

Effect of Trans Fatty Acids Intake on Blood Lipid Profile of Workers in East Kalimantan, Indonesia

Sartika RAD

Center for Epidemiological Research & Surveillance, Faculty of Public Health
University of Indonesia, Depok, West Java, Indonesia, 16424

ABSTRACT

Introduction: In experiencing epidemiologic transition, Indonesia faces emerging nutrition problems. **Methods:** Among the dietary lifestyle changes over the years is the rising consumption of fats and oils, including trans fatty acids. Intake of these fatty acids from ruminant meats, hydrogenated vegetable oils and fried foods is known to have detrimental effects on serum lipoprotein cholesterol levels. The purpose of this study was to determine the effects of trans fatty acid intake on blood lipid profile. A prospective cohort study was conducted on 388 workers at an on-shore oil company in East Kalimantan. **Results:** The mean intake of trans fatty acid was 0.48% of the total dietary calories. Fried foods contributed most to the total trans fatty acid consumed at 0.20% of the total calories. Trans fatty acid intake from ruminant products, and margarine/hydrogenated vegetable oil products were 0.09% and 0.06% of calories, respectively. The high consumption of fried foods is associated with risks of hypertriglyceridemia (RR: 1.41, 95%CI 1.06; 1.86), high ratio of total cholesterol/HDL-C (RR: 1.98, 95%CI 1.00; 3.98) and dyslipidemia (RR: 1.35, 95%CI 1.00;1.84). Every additional one percent of saturated fatty acid intake is associated with an increase in trans fatty acids amounting to 0.03% of total calories ($r = 0.320$, $p = 0.000$). **Conclusion:** These results suggest that a reduction in consumption of fried foods will be of benefit as it will reduce intake of both saturated and trans fatty acids. Further studies are recommended to determine the intake of trans fatty acids and their health effects on other population groups in Indonesia.

Keywords: Blood lipid profile, fried foods, Indonesian workers, trans fatty acids

INTRODUCTION

Indonesia, in experiencing epidemiologic transition, faces the double burden of infectious diseases and non-communicable diseases. The National Health Survey (2005) showed that the mortality rate from heart diseases for above 15 years was 6.0% in 2002. The level rose to 8.4% in 2005. The

increase in coronary heart disease mortality may be attributed to lifestyle changes. There has been an increase in the consumption of fats (more than 30% of calories), saturated fatty acids (>10% of calories) and cholesterol (>300 mg) per day (Lichtenstein *et al.*, 2006). Despite the low fat content in the Indonesian diet generally, cases of heart disease continue to increase and has become one of the leading

* Correspondence author: Ratu Ayu Dewi Sartika; Email: ayu_fkm_ui@yahoo.com

causes of death (National Health Survey, 2005).

A study on Indonesian diet showed that the main source of fat was fried foods (80-90%) (Sartika, 2009). The presence of trans fatty acids in fried cassava root amounted to 0.73% of total fat, probably derived from the cooking oil used for deep frying of the cassava. There is lack of data on the trans fatty acid content in Indonesian foods and the mean intake of trans fatty acids by the Indonesian people. It is suspected that the intake of trans fatty acids from fried foods, ruminant products, and margarine/hydrogenated vegetable oil products in the Indonesian diet might be high. The purpose of this study was to determine the average intake of trans fatty acid and its effect on blood lipid profile in a sample of Indonesian workers in East Kalimantan.

METHODS

This was a prospective cohort study undertaken and completed in 2006. The study subjects comprised workers operating in an on-shore oil company in East Kalimantan, Indonesia. Results of initial surveys conducted in 2005 showed the prevalence of Coronary Artery Disease (CAD) among workers to be 1.32% in 2003, rising to 3.92% in 2004 and 7.9% in 2005. Oomen *et al.* (2001) found that the intake of trans fatty acid predicted a risk factor of CAD.

The inclusion criteria were that they had worked for a period of one year and longer, while the exclusion criteria were being pregnant, not willing to participate in this study, and those having diabetes and other chronic diseases of the kidney, liver, gastrointestinal tract and hypothyroid which can increase total cholesterol and low cholesterol lipoprotein (LDL-C). Of the total of 410 subjects, 388 were eligible and were included in the study.

