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ORIGINAL ARTICLE

Can dietary saturated fat be beneficial in prevention of stroke risk? A meta-analysis

Pengfei Cheng[1] · Jingxia Wang[1] · Weihua Shao[2] · Meiling Liu[3] · Hui Zhang[1]

Abstract   We conducted a meta-analysis to summarize available evidence regarding the relation between saturated fatty acid (SFA) intake and stroke risk. We searched multiple electronic databases through February 2016. Log relative risks (RRs) with 95 % confidence intervals (CIs) of the highest versus the lowest for cohort studies were weighed by the inverse variance method to obtain combined RRs. 15 prospective studies including 476,569 individuals and 11,074 strokes were included. Higher SFA intake was associated with reduced overall stroke risk [RR = 0.89 (95 % CI 0.82–0.96)] and fatal stroke risk [RR = 0.75 (95 % CI 0.59–0.94)]. Subgroup analysis indicated that higher SFA intake was associated with reduced stroke risks for East-Asians [RR = 0.79 (95 % CI 0.69–0.90)], for dose <25 g/day [RR = 0.81 (95 % CI 0.71–0.92)], for males [RR = 0.85 (95 % CI 0.75–0.96)], and for individuals with body mass index (BMI) <24 [RR = 0.75 (95 % CI 0.65–0.87)], but not for non East-Asians, females, and individuals with dose ≥25 g/day and BMI ≥24. This meta-analysis reveals that higher SFA intake is inversely associated with risk of stroke morbidity and mortality with race, sex, and BMI as key factors influencing this risk. There seems to be a threshold of SFA intake for inverse relation of SFA intake with stroke. However, the stroke-reducing or -increasing effects for specific subtypes and specific food sources of SFA can be concealed. Functions of specific subtypes of SFA (e.g. lignoceric acid) and specific food sources of SFA (i.e. plant vs. animal) in relation to stroke need to be clarified in further studies.

Introduction

Saturated fatty acids (SFAs) have no double bonds. A reduction in SFA was regularly recommended as a key intervention to reduce incidence and mortality of cardio-vascular disease (CVD). However, a recent meta-analysis [1] indicates that SFA intake has no association with coronary disease, and another meta-analysis [2] including eight prospective studies suggests no association between SFA and stroke risk. Some evidence [3] has found that all SFAs raise high-density lipoprotein cholesterol (HDL-C). In recent years, several large prospective studies [4–9] were added and reported inconsistent results of SFA intake in relation to stroke risk. For instance, four of these studies [5–8] suggest no associations between SFA intake and stroke risk, while one study [4] suggests SFA intake is inversely associated with intraparenchymal hemorrhage and ischemic stroke, and another study [9] indicates SFA intake is inversely associated with deep intraparenchymal hemorrhage and lacunar infarction. Although one recent meta-analysis [10] indicates that SFA intake is not associated with ischemic stroke, there is still lacking the summary evidence on total stroke and hemorrhagic stroke. In

✉ Pengfei Cheng
    chengpengfei@jmsu.edu.cn

[1]   Department of Histology and Embryology, School of Basic Medical Sciences, Jiamusi University, Jiamusi, Heilongjiang, China

[2]   Department of Respiratory Medicine, The First Affiliated Hospital of Chongqing Medical University, Chongqing, China

[3]   Department of Gastroenterology, The First Affiliated Hospital of Chongqing Medical University, Chongqing, China

addition, the pivotal factors such as fatal stroke risk and body mass index (BMI) were not evaluated. Thus, there is a requirement to clarify the roles of SFA in relation to stroke risk. Here, we conducted a meta-analysis of prospective cohort studies to clarify the associations between SFA intake and stroke risk based on the epidemiologic characteristics of study population.

Methods

Search strategy and selection criteria

References for this meta-analysis were identified through searches of PubMed, Embase, and Web of Knowledge for studies of SFA with the search terms: ("fat" OR "fatty acids") combined with ("stroke" OR "cerebrovascular disease" OR "cerebrovascular accident" OR "cerebrovascular event" OR "TIA" OR "transient ischemic attack"). Reference lists of retrieved studies and review articles were hand searched to identify any eligible resources. There were no language restrictions. We included studies that met the following criteria (1) a prospective cohort design was used; (2) relative risks (RRs) and their corresponding 95 % confidence intervals (CIs) of stroke relating to SFA intake were provided; (3) covariates (such as alcohol, smoking, and blood pressure) were controlled in the multivariate analysis; and (4) only the most recent publication, or the one with the longest follow-up period, was included when duplicate reports based on the same cohort occurred. We excluded articles that met the following criteria: (1) non prospective cohort study design was used, (2) reviews, (3) non-human studies, and (4) conference abstracts or reports lacking of RRs and the corresponding 95 % CIs of stroke relating to SFA intake.

