

**Conference Summary
or
Another Inconvenient Truth**

Michael Clearfield DO
Dean Touro University College of
Osteopathic Medicine

Obesity Summary Objectives

- Review the previous presentations and how they underscore the need to understand obesity and its ramifications to our future well being as a global society
- Understand potential interaction between obesity, metabolic syndrome and global warming
- Understand, how we as the American College of Osteopathic Internists can make a difference

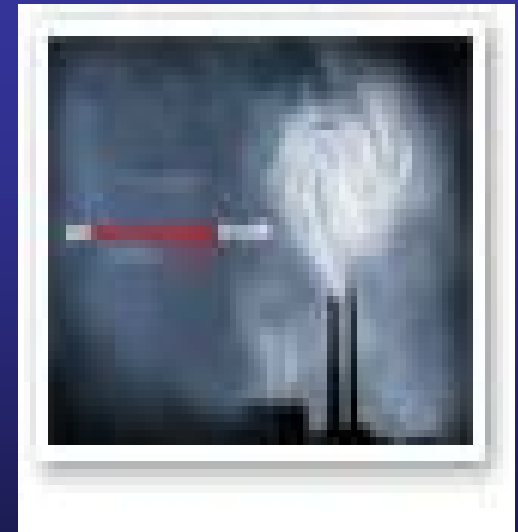
Obesity Summary

- Is bad
- Is prevalent
- Is increasing
- Is hard to treat

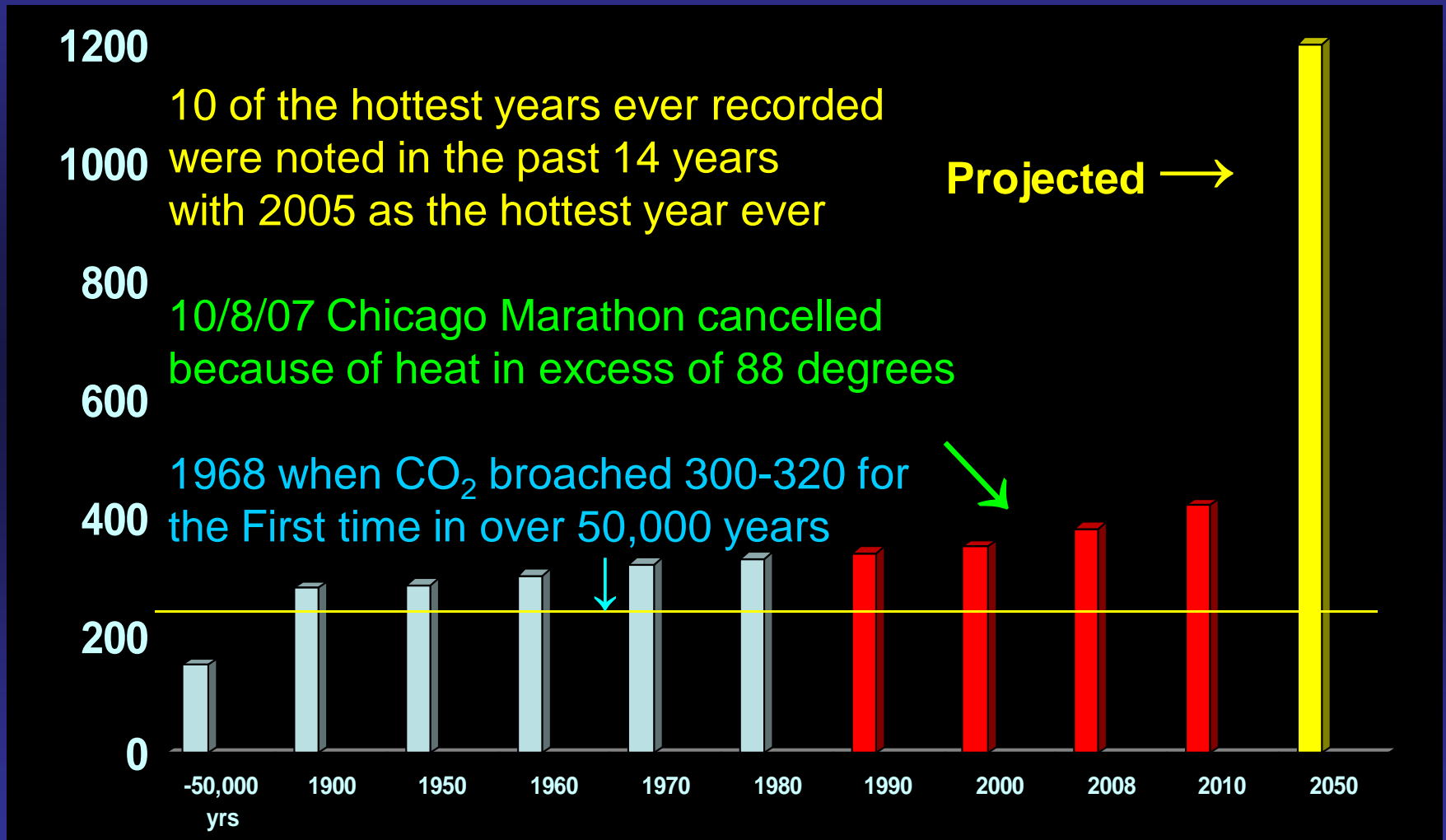
Global Warming

An Inconvenient Truth

- U.N. Intergovernmental Panel on Climate Change (IPCC)
- 600 scientists from 40 countries
- Concluded for the first time that evidence of the earth's rising temperature was “unequivocal” & this warming was greater than 90% due to human activity
- Even if all greenhouse-gas emissions ended today, the earth would continue to warm through the rest of the century because of the amount of carbon already in the atmosphere



CO₂ Content in the Atmosphere



Another Inconvenient Truth

- Over the past 40 years despite advances in coronary artery bypass & percutaneous intervention strategies, advanced therapies for myocardial infarction, unstable angina and heart failure...

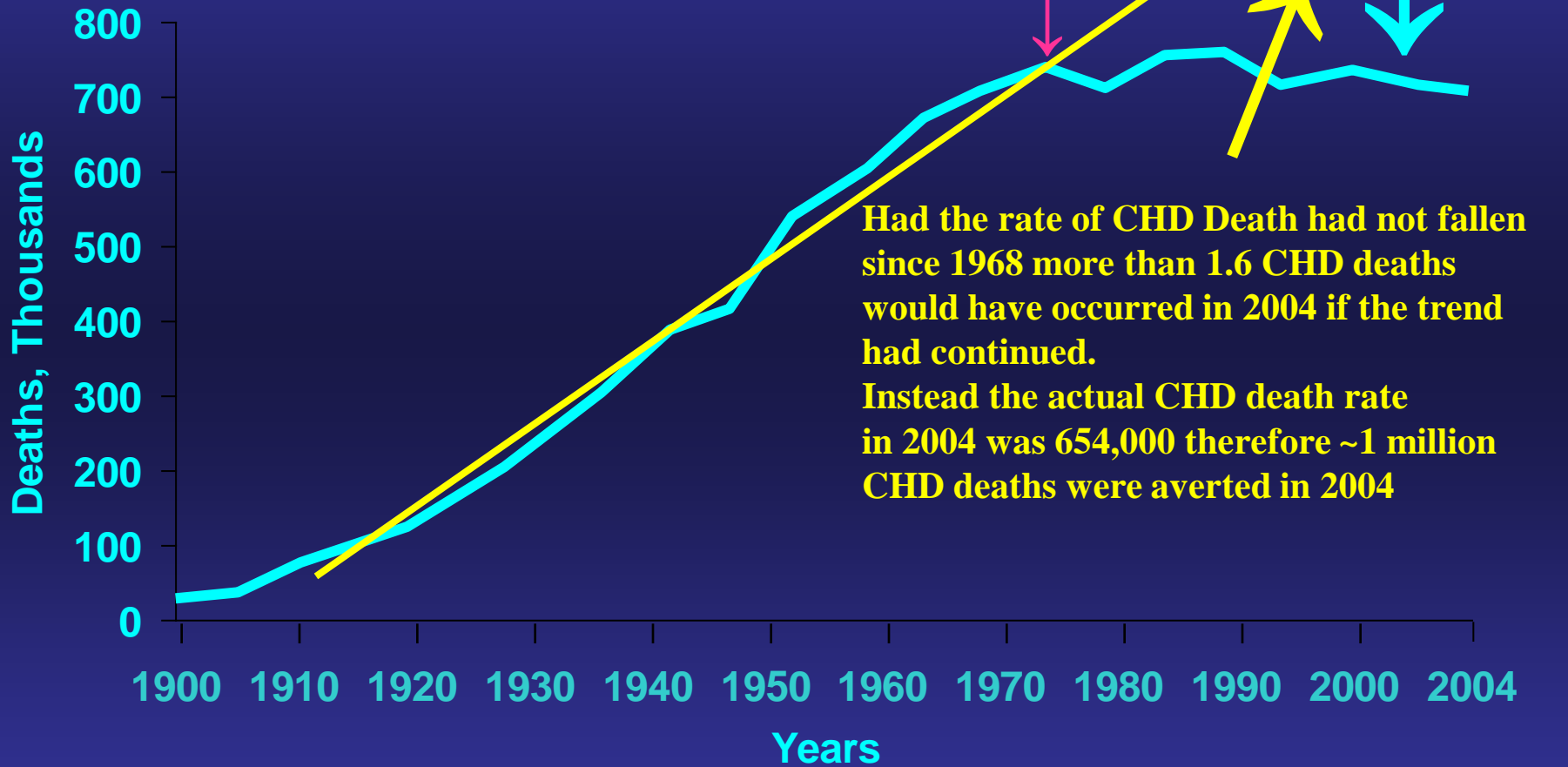
as well as age adjusted reductions in total cholesterol, systolic blood pressure, smoking prevalence and physical inactivity...

Cardiovascular disease still remains the number one killer of adults in the United States

Deaths From Heart Disease

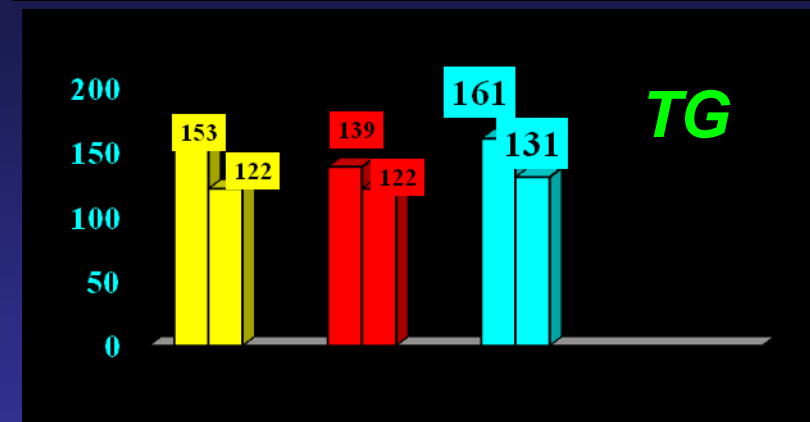
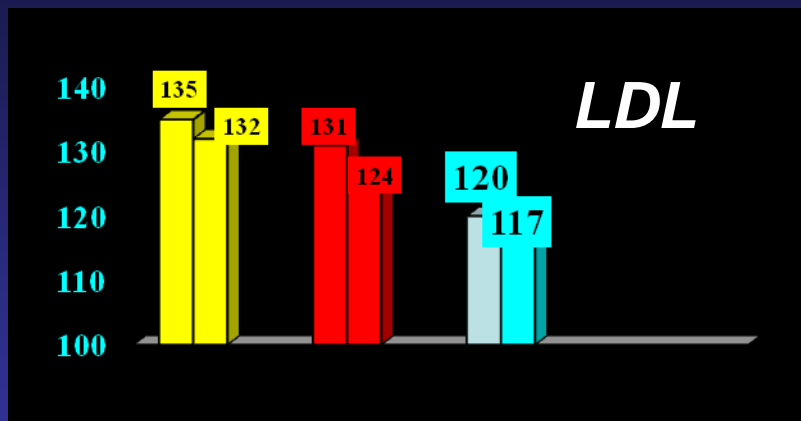
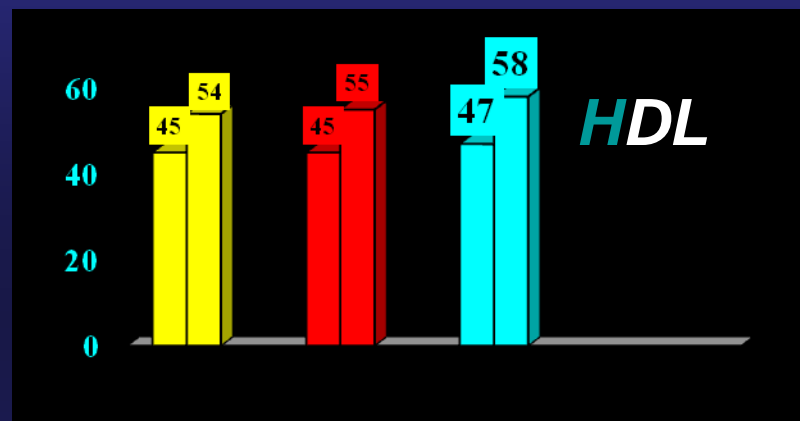
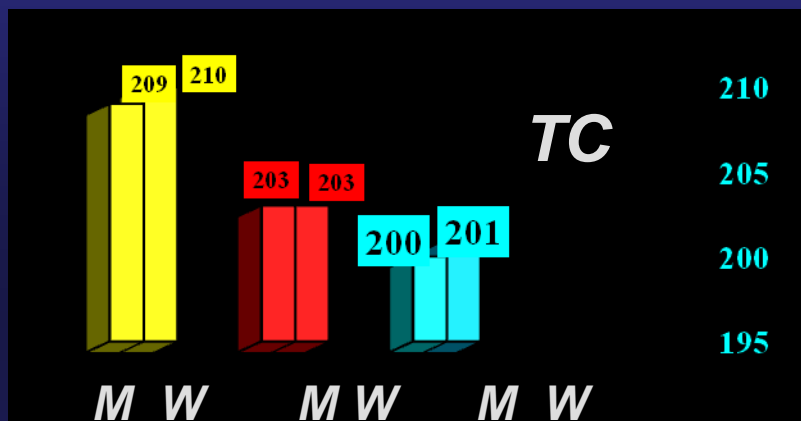
Progress since 1968

