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

Professor TD Noakes  OMS, MBChB, MD, DSc, PhD (hc), FACSM, (hon) FFSEM (UK)

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

[Logos of various organizations]
GOOD CALORIES, BAD CALORIES

FATS, CARBS, AND THE SCIENCE OF DIET

GARY TAUROG

GOOD CALORIES, BAD CALORIES

FATS, CARBS, AND THE SCIENCE OF DIET

GARY TAUROG

The Cholesterol Conspire

by

Russell L. Smith, M.D.
in consultation with
Edward R. Pinto, M.D.

“Saturated fat and cholesterol are the cause of coronary heart disease—probably the greatest scientific deception of any century.”

Ben Goldacre
Bestselling author of Bad Science

Bad Pharma

How drug companies mislead doctors and harm patients

364 pages
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TODAY

2.5 - 3.5 MILLION YEARS AGO

Omnivore

Vegetarian

Homo sapiens

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

This change occurred as humans became the best mid-day persistence hunters in the animal kingdom.
PLAINS INDIANS HUNTING BUFFALO BEFORE THE ARRIVAL OF THE WHITE MAN

MODERN PLAINS INDIANS HUNTING
THE WHITE MAN’S DIET
# THE COUNTRIES WITH THE HIGHEST PERCENTAGE OF OBESE ADULTS

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

Data from World Health Organization. Source: See www.robbrooks.net/rob-brooks/1317 for the full 137 countries
ANCEL KEYS (1904-2004)

CHD (deaths per 1000)

Percent calories from fat

Japan

Italy

England & Wales

Canada

Australia

US

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.
“….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.”

“…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.**”
“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.
• 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.
“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
“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
“...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”.

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.

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


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-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.

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.

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MILLIONS OF YEARS
28 YEARS
INFLUENCE OF 1977 DIETARY GUIDELINES ON % OBESITY IN USA

Dietary goals for Americans released

Percent with BMI > 30kg.m²

Year


18-29 years
30-44 years
45-64 years
65 years & over
18-29 years

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

% of Americans with diabetes

1980 2010

% of US children who are obese

1980 2010

% of US adults who are obese

1980 2010

Data from USDA, CDC, US Census Bureau
Economic considerations drove the adoption of the current dietary guidelines without proper scientific evaluation or proof.

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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 Woman's Health Initiative Randomized Controlled Dietary Modification Trial (WHIRCDMT) of which my opponent was Project Leader proves that the prescription of a high carbohydrate diet to persons with either known heart disease or diabetes constitutes medical malpractice.

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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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 condition of carbohydrate resistance


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

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated blood glucose concentrations</td>
</tr>
<tr>
<td>Elevated blood insulin concentrations</td>
</tr>
<tr>
<td>Elevated HbA1c concentrations</td>
</tr>
<tr>
<td>Elevated blood triglyceride concentrations</td>
</tr>
<tr>
<td>Reduced blood HDL-cholesterol concentrations (HDL-C)</td>
</tr>
<tr>
<td>Increased small LDL-cholesterol particles (LDL-C P)</td>
</tr>
<tr>
<td>Increased blood uric acid concentrations</td>
</tr>
<tr>
<td>Increased blood ultrasensitive CRP concentrations</td>
</tr>
</tbody>
</table>

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)

OBESITY, DIABETES, HYPERTENSION, GOUT, METABOLIC SYNDROME ARE SEPARATE/DISTINCT DISEASES
<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Hazard Ratio (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>2.04 (1.76 – 2.35)</td>
</tr>
<tr>
<td>Age</td>
<td>1.87 (1.73 – 2.02)</td>
</tr>
<tr>
<td>Current smoking</td>
<td>1.79 (1.66 – 1.94)</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>1.31 (1.26 – 1.37)</td>
</tr>
<tr>
<td>Total [Cholesterol]</td>
<td>1.22 (1.17 – 1.27)</td>
</tr>
<tr>
<td>[Triglyceride]</td>
<td>1.19 (1.15 – 1.23)</td>
</tr>
<tr>
<td>[HDL-Cholesterol]</td>
<td>0.83 (0.78 – 0.87)</td>
</tr>
</tbody>
</table>

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

Hemoglobin A1C concentrations (%)

Age-adjusted relative risk (95% CI)

0
1
2
3
4
5
6
7
8

Coronary heart disease events

All-cause mortality

Carbohydrate resistance
“Pre-diabetes”

Total Cholesterol
Hazard Ratio

<5.0
5.5-5.9
6.0-6.4
6.5-6.9
>7.0

Known diabetes

Hemoglobin A1c concentrations (%)

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

Ischemic heart disease

Myocardial infarction

Overall log rank p<0.001

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

Fasting glucose levels within the high normal range predict 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
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.

