CHOLESTEROL IS NOT AN IMPORTANT RISK FACTOR FOR HEART DISEASE AND CURRENT DIETARY RECOMMENDATIONS DO MORE HARM THAN GOOD

@ProfTimNoakes

Slides available on www.health.uct.ac.za

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MRC/UCT Research Unit for Exercise Science and Sports Medicine,
University of Cape Town and
Sports Science Institute of South Africa

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FATS, CARBS, AND THE
SCIENCE OF DIET

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*"Saturated fat and cholester
cause of coronary heart dis
greatest scientific deception o
any century."*

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Economic considerations drove the adoption of the current dietary guidelines without proper scientific evaluation or proof.

Within 5 years of the widespread adoption of these guidelines rates of diabetes and obesity increased explosively.

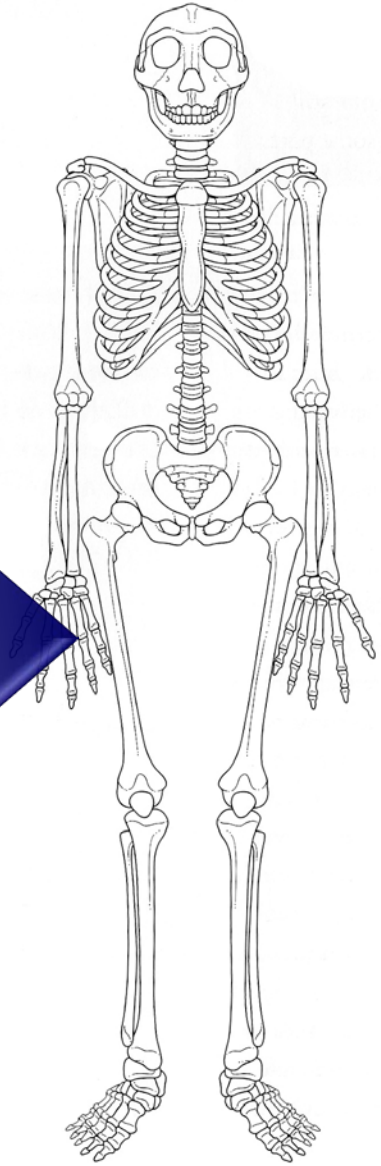
The presence of the genetic predisposing condition, carbohydrate-resistance, explains why large numbers of persons in predisposed populations become obese and diabetic when exposed to a high carbohydrate diet.

A high fat diet reverses all known coronary risk factors in persons with carbohydrate-resistance whereas a high carbohydrate diet worsens those factors.

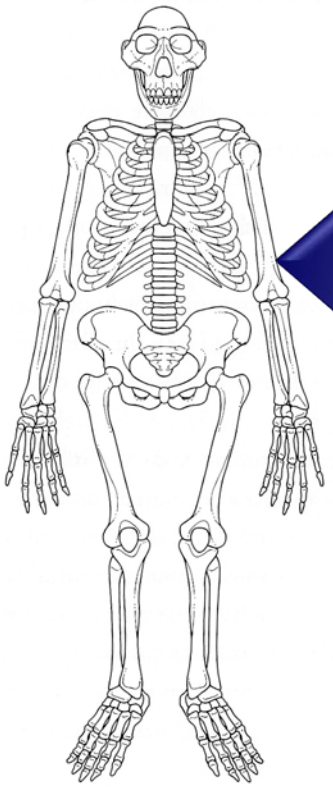
TODAY

2.5 - 3.5 MILLION YEARS AGO

Omnivore



Homo sapiens

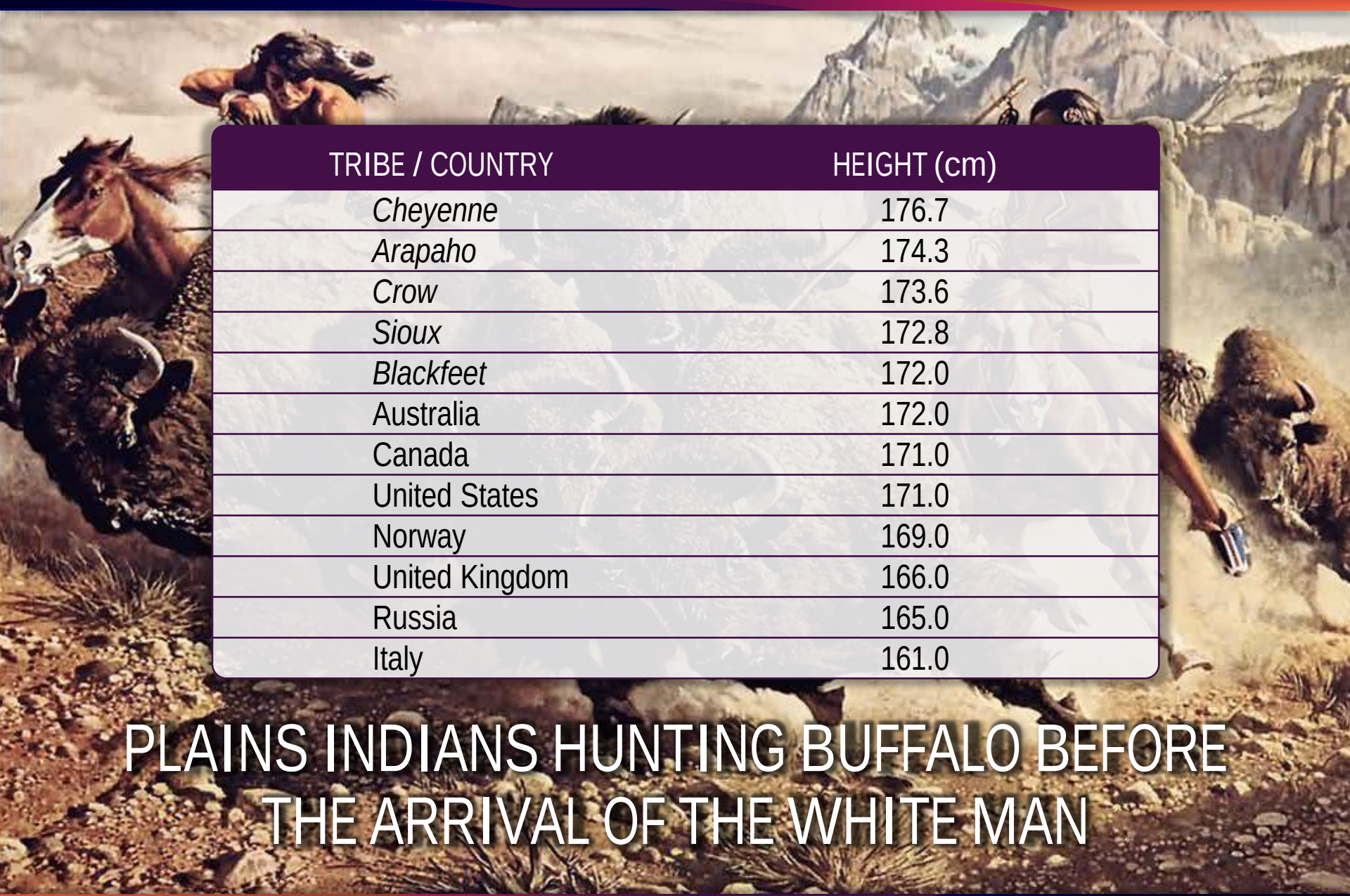


Australopithecus Africanus

Vegetarian

This change occurred as humans became the best mid-day persistence hunters in the animal kingdom

For 3.5 million years we have done very well without being told what we should eat



TRIBE / COUNTRY	HEIGHT (cm)
Cheyenne	176.7
Arapaho	174.3
Crow	173.6
Sioux	172.8
Blackfeet	172.0
Australia	172.0
Canada	171.0
United States	171.0
Norway	169.0
United Kingdom	166.0
Russia	165.0
Italy	161.0

PLAINS INDIANS HUNTING BUFFALO BEFORE THE ARRIVAL OF THE WHITE MAN

Steckel RH, Prince JM. Tallest in the world: Native Americans of the Great Plains in the nineteenth century. Am Econ Rev 2001; 91: 287-294.

MODERN PLAINS INDIANS HUNTING THE WHITE MAN'S DIET

THE COUNTRIES WITH THE HIGHEST PERCENTAGE OF OBESE ADULTS

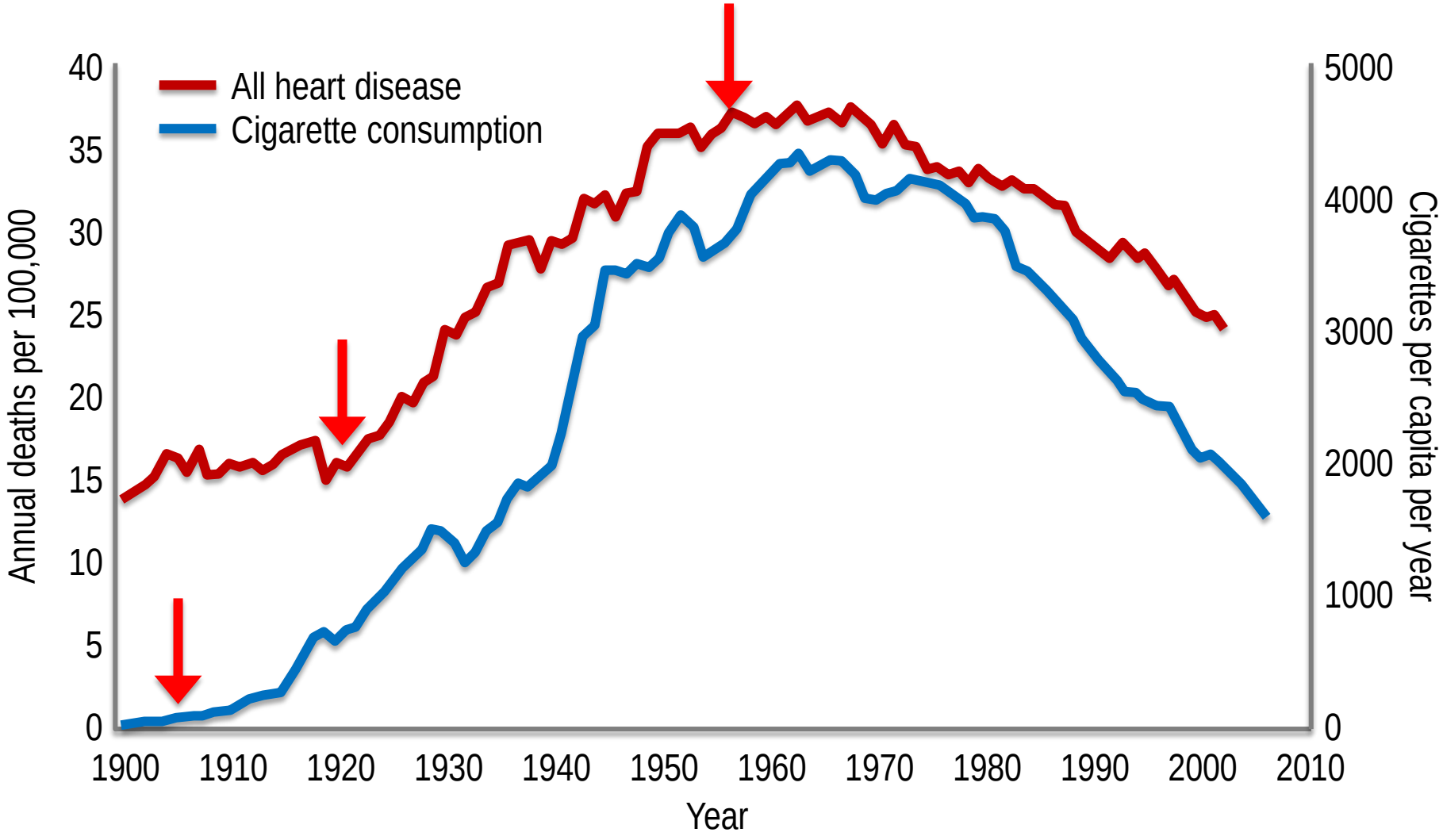
Rank	Country	Adult obesity (%)
1	Nauru	78.7
2	Samoa	74.8
3	Tokelau	63.2
4	Kiribati	50.3
5	Marshall Islands	46.0
6	Federated States of Micronesia	44.0
7	French Polynesia	40.4
8	Saudi Arabia	36.1
9	Panama	33.9
10	United States	33.7
11	United Arab Emirates	32.8
12	Iraq	32.2
13	Mexico	29.4
14	Kuwait	29.0
15	Egypt	28.9
16	Bahrain	28.5
17	New Zealand	25.4
18	Macedonia	25.3
19	Seychelles	25.1
20	Australia	24.8
21	United Kingdom	24.0

Data from World Health Organization. Source: See www.robbrooks.net/rob-brooks/1317 for the full 137 countries

ANCEL KEYS (1904-2004)

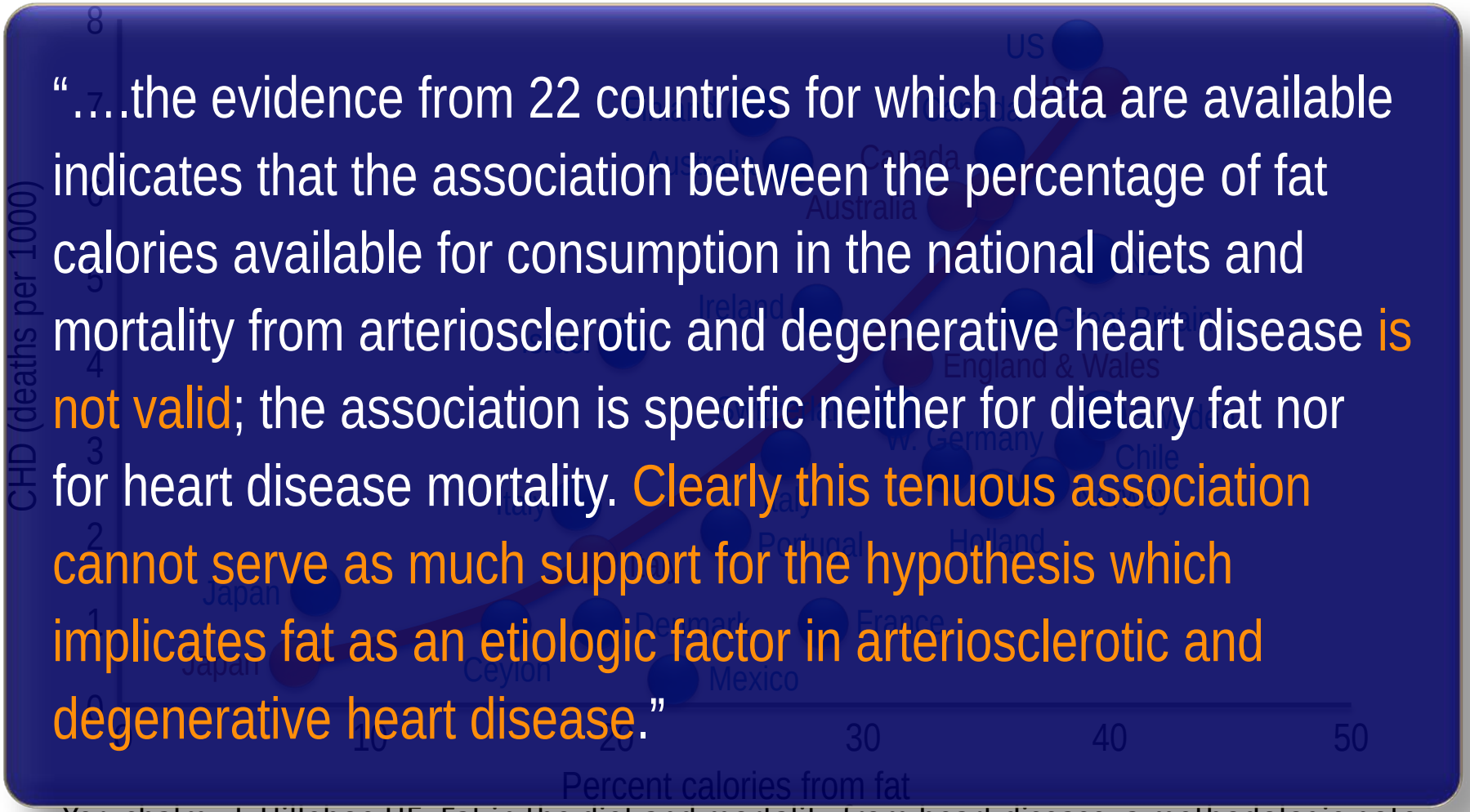
Keys A. Atherosclerosis: a problem in newer public health. *J Mt Sinai Hosp N Y* 1953; 20: 118-139.

