OSN NURSING SYMPOSIUM: STROKE

5th Annual OSN Symposium
St. Charles – Bend

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Epidemiology

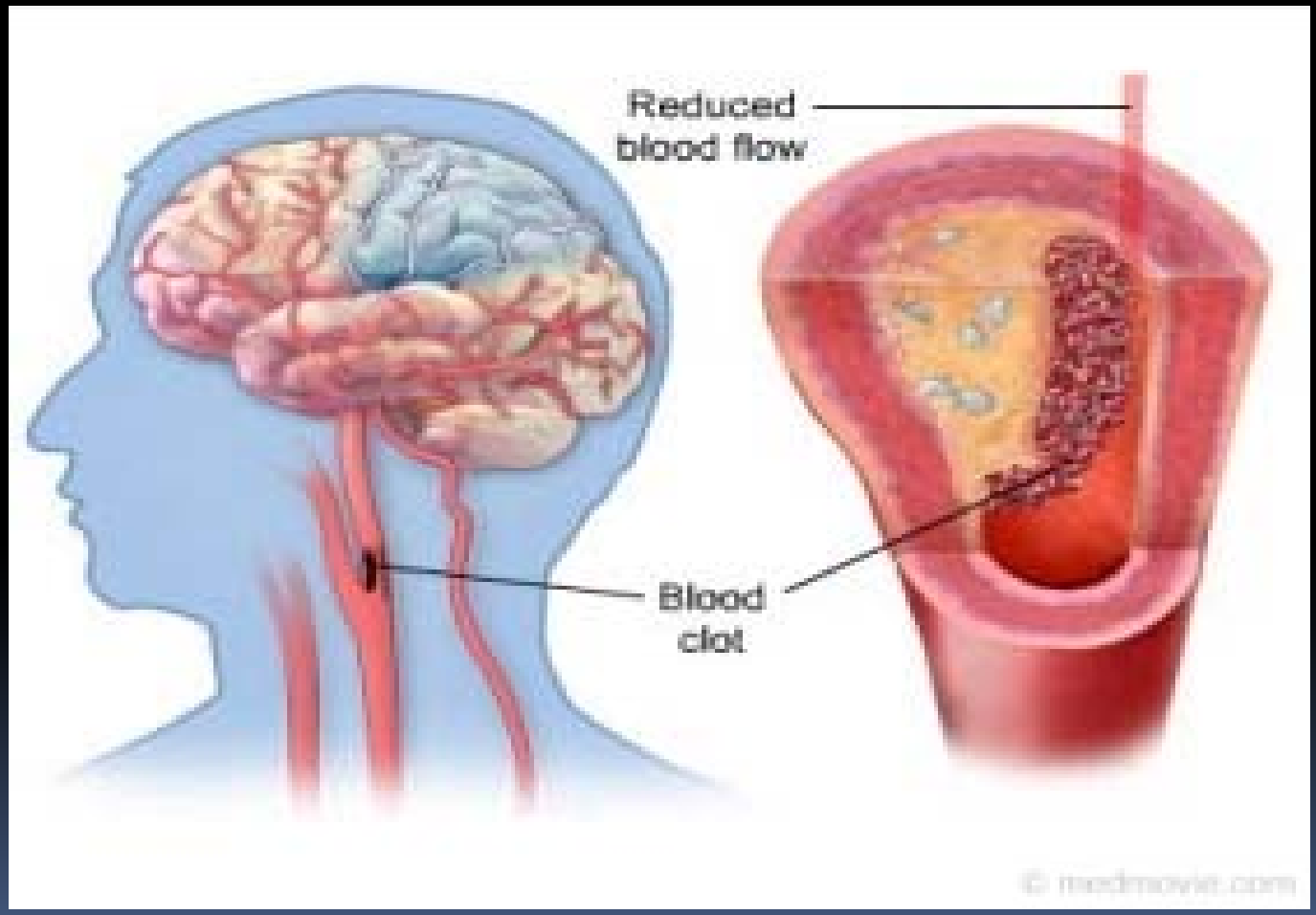
- 795,000 suffer a new or recurrent stroke each year
  - 1 every 40 seconds
- Stroke kills more than 137,000 each year
  - 1 of every 18 deaths
  - Every 4 minutes someone dies of a stroke
  - Of deaths: 40% men & 60% women
- $73.7 billion in 2010 for stroke related medical costs & disability

