# Food for you Heart

**Amir Lerman, MD**
**Professor of Medicine**
**Cardiovascular Division**
**Mayo Clinic, Rochester, MN**

MAYO CLINIC

mayo

CP992924-2

# Problem Statement(s)

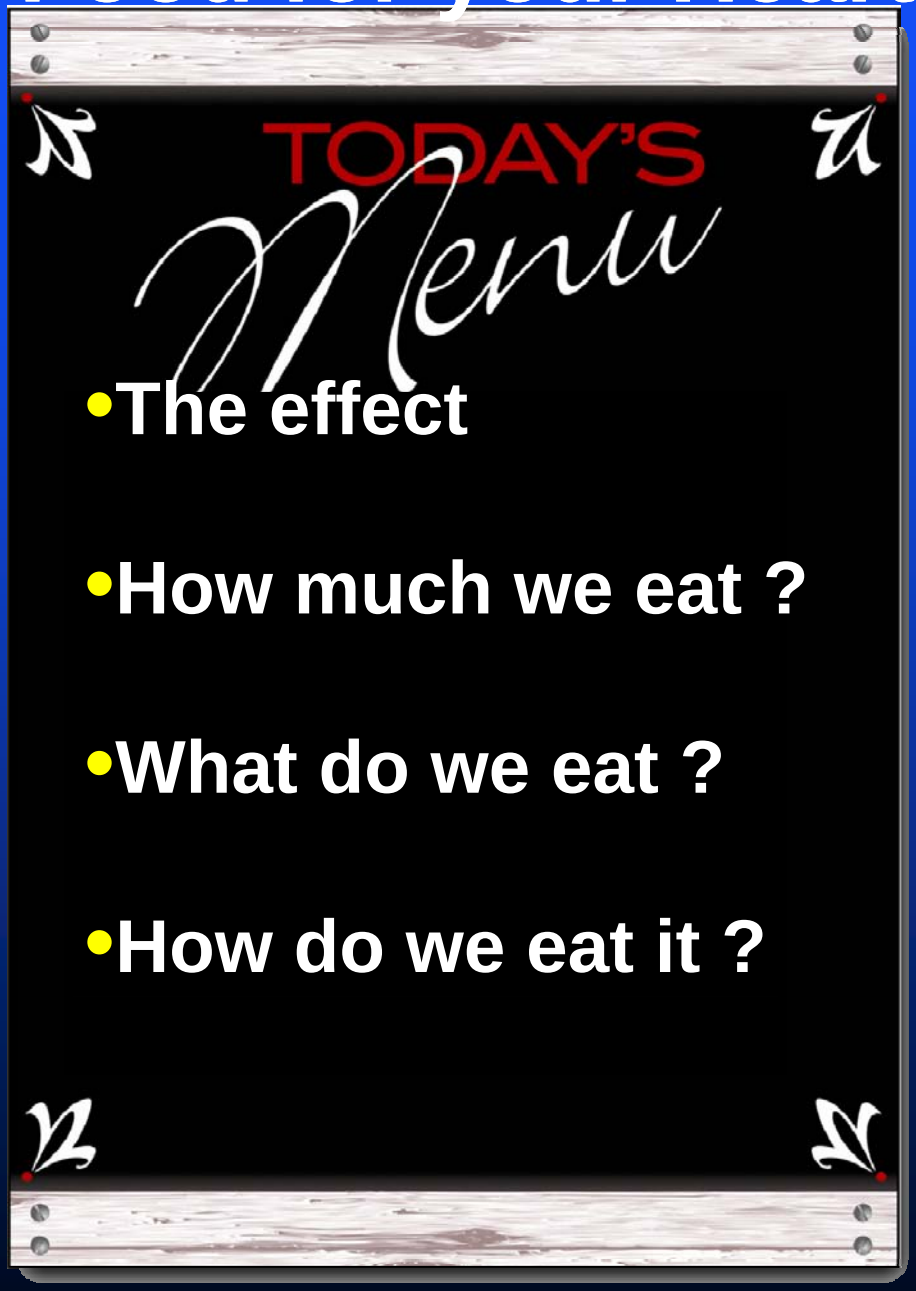
**Discrepancy between epidemiological data and outcomes data**

- Most data is retrospective or cohort in nature
- RCT data is difficult to accomplish in large numbers, well-controlled study and blinded

**Discrepancy between studies examining "surrogate" CVD markers and studies measuring CVD outcomes**

- Few studies have supported one single food as being overtly beneficial
- Difficulty in maintaining calorie neutral studies
- Most positive studies highlight "diets" as being beneficial without being to link mechanism to outcome

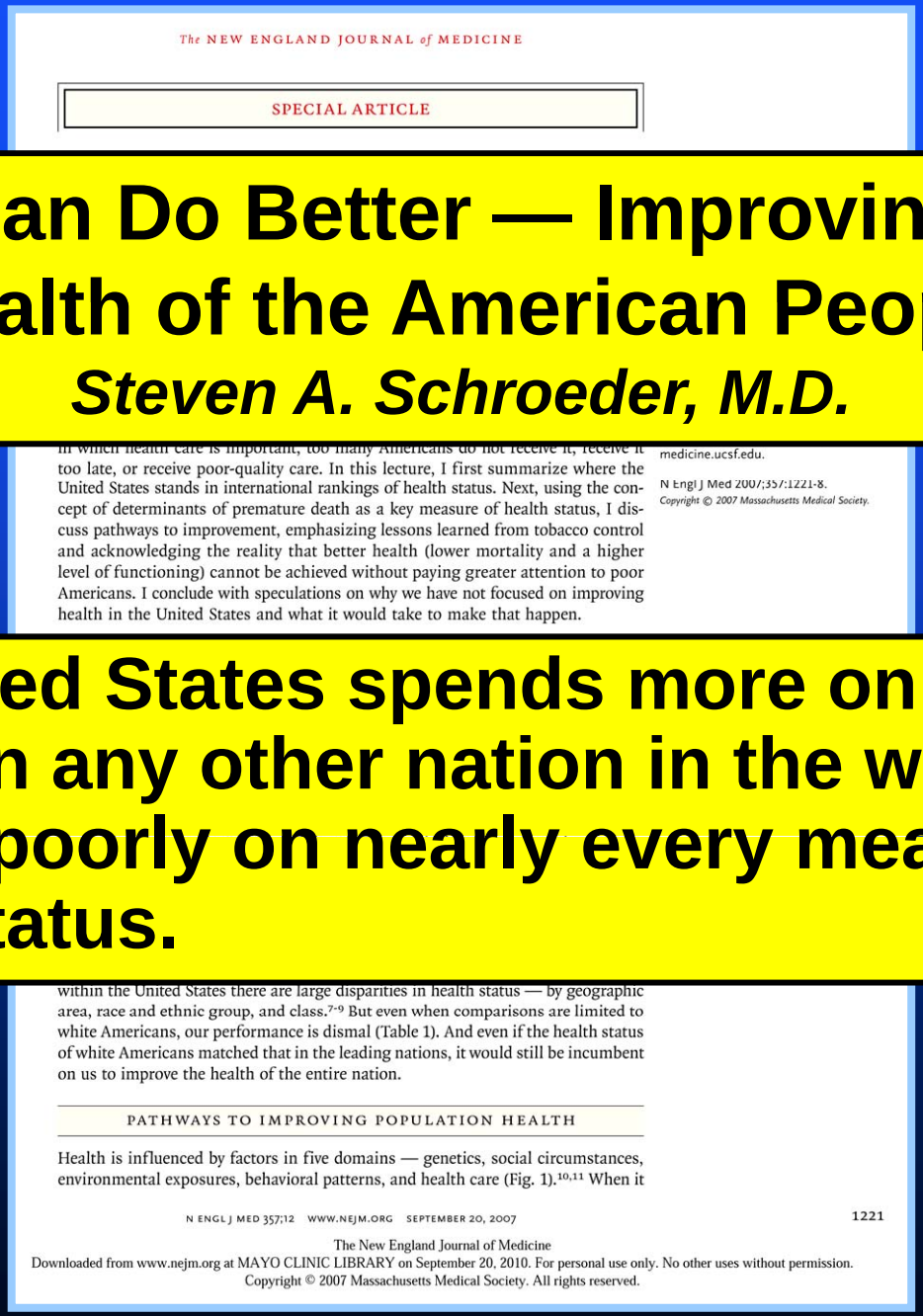
# Food for your Heart




- The effect
- How much we eat ?
- What do we eat ?
- How do we eat it ?

# We Can Do Better — Improving the Health of the American People

***Steven A. Schroeder, M.D.***



The NEW ENGLAND JOURNAL of MEDICINE

SPECIAL ARTICLE

in which health care is important, too many Americans do not receive it, receive it too late, or receive poor-quality care. In this lecture, I first summarize where the United States stands in international rankings of health status. Next, using the concept of determinants of premature death as a key measure of health status, I discuss pathways to improvement, emphasizing lessons learned from tobacco control and acknowledging the reality that better health (lower mortality and a higher level of functioning) cannot be achieved without paying greater attention to poor Americans. I conclude with speculations on why we have not focused on improving health in the United States and what it would take to make that happen.

medicine.ucsf.edu.

N Engl J Med 2007;357:1221-8.



**The United States spends more on health care than any other nation in the world, yet it ranks poorly on nearly every measure of health status.**

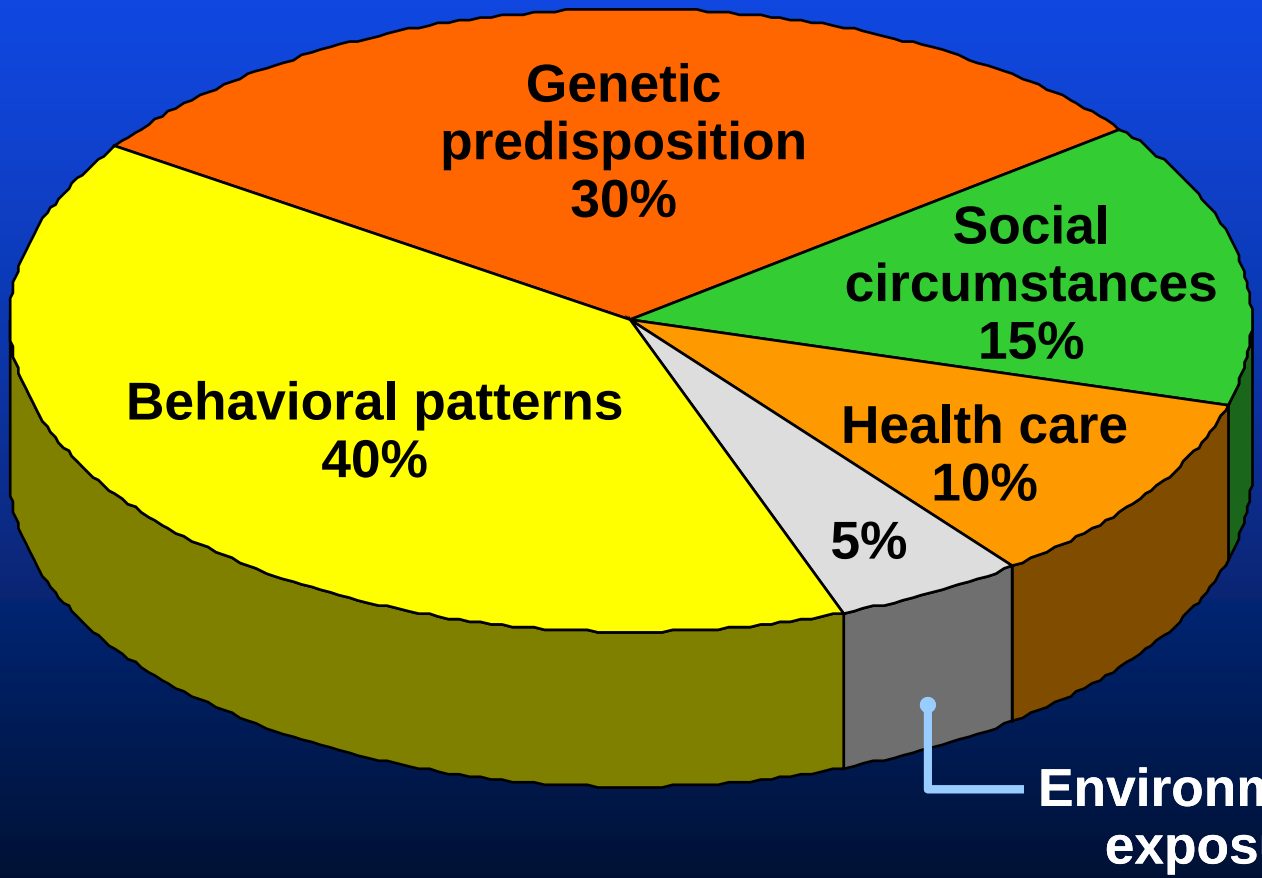
within the United States there are large disparities in health status — by geographic area, race and ethnic group, and class.[7-9] But even when comparisons are limited to white Americans, our performance is dismal (Table 1). And even if the health status of white Americans matched that in the leading nations, it would still be incumbent on us to improve the health of the entire nation.

## PATHWAYS TO IMPROVING POPULATION HEALTH

Health is influenced by factors in five domains — genetics, social circumstances, environmental exposures, behavioral patterns, and health care (Fig. 1).[10,11] When it

# Determinants of Health and Their Contribution to Premature Death

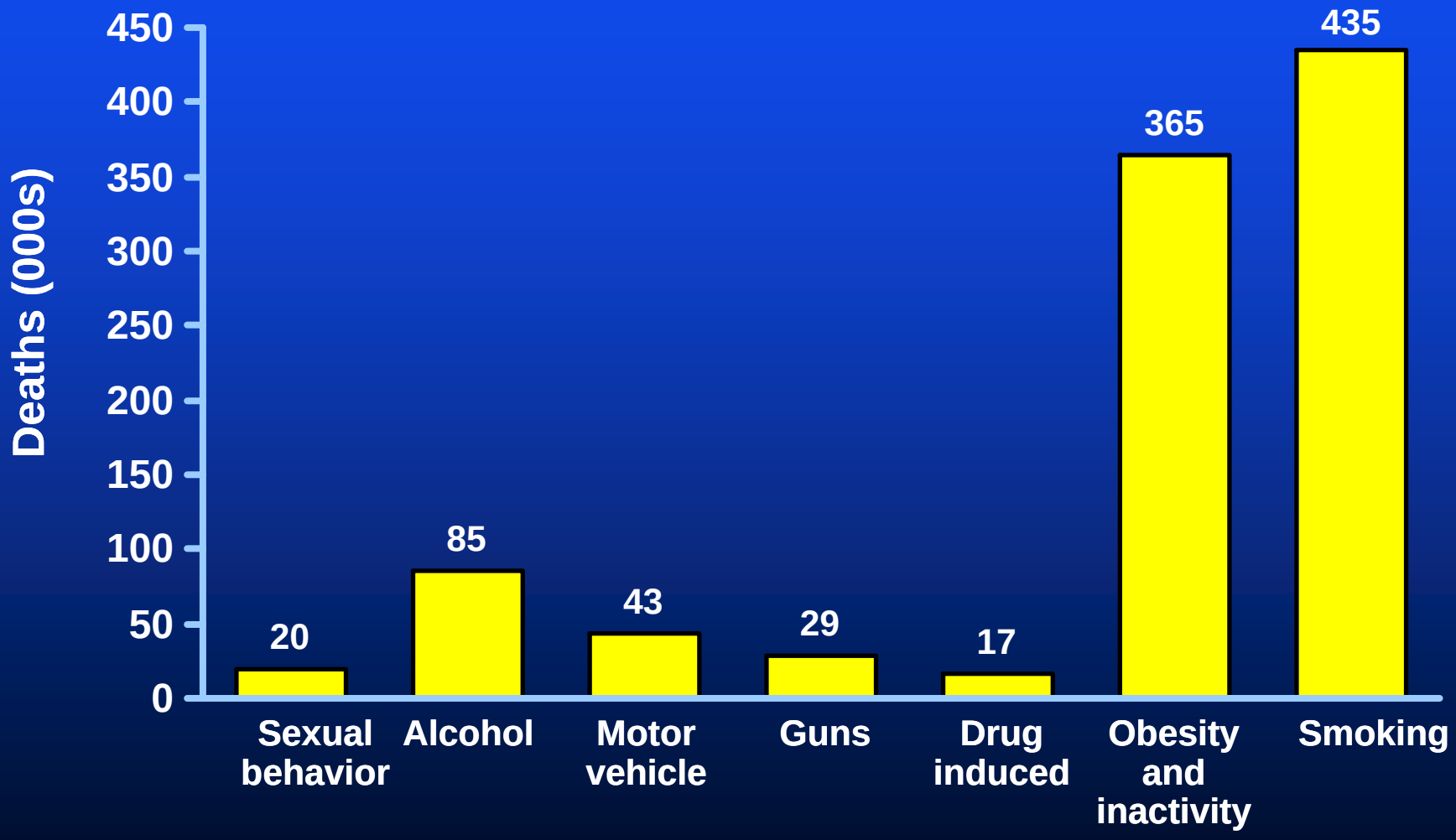
## Proportional Contribution to Premature Death




Schroeder SA: NEJM 357:1221, 2007

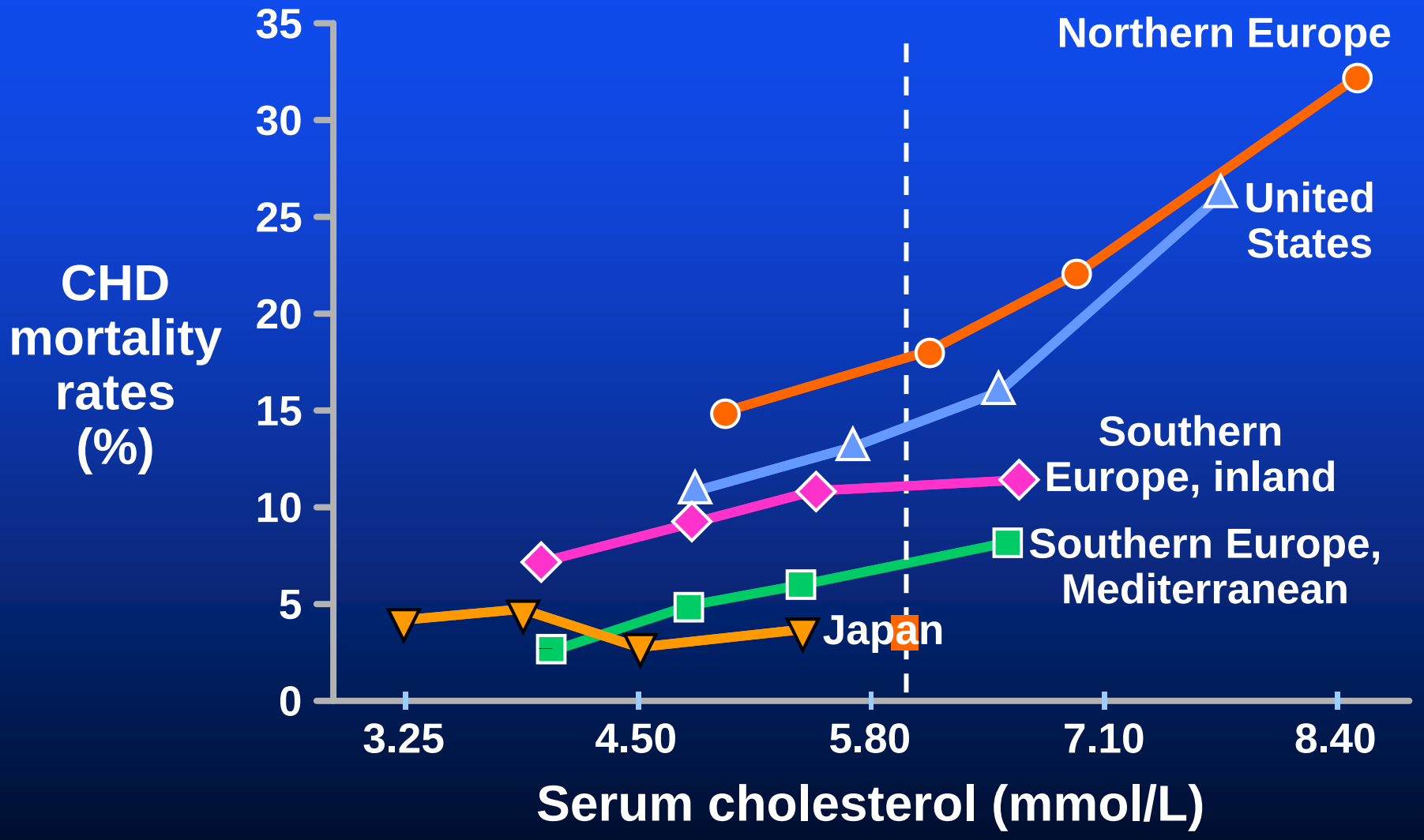
MAYO CLINIC

# Numbers of U.S. Deaths from Behavioral Causes – 2000




Schroeder SA: NEJM 357:1221, 2007

# Coronary Heart Disease Mortality in the 7-Countries Study




De Lorgeril M: Current Opinion in Cardiology 15:364, 2000

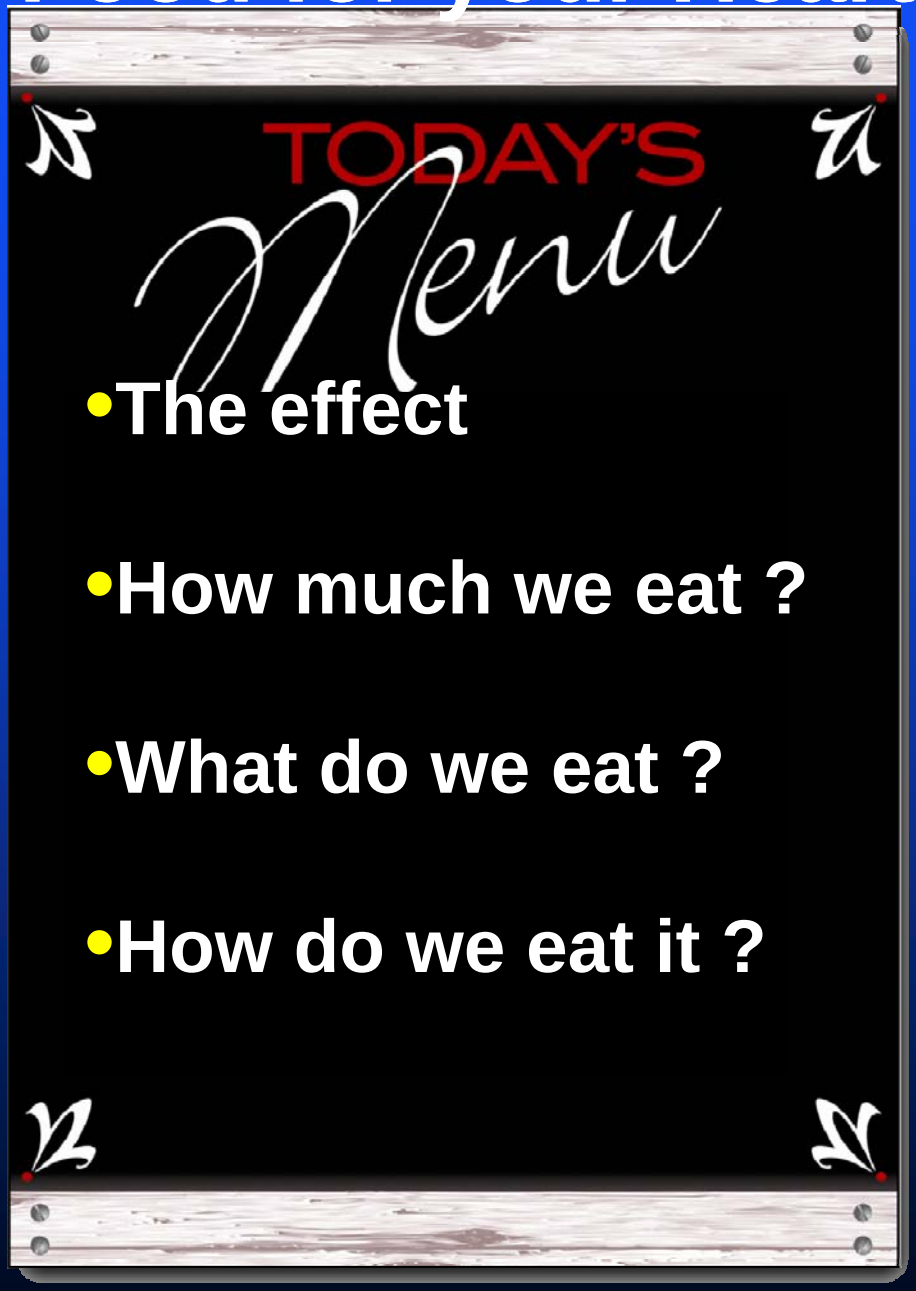
CP1159481-1

# Dietary Pattern

| | Olive oil and vegetables | Pasta and meat | Eggs and sweets |
|---|---|---|---|
| Cholesterol | 213 | 217 | 215 |
| Blood pressure | 134 | 136 | 136 |
| TG | 102 | 97 | 95 |
| HDL | 59 | 59 | 59 |
| CRP | 1.17 | 1.3 | 1.33 |
| CV risk (men) | 3.44 | 3.75 | 3.67 |
| CV risk (women) | 0.88 | 0.93 | 0.89 |

