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Dietary Cholesterol and Cardiovascular Risk: A Science Advisory From the American Heart Association

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Abstract

The elimination of specific dietary cholesterol target recommendations in recent guidelines has raised questions about its role with respect to cardiovascular disease. This advisory was developed after a review of human studies on the relationship of dietary cholesterol with blood lipids, lipoproteins, and cardiovascular disease risk to address questions about the relevance of dietary cholesterol guidance for heart health. Evidence from observational studies conducted in several countries generally does not indicate a significant association with cardiovascular disease risk. Although meta-analyses of intervention studies differ in their findings, most associate intakes of cholesterol that exceed current average levels with elevated total or low-density lipoprotein cholesterol concentrations. Dietary guidance should focus on healthy dietary patterns (eg, Mediterranean-style and DASH [Dietary Approaches to Stop Hypertension]–style diets) that are inherently relatively low in cholesterol with typical levels similar to the current US intake. These patterns emphasize fruits, vegetables, whole grains, low-fat or fat-free dairy products, lean protein sources, nuts, seeds, and liquid vegetable oils. A recommendation that gives a specific dietary cholesterol target within the context of food-based advice is challenging for clinicians and consumers to implement; hence, guidance focused on dietary patterns is more likely to improve diet quality and to promote cardiovascular health.

Historically, nutrition guidelines for reducing cardiovascular disease (CVD) risk and achieving optimal plasma lipoprotein profiles have included recommendations to limit dietary cholesterol.[1,2] However, contemporary guidelines for CVD risk reduction from the American Heart Association (AHA) and American College of Cardiology (ACC)[3,4] and the "2015–2020 Dietary Guidelines for Americans" (DGA)[5] have not issued explicit guidance for dietary cholesterol. Because of the inconsistencies in the evidence base and the inherent difficulty in conducting and interpreting studies to isolate the independent effect of dietary cholesterol on CVD risk, controversy has ensued about whether dietary cholesterol should be a target for CVD prevention and management. This science advisory has 2 aims: to review the key human studies to date that have assessed the relationship of dietary cholesterol with plasma and lipoprotein cholesterol concentrations and CVD risk, and to explore methodological issues that contribute to the continued controversy on this topic, including questions on the impact of egg intake.

Current Dietary Cholesterol Recommendations

The "2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk" did not include a recommendation for dietary cholesterol and concluded, "There is insufficient evidence to determine whether lowering dietary cholesterol reduces LDL-C [low-density lipoprotein cholesterol]."[3] Similarly, the 2015 Dietary Guidelines Advisory Committee did not recommend limiting dietary cholesterol to <300 mg/d as presented in prior editions of the DGA.[6] The 2015 Dietary Guidelines Advisory Committee, with a focus on dietary patterns, provided nuanced statements related to cholesterol intake:

> Previously, the Dietary Guidelines for Americans recommended that cholesterol intake be limited to no more than 300 milligrams per day. The 2015 DGAC [Dietary Guidelines Advisory Committee] will not bring forward this recommendation because available evidence shows no appreciable relationship between consumption of dietary cholesterol and serum cholesterol, consistent with the conclusions of the AHA/ACC report. Cholesterol is not a nutrient of concern for overconsumption.[7]

At the time of the 2015 Dietary Guidelines Advisory Committee, the mean dietary intake in the United States for those ≥1 year of age was <300 mg/d. Because this level did not meet the criterion of a nutrient of concern, the importance of a numerical limitation was diminished. However, the 2015 DGA report stated that the lack of an explicit limit for dietary cholesterol did not imply that dietary cholesterol is unimportant in the adoption of healthy eating patterns. The report also emphasized that adherence to US Department of Agriculture–recommended eating patterns will naturally contribute to low amounts of dietary cholesterol because dietary cholesterol commonly coexists with major food sources of saturated fat. All of the US Department of Agriculture–recommended eating patterns included in the 2015 to 2020 report have <300 mg/d cholesterol across calorie levels ≤3200 kcal/d.[8] Similarly, the multisociety 2018 Guideline on the Management of Blood Cholesterol[4] brought forward the "2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk,"[3] which did not include a recommendation for dietary cholesterol but recommended healthy eating patterns involving relatively low levels of dietary cholesterol.

Two recent guidelines, one for primary prevention and one for secondary prevention, have addressed the issue of dietary cholesterol. The "2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease" cited studies of the benefit of plant-based rather than animal-based protein and concluded, "A diet containing reduced amounts of cholesterol and sodium can be beneficial to decrease atherosclerotic CVD risk" without further distinction.[9] The 2015 "National Lipid Association Recommendations for Patient-Centered Management of Dyslipidemia," focused on individuals with established hypercholesterolemia, recommend limiting dietary cholesterol to <200 mg/d to lower LDL cholesterol and non–high-density lipoprotein (HDL) cholesterol concentrations.[10] The National Lipid Association Expert Panel concluded that "dietary cholesterol modestly increases total cholesterol, and LDL-cholesterol on average, although hypo- and hyper-responders do exist in the population."[10]

Current Cholesterol Intake in the United States

According to the 2013 to 2014 NHANES (National Health and Nutrition Examination Surveys), the mean cholesterol intake in US adults ≥20 years of age was 293 mg/d (95% CI, 284–302), corresponding to a cholesterol density of 137 mg/1000 kcal. However, there are racial/ethnic differences. Mexican Americans have the highest intake with a mean of 338 mg/d (153 mg/1000 kcal), whereas the intake for non-Hispanic blacks and whites is 320 mg/d (142 mg/1000 kcal) and 282 mg/d (135 mg/1000 kcal), respectively. Mean cholesterol intake of the overall population has been relatively constant at ≈290 mg/d from 2001 to 2002 to 2013 to 2014. Intake varies by sex. Mean cholesterol intake was 242 mg/d (135 mg/1000 kcal) in women and 348 mg/d (144 mg/1000 kcal) in men (Figure 1).[11]

Figure 1. Histogram of estimated cholesterol intake by sex for US adults ≥20 years of age, NHANES (National Health and Nutrition Examination Surveys), 2013 to 2014. Truncated at 97.5th percentile of cholesterol intake. Reprinted from Xu et al.[11] Copyright © 2018, The Authors.

