Objectives of the IEM

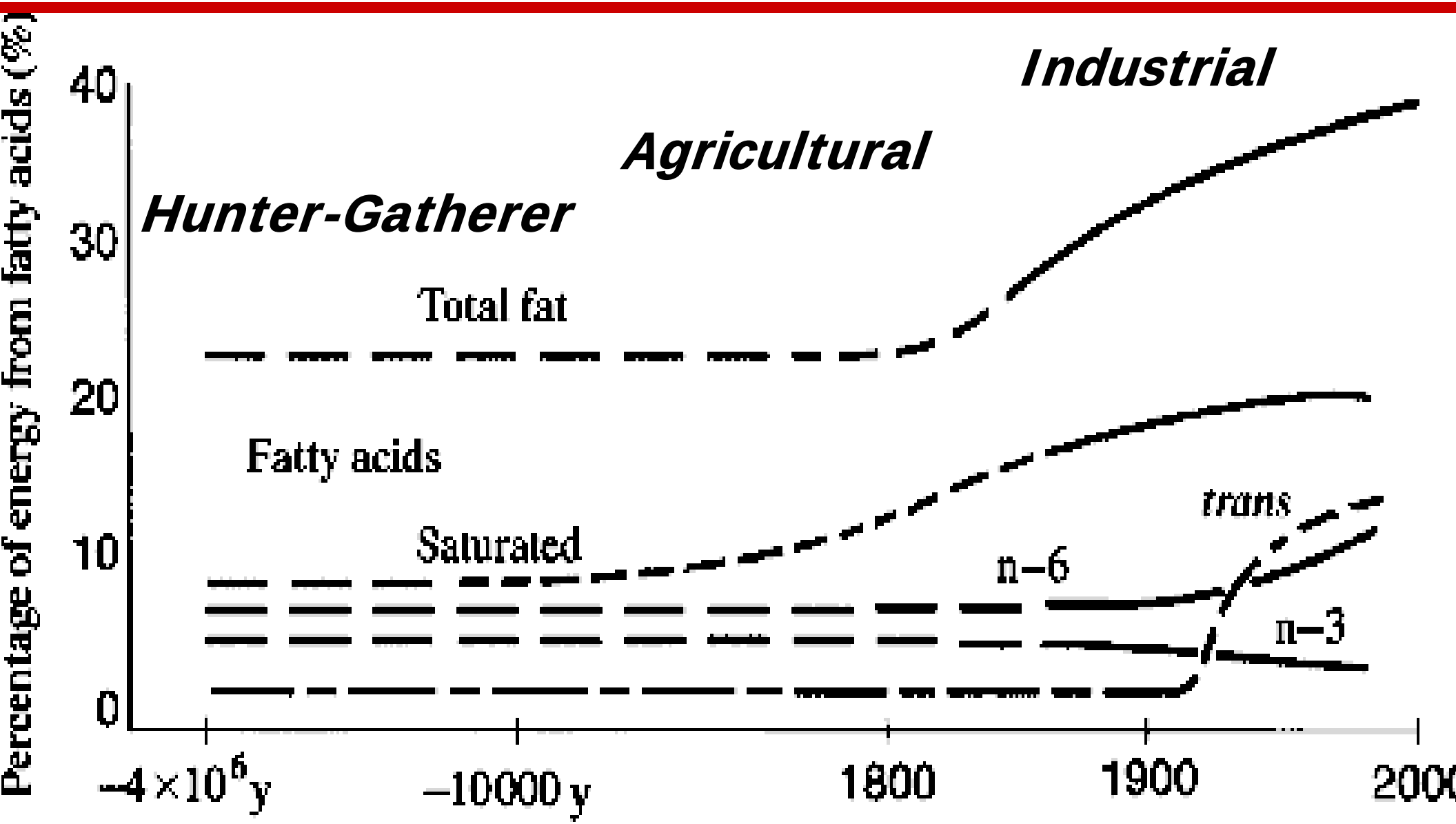
Review recommendations and guidelines on fats, update our current knowledge on health implications of the fat quality of the diet;

Review current consumer behavior: what do consumers know about fats and health and what do they do with this information.

Are consumers confused about what types of foods and fats they should eat, examine if current dietary fat intakes are or not inline with a healthy, recommended, fat intake.

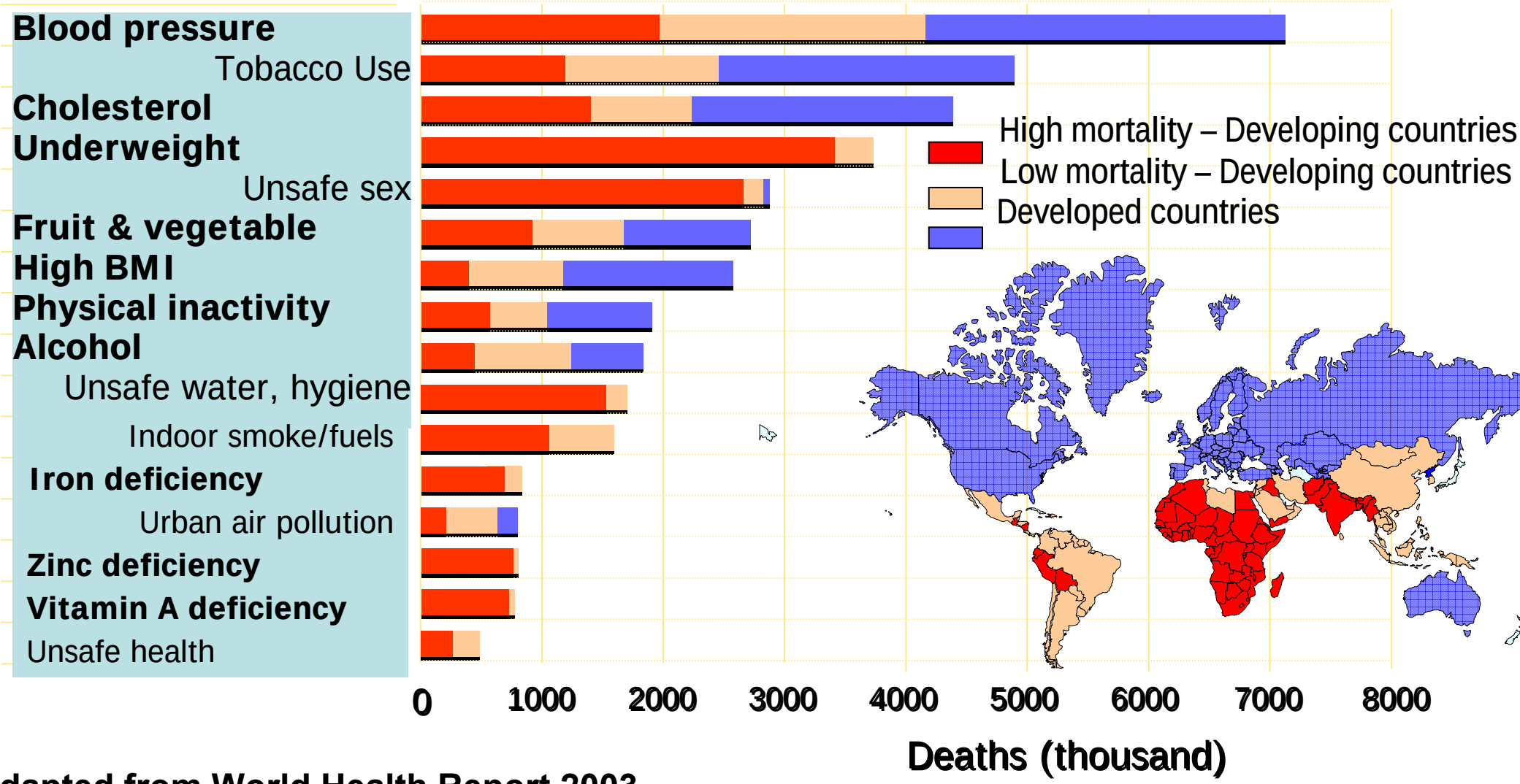
Call to action from IEM experts together with input of others to help consumers to make healthier choices with respect to the fat quality of the diet

Changes in dietary fat sources during Evolution



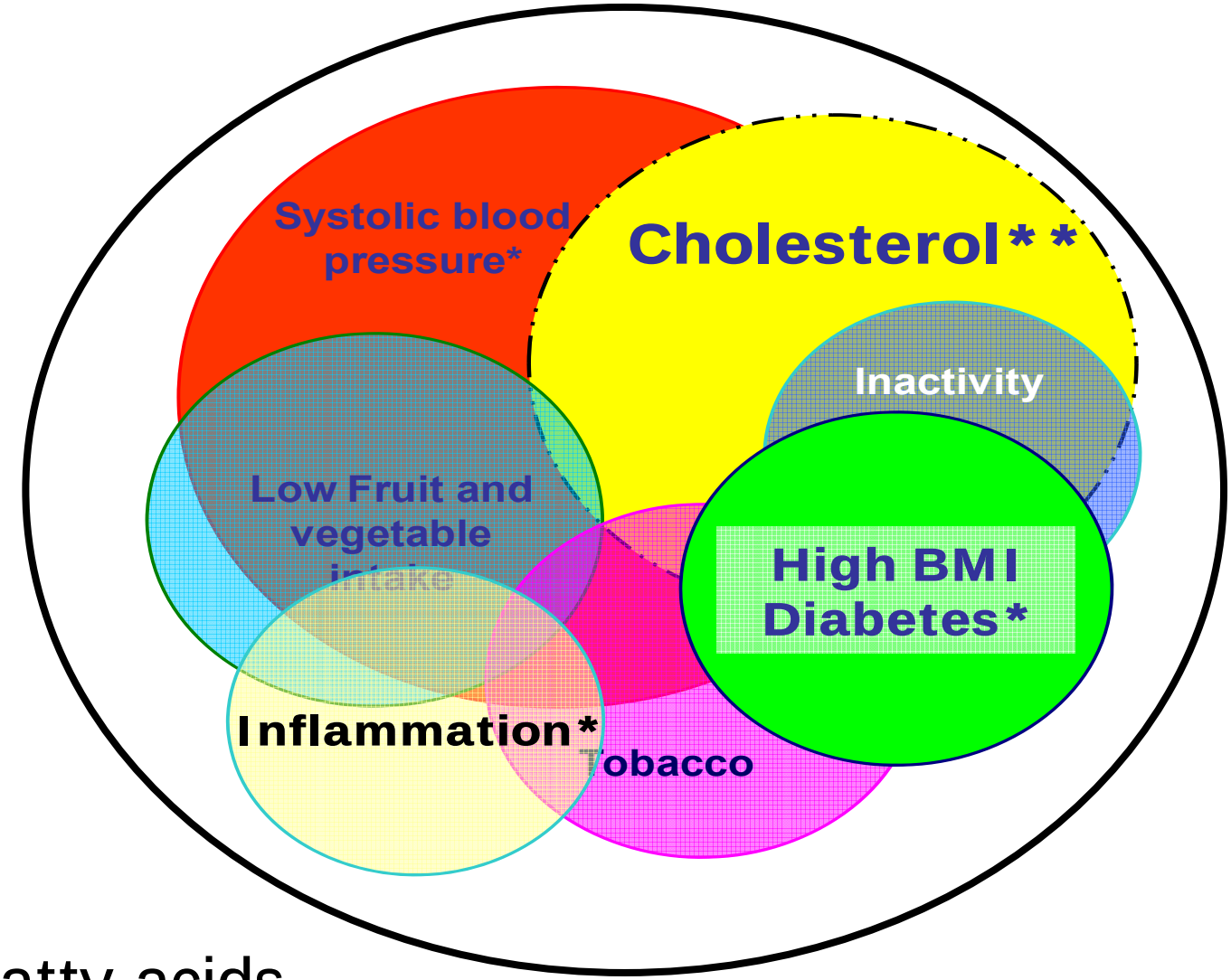
Simopoulos AP. Am J Clin Nutr. 1999;70:560-9S.