Peak Rate Noted in 1968



American Heart Association (AHA). *Heart Disease and Stroke Statistical—2004 Update*. Dallas, Tex: AHA; 2003. Available at: www.americanheart.com/presenter.jhtml?identifier=3018163. Accessed January, 2006.

30 Year Lipid Trends (Men/Women) NHANES (1976-2006)



NHANES II 1976-1980
 NHANES III (1988-1994)
 NHANES IV (1999-2006)

***Although* Improvements in lipids, BP,
cigarette smoking CVD still remains
number one killer in America**

LDL lowered to 120 (m) & 117 (w)

HDL raised to 47 (m) & 58 (w)

***This still leaves 69% with LDL > 100 mg/dl
and 19% with HDL < 40 mg/dl***

***Besides further reductions in LDL and further
increases of HDL what else should be
considered to lower the residual CVD risk,
such that CVD is no longer the number one
killer of Americans?***

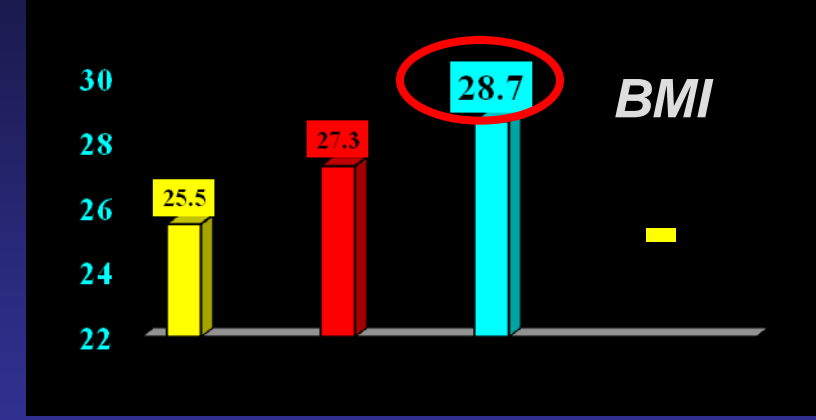
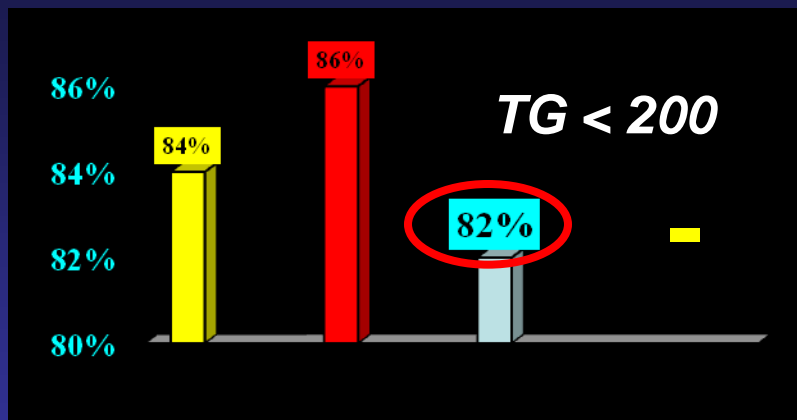
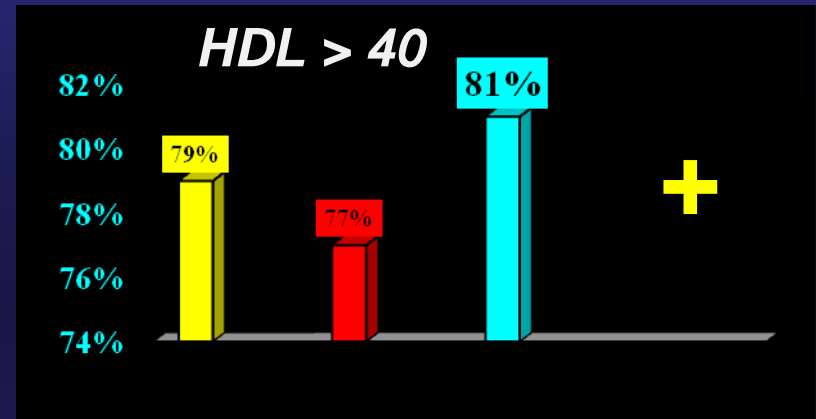
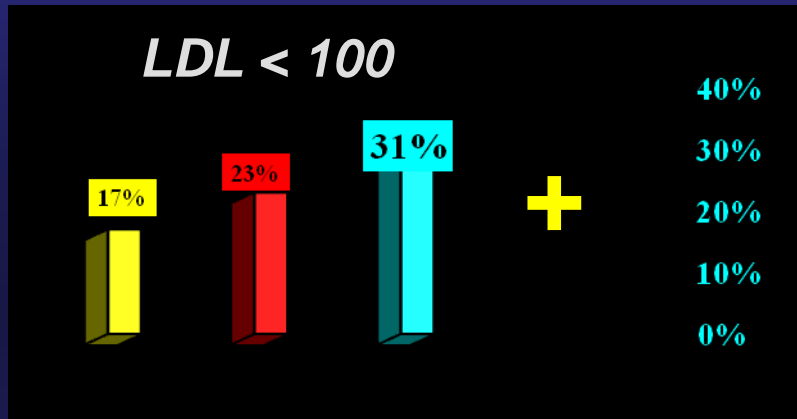
First Hint

Obesity +
Metabolic Syndrome

ATP III: Components of Metabolic Syndrome

- **Abdominal Obesity**
- Atherogenic Dyslipidemia
- Elevated Blood Pressure
- Insulin Resistance \pm glucose intolerance
- Pro-inflammation

30 Year Lipid Trends from NHANES (1976-2006)



Cohen AJC 2010;106:969

NHANES II 1976-1980
 NHANES III (1988-1994)
 NHANES IV (1999-2006)

Silent Progression of Vascular Disease: Who is at Risk?

20th Century



21st Century



1/3 die
before
patients

Happy?



....self induced diseases amplify the genetics of atherosclerosis

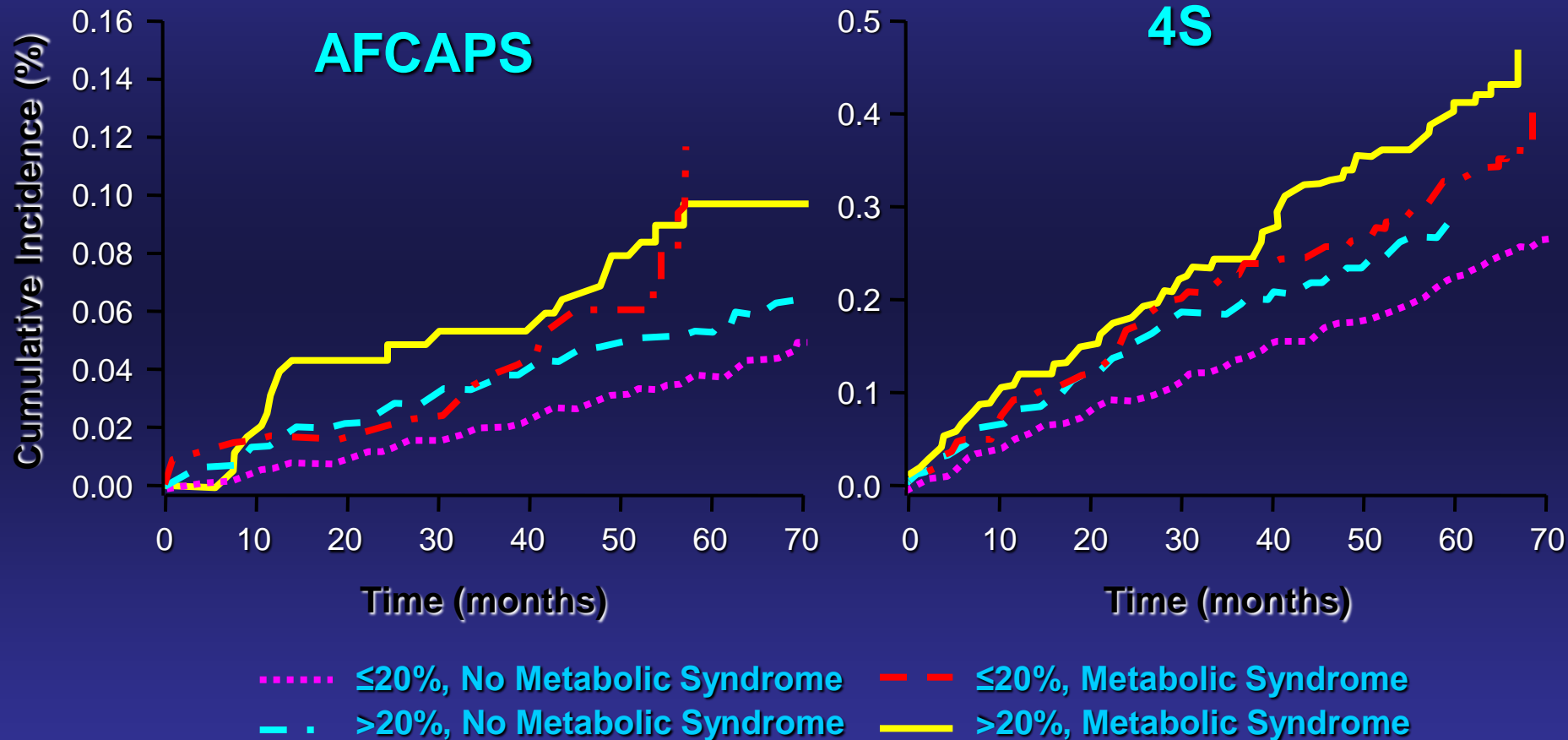
International Diabetes Federation Classification of Metabolic Syndrome

Endorsed by 2009 Canadian Guidelines

**Central obesity (waist circumference) +
Plus two of the following factors:**

- TG > 1.7 mmol/L (150 mg/dl)
- HDL-C < 1.03 mmol/L (40 mg/dl) men
- HDL-C < 1.3 mmol/L (50 mg/dl) women
- BP > 130/85 or treatment for hypertension
- FPG > 5.6 mmol/L (100 mg/dl)

