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Palm oil and cardiovascolar disease: what scientific evidence ?

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OUTLINE

1. Introduction

General information on palm oil

2. Scientific evidence on palm oil consumption and cardiovascular diseases

3. Scientific evidence on saturated fatty acids and cardiovascular diseases – old and new studies

4. Conclusions

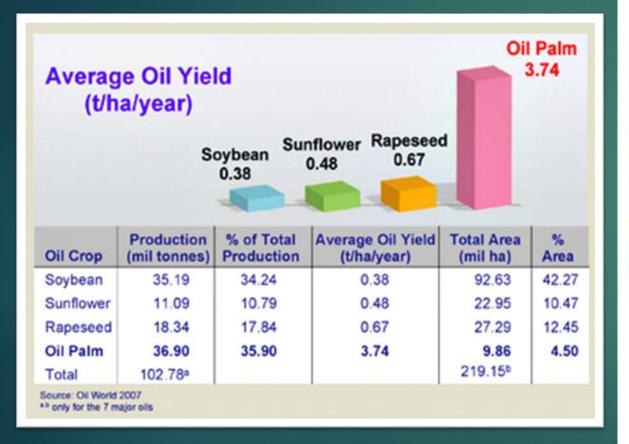
Palm Oil

- Vegetable oil from the fruit of the palm tree (Elaeis guineensis) originating West Africa
- Widespread throughout the tropical areas of America and South East Asia.



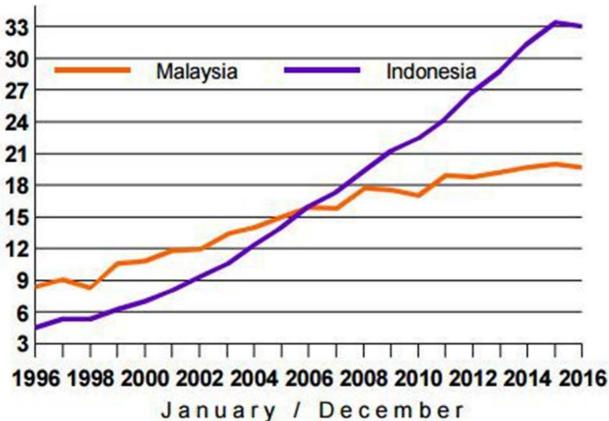
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Palm oil productivity



PALM OIL : Production in Key Countries (Mn T)

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Fatty acid composition

Guillén N et al, Extra Virgin Olive Ol and Mice Lacking Apolipoprotein E

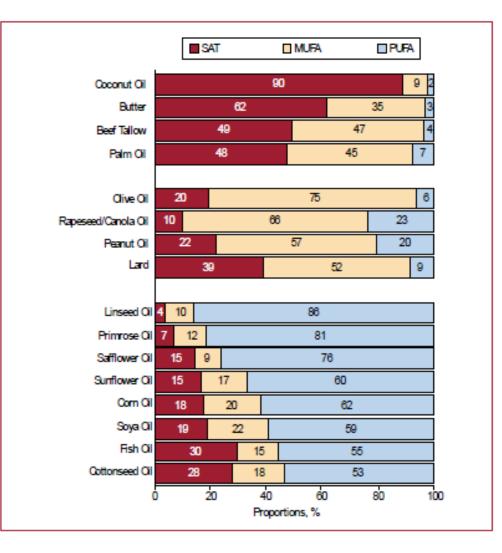


Figure 1. Percentage of the different types of fatty acids present in the different oils and fats. The most frequently consumed oils in the world are as follows: soybean (used by 20% of the world population), paim (18%), surflower (13%), and cotza/ candia (12%). Adapted from Suma et al.¹³ with permission from the Spanish Society of Obesity. MLFA indicates monounsaturated fat; PUFA polyunsaturated fat; SAT, saturated fat.









I Nostri Biscotti Senza Olio di Palma



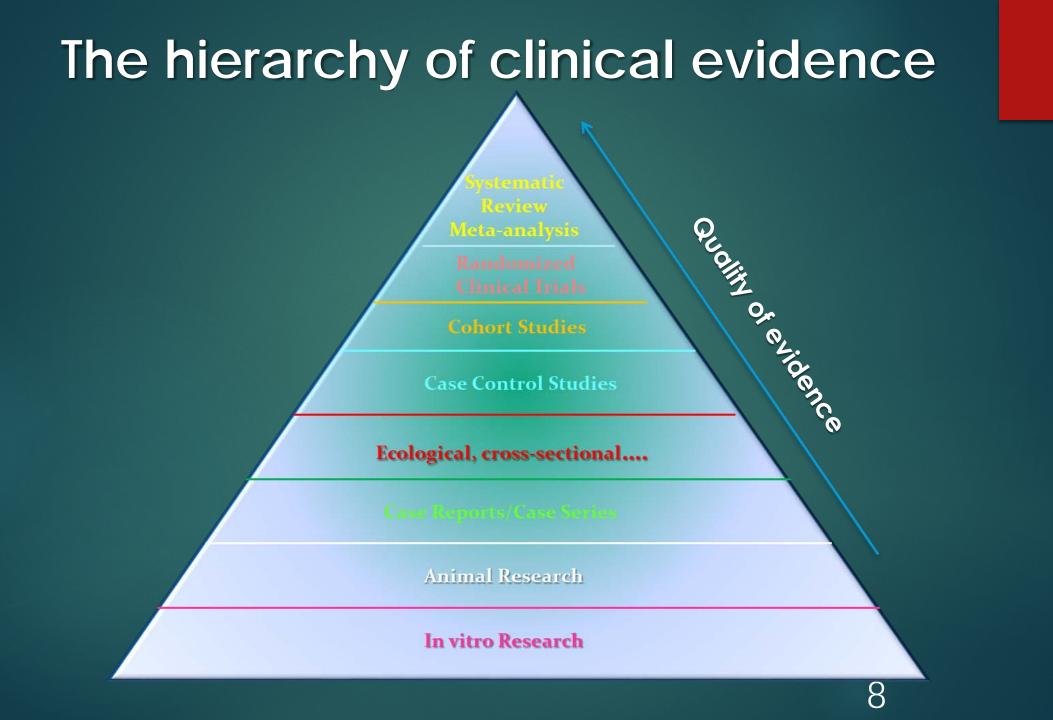




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2. Scientific evidence on palm oil consumption and cardiovascular diseases





The American Journal of Clinical Nutrition

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Palm oil and blood lipid–related markers of cardiovascular disease: a systematic review and meta-analysis of dietary intervention trials^{1–3}

Elena Fattore, Cristina Bosetti, Furio Brighenti, Carlo Agostoni, and Giovanni Fattore

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What evidence on the effect of palm oil on blood lipid markers of cardiovascular disease risk?