Deaths attributable to 16 leading risk factors: all countries



Adapted from World Health Report 2003

Nutrition for life long health and wellbeing



* Fats & fatty acids

Joint WHO/FAO expert consultation on diet, nutrition and the prevention of chronic diseases

Geneva, Switzerland
28 January – 1 February 2002

Diet, nutrition and the prevention of chronic diseases

Report of the Joint WHO/FAO expert consultation

www.who.int/hpr/nutrition/ExpertConsultationGE.htm

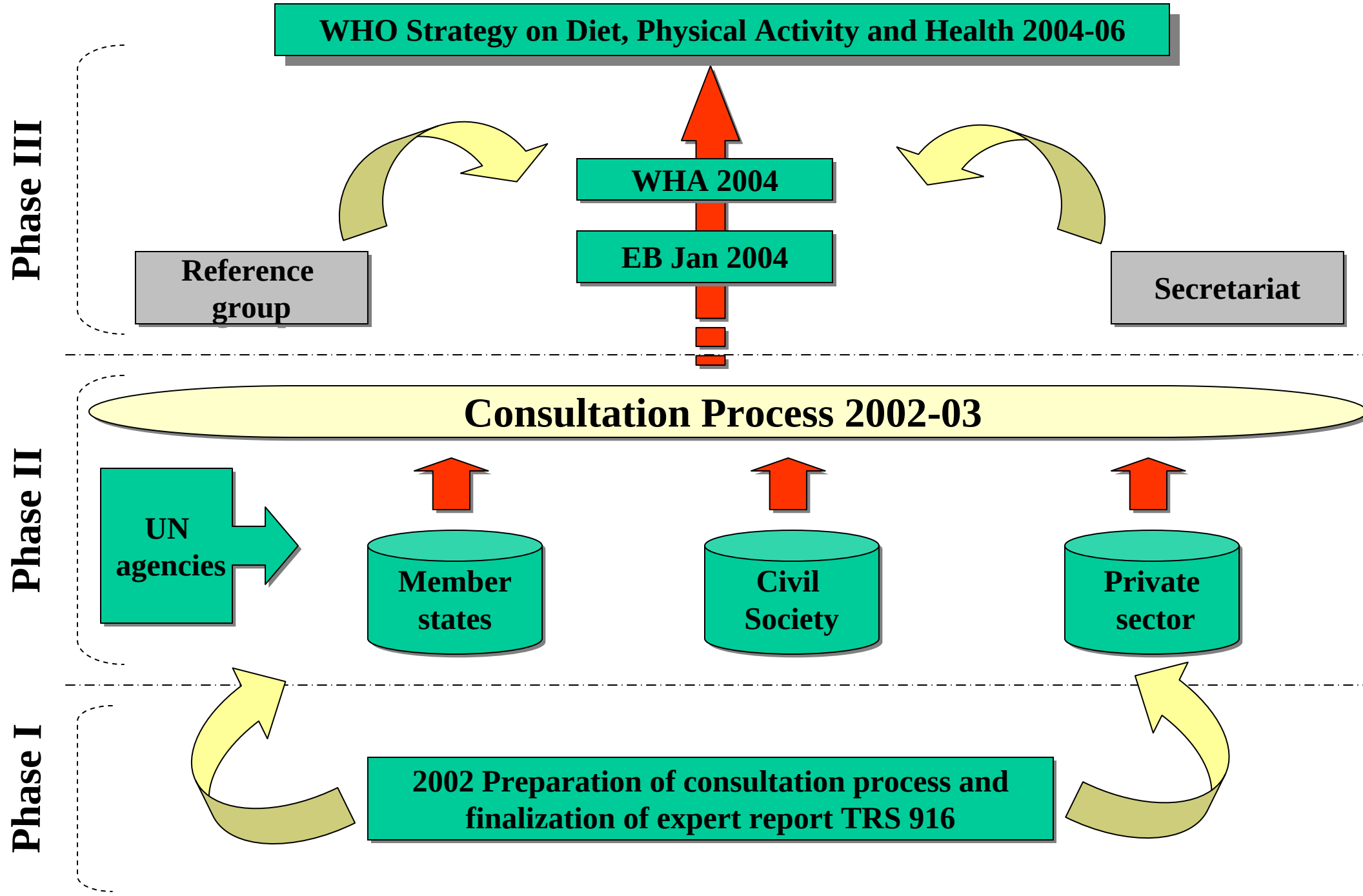
Principal nutrient/food changes

Reduce

- Total fat
- Saturated Fats (C14,C16)
- Trans fatty acids
- Free sugars
- Refined starches
- Sodium/salt
- Preserved meats

Increase

- Vegetables,
- Fruits, legumes
- Fibre/NSP
- ω - 3 fatty ac (LNA,EPA,DHA)
- Iron/iodine
- Zinc/folate?
- PHYSICAL ACTIVITY


Phase III
WHO Strategy on Diet, Physical Activity and Health 2004-06
WHA 2004
EB Jan 2004
Reference group
Secretariat
Phase II
Consultation Process 2002-03
UN agencies
Member states
Civil Society
Private sector
Phase I
2002 Preparation of consultation process and finalization of expert report TRS 916

Recommendations In Need of Updates

- Total Fat
- Saturated Fat (C12, C14, C16, C18)
- n-6 PUFA
- n-3 PUFA (ALA, EPA+DHA)
- Trans Fat

Newer Recommendations post FAO/WHO 1994 (Fats and Oils)

Table 2.1 Overview of dietary recommendations and criteria for total fat intake

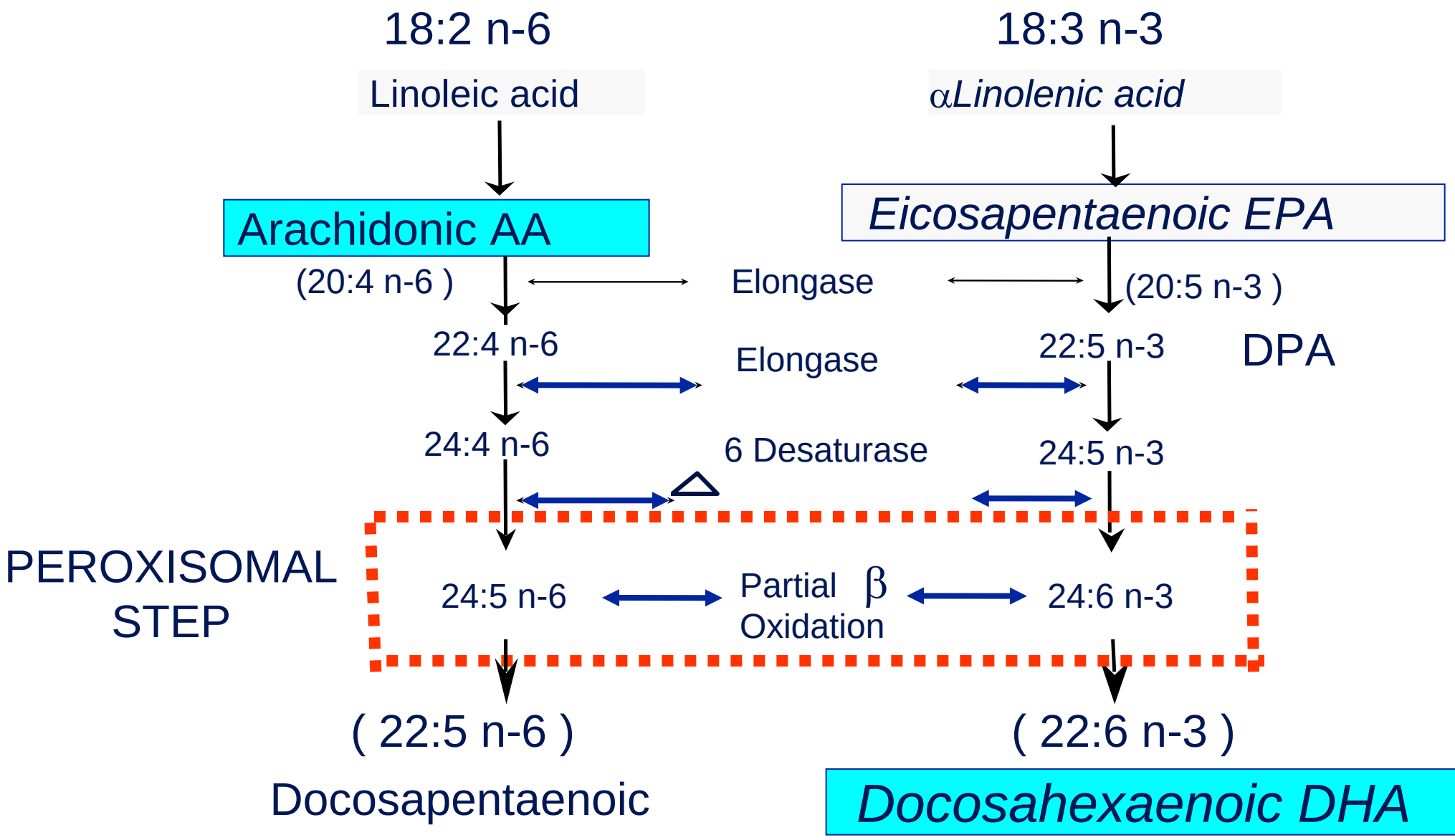
Report	Adults	Infant 0–6 months	Infant 6–12 months	Child 1–3 years	Child and Adolescent 4–18 years[1]	Pregnant women	Lactating women
FAO/WHO	15–35 en% *Sedentary:* 30 en% UL (D,P)	Similar to human milk (M)	30–40 en%	30–40 en%	-	>20 en%	>20 en%
India	20–50 g/d	-	-	25 g/d	-	30 g/d	40 g/d
Eurodiet	<30 en% PG (D)	-	-	-	-	-	-
Netherlands	20 (P)–40 (P,M) en% AI	45–50 en% (M)	40 en%	25–40 en% (M)	20–40 en%	20–40 en%[2]	20–40 en%[2]
DACH	30 en% (D,P)	45–50en%	-	30–40 en%[3] (P)	30 en%	30–35 en%	30–35 en%
US (IOM)	20–35 en% AMDR (D)	31 g/d AI	30 g/d AI	30–40 en% AMDR	20–35 en% AMDR	-	-
AU/NZ	20–35 en% (D,P) *Overweight*: 20–25 en% (D)	31 g/d AI (M)[4]	30 g/d AI (M)[4]	-	-	-	-
China	20–30 en%	45–50en%	35–40 en%	30–35 en%	25–30 en%	-	-

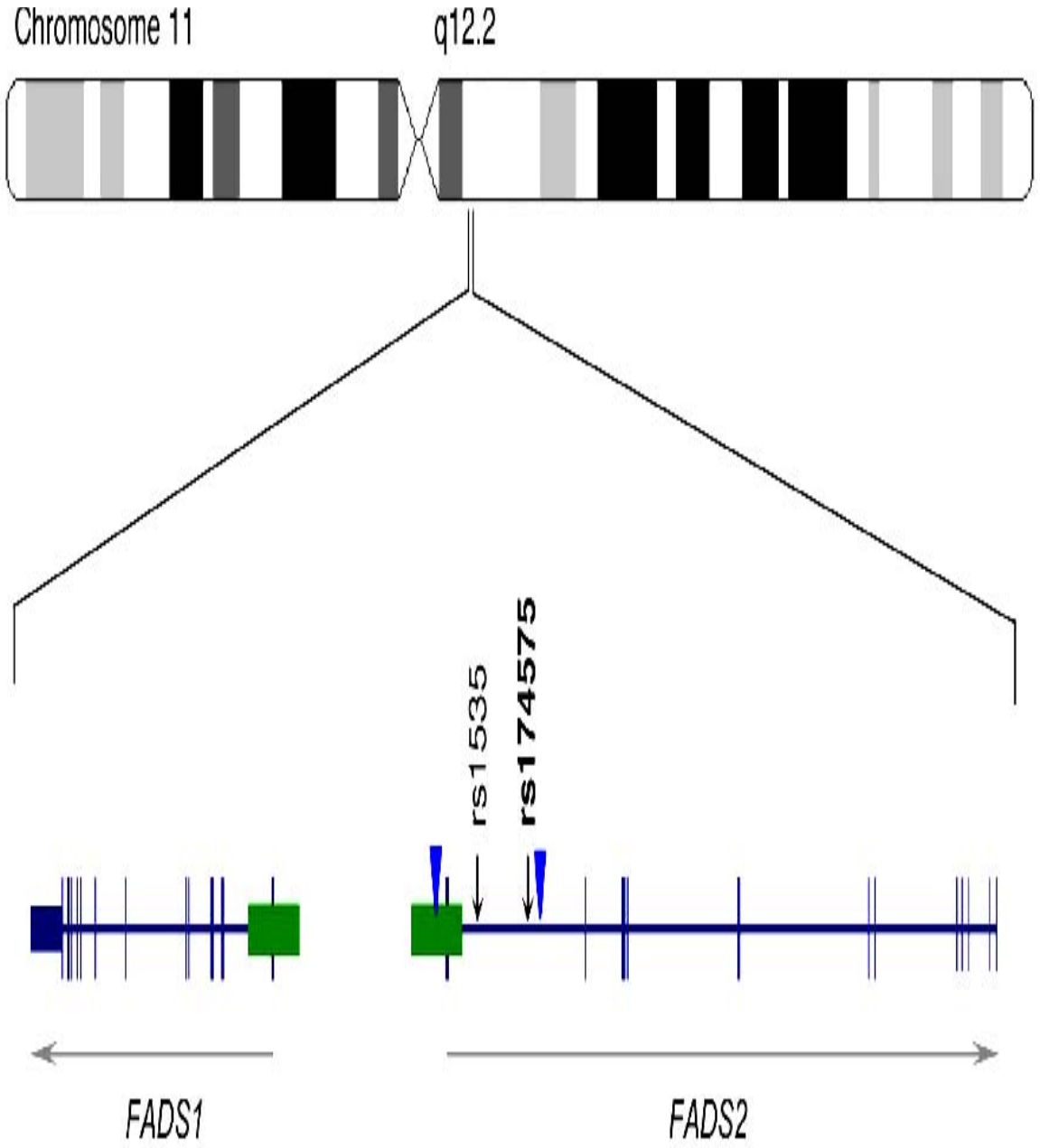
When no criterion is reported in the table, the criterion on which the recommendation is based has not been provided. These are probably similar to the criterion used for adults.

Table 1. Types of dietary reference intakes (DRIs)

DRI	Definition and Description	Historic Use for Fats and Fatty Acids
Estimated Average Requirement (EAR)	Intake that meets the nutrient needs of half of the healthy individuals in a life stage or gender group. *Reflects the estimated average (median) requirement and is particularly appropriate for applications related to planning and assessing intakes for groups of persons.*	Not traditionally used for fats and fatty acids.
Recommended Dietary Allowance (RDA)	A value based on observed or experimentally determined estimates of nutrient intake by a group of people who are apparently healthy and assumed to be maintaining an adequate nutritional state. *The RDA is calculated from the EAR and covers the requirements for 97–98 percent of the population. It is dependent upon estimating the variance around the EAR and reflects a point estimate defined as two standard deviations above the EAR.*	Not traditionally used for fats and fatty acids.
Tolerable Upper Intake Level (UL)	Highest average intake that is likely to pose no risk.	Has been used for total fat, saturated fat, total polyunsaturated fat, ALA, EPA+DHA, and dietary cholesterol .
Adequate Intake (AI)	Used when an EAR/RDA cannot be developed; average intake level based on observed or experimental intakes.	Has been used for total fat, linoleic acid, alpha-linolenic acid, and EPA and DHA.
Acceptable Macronutrient Distribution Range (AMDR)	An intake range for an energy source associated with reduced risk of chronic disease.	Has been used for total fat, linoleic acid, and alpha-linolenic acid.

