

Medical Nutrition Therapy for Cardiovascular Disease

Farzad Shidfar

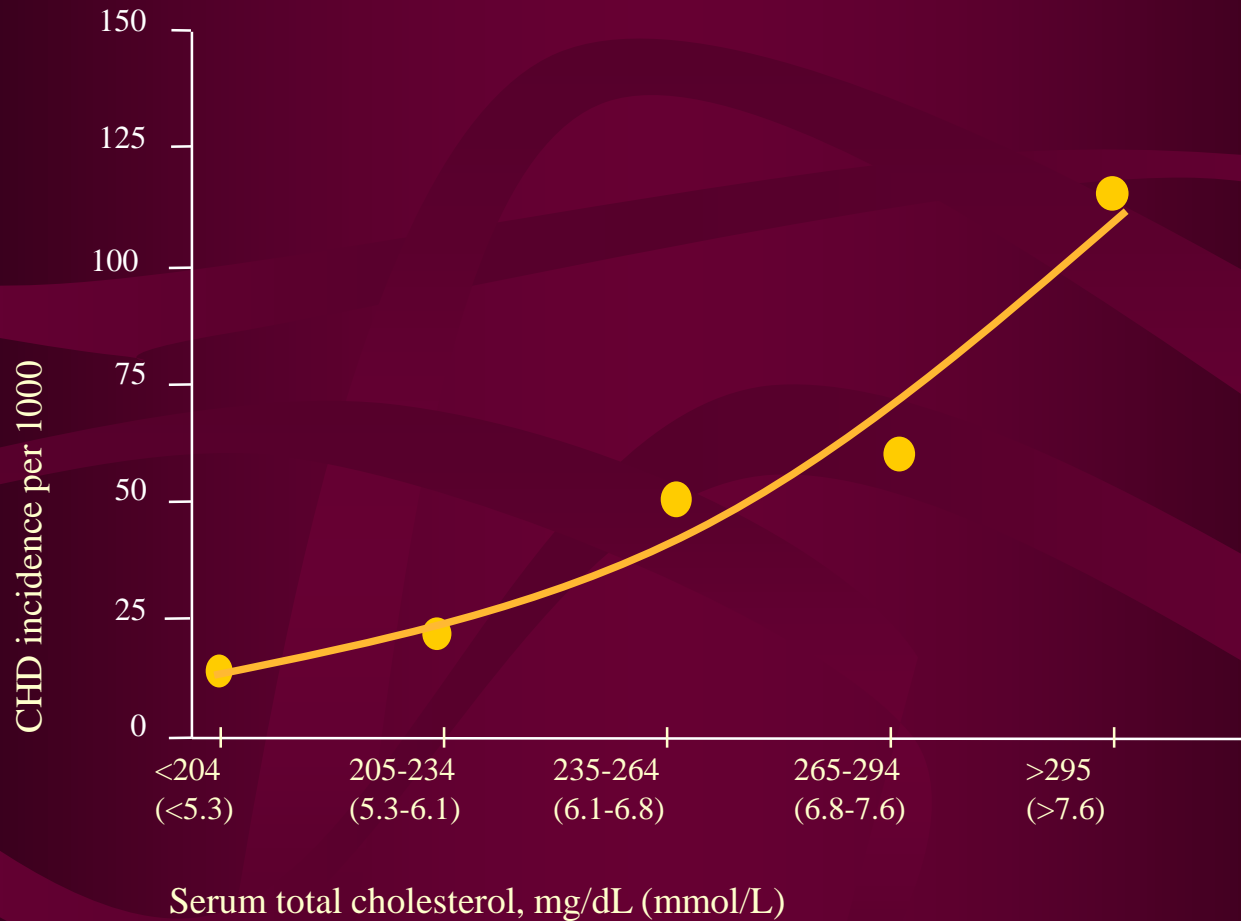


Review Article

Cardiovascular Disease in Iran in the Last 40 Years: Prevalence, Mortality, Morbidity, Challenges and Strategies for Cardiovascular Prevention

Four decades ago, Iran encountered rapid sociodemographic and economic transitions. This review was carried out to investigate the trend of cardiovascular disease (CVD) prevalence, mortality and morbidity, relevant challenges and suggestions for prevention of CVD. In Iran, the most prevalent causes of death have transited from infectious and diarrheal diseases in 1960 to CVD few decades ago. CVD was the first leading cause of mortality and a million disability adjusted life years (DALYs) led to 46% of all deaths and 20%-23% of the burden of disease in Iran. Ischemic heart disease and stroke are considered the first and second cause of death and DALYs in Iran, respectively. CVD rising epidemic might be related to socioeconomic and cultural changes, nutrition transition, inadequate physical activity, industrialization and urbanization and increasing life expectancy, increasing metabolic and physical risk factors, low accessibility and affordability to primary care and treatment, and low compliance because of economic and psychological problems. Thus, planning and implementing strategies for prevention and control of the disease and its risk factors are on top of the ministry of health agenda in the recent years. Health promotion strategies to prevent and control CVD risk factors, early detection of the disease and treatment of acute and chronic CVD events are essential elements for reducing the burden of CVD in Iran.