The data were collected through individual interviews using a pre-tested questionnaire. The data included indi-

vidual socio-economic characteristics, knowledge about nutrition and health, family medical history, exercise and smoking habits, and dietary intake.

Educational level measured using a standardised questionnaire, was classified into two categories: low (≤ 9 years of schooling) and high (> 9 years of schooling). Nutrition and health knowledge consisted of the impact of fried foods on health, food sources of fat, and how to prevent the risk factors of heart disease. The score ranged from 0-10 with 0-5 (below the mean) categorised as 'poor' and 5-10 (above the mean) as 'good'. Information on familial diseases was answered 'yes' or 'no' (Lichtenstein *et al.*, 2006). Exercise routine was classified into regular and irregular. Regular exercises of the respondent were exercises and aerobics done at home or the office continuously and rhythmically 3-7 times/week, for a 30- to 60-minute duration (Varady & Jones, 2005). Information on smoking habit was classified as current smoker, never smoker and former smoker. The former smoker in this study was a subject who had previously smoked and stopped smoking for more than a year (Houterman *et al.*, 2001).

Dietary intake was assessed using a semi-quantitative food frequency questionnaire to assess the habitual consumption of 66 food items during the past one year. For each item, an open-ended numerical response was entered into one of the following categories: per day, per week, per month, per year or never. The food items included fried foods, ruminant meats and margarine/hydrogenated vegetable oil (HVO) products. Trans fatty acid intake was grouped into 'high consumption' (≥ 4 servings/week) and 'low consumption' (< 4 servings/week). In addition, a 24-hour recall interview using a computer-assisted dietary system to estimate total intake of nutrients including trans fatty acid intake was conducted. The data was collected by the researcher and two trained nutritionists.

The nutrient and energy intake was quantified for each individual using an extended version of the Nutrisoft (2003), which contains the Indonesian food composition table. However, this database does not include trans fatty acids. In order to determine the trans fatty acids content in different types of food (fried food, ruminant products and margarine/HVO products), the researcher determined the trans fatty acid content of the foods identified to be commonly consumed by the study subjects. These consisted of 30 examples of fried foods, 20 ruminant products and 20 margarine/HVO products tested in a certified agricultural laboratory at the Bogor Agricultural Institute. The trans fatty acid content of the diets was determined by gas chromatographic separation of fatty acid methyl esters. In addition, for comparative purposes, the researcher also used the Food Balance Sheet from the US Department of Agriculture, as well as other sources (USDA, 2002). Based on the 24-hour recall diet, we calculated the daily total intake of trans fatty acids from each food group (grams).

Blood lipid profile (total cholesterol, LDL-C, HDL-C and triglyceride) was determined by a photometer 'Cobas Mira/Micros'. The instrument was calibrated periodically by a licensed technician from the instrument distributor. The blood results were classified as follows: hypercholesterolemia (>200 mg/dl) and normal (≤ 200 mg/dl); LDL-C 'abnormal' (>130 mg/dl) and normal (≤ 130 mg/dl); hypertriglyceridemia (>160 mg/dl) and normal (≤ 160 mg/dl); HDL-C 'low' (<40 mg/dl) and normal (≥ 40 mg/dl); the ratio of total cholesterol/HDL-C 'high' (>5 mg/dl) and low (< 5 mg/dl); the ratio of LDL-C/HDL-C 'high' (>3.5 mg/dl) and low (< 3.5 md/dl) (Executive Summary of the Third Report of the National Cholesterol Education Program, 2001).

Body weight was measured using a 'Tanita TBF-310', with a range of 0-270 kg and a standard deviation of 0.2 kg while height was determined by a microtoise tape

(Xenical Orlistat) with an accuracy of 0.1 cm. The body mass index (kg/m^2) of the subjects was calculated and categorised into 5 groups: underweight (<18.5 kg/m^2); normal (18.5 - 22.9 kg/m^2); risk of obesity (23 - 24.9 kg/m^2); obesity type 1 (25 - 29.9 kg/m^2); and obesity type 2 (≥ 30 kg/m^2) (WHO, 2004).