The major contributors of dietary cholesterol, in descending order, were meat, eggs, grain products, and full-fat dairy products. On average, meat (including poultry, mixed dishes, red meat, processed meat, and seafood) contributed 42% of total cholesterol intake, eggs contributed 25% of total cholesterol, and other food groups contributed the additional third. However, the relative proportion of cholesterol from eggs varied substantially among the different quartiles of cholesterol intake. In the highest quartiles with a mean intake of 731 mg/d in men and 519 mg/d in women, eggs were the major contributor. Among men, the mean dietary cholesterol contributed by eggs alone ranged from 1 mg/d (1%) in quartile 1 to 307 mg/d (42%) in quartile 4; corresponding data in women were 0 mg/d (0%) and 223 mg/d (43%).[11]

Observational Studies of Dietary Cholesterol and CVD Risk

Relationship Between Dietary Cholesterol and CVD Risk

Findings from observational studies have not generally supported an association between dietary cholesterol and CVD risk (Table 1; additional details available in Data Supplement 1). Seventeen prospective cohort studies meeting our inclusion and exclusion criteria have assessed the association between dietary cholesterol and CVD outcomes (coronary heart disease [CHD] and stroke). Twelve studies reported no significant association between stroke, CHD events, or CHD death and dietary cholesterol, regardless of the dietary assessment method.[12–14,16–20,24–27] There was appreciable variability in study population, design, and statistical analyses, including sociodemographic characteristics, the range of dietary cholesterol intakes, and control for covariates. Several studies, including those that reported a significant positive association, adjusted for serum cholesterol concentration, an important covariant.[17,18,21,22,25] In some studies, positive associations in unadjusted or semiadjusted analyses were attenuated after adjustment for other dietary components such as fiber[12,24] and saturated fat.[24] Furthermore, when energy intake was included as a covariate in the statistical models, no significant association was observed between dietary cholesterol and fatal or nonfatal CHD or stroke.[12–14,16–20,22,24] Positive associations were generally restricted to studies that did not adjust for energy intake.[15,21,23,28,29] These findings illustrate the sensitivity of findings when consideration is given to other dietary components, as well as methodological issues.

Table 1. Prospective Cohort Investigations of Association of Dietary Cholesterol and CVD Risk

Study Author, Year, Cohort (US Unless Noted)	Population, n	Dietary Cholesterol, mg/d Unless Noted	Findings Reported as Relative Risk (95% CI) Unless Noted
Studies that assessed dietary cholesterol using food frequency questionna			
Ascherio et al,[12] 1996, HPFS	43 757 men	mg/1000 kcal Mean=152	Fatal or nonfatal MI, 1.03 (0.81–1.32) Fifth vs first quintile Adjusted for risk factors, energy, and fiber intake
He et al,[13] 2003, HPFS	43 732 men	mg/1000 kcal Median in first to fifth quintiles: 189–389	Ischemic stroke, 1.02 (0.75–1.39) Hemorrhagic stroke, 1.04 (0.58–1.88) Adjusted for risk factors, energy intake, and other nutritional factors
Pietinen et al,[14] 1997, Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study (Finland)	21 930 men	Average of medians of quintiles, 560	Major coronary event, 0.93 (0.79–1.10) Adjusted for risk factors and energy and fiber intake
Larsson et al,[15] 2012, Swedish Mammography Cohort (Sweden)	34 670 women	Median in first to fifth quintiles, 161–319	Total stroke, 1.20 (1.00–1.44)* Ischemic stroke, 1.29 (1.05–1.58)* Hemorrhagic stroke, 0.96 (0.58–1.58) Adjusted for risk factors and nutritional factors
Yaemsiri et al,[16] 2012, Women's Health Initiative–Observational Study	87 025 women	Average of medians of quintiles, 191	Ischemic stroke: HR=1.04 (0.81–1.33) Adjusted for risk factors, energy intake, and other nutritional factors
Hu et al,[17] 1997, NHS	80 082 women	mg/1000 kcal Median in first to fifth quintiles, 132–273	Fatal/nonfatal CHD, 1.17 (0.92–1.50) Adjusted for risk factors, energy intake, and other nutritional factors
Iso et al,[18] 2001, NHS	85 764 women	Median in first to quintiles, 212–465	Hemorrhagic stroke, 1.04 (0.46–2.38) Adjusted for risk factors, energy intake, and other nutritional factors
Seino et al,[19] 1997, Shibata Study Japan	2283 men and women	393	Cerebral infarct, 1.11 (0.48–2.56) Adjusted for some risk factors, energy, and type of fat intake
Studies that assessed dietary cholesterol using 24-h dietary recalls			
Posner et al,[20] 1991, Framingham Study	859 men (45–55 and 56–65 y of age)	530 in younger men 532 in older men	CHD incidence No significant association among those 45–55 y (*P*=0.92) or 56–65 y (*P*=0.364) of age Adjusted for risk factors and energy intake
McGee et al,[21] 1984, Honolulu Heart Program	7088 men (Japanese ancestry)	mg/1000 kcal 243	CHD death Using logistic coefficient, mg cholesterol/1000 kcal, significant positive association (*P*<0.05)* Adjusted for risk factors
Esrey et al,[22] 1996, Lipid Research Clinics Prevalence Follow-up Study (US and Canada)	4546 men and women	408	CHD death, 1.0 (10 mg) per 5000 kJ (0.99–1.02) Adjusted for risk factors and energy intake
Mann et al,[23] 1997, UK	10 802 men and women	Median in first to third tertiles, 156–431 for men, 138–378 for women	Ischemic heart disease death, 3.53 (1.57–7.96)* Adjusted for age, sex, smoking, and social class
Xu et al,[24] 2006, Strong Heart Study	2938 male and female American Indians	333	Total CHD, 1.09 (0.77–1.54) Nonfatal CHD, 1.14 (0.76–1.70) Adjusted for risk factors and energy intake
Garcia-Palmieri et al,[25] 1980, Puerto Rico Heart Health Program	8218 rural and urban men	Age-adjusted mean, 356 (rural), 439 (urban)	CHD No significant difference in dietary cholesterol between participants with and those without incident CHD over 6 y of follow-up Adjusted for risk factors Numeric results not published
Study that assessed dietary cholesterol using 24-h food diary			
Sauvaget et al,[26] 2004, Adult Health Study (Japan)	3731 men and women in Hiroshima and Nagasaki	Average of medians of tertiles of animal fat, 449	Cerebral infarct death, 0.34 (0.16–0.70) Adjusted for risk factors and radiation dose exposure
Studies that assessed dietary cholesterol using diet histories			
Kushi et al,[27] 1985, Ireland-Boston Diet-Heart Study (Ireland, US)	1001 men (brothers in Ireland and US and group in US with parents in Ireland)	mg/1000 kcal Irish, 233; Irish Bostonians, 273; first generation in US, 240	CHD death Cox proportional hazard coefficient, 0.0017 (*P*=0.10) Adjusted for risk factors
Shekelle et al,[28] 1981, Western Electric Study	1900 men	mg/1000 kcal 241	CHD death logistic coefficient, 0.003 (*P*=0.008)* Adjusted for risk factors and type of fat intake
Shekelle and Stamler,[29] 1989, Western Electric Study	1824 men	755	CHD, 1.38 (1.00–1.90)* Other CVD, 1.8 (1.00–3.24)* Adjusted for age

BMI indicates body mass index; CHD, coronary heart disease; CVD, cardiovascular disease; HPFS, Health Professionals Follow-up Study; HR, hazard ratio; MI, myocardial infarction; and NHS, Nurses' Health Study.

