatty acids from restaurants/fast food could save between 1800 and 3500 deaths – inequalities would be reduced by 600 (3%) to 1500 (7%). A total ban would have the greatest net cost savings of about £265 million as compared to £80-147m for the other policies.
Rubinstein et al. 2015[43] Argentina	Policy model	Entire population of Argentina	To estimate the impact of Argentine policies to reduce TFA on CHD, DALYs and associated healthcare costs from 2004 to 2015	Progressive TFA policies actually implemented (voluntary labelling (2004), mandatory labelling (2006), 2% TFA limit in fats (2012) and 5% TFA limits in all foods (2014-15): Three scenarios: Scenario 1: Based only on the effect of TFA replacements on the ratio	CHD events averted. DALYs saved and costs saved	Based on projected changes in lipid profile, 301 deaths, 1066 acute CHD events and 5237 DALYs would be averted. US$17 million in health-care costs annually would be avoided. Based on the adverse effects of TFA intake reported in prospective cohort studies, 1517 deaths, 5373 acute CHD events and 26,394 DALYs would be averted annually. US$87 million in health-care costs annually would be avoided.

				of TC/HDL-CScenario 2: Scenario 1 plus the effects of TFA replacements on other CHD biomarkers in controlled trialsScenario 3: Based on the observed relationship of TFA replacements with clinical CHD events in prospective cohort studies		
Restrepo & Rieger[44] 2016 Denmark	Empirical model	Populations of OECD countries from 1990 to 2012	To assess whether Denmark's trans fat policy reduced deaths caused by CVD using synthetic control methods to simulate the CVD mortality trajectory (using that of other OECD countries) that Denmark would have witnessed in the absence of the policy	Trans fat ban (2% upper limit) in all products	CVD mortality in Denmark, with and without the TFA policy. Mortality attributed to CVD.	Before the trans fat policy was implemented, CVD mortality rates in Denmark closely tracked those of a weighted average of other OECD countries (i.e., the synthetic control group). In the years before the policy, the annual mean was 441.5 deaths per 100,000 people in Denmark and 442.7 in the synthetic control group. In the 3 years after the policy was implemented, mortality attributable to CVD decreased on average by about 14.2 deaths per 100,000 people per year in Denmark relative to the synthetic control group.

TFA = trans fatty acid, CVD = cardiovascular disease, CHD = coronary heart disease, QALY = quality adjusted life year, SEC = socio-economic circumstances, DALY = disability adjusted life year, TC/HDL-C = total cholesterol/high density lipoprotein cholesterol, OECD= Organisation for Economic Cooperation and Development

Quality of modelling studies

Supplementary Table 3 provides an overview of the main assumptions of the models included in the review. There are several limitations associated with the assumptions of the various models. The time horizons used for the models were short. In one study [40] the deaths averted were only examined for one year. Longer time horizons would be more appropriate as the implementation of a TFA policy would not result in instantaneous effects on CVD. Some of the modelling studies did not address current levels of TFA intake, which may in part be explained by limited data availability; however, this has significant implications in terms of model assumptions. For example, in the UK average TFA intakes are already low (0.8% total energy) [20] so it may not be possible to reduce TFA by an additional 1% of total energy intake, as was examined in three of the models[38, 40, 41]. TFA policies would only apply to industrially produced TFA rather than all TFA given the lack of feasibility in terms of implementing a policy that addresses ruminant TFA levels in the food supply. It is therefore unlikely that TFA intakes can be reduced to 0% of total energy intake in the absence of avoiding all ruminant products. Moreover, another area of uncertainty in the models is the assumption that reducing ruminant TFA would have the same effect on CVD risk as reducing industrially produced TFA — existing evidence on the cardiovascular effects of rTFA remains mixed and uncertain [45, 46].

TFA interventions in the ‘real world’ (2013–2016)

Five new studies that met our inclusion criteria were identified through our searches that examined the impact of TFA policies in a ‘real world’ context. These studies examined voluntary self-regulation (Costa Rica)[47], mandatory labelling (US)[48], mandatory labelling in addition to voluntary TFA limits (Canada) [10, 49] and mandatory TFA limits in restaurants (NYC)[50]. These studies examined a variety of outcomes, including TFA levels in foods[10], TFA intakes[47-49], TFA levels in breast milk [49] and hospital admissions due to myocardial infarction (MI) or stroke [50]. In addition, three studies [10, 47, 48] examined changes in other fats (including saturated fat) in response to TFA regulation. Table 10 provides an overview of the studies included in the review. In terms of the overall quality of the studies included in this section of the review, all but one of the studies[48] included in the review were weak. The main contributing factors to the studies being deemed weak by the EPHPP quality assessment tool were related to the high selection bias and data collection methods that lacked strong validity and/or reliability. However, it is important to note that these studies were examining real policies that have been implemented worldwide. It is not possible to use more rigorous study designs, such as randomized control trials, to examine these policies in a ‘real world’ context. We only included studies with pre and post TFA policy implementation data to ensure that we included higher quality studies; however, these studies still had limitations.

Table 10. Overview of the 'real life' TFA policy studies included in the systematic review

Authors, Year and Country	Study design	Study population and sample size	Aim of study	TFA policy	Main outcomes assessed	Main findings	Study quality
				Voulntary self-regulation			
Monge-Rojas et al.[47] 2013 Costa Rica	Pre-post cross-sectional	Adolescents aged 12 to 17 years living in San Jose, Costa Rica in 1996 (n=276) and 2006 (n=133)	To identify how dietary intake and food sources of SFA, PUFA and TFA in the diet of Costa Rican adolescents changed from 1996 to 2006	Voluntary self-regulation	Total fat, SFA, TFA, PUFA and MUFA	In 1996, 100% of adolescents exceeded the WHO TFA recommendations (<1% of total energy intake from TFA). By 2006, 68% of adolescents exceeded the limit. Both SFA and TFA were significantly lower in 2006 (9.5% and 1.3% respectively) as compared to adolescents in 1996 (12.1% and 2.1%). Total fat and MUFAs (8.2% to 10.6%) and PUFAs intakes increased from 1996 to 2006 (5.5% to 7.5%).	Weak
				Mandatory TFA limits in restaurants			
Brandt et al.[50*] 2015 USA	Pre-post cohort study	NYC residents Census data from 2000 and 2010 were used for hospital admissions. 2004 NYC HANES data were used to examine restaurant usage per week.	To assess MI and stroke rates before and after the TFA limits in New York City restaurants	Mandatory 5% TFA limits in restaurants	Hospital admission due to MI or stroke and restaurant usage	After 2007, younger age groups (25-34 and 35-44) experienced an additional decline in stroke, but not MI, that was greater than would have been expected based on temporal trends. Younger age groups also reported higher mean restaurant use in NYC HANES.	Weak
				Mandatory TFA labelling on packaged foods			
Storey &	Interrupted time series	Children aged 6-11 years,	To examine the trend in energy and	Mandatory TFA	Energy and fatty acid intake.	Overall, intakes of total energy, total fat, SFAs, and monounsaturated fatty acids (MUFAs) decreased	Moderate

Andersen[48] 2015 USA	(2005-6, 2007-8, 2009-10).	adolescents aged 12-18 years and adults aged under 19 years. Pregnant and lactating women were excluded 2005-2006: children (n=1009), adolescents (n=1853), adults (n= 4367) 2007-2008: children (n=1117), adolescents (n=999), adults (n= 5411) 2009-2010: children (n=1147), adolescents (n=1097), adults (n= 5784)	fat intakes as well as SFA and TFA intakes from French fries among children, adolescents, and adults using the 3 most recent waves of NHANES.	labelling on packaged foods	Intakes of SFAs and TFAs from fried French fried potatoes	significantly between 2005–2006 and 2009–2010 among children and adolescents; however, PUFA intakes did not change. Among adults, intakes of total fat, SFAs, and MUFAs decreased; however, total energy and PUFA intake did not change. Intakes of SFAs and TFAs from fried French fried potatoes decreased significantly between 2005–2006 and 2009–2010 among children (SFA 0.3g/d vs 0.2g/d; TFA 0.4g/d vs 0.01 g/d), adolescents (SFA 0.5g/d vs 0.2g/d; TFA 0.6g/d vs 0.01 g/d), and adults (SFA 0.3g/d vs 0.1g/d; TFA 0.3g/d vs 0.01 g/d).	
Mandatory TFA Labelling + Voluntary TFA limits							
Ratnayake et al.[49] 2014	Pre-post cross sectional study (1992,	TFA content in breast milk samples collected in 2009 (n=153), 2010 (n=309), and	To assess the impact of the efforts in Canada to reduce industrial TFAs in foods, the	Mandatory TFA labelling + voluntarily TFA limits	TFA intake of breastfeeding women and TFA content of breast	TFA contents of total milk fat were 2.8% in 2009, 2.3% in 2010 and 2% in 2011 as compared to 7.2% found previously for Canadian breastmilk in 1992. TFA intake of Canadian breastfeeding mothers was 0.9%,	Weak