Metabolism of n-6 and n-3 LCPs





genetic polymorphism (rs174575) in *FADS2* delta 6 Desaturase activity mediates human milk effect on infant's IQ

LA and ALA should be considered essential and indispensable since they cannot be synthesized by humans. While DHA and AA can be synthesized from ALA and LA respectively, they should be considered dispensable although a dietary supply may be necessary for long-term health.

Given the limited and highly variable formation of DHA from ALA (1-5 %) and because of its critical role in normal retinal and brain development in the human they should be considered conditionally essential during early development, can be provided by human milk.

Similarly they might be considered necessary for life-long health considering intakes required for the prevention of CVDs and other chronic disease

Summary of childhood data

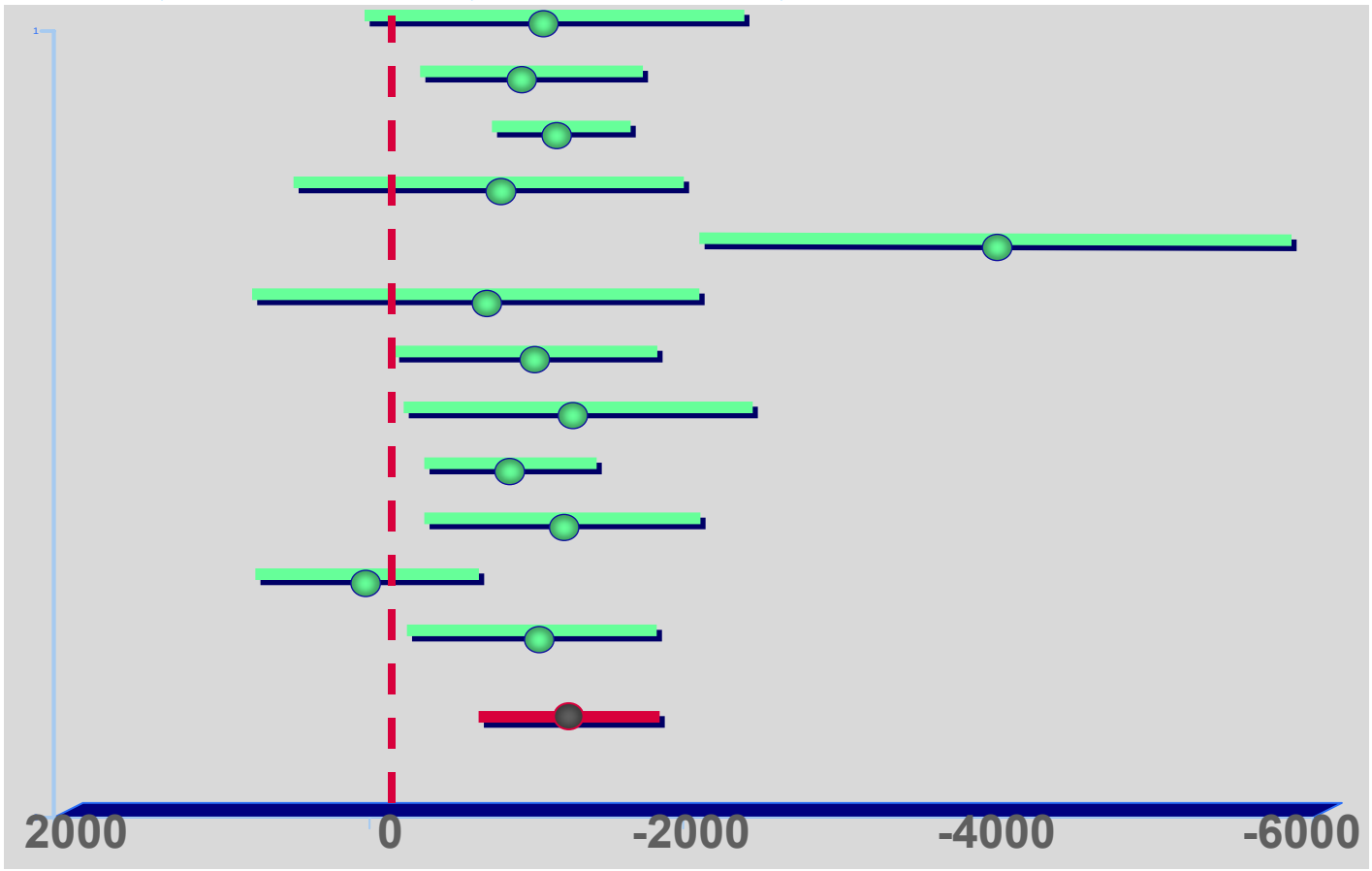
- Inconclusive evidence of effect of fish oil on ability and behaviour among school-age children
- Studies mostly limited to children with neurodevelopmental disorders
- Issue of importance for public health
- Large RCTs are urgently needed

What research is needed to develop/ strengthen n-3 LCP recommendations for children

- Address age specific proposed outcomes (depression, aggressiveness, mood swings, attentiveness, learning).
- Assess the impact on learning/behavior in school since this is of great social/public/economic interest.
- The functional effects may also be specific to given DHA/EPA/ and combinations. Evidence of biochemical inter conversion not sufficient to claim functional equivalence
- Studies to define optimal combination of n-3 LCPs and dose need to be conducted.

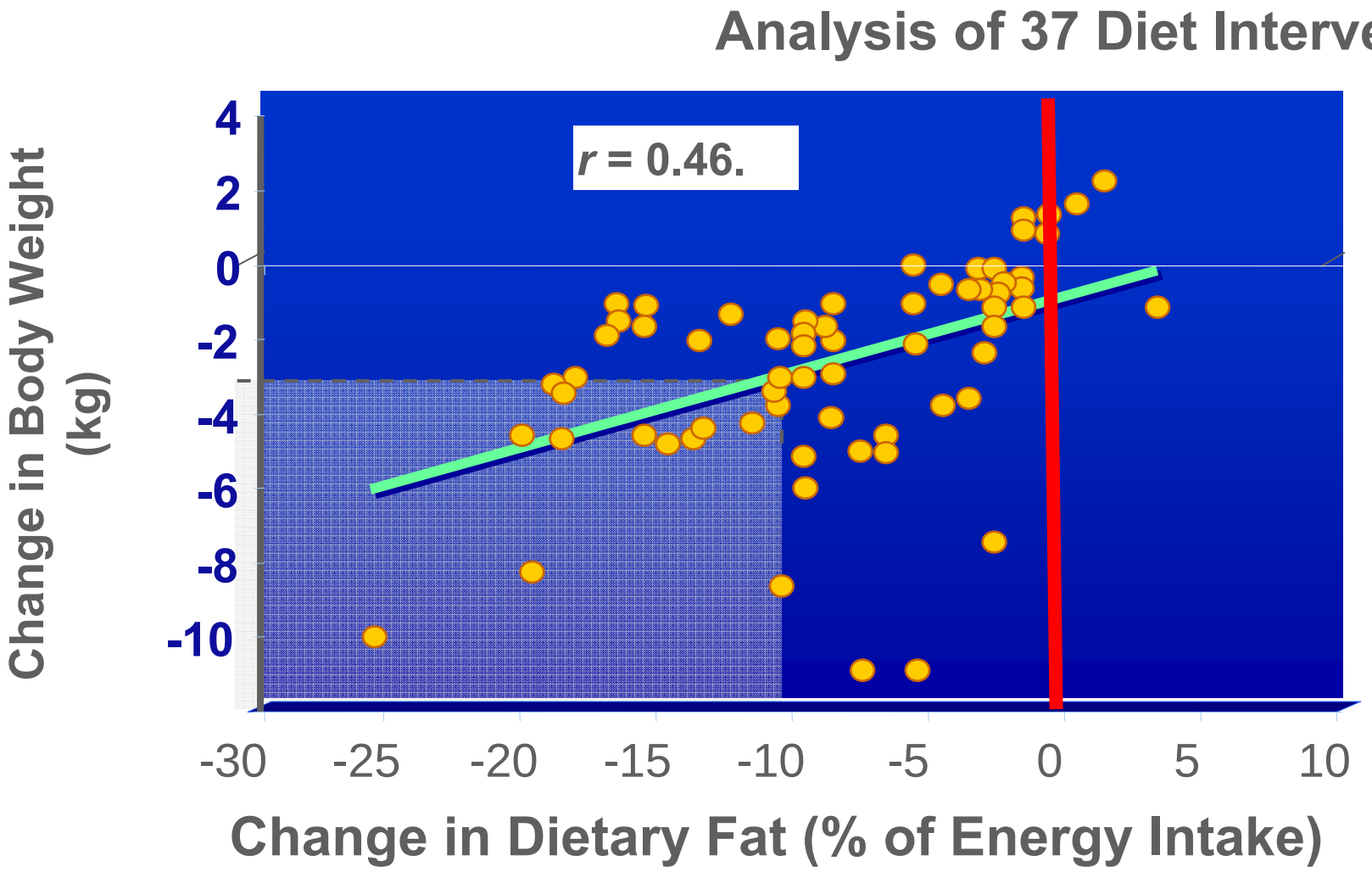
Ad Libitum Low-Fat Diets Decrease Daily Energy Intake

Meta-analysis of 12 Intervention Trials



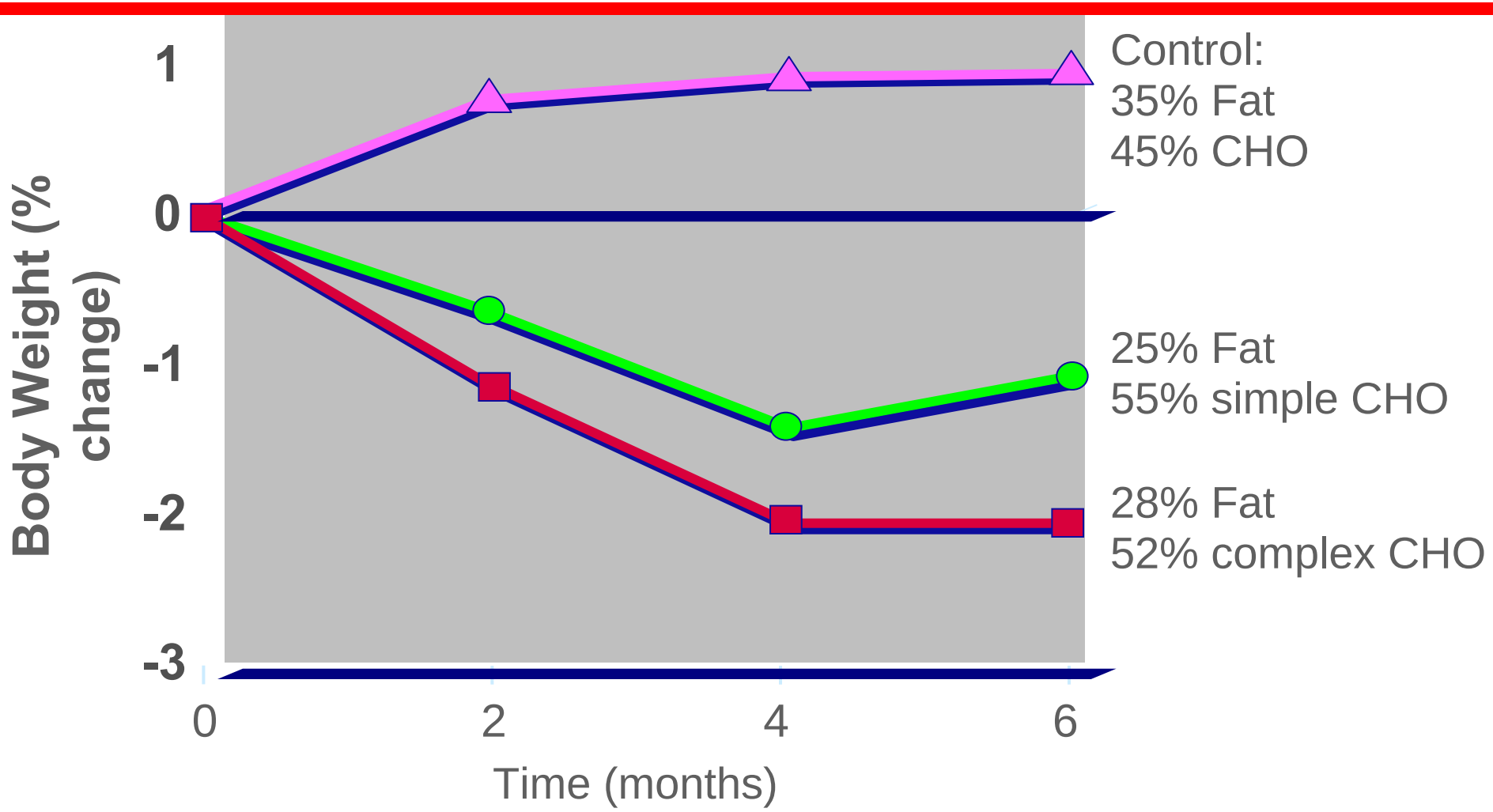
Astrup et al. *Int J Obes Relat Metab Disord* 2000;24:1545.

Decreasing Dietary Fat is Associated with a Decrease in Body Weight



Yu-Poth et al. *Am J Clin Nutr* 1999;69:632.

