



Professor Grant Schofield

Public Health

Auckland University of Technology

Auckland

Friday, June 9, 2017

(Room 2)

16:30 - 17:25 WS #72: A New Understanding of Dietary Carbs and Fats

17:35 - 18:30 WS #84: A New Understanding of Dietary Carbs and Fats
(Repeated)

A changing view of carbs and fat

Grant Schofield

Professor of Public Health

AUT University, Auckland NZ



@grantsnz

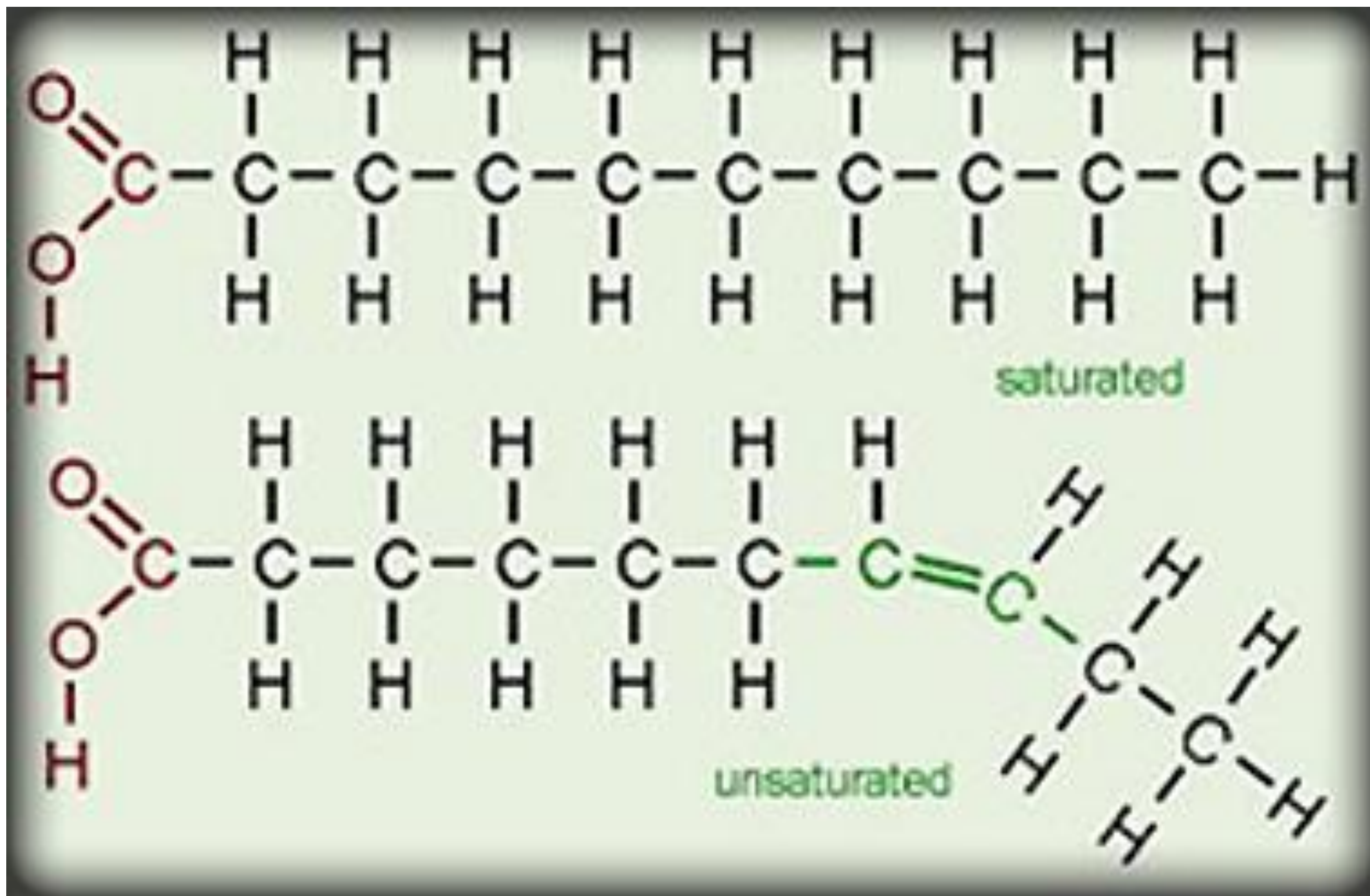


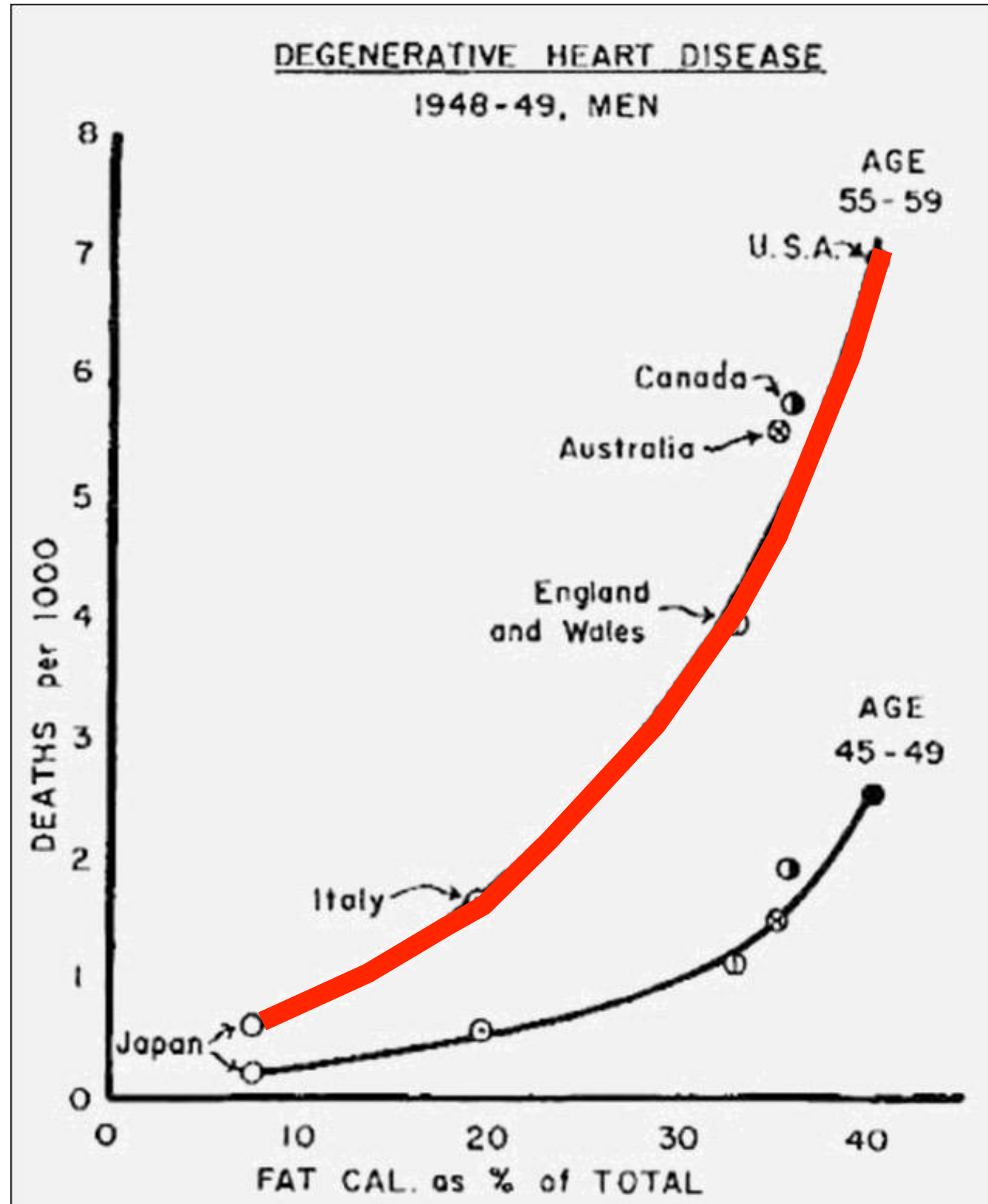
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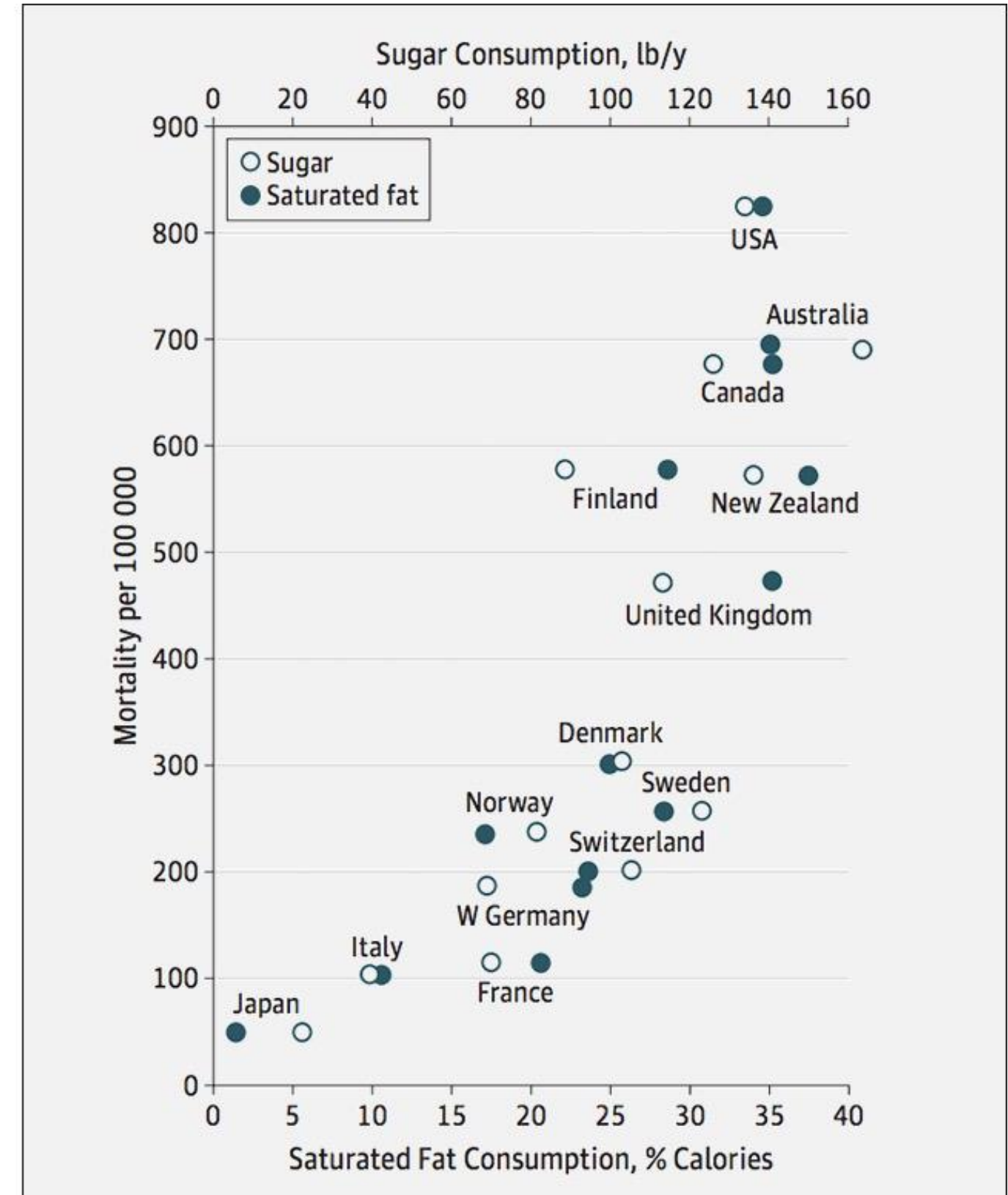
1. Ancestral Keys changed the way
we see the world







What Ancel Keys said.



What Ancel Keys didn't say.

Saturated fat does not clog the arteries: coronary heart disease is a chronic inflammatory condition, the risk of which can be effectively reduced from healthy lifestyle interventions

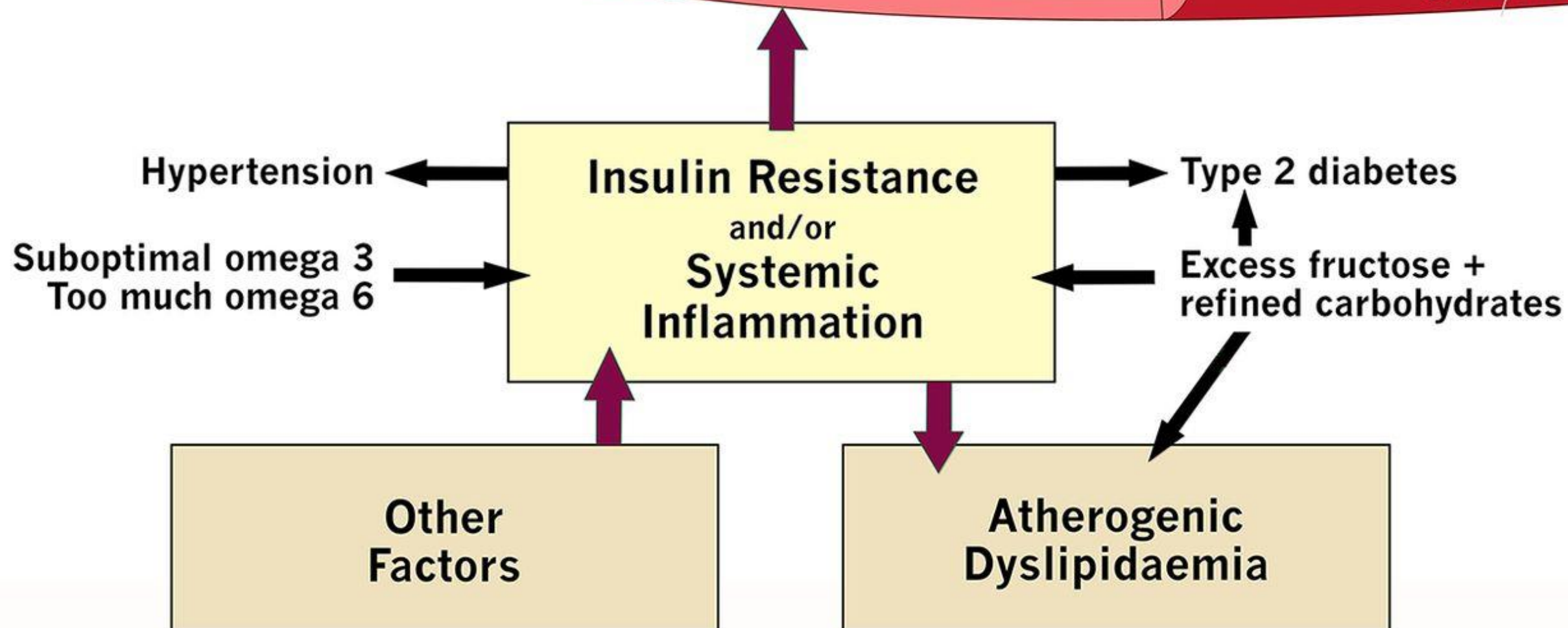
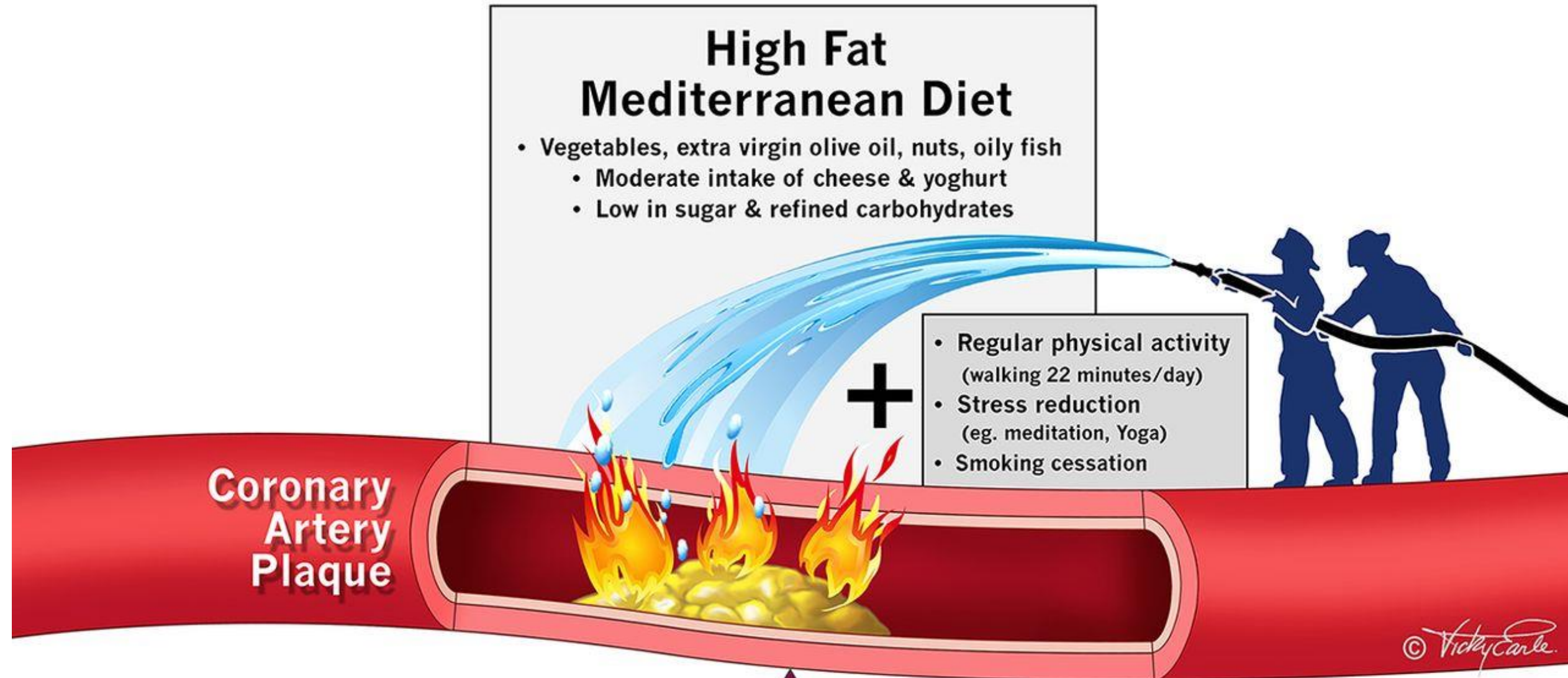
Aseem Malhotra,¹ Rita F Redberg,^{2,3} Pascal Meier^{4,5}

Coronary artery disease pathogenesis and treatment urgently requires a paradigm shift. Despite popular belief among doctors and the public, the conceptual model of dietary saturated fat clogging a pipe is just plain wrong. A landmark systematic review and meta-analysis of observational studies showed no association between saturated fat consumption

testing.⁴ When plaques rupture (analogous to a pimple bursting), coronary thrombosis and myocardial infarction can occur within minutes. The limitation of the current plumbing approach ('unclogging a pipe') to the management of coronary disease is revealed by a series of randomised controlled trials (RCTs) which prove that

7500 high-risk patients. Furthermore, the Lyon Heart study showed that adopting a Mediterranean diet in secondary prevention improved hard outcomes for both recurrent myocardial infarction (NNT=18) and all-cause mortality (NNT=30), despite there being no significant difference in plasma low-density lipoprotein (LDL) cholesterol between the two groups. It is the alpha linoleic acid, polyphenols and omega-3 fatty acids present in nuts, extra virgin olive oil, vegetables and oily fish that rapidly attenuate inflammation and coronary thrombosis.⁶ Both control diets in these studies were relatively healthy, which make it highly likely that even larger benefits would be observed if the Mediterranean diets discussed above were compared with a typical western diet.