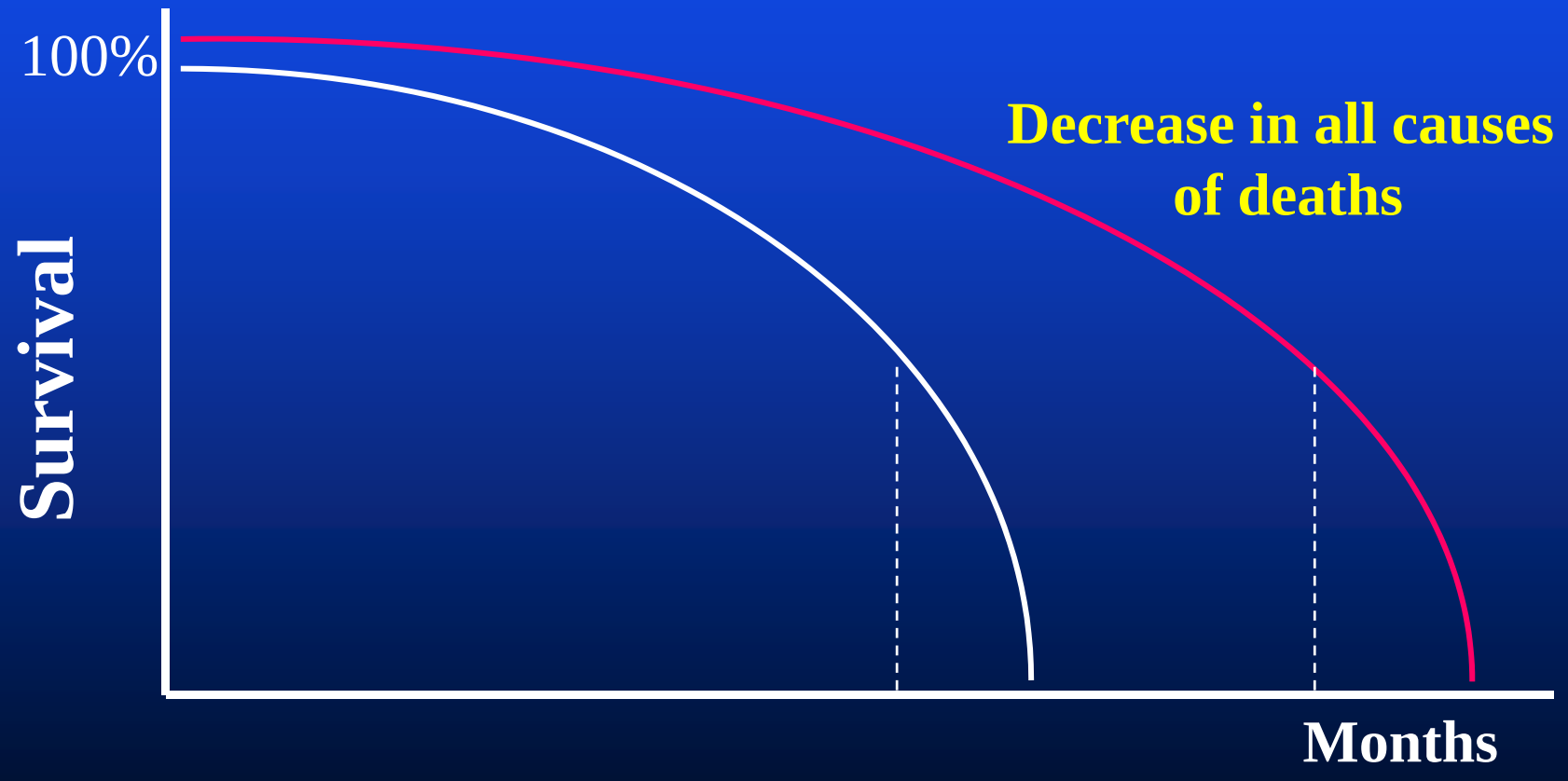
Centritto F et al: Nutrition, Metabolism & CV Dis 19:697, 2009

# Food for your Heart




- The effect
- How much we eat ?
- What do we eat ?
- How do we eat it ?

# Probability of survival in caloric restricted animals




MAYO CLINIC

# Diet, Obesity, and Cardiovascular Risk

## Growing Prevalence of Obesity and Diabetes in American Adults

- Two-thirds overweight (BMI > 25)
- > 30% frankly obese (BMI > 30)
- 30-40% metabolic syndrome
- 8-10% diabetic
- CHD mortality 2.6-3.0 higher

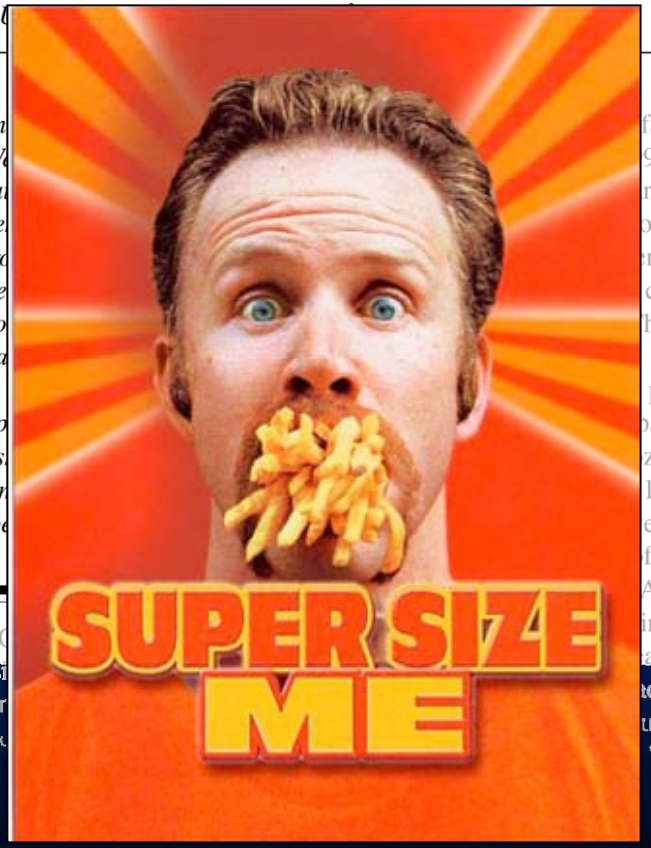
*Center for Disease Control and Prevention

# Research Article

## THE ECOLOGY OF EATING: Smaller Portion Sizes in France Than in the United States Help Explain the French Paradox

Paul Rozin,[1] Kimberly Kabnick,[1] Erin Pete,[1] Claude Fischler,[2] and Christy Shields[2]

**Abstract**—*Part of the "French paradox" can that the French eat less than Americans. W portion sizes are smaller in comparable resta dividual portions of foods (but not other ite portions specified in cookbooks, and in the pr eat" restaurants in dining guides. We also pre tions at McDonald's, that the French take lo cans. Our results suggest that in the doma generally, more attention should be paid to though their mechanism of operation is transp vealing of fundamental psychological proces the French eat less than Americans, they seer riod of time, and hence have more food exp have their cake and eat it as well.*




favorite explanation of the French para-
92).
radox only if one assumes that level of
or blood cholesterol) is the major cause
nt analyses suggest that the importance
cardiovascular disease has been greatly
here are many other possible accounts
1999), including (a) genetically based
possibility that life in France may be
particular that life around food may be
zin et al., 1999); (c) differences in atti-
less focused on outside influences such
er, 1988); (d) the fact that the French
f eating; and (e) the possibility that the
Americans.

Unlike smoking, eating is a necessity. (
health strategy for reducing the risks of obes
be easy to restrict food intake, dieting has, or
successful strategy for most Americans (Wad

ing difference between the French and
aner than Americans. The mean body
cross males and females) is 24.4 for
unication, S. Herceberg, 2002), as com-

# Restaurant Portion Sizes

| Restaurant | | No. of items sampled/no. larger in U.S. | Mean size ratio (U.S./France) | Range of ratios |
|---|---|---|---|---|
| In Paris | In Philadelphia | | | |
| **Identical chains** | | | | |
| McDonald's | McDonald's | 6/4 | 1.28 | 1.0-1.94 |
| Hard Rock Cafe | Hard Rock Cafe | 2/0 | 0.92 | 0.84-0.99 |
| Pizza Hut | Pizza Hut | 2/2 | 1.32 | 1.25-1.38 |
| Häagen Dazs | Häagen Dazs | 2/2 | 1.42 | 1.37-1.48 |
| **Comparable restaurants** | | | | |
| French: local bistro | French: local bistro | 1/1 | 1.17 | – |
| Quick | Burger King | 5/4 | 1.36 | 0.73-1.81 |
| Local Chinese | Local Chinese | 6/4 | 1.72 | 0.87-2.78 |
| Italian: Bistro Romain | Olive Garden | 3/2 | 1.02 | 0.50-1.45 |
| Crepes: local | Crepes: local | 4/2 | 1.04 | 0.70-1.39 |
| Local ice cream* | Local ice cream* | 2/2 | 1.24 | 1.08-1.41 |
| Pizza: local | Pizza: local | 2/2 | 1.32 | 1.17-1.46 |

*Berthillon in Paris, Bassett's in Philadelphia

Rozin P et al: Psych Sci 14:450, 2003

Epidemiology and Prevention

# Dietary Intervention to Reverse Carotid Atherosclerosis

Iris Shai, RD, PhD*; J. David Spence, MD*; Dan Schwarzfuchs, MD; Yaakov Henkin, MD; Grace Parraga, PhD; Assaf Rudich, MD, PhD; Aaron Fenster, PhD; Christiane Mallett, MSc; Noah Liel-Cohen, MD; Amir Tirosh, MD, PhD; Arkady Bolotin, PhD; Joachim Thiery, MD; Georg Martin Fiedler, MD; Matthias Blüher, MD; Michael Stumvoll, MD; Meir J. Stampfer, MD, DrPH; for the DIRECT Group

***Background***—It is currently unknown whether dietary weight loss interventions can induce regression of carotid atherosclerosis.

**Background: It is currently unknown whether dietary weight loss interventions can induce regression of carotid atherosclerosis.**

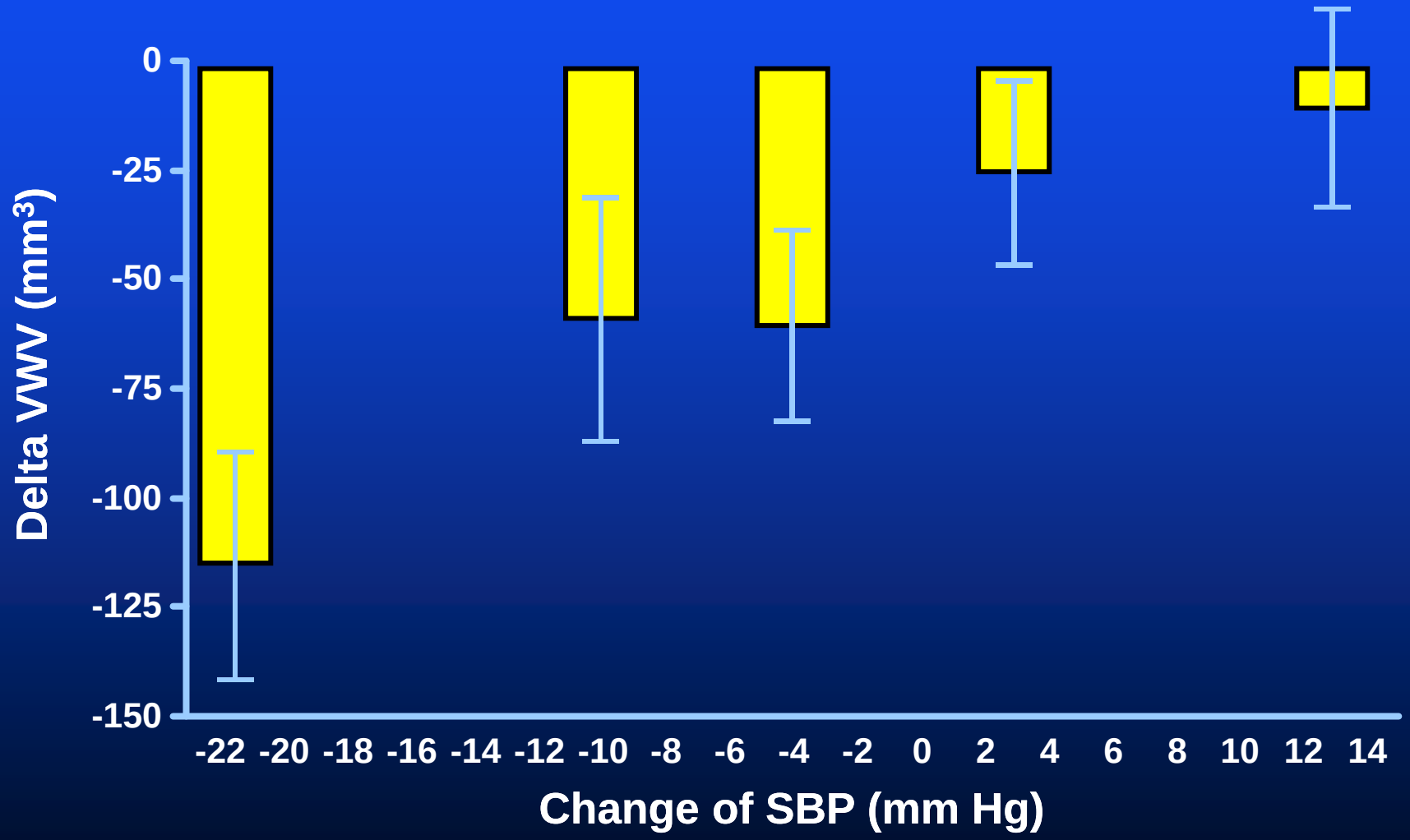
exhibited progression (mean increase, +89.6 $mm^3$; 95% confidence interval, +66.6 to +112.6 $mm^3$) had achieved greater weight loss (−5.3 versus −3.2 kg; $P$=0.03), greater decreases in systolic blood pressure (−6.8 versus −1.1 mm Hg; $P$=0.009) and total homocysteine (−0.06 versus +1.44 μmol/L; $P$=0.04), and a higher increase of apolipoprotein A1 (+0.05 versus −0.00 g/L; $P$=0.06). In multivariate regression models, only the decrease in systolic blood pressure remained a significant independent modifiable predictor of subsequent greater regression in both carotid VWV ($\beta$=0.23; $P$=0.01) and intima-media thickness ($\beta$=0.28; $P$=0.008) levels.

**Conclusions: Two-year weight loss diets can induce a significant regression of measurable carotid VWV. The effect is similar in low-fat, Mediterranean, or low-carbohydrate strategies and appears to be mediated mainly by the weight loss-induced decline in blood pressure.**

Medical School, and Departments of Epidemiology and Nutrition, Harvard School of Public Health, Boston, Mass (M.J.S.).
*The first 2 authors contributed equally to this work.
Guest Editor for this article was Paul W. Armstrong, MD.
**The online-only Data Supplement is available with this article at http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.109.879254/DC1.**
Correspondence to Iris Shai, RD, PhD, The S. Daniel Abraham International Center for Health and Nutrition, Department of Epidemiology and Health Systems Evaluation, Ben-Gurion University of the Negev, PO Box 653, Beer-Sheva 84105, Israel. E-mail irish@bgu.ac.il

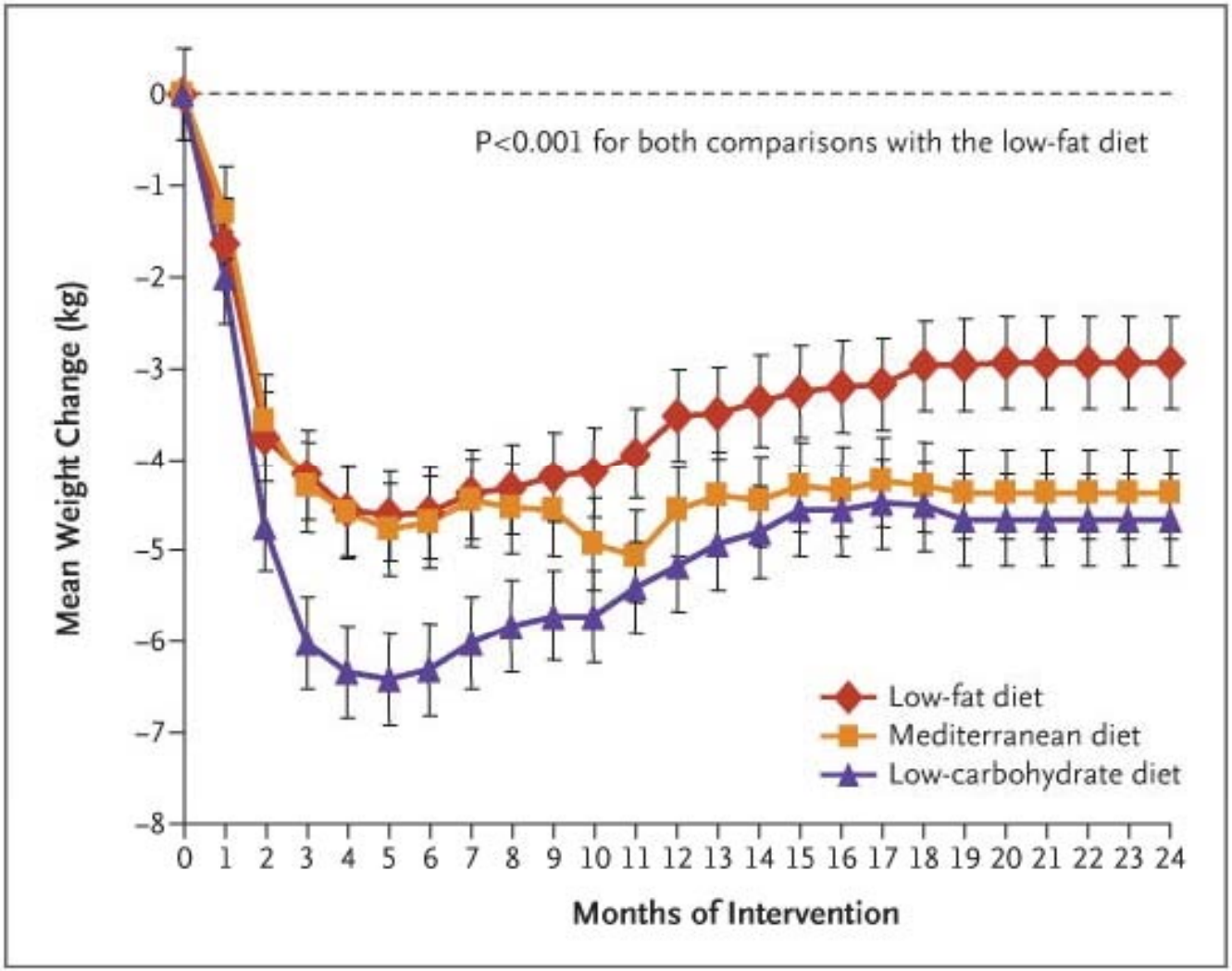
*Circulation* **is available at http://circ.ahajournals.org** **DOI: 10.1161/CIRCULATIONAHA.109.879254**