Relationship Between Dietary Macronutrient Composition and Body Weight

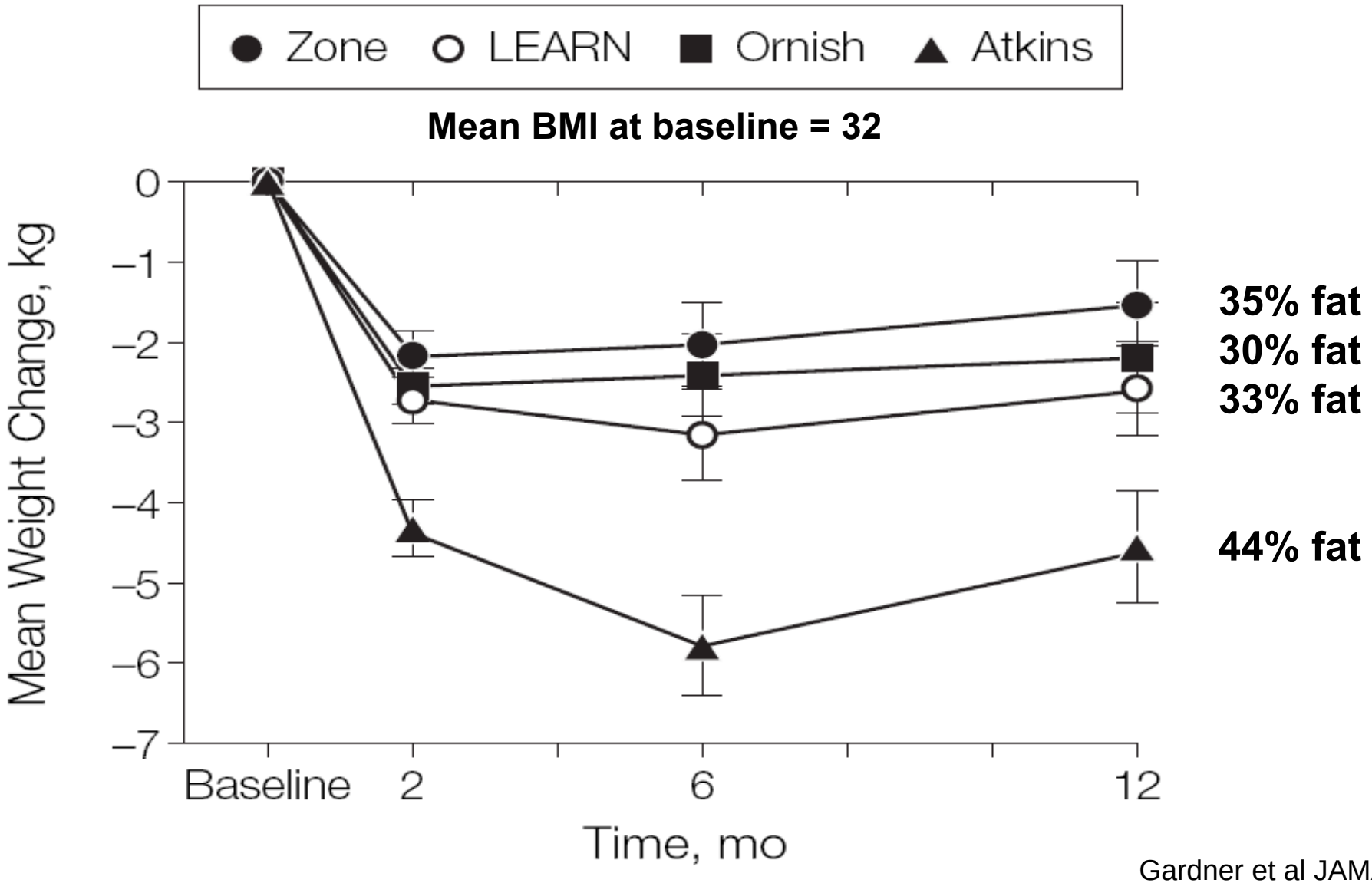


Saris WHM et al.,Int J Obes 24:1310,2000

Weight Loss at 6-Months in RCTs of Low-fat vs Low-Carbohydrate Diets

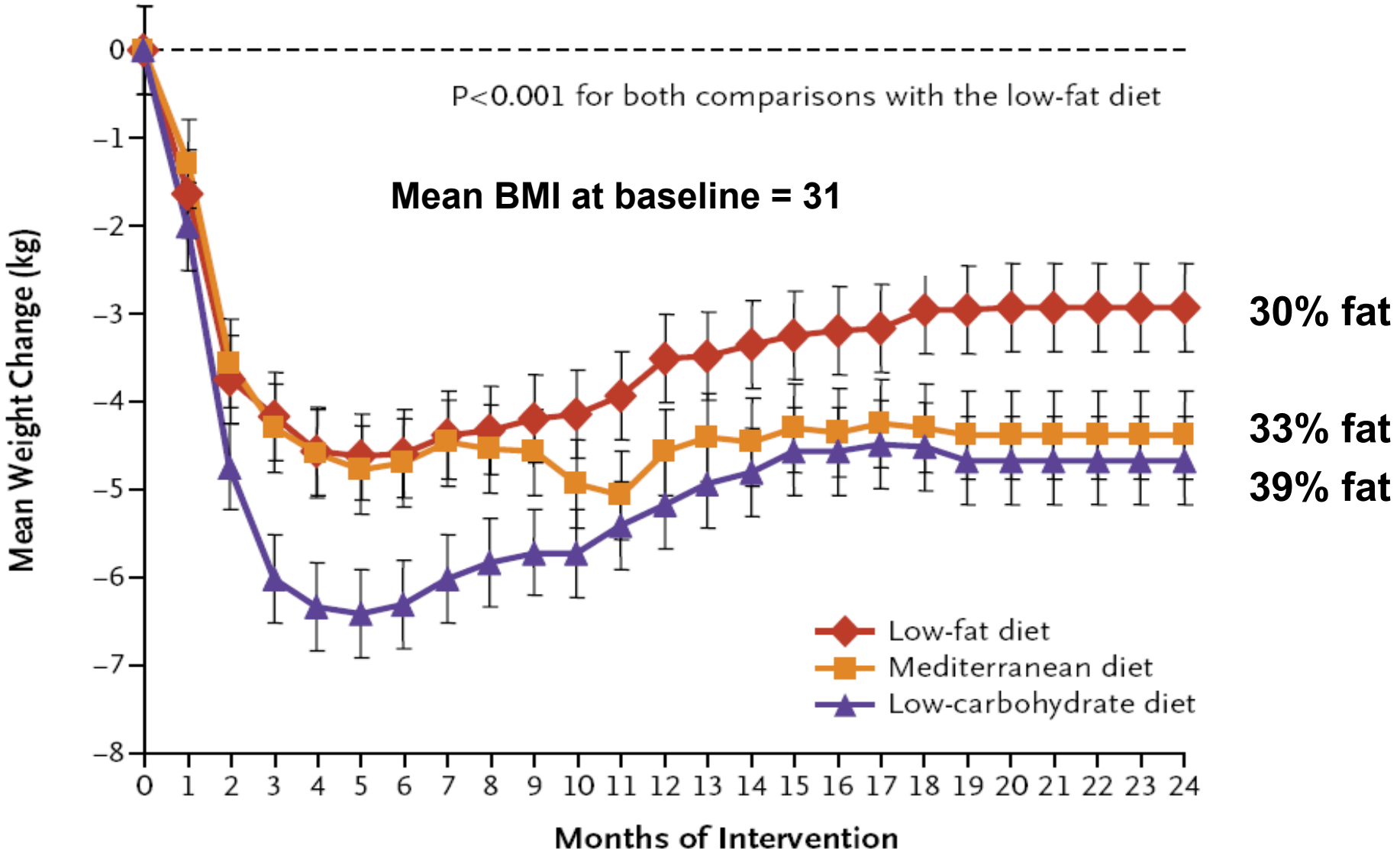
		Weight Loss (kg)		Difference
Study	n	Low-fat	Low-carb	(kg)
Samaha (2003)	132	-1.9	-5.8	3.9
Brehm (2003)	42	-3.9	-8.5	4.6
Foster (2003)	63	-5.3	-9.6	4.3
Yancy (2004)	120	-6.5	-12.0	5.5

Randomized Dietary Trials of Equal Intensity Intervention



Gardner et al JAMA 2007

Randomized Dietary Trials of Equal Intensity Intervention



Shai et al NEJM 2008

Prospective Cohort Studies

Large scale trials that have repeated data, such as repeated cross-sectional studies and prospective cohort studies have yielded mixed results.

Framingham data; total calories as well as % calories from fat decreased from the cohorts sampled in the 50's and 60's to the 1984-88 cohort yet prevalence of excess weight increased

Health Professional's FU study of > 16,000 men; higher total fat consumption was associated with increases in waist circ; after controlling for delta BMI effect of total fat disappeared whereas effect of TFA remained

Summary of the prospective cohort studies,

Many studies do show a positive relationship between dietary fat intake and weight gain, data are overall inconclusive in relating dietary fat and body weight.

Strengths include sample size (from 360 to over 41,000 participants), prospective nature as well a longer-term duration and as such, ability to evaluate more chronic effects of diet.

Data are still limited by self-reported intake.

Intervention Trials

Smaller intervention studies have found that the low CHO higher fat diets “Atkins diet” often lead to more weight loss than either low fat or more traditional weight loss diets. However effect is often lost at or beyond 12 months.

Recent 2 yr long RCT of low CHO, Mediterranean, or low-fat diets showed greater weight loss on low CHO and Med diets

Very large (n >48,000) intervention trial, WHI is supportive of connection between lower fat and weight loss.

Limitations lack of specific comparison of dietary fat as the low-CHO are also higher in protein, often a high attrition rate

Total fat intake affects weight gain / loss ?

PRO

- Ecologic studies and animal experiments.
- Fat content of foods affects energy density, one of the components of dietary quality.
- Astrup meta-analysis, trials 2-12 months.

CON

- Energy density is complex, and fat content not a major determinant of long term weight loss in trials of different energy density .
- Women's Healthy Eating & Living Trial and Women's Health Initiative, two very large and long duration RCTs of low fat, both showed minimal effects on weight.
- Multiple well-controlled RCTs of equal intensity interventions (true causal inference) show greater weight loss on high fat diets.

WHO TRS 916 Report : strength of evidence on factors and risk of developing CVD

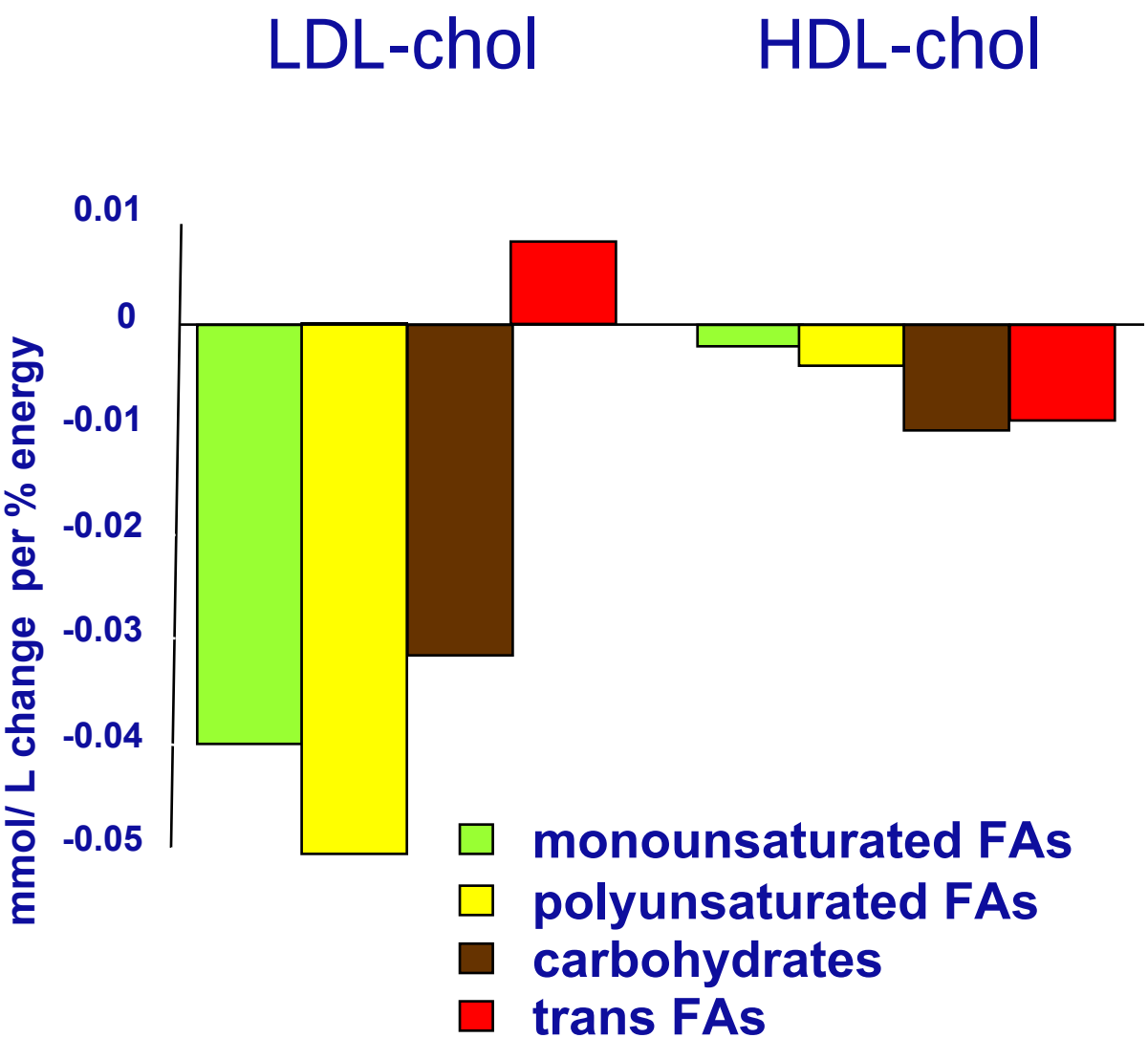
Evidence	Decreased risk	No relationship	Increased risk
Convincing	Regular physical activity **Linoleic acid 18:2n-6** **Fish and fish oils (EPA &DHA)** Vegetables & fruits (including berries) Potassium Low to moderate alcohol intake	Vitamin E Supplements	**Myristic and palmitic acids 14:0 16:0** **Trans fatty acids** High sodium intake Overweight High alcohol intake
Probable	**α-Linolenic acid 18:3 n-3** **Oleic acid 18:1 n-9** Fibre Nuts (unsalted) **Plant sterols/stanols** Folate	**Stearic acid 18:0**	**Dietary cholesterol** Unfiltered boiled coffee
Possible	Flavonoids Soy products		**Fats rich in lauric acid** Impaired fetal nutrition Beta-carotene supplement