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P = **Population:** general population (humans)

I = Intervention: diets rich in palm oil, palm olein or palmitic acid at the sn-1,3 position

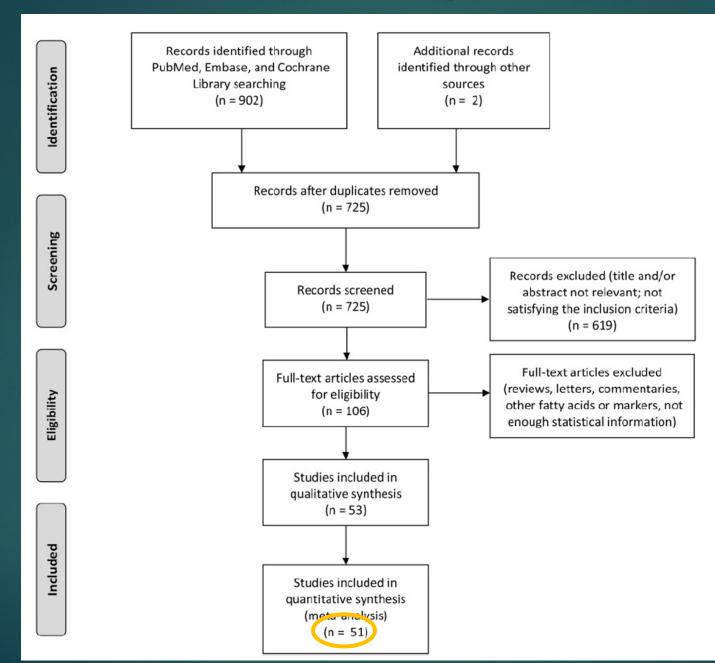
C = Comparator: diets rich other dietary fats
•stearic acid
•myristic and/or lauric acid
•monounsaturated fatty acids (MUFA), mainly in oleic acid
•polyunsaturated fatty acids (PUFA), mainly linoleic acid
•partially hydrogenated fatty acids (TRANS)
•interesterified (IE) palm oil or with palmitic acid occurring in sn-2 position

O = **Outcomes** TC, LDL-C , HDL-C TC/HDL-C ratio, LDL-C/HDL-C, TAG, Apo AI, Apo B, VLDL, Lp(a)

Inclusion Criteria:

- Control and intervention diets, and the exchange of the test fat, should be iso-energetic
- Estimate of mean values, and a corresponding measure of dispersion for the outcome
- Intervention duration over 2 weeks

Flow chart study selection



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Results Palm Oil (PO) meta-analysis (1)

B LDL-C (mg/dL): PO vs myristic/lauric acid

tudy		N, mean	N, mean	Study ID	WMD (95% CI)	N, mean (SD); Treatment	N, mean (SD); Control
)	WMD (95% CI)	(SD); Treatment	(SD); Control				
				Mattson 1985 (57)	24.00 (-2.66, 50.66)		20, 119 (35.8
				Bonanome 1988 (53)	- 21.27 (0.88, 41.66)		11, 119 (25.7
g 1991 (37) (-25.52 (-46.32, -4.73)	27, 97.4 (29.8)	27, 123 (46.4)	Denke 1992 (55)	► 23.98 (10.33, 37.62)		14, 128 (17)
enke 1992 (55)	8.89 (-6.60, 24.39)	14, 152 (19.7)	14, 143 (22)	Nestel 1992 (49)	10.00 (-3.87, 23.87)		27, 151 (26)
			-26/2226/2222222222222	Ng 1992 (38)	-1.00 (-14.59, 12.59)		33, 134 (31)
eber 1992 (56)	-14.00 (-34.83, 6.83)	13, 115 (25.2)	13, 129 (28.8)	Ng 1992 (38)	-2.00 (-15.77, 11.77)		33, 127 (30)
holstrup 1994 (63)	1.16 (-9.61, 11.93)	12, 86.6 (14.7)	12, 85.5 (12.1)	Zock 1994 (68)	14.69 (5.47, 23.92)		69, 101 (27.5
				Nestel 1994 (48)	2.90 (-7.14, 12.94)		68, 154 (23.7
holstrup 1994 (62)	-4.25 (-20.95, 12.44)	15, 114 (21)	15, 119 (25.5)	Choudhury 1995 (45)	-3.09 (-27.62, 21.43)		21, 132 (37.1
ock 1994 (68)	-4.25 (-13.94, 5.43)	69, 115 (27.8)	69, 119 (30.2)	Sundram 1995 (39)	4.64 (-3.14, 12.42)		23, 94.4 (1.9
				Temme 1996 (66)	8.51 (-9.02, 26.04)		32, 135 (36.3
undram 1994 (85)	-11.99 (-23.95, -0.03)	17, 92.4 (17.8)	17, 104 (17.8)	Noakes 1996 (52)	- 15.85 (0.11, 31.60)		23, 146 (22.4
chwab 1995 (75)	3.09 (-10.32, 16.50)	15, 113 (19.5)	15, 110 (18)	Sundram 1997 (41)	-0.77 (-15.53, 13.98)		27, 123 (27.1
				Cater 1997 (54)	25.14 (3.83, 46.44)	9, 169 (27.1)	9, 144 (18.2)
emme 1996 (66)	-5.03 (-23.04, 12.99)	32, 143 (35.2)	32, 148 (38.3)	Choudhury 1997 (46)	- 10.44 (-7.84, 28.72)	42, 143 (43.7)	42, 132 (41.8
undram 1997 (41)	-16.24 (-30.31, -2.18)	27, 122 (28.2)	27, 138 (24.4)	Cuesta 1998 (71)	29.78 (4.10, 55.45)	14, 167 (36.3)	14, 138 (32.9
				Montoya 2002 (72)	- 16.63 (-1.44, 34.69)	24, 152 (33.3)	24, 135 (30.5
nook 1999 (58)	8.89 (-10.52, 28.31)	16, 98.6 (30.9)	17, 89.7 (25.5)	Montoya 2002 (72)	- 16.63 (-3.04, 36.30)	17, 164 (32.9)	17, 148 (25.1
verall	-4.70 (-10.28, 0.87)			Vega-Lopez 2006 (59)	25.00 (3.81, 46.19)	15, 165 (35)	15, 140 (23)
Y				Mensink 2008 (65)	- 13.53 (-4.81, 31.87)	44, 148 (44.1)	44, 135 (43.7
				Overall 🔷	10.75 (6.60, 14.89)		
-46.3 0 4	16.3						
-40.5 0 4	0.0			-55.4 0	55.4		
Favors PO Fa	vors myristic/lauric a	acid		Favors PO	Favors MUFAs		

С

LDL-C (mg/dL): PO vs MUFAs

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Results Palm Oil (PO) meta-analysis (2)