Permission and other administrative requirements for conducting the study were obtained from the relevant institutions. Ethical clearance was obtained from the Ethics Committee of the Faculty of Public Health, University of Indonesia. Prior to the study, all the subjects were informed about the purpose of the study and requested to give written consent of their voluntary participation. All information from respondents was held confidential.

Descriptive data analysis was used to show proportions of categorical data and mean value of continuous data (univariate analysis). Chi-square test with significant level of $p < 0.05$ was performed to identify associations between two categorical data (bivariate analysis). All analyses were conducted with STATA 9.1.

RESULTS

The mean age of the subjects was 46 years with a range of 26.2 and 54.8 years. Almost all the subjects were male (97.4%), and most of them had more than 9 years of schooling (91.2%) (Table 1). About 60.8% of the subjects had 'poor' knowledge of nutrition and health, while 15.2% had a family history of coronary heart disease.

The proportion of 'never smokers' was 44.9% followed by the 'smokers' (37.9%) and 'former smokers' (17.3%). The mean of body mass index (BMI) of the subjects was 25.07 kg/m^2 with a range of 17.37 kg/m^2 and 37.89 kg/m^2 . Obesity is a common problem among the subjects as 42.5% were 'obese type 1' followed by 'risk of obesity' (32%). Most of the respondents reported that they did not exercise regularly (74%). The mean intake of energy was 1827.53 Kcal while data amounted to 31.3% of total calorie intake.

Table 1. Background characteristics of respondents

Characteristics	Response (n=388)
Age (mean \pm SD, year)	46.0 \pm 1.7
Sex: male (%)	97.4
Educational level: >9 years of schooling (%)	91.2
Level of nutrition and health knowledge: poor (%)	60.8
Family history of CHD (%)	15.2
Smoking habit: never (%)	44.9
BMI: 'obese type 1 (25–29.9 kg/m ²) (%)	42.5
Exercise habit: irregular (%)	74.0
Intake of energy (mean \pm SD, %)	31.3 \pm 0.5
Intake of SAFA (mean \pm SD, %)	14 \pm 0.3
Intake of MUFA (mean \pm SD, %)	7.7 \pm 0.2
Intake of PUFA (mean \pm SD, %)	5.5 \pm 0.2
Hypercholesterolemia (%)	65.2
LDL-C 'abnormal' (%)	42.1
Hypertriglyceridemia (%)	39.8
HDL-C: 'low' (%)	6.3
The ratio of total cholesterol/HDL-C: 'high' (%)	15.0
The ratio of LDL-C/HDL-C: 'high' (%)	8.3

Table 2. Mean intake of *trans* fatty acid from fried foods, ruminant and margarine/HVO products

	n	Mean	SEM*	95%CI
Trans from fried food (% of calories)	373	0.20	0.02	0.17; 0.24
Trans from ruminants (% of calories)	301	0.09	0.01	0.08; 0.11
Trans from margarine (% of calories)	157	0.06	0.01	0.04; 0.08
Trans fatty acid total (% of calories)	386	0.48	0.03	0.42; 0.52

*Standard error of the mean

The percentage of energy derived from SAFA, MUFA and PUFA were 13.98%, 7.70% and 5.51%, respectively.

Blood lipid profiles

The prevalence of hypercholesterolemia, LDL-C 'abnormal' and hypertriglyceridemia were 65.21%, 42.15% and 39.82% respectively. The prevalence of HDL-C 'low' was at 6.3%, whereas the ratio of total cholesterol/HDL-C and the ratio of LDL-C/HDL-C were 'high' at 15% and 8.3% respectively (Table 1).