CONCLUSION:
Their abnormal carbohydrate metabolism explains why those with carbohydrate resistance develop obesity, diabetes and coronary heart disease when eating a high carbohydrate diet.
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.
A HIGH FAT DIET REVERSES ALL CORONARY RISK FACTORS MORE EFFECTIVELY THAN A LOW FAT DIET

“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”.

THE DIETARY CARBOHYDRATE HYPOTHESIS FOR HEART DISEASE
Hyperglycaemic Hyperinsulinaemic Atherogenic Dyslipidaemia (HHAD)

LOW OMEGA 3, HIGH OMEGA 6

ONE CAUSE, ONE TREATMENT FOR ALL CONDITIONS

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

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

High carbohydrate (fructose) diet *
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.

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.
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.

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.
48,836 post-menopausal women were assigned to a low-fat dietary pattern, where they reduced energy from fat to 20% and from saturated fat to 7%, and increased fruit and vegetable intake to at least five servings per day and grains to at least six servings per day. 60% of the subjects were assigned to self-selected dietary behavior. Control subjects received a copy of Dietary Guidelines for Americans “as well as other health-related material but had no contact with nutritional interventionists.” Subjects were followed for 8.1 years.
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?
Low-Fat Dietary Pattern and Risk of Cardiovascular Disease
The Women's Health Initiative Randomized Controlled Dietary Modification Trial

Objective: To test the hypothesis that a low-fat diet, in addition to improving other health-related outcomes, reduces the risk of CVD.

Methods: A randomised controlled trial with 48,835 postmenopausal women aged 50 to 79 years, with a mean age of 67 years and a mean BMI of 30 kg/m², were randomly assigned to one of three dietary groups: low-fat, moderate-fat, or high-fat. The diet was followed for 8 years.

Results: The low-fat diet group had a 30% reduction in the risk of CVD compared to the moderate-fat group. The risk was reduced by 25% in the high-fat group compared to the low-fat group.

Conclusion: A low-fat diet reduces the risk of CVD by 30%, and a moderate-fat diet reduces the risk by 25%, compared to a high-fat diet.

Clinical Trials Registration: ClinicalTrials.gov Identifier: NCT0000001

See also pp. 123, 445, and 694.
This is entirely predictable as a high carbohydrate diet produces HHAD (hyperglycaemic hyperinsulinaemic atherogenic dyslipidaemia) in those who are metabolically vulnerable.
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 may not reduce the risk of breast cancer, heart disease, or stroke, and did not change the risk of colorectal cancer, heart disease, or stroke, according to researchers at the National Institutes of Health's Women's Health Initiative.

The study was designed to evaluate a low-fat diet for reducing colorectal cancer. However, investigators also evaluated the effect of the diet on a variety of cardiovascular disease. The results from the study were reported in three papers in the February 8 edition of the Journal of the American Medical Association.

Among the 48,835 women who participated in the trial, there were no differences in the rates of colorectal cancer, heart disease, or stroke among those who followed a low-fat dietary plan and the control group. The women 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 occurred by chance.
“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.
News from the Women's Health Initiative
Reducing Total Fat Intake May Not Affect on Risk of Breast Cancer, Risk of Colorectal Cancer, Heart Disease, Stroke

Following an eating pattern lower in total fat did not significantly reduce the risk of breast cancer, heart disease, or stroke, and did not reduce colorectal cancer risk in healthy postmenopausal women, according to the latest clinical findings from the National Institutes of Health's Women's Health Initiative (WHI). The study was designed to evaluate a low-fat dietary pattern for heart disease and colorectal cancer. However, investigators also evaluated the data to see whether it could protect against cardiovascular disease. The results from the largest ever clinical trial focused on dietary fats were reported in three papers in the February 8 edition of the Journal of the American Medical Association.

Among the 48,835 women who participated in the trial, there were no significant differences in the rates of colorectal cancer, heart disease, or stroke between women who followed a low-fat dietary plan and the comparison group with no dietary changes. 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 happened by chance.
“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
“It is difficult to get a man to understand something, when his salary depends upon his not understanding it”.