LDL CHOLESTEROL RISK HAS BEEN



2. New views and evidence are
emerging

JAMA Internal Medicine | Special Communication

Sugar Industry and Coronary Heart Disease Research

A Historical Analysis of Internal Industry Documents

Cristin E. Kearns, DDS, MBA; Laura A. Schmidt, PhD, MSW, MPH; Stanton A. Glantz, PhD

Early warning signals of the coronary heart disease (CHD) risk of sugar (sucrose) emerged in the 1950s. We examined Sugar Research Foundation (SRF) internal documents, historical reports, and statements relevant to early debates about the dietary causes of CHD and assembled findings chronologically into a narrative case study. The SRF sponsored its first CHD research project in 1965, a literature review published in the *New England Journal of Medicine*, which singled out fat and cholesterol as the dietary causes of CHD and downplayed

 [Invited Commentary](#)

 [Author Audio Interview](#)

 [Supplemental content](#)

Together with other recent analyses of sugar industry documents about the hazards of sucrose while promoting fat as the dietary

Metabolic disease

**Its about the glycemc
environment**

glucose, insulin, and
insulin resistance

Atherogenic dyslipidemia
is [primarily] **driven by the**
glycemic environment,
especially sugar and refined
carbohydrate in the context
of insulin resistance

**A balanced diet [in the
context of whole food] is
one which normalises
blood glucose and
prevents the hyper-
secretion of insulin**



[Online First >](#)

Viewpoint | September 28, 2016

Lowering the Bar on the Low-Fat Diet **FREE**

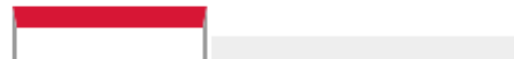
ONLINE FIRST

David S. Ludwig, MD, PhD¹

[\[+\] Author Affiliations](#)

JAMA. Published online September 28, 2016. doi:10.1001/jama.2016.15473

Text Size: [A](#) [A](#) [A](#)





Re-evaluation of the traditional diet-heart hypothesis: analysis of recovered data from Minnesota Coronary Experiment (1968-73)

Christopher E Ramsden,^{1,2} Daisy Zamora,³ Sharon Majchrzak-Hong,¹ Keturah R Faurot,² Steven K Broste,⁴ Robert P Frantz,⁵ John M Davis,^{3,6} Amit Ringel,¹ Chirayath M Suchindran,⁷ Joseph R Hibbeln¹

¹Section on Nutritional Neuroscience, Laboratory of Membrane Biochemistry and Biophysics, National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health, Bethesda, MD, USA

²Department of Physical Medicine and Rehabilitation, Program on Integrative Medicine, University of North Carolina, Chapel Hill, NC, USA

³Department of Psychiatry, University of North Carolina, Chapel Hill, NC, USA

⁴Medtronic, Minneapolis, MN, USA

⁵University of Minnesota, Minneapolis, MN, USA

⁶University of Minnesota, Minneapolis, MN, USA

⁷University of Minnesota, Minneapolis, MN, USA

ABSTRACT

OBJECTIVE

To examine the traditional diet-heart hypothesis through recovery and analysis of previously unpublished data from the Minnesota Coronary Experiment (MCE) and to put findings in the context of existing diet-heart randomized controlled trials through a systematic review and meta-analysis.

DESIGN

The MCE (1968-73) is a double blind randomized controlled trial designed to test whether replacement of saturated fat with vegetable oil rich in linoleic acid reduces coronary heart disease and death by lowering serum cholesterol. Recovered MCE unpublished

oil polyunsaturated margarine). Control diet was high in saturated fat from animal fats, common margarines, and shortenings.

MAIN OUTCOME MEASURES

Death from all causes; association between changes in serum cholesterol and death; and coronary atherosclerosis and myocardial infarcts detected at autopsy.

RESULTS

The intervention group had significant reduction in serum cholesterol compared with controls (mean change from baseline -13.8% v -1.0% ; $P<0.001$). Kaplan Meier graphs showed no mortality benefit for the intervention group in the full randomized cohort or

Insulin Causes Endothelial Dysfunction in Humans

Sites and Mechanisms

Guido Arcaro, MD; Anna Cretti, MD; Sara Balzano, MD; Alessandro Lechi, MD;
Michele Muggeo, MD; Enzo Bonora, MD, PhD; Riccardo C. Bonadonna, MD

Background—Insulin resistance is often accompanied by hyperinsulinemia and may predispose to atherosclerosis. Endothelium plays a central role in atherogenesis. The in vivo effects of hyperinsulinemia on endothelial function of large conduit arteries are unknown.

Methods and Results—Twenty-five healthy subjects were enrolled for study. In study A (n=9), subjects underwent both a time-control saline study and a euglycemic low-dose insulin (insulin ~ 110 pmol/L) clamp for 6 hours. Study B (n=5) was identical to study A except that the euglycemic clamp was performed at high physiological insulin concentrations (~ 440 pmol/L). In study C (n=7), subjects underwent two 4-hour euglycemic insulin (~ 110 pmol/L) clamps with and without the concomitant infusion of an antioxidant (vitamin C). In study D (n=4), two saline time-control studies were performed with and without the concomitant infusion of vitamin C. In all studies, both at baseline and throughout the experimental period, endothelium-dependent (flow-mediated) and endothelium-independent (nitroglycerin-induced) vasodilation was assessed in femoral and brachial arteries by echo Doppler. Both low (study A) and high physiological (study B) hyperinsulinemia abolished endothelium-dependent vasodilation, whereas endothelium-independent vasodi-

Hi guys,

This is a form of analysis called Mendelian randomisation, which has some strong underlying assumptions, so would take with a grain of salt... As I understand it, every individual in a cohort study has a predicted genetic risk factor score assigned to them (LDL, HDL, BP, Gluc, Insulin) from the findings of a cross-sectional study which links genes with risk factors. The predicted scores are then associated with the disease. The idea is that a form of randomisation occurs when we receive our genes from our parents, so that if these genes have all their effects on CVD through the risk factor that they predict, then this two stage regression gives the causal effect of the gene.

The main assumptions of the method are that the genes don't influence the outcome (CVD), except through the risk factor. It is impossible to verify this...

It seems (genetically) predicted glucose, insulin, triglycerides and LDL are all significantly associated with CVD... This suggests that these factors are

Systematic Review of the Association between Dairy Product Consumption and Risk of Cardiovascular-Related Clinical Outcomes^{1–3}

Jean-Philippe Drouin-Chartier,⁴ Didier Brassard,⁴ Maude Tessier-Grenier,⁴ Julie Anne Côté,⁵ Marie-Ève Labonté,⁶ Sophie Desroches,⁴ Patrick Couture,^{4,7} and Benoît Lamarche^{4*}

⁴Institute of Nutrition and Functional Foods, Laval University, Quebec City, Quebec, Canada; ⁵Institut Universitaire de Cardiologie et de Pneumologie de Québec, Quebec City, Quebec, Canada; ⁶Department of Nutritional Science, Faculty of Medicine, University of Toronto, Toronto, Ontario, Canada; and ⁷CHU de Québec-Université Laval, Quebec City, Quebec, Canada

ABSTRACT

The objective of this systematic review was to determine if dairy product consumption is detrimental, neutral, or beneficial to cardiovascular health and if the recommendation to consume reduced-fat as opposed to regular-fat dairy is evidence-based. A systematic review of meta-analyses of prospective population studies associating dairy consumption with cardiovascular disease (CVD), coronary artery disease (CAD), stroke, hypertension, metabolic syndrome (MetS), and type 2 diabetes (T2D) was conducted on the basis of the PRISMA (Preferred Reporting

Cardiovascular disease

Total dairy

O'Sullivan et al., 2013 (15)
Cin et al., 2012 (19)
Alexander et al., 2016 (24)

High-fat

Low-fat

Dairy fat

Milk

Flanagan et al., 2004 (5)
Rondanelli-Gatti et al., 2011 (8)
O'Sullivan et al., 2013 (15)
Alexander et al., 2016 (24)

Cheese

O'Sullivan et al., 2013 (15)
Alexander et al., 2016 (24)

Yogurt

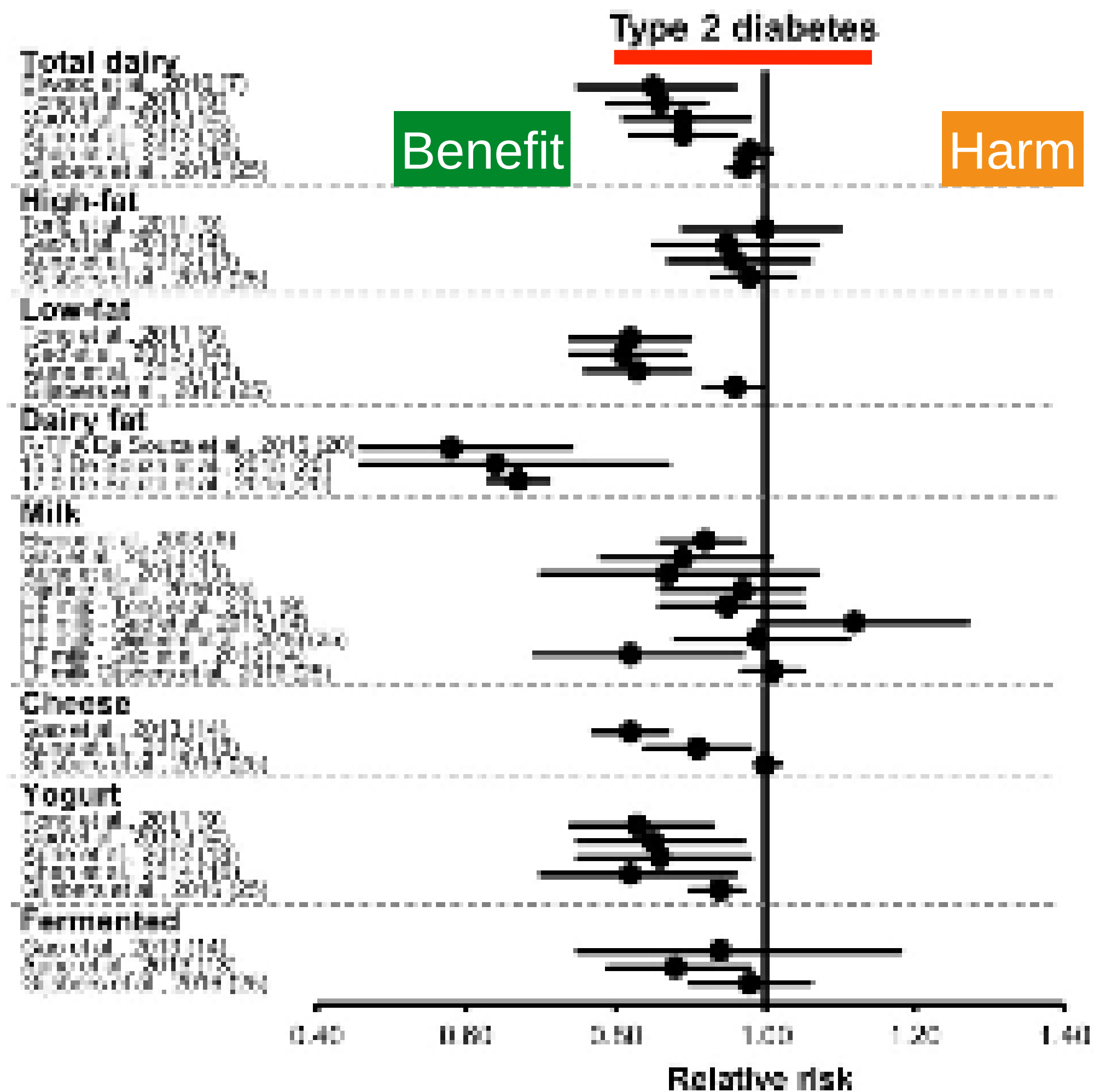
Alexander et al., 2016 (24)

Fermented

Benefit

Harm





3. Old v new?

Fats, Oils & Sweets
USE SPARINGLY

KEY

● Fat (naturally occurring and added)

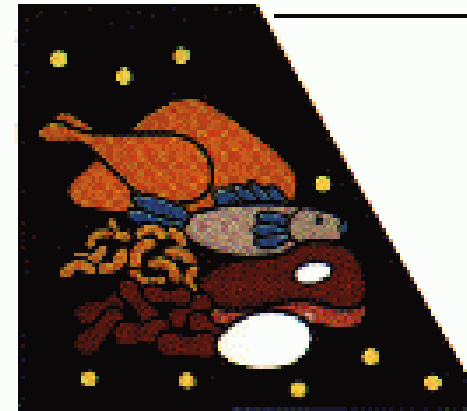
▼ Sugars (added)

These symbols show fats and added sugars in foods.

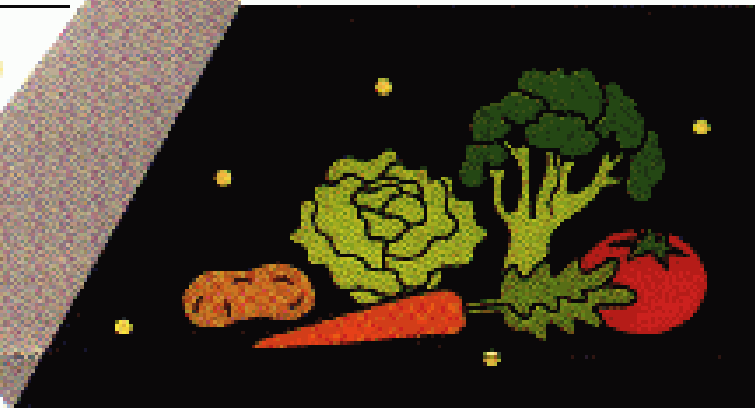
**Milk, Yogurt &
Cheese Group**
2-3 SERVINGS



**Meat, Poultry, Fish, Dry Beans,
Eggs & Nuts Group**
2-3 SERVINGS



Vegetable Group
3-5 SERVINGS



Fruit Group
2-4 SERVINGS



**Bread, Cereal,
Rice & Pasta
Group**
**6-11
SERVINGS**



openheart Evidence from randomised controlled trials did not support the introduction of dietary fat guidelines in 1977 and 1983: a systematic review and meta-analysis

Zoë Harcombe,¹ Julien S Baker,¹ Stephen Mark Cooper,² Bruce Davies,³ Nicholas Sculthorpe,¹ James J DiNicolantonio,⁴ Fergal Grace¹

To cite: Harcombe Z, Baker JS, Cooper SM, *et al*. Evidence from randomised controlled trials did not support the introduction of

ABSTRACT

Objectives: National dietary guidelines were introduced in 1977 and 1983, by the US and UK governments, respectively, with the ambition of

KEY MESSAGES

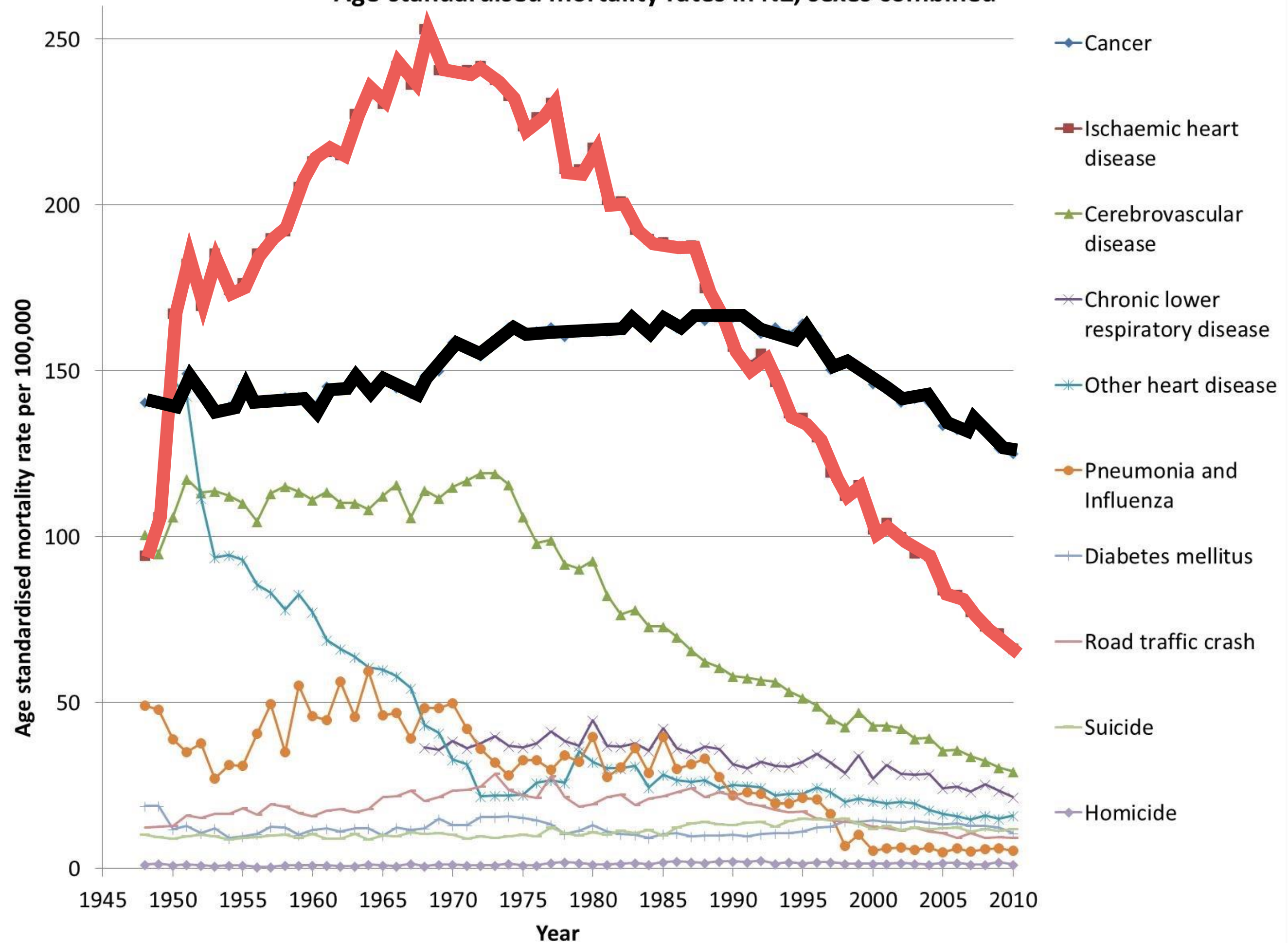
What is already known about this subject?