LDL-C (mg/dL): PO vs PUFAs

D

Study ID	WMD (95% CI)	N, mean (SD); Treatment	N, mean (SD); Control
Mattson 1985 (57)	23.00 (-4.86, 50.86)	20, 143 (49.2)	20, 120 (40.2)
Marzuki 1991 (36)	-1.55 (-10.67, 7.58)	110, 91.6 (32.4)	110, 93.2 (36.5)
Ghafoorunissa 1995 (76)	-8.00 (-27.90, 11.90)	12, 71 (31.5)	12, 79 (15.6)
Ghafoorunissa 1995 (76)	4.00 (-14.22, 22.22)	12, 104 (20.8)	12, 100 (24.6)
Ghafoorunissa 1995 (76)	-1.00 (-17.27, 15.27)	12, 98 (17)	12, 99 (23.2)
Zhang 1997 (69) -	-7.15 (-16.19, 1.88)	30, 82.4 (20.5)	60, 89.5 (20.9)
Zhang 1997 (69)	-10.83 (-18.21, -3.44) 50, 146 (17.9)	52, 157 (20.1)
Muller 1998 (78)	11.21 (-3.26, 25.69)	27, 112 (29)	27, 101 (25.1)
Montoya 2002 (72)	- 22.62 (6.34, 38.90)	24, 152 (33.3)	48, 129 (33.1)
Montoya 2002 (72)	— 37.32 (19.79, 54.84)	17, 164 (32.9)	34, 127 (23.6)
Scholtz 2004 (82)	- 8.51 (-11.59, 28.61)	36, 135 (33.7)	20, 126 (38.4)
Pedersen 2005 (77)	11.21 (-3.26, 25.69)	27, 112 (29)	27, 101 (25.1)
Vega-Lopez 2006 (59)	20.00 (-1.48, 41.48)	15, 165 (35)	15, 145 (24)
Utarwuthipong 2009 (83)	12.60 (0.46, 24.74)	32, 169 (25.4)	32, 156 (24.1)
Overall	7.27 (-0.15, 14.70)		
-54.8 0	54.8		
Favors PO	Favors PUFAs		

E LDL-C (mg/dL): PO vs trans fatty acids

Study		N, mean	N, mean
ID	WMD (95% CI)	(SD); Treatment	(SD); Control
Heber 1992 (56)	4.00 (-14.07, 22.07)	13, 115 (25.2)	13, 111 (21.6)
Nestel 1992 (49)	-4.00 (-18.69, 10.69)	27, 161 (26)	27, 165 (29)
Nestel 1995 (50)	15.85 (-2.63, 34.34)	27, 171 (36.3)	27, 155 (32.9)
Noakes 1996 (52)	11.60 (-5.32, 28.52)	23, 162 (31.3)	23, 150 (27.1)
Sundram 1997 (41)	-25.52 (-43.11, -7.93)	27, 122 (28.2)	27, 147 (37.1)
Muller 1998 (78)	0.77 (-14.19, 15.74)	27, 112 (29)	27, 111 (27.1)
Mutalib 1999 (81)	 36.72 (17.82, 55.63)	15, 138 (32.9)	28, 102 (24.1)
Pedersen 2005 (77)	0.77 (-14.19, 15.74)	27, 112 (29)	27, 111 (27.1)
Vega-Lopez 2006 (59)	3.00 (-19.68, 25.68)	15, 165 (35)	15, 162 (28)
Sundram 2007 (42)	-8.51 (-19.99, 2.97)	30, 119 (20.9)	30, 128 (24.4)
Teng 2010 (44) 🗕	-11.21 (-15.88, -6.55)	82, 109 (12.6)	41, 120 (12.4)
Overall	1.07 (-7.51, 9.64)		
1	1		
-55.6 0	55.6		
Favors PO	Favors trans fatty acid	ls	

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Results Palm Oil (PO) meta-analysis (3)

B HDL-C (mg/dL): PO vs myristic/lauric acid

Study		N, mean	N, mean	Study ID
ID	WMD (95% CI)	(SD); Treatment	(SD); Control	Mattson 1985
Ng 1991 (37) (-	-10.44 (-16.22, -4.66)	27, 41.8 (10.4)	27, 52.2 (11.2)	Bonanome 19 Denke 1992 (5
Denke 1992 (55)	-2.32 (-8.34, 3.70)	14, 34.8 (8.12)	14, 37.1 (8.12)	Nestel 1992 (4 Ng 1992 (38)
Heber 1992 (56)	-1.00 (-13.55, 11.55)	13, 41 (18)	13, 42 (14.4)	Ng 1992 (38)
Tholstrup 1994 (63)	-3.48 (-9.40, 2.44)	12, 39.1 (6.7)	12, 42.5 (8.04)	Zock 1994 (68 Nestel 1994 (4
Tholstrup 1994 (62)	-7.73 (-13.65, -1.81)	15, 38.3 (7.49)	15, 46 (8.99)	Choudhury 19
Zock 1994 (68)	-5.03 (-9.55, -0.50)	69, 58.8 (12.8)	69, 63.8 (14.3)	Sundram 1995 Temme 1996
Sundram 1994 (85)	-3.87 (-9.75, 2.02)	17, 41.8 (7.73)	17, 45.6 (9.67)	Noakes 1996
Schwab 1995 (75)	- 1.16 (-6.90, 9.22)	15, 58.8 (12)	15, 57.6 (10.5)	Sundram 1997 Cater 1997 (54
Temme 1996 (66)	-4.64 (-12.71, 3.43)	32, 56.8 (15.5)	32, 61.5 (17.4)	Choudhury 19 Cuesta 1998 (
Sundram 1997 (41)	- 3.09 (-1.55, 7.74)	27, 48.7 (8.51)	27, 45.6 (8.89)	Montoya 2002
Snook 1999 (58)	-5.41 (-14.54, 3.71)	16, 50.3 (12.4)	17, 55.7 (14.3)	Montoya 2002 Sanchez-Muni
Overall	-3.70 (-6.26, -1.15)		949 946 989956	Vega-Lopez 2
				Mensink 2008 Overall
				e reidin
-16.2 0	16.2			
Favors myristic/lauric	Favors PO			
acid				Favo

HDL-C (mg/dL): PO vs MUFAs

Study ID	WMD (95% CI)	N, mean (SD); Treatment	N, mean (SD); Control
Mattson 1985 (57)	- 1.00 (-4.54, 6.54)	20, 39 (8.94)	20, 38 (8.94)
Bonanome 1988 (53)	-1.55 (-15.21, 12.12)		11, 43.7 (19.2)
Denke 1992 (55)		14, 34.8 (8.12)	14, 32.1 (6.96)
Nestel 1992 (49)	4.00 (0.80, 7.20)	27, 42 (6)	27, 38 (6)
Ng 1992 (38)	0.00 (-3.63, 3.63)	33, 37 (8)	33, 37 (7)
Ng 1992 (38)	0.00 (-3.63, 3.63)	33, 46 (7)	33, 46 (8)
Zock 1994 (68)	0.77 (-3.30, 4.84)	69, 58.8 (12.8)	69, 58 (11.6)
Nestel 1994 (48)	1.16 (-2.63, 4.95)	34, 44.1 (9.28)	68, 42.9 (9.02)
Choudhury 1995 (45)	4.25 (-2.04, 10.55)	21, 35.2 (12.8)	21, 30.9 (7.35)
Sundram 1995 (39)	► 0.00 (-6.60, 6.60)	23, 47.6 (10.8)	23, 47.6 (12)
Temme 1996 (66)	1.16 (-6.23, 8.55)	32, 56.8 (15.5)	32, 55.7 (14.7)
Noakes 1996 (52)	1.16 (-6.10, 8.42)	23, 48.3 (12.8)	23, 47.2 (12.4)
Sundram 1997 (41)	0.39 (-3.85, 4.63)	27, 48.7 (8.51)	27, 48.3 (7.35)
Cater 1997 (54)	-0.77 (-8.49, 6.94)	9, 35.2 (6.19)	9, 36 (10.1)
Choudhury 1997 (46)	2.32 (-3.80, 8.44)	42, 47.6 (14.7)	42, 45.2 (13.9)
Cuesta 1998 (71)	11.21 (-0.62, 23.05)		14, 75.4 (17.8)
Montoya 2002 (72)	■ 0.39 (-6.67, 7.44)	24, 46 (13.9)	24, 45.6 (10.8)
Montoya 2002 (72)	- 0.00 (-7.54, 7.54)	17, 57.6 (11.2)	17, 57.6 (11.2)
Sanchez-Muniz 2002 (73)	— 11.21 (-0.62, 23.05)	,	14, 75.4 (17.8)
Vega-Lopez 2006 (59)	2.00 (-3.73, 7.73)	15, 50 (8)	15, 48 (8)
Mensink 2008 (65)	2.71 (-3.93, 9.34)	44, 62.6 (16.6)	44, 59.9 (15.1)
Overall	1.54 (0.38, 2.71)		
-23 0	23		
Favors MUFAs	Favors PO		