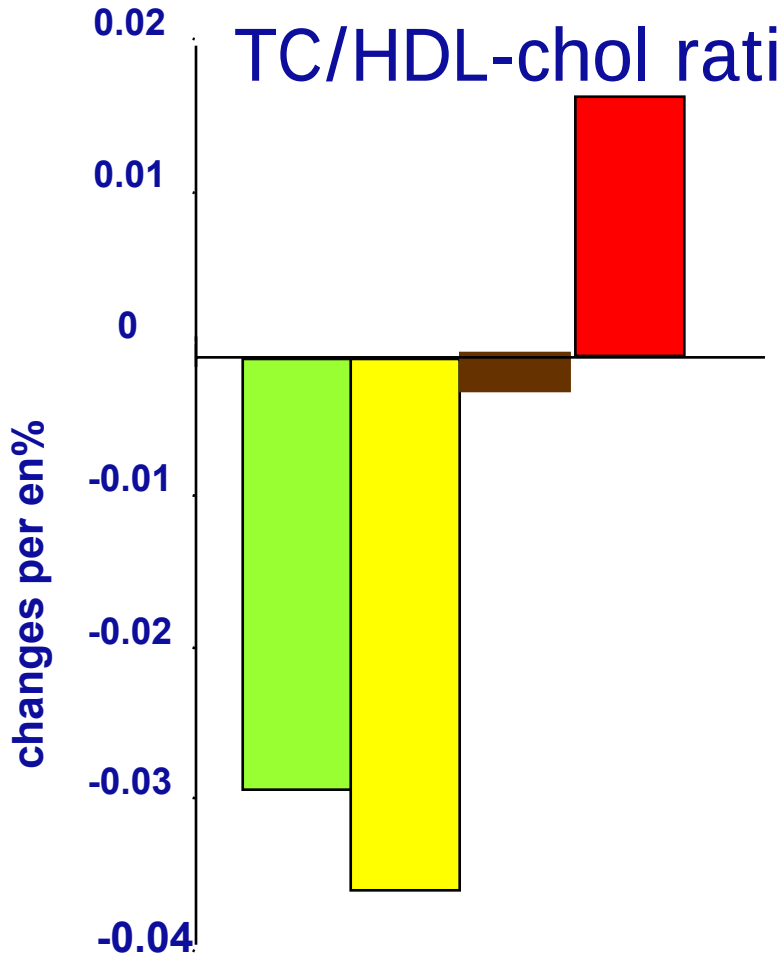
TRS 916 WHO 2003

Mechanisms by Which Dietary Fatty Acids Potentially Influence Risk of Coronary Heart Disease

- Effects on Lipoprotein and Cholesterol metabolism receptor systems, gene expression and regulation (LDL, HDL, Lp(a), TG)
- Prostanoids:(Eicosanoids and Docosanoids) related functions Inflammation/cytokines
- Blood pressure / arterial stiffness
- Thrombosis and thrombolytic mechanisms
- Oxidative stress and re-perfusion injury
- Endothelial function and adhesion molecules
- Cardiac Rhythm (arrhythmias)
- Insulin Sensitivity

Effect on lipoproteins of replacing saturated fat with other fatty acids or carbohydrates





Source: Mensink et al Am J Clin Nutr 2003

American Journal of Epidemiology

Vol. 161, No. 7
Printed in U.S.A.
DOI: 10.1093/aje/kwi085

Dietary Fat Intake and Risk of Coronary Heart Disease in Women: 20 Years of Follow-up of the Nurses' Health Study

Kyungwon Oh[1], Frank B. Hu[1,2,3], JoAnn E. Manson[2,3,4], Meir J. Stampfer[1,2,3], and Walter C. Willett[1,2,3]

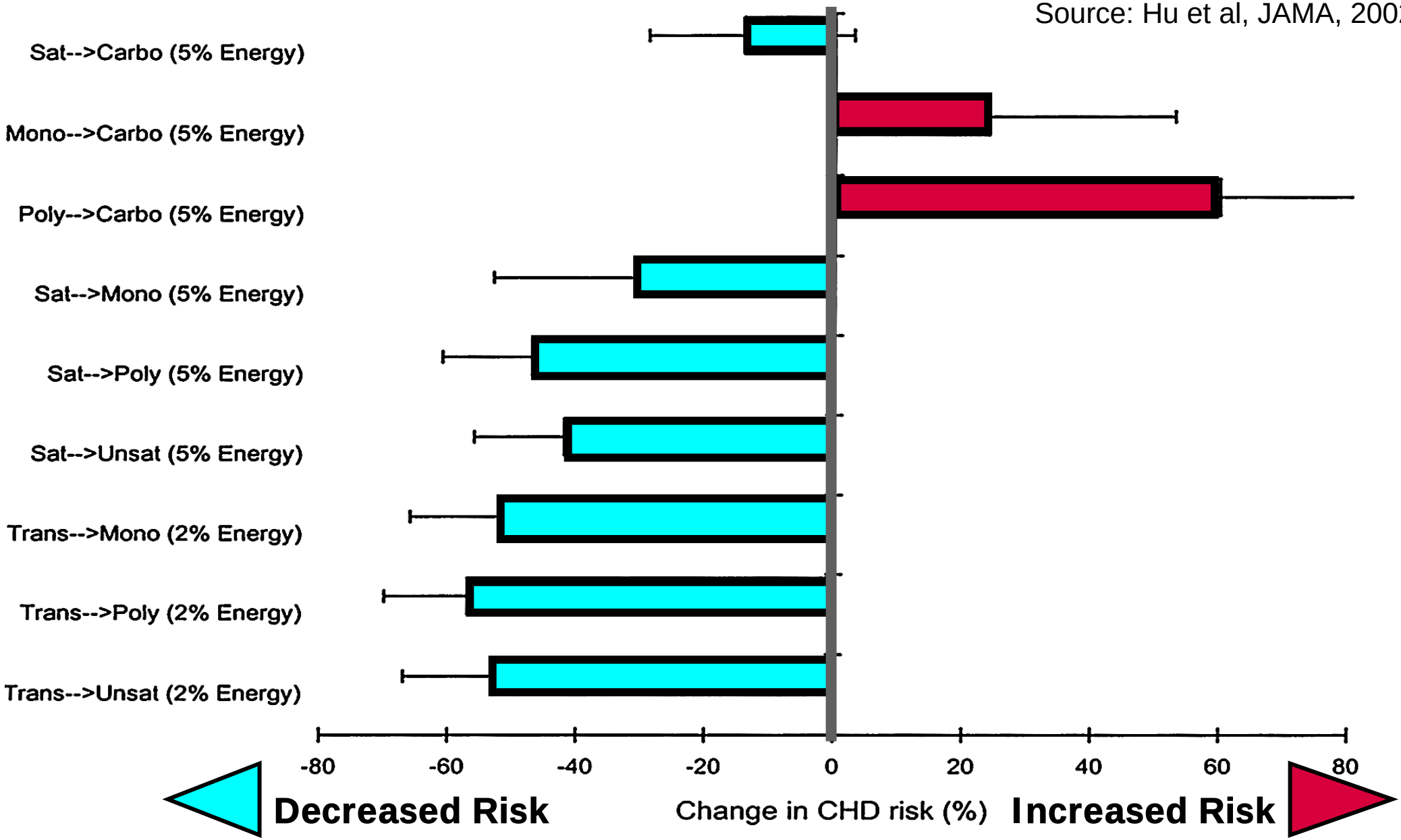
In summary, our results provide evidence that high intake of trans-fat increases the risk of CHD in women, the effects are stronger among younger women.

Our findings also support a benefit of polyunsaturated fat intake, at least up to approximately 7 percent of energy, in preventing CHD, particularly among women who are younger or overweight.

Am J Epidemiol 2005;161:672–679

Nurses' Health Study: changes in risk of coronary heart disease associated with iso-energetic diet substitutions

Source: Hu et al, JAMA, 2002



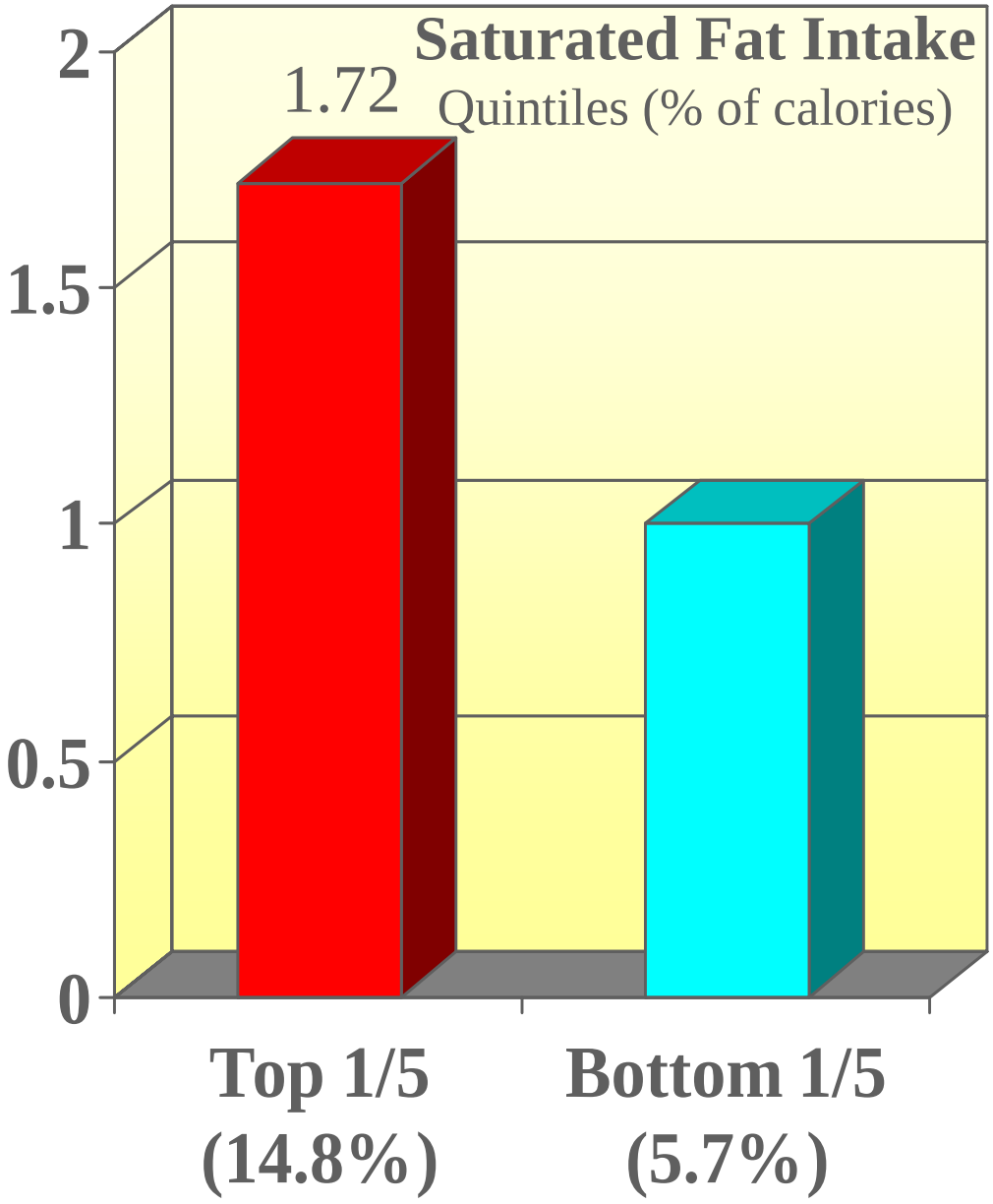
Changes in serum lipids (mmol/L by replacing 1% E individual fatty ac for carbohydrate based on meta-analysis [EFSA J (2004) 81, 1-49]

Fatty acid (1% energy exchange)	Total cholesterol	LDL cholesterol	HDL cholesterol	Total:HDL cholesterol
Lauric acid (12:0)	+0.069	+0.052	+0.027	-0.037
Myristic acid (14:0)	+0.059	+0.048	+0.018	-0.003
Palmitic acid (16:0	+0.041	+0.039	+0.010	+0.005
Stearic acid (18:0)	-0.010	-0.004	+0.002	-0.013
Elaidic acid (18:1 trans)	+0.031	+0.040	0.000	+0.022
Oleic acid (18:1 cis)	-0.006	-0.009	+0.008	-0.026
Linoleic acid) (18:2n-6)	-0.021	-0.019	+0.006	-0.032