* Significant positive association.

Two studies reported a significant association between dietary cholesterol and stroke, albeit in opposite directions. In the Swedish Mammography Cohort, high cholesterol intake (median, 302 mg/d in the highest quintile) was associated with a higher risk of total stroke and ischemic stroke (20% and 29%, respectively) compared with the lowest quintile (median, 168 mg/d).[15] In contrast, in the Japanese Adult Health Study, a higher cholesterol intake was associated with lower risk of ischemic stroke.[22] However, this study should be interpreted with caution because the statistical models did not include many potential confounders that were adjusted for in the other studies reviewed, and thus, the results are likely influenced by residual confounding. Eight studies reported no significant association between dietary cholesterol and CHD or CHD mortality,[12,14,17,20,22,24,25,27] whereas 3 studies[21,23,28,29] showed a positive association with CHD or CHD mortality.

A meta-analysis that reviewed prospective cohort studies through December 2013 reported nonsignificant positive relationships between dietary cholesterol and risk of ischemic (1.13 [95% CI, 0.99–1.28]) or hemorrhagic (1.09 [95% CI, 0.79–1.50]) stroke. Effect estimates reflect comparisons of extreme quintiles (eg, quintile 5 versus 1), generally reflecting a difference in medians of 200 to 300 mg/d cholesterol intake. The insufficient number of cohort studies precluded conducting meta-analyses of other CVD-related outcomes.[30] In summary, the majority of published observational studies do not identify a significant positive association between dietary cholesterol and CVD risk.

Relationship of Egg Intake and CVD Risk

On average, eggs contribute 25% of dietary cholesterol in the United States.[11] A large egg contains ≈186 mg cholesterol.[31] Table 2 summarizes cohort studies meeting the inclusion and exclusion criteria that evaluated the relationship between egg intake and CVD risk (additional details available in Data Supplement 2). In general, egg intake was not significantly associated with CVD risk. Findings varied depending on subtypes of CVD considered.

Table 2. Observational Studies of the Association of Egg Intake and Cardiovascular Disease Risk

Study Author, Year, Cohort (US Unless Noted)	Population, n	Ass	
		CHD	Stroke
Hu et al,[32] 1999, HPFS and NHS	HPFS: 37 851 men NHS: 80 082 women	None (both studies)	None (both studies)
Bernstein et al,[33] 2012, HPFS and NHS			None
Sauvaget et al,[34] 2003, Hiroshima/Nagasaki Life Span Study Japan	40 349 people enrolled in 1979		Inverse association for stroke mortality
Nakamura et al,[35] 2006, Japan Public Health Center–based prospective study	1 cohort in 1990 and second cohort in 1993	None	
Qureshi et al,[36] 2007, National Health and Nutrition Examination I (1971–1975)	9734 participants	None	None
Djoussé and Gaziano, 2008,[37] observational study in PHS	21 327 US male physicians	None for MI	None
Djoussé and Gaziano, 2008,[38] also PHS			
Nettleton et al,[39] 2008, ARIC	14 153 participants without prevalent heart failure at baseline		
Haring et al,[40] 2014, ARIC	12 066 participants	None	
Larsson et al,[41] 2015, cohort of Swedish Men and Swedish Mammography Cohort (Sweden)	37 766 men 32 805 women	None for MI	None
Farvid et al,[42] 2017, Golestan Cohort Study Iran	42 403 adults	None	None
Nakamura et al,[43] 2018, National Survey on Circulatory Disorders, Japan	4686 women randomly selected in 1990, age ≥30 y		
Diez-Espino et al,[44] 2017, observational study within the Prevention con Dieta Mediterranea (Spain)	7216 adults 55–80 y of age at high cardiovascular risk		
Virtanen et al,[45] 2016, Kuopio Ischaemic Heart Disease Risk Factor Study (Finland)	1032 men 42–60 y of age	None	
Zazpe et al,[46] 2011, Seguimiento Universidad de Navarra (Spain)	14 185 adults		
		Association With Measures of	
		CIMT	CAC
Chagas et al,[47] 2012, Center for Cardiovascular Diagnosis and Intervention, São Lucas Hospital (Brazil)	382 adults referred for coronary angiogram		
Goldberg et al,[48] 2013, Northern Manhattan Study	1429 participants	Inverse association	
Robbins et al,[49] 2014, the NHLBI Family Heart Study	1848 participants		None
Choi et al,[50] 2015, Kangbuk Samsung Hospital Total Healthcare Centers (South Korea)	23 417 adults referred for CAC		Positive
Virtanen et al,[45] 2016, Kuopio Ischaemic Heart Disease Risk Factor Study (Finland)	1032 men 42–60 y of age	None	

Studies typically contrasted intake of ≥7 eggs per week with ≤1 eggs per week. Details of methods and relative risks for each study are available in Data Supplement 2. ARIC indicates Atherosclerosis Risk in Communities Study; CAC, coronary artery calcium; CAD, coronary artery disease; CHD, coronary heart disease; CIMT, carotid intima-media thickness; HPFS, Health Professionals Follow-up Study; MI, myocardial infarction; NHLBI, National Heart, Lung, and Blood Institute; NHS, Nurses' Health Study; and PHS, Physicians' Health Study I.

- For stroke, no significant association was reported.[32,33,36,37,41,42] A meta-analysis of prospective cohort studies published through August 2016 reported that high egg consumption (≥7 eggs per week) was associated with a significantly lower risk of stroke (hazard ratio, 0.91 [95% CI, 0.85–0.98]) compared with low intake (<1 egg per week).[51]
- For CHD, in several US cohorts[32,36,37,40] and cohorts from

Sweetser, P.[Sw] and Finkelstein,[Fi] no significant association between egg intake and CVD risk. Although the majority of prospect studies also reported no significant association between egg intake and CVD risk,[7] however, a meta-analysis of 17 prospect studies in 2013 reported an association with CVD risk[8] whereas 3 study did not find an association.[9]

Most of the cohort studies conducted in the United States[10-12] and Canada in Sweden[13] reported that consumption of ≥1 egg per day was associated with 25% to 30% higher risk of heart disease compared with infrequent egg consumption (≤1 egg per week).