# 2-Year Change in Carotid Vessel Wall Volume Across Quintiles of Change in Systolic Blood Pressure




Shai et al: Circ 121:1200, 2010

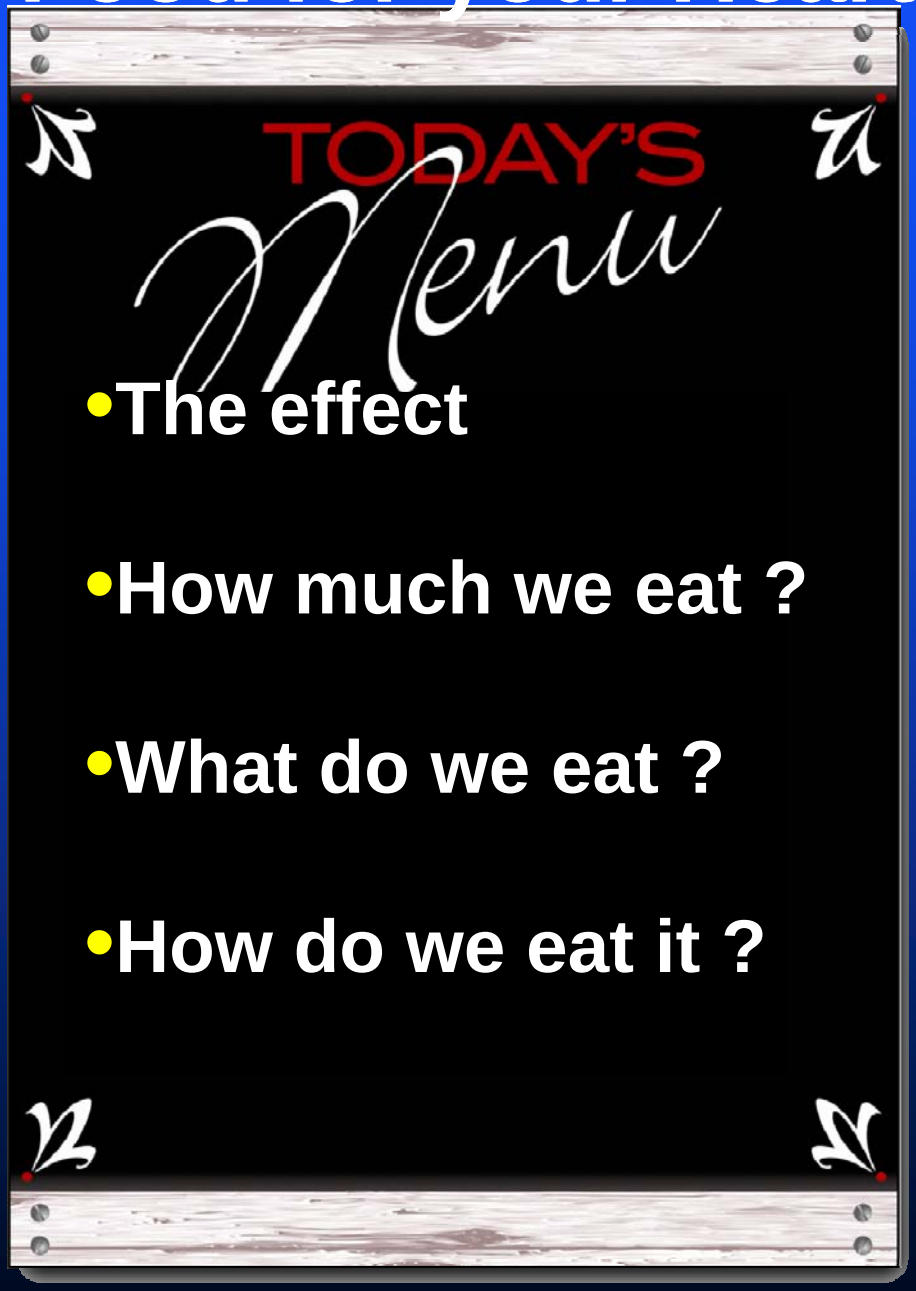
# Weight Changes during 2 Years According to Diet Group.





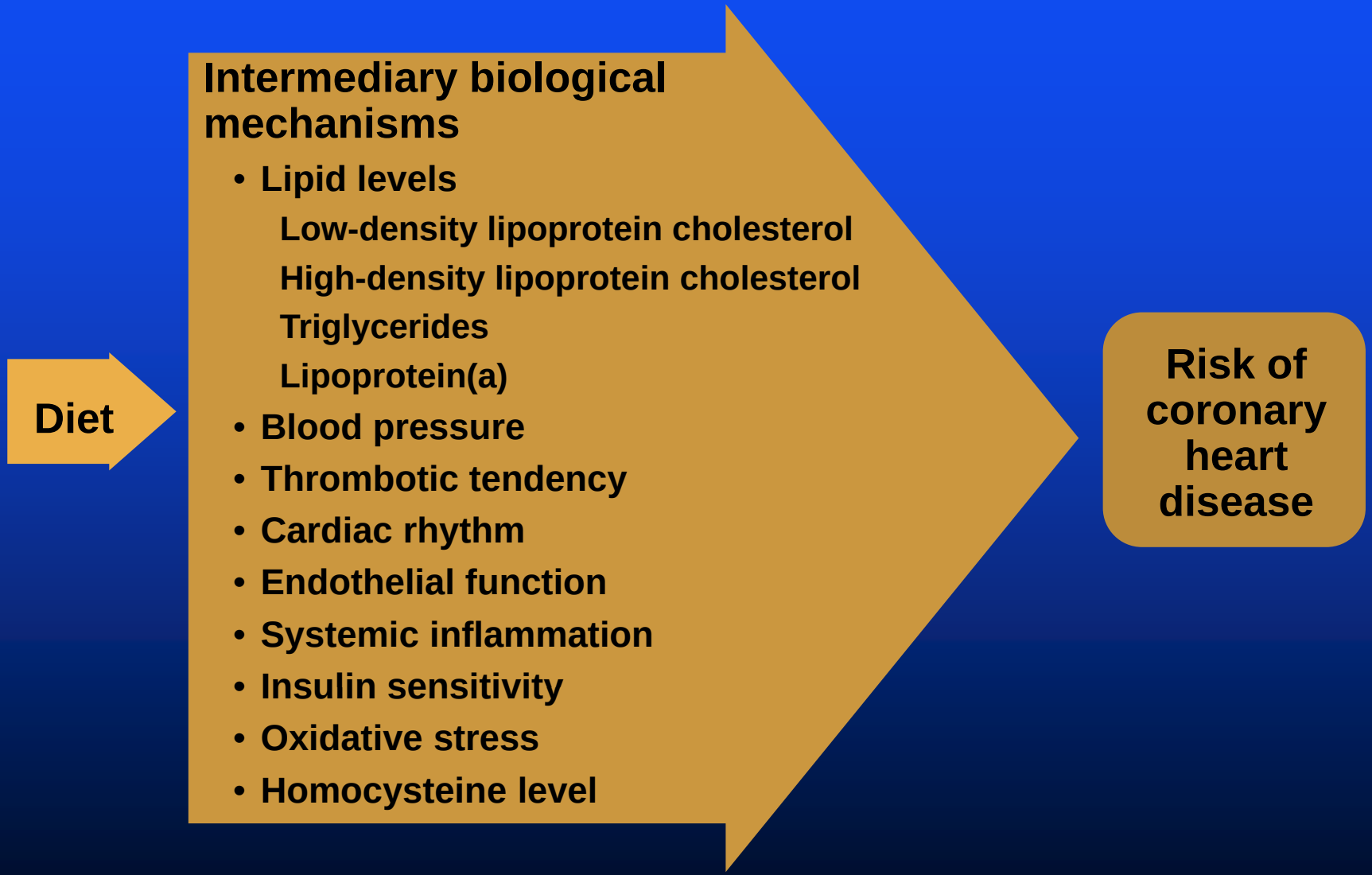

Shai I et al. N Engl J Med 2008;359:229-241.

# Food for your Heart



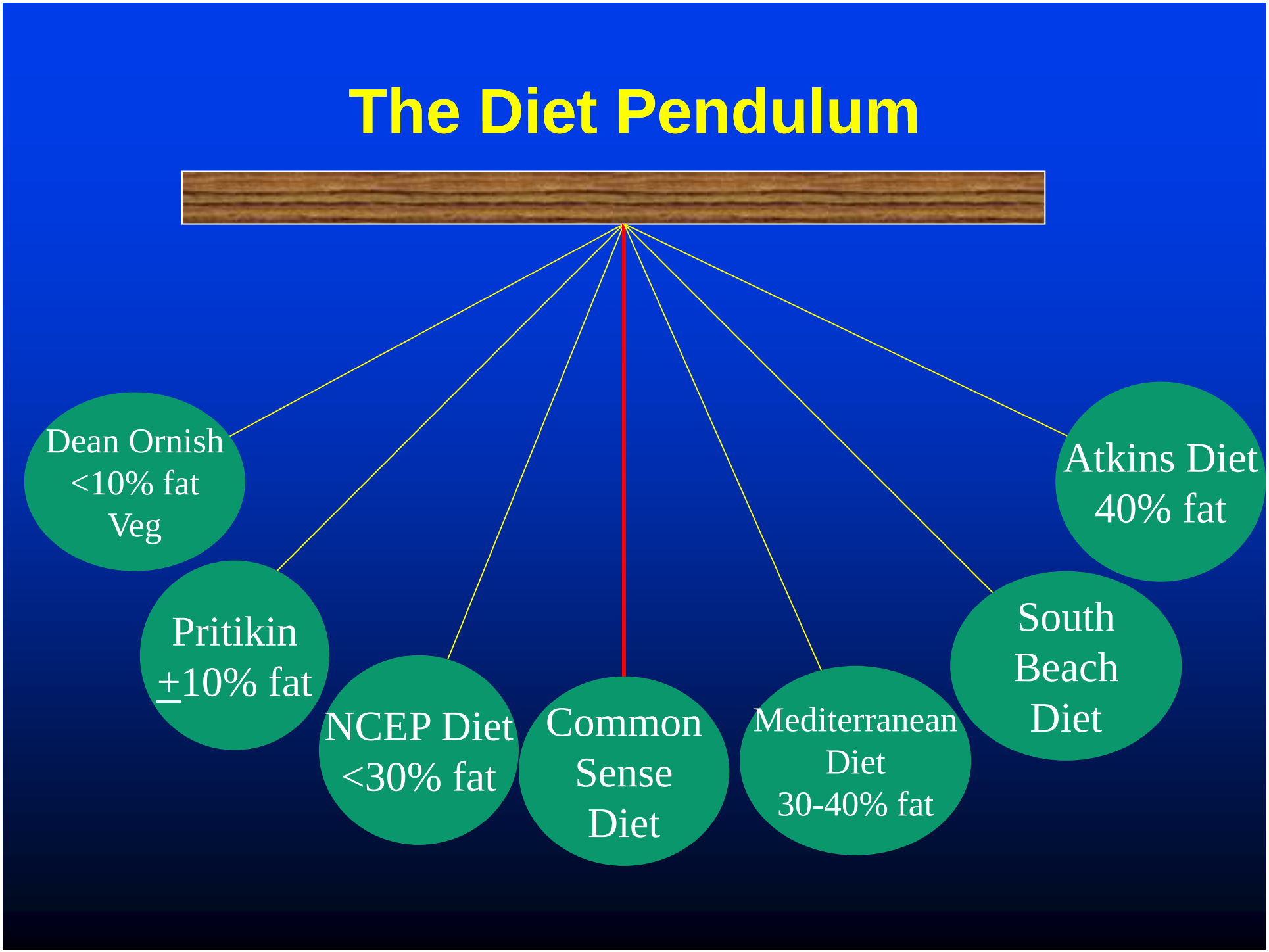

- The effect
- How much we eat ?
- What do we eat ?
- How do we eat it ?

# Mechanisms by Which Diet Potentially Influences Risk of Coronary Heart Disease




Hu and Willett: JAMA 288(20):2569, 2002

# The Diet Pendulum



Dean Ornish
<10% fat
Veg

Pritikin
±10% fat

NCEP Diet
<30% fat

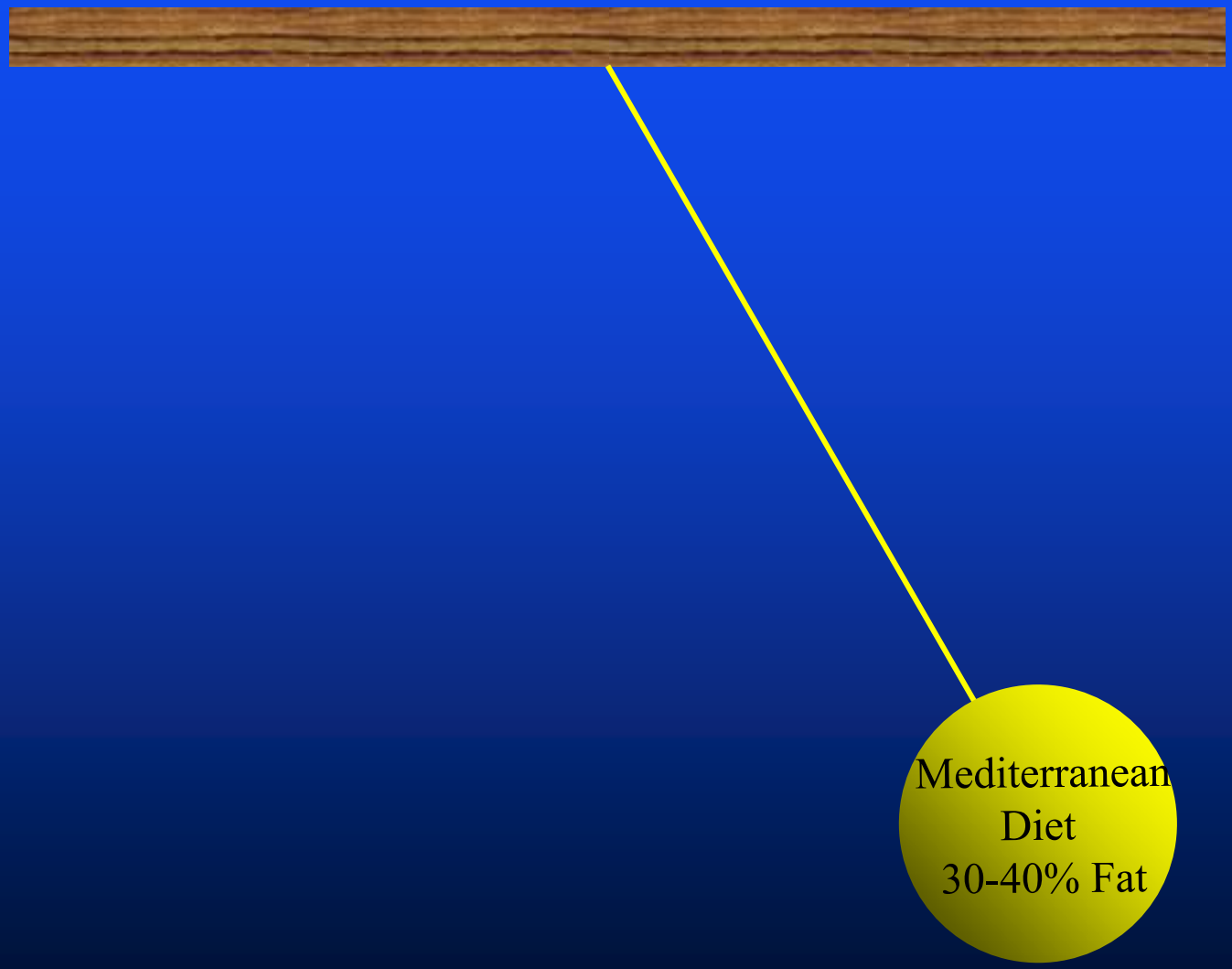
Common
Sense
Diet

Mediterranean
Diet
30-40% fat

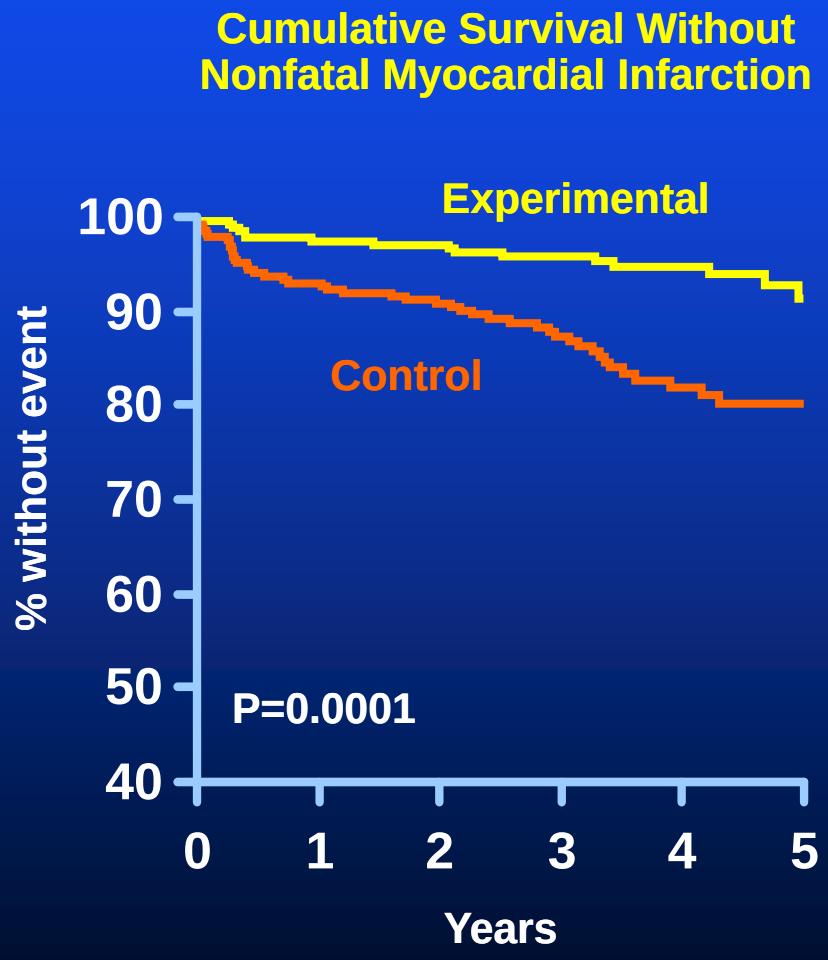
South
Beach
Diet

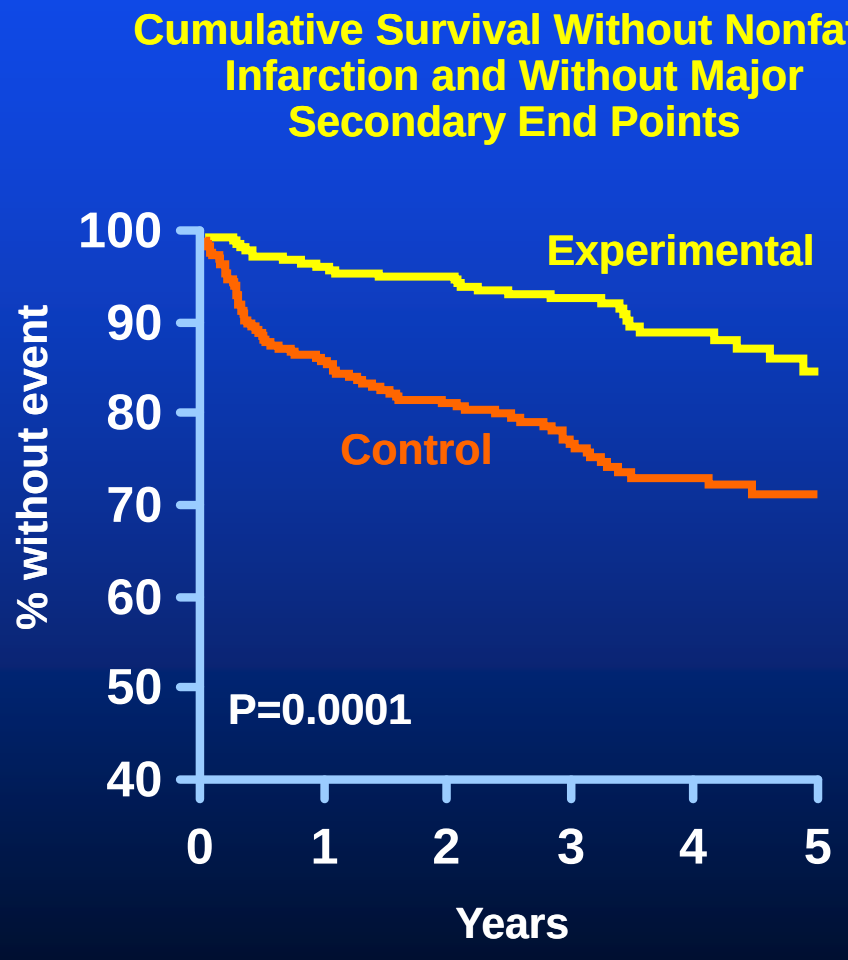
Atkins Diet
40% fat

# The Diet Pendulum



# Mediterranean Diet and CV Events




MAYO CLINIC

de Lorgeril M et al: Circulation 99:779, 1999

# Lyon Diet Heart Study

**605 men/women (302 treatment, 303 control)**

- **diet - Mediterranean vs usual care**
- **treatment - margarine, omega-3 FA (α linolenic acid)**
  - **↓total cardiac mortality 65%**
  - **↓sudden death 64% (0% sudden death first 2 yrs)**
- **lipids did not change**
- **major benefit - omega-3 FA (α linolenic acid)**
  - **(+) better diet - ? Wine**

**Ref: Lyon Heart Study, Circulation 1999;99:779-85**

# Mediterranean Diet (Greece)

- 22,000 adults - 20-86 yrs
- Follow-up 4 yrs
- Interviewed -
  - what they ate and drank
  - portion sizes
  - how often they ate
  - smoking habits
- Measured - ht, wt, waist circumference
- Rated - score of 0-9, how closely they followed traditional Mediterranean diet

N Engl J Med 2003;348:2599-2608

# Mediterranean Diet (Greece)

- A 2-point increase on diet adherence score
  - 25% ↓ death all cause
  - 33% ↓ heart disease death
  - 24% ↓ death from cancer
- Individual foods alone - no effect on risk ↓

  (Total Diet)