Data extraction

After the initial assessment for eligibility, P.F.C. and W.H.S. independently completed a data extraction form. Any disagreement about the data was resolved by a third reviewer (M.L.L). We extracted the following information from the selected publications: first author's name, publication year, cohort name, country, age, sex, size of study population, number of stroke events, methods for saturated fat intake assessment, BMI, length of follow-up, levels of SFA intake, outcome assessment (fatal or non-fatal), hazard ratios (HRs) or RRs of stroke and corresponding 95 % CIs for SFA intake, and covariates adjusted in the statistical analysis. The Newcastle-Ottawa Scale [11] was used for the study quality assessment in this meta-analysis, and

we defined a high-quality study as a study with ≥ 7 awarded stars.

Statistical analysis

For calculating the risk of stroke morbidity and mortality, log RRs of the highest versus the lowest for cohort studies were combined by an inverse variance method. RRs for association of SFA intake and stroke that were often differently reported by each study (such as per-unit or per^{-1}-SD change or comparing quintiles, quartiles, thirds, and other groupings) were transformed, using methods previously described [1]. For obtaining more robust RRs of stroke for SFA intake, we referred to one previous meta-analysis, [2] and RRs with max multivariates adjusted were extracted for each study. In addition, we retrieved all supplemental files of the included studies for RRs of total stroke, specific types of stroke, and specific sex of stroke if available. Statistical heterogeneity was evaluated with Q and I^2 statistics [12]. An I^2 of 25, 50, and 75 % represented low, moderate, and high heterogeneity, respectively. We used the fixed effects model for statistics when $I^2 < 50 \%$; otherwise, we used the random effects model for calculating pooled RRs of stroke for SFA intake.

We performed analyses stratified by length of follow-up, race, dose, sex, stroke type, BMI, score of study quality, and dietary assessment methods. Sensitivity analysis was conducted to detect the potential bias of studies in this meta-analysis. Evidence of publication bias was assessed using funnel plots and the Egger's test. Data analysis was performed with STATA software package (version 12.0) (StataCorp, College Station, TX, USA), and $P < 0.05$ was considered as statistically significant.

Results

Study characteristics

1828 records were identified through database searching, and 2 records [13, 14] were retrieved from a previous meta-analysis. After screening the abstracts, 140 qualified for full review (Fig. 1). Finally, 15 prospective studies [4–9, 13–21] were included (two studies [7, 22] were based on the same cohort, the one [7] with the longest follow-up period was included in this meta-analysis), published between 1984 and 2013 including 11,074 stroke events among 476,569 participants. Five studies were from USA, [8, 13, 15, 17, 18], five from Japan, [4, 9, 16, 19, 20], two from Sweden, [6, 7], one from Israel, [14], one from United Kingdom, [5], and one from Greece [21]. Age ranged from 20 to 89 years. Number of participants ranged from 832 to
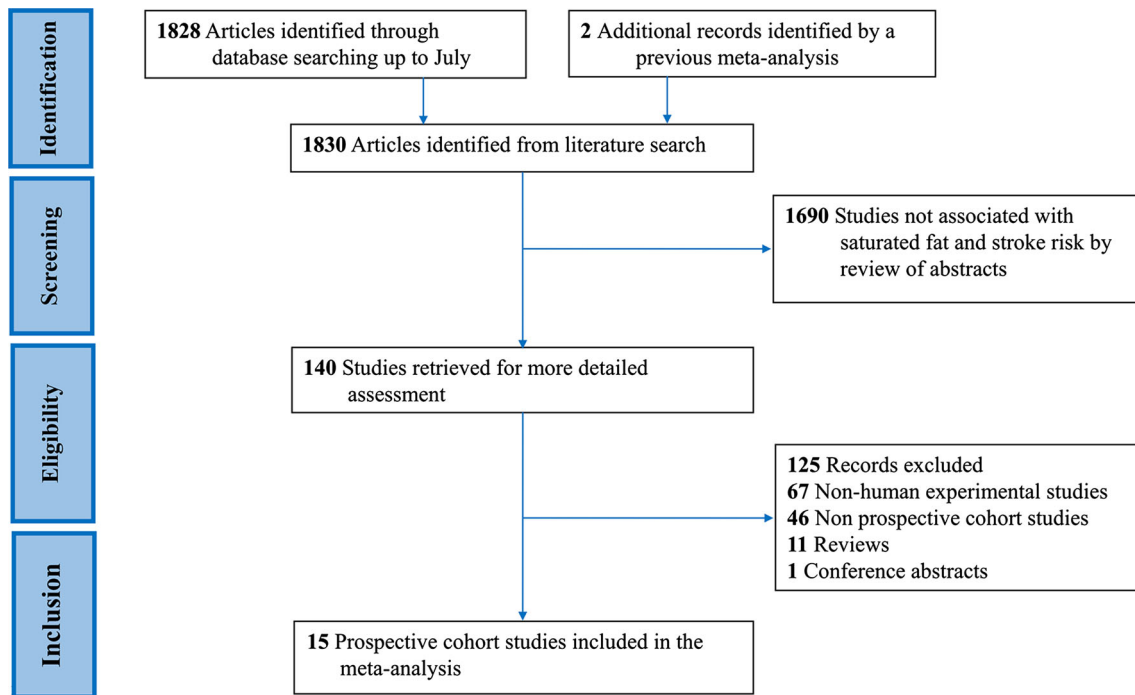
Fig. 1 Flow chart of selection of studies