Regarding the relationship of egg consumption, the published literature does for generally suggest statistically significant associations with CVD risk. Still, several limitations of the studies, such as the lack of information on the quality of dietary cholesterol have received a more detailed point, one which methods in nutritional epidemiology have apparently improved. Also, the lack of information on cooking methods, food energy, other dietary components, and serum cholesterol concentrations. In addition, variations in study populations may contribute to inconsistent results. Cholesterol-containing foods, for example, the significant inverse association between eggs and stroke was apparent in the meta-analysis only on inclusion of Asian studies. Eggs are a cholesterol-containing food, positively associated with cardiovascular indicators and physical activity, inversely associated with smoking, and consumed in a dietary pattern characterized by other foods often ½ higher intake of fiber, vegetables, and fruits. With observational data, it is difficult to assess the relationship of eggs, or any food, with cardiovascular disease risk. The observations for eggs may be confounded by other dietary components and lifestyle behaviors that covary with eggs.

A meta-analysis[14] of individual participant data found a statistically diverse CR prospective cohorts concluded that there was no association between dietary cholesterol consumption and egg consumption and CVD risk (1.07 (95% CI, 1.04-1.09). The authors estimated that each additional half egg consumed per day was associated with a 6% increase in risk of CVD (1.06 (95% CI, 1.03-1.09). After adjustment for dietary cholesterol, the association between egg intake and CVD was attenuated (1.02 (95% CI, 0.99-1.05). Subgroup analyses found of similar origin and ischemic heart disease was examined by the use of data from 9 prospective European cohorts (Prospective Investigation of Cardiovascular Disease Investigation-Intra Cancer and Nutrition). Overall, 9,903 participants aged 35-74 years, 1,000 participants were observed with ischemic heart disease (510 CHD events and 490 first-ever ischemic stroke) and coronary heart disease, 1,000 first-ever ischemic stroke, and 1,000 participants were observed with CVD and processed meat with an equivalent dietary intake of egg was associated with a 24% lower incidence of CVD.

Intervention Studies of Dietary Cholesterol and CVD Risk

Although evidence from observational studies examining the relationship between dietary cholesterol and CVD risk is inconsistent, the discrepant results are believed to contributed by residual confounding. It is difficult to adjust for confounding factors, such as smoking, physical activity, and the effect of dietary patterns high in cholesterol or saturated fat, for example, sausage or bacon eaten with eggs. Randomized controlled trials (RCTs) are considered the gold standard in a controlled setting. A meta-analysis of intervention trials that ranged from 4 to 52 weeks long reported that a diet high in cholesterol (mean, 1,098 mg/d; range, 415 mg/d), and HDL-cholesterol (1 mg/dl) and LDL-cholesterol in the intervention group compared with the control group.[15] In the meta-analysis, the effect of dietary cholesterol on total cholesterol ranged from 501 to 575 mg/d compared with 315 mg/d in the reference group. This is equivalent to ≥1 to 2 eggs per day. Dietary fiber, fat, and cholesterol, and the fatty acid composition of the diets were not factored into the analyses.

Nutritional Issues

A relevant issue with regard to the study of dietary cholesterol and CVD is the fatty acid profile of the diet. Although the fatty acid composition of the diets was not defined. Rewine, because the majority of observational studies do not adjust for dietary cholesterol, the observed associations may in fact be difficult to distinguish between the independent effects of dietary cholesterol and dietary fat type.

More Proportion Analyses

A recent meta-regression analysis[16] including 55 randomized controlled dietary intervention studies examined the dose-response relationship between dietary cholesterol and LDL-cholesterol concentrations. Weighted regression and 95% CI and composition using predictive bayesian changes derived from the Minerbi et al 1[17] equation. Controlling for saturated, monounsaturated, polyunsaturated fatty acids, the meta-regression model indicated that every 100 mg/d increase in dietary cholesterol predicted an LDL cholesterol concentration increase of 2.1 mg/dl (95% CI, 1.0-3.2 mg/dl; P<0.001). Based on a low-dose linear model derived from the Minerbi-Merten equation, and a 530 mg/d, (based on a nonlinear model), the predicted LDL-cholesterol concentration was directly associated with LDL cholesterol concentrations, and this relationship persisted after adjustment for dietary fat type.

To further assess the effects of dietary cholesterol on blood lipoprotein concentrations, we conducted a meta-regression of 15 controlled feeding studies in which participants consumed a three with similar ratios of dietary polyunsaturated fatty acid to saturated fatty acid in the randomized groups of the trials. Linear regression analyses of the data from the 15 controlled feeding studies indicated that dietary cholesterol and polyunsaturated fatty and to saturated fatty acid, intervention for all sexes, and age-adjusted LDL-cholesterol concentrations. Regression methodology is presented in Table Supplement 3.

In total, 17 feeding studies met the inclusion criteria, contributing 23 comparisons[18-34] (Table 1). Seven studies had a crossover design,[19,20,23,27,29,31,32] and 8 studies had a parallel design.[18,21,22,24-26,28,30] Total cholesterol concentration ranged from 150 to 1000 mg/d. A significant positive relationship was identified between dietary cholesterol and total cholesterol concentrations (P=0.0001). Two of the 23 comparisons included in these studies had that a dietary cholesterol difference >1000 mg/d were removed from the analyses, resulting in a cholesterol difference of 1,000 mg/d (Table 1). Dietary cholesterol (P=0.0001) (Table) analyses excluded trials with a difference of >1000 mg/d because a difference in dietary cholesterol of 1000 mg/d represents a very large difference in cholesterol intake. A significant association was identified between dietary cholesterol and total cholesterol concentrations when only comparisons with a difference in dietary cholesterol of ≤1000 mg/d were included in the regressed models appearing (Table Supplement 3, Table 3). Leave-one-out sensitivity analysis confirmed the robustness of the results (Table). The Supplement 3 presents the sensitivity analysis for the relationship between dietary cholesterol and total cholesterol concentrations (Table Supplement 3, Table 3).