N Engl J Med 2003;348:2599-2608

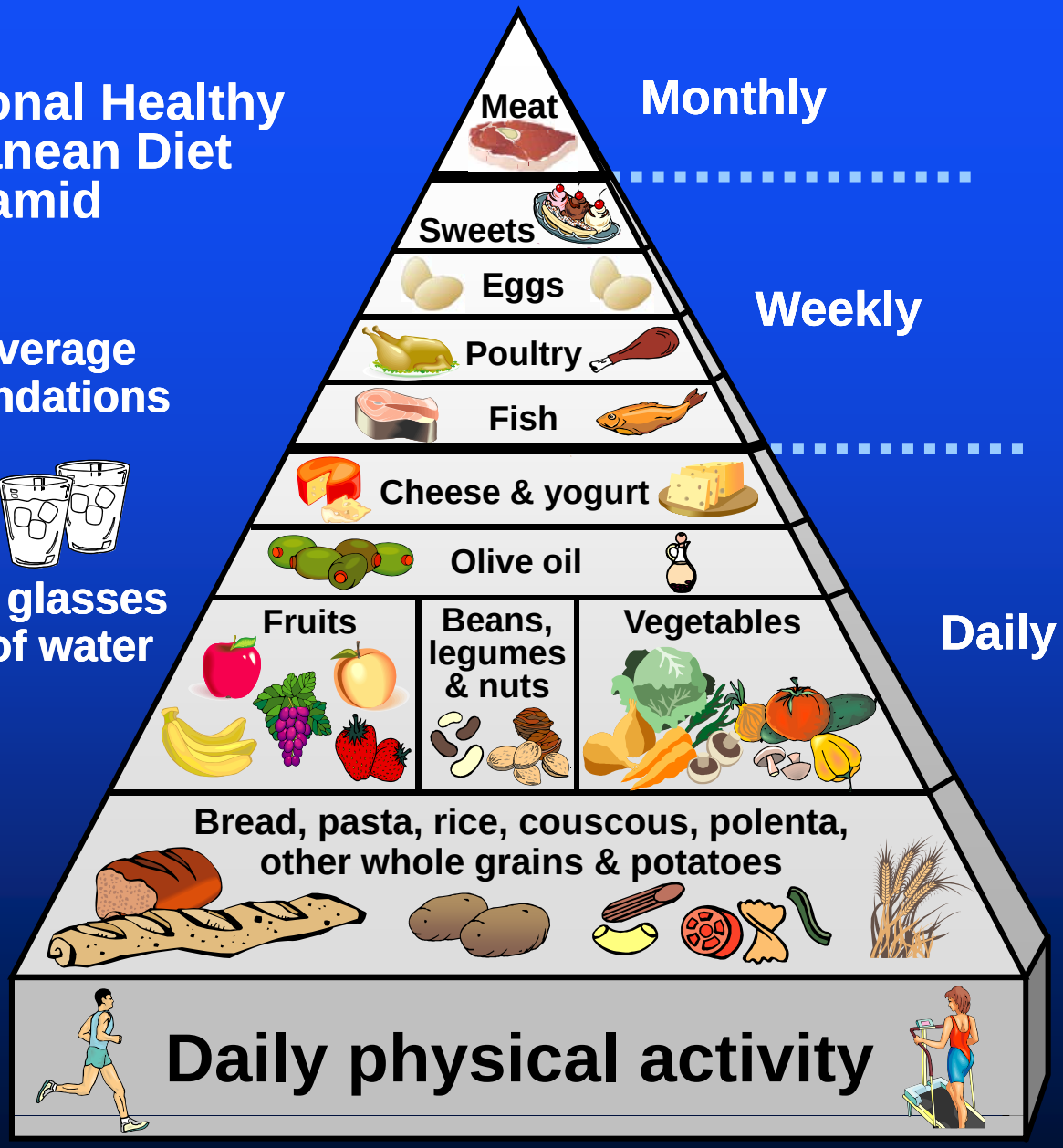
# The Traditional Healthy Mediterranean Diet Pyramid

Wine in moderation

Daily beverage recommendations



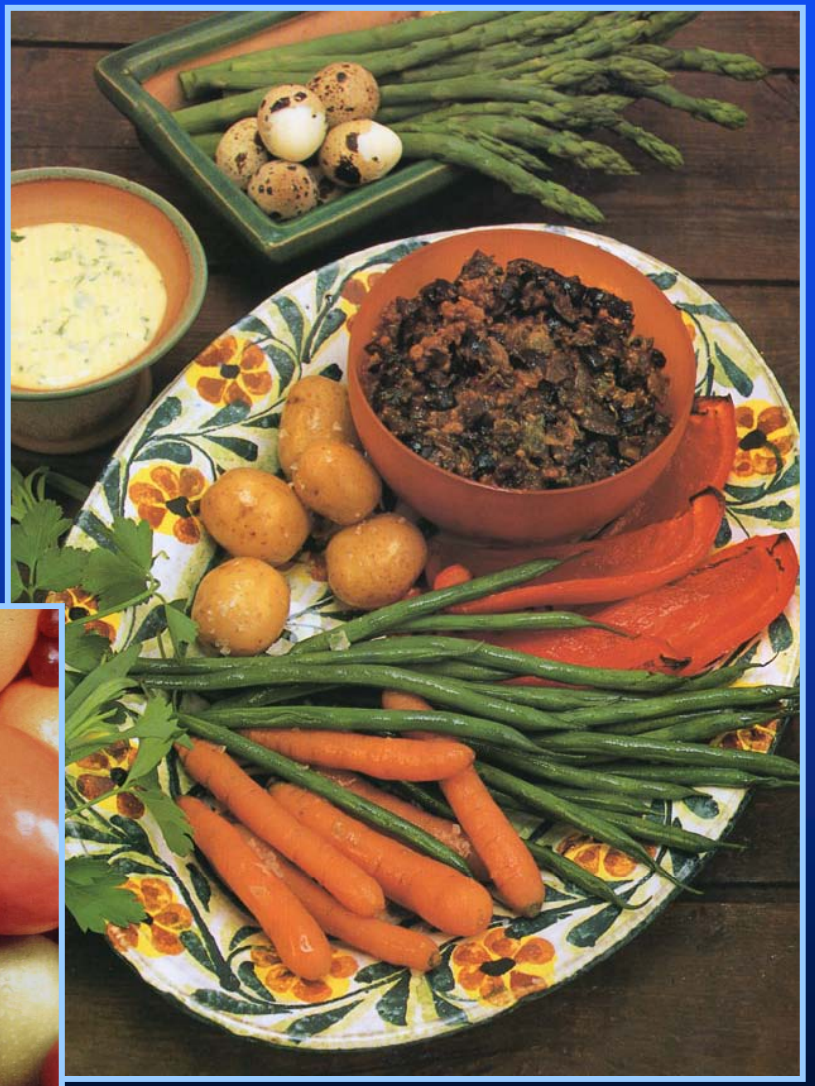
6 glasses of water



# Mediterranean Diet – Health Effects

- Vegetables and fruits
  - High in phytochemicals
  - Low in calories
  - Associated with low risk of CHD*

*Ann Intern Med 134:1106, 2001



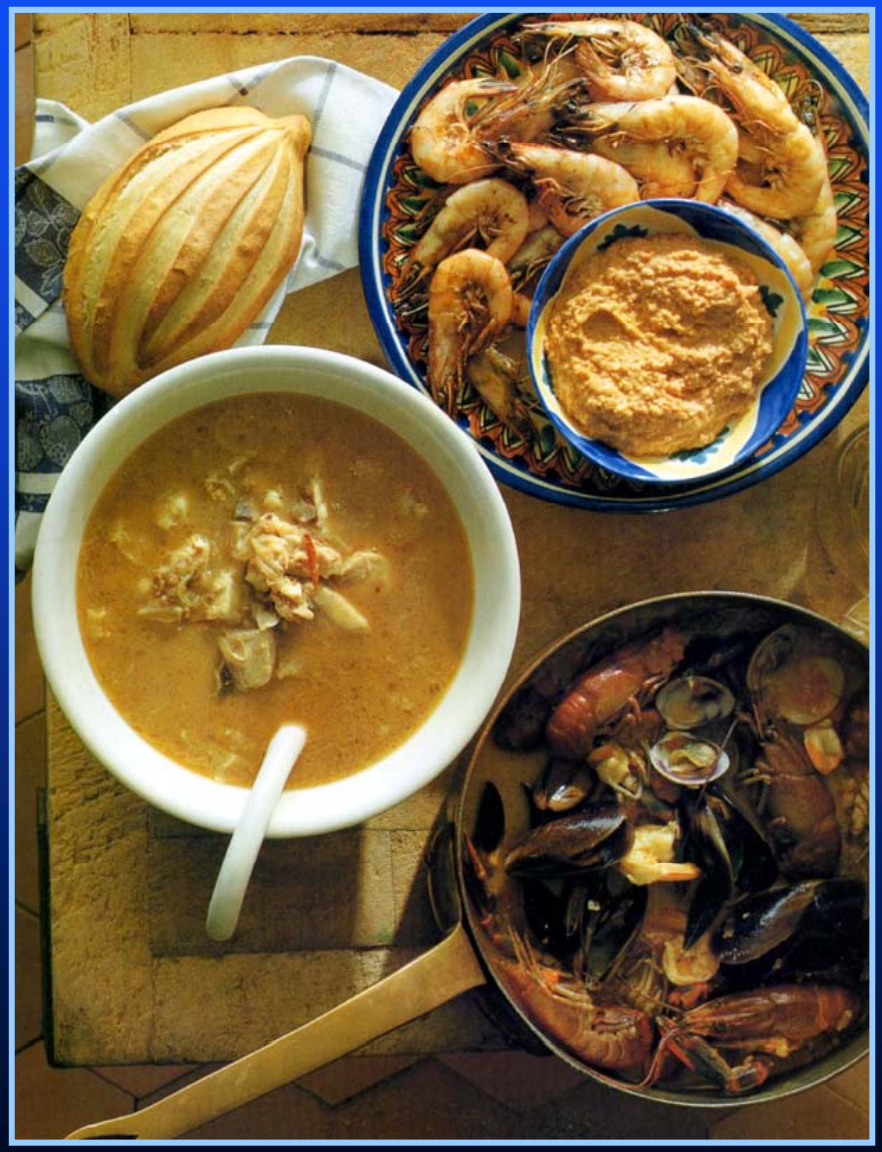
CP1059685-21

# Mediterranean Diet – Health Effects

- Olive oil
  - Monounsaturated fat
    - Lowers total and LDL cholesterol
    - Doesn't lower HDL cholesterol
    - Resistant to oxidation
    - Associated with reduced risk of CHD

# Mediterranean Diet – Health Effects

- Fish and shellfish – omega-3 fatty acids
  - Anti-arrhythmic effect
  - Antithrombotic effect
  - Lowers triglycerides
  - Lowers blood pressure
  - Anti-inflammatory effect
- Associated with reduced risk of CHD and sudden death (DART* and GISSI-Prevenzione** trials)



CP1059685-18

* Lancet 16:1450, 1989

** Lancet 354:447, 1999

# Fish and Omega-3 Fatty Acid Intake
## Secondary Prevention Trial

**GISSI - Prevenzione Trial**

- 11,324 pts (mainly men) post-MI
- 1 g fish oil omega-3/day - 2 yrs
  - ↓ 20% all-cause mortality
  - ↓ 45% sudden death

Ref JAMA 2002;287(14):1815-21

# Prevention of Fatal Cardiac Arrhythmias

**N-3 - α linolenic acid (omega-3)**

- **↑electrical stimulus to elicit action potential (↑ 50%)**
- **prolongs relative refractory time (> 150%)**
- **modulates sodium and calcium channels in myocytes**
- **similar anticonvulsant activity in brain cells**

**Kang J, et al Circulation 1996;94(7):1774-80**

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

# n–3 Fatty Acids and Cardiovascular Events after Myocardial Infarction

**Background** **Results from prospective cohort studies and randomized, controlled trials have provided evidence of a protective effect of n-3 fatty acids against CV diseases. We examined the effect of the marine n-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) and of the plant-derived alpha-linolenic acid (ALA) on the rate of CV events among patients who have had an MI.**

events and cardiac interventions. Data were analyzed according to the intention-to-treat principle, with the use of Cox proportional-hazards models.

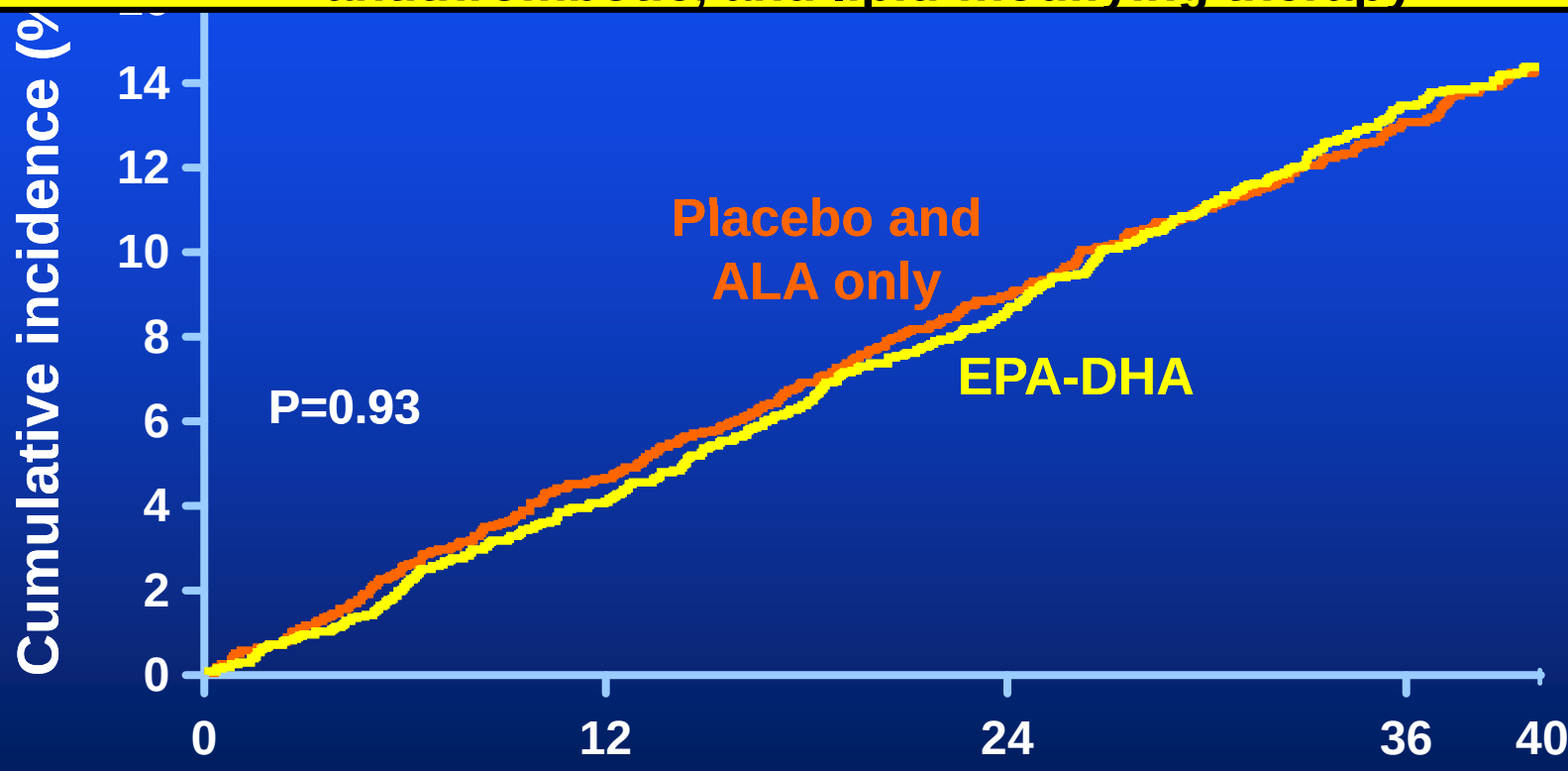
**RESULTS**

The patients consumed, on average, 18.8 g of margarine per day, which resulted in additional intakes of 226 mg of EPA combined with 150 mg of DHA, 1.9 g of ALA, or both, in the active-treatment groups. During the follow-up period, a major cardiovascular event occurred in 671 patients (13.9%). Neither EPA–DHA nor ALA reduced this primary end point (hazard ratio with EPA–DHA, 1.01; 95% confidence interval [CI], 0.87 to 1.17; P=0.93; hazard ratio with ALA, 0.91; 95% CI, 0.78 to 1.05; P=0.20). In the prespecified subgroup of women, ALA, as compared with placebo and EPA–DHA alone, was associated with a reduction in the rate of major cardiovascular events that approached significance (hazard ratio, 0.73; 95% CI, 0.51 to 1.03; P=0.07). The rate of adverse events did not differ significantly among the study groups.

**CONCLUSIONS**

Low-dose supplementation with EPA–DHA or ALA did not significantly reduce the rate of major cardiovascular events among patients who had had a myocardial infarction and who were receiving state-of-the-art antihypertensive, antithrombotic, and lipid-modifying therapy. (Funded by the Netherlands Heart Foundation and others; ClinicalTrials.gov number, NCT00127452.)

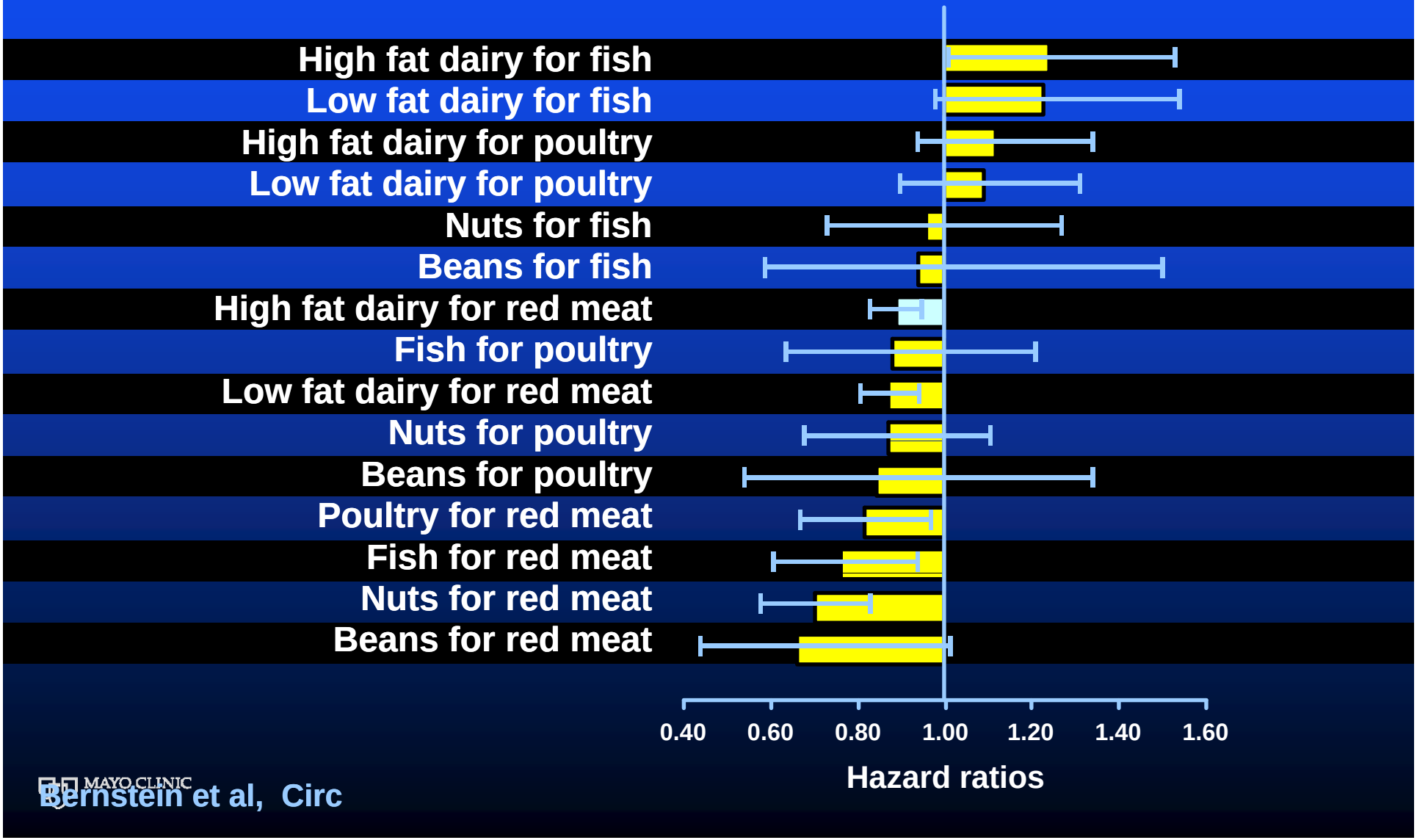
**Conclusions** **Low-dose supplementation with EPA-DHA or ALA did not significantly reduce the rate of major CV events among patients who had had an MI and who were receiving state-of-the-art antihypertensive, antithrombotic, and lipid-modifying therapy.**




| No. at risk | | | | | |
|---|---|---|---|---|---|
| EPA-DHA | 2,404 | 2,290 | 2,162 | 1,715 | 1,424 |
| Placebo | 2,433 | 2,299 | 2,163 | 1,714 | 1,425 |