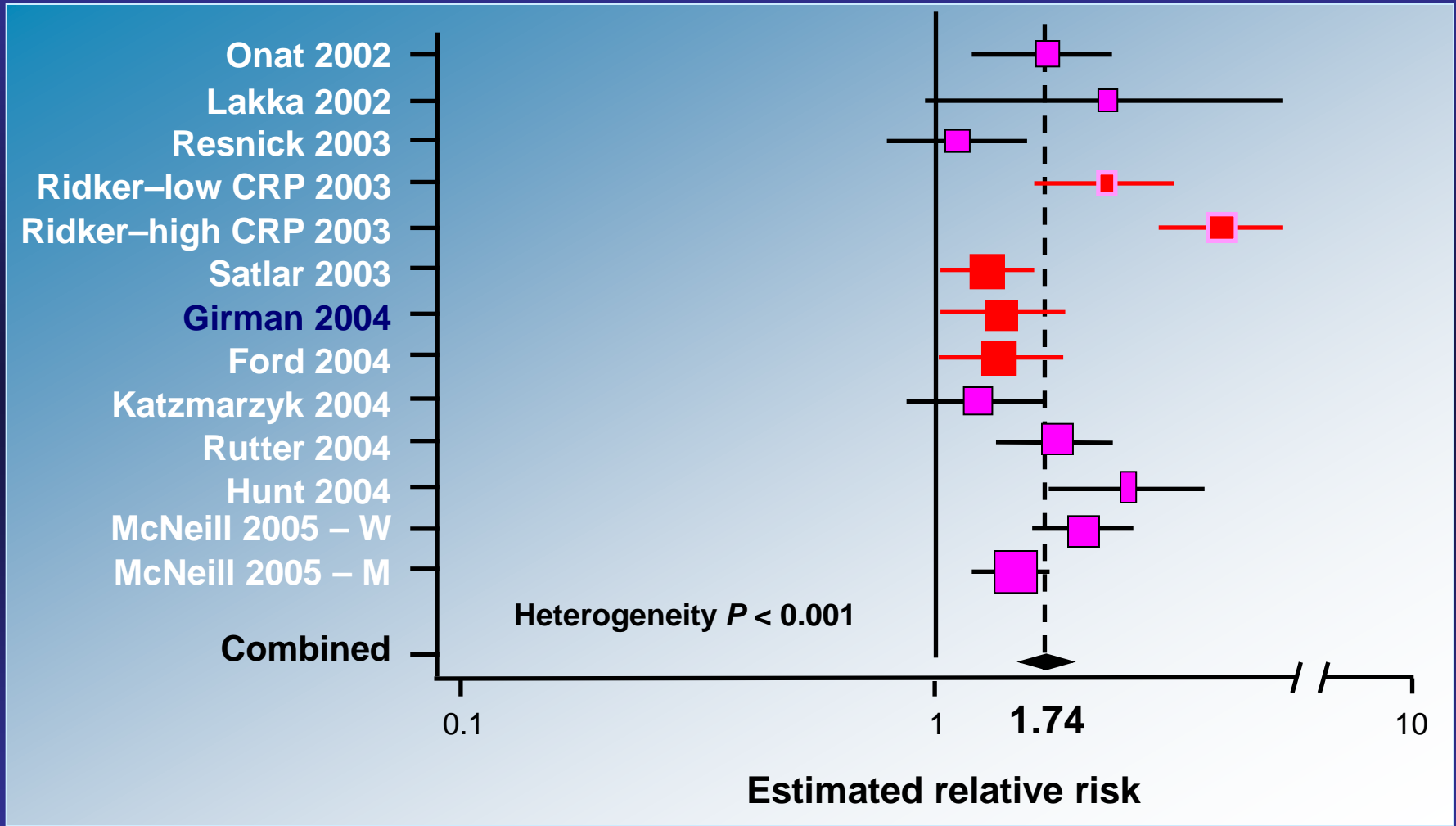
Metabolic Syndrome Increases Risk for MACE Regardless of Framingham Estimated Risk



MACE = major adverse coronary events

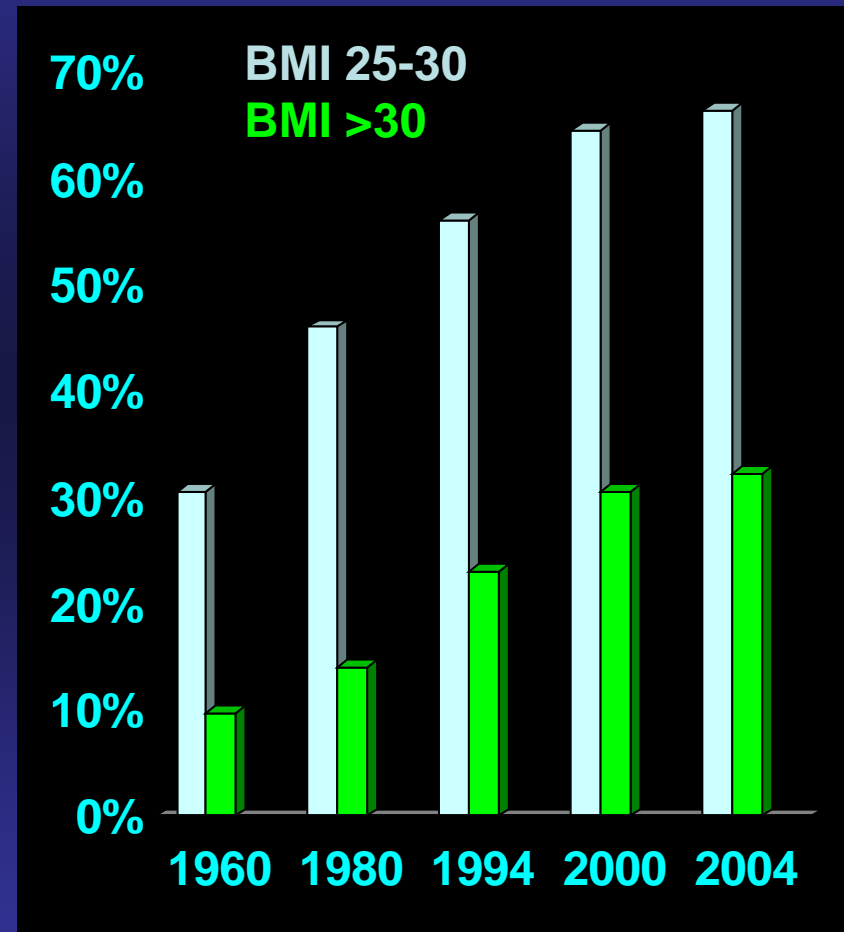
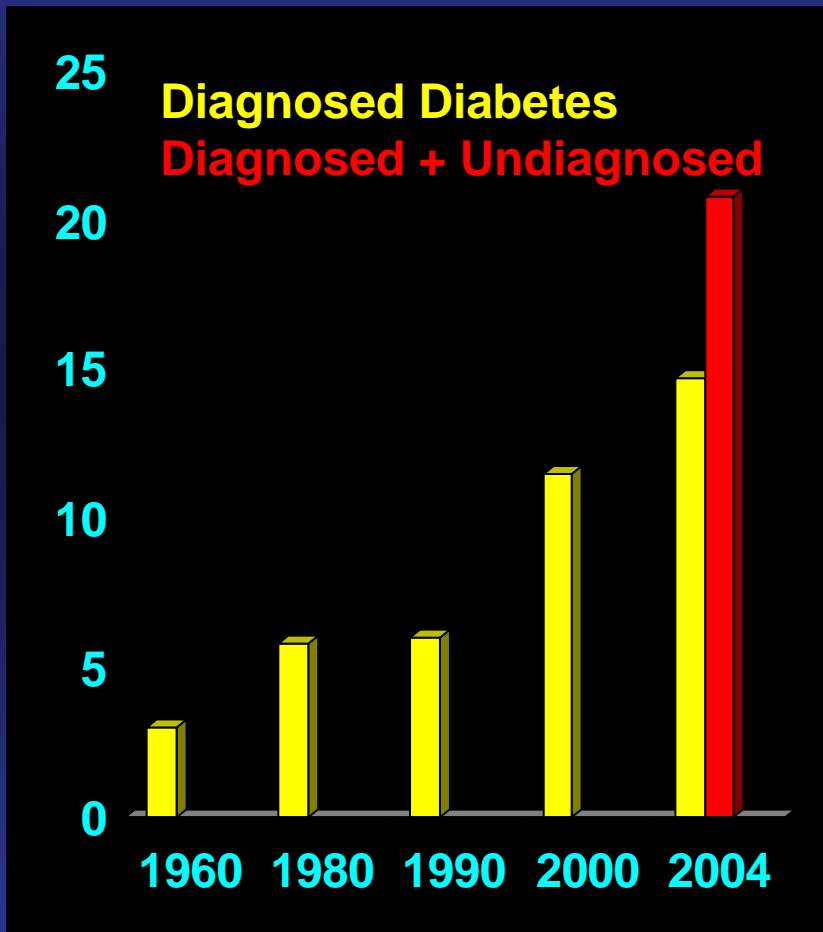
Girman CJ, et al. Am J Cardiol. 2004;93:136-141.