87,025, and the number of stroke events ranged from 72 to 3192. Three studies [13, 15, 19] used 24-h recall method, Eleven studies [4–6, 8, 9, 14, 16–18, 20, 21] used food frequency questionnaire (FFQ), and one study [7] used diet history method to evaluate dietary intake of SFA. Average length of follow-up ranged from 7.6 to 23 years. The average dietary SFA intake available in the high categories ranged between 15.4 and 24.9 g/day among East Asian cohorts and 22.1 and 36 g/day among non East Asian cohorts (Table 1). RRs of total stroke, intraparenchymal haemorrhage, and ischaemic stroke with max multivariate adjusted for two included studies [4, 9] were retrieved from the "Results" section rather than routinely from the data sources of tables, with the permission of Boards of Japan Collaborative Cohort Study for Evaluation of Cancer Risk (JACC); and Japan Public Health Center-based Prospective Study (JPHC) unpublished RRs of subarachnoid hemorrhage with max multivariate adjusted were provided by authors of these two studies. RRs of total stroke, specific type of stroke, and specific sex of stroke were retrieved from the supplemental files of two included studies [7, 19]. All studies report RRs adjusted for age, hypertension or measured blood pressure; two studies [13, 16] did not report RRs adjusted for diabetes; and only one study [16] did not report RRs adjusted for smoking. Of these 15 studies, 12 studies [4–9, 14, 15, 17–19, 21] were awarded as lower quality studies, while three studies [13, 16, 20] were awarded as high quality studies.

Primary outcome

Higher SFA intake was associated with a reduced overall stroke risk [0.89 (95 % CI 0.82–0.96)] (Fig. 2) and a fatal stroke risk [0.75 (95 % CI 0.59–0.94)] (Table 2).

Secondary outcome

Subgroup analysis indicated that higher SFA intake was positively associated with reduced stroke risks for follow-up duration ≥14 years [RR = 0.77 (95 % CI 0.68–0.88)], for East-Asians [RR = 0.79 (95 % CI 0.69–0.90)], for dose <25 g/day [RR = 0.81 (95 % CI 0.71–0.92)] (Fig. 3), for males [RR = 0.85 (95 % CI 0.75–0.96)], for ischemic stroke [RR = 0.90 (95 % CI 0.82–0.99)], for hemorrhagic stroke [RR = 0.76 (95 % CI 0.62–0.92)], for BMI <24 [RR = 0.75 (95 % CI 0.65–0.87)], for quality score ≥7 stars [RR = 0.89 (95 % CI 0.82–0.96)], and for FFQ [RR = 0.91 (95 % CI 0.83–0.99)]. However, higher SFA intake demonstrated no associations with stroke risk for follow-up duration <14 years [RR = 0.95 (95 % CI 0.87–1.04)], for non East-Asians [RR = 0.94 (95 % CI 0.86–1.02)], for dose ≥25 g/day [RR = 1.02 (95 % CI 0.89–1.15)], for females [RR = 1.04 (95 % CI 0.91–1.18)], for BMI ≥24 [RR = 0.94 (95 % CI 0.84–1.04)], for quality score <7 stars [RR = 0.90 (95 % CI 0.66–1.24)], and for 24 h recall [RR = 0.76 (95 % CI 0.51–1.14)] (Table 2).