Kromhout et al: NEJM, 2010

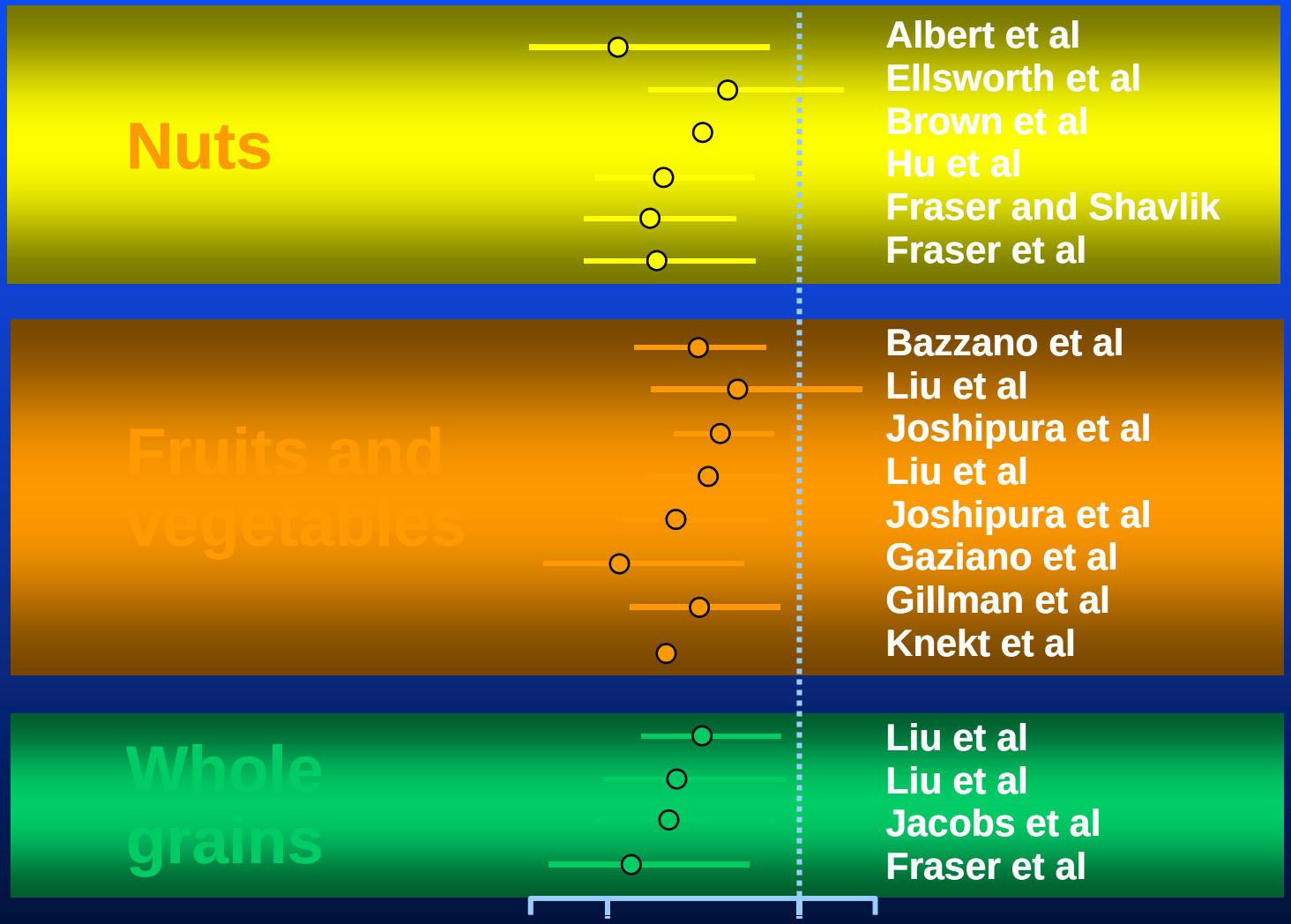
# RRs and 95% CIs for CHD Associated with Replacement of a Major Dietary Protein Source with Another




MAYO CLINIC

Bernstein et al, Circ

# Prospective Cohort Studies of Cardiovascular Disease and Consumption of Nuts, Fruits and Vegetables or Whole Grains

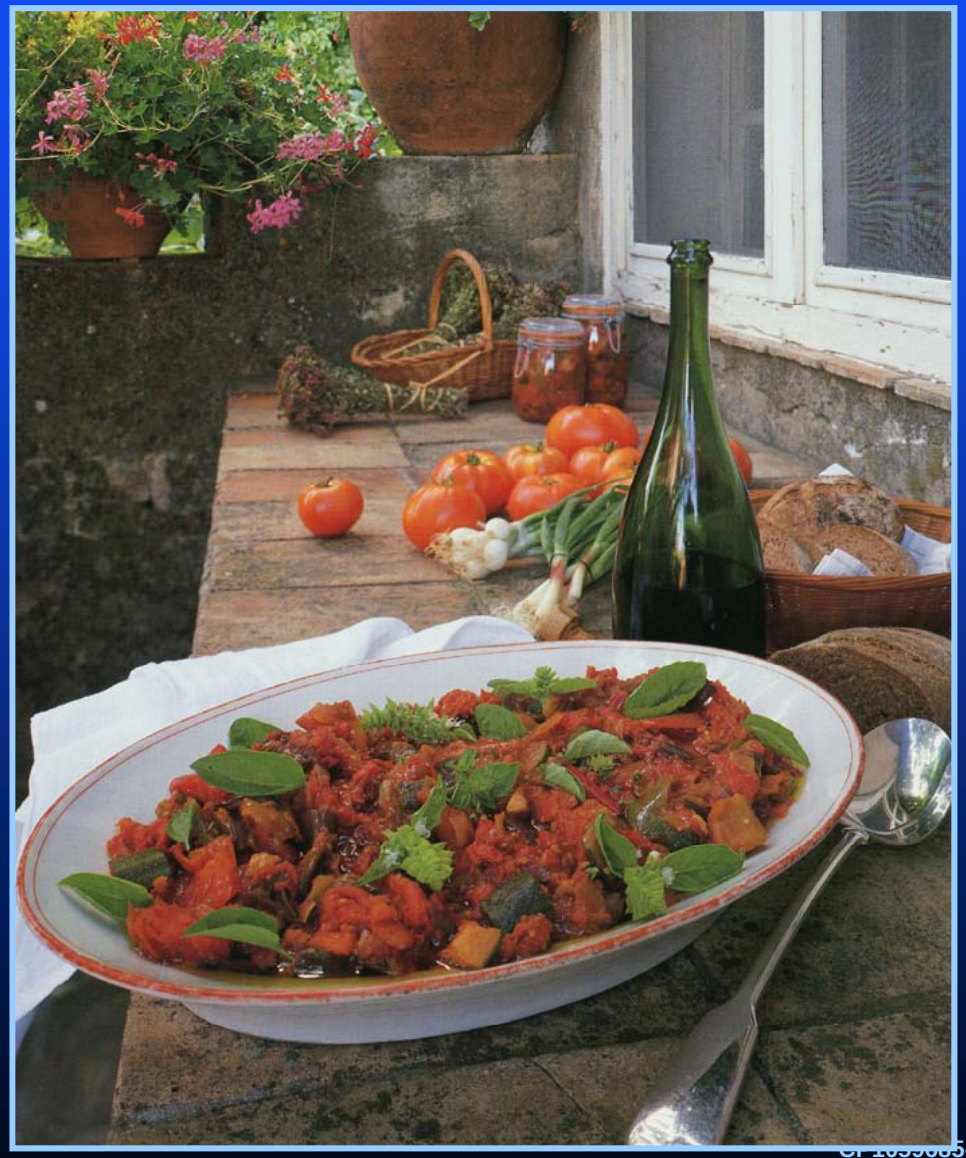



Hu and Willett:
JAMA 288(20):2569, 2002

MAYO CLINIC

# Mediterranean Diet – Health Effects

- **Wine**
  - **Raises HDLc**
  - **Inhibits platelet aggregation**
  - **High in phenolic antioxidants**
  - **Alcohol associated with reduced risk of CHD**



Journal of the American College of Cardiology
© 2010 by the American College of Cardiology Foundation
Vol. 55, No. 13, 2010
ISSN 0735-1097/10/$36.00

**QUARTERLY FOCUS ISSUE: PREVENTION/OUTCOMES**

**Editorial Comment**

# Alcohol and Cardiovascular Mortality

## Common Sense and Scientific Truth*

Arthur L. Klatsky, MD

*Oakland, California*

intake was less harmful or innocuous led to guidelines for a reasonable upper drinking limit. Probably the most famous

See page 1328

of these was Anstie's Rule of a "sensible limit" of 45 ml of ethyl alcohol/day, or approximately 3 standard-sized drinks (1). In the mid-19th century, Anstie, a prominent public health activist, no doubt intended his guideline for mature men, but he recognized variation in alcohol tolerance. The use of "sensible" rather than "safe" acknowledges that no amount of alcohol is safe for everyone.

### Scientific Evidence Develops

Evidence of possible benefit from moderate alcohol drinking was a 20th century development. One population report (2) of an alcohol–mortality J-curve relationship preceded others by one-half century. In Pearl's Baltimore study of 5,248 subjects, "heavy/steady" drinkers had the highest mortality, "abstainers" were next, and "moderate" drinkers had the lowest mortality. With no explanation, and in U.S. Prohibition days, his predictably cautious interpretation was that moderate drinking was "not harmful." A major contribution was the realization that comparing all drinkers with abstainers masked the J-curve. Memorably, he said, "one cannot judge the role of diet by starvation or excess" (2).

CAD dominates statistics for all cardiovascular (CV) disease, the alcohol relation is similar for CAD and CV.

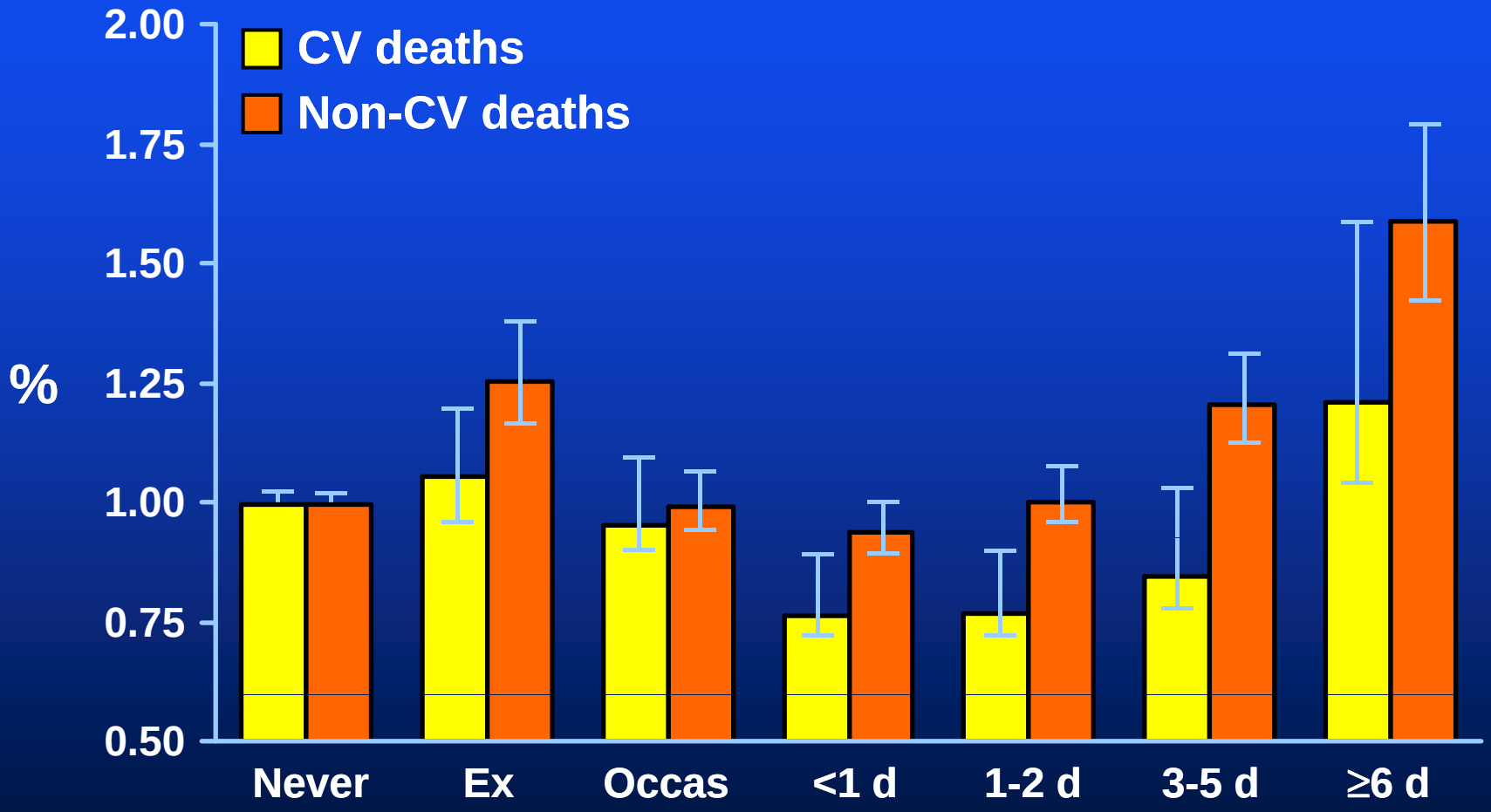
The alcohol–CAD reports have been examined intensely for methodological flaws (7–9). Reasonable fear of problems consequent to encouragement of moderate drinking contributes to reluctance to accept that there is any benefit from alcohol. Skepticism is fueled because some studies failed to separate ex-drinkers, including "sick quitters," from lifelong abstainers in the referent group, thus exaggerating apparent benefits of lighter drinking. Although studies using lifelong abstainers or infrequent drinkers as referents confirmed apparent protection (4), the absence of prospective randomized trials with CV events as the outcome allows residual uncertainty about CAD protection by alcohol. Consequently there has been a point-counterpoint debate in the published medical reports (8–11).

### A National Study

The analysis reported by Mukamal et al. (12) in this issue of the *Journal* fits nicely into this context. This characteristically elegant presentation from a leading group in the alcohol epidemiology field confirms a U-shaped relationship between alcohol intake and CV mortality in a large nationwide study population. As expected, most of the apparent benefit in light–moderate drinkers is due to lower risk of CAD death.

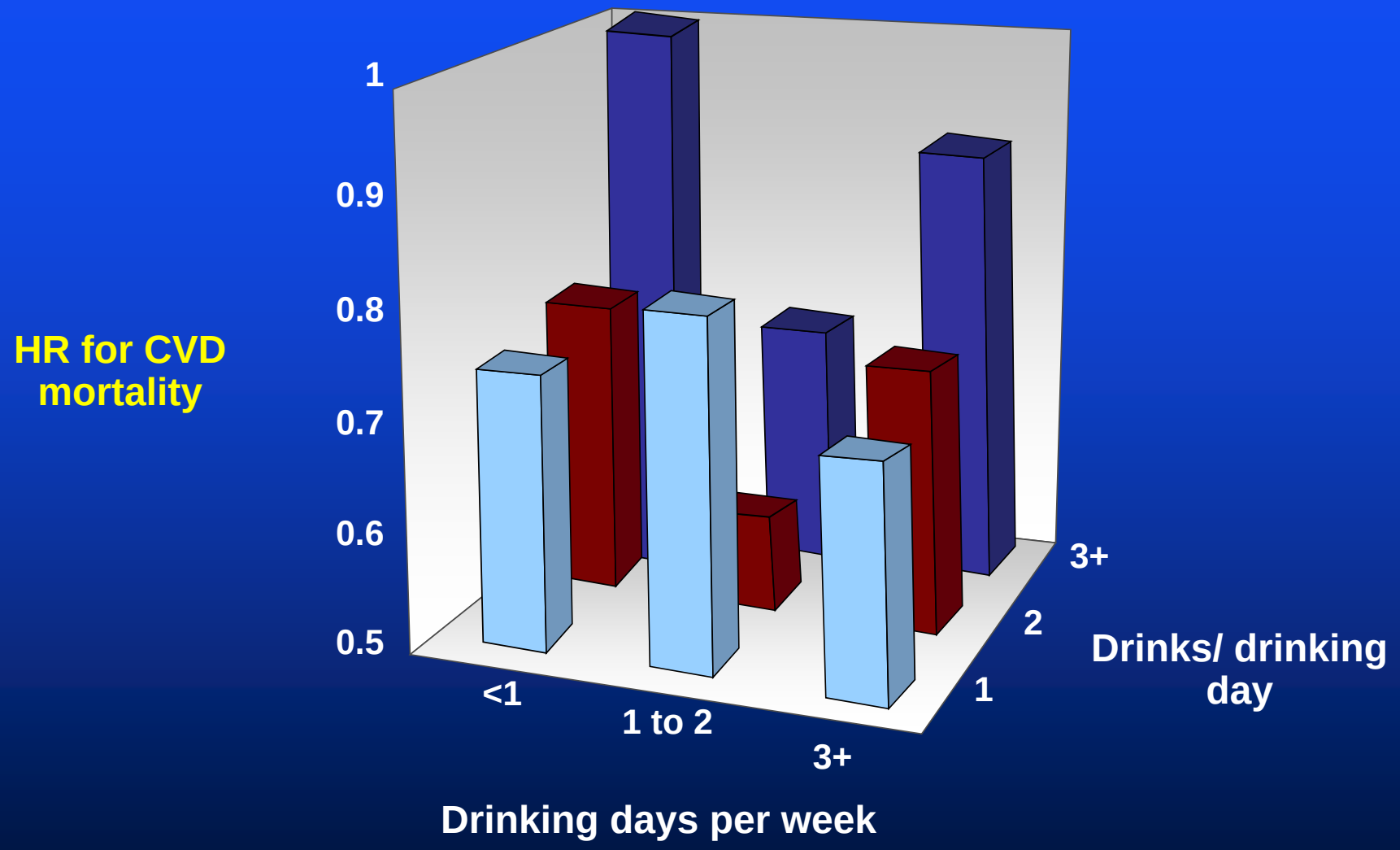
The analysis (12) adds to the case that the inverse relationship of light–moderate drinking to CV mortality is

# CV and Non-CV Deaths




Klatsky AL: JACC 55(13):1336, 2010

# Alcohol and Mortality




Cardiovascular mortality according to quantity and frequency of alcohol consumption-adjusted summary hazard ratios are shown relative to abstainers from alcohol

MAYO CLINIC

Mukamal: JACC, 2010

Journal of the American College of Cardiology Vol. 55, No. 13, 2010

**QUARTERLY FOCUS ISSUE: PREVENTION/OUTCOMES**

# Alcohol Consumption and Mortality in Patients With Cardiovascular Disease

## A Meta-Analysis

Simona Costanzo, ScD, Augusto Di Castelnuovo, ScD, Maria Benedetta Donati, MD, PhD, Licia Iacoviello, MD, PhD, Giovanni de Gaetano, MD, PhD

*Campobasso, Italy*

**Results** The meta-analysis on cardiovascular mortality showed a J-shaped pooled curve with a significant maximal protection (average 22%) by alcohol at approximately 26 g/day. In the meta-analysis on mortality for any cause, J-shaped pooled curves were observed in the overall analysis (average maximal protection of 18% in the range of 5 to 10 g/day) and in all subgroups according to either the type of patients or the characteristics of the studies.

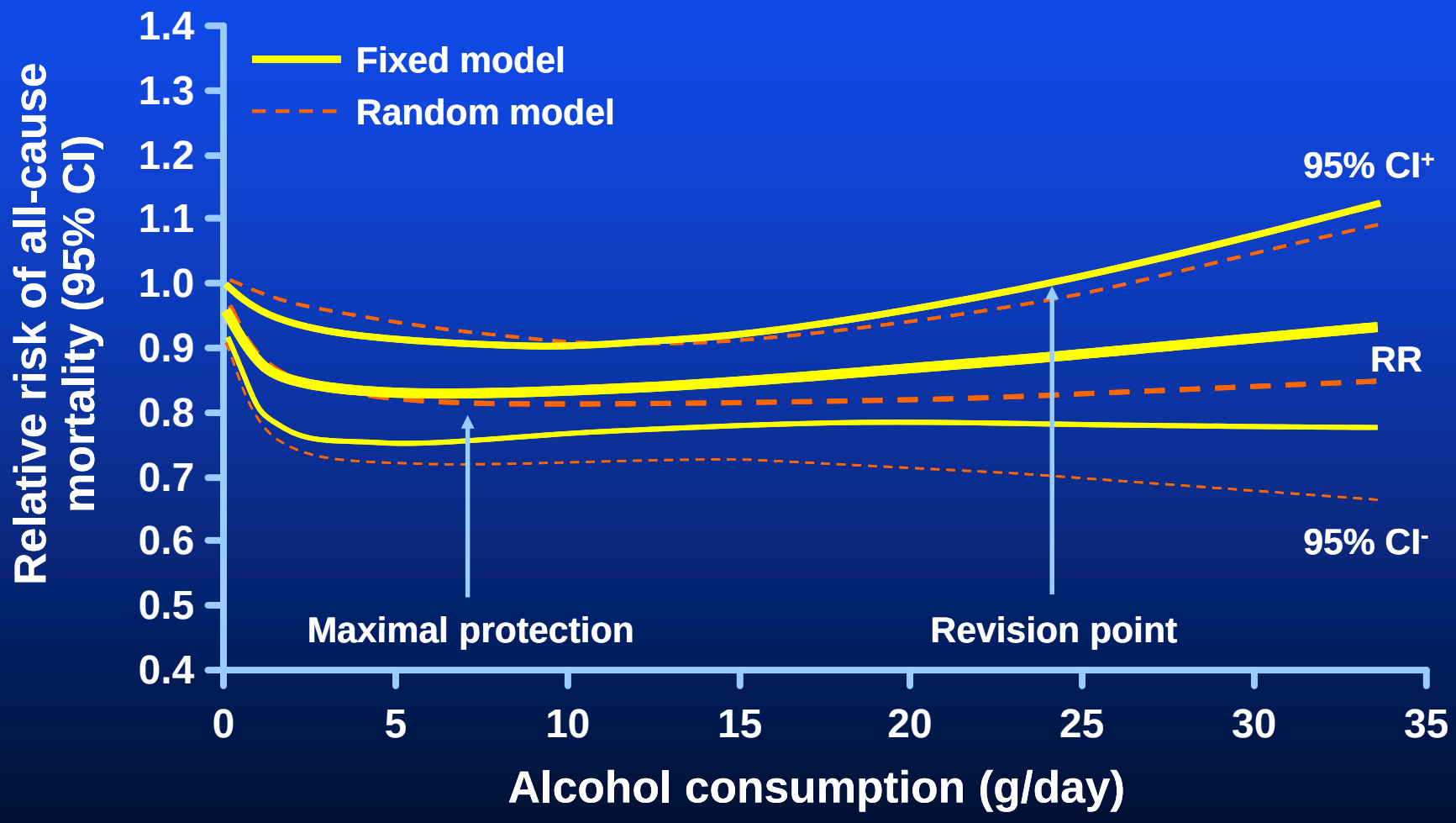
**Conclusions** In patients with cardiovascular disease, light to moderate alcohol consumption (5 to 25 g/day) was significantly associated with a lower incidence of cardiovascular and all-cause mortality. (J Am Coll Cardiol 2010;55: 1339–47) © 2010 by the American College of Cardiology Foundation

Moderate, regular alcohol consumption by apparently healthy people is associated with lower cardiovascular morbidity and mortality than in abstainers (1–6). Mechanisms supporting this include beneficial regulation of lipids and fibrinolysis, decreased platelet aggregation and coagulation factors, beneficial effects on endothelial function, and inflammation and insulin resistance (7–9). The proposed mechanisms of the beneficial role of drinking in moderation in healthy people may be similarly effective in people with a history of cardiovascular disease (CVD).