Canada	2009, 2010, 2011)	2011 (n=177) from breastfeeding mothers in 10 major Canadian cities. These were compared to breastmilk samples 1992 (n=198).	concentration of TFAs in human breast milk samples was measured	(2% for oils/fats and 5% for packaged food) in foods	milk	0.5%, and 0.3% of total energy in 2009, 2010, and 2011, respectively.	
Arcand et al.[10] 2014 Canada	Pre-post cross sectional study	3 databases were used. Packaged foods: 1) Foods (n=5544) from 3 grocery stores in Toronto, 1 in Calgary (2010-11) and 2) foods from the Trans Fat Monitoring Program (TFMP) database (2006-9) of foods that contribute the most to the Canadian diet. Restaurant foods: 4272 foods from 85 Canadian restaurants with >20 outlets nationwide (2010).	To conduct an updated assessment of TFA levels in the food supply and to determine whether they have been replaced by SFAs	Mandatory TFA labelling + voluntarily TFA limits (2% for oils/fats and 5% for packaged food) in foods	Foods that met recommended limits for TFA and TFA in foods.	In 2005-2006, 75% of foods met the TFA limits as compared to 95.4% of packaged foods and 96.1% of restaurant foods in 2010-2011. Foods with the greatest improvements were: croissants (25% to 100%), pies (36% to 98%), cakes (43% to 90%), garlic spreads (33% to 100%), and garlic bread (55% to 91%). However, the number of products meeting the TFA limits decreased in 3 product categories: coffee whiteners (53% to 33%) and lard (100% to 75%) and vegetable (50% to 40%) shortenings. Packaged foods 2010-2011: Foods that exceeded the recommended TFA limits were: dairy free cheeses (100%), frosting (72%), coffee whiteners (66.7%), Mexican meal kits (62.5%), lard and shortening (55.5%), shortbread cookies (41.7%) and refrigerated dough (50%). Many packaged foods that exceeded the TFA limits contained very high mean quantities of TFA as a % of total fat: coffee whiteners (38.3%), yeast doughnuts (35%), popcorn (33.9%), frosting (28.6%), cake doughnuts (27.7%), dairy-free cheese and spreads (27.5%) and sugar wafer cookies (25.0%). Restaurant foods 2010: biscuits and scones (47.4%) and cookies (14.7%) were categories with the highest proportion of foods exceeding the TFA limits. SFAs: Most food categories did not have increases in SFA	Weak

						between those foods that met the TFA limit. Packaged food exceptions included: chocolate chip, chocolate-covered and sandwich cookies, brownies, squares, cakes with pudding/mousse, dessert toppings and lard/shortening. Restaurant food exceptions included: cookies and desserts and other baked goods.	

*The full-text of this article was not available. These findings are based on a published abstract.

TFA = trans fatty acid, SFA = saturated fatty acid, PUFA = polyunsaturated fatty acid, MUFA = monounsaturated fatty acid, WHO = World Health Organization, NYC =, New York City, (N)HANES = (National) Health and Nutrition Examination Survey, MI = myocardial infarction

The impact of TFA interventions in the 'real world' (2013–2016)

All five studies found a positive impact of TFA policies; however, given the differences in outcome measures reported we were unable to pool these results. Overall, mandatory TFA labelling coincided with reductions in the TFA levels in foods in both Canada in the US[10, 48]. For example, in Canada 75% of foods met the voluntary TFA limits (2% of total fat in fats/oils and 5% in packaged foods) in 2005–06 when the TFA policy first came into effect but by 2010–11, 95.4% of all packaged and 96.1% of restaurant foods met the recommended limits[10]. However, it is important to note that TFA levels remained high in some product categories. For example, TFA content in coffee whiteners was on average 38.3%[10]. Moreover, in two product categories (coffee whiteners and lard/vegetable shortenings) the number of products meeting the voluntary TFA limits actually declined between 2005–09 and 2010–11[10].

The three studies[47-49] examining the association between TFA policies and intakes found reductions in TFA intakes after the policy implementation. In Costa Rica, where there was industry self-regulation, the number of adolescents that exceeded the WHO recommendations (less than 1% of total energy intake from TFA) for TFA consumption declined by 32 percentage points between 1996 and 2006; however, the percentage of adolescents exceeding the intake limit remained high (68%)[47]. A study in the US examining TFA intakes from French fries found a reduction in intakes between 2005–6 and 2009–10[48] while a Canadian study found a reduction in TFA intakes in breastfeeding women (from 0.9% of total energy in 2009 to 0.3% in 2011) as well as reductions in TFA levels in their breast milk (from 7.2% of total milk fat in 1992 to 2% in 2011)[49].

In addition to the studies examining TFA levels in foods and their intake, one study looked at the impact of New York City's (NYC) TFA limits in restaurants on hospital admissions due to MI or stroke. That study found that younger age groups, which also reported higher restaurant use, experienced an additional decline in stroke, but not MI, which was greater than would have been expected based on temporal trends after the policy implementation[50].

There is often a concern that if TFA is removed from a product it will be replaced with saturated fatty acids (SFA), decreasing the potential health benefits of product reformulation. This could potentially lead to increased SFA intakes; however, we did not find evidence to support this possibility. Two studies [47, 48] found a decrease in SFA intakes coinciding with TFA policy. Table 11 provides an overview of the changes in fatty acid intakes after the introduction of a TFA policy. A third study in Canada that compared the SFA levels in foods that met the voluntary TFA limits to those that did not, found that SFA levels were not higher in the low TFA products for the majority of food categories; however, there were exceptions[10]. In Australia, FSANZ data also indicated there was no appreciable change in SFA level between 2008 and 2013 in the food products examined.[3] However, as discussed in section 1.5, there was also no meaningful change in TFA in products in Australia across this time span.

Table 11. Changes in the consumption of fatty acids following the implementation of TFA intervention

Policy Intervention	Authors	TFA	SFA	MUFA and/or PUFA	Total Fat
Voluntary self-regulation	Monge-Rojas et al.[46] 2013 **Costa Rica**	↓	↓	↑	↓
Mandatory TFA limits (bans)	Storey & Anderson[47] 2015 **USA**	--	↓	↓ MUFA NC PUFA	↓
Mandatory TFA Labelling + Voluntary Limits	Ratnayake et al.[48] 2014 **Canada**	↓	--	--	--

Synthesis of the evidence from 2000–2016

Supplementary Table 4 provides a summary of the studies (n=14) from the previous 2000–2012 review of the impact of TFA policies that had a pre and post study designs[34]. The findings of the updated review are consistent with those from the earlier review. Supplementary Tables 5 and 6 update the synthesized evidence from the 2013 review on the number of TFA free products and the changes in the fatty acid composition of foods after the implementation of TFA policy, respectively. TFA policies coincide with reduction in TFA levels in foods as well as intakes. Moreover, it is likely that they contribute to reductions in CVD risk and are cost saving, as demonstrated in the modelling studies included in this review. There is also some evidence from the NYC study to suggest that admissions due to stroke decreased in younger populations after the implementation of the TFA limit in restaurant foods[50]. The totality of the evidence from both modelling studies as well as the studies conducted in 'real world' settings suggest that stronger TFA policies will have a larger impact on TFA levels in foods, their intake and CVD outcomes.