Table 1 Baseline characteristics of included studies

First author (Year) [References]	Cohort name (country)	Age (sex)	Number of participants (stroke events)	Method of dietary assessment	BMI	Follow-up (years)	Dietary SF (g/day) Per-unit or per^{-1}-SD change		Fatal or non-fatal strokes	Maximum adjustment available
							High	Low		
McGee (1984) [13]	Honolulu Heart Program (USA), based on men of Japanese ancestry	45–68 (M)	7088 (492)	24-h recall	NA	10	Mean: 12.7 % of total energy (age-adjusted)		Mixed	Age, total energy intake, SBP, BMI, smoking, family history of MI, physical activity, intakes of PUFAs, alcohol, protein, carbohydrate, vegetables, and cholesterol
Goldbourt (1993) [14]	Israeli Ischemic Heart Disease prospective Study (Israel)	≥40 (M)	11,876 (362)	FFQ	NA	23	NA		Fatal	Age, SBP, total cholesterol, smoking, diabetes, QI, CHD baseline
Gillman (1997) [15]	Framingham Heart Study (USA)	45–65 (M)	832 (73)	24-h recall	≥24	20	Mean: 15.0 % of total energy	1 % increase	Mixed	Age, energy, SBP, smoking, glucose intolerance, BMI, physical activity index, LVH, energy, alcohol, fruits and vegetables
Seino (1997) [16]	Shibata Study (Japan)	40–89 (M/F)	2,283 (75)	FFQ	<24	15.5	15.4 (Q4)	7.2 (Q1)	Mixed	Age, sex, DBP, atrial fibrillation
Iso (2001) [17]	Nurses' Health Study (USA)	34–59 (F)	85,764 (690)	FFQ	NA	14	36 (Q5)	20 (Q1)	Mixed	Age, smoking, time interval, BMI, alcohol, menopausal status, hormone use, exercise, aspirin use, multivitamin use, vitamin E use, n-3 fatty acid intake, calcium intake, hypertension, diabetes, high cholesterol levels, intakes of cholesterol, monosaturated fat, polyunsaturated fat (linoleic acid), vegetable protein, intakes of saturated fat, trans unsaturated fat, or animal protein
He (2003) [18]	Health Professional Study (USA)	40–75 (M)	43,732 (725)	FFQ	NA	14	31 (Q5)	17 (Q1)	Mixed	Age, BMI, physical activity, hypertension, smoking, aspirin, multivitamins, alcohol, potassium, fiber, vitamin E, fruit and vegetables, total energy intake, and hypercholesterolaemia at baseline. Additional adjustments based on other fat categories
Iso (2003) [19]	Ikawa, Ishizawa, Yao, Noichi, and Kyowa Study (Japan)	40–69 (M/F)	4775 (295)	24-h recall	<24	14.3	17.1 (Q4)	5.2 (Q1)	Mixed	Age, sex, total energy intake and BMI, hypertension, diabetes, serum total cholesterol, smoking, ethanol intake, and menopausal status
Sauvaget [20] (2004)	Adult Health Study (Japan)	35–89 (M/F)	3731 (90)	FFQ	<24	14	21 (T3)	7 (T1)	Fatal	Age and sex, radiation dose, city, BMI, smoking, alcohol, hypertension and diabetes
Yamagishi (2010) [4]	Japan Collaborative Cohort Study for Evaluation of Cancer Risk (Japan)	40–79 (M/F)	58,453 (976)	FFQ	<24	14.1	F: 19.8 (Q5) M: 20.3 (Q5)	F: 9.4 (Q1) M: 9.2 (Q1)	Fatal	Age, sex, hypertension and diabetes, smoking, alcohol consumption, BMI, mental stress, walking, sports, education, and intakes of total energy, animal protein, cholesterol, $n-3$ and $n-6$ polyunsaturated fatty acids, vegetables, and fruit