CHANGES IN CIGARETTE CONSUMPTION MATCHES THE CHANGING INCIDENCE OF HEART DISEASE



Data from US National Vital Statistics and the Centre for Disease Control and Prevention.

COUNTRIES WHERE DATA WERE AVAILABLE WHEN KEYS PUBLISHED



"….the evidence from 22 countries for which data are available indicates that the association between the percentage of fat calories available for consumption in the national diets and mortality from arteriosclerotic and degenerative heart disease is not valid; the association is specific neither for dietary fat nor for heart disease mortality. Clearly this tenuous association cannot serve as much support for the hypothesis which implicates fat as an etiologic factor in arteriosclerotic and degenerative heart disease."

Yerushalmy J, Hilleboe HE. Fat in the diet and mortality from heart disease; a methodologic note. *N Y State J Med* 1957; 57: 2343-2354.

"….the evidence from 22 countries for which data are available indicates that the association between the percentage of fat calories available for consumption in the national diets and mortality from arteriosclerotic and degenerative heart disease is not valid; the association is specific neither for dietary fat nor for heart disease mortality. Clearly this tenuous association cannot serve as much support for the hypothesis which implicates fat as an etiologic factor in arteriosclerotic and degenerative heart disease."


RICHARD NIX
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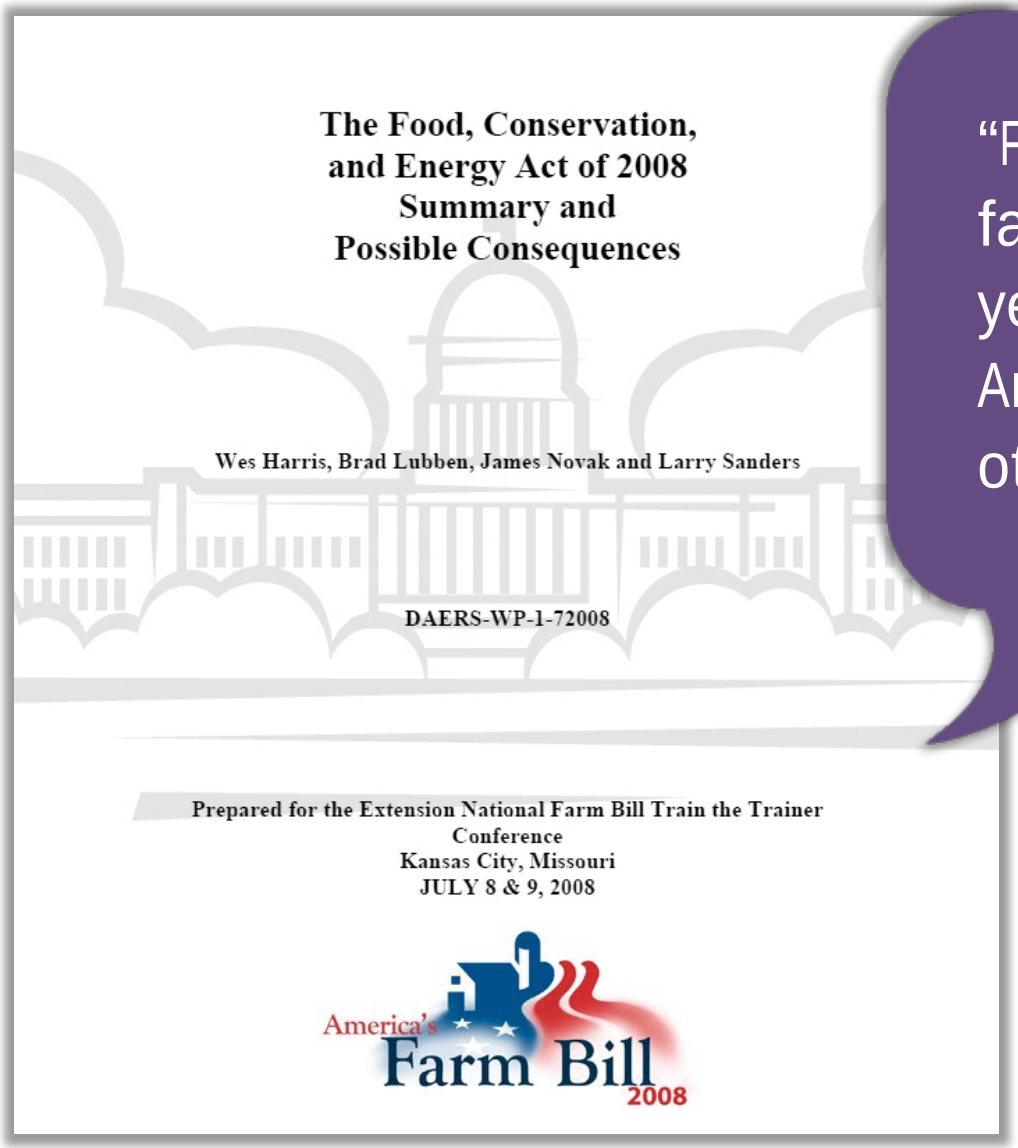
Wes Harris, Brad Lubben
DAERS-
Prepared for the Extension National Farm Bill Train the Trainer
Conference
Kansas City, Missouri
JULY 8 & 9, 2008
America's
Farm Bill
2008
"Food Bill" insures that US farmers receive $5 billion per year to grow corn and soy. An additional $5 billion for other farmers.



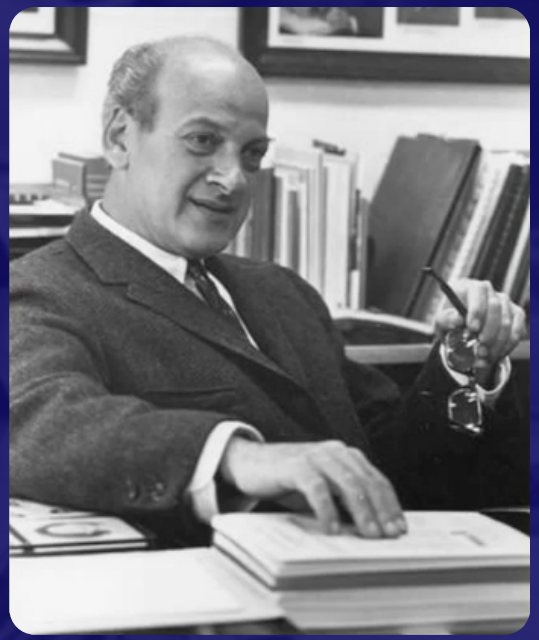
"Food Bill" insures that US farmers receive $5 billion per year to grow corn and soy. An additional $5 billion for other farmers.

UNITED STATES SENATE SELECT COMMITTEE ON NUTRITION AND HUMAN NEEDS (1968-1977)

- Reduce consumption of fat
- Switch from saturated fat to vegetable fats
- Reduce cholesterol to 1 egg per day
- Eat more carbohydrate, especially grains

The McGovern Report was written by a junior staffer, a vegan, who had no training in the nutritional sciences.

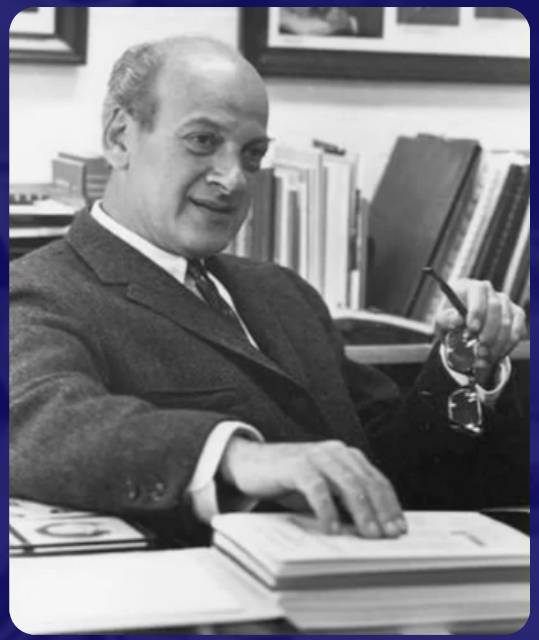
UNITED STATES SENATE SELECT COMMITTEE ON NUTRITION AND HUMAN NEEDS (1968-1977)



"What right has the federal government to propose that the American people conduct a vast nutritional experiment, with themselves as subjects, on the strength of so very little evidence?"

Philip Handler, National Academy of Science

UNITED STATES SENATE SELECT COMMITTEE ON NUTRITION AND HUMAN NEEDS (1968-1977)



"Resolution of this dilemma turns on a value judgment. The dilemma so posed is not a scientific question; it is a question of ethics, morals, politics. Those who argue either position strongly are expressing their values; they are not making scientific judgments".

Philip Handler, National Academy of Science

UNITED STATES SENATE SELECT COMMITTEE ON NUTRITION AND HUMAN NEEDS (1968-1977)

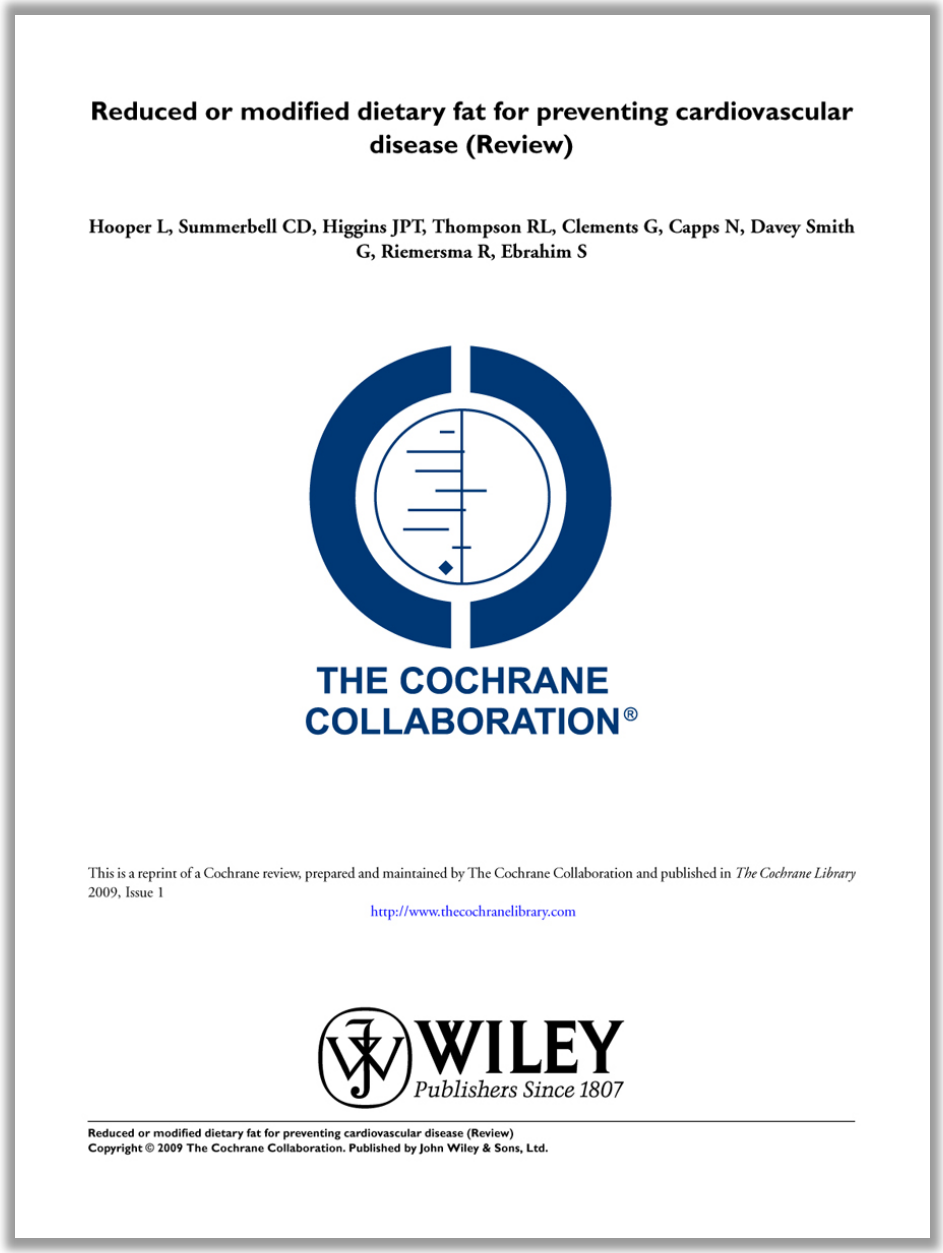
"…a trial of the low fat diet recommended by the McGovern Committee and the American Heart Association has never been carried out. It seems that the proponents of this dietary change are willing to advocate an untested diet to the nation on the basis of suggestive evidence obtained in tests of a different diet. This illogic is presumably justified by the belief than benefits will be obtained, vis-à-vis CHD prevention, by any diet that causes a reduction in plasma lipid levels".

Ahrens EH. Dietary fats and coronary heart disease: unfinished business. *Lancet* 1979; 2: 1345-1348.

2010:

During 5-23 y of follow-up of 347,747 subjects, 11,006 developed CVD or stroke. Intake of saturated fat was not associated with an increased risk of CHD, stroke or CVD. Consideration of age, sex and study quality did not change the results.