► Dietary recommendations were introduced in the

PASCAL'S WAGER

	YOU'RE RIGHT!	YOU'RE WRONG!
BELIEF		
ATHEISM		

Age-standardised mortality rates in NZ, sexes combined



VALUE PACK

UNCLE
TOBYS



HONEY
Cheerios[®]
4 WHOLEGRAINS
CORN • WHEAT • OATS • RICE

69% WHOLEGRAIN
NO ARTIFICIAL COLOURS OR FLAVOURS

Energy 490kJ or 6%	Fat 1.2g or 2%	Sat Fat 0.2g or +1%	Sugars 6.0g or 7%	Sodium 90mg or 4%	Fibre 2.0g or 7%
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Per 30g serve

570g e

MADE WITH
NATURAL
HONEY





ORIGINAL ARTICLE

Food consumption and the actual statistics of cardiovascular diseases: an epidemiological comparison of 42 European countries

Pavel Grasgruber*, Martin Sebera, Eduard Hrazdina, Sylva Hrebickova and Jan Cacek

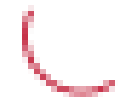
s, health expenditure showed by far the highest correlation coefficients. T

Abstract

Background: The aim of this ecological study was to identify the main nutritional factors related to the prevalence of cardiovascular diseases (CVDs) in Europe, based on a comparison of international statistics.

Design: The mean consumption of 62 food items from the FAOSTAT database (1993–2008) was compared with the actual statistics of five CVD indicators in 42 European countries. Several other exogenous factors (health expenditure, smoking, body mass index) and the historical stability of results were also examined.

Keywords: CVD, Europe, food consumption, health expenditure, smoking, body mass index, historical stability, results



ORIGINAL ARTICLE

Food consumption and the actual statistics of cardiovascular diseases: an epidemiological comparison of 42 European countries

Pavel Grasgruber*, Martin Sebera, Eduard Hrazdira, Sylva Hrebickova and Jan Cacek

Faculty of Sports Studies, Masaryk University, Brno, Czech Republic

Abstract

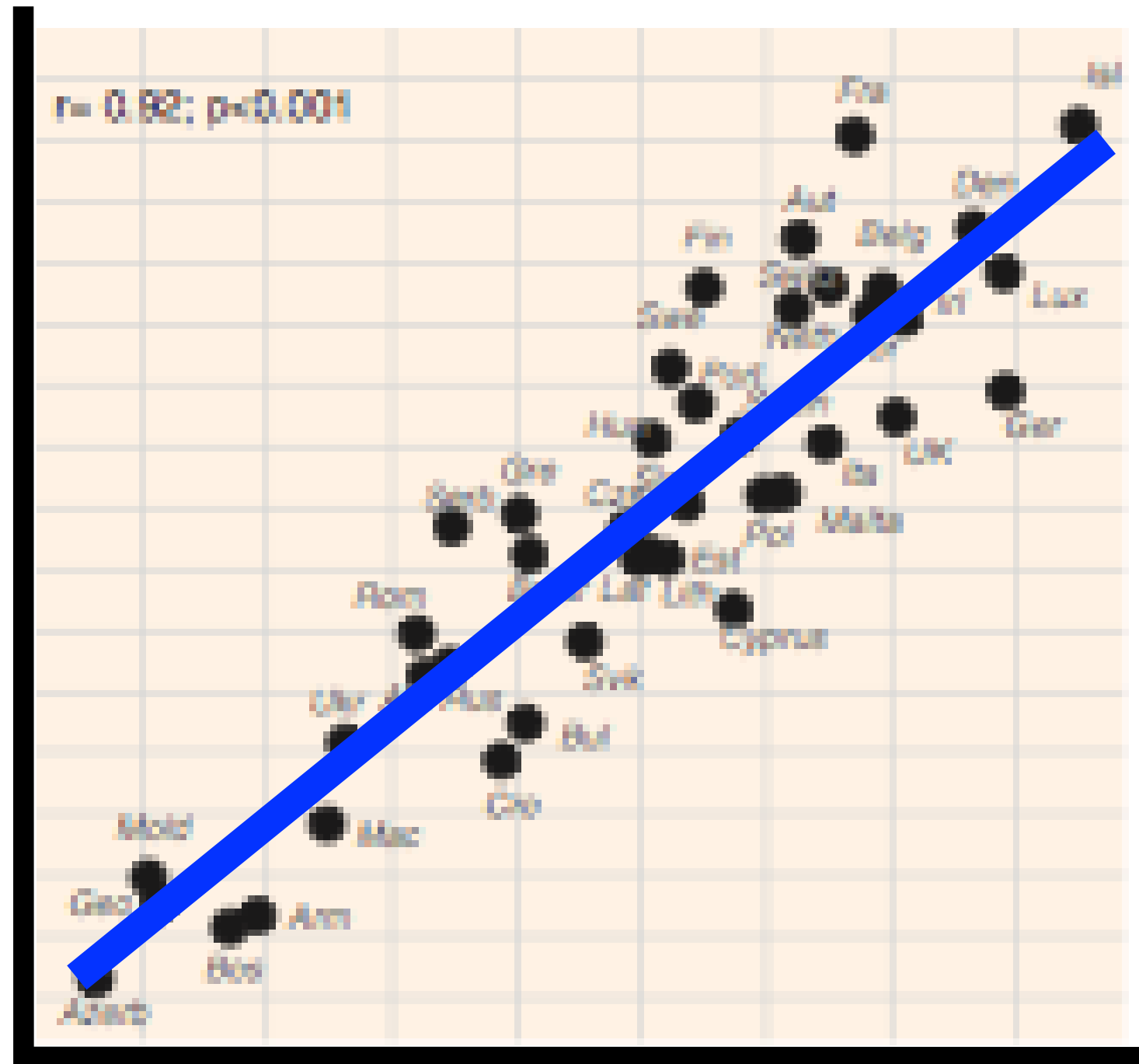
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Design: The mean consumption of 62 food items from the FAOSTAT database (1993–2008) was compared with the actual statistics of five CVD indicators in 42 European countries. Several other exogenous factors (health expenditure, smoking, body mass index) and the historical stability of results were also examined.

Results: We found exceptionally strong relationships between some of the examined factors, the highest being a correlation between raised cholesterol in men and the combined consumption of animal fat and animal protein ($r = 0.92$, $p < 0.001$). The most significant dietary correlate of low CVD risk was high total fat and animal protein consumption. Additional statistical analyses further highlighted strong positive links for dairy (cheese) and

The cholesterol story fits?

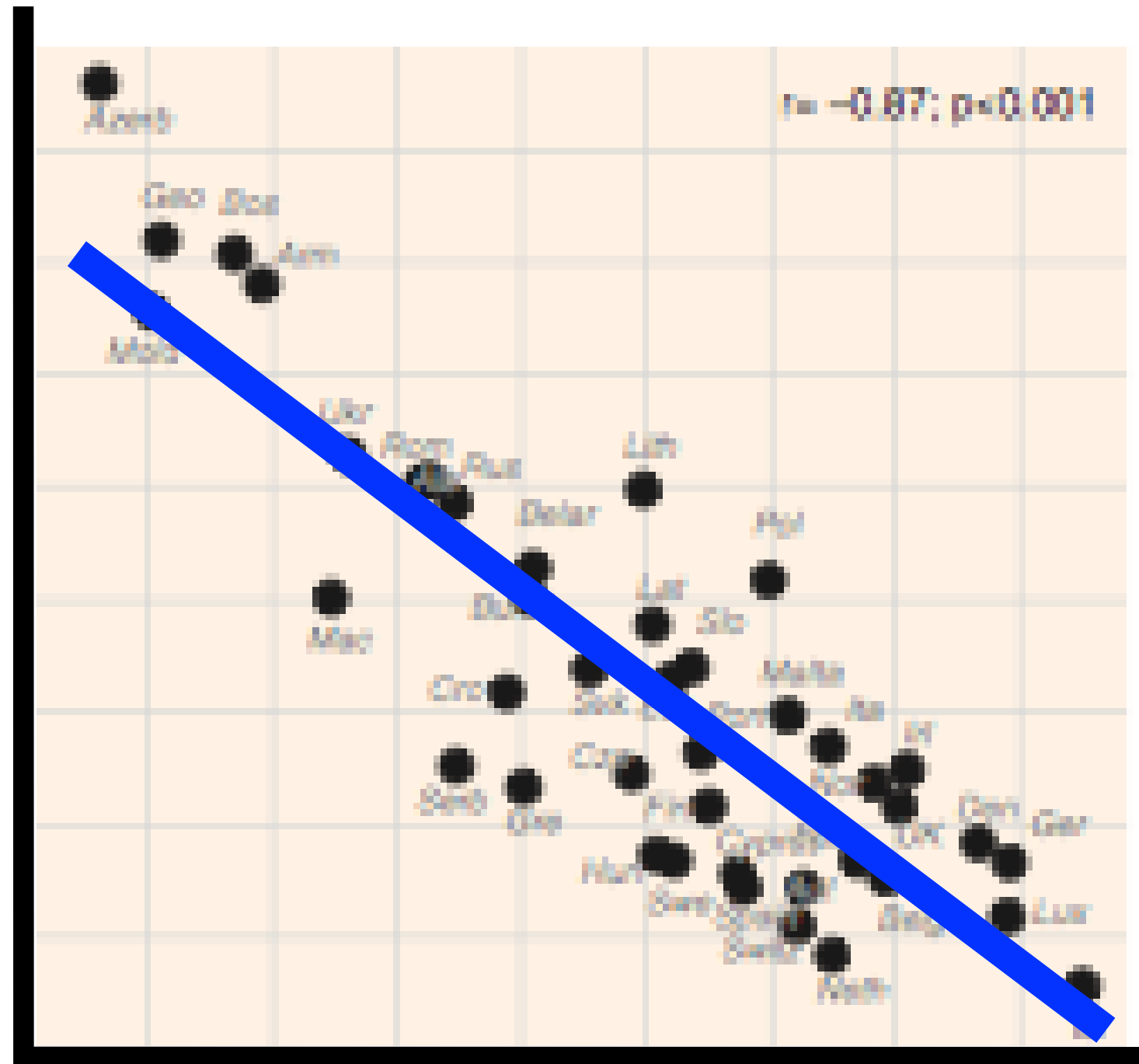
Total fat and animal
protein



Prev. of raised cholesterol

The cholesterol story fits?

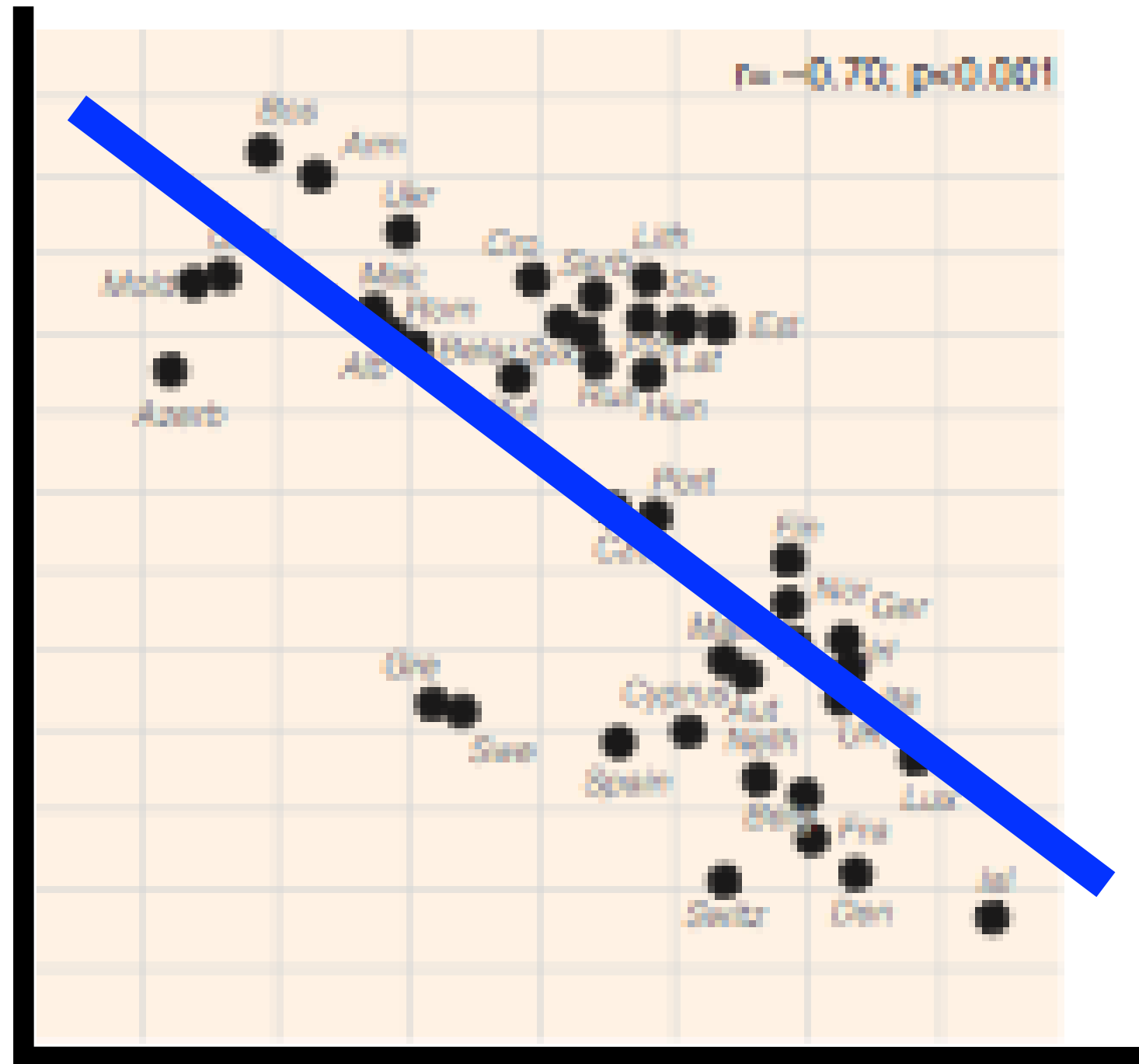
Total carbs



Prev. of raised cholesterol

The cholesterol story fits?

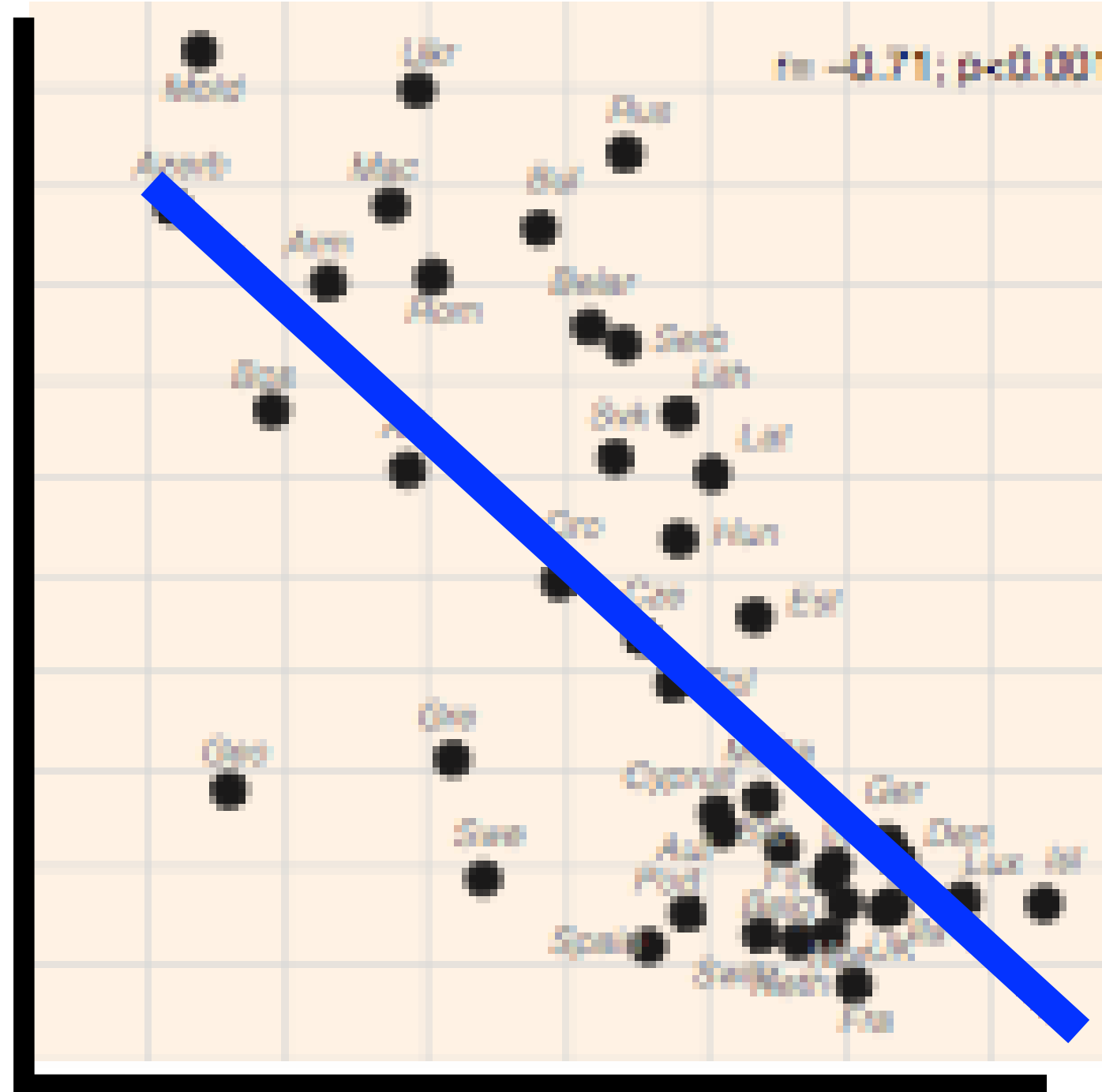
Prev. of raised BP



Prev. of raised cholesterol

The cholesterol story fits?