Metabolic Syndrome Increases CV Risk

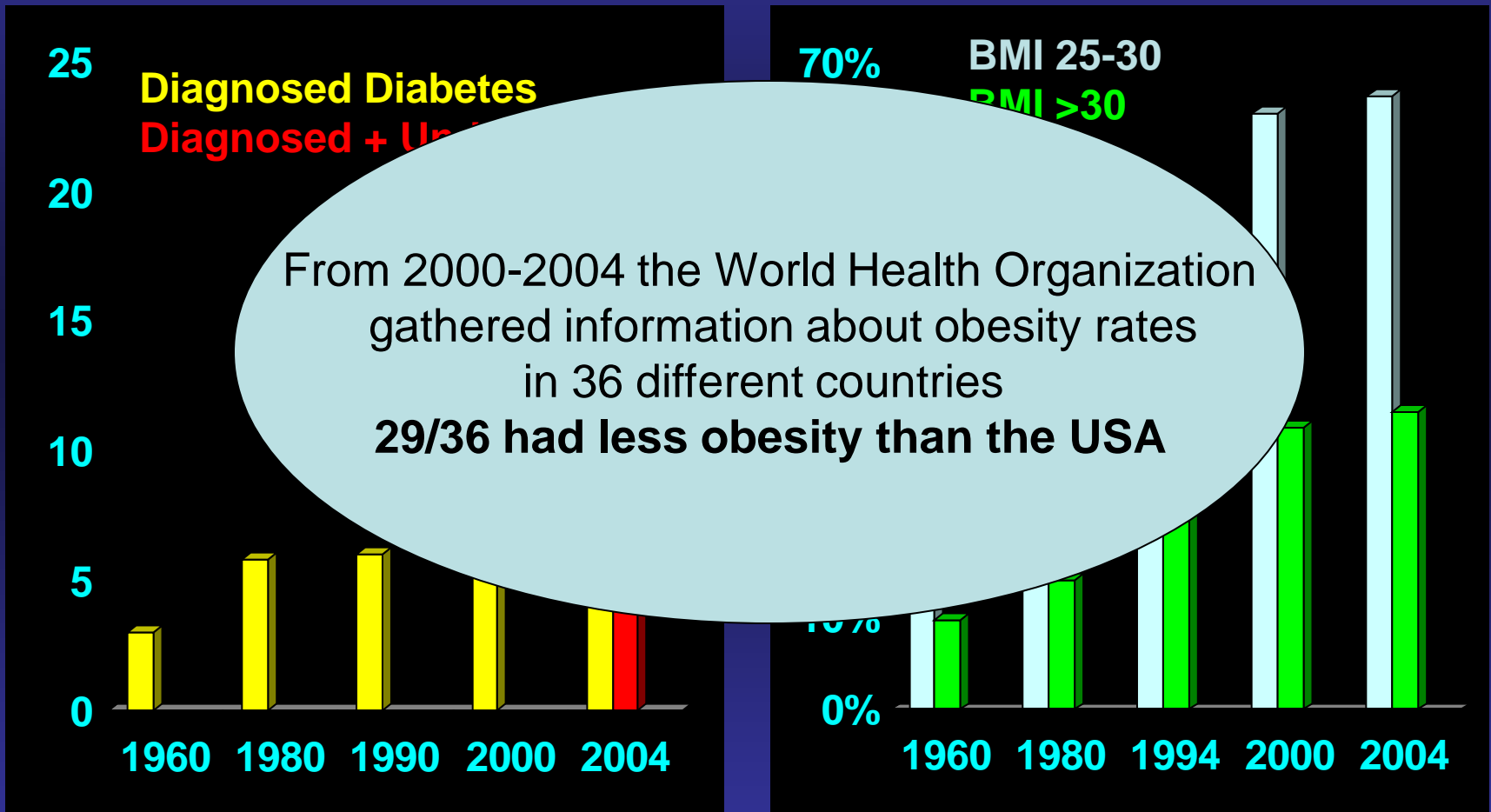


■ Original ATP III definition
■ Modified ATP III definition
 W = Women; M = Men

Twin Epidemics of Obesity and Diabetes



Twin Epidemics of Obesity and Diabetes

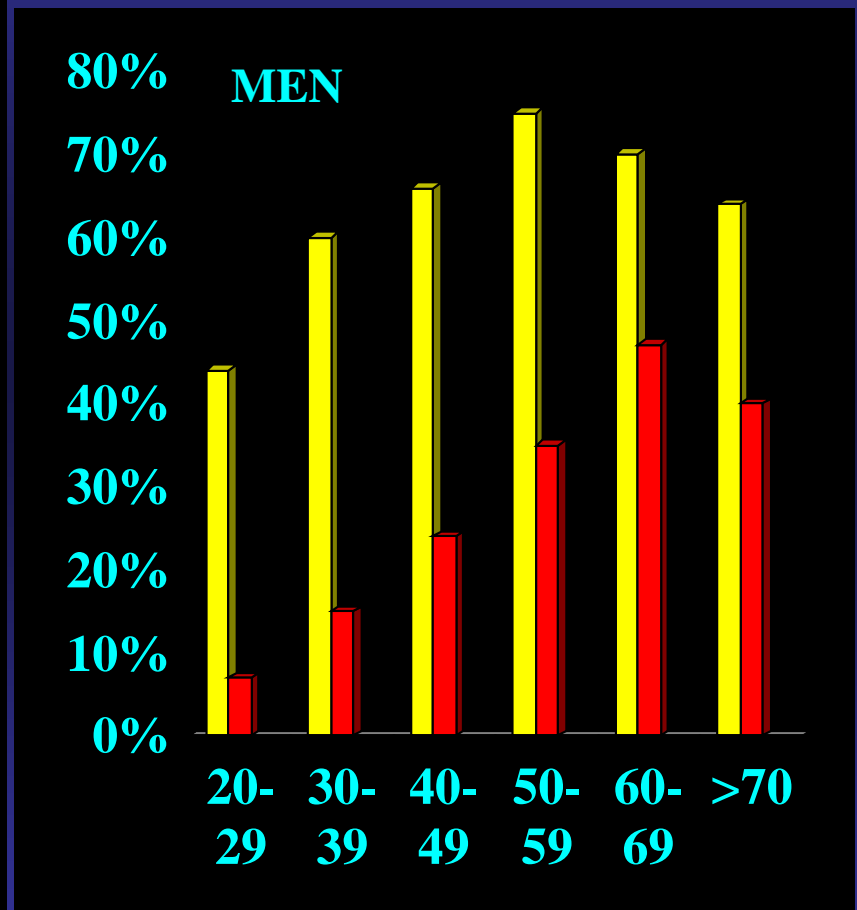
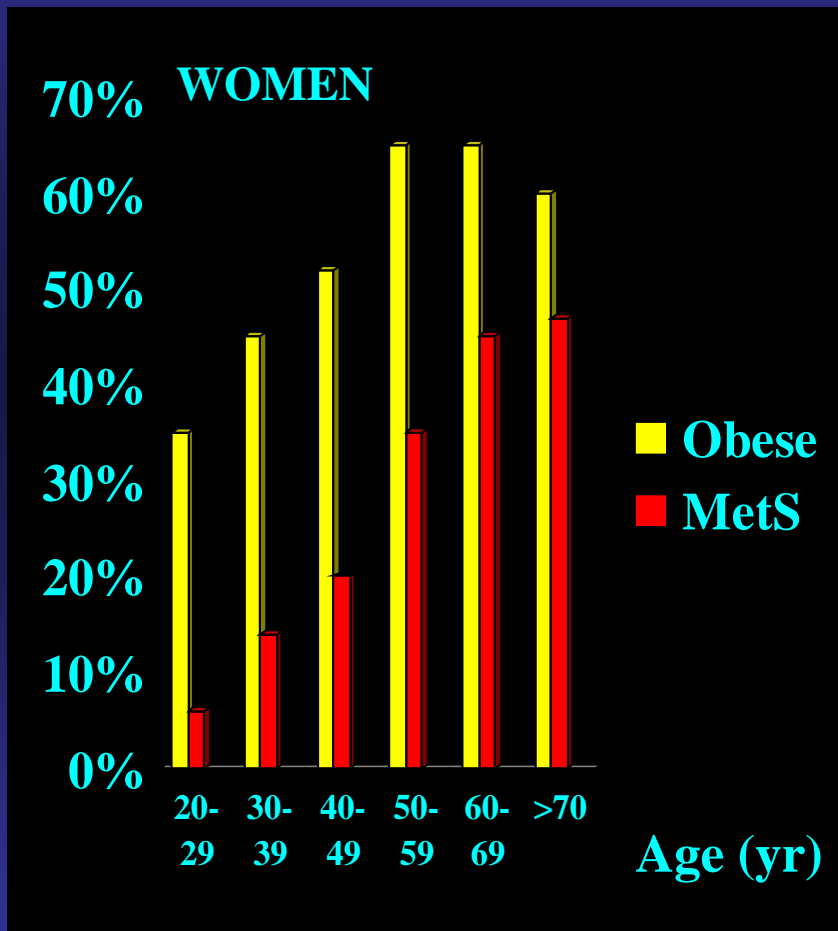


National Center for Chronic Disease Prevention National Center for Health Statistics

Flegal JAMA 2002;288:1723 Flegal Int J Obesity 1998;22:39

World Health Organization, World Health Statistics 2005 <http://www3.who.int/statistics/>

Twin Epidemics: Parallels in Prevalence Overweight/Obesity and Metabolic Syndrome



Is all Obesity the Same?

Or...The importance of identifying the overweight person who will benefit most from weight reduction.

- Is insulin resistance the Key?
- Insulin resistance is associated with increased risk DM, HBP, CVD
 - 5% people with BMI < 25 have IR
 - 22% people with BMI 25-30 have IR
 - 60 % people with BMI > 30 have IR
- Conclusion: Insulin Resistance found in 5% of normal weight and not found in 40% of obese people. Therefore insulin resistance is more than weight alone.

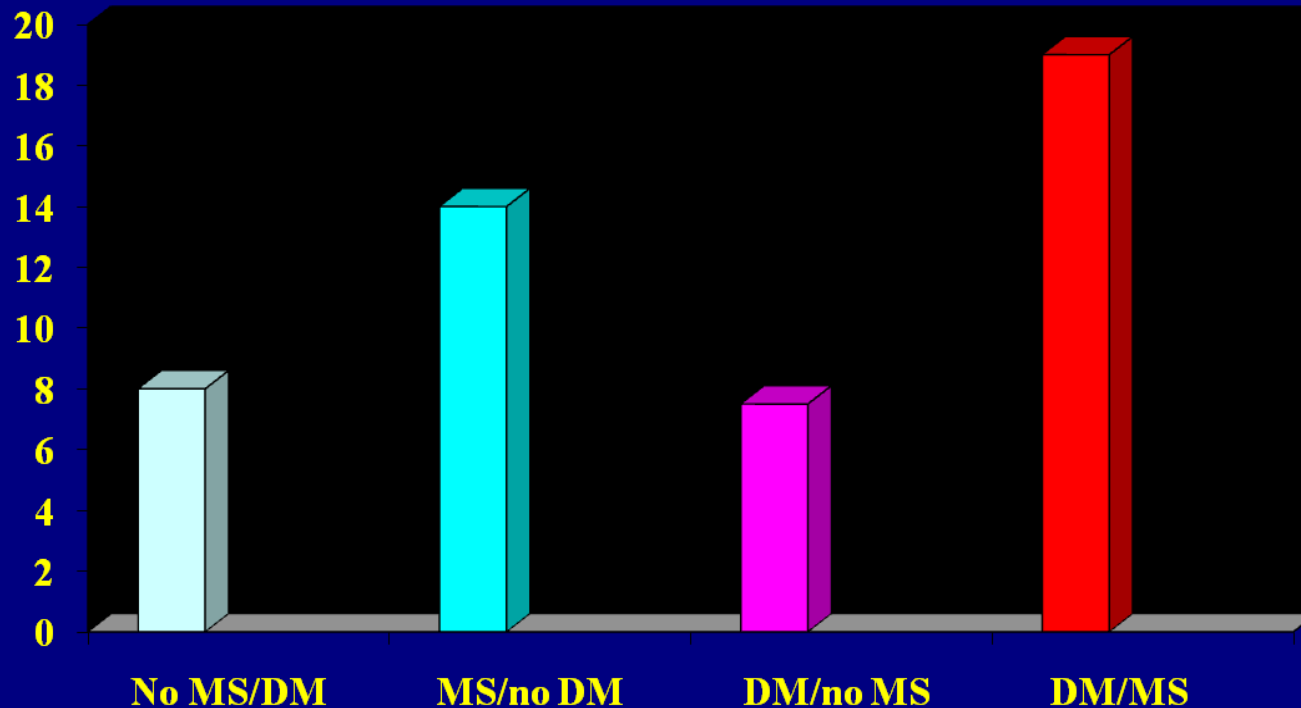
Thrifty Gene

- Thrifty gene (*or pig out gene*) 825T allele of B3 subunit of heteromeric G-protein (*GNB3*)
- Associated with development of obesity and metabolic syndrome in absence of regular exercise
- Highest thrifty gene prevalence in people of African descent (80-90%), next Asian (50%) and lowest in Caucasians (30%)
- Furthermore Asian Indians are predisposed to the deadly lipid tetrad (↑small dense LDL, ↑TG, ↑Lp(a), ↓HDL)

Prevalence Metabolic Syndrome in Adults aged > 50

- Prevalence of metabolic syndrome in those with type II DM is 87%
- Individuals with diabetes but without metabolic syndrome had a similar prevalence of CHD as those with neither DM or metabolic syndrome
- Individuals with metabolic syndrome without diabetes had a higher CHD prevalence than those with DM alone
- Those with both DM and metabolic syndrome represented 15% of the population and had highest prevalence of CHD

CHD Prevalence in Adults > 50 yrs in Diabetes and/or Metabolic Syndrome



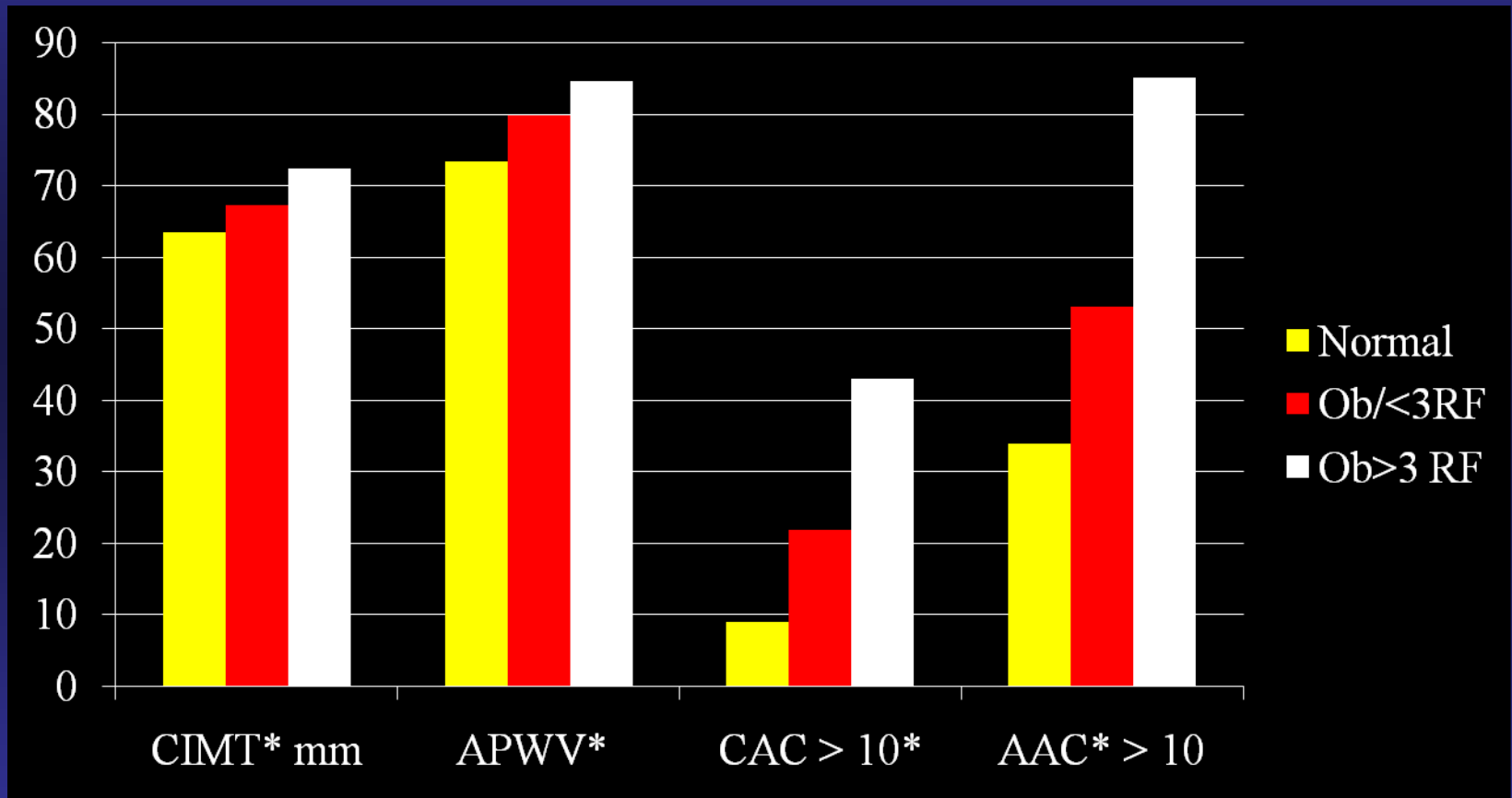
Study of Women's Health Across the Nation (SWAN)

