

Table. PUFAs and CHD/CVD: Reviews

Author/ Year	De- sign Type	Class	Quality (+,-,Ø)	Purpose/ Population Sample Size	Regimen	Primary Outcome Measures Results	Author's Conclusions/ Reviewer's Comments (<i>Italicized</i>)
Kris-Etherton et al., 2004	Re- view	M	NA	<p>Purpose: To review human studies for effects of PUFA on CVD risk factors</p> <p>Inclusions: Epidemiologic and clinical investigations</p> <p>Exclusions: None given</p>	<p>Study Protocol: Not reported</p> <p>Data Collection: Not reported</p>	<p>Outcome Measures: CVD risk factors</p> <p>Actual Sample: Not reported</p> <p>Results: Epidemiological studies, in general, not supportive of incr PUFA intake assoc with adverse effects on CVD end points</p> <p>Clinical investigations suggest linoleic acid may decr blood chol, especially LDL, by upregulating LDL receptors</p> <p>Clinical diet studies show that diets low in SFA and rich in PUFA decr LDL by 13-15%; assoc with 25-43% decr in CVD events</p> <p>Limited clinical studies suggest PUFA more favorable effects on postprandial TG and lipoprotein metabolism than MUFA or SFA</p> <p>Controlled clinical trials suggest PUFA decr TC and LDL, SFA increase TC and LDL and MUFA neutral effect</p> <p>Epidemiological and clinical</p>	<p>Author's Conclusions: "Epidemiologic studies have shown a beneficial association between polyunsaturated fatty acid (PUFA), specifically linoleic acid (C18:2, n-6), intake and cardiovascular disease morbidity and mortality. Clinical studies have shown that n-6 PUFAs have the most potent cholesterol-lowering effects of the individual fatty acid classes, and emerging evidence suggests that PUFAs have favorable effects on postprandial lipemia. However, some studies suggest that high intakes of linoleic acid may have adverse effects on pro-inflammatory cytokines and adhesion molecules.</p>

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						<p>studies suggest limited evidence that linoleic acid may adversely affect certain CVD risk factors such as endothelial activation and oxidative stress</p> <p>NHANES studies suggest PUFA intake incr over time. PUFA contribute approx 19-22% of energy from fat in adult diets</p>	<p>Research is needed to establish the optimal level of dietary PUFAs that maximally affects the greatest number of health risk factors”</p> <p>Reviewer's Comments: <i>None</i></p>
Hu and Willett, 2002	Sys-tem-atic review	M	Ø	<p>Purpose: To review metabolic, epidemiologic and clinical trial evidence regarding diet and CHD prevention</p> <p>Inclusions: Epidemiologic and clinical investigations; dietary factors and CHD</p> <p>Exclusions: Not given</p>	<p>Study Protocol: Searched MEDLINE through May 2002 for epidemiologic and clinical investigations of various dietary factors (fat, chol, n-3 FA, TFA, CHO, glycemic index, fiber, folate, specific foods and dietary patterns) and CHD</p> <p>Data Collection: Data examined for relevance and quality and extracted by 1 author</p> <p>Emphasized controlled trials with clinical end points, but gave substantial weight to</p>	<p>Outcome Measures: CHD prevention</p> <p>Actual Sample: 147 studies</p> <p>Results: Numerous controlled feeding studies report that: SFA incr and PUFA decr TC and LDL; SFA, MUFA and PUFA incr HDL when they replace CHO in diet; TG levels incr when dietary FA replaced by CHO</p> <p>In numerous controlled metabolic studies, TFA shown to raise LDL levels and lower HDL relative to <i>cis</i>-unsaturated FA and incr in TC/HDL ratio for TFA approx twice that for SFA</p>	<p>Author's Conclusions: “Compelling evidence from metabolic studies, epidemiologic investigations, and clinical trials in the past several decades converges to indicate that at least 3 dietary strategies are effective in preventing CHD: substitute unsaturated fats (especially polyunsaturated fat) for saturated and <i>trans</i>-fats; increase consumption of omega-3 fatty acids from fish oil or plant sources; and</p>

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					large prospective cohort studies that reported disease outcomes and metabolic studies with established intermediate end points	Combining results of 4 prospective studies, pooled RR of CHD assoc with diff of 2% energy in TFA intake (assessed at baseline) 1.25 (95% CI, 1.11-1.40)	consume a diet high in fruits, vegetables, nuts and whole grains and low in refined grains....Evidence is now clear that diets including nonhydrogenated unsaturated fats as the predominant form of dietary fat, whole grains as the main form of carbohydrate, and an abundance of fruits and vegetables, and adequate omega-3 fatty acids can offer significant protection against CHD" <i>Reviewer's Comments:</i> <i>Review looks at all types of dietary advice and CHD; does not specifically address corn oil</i>