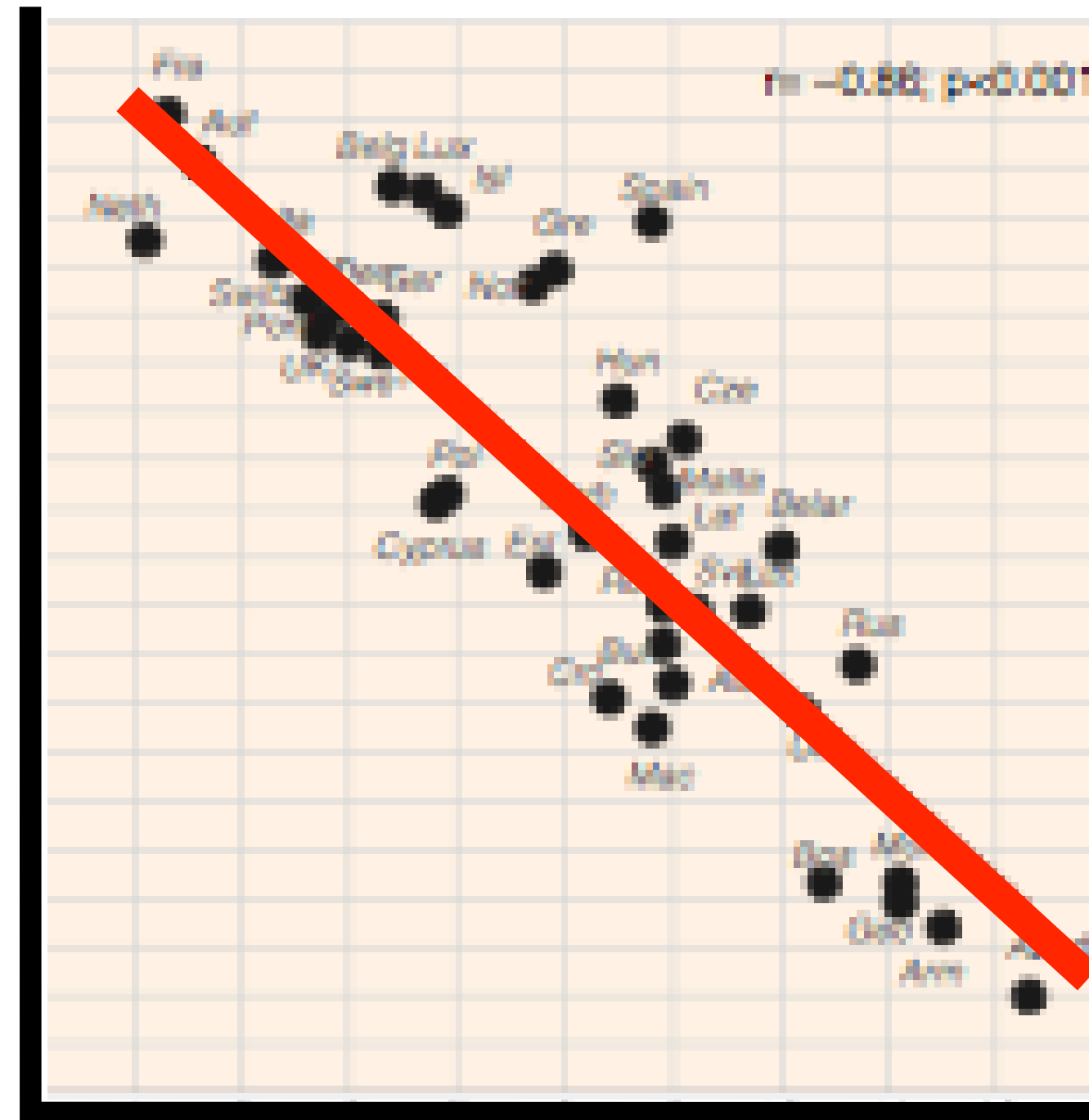
Total CVD mortality
(women)



Prev. of raised cholesterol

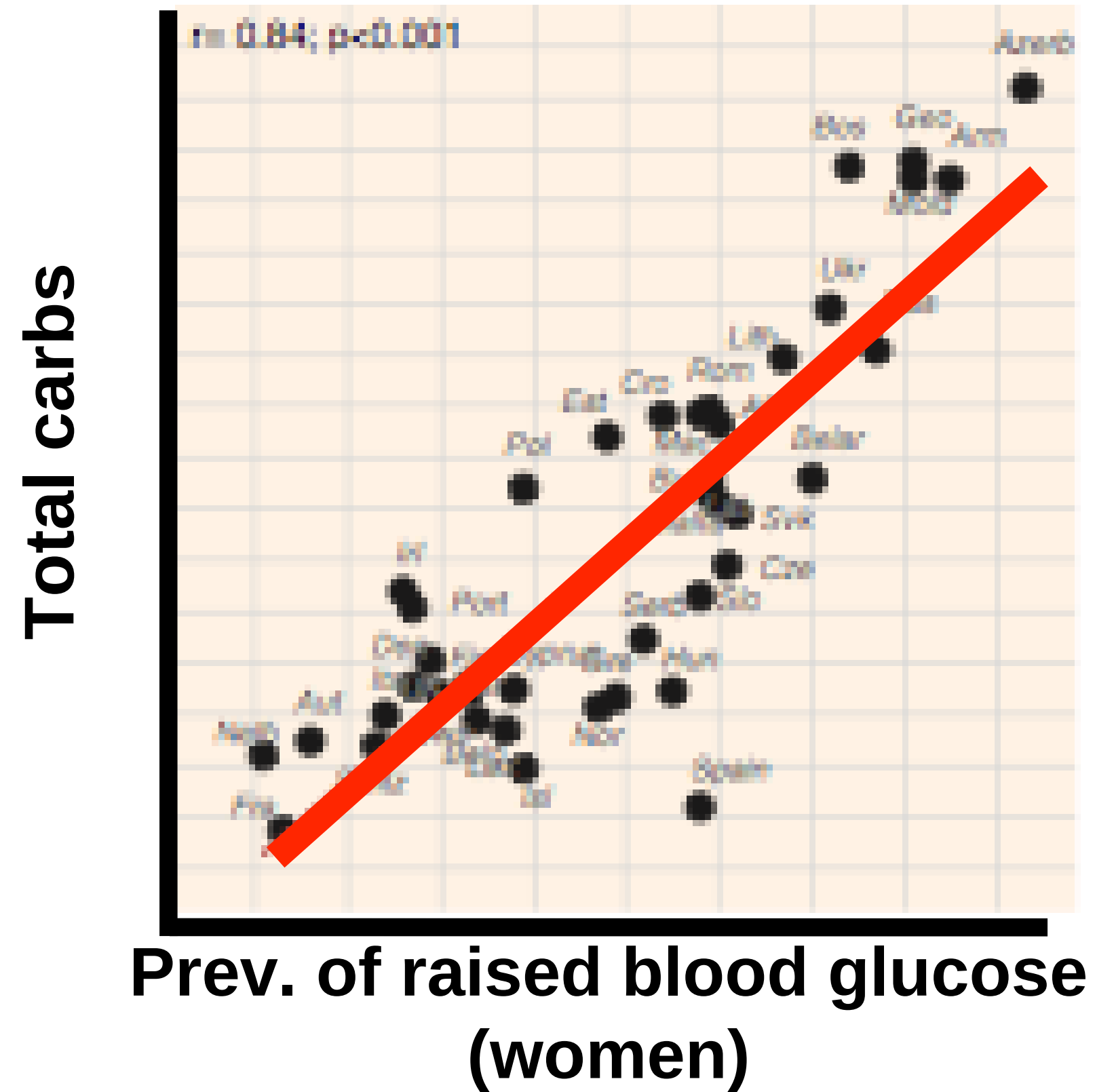
Blood sugar?

**Total fat and animal
protein**



Prev. of raised blood glucose

Blood sugar?



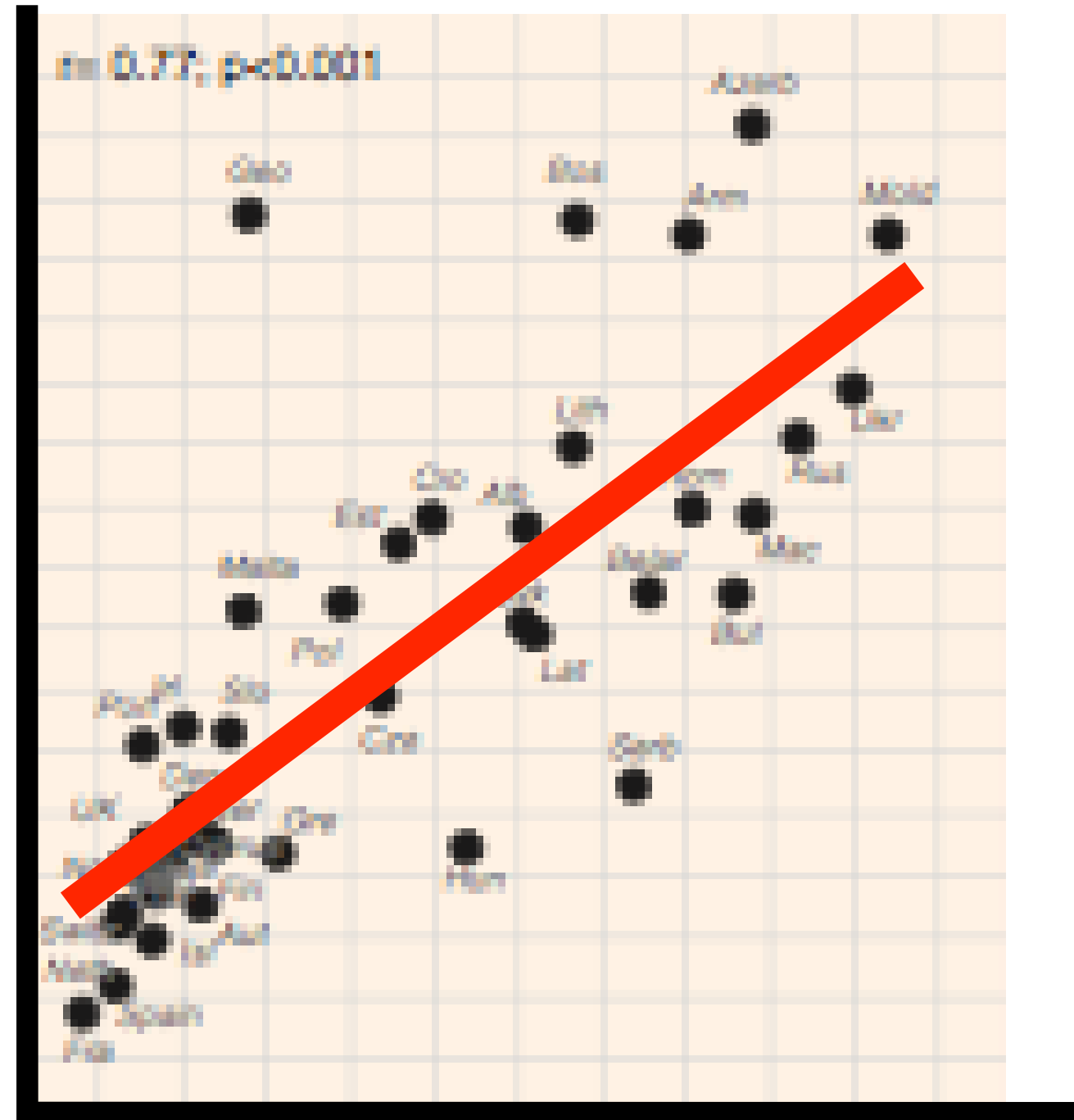
Blood sugar?

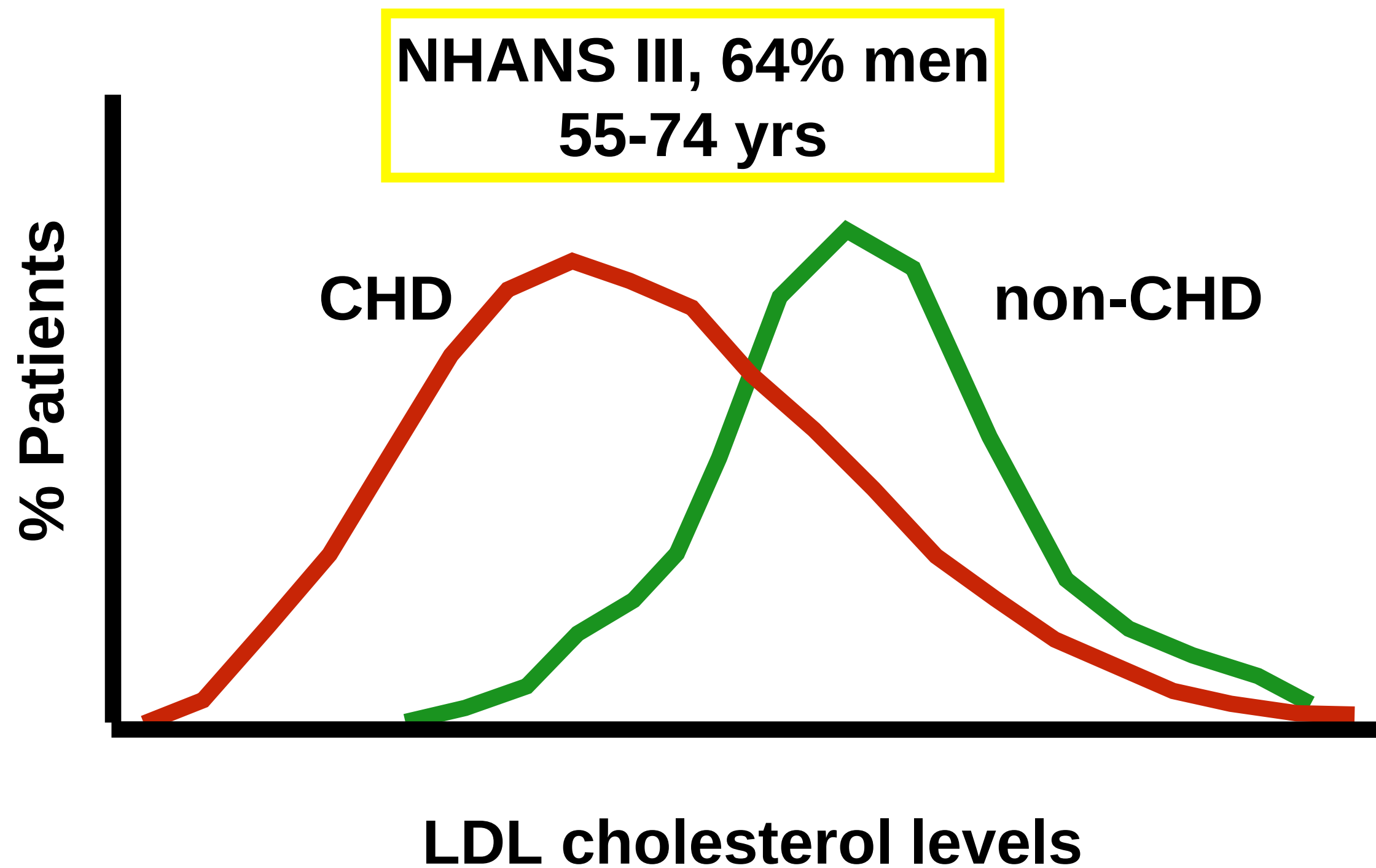


Prev. of raised blood pressure (women)

Blood sugar?

Total carbs





Malmo diet and cancer cohort

Sweden

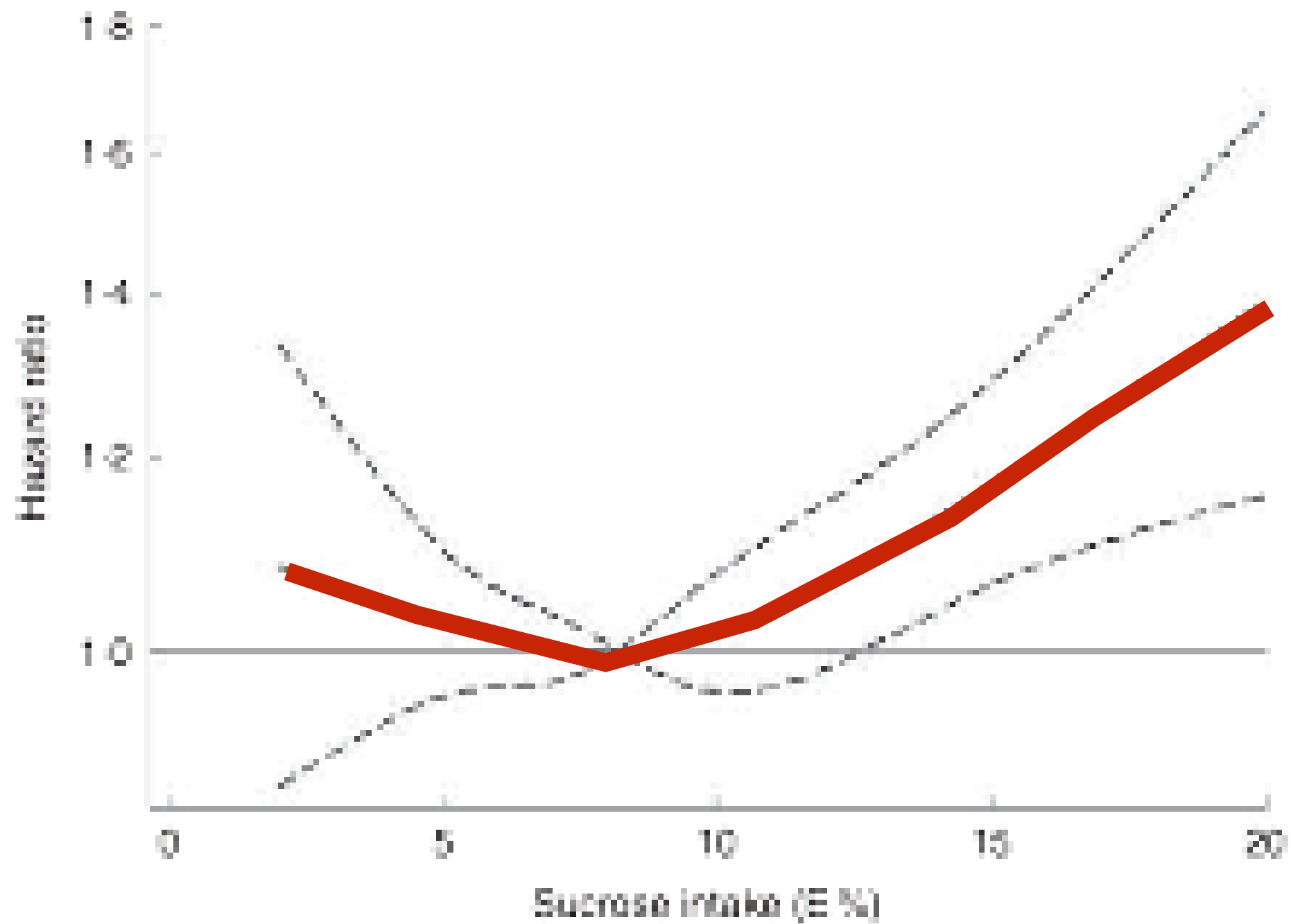
N=26 190

No diabetes orCVD

17 years follow up
457 131 person yrs

2493 incident
coronary events

Malmo diet and cancer cohort



4.. Here's the more sophisticated understanding of glucose, insulin, and metabolic disease