- 475 women (mean age 51 yrs) divided in 3 gps:
 - Healthy normal weight
 - Metabolically benign overweight/obese (less <3 risk metabolic syndrome components (with CRP > 3 substituting for waist since already overweight/obese)
 - This phenotype fairly rare < 1.5% US population
 - At risk overweight/obese (≥ 3 metabolic syndrome components including CRP > 3)
- Evaluated for CIMT, aortic pulse wave velocity, CAC, aortic calcification

SWAN Baseline Data

	Normal (n=145)	Overweight/obese meta benign (n=260)	Overweight/obese at risk (n=70)
BMI	22.7	30.8	34.6
LDL	113	121	124
HDL	63	59	45
HDL < 50	17%	20%	83%
TG	87	96	173
TG > 150	8%	11%	64%
Glucose	84	88	102
Glucose > 100	6%	7%	64%
CRP	.8	2.1	6.1
CRP > 3	15%	35%	84%
BP	110/70	119/76	130/81
10 yr FRS	8.6	9.5	12.9

Subclinical Atherosclerosis Outcomes in SWAN



*CIMT x 10, APWV cm/sec divided by 10,
CAC & AAC both in % with Agatston score > 10

Khan Atheroscl 2011;217:179

SWAN Results

- Metabolically benign overweight/obese women appeared to have significantly greater subclinical CVD than normal weight
 - Further adjustment for BMI attenuated the significance aortic pulse wave velocity, CAC/AAC
 - Many of these benign women actually have metabolic syndrome since they had 2 risk factors plus weight and/or had CRP > 3 in 35% of subjects

Metabolic Risk Factors, Weight and CVD

- Prior evaluations suggested metabolically benign overweight/obese patients are not at increased risk CV events over 3-11 years compared to normal weight
- Health Profession Follow-up in men and Nurses Health Study in women demonstrate a 40-200% increase risk CVD in overweight/obese when followed over 16 yrs

Song AJC 2007;100:1654

Katznarzyk Diab Care 2006;30:2145

Obes Res 1998;6 suppl 2:51S

ATP III:

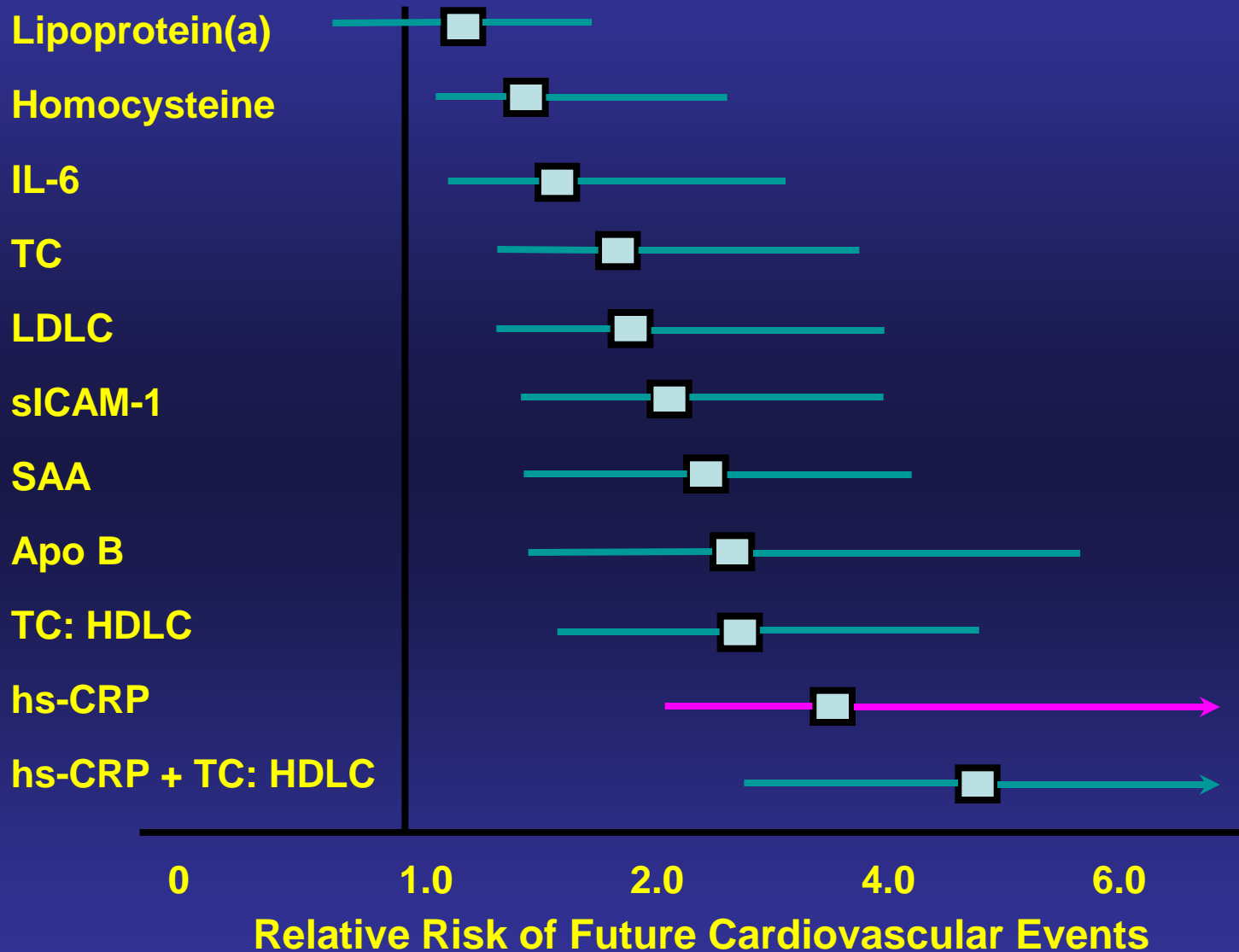
Components of Metabolic Syndrome

- Abdominal Obesity
- Atherogenic Dyslipidemia
- Elevated Blood Pressure
- Insulin Resistance \pm glucose intolerance
- **Pro-inflammation**
 - *Obese subjects with metabolic syndrome had significantly greater amounts of small dense LDL particles and higher CRP than obese subjects without metabolic syndrome*

Does it make sense? Emerging Risk Factors

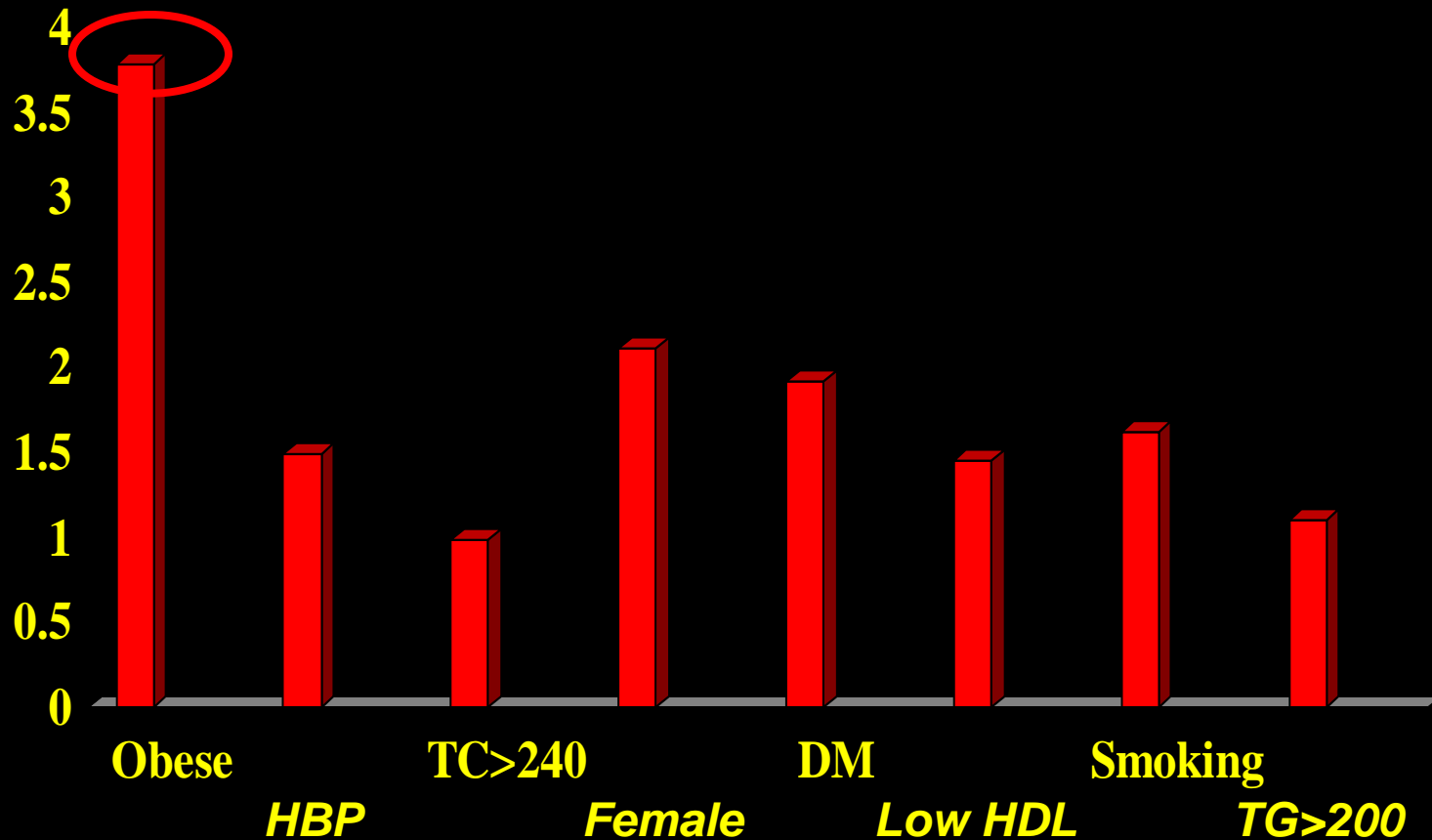
<i>Inflammation</i>	Prospective Studies	Commerical Assay	Additive to Lipids	Additive to FRS
Hs-CRP	++++	+++	+++	++
sICAM-1	++	+/-	+	-
SAA	++	-	+	-
Interleukin-6	++	-	+	-
Interleukin-18	++	-	+	-
Myeloperoxidase	+	-	+/-	-
sCD40 ligand	+	-	-	-
LpPLA2	+	+/-	+/-	-

Risk Factors for Future Cardiovascular Events: WHS



Attributable Risk CRP > 3mg/L from CV Risk Factors

Weighted multiple logistic regression analysis



How to lower CRP

Best Results

- **Exercise & weight loss**
- Statins

Mixed results

- Alcohol in low amounts
- Multivitamins
- Omega 3 fatty acids
- Fibrates
- Niacin
- PPAR gamma (TZD)
- Clopidogrel, abciximab,
- CCB and nitrates
- ARB
- Rimonabant

No CRP Effect (*neutral effect*)