Relationship Between Cholesterol and CHD Risk: Framingham Study



Benefits of Cholesterol Lowering

Meta-analysis of 38 primary and secondary intervention trials

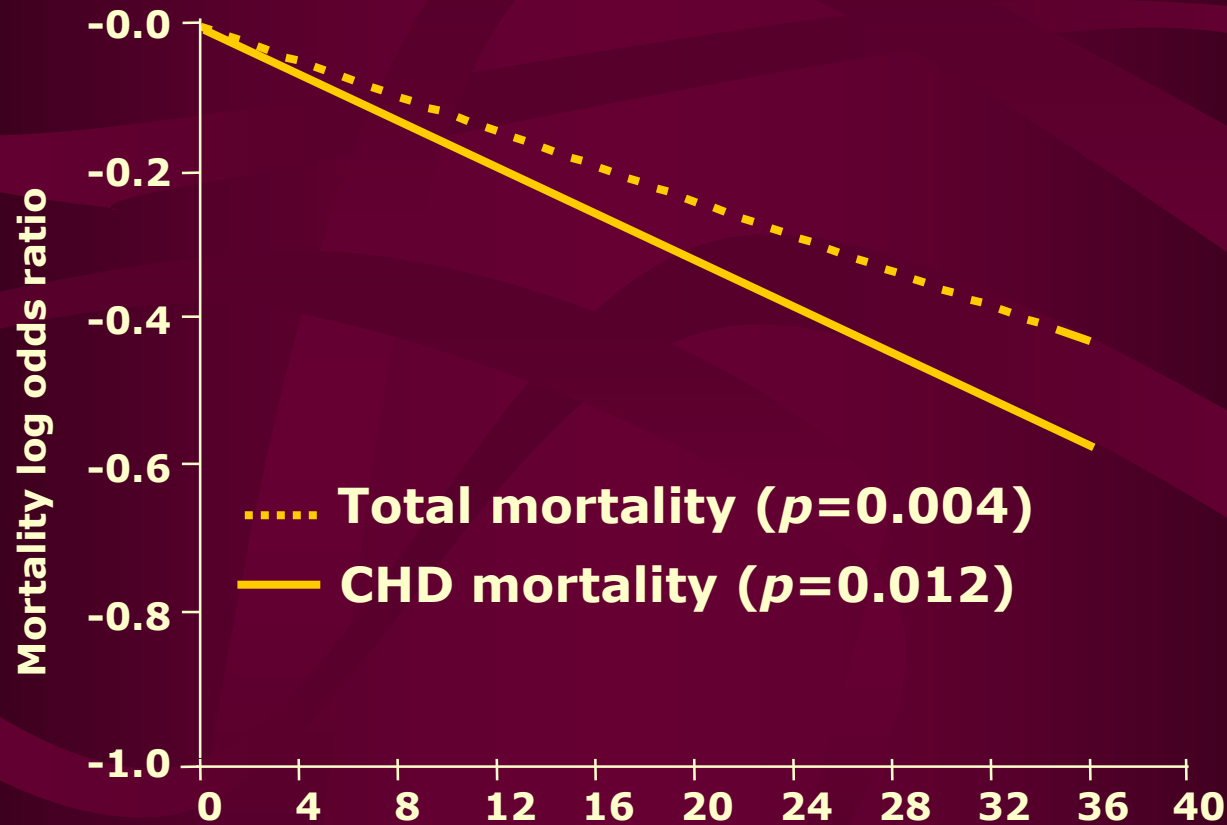


Table 1. Age standardized DALY rates per 100,000 in 1990, 2005, and 2015 for all causes in both sexes in 16 countries and the SDI category to which each country belongs.

	1990	2005	2015	SDI category
Afghanistan	96,500 (84,800 – 109,300)	88,700 (77,300 – 101,000)	82,000 (71,000 – 94,500)	Low SDI
Armenia	36,900 (34,000 – 40,100)	32,600 (29,800 – 35,700)	27,400 (24,600 – 30,500)	High-Middle SDI
Azerbaijan	46,900 (43,800 – 50,400)	39,700 (36,400 – 43,100)	30,800 (27,600 – 34,300)	High-Middle SDI
Bahrain	33,900 (30,100 – 38,000)	26,700 (23,500 – 30,300)	22,400 (18,900 – 26,100)	High-Middle SDI
Iran	47,200 (42,200 – 52,000)	33,400 (29,000 – 38,100)	28,400 (24,300 – 32,900)	High-Middle SDI
Iraq	47,500 (42,100 – 53,200)	47,300 (41,500 – 54,600)	43,400 (36,900 – 50,300)	Middle SDI
Kazakhstan	40,900 (38,000 – 44,300)	46,300 (43,200 – 49,600)	35,600 (32,100 – 39,300)	High-Middle SDI
Kuwait	25,300 (22,200 – 28,900)	23,300 (20,500 – 26,500)	20,000 (17,000 – 23,300)	High SDI
Oman	34,600 (29,400 – 40,200)	27,100 (23,700 – 30,600)	25,300 (21,500 – 29,100)	High-Middle SDI
Pakistan	56,100 (51,900 – 60,500)	55,500 (51,100 – 60,400)	46,700 (42,100 – 52,100)	Low-Middle SDI
Qatar	29,500 (26,100 – 33,000)	26,300 (22,800 – 29,900)	21,500 (18,100 – 25,500)	High-Middle SDI
Russia	38,400 (35,300 – 41,900)	47,000 (44,000 – 50,000)	34,600 (31,500 – 38,000)	High SDI
Saudi Arabia	31,200 (27,900 – 35,300)	24,000 (21,200 – 27,200)	21,500 (18,500 – 24,700)	High-Middle SDI
Turkey	42,600 (39,200 – 46,300)	27,200 (24,200 – 30,500)	22,700 (19,600 – 26,100)	High-Middle SDI
Turkmenistan	49,800 (46,300 – 53,800)	44,900 (41,200 – 49,000)	36,100 (32,800 – 39,700)	High-Middle SDI
United Arab Emirates	36,000 (30,700 – 41,600)	27,700 (24,300 – 31,300)	26,800 (22,500 – 31,900)	High SDI
Figures in parentheses show the 95% uncertainty intervals. DALY: disability-adjusted life years. SDI: socio-demographic index.				

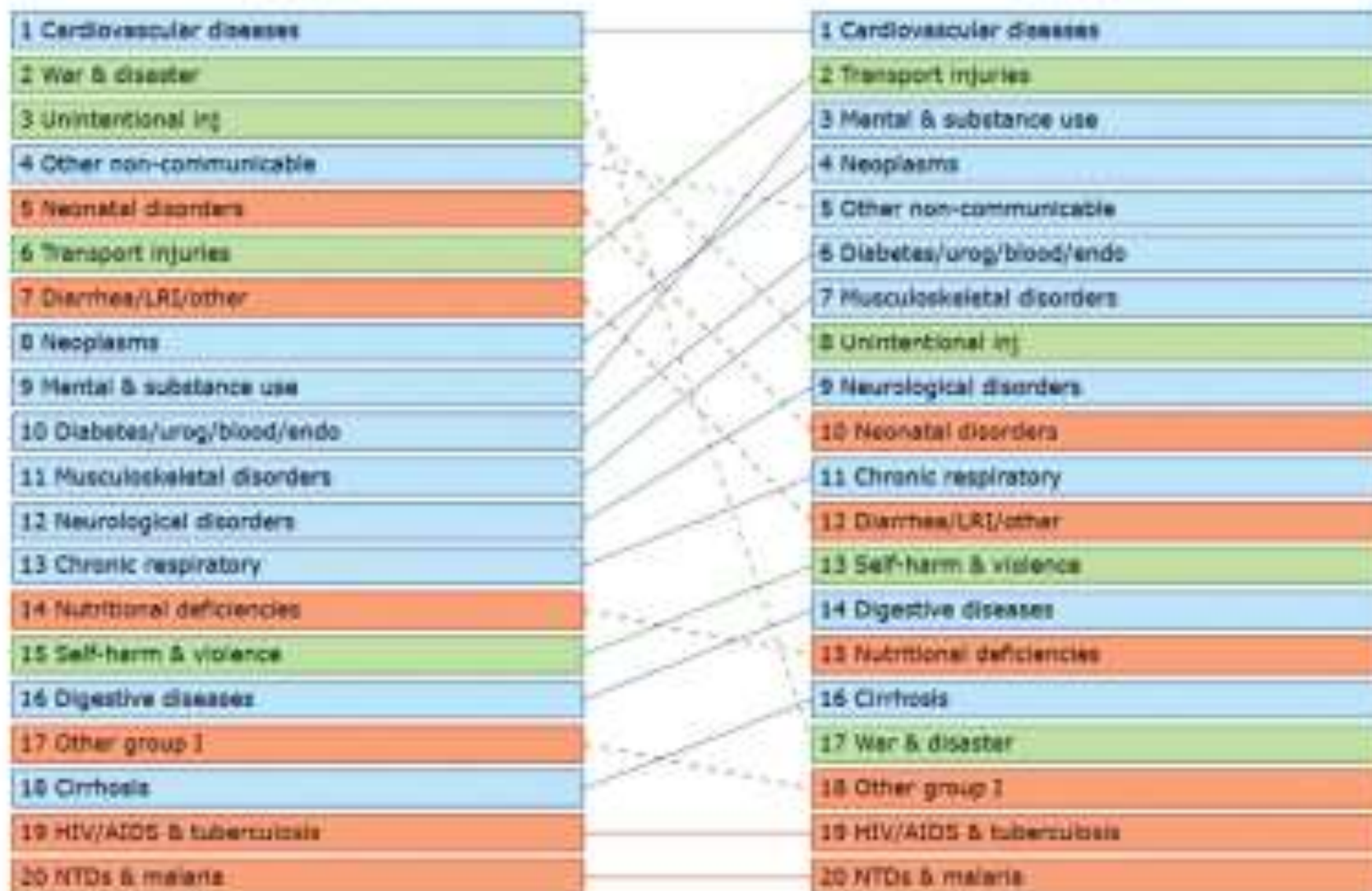
Disability-Adjusted Life-Years (DALYs) for 315 Diseases and Injuries and Healthy Life Expectancy (HALE) in Iran and its Neighboring Countries, 1990–2015: Findings from Global Burden of Disease Study 2015