Heart Disease and Stroke Statistics. 2011 Update

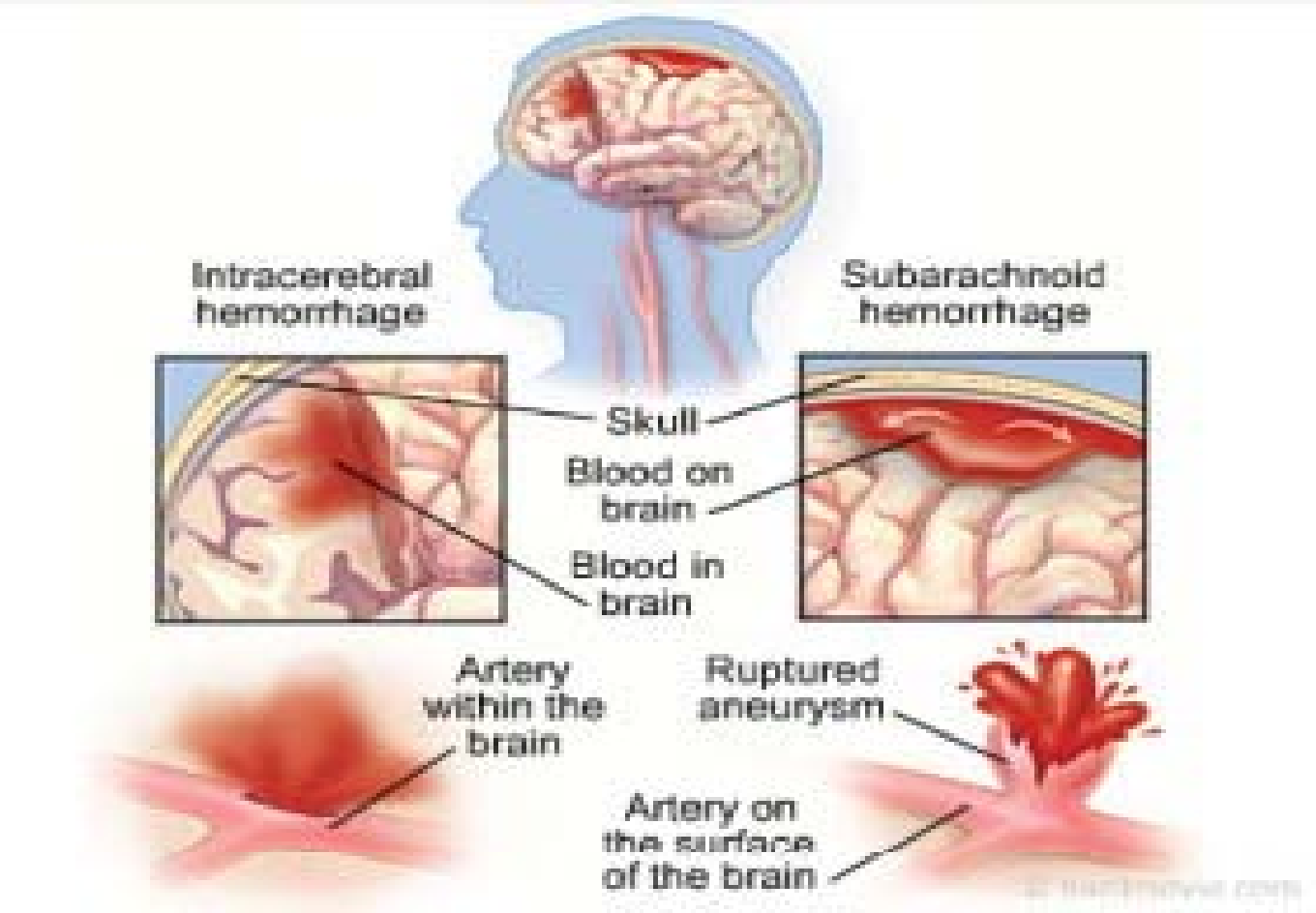
Stroke types

- Ischemic
  - Associated with atherosclerosis
- Hemorrhagic
  - Associated with Hypertension
- TIA