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С

Results Palm Oil (PO) meta-analysis (4)

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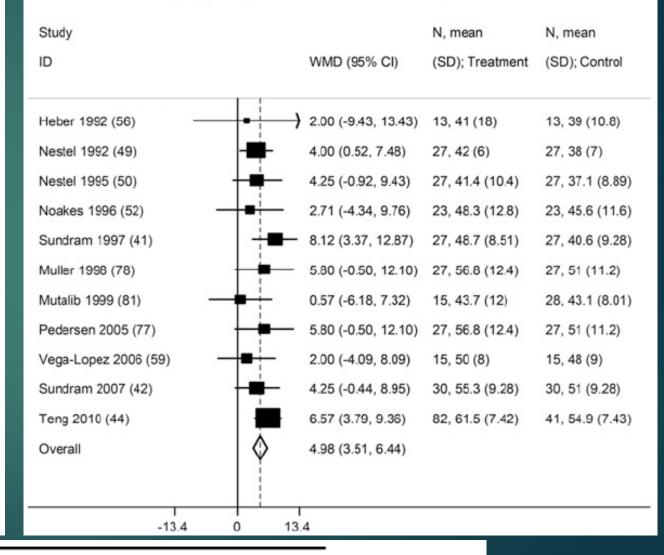
HDL-C (mg/dL): PO vs PUFAs

D

Study ID	WMD (95% CI)	N, mean (SD); Treatment	N, mean (SD); Control
Mattson 1985 (57)	4.00 (-1.54, 9.54)	20, 39 (8.94)	20, 35 (8.94)
Marzuki 1991 (36)	0.39 (-2.83, 3.60)	110, 51.4 (12.2)	110, 51 (12.2)
Ghafoorunissa 1995 (76)	- 2.00 (-7.74, 11.74)	12, 36 (13.2)	12, 34 (11.1)
Ghafoorunissa 1995 (76) -	-3.00 (-9.52, 3.52)	12, 30 (7.97)	12, 33 (8.31)
Ghafoorunissa 1995 (76)	3.00 (-1.44, 7.44)	12, 32 (5.2)	12, 29 (5.89)
Zhang 1997 (69)	5.41 (2.09, 8.74)	30, 38.7 (7.73)	60, 33.3 (7.26)
Zhang 1997 (69)	1.16 (-2.55, 4.87)	50, 49.9 (8.72)	52, 48.7 (10.4)
Muller 1998 (78)	1.55 (-4.66, 7.75)	27, 56.8 (12.4)	27. 55.3 (10.8)
French 2002 (35)	7.73 (-6.57, 22.03)	6, 58 (15.2)	6, 50.3 (9.47)
Montoya 2002 (72)	-0.00 (-6.29, 6.29)	24, 46 (13.9)	48, 46 (10.3)
Montoya 2002 (72)	3.09 (-3.40, 9.59)	17, 57.6 (11.2)	34, 54.5 (11)
Zhang 2003 (70)	-1.16 (-5.06, 2.74)	20, 45.6 (6.96)	22, 46.8 (5.8)
Scholtz 2004 (82)	- 1.16 (-10.59, 12.91)	36, 38.7 (14)	20, 37.5 (24.7)
Pedersen 2005 (77)	1.55 (-4.66, 7.75)	27, 56.8 (12.4)	27, 55.3 (10.8)
Vega-Lopez 2006 (59)	1.00 (-5.09, 7.09)	15, 50 (8)	15, 49 (9)
Utarwuthipong 2009 (83)	4.15 (-1.89, 10.19)	32, 58.4 (12.7)	32, 54.3 (11.9)
Overall	1.82 (0.54, 3.10)		
-22 0	22		
Favors PUFAs	Favors PO		

HDL-C (mg/dL): PO vs trans fatty acids

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Conclusions

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Palm oil and blood lipid–related markers of cardiovascular disease: a systematic review and meta-analysis of dietary intervention trials^{1–3}

Elena Fattore, Cristina Bosetti, Furio Brighenti, Carlo Agostoni, and Giovanni Fattore

ABSTRACT

Background: Palm oil (PO) may be an unhealthy fat because of its high saturated fatty acid content.

Objective: The objective was to assess the effect of substituting PO for other primary dietary fats on blood lipid-related markers of coronary heart disease (CHD) and cardiovascular disease (CVD). Design: We performed a systematic review and meta-analysis of dietary intervention trials. Studies were eligible if they included original data comparing PO-rich diets with other fat-rich diets and analyzed at least one of the following CHD/CVD biomarkers: total cholesterol (TC), low-density lipoprotein (LDL) cholesterol, highdensity lipoprotein (HDL) cholesterol, TC/HDL cholesterol, LDL cholesterol/HDL cholesterol, triacylglycerols, apolipoprotein A-I and B, very-low-density lipoprotein cholesterol, and lipoprotein(a). Results: Fifty-one studies were included. Intervention times ranged from 2 to 16 wk, and different fat substitutions ranged from 4% to 43%. Comparison of PO diets with diets rich in stearic acid, monounsaturated fatty acids (MUFAs), and polyunsaturated fatty acids (PUFAs) showed significantly higher TC, LDL cholesterol, apolipoprotein B, HDL cholesterol, and apolipoprotein A-I, whereas most of the same biomarkers were significantly lower when compared with diets rich in myristic/lauric acid. Comparison of PO-rich diets with diets rich in trans fatty acids showed significantly higher concentrations of HDL cholesterol and apolipoprotein A-I and significantly lower apolipoprotein B, triacylglycerols, and TC/HDL cholesterol. Stratified and meta-regression analyses showed that the higher concentrations of TC and LDL cholesterol, when PO was substituted for MUFAs and PUFAs, were not significant in young people and in subjects with diets with a lower percentage of energy from fat.

Conclusions: Both favorable and unfavorable changes in CHD/ CVD risk markers occurred when PO was substituted for the primary dietary fats, whereas only favorable changes occurred when PO was substituted for *trans* fatty acids. Additional studies are needed to provide guidance for policymaking. *Am J Clin Nutr* 2014;99:1331–50. morbidity and mortality (5). These recommendations were put forward because dietary SFAs increase blood total cholesterol (TC) and LDL cholesterol, which are known risk factors for CHD and CVD (6). However, not all studies have supported the relation between SFAs and CHD or CVD (7–11), and research on individual dietary fats has shown that different SFAs can exert different effects on cholesterolemia (12) and not only the type of fatty acid, but also the triacylglycerol structure, plays a role (13). In addition, conflicting results have recently emerged regarding the benefit of substituting SFAs with PUFAs on major cardiovascular outcomes (14–16).