Archives of Iranian Medicine, Volume 20, Number 7, July 2017 403

1990 rank

2015 rank

(A)



1990 rank

2015 rank

(B)

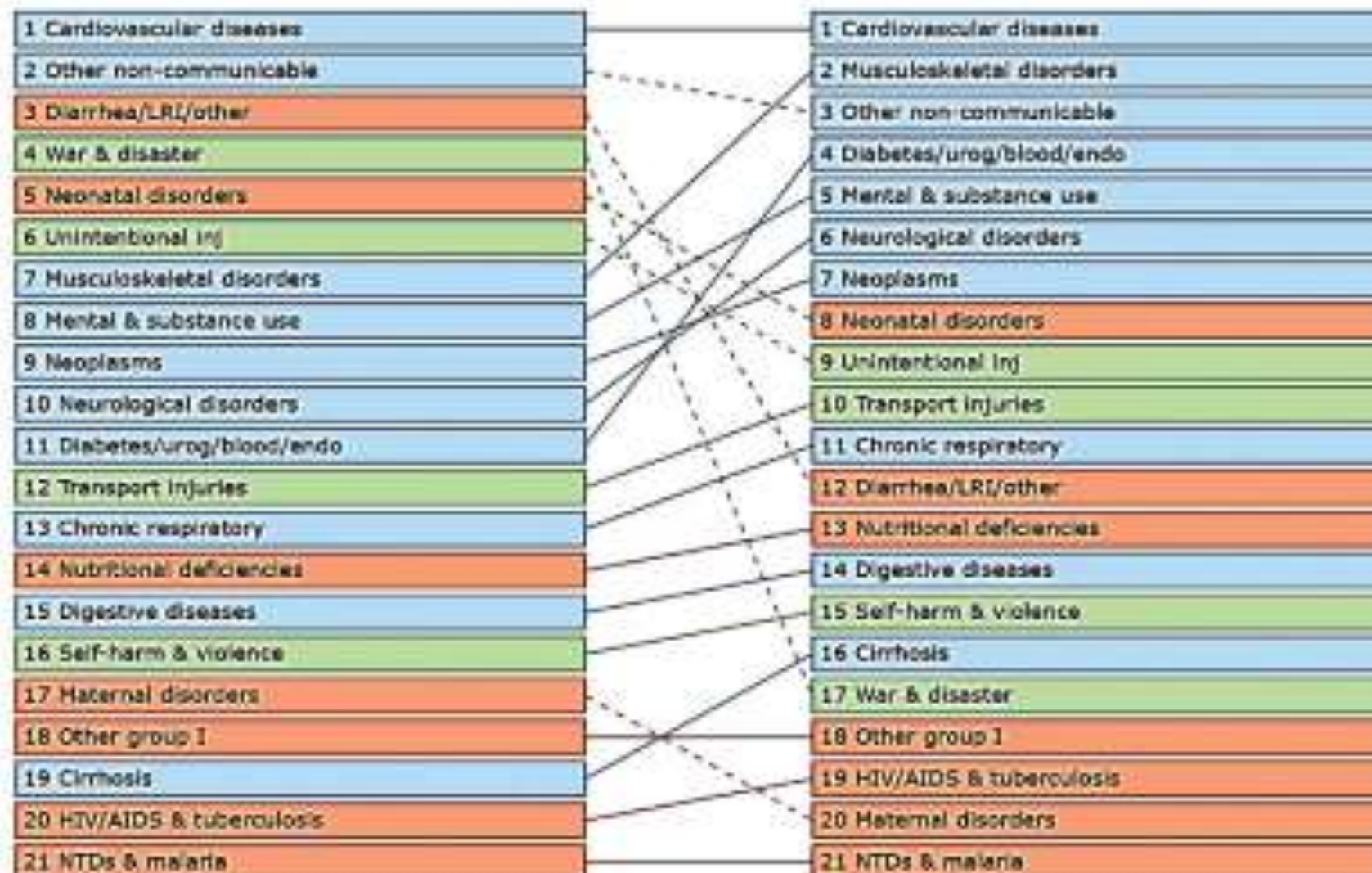


Figure 2. Leading causes of age standardized DALY rates per 100,000 in 1990 and 2015 in Iran. A) Men, B) Women. (Group 1 diseases are presented in red, non-communicable diseases in blue, and injuries in green).