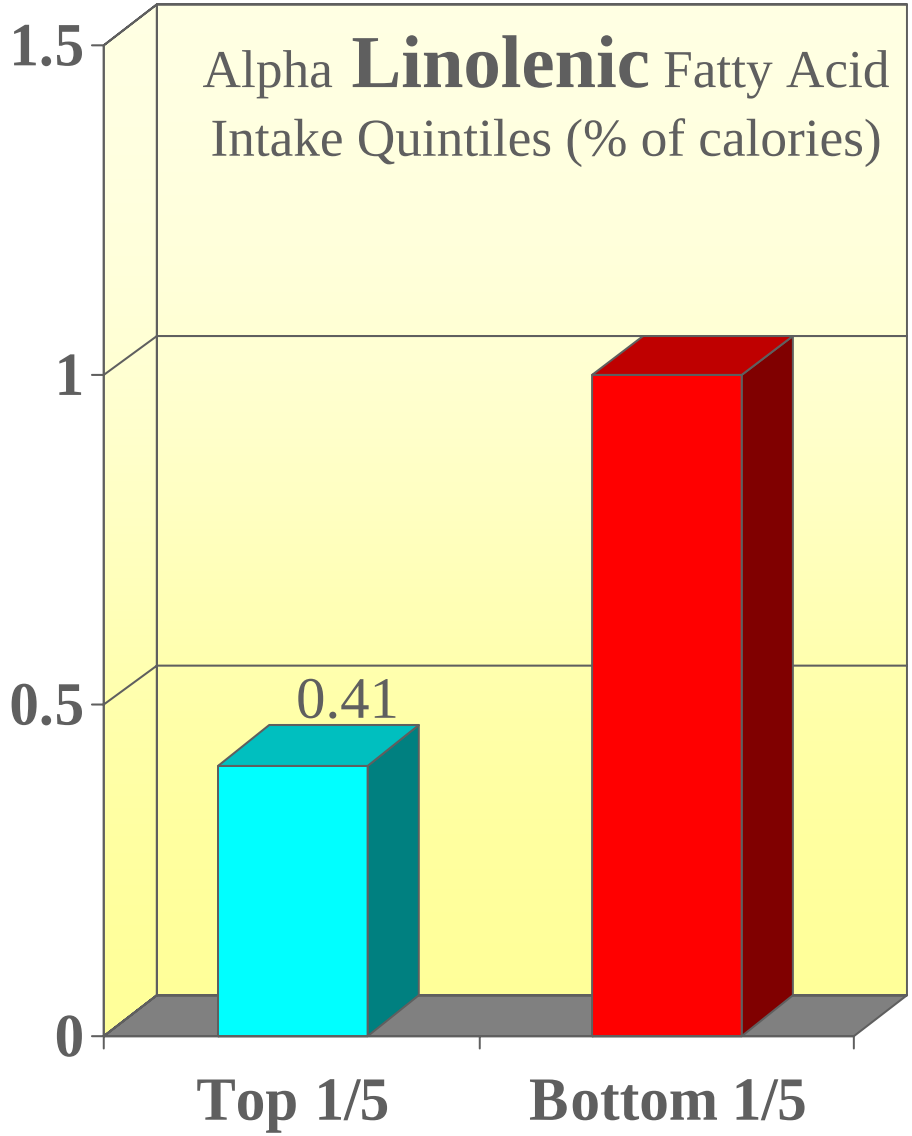
Dietary cholesterol

- Very high intakes (1200mg/d) down-regulate the LDL receptor and lead to large increases in LDL cholesterol
- Over the range of practical intake (0-400mg/d) each 100mg leads to an increase of about 0.056 mmol/L in plasma LDL cholesterol and 0.008 mmol/L in HDL cholesterol (Weggemans et al. 2001)
- Individuals with the ε4 allele for apolipoprotein are more sensitive to dietary cholesterol and may show a 10% increase in plasma cholesterol following an additional 300mg/d

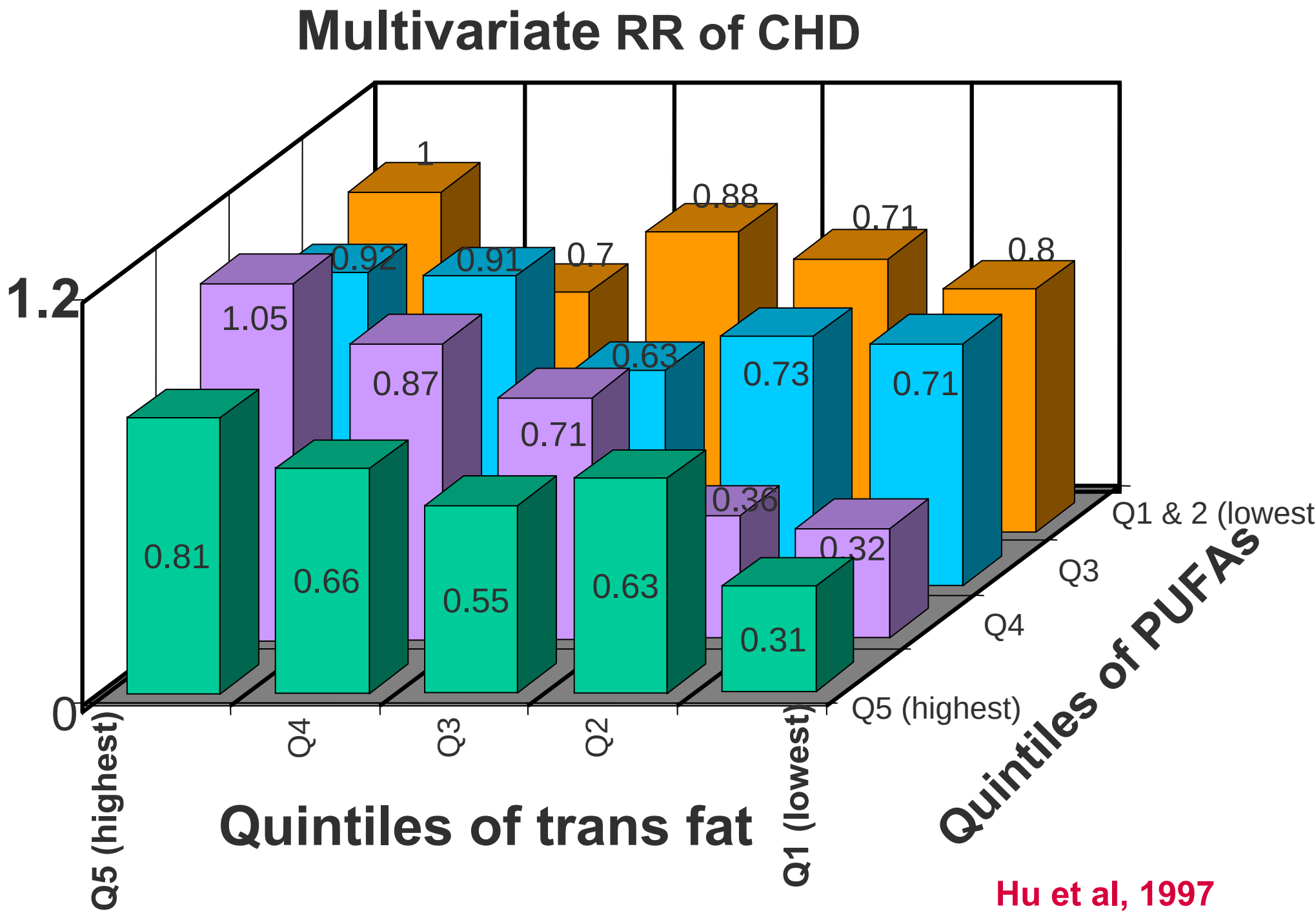
Relative risk was after adjusting for dietary fiber intake.



Ascherio et al *BMJ* 1996

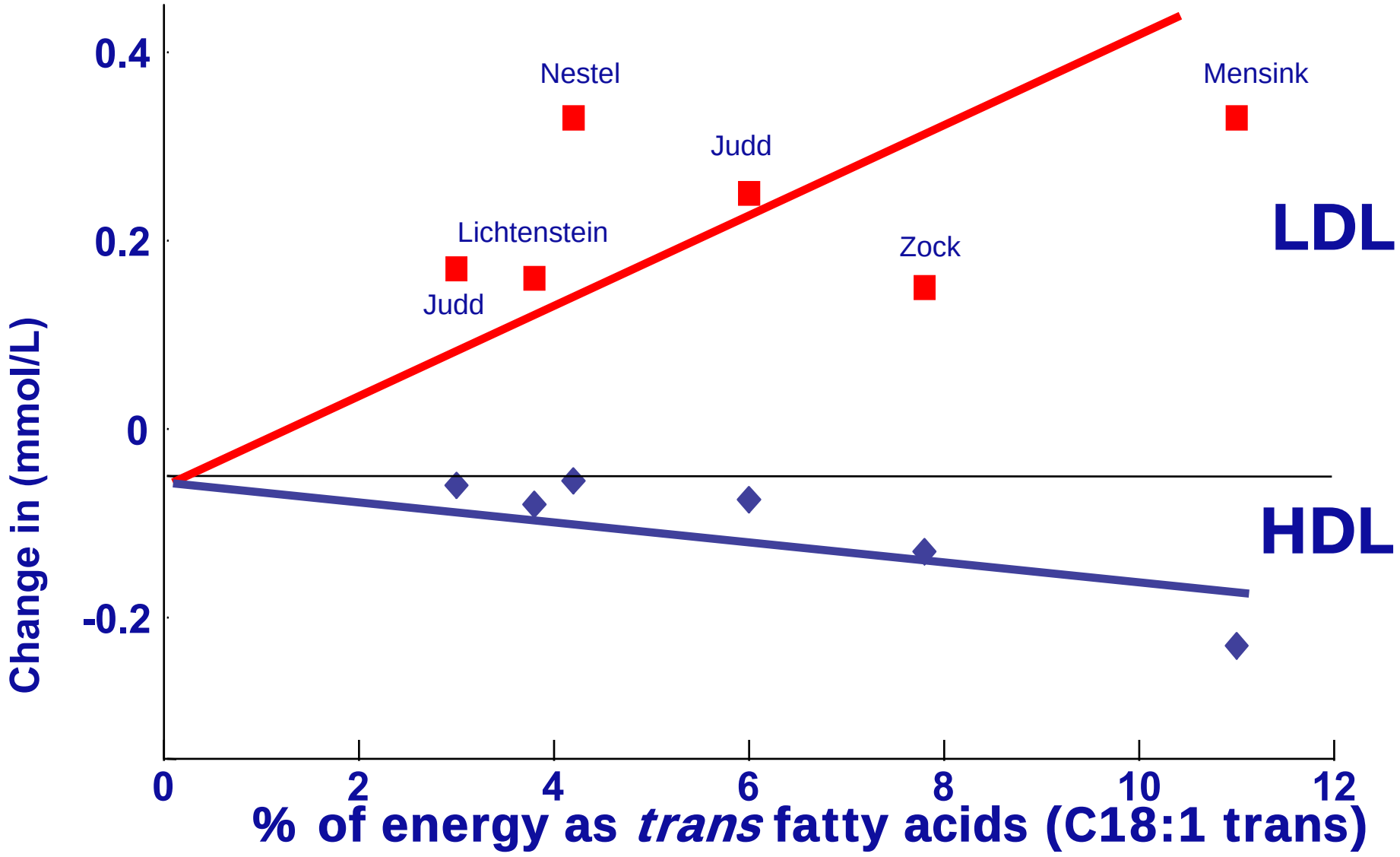


a 1% increase in calories from linolenic acid (2-3 grams/day).


Multivariate RR of CHD
1.2
0
1
0.92
0.91
0.7
0.88
0.71
0.8
1.05
0.87
0.63
0.73
0.71
0.71
0.36
0.32
0.81
0.66
0.55
0.63
0.31
Q5 (highest)
Q4
Q3
Q2
Q1 (lowest)
Quintiles of trans fat
Q1 & 2 (lowest
Q3
Q4
Q5 (highest)
Quintiles of PUFAs

Hu et al, 1997

Adverse effects of trans FAs on blood cholesterol



Zock et al Am J Clin Nutr, 1995

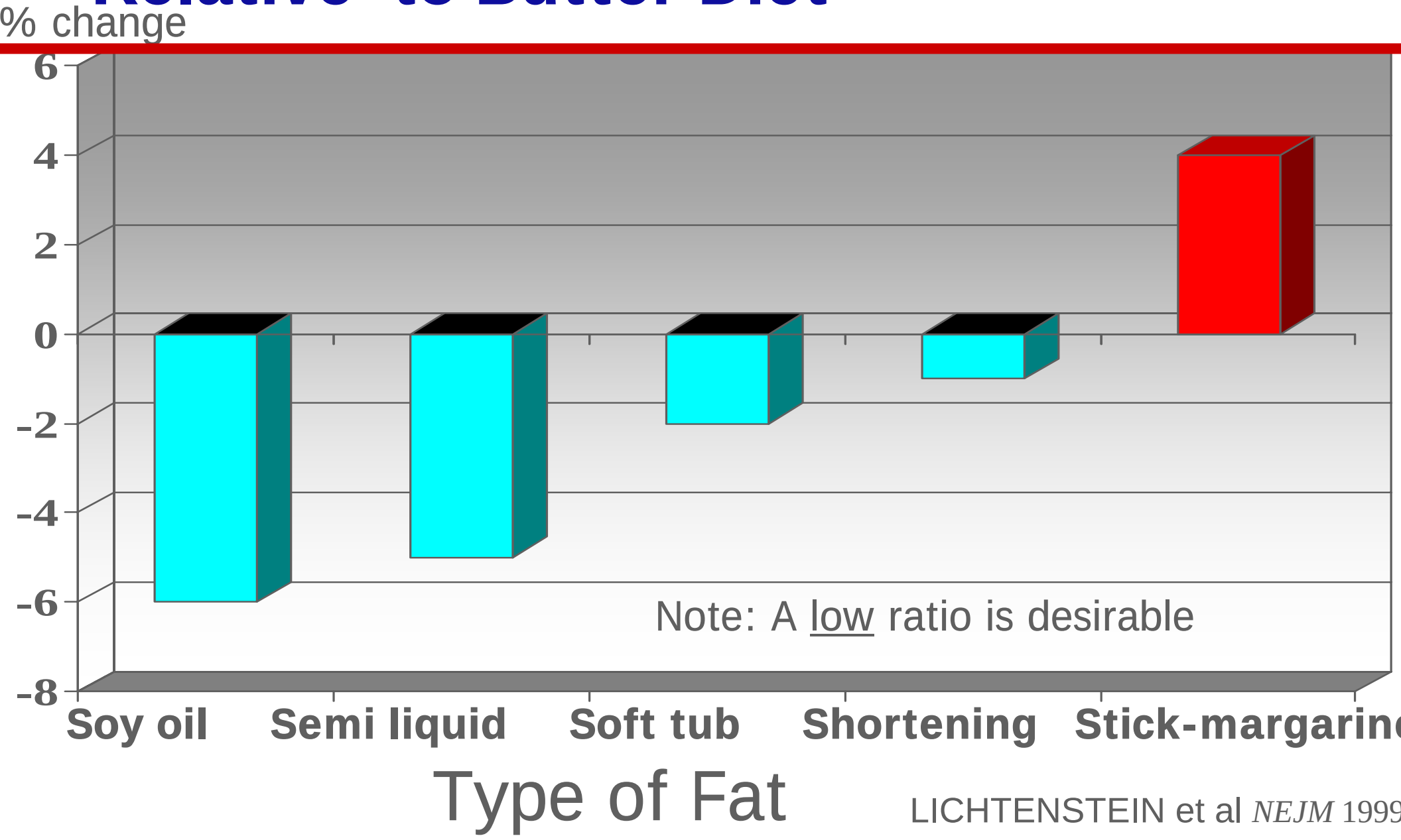
Quality of the added dietary Fat

	Trans Fat Intake (% of cal)
•10% of fat calories from butter	**1.25%**
•10% of calories from stick margarine	**6.72%**
•10% of calories from shortening	**4.15%**
•10% of calories from soft tub margarine	**3.30%**
•10% of cal. from semiliquid margarine	**0.91%**
•10% of calories from soft tub margarine	**0.55%**

Saturated fat intake was 7-8% of calories in all diets except the butter diet where it was 16.7% of calories.