Trans fatty acids intake

Most of the respondents consumed two main sources of foods that contained *trans* fatty acid. Fried foods provided the highest contribution to total *trans* fatty acid intake. Based on a 24-hour recall diet, the geometric mean of *trans* fatty acid intake was 0.48% of calories (95% CI 0.42; 0.52), with a range of 0.004% - 2.87% of calories. Table 2 shows that there were significant differences between *trans* fatty acid intake from fried foods (0.20% of calories), ruminants (0.09% of calories) and margarine/HVO products (0.06% of

Table 3. Distribution of respondents by intake of fried foods, ruminants and margarine/HVO products

<i>Food intake</i>	<i>n= 388</i>	<i>Percentage (%)</i>
Fried foods		
≤ 2-3 servings/month	17	4.38
1-4 servings/week	132	34.02
≥5 servings/week	239	61.60
Ruminants		
≤ 2-3 servings/month	18	4.64
1-4 servings/week	254	65.46
≥5 servings/week	116	29.90
Margarine/HVO products		
≤ 2-3 servings/month	249	64.18
1-4 servings/week	120	30.93
≥5 servings/week	19	4.90

calories). The results from the semi-quantitative FFQ showed that fried food was most frequently consumed at ≥5 servings/week (61.6%), followed by ruminant products consumption at 1-4 servings/week (65.5%) and from margarine/HVO products at ≤2-3 servings/month (64.2%) (Table 3).

The Pearson correlation showed a significant positive correlation ($r = 0.320$, $p = 0.000$) between the intake of saturated fatty acids and trans fatty acids. Trans fatty acid intake tends to increase with increasing intake of saturated fatty acids (Figure 1). The coefficient of saturated fatty acid intake was 0.03, meaning that for every additional one percent of saturated fatty acid intake, the intake of trans fatty acids increases by an amount of 0.03% of total calories consumed.

Trans fatty acid intake from fried foods, ruminants and margarine/HVO products were grouped into 'high consumption' (≥ 4 servings/week) and 'low consumption' (< 4 servings/week). Table 4 shows that the relative risks of hypertriglyceridemia (RR:1.41, 95%CI 1.06;1.86), high total cholesterol/HDL-C ratio (RR:1.98, 95%CI 1.00;3.98) and dyslipidemia (RR:1.35, 95%CI

1.00;1.84) are associated with trans fatty acid intake.

DISCUSSION

The main sources of trans fatty acid among the subjects were hydrogenated food products of vegetable fat, such as margarine, shortening, cooking oil, and prepared food using HVO (hydrogenated vegetable oil). Other sources were microbial bio-hydrogenating process of multiple unsaturated fatty acids in ruminant abdomen. The formation of trans fatty acid also occurs during heating in the refinery process and deep frying process, where the transformation process from cis to trans fat begins at 180°C and increases with increasing temperature.

The subjects showed that the geometric mean of trans fatty acid intake was 0.48% of calories (95% CI 0.42; 0.52), whereas the arithmetic mean was 0.71% of calories, with a range of 0% and 2.87% of calories. The median intake of trans fatty acid intake was 0.59%. By comparison, the mean intake of trans fatty acid in West Europe was 0.5-2.1%