LDL cholesterol

- **Strongly associated with atherosclerosis and CVD events**
- **10% increase results in an approximate 20% increase in CHD risk**
- **Most of the cholesterol in plasma is found in LDL particles**
- **Smaller denser LDL are more atherogenic than larger, less dense particles**
- **Risk associated with LDL-C is increased by other risk factors:**
 - **low HDL-C**
 - **smoking**
 - **hypertension**
 - **diabetes and the metabolic syndrome**

HDL cholesterol

- HDL-C has a protective effect for risk of atherosclerosis and CHD
- Epidemiological studies show the lower the HDL-C level, the higher the risk for atherosclerosis and CHD
 - low level (<40 mg/dL, 1 mmol/L) increases risk
- HDL-C tends to be low when triglycerides are high
- HDL-C is lowered by smoking, obesity and physical inactivity
- ApoA-I is the major apolipoprotein in HDL and an elevated ApoA-I is linked to reduced CVD risk

Cardiovascular Risk Factors

- Category I—cigarette smoking, LDL cholesterol, high-fat diet, hypertension
- Category II—diabetes mellitus, physical inactivity, HDL cholesterol, TG, obesity
- Category III—psychosocial factors, lipoprotein a, homocysteine
- Category IV—age, male gender, low socioeconomic status, family history

Assessing risk

- 10 year risk algorithm categorization:
 - 1- very high risk (more than 30% chance of developing CHD or have a recurrent event within 10 years.
 - 2-high risk (20-30% chance of new CHD or have a recurrent event within 10 years)
 - 3-low risk (less than a 10% risk

NCEP/Framingham risk scores: Estimate of 10-yr Hard CHD risk in men without CHD

Age (y)	20–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79
Points	–9	–4	0	3	6	8	10	11	12	13

Total-C (mg/dL)	Points				
	20–39	40–49	Age (y) 50–59	60–69	70–79
<160	0	0	0	0	0
160–199	4	3	2	1	0
200–239	7	5	3	1	0
240–279	9	6	4	2	1
? 280	11	8	5	3	1

HDL-C (mg/dL)	Points
? 60	–1
50–59	0
40–49	1
<40	2

Systolic BP (mm Hg)	Points	
	Untreated	Treated
<120	0	0
120–129	0	1
130–139	1	2
140–159	1	2
? 160	2	3

Point total:	<0	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	>17
10-yr risk (%)	<1	1	1	1	1	1	2	2	3	4	5	6	8	10	12	16	20	25	? 30

Therapeutic Lifestyle Changes in LDL-Lowering Therapy: Major Features

- Saturated fats <7% of total calories
- Dietary cholesterol <200 mg per day
- Plant stanols/sterols (2 g per day)
- Viscous (soluble) fiber (10–25 g per day)
- Weight reduction
- Increased physical activity

Therapeutic Lifestyle Changes

Nutrient Composition of TLC Diet

<u>Nutrient</u>	<u>Recommended Intake</u>
• Saturated fat	Less than 7% of total calories
• Polyunsaturated fat	Up to 10% of total calories
• Monounsaturated fat	Up to 20% of total calories
• Total fat	25–35% of total calories
• Carbohydrate	50–60% of total calories
• Fiber	25–30 grams per day
• Protein	Approximately 15% of total calories
• Cholesterol	Less than 200 mg/day
• Total calories (energy) expenditure	Balance energy intake and to maintain desirable body weight/ prevent weight gain

NCEP ATP III Guidelines

Patients with	Initiate TLC* if LDL - C	Drug therapy considered if LDL - C	LDL - C treatment goal
0 - 1 risk factors	≥ 160 mg/dL [†]	≥ 190 mg/dL (160 - 189 mg/dL: drug optional)	< 160 mg/dL [†]
≥ 2 risk factors (10 - year risk $\leq 20\%$)	≥ 130 mg/dL [†]	10-yr risk 10-20%: ≥ 130 mg/dL 10-yr risk $< 10\%$: ≥ 160 mg/dL	< 130 mg/dL [†]
CHD and CHD risk equivalents (10 - year risk $> 20\%$)	≥ 100 mg/dL [†]	≥ 130 mg/dL (100 - 129 mg/dL: drug optional)	< 100 mg/dL [†]

[†] 100 mg/dL = 2.6 mmol/L; 130 mg/dL = 3.4 mmol/L; 160 mg/dL = 4.1 mmol/L; 190 mg/dL = 5 mmol/L

* TLC: therapeutic lifestyle changes

Summary Statin Initiation Recommendations to

Heart-healthy lifestyle habits are the foundation of ASCVD prevention
(See 2013 AHA/ACC Lifestyle Management Guideline)

Age ≥ 21 y and a candidate for statin therapy

Yes

Clinical
ASCVD

Yes

Age ≤ 75 y
High-intensity statin
(Moderate-intensity statin if not candidate for high-intensity statin)

Yes

Age > 75 y **OR** if not candidate for high-intensity statin
Moderate-intensity statin

No

Definitions of High- and Moderate-Intensity Statin Therapy*

(See Table 5)

High

Daily dose lowers LDL-C by approx. $\geq 50\%$

Moderate

Daily dose lowers LDL-C by approx. 30% to $< 50\%$

LDL-C ≥ 190 mg/dL

Yes

High-intensity statin
(Moderate-intensity statin if not candidate for high-intensity statin)

No

Regularly monitor adherence to lifestyle and drug therapy with lipid and safety assessments
(See Fig 5)