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: 535-546.



Reduced or modified dietary fat for preventing cardiovascular disease (Review)

Hooper L, Summerbell CD, Higgins JPT, Thompson RL, Clements G, Capps N, Davey Smith G, Riemersma R, Ebrahim S

THE COCHRANE COLLABORATION®

This is a reprint of a Cochrane review, prepared and maintained by The Cochrane Collaboration and published in *The Cochrane Library* 2009, Issue 1

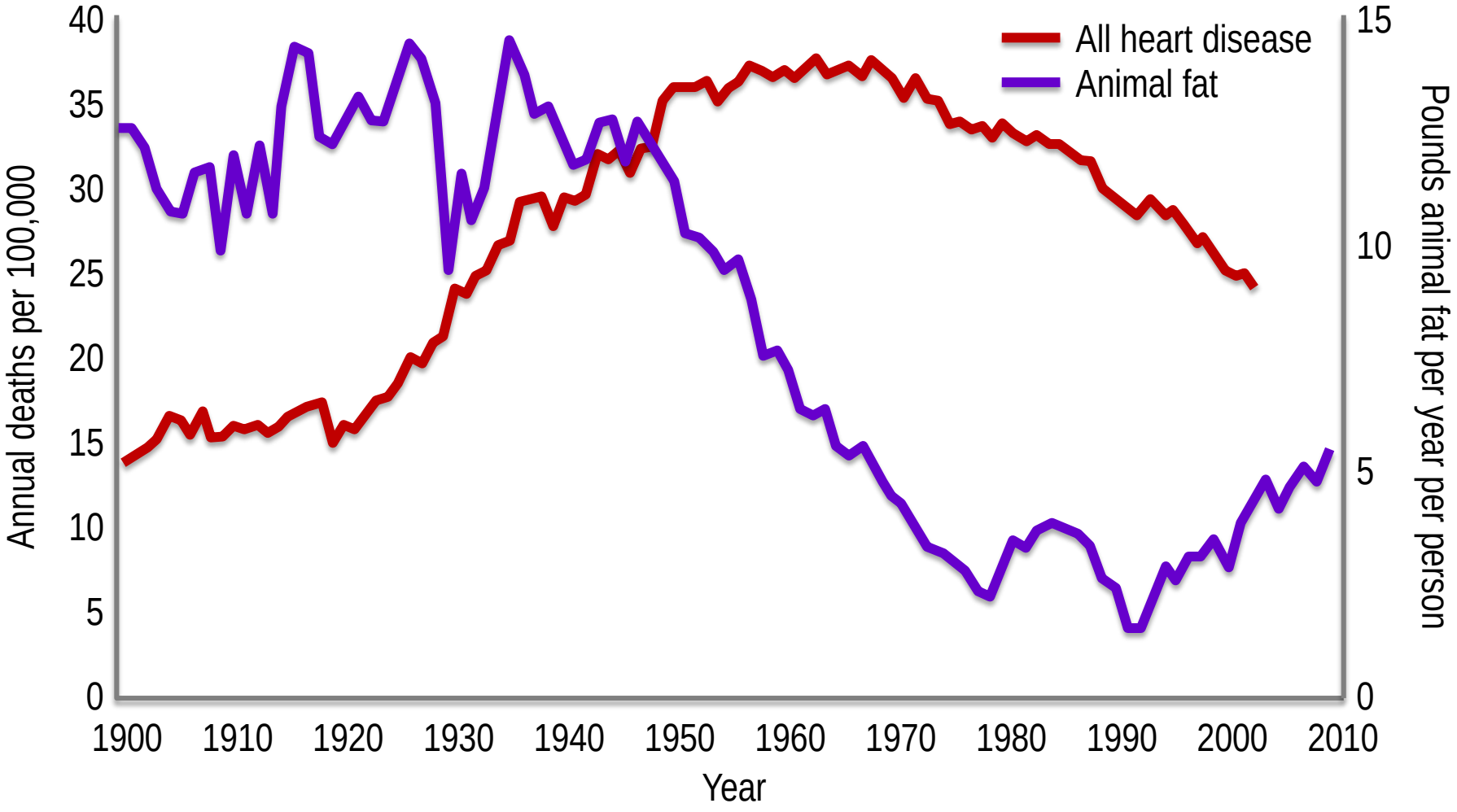
http://www.thecochranelibrary.com

WILEY Publishers Since 1807

2011:

There were **no clear effects of dietary fat changes** on total mortality or cardiovascular mortality.

CONSUMPTION OF ANIMAL FAT IN USA FALLS AS INCIDENCE OF HEART DISEASE RISES



Data from US National Vital Statistics.

Economic considerations drove the adoption of the current dietary guidelines without proper scientific evaluation or proof.

Within 5 years of the widespread adoption of these guidelines rates of diabetes and obesity increased explosively.

The presence of the genetic predisposing condition, carbohydrate [illegible] explains why large numbers of predisposed [illegible] diabetic when exposed to a high carbohydrate diet.

A high fat diet reverses all known coronary risk factors in [illegible] resistance whereas a high carbohydrate diet worsens those factors.

CONCLUSION:

Keys was wrong.

Fat in the diet does not cause heart disease.

Diet-heart hypothesis is wrong.

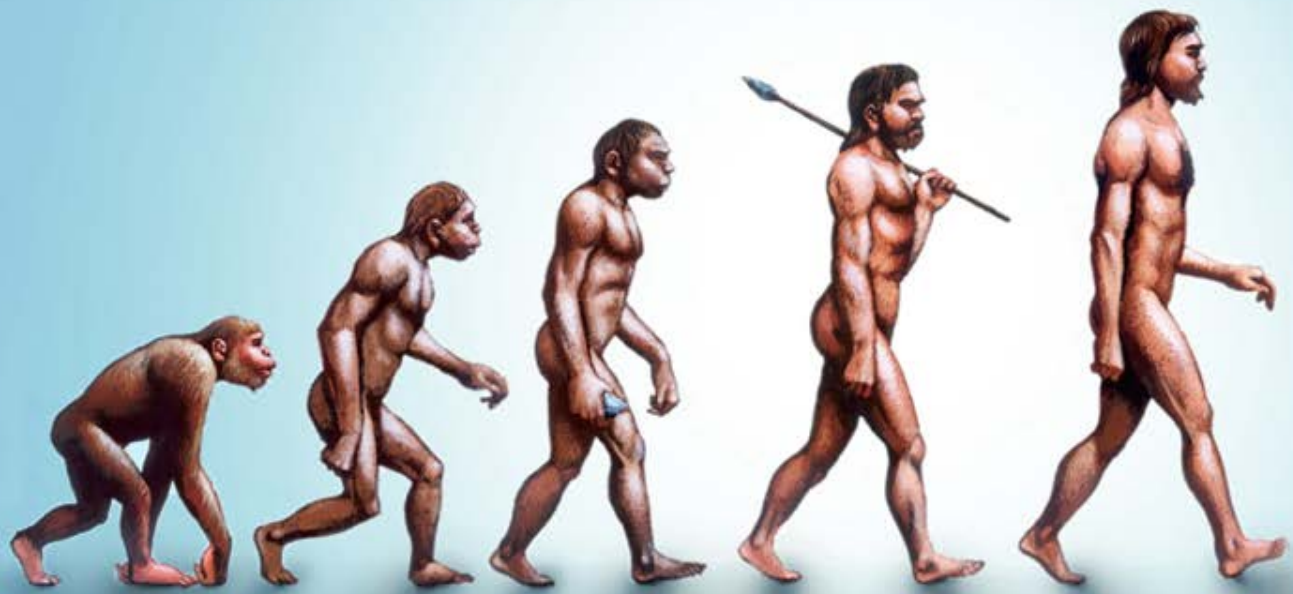
Economic considerations drove the adoption of the current dietary guidelines without proper scientific evaluation or proof.

Within 5 years of the widespread adoption of these guidelines rates of diabetes and obesity increased explosively.

The presence of the genetic predisposing condition, carbohydrate-resistance, explains why large numbers of persons in predisposed populations become obese and diabetic when exposed to a high carbohydrate diet.

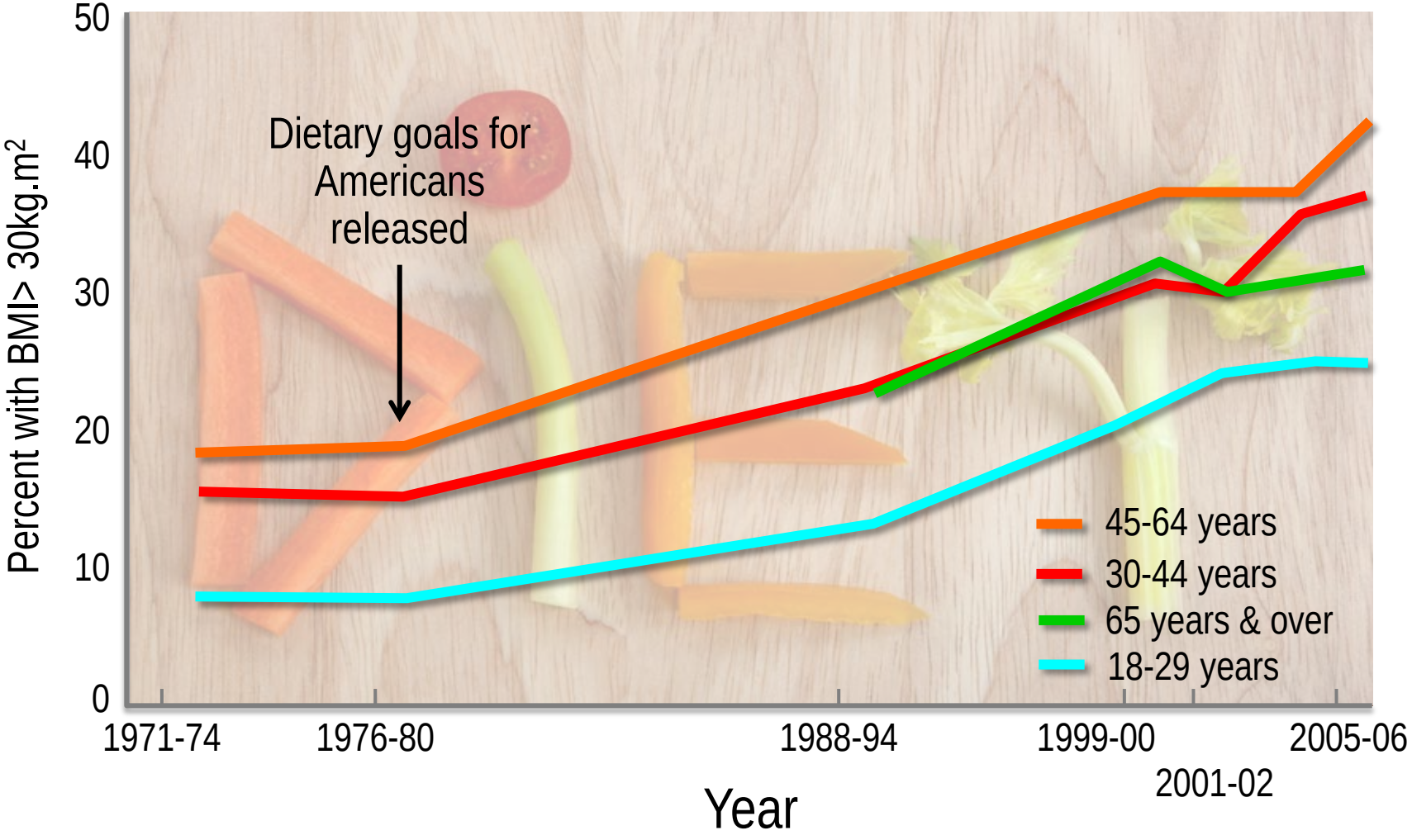
A high fat diet reverses all known coronary risk factors in persons with carbohydrate-resistance whereas a high carbohydrate diet worsens those factors.