Table 1 continued

First author (Year) [References]	Cohort name (country)	Age (sex)	Number of participants (stroke events)	Method of dietary assessment	BMI	Follow-up (years)	Dietary SF (g/day) Per-unit or per⁻¹-SD change High	Low	Fatal or non-fatal strokes	Maximum adjustment available
Atkinson (2011) [5]	Caerphilly cohort (UK)	45–59 (M)	2,710 (225)	FFQ	≥24	18	NA		Mixed	Age, energy, Smoking, adult social class, marital status, alcohol intake, vitamin C intake, vegetable fiber intake. Blood pressure, cholesterol, BMI, fasting glucose, diabetes, atrial fibrillation
Leosdottir (2007) [22] Wallstrom (2012) [7]	Malmö Diet and Cancer Study (Sweden)	44–73 (M/F)	28,098 (755)	Diet history method	≥24	13.5	M: 22.7 (Q5) F: 22.1 (Q5)	M: 13.0 (Q1) F: 12.9 (Q1)	Mixed	Age, smoking, alcohol, socio-economic status, marital status, physical activity, BMI, fiber intake, and blood pressure. Total fat intake for the ratio between unsaturated and saturated fats
Yaemsiri (2012) [8]	Women's Health Initiative Observational Study (USA)	50–79 (F)	87,025 (1049)	FFQ	≥24	7.6	26.1 (Q5)	12.9 (Q1)	Mixed	Age, race, education, income, smoking, hormone use, total metabolic equivalent task hours per week, alcohol, coronary heart disease, atrial fibrillation, diabetes, aspirin use, use of antihypertensive medication, use of cholesterol-lowering medication, BMI, SBP, and energy, dietary vitamin E, fruits and vegetable intake, and fiber
Larsson (2012) [6]	Swedish mammography cohort (Sweden)	49–83 (F)	34,670 (1680)	FFQ	≥24	10.4	35.5 (Q5)	19.5 (Q1)	Mixed	Age, smoking, education, BMI, physical activity, hypertension, diabetes, aspirin use, myocardial infarction, alcohol, protein, dietary fiber, specific types of fat and cholesterol
Misirli (2012) [21]	European Prospective Into Cancer and Nutrition (Greece)	20–87 (M/F)	23,601 (395)	FFQ	NA	10.6	Mean: 29.5 per increment		Mixed and fatal	Age, sex, education, smoking status, BMI, physical activity, hypertension, diabetes, and total energy intake
Yamagishi (2013) [9]	Japan Public Health Center-based prospective Study (Japan)	45–74 (M/F)	81,931 (3192)	FFQ	<24	11.1	24.9 (Q5)	9.6 (Q1)	Mixed	Age, sex, energy intake, cohort, smoking, alcohol, BMI, sports at leisure time, walking and standing time, mental stress; energy-adjusted intakes of carbohydrate, protein, cholesterol, vegetables, fruit, and calcium; treatment for hypertension and diabetes

BMI body mass index, *CHD* coronary heart disease, *DBP* diastolic blood pressure, *F* female, *FFQ* food frequency questionnaire, *LVH* left ventricular hypertrophy, *M* male, *MI* myocardial infarction, *NA* not available, *PUFA* polyunsaturated fatty acid, *Q* quartile/quintile, *QI* Quetelet index, *SBP* systolic blood pressure, *SF* saturated fat, *T* tertile, *TUF* trans-unsaturated fat, *USA* United States of America
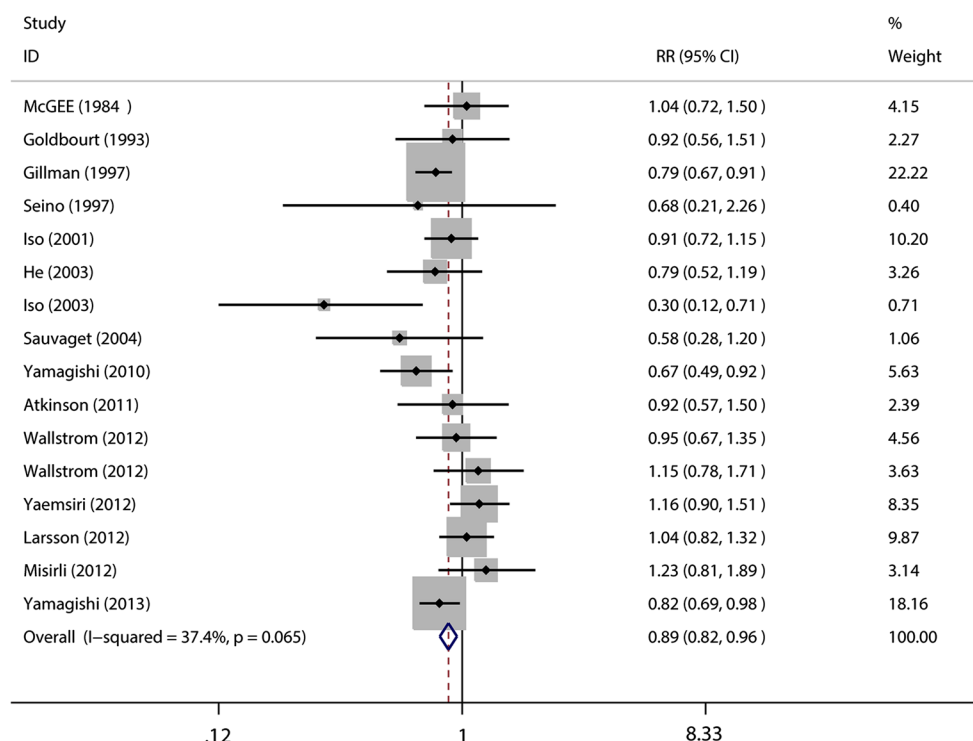
Fig. 2 Forest plot of relative risks (RRs) for saturated fatty acid intake and stroke risk. *CI* confidence interval, *RR* relative risk

Publication bias

The statistical analysis indicated that there was no publication bias for intake of SFA in relation to stroke risk according to the Egger's test ($P = 0.618$), and the funnel plots were basically symmetrical by visual inspection (Fig. 4a).