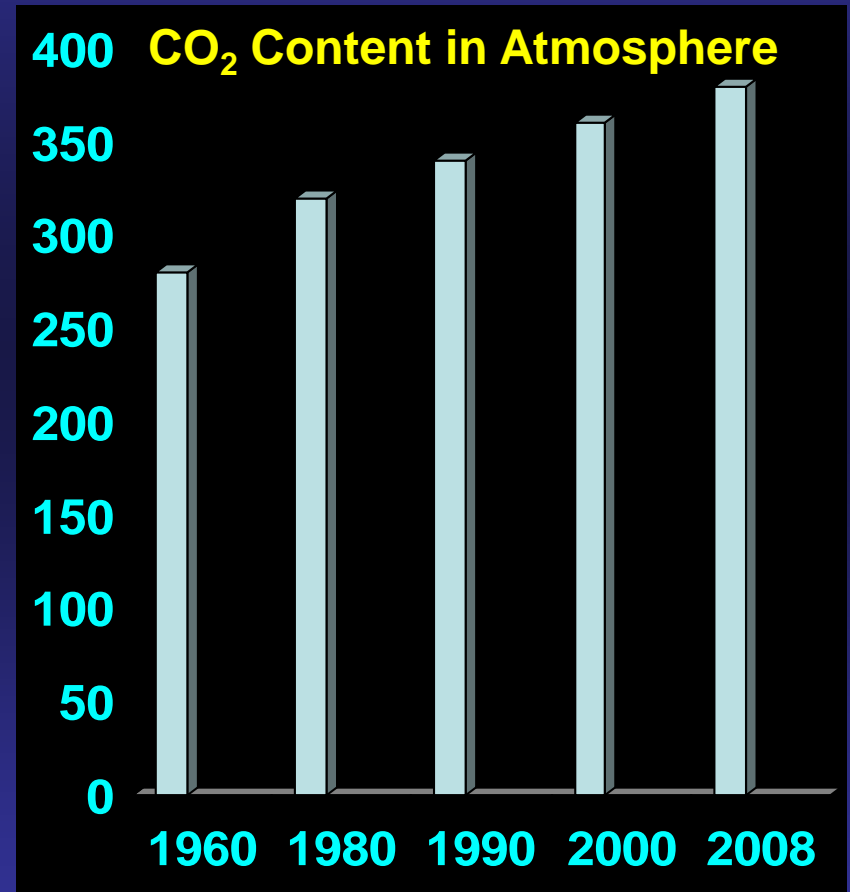
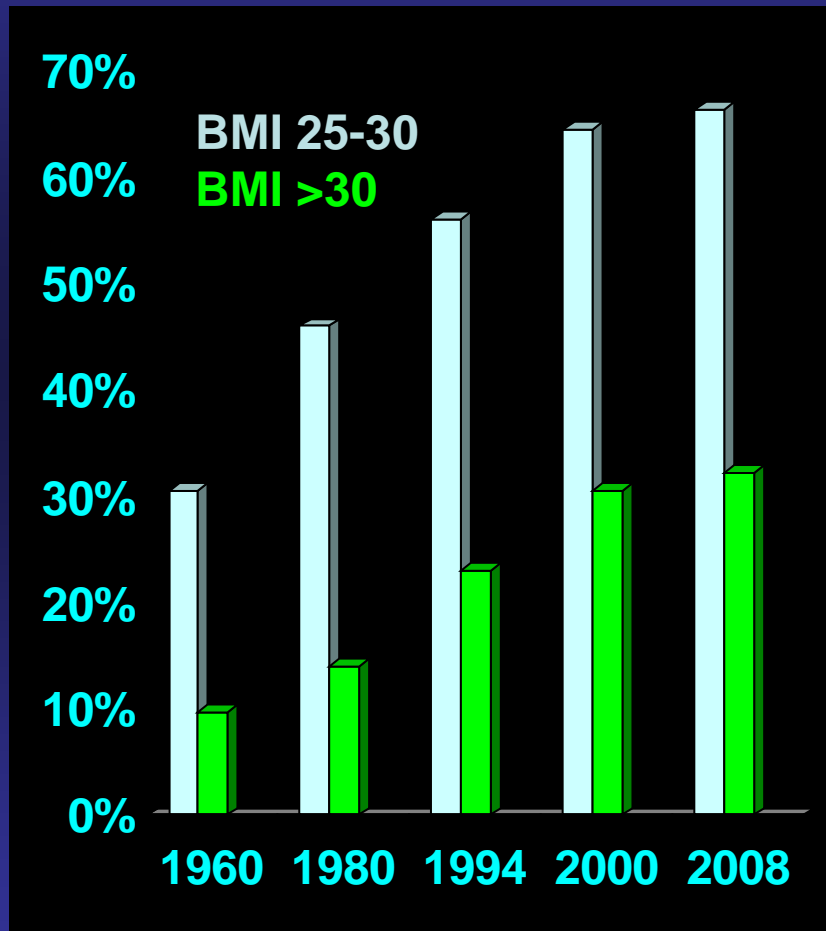
- Aspirin \pm
- COX-1 and 2
- Ezetimibe (monotherapy)
- Stanol ester \pm
(monotherapy)
- *Both ezetimibe and stanols may \downarrow CRP when added to statin)*

Second Hint

Climate Change and
Pollution

Twin Epidemics of Obesity and Global Warming

Two Inconvenient Truths



National Center for Chronic Disease Prevention National Center for Health Statistics
Flegal JAMA 2002;288:1723 Flegal Int J Obesity 1998;22:39 *Inconvenient Truth* 2006

Air Pollution and Atherosclerosis

- Air pollution is a heterogeneous mixture of gases and vapors interacting with solid and liquid particulate matter in atmosphere
- Although both gaseous (eg, ozone) and particulate pollutants are linked, evidence is strongest for particulate matter (PM) as the greater risk for CV disease.
 - Most data to date (hundreds of studies) associate CV risk with PM $<2.5 \mu\text{m}$ (PM_{2.5}) {size $\sim 1/50$ the width of a human hair or half size of RBC}
 - Approximate range in US in 2010 = 5-35 $\mu\text{g}/\text{m}^3$ with annual mean Environmental Protection Agency standard of 15 $\mu\text{g}/\text{m}^3$



Fine Particulate Matter vs Ozone and CVD Mortality

- 448,850 subjects followed from 96 metropolitan areas for 18 yrs
 - Long term exposure to fine particulate matter ($<2.5 \mu\text{m}$) was risk factor for CVD & CV mortality
- Ozone is a single pollutant whose exposure did not increase CVD when corrected for $\text{PM}_{2.5}$ but ozone did increase the risk for respiratory death

Particulate Matter and Plaque

- Numerous cross-sectional observations and one prospective epidemiologic study have demonstrated an association between air pollution and progression of atherosclerosis during a prospective follow-up
- Several studies have demonstrated greater plaque burden, decreased endothelial function and more advanced lesions in animal models who had PM_{2.5} instilled into lungs intermittently at 85 µg/m³ but averaging 15 µg/m³ over 6 months

Exposure to Air Pollution is Associated with Adverse CV Events

- Brief exposure to diesel exhaust (equivalent to exposure of driving in traffic) promotes myocardial ischemia and inhibits endogenous fibrinolytic capacity in those with baseline CAD
- The risk after brief exposure appears to persist for several days and is related to pulmonary oxidative stress, decreased O² carrying capacity caused by ↑ levels carbon monoxide, enhanced systemic inflammation, and altered hemostatic-fibrinolytic balance

Diesel Fuel and Atherosclerosis

- Prevalence atherosclerosis increased with exposure diesel engine exhaust
 - Due to carbon monoxide (CO), nitrogen dioxide (NO₂) aldehydes, particulate matter (PM) ultrafine <.1 μm and fine PM_{2.5}
- Numerous studies have shown total and CV mortality increased in populations living near major roadways

Acute vs Chronic Exposure

- Short term exposure over a few hours to days increases risk of MI, stroke, HF exacerbation, arrhythmias, SCD
 - Risk approximate 1% increase CV mortality per 10 $\mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$ or about a 1 standard deviation increase in ambient levels in US
- Chronic exposure (yrs) lead to 10-20% increase in CV mortality per 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$
 - Suggests cumulative exposure promotes development of chronic underlying disease state that exponentially augments future CV risk over yrs by enhancing progression and vulnerability of atherosclerotic plaque

Exposure to Air Pollution is Associated with Adverse CV Events

- WHO estimates that air pollution responsible for 800,000 premature deaths worldwide/yr
- Long term exposure to air pollution ↑ risk for MI, stroke, ventricular arrhythmia and HF
- The acute transient effects of particulate air pollution needs to be distinguished from the heightened long term chronic risk of CVD associated with living in areas with high levels of air pollution

Jerrett *Epidem* 2005;16:727
Miller *NEJM* 2007;356:447
<http://www..who.int/whr/2002/en/>.

Exposure to Air Pollution is Associated with Adverse CV Events

Harvard Six Cities Study (1977-1988)

Characteristics of the Study Population and Mean Air-Pollution Levels in Six Cities

CHARACTERISTIC	PORTAGE, WIS.	TOPEKA, KANS.	WATERTOWN, MASS.	HARRIMAN, TENN.	ST. LOUIS	STEUBENVILLE, OHIO
No. of participants	1,631	1,239	1,336	1,258	1,296	1,351
Person-years of follow-up	21,618	16,111	19,882	17,836	17,715	17,914
No. of deaths	232	156	248	222	281	291
Deaths/1000 person-years	10.73	9.68	12.47	12.45	15.86	16.24
Female sex (%)	52	56	56	54	55	56
Smokers (%)	36	33	40	37	35	35
Former smokers (%)	24	25	25	21	24	23
Average pack-years of smoking						
Current smokers	24.0	25.6	25.2	24.5	30.9	28.0
Former smokers	18.0	19.7	21.8	21.1	22.0	25.0
Less than high-school education (%)	25	12	22	35	45	30
Average age (yr)	48.4	48.3	48.5	49.4	51.8	51.6
Average body-mass index	26.3	25.3	25.5	25.1	26.0	26.4
Job exposure to dust or fumes (%)	53	28	38	50	40	48
Total particles ($\mu\text{g}/\text{m}^3$)	34.1	56.6	49.2	49.4	72.5	89.9
Inhalable particles ($\mu\text{g}/\text{m}^3$)	18.2	26.4	24.2	32.5	31.4	46.5
Fine particles ($\mu\text{g}/\text{m}^3$)	11.0	12.5	14.9	20.8	19.0	29.6
Sulfate particles ($\mu\text{g}/\text{m}^3$)	5.3	4.8	6.5	8.1	8.1	12.8
Aerosol acidity (nmol/m^3)	10.5	11.6	20.3	36.1	10.3	25.2
Sulfur dioxide (ppb)	4.2	1.6	9.3	4.8	14.1	24.0
Nitrogen dioxide (ppb)	6.1	10.6	18.1	14.1	19.7	21.9
Ozone (ppb)	28.0	27.6	19.7	20.7	20.9	22.3

*Air-pollution values were measured in the following years: total particles, sulfur dioxide, nitrogen dioxide, and ozone, 1977 through 1985; inhalable and fine particles, 1979 through 1985; sulfate particles, 1979 through 1984; and aerosol acidity, 1985 through 1988.

Dockery D et al. N Engl J Med 1993;329:1753-1759



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JOURNAL of MEDICINE

Exposure to Air Pollution is Associated with Adverse CV Events

Harvard Six Cities Study

Characteristics of Study Population and Air Pollution Levels in Six Cities

CHARACTERISTIC	YONKONG, CHINA	CHENGBAI, CHINA	CHENGBAI, CHINA	CHENGBAI, CHINA	CHENGBAI, CHINA	CHENGBAI, CHINA
No. of persons						1,351
Person-years						7,914
No. of deaths						91
Deaths/100 person-years						16.24
Female sex (%)						56
Smokers (%)						35
Former smokers (%)						23
Average pack-years of smoking						
Current smokers				30.9	28.0	
Former smokers				21.1	22.0	25.0
Less than high-school education (%)	25	12	22	35	45	30
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Ozone (ppb)	28.0	27.6	19.7	20.7	20.9	22.3

Cardiovascular deaths increased 28% with each $10 \mu\text{g}/\text{m}^3$ increase in fine particulate air pollution measuring less than $2.5 \mu\text{g}$ ($\text{PM}_{2.5}$)

*Air-pollution values were measured in the following years: total particles, sulfur dioxide, nitrogen dioxide, and ozone, 1977 through 1985; inhalable and fine particles, 1979 through 1985; sulfate particles, 1979 through 1984; and aerosol acidity, 1985 through 1988.

Dockery D et al. N Engl J Med 1993;329:1753-1759
 Laden Am J Resp Crit Care Med 2006;173:667
 Pope JA MA 2002;287:1132



Exposure to Air Pollution is Associated with Adverse CV Events

Harvard Six Cities Study

Characteristics of ... in Six Cities

Exposure to PM_{2.5} may modulate atherosclerosis similar to other risk factors (ie DM, lipids, cigarettes etc)
Stimulating immune response with macrophages,

1/3 of US population live in areas of unhealthful levels of ozone

- More than 93.7 million US citizens live in areas with unhealthful short-term levels of particulate pollution. And >200 million are currently overweight or obese

CHARACTE

No. of

Person

No. of

Death

Female

Smokers

Former sm

Average pack

smok

Curre

For

Less

A

A

J

T

In

Fin

Sulfur

Aerosol

Sulfur di

Nitrogen di

Ozone (ppb)

*Air-pollution values were ...
1977 through 1985; inhalable and ...
1985 through 1988.