MILLIONS OF YEARS



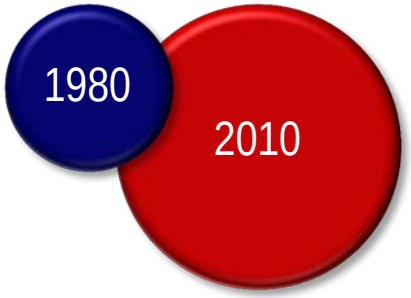
28 YEARS

INFLUENCE OF 1977 DIETARY GUIDELINES ON % OBESITY IN USA

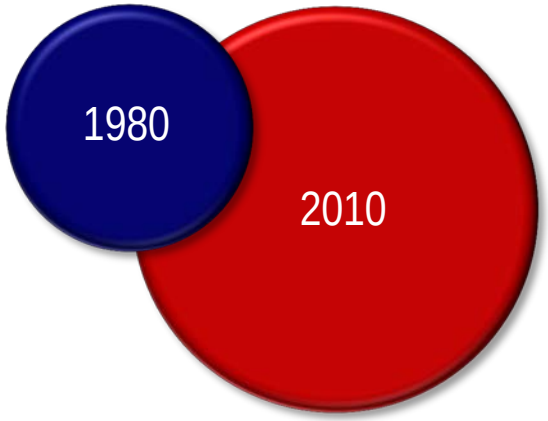


DIABETES AND OBESITY RATES IN THE US HAVE SORED SINCE THE ADOPTION OF THE 1977 DIETARY GUIDELINES

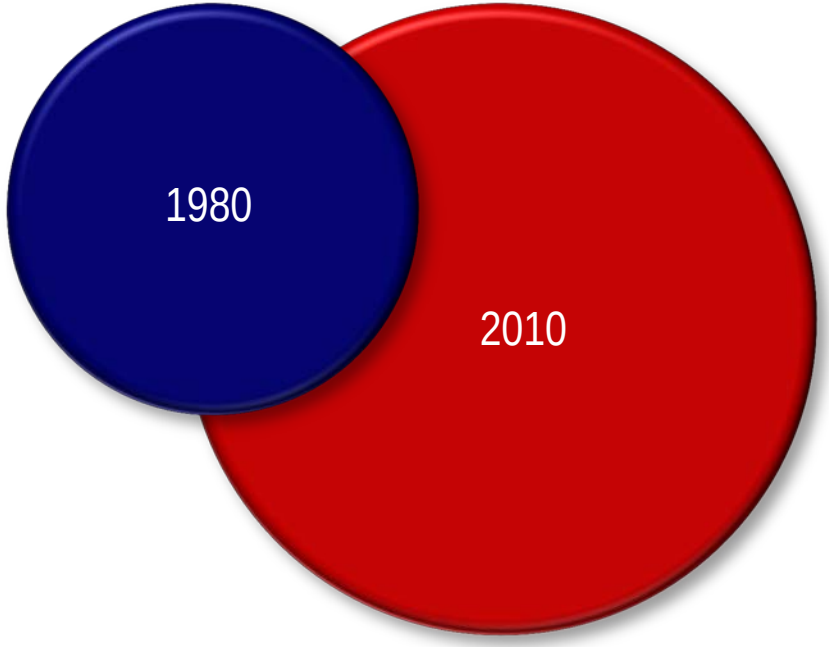
% of Americans with diabetes



% of US children who are obese

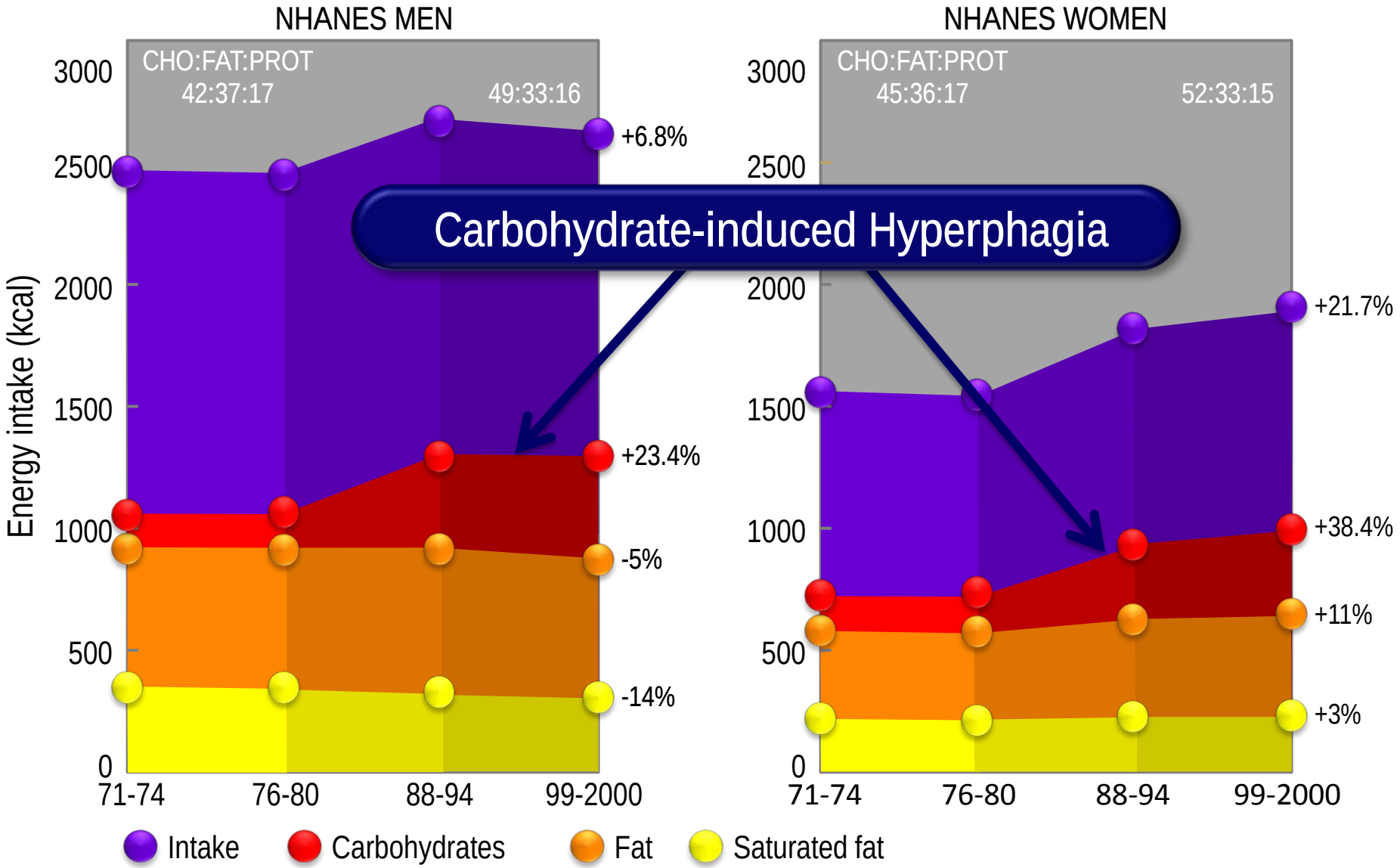


% of US adults who are obese



Data from USDA, CDC, US Census Bureau

CHANGES IN US MACRONUTRIENT INTAKES – 1971 - 2000



Hite AH, Feinman RD, Guzman GE, et al. In the face of contradictory evidence: report of the Dietary Guidelines for Americans Committee. *Nutrition* 2010; 26: 915-924.

Economic considerations drove the adoption of the current dietary guidelines without proper scientific evaluation or proof.

Within 5 years of the widespread adoption of these guidelines rates of diabetes and obesity increased explosively.

CONCLUSION:

Explosive increase in rates of obesity and Type II diabetes in the US has been caused by an increased carbohydrate intake resulting from the 1977 Dietary Guidelines

Economic considerations drove the adoption of the current dietary guidelines without proper scientific evaluation or proof.

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The presence of the genetic predisposing condition, carbohydrate-resistance, explains why large numbers of persons in predisposed populations become obese and diabetic when exposed to a high carbohydrate diet.

A high fat diet reverses all known coronary risk factors in persons with carbohydrate-resistance whereas a high carbohydrate diet worsens those factors.

WHY DOES OBESITY OCCUR ONLY IN SOME WHEN ALL EAT HIGH CARBOHYDRATE DIETS?

Largest man in the world in 1903

American police officer in 2012

Obesity cannot be due simply to doing too little exercise.

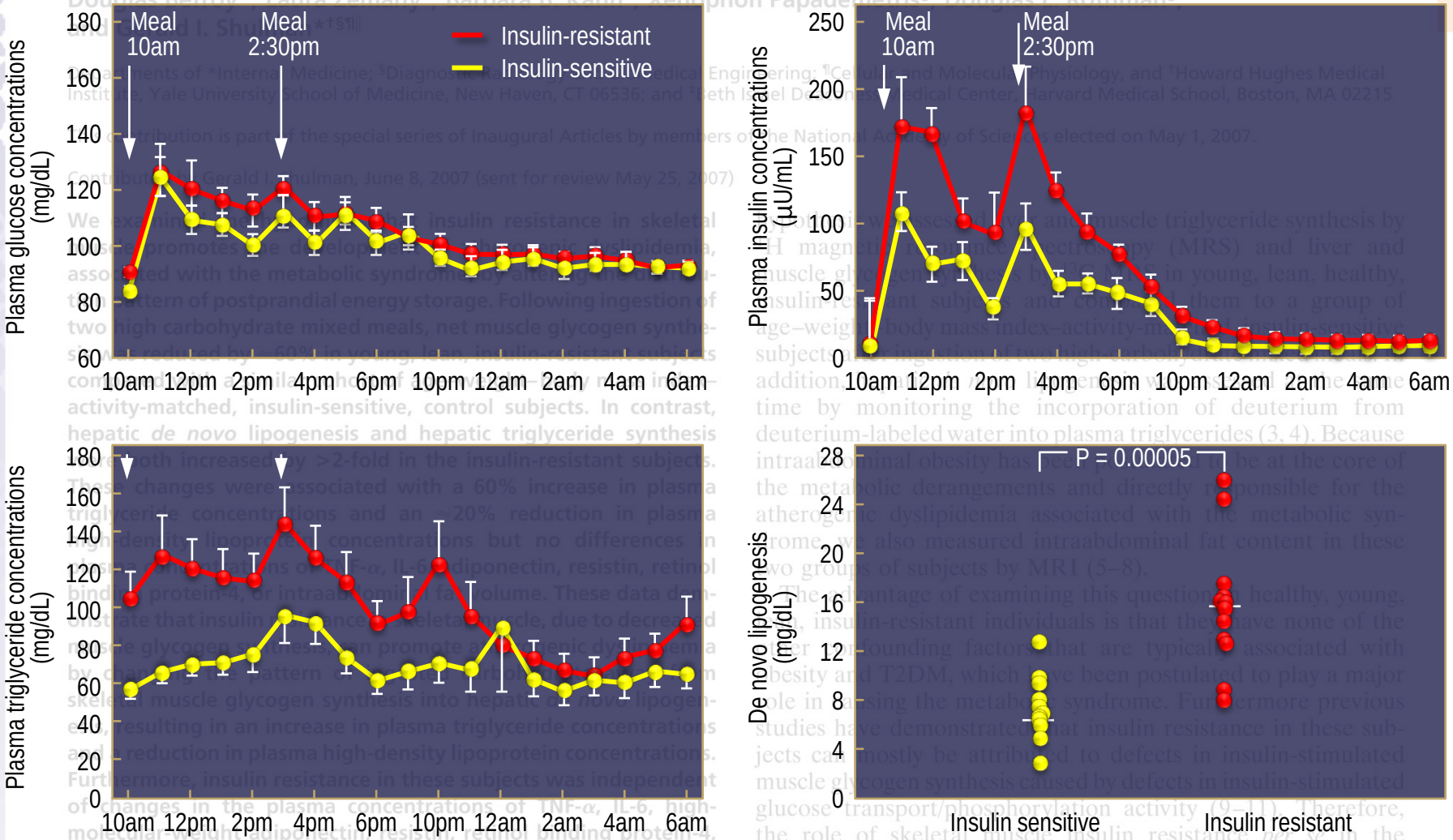
In a homeostatically-regulated system, any reduction in energy expenditure will be matched by an exactly equal reduction in energy intake.

Conversely any sustained increase in energy consumption should be matched by an increase in energy expenditure.

Hence the problem must be that the homeostat has been broken by the 1977 Dietary Guidelines.

THE CONDITON OF CARBOHYDRATE RESISTANCE

KF Petersen, S Dufour, DB Savage. PNAS. 104; 12587–12594, 2007.



Petersen KF, Dufour S, Savage DB, et al. The role of skeletal muscle insulin resistance in the pathogenesis of the metabolic syndrome. *Proc Natl Acad Sci U S A* 2007; 104: 12587-12594.

The metabolism of every human is not the same.
Those with carbohydrate resistance are unable to metabolize carbohydrate safely.

METABOLIC PROFILE OF PERSONS WITH CR INGESTING A HIGH CARBOHYDRATE DIET

- Elevated blood glucose concentrations
- Elevated blood insulin concentrations
- Elevated HbA1c concentrations
- Elevated blood triglyceride concentrations
- Reduced blood HDL-cholesterol concentrations (HDL-C)
- Increased small LDL-cholesterol particles (LDL-C P)
- Increased blood uric acid concentrations
- Increased blood ultrasensitive CRP concentrations
- Additional features:
  - Fatty liver
  - Obesity
  - Hypertension

BLOOD RISK FACTORS FOR CORONARY HEART DISEASE

- Total Cholesterol
- Ultrasensitive CRP
- Fibrinogen
- Glucose
- HbA1c
- Homocysteine
- HDL-cholesterol
- LDL-Cholesterol
- LDL- Cholesterol particle size or number
- Lp (a)
- Insulin
- Omega 6 to Omega 3 ratios
- Triglycerides
- Uric Acid

THE DIETARY FAT HYPOTHESIS FOR HEART DISEASE

Atherogenic Dyslipidaemia (AD)

High fat diet

Atherogenic Dyslipidaemia

Increased LDL cholesterol

Increased triglycerides

Reduced HDL cholesterol

Arterial "clogging"

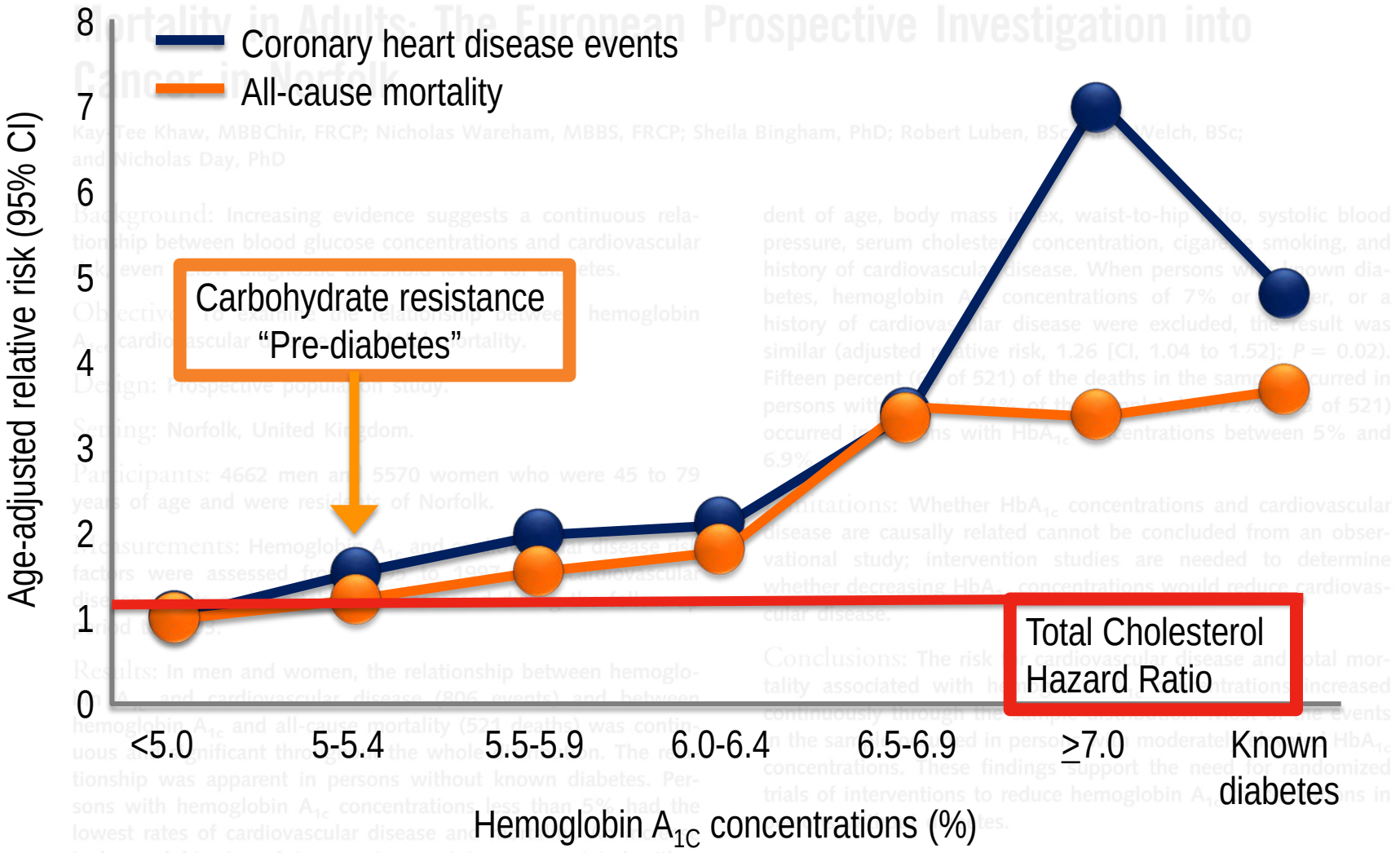
OBESITY, DIABETES, HYPERTENSION, GOUT, METABOLIC SYNDROME ARE SEPARATE/DISTINCT DISEASES

RELATIVE IMPORTANCE (BASED ON HAZARD RATIO) OF DIFFERENT RISK FACTORS FOR CORONARY HEART DISEASE

RISK FACTOR	HAZARD RATIO (RANGE)
Diabetes	2.04 (1.76 – 2.35)
Age	1.87 (1.73 – 2.02)
Current smoking	1.79 (1.66 – 1.94)
Systolic blood pressure	1.31 (1.26 – 1.37)
Total [Cholesterol]	1.22 (1.17 – 1.27)
[Triglyceride]	1.19 (1.15 – 1.23)
[HDL-Cholesterol]	0.83 (0.78 – 0.87)

Di AE, Gao P, Pennells L, et al. Lipid-related markers and cardiovascular disease prediction. *JAMA* 2012; 307: 2499-2506.