Sensitivity analysis

The results of sensitivity analysis demonstrated that higher SFA intake was positively associated with a reduced stroke risk when omitting each study at a time (Fig. 4b).

Discussion

This meta-analysis identified 15 prospective studies [4–9, 13–21] including 476,569 individuals and 11,074 stroke events, indicating that higher SFA intake is inversely associated with risk of overall stroke, stroke mortality, ischaemic stroke, and haemorrhagic stroke, although one previous meta-analysis [2] including eight prospective studies suggests no association between SFA and stroke risk. The potential biological mechanisms of reduced stroke risks by higher intake of SFA are complicated. All SFAs raise HDL-C, with carbohydrate consumption as the

reference, lauric (12:0), myristic (14:0), and palmitic (16:0) acid raise total cholesterol and LDL-C, whereas stearic acid (18:0) does not [3]. There is evidence showing that the elevation of baseline triglyceride levels is positively associated with the incidence of stroke, [23] while increased HDL-C levels are associated with reduced risk of ischemic stroke [24]. Thus, based upon our results, individuals seem to benefit more from the HDL-C raising effects of higher SFA intake.

Some recent studies [25–27] demonstrated that total very-long chain SFAs in plasma were associated with decreased risks of coronary heart disease and diabetes (main risk factors for stroke); another study [28] indicated that higher levels of circulating palmitic acid (16:0) were associated with a higher risk of AF, while higher levels of circulating stearic acid (18:0), arachidic acid (20:0), behenic acid (22:0), and lignoceric acid (24:0) were each associated with a lower risk of AF. Very long-chain SFAs (saturated fats with a chain length of 20 or more carbon atoms) are derived from dietary sources, such as peanuts, macadamia nuts, and canola oil, and may also be produced endogenously [29, 30]. Some short-term feeding trials have shown that dietary intake of macadamia nuts increase the circulating levels of arachidic and behenic acids [31] and dietary intake of peanut butter can increase circulating behenic and lignoceric acids [32]. A recent meta-analysis [33] including nine independent prospective cohorts

Table 2 Saturated fatty acid intake and stroke risk

Outcome	Studies (N)	RR	95 % CI		P value	I² for heterogeneity
Total stroke	15	0.89	0.82	0.96	0.002*	37.4
Fatal stroke risk	4	0.75	0.59	0.94	0.014*	0
Follow-up duration						
<14 years	9	0.95	0.87	1.04	0.304	16.6
≥14 years	6	0.77	0.68	0.88	<0.001*	20.4
Race						
Non East-Asian	9	0.94	0.86	1.02	0.143	17.2
East-Asian	6	0.79	0.69	0.90	0.001*	42.4
Dose						
≤25 g/day	6	0.81	0.71	0.92	0.001	45.3
>25 g/day	5	1.02	0.89	1.15	0.808	2.7
Sex						
Female	4	1.04	0.91	1.18	0.587	0
Male	6	0.85	0.75	0.96	0.008*	0
Stroke type						
Ischemic	10	0.90	0.82	0.99	0.025*	35.9
Hemorrhagic	6	0.76	0.63	0.93	0.007*	42.5
BMI						
<24	5	0.75	0.65	0.87	<0.001*	34.1
≥24	5	0.94	0.84	1.04	0.212	41.5
Quality score						
<7 stars	3	0.90	0.66	1.24	0.531	9.5
≥7 stars	12	0.89	0.82	0.96	0.002*	44.8
Dietary assessment						
24 h recall	3	0.76	0.51	1.14	0.182	69.8
FFQ	11	0.91	0.83	0.99	0.036*	26.9
Diet history method	1	1.03	0.80	1.34	–	–

CI confidence interval, *FFQ* food frequency questionnaire, *N* number, *RR* relative risk

* $P < 0.05$

indicated that nut consumption is inversely associated with risk of stroke. Very long-chain SFAs are known to modulate retinal function, anti-inflammatory reactions, [34], and peroxisome-related functionality in the etiology of CVD [35]. Unfortunately, the included studies in this meta-analysis did not report the association between specific subtype of SFA and stroke risk. Thus, the stroke-reducing or stroke-increasing effects for specific subtype of SFA can be concealed.