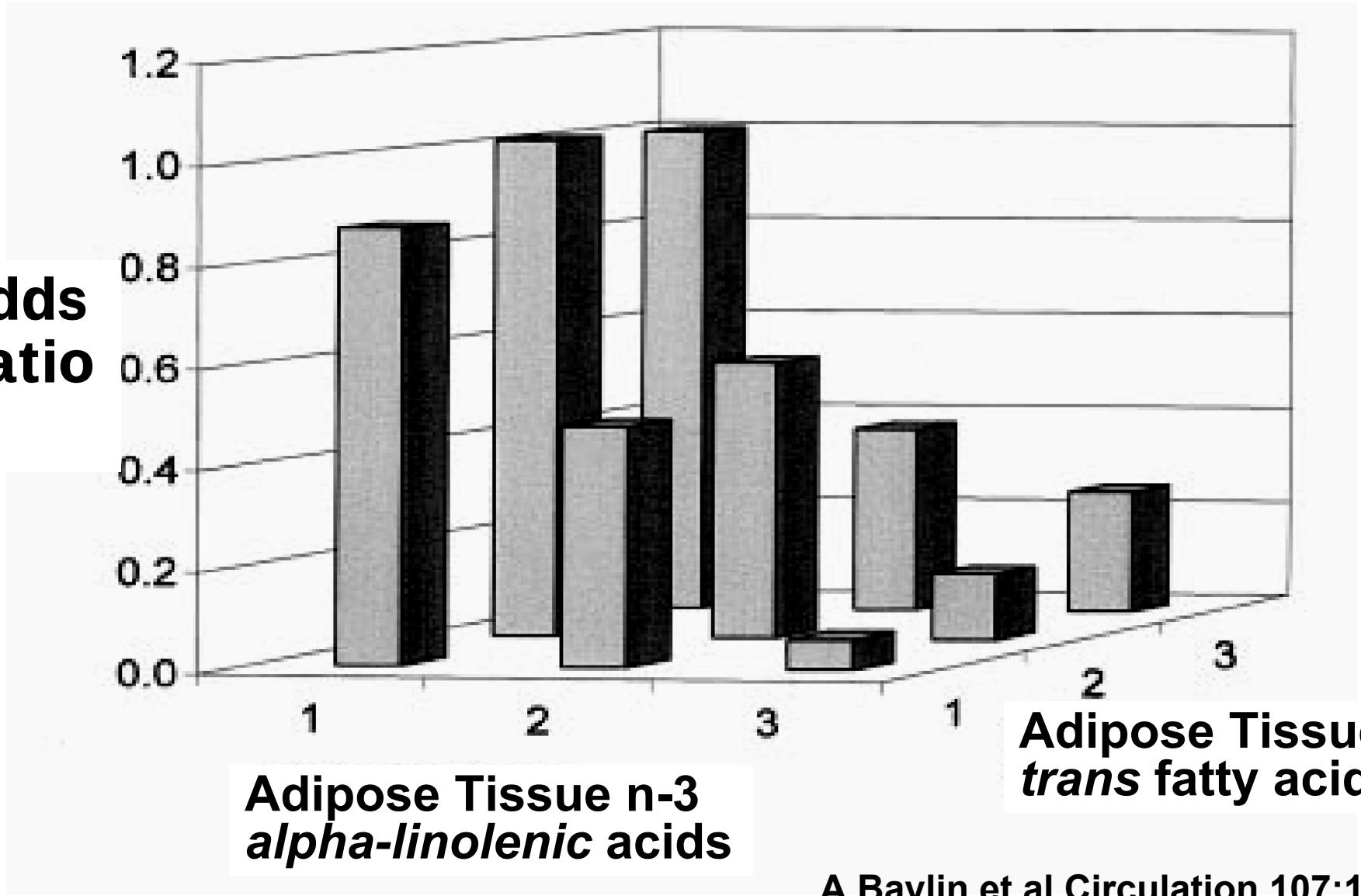
LICHTENSTEIN et al *NEJM* 1999

% Change in Total Chol / HDL Cholesterol Relative to Butter Diet



LICHTENSTEIN et al *NEJM* 1999

ORs for Risk of Nonfatal Acute MI by tercile of Linolenic & Trans FA content of Adipose Tissue in Costa Rica



A Baylin et al Circulation 107:1586-91 2003

Conclusions

1. CHD rates can be significantly reduced by dietary changes, but this is not be achieved by replacing saturated fat with carbohydrate.
2. We should reconsider recommendations to lower % of energy from fat and reduction of fat or “fatty foods” as a way to loose weight.
3. Advice about dietary fat should focus on replacement of saturated and trans fat with PUFA rich vegetable oils, including sources of n-3 fatty acids.

Changing Sats and Trans for PUFA is beneficial / with CHO it is not !

- Different types of evidence consistently show that replacing saturated and trans fats with omega-6 (LA) or omega-3 (LNA) reduce CHD and mortality. Sats replaced by CHO no benefit !!!
- The evidence also indicates that fish long chain omega-3 (EPA+DHA) reduce CHD mortality
- Omega-3 PUFA (both 18 and ≥ C20) intake is often below recommended levels and should be increased for most populations.

Recommendations for PUFA and trans

- **General international agreement on absolute levels.**
  - Total PUFA: 4-15 en% (8-10 en% most common)
  - Linoleic acid n-6 : up to 14 en% (8 en% most common)
  - Linolenic acid n-3: 0.2 to 1.0 en%
  - EPA+DHA : 200-500 mg/day
- **Trans fatty acids: as low as possible, lower than 1-2 %**
- **Some give recommendations for omega-6:omega-3 ratio, others do not. Most often ~ 5:1**
- **In many societies the intake of Omega 3 is lower and that of trans is higher than recommended**

Denmark introduced legislation, January 1, 2004, restricting the use of **industrially produced trans fatty acids to a maximum of 2 percent of the fat in any food product.**

The experience with the Danish surveillance system and the results of our study demonstrate that **this health risk can be eliminated without any noticeable effect for the consumer.**

S. Stender et al NEJM 354:15 2006

June 25, 2005

The New York Times

ARTHUR OCHS SULZBERGER JR., *Publisher*

Founded in 1851

ADOLPH S. OCHS
Publisher 1896-1935

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As Serious as a Heart Attack

Most people who pay attention to their diets know that partially hydrogenated oil contains trans fat that clogs the arteries and reduces the "good" cholesterol that helps unclog them. Beginning next year, companies must disclose trans fat amounts on food labels. But it is already clear that the Food and Drug Administration is going to have to do more to protect the public from heart-threatening fats.

One problem, detailed in a report from the Center for Science in the Public Interest, is that some companies that don't use trans fat nevertheless use other dangerous oils. Other companies, searching for trans-fat alternatives, are turning to unhealthy fats. The most popular is palm oil, a saturated fat that is widely believed to promote heart disease and whose main distinction is that it is less harmful than trans fat.

Some companies that make products with palm oil, including Newman's Own Organics popcorn and cookies, emphasize on their packages that their products are trans-fat free and note the relative advantage of palm oil over trans fat. But all this does is create the false impression that palm oil is good for you. The F.D.A. should act quickly to stop labels that could mislead consumers. The agency should also encourage the use of healthier alternatives like certain safflower and sunflower oils and promising new blends.

The ultimate aim, however, should be to end the widespread use of partially hydrogenated oils. As things now stand, the F.D.A. acknowledges that trans fats are unhealthy at any level, and yet maintains that the partially hydrogenated oils that contain them are basically safe. The agency can't have it both ways. Public health would be greatly improved if the F.D.A. prohibited their use.

To the Editor:

Re "Justices Uphold Taking Property for Development" (front page, June 24):

The Supreme Court's decision involving New London, Conn., granting [illegible] employers to discriminate against older workers, and they continue to do so even though there have been numerous ideas put forth about how important it is to overcome that prejudice ever since the implications of the coming retirement of the baby boomers have become clear.

Those of us who studied the subject devoted a great deal of effort to explaining ways to keep older workers in the work force. But corporate America does not seem interested, and the barriers to securing rewarding employment after 50 remain in place and show no signs of disappearing. So perhaps Mr. Tierney can tell us how to get business to listen.

BEVERLY GOLDBERG
New York, June 21, 2005

The writer is vice president and director of publications at the Century Foundation and the author of a book about the graying of the work force.

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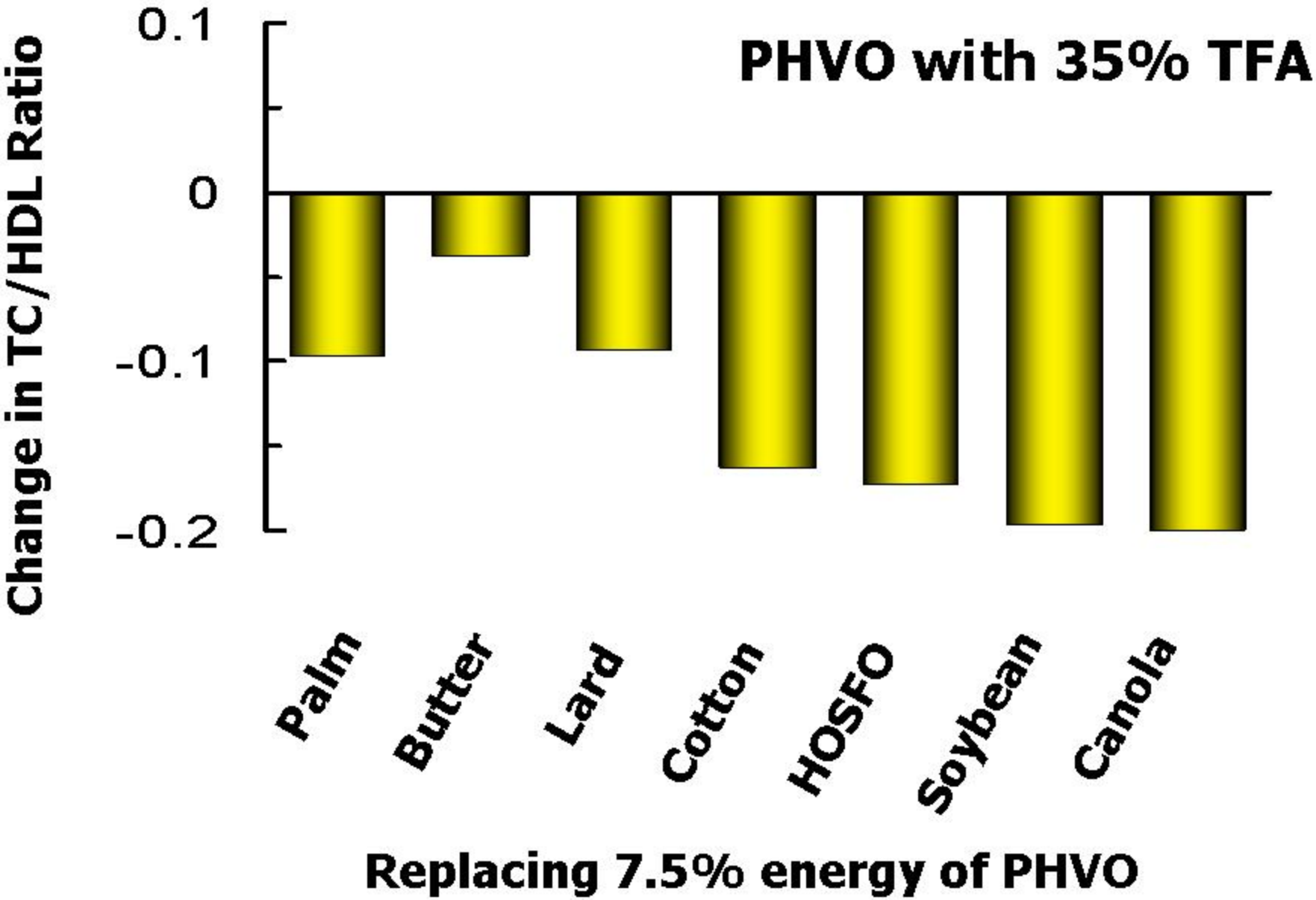
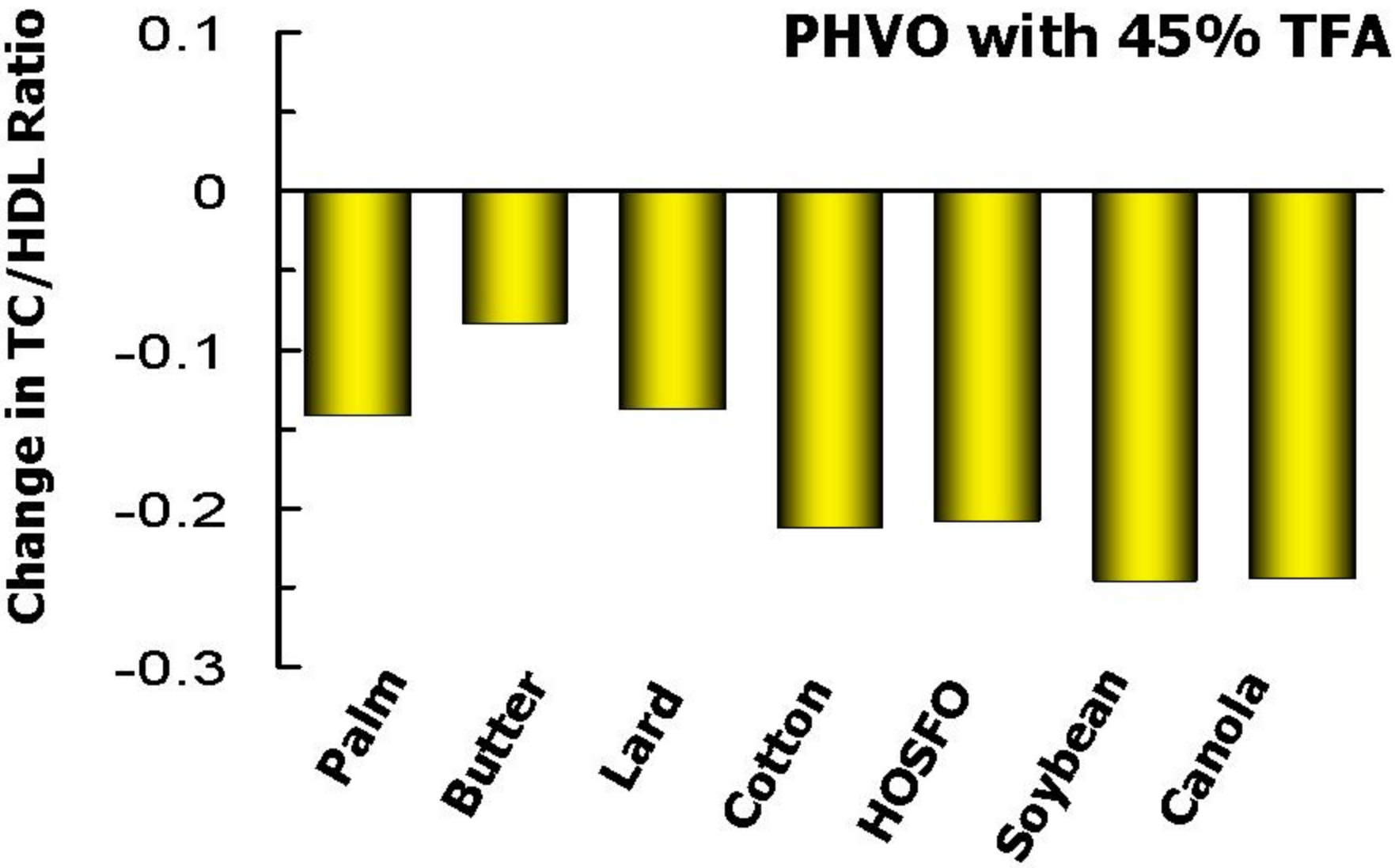