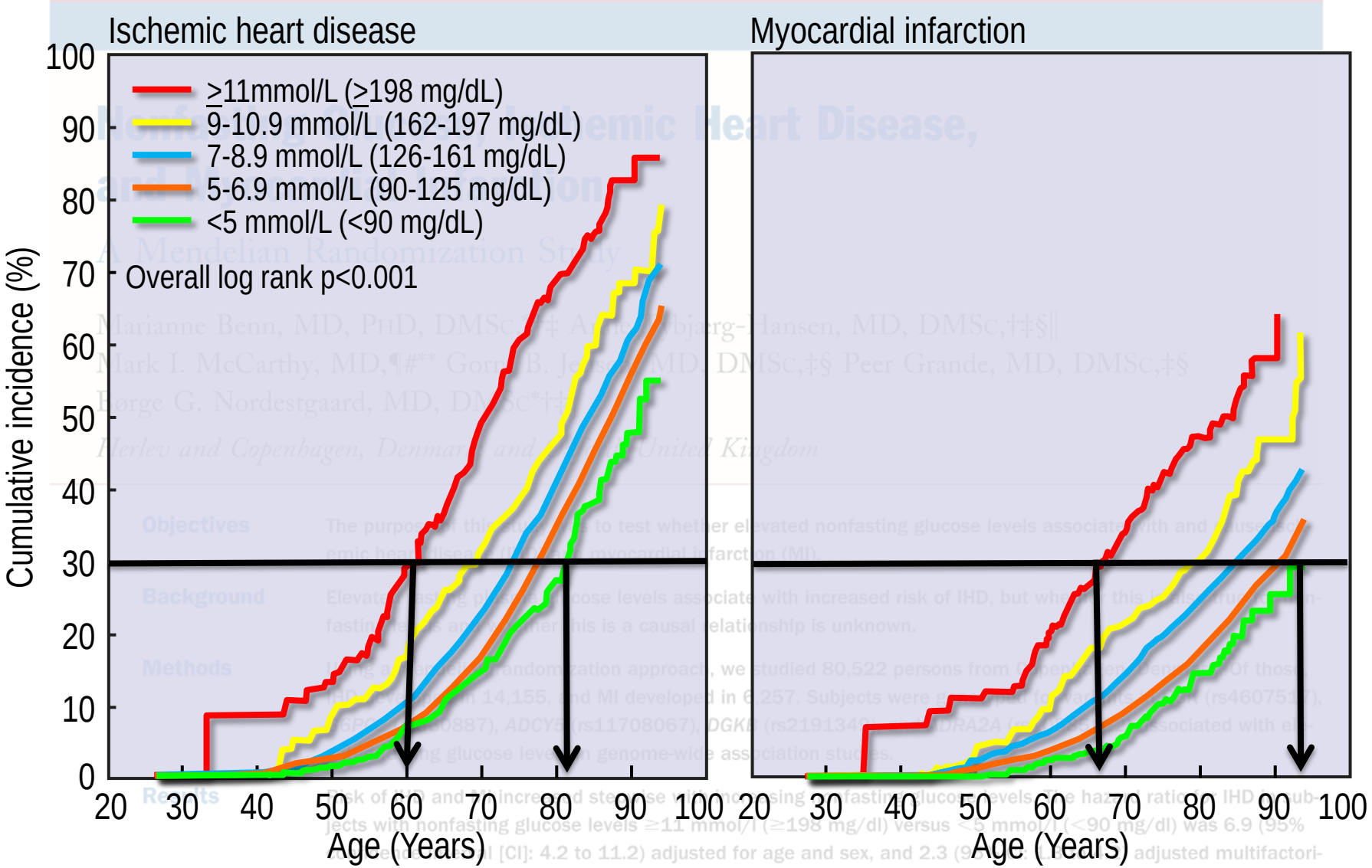
PREDICTIVE VALUE OF HbA1c FOR CORONARY HEART DISEASE EVENTS AND ALL-CAUSE MORTALITY



Khaw KT, Wareham N, Bingham S, et al. Association of hemoglobin A1c with cardiovascular disease and mortality in adults: the European Prospective Investigation into Cancer in Norfolk. *Ann Intern Med* 2004; 141: 413-420.

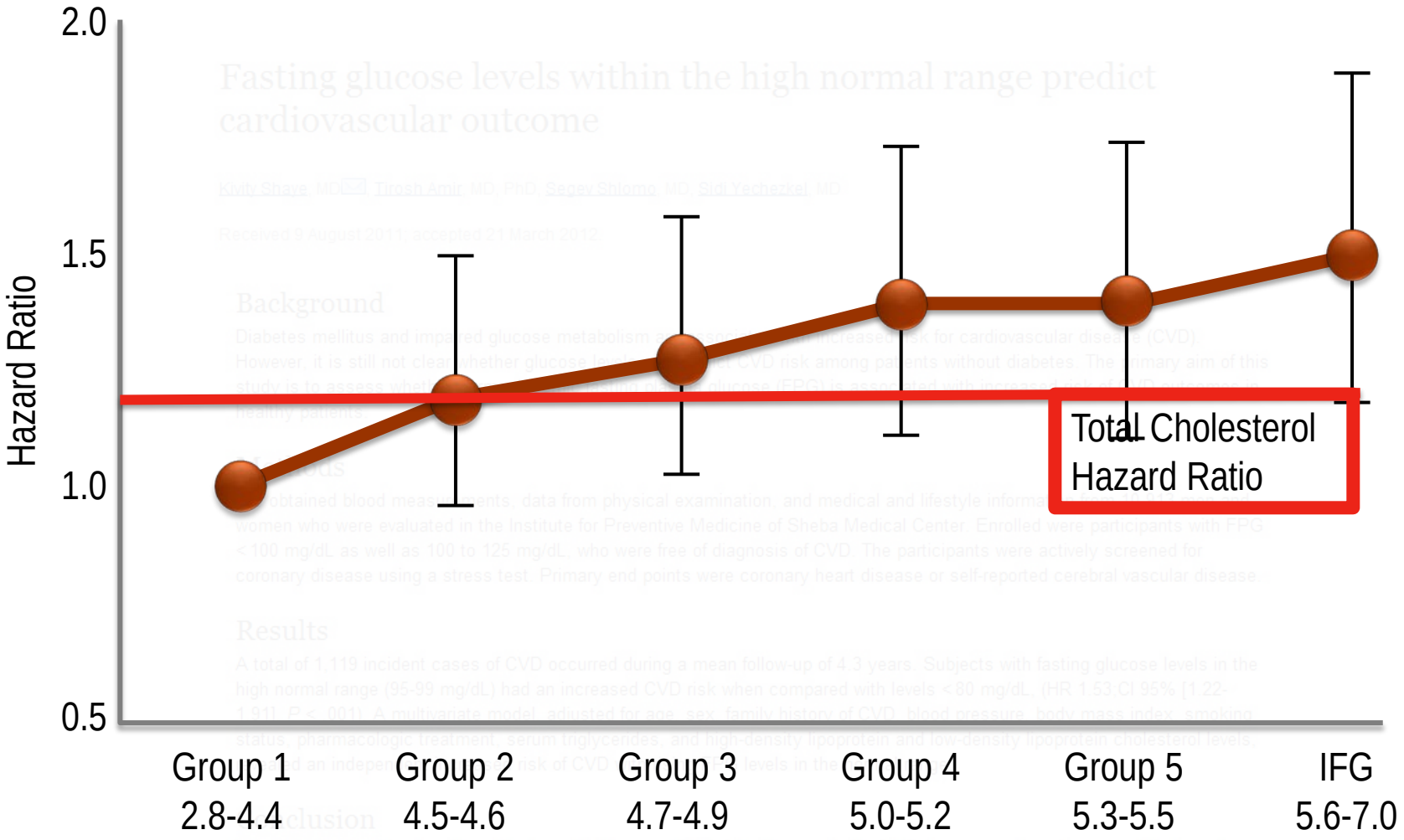
Risk was “independent of age, body mass index, waist-to-hip ratio, systolic blood pressure, serum cholesterol concentration, cigarette smoking, and history of cardiovascular disease”.

CUMULATIVE INCIDENCE OF IHD FOR DIFFERENT RANDOM BLOOD GLUCOSE CONCENTRATIONS



Benn M et al. Non-fasting glucose, ischemic heart disease and myocardial infarction. Journal of the American College of Cardiology 59; 2012.

BLOOD GLUCOSE (mmol/L) IN THE NORMAL RANGE PREDICTS CARDIOVASCULAR OUTCOME



Shaye K, Amir T, Shlomo S, Yechezkel S. Fasting glucose levels within the high normal range predict cardiovascular outcome. *Am Heart J* 2012; 164: 111-116

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Within 5 years of the widespread adoption of these guidelines rates of diabetes and obesity increased explosively.

The presence of the genetic predisposing condition, carbohydrate-resistance, explains why large numbers of persons in predisposed populations become obese and diabetic when exposed to a high carbohydrate diet.

CONCLUSION:

Their abnormal carbohydrate metabolism explains why those with carbohydrate resistance develop obesity, diabetes and coronary heart disease when eating a high carbohydrate diet.

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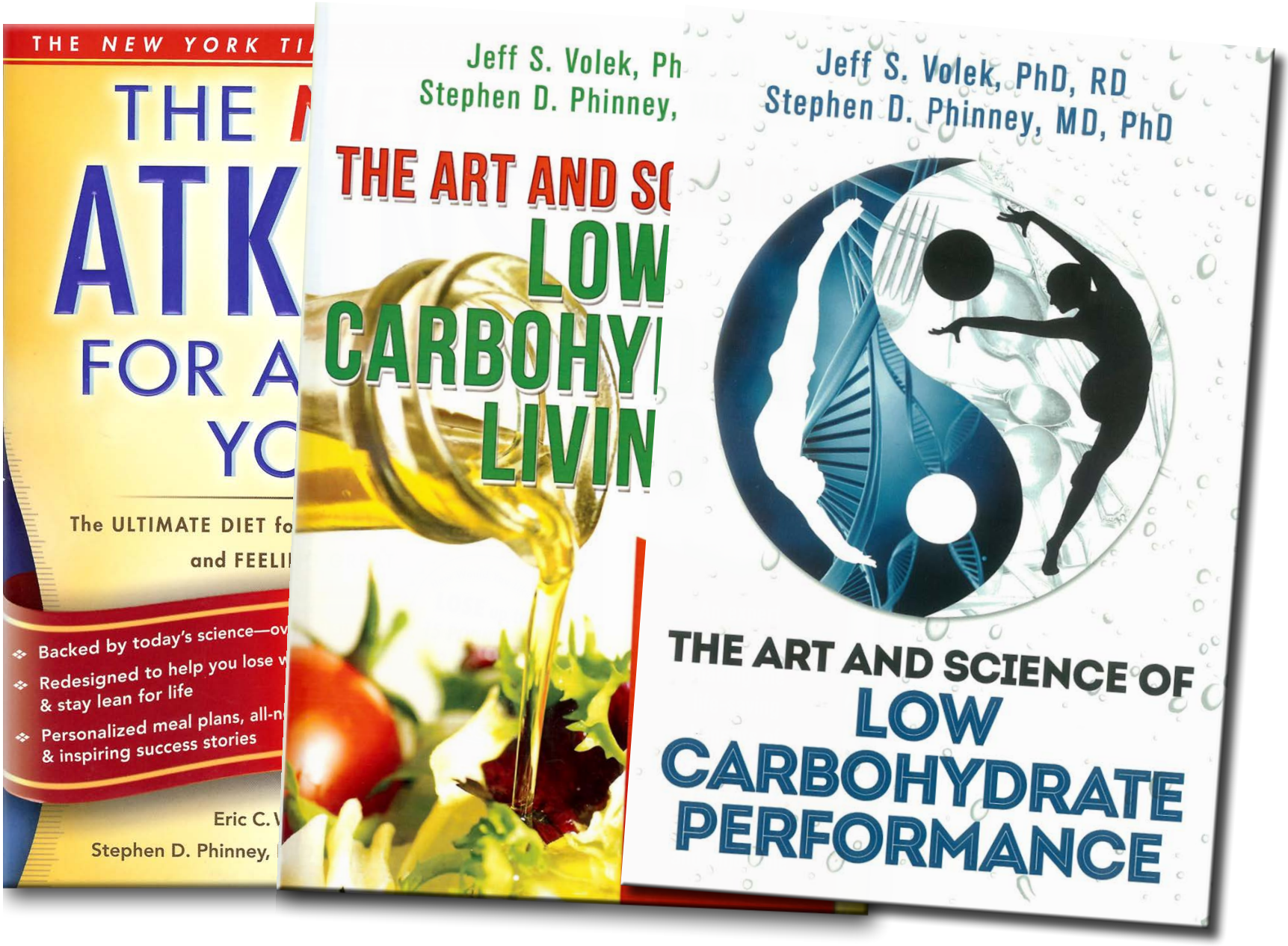
A high fat diet reverses all known coronary risk factors in persons with carbohydrate-resistance whereas a high carbohydrate diet worsens those factors.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Personalized meal plans, all-n
& inspiring success stories
Eric C. V
Stephen D. Phinney,
Jeff S. Volek, Ph
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THE ART AND SC
LOW
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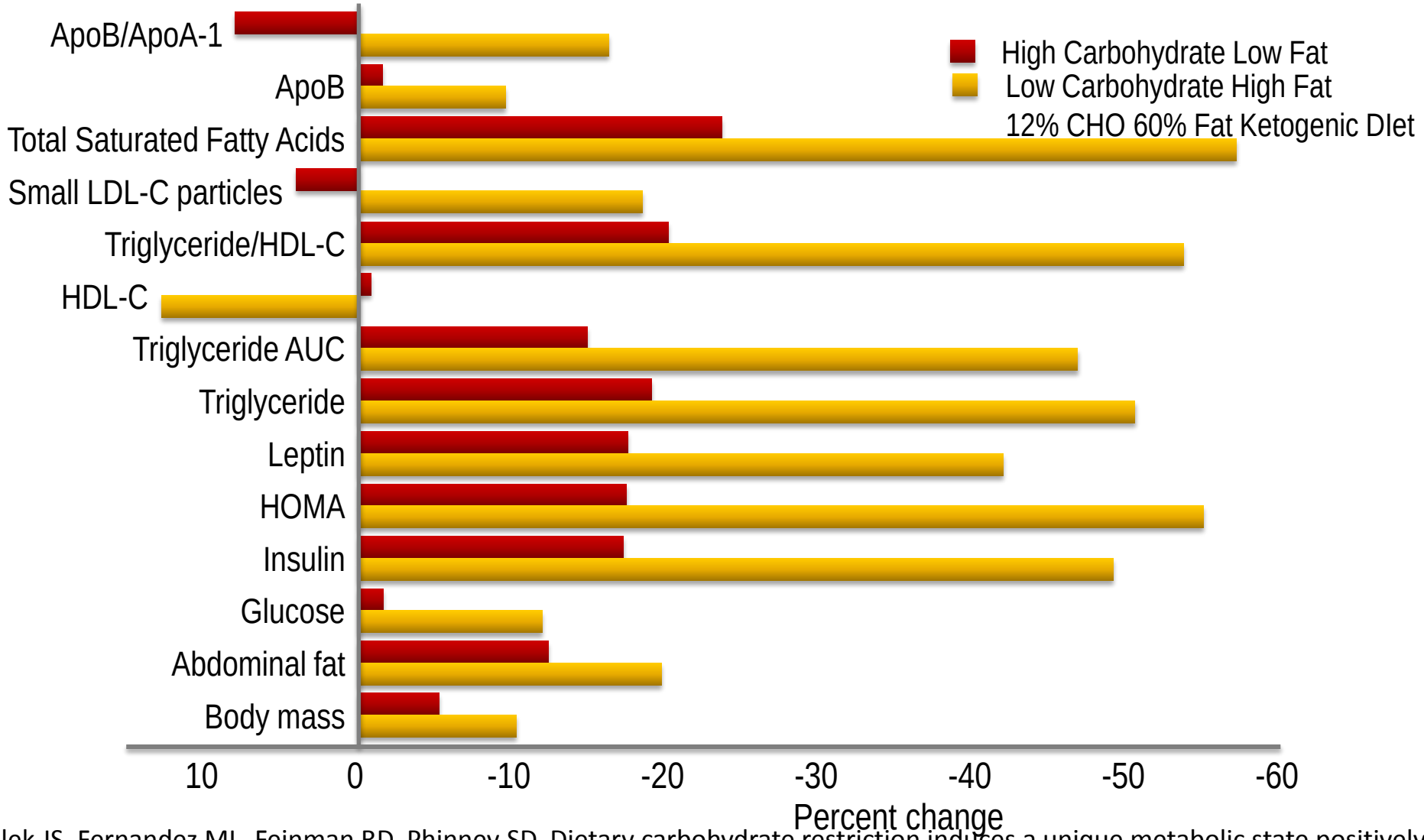
Jeff S. Volek, PhD, RD
Stephen D. Phinney, MD, PhD
THE ART AND SCIENCE OF
LOW
CARBOHYDRATE
PERFORMANCE