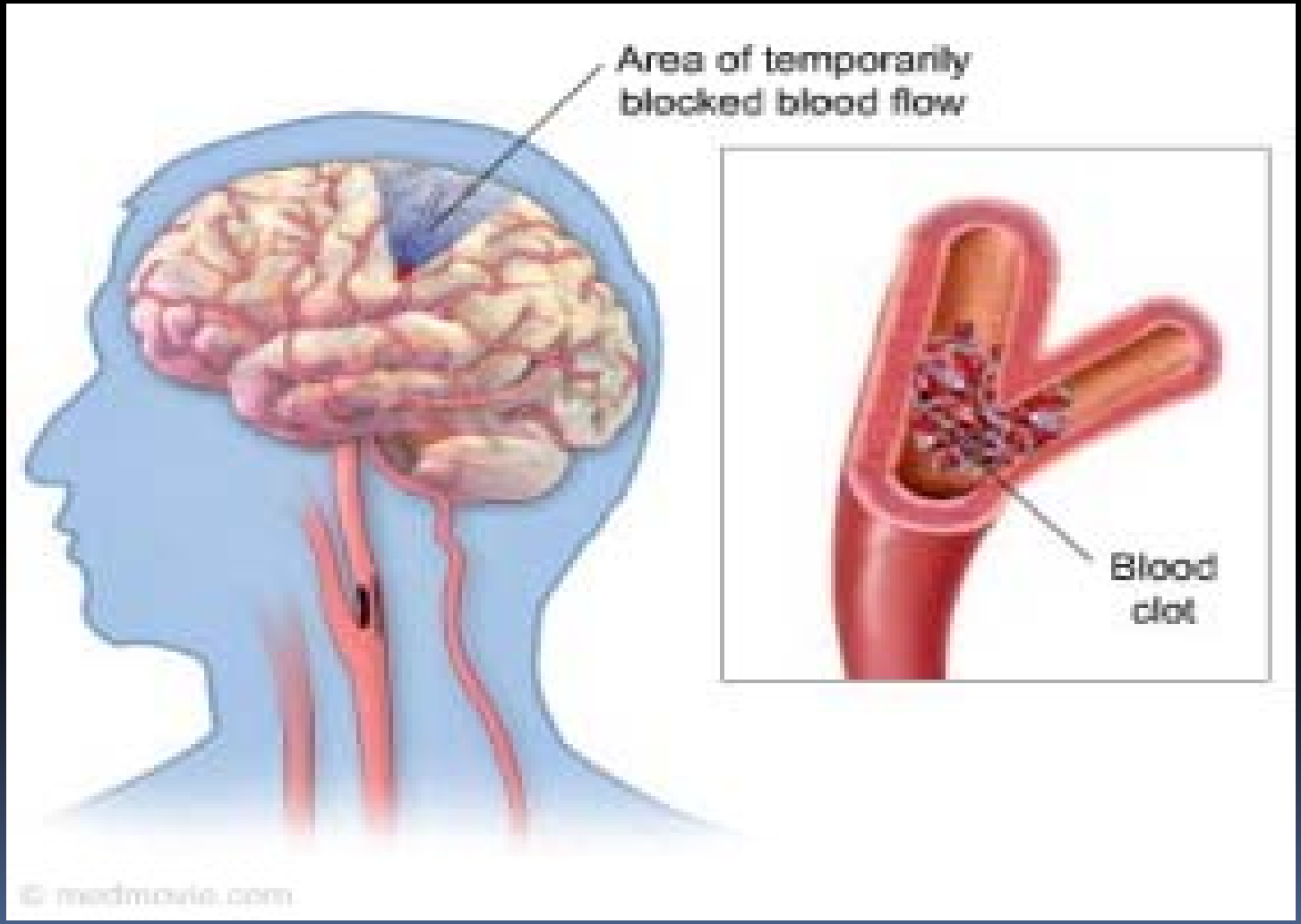
Ischemic Stroke - 87% of all stroke



Hemorrhagic Stroke



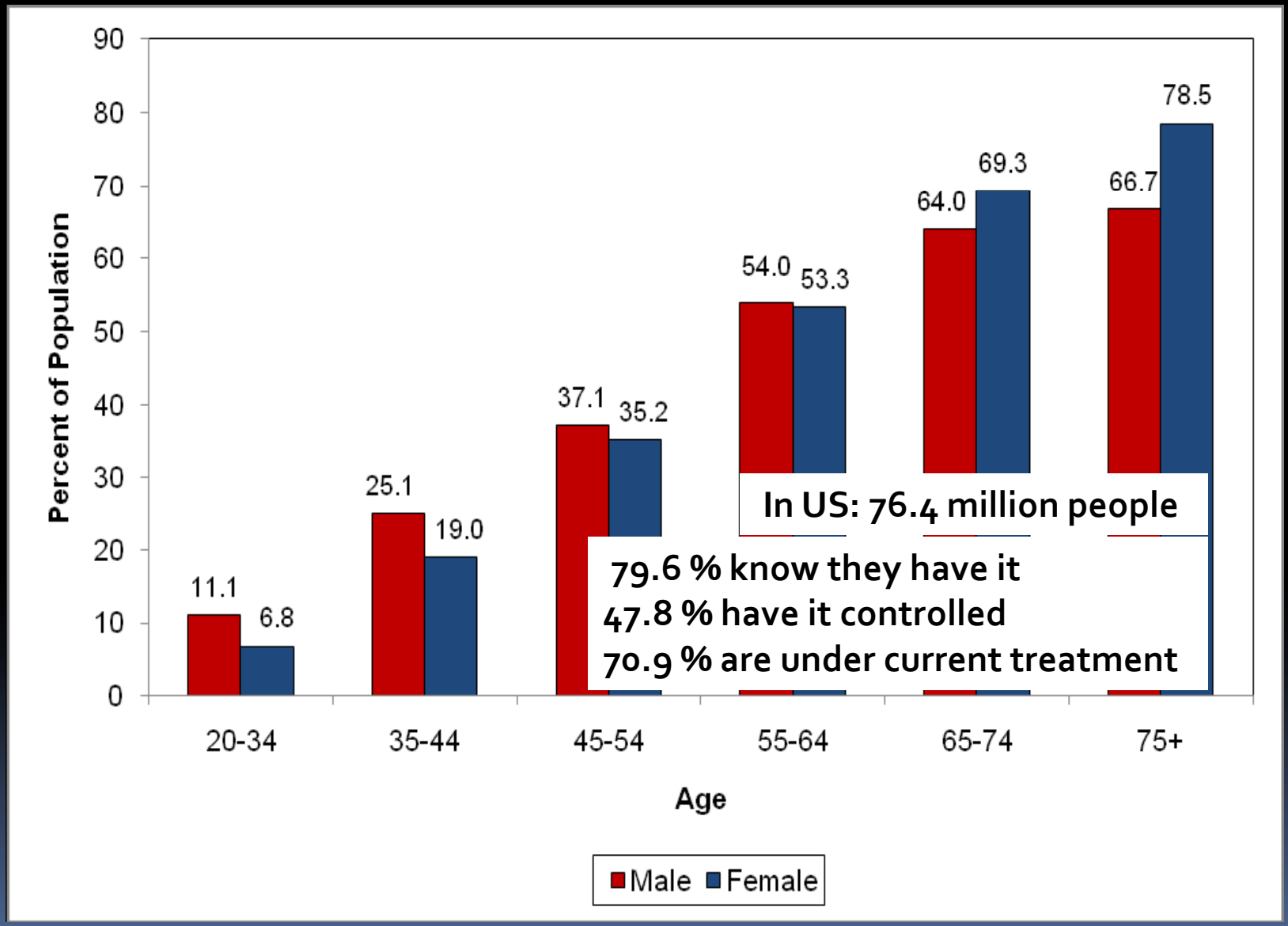
TIA



Risk Factors for Stroke

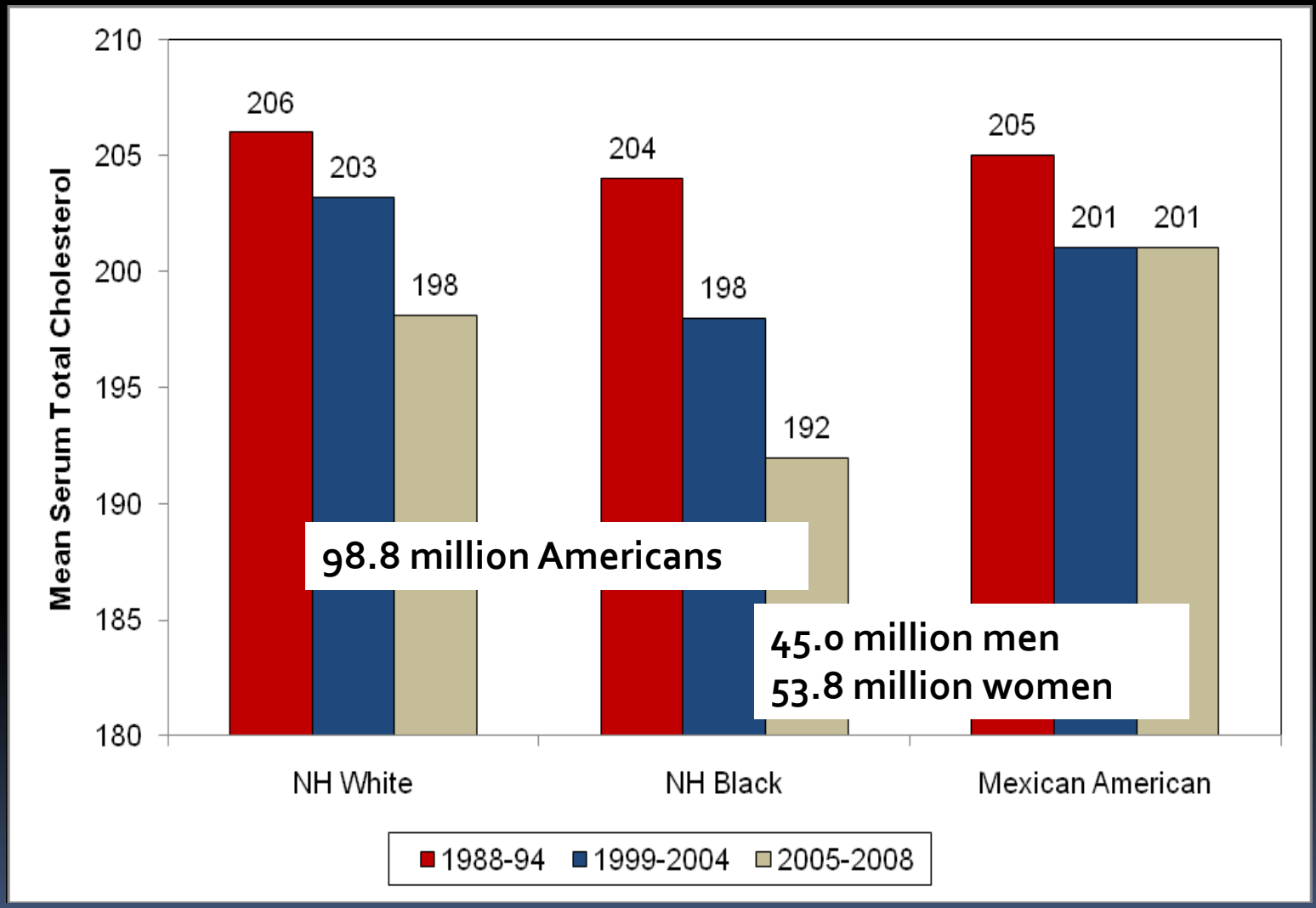
- The Big Killers and how they work