Overall, during the past several years, a more complex picture concerning the risk factors for CVD has been developed. In addition to the major traditional serum/plasma markers of CHD risk (ie, TC, LDL cholesterol, HDL cholesterol, and triacylglycerols), other lipid-related biomarkers, such as apolipoprotein A-I and -B, which are the main protein components of HDL cholesterol and LDL cholesterol, respectively, and lipoprotein(a), have been suggested to be valid, if not better, risk predictors (17–20).

Palm oil (PO), a vegetable oil obtained from the fruit of the palm tree (*Elaeis guineensis*), is composed of \sim 50% palmitic acid, 40% oleic acid, and 10% linoleic acid. Palmitic acid, in addition to being the most abundant constituent of PO, is the main SFA that naturally occurs in animal and vegetable fats and is the main component of human milk fats (21). Over the past few years, PO use has significantly increased, despite debates over whether it is a potential unhealthy fat because of its relatively high palmitic

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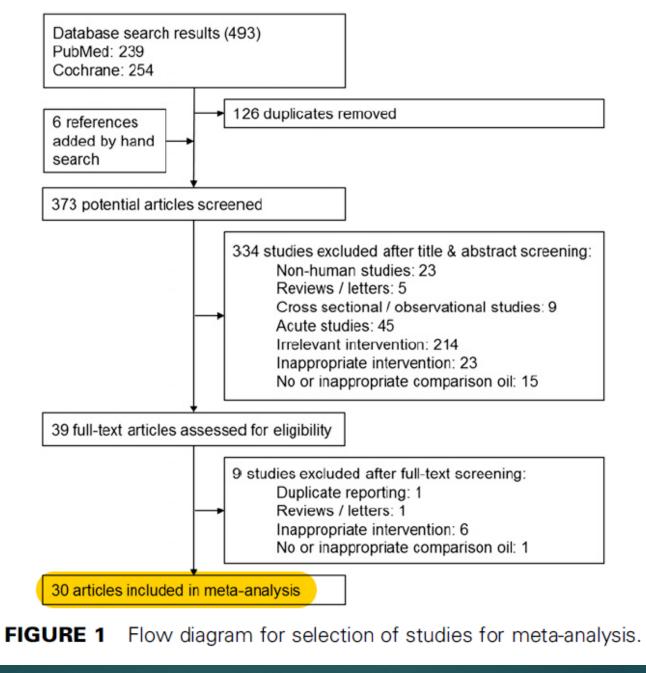
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Palm Oil Consumption Increases LDL Cholesterol Compared with Vegetable Oils Low in Saturated Fat in a Meta-Analysis of Clinical Trials¹⁻³

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Sun et al., Journal of Nutrition 2015

Results (Sun et al., Journal of Nutrition 2015) (1)

LDL-C PO vs vegetable oils

HDL-C PO vs vegetable oils

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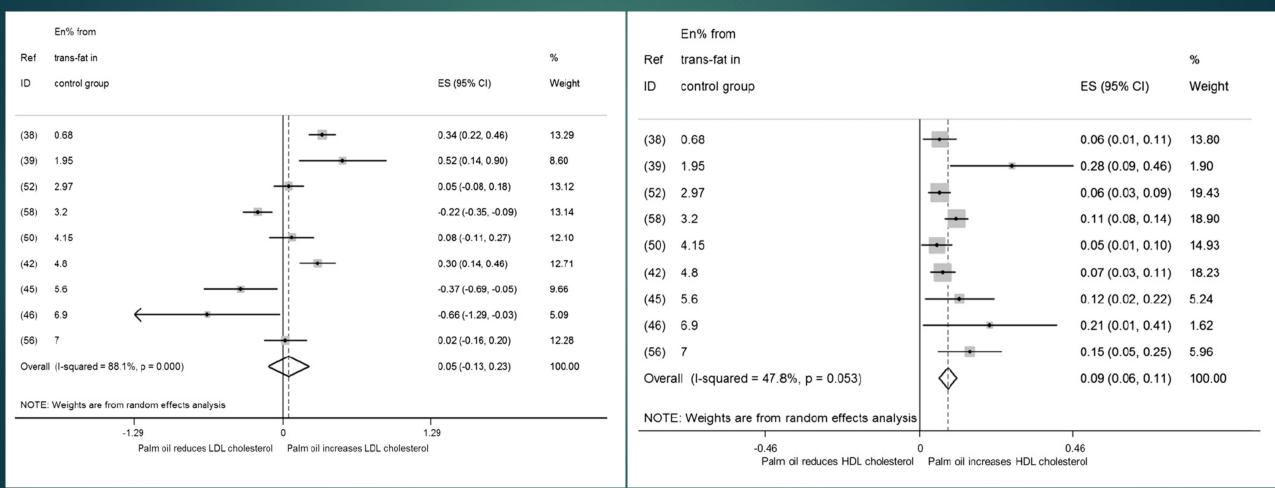
Ref ID	Control oil	En% from test oil		ES (95% CI)	% Weight	Ref ID	Control oil	En% from test oil		ES (95% CI)	% Weight
(35)	peanut	17.5	+	-0.21 (-0.57, 0.16)	3.38	(35)	peanut	17.5		0.05 (-0.07, 0.17)	1.66
(53)	peanut	18-19.5		-0.28 (-0.40, -0.16)	5.09	(53)	peanut	18-19.5		0.03 (-0.02, 0.08)	5.92
(35)	peanut	20 -		0.04 (-0.16, 0.23)	4.63	(35)	peanut	20		0.00 (-0.05, 0.05)	5.71
(32)	olive	17		-0.08 (-0.47, 0.31)	3.19	(32)	olive	17		0.11 (0.02, 0.20)	2.54
(47)	olive	17		0.22 (0.12, 0.32)	5.19	(47)	olive	17	+	0.02 (-0.01, 0.05)	8.55
(51)	olive	20	+	0.14 (-0.03, 0.31)	4.78	(51)	olive	20		0.03 (-0.02, 0.08)	5.74
(41)	olive	23 —	• •	-0.04 (-0.25, 0.18)	4.46	(41)	olive	23		0.00 (-0.05, 0.05)	5.48
(57)	canola	20	+ • · ·	0.12 (-0.07, 0.31)	4.67	(57)	canola	20		0.00 (-0.08, 0.08)	3.12
(48)	canola	NR –	- B ¹	0.17 (-0.12, 0.46)	3.91	(48)	canola	NR		0.12 (0.01, 0.23)	1.86
(48)	canola	NR		0.26 (0.02, 0.50)	4.27		canola	NR		0.00 (-0.07, 0.07)	3.50
(36)	soybean	NR —	• · · ·	-0.04 (-0.19, 0.11)	4.94		soybean	NR		0.01 (-0.03, 0.05)	
(44)	linoleic sunflower	12		0.53 (0.19, 0.87)	3.58		linoleic sunflower	18-20		0.06 (0.01, 0.11)	5.30
(52)	linoleic sunflower	18-20	- <u>-</u>	0.18 (-0.06, 0.42)	4.29	(42)	oleic sunflower	20	+	0.03 (0.01, 0.05)	9.81
(42)	oleic sunflower	20	+	0.41 (0.20, 0.62)	4.50	(55)	oleic sunflower	28.4	-	0.29 (0.03, 0.55)	0.40
(55)	oleic sunflower	28.4		0.77 (0.41, 1.13)	3.44	(43)	oleic sunflower	28.5		0.29 (0.03, 0.55)	0.40
(34)	oleic sunflower	40		0.62 (0.18, 1.06)	2.88	(34)	oleic sunflower	40	· • ·	0.07 (0.00, 0.14)	3.84
(30)	oleic sunflower	43		0.72 (0.34, 1.10)	3.28	(30)	oleic sunflower	43		-0.03 (-0.13, 0.07)	
(31)	oleic sunflower	43		0.65 (0.40, 0.90)	4.20	(31)	oleic sunflower	43		-0.02 (-0.13, 0.09)	
(33)	oleic sunflower	NR		0.27 (0.01, 0.53)	4.14	(33)	oleic sunflower	NR	+	0.06 (-0.01, 0.13)	
(40)	corn	22.5 -	* * *	0.65 (-0.12, 1.42)	1.47		corn	22.5		0.08 (-0.15, 0.31)	
(54)	oleic safflower	40		0.55 (0.25, 0.85)	3.85		oleic safflower	40	•	-0.04 (-0.23, 0.15)	
(37)	oleic & linoleic safflower	r 40		0.61 (0.04, 1.18)	2.20	. ,	oleic & linoleic safflowe				
(50)	soybean & canola	20		0.58 (0.05, 1.11)	2.38		soybean & canola	20		0.04 (-0.03, 0.11)	
(49)	soybean & rice bran	20		0.74 (0.06, 1.42)	1.75		soybean & rice bran	20		0.13 (-0.30, 0.56)	
(46)	canola+sunflower	21.2	•	-0.02 (-0.25, 0.21)	4.34		canola+sunflower	21.2			
(29)	soybean+sunflower	NR -	•	-0.05 (-0.15, 0.06)	5.17		soybean+sunflower	NR		-0.06 (-0.10, -0.03)	
Overa	all (I-squared = 83.2%, p	= 0.000)	\Diamond	0.24 (0.13, 0.34)	100.00		all (I-squared = 49.8%, p		6	0.02 (0.01, 0.04)	100.00
NOTE	: Weights are from rand	om effects analysis					E: Weights are from rand			0.02 (0.01, 0.04)	100.00
		-1.42	0 1.4	2				1			
		Palm oil reduces LDL cholesterol	Palm oil increases LDL cholesterol					-0.562	0 0.5	62	
								Palm oil reduces HDL cho	lesterol Palm oil increases HDL cholesterol		