A HIGH FAT DIET REVERSES ALL CORONARY RISK FACTORS MORE EFFECTIVELY THAN A LOW FAT DIET



Volek JS, Fernandez ML, Feinman RD, Phinney SD. Dietary carbohydrate restriction induces a unique metabolic state positively affecting atherogenic dyslipidemia, fatty acid partitioning, and metabolic syndrome. *Prog Lipid Res* 2008; 47: 307-318.

"Meta-analysis … on data obtained in 1,141 obese patients, showed the low carbohydrate diet to be associated with significant decreases in body weight, body mass index, abdominal circumference, systolic blood pressure, diastolic blood pressure, plasma triglycerides, fasting plasma glucose, glycated haemoglobin, plasma insulin and plasma C-reactive protein, as well as an increase in high-density lipoprotein cholesterol. Low-density lipoprotein cholesterol and creatinine *did not change significantly*, whereas limited data exist concerning plasma uric acid".

Santos FL et al. **Systematic review and meta-analysis of clinical trials of the effects of low carbohydrate diets on cardiovascular risk factors.** *Obes Rev* 2012; 13: 1048-1066.

THE DIETARY CARBOHYDRATE HYPOTHESIS FOR HEART DISEASE

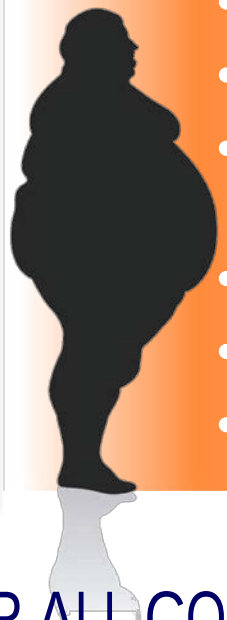
Hyperglycaemic Hyperinsulinaemic Atherogenic Dyslipidaemia (HHAD)

High carbohydrate (fructose) diet

- ↑ Glucose
- ↑ Insulin
- ↑ Triglycerides
- ↓ HDL-C
- ↑ Small LDL-C particles
- ↑ Uric acid*
- ↑ CRP

Fatty liver

Low Omega 3, high Omega 6



- Arterial inflammation
- Metabolic syndrome
- Coronary heart disease/stroke
- Obesity
- Diabetes
- Hypertension

ONE CAUSE, ONE TREATMENT FOR ALL CONDITIONS

CONCLUSION:

A high fat diet reverses (almost) all coronary risk factors. This is the converse of what is taught at all medical schools around the world.

A high fat diet reverses all known coronary risk factors in persons with carbohydrate-resistance whereas a high carbohydrate diet worsens those factors.

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Within 5 years of the widespread adoption of these guidelines rates of diabetes and obesity increased explosively.

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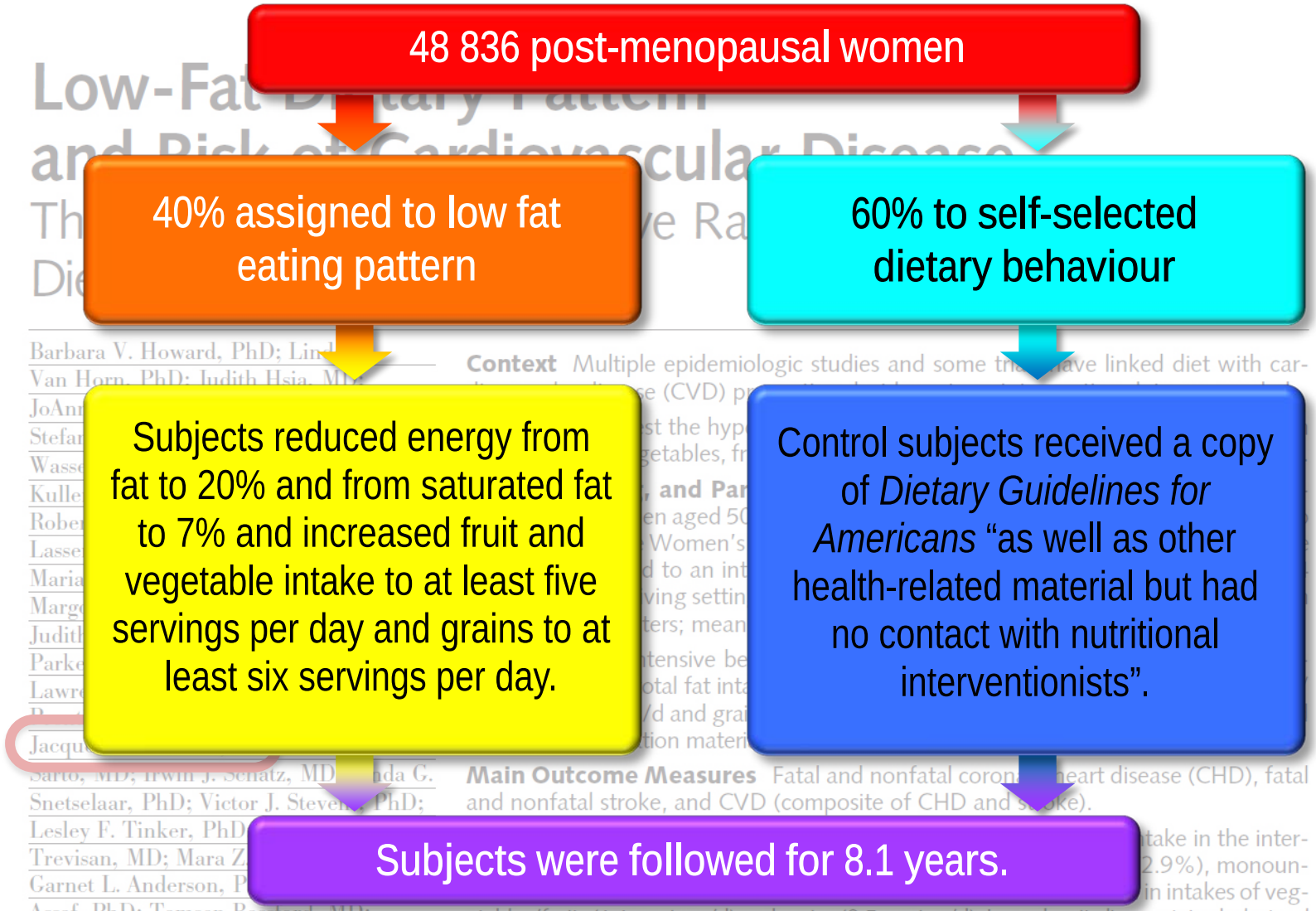
A high fat diet reverses all known coronary risk factors in persons with carbohydrate-resistance whereas a high carbohydrate diet worsens those factors.

The 48 836-person Woman's Health Initiative of which my opponent was the Project Director proves that the 1977 US Dietary Guidelines accelerate disease progression in persons with either known heart disease or diabetes. Thus his landmark study provides the definitive evidence disproving Keys' false diet-heart hypothesis.

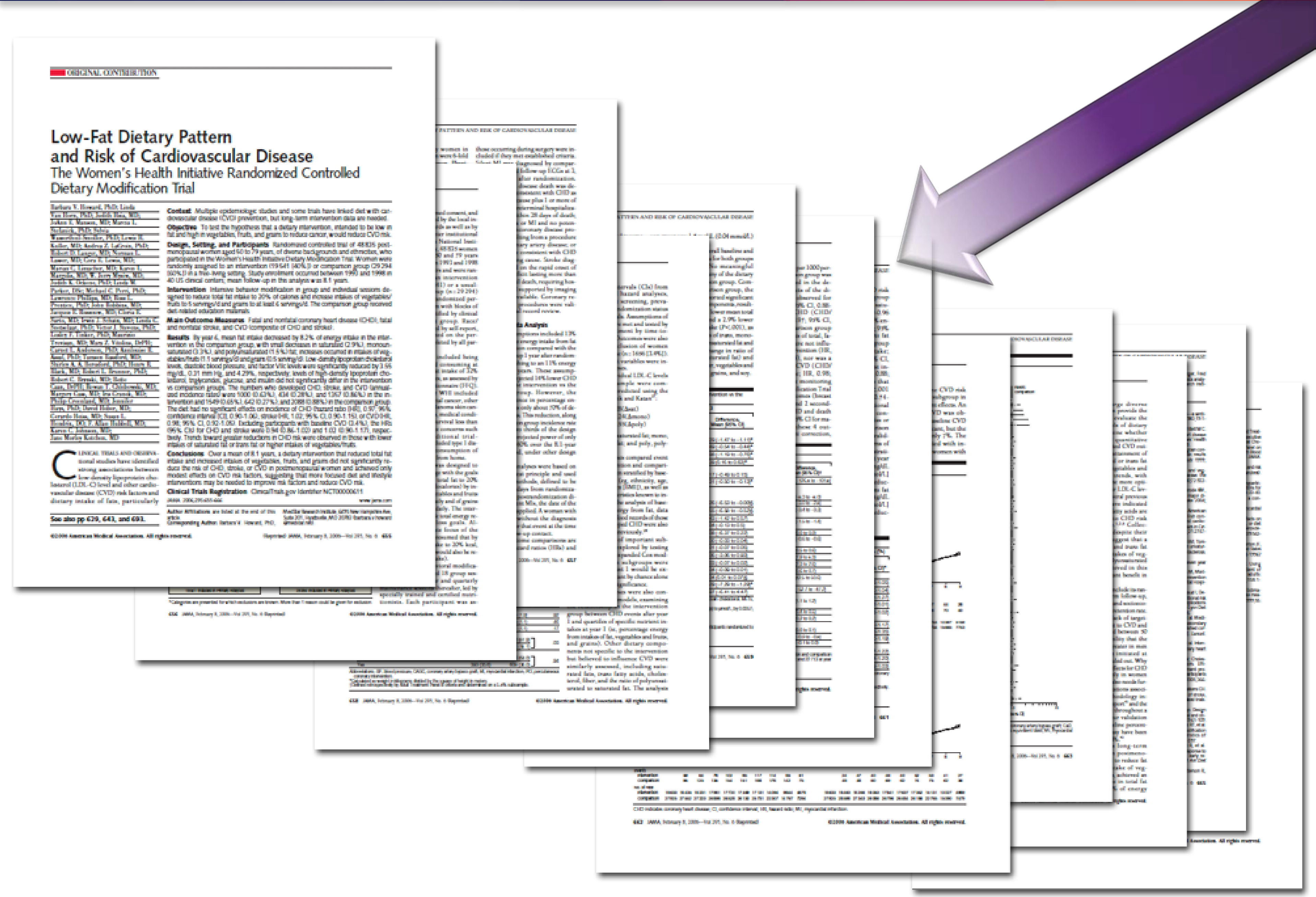
WOMEN'S HEALTH DIETARY MODIFICATION TRIAL



Howard BV, Van HL, Hsia J, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. *JAMA* 2006; 295: 655-666.

Conclusion: The study “did not significantly reduce the risk of coronary heart disease, stroke, or cardiovascular disease in postmenopausal women and achieved only modest effects on cardiovascular risk factors”.

But was that all they found?


ORIGINAL CONTRIBUTION
Low-Fat Dietary Pattern and Risk of Cardiovascular Disease
The Women's Health Initiative Randomized Controlled Dietary Modification Trial

was 0.86 to 1.12. After women with history of CVD at baseline were removed (n=1656 [3.4%]), HRs (95% CIs) for major CHD, composite CHD, stroke, and total CVD were 0.93 (0.83-1.05), 0.94 (0.86-1.02), 1.02 (0.90-1.15), and 0.96 (0.89-1.03), respectively. The HR for the 3.4% of women with CVD at baseline was 1.26 (95% CI, 1.03-1.54). We considered the potential confounding effects of changing medication use during the trial by examining use of statins, aspirin, and angiotensin-converting enzyme inhibitors at year 6. All differences in medication use between groups were less than 1%.