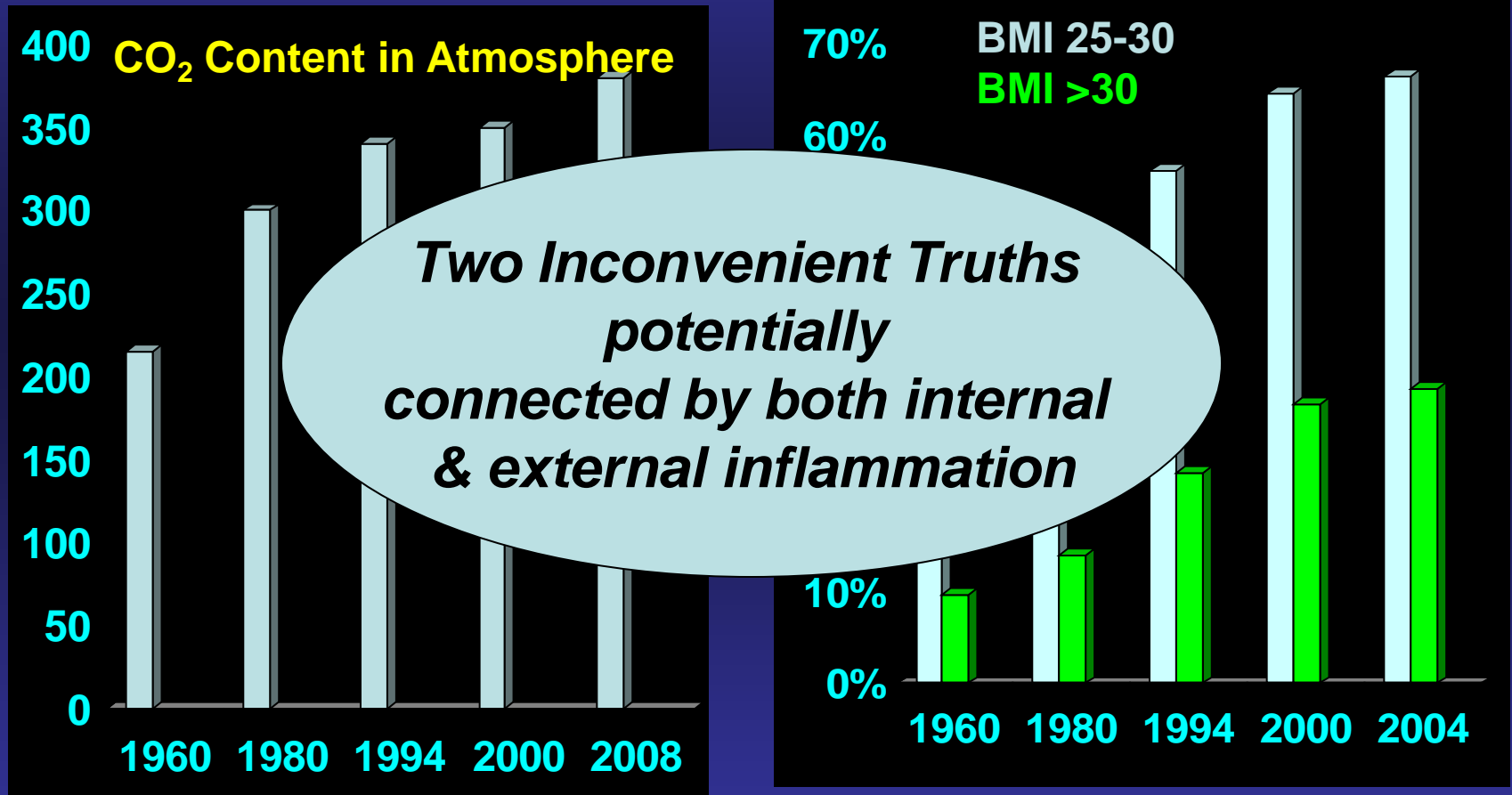
The NEW ENGLAND
JOURNAL of MEDICINE

Dockery D et al. N Engl J Med 1993;329:1753-1759

Sun JAMA 2005;294:3003

Sun Inhal Toxicol 2008;20:127

Obesity and Global Warming



Third Hint

**All calories may not be
created equal**

30-40 Year Trends

- We as a population weigh 25 pounds more than we did 30 years ago.
- In 1982 AHA, AMA and USDA recommended to decrease fat intake from 40% to 30% of calories
- We succeeded in doing this but our weight has gone up by 25 pounds.

30-40 Year Trends

Today we eat more calories

- Teen boys eat 275 more calories than 30 years ago
 - Of these calories ~ 5g are from fat = 45 calories and ~57g from carbohydrates = 228 calories
- Adult males eat 187 more calories a day
- Adult females 335 more calories a day

30-40 Year Trends

More Liquid Carbohydrate Intake

- 41% increase in soft drinks and 35% increase in fruit drinks
- Average can of soda = 150 calories x 365 days then divide by 3500 (number of calories per pound) = 15.6 pounds per yr
 - First Coke 1915 6.5 oz (8lb/yr) →
 - 1955 10 oz (13lb/yr) →
 - 1960 12 oz (16 lb/yr) →
 - 1992 20 oz (26 lb/yr)

Why is Coke Bad?

- Soda 41% increase in consumption of drinks and 35% increase

***55 mg sodium per can of Coke
+ caffeine (mild stimulant and diuretic)
increases free water clearance thus
the increased sodium and the decrease in water
causes increased thirst.***

***The increased salt is masked by
the sugar to allow us to take in more***

(13lb/yr)

oz (26 lb/yr)

oz

1992 20

COKE FOR INFANTS!

*Half is sugar
(corn syrup & sucrose)
A baby milk shake*

*Coca Cola is 10.5% sucrose
What's the difference?*

The start of a vicious cycle

- Leptin (fat cell hormone) which tells us we have had enough and we stop eating correctly

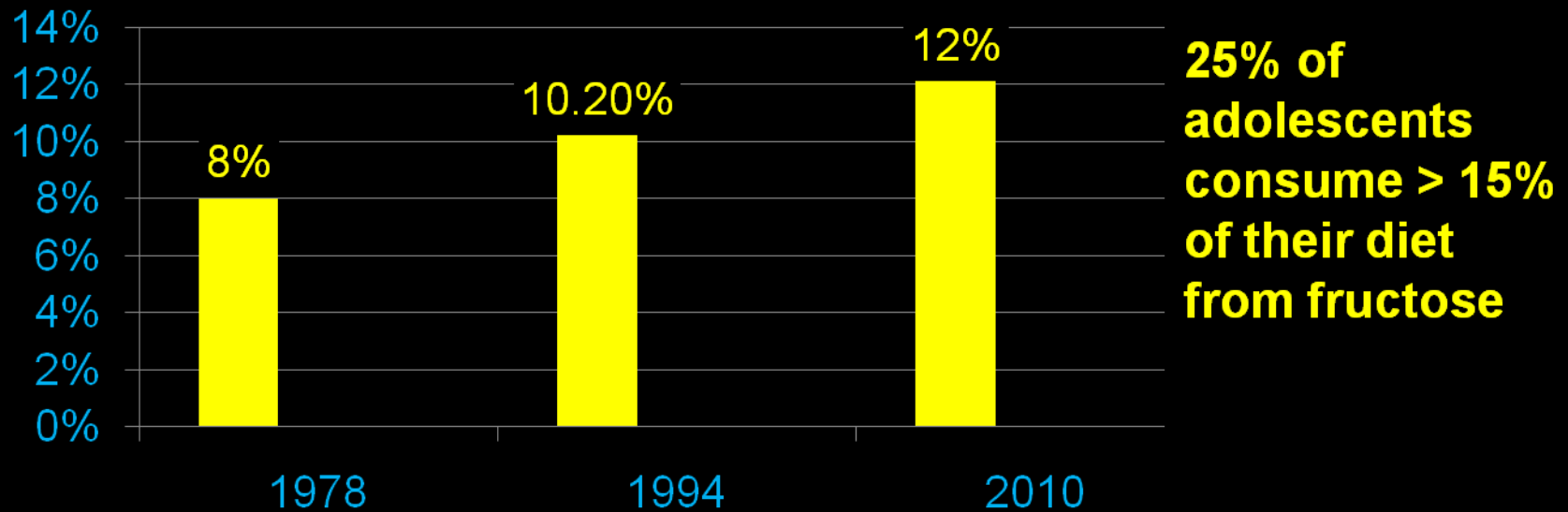
The earlier you expose a child to sweets the more they crave it later.

The more sugar a pregnant women eats the more gets across the placenta causing developmental programming which can change a newborns adiposity before birth

High Fructose Corn Syrup

- HFCS is main component of soft drinks because it's 20% sweeter than regular table sugar (*sucrose*)
 - Glucose is 26% less sweet than sucrose
 - HFCS is also cheaper
 - Current US consumption of HFCS = 63 lbs per person/yr
 - HFCS is 42-55% fructose
 - HFCS is one glucose (6 member ring) and one fructose (5 member ring)
 - HFCS is essentially equivalent to sucrose which is 50% fructose connected to glucose by an ether linkage

Fructose Increasing in American Diet



**▲ Consumption fruits & vegetables decreased from 24 g in 1978 to 15 g/d today
And fiber reduced from 100g to 15g
Why is this trend related to obesity?**



Glucose Metabolism

- *Example of how glucose metabolized ie 120 calories (2 slices of white bread)*
 - 80% glucose used by every cell for energy
 - 20% or 24 calories to liver
 - Glucokinase converts glucose to glucose-6-phosphate then to glycogen
 - Most of glucose is stored in liver as glycogen
 - No amount of glycogen will hurt the liver since glycogen is non-toxic form of glucose storage

Glucose Metabolism II

- ***How 120 calories from glucose metabolized***
A small amount G-6-P not stored as glycogen gets converted to fructose 6-P & eventually metabolized to pyruvate then converted to acetyl CoA in mitochondria to enter Krebs cycle = ATP & CO²
 - Small amount acetyl CoA from TCA exits as citrate and leaves mitochondria via citrate shuttle and results in de novo lipogenesis by forming acyl CoA which is converted to VLDL (so maybe a ½ to 1 calorie from the original 120 used for VLDL)

Fructose Metabolism

120 calories (glass of OJ) from sucrose (half glucose & half fructose)

- All 60 calories from fructose to liver (only liver can metabolize fructose) thru GLUT 5 metabolized by fructokinase to form fructose-1 phosphate and in the process ATP gives up one phosphate to create ADP
- The 60 calories from glucose (12 to liver and 48 to body)

Fructose Metabolism II

- For the liver to metabolize the 72 calories (3x calorie from glucose alone) a lot of phosphate will be lost thru scavenger pathway resulting in end product of uric acid
 - Therefore, high fructose consumption increases uric acid and possibly leads to gout and SBP
 - Uric acid can then block ENOS which predisposes to high blood pressure
 - Obese adolescents with HBP, allopurinol lowered BP

Low Fat Diet Myth

- The typical low fat diet isn't really low fat if it is substituting fructose for fat
- Fructose although a carbohydrate is metabolized like fat, such that 30% of its calories is converted to fat



Parks J Nutr 2004;134: 1333

Schwarz Diabetes 2005;54:1907



Fructose *is not* Glucose

- Fructose increases calorie loads to liver
- Acute fructose ingestion does not stimulate insulin because no fructose receptor on Beta cell, if insulin doesn't go up then leptin won't go up (brain doesn't recognize you ate something)
 - Fructose does not suppress gherlin (hunger hormone)
- Fructose is 7 times more likely than glucose to form Advanced Glycation End Products

*Bray Am J Clin Nutr 2004 Wei J Nutr Biochem 2006
Johnson Am J Clin Nutr 2007 Rutledge Nutr Rev 2007*

Fructose at the Center of Obesity Epidemic

- Fructose consumption has increased in past 30 years coinciding with obesity epidemic
- A calorie is not a calorie (fructose is not glucose)
- Hepatic fructose metabolism leads to metabolic syndrome:
 - Hypertension
 - De novo lipogenesis, dyslipidemia, steatosis
 - Inflammation
 - Insulin resistance
 - Obesity
 - CNS leptin resistance promoting continuous consumption
- Fructose is a chronic hepatotoxin (*alcohol without the buzz*)