Discussion

The evidence of the impact of TFA policies suggests that although all policy approaches will likely lead to reductions in TFA levels in foods and subsequent intakes, stronger policies will likely have an even more pronounced effect. Although the evidence suggests that labelling policies lead to the reduction of TFA in the food supply, high TFA products remain. In Canada, although most foods have low levels of TFA, foods with very high levels of TFA continue to be available in the food supply. It is likely that the same situation exists in other countries worldwide. In order to ensure that all foods have low levels of TFA, 'bans' or mandating upper limits on the allowable quantity of TFA are likely necessary. In recognition of the need to ensure that the food supply as a whole is low in TFA, the US Food and Drug Administration (FDA) has removed the "generally recognized as safe" (GRAS) status from partially hydrogenated oils in the US which, once implemented, will essentially act as a countrywide iTFA ban.

Given the results of the modelling studies included in this review, it is likely that policies that set TFA limits will likely have the greatest impact on low socioeconomic groups. Labelling policies may not deliver for lower socioeconomic groups given that high TFA products will likely remain in the food supply and these foods tend to be cheaper than their low TFA counterparts[51, 52]. In order to reach the populations that would benefit the most from a reduction in TFA intakes, bans or upper limits will likely be necessary.

6 References

1. Food Standards Australia and New Zealand. Trans fatty acids reports. 2007. [Access Date: 5/4/2016]. Available from: http://www.foodstandards.gov.au/publications/Pages/transfattyacidsrepor4561.aspx
2. Food Standards Australia and New Zealand. Trans fatty acids reports. 2009. [Access Date: 5/4/2016]. Available from: http://www.foodstandards.gov.au/publications/Pages/transfattyacidsrepor4560.aspx
3. Food Standards Australia and New Zealand. 2013 survey of the trans fatty acid content of Australian and New Zealand foods. 2013. [Access Date: 5/4/2016]. Available from: http://www.foodstandards.gov.au/consumer/labelling/review/Documents/SD1%20Analytical%20survey.pdf
4. Mansour MP, Sinclair AJ. The trans fatty acid and positional (sn-2) fatty acid composition of some Australian margarines, dairy blends and animal fats. Asia Pacific journal of clinical nutrition. 1993;2(4):155-63.
5. McCarthy J, Barr D, Sinclair A. Determination of trans fatty acid levels by FTIR in processed foods in Australia. Asia Pacific journal of clinical nutrition. 2008;17(3):391-6.
6. Wijesundera C, Richards A, Ceccato C. Industrially produced trans fat in foods in Australia. J Amer Oil Chem Soc. 2007;84:433-42.
7. Australian Bureau of Statistics. Australian Health Survey: User's Guide, 2011-2013, 24-hour dietary recall. 2014. [Access Date: 5/4/2016]. Available from: http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/0D6B1FE95EAB8FF3CA257CD2001CA113?opendocument
8. Gebauer SK, Chardigny JM, Jakobsen MU, Lamarche B, Lock AL, et al. Effects of ruminant trans fatty acids on cardiovascular disease and cancer: a comprehensive review of epidemiological, clinical, and mechanistic studies. Advances in nutrition (Bethesda, Md). 2011;2(4):332-54.
9. New South Wales Food Authority. Report on trans fatty acids in foods2008 5/4/2016. Available from: http://foodauthority.nsw.gov.au/aboutus/science/market-analysis/trans-fats
10. Arcand J, Scourboutakos MJ, Au JT, L'Abbe MR. Trans fatty acids in the Canadian food supply: an updated analysis. The American journal of clinical nutrition. 2014;100(4):1116-23.
11. Stender S, Astrup A, Dyerberg J. Tracing artificial trans fat in popular foods in Europe: a market basket investigation. BMJ open. 2014;4(5):e005218.
12. Roe M, Pinchen H, Church S, Elahi S, Walker M, et al. Trans fatty acids in a range of UK processed foods. Food chemistry. 2013;140(3):427-31.
13. Temme EH, Millenaar IL, Van Donkersgoed G, Westenbrink S. Impact of fatty acid food reformulations on intake of Dutch young adults. Acta cardiologica. 2011;66(6):721-8.
14. Karupaiah T, Tan HK, Ong WW, Tan CH, Sundram K. Trans fatty acid content in Malaysian supermarket foods: a field-to-laboratory approach in assessing food risk. Food additives & contaminants Part A, Chemistry, analysis, control, exposure & risk assessment. 2014;31(8):1375-84.
15. Dhaka V, Gulia N, Ahlawat KS, Khatkar BS. Trans fats-sources, health risks and alternative approach - A review. Journal of food science and technology. 2011;48(5):534-41.
16. Wang Q, Afshin A, Yakoob MY, Singh GM, Rehm CD, et al. Impact of Nonoptimal Intakes of Saturated, Polyunsaturated, and Trans Fat on Global Burdens of Coronary Heart Disease. Journal of the American Heart Association. 2016;5(1)
17. Pereira RA, Duffey KJ, Sichieri R, Popkin BM. Sources of excessive saturated fat, trans fat and sugar consumption in Brazil: an analysis of the first Brazilian nationwide individual dietary survey. Public health nutrition. 2014;17(1):113-21.
18. Ratnayake WM, L'Abbe MR, Farnworth S, Dumais L, Gagnon C, et al. Trans fatty acids: current contents in Canadian foods and estimated intake levels for the Canadian population. Journal of AOAC International. 2009;92(5):1258-76.
19. Mozaffarian D, Abdollahi M, Campos H, Houshiarrad A, Willett WC. Consumption of trans fats and estimated effects on coronary heart disease in Iran. European journal of clinical nutrition. 2007;61(8):1004-10.
20. Pot GK, Prynne CJ, Roberts C, Olson A, Nicholson SK, et al. National Diet and Nutrition Survey: fat and fatty acid intake from the first year of the rolling programme and comparison with previous surveys. The British Journal of Nutrition. 2012;107:405-15.
21. Honors MA, Harnack LJ, Zhou X, Steffen LM. Trends in fatty acid intake of adults in the Minneapolis-St Paul, MN Metropolitan Area, 1980-1982 through 2007-2009. Journal of the American Heart Association. 2014;3(5):e001023.