Diabetes
LDL-C 70-189 mg/dL
Age 40-75 y

Yes

Moderate-intensity statin

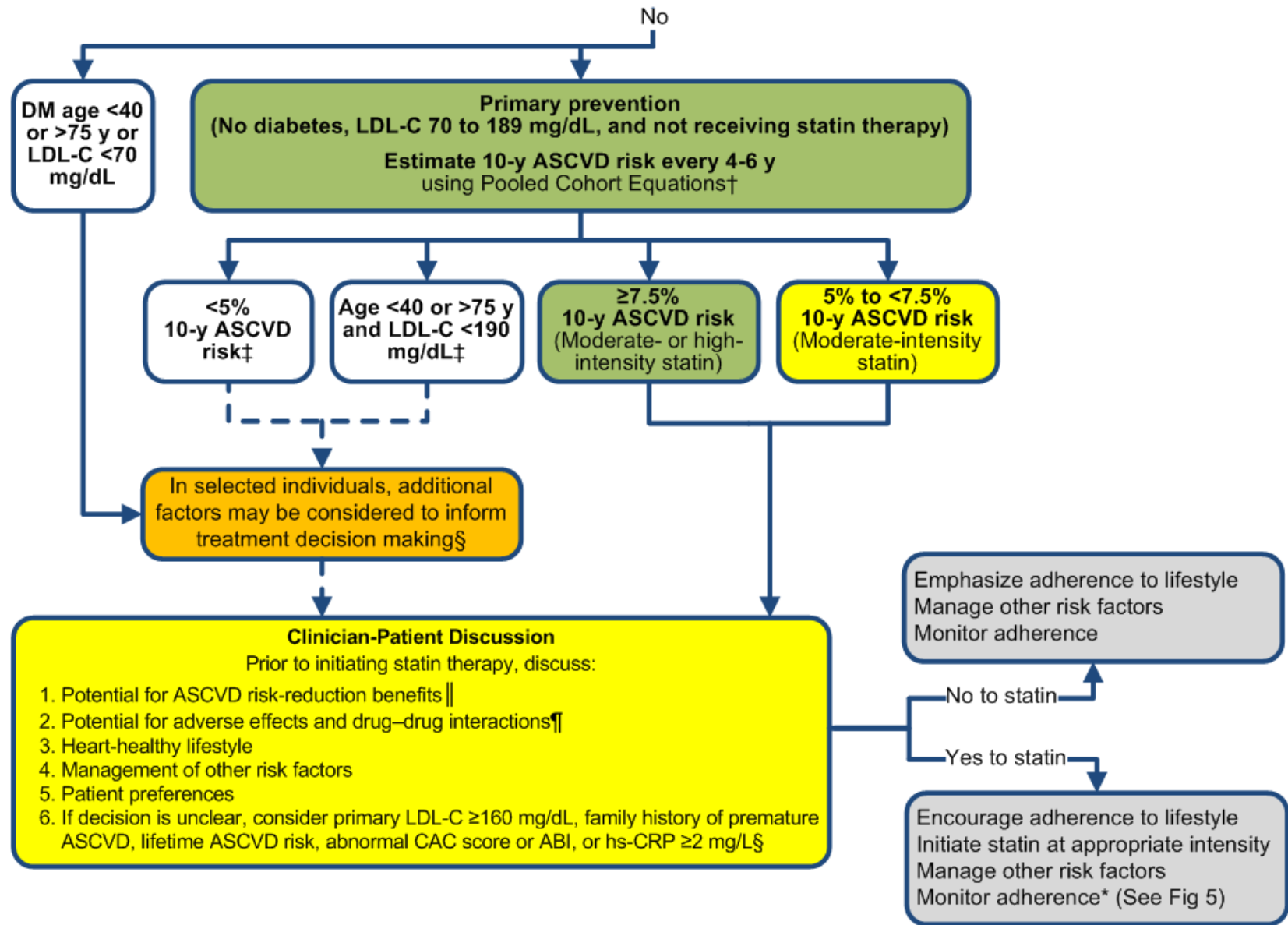
Yes

Estimated 10-y ASCVD risk $\geq 7.5\%$ †
High-intensity statin

No



Summary of Statin Initiation Recommendations to Reduce ASCVD Risk (Revised Figure)



Intensity of Statin Therapy

Table 5. High- Moderate- and Low-Intensity Statin Therapy (Used in the RCTs reviewed by the Expert Panel)*

High-Intensity Statin Therapy	Moderate-Intensity Statin Therapy	Low-Intensity Statin Therapy
Daily dose lowers LDL-C on average, by approximately $\geq 50\%$	Daily dose lowers LDL-C on average, by approximately 30% to $<50\%$	Daily dose lowers LDL-C on average, by $<30\%$
Atorvastatin (40[†])–80 mg Rosuvastatin 20 (40) mg	Atorvastatin 10 (20) mg Rosuvastatin (5) 10 mg Simvastatin 20–40 mg[‡] Pravastatin 40 (80) mg Lovastatin 40 mg <i>Fluvastatin XL 80 mg</i> Fluvastatin 40 mg bid <i>Pitavastatin 2–4 mg</i>	<i>Simvastatin 10 mg</i> Pravastatin 10–20 mg Lovastatin 20 mg <i>Fluvastatin 20–40 mg</i> <i>Pitavastatin 1 mg</i>

*Individual responses to statin therapy varied in the RCTs and should be expected to vary in clinical practice. There might be a biologic basis for a less-than-average response.

[†]Evidence from 1 RCT only: down-titration if unable to tolerate atorvastatin 80 mg in IDEAL (Pedersen et al).

[‡]Although simvastatin 80 mg was evaluated in RCTs, initiation of simvastatin 80 mg or titration to 80 mg is not recommended by the FDA due to the increased risk of myopathy, including rhabdomyolysis.

Dietary Factors

- Saturated Fatty Acids
- Omega-6 Polyunsaturated Fatty Acids
- Omega-3 Polyunsaturated Fatty Acids
- Monounsaturated Fatty Acids
- Trans Fatty Acids
- Total Fat
- Dietary Cholesterol
- Fiber
- Alcohol
- Coffee
- Antioxidants
- Soy Protein/Stanoles

Saturated fatty acids

- When substituted for CHO or other fatty acids: elevates both LDL-C and HDL-C
- Dose-response between SFA and LDL-C
- Myristic acid > palmitic acid > lauric acid
- 1% increase in total energy from SFA



2.7 mg/dl increase in plasma Chol

- Myristic acid sources: butterfat , coconut oils and palm kernel oil
- Palmitic acid sources: animal foods
- Lauric acid sources: palm kernel and coconut oils

Public health guidelines should recommend reducing saturated fat consumption as much as possible: YES

Isocaloric replacement of 5% of energy from SFA with PUFA, MUFA from plant sources, or carbohydrates from whole grains was associated with a 25%, 15%, and 9% lower risk of coronary heart disease (CHD), respectively (PUFA—HR: 0.75; 95% CI: 0.67, 0.84; $P < 0.0001$; MUFA—HR: 0.85; 95% CI: 0.74, 0.97; $P = 0.02$; carbohydrates from whole grains—HR: 0.91; 95% CI: 0.85, 0.98; $P = 0.01$).

TABLE 1 Recommendations for saturated fat and evidence ratings from the AHA/ACC, National Lipid Association, and 2010 and 2015 Dietary Guidelines Advisory Committees¹

Recommendation	Evidence rating	
	ACC/AHA COR	ACC/AHA LOE
2019 AHA/ACC Guideline on the Primary Prevention of Cardiovascular Disease		
• Replacement of saturated fat with dietary monounsaturated and polyunsaturated fats can be beneficial to reduce ASCVD risk	IIa ²	B-NR ³
2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk		
• Aim for a dietary pattern that achieves 5% to 6% of calories from SFA	I ²	A ³
• Reduce percent of calories from SFA	I ²	A ³
2015 National Lipid Association Recommendations for Patient-Centered Management of Dyslipidemia: part 2	Grade	Strength of recommendation
• Dietary SFA may be partially replaced with unsaturated fats (MUFA and PUFA), as well as proteins, to reach a goal of <7% of energy from SFA	A ⁴	Moderate ⁵
Scientific Report of the 2015 Dietary Guidelines Advisory Committee	DGAC evidence grade	
• Strong and consistent evidence from RCTs and statistical modeling in prospective cohort studies shows that replacing SFA with PUFA reduces the risk of CVD events and coronary mortality	Strong	
Scientific Report of the 2010 Dietary Guidelines Advisory Committee	Scientific evidence	
• Strong evidence indicates that intake of dietary SFA is positively associated with intermediate markers and endpoint health outcomes for 2 distinct metabolic pathways: 1) increased serum total and LDL cholesterol and increased risk of CVD and 2) increased markers of insulin resistance and increased risk of type 2 diabetes	Strong	

¹ACC, American College of Cardiology; AHA, American Heart Association; ASCVD, atherosclerotic cardiovascular disease; COR, Classification of Recommendation; CVD, cardiovascular disease; DGAC, Dietary Guidelines Advisory Committee; LOE, Level of Evidence; RCT, randomized controlled trial.

²COR—class I (strong): benefit >>> risk; class IIa (moderate): benefit >> risk.

³LOE—level A: high-quality evidence; level B-NR (nonrandomized): moderate-quality evidence.

⁴Strength of recommendation—grade A: strong.

⁵Quality of evidence—moderate. RCTs with minor limitations.