Results (Sun et al., Journal of Nutrition 2015) (1)



LDL-C PO vs partially hydrogenated vegetable oils

HDL-C PO vs partially hydrogenated vegetable oils



Conclusions

Palm Oil Consumption Increases LDL Cholesterol Compared with Vegetable Oils Low in Saturated Fat in a Meta-Analysis of Clinical Trials^{1–3}

Ye Sun,^{4,7} Nithya Neelakantan,⁴ Yi Wu,⁴ Rashmi Lote-Oke,⁴ An Pan,⁴ and Rob M van Dam^{4–7}*

⁴Saw Swee Hock School of Public Health and ⁵Department of Medicine, Yong Loo Lin School of Medicine, National University of Singapore and National University Health System, Singapore; ⁶Department of Nutrition, Harvard School of Public Health, Boston, MA; and ⁷NUS Graduate School for Integrative Sciences and Engineering, National University of Singapore, Singapore

Abstract

Background: Palm oil contains a high amount of saturated fat compared with most other vegetable oils, but studies have reported inconsistent effects of palm oil on blood lipids.

Objective: We systematically reviewed the effect of palm oil consumption on blood lipids compared with other cooking oils using data from clinical trials.

Methods: We searched PubMed and the Cochrane Library for trials of at least 2 wk duration that compared the effects of palm oil consumption with any of the predefined comparison oils: vegetable oils low in saturated fat, *trans* fat–containing partially hydrogenated vegetable oils, and animal fats. Data were pooled by using random-effects meta-analysis.

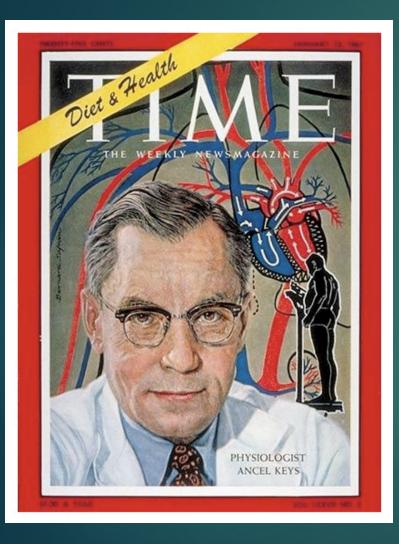
Results: Palm oil significantly increased LDL cholesterol by 0.24 mmol/L (95% CI: 0.13, 0.35 mmol/L; $l^2 = 83.2\%$) compared with vegetable oils low in saturated fat. This effect was observed in randomized trials (0.31 mmol/L; 95% CI: 0.20, 0.42 mmol/L) but not in nonrandomized trials (0.03 mmol/L; 95% CI: -0.15, 0.20 mmol/L; *P*-difference = 0.02). Among randomized trials, only modest heterogeneity in study results remained after considering the test oil dose and the comparison oil type ($l^2 = 27.5\%$). Palm oil increased HDL cholesterol by 0.02 mmol/L (95% CI: 0.01, 0.04 mmol/L; $l^2 = 49.8\%$) compared with vegetable oils low in saturated fat and by 0.09 mmol/L (95% CI: 0.06, 0.11 mmol/L; $l^2 = 47.8\%$) compared with *trans* fat–containing oils.

Conclusions: Palm oil consumption results in higher LDL cholesterol than do vegetable oils low in saturated fat and higher (HDL cholesterol than do *trans* fat–containing oils in humans.) The effects of palm oil on blood lipids are as expected on the basis of its high saturated fat content, which supports the reduction in palm oil use by replacement with vegetable oils low in saturated and *trans* fat. This systematic review was registered with the PROSPERO registry at http://www.crd.york. ac.uk/PROSPERO/display_record.asp?ID=CRD42012002601#.VU3wvSGeDRZ as CRD42012002601. *J Nutr* 2015;145:1549–58.