The abuse of alcohol is unquestionably harmful (2,3,6,10); in fact, the relationship between alcohol consumption and ischemic cardiovascular events or all-cause curve attributed to a dose-related combination of beneficial and harmful effects (1,11,12). The nonlinear J-shaped dose-response curve supports the hazards of excess drinking, but also indicates the potential windows of alcohol consumption that may confer a net beneficial effect, at least in terms of survival for apparently healthy subjects.

The 2006 Diet and Lifestyle Recommendations Scientific Statement from the American Heart Association Nutrition Committee (13) advises: "If you consume alcohol, do so in moderation (equivalent of no more than one drink for women or two drinks for men per day)." This is widely accepted within the scientific community, definitely when referring to healthy people. However, some concern has

# Alcohol Consumption in Relation to All-Cause Mortality in Cardiovascular Disease Patients




Costanzo S et al: JACC 55:1339, 2010

# Omega-3 Fish Oil

**AHA**

- everyone eat fish (3 oz) 2 x wk
- with CAD - take 1 gram fish oil - Omega-3 (EPA and DHA)

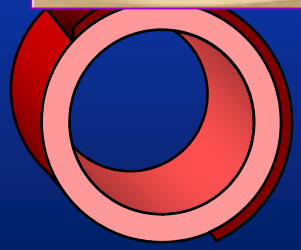
**Are fish oil supplements safe?**

- yes - top 16 brands
  - proper dose
  - no spoilage
  - no sig mercury, PCBs or Dioxin

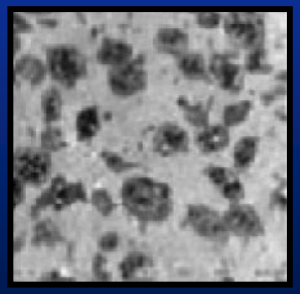
Consumer Reports, July 2003

# Desert: Health-Relevant Effect of Chocolate

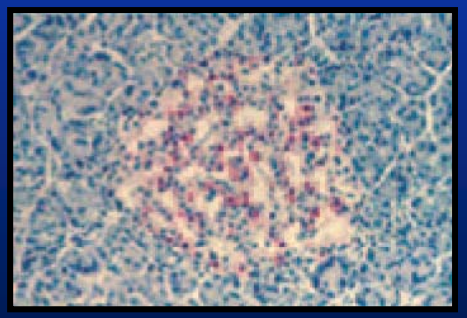
Blood pressure reduction

Improved vascular function



Reduced platelet reactivity



Improved insulin sensitivity

Anti depressant

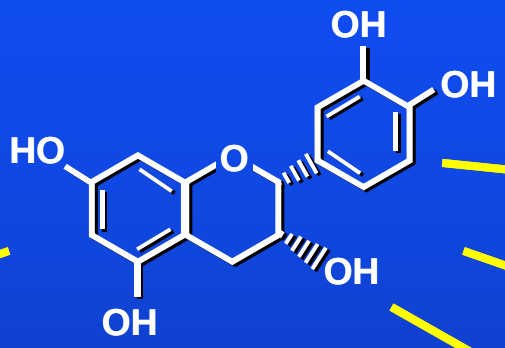
Other: anti-inflammatory

Mobilization of Functional EPCs

Modified from Corti et al: Circ, 2009

# Desert: Health-Relevant Effect of Chocolate

Blood pressure reduction

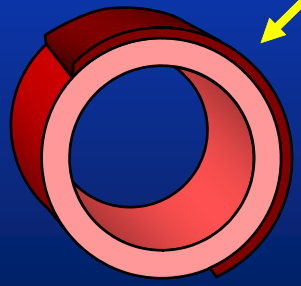



Epicatechins

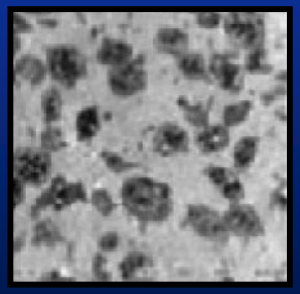
Anti depressant

Other: anti-inflammatory

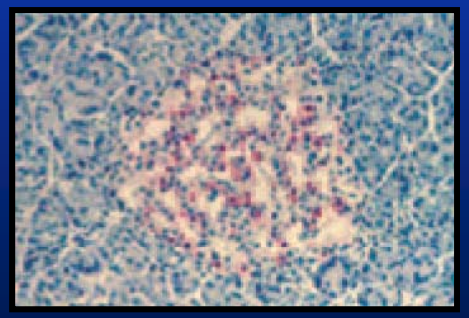
Mobilization of Functional EPCs



Improved vascular function



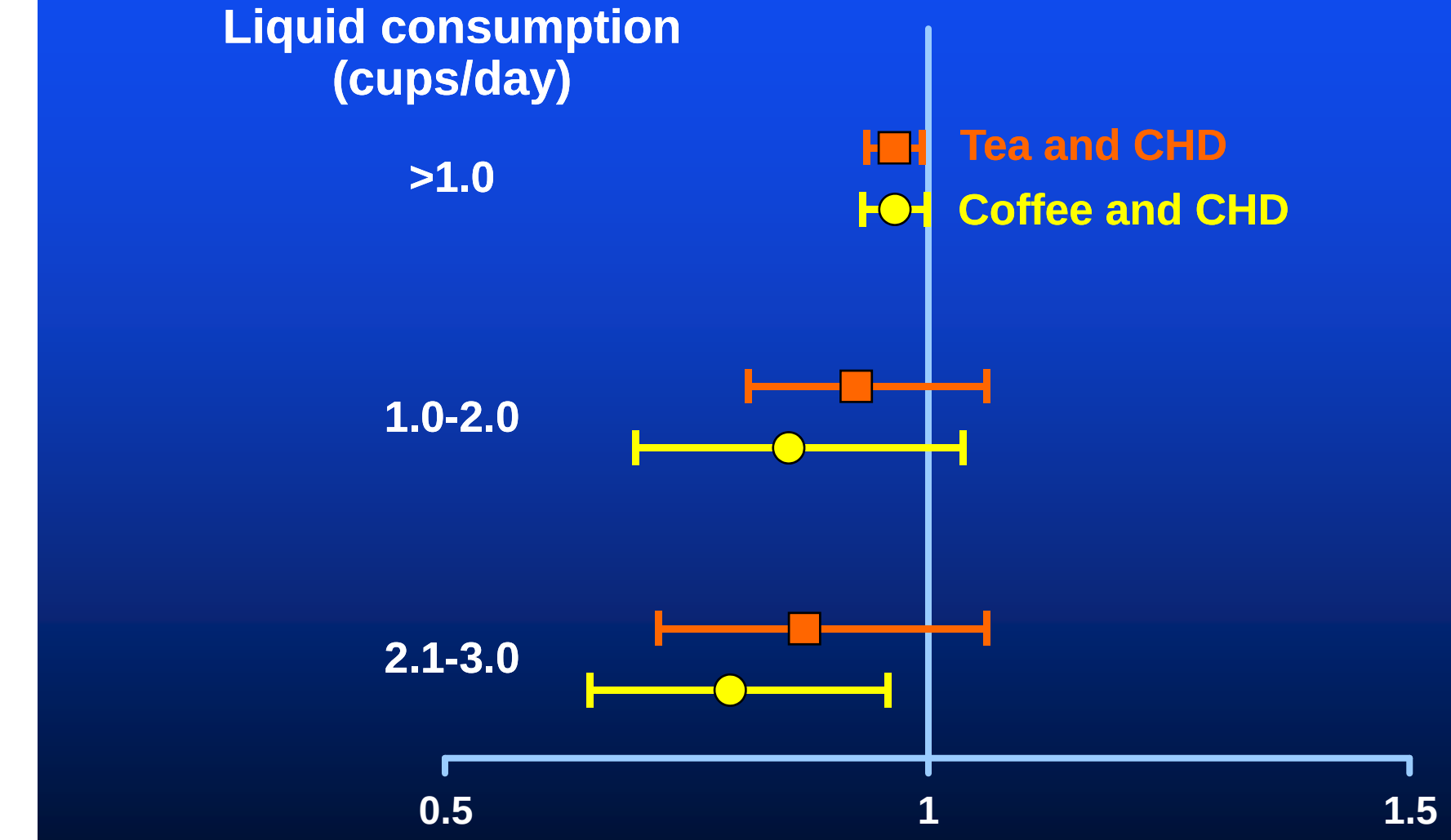
Reduced platelet reactivity



Improved insulin sensitivity

Modified from Corti et al: Circ, 2009

# Coffee or Tea Consumption and CHD



## *2. EFFECT OF INGREDIENTS OF POLYMEAL IN REDUCING RISK OF CVD*

| Ingredients | % Reduction (95% CI) in Risk of CVD | Source |
|---|---|---|
| Wine (150 ml/d) | 32 (23 to 41) | DiCastelnuovo, 2002 (MA) |
| Fish (114 g x 4 w) | 14 (8 to 19) | Whelton, 2004 (MA) |
| Dark Chocolate (100 g/d) | 21 (14 to 27) | Taubert, 2003 (RCT) |
| Fruit/Vegetables (400 g/d) | 21 (14 to 27) | John, 2002 (RCT) |
| Garlic (2.7 g/d) | 25 (21 to 27) | Ackerman, 2001 (MA) |
| Almonds (68 g/d) (RCT) | 12.5 (10.5 to 13.5) | Jenkins, Sabate. 2002,03 |
| **Combined Effect** | **76 (63 to 84)** | |

MA = meta-analysis; RCT = randomized controlled trial

OH Franco et al., BMJ 2004; 329:1447

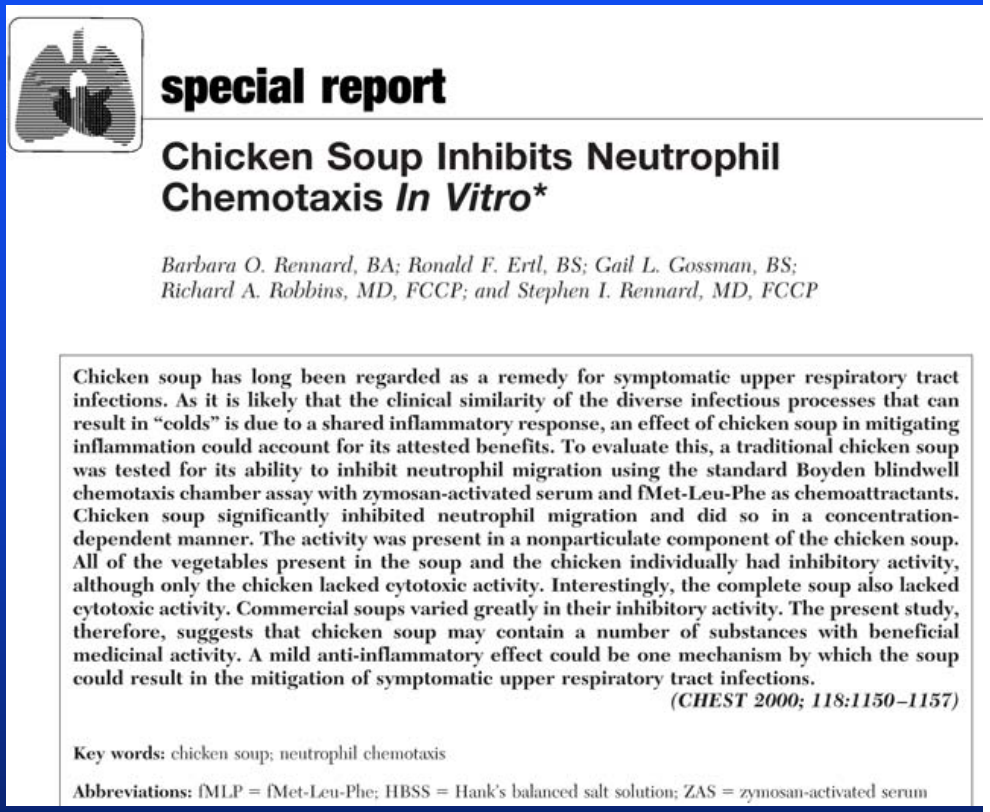
**Polypill - NJ Wald et al., BMJ 2003; 326:1419**

Statin, ASA, Folic Acid, BP (ACE-I, β-blocker, Thiazide) - **% Reduction 85%**

# Food for your Heart

## Menu

- The effect
- How much we eat ?
- What do we eat ?
  - Types of diets:
  - Epidemiology data
  - Prospective studies
  - Myth: Chicken soup…



special report

Chicken Soup Inhibits Neutrophil Chemotaxis *In Vitro**

*Barbara O. Rennard, BA; Ronald F. Ertl, BS; Gail L. Gossman, BS; Richard A. Robbins, MD, FCCP; and Stephen I. Rennard, MD, FCCP*

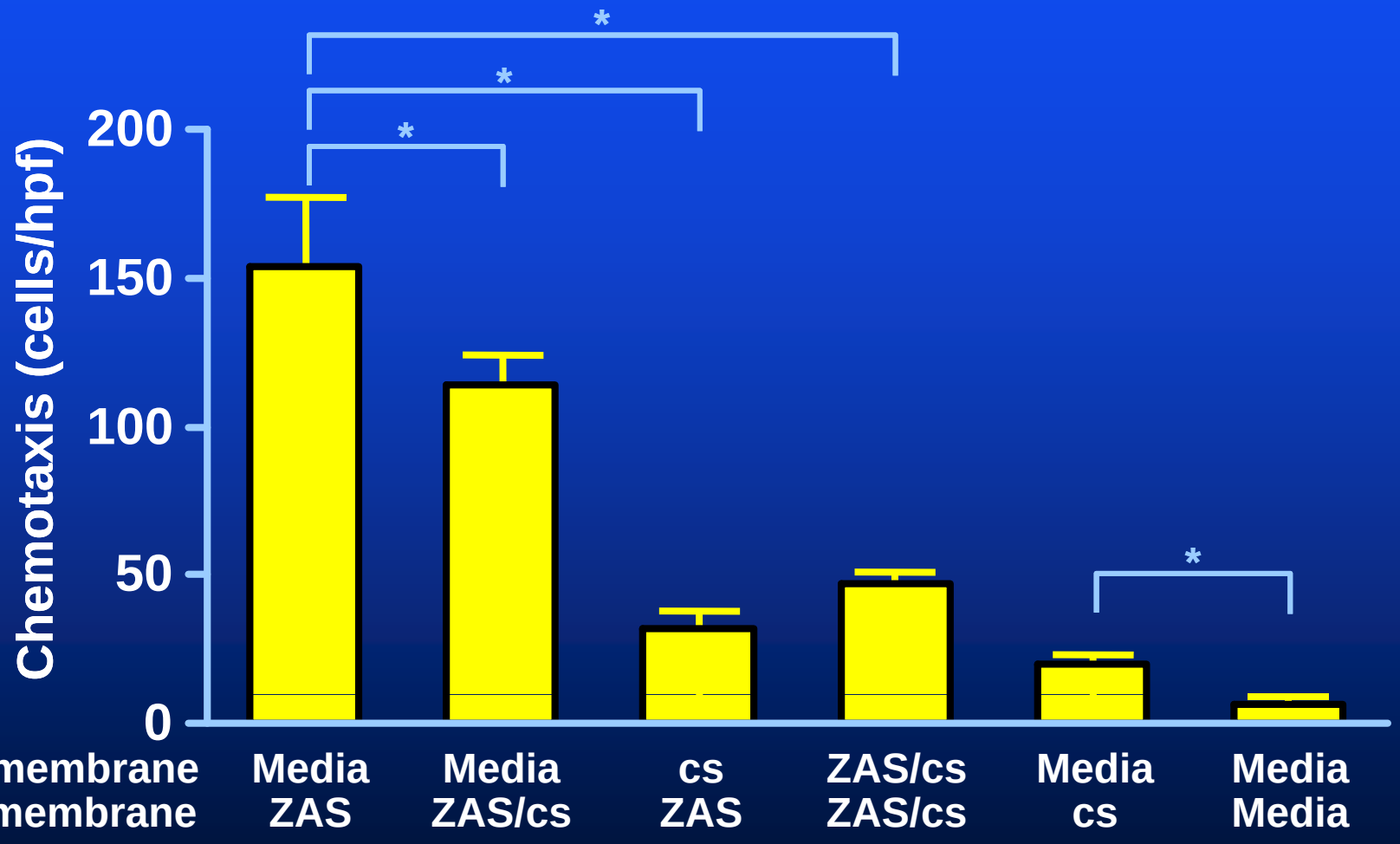
Chicken soup has long been regarded as a remedy for symptomatic upper respiratory tract infections. As it is likely that the clinical similarity of the diverse infectious processes that can result in "colds" is due to a shared inflammatory response, an effect of chicken soup in mitigating inflammation could account for its attested benefits. To evaluate this, a traditional chicken soup was tested for its ability to inhibit neutrophil migration using the standard Boyden blindwell chemotaxis chamber assay with zymosan-activated serum and fMet-Leu-Phe as chemoattractants. Chicken soup significantly inhibited neutrophil migration and did so in a concentration-dependent manner. The activity was present in a nonparticulate component of the chicken soup. All of the vegetables present in the soup and the chicken individually had inhibitory activity, although only the chicken lacked cytotoxic activity. Interestingly, the complete soup also lacked cytotoxic activity. Commercial soups varied greatly in their inhibitory activity. The present study, therefore, suggests that chicken soup may contain a number of substances with beneficial medicinal activity. A mild anti-inflammatory effect could be one mechanism by which the soup could result in the mitigation of symptomatic upper respiratory tract infections.

*(CHEST 2000; 118:1150–1157)*

**Key words:** chicken soup; neutrophil chemotaxis

**Abbreviations:** fMLP = fMet-Leu-Phe; HBSS = Hank's balanced salt solution; ZAS = zymosan-activated serum

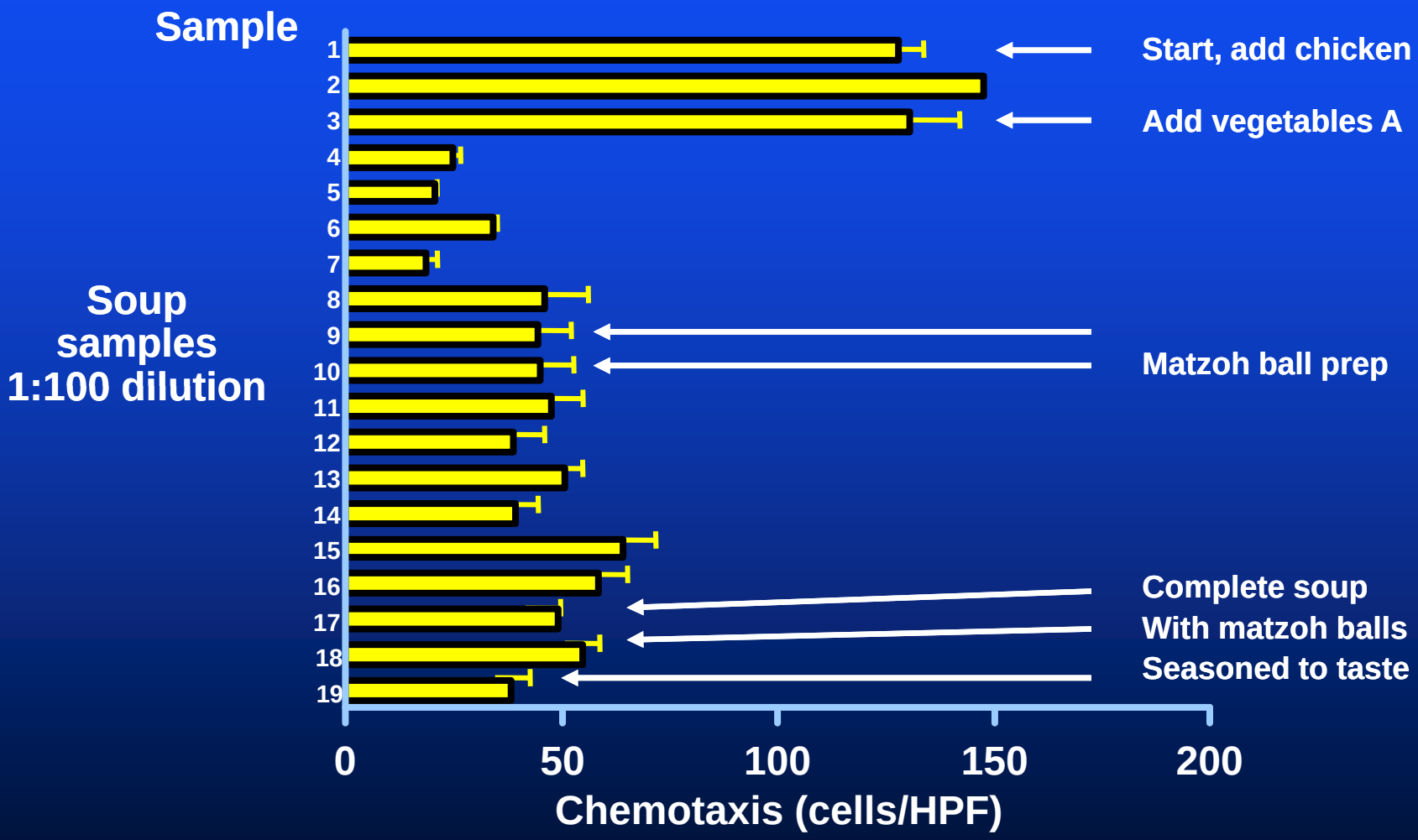
# Inhibition of Neutrophil Chemotaxis by Chicken Soup