  - Hypertension
  - Hypercholesterolemia
  - Diabetes
  - Smoking

Prevalence of Hypertension: Adults >20 & gender



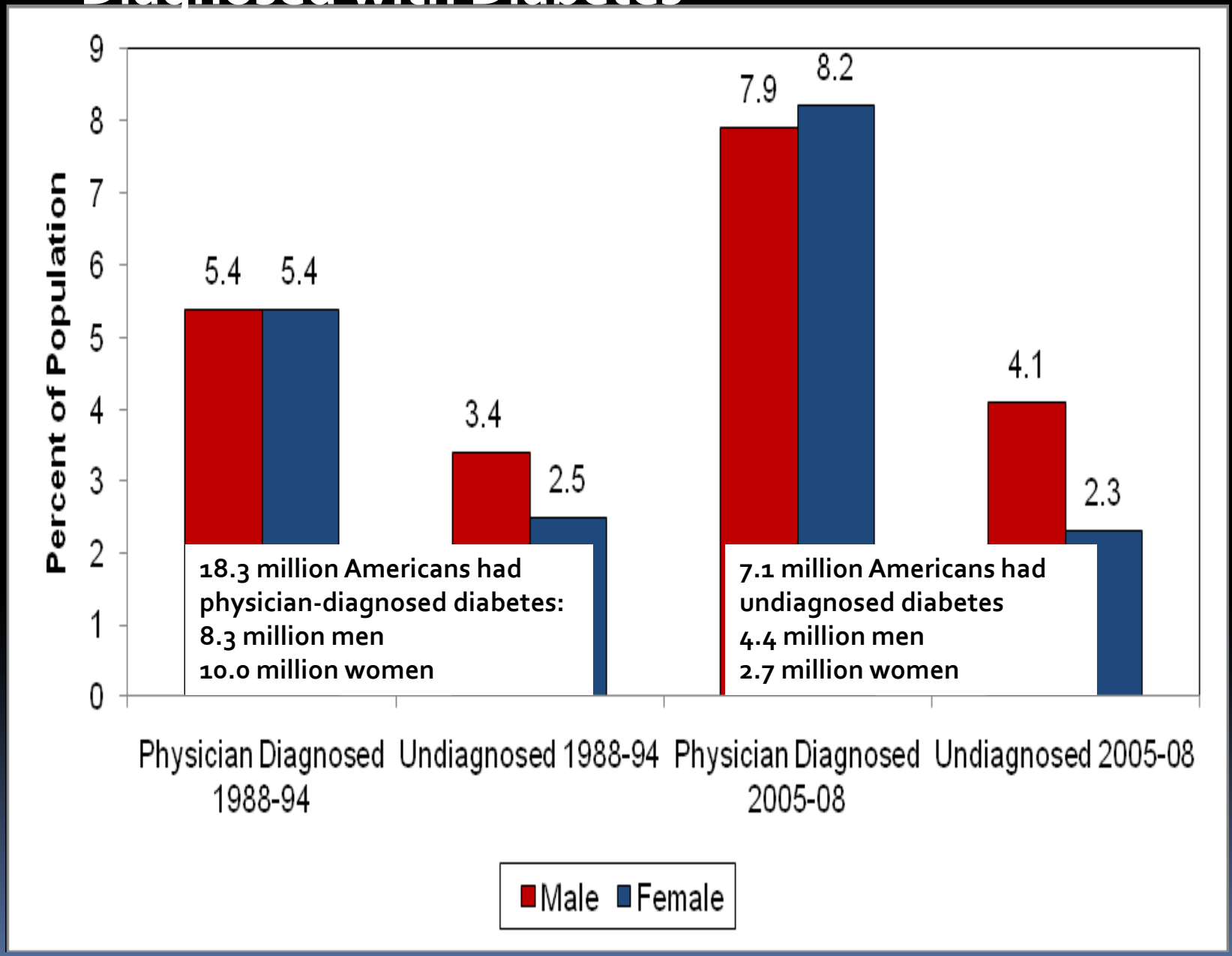
Prevalence of High Blood Pressure in Adults Age 20 and Older by Age and Sex. NHANES: 2005–08