2021 ESC Guidelines on cardiovascular disease prevention in clinical practice

Risk of CHD is reduced when dietary saturated fats are replaced appropriately (Figure 10). This is also the case when replacing meat and dairy foods.^{406,407} Polyunsaturated fats (-25%), monounsaturated fats (-15%), and to a lesser extent carbohydrates from whole grains (-9%), were all associated with reduced CHD risk when isocalorically substituted for dietary saturated fat.^{408,409}

Reducing saturated fatty acid intake to less than 10% of energy may have additional benefits.⁴⁰⁵ However, the LDL-C-lowering effect of substituting polyunsaturated fatty acids (PUFAs) for saturated fatty acids may be less in obese (5.3%) than in normal-weight persons (9.7%).⁴²¹

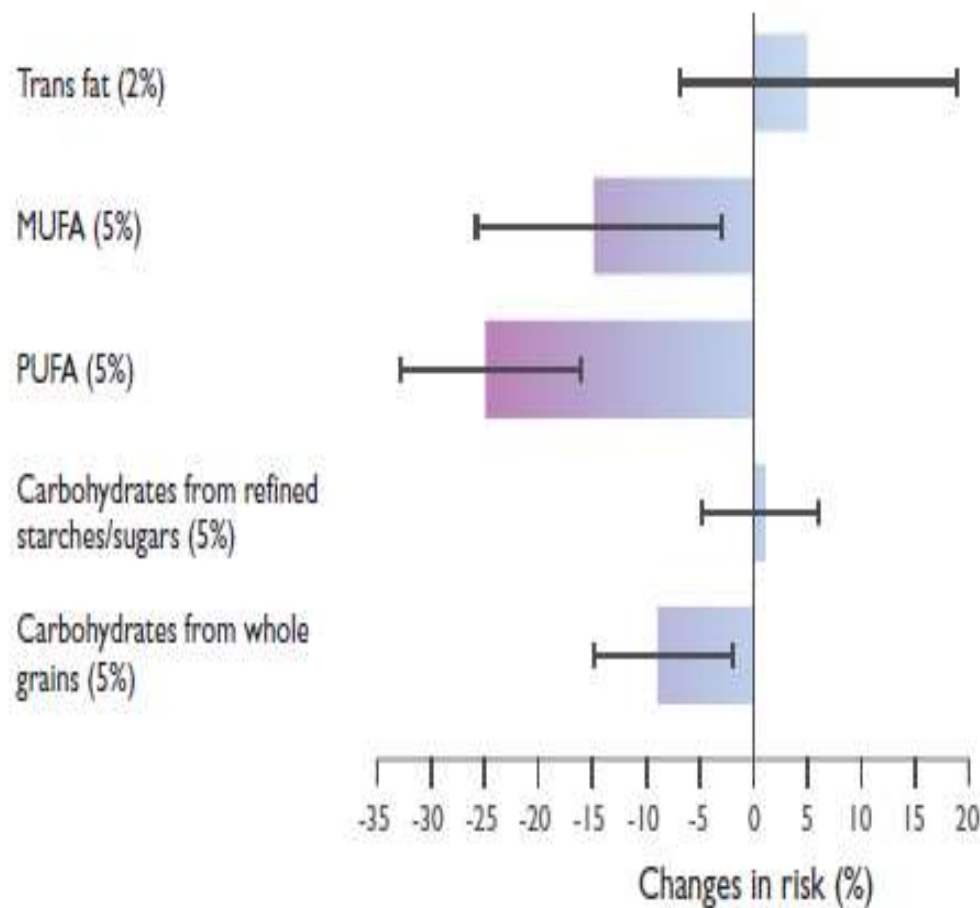


Figure 10 Estimated percentage change in risk of coronary heart disease associated with isocaloric substitutions of saturated fat for other types of fat or carbohydrates. Reproduced from Sacks *et al.*⁴⁰⁹ MUFA = monounsaturated fatty acid; PUFA = polyunsaturated fatty acid.



Review

The Short Overview on the Relevance of Fatty Acids for Human Cardiovascular Disorders

The American Heart Association/American College of Cardiology guideline has recommended to decrease intake of saturated FA (SFA) to 5% to 6% of total daily energy (calorie) intake to reduce the risk of CVD.

The scientific rationale for decreasing SFA in the diet has been and remains based on well-established effects to raise low-density lipoprotein (LDL) cholesterol, along with a reduction in non-high density lipoprotein (HDL) cholesterol, a leading causes of atherosclerosis

stearic SFA does not have a significant influence on lipid metabolism.

Stearic acid induces apoptosis and necrosis of endothelial cells more strongly than does palmitic or myristic FA. A hypothesis that intracellular accumulation of stearic FA can be proinflammatory and lipotoxic.

A change in the proportions of the ingested-with-food FA that affect the ratio of HDL cholesterol to LDL cholesterol may be more important than the simple limiting of SFA, at least myristic and stearic ones, both of which influence the HDL cholesterol level.

Table 4. The influence of fatty acids on the cardiovascular system.

Fatty Acids	The Influence on the Cardiovascular System	Effect of Increasing the Level	Effect of Reducing the Level
Myristic acid (C14:0)	-	↑ the risk of developing IHD; ↑ total cholesterol concentration [32,39]	
Palmitic acid (C16:0)	-	↑ production of pro-inflammatory cytokines and oxidants; promotes inflammation and the development of CVD [31,32,34–37]	↓ LDL, glucose, arterial blood pressure normalizes [30]
Stearic acid (C18:0)	±	not correlate with a higher risk of IHD and MI; induces apoptosis and necrosis of endothelial cells; ↑ the risk of developing IHD [27,44–48]	does not have a significant influence on lipid metabolism [10,41]
Palmitoleic acid (C16:1)	±	↓ cholesterol and TG concentrations; ↑ HDL cholesterol concentrations [56,57]; ↑ the risk of developing IHD [32]	
Oleic acid (C18:1)	±	can improve the blood lipid profile [65]; ↓ the risk of atherosclerosis [69]; ↑ TG concentrations; ↑ markers of inflammation; ↑ the risk of developing heart failure [36,71]	
Linoleic acid (C18:2)	+	↓ risk developing IHD and death from IHD [140,141]; does not affect the concentrations of inflammatory biomarkers [146,147]	↑ risk of arterial hypertension [154–156]