22. Hulshof KF, van Erp-Baart MA, Anttolainen M, Becker W, Church SM, et al. Intake of fatty acids in western Europe with emphasis on trans fatty acids: the TRANSFAIR Study. European journal of clinical nutrition. 1999;53(2):143-57.
23. Shrapnel B. Should trans fats be regulated. Nutrition & Dietetics. 2012;69:256-59.
24. Blewett N, Goddard N, Pattigrew S, Reynolds C, Yeatman H. Labelling Logic: Review of Food Labelling Law and Policy. 2011. [Access Date: August 2014]. Available from: http://www.foodlabellingreview.gov.au/internet/foodlabelling/publishing.nsf/content/home
25. Food Standards Australia and New Zealand. Technical evaluation for labelling review recommendation 13: Mandatory declaration of trans fatty acids in the nutrition information panel executive summary2014 5/4/2016. Available from: http://www.foodstandards.gov.au/publications/Pages/Technical-evaluation-for-labelling-review-recommendation-13.aspx
26. World Health Organization. Scientific update on trans fatty acids (TFA). 2009. [Access Date: 5/4/2016]. Available from: http://www.who.int/nutrition/topics/trans_fatty_acids/en
27. Backholer K, Peeters A. The impact of trans fat regulation on social inequalities in coronary heart disease in Australia. The Medical journal of Australia. 2013;199(3):168.
28. Stender S, Astrup A, Dyerberg J. Ruminant and industrially produced trans fatty acids: health aspects. Food & nutrition research. 2008;52
29. Downs SM, Loeh S, Wu JH. Trans fatty acids: a summary of the evidence relating consumption to cardiovascular outcomes and the efficacy of prevention policy to reduce levels in the food supply. In: Bendich A, Deckelbaum R, editors. Preventive Nutrition 5th Edition: The Comprehensive Guide for Health Professionals. 5th ed: Springer; 2016
30. Micha R, Khatibzadeh S, Shi P, Fahimi S, Lim S, et al. Global, regional, and national consumption levels of dietary fats and oils in 1990 and 2010: a systematic analysis including 266 country-specific nutrition surveys. BMJ. 2014;348
31. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet (London, England). 2012;380(9859):2095-128.
32. Barendregt J, Veerman JL. Categorical versus continuous risk factors, and the calculation of potential impact fractions. . Journal of Epidemiology and Community Health. 2010;64:209-12.
33. Barendregt J. The effect size in uncertainty analysis. Value in Health. 2010;13(4):388-91.
34. Downs SM, Thow AM, Leeder SR. The effectiveness of policies for reducing dietary trans fat: a systematic review of the evidence. Bulletin of the World Health Organization. 2013;91(4):262-9H.
35. Armijo-Olivo S, Stiles CR, Hagen NA, Biondo PD, Cummings GG. Assessment of study quality for systematic reviews: a comparison of the Cochrane Collaboration Risk of Bias Tool and the Effective Public Health Practice Project Quality Assessment Tool: methodological research. Journal of Evaluation in Clinical Practice. 2012;18:12-18.
36. Effective Public Health Practice Project. Quality Assessment Tool for Quantitative Studies Dictionary. 25/5/2016]. Available from: http://www.ephpp.ca/PDF/QADictionary_dec2009.pdf
37. NHMRC. How to use the evidence: assessment and application of scientific evidence. 2000. [Access Date. Available from: https://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/cp69.pdf
38. Barton P, Andronis L, Briggs A, McPherson K, Capewell S. Effectiveness and cost effectiveness of cardiovascular disease prevention in whole populations: modelling study. BMJ (Clinical research ed). 2011;343:d4044.
39. O Flaherty M, Flores-Mateo G, Nnoaham K, Lloyd-Williams F, Capewell S. Potential cardiovascular mortality reductions with stricter food policies in the United Kingdom of Great Britain and Northern Ireland. Bulletin of the World Health Organization. 2012;90:522-31.
40. O'Keeffe C, Kabir Z, O'Flaherty M, Walton J, Capewell S, et al. Modelling the impact of specific food policy options on coronary heart disease and stroke deaths in Ireland. BMJ open. 2013;3
41. Pearson-Stuttard J, Critchley J, Capewell S, O'Flaherty M. Quantifying the Socio-Economic Benefits of Reducing Industrial Dietary Trans Fats: Modelling Study. PloS One. 2015;10:e0132524.
42. Allen K, Pearson-Stuttard J, Hooton W, Diggle P, Capewell S, et al. Potential of trans fats policies to reduce socioeconomic inequalities in mortality from coronary heart disease in England: cost effectiveness modelling study. BMJ (Clinical research ed). 2015;351:h4583.
43. Rubinstein A, Elorriaga N, Garay OU, Poggio R, Caporale J, et al. Eliminating artificial trans fatty acids in Argentina: estimated effects on the burden of coronary heart disease and costs. Bulletin of the World Health Organization. 2015;93(9):614-22.
44. Restrepo BJ, Rieger M. Denmark's Policy on Artificial Trans Fat and Cardiovascular Disease. American Journal of Preventive Medicine. 2016;50:69-76.

45. Gayet-Boyer C, Tenenhaus-Aziza F, Prunet C, Marmonier C, Malpuech-Brugère C, et al. Is there a linear relationship between the dose of ruminant trans-fatty acids and cardiovascular risk markers in healthy subjects: results from a systematic review and meta-regression of randomised clinical trials. The British Journal of Nutrition. 2014;112:1914-22.
46. Stender S. In equal amounts, the major ruminant trans fatty acid is as bad for LDL cholesterol as industrially produced trans fatty acids, but the latter are easier to remove from foods. The American journal of clinical nutrition. 2015;102(6):1301-2.
47. Monge-Rojas R, Aragón MC, Chinnock A, Campos H, Colón-Ramos U. Changes in dietary intake and food sources of saturated and cis and trans unsaturated fatty acids in Costa Rican adolescents: 1996 versus 2006. Nutrition (Burbank, Los Angeles County, Calif). 2013;29:641-45.
48. Storey ML, Anderson PA. Changes in mean intake of fatty acids and intake of saturated and trans fats from potatoes: NHANES 2005-2006, 2007-2008, and 2009-2010. Advances in nutrition (Bethesda, Md). 2015;6:376S-82S.
49. Ratnayake WMN, Swist E, Zoka R, Gagnon C, Lillycrop W, et al. Mandatory trans fat labeling regulations and nationwide product reformulations to reduce trans fatty acid content in foods contributed to lowered concentrations of trans fat in Canadian women's breast milk samples collected in 2009–2011. The American journal of clinical nutrition. 2014:ajcn.078352.
50. Brandt EJ, Myerson R, Perraillon MC, Polonsky T. Abstract 17878: Hospital Admission Rates for Myocardial Infarction and Stroke in New York City Before and After the Ban on Trans Fatty Acids. Circulation. 2015;132:A17878-A78.
51. Ricciuto L, Lin K, Tarasuk V. A comparison of the fat composition and prices of margarines between 2002 and 2006, when new Canadian labelling regulations came into effect. Public health nutrition. 2009;12:1270-75.
52. Silveira BM, Kliemann N, Silva DP, Colussi CF, Proença RPdC. Availability and price of food products with and without trans fatty acids in food stores around elementary schools in low- and medium-income neighborhoods. Ecology of Food and Nutrition. 2013;52:63-75.
53. Colón-Ramos U, Baylin A, Campos H. The relation between trans fatty acid levels and increased risk of myocardial infarction does not hold at lower levels of trans fatty acids in the Costa Rican food supply. The Journal of Nutrition. 2006;136:2887-92.
54. Monge-Rojas R, Colón-Ramos U, Jacoby E, Mozaffarian D. Voluntary reduction of trans-fatty acids in Latin America and the Caribbean: current situation. Revista Panamericana De Salud Pública = Pan American Journal of Public Health. 2011;29:126-29.
55. Lee JH, Adhikari P, Kim SA, Yoon T, Kim IH, et al. Trans fatty acids content and fatty acid profiles in the selected food products from Korea between 2005 and 2008. J Food Sci. 2010;75(7):C647-52.
56. Niederdeppe J, Frosch DL. News coverage and sales of products with trans fat: effects before and after changes in federal labeling policy. American Journal of Preventive Medicine. 2009;36:395-401.
57. Mozaffarian D, Jacobson MF, Greenstein JS. Food reformulations to reduce trans fatty acids. The New England Journal of Medicine. 2010;362:2037-39.
58. Van Camp D, Hooker NH, Lin C-TJ. Changes in fat contents of US snack foods in response to mandatory trans fat labelling. Public health nutrition. 2012;15:1130-37.
59. Vesper HW, Kuiper HC, Mirel LB, Johnson CL, Pirkle JL. Levels of plasma trans-fatty acids in non-Hispanic white adults in the United States in 2000 and 2009. JAMA. 2012;307:562-63.
60. Friesen R, Innis SM. Trans fatty acids in human milk in Canada declined with the introduction of trans fat food labeling. The Journal of Nutrition. 2006;136:2558-61.
61. Ratnayake WMN, L'Abbe MR, Mozaffarian D. Nationwide product reformulations to reduce trans fatty acids in Canada: when trans fat goes out, what goes in? European journal of clinical nutrition. 2009;63:808-11.
62. Angell SY, Silver LD, Goldstein GP, Johnson CM, Deitcher DR, et al. Cholesterol control beyond the clinic: New York City's trans fat restriction. Annals of Internal Medicine. 2009;151:129-34.
63. Angell SY, Cobb LK, Curtis CJ, Konty KJ, Silver LD. Change in trans fatty acid content of fast-food purchases associated with New York City's restaurant regulation: a pre-post study. Annals of Internal Medicine. 2012;157:81-86.
64. Leth T, Jensen HG, Mikkelsen AA, Bysted A. The effect of the regulation on trans fatty acid content in Danish food. Atherosclerosis Supplements. 2006;7:53-56.
65. Benincá C, Zanoelo EF, de Lima Luz LF, Spricigo CB. Trans fatty acids in margarines marketed in Brazil: Content, labeling regulations and consumer information. European Journal of Lipid Science and Technology. 2009;111:451-58.