Total Cholesterol: Adults >20 by race and year



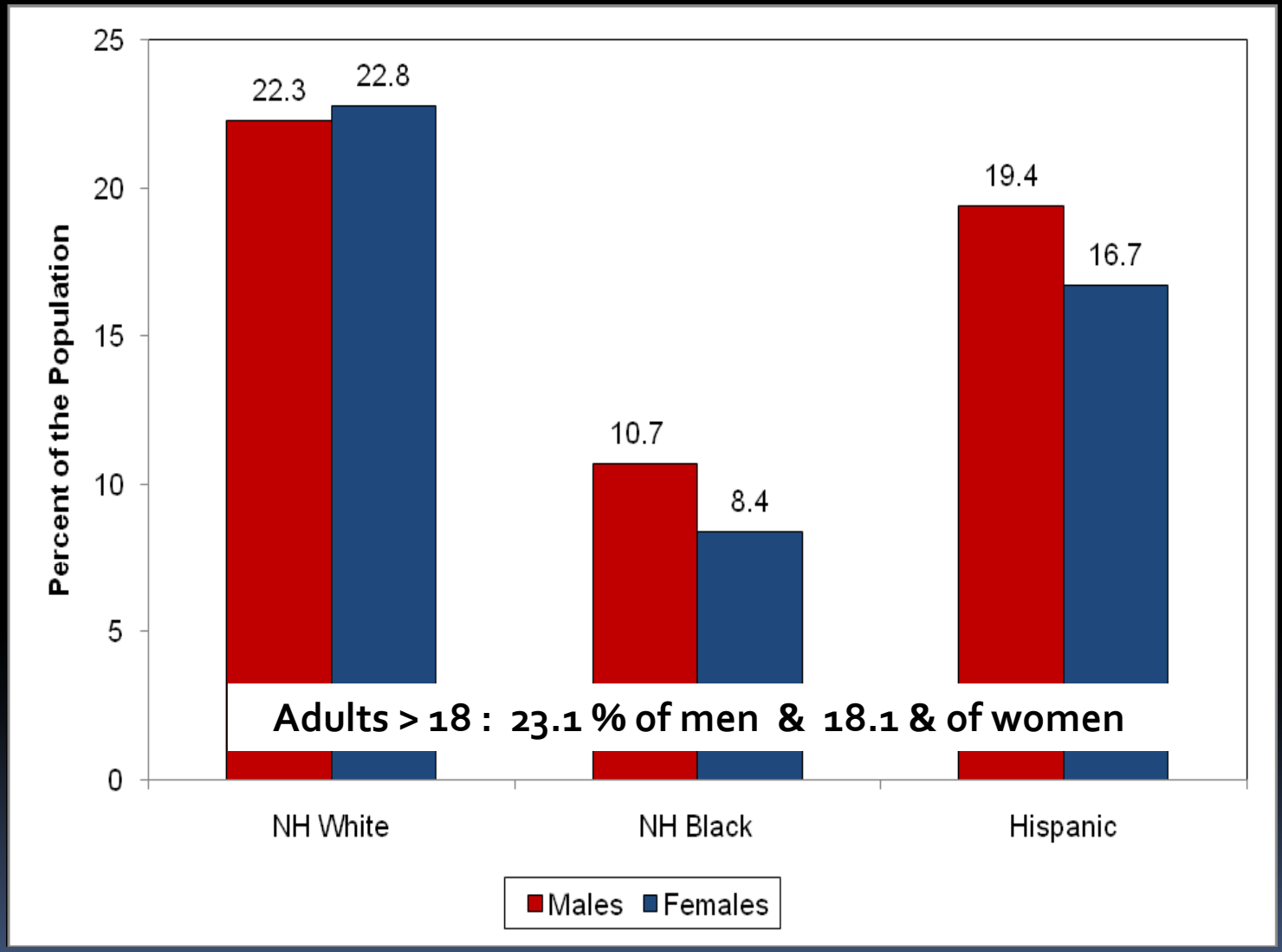
Trends in mean total serum cholesterol among adults ages ≥20 by race and survey year, (NHANES: 1988–1994, 1999–2004 and 2005–2008).