The NEW ENGLAND JOURNAL of MEDICINE

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ORIGINAL ARTICLE

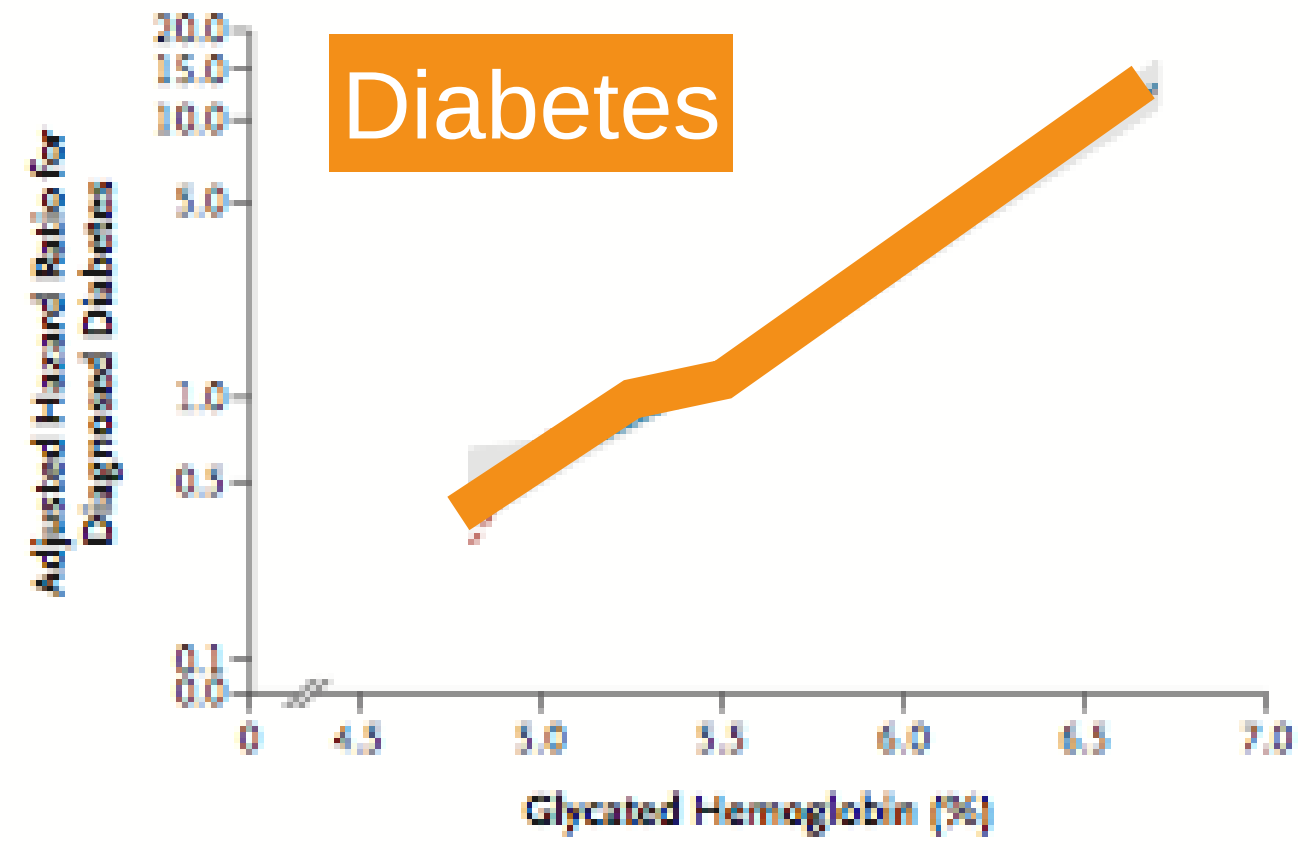
Glycated Hemoglobin, Diabetes, and Cardiovascular Risk in Nondiabetic Adults

Elizabeth Selvin, Ph.D., M.P.H., Michael W. Steffes, M.D., Ph.D., Hong Zhu, B.S., Kunihiro Matsushita, M.D., Ph.D., Lynne Wagenknecht, Dr.P.H., James Pankow, Ph.D., M.P.H., Josef Coresh, M.D., Ph.D., and Frederick L. Brancati, M.D., M.H.S.
N Engl J Med 2010; 362:800-811 | [March 4, 2010](#) | DOI: 10.1056/NEJMoa0908359

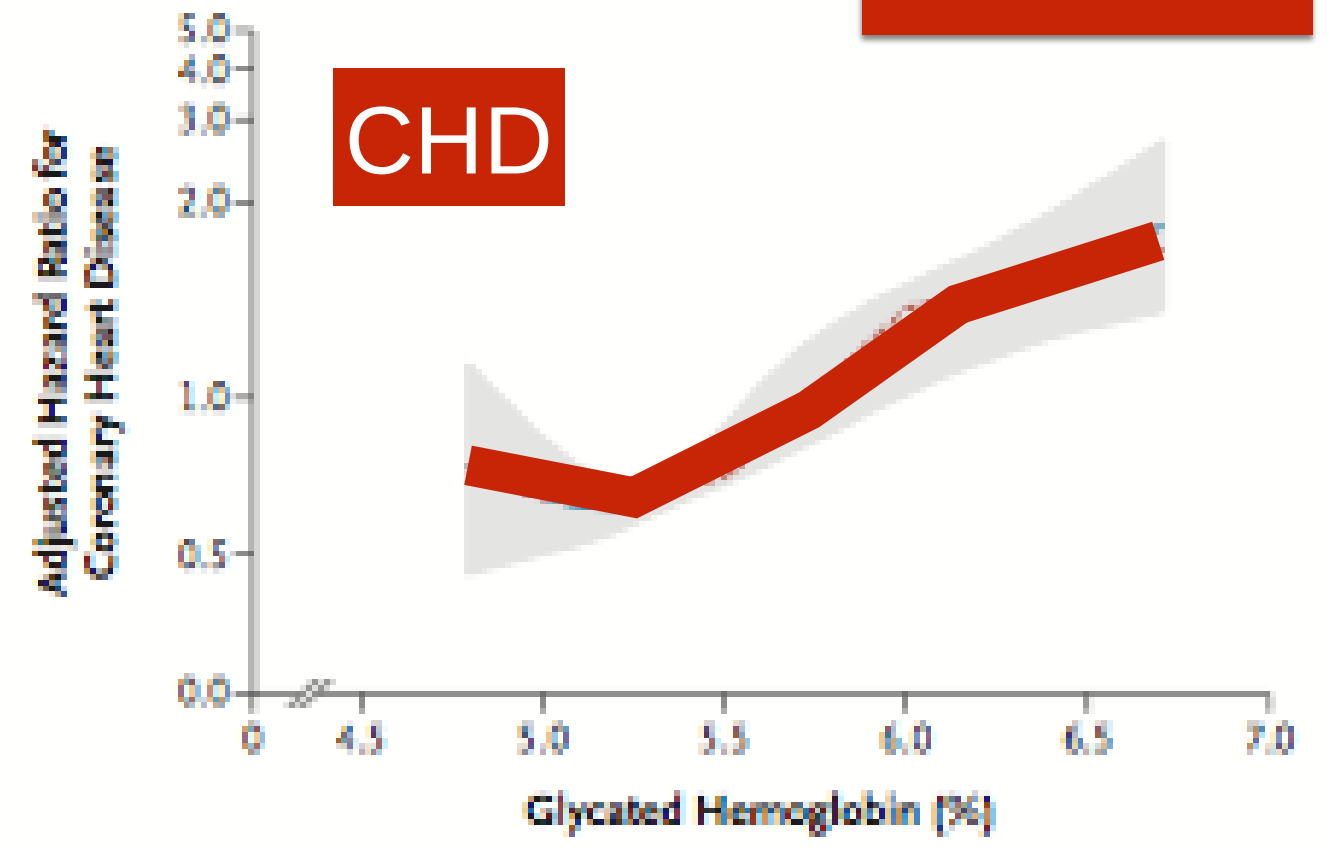
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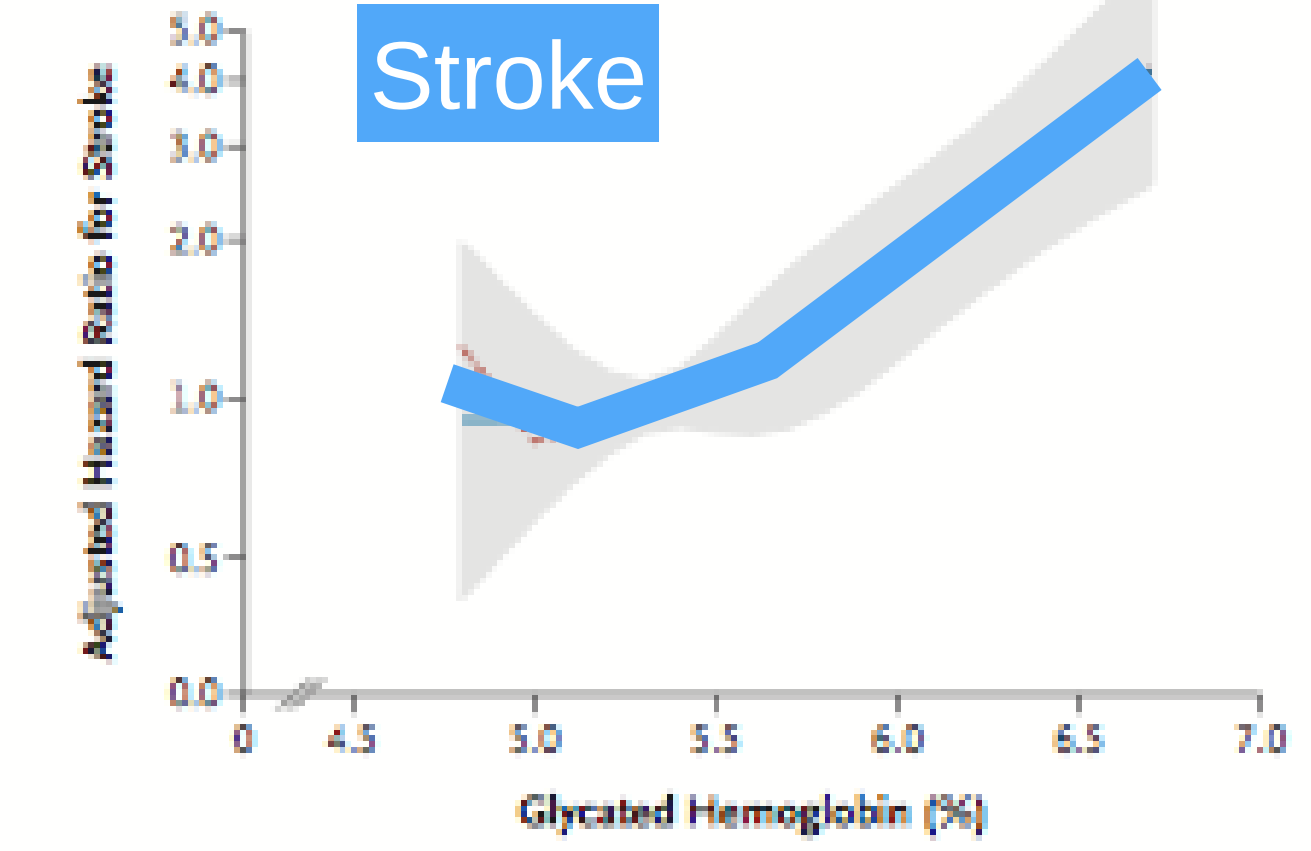
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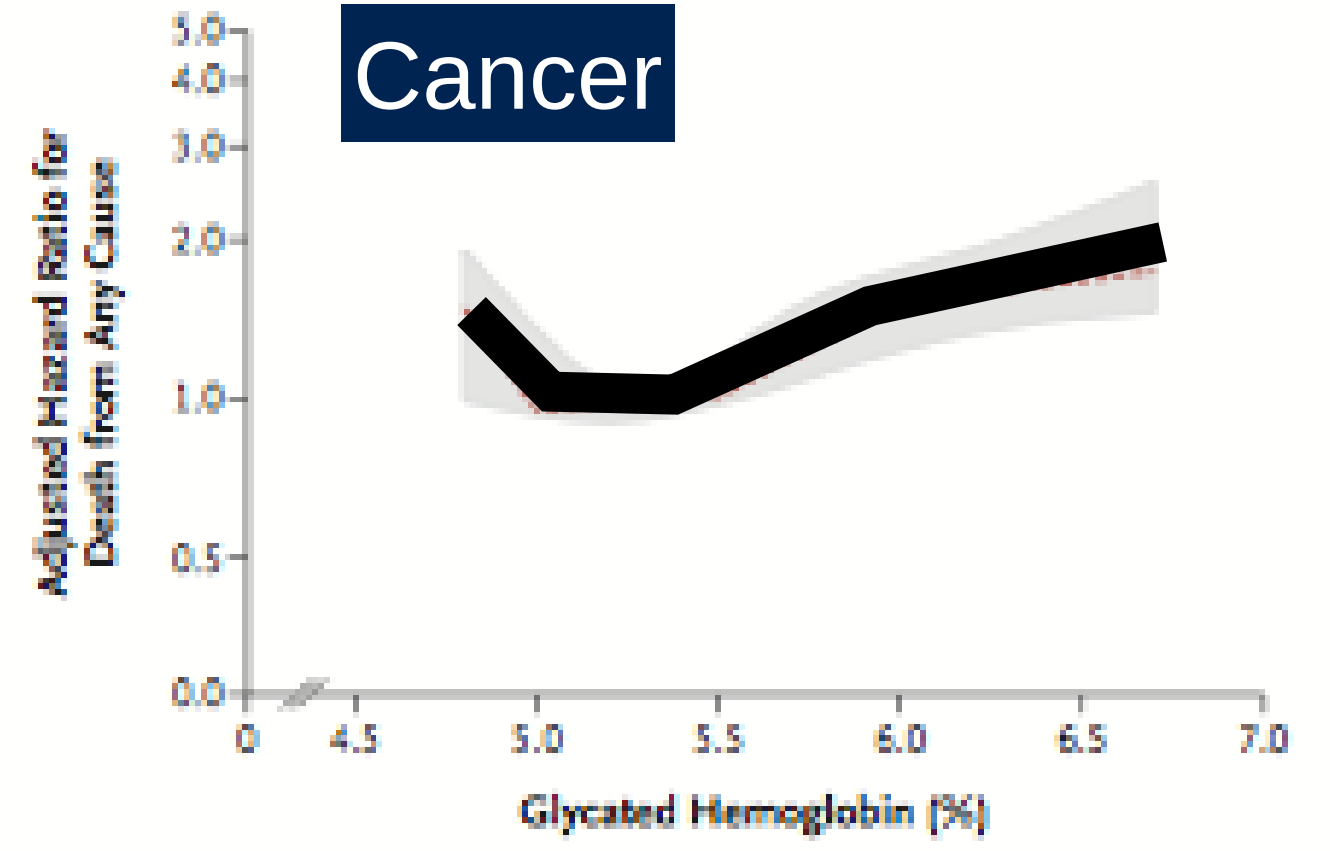
B



C



D





Hemoglobin A_{1c} in Nondiabetic Patients: An Independent Predictor of Coronary Artery Disease and Its Severity

Naveen Garg, DM; Nagaraja Moorthy, DM; Aditya Kapoor, DM;
Satyendra Tewari, DM; Sudeep Kumar, DM; Archana Sinha, MSc;
Anshika Shrivastava, MSc; and Pravin K. Goel, DM

1141 angiography Non-diabetic

HbA1C

CVD presence
adjusted odds

5
4
3
2
1
0

<5.5%

<5.7%

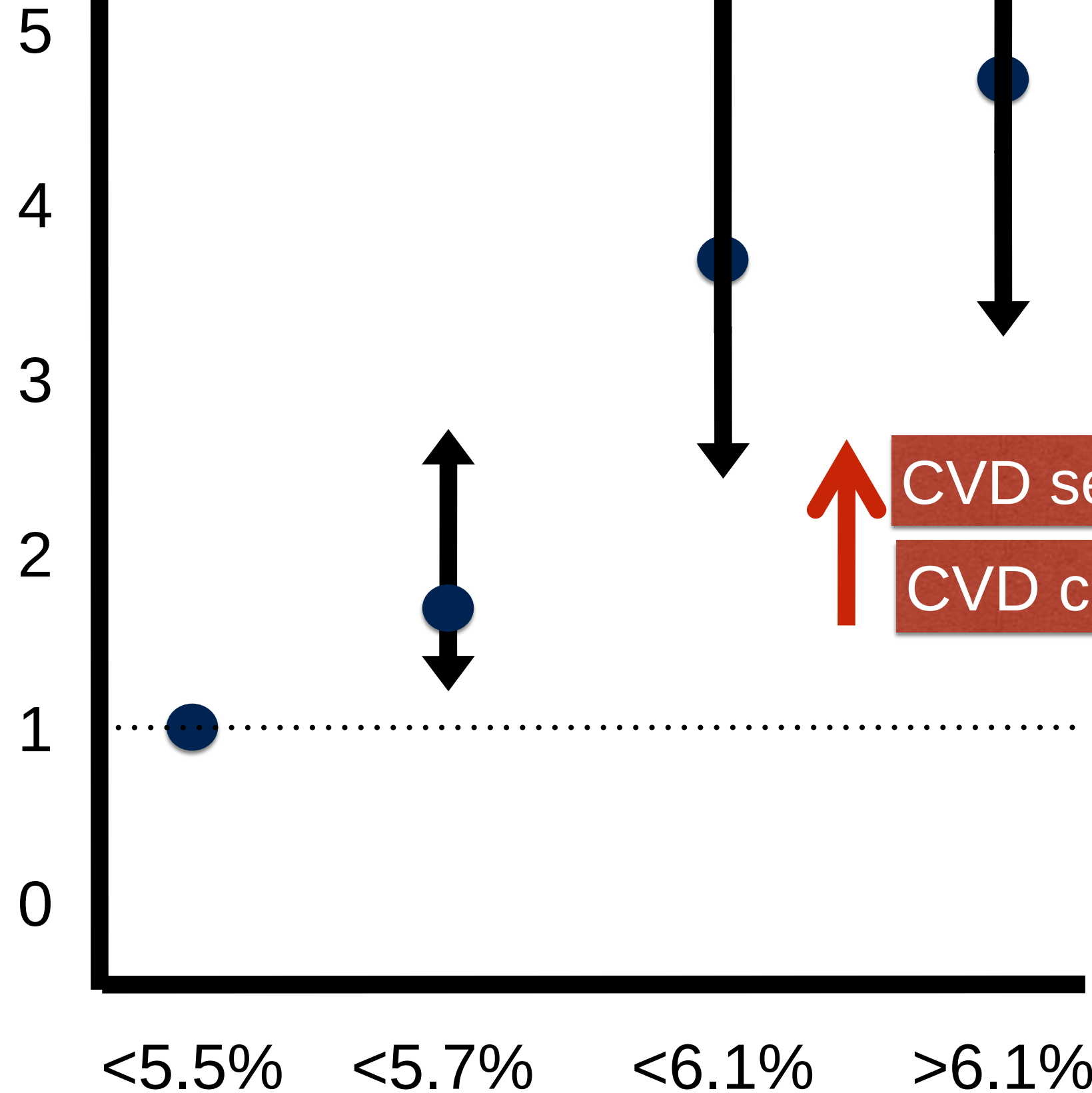
<6.1%

>6.1%

HbA1C quartile

CVD severity

CVD complexity



C-peptide as a risk factor of coronary artery disease in the general population

Antonio Cabrera de León^{1,2,3}, José Gregorio Oliva García⁴, Itahisa Marcelino Rodríguez^{1,3}, Delia Almeida González^{3,5}, José Juan Alemán Sánchez^{1,3}, Buenaventura Brito Díaz^{1,3}, Santiago Domínguez Coello^{1,3}, Vicente Bertomeu Martínez^{3,6}, Armando Aguirre Jaime¹ and María del Cristo Rodríguez Pérez^{1,3}