α -Linolenic acid (C18:3)	+	↓ the risk of death from IHD; ↓ the levels of LDL and total cholesterol; ↑ the levels of HDL [80–82];	↑ development of IHD [88]
γ -Linolenic acid (C18:3)	+	↓ concentrations of TG, total cholesterol and LDL; ↑ of the HDL concentrations [145]; ↓ development of arterial hypertension [146]	↑ TG concentrations [144]
Arachidonic acid (C20:4)	-	↑ of biomarkers of inflammation; risk of CVD [132–134]; ↑ risk of arterial hypertension [153–156]; ↑ the risk of atherosclerosis [151,152];	
Eicosapentaenoic acid (C20:5)	+	can improve the blood lipid profile [90,107]; ↓ vascular inflammation; ↓ risk developing of major ischemic events, and death from IHD [91,107]	↑ risk developing of IHD [124]
Docosapentaenoic acid (C22:5)	+	↓ prevalence of CVD; ↓ developing of congestive heart [119,120]	↑ developing of plaques enriched with lipids; promote the frequency of unstable plaque formation leading to the development of acute coronary syndrome and MI [121]; ↑ risk developing of IHD [124]
Docosahexaenoic acid (C22:6)	+	↓ concentrations of TG; ↑ the HDL concentrations [109]; ↓ inflammation markers [110,111]	↑ endothelial dysfunction [112,113]; ↑ risk developing of IHD [124]

«-»—the adverse effect, «+»—the beneficial effect, «±»—the questionable impact, «↓»—the decrease, «↑»—the increase, IHD—ischemic heart disease, CVD—cardiovascular disease, LDL—low-density lipoproteins, HDL—high-density lipoprotein cholesterol, MI—myocardial infarction, TG—triglyceride.

Omega-6 fatty acids

- When substitute for CHO: LDL ↓ HDL ↑

- When substitute for SFA in a low fat diet:

LDL ↓ HDL ↓

Replacement of saturated fat 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.

Eliminating SFA is twice as effective in lowering Chol levels as is increasing PUFA

1% increase in omega -6 → lower CHOL by 1.4 mg/dl , lower VLDL, apoB, and HDL

ω -6 PUFA exert pro-inflammatory properties.

Increasing ω -6 PUFA reduces serum total cholesterol over at least one year, but not other lipid fractions in the blood.

According to the Polish Forum of Cardiovascular Disease Prophylactic Program, the proportion of ω -6 to ω -3 acids should be 4:1

Omega-3 fatty acids

- EPA and DHA associated with a decreased CVD risk.
- High omega-3 fish (salmon, tuna, mackerel, sardines) twice a week or 1 g EPA and DHA supplements
- Hyper TG patients need 2 to 4 g of EPA and DHA per day for effective lowering .



ELSEVIER

Available online at www.sciencedirect.com

Nutrition, Metabolism & Cardiovascular Diseases

journal homepage: www.elsevier.com/locate/nmcd

REVIEW

Impact of omega-3 polyunsaturated fatty acids on vascular function and blood pressure: Relevance for cardiovascular outcomes

**Table 1** Effects of omega-3 polyunsaturated fatty acids on blood vessels.**1. Endothelium and vascular smooth muscle cells (VSMC)**

- a. Increased nitric oxide availability
- b. Anti-oxidant activity
- c. Decreased endothelin-1
- d. Stimulation of endothelial progenitor cells
- e. Generation of epoxides
- f. Direct VSMC relaxation
- g. Production of adiponectin from perivascular adipocytes
- h. Flow-mediated vasodilatation

2. Inflammation and thrombosis

- a. Reduction of pro-inflammatory and prothrombotic eicosanoids
- b. Increased production of SPM (resolvins, protectins, and maresins)
- c. Reduction of proinflammatory cytokines (IL-1, IL-2, and TNF- α) and adhesion molecules
- d. Downregulation of AP-1 and NF κ B
- e. Inhibition of inflammasomes

3. Plaque formation and stability

- a. Reduced carotid intima-media thickness
- b. Thickening of plaque fibrous cap
- c. Inhibition of metalloproteinases activity
- d. Increased HDL-cholesterol
- e. Decreased lipoprotein(a)

4. Arterial stiffness

- a. Decreased pulse wave velocity
- b. Reduction of brachial artery diameter

Each statement is explained in text together with the related references.

Abbreviations: SPM, specialized proresolving mediators; IL, interleukine; TNF, tumor necrosis factor; AP1, activator protein-1; NF κ B, nuclear factor κ -light-chain-enhancer of activated B-cells; HDL, high-density lipoprotein.



Review

Cardioprotective mechanism of omega-3 polyunsaturated fatty acids



Omega-3 polyunsaturated fatty acids (PUFAs), such as eicosapentaenoic acid and docosahexaenoic acid, are widely regarded as cardioprotective. Several large-scale, randomized clinical trials have shown that dietary intake of omega-3 PUFAs improves the prognosis of patients with symptomatic heart failure or recent myocardial infarction. Therefore, dietary consumption of omega-3 PUFA is recommended in international guidelines for the general population to prevent the occurrence of cardiovascular diseases (CVDs). However, the precise mechanisms underlying the cardioprotective effects of omega-3 PUFAs are not fully understood. Omega-3 PUFAs can be incorporated into the phospholipid bilayer of cell membranes and can affect membrane fluidity, lipid microdomain formation, and signaling across membranes. Omega-3 PUFAs also modulate the function of membrane ion channels, such as Na and L-type Ca channels, to prevent lethal arrhythmias. Moreover, omega-3 PUFAs also prevent the conversion of arachidonic acid into pro-inflammatory eicosanoids by serving as an alternative substrate for cyclooxygenase or lipoxygenase, resulting in the production of less potent products. In addition, a number of enzymatically oxygenated metabolites derived from omega-3 PUFAs were recently identified as anti-inflammatory mediators. These omega-3 metabolites may contribute to the beneficial effects against CVDs that are attributed to omega-3 PUFAs.

Benefits of fish oil supplementation

- In the Diet and Reinfarction Trial (DART) in 2033 men with CHD increased intake of fish or use of 2 fish oil caps/day reduced CHD mortality 29% over 2 years
- In GISSI 11324 men and woman with CHD use of 1 gr. of n-3 PUFA decreased CVD events including mortality 15%

Omega-3 sources

➤ Plant sources

Flax seed oil

Canola oil

Soy bean oil

Walnut , butternuts

Red and black currant seed

➤ Animal sources

- Fish special fatty fish such as salmon , sardines ,herring and mackerel

Dietary calcium intake and risk of cardiovascular disease, stroke, and fracture in a population with low calcium intake

Sung Hye Kong,¹ Jung Hee Kim,¹ A Ram Hong,¹ Nam H Cho,² and Chan Soo Shin¹

Background: The role of dietary calcium intake in cardiovascular disease (CVD), stroke, and fracture is controversial. Most previous reports have evaluated populations with high calcium intake.

Objective: We aimed to evaluate whether high dietary calcium intake was associated with the risk of CVD, stroke, and fracture in a population with low calcium intake.

Design: In a prospective cohort study beginning in 2001 in Ansung-Ansan, Korea, 2158 men and 2153 women aged >50 y were evaluated for all-cause mortality, CVD, stroke, and fractures over a median 9-y follow-up.