Diagnosed with Diabetes



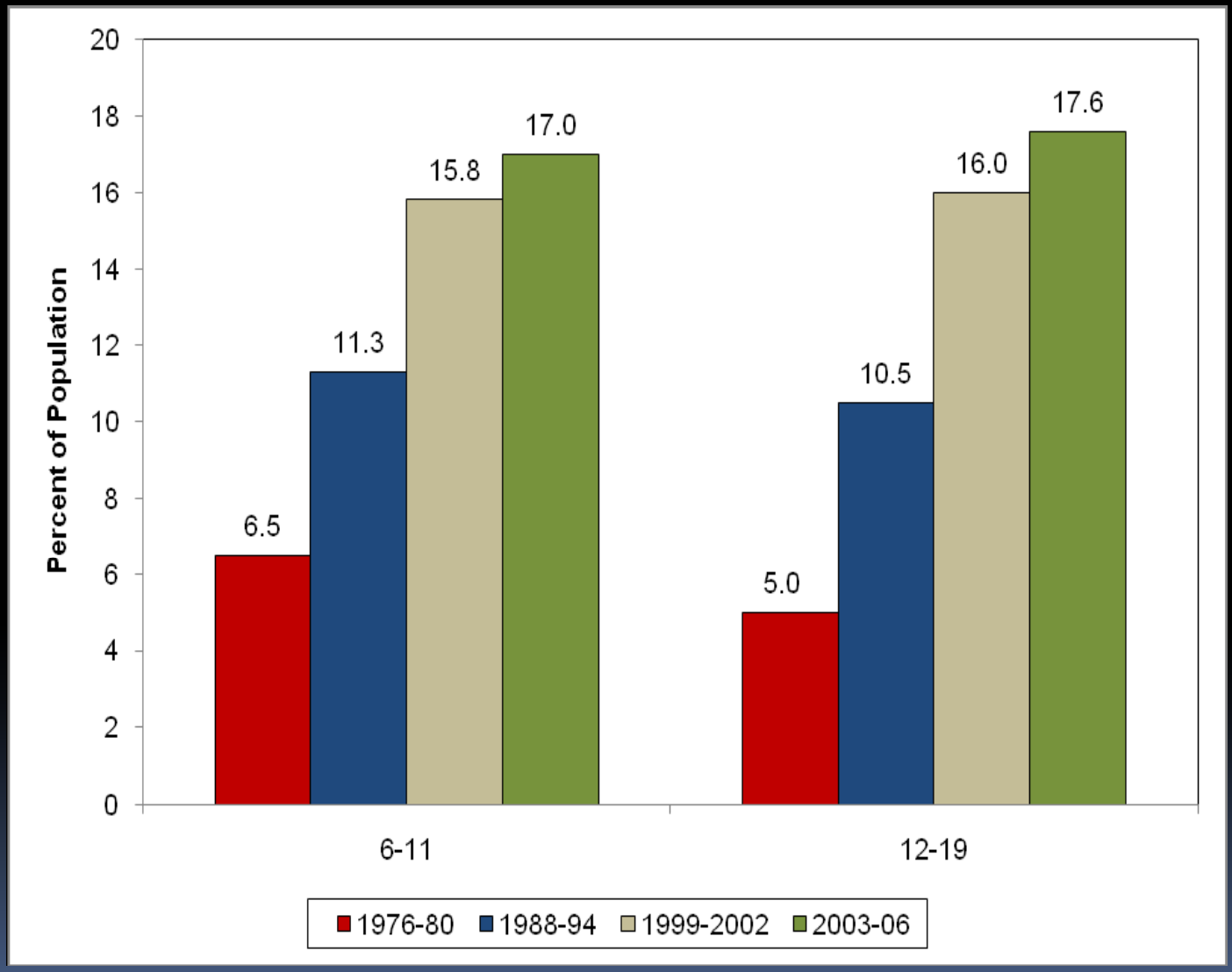
Trends in Diabetes Prevalence in Adults Age 20 and Older by Sex ; NHANES: 1988–94 and 2005–08