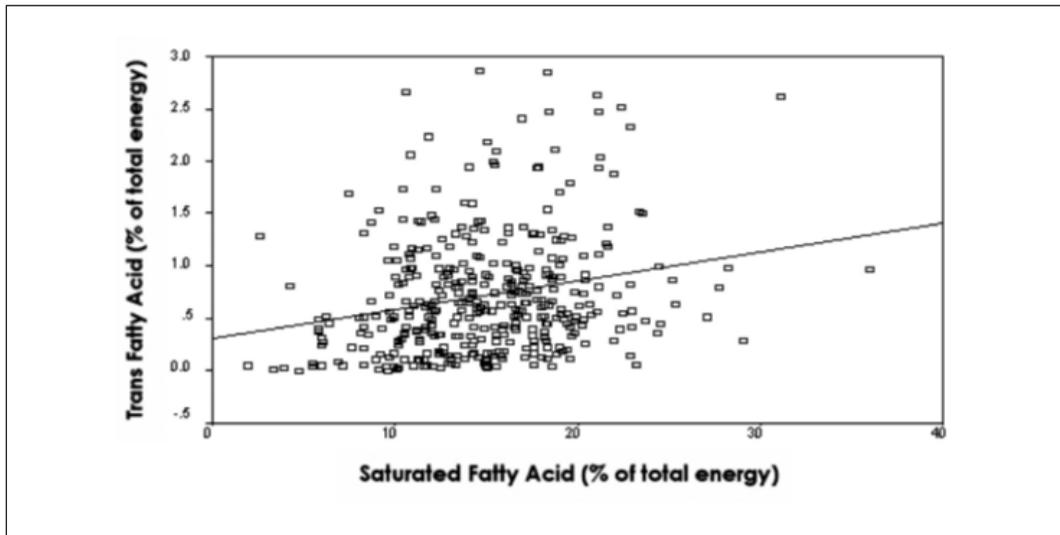


Figure 1. The relationship between intakes of saturated fatty acid and trans fatty acid (% of total energy).

Source: y (trans fatty acid intake) = $-0.02 + 0.03$ saturated fatty acid intake

Table 4. The relationship between trans fatty acid intake (fried foods, ruminant and margarine/HVO products) and blood lipid profile

Blood lipid profile	Fried food (n=373)		Ruminant (n=301)		Margarine(n=157)	
	RR (95%CI)	p value	RR (95%CI)	p value	RR (95%CI)	p value
Hypercholesterolemia	1.11 (0.84;1.47)	0.44	1 (0.71;1.41)	0.98	1.02 (0.71;1.46)	0.91
LDL-C 'high'	1.04 (0.79;1.36)	0.77	1.11 (0.80;1.55)	0.50	1.41 (1.00;1.99)	0.05
HDL-C 'low'	0.72 (0.41;1.25)	0.24	0.95 (0.49;1.81)	0.87	1.99 (0.91;4.35)	0.09
Hypertriglyceridemia	1.41 (1.06;1.86)	0.01*	0.82 (0.58;1.14)	0.24	0.73 (0.51;1.06)	0.09
Ratio of total cholesterol /HDL-C 'high'	1.98 (1.00;3.98)	0.04*	0.79 (0.49;1.28)	0.35	0.66 (0.40;1.10)	0.11
Ratio of LDL-C/ HDL-C 'high'	1.48 (0.95;2.32)	0.08	0.85 (0.48;1.50)	0.58	0.62 (0.32;1.22)	0.17
Dyslipidemia	1.35 (1.00;1.84)	0.04*	1.19 (0.81;1.75)	0.35	0.84 (0.50;1.25)	0.38

Note: * statistically significant ($p < 0.05$)

of calories (Hulshof *et al.*, 1999), while that in the United States was 2%-3% of calories (Allison *et al.*, 1999). The levels might be lower in these countries due to current stricter regulations. The American Heart Association (AHA) recommended that trans fatty acid intake should be not more than 1% of calories (Lichtenstein *et al.*, 2006).

The geometric mean of trans fatty acid derived from fried food (0.20% of calories) is higher than from ruminant products (0.09% of calories) and margarine/HVO products (0.06% of calories). The study showed that the highest content of trans fatty acid was found in fried foods (fried *tempe*, fried chicken, fried ox lung, fried egg), ruminant products (*rawon* meat, oxtail soup, beef cheese burger, meat *satay*, *rica-rica* meat) and margarine/HVO products (chocolates, biscuits, croissant, black forest cake, *sukro*, peanut).

Cooking through deep-frying is one of the most common methods in Indonesia. This is reflected in the annual increase in consumption of vegetable oils. Foods fried in normal temperature will absorb fat (8 to 25% oil), develop a golden colour, a crisp texture and good flavour. However, deep fried cooking not only produces fumes containing a mutagenic compound but also generates non-volatile hazardous compounds such as trans fatty acids, hydroperoxides and aldehydes. Sartika (2009) found that the initial formation of elaidic (trans fat) during the deep-frying process is followed by a decrease in oleic acid (cis).

This study found that trans fatty acids from fried foods increased triglyceride and the ratio of LDL/HDL cholesterol more than did ruminant and margarine/HVO products. De Roos, Schouten & Katan (2001) found that the LDL/HDL ratio was significantly higher in a diet of trans fatty acids than in saturated fatty acids. Changes in the ratio of LDL-C/HDL-C are better predictors of CVD risk than are changes in LDL-C alone (Mensink *et al.*, 2003).