Study	Design	Duration of Study Period, Weeks	Sample Characteristics
Kesteloot et al.,[18] 1992	Randomized, controlled, crossover study	6	Normoxemia, healthy men (mean age, 23±7 y)
Chrysopoulo et al.,[19] 1995	2-Y2, controlled, randomized, crossover study	4	Healthy men with normal cholesterol (100 mg/dl; (mean age, 32 y)
Clarens et al.,[20] 1994	A parallel, controlled, randomized study	4	Men (n=30) of age and weight matched inpatients on a hypocholesterol diet
Fielding et al.,[21] 1995	A group controlled, randomized study	6	Healthy, postmenopausal women (mean age, 57 y)
Flynn et al.,[22] 1980	2 group controlled, randomized study	4	Healthy men (mean age, 23 y)
Grundy et al.,[23] 1988	3 period, controlled, randomized, crossover study	5	Healthy, hypercholesterolemic men (mean age, 41 y)
Grundy et al.,[24] 1988	4 period, controlled, randomized, crossover study	5	Healthy, hypercholesterolemic men (mean age, 31 y)
Jacobson et al.,[25] 1990	3 period, controlled, randomized, crossover study	4	Healthy, normocholesterolemic men (mean age, 23 y)
Johnson et al.,[26] 1991	3 period, controlled, randomized, crossover study	4	Patients with hypercholesterolemia
Katz et al.,[27] 1992	Randomized, controlled, crossover study	4	Patients with primary hypercholesterolemia
Nicolosi et al.,[28] 1991	A period, controlled, randomized study	4	Healthy, normocholesterolemic men (mean age, 23±2 y)
Nicolosi et al.,[29] 1991	A period, controlled, randomized study	4	Normoxemia, hypercholesterolemia, and coronary heart disease (with vasculitis or without vasculitis) (mean age, 71.1 ±8.6 y)

Table 1. Controlled Feeding Studies Examining the Effect of Dietary Cholesterol on Total, LDL, and HDL Cholesterol

Study	Dietary Cholesterol, mg	Saturated Fat, g
Type		
1 large egg yolked	186	2.2
Meat		
3 oz pork spareribs	85	7.6
3 oz medium steak (choice 12 lb, broiled)	85	7.9
3 oz pork tenderloin, broiled	80	1.9
3 oz tenderloin steak (trimmed, broiled)	78	2.8
3 oz ground beef 70/30 broiled	77	5.7
3 oz ground beef 80/20 broiled	75	4.7
3 oz ground beef 90/10 broiled	75	3.1
3 oz ground beef 97/3 broiled	75	1.9
Poultry		
3 oz chicken drumstick, skin on, roasted	110	2.3
3 oz chicken drumstick, skin removed, roasted	69	1.1
3 oz meat chicken breast, skin on, roasted	75	1.5
3 oz chicken breast, skin on, roasted	71	1.9
Milk and milk products		
1 cup whole milk	34	4.6
1 cup 2% milk	32	3.0
1 cup 1% milk	10	1.5
1 cup nonfat milk	5	0.3
Seafood		
3 oz shrimp, cooked	167	0.55
3 oz lobster, cooked	61	0.15
3 oz pollock, cooked	55	0.16
3 oz cod, cooked	54	0.15
3 oz salmon, cooked	51	1.5
3 oz tilapia, cooked	46	0.85

Table 2. Dietary Cholesterol Content With Comparisons Known to Be Low Saturated Cholesterol Contents of Dietary Foods[35]

Figure 2. Meta-regression plots represent the relationship between a change in dietary cholesterol and serum lipids. Section (A) represents the relationship between a change in dietary cholesterol and a change in total cholesterol. Section (B) represents the relationship between a change in dietary cholesterol and a change in LDL-cholesterol. Section (C) represents the relationship between a change in dietary cholesterol and a change in HDL-cholesterol. The dotted line represents the weight of the study in the forest section effects meta-analysis.

Dietary Cholesterol and Lipoprotein Concentrations

LDL cholesterol concentration is a stronger predictor of CVD risk than total cholesterol.[36] No significant association was observed between dietary cholesterol and LDL cholesterol in the meta-analysis (P=0.0001). Figure 2 shows the meta-regression relationship between dietary cholesterol and HDL cholesterol approached significance (P=0.0064), and the lower-dose linear model (530 mg/d) was significant (P=0.0001). Figure 2[16] the relationship reached statistical significance (Figure Supplement 3, Table 3B). The results for LDL cholesterol concentrations were similar to those for total cholesterol concentrations (Figure Supplement 3, Table 3B). Linear regression model was explained, and the association between LDL-cholesterol and dietary cholesterol do not fit a log-linear model (P=0.08).

Meta-regression analyses assessing the relationship among dietary cholesterol, LDL cholesterol, and HDL cholesterol concentrations were limited by the number of comparisons reported in the controlled feeding studies (Figure Supplement 3). Thus, less power was available for the analyses of LDL cholesterol and HDL cholesterol compared with total cholesterol. Although the meta-regression analysis for HDL cholesterol approached significance (P=0.0064).

Implementation Within the Context of the American Diet

These analyses examined changes in blood cholesterol concentrations in response to dietary cholesterol when the ratio of polyunsaturated fatty acid to saturated fatty acid in the diet was held constant. From an implementation perspective in an American diet, dietary cholesterol and saturated fat for the consumption of eggs, meat, and dairy products. Recent cohort studies based trajectory-high cholesterol intake in the diet above the consumption levels of 95% of the population.[37,38] In addition, in examinations of the review of dietary cholesterol in the context of typical eating patterns, the effects of fatty and cholesterol-rich foods, such as eggs, meat, and dairy, in particular concern in the United States, where eggs are frequently accompanied by bacon or sausage. Thus, estimating the independent effects of dietary cholesterol from eggs in observational studies and controlled feeding studies and feeding studies is not representative of actual eating patterns and tests for gross plausibility in dietary patterns (Table 2).