THE UPTON SINCLAIR THEOREM
Figure 3. Risk of Composite Coronary Heart Disease (CHD) in Various Subgroups

<table>
<thead>
<tr>
<th>Race or Ethnic Group</th>
<th>No. of Cases of CHD (Annexed %)</th>
<th>P Value for Interaction</th>
<th>Favor of Intervention</th>
<th>Favor of Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>808 (0.65) 1330 (0.68)</td>
<td>.11</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Black</td>
<td>119 (0.79) 180 (0.62)</td>
<td>.33</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Hispanic</td>
<td>20 (0.35) 30 (0.35)</td>
<td>.007</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>American Indian/Alaskan Native</td>
<td>1 (0.14) 7 (0.78)</td>
<td>.58</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Asian or Pacific Islander</td>
<td>8 (0.24) 15 (0.28)</td>
<td>.77</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Unknown/Other</td>
<td>14 (0.72) 17 (0.56)</td>
<td>.039</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Age Group at Screening, y</td>
<td>50-59 80-69 70-79</td>
<td>-</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Family History of Premature MI</td>
<td>No 556 (0.72) 918 (0.54)</td>
<td>.77</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Yes</td>
<td>270 (1.08) 419 (1.10)</td>
<td>-</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Waist Circumference, cm</td>
<td>≤30.5 31-35.9 36+</td>
<td>.19</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Physical Activity, METS per Week</td>
<td>≤2.6 2.7-11.2 11.3+</td>
<td>.37</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Hypertension (Treated or BP ≥140/90 mm Hg)</td>
<td>No 295 (0.28) 514 (0.42)</td>
<td>.03</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Yes</td>
<td>424 (1.04) 915 (1.52)</td>
<td>-</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>B Vitamins Use</td>
<td>No 862 (0.62) 1344 (0.60)</td>
<td>.81</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Yes</td>
<td>126 (1.06) 205 (1.48)</td>
<td>-</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Treated for Diabetes (FBG or reactions)</td>
<td>No 834 (0.65) 1302 (0.57)</td>
<td>.71</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Yes</td>
<td>166 (1.05) 247 (2.44)</td>
<td>-</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Baseline % Energy From Fat*</td>
<td>&lt;33.84 33.85-40.90 40.90-48.80</td>
<td>.087</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Baseline Energy From Saturated Fat, %</td>
<td>≤10.0 10.0-15.2 15.3-19.4</td>
<td>.96</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Randomized to Hormone Therapy Trial</td>
<td>Active 94 (0.73) 145 (0.72)</td>
<td>.40</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Placebo</td>
<td>80 (0.70) 150 (0.70)</td>
<td>-</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Randomized to Ca2+ Trial</td>
<td>Active 236 (0.61) 377 (0.56)</td>
<td>.50</td>
<td>●</td>
<td>●</td>
</tr>
<tr>
<td>Placebo</td>
<td>232 (0.58) 364 (0.57)</td>
<td>-</td>
<td>●</td>
<td>●</td>
</tr>
</tbody>
</table>

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

### No. of Cases of CHD (Annualized %)

<table>
<thead>
<tr>
<th>Statin Use</th>
<th>Intervention</th>
<th>Comparison</th>
<th>P Value for Interaction</th>
<th>Favors</th>
<th>Favors</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>862 (0.58)</td>
<td>1344 (0.60)</td>
<td>.81</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>138 (1.49)</td>
<td>205 (1.45)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Physical Activity, METS per Week</th>
<th>Intervention</th>
<th>Comparison</th>
<th>P Value for Interaction</th>
<th>Favors</th>
<th>Favors</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤2.6</td>
<td>885 (0.61)</td>
<td>1377 (0.68)</td>
<td>.77</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2.7.11.2</td>
<td>95 (0.98)</td>
<td>144 (0.91)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥11.0</td>
<td>1252 (0.77)</td>
<td>524 (0.77)</td>
<td>.27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension (Treated or BP &gt;140/90 mm Hg)</td>
<td>Intervention</td>
<td>Comparison</td>
<td>P Value for Interaction</td>
<td>Favors</td>
<td>Favors</td>
</tr>
<tr>
<td>No</td>
<td>290 (0.68)</td>
<td>514 (0.42)</td>
<td>.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>424 (0.68)</td>
<td>915 (0.12)</td>
<td></td>
<td></td>
<td></td>
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### History of CVD (MI, CABG/PCI, or Stroke)

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<tr>
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<td>806 (0.53)</td>
<td>1292 (0.57)</td>
<td>.006</td>
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*pConclusions do not change if results from 4 day food records are used.*

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### Risk of Composite Coronary Heart Disease (CHD) in Various Subgroups

**Figure 3**

#### No. of Cases of CHD

#### Statin Use

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

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 A1c level (−0.36% vs −0.09%; P < .001), systolic blood pressure (−2.92 vs −2.48 mm Hg; P = .01), diastolic 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). Reducions 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 A1c 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

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 measurable effect on development of arterial disease and its complications in persons with Type 2 Diabetes. Continuing the trial was considered “pointless”.
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.

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.

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.
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.

The 48,836-person Woman’s Health Initiative of which my opponent was the Project Leader proves that the 1977 US Dietary Guidelines accelerate disease progression in persons with 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.