JN THE JOURNAL OF NUTRITION

3. Scientific evidence on saturated fatty acids and cardiovascular diseases – old and new studies

Ancel Keys The Seven Countries Study



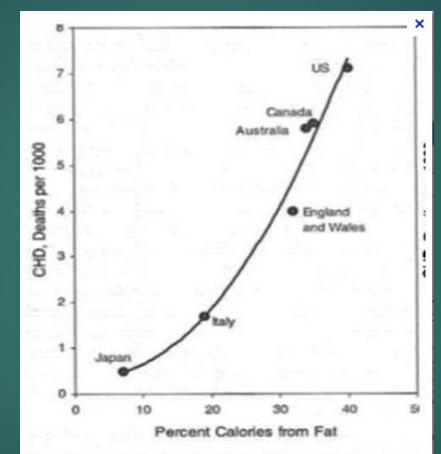
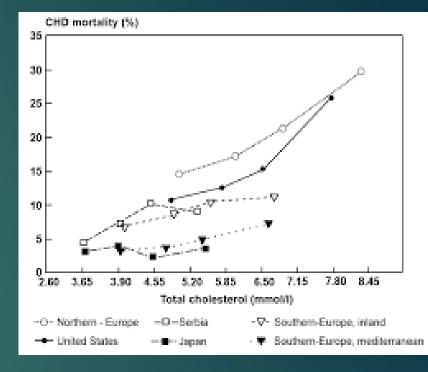


Figure 1A. Correlation between the total fat consumption as a percent of total calorie consumption, and mortality from coronary heart disease in six countries. Data from Keys.¹



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The Lipid Theory

Downloaded from http://openheart.bmj.com/ on October 25, 2017 - Published by group.bmj.com

Meta-analysis

openheart Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977

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Dietary Intervention & Heart Deaths

Study name				Heart Deat	hs / Total		Risk ratio	and 9	6% CI	
	Risk ratio	Lower limit	Upper limit	Interventio	n Control					
Rose Com Oil (1965)	4.643	0.580	37,149	5/28	1/26		- 1			·
Rose Olive Oil (1965)	3.000	0.333	26.992	3/26	1/28		<u> </u>	-	<u> </u>	
Research Committee Low-Fat (1965)	0.891	0.490	1.620	17/123	20 / 129			-		
MRC Soybean OI (1968)	1.053	0.634	1.748	27 / 199	25 / 194			•		
LA Veterans Dayton (1969)	0.816	0.552	1.206	41/424	50 / 422			-		
Leren, Oslo heart study (1970)	0.840	0.669	1.056	79/206	94 / 206					
Woodhill, Sydney heart study (1978)	1.501	0.930	2.425	35/221	25 / 237					
	0.989	0.784	1.247					<u>+</u>		
						0.01	0.1	1	10	100
						Fav	ours Intervention	,	lav our i Contro	

Meta Analysis Random Effects Method

Figure 3 Estimates of CHD mortality (95% CIs) from meta-analysis.

University of the Wast of Scotland, Hamilton, Lanarkshire, UK ²Cardiff School of Sport, Cardiff Metropolitan University, Cardiff, UK ³University of South Wales, were untested in any trial prior to being introduced. **Conclusions:** Dietary recommendations were introduced for 220 million US and 56 million UK citizens by 1983, in the absence of supporting evidence from RCTs. sumption to 30% of total energy intake and (2) reduce saturated fat consumption to 10% of total energy intake.

The recommendations were an attempt to address the incidence of coronary heart disease (CHD). Both documents acknowle

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Re-evaluation of the traditional diet-heart hypothesis: analysis of recovered data from Minnesota Coronary Experiment (1968-73)

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ABSTRACT

OBJECTIVE

To examine the traditional diet-heart hypothesis through recovery and analysis of previously unpublished data from the Minnesota Coronary Experiment (MCE) and to put findings in the context of existing diet-heart randomized controlled trials through a systematic review and meta-analysis.

DESIGN

The MCE (1968-73) is a double blind randomized controlled trial designed to test whether replacement of saturated fat with vegetable oil rich in linoleic acid reduces coronary heart disease and death by lowering serum cholesterol. Recovered MCE unpublished documents and raw data were analyzed according to hypotheses prespecified by original investigators. Further, a systematic review and meta-analyses of randomized controlled trials that lowered serum cholesterol by providing vegetable oil rich in linoleic acid in place of saturated fat without confounding by concomitant interventions was conducted.

SETTING

One nursing home and six state mental hospitals in Minnesota, United States.

PARTICIPANTS

Unpublished documents with completed analyses for the randomized cohort of 9423 women and men aged 20-97; longitudinal data on serum cholesterol for the 2355 participants exposed to the study diets for a year or more; 149 completed autopsy files.

INTERVENTIONS

Serum cholesterol lowering diet that replaced saturated fat with linoleic acid (from corn oil and corn oil polyunsaturated margarine). Control diet was high in saturated fat from animal fats, common margarines, and shortenings.

MAIN OUTCOME MEASURES

Death from all causes; association between changes in serum cholesterol and death; and coronary atherosclerosis and myocardial infarcts detected at autopsy.

RESULTS

The intervention group had significant reduction in serum cholesterol compared with controls (mean change from baseline -13.8% v -1.0%; P<0.001). Kaplan Meier graphs showed no mortality benefit for the intervention group in the full randomized cohort or for any prespecified subgroup. There was a 22% higher risk of death for each 30 mg/dL (0.78 mmol/L) reduction in serum cholesterol in covariate adjusted Cox regression models (hazard ratio 1.22, 95% confidence interval 1.14 to 1.32; P<0.001). There was no evidence of benefit in the intervention group for coronary atherosclerosis or myocardial infarcts. Systematic review identified five randomized controlled trials for inclusion (n=10808). In metaanalyses, these cholesterol lowering interventions showed no evidence of benefit on mortality from coronary heart disease (1.13, 0.83 to 1.54) or all cause mortality (1.07, 0.90 to 1.27).

CONCLUSIONS

Available evidence from randomized controlled trials shows that replacement of saturated fat in the diet with linoleic acid effectively lowers serum cholesterol but does not support the hypothesis that this translates to a lower risk of death from coronary heart disease or all causes. Findings from the Minnesota

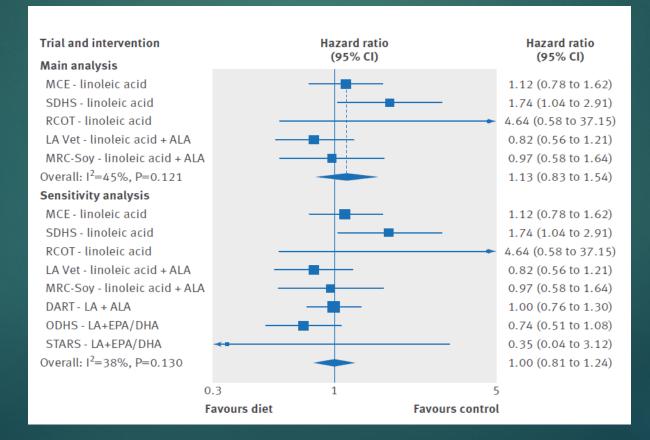
RESEARCH

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Re-evaluation of the traditional diet-heart hypothesis: analysis of recovered data from Minnesota Coronary Experiment (1968-73)

Christopher E Ramsden,^{1,2} Daisy Zamora,³ Sharon Majchrzak-Hong,¹ Keturah R Faurot,² Steven K Broste,⁴ Robert P Frantz,⁵ John M Davis,^{3,6} Amit Ringel,¹ Chirayath M Suchindran,⁷ Joseph R Hibbeln¹



Associations of fats and carbohydrate intake with

cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study

Interpretation High carbohydrate intake was associated with higher risk of total mortality, whereas total fat and individual types of fat were related to lower total mortality. Total fat and types of fat were not associated with cardiovascular disease, myocardial infarction, or cardiovascular disease mortality, whereas saturated fat had an inverse association with stroke. Global dietary guidelines should be reconsidered in light of these findings.