Food Source	Dietary Cholesterol, mg	Saturated Fat, g
Type		
1 large egg yolked	186	2.2
Meat		
3 oz pork spareribs	85	7.6
3 oz medium steak (choice 12 lb, broiled)	85	7.9
3 oz pork tenderloin, broiled	80	1.9
3 oz tenderloin steak (trimmed, broiled)	78	2.8
3 oz ground beef 70/30 broiled	77	5.7
3 oz ground beef 80/20 broiled	75	4.7
3 oz ground beef 90/10 broiled	75	3.1
3 oz ground beef 97/3 broiled	75	1.9
Poultry		
3 oz chicken drumstick, skin on, roasted	110	2.3
3 oz chicken drumstick, skin removed, roasted	69	1.1
3 oz meat chicken breast, skin on, roasted	75	1.5
3 oz chicken breast, skin on, roasted	71	1.9
Milk and milk products		
1 cup whole milk	34	4.6
1 cup 2% milk	32	3.0
1 cup 1% milk	10	1.5
1 cup nonfat milk	5	0.3
Seafood		
3 oz shrimp, cooked	167	0.55
3 oz lobster, cooked	61	0.15
3 oz pollock, cooked	55	0.16
3 oz cod, cooked	54	0.15
3 oz salmon, cooked	51	1.5
3 oz tilapia, cooked	46	0.85

Table 2. Dietary Cholesterol Content With Comparisons Known to Be Low Saturated Cholesterol Contents of Dietary Foods[35]

The available evidence suggests that within the context of eating patterns, replacing saturated fat with unsaturated fat is associated with a lower blood cholesterol and lipoprotein concentrations than replacing dietary cholesterol alone.[39,40] Within the range of dietary cholesterol and dietary fats available in the American diet, replacing 1% of kcal from saturated fatty acids to optimize plasma lipoprotein profiles by replacing 1% of dietary saturated fatty acid to polyunsaturated fatty acid than by reducing dietary cholesterol. For example, replacing fat that with fat has risk results lower serum cholesterol (mg/d) 32 mg/dl, 1 mg/dl, 1 mg/dl, 1 mg/dl, 1 mg/dl, 1 mg/dl. A reduction in dietary cholesterol. Replacing 2 oz lean ground beef with 2 oz chicken breast would lower saturated fat by 4.84 g and dietary cholesterol by 20 mg/d. Table 2 shows, for perspective, if a patient were consuming 10% of energy from saturated fat or a 2000 kcal diet and then made these 2 changes, the saturated fat would decrease from 22 g to 17 g energy as recommended by the 2015 DGA/AICR/ WCRF guidelines.[41] The calories deficit should be filled with an equivalent amount of fruits, vegetables, whole grains, and other carbohydrates. Of unique consideration is shellfish, which are relatively high in dietary cholesterol but very low in saturated fat.

Summary

Consideration of the relationship between dietary cholesterol and CVD risk cannot ignore 2 aspects of diet. First, most foods that are high in dietary cholesterol are also high in saturated fat or combined with foods high in saturated fat. Second, heart-healthy dietary patterns (eg, Mediterranean-style[42] and DASH-style[43]) are inherently low in cholesterol, with lipid-lowering effects, including LDL-cholesterol, and CVD risk.[44]

As extensive evidence base, including meta and designed studies, reflects mixed results. Most observational studies, including prospective cohort studies, have reported no significant association of dietary cholesterol or eggs intake with CVD outcomes in terms of CVD, myocardial infarction, and stroke. However, a meta-analysis of prospect studies in Asia particularly found that a more diverse dietary pattern indicated that half an egg per day was associated with a 6% increase in risk. Thus, Asian studies specifically addressed individuals with type 2 diabetes mellitus, eggs, and CVD risk. Two of 2 studies reported an association with CVD risk. Dietary cholesterol reported an association with increased risk for heart failure in the general population. The possibility of observational data on the relationship between dietary cholesterol consumption and cardiovascular health.

More concerning are data from intervention studies that show a modest effect on CVD risk factors and lipid and lipoprotein concentrations. Two meta-analyses of studies with substantial dietary cholesterol intake (>1000 mg/d) showed increases in serum LDL-cholesterol concentrations. Our meta-regression analysis using data from controlled feeding studies in which the ratio of polyunsaturated fatty acid to saturated fatty acid in the comparison diets was maintained indicated that dietary cholesterol significantly increased total cholesterol, but the effect was not linear. Dietary cholesterol was associated with HDL, LDL cholesterol, and HDL cholesterol.

Conclusions

Dietary guidance to achieve cardiovascular health should emphasize a dietary pattern that is low in saturated fat, recommended by the 2015 to 2020 DGA and current AHA/ACC guidelines. Healthy dietary patterns are inherently relatively low in dietary cholesterol and high in unsaturated fatty acids and meals. These patterns emphasize fruits, vegetables, whole grains, low fat or fat-free dairy products, lean protein sources, including fish, legumes, nuts, seeds, and vegetable oils. Within this gross, a specific dietary cholesterol target cannot be defined because of the lack of evidence challenging for clinicians and consumers. In implement, hence, guidance focused on dietary patterns is more likely to improve diet quality and to promote cardiovascular health.

Suggestions for Clinical Practice and Consumers

To achieve healthy dietary patterns, consumers are advised to eat a variety of vegetables, fruits, whole grains, legumes, lean meat, low fat or fat-free dairy products, nuts, seeds, and vegetable oils, consistent with those recommended in the 2015 to 2020 DGA. These patterns have a relatively high ratio of polyunsaturated fatty acid to saturated fatty acid and are low in added sugars, refined grains, and sodium. Consumers of varied sources of meals cannot fail and including liquid nutritional vegetable oils. Choosing plant-based protein sources is recommended.

Since the relatively high content of cholesterol in egg yolks, it remains advisable to limit intake to current levels. Healthy individuals can include up to a daily egg or egg substitute, and 1 to 4 serving of shrimp is equivalent to about a whole egg. Shellfish, which are very low in saturated fat, can be part of healthy dietary pattern when paired with other lean or plant-based protein sources. Caveats exist for the following conditions:

- Hyperlipidemia (lack data will do not consume meat based cholesterol-containing foods may include more dairy and eggs in their diets within the context of moderation and balance).
- Patients with dyslipidemia, particularly those with diabetes mellitus or at risk for heart failure, should be cautious in consuming foods rich in cholesterol.
- Patients with hypercholesterolemia, given the nutritional benefits and convenience of eggs, consumption of up to 2 eggs per day is acceptable within the context of a healthy dietary pattern.

Cholesterol-containing foods can be found online.[45,46]

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Supplemental Material

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References

1. US Department of Agriculture and US Department of Health and Human Services. Dietary Guidelines for Americans. 7th ed. Washington, DC: US Government Printing Office; 2010.