**Bray Am J Clin Nutr 2004 Wei J Nutr Biochem 2006
Johnson Am J Clin Nutr 2007 Rutledge Nutr Rev 2007**

CHOPPS Program

- Soda 41% increase in soft drinks and 35% increase in

Removal of carbonated (fructose) drinks from schools in England (CHOPPS Program) for a year found the prevalence of obesity remained the same (-0.2%) Compared to keeping soda in the schools where rate of obesity continued to rise (+7.5%)

James BMJ 2004;328:1237

(15... 20
oz (26 lb/yr)

(CHOPPS Program = Christchurch obesity prevention project in schools)

Changes in Diet and Lifestyle and Long Term Weight Gain

- 120,877 men and women free of chronic diseases and obesity followed up from 1986-2006
- With each four year period participants gained an average of 3.35 lb on the basis of increasing daily servings

Mozaffarian NEJM 2011;364:2392

Diet component	Additional lb
Potato Chips	1.69 lb
Potatoes	1.28 lb
Sugar sweetened beverages	1.00 lb
Red meats	0.95 lb

Inversely related	Minus lb
Vegetables	-0.22 lb
Whole grains	-0.37 lb
Fruits	-0.49 lb
Nuts	-0.57 lb
Yogurt	-0.82 lb

Changes in Diet and Lifestyle and Long Term Weight Gain (Part 2)

- 120,877 men and women free of chronic diseases and obesity followed up from 1986-2006
- With each four year period participants gained an average of 3.35 lb on the basis of increasing daily servings

Other components	Additional lb
Alcohol	0.41/drink/day
Smoking new quitters	5.17 lb
Former smoker	0.14 lb
Sleep (<6 or >8 hrs)	0.31 lb
Watch TV	0.31 lb

Inversely related	Minus lb
Physical activity	-1.76 lb

When a calorie is not a calorie II

- 120.8
- wo
- dise
- follow
- 20
- 9
- 3.35
- increasing
- servings

When you sit down to a meal, your brain is looking for nutrients, not calories and will

High fiber foods expand in the stomach, slowing digestion and augmenting satiety. SO taking in right calories can help prevent the need for more calorie intake later

Changes in Diet and Lifestyle

Nuts versus French Fries

NUTS

- *Take longer to chew*
 - *Contain fat and fiber that take longer to digest*
 - *Your stomach stays fuller, and you feel satisfied longer*
 - **SO YOU EAT LESS AT YOUR NEXT MEAL**
- Net loss of 0.57 pounds*

servings

FRENCH FRIES

- *Cooked starch is quickly broken down*
- *Causes spike in glucose and insulin secretion leading to hunger*
- **SO YOU EAT MORE AT YOUR NEXT MEAL**

Net gain of 3.35 pounds

What Can We DO?

**An Osteopathic
Approach**

If you eat it you have to burn it off. True or false?

- If true... why is there now an epidemic of obesity in six month olds
- Very few to no one chooses to be obese, this is a problem of a system gone out of control.
- Our neurohumoral feedback systems do not seem to be working correctly

Hungry Brain Syndrome

- The inherent directive from the brain to eat and defend against loss of fat emerged early in our evolution due to the uncertainty of that next meal.
 - This process is also related to 24 hour (day/night) cycle which is up-regulated with sleep disturbances and stress which decrease metabolism but increase the yen for high calorie food
- However, the myth that obesity is caused by a loss of willpower or lack of motivation is simply a misconception.

Fat Phobia Scale

Listed are 14 pairs of adjectives sometimes used to describe obese or fat people. For each adjective pair please place an X on the line closest to the adjective that best describes your feelings and beliefs

	5	4	3	2	1	
Lazy						Industrious
No will power						Has will power
Attractive						Unattractive
Good self control						Poor self control
Fast						Slow
Having endurance						Having no endurance
Active						Inactive
Weak						Strong
Self Indulgent						Self sacrificing
Dislikes food						Likes food
Shapeless						Shapely
Undereats						Overeats
Insecure						Secure
Low self esteem						High self esteem

Obesity Bias in Health Care Professions

- Using the fat phobia scale Primary Care Physicians, Physician Assistants, Nurses and Dietitians all viewed obese patients as awkward, unattractive, lazy, weak willed and noncompliant
- Bias and negative attitude toward obese patients creates a barrier in the treatment of obesity

*Wolf J Phys Asst Edu 2010;21:37
Foster Obes Res 2003;11:1168
Puhl J Am Diet Assoc 2009;109:438
Poon J Clin Nurs 2009;18:2355*

Self efficacy of Family Medicine Practitioners to Treat Obesity

- Self efficacy is a belief in one's ability to organize and execute a course of action required to produce a given result
- 29 FP were analyzed before and after an interactive course designed to enrich their knowledge of obesity management
 - Post course efficacy scores were significantly improved

Self efficacy of Family Medicine Practitioners to Treat Obesity

Content of interactive course on obesity:

- Epidemiology
- Pathogenesis and metabolic factors
- Nutrition and diet
- Self control and behavior modification
- Physical activity
- Drug and surgical treatment
- *Metabolic syndrome*
- *Mechanism of hypertension in obesity*
- *Diabetic obese patient*
- *Infertility in obese women*

AOA Resolution

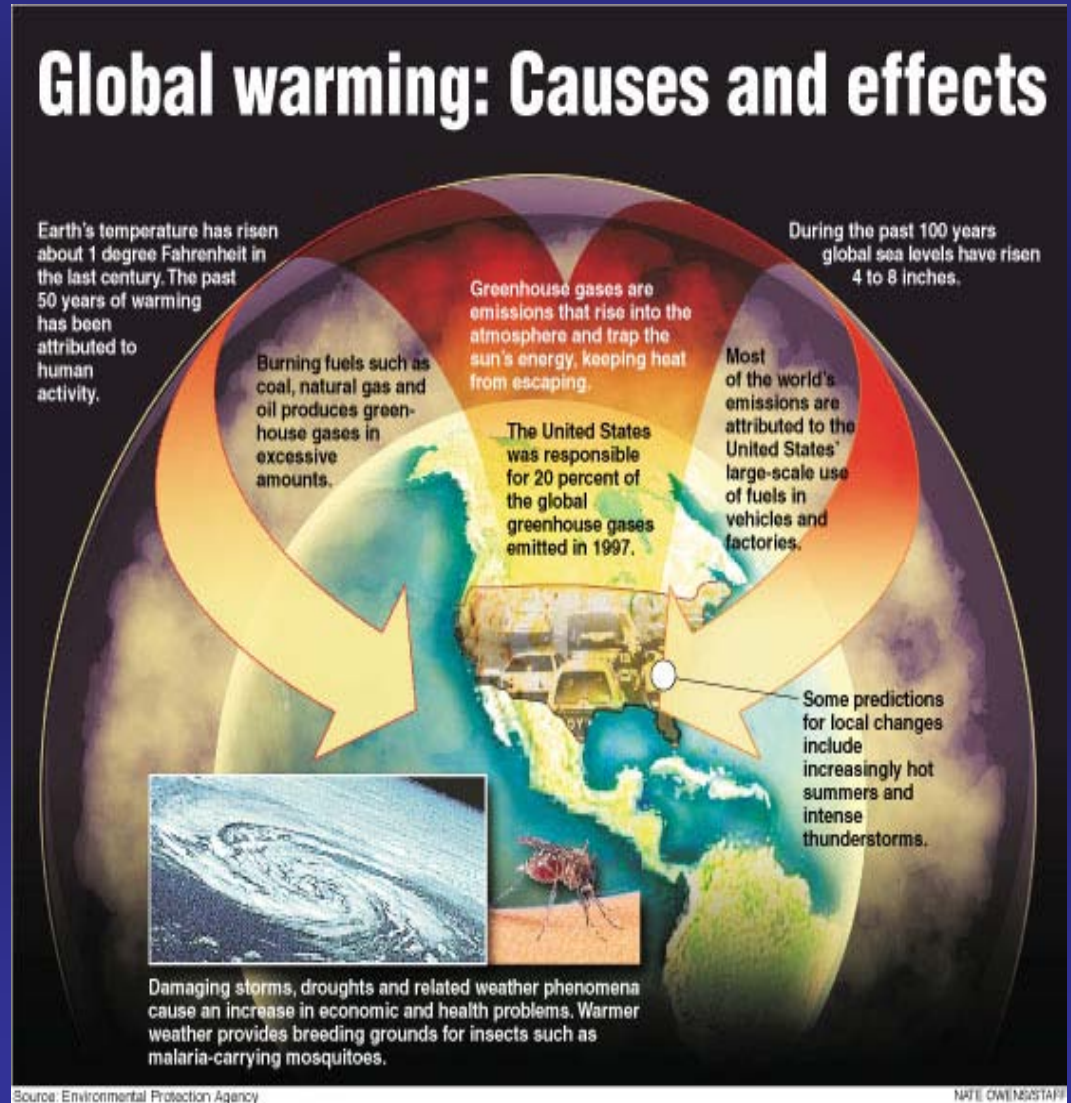
- At the 2011 AOA House of Delegates a resolution was approved to have all DOs address the obesity epidemic in their practices
- A pilot study is being implemented to determine whether an educational program will change medical students attitudes which will change behavior and ultimately change their patients outcomes

Easy to Use Recommendations For Lifestyle Intervention

- Get rid of all sugar liquids-only water and milk
- Eat your carbohydrate with fiber (fruit, vegetables)
- Wait 20 minutes for second portion
- Buy your screen time minute for minute with physical activity

Global Risks with Warming

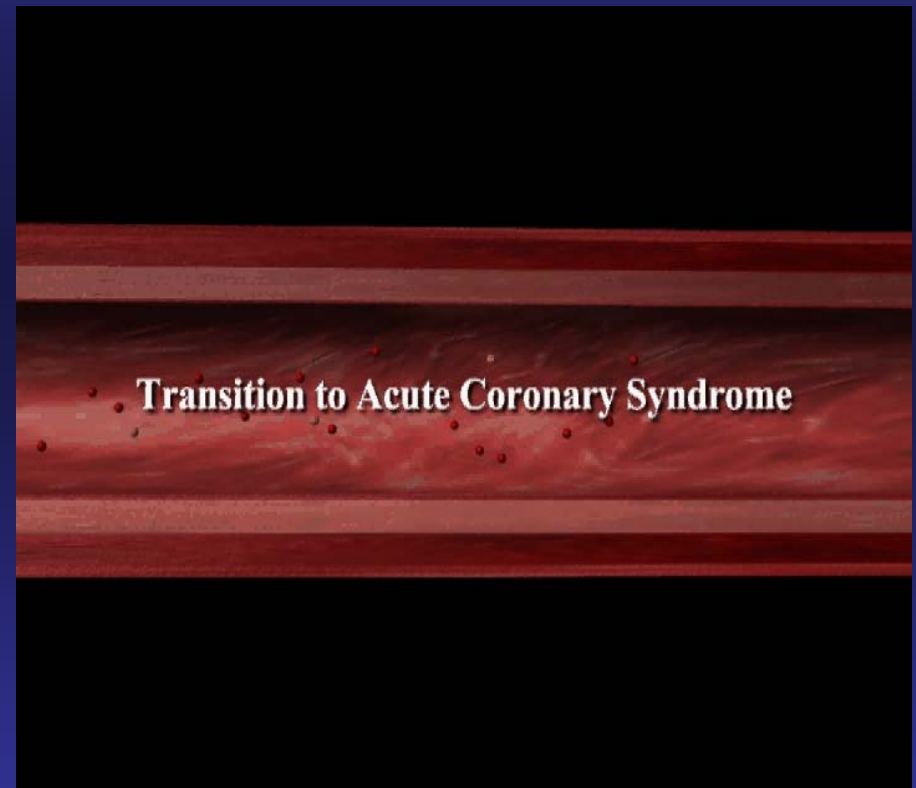
- As the earth's atmosphere warms and CO₂ levels rise... glaciers & polar ice caps melt
 - Increasing temperatures predispose to catastrophic hurricanes, tornados and paradoxically draughts



Global Risks with Warming and Atherosclerosis

Icecaps and Endothelium

- As the earth's atmosphere warms & CO₂ levels rise. causing melting & cracks in the icecaps
- While also increasing oxidative stress and systemic inflammation...
- And when combined with an obesity mediated atherogenic milieu...



Dual Inconvenient Truths

- Combination of air pollution when superimposed on obesity and its metabolic abnormalities results in the dual inconvenient truths of increased CV risk & mortality and global warming.

Nature Abhors a Vacuum

So as the Glaciers melt away...



Dual Inconvenient Truths

Nature Abhors a Vacuum



As the glaciers melt away...

The space previously occupied will be taken up by our expanding waistlines