The background SFA intake was about 12 % (range 9–16 %) in North American studies, while it was about 9 % (range 5–14 %) in Asian studies, and consistently <7 % in Japanese cohorts [9, 10]. For this meta-analysis, the average dietary SFA intake available in the high categories ranged between 15.4 and 24.9 g/day among Japanese cohorts, 22.1 and 36 g/day among non East-Asian cohorts, and 26.1 and 36 g/day among North American cohorts. Based on our results of dose subgroup, there seems to be a threshold of SFA intake (less than 25 g/day;

however, the accuracy of this value still needs to be confirmed) for inverse relation of SFA intake with stroke. Compared with other industrialized countries, however, Japan consumes low amounts of fat from animal sources [36]. One Greece cohort study [21] included in this meta-analysis was based on traditional Mediterranean diet, which is characterized by high consumption of legumes, nuts, cereals, vegetables and fruits; moderate-to-high consumption of fish; low consumption of dairy products and meat. Thus, the effect of SFA might not be uniform across ethnic populations, intake levels, or possibly food sources. Further studies are needed to evaluate the association between specific food sources of SFA (i.e. plant vs. animal) and risk of stroke.

In the subgroup analysis, participants with longer duration of SFA intake demonstrated a reduced risk of stroke, and studies with a higher quality score provided homogeneous results. Sex, [37] and BMI [38] are factors influencing the incidence of stroke; our subgroup analysis
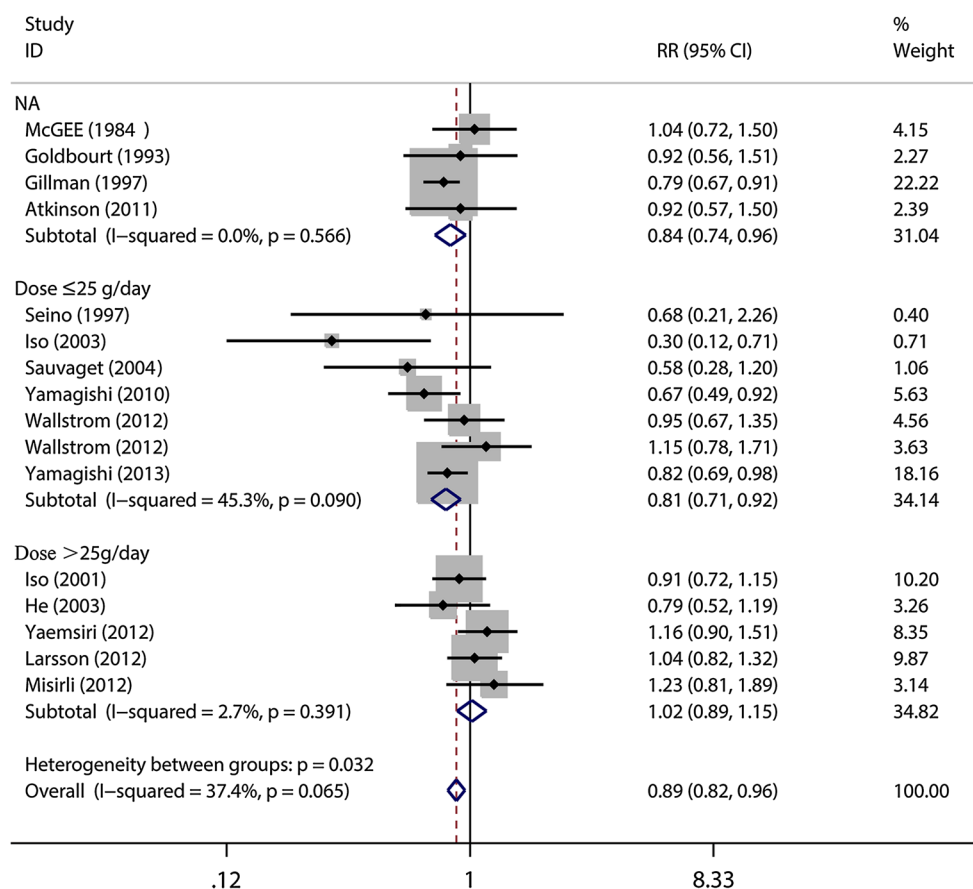
Fig. 3 Forest plot of relative risks (RRs) for saturated fatty acid intake and stroke risk in dose subgroup. *CI* confidence interval, *NA* not available, *RR* relative risk