Rennard et al: Chest 118:1150, 2000 *P<0.05

MAYO CLINIC

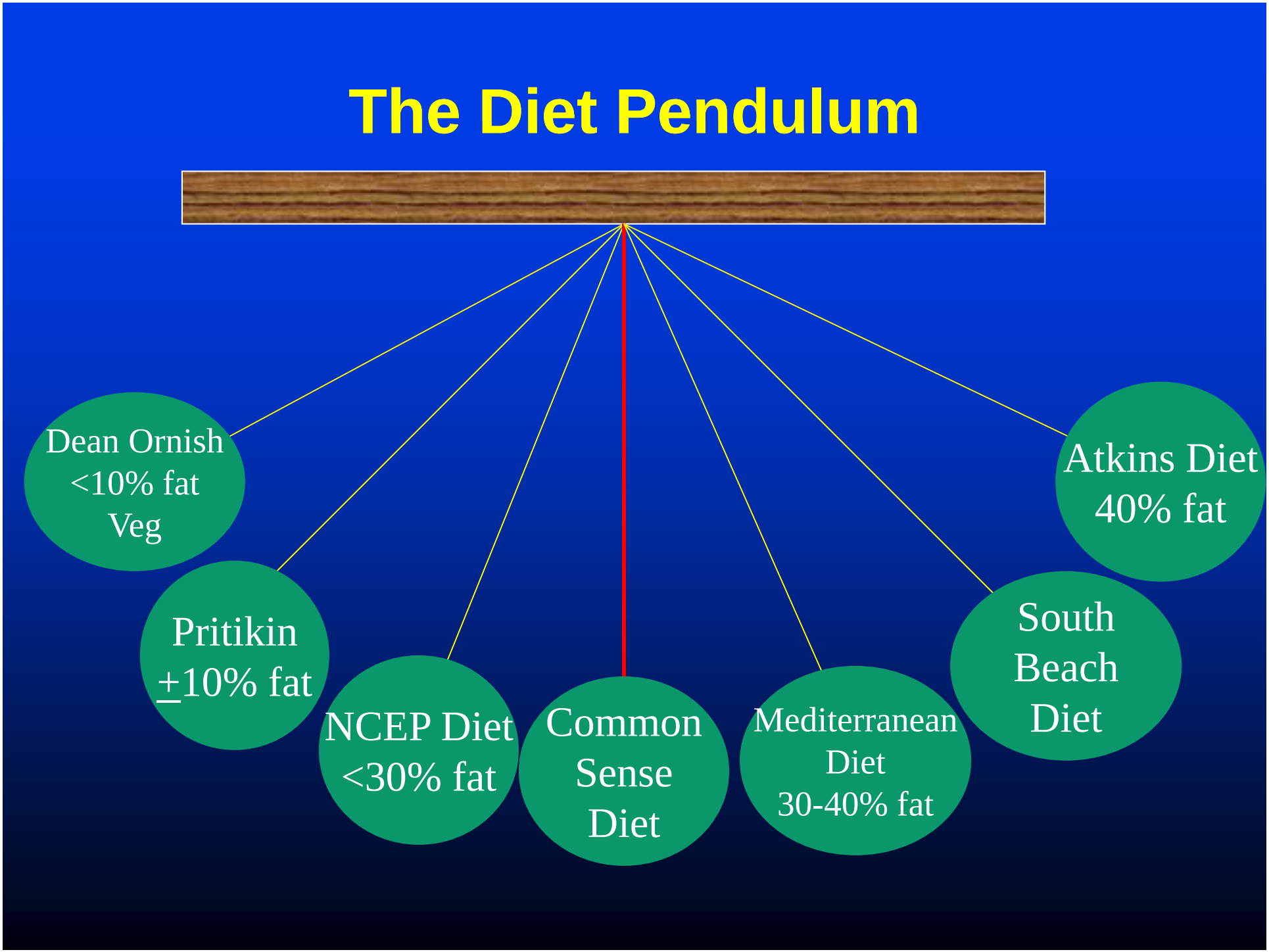
# Acquisition of Chemotactic Inhibitory Activity During Preparation of the Soup

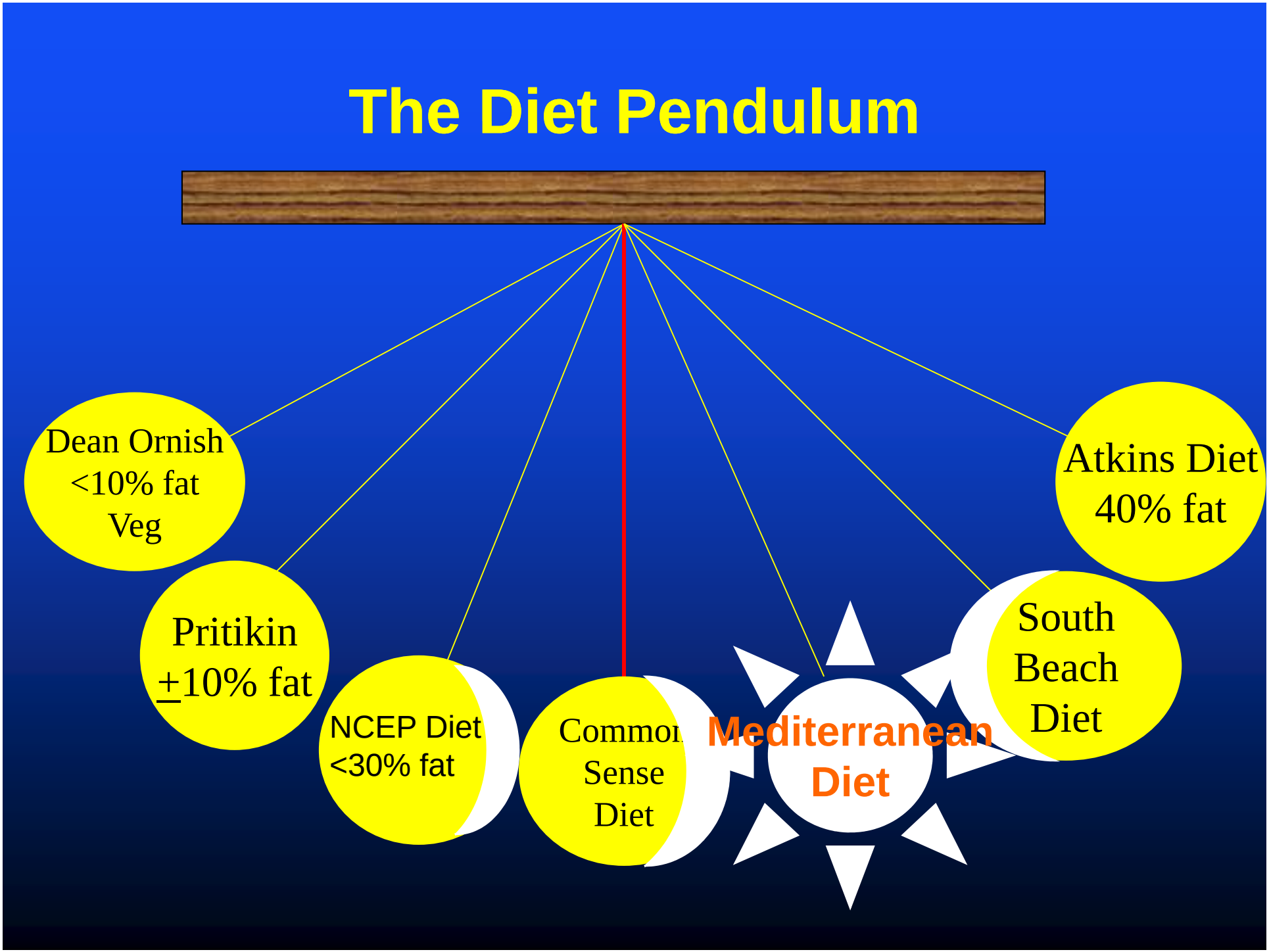



Rennard et al: Chest 118:1150, 2000

MAYO CLINIC

# The Diet Pendulum




The Diet Pendulum
Dean Ornish
<10% fat
Veg
Pritikin
±10% fat
NCEP Diet
<30% fat
Common
Sense
Diet
Mediterranean
Diet
South
Beach
Diet
Atkins Diet
40% fat

# Food for your Heart

## Menu

- The effect
- How much we eat ?
- What do we eat ?
  - Types of diets:
  - Epidemiology data
  - Prospective studies
  - Legend

# Torino & Mayo

M·E·N·U

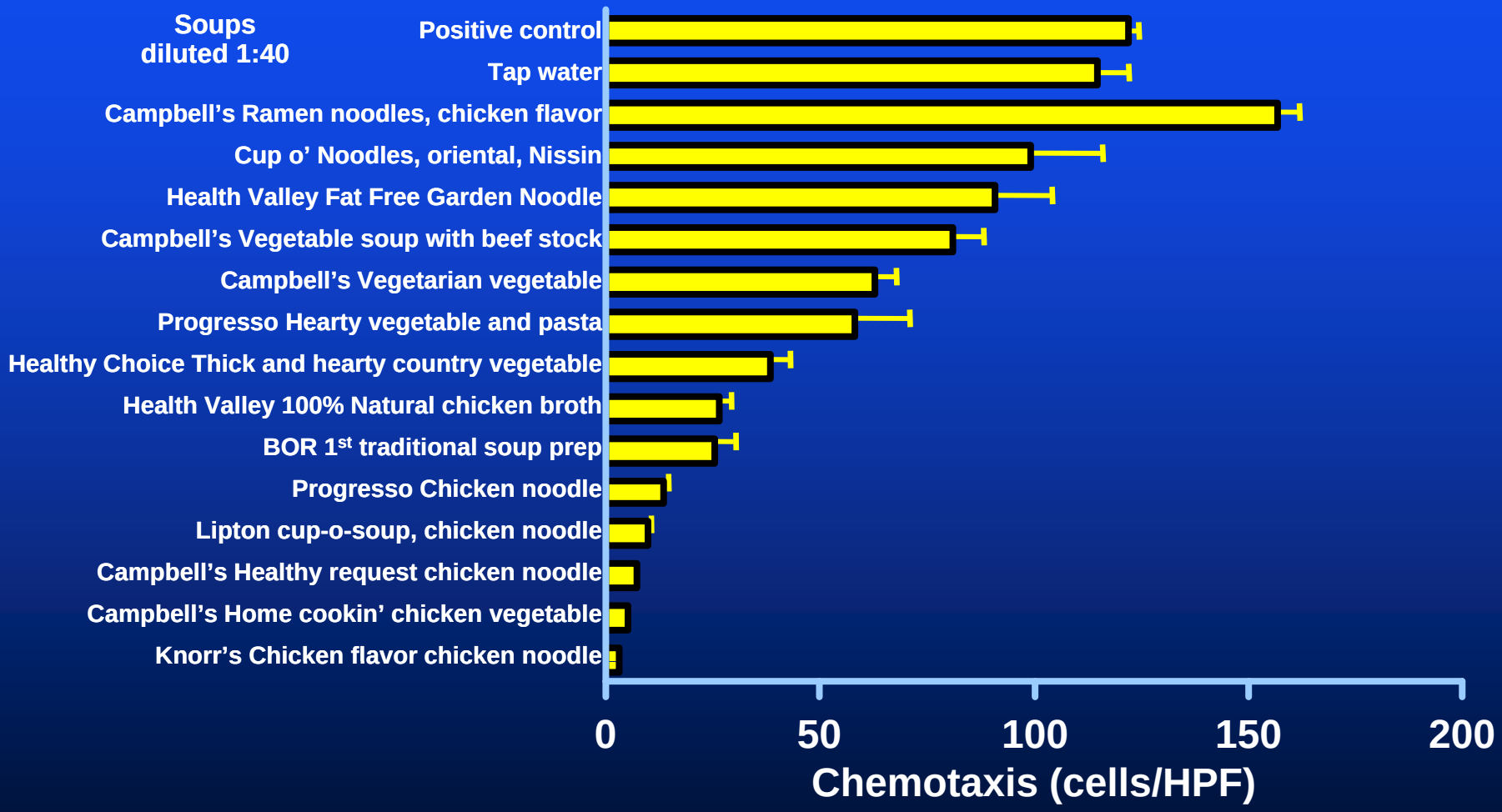
Appetizer

- *Drinks*
- *Appetizers*
- *Soup*
- *Entrée*
- *Desert*

IN CELEBRATION OF

# Torino &

# Commercially Available Soups and Neutrophil Chemotaxis




Rennard et al: Chest 118:1150, 2000

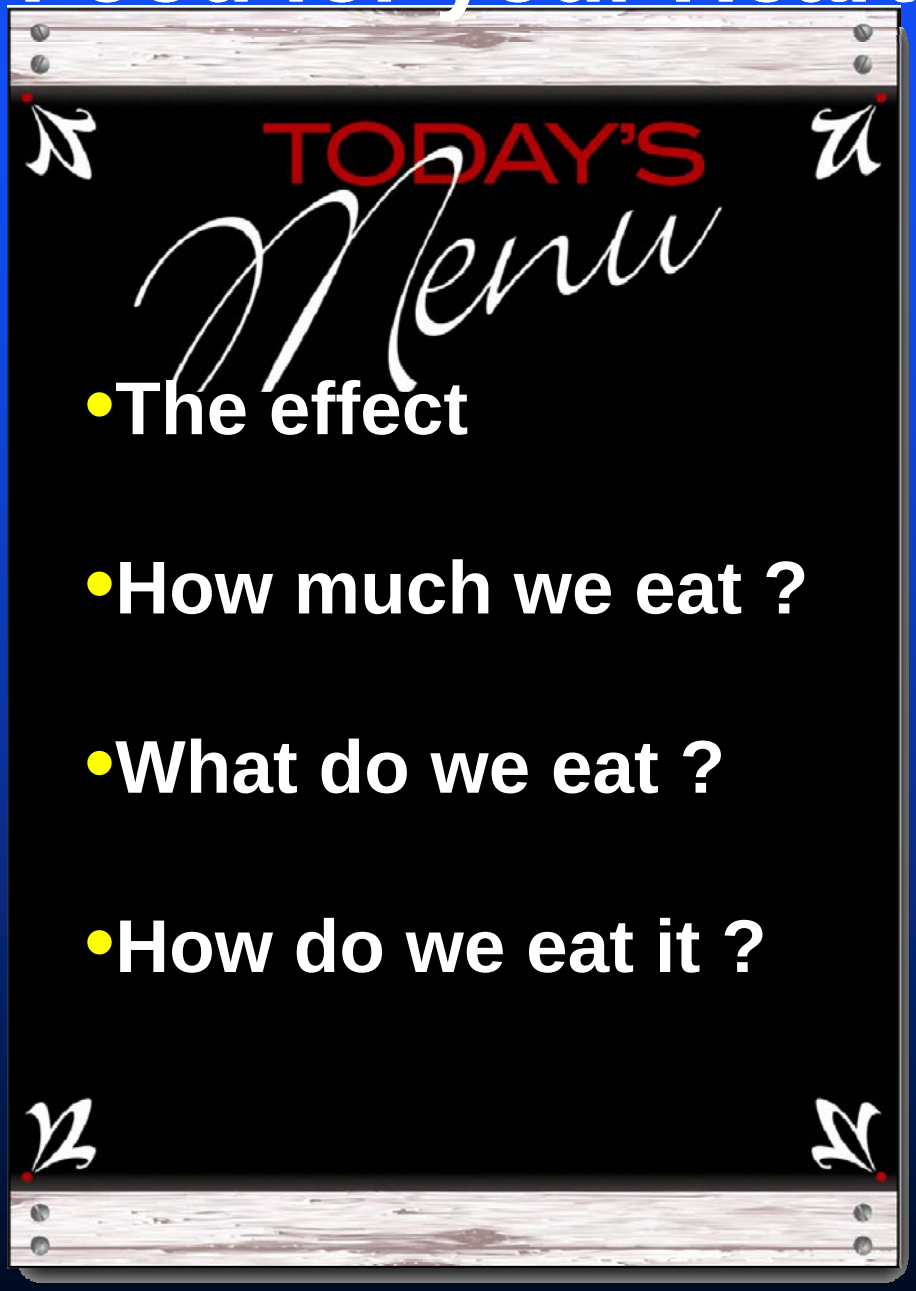
MAYO CLINIC

# The real Data…

| Food Group | Epi Data | RCT Data | CVD Effect | Proposed Mechanism(s) |
|---|---|---|---|---|
| **Mediterranean Diet** | **+++** | **+++** | **++** | **Improved lipids, anti-oxidants, endothelial function, platelet aggregation** |
| **MUFA/PUFA** | **++** | **+/-** | **NC** | **Mixed data on improved lipid profiles and/or endothelial function** |
| **Fish** | **+/-** | **++** | **+** | **Improves lipid profiles and BP** |
| **Olive Oil** | **+** | **+/-** | **NC** | **Improves lipid profiles, BP, and endothelial function** |
| **Plant-Based Foods** | **+++** | **++** | **+/-** | **Improves lipid profiles, BP, and endothelial function; reduces ROS** |
| **Fiber** | **++** | **+** | **+** | **Improves lipid profiles, BP, and endothelial function; reduces ROS** |
| **Garlic** | **+/-** | **+/-** | **NC** | **Improves lipid profiles** |
| **Green Tea** | **++** | **+** | **+** | **Reduced ROS; improve endothelial function** |
| **Alcohol** | **++** | **+** | **++** | **Improves lipid profiles, BP, and endothelial function; +/- impact on ROS** |
| **Nuts** | **++** | **++** | **+** | **+/- impact on ROS and lipid profiles; improves BP** |
| **Chocolate** | **++** | **++** | **++** | **Improves lipid profiles, BP, and endothelial function; reduces ROS** |
| **“DASH” Diet** | **+++** | **+++** | **++** | **Improves BP and lipid profiles** |
| **Low Glycemic Diet** | **+** | **+** | **+** | **Improves BP** |
| **Low Calorie Diets** | **???** | **???** | **???** | **Weight loss = improved cardiac health?** |

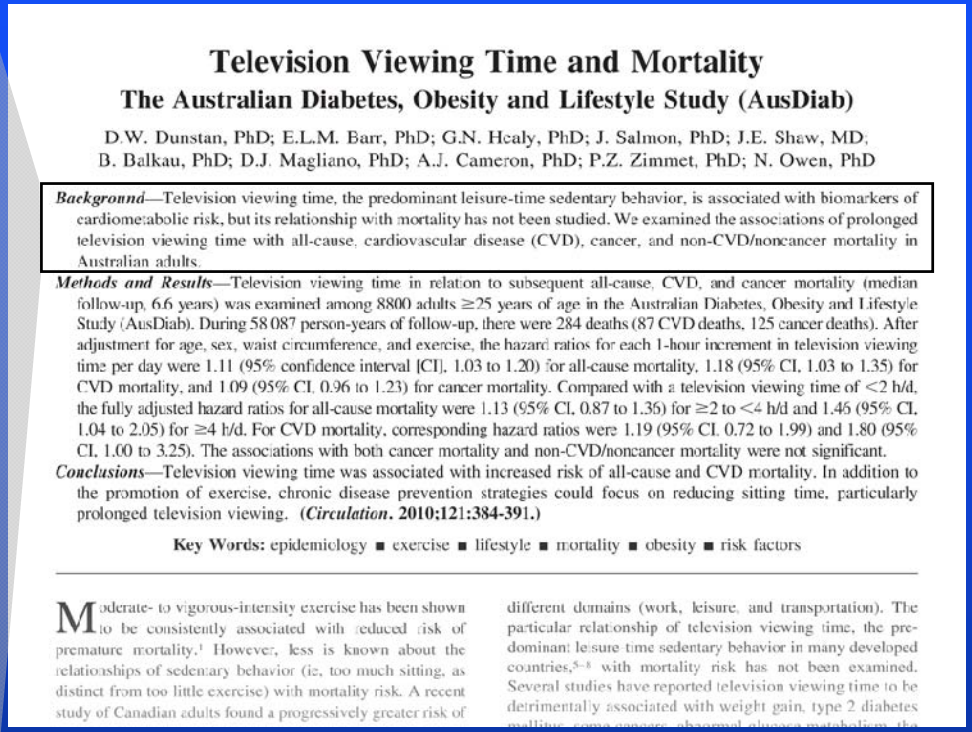
# Food for your Heart

# Food for your Heart




- The effect
- How much we eat ?
- What do we eat ?
- How do we eat it ?

**Background – Television viewing time, the predominant leisure-time sedentary behavior, is associated with biomarkers of cardiometabolic risk, but its relationship with mortality has not been studied. We examined the associations of prolonged television viewing time with all-cause, cardiovascular disease (CVD), cancer, and non-CVD/noncancer mortality in Australian adults.**



# Television Viewing Time and Mortality

## The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)

D.W. Dunstan, PhD; E.L.M. Barr, PhD; G.N. Healy, PhD; J. Salmon, PhD; J.E. Shaw, MD; B. Balkau, PhD; D.J. Magliano, PhD; A.J. Cameron, PhD; P.Z. Zimmet, PhD; N. Owen, PhD

***Background***—Television viewing time, the predominant leisure-time sedentary behavior, is associated with biomarkers of cardiometabolic risk, but its relationship with mortality has not been studied. We examined the associations of prolonged television viewing time with all-cause, cardiovascular disease (CVD), cancer, and non-CVD/noncancer mortality in Australian adults.

***Methods and Results***—Television viewing time in relation to subsequent all-cause, CVD, and cancer mortality (median follow-up, 6.6 years) was examined among 8800 adults ≥25 years of age in the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). During 58 087 person-years of follow-up, there were 284 deaths (87 CVD deaths, 125 cancer deaths). After adjustment for age, sex, waist circumference, and exercise, the hazard ratios for each 1-hour increment in television viewing time per day were 1.11 (95% confidence interval [CI], 1.03 to 1.20) for all-cause mortality, 1.18 (95% CI, 1.03 to 1.35) for CVD mortality, and 1.09 (95% CI, 0.96 to 1.23) for cancer mortality. Compared with a television viewing time of <2 h/d, the fully adjusted hazard ratios for all-cause mortality were 1.13 (95% CI, 0.87 to 1.35) for ≥2 to <4 h/d and 1.45 (95% CI, 1.04 to 2.05) for ≥4 h/d. For CVD mortality, corresponding hazard ratios were 1.19 (95% CI, 0.72 to 1.99) and 1.80 (95% CI, 1.00 to 3.25). The associations with both cancer mortality and non-CVD/noncancer mortality were not significant.