Abstract

Diabetes & Vascular Disease Research
1–9

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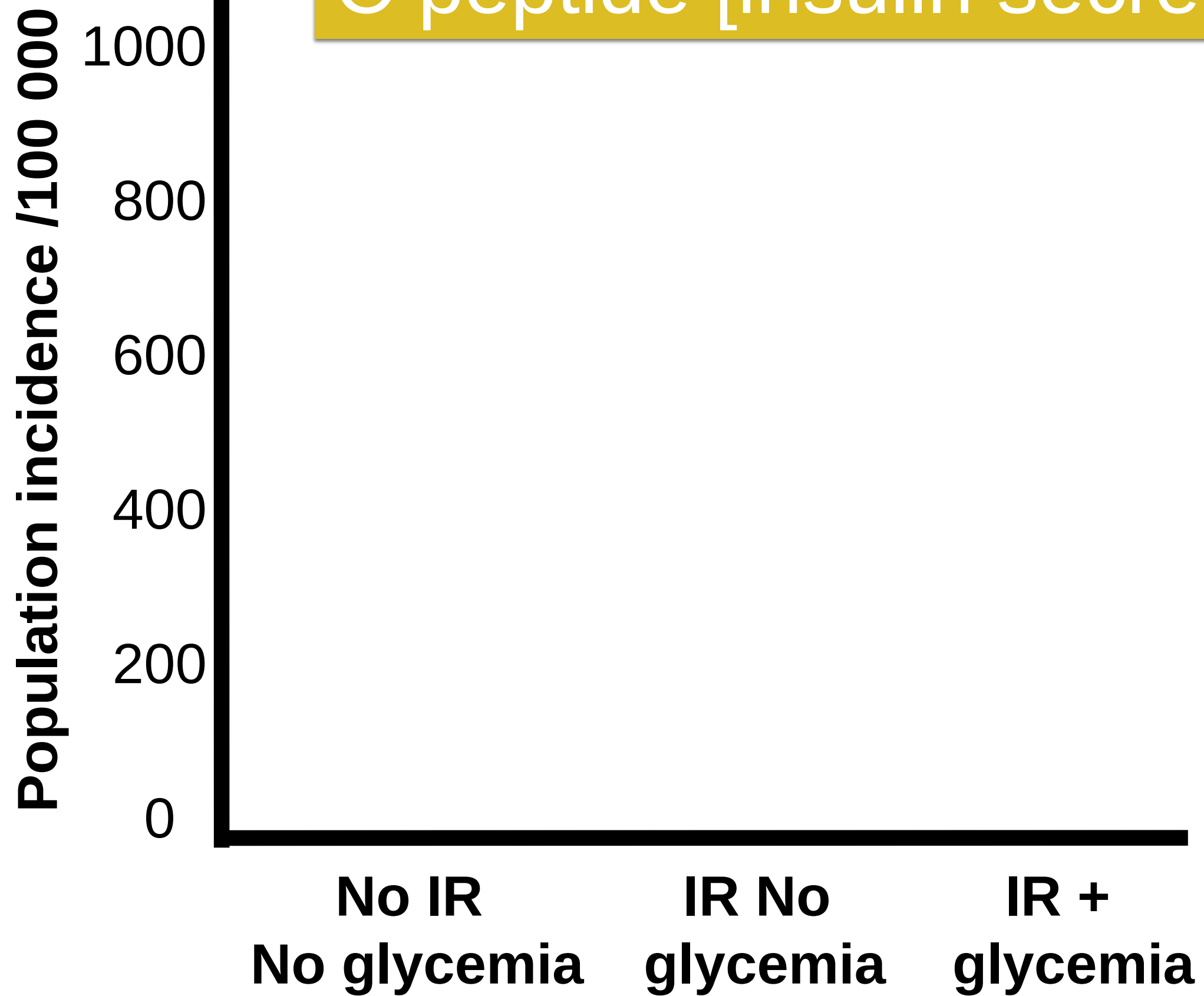
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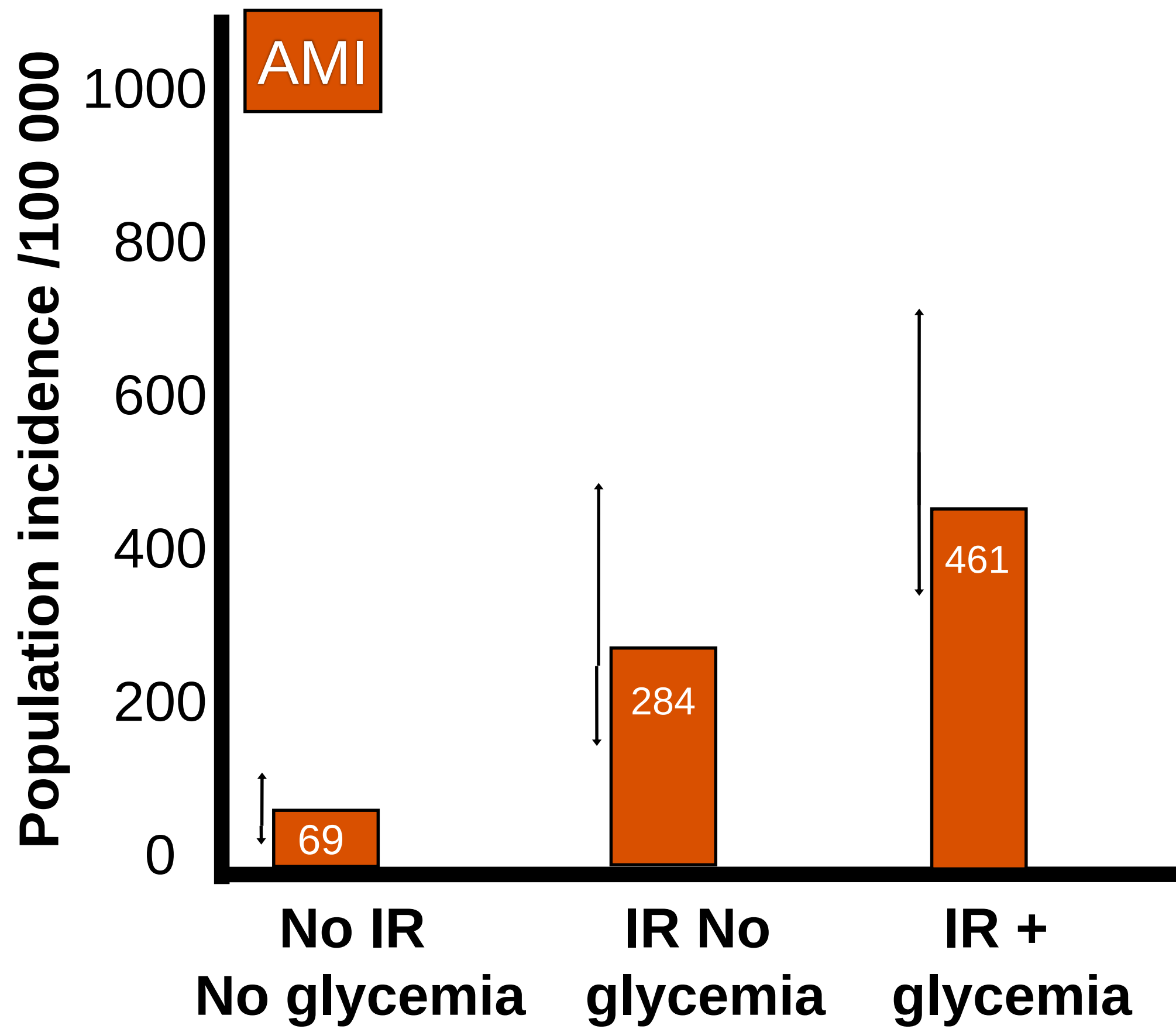
DOI: 10.1177/1479164114564900

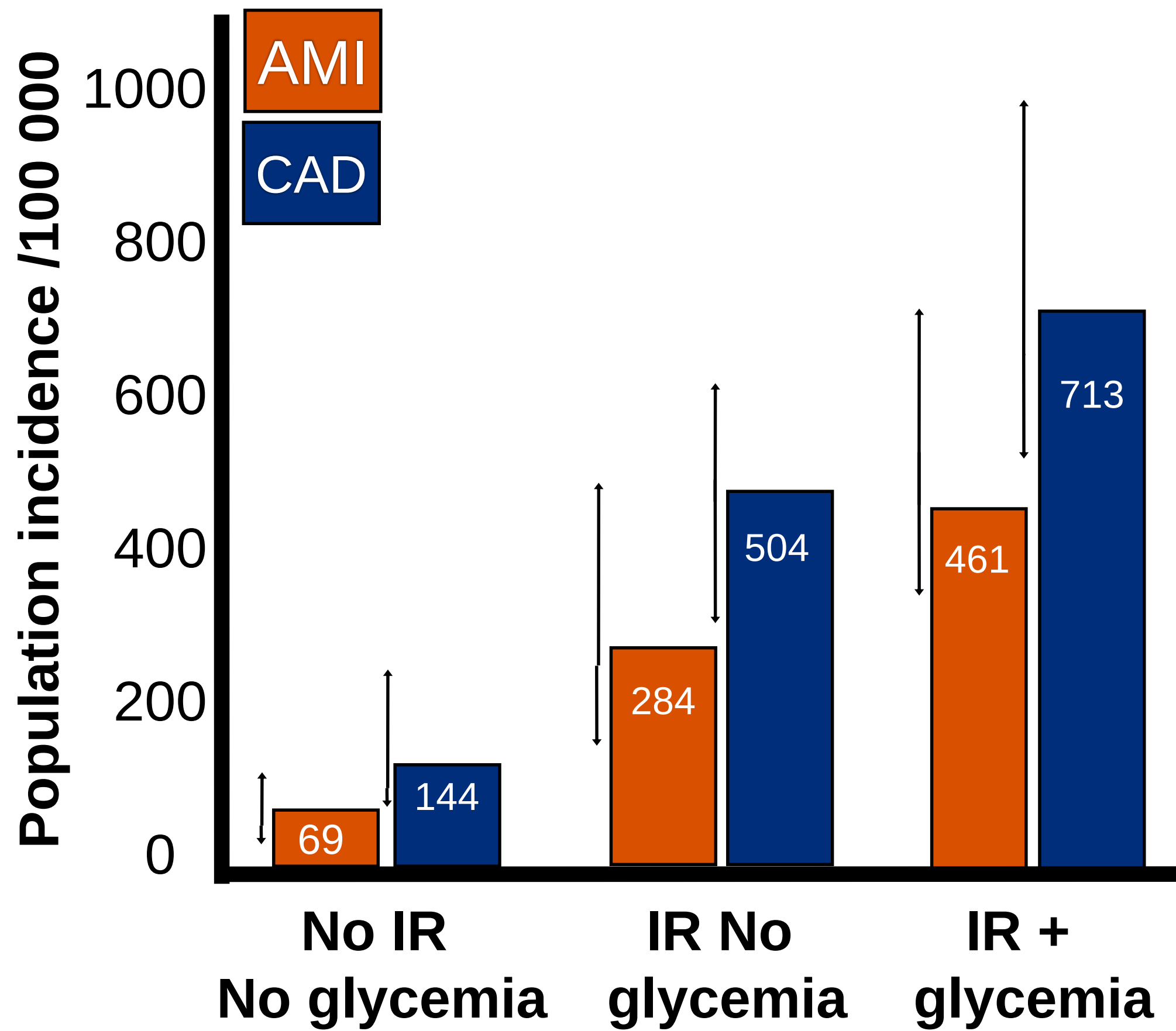
dvr.sagepub.com



C peptide [insulin secretion]









4 grams glucose
Total amount of blood sugar
in a normal adult
(83 mg/dL)

240 grams = 57 teaspoons sugar
ADA daily recommendations
for diabetics.



Inflammation
Insulin
resistance

Stress

Poor sleep

Too much exercise

Low physical activity

Genes/ethnicity
Age?

Obesity

High insulin

High trans fat

Smoking

High sugar die High alcohol
diet

Pollution

environmental toxins

Too much/too little sun

High iron

Poor gut
microbes

High insulin

NEUROPSYCHIATRIC

Alzheimers / other dementias
Peripheral neuropathy
Retinopathy
Neuro-psychiatric disorders
Parkinson's disease
Autism

GASTROINTESTINAL

Diabetes: Type 2 / Gestational
Hypertriglyceridaemia
Non-alcoholic fatty liver disease
Ulcerative colitis

ENDOCRINE

Chronic inflammation
Fatty liver
Obesity
PCOS

CANCER

Breast, ovarian colon bladder,
pancreas, liver, prostate

UROLOGY Nephropathy, erectile dysfunction

Tinnitus
Vertigo
Meniere's disease

Periodontal disease

CIRCULATORY

Atherosclerosis
Cardiomyopathy
Endothelial dysfunction
(microvascular disease and
peripheral vascular disease)
Stroke
Thrombosis (DVT)
Hypertension

SKELETAL

Osteoporosis

INFLAMMATION

Osteoarthritis
Rheumatoid arthritis



5. Saturated fat is still seen as a major problem, but is it warranted?

Lipids (2008) 43:65–77

DOI 10.1007/s11745-007-3132-7

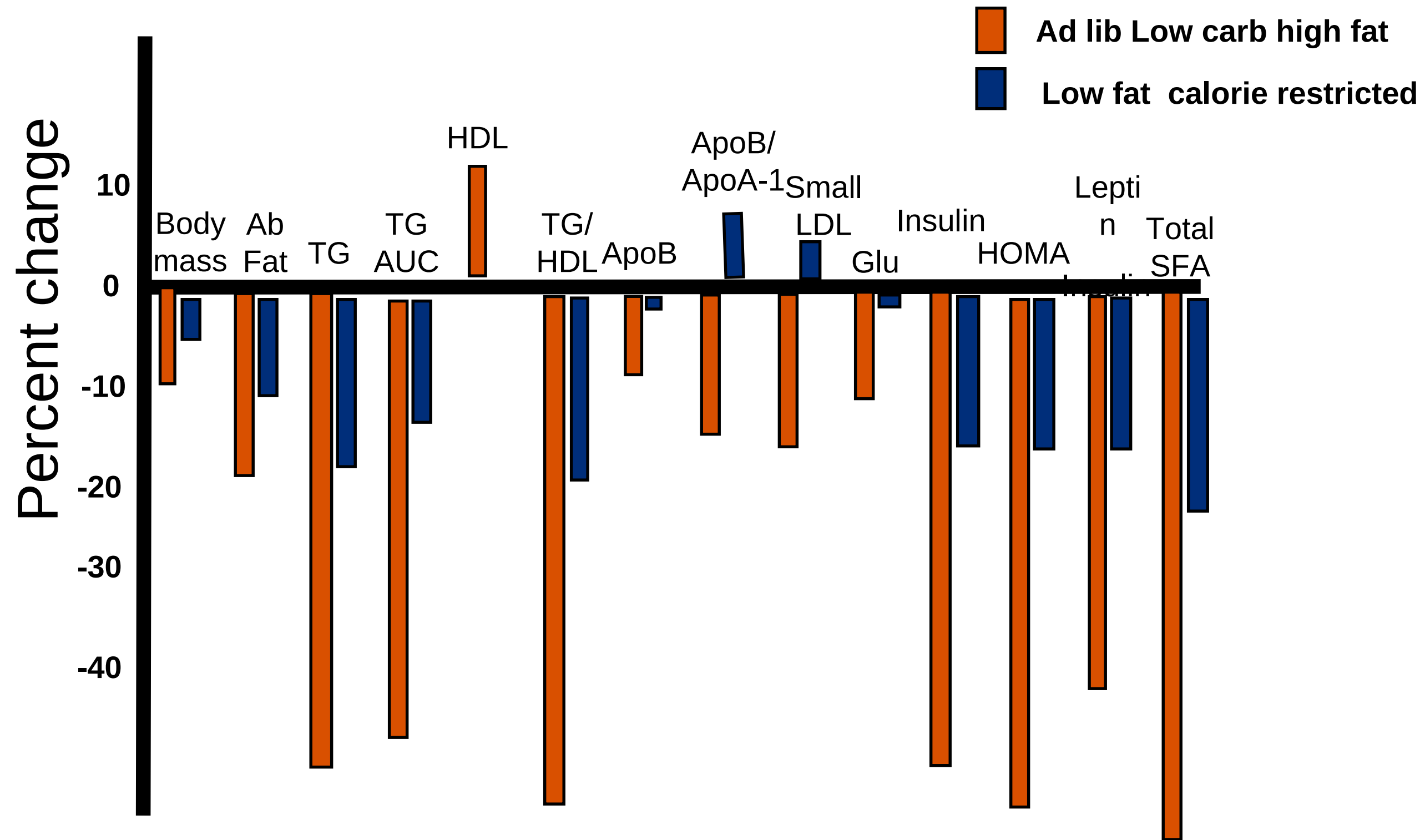
ARTICLE

Comparison of Low Fat and Low Carbohydrate Diets on Circulating Fatty Acid Composition and Markers of Inflammation