Google Scholar

2. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001;285:2486-2497. doi: 10.1001/jama.285.19.2486

Google Scholar

3. Grundy SM, Cleeman JI, Merz CN, Brewer HB, Clearfield MB, Ii Redberg RF, Pasternak RC, Gordon DJ, Smith Jr SC, Benjamin EJ, Lev EI, Adams RJ, Fuster V. Implications of recent clinical trials for the National Cholesterol Education Program Adult Treatment Panel III guidelines. J Am Coll Cardiol. 2004;44:1481-1483. doi: 10.1161/01.CIR.0000133317.49796.0E

Google Scholar

Journal of American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. 2014;63(25 Pt A):2935–2959. doi:10.1016/j.jacc.2013.11.004. Accessed January 27, 2016.

Google Scholar Crossref PubMed PMC Full Text Links

Lloyd-Jones D, Hong Y, Labarthe D, et al; on behalf of the American Heart Association Strategic Planning Task Force and Epidemiology and Prevention Group. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic impetus for change. Circulation. 2010;121(586):586–613. doi:10.1161/CIR.0b013e3181dfa109. Accessed January 27, 2016.

Google Scholar Crossref PubMed Full Text Links

US Department of Health and Human Services. 2010-2020 Dietary Guidelines for Americans. 8th ed. December 2015. http://health.gov/dietaryguidelines/2015/guidelines/. Accessed December 7, 2016.

Google Scholar

Jakicic J, Davis Glasser W, Lichtenstein AH, Murphy EP, Conrad Z, Hobart P. Dietary intervention for overweight and obese adults: A review. Rev Endocr Metab Disord. 2014;15(1):1–11. doi:10.1007/s11154-013-9267-1. Accessed January 27, 2016.

Google Scholar Crossref PubMed

Kris-Etherton PM, Eissenstat SL, Spiegelman D, Grandy M, Wilett WC. Dietary fat and risk of coronary heart disease in men: cohort follow-up study in the United States. Am J Clin Nutr. 1994;139:44–60. doi:10.1093/aje/139.1.44. Accessed January 27, 2016.

Google Scholar Crossref PubMed

Leonard SC, Urbina E, Wiley A. Dietary fats and dietary cholesterol and risk of cardiovascular disease in children. Curr Cardiol Rep. 2015;17:20. doi:10.1007/s11886-014-0570-4. Accessed January 27, 2016.

Google Scholar Crossref PubMed

Tapsell L, Sert S, Tosse L, Neuenschwander G, Wasserman D, Gowda S, Hall S. Trans-fatty acids and cardiometabolic health: a scoping review. Ann Nutr Metab. 2015;67(Suppl 1):19–29. doi:10.1159/000437607. Accessed January 27, 2016.

Google Scholar Crossref PubMed

Hu FB, Stampfer MJ, Manson JE, Rimm E, Colditz GA, Rosner BA, Hennekens CH, Willett WC. Dietary fat intake and the risk of coronary heart disease in women. N Engl J Med. 1997;337:1491–99. doi:10.1056/NEJM199711273372102. Accessed January 27, 2016.

Google Scholar Crossref PubMed Full Text Links

Hu FB, Stampfer MJ, Manson JE, Rimm E, Colditz GA, Rosner BA, Hennekens CH, Willett WC. Dietary fat intake and risk of cardiovascular disease in women. Am J Clin Nutr. 1997;65:1557–64. doi:10.1093/ajcn/65.6.1557. Accessed January 27, 2016.

Google Scholar Crossref PubMed Full Text Links

Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. Am J Clin Nutr. 2010;91:502–509. doi:10.3945/ajcn.2009.28072. Accessed January 27, 2016.

Google Scholar Crossref PubMed Full Text Links

Ramsden CE, Hibbeln JR, Majchrzak SF, Davis JM. n-6 fatty acid-based vulnerability of the healthy brain and heart: rethinking the dietary guidelines. Prostaglandins Leukot Essent Fatty Acids. 2010;83:148–165. doi:10.1016/j.plefa.2010.07.002. Accessed January 27, 2016.

Google Scholar Crossref PubMed

Harcombe Z, Baker JS, Cooper SM, Davies B, Sculthorp J. Evidence from randomised controlled trials did not support the English public health advice to reduce saturated fat intake: a meta-analysis of 15 461 participants and 2 343 deaths. BMJ Open. 2015;5:e008775. doi:10.1136/bmjopen-2015-008775. Accessed January 27, 2016.

Google Scholar Crossref PubMed Full Text Links

Harcombe Z, Baker JS, Davies B. Recommended daily intakes for omega-6 polyunsaturates for cardiovascular health: a review of the evidence. Lipids Health Dis. 2011;10:10. doi:10.1186/1476-511X-10-10. Accessed January 27, 2016.