7 Supplementary tables and figures

Supplementary Table 1. Summary of FSANZ reports on TFA level in foods

Year	Aim(s) for the survey	Data sources	Recommendations and government responses
2007	• At the request of the Ministerial Council, review TFA level in foods to and estimated Australian population TFA intake	• ~330 samples were analysed between 2001 and 2005. • Samples were collected primarily from NSW, VIC and SA. • About 50 different types of processed and takeaway foods were included.	• Non-regulatory approaches to reducing level of TFA in the food supply would be most appropriate. • TFA level should be reassessed in 2009 to further assess the need to consider regulatory action.
2009	• Reassessed TFA level in foods and estimated Australian population TFA intake. • Evaluated outcomes of voluntary initiatives undertaken by quick service restaurant (QSR) to reduce TFA.	• 456 samples were analysed between 2008 and 2009. • Samples were collected from NSW, SA and WA. • 36 different types of processed and takeaway foods were included. • QSR chains provided self-reported progress on TFA reduction.	• Status quo of non-regulatory approaches to reducing TFA in the food supply should be retained.
2013	• The 'Food Labelling Logic' review recommended mandatory labelling of TFA should be introduced if manufactured TFA had not been phased out of the food supply by January 2013.	• 500 samples were analysed in October, 2013. • Samples were collected from NSW, WA, SA, TAS, QLD and VIC. • 39 different types of processed and takeaway foods were included. Samples were selected to include different quality products and range of prices. • QSRs, edible oil and spread manufacturer/suppliers, packaged food manufacturers, and supermarket chains were surveyed and provided self-reported activities to reduce TFA.	• Additional data is needed (including cost and benefits analysis of a threshold labelling of TFA and other approaches, potential impact on consumer purchase, blood cholesterol levels, products reformulations and industry costs) to inform further considerations of mandatory TFA labelling. • The Ministerial Forum questioned whether a deadline for a complete phase out of TFA was actually required.

Supplementary Table 2. TFA as % of total fat in Australian processed and takeaway foods in 2013

Product type	n	Mean	Standard deviation	Minimum	Maximum
Takeaway foods					
Chicken nuggets/products	16	1.35	0.40	0.9	2.4
Crumbed/battered fish fillets	16	1.81	0.80	.8	3.3
Falafel	9	1.31	0.64	.6	2.6
Fried noodle dishes	6	1.03	0.62	0	1.6
Hot chips/potato products	18	1.04	.78	0	2.5
Pizza	15	1.79	1.36	0	3.8
Restaurant style takeaway dishes	14	2.02	1.49	0	5.2
Spring rolls	9	1.6	1.10	0	3.3
Snack Food					
Extruded snacks	10	1.04	0.75	0	2.6
Popcorn	6	6.75	7.54	0	18.1
Potato crisps and corn chips	13	0.82	0.29	0.3	1.3
Fats & Oils					
Edible oil spreads	13	1.19	0.69	0.6	3.3
Oil based dressing	8	0.89	0.47	0.4	1.7
Vegetable oil	10	0.73	0.5	0	1.7
Meat Products					
Meat pies	9	4.44	2.21	0.9	8.1
Sausage rolls	11	4.82	3.02	0.4	10
Bakery Products					
Chocolate chip biscuits	13	1.72	1.02	0.5	3.7
Choc coated or filled biscuits	10	1.01	0.31	0.5	1.3
Sweet biscuits	10	0.88	0.62	0	2

Cream biscuits	11	1.68	1.66	0.5	6.4
Croissants	8	4.81	1.39	2.5	6.7
Custard baked goods	9	4.4	2.37	1	7.4
Desserts	10	2.69	1.69	0.7	6.4
Doughnut	13	1.99	1.39	0	4.7
Pikelets or pancakes	8	0.4	0.57	0	1.2
Prepared pastry	8	6.26	3.16	0.7	10.8
Rice crackers and crispbreads	10	0.57	0.62	0	1.6
Standard savoury biscuits	8	0.8	0.52	0	1.4
Scones	8	2.40	2.67	0	5.9
Shelf stable cakes without cream	10	1.12	0.89	0	2.5
Shelf stable cream filled cakes	8	0.48	0.49	0	1.3
Sweet muffins and banana bread	11	1.65	1.15	0.6	4.9
Others					
Snack bars	10	0.39	0.69	0	2
Dry mix pasta	8	1.89	1.96	0	4.7
Nut based spreads	10	0.4	0.40	0	1.3
Sauces	8	0.8	1.73	0	5
Toasted style muesli cereal	8	0.1	0.14	0	0.3
Confectionery	10	0.6	0.66	0	1.8
Dips	8	1.5	0.79	0.8	2.7

Supplementary Table 3. The underlying assumptions of the included modelling studies

Authors, Year and Country	Model Assumptions
O'Keeffe et al.[40] 2013 Republic of Ireland	• Combined changes in RR for individuals are multiplicative. • Changes between current food component consumption and the expected two scenarios examined will be made by all individuals within the population changing consumption by the same amount. • Reductions in unit change in RR refer to a unit change in food component consumption or proximal risk factors following a dose-response relationship (i.e. a change in consumption of fruit and vegetables from 2 to 3 portions a day has the same effect on RR as a change in consumption from 8 to 9 portions a day).
Allen et al.[42] 2015 England	• Future consumption would remain constant. • Continuing declines in incidence of and mortality from CHD. • The policies modelled varied in assumed coverage: 100% coverage for a total ban, at most 49% for labelling, and at best 40% for a restaurant/fast food ban.
O'Flaherty et al.[39] 2012 United Kingdom	• Effects of food policies would be quantitatively similar to those in other countries. • Assumed trans fat could be replaced by an equal mixture of good MUFAs and PUFAs, not by saturated fat. • Assumed changes in dietary variable would be similar across all age groups. • Effects of dietary changes on mortality would wane with increasing age, as with cholesterol and blood pressure.
Barton et al.[38] 2011 England and Wales	• Benefits apply consistently for men and women across age, social and risk groups. • No attempt to consider recurrent events or subsequent deaths. • Assumes that CVD rates would remain constant if there was no intervention.
Rubinstein et al.[43] 2015 Argentina	• 80% of sudden deaths were due to CHD. • The reduction in CHD deaths was proportional to the difference in estimated CHD risk. • Baseline consumption of 1.5% of total energy intake as TFA in 2004.

Restrepo & Rieger [44] 2016 Denmark	• Compared Denmark's annual CVD mortality rates over time with those of a synthetic control group, which was composed of a weighted average of other OECD countries that did not implement a policy restricting iTFA in food
Pearson-Stuttard et al.[41] 2015 England and Wales	• Used an area level categorization of socioeconomic status. This may be sub-optimal for analysing trends within individuals. • The value for the 85+ age group was extrapolated using the reducing mortality reduction figures from the younger age groups. • Mortality reduction for a 0.25% reduction in TFA intake was estimated using linear extrapolation. • Assumed also that the elimination of ruminant TFA would not be feasible. • Assumed no future decline in case fatality, which is a conservative estimate when modeling a 2030 scenario.