Results: During follow-up, 242 and 100 deaths, 149 and 150 CVD events, 58 and 82 stroke events, and 211 and 292 incident fractures occurred in men and women, respectively. The first quartiles of energy-adjusted dietary calcium intake were 249 mg/d (IQR: 169 mg/d) in men and 209 mg/d (IQR: 161 mg/d) in women. Both men and women with higher dietary calcium intake tended to have higher fat, protein, sodium, phosphorus, fruit, and vegetable intakes. In men, outcomes were not significantly associated with dietary calcium intake with or without adjustments, and CVD risk tended to increase with increasing energy-adjusted dietary calcium intake, but this was not statistically significant ($P = 0.078$ and $P = 0.093$ with and without adjustment, respectively). In women, CVD risk and dietary calcium intake showed a U-shaped association; the HRs (95% CIs) without adjustment relative to the first quartile were 0.71 (0.47, 1.07), 0.57 (0.36, 0.88), and 0.52 (0.33, 0.83) for quartiles 2, 3, and 4, respectively, and the values after adjustment were 0.70 (0.45, 1.07), 0.51 (0.31, 0.81), and 0.49 (0.29, 0.83) for quartiles 2, 3, and 4, respectively.

Conclusion: In Korean women, increased dietary calcium intake was associated with a decreased CVD risk, but it did not influence the risk of stroke or fracture.

Am J Clin Nutr doi:

<https://doi.org/10.3945/ajcn.116.148171>.

Omega 9:

Oleic acid substituting for CHO : no appreciable effect on blood lipids, but replacing SFA with MUFA lowers TC, LDL-c and TG (same extent as PUFA)

MUFA (> 15 % kcal) , TF (> 35 % kcal) : HDL-c does not change or increase slightly compared with levels with a lower-fat diet.

Main acceptor of reactive oxygen species (ROS) in models of oxidative stress. The strongest oxidants for oleic FA are the superoxide anion-radical, nitrogen dioxide, and ozone.

Oleic acid can improve the blood lipid profile, maintain healthy body weight , and even prevent palmitic-SFA-promoted mitochondrial dysfunction, insulin resistance, and inflammation-related signaling in neuronal cells and skeletal muscle

Enrichment of LDL particles with oleic acid can shorten the stay of LDL particles in the artery wall and consequently may lower the risk of atherosclerosis

- AHA recommends no more than 1% of calories from trans fatty acids (about 1-3 g per day).

Dietary Chol.

Raises total Chol & LDL-c but to a lesser extent than SFA.

**25 mg increase in dietary cholesterol
increases 1mg/dl cholesterol**

Dietary Chol.

- **cholesterol response to dietary cholesterol.**
- **threshold for a plasma**
when Chol intakes reach 500 mg/day ,
only small increments in blood Chol
occur.
- **hypo responder and hyper responder**

Dietary Chol.

- **Dietary saturated fatty acids and Chol :
Synergistic on LDL-C**

**decrease LDL receptor synthesis and
activity, Chylomicron size**

Dietary Fiber

- Soluble fibers lower LDL –c
- The quantity of fiber needed to produce the lipid lowering effect varies by food sources; higher legumes are needed than of pectin or gums.

Quantity of Soluble Fiber Needed Daily to Produce Lipid-Lowering Effect

- Pectin: 6 to 40 g
- Gums: 8 to 36 g
- Dried beans or legumes: 100 to 150 g
- Dry oat bran: 25 to 100 g
- Oatmeal: 57 to 140 g
- Psyllium: 10 to 30 g

Dietary Fiber

- Proposed mechanisms:

- 1- bind to bile acids

- 2-fermentation of fiber by bacteria in the colon, produces acetate, propionate and butyrate, which inhibits Chol. synthesis.

Nuts, Soy, Phytosterols, Garlic

- Nurses' Health Study: five 1oz servings of nuts per week associated with 40% lower risk of CHD events
- Metaanalysis of 38 trials of soy protein showed 47g intake lowered total, LDL-C, and trigls 9%, 13%, and 11%
- Phytosterol-supplemented foods (e.g., stanol ester margarine) lowers LDL-C avg. 10%
- Meta-analysis of garlic studies showed 9% total cholesterol reduction (1/2-1 clove daily for 6 months).

ATP III

Dietary Component	Dietary Change	Approx. % LDL ↓
Saturated Fatty Acids	<7% kcal	8-10
Cholesterol	<200mg/d	3-5
Weight Reduction	Lose 10 pounds	5-8
Soluble Fibre	5-10g/d	3-5
Plant Sterols	2g/d	6-15

Cumulative Potential Effect	20-30
------------------------------------	--------------

Living Under the Umbrella of Good Cardiovascular Health



Physician Nutrition Check List for Heart Health → “The 5 F’s”



Fruits & Vegetables



Unsaturated Fats

Fish

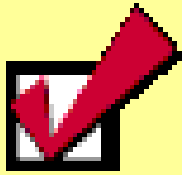


Fibre



Food Portions

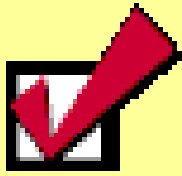




Fruits and Vegetables

- Current recommendations are to aim for 7-10 fruits and vegetable servings everyday

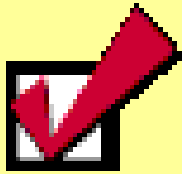




Unsaturated Fats

- Limit red meat to < 1 x per week
- Choose a soft non-hydrogenated margarine
- Choose low fat dairy products 1% M.F. or less for milk, yogurt and cottage cheese, <20% M.F. for cheese
- Choose olive and canola oil for cooking
- Snack on a ¼ cup of nuts daily

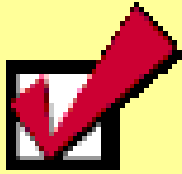




Fish

- Aim for 2-3 meals of fish per week
- Salmon and trout are high in omega-3 fats





Fibre

- Recommendations for daily Fibre intake:

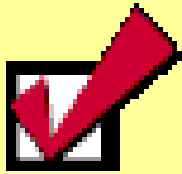
Men: 30-38 grams per day

Women: 21-27 grams per day

Soluble Fibre recommendation: 10-15grams per day

- Sources include: psyllium, oatbran, oatmeal, beans, lentils, some fruits and vegetables





Food Portions

- Eat frequently throughout the day so it is easier to control food portions
- Measure foods at home and become familiar with recommended portion sizes



Get a **HAND**le on Heart Healthy Nutrition

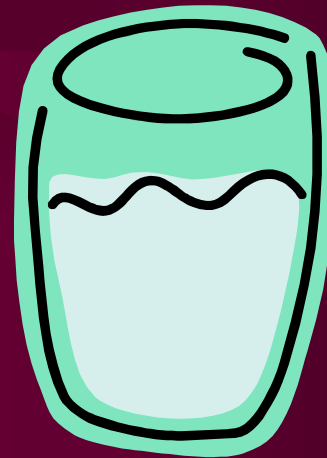


Fruits & Vegetables



Nutrition Management of CHF

- Low Salt Diet:
2400mg sodium per day
- Fluid restriction:
1.5L fluid per day



Heart Failure Society of America

Education modules available online at

www.hfsa.org/pdf/module2.pdf