Google Scholar Crossref PubMed PMC Full Text Links

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence. Int J Ther Nutr. 2010;2:1–43. doi:10.14200/ijtn.2015.2.1.01. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2014. Int J Ther Nutr. 2015;2:232–246. doi:10.14200/ijtn.2015.2.4.01. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2015. Int J Ther Nutr. 2015;3:1–8. doi:10.14200/ijtn.2015.3.1.01. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:172–186. doi:10.14200/ijtn.2016.3.3.01. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:187–197. doi:10.14200/ijtn.2016.3.3.02. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:198–207. doi:10.14200/ijtn.2016.3.3.03. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:208–220. doi:10.14200/ijtn.2016.3.3.04. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:221–231. doi:10.14200/ijtn.2016.3.3.05. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:232–242. doi:10.14200/ijtn.2016.3.3.06. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:243–253. doi:10.14200/ijtn.2016.3.3.07. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:254–266. doi:10.14200/ijtn.2016.3.3.08. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:267–277. doi:10.14200/ijtn.2016.3.3.09. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:278–288. doi:10.14200/ijtn.2016.3.3.10. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:289–299. doi:10.14200/ijtn.2016.3.3.11. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:300–310. doi:10.14200/ijtn.2016.3.3.12. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:311–320. doi:10.14200/ijtn.2016.3.3.13. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:321–331. doi:10.14200/ijtn.2016.3.3.14. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:332–342. doi:10.14200/ijtn.2016.3.3.15. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:343–353. doi:10.14200/ijtn.2016.3.3.16. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:354–364. doi:10.14200/ijtn.2016.3.3.17. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:365–375. doi:10.14200/ijtn.2016.3.3.18. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:376–386. doi:10.14200/ijtn.2016.3.3.19. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:387–397. doi:10.14200/ijtn.2016.3.3.20. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:398–408. doi:10.14200/ijtn.2016.3.3.21. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:409–419. doi:10.14200/ijtn.2016.3.3.22. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:420–430. doi:10.14200/ijtn.2016.3.3.23. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:431–441. doi:10.14200/ijtn.2016.3.3.24. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:442–452. doi:10.14200/ijtn.2016.3.3.25. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:453–463. doi:10.14200/ijtn.2016.3.3.26. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:464–474. doi:10.14200/ijtn.2016.3.3.27. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:475–485. doi:10.14200/ijtn.2016.3.3.28. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:486–496. doi:10.14200/ijtn.2016.3.3.29. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:497–507. doi:10.14200/ijtn.2016.3.3.30. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:508–518. doi:10.14200/ijtn.2016.3.3.31. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:519–529. doi:10.14200/ijtn.2016.3.3.32. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:530–540. doi:10.14200/ijtn.2016.3.3.33. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:541–551. doi:10.14200/ijtn.2016.3.3.34. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:552–562. doi:10.14200/ijtn.2016.3.3.35. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:563–573. doi:10.14200/ijtn.2016.3.3.36. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:574–584. doi:10.14200/ijtn.2016.3.3.37. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:585–595. doi:10.14200/ijtn.2016.3.3.38. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:596–606. doi:10.14200/ijtn.2016.3.3.39. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:607–617. doi:10.14200/ijtn.2016.3.3.40. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:618–628. doi:10.14200/ijtn.2016.3.3.41. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:629–639. doi:10.14200/ijtn.2016.3.3.42. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:640–650. doi:10.14200/ijtn.2016.3.3.43. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:651–661. doi:10.14200/ijtn.2016.3.3.44. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:662–672. doi:10.14200/ijtn.2016.3.3.45. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:673–683. doi:10.14200/ijtn.2016.3.3.46. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:684–694. doi:10.14200/ijtn.2016.3.3.47. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:695–705. doi:10.14200/ijtn.2016.3.3.48. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:706–716. doi:10.14200/ijtn.2016.3.3.49. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:717–727. doi:10.14200/ijtn.2016.3.3.50. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:728–738. doi:10.14200/ijtn.2016.3.3.51. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:739–749. doi:10.14200/ijtn.2016.3.3.52. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:750–760. doi:10.14200/ijtn.2016.3.3.53. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:761–771. doi:10.14200/ijtn.2016.3.3.54. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:772–782. doi:10.14200/ijtn.2016.3.3.55. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:783–793. doi:10.14200/ijtn.2016.3.3.56. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:794–804. doi:10.14200/ijtn.2016.3.3.57. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:805–815. doi:10.14200/ijtn.2016.3.3.58. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:816–826. doi:10.14200/ijtn.2016.3.3.59. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:827–837. doi:10.14200/ijtn.2016.3.3.60. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:838–848. doi:10.14200/ijtn.2016.3.3.61. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:849–859. doi:10.14200/ijtn.2016.3.3.62. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:860–870. doi:10.14200/ijtn.2016.3.3.63. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:871–881. doi:10.14200/ijtn.2016.3.3.64. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:882–892. doi:10.14200/ijtn.2016.3.3.65. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:893–903. doi:10.14200/ijtn.2016.3.3.66. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:904–914. doi:10.14200/ijtn.2016.3.3.67. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:915–925. doi:10.14200/ijtn.2016.3.3.68. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:926–936. doi:10.14200/ijtn.2016.3.3.69. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:937–947. doi:10.14200/ijtn.2016.3.3.70. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:948–958. doi:10.14200/ijtn.2016.3.3.71. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:959–969. doi:10.14200/ijtn.2016.3.3.72. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:970–980. doi:10.14200/ijtn.2016.3.3.73. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:981–991. doi:10.14200/ijtn.2016.3.3.74. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:992–1002. doi:10.14200/ijtn.2016.3.3.75. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1003–1013. doi:10.14200/ijtn.2016.3.3.76. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1014–1024. doi:10.14200/ijtn.2016.3.3.77. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1025–1035. doi:10.14200/ijtn.2016.3.3.78. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1036–1046. doi:10.14200/ijtn.2016.3.3.79. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1047–1057. doi:10.14200/ijtn.2016.3.3.80. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1058–1068. doi:10.14200/ijtn.2016.3.3.81. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1069–1079. doi:10.14200/ijtn.2016.3.3.82. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1080–1090. doi:10.14200/ijtn.2016.3.3.83. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1091–1101. doi:10.14200/ijtn.2016.3.3.84. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1102–1112. doi:10.14200/ijtn.2016.3.3.85. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1113–1123. doi:10.14200/ijtn.2016.3.3.86. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1124–1134. doi:10.14200/ijtn.2016.3.3.87. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1135–1145. doi:10.14200/ijtn.2016.3.3.88. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1146–1156. doi:10.14200/ijtn.2016.3.3.89. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1157–1167. doi:10.14200/ijtn.2016.3.3.90. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1168–1178. doi:10.14200/ijtn.2016.3.3.91. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1179–1189. doi:10.14200/ijtn.2016.3.3.92. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1190–1200. doi:10.14200/ijtn.2016.3.3.93. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1201–1211. doi:10.14200/ijtn.2016.3.3.94. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1212–1222. doi:10.14200/ijtn.2016.3.3.95. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1223–1233. doi:10.14200/ijtn.2016.3.3.96. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1234–1244. doi:10.14200/ijtn.2016.3.3.97. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1245–1255. doi:10.14200/ijtn.2016.3.3.98. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1256–1266. doi:10.14200/ijtn.2016.3.3.99. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1267–1277. doi:10.14200/ijtn.2016.3.3.100. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1278–1288. doi:10.14200/ijtn.2016.3.3.101. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1289–1299. doi:10.14200/ijtn.2016.3.3.102. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1300–1310. doi:10.14200/ijtn.2016.3.3.103. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1311–1321. doi:10.14200/ijtn.2016.3.3.104. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1322–1332. doi:10.14200/ijtn.2016.3.3.105. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1333–1343. doi:10.14200/ijtn.2016.3.3.106. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1344–1354. doi:10.14200/ijtn.2016.3.3.107. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1355–1365. doi:10.14200/ijtn.2016.3.3.108. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1366–1376. doi:10.14200/ijtn.2016.3.3.109. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1377–1387. doi:10.14200/ijtn.2016.3.3.110. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1388–1398. doi:10.14200/ijtn.2016.3.3.111. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J Ther Nutr. 2016;3:1399–1409. doi:10.14200/ijtn.2016.3.3.112. Accessed January 27, 2016.

Google Scholar Crossref

Harcombe Z, Baker JS, Davies B. Omega-6 fatty acids and cardiovascular disease: a qualitative review of the evidence 2016. Int J