HS Students who Smoke by race & gender



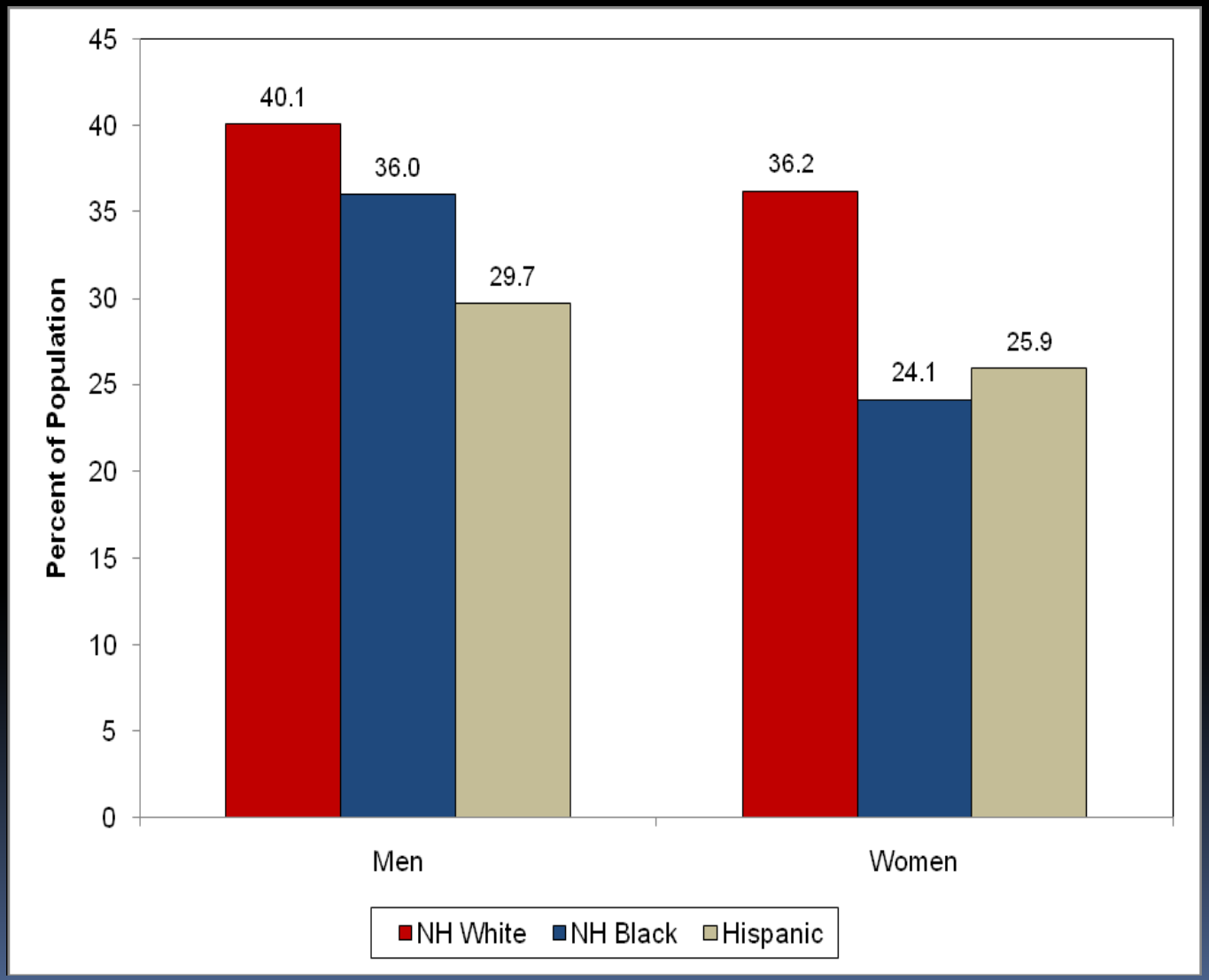
Prevalence of High School Students in Grades 9–12 Reporting Current Cigarette Smoking by Race/Ethnicity and Sex; YRBSS: 2009.

Trends in Overweight US Youth by age & year



Trends in the prevalence of overweight among US children and adolescents by age and survey year. Source: Health, United States, 2009 (NCHS).

Leisure-time Physical Activity: Adults >18 by race & gender



Prevalence of regular leisure-time physical activity among adults > 18 years of age by race/ethnicity and sex (NHIS: 2009).

Contribution of Hypertension

- Prolonged injury/trauma/irritation to the intima due to shearing forces
- Stimulates platelet aggregation
- Stimulates inflammatory response
- Decreased blood flow in narrow arteries in the brain can result in ischemic stroke
- Increased damage to the intima and loss of elastin in arterial smooth muscle will allow for rupture of the vessel resulting in hemorrhagic stroke

Contribution of Hypercholesterolemia

- Most specifically
  - high LDL cholesterol high total cholesterol
- Provides substrate for atherosclerosis process

Contribution of Diabetes

- Associated with increased LDL & reduced HDL cholesterol, hypertension
- Elevated glucose levels damage the basement membranes of blood vessels causing thickening
- Increases platelet aggregation by secretion of Thromboxane A2 (self stimulating)
  - TXA2 is a potent vasoconstrictor
- Provides substrate to damaged brain cells & increases the acidic environment after the stroke has occurred

Contribution of Smoking

- Tobacco in any form is Atherogenic
- 4000 elements w/ nicotine & carbon monoxide the most documented to cause damage to blood vessels
- Generates superoxide anions, increases production and bioavailability of nitric oxide and increases production and release of endothelin.
- Results in endothelia dysfunction, increased tendency to form thromboses and accelerates atherosclerosis
- Nicotine increases LDL cholesterol & triglycerides and decreases HDL cholesterol
- Vasospasm, increases platelet aggregation & decreases oxygen supply to tissues
- Increases catecholamines which increases BP and HR; and suppresses lymphoid tissue, specifically T-cell tissue areas
- Accelerates inflammation
  - Increased ICAMS on Monocytes where they can attach to endothelial selectins within 30 minutes of starting to smoke

Vulnerability d/t Risk Factors

Modified Shwartzman Reaction

- Blood vessel activation
- Thrombogenesis
- Ischemia in a Vascular Territory

- But NOT the microvascular hemorrhages seen with a true schwartzman Reaction

Early Blood Vessel Activation

Inflammatory response of vessel endothelium

- Inflammation
  - Increased permeability of vessel walls – changes in gap junctions
  - Increased endothelial binding of neutrophils & monocytes
  - Vasoactive chemicals promote dilation
  - Chemokines – chemotaxis
  - Margination, pavementation, & emigration of neutrophils & monocytes into tissue
    - PMNs & macrophages - phagocytize
- Acute Phase Response

Big Concept – Acute Phase Response

- IL-1, IL-6 & TNF-alpha
  - Acute Phase Reactants released by liver d/t
    - signal from monocytes by IL-6
    - injured brain cells
    - Increase secretion of IL-6 creates more:
      - C-reactive protein
      - Fibrinogen
      - Mannan-binding Lectin (can start complement cleavage)
- Local activation of vascular endothelium leading to thrombogensis

Thrombogenesis

Inflamed vascular endothelium foundation for:

- Accumulation of platelets, fibrin & thrombus
- Sub-intimal layers (fibrous & smooth muscle) overgrow
- Foam cells migrate into these layers forming fatty plaques
  - Foam cells induced by TNF-alpha
- Plaques provide framework for platelets to adhere
- Fibrin & Thrombin deposition & clot forms

Big Concepts

Platelet Function in thrombosis

- W/ injury Platelets adhere to collagen in exposed connective tissue
- Platelets catalyze reactions between coagulation factors accelerating the formation of thrombin from prothrombin
- Not only create the plug, but also promote clot formation

Thrombus – now what?