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Sacks and Katan, 2002	Re- view	R	Ø	<p>Purpose: To review RCTs that determined effects of diet on plasma lipoproteins and CVD and discuss effect of amt and type of dietary fats and CHO on plasma lipid risk factors, effects on CVD predicted by these effects on lipid risk factors and observed effects of dietary fats on CVD</p> <p>Inclusions: RCT; effects of dietary fats on plasma lipoprotein conc; effects of dietary TX on coronary death, MI, stroke and all-cause mortality</p> <p>Exclusions: Trials that combined dietary therapy with other TX; primarily used coronary arteriography rather than coronary death or MI as study end point</p>	<p>Study Protocol: Not given</p> <p>Data Collection: Not given</p>	<p>Outcome Measures: Plasma lipoproteins CVD</p> <p>Actual Sample: 4 high PUFA diet trials 2 low fat diet trials 1 Mediterranean diet trial 1 Indian vegetarian diet trial 3 fish/FO diet trials</p> <p>Results: 30% fat, 20% fat and Mediterranean diets all lower LDL, but low fat diets decr HDL proportionately more than they decr LDL, and they incr TG; TC:HDL incr on 30% or 20% fat diets but decr only on Mediterranean diet</p> <p>Predicted effects of dietary therapies on coronary disease: Men: 20% or 30% fat diet - no change in incidence High unsaturated fat diet - decr incidence by 19% Women: 20% or 30% fat diet - incr incidence High unsaturated fat diet - decr incidence by 16%</p>	<p>Author's Conclusions: "...several dietary approaches reduced coronary events in randomized clinical trials. Replacing saturated fat with polyunsaturated fat prevented coronary events in men. In men who had an acute myocardial infarction, a Mediterranean diet and fatty fish improved survival. None of these trials had much impact on total fat intake but rather increased vegetable oils, n-3 fatty acids, or many other plant foods or nutrients that are linked to coronary prevention. The reductions in rates of CVD caused by these dietary therapies compare favorably with drug treatments for coronary patients"</p> <p><i>Reviewer's</i></p>

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						<p>4 RCTs support relationship found in trials of drug therapy that coronary events reduced by 2% for every 1% reduction in TC</p> <p>Replacement of SFA with PUFA in diet reduced coronary event rates in 4 RCT (sig in 3 of them)</p> <p>Positive outcomes of RCTs suggest oxidizability of PUFAs may be less of factor in genesis of atherosclerosis and CAD in humans than previously thought</p> <p>TFA have worst effects on blood lipids among all dietary FA, even saturates</p> <p>Most favorable overall changes in plasma lipid conc produced by replacing SFA and <i>trans</i> unsaturated fat with unhydrogenated MUFA and PUFA</p>	Comments: <i>None</i>
Hu et al., 2001	Re- view	R	Ø	Purpose: To review evidence from epidemiologic studies and dietary intervention trials addressing relationship bet dietary fat intake and	Study Protocol: Not given Data Collection: Not given	Outcome Measures: CHD risk Actual Sample: Not given (143 references) Results: Sig positive assoc bet SFA intake	Author's Conclusions: "Although the ideal randomized trial of dietary fat and primary prevention of CHD in free-living populations has not been and may

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				<p>risk of CHD, with particular emphasis on diff major types of fat, n-3 FA and optimal balance bet n-3 and n-6 FA</p> <p>Inclusions: Not given</p> <p>Exclusions: Not given</p>		<p>and risk of CHD found in 2 studies, but not in 7 others</p> <p>Sig inverse assoc bet PUFA intake and CHD found in only 1 study, but not in 6 others</p> <p>Numerous metabolic studies show strong chol-lowering effects for veg oils rich in linoleic acid when substituted for dietary SFA</p> <p>Dietary intervention trials using high-PUFA diets more effective than those using low fat high CHO diets in lowering total serum chol as well as rates of CHD</p> <p>Results indicate substantial benefit to substituting PUFA (such as unhydrogenated soybean or corn oil) for TFA (such as hard margarine) in diet</p> <p>Ecological studies suggested inverse assoc bet MUFA intake and total mortality, as well as with CHD death</p> <p>Results of 2 RCTs indicate that right types of FA and other components of diet more imp than total amt of fat in reducing coronary risk</p>	<p>never be conducted due to feasibility concerns, the existing evidence powerfully supports the strategy of replacing saturated and trans fats with unhydrogenated unsaturated fats....Compelling evidence indicates the greater importance of types of fat than total amount of fat with respect to risk of CHD, although the optimal mixture of different fatty acids remains unsettled"</p> <p>Reviewer's Comments: <i>None</i></p>

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						<p>Dietary intervention trials in general support benefit of replacing SFA with PUFA</p> <p>Controlled clinical trials show that replacing SFA with PUFA more effective in lowering serum chol and reducing risk of CHD than simply reducing total fat consumption</p>	
Mock, 1959	Re- view	M	NA	<p>Purpose: To define etiology of coronary artery disease in Americans</p> <p>Inclusions: Not given</p> <p>Exclusions: Not given</p>	<p>Study Protocol: Not given</p> <p>Data Collection: Not given</p>	<p>Outcome Measures: Not given</p> <p>Actual Sample: Not given</p> <p>Results: No summary of results provided</p>	<p>Author's Conclusions: "While there appears, then, to be no general agreement as to the nature of the mechanism by which unsaturated fats lower cholesterol, or even the amounts necessary to do this, many products containing these and many other 'lipotropic factors' have been introduced into the market. While awaiting clarification of these issues, it is perhaps well to recall that no one has yet proven that the</p>

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							reduction of a patient's serum cholesterol will cause his arteriosclerosis to regress or will prevent future coronary occlusions" <i>Reviewer's Comments:</i> <i>None</i>

APPENDIX P2 PUFAR Review Table