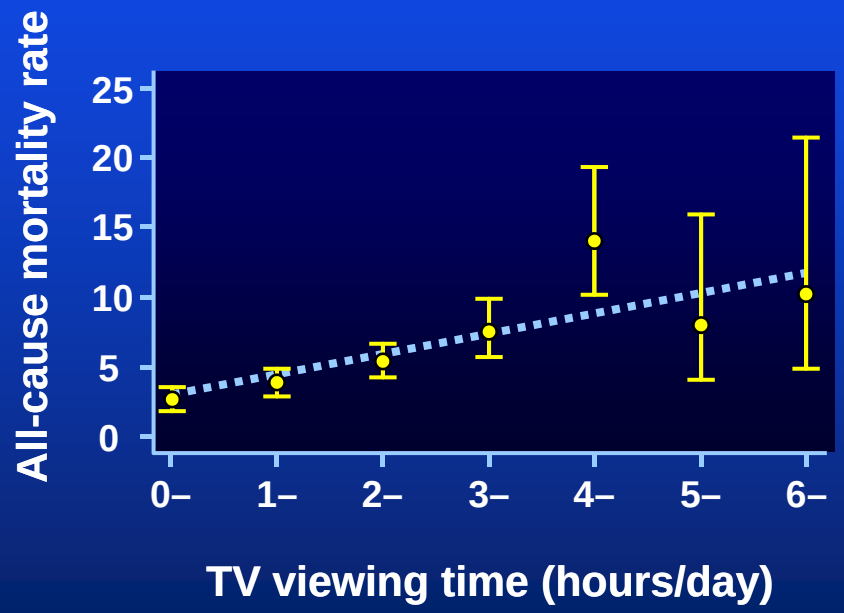
***Conclusions***—Television viewing time was associated with increased risk of all-cause and CVD mortality. In addition to the promotion of exercise, chronic disease prevention strategies could focus on reducing sitting time, particularly prolonged television viewing. (***Circulation***. **2010;121:384-391.**)

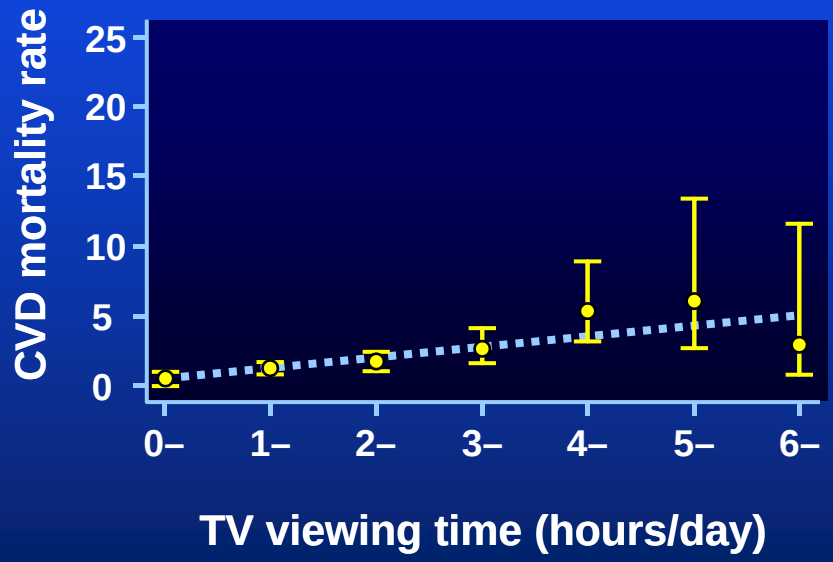
**Key Words:** epidemiology ■ exercise ■ lifestyle ■ mortality ■ obesity ■ risk factors

Moderate- to vigorous-intensity exercise has been shown to be consistently associated with reduced risk of premature mortality.[1] However, less is known about the relationships of sedentary behavior (ie, too much sitting, as distinct from too little exercise) with mortality risk. A recent study of Canadian adults found a progressively greater risk of

different domains (work, leisure, and transportation). The particular relationship of television viewing time, the predominant leisure time sedentary behavior in many developed countries,[5–8] with mortality risk has not been examined. Several studies have reported television viewing time to be detrimentally associated with weight gain, type 2 diabetes

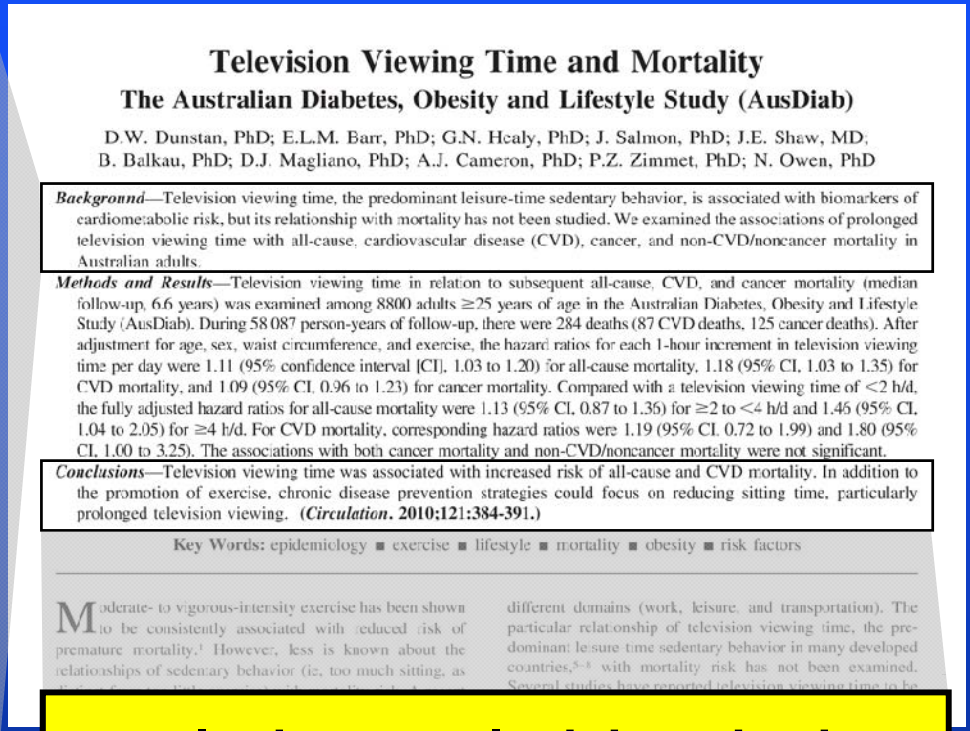
# Unadjusted All-Cause CVD Mortality Rates per 1,000 Person-Years According to Television Viewing Time




Dunstan et al: Circ 121:384, 2010

**Background – Television viewing time, the predominant leisure-time sedentary behavior, is associated with biomarkers of cardiometabolic risk, but its relationship with mortality has not been studied. We examined the associations of prolonged television viewing time with all-cause, cardiovascular disease (CVD), cancer, and non-CVD/noncancer mortality in Australian adults.**



## Television Viewing Time and Mortality

### The Australian Diabetes, Obesity and Lifestyle Study (AusDiab)

D.W. Dunstan, PhD; E.L.M. Barr, PhD; G.N. Healy, PhD; J. Salmon, PhD; J.E. Shaw, MD; B. Balkau, PhD; D.J. Magliano, PhD; A.J. Cameron, PhD; P.Z. Zimmet, PhD; N. Owen, PhD

***Background***—Television viewing time, the predominant leisure-time sedentary behavior, is associated with biomarkers of cardiometabolic risk, but its relationship with mortality has not been studied. We examined the associations of prolonged television viewing time with all-cause, cardiovascular disease (CVD), cancer, and non-CVD/noncancer mortality in Australian adults.

***Methods and Results***—Television viewing time in relation to subsequent all-cause, CVD, and cancer mortality (median follow-up, 6.6 years) was examined among 8800 adults ≥25 years of age in the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). During 58 087 person-years of follow-up, there were 284 deaths (87 CVD deaths, 125 cancer deaths). After adjustment for age, sex, waist circumference, and exercise, the hazard ratios for each 1-hour increment in television viewing time per day were 1.11 (95% confidence interval [CI], 1.03 to 1.20) for all-cause mortality, 1.18 (95% CI, 1.03 to 1.35) for CVD mortality, and 1.09 (95% CI, 0.96 to 1.23) for cancer mortality. Compared with a television viewing time of <2 h/d, the fully adjusted hazard ratios for all-cause mortality were 1.13 (95% CI, 0.87 to 1.35) for ≥2 to <4 h/d and 1.45 (95% CI, 1.04 to 2.05) for ≥4 h/d. For CVD mortality, corresponding hazard ratios were 1.19 (95% CI, 0.72 to 1.99) and 1.80 (95% CI, 1.00 to 3.25). The associations with both cancer mortality and non-CVD/noncancer mortality were not significant.

***Conclusions***—Television viewing time was associated with increased risk of all-cause and CVD mortality. In addition to the promotion of exercise, chronic disease prevention strategies could focus on reducing sitting time, particularly prolonged television viewing. (***Circulation***. **2010;121:384-391.**)

**Key Words:** epidemiology ■ exercise ■ lifestyle ■ mortality ■ obesity ■ risk factors

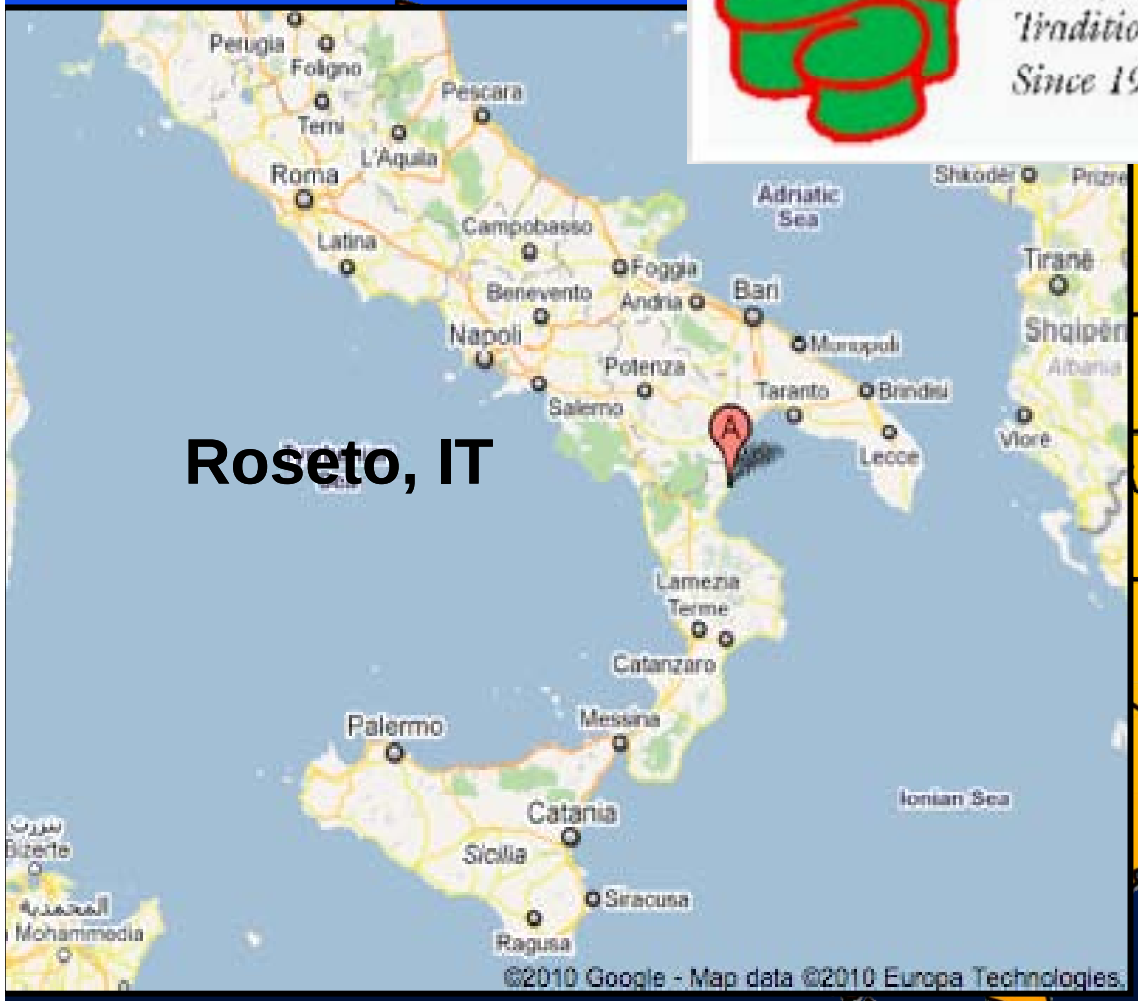
Moderate- to vigorous-intensity exercise has been shown to be consistently associated with reduced risk of premature mortality.[1] However, less is known about the relationships of sedentary behavior (ie, too much sitting, as different domains (work, leisure, and transportation). The particular relationship of television viewing time, the predominant leisure time sedentary behavior in many developed countries,[5–8] with mortality risk has not been examined. Several studies have reported television viewing time to be

**Conclusions – television viewing time was associated with increased risk of all-cause and CVD mortality. In additional to the promotion of exercise, chronic disease provention strategies could focus on reducing sitting time, particularly prolonged television viewing.**

ROSETO

*Family Values*
*Traditional Spirit*
*Since 1912*

**Roseto, PA**




**Roseto, IT**

MAYO CLINIC

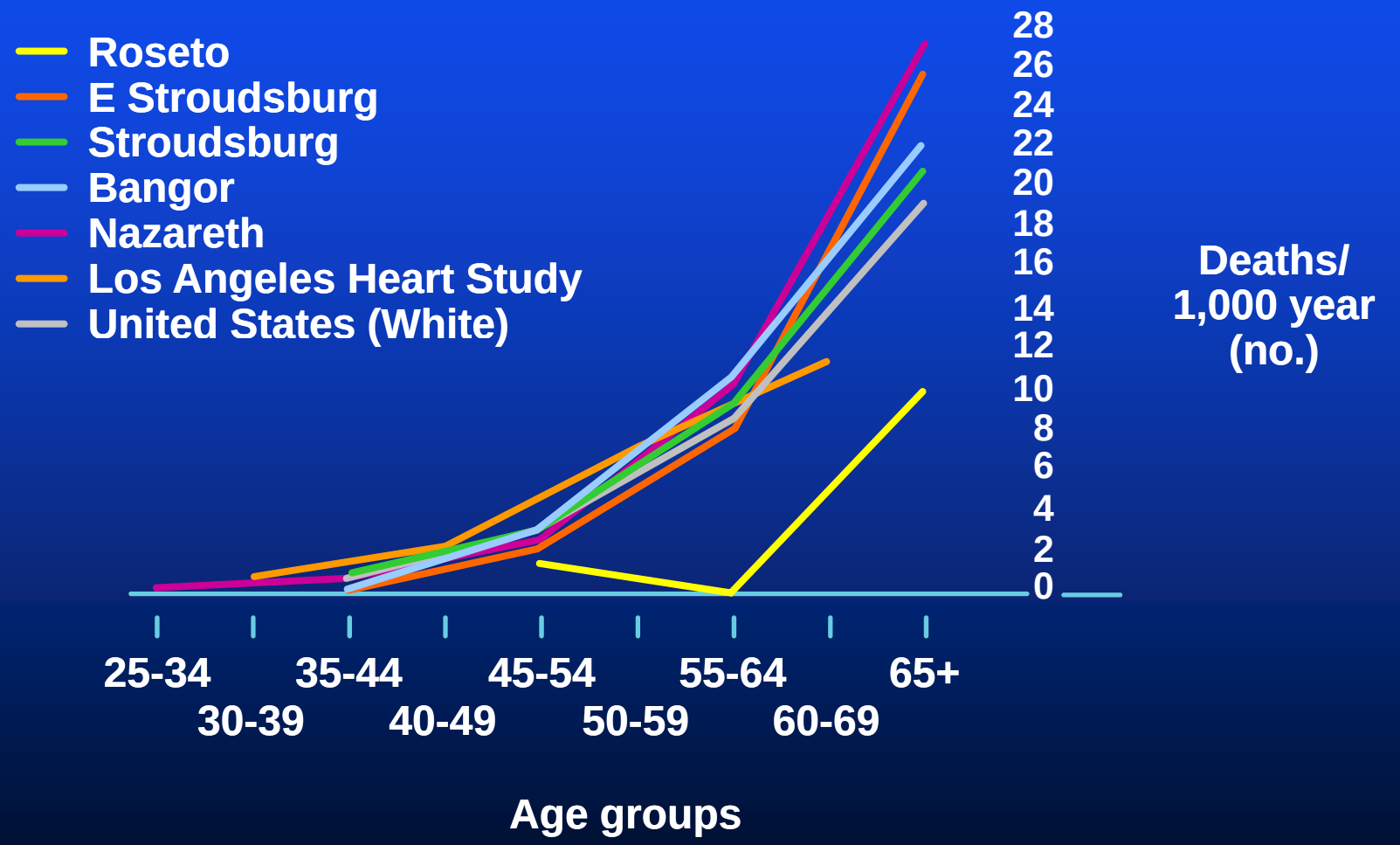
# Predictors of Myocardial Infarction Over a Span of 30 Years in Roseto, Pennsylvania

STEWART WOLF

**Abstract**—Predictors of myocardial infarction with or without survival were sought in a 30-year study of Roseto, Pennsylvania, a nearly exclusively Italian community of approximately 1,600, compared to the immediately adjacent town of Bangor with a population of approximately 5,000. At the start of the study the death rate from myocardial infarction among men in Roseto was less than half that in Bangor despite an equal prevalence of the usual risk factors, mainly smoking and diet. The communities were followed prospectively for 30 years during a striking social change in Roseto toward less family and community cohesion and more commitment to individual goals and adherence to materialistic values. During this period the prevalence of and mortality from myocardial infarction increased sharply to equal the situation in Bangor. The predictive values of measurements made of Rosetans during individual examinations in 1962–63 were tested against the outcome in 1990. Those who experienced fatal myocardial infarction and those who had a well documented infarction and survived were matched with and compared to controls. Although subjects with cholesterol concentration above 200 were twice as likely to experience myocardial infarction as those with concentrations below 200, less than 20% of those whose cholesterol concentration was above 200 experienced any evidence of myocardial infarction over the nearly 30-year period. Moreover, there were no significant differences between the coronary patients, with or without survival, and their sex, age, and cholesterol matched controls; nor were smoking, evidence of hypertension, diabetes, or obesity predictive of significant differences between the two groups. These data lead to the inference that while those with the conventional risk factors are more likely to develop myocardial infarction than are those without the risk factors, an even larger proportion of the population may have the risk factors and not succumb to myocardial infarction over a period of nearly three decades.

*Integrative Physiological and Behavioral Science*, July-September, 1992, Vol. 27, No. 3, 246-257

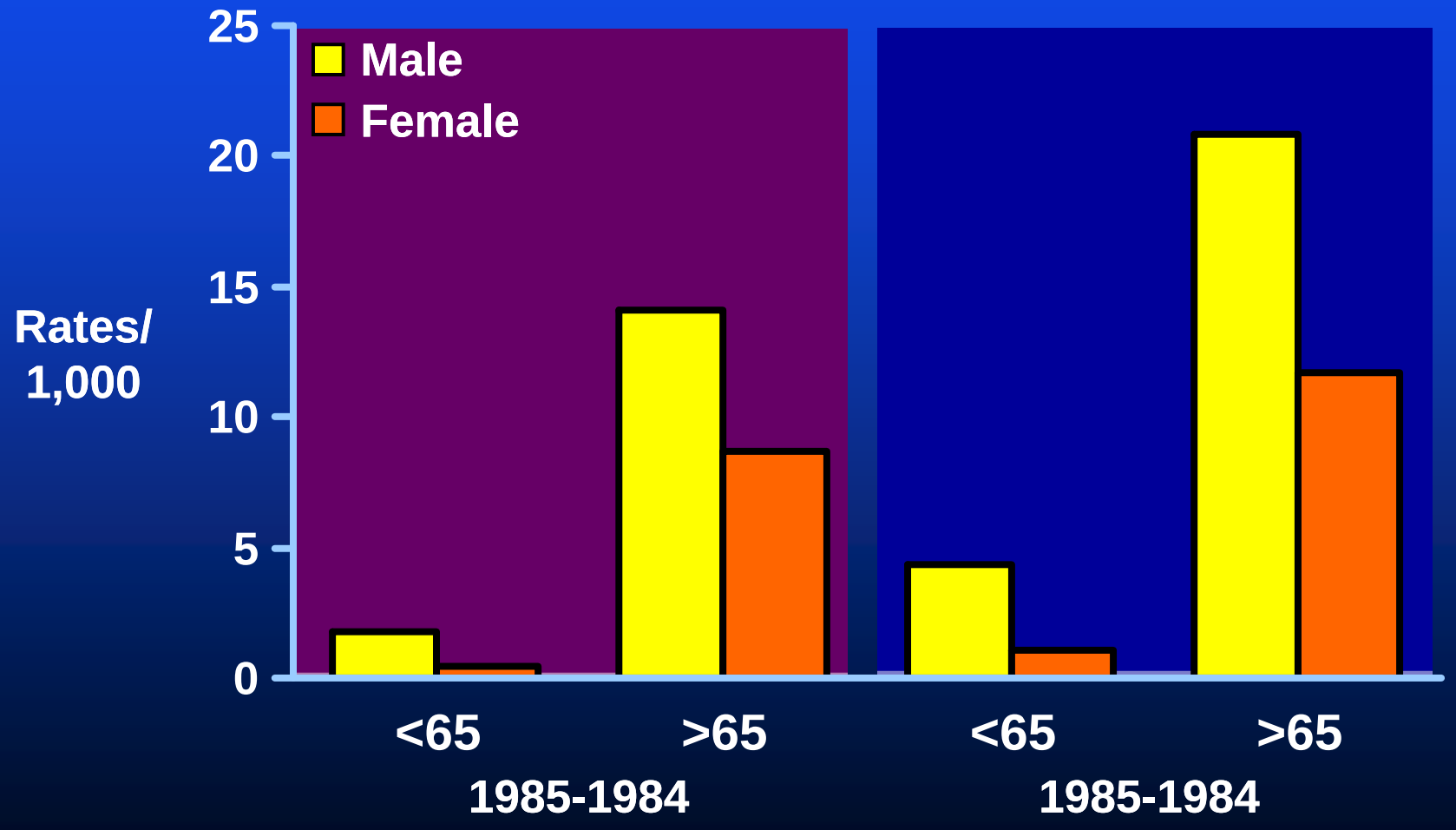
# Comparison of Death Rates from Coronary Heart Disease in Males




MAYO CLINIC

# Mortality Rtes/1,000 from Myocardial Infarction

## Roseto




MAYO CLINIC

# Bon Appetite