Mozaffarian D et al ECJN 2008



Mozaffarian D et al ECJN 2008

Ruminant or industrial sources of *trans* fatty acids: public health issue or food label skirmish?[1,2]

Walter Willett and Dariush Mozaffarian

***Am J Clin Nutr Marzo* 2008;87:515– 6.**

- The dairy and beef industries have reason to wish that ruminant TFA would be excluded in the *trans* fat category on food labels or in regulations limiting TFA intakes.

- Not to include all sources of TFA on food labels would, however, create regulatory complexity, especially for prepared dishes with mixed animal and vegetable constituents. **At present there is no compelling evidence to exclude natural TFA from the total TFA on food labels.**

- To its credit, **the food industry appears to be generally taking advantage of the costs and effort of reformulation to make an overall healthier product;** avoiding replacing one problem with another.

Annie Motard-Bélangeret al Am J Clin Nutr 2008;87:593–9.

Ruminant TFAs

Variable **TFAs % E**	Control **0.8 %**	Moderate **1.5 %**	High **3.7 %**	iTFA **3.7 %**
Weight (kg)	74.2 ± 8.8[4]	74.3 ± 8.7	74.6 ± 8.6	74.6 ± 9.3
Waist girth (cm)	81.1 ± 9.0	80.7 ± 8.7[5]	81.3 ± 8.6	81.2 ± 8.9[6]
Cholesterol (mmol/L)	4.77 ± 0.93	4.72 ± 0.88	4.92 ± 0.98[6]	4.88 ± 0.95[6]
VLDL-C (mmol/L)[7]	0.21 ± 0.16	0.22 ± 0.15	0.23 ± 0.18	0.24 ± 0.22
LDL-C (mmol/L)	3.27 ± 0.80	3.22 ± 0.83	3.47 ± 0.90[5,6]	3.42 ± 0.89[6]
HDL-C (mmol/L)	1.25 ± 0.24	1.28 ± 0.28	1.22 ± 0.26[6]	1.23 ± 0.24
HDL_2-C (mmol/L)	0.59 ± 0.21	0.59 ± 0.21	0.54 ± 0.19[5]	0.56 ± 0.18
HDL_3-C (mmol/L)	0.66 ± 0.14	0.69 ± 0.13	0.68 ± 0.11	0.67 ± 0.13
TG (mmol/L)[7]	0.98 ± 0.45	0.95 ± 0.41	0.99 ± 0.43	0.97 ± 0.54
ApoB (g/L)	0.94 ± 0.23	0.91 ± 0.21	0.96 ± 0.23[6]	0.94 ± 0.22[6]
ApoA1 (g/L)	1.51 ± 0.19	1.53 ± 0.18	1.49 ± 0.18[6]	1.52 ± 0.18
Total/HDL-C[7]	3.97 ± 1.16	3.86 ± 1.16	4.23 ± 1.32[6]	4.16 ± 1.39[6]
LDL-C/HDL-C	2.75 ± 1.00	2.67 ± 1.01	3.02 ± 1.15[5,6]	2.94 ± 1.17
ApoB/apoA1[7]	0.63 ± 0.21	0.60 ± 0.17	0.65 ± 0.19	0.63 ± 0.17
CRP (mg/L)[7]	0.99 ± 1.49	0.79 ± 1.37	0.72 ± 0.69	0.74 ± 0.96

Outcomes to assess health effects of dietary FA

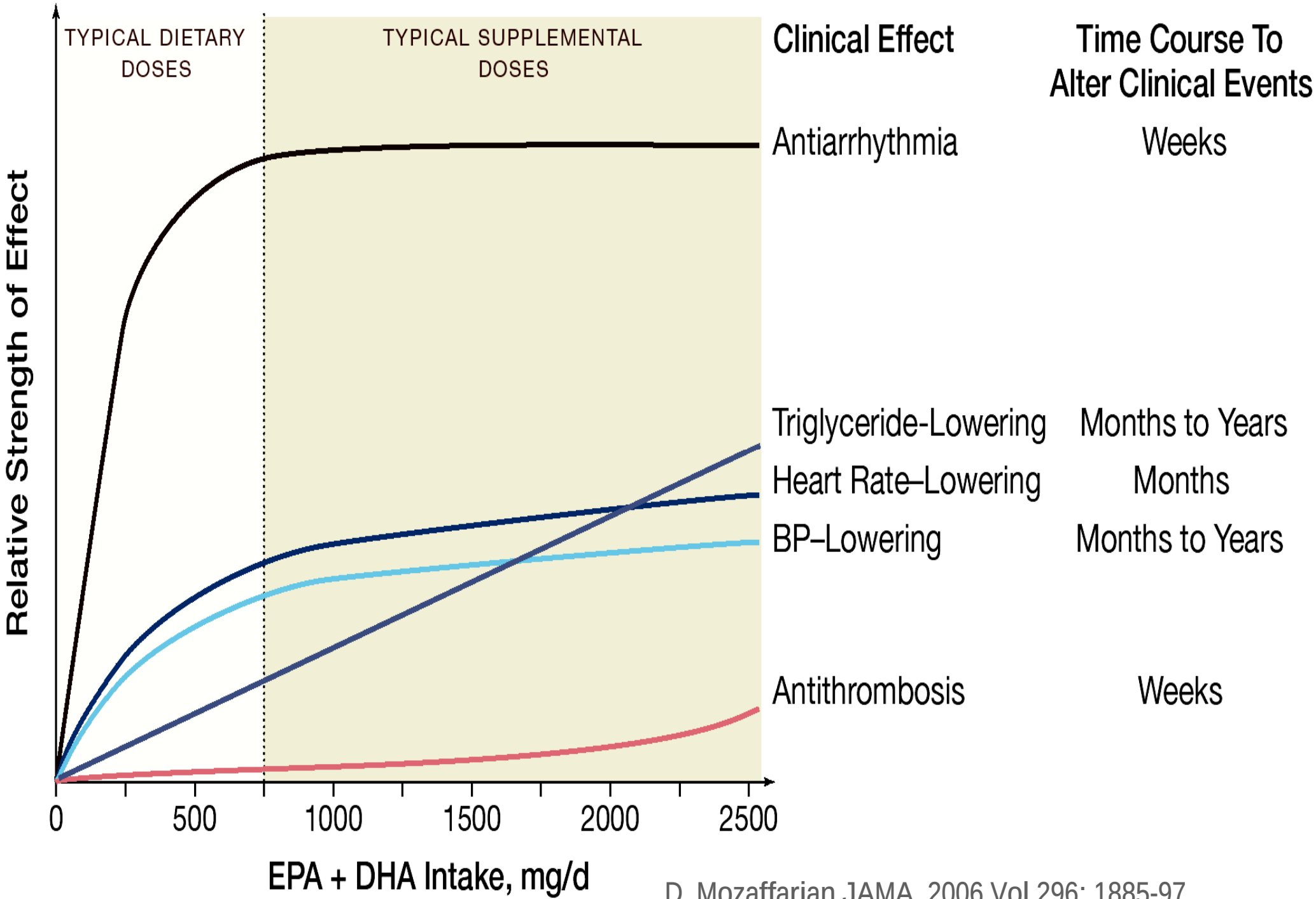
- **Lipoproteins and Cholesterol (LDL, HDL, Lp(a), TG)**
- **Coronary heart disease events Fatal and non Fatal**
- ***Post prandial lipemia***
- ***Glucose/Insulin responses; insulin sensitivity***
- **Cancer outcomes (Colo rectal, prostate, breast, edometrial, ovarian)**
- ***Oxidative Stress***
- ***Inflammation***
- **Blood Pressure**
- **Arterial Stiffness, Endotelial Function**
- ***Fibrinogen, Factor VII, Fibrinolytic activity***

Consumption of Fish or Fish Oil: Effects on CVDs

Outcome	Clinical Effect	Strength of Evidence	Comment
CHD mortality			
CHD death	≈ 35% decrease	Strong	Probable threshold of effect—most risk reduction occurs with modest intake (≈ 250 mg/d EPA + DHA), with little additional benefit with higher intakes[2-4,6-17,45-51]*
Sudden death	≈ 50% decrease	Strong	
Ischemic stroke	≈ 30% decrease	Moderate	Strong evidence from prospective cohort studies[53,54]; no RCTs
Nonfatal CHD			
Nonfatal MI	Modest benefit?	Equivocal	Possible benefits at very high intakes (≈ 2 g/d n-3 PUFAs)[17,50]
Progression of atherosclerosis	Modest benefit?	Equivocal	Mixed results in cohort studies[55] and RCTs[56-58]
Postangioplasty restenosis	Modest benefit?	Equivocal	Possible benefits in a meta-analysis of RCTs[59]
Recurrent ventricular tachyarrhythmias	Modest benefit?	Equivocal	Mixed results in 3 RCTs[60-62]
Atrial fibrillation	≈ 30%+ decrease	Limited	Mixed results in 2 cohort studies[63,64]; benefit in 1 RCT[65]
Congestive heart failure	≈ 30% decrease	Limited	Benefit in 1 prospective cohort study[66]

Abbreviations: CHD, coronary heart disease; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; MI, myocardial infraction; n-3 PUFA, n-3 polyunsaturated fatty acid; RCT, randomized clinical trial.
*See Figure 1.

D. Mozaffarian JAMA, 2006 Vol 296: 1885-97



D. Mozaffarian JAMA, 2006 Vol 296: 1885-97

FAO/WHO 2008 : strength of evidence on risk of developing CHD (Fatal & Non fatal events)

Evidence	Decreased risk	No relationship	Increased risk
Convincing	**Fish and fish oils (EPA + DHA)**	Vitamin E	**Trans fatty acids**
Probable	***α-Linolenic acid 18:3 n-3*** ***Linoleic acid 18:2n-6*** **Plant sterols/stanols**	***Oleic 18:1 n-9 MUFA***	***Myristic & palmitic 14:0 16:0*** **Dietary cholesterol**
Possible	Mediterranean/Asian diets Soy products Nuts (unsalted)		***Fats rich in Stearic acid 18:0*** Impaired fetal nutrition Beta-carotene supplement

It gets worse: Contains partially hydrogenated vegetable oil & saturated fat.