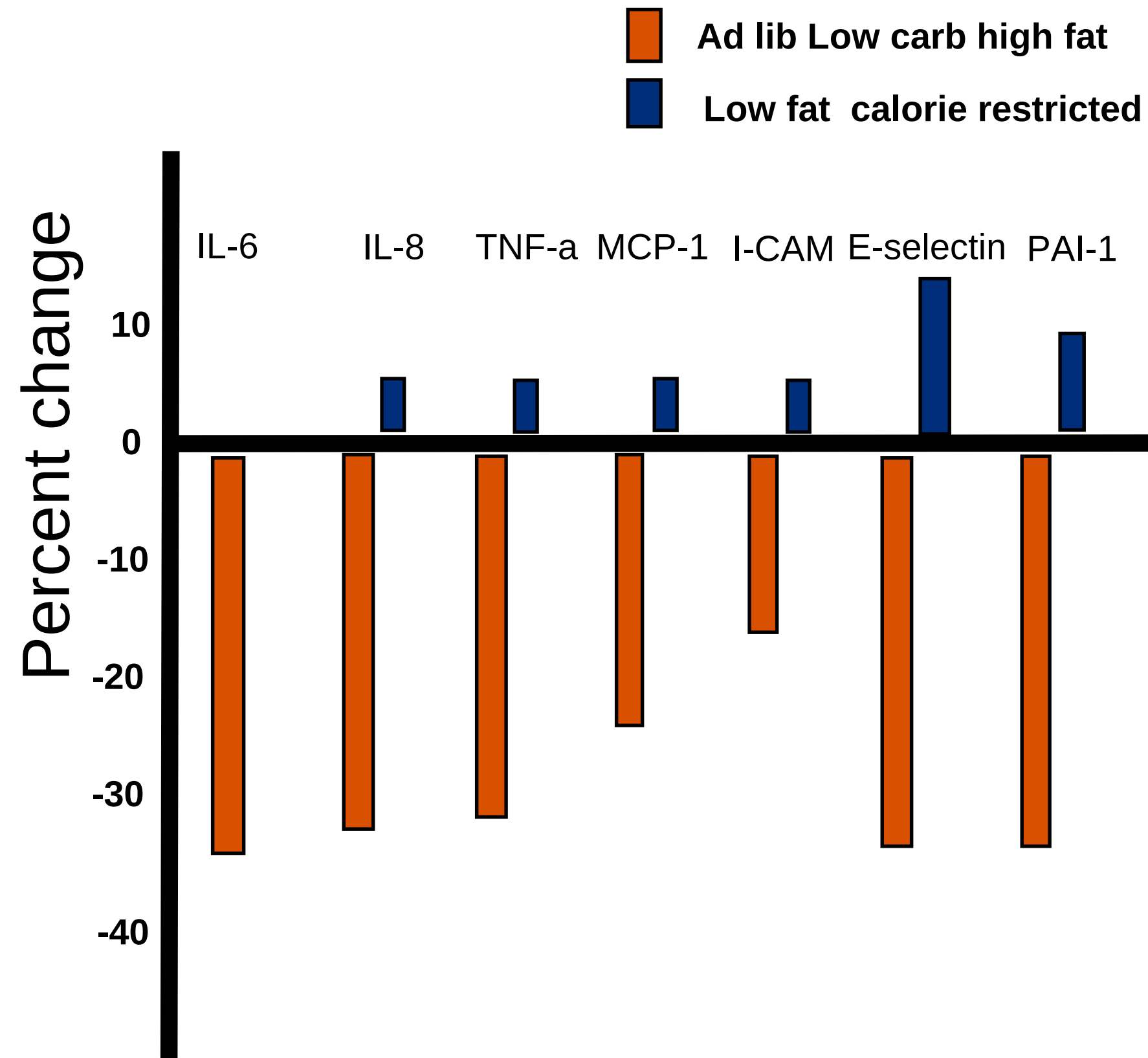
**Cassandra E. Forsythe · Stephen D. Phinney · Maria Luz Fernandez ·
Erin E. Quann · Richard J. Wood · Doug M. Bibus · William J. Kraemer ·
Richard D. Feinman · Jeff S. Volek**

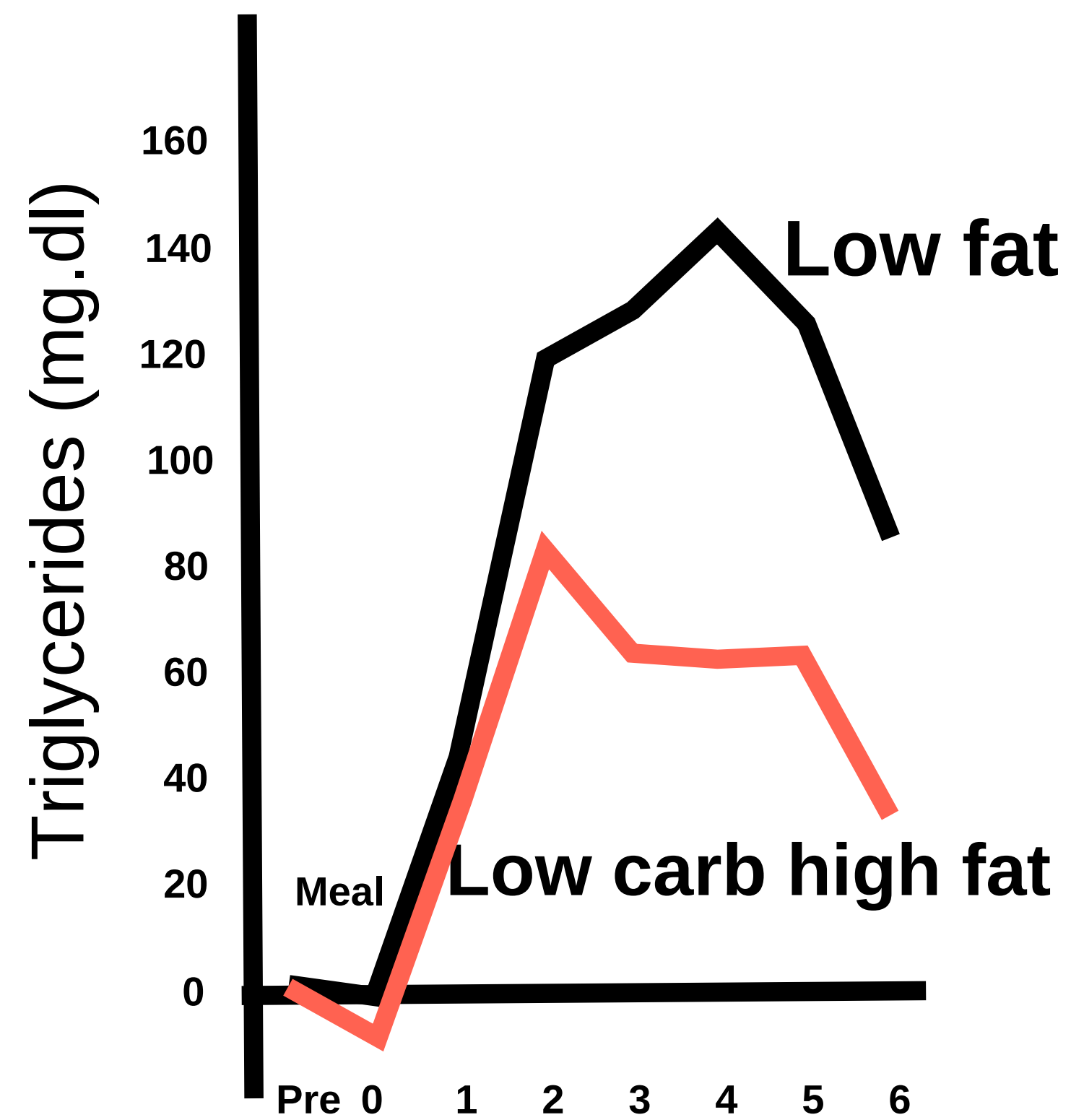
Received: 5 July 2007 / Revised: 24 October 2007 / Accepted: 25 October 2007 / Published online: 29 November 2007
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Eat more fat?

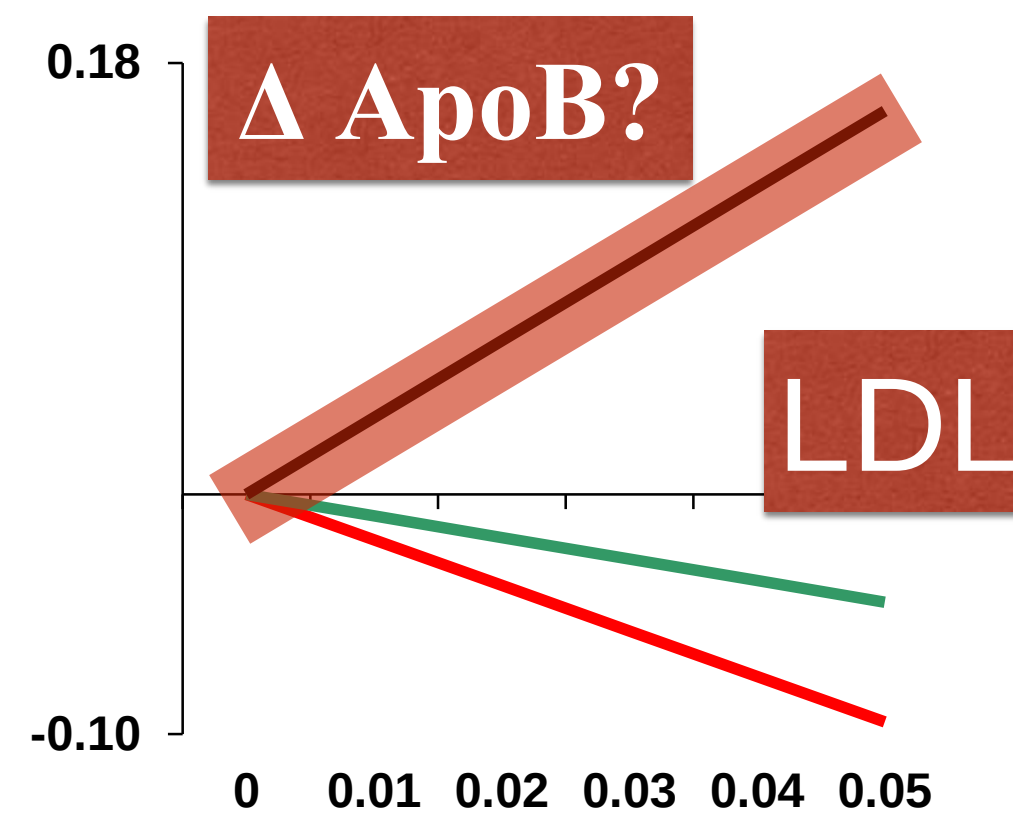
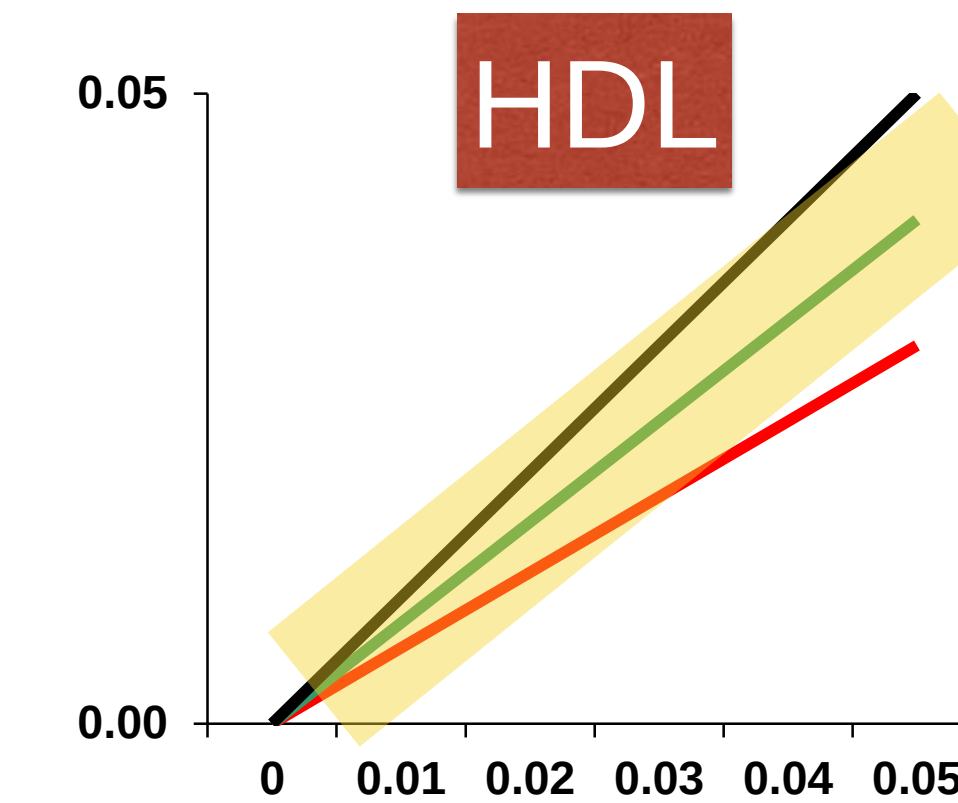
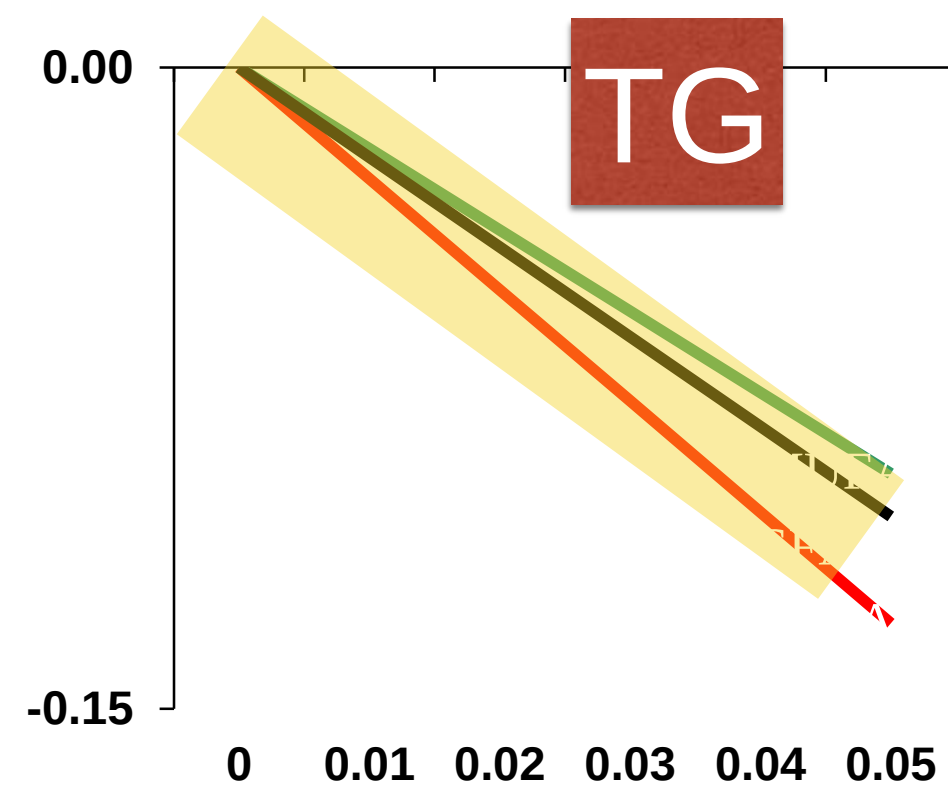


Eat more fat?





ApoB



Q J Med 2006; **99**:1–14
doi:10.1093/qjmed/hci154

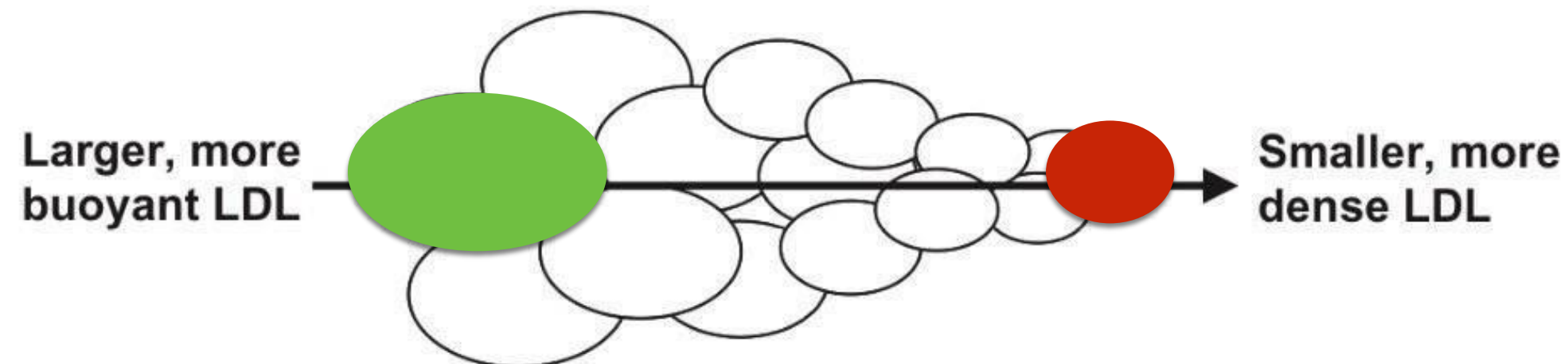
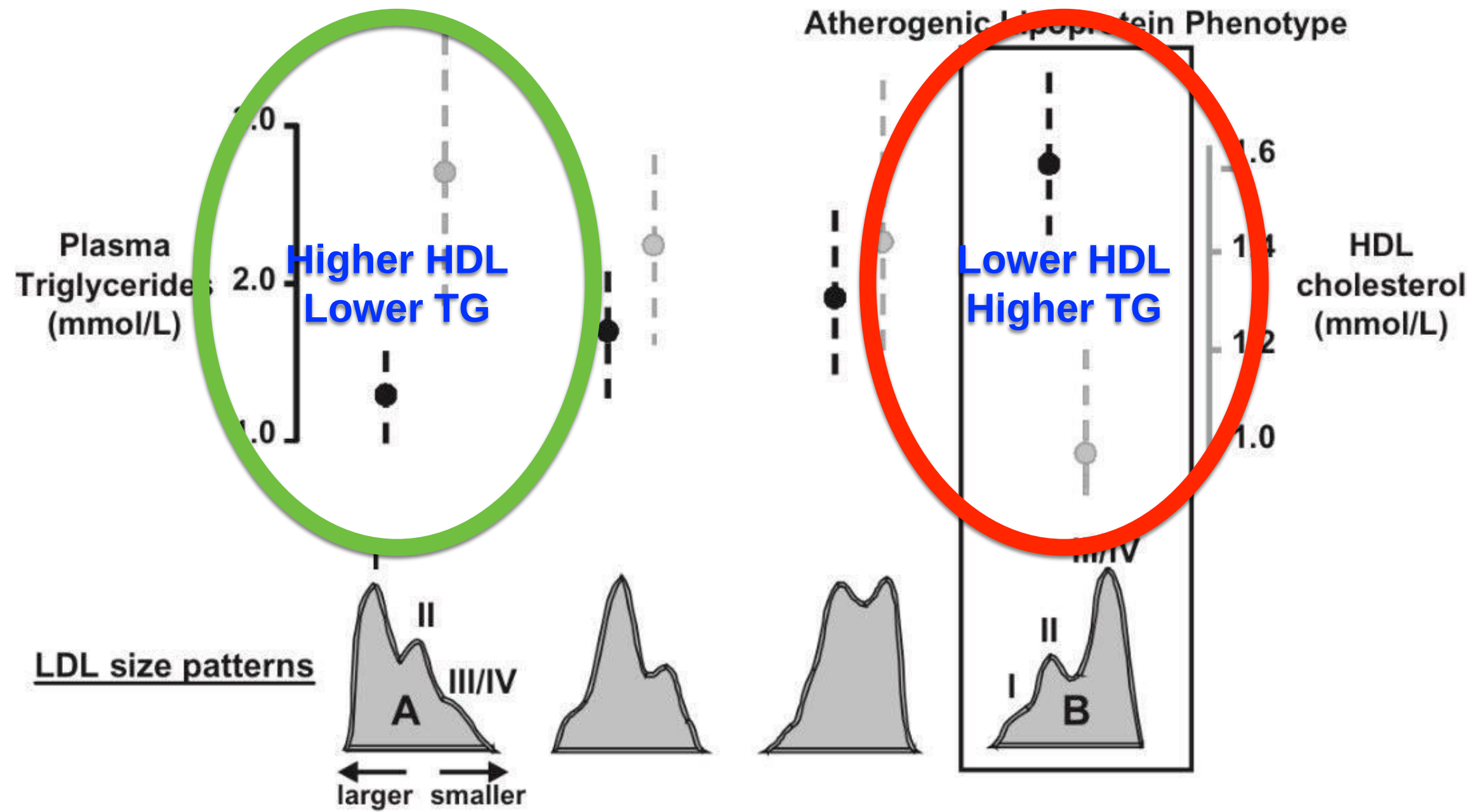
Review