In examining trends over tim (FIGURE 2), there was no apparent fluence of the dietary intervention stroke at any point up to 9 years of low-up. There appeared to be a sli nonsignificant trend toward creased CHD rates in the intervent group in the later years, and this more pronounced for women with CVD at baseline. Likewise, expl atory analyses of CHD outcomes groups stratified by race/ethnicity, BMI, waist circumference, smoki statin use, diabetes, randomizatio the HT and CaD trials, and baseline

intake revealed no significant interactions between the intervention group and any of these variables (FIGURE 3), either in the group as a whole or if women with baseline CVD were excluded. A significant interaction was observed between the intervention effect and baseline disease ($P=.006$).

Ad l Analyses

When t of the intervention was ass adherence criteria based o n in intervention activitie id not change. Tra

served toward reduction of CHD risk among those in the intervention group who reached the lowest levels of saturated fat (HR, 0.81; 95% CI, 0.69-0.96 in the group that consumed <6.1% energy; $P<.001$ [adjusted HR, 0.82; 95% CI, 0.67-0.99; $P=.05$]) and *trans* fat (HR, 0.81; 95% CI, 0.69-0.95 in group consuming <1.1% energy intake; $P<.001$ [adjusted HR, 0.84; 95% CI, 0.69-1.02; $P=.10$]) or the highest intakes of vegetables and fruits (HR, 0.88; 95% CI, 0.76-1.03 in the group that consumed ≥6.5 servings/d; $P<.001$

Table 4. Clinical Outcomes (Annualized

Mean follow-up time, mo						
Major CHD (nonfatal MI or CHD death)†‡						
Nonfatal MI						
CHD death						
CABG/PCI						
Composite CHD (nonfatal MI, CHD death, or CABG/PCI)	1000					
Stroke	434 (0.28)	542 (0.27)	1.02 (0.90-1.15)	364 (0.25)	564 (0.25)	1.02 (0.90-1.17)
Fatal§	55 (0.03)	66 (0.03)	0.97 (0.69-1.36)	46 (0.03)	73 (0.03)	0.94 (0.65-1.35)
Hemorrhagic	67 (0.04)	113 (0.05)	0.60 (0.44-0.82)	62 (0.04)	106 (0.05)	0.58 (0.43-0.78)

"...THE HR FOR THE 3.4% OF WOMEN WITH CVD AT BASELINE WAS 1.26 (95% CI 1.03-1.54)...."

This is entirely predictable as a high carbohydrate diet produces HHAD (hyperglycaemic hyperinsulinaemic atherogenic dyslipidaemia) in those who are metabolically vulnerable.

For Immediate Release: February 7, 2006

National Heart Lung and Blood Institute
People Science Health

News from the Women's Health Initiative: Reducing Total Fat Intake May Have Small Effect on Risk of Breast Cancer, No Effect on Risk of Colorectal Cancer, Heart Disease, or Stroke

Following an eating pattern lower in total fat
breast cancer, heart disease, or stroke, and
healthy postmenopausal women, according t
National Institutes of Health's Women's Healt

The study was designed to evaluate a low-fat
cancer. However, investigators also evaluate
cardiovascular disease. The results from the
reported in three papers in the February 8 ed
Association.

Among the 48,835 women who participated i
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who followed a low-fat dietary plan and the c
dietary patterns. Although the women in the study who reduced their total fat intake had a 9 percent lower risk of breast cancer than did women who made no dietary changes, the difference was not large enough to be statistically significant meaning it could have

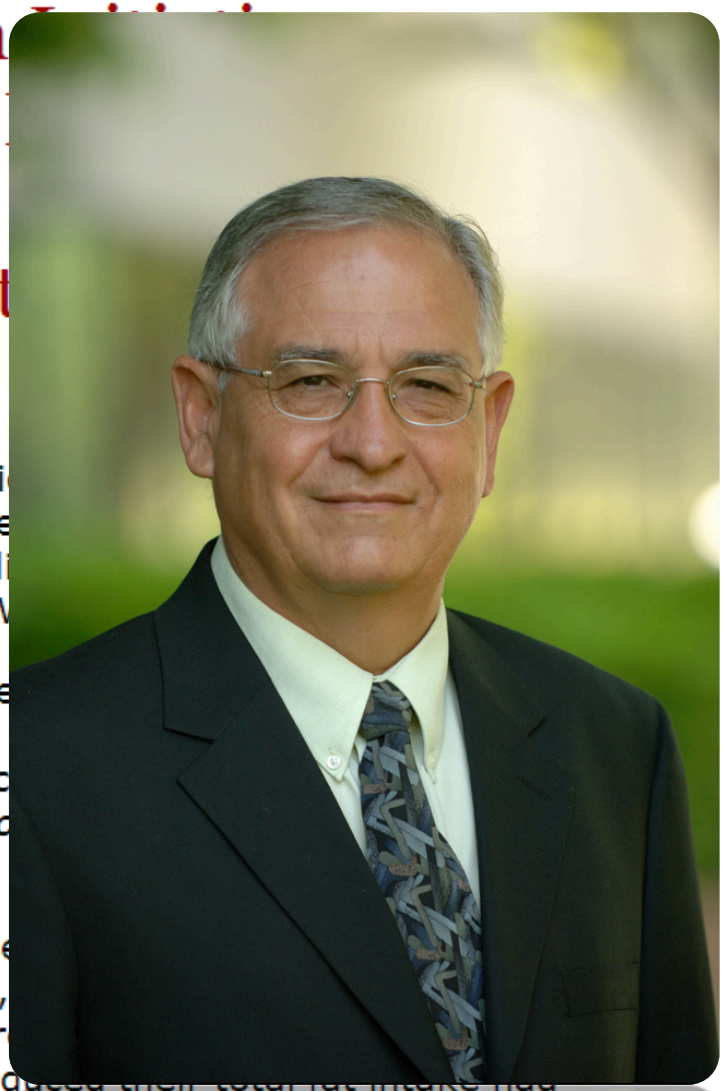
“The results of this study do not change established recommendations on disease prevention. Women should continue to … work with their doctors to reduce their risks for heart disease including following a diet low in saturated fat, trans fat and cholesterol”.

E Nabel, Director, NHLBI.

For Immediate Release: February 7, 2006

National Heart Lung and Blood Institute
People Science Health

News from the Women's Health
Reducing Total Fat Intake May
Effect on Risk of Breast Cancer,
Risk of Colorectal Cancer, Heart
Stroke



Following an eating pattern lower in total fat did not signifi
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“This study shows that just reducing total fat intake does not go far enough to have an impact on heart disease risk. While the participants’ overall change in LDL “bad” cholesterol was small, we saw trends towards greater reductions in cholesterol and heart disease risk in women eating less saturated and trans fat”.

J Rossouw, Project Director, WHIRCDMT

THE UPTON SINCLAIR THEOREM

"It is difficult to get a man to understand something, when his salary depends upon his not understanding it".

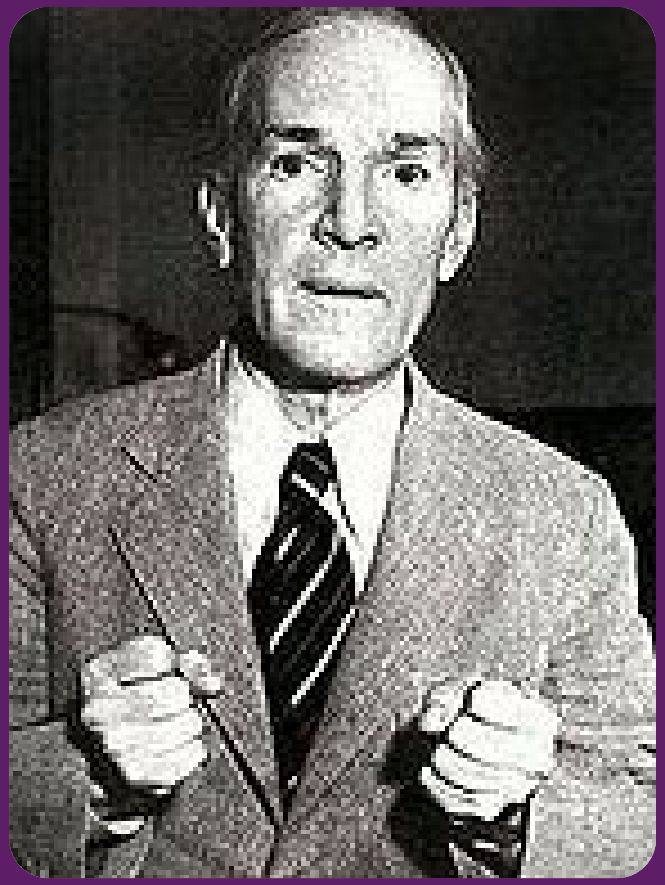
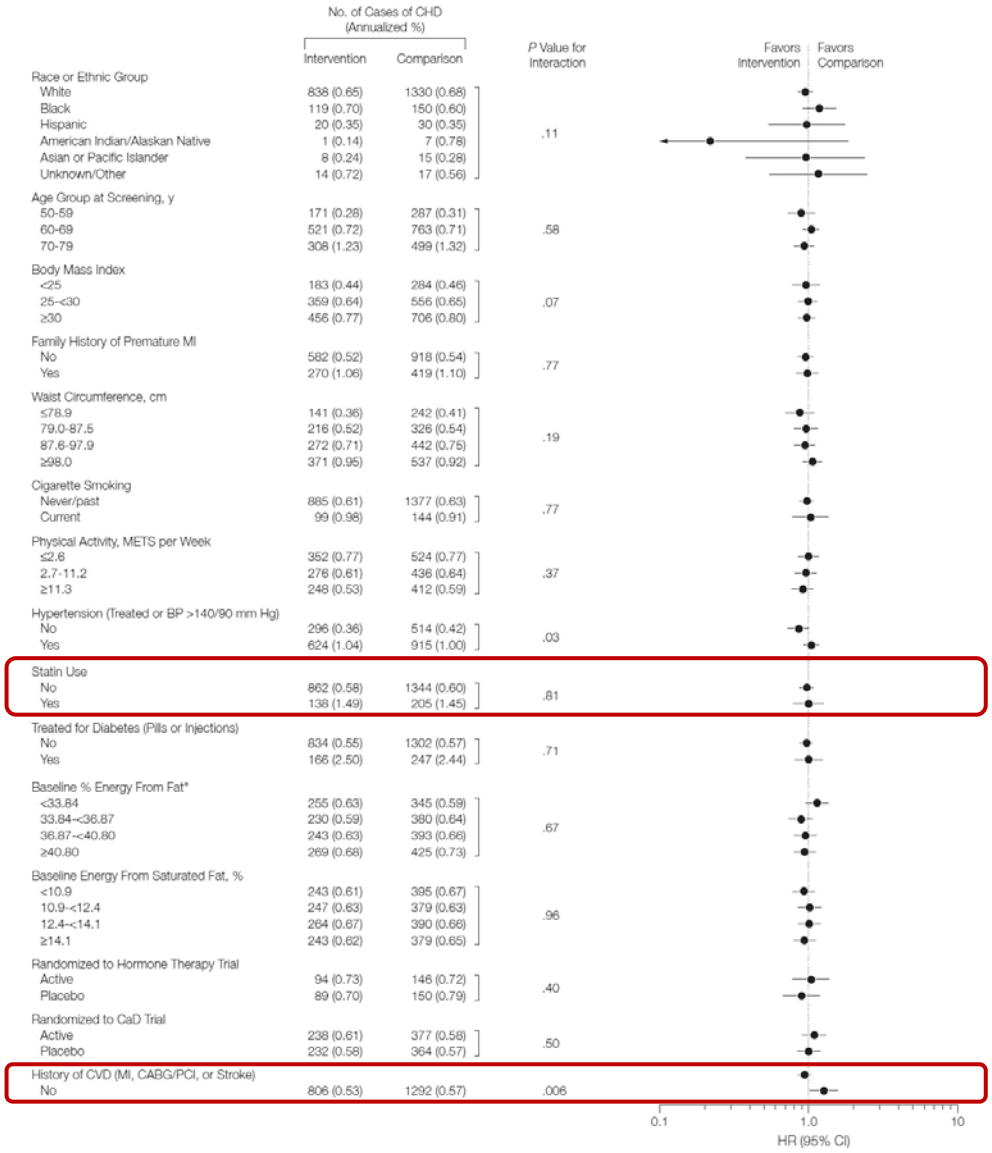


Figure 3. Risk of Composite Coronary Heart Disease (CHD) in Various Subgroups



Body mass index was calculated as weight in kilograms divided by the square of height in meters. BP indicates blood pressure; CABG, coronary artery bypass graft; CaD, calcium and vitamin D; CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio; HT, hormone therapy; METs, metabolic equivalent tasks; MI, myocardial infarction; PCI, percutaneous coronary intervention.
*Conclusions do not change if results from 4-day food records are used.

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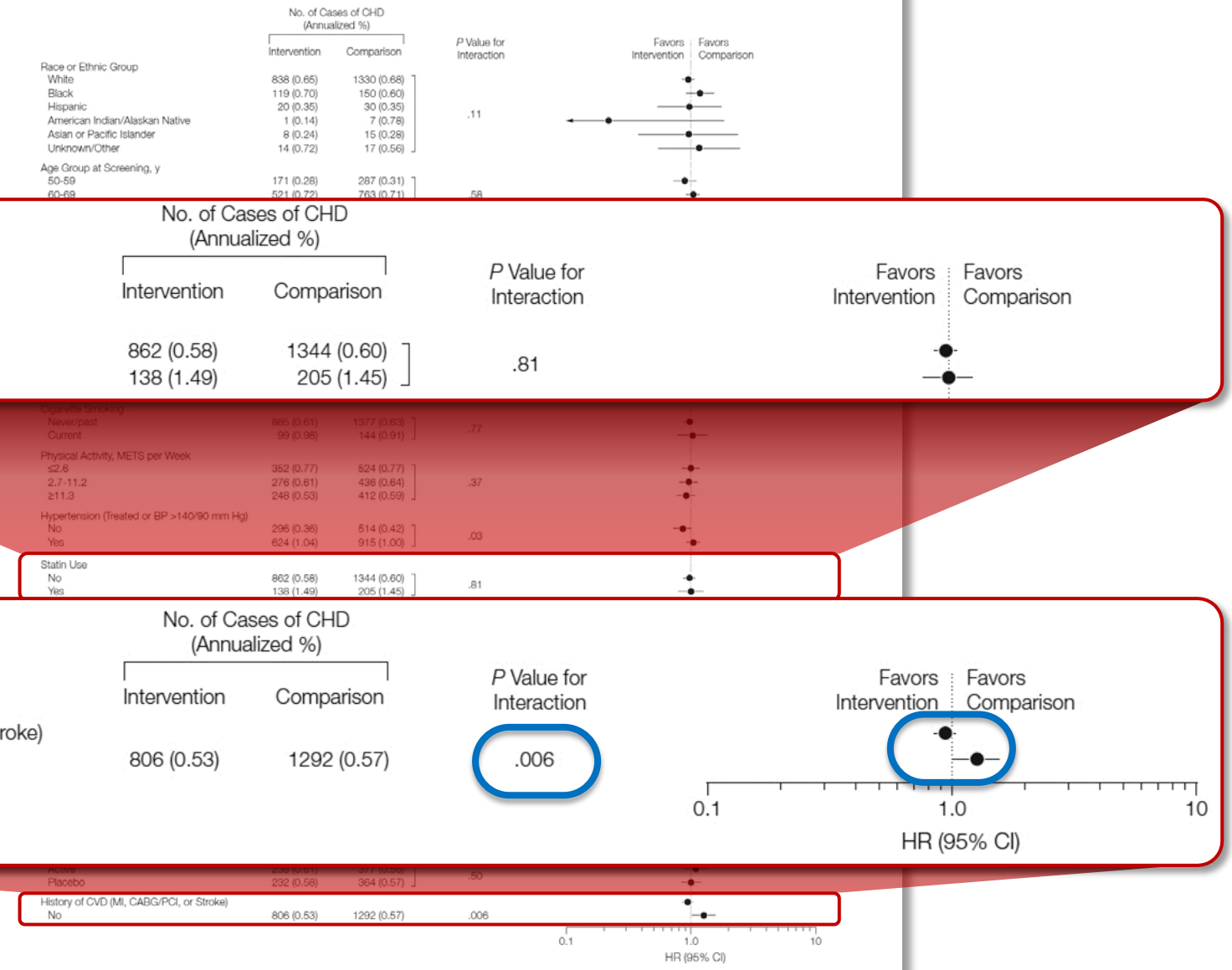