RR = relative risk, CHD = coronary heart disease, MUFA = monounsaturated fatty acid, PUFA = polyunsaturated fatty acid, CVD = cardiovascular disease, TFD = trans fatty acid, OECD = Organisation for Economic Cooperation and Development

Supplementary Table 4. Summary of the relevant results of studies from the 2000-2012 review that used a pre/post study design (adapted from [34])

Country, Authors, Year	Study Type*	Study population/sample	Aim & Main outcomes	Relevant Results
		Voluntary TFA Self-regulation		
Netherlands[a] Temme et al. 2011[13]	Pre-post test	750 Dutch participants aged 19-30 years	Aim: To estimate the impact of reformulations in task force food groups by estimating intakes of TFA in young Dutch Outcome: Usual intakes of fatty acids before and after task force activities	Changes in TFA intakes: TFA decreased from 1.0% of energy intake before to 0.8% after reformulation. Contribution of task force foods to TFA consumption declined from 45% to 29% after reformulation. Pastry, cakes, biscuits and snacks contributed the most to the decrease in TFA. The TFA intakes from fats and margarines were not different. Changes in TFA levels: TFA content of packaged potatoes and bread were 0.3g/100g lower, cookies/ biscuits were 0.8g/100g lower and snacks and salads were 0.6 g/100g lower. SFA: SFA intakes did not change. MUFA and PUFA contents did not change significantly with the exception of a slight decrease in PUFA content in cookies/biscuits. No changes to total fat.
Costa Rica[b] Colon-Ramos et al. 2006[53]	Case-control	1797 case control pairs from metropolitan Costa Rica in 1994-1999 (before industrial modifications of TFA) and in 2000-2003 (after industrial modification of TFA)	Aim: To assess the risk of nonfatal acute MI before and after the TFA reduction in the food supply Outcome: Subcutaneous adipose tissue sample for fatty acid, FFQ + questions about oils and fats used (confirmed by visual identification at home visits)	Changes in TFA in tissue: Median values for quintiles of TFA were higher in 1994-1999 as compared to 2000-2003. There was a decrease over time of TFA in the tissue of those who reported using soybean oil. Changes in MI risk: Prior to self-regulation total TFA content in adipose tissue was associated with increased risk of MI, after controlling for several confounders; this relationship was not seen in the period 2000-2003.

Country, Authors, Year	Study Type*	Study population/sample	Aim & Main outcomes	Relevant Results
Americas[c] Monge-Rojos et al. 2011[54]	Pre-post test	Self-reported surveys by corporations (n=12) that had signed "trans-fat-free Americas" declaration. 3 provided all data requested; the remaining companies completed portions of the survey or refused to provide data (n=6)	Aim: To assess progress towards the goal of achieving a "trans-fat-free Americas" Outcome: % and type of fats/oils used to replace TFA; current and past (2006) TFA amounts in specific foods; description of obstacles	Changes to TFA levels: Of the 3 companies that provided data, some progress had been made to reduce TFA however high quantities remained in cookies/crackers and seasonings/sauces. PepsiCo had virtually eliminated TFA in cookies/crackers sold in Mexico and the Caribbean but in North America those products still contained 11-28gTFA/100g. The Brazilian Association of Food Industries reported reductions ranging from 25-92% in oils and fats to 100% in breakfast cereals; TFA in seasoning and sauces averaged 11.5gTFA/100g. McDonalds reported 100% reductions in TFA in the oils used in Brazil. Other companies reported that they had made efforts to reduce TFA but did not provide specific data.
Mandatory TFA labelling				
Korea[d] Lee et al. 2010[55]	Pre-post test	21 food products within 7 different categories of food from local markets and fast food restaurants in both 2005 and 2008	Aim: To examine the impact of mandatory TFA regulation on TFA levels in food products Outcome: Fatty acid composition of food products sampled in 2005 (pre-regulation) and 2008 (post-regulation)	Pre-regulation levels: TFA ranged from 0.6% to 44.6% of total fatty acids. Post-regulation levels: TFA levels significantly (p<.05) decreased with the exception of 1 breakfast cereal and fried chicken. TFA levels were <1% in breakfast cereals, French fries and fried chicken in 2008. Cream filled biscuits and cakes had TFA levels ranging from non-detectable to 5.4%. Pre/post changes savoury snacks: TFA content in French fries decreased by 91-98% and fried chicken by 50-96%. Pre/post changes baked goods: TFA reduction ranged from 69-89% in cream filled biscuits and 88-97% in cream filled cakes. SFA: SFA significantly increased by up to 29-135% in biscuits and 48-69% in cakes respectively (p<.05). Unsaturated fatty acids increased by 35-185% in French fries and 17-76% in fried chicken.

Country, Authors, Year	Study Type*	Study population/sample	Aim & Main outcomes	Relevant Results
United States[e] Niederdeppe & Frosch 2009[56]	Pre-post test	Sales of 7 TFA containing products in a major Los Angeles County grocery store chain between 2005 and 2007 (n=11,997 store weeks)	Aim: To assess whether news coverage influenced sales of products containing TFA in between Dec 2004 (before labelling regulation) and June 2007 (after labelling regulation) Outcome: Weekly unit sales/price data of 7 TFA containing products, average number of TFA stories per week across five news outlets, trends in product-purchase patterns	Pre-regulation: Little support (2 of the 7 products) for news effects on TFA product unit sales. Only stick margarine and hot dogs showed an effect. Post-regulation: Strong support (5 of the 7 products) for news effects on TFA product unit sales. Interactions between unit sales and TFA news were significant (negative) for the two products with the highest TFA content (Crisco and buttered popcorn). Effects dissipated after 3 weeks.
United States Mozaffarian et al. 2010[57]	Pre-post test	83 reformulated products (58 supermarket foods and 25 restaurant foods) identified based on consumer magazines, health newsletters, a non-profit organisation database and FDA food composition databases	Aim: To assess the levels of TFA and SFA in major brand name US supermarket and restaurant foods reformulated to reduce TFA from 1993 to 2006 and 2008 to 2009. Outcome: TFA, SFA and total fat content pre- and post-reformulation	Pre/post changes in supermarket foods: TFA was reduced to <0.5g/serving in 95% of supermarket foods. Average reductions were 1.8g/serving (84%) in supermarket foods. Pre/post changed in restaurant foods: TFA was reduced to <0.5g/serving in 80% of restaurant foods. Average reductions were 3.3g/serving (92%) in restaurant foods. SFA: 65% of supermarket products and 90% of restaurant products had SFA levels that were lower, unchanged or only marginally (< 0.5g) higher than before reformulation. The average SFA content in supermarket foods increased slightly (< 0.5g; attributed to 1/3 of products) and decreased in restaurant foods. Reduction in TFA nearly always exceeded any increase in SFAs. Overall the content of both fats combined was reduced in 90% (average reduction of 1.2g/serving) of supermarket foods and 96% (average of 3.9g/serving) of restaurant products.

Country, Authors, Year	Study Type*	Study population/sample	Aim & Main outcomes	Relevant Results
United States Van Camp et al. 2012[58]	Pre-post test	5012 chip and cookie products in 2001-02 and 2008-09	Aim: To assess the impact of mandatory TFA labelling on US snack foods Outcome: TFA media citations, changes in lipid ingredients used, change in reported TFA & SFA content and use of '0g TFA' declaration	Pre/post changes savoury snacks: A 45% reduction in PHVO use in chips and (sunflower oil main replacement). Only 1% of chip introductions reported >0g TFA. Pre/post changes baked goods: A 42% reduction in PHVO use in cookies (palm oil main replacement). Cookie introductions containing palm and palm kernel oil increased by 41% and 5% respectively. 9% of cookie introductions reported >0g TFA. SFA: No difference in SFA levels in chips. On average, increase of 0.49g SFA/30g serving of cookies but no increase in total fat.
United States Vesper et al. 2012[59]	Pre-post test	229 non-Hispanic white participants in 2000 and 292 from 2009	Aim: To assess the possible impact of TFA regulation on TFA levels in the blood Outcome: TFA in the blood in 2000 vs 2009, LDL, HDL cholesterol and triglycerides	Pre/post changes in blood: TFA levels in the blood decreased from 43.7 µmol/L in 2000 to 19.4 µmol/L in 2009 (58% decrease). Pre/post changes in cholesterol/triglycerides: Levels of LDL-C were lower in 2009 (119.2mg/dL) compared to 2000 (128.2 mg.dL). Levels on HDL-C were higher in 2009 (55.8mg/dL) as compared to 2000 (49.6mg//dL). Triglyceride levels were lower (131mg/dL) in 2000 as compared to 2009 (109.3mg/dL).