QJM

Low-density lipoprotein size and cardiovascular risk assessment

M. RIZZO¹ and K. BERNEIS²

From the ¹Department of Clinical Medicine and Emerging Diseases, University of Palermo, Italy, and ²Department of Endocrinology and Diabetology, University Hospital Zurich, Switzerland



Meta-analysis

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

Hooper L, Summerbell CD, Thompson R, Sills D, Roberts FG, Moore H, Davey Smith G



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Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies^{1,2,3}

Marianne U Jakobsen, Eilis J O'Reilly, Berit L Heitmann, Mark A Pereira, Katarina Bälter, Gary E Fraser, Uri Goldbourt, Göran Hallmans, Paul Knekt, Simin Liu, Pirjo Pietinen, Donna Spiegelman, June Stevens, Jarmo Virtamo, Walter C Willett, and Alberto Ascherio

This Article

First published February 11, 2009, doi: 10.1093/ajcn.2008.27124

Am J Clin Nutr May 2009, vol. 89 no. 5 1425–1435

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89/5/1425 mos

Meta-analysis

Hooper Cocrane
review RCTs

All cause
mortality

CVD
mortality

CV events
combined

Fat intake



RR 0.86
(0.77-0.96)
Mod evid



Modified fat
intake



Reduced fat
intake



Jackobsen
Prospective

Saturated fat
intake



Substitutions



Cochrane review meta analysis: Combined CV events

Intervention

N=21791

Cases=1774

81.4/1000

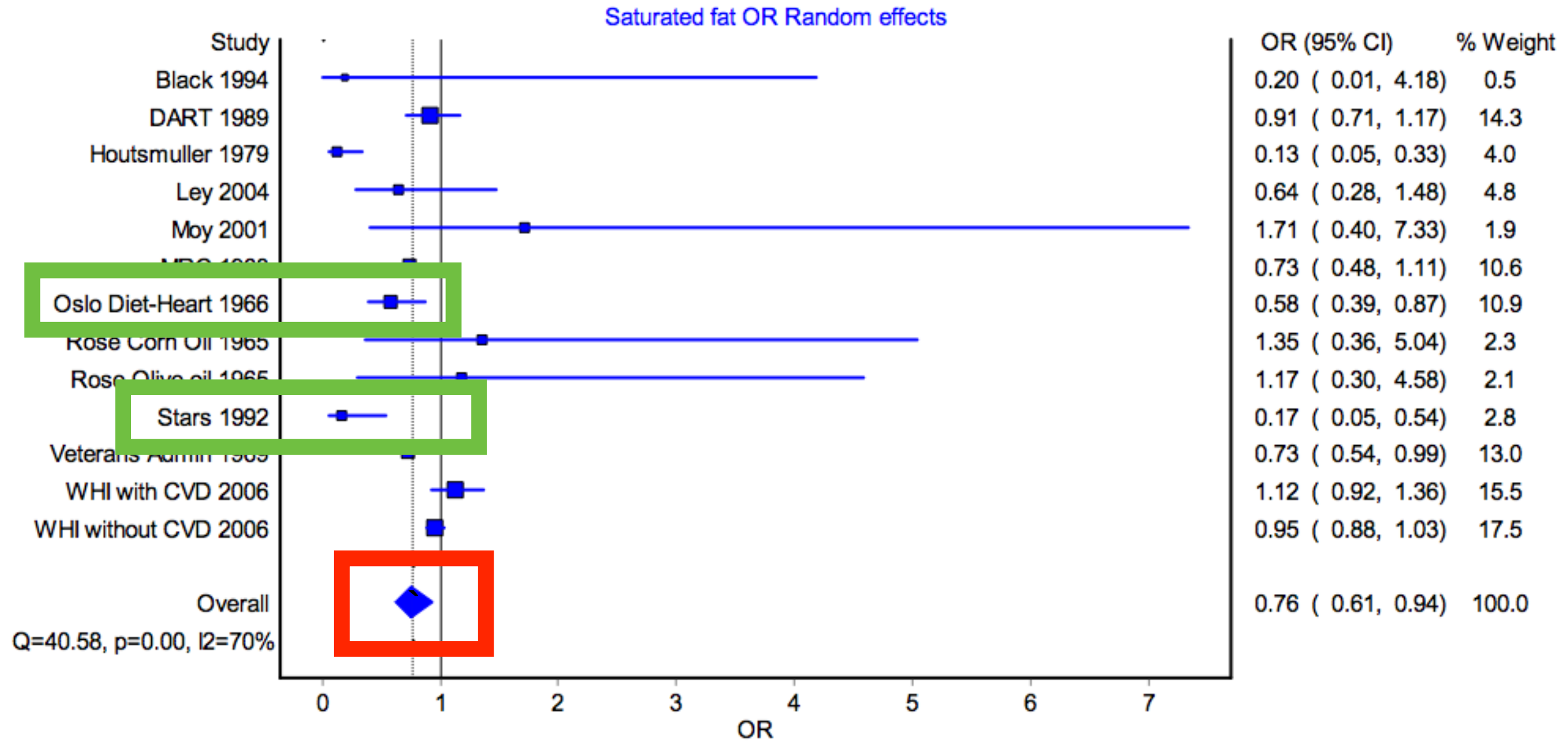
Control

N=31509

Cases=2603

82.6/1000

Meta-analysis



PLoS One. 2013; 8(7): e69930.

Published online 2013 Jul 26. doi: [10.1371/journal.pone.0069930](https://doi.org/10.1371/journal.pone.0069930)

A Re-Analysis of the Cochrane Library Data: The Dangers of Unobserved Heterogeneity in Meta-Analyses

[Evangelos Kontopantelis](#)^{1,2,3,*} [David A. Springate](#)^{1,2} and [David Reeves](#)^{1,2}

Tim Friede, Editor

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Abstract

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Background

Heterogeneity has a key role in meta-analysis methods and can greatly affect conclusions. However, true levels of heterogeneity are unknown and often researchers assume homogeneity. We aim to: a) investigate the prevalence of unobserved heterogeneity and the validity of the assumption of homogeneity; b) assess the performance of various meta-analysis methods; c) apply the findings to published meta-analyses.

Methods and Findings

We accessed 57,397 meta-analyses, available in the Cochrane Library in August 2012. Using simulated data we assessed the performance of various meta-analysis methods in different scenarios

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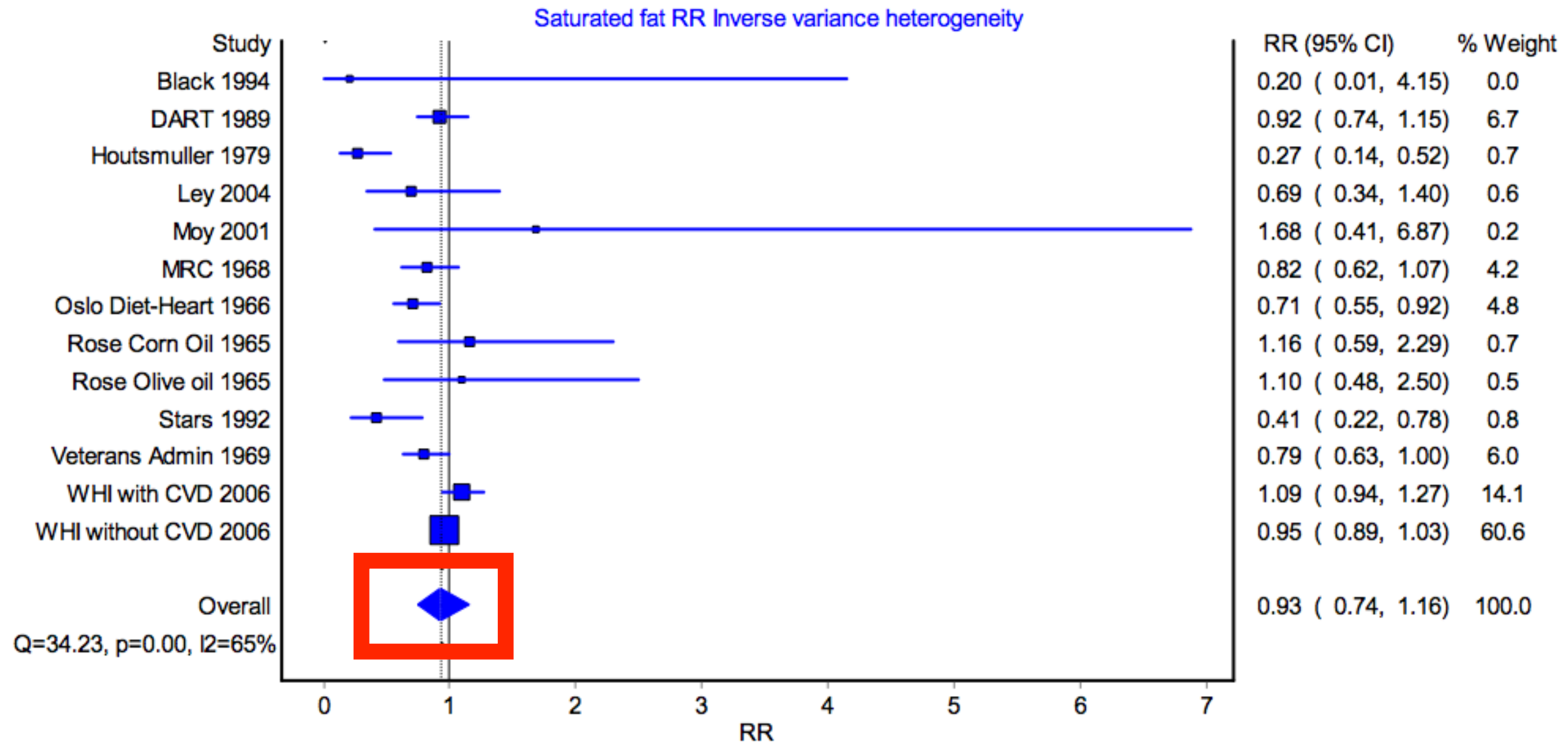
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Prospective Values of

Meta-analysis



Substitutions asked

Meta-analysis



Carbs for **saturated fat**



Monounsaturated fat for **saturated fat**



Polyunsaturated fat for **saturated fat**



Polyunsaturated fat for **carbs**



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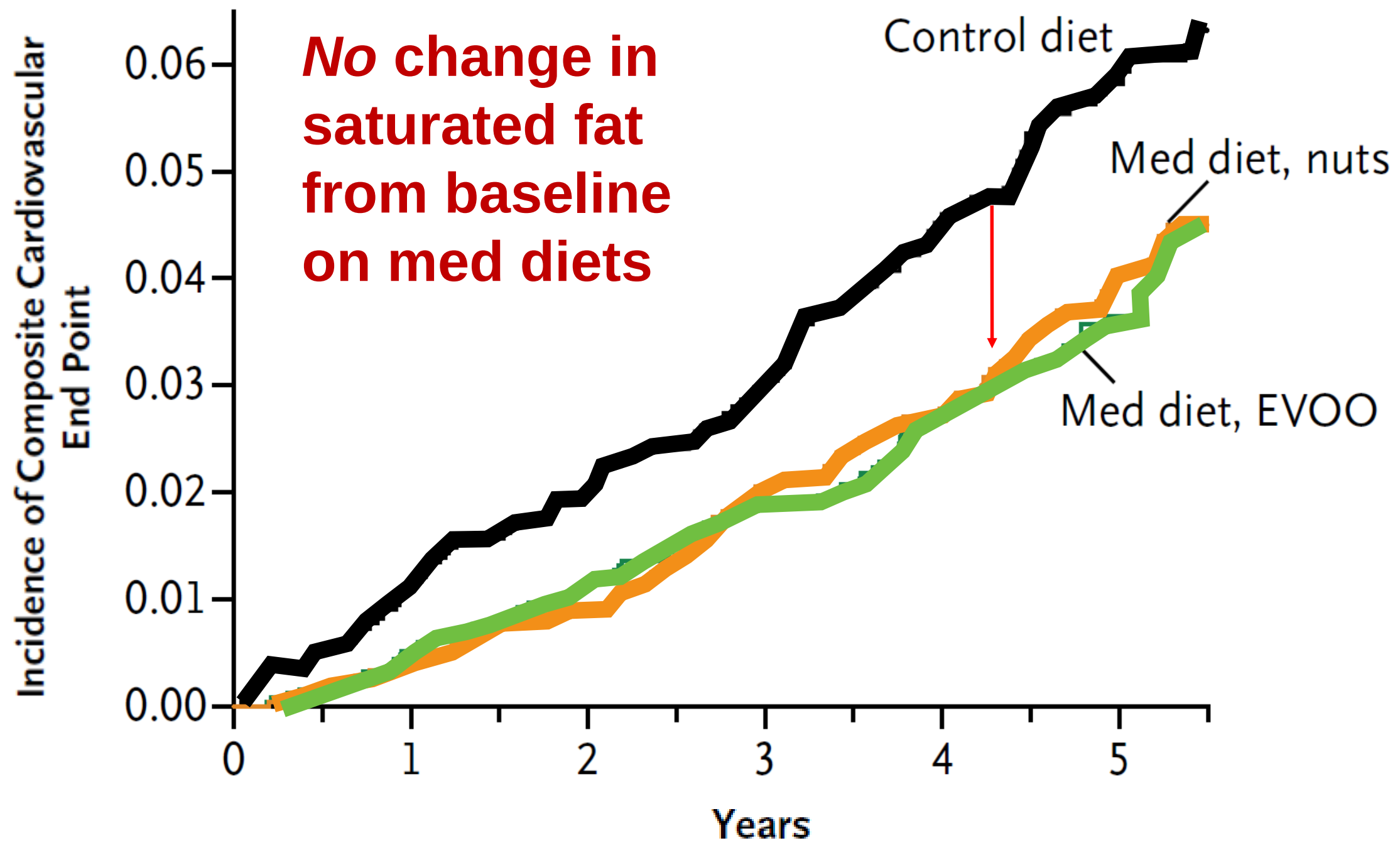
[A Correction Has Been Published >](#)

Primary Prevention of Cardiovascular Disease with a Mediterranean Diet

Ramón Estruch, M.D., Ph.D., Emilio Ros, M.D., Ph.D., Jordi Salas-Salvadó, M.D., Ph.D., Maria-Isabel Covas, D.Pharm., Ph.D., Dolores Corella, D.Pharm., Ph.D., Fernando Arós, M.D., Ph.D., Enrique Gómez-Gracia, M.D., Ph.D., Valentina Ruiz-Gutiérrez, Ph.D., Miquel Fiol, M.D., Ph.D., José Lapetra, M.D., Ph.D., Rosa Maria Lamuela-Raventos, D.Pharm., Ph.D., Lluís Serra-Majem, M.D., Ph.D., Xavier Pintó, M.D., Ph.D., Josep Basora, M.D., Ph.D., Miguel Angel Muñoz, M.D., Ph.D., José V. Sorlí, M.D., Ph.D., José Alfredo Martínez, D.Pharm, M.D., Ph.D., and Miguel Angel Martínez-González, M.D., Ph.D. for the PREDIMED Study Investigators

N Engl J Med 2013; 368:1279-1290 | [April 4, 2013](#) | DOI: 10.1056/NEJMoa1200303

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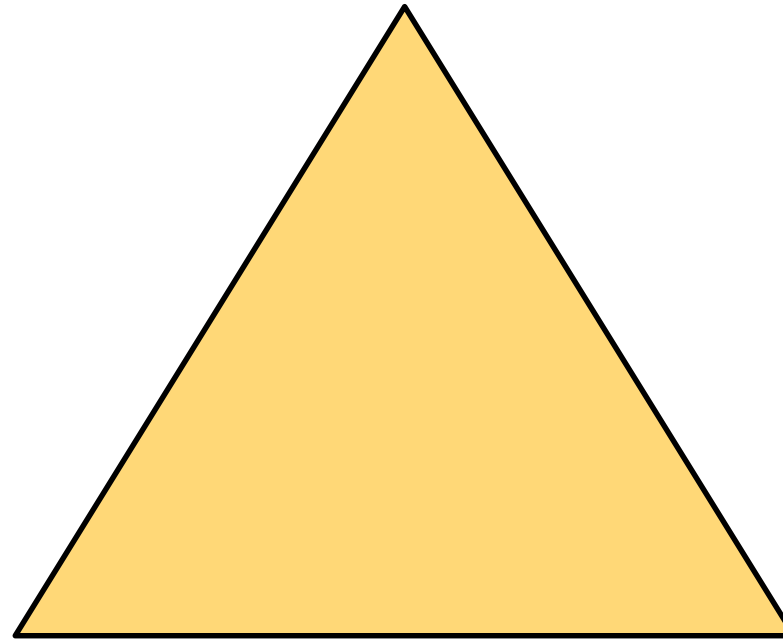


6. Moving forward constructively and scientifically

Evidence?

Science

Biology Epidemiology Experiments



Culture

Practice

Preventative health/wellness

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Nutrition

Food and Nutrition Guidelines

Evaluation of the Food and
Nutrition Guidelines series

Food and Nutrition Guidelines

This series of 5 population-specific food and nutrition guidelines background papers provide the Ministry's evidence base for nutrition policy advice and support the implementation of strategies such as the New Zealand Health Strategy.

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Rel

**Plant based
flexi-tarianism**

**Carbohydrate
restricted whole food**

Mediterranean

6 things in Common

1. They Are Low in Added Sugar



2. They Eliminate Refined Carbohydrates



3. They Eliminate Industrial Vegetable Oils

4. They Eliminate Artificial Trans Fats

5. They Are High in Vegetables and Fibre



6. They Focus on Foods Instead of Calories



The REAL food guidelines

Real food for real people, based on real evidence

The REAL food guidelines

Real food for real people, based on real evidence

1. Enjoy nutritious foods everyday including plenty of fresh vegetables and seasonal fruit.
2. Buy and prepare food from whole unprocessed sources of dairy, nuts, seeds, eggs, meat, fish and poultry.
3. Keep sugar, added sugars, and processed foods to a minimum in all foods and drinks.
4. If you drink alcohol, keep your intake low.
- 5. Prepare, cook, and eat minimally processed traditional foods with family, friends, and your**

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- 4. If you drink alcohol, keep your intake low.**

A note to physicians: Advise to restrict refined grains and carbohydrates to the degree necessary to maintain stable BGL and to prevent hyper-secreting insulin even with stable BGL