Trans fatty acid intake had negative effects compared with saturated fatty acids in increasing the total cholesterol and LDL-C levels and in decreasing HDL-C levels (Judd *et al.*, 2002). Muller *et al.* (2003) found that HDL cholesterol was significantly higher after intake of saturated fatty acid than after a unsaturated fatty acid diet. High stearic acid soybean oil (saturated fatty acid) tended to raise LDL cholesterol, lower HDL cholesterol, and increase the ratio of total to HDL cholesterol in comparison with unsaturated fatty acids (Hunter, Zhang & Kris-Etherton, 2010). Lichtenstein *et al.* (1999) concluded that the consumption of products that are low in trans fatty acids and saturated fat has beneficial effects on serum lipoprotein cholesterol levels.

Motard-Belanger, Charest & Grenier (2008) reported that plasma LDL-C and HDL-C were significantly higher and lower after consumption of a high trans fatty acid diet from ruminants compared to a moderate trans fatty acid diet. In a study by Chardigny, Destailats & Malpuech-Brugère (2008), the industrial trans fatty acid diet lowered HDL-C, LDL-C and triacylglycerol concentrations compared with dairy trans fatty acid only in women. In addition to HDL cholesterol, LDL cholesterol is a known risk factor for heart disease that is affected by the consumption of trans fatty acids from industrial sources (Mozaffarian *et al.*, 2006; Mensink *et al.*, 2003). Oomen *et al.* (2001) in the Zutphen Elderly Study found that the intakes of both ruminant and industrially produced trans fatty acids predicted a higher risk of CAD.

This study did not find any significant interactions between consumption of ruminant and margarine/HVO products and total cholesterol, LDL-C, triglyceride and HDL-C. Fried foods, ruminants and margarine are a source of trans fatty acids in the Indonesian diet. Generally, consumption of fried foods prevails in almost all segments of the Indonesian society, while consumption of ruminant products and

margarine are more frequent in the middle to high income households.

The eating patterns in the study area included high-fat consumption (31.3% of calories), which is higher than that found in the general Indonesian population of 26.5% (Sartika, 2009). The main sources of the dietary fat of the subjects were fried foods, coconut milk, ruminants and margarine. According to the AHA, total fat intake should be no more than 30% of calories and should improve the fatty acid composition to prevent chronic diseases (Lichtenstein *et al.*, 2006).

The intake of saturated fat showed a positive correlation with trans fatty acid intake, indicating that in these subjects, their intake of trans fat would increase along with the increase in intake of saturated fats ($r = 0.32$ and $p = 0.000$). Sources of saturated fats in this population were fried foods, ruminant products (meat, milk and processed milk), food with coconut milk (*kalio*, *rendang*, curry, *lodeh* soup, etc), and margarine. The recommendation by the AHA limits trans fatty acid intake to 1% of calories (Lichtenstein *et al.*, 2006). Hu, Manson & Willet (2001) state that the replacement of saturated fatty acid and trans fatty acid with monounsaturated and polyunsaturated fats is more effective in preventing coronary heart disease than reducing overall fat intake.

Many factors contribute to heart disease risks including family history, smoking, lack of physical activity and high blood cholesterol. A high prevalence of dyslipidemia was found among the oil-station workers in East Kalimantan and this could be attributed to their high fat intake including frequent intake of saturated and trans fatty acids.

CONCLUSION AND RECOMMENDATIONS

A positive correlation between intake of trans fatty acids with saturated fatty acid intake

showed that a recommendation to reduce saturated fatty acid intake would be still relevant and in line with the decrease in trans fatty acid intake, especially reducing fried foods. Other studies are recommended to determine trans fatty acids intake and their health effects in different population segments in Indonesia.