- Can stay put and occlude vessel
  - Decreasing blood flow that fits a known vascular territory

OR

- Can be embolus
  - Decreasing blood flow that fits a known vascular territory
- Location, Location, Location
- Real Estate of the Brain – Eloquent Areas
  - Eloquence is in the eye of the beholder
  - Senses, movement & speech areas

Pathophysiology of Risk Factors

End point: Atherosclerosis – structural change in arteries

- inc # of smooth muscle cells
- collection of lipids in the intima in response to trauma or irritation of the intima
- injury stimulates platelet aggregation and the inflammatory response w/ chemotaxis of neutrophils and monocytes to area and integration into vessel wall as foam cells
- medial smooth muscle proliferates & migrates into the intima; forming the plaque
- LDL cholesterol leaks into the vessel wall
- a fibrous cap forms over the plaque
- the lesion slowly grows to decrease vessel diameter
- additional injury may cause rupture of the plaque resulting in thrombus formation and acute occlusion.

Pathophysiology of Stroke
animated

- Creation of blood vessel narrowing
  - http://www.youtube.com/watch?v=Wo_qxVr2cBs&feature=related
- Creation of plaque and plaque rupture
  - http://www.youtube.com/watch?v=GL37q6euND4&feature=related

Pathophysiology of Stroke

Ischemic Cascade

- Series of events when the supply of oxygen is interrupted
- Begins w/in seconds of an ischemic event like a stroke
- Can continue after the normal flow of blood is restored
- This is time delineated, meaning there is a linear timeline for the events to occur

Pathophysiology of Stroke

Ischemia

- CBF decreasing to less than 30 ml/100gm/min (normal CBF = 30-50 ml/100gm/min)

Neurological deficits are seen

- CBF is less than 20 ml/100gm/min
  - abnormal electrical activity occurs –rhythmic
  - cellular function ceases d/t lack of ATP
  - Ion pump failure (Na+/K+ ATPase pump)
- Cellular Death with CBF < 10 ml/100gm/min
  - Stepwise cellular death in core
  - Penumbra at risk – 50% goes on to die

Blood Brain Barrier

Tight junctions between cells that are always maintained

- Ischemia induces Matrix Metalloproteases (MMPs) to breakdown cellular matrix of basal lamina between cells of the blood vessel and the podocytes of the astrocytes
- TNF-alpha activates MMPs & increases vascular permeability
- Immune privileged area of brain is exposed as novel to immune system effector cells
  - Immigration of WBCs and promotion of vasogenic edema.

Late Blood Vessel Activation

- Selectins in vascular endothelium support adhesion & diapedesis
  - In response to IL-1 and TNF-alpha
- W/in hrs of ischemia onset ICAMs
  - Both on the vessel wall & on the neutrophil
  - Increase accumulation of neutrophils at the site of the injury
- IL-1, IL-6 and TNF-alpha
  - Promote systemic inflammation
  - Promoting neutrophils & monocytes to infiltrate the brain tissue through open BBB; start phagocytosis

Cellular Excitotoxicity

- Local cellular energy deficits (No ATP) result in depolarization of neurons and glia
- Depolarization stuns the calcium channels allowing excitotoxic glutamate to flow out of cell
  - Peri-infarct wave depolarizations deplete ATP more
  - Speeds conversion of penumbra to core
- Glutamate self stimulates NMDA receptors allowing inflow of Na+ & Cl- into cell & leads to osmotic lysis
- AKA Necrosis

Necrosis – Hallmark Features

- Cellular calcium increases & continues to rise
- The cell releases amino acids to correct them
- Intracellular acidosis
- The cell wall breaks down
- Organelles in the cell start to die
- The cell itself dies
- Releasing toxins into the surrounding area
- Damaging neighboring cells
  - including cells with an adequate supply of oxygen

Apoptosis - Hallmark Features

- Fas-receptor & Fas-Ligand
- http://www.youtube.com/watch?v=hqhxnWty5jc&feature=related
- Caspases, BcL-2 proteins (BAD, BID BAX)
- Mitochondrial Membrane Transition Pore
- Caspases – NF-kB – all roads lead to Rome
- DNA – 200 base pair ladders
- How soon does this happen?
  - Less than 30 minutes both can be seen on tissue samples

Hemorrhage & Hypertension

- Why does this happen?
- What protein components are not intact in the vessel wall?
- How thick is the vessel wall?
- What is the BBB like?
- Blood Flow & onset of symptoms
  - Ischemia at 30ml/100gm/min
  - Deficits at 20ml/100gm/min
  - Electrical activity of the brain changes,

Stroke

- Learn Stroke
- http://www.learnstroke.com/
  - LearnStroke.com - Stroke Education tool
  - NET SMART for APNs, PAs, & Genera Nursing Staff
- **Stroke Rap**
- http://www.vimeo.com/22111491

ARE you FAST?

- http://youtu.be/bCvIMy_dTmQ
  - Fast cartoon

So What?

- What does your front door look like?
- LA Stroke Screening
  - Basic 3 + some other items like age, hx of seizure, onset of symptoms, ambulatory & blood glucose between 60 - 400
- Cincinnati Stroke Scale
  - Basic 3