indicated that higher SFA intake was associated with reduced stroke risk for males and for individuals with lower BMI. Our results demonstrated that higher SFA intake was associated with reduced stroke risk for FFQ subgroup. Three studies [13, 15, 19] included in this meta-analysis used 24 h recall method, which is relatively easy to collect, but the information may not well reflect long-term dietary patterns. However, most included (eleven) studies [4–6, 8, 9, 14, 16–18, 20, 21] in this meta-analysis used FFQ, which can assess long-term diets. Sensitivity analysis demonstrates that high SFA intake is persistently associated with a reduced stroke risk when omitting each study in turn. The heterogeneity between studies in this meta-analysis was at a low level ($I^2 = 37.4$ %). When we conducted the sex subgroup analysis, the heterogeneity decreased to zero in both male and female subgroups. Thus, sex is considered to be a source of heterogeneity.

Study limitations: first, one limitation inherited in observational studies is the possibility of differing degrees of adjustment for potential confounders. Although majority of studies adjusted for known risk factors for stroke, such as age, smoking, hypertension or measured blood pressure,

diabetes, and other dietary factors, the possibility of residual confounding cannot be excluded. Second, this meta-analysis included East-Asians and non East-Asians; the upper limit of normal BMI for Asian populations is 23 kg/m^2, and an upper limit of normal BMI issued by World Health Organization guidelines is 25 kg/m^2 [39]; in the stratification analysis for BMI, for obtaining more conservative results, we used a midpoint of BMI (24) for subgroup dividing to minimize the bias of observational results.

Conclusion

This meta-analysis of prospective studies reveals that higher SFA intake is inversely associated with risk of stroke morbidity and mortality with race, sex, and BMI as key factors influencing this risk. There seems to be a threshold of SFA intake for inverse relation of SFA intake with stroke. However, the stroke reducing or increasing effects for specific subtypes and specific food sources of SFA can be concealed. Functions of specific subtypes of SFA (e.g. lignoceric acid) and specific food sources of SFA
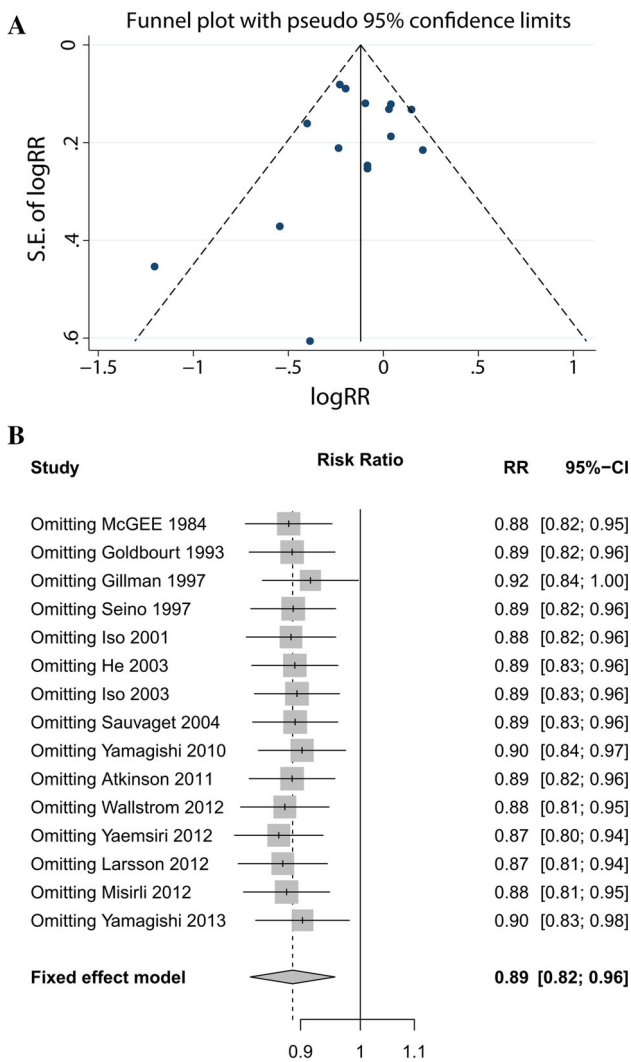
A

B

Fig. 4 a Publication bias for saturated fatty acids and stroke risk.
b Sensitivity analysis for saturated fatty acid intake and stroke risk. *CI*
confidence interval, *RR* relative risk, *S.E.* standard error

(i.e. plant vs. animal) in relation to stroke need to be
clarified in further studies. This meta-analysis adds to
recent high-profile scientific evidences that have called for
a re-evaluation of dietary guidelines for saturated fat intake
and a re-appraisal of the effects of saturated fat on health.

Compliance with ethical standards

Conflict of interest None.

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