REFERENCES

- Allison DB, Egan SK, Barraj LM, Caughman C, Infante M & Heimbach JT (1999). Estimated intakes of trans fatty and other fatty acids in the US population. *J Am Diet Assoc* 99(2): 166-74.
- Chardigny J-M, Destaillets F & Malpuech-Brugère C (2008). Do trans fatty acids from industrially produced sources and from natural sources have the same effect on cardiovascular diseases risk factors in healthy subjects? Results of the Trans Fatty Acids Collaboration (TRANSFACT) study. *Am J Clin Nutr* 87: 558-66.
- de Roos NM, Schouten EG & Katan MB (2001). Consumption of a solid fat rich in lauric acid results in a more favorable serum lipid profile in healthy men and women than consumption of a solid fat rich in trans fatty acids. *J Nutr* 131: 242-5.
- Executive Summary of the Third Report of the National Cholesterol Education Program (2001). Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* 285 (19): 2486-2497.
- Houterman S, Verschuren WMM, Oomen CM, Boersma-Cobbaert CM & Kromhout D (2001). Trends in total and high density lipoprotein cholesterol and their determinants in The Netherlands between 1993 and 1997. *Int J Epidemiol* 30(5): 1063-70.

- Hulshof KFAM, van Erp-Baart MA, Anttolainen M, Becker W, Church SM, Couet C & Hermann-Kunz E *et al.* (1999). Intake of fatty acids in Western Europe with emphasis on trans fatty acids: the TRANSFAIR Study. *Eur J Clin Nutr* 53: 142-57.
- Hunter JE, Zhang J & Kris-Etherton PM (2010). Cardiovascular disease risk of dietary stearic acid compared with trans, other saturated, and unsaturated fatty acids: a systematic review. *Am J Clin Nutr* 91: 46-63.
- Hu FB, Manson JE & Willett WC (2001). Types of dietary fat and risk of coronary heart disease: a critical review. *J Coll Nutr* 20(1): 5-19.
- Judd JT, Baer DJ, Clevidence BA, Kris-Etherton P, Muesing RA & Iwane M (2002). Dietary cis and trans mono-unsaturated and saturated FA and plasma lipids and lipoproteins in men. *Lipids* 37: 123-131.
- Lichtenstein AH, Ausman LM, Jalbert SM & Schaefer EJ (1999). Effects of different forms of dietary hydrogenated fats on serum lipoprotein cholesterol levels. *N Engl J Med* 340(25): 1933-40.
- Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA *et al.* (2006). Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation* 114(1): 82-96.
- Mensink RP, Zock PL, Kester AD & Katan MB (2003). Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr* 77: 1146-55.
- Motard-Belanger A, Charest A & Grenier G (2008). Study of the effect of trans fatty acids from ruminants on blood lipids and other risk factors for cardiovascular disease. *Am J Clin Nutr* 87: 593-9.
- Mozaffarian D, Katan MB, Ascherio A, Stampfer MJ & Willett WC (2006). Trans fatty acids and cardiovascular disease. *N Engl J Med* 354: 1601-13.
- Muller H, Lindman AS, Brantsaeter AL & Pedersen JI (2003). The serum LDL/HDL cholesterol ratio is influenced more favorably by exchanging saturated with unsaturated fat than by reducing saturated fat in the diet of women. *J Nutr* 133(1): 78-83.
- National Health Survey (2005). Morbidity and Disability Study. Jakarta: Ministry of Health; Indonesia.
- Oomen CM, Ocke MC, Feskens EJM, van Erp-Baart A, Kok FJ & Kromhout D (2001). Association between trans fatty acid intake and 10-year risk of coronary heart disease in the Zutphen Elderly Study: a prospective population-based study. *Lancet* 357: 746-51.
- Sartika RAD (2009). Influence of deep frying in forming of trans fatty acid. *Makara, Sains* 13: 23-28.
- US Department of Agriculture, Agricultural Research Service (2002). USDA Nutrient Database for Standard Reference.
- Varady KA & Jones PJH (2005). Combination of diet and exercise interventions for the treatment of dyslipidemia: an effective preliminary strategy to lower cholesterol levels? *J Nutr* 135(8): 1829-35.
- WHO Expert Consultation (2004). Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 363: 157-163.