Association of dietary nutrients with blood lipids and blood pressure in 18 countries: a cross-sectional analysis from the PURE study

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Interpretation Our data are at odds with current recommendations to reduce total fat and saturated fats. Reducing saturated fatty acid intake and replacing it with carbohydrate has an adverse effect on blood lipids. Substituting saturated fatty acids with unsaturated fats might improve some risk markers, but might worsen others. Simulations suggest that ApoB-to-ApoA1 ratio probably provides the best overall indication of the effect of saturated fatty acids on cardiovascular disease risk among the markers tested. Focusing on a single lipid marker such as LDL cholesterol alone does not capture the net clinical effects of nutrients on cardiovascular risk.

Conclusions

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There is not a scientific evidence on advantage/disadvantage from substitution of palm oil by other main dietary fats (trans fatty acids excluded)

A more complex picture has emerged about the lipid biomarkers related to cardiovascular diseases: total serum cholesterol or LDLcholesterol seem to be not valid predictors.

The soundness of the scientific evidence underpinning current and previous dietary advice on dietary fat intake for total fats and SFA has been questioned (no causal relation)

Thank you for your attention !

Review

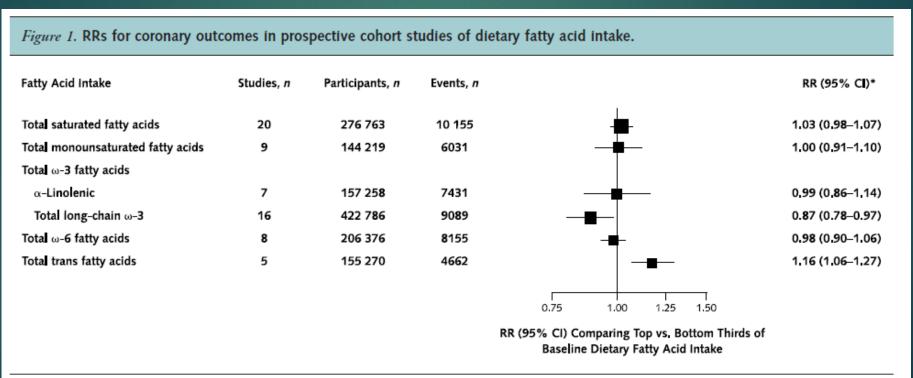
Annals of Internal Medicine

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Association of Dietary, Circulating, and Supplement Fatty Acids With Coronary Risk

A Systematic Review and Meta-analysis

Rajiv Chowdhury, MD, PhD; Samantha Warnakula, MPhil*; Setor Kunutsor, MD, MSt*; Francesca Crowe, PhD; Heather A. Ward, PhD; Laura Johnson, PhD; Oscar H. Franco, MD, PhD; Adam S. Butterworth, PhD; Nita G. Forouhi, MRCP, PhD; Simon G. Thompson, FMedSci; Kay-Tee Khaw, FMedSci; Dariush Mozaffarian, MD, DrPH; John Danesh, FRCP*; and Emanuele Di Angelantonio, MD, PhD*



Size of the data marker is proportional to the inverse of the variance of the RR. RR = relative risk.

* Pooled estimate based on random-effects meta-analysis. Corresponding forest plots, I^2 estimates, and pooled RRs based on fixed-effects meta-analysis are provided in **Supplement 1**, available at www.annals.org.

RESEARCH





Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies

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org/10.1136/bm(.h3978)

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ABSTRACT OBJECTIVE

To systematically review associations between intake of saturated fat and trans unsaturated fat and all cause mortality, cardiovascular disease (CVD) and associated mortality, coronary heart disease (CHD) and associated mortality, ischemic stroke, and type 2 diabetes.

DESIGN

Systematic review and meta-analysis.

DATA SOURCES

Medline, Embase, Cochrane Central Registry of Controlled Trials, Evidence-Based Medicine Reviews, and CINAHL from Inception to 1 M ay 2015, supplemented by bibliographies of retrieved articles and previous reviews.

ELIGIBILITY CRITERIA FOR SELECTING STUDIES

Observational studies reporting associations of saturated fat and/or trans unsaturated fat (total, industrially manufactured, or from ruminant animals) with all cause mortality, CHD/CVD mortality, total CHD, ischemic stroke, or type 2 diabetes.

DATA EXTRACTION AND SYNTHESIS

Two reviewers independently extracted data and assessed study risks of bias. Multivariable relative risks were pooled. Heterogeneity was assessed and quantified. Potential publication bias was assessed and subgroup analyses were undertaken. The GRADE approach was used to evaluate quality of evidence and certainty of conclusions.

dol: 10.1136/bmj.h3978 Accepted: 15 July 2015

WHAT IS ALREADY KNOWN ON THIS TOPIC

Contrary to prevailing dietary advice, authors of a recent systematic review and meta-analyses claim that there is no excess cardiovascular risk associated with intake of saturated fat, and the US has recently taken policy action to remove partially hydrogenated vegetable oils from its food supply

RESULTS

For saturated fat, three to 12 prospective cohort studies for each association were pooled (five to 17 comparisons with 90501-339090 participants). Saturated fat intake was not associated with all cause mortality (relative risk 0.99, 95% confidence interval 0.91 to 1.09), CVD mortality (0.97, 0.84 to 1.12), total CHD (1.06, 0.95 to 1.17), ischemic stroke (1.02, 0.90 to 1.15), or type 2 diabetes (0.95, 0.88 to 1.03). There was no convincing lack of association between saturated fat and CHD mortality (1.15, 0.97 to 1.36; P=0.10). For trans fats, one to six prospective cohort studies for each association were pooled (two to seven comparisons with 12 94 2-230 135 participants). Total trans fat intake was associated with all cause mortality (1.34, 1.16 to 1.56), CHD mortality (1.28, 1.09 to 1.50), and total CHD (1.21, 1.10 to 1.33) but not ischemic stroke (1.07, 0.88 to 1.28) or type 2 diabetes (1.10, 0.95 to 1.27). Industrial, but not ruminant, trans fats were associated with CHD mortality (1.18 (1.04 to 1.33) v 1.01 (0.71 to 1.43)) and CHD (1.42 (1.05 to 1.92) v 0.93 (0.73 to 1.18)). Ruminant trans-palmitoleic acid was inversely associated with type 2 diabetes (0.58, 0.46 to 0.74). The certainty of associations between saturated fat and all outcomes was "very low." The certainty of associations of trans fat with CHD outcomes was "moderate" and "very low" to "low" for other associations.

CONCLUSIONS

Saturated fats are not associated with all cause mortality, CVD, CHD, ischemic stroke, or type 2 diabetes, but the evidence is heterogeneous with methodological limitations. Trans fats are associated with all cause mortality, total CHD, and CHD mortality, probably because of higher levels of intake of industrial trans fats than ruminant trans fats. Dietary guidelines must carefully consider the health effects of recommendations for alternative macronutrients to replace trans fats and saturated fats. No associations between saturated fatty acids and cardiovascular outocomes Positive association with partially hydrogenated fatty acids

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