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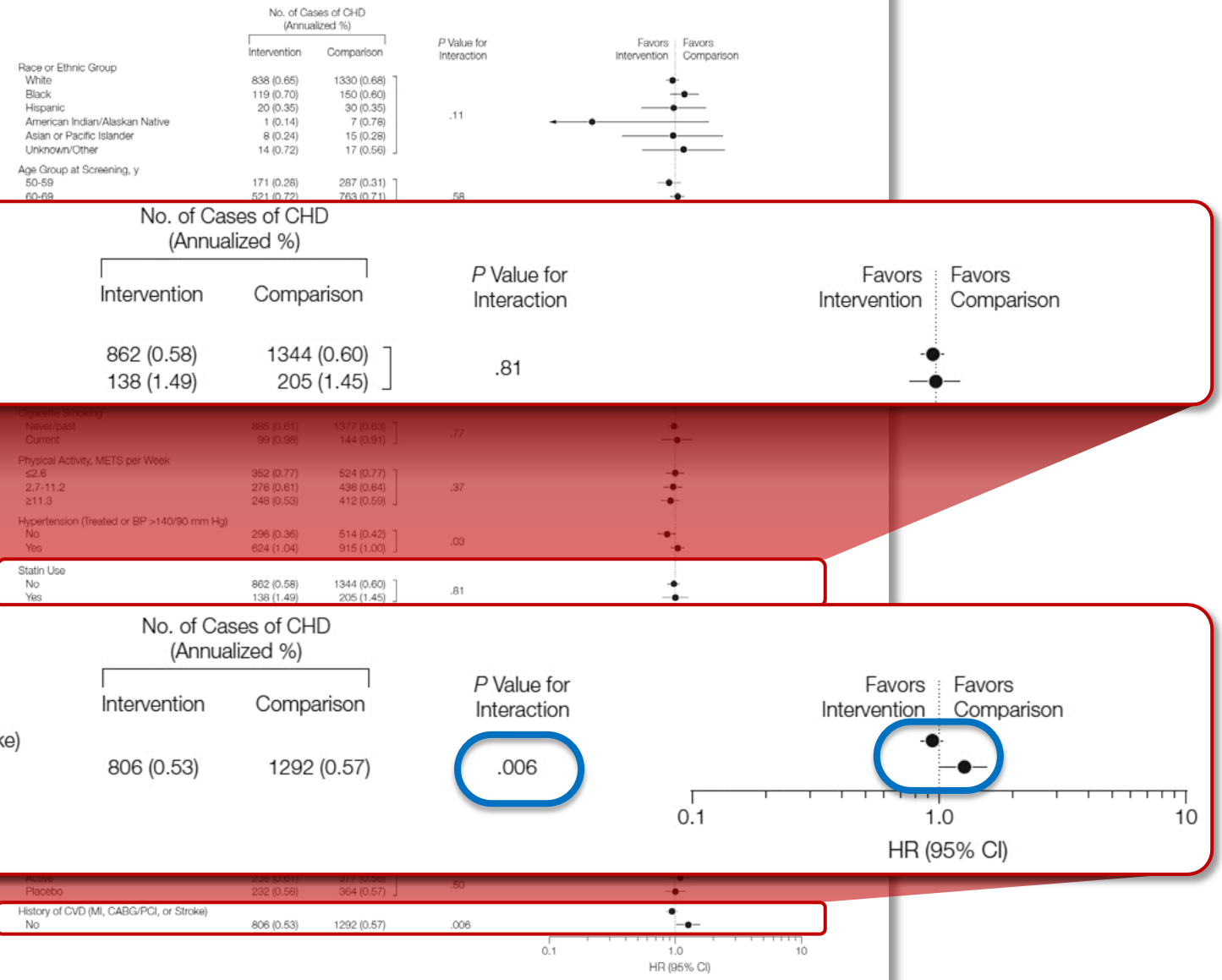

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*Conclusions do not change if results from 4-day food records are used.

Effects of a low-fat dietary intervention on glucose, insulin, and insulin resistance in the Women's Health Initiative (WHI) Dietary Modification trial[1–3]

James M Shikany, Karen L Margolis, Mary Pettinger, Rebecca D Jackson, Marian C Limacher, Simin Liu, Lawrence S Phillips, and Lesley F Tinker

The American Journal of Clinical Nutrition

ABSTRACT

Background: Glycemic effects of the Women's Health Initiative (WHI) low-fat dietary intervention are unknown.

Objective: Our objective was to analyze the effects of the WHI low-fat dietary intervention on serum glucose and insulin and insulin resistance up to 6 y after random assignment.

Design: Postmenopausal WHI Dietary Modification trial intervention (DM-I) and comparison (DM-C) participants with blood measures at least at baseline and year 1 (n = 2263) were included. Anthropometric measures, dietary assessments, serum glucose and insulin concentrations, homeostasis model assessment of insulin resistance (HOMA-IR) measures, and quantitative insulin sensitivity check index (QUICKI) values were obtained at baseline, year 1, year 3, and year 6. Changes in measures were compared between groups at years 1, 3, and 6 overall and within stratified analyses.

Results: Mean (±SD) differences in changes at year 1 between the DM-I and DM-C groups were as follows: glucose, −1.7 ± 17.9 mg/dL; insulin, −0.7 ± 5.1 μIU/mL; HOMA-IR, −0.2 ± 1.9; and QUICKI, 0.004 ± 0.019 (all $P < 0.05$). Similar findings resulted from repeated-measures analyses comparing the intervention and comparison groups over the 6 y. Whereas normoglycemic women at baseline had a decrease in glucose at year 1 that was 1.9 ± 17.2 mg/dL greater in the DM-I than in the DM-C group, diabetic women had an increase in glucose that was 7.9 ± 20.3 mg/dL greater in the DM-I than in the DM-C group (P for interaction <0.001).

Conclusions: A low-fat diet was not significantly associated with adverse glycemic effects up to 6 y after random assignment in postmenopausal women. However, diabetic women experienced adverse glycemic effects of the low-fat diet. This trial is registered at clinicaltrials.gov as NCT00000611. *Am J Clin Nutr* 2011;94:75–85.

INTRODUCTION

The optimal macronutrient content of the diet for human health remains a major controversy in nutritional science. Low-fat diets in general, and the Women's Health Initiative (WHI) low-fat dietary intervention in particular, have been criticized for their potential to substitute unhealthy carbohydrates for fat, potentially contributing to hyperglycemia, hyperinsulinemia, and insulin resistance (1).

The WHI Dietary Modification (DM) trial was designed to test the effects of a dietary pattern low in total fat, along with increased vegetables, fruit, and grains, on primarily breast cancer and colorectal cancer incidence in postmenopausal women during a mean follow-up of 8.1 y. Despite the increased intake of carbohydrate in the intervention group, and question of associated increased risk of diabetes, no increase in diabetes risk was observed. Subgroup analysis suggested that greater decreases in percentage of energy from total fat reduced diabetes risk (P for trend = 0.04); however, that finding was not statistically significant after adjustment for weight loss—a common effect of eating a low-fat diet (2).

Details of the effects of the WHI diet intervention on glucose, insulin, and insulin resistance have not been reported. The aim of this report was to analyze the effect of the overall diet intervention, and the specific effects of fiber and whole grain intakes, and dietary glycemic index (GI) and glycemic load (GL) on glucose, insulin, and insulin resistance in the WHI DM trial.

SUBJECTS AND METHODS

WHI DM trial

Recruitment

Details of the study design and methods were published previously (3). All women provided written informed consent, and

[1] From the Division of Preventive Medicine, School of Medicine, University of Alabama at Birmingham, Birmingham, AL (JMS); HealthPartners Research Foundation, Minneapolis, MN (KLM); the Division of Public Health Sciences, Fred Hutchinson Cancer Research Center, Seattle, WA (MP and LFT); the Department of Internal Medicine, Division of Endocrinology, Diabetes and Metabolism, The Ohio State University, Columbus, OH (RDJ); the Division of Cardiovascular Medicine, University of Florida College of Medicine, Gainesville, FL (MCL); the Department of Epidemiology, University of California, Los Angeles, Los Angeles, CA (SL); and the Atlanta VA Medical Center and Division of Endocrinology, Department of Medicine, Emory University School of Medicine, Atlanta, GA (LSP).

[2] Supported by the National Heart, Lung, and Blood Institute, National Institutes of Health, US Department of Health and Human Services (contract numbers N01WH22110, 24152, 32100-2, 32105-6, 32108-9, 32111-13, 32115, 32118-32119, 32122, 42107-26, 42129-32, and 44221).

[3] Address correspondence to JM Shikany, Division of Preventive Medicine, School of Medicine, University of Alabama at Birmingham, 1530 3rd Avenue S, MT 610, Birmingham, AL 35294. E-mail: jshikany@dopm.uab.edu.

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Shikany JM, Margolis KL, Pettinger M, et al. Effects of a low-fat dietary intervention on glucose, insulin, and insulin resistance in the Women's Health Initiative (WHI) Dietary Modification trial. *Am J Clin Nutr* 2011; 94: 75-85.

"….women with diabetes at baseline did experience adverse glycemic effects of the low-fat diet, which indicated that **caution should be exercised in recommending a reduction in overall dietary fat in women with diabetes** unless accompanied by additional recommendations to guide carbohydrate intake".

Shikany JM et al. *Am J Clin Nutr* 2011; 94: 75-85.

ORIGINAL INVESTIGATION

Long-term Effects of a Lifestyle Intervention on Weight and Cardiovascular Risk Factors in Individuals With Type 2 Diabetes Mellitus

Four-Year Results of the Look AHEAD Trial

The Look AHEAD Research Group

Background: Lifestyle interventions produce short-term improvements in glycemia and cardiovascular disease (CVD) risk factors in individuals with type 2 diabetes mellitus, but no long-term data are available. We examined the effects of lifestyle intervention on changes in weight, fitness, and CVD risk factors during a 4-year study.

Methods: The Look AHEAD (Action for Health in Diabetes) trial is a multicenter randomized clinical trial comparing the effects of an intensive lifestyle intervention (ILI) and diabetes support and education (DSE; the control group) on the incidence of major CVD events in 5145 overweight or obese individuals (59.5% female; mean age, 58.7 years) with type 2 diabetes mellitus. More than 93% of participants provided outcomes data at each annual assessment.

Results: Averaged across 4 years, ILI participants had a greater percentage of weight loss than DSE participants (−6.15% vs −0.88%; $P<.001$) and greater improvements in treadmill fitness (12.74% vs 1.96%; $P<.001$), hemoglobin A_{1c} level (−0.36% vs −0.09%; $P<.001$), systolic (−2.92 vs −2.48 mm Hg; $P=.01$) blood pressure, and levels of high-density lipoprotein cholesterol (3.67 vs 1.97 mg/dL; $P<.001$) and triglycerides (−25.56 vs −19.75 mg/dL; $P<.001$). Reductions in low-density lipoprotein cholesterol levels were greater in DSE than ILI participants (−11.27 vs −12.84 mg/dL; $P=.009$) owing to greater use of medications to lower lipid levels in the DSE group. At 4 years, ILI participants maintained greater improvements than DSE participants in weight, fitness, hemoglobin A_{1c} levels, systolic blood pressure, and high-density lipoprotein cholesterol levels.

Conclusions: Intensive lifestyle intervention can produce sustained weight loss and improvements in fitness, glycemic control, and CVD risk factors in individuals with type 2 diabetes. Whether these differences in risk factors translate to reduction in CVD events will ultimately be addressed by the Look AHEAD trial.

Trial Registration: clinicaltrials.gov Identifier: NCT00017953

Look AHEAD Research Group, Wing RR. Long-term effects of a lifestyle intervention on weight and cardiovascular risk factors in individuals with type 2 diabetes mellitus: four-year results of the Look AHEAD trial. *Arch Intern Med* 2010; 170: 1566-1575.

The Look AHEAD Trial was terminated prematurely in October 2012 after 11.5 years as it was found that even when combined with exercise, the Prudent diet had no measureble effect on development of arterial disease and its complications in persons with Type 2 Diabetes. Continuing the trial was considered “pointless”.

CONCLUSION:

The WHI provides the definitive evidence from a randomized controlled clinical trial that disproves the diet-heart hypothesis. Following the 1977 US "Prudent" Diet Guidelines worsens the outcome of those who are the most vulnerable because they have either heart disease or diabetes.

The 48 836-person Woman's Health Initiative proves that the 1977 US Dietary Guidelines accelerate disease progression in persons with either known heart disease or diabetes. Thus the research of my opponent provides the definitive evidence that disproves Keys' false diet-heart hypothesis.

Economic considerations drove the adoption of the current dietary guidelines without proper scientific evaluation or proof.

Within 5 years of the widespread adoption of these guidelines rates of diabetes and obesity increased explosively.

The presence of the genetic predisposing condition, carbohydrate [illegible] explains why large numbers of [illegible] predisposed [illegible] become obese and diabetic when [illegible] carbohydrate diet.

A high fat diet reverses all known coronary risk factors in persons with [illegible] carbohydrate-resistance [illegible] worsens those

The 48 836-person Woman's Health Initiative of which my opponent was the Project Leader proves that the 1977 US Dietary Guidelines [illegible] accelerate disease progression in either known heart disease or diabetes. Thus that landmark study provides the definitive evidence disproving Keys' false diet-heart hypothesis.

CONCLUSION:

The Diet Heart Hypothesis of Ancel Keys is WRONG.

Its widespread promotion in the name of good science represents the single greatest error in medicine in the past 60 years.