Mandatory TFA Labelling + Voluntary TFA limits				
Canada[f] Friesen et al. 2006[60]	Pre-post test	Breast milk samples from women (n=87) who gave birth between 2004 and 2006 Breast milk samples from women (n=103) collected in 1998	Aim: To compare TFA in the breast milk before (1998) and after (2004-05) the Canadian TFA regulation Outcome: TFA in the breast milk of women in 2004-05 as compared to samples taken from women in 1998	Change in TFA levels: TFA was 35% lower in 2004-06 than 1998. TFA levels progressively decreased in 3 time points at 5 month intervals between 2004 and 2006. The estimated intake of TFA (using breast milk) was 4.0g/person/day in 1998 as compared to 2.2g/person/day in 2005. SFA: MUFA and PUFA did not change but SFA were slightly higher.
Canada Ricciuto et al. 2009[51]	Pre-post test	229 margarines sold in 9 Greater Toronto Area (GTA) supermarkets in 2002 and 274 margarines sold in 10 GTA supermarkets in 2006	Aim: To examine the effectiveness of TFA labelling regulation on the fat composition and prices of margarines Outcome: Comparison of price and fatty acid composition in 2002 and 2006	Pre/post changes: Products with ≤0.2g/10g of TFA increased significantly from 31% to 69%. TFA of 13 of 18 margarines on the market in both years decreased over the time period from 0.1 to 1.3g/10g serving. TFA content of 3 products increased from 2002 to 2006. SFA: Average amounts of TFA and MUFA decreased, PUFAs increased and SFA did not change significantly between two time points. Of the 13 products reformulated, SFA content increased and in three products equalled or exceeded TFA decrease. Price: The average price of products labelled as TFA free in 2006 rose by 28% as compared to 10% in those without claims. Products with low TFA and SFA levels were more expensive than those with higher TFA levels.

Canada Ratnayake et al. 2009[61]	Interrupted time series	221 individual manufactured and restaurant foods sold in major grocery/restaurants in Canada (2005-07) that were likely to contain TFA (i.e., cookies, crackers, breakfast bars, frozen potato products, margarines, etc.)	Aim: To assess the TFA and SFA levels of both grocery and restaurant foods that likely contain TFA in Canada in 2005-07 Outcome: Number of products containing TFA, number of reformulated products, changes in fatty acids and total fat content of reformulated products	Post-regulation levels: 42% of products contained TFA in initial assessment: 12 with 5-10% TFA and 80 with >10% TFA. Of the TFA containing products, 8 were discontinued, 12 were reformulated, but not yet assessed, and 7 were measured for the first time in 2007. Of the remaining products, nearly 75% were reformulated during the study period to decrease TFA content. TFA levels decreased from 26 ± 13% to 2 ± 4%. SFA: Of products tested more than once to assess reformulation, none had an increase in SFA or TFA; 1 had no change but all others had lower TFA + SFA and increased cis-unsaturated fat content. Average TFA + SFA was reduced from 53% ± 12% to 30± 19%. Average absolute change in total fat content was 0.8 ±3.0%.

Canada Ratnayake et al. 2009[18]	Interrupted time series	1120 Samples in 31 different food categories were collected from 2005-2009 from major grocery stores, fast food chains and cafeterias across Canada. Foods collected had previously been identified as having high quantities of TFA 2004 Canadian Community Health Survey data on TFA and SFA intakes (n=33,000)	Aim: To provide results of the TFA monitoring program for the period 2005- 2009 Outcome: Fatty acid composition of selected foods	Post-regulation levels: From 2005-2009, of the 1120 samples analysed 76% met the recommended TFA limits. In 2005 and 2006 only 58% of samples met the limits, in 2007 68%, in 2008 77% and 2009 78% met the limit. Post-regulation changes in baked goods: Only 45% of brownies, 43% cakes, 25% of croissants, 45% of Danishes, 55% of garlic breads, 36% pies, and 67% of tarts contained TFA <5%. Only 29% of donuts sampled between 2005 and 2008 contained <5% TFA. Post-regulation changes in savoury snacks: 100% of pizzas, 79% of French fries and chicken products, 83% of fish products, 89% of muffins and 75% of onion rings sampled <5% TFA. Post-regulation changes in margarines/spreads: 62% of tub margarines, 0% of print margarines and 50% of vegetable shortenings met the recommended limits. TFA in margarines between 2005-2007 were on average 39.3%. Most (70%) of the products sold in cafeterias met the limit with the exception of some products (margarines, onion rings and fish products). SFA: TFA reductions were achieved in most products without increasing SFA but increasing MUFA and PUFAs. However, in crackers, cookies, frozen chicken products and garlic spreads TFA reductions were associated with an increase in SFA and unsaturated fatty acids. The sum of TFA + SFA in these products did not increase. Reformulated donuts that reduced TFA had SFA levels nearly double those with TFA levels >5%. Changes in TFA intakes: TFA has decreased from 8.4g/day in the mid 90s to 3.4g/day in 2008 (still above WHO recommendations). On average, there has been a 30% decrease in TFA intakes between 2004 and 2008. SFA intakes have not increased during this period.

Mandatory TFA limits (local bans)				
New York City (NYC), USA[g] Angell et al. 2009[62]	Interrupted time series	478 restaurants in 2005, 1021 restaurants in 2006, 996 food establishments in 2007 (after passage but prior to effective date) Fast food nutrition (n=12) information of major chains pre and post regulation	Aim: To examine the effectiveness of NYC TFA restrictions Outcome: Inspector assessed restaurant compliance with TFA ban, fatty acid composition of selected foods	Pre-regulation compliance 2005-2007: 50% of restaurants used fats containing TFA to prepare food. Following an education campaign aimed at restaurants, the 2006 survey found that 51% of restaurants were still using fats containing TFA. In 2007 TFA use had decreased to 43%. Post-regulation compliance 2008: 99% of all restaurants were compliant with Phase 1. 6 months after the 2nd phase of regulation 92% compliance. Changes in use: 98% of restaurants were not using oils/spreads containing TFA in November 2008 compared to 50% in 2005. SFA: On average, French fries in fast food chains decreased SFA by 10.5%, TFA by 97.9% and total TFA + SFA grams by 54%.
New York City, USA Angell et al. 2012[63]	Pre-post test	6969 purchases in 2007 and 7885 purchases in 2009 at 168 randomly selected NYC restaurant locations of 11 fast-food chains in 2007 and 2009	Aim: To examine the effect of NYC TFA restriction on the TFA and SFA content of fast-food purchases Outcome: Change in mean grams of TFA, SFA, TFA + SFA and TFA/1000kcal overall and by fast-food chain type	Post-regulation changes in TFA levels: Mean TFA/purchase decreased by 2.4g (2.9g vs 0.5g; p<.001) and mean TFA/1000kcal decreased by 2.7g/1000kcal. TFA/purchase significantly decreased in 3 of 5 chain types and increased in one (0.2g vs 0.3g). Purchases with 0g of TFA increased by 86% from 32% to 59%. The maximum TFA content of a single purchase decreased from 28g to 5g. SFA: SFA increased by 0.6g (p=.011) and mean TFA + SFA decreased by 1.9g (p<.001). The maximum TFA + SFA content in a single purchase decreased from 96g to 60g.

Mandatory TFA limits (national bans)				
Denmark[h] Leth et al. 2006[64]	Pre-post test	253 samples of both imported and domestically produced products at the end of 2002 to early 2003 (pre-ban) 148 samples of both imported and domestically produced products from Nov 2004 to Feb 2005 (post-ban)	Aim: To assess the effectiveness of Denmark's TFA ban Outcome: TFA content of food samples known to be high in TFA in both 2002-03 and 2004-05	TFA intakes: TFA decreased from 4.5g/day in 1976, to 1.5g/day in 1995 and were virtually eliminated in 2005 (post-ban). Pre-regulation levels: Highest TFA levels were found in frying fat of chain restaurants (20% of fat from TFA), popcorn (30% of fat from TFA) and in various cakes, biscuits and pastries (10% TFA of total fat). 25% of samples had >2% of fat from TFA. Chocolate and confectionery items contained virtually no TFA, with the exception of caramels. Industrial bakery products contained high amounts of TFA, 43% cookie products had levels >2%; 26% potato products contained > 2% TFA and 80% ready made French fries >2% TFA. Post-regulation levels: Fewer samples had levels of TFA >2%. Those that did have levels >2% ranged between 2-6% TFA, some with milk ingredients (meaning some of the TFA was likely naturally occurring TFA). In a couple of potato and cake products higher levels of TFA were found and steps have been taken by authorities to correct this.

*Study Quality based on NHMRC (2000) designation of levels of evidence: Case-control studies (III-2), Interrupted time series (III-3), Pre/post, post-test and cross-sectional (IV)

PHVO = Partially hydrogenated vegetable oils; MUFA = Monounsaturated fatty acids; PUFA = Polyunsaturated fatty acids; SFA = Saturated fatty acids; MI =Myocardial Infarction

[a] 2004 Product Board for Margarine, fats and oils set up the Task Force for Responsible Fatty Acid Composition to reduce use of PHVOs setting limit of 5% TFA in frying oils

[b] Voluntary industrial modification of partially hydrogenated soybean oil

[c] Voluntary reformulation of products; 12 major companies in the Americas signed a declaration of their intentions to help achieve a "trans-fat-free Americas"

[d] TFA content <0.2g/serving labelled as TFA free

[e] TFA content <0.5g/serving labelled as TFA free

[f] TFA content <0.2g/serving and TFA+SFA combined < 2g/10g (≤ 15% of energy) serving labelled as TFA free. Voluntary limit of 2% total fat from TFA in fats, oils and spreads and 5% total fat from in foods.

[g] Ban (<0.5g/serving) of artificially produced TFAs in all licensed food establishments. First phase – oils and spreads, second phase – all products including bakery items.

[h] Ban of TFA in all foods sold in the country; a maximum of 2% total fat from TFA.

Supplementary Table 5. The percentage of foods classified as TFA free post policy interventions (adapted from[34])

Policy intervention	Food categories	% TFA free[a]
National bans[64]	Overall	Virtually eliminated
Mandatory labelling + voluntary limits[10, 61]	Margarines/spreads	0-85%
	Bakery Products	25-100%
	Restaurant food (including restaurants in institutions)	50-100%
	Overall	76-97%
Mandatory labelling[10, 61, 65]	Margarines/spreads	67-79%
	Fried restaurant foods	80%
	Supermarket foods	95%
	Bakery Products	42-77%
	Savoury snacks	40-100%
Voluntary TFA limits[13]	Restaurant frying oil	45%

[a]Studies conducted in the United States classify <0.5g/serving as TFA free whereas other countries classify products <0.2g/serving as TFA free. In Canada, additional requirements include a combined TFA + SFA ≤15% of energy

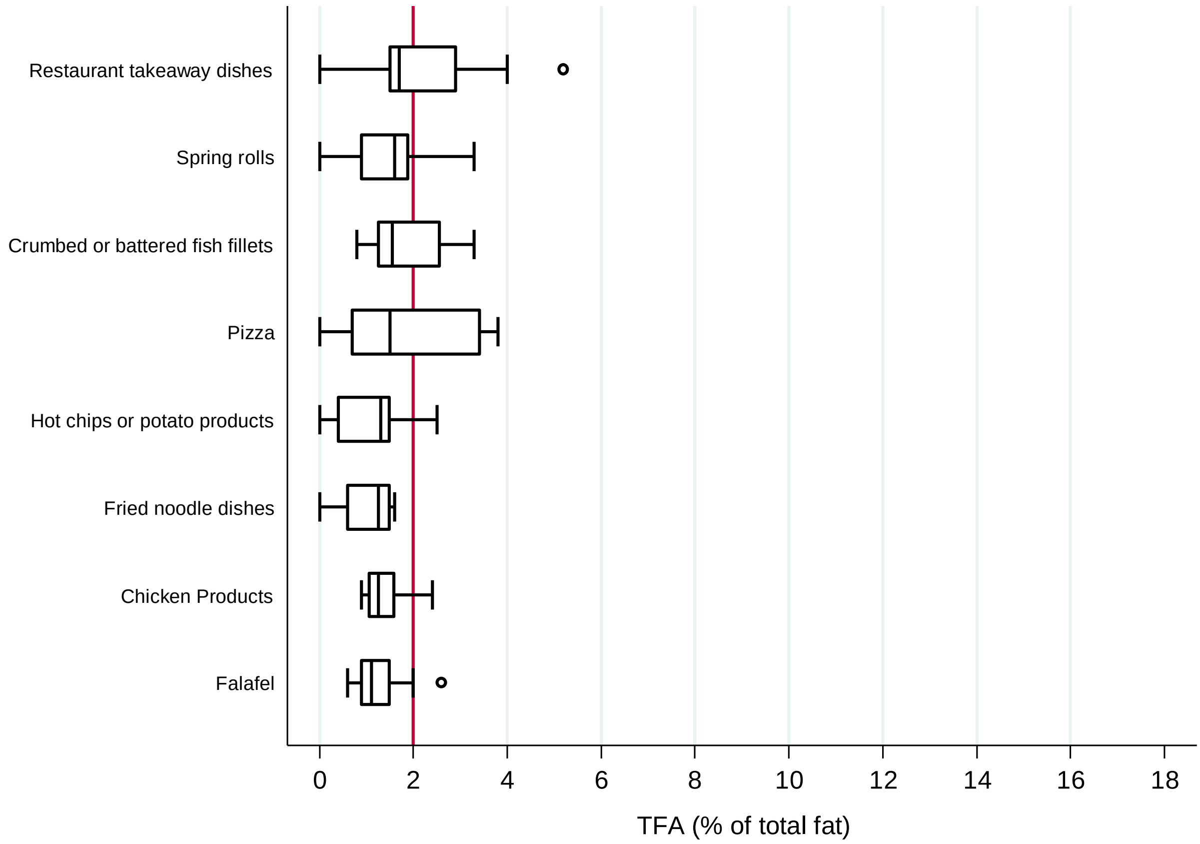
Supplementary Table 6. Changes in the fatty acid composition of foods after the introduction of a TFA policy (adapted from Downs 2013,[34])

Policy Intervention	Authors	TFA	SFA	MUFA and/or PUFA	Total Fat*
Mandatory TFA Labelling	Lee et al (2010)[(54)]	↓	↑ bakery products	↑ restaurant food	↓
	Mozaffarian et al. (2010)[(56)]	↓	↑ supermarket foods ↓ restaurant foods	Not included	↓ (SFA+TFA)
	Van Camp et al. (2012)[(57)]	↓	↑ bakery products	↑ oils high in PUFA& MUFA in chips	NC
	Storey & Anderson (2015) [(15)]	↓	↓	--	--
Mandatory TFA limits (bans)	Angell et al. (2009)[(47)]	↓	↓	Not included	↓ (SFA+TFA)
	Angell et al. (2012)[(62)]	↓	↑	Not included	↓ (SFA+TFA)
Mandatory TFA Labelling + Voluntary Limits	Ricciuto et al. (2009)[(50)]	↓	NC	↓ MUFA ↑ PUFA	↓ (significance not assessed)
	Ratnayake et al. (2009)[(60)]	↓	↓	↑	NC
	Ratnayake et al. (2009)[(17)]	↓	↑ crackers, cookies, and garlic spreads& donuts	↑	NC
Voluntary TFA Self-regulation	Temme et al. (2011)[(13)]	↓	NC	NC ↓ decrease in biscuits	NC

*Change in total fat calculated by adding fatty acids when not reported by authors; in these cases, significance was not assessed. For studies that did not examine MUFA and PUFA, SFA + TFA changes are reported.

MUFA = Monounsaturated fatty acids; PUFA = Polyunsaturated fatty acids; SFA = Saturated fatty acids; TFA = Trans fatty acids; NC = No change

Supplementary Figure 1. Products sampled from takeaway shops



Supplementary Figure 2. Products sampled from independent retailers and supermarkets