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Author: Kenneth A. Jamerson, M.D., 2009

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### Identifying Issues and Overcoming Barriers: Hypertension in African Americans

Kenneth A Jamerson, M.D.
Professor of Cardiovascular Medicine
University of Michigan Health System
Medical Director, Program for Multi-cultural Health

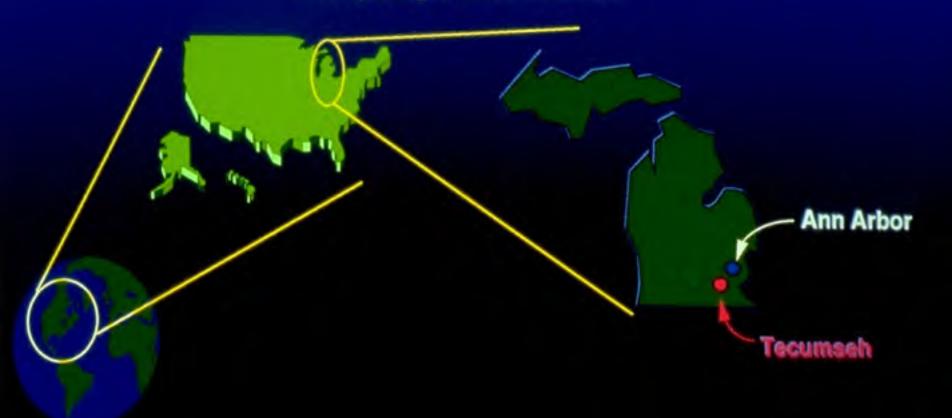


#### Hypertension in African Americans

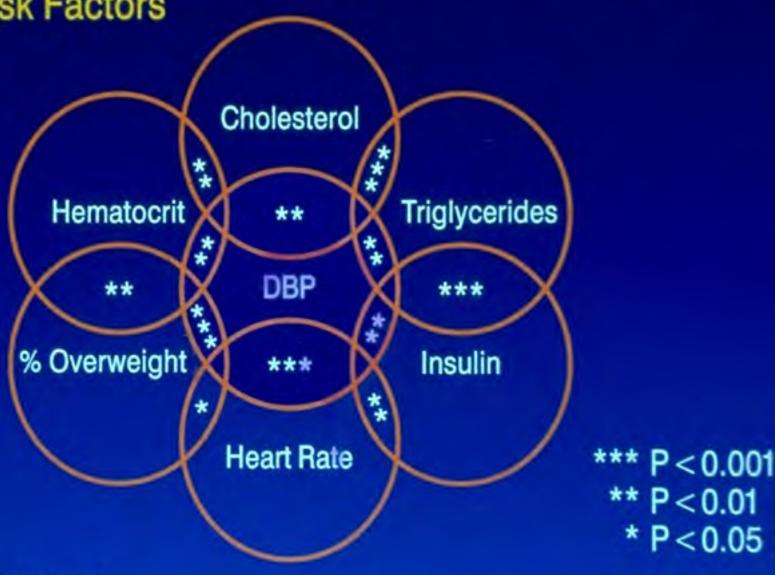
- African Americans develop HTN earlier in life and their average BPs are much higher
- Prevalence of HTN in African Americans in US is among the highest in world
- African American have increased Target Organ Damage compared with whites
  - 4.2X greater rate of ESRD
  - 1.8X greater rate of fatal stroke
  - 1.5X greater rate of heart disease death

### The Tecumseh Blood Pressure Study

A prospective epidemiological study of the antecedents of hypertension and cardiovascular disease in 1100 young men and women



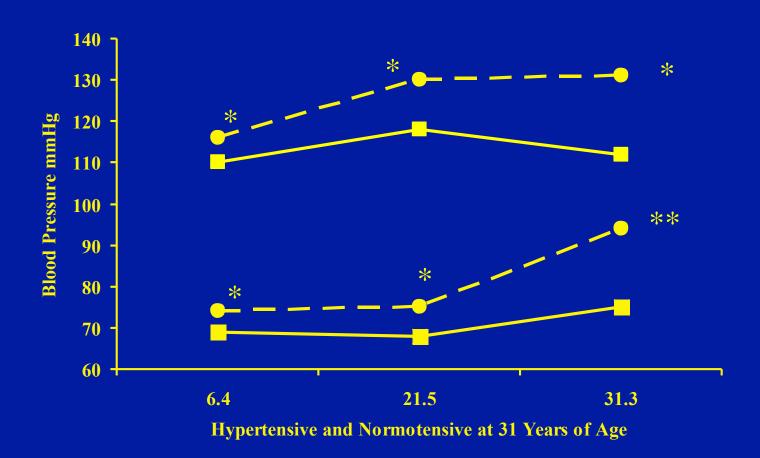
Tecumseh BP Study: Association of DBP and Other CHD Risk Factors



n = 124 (aged 18-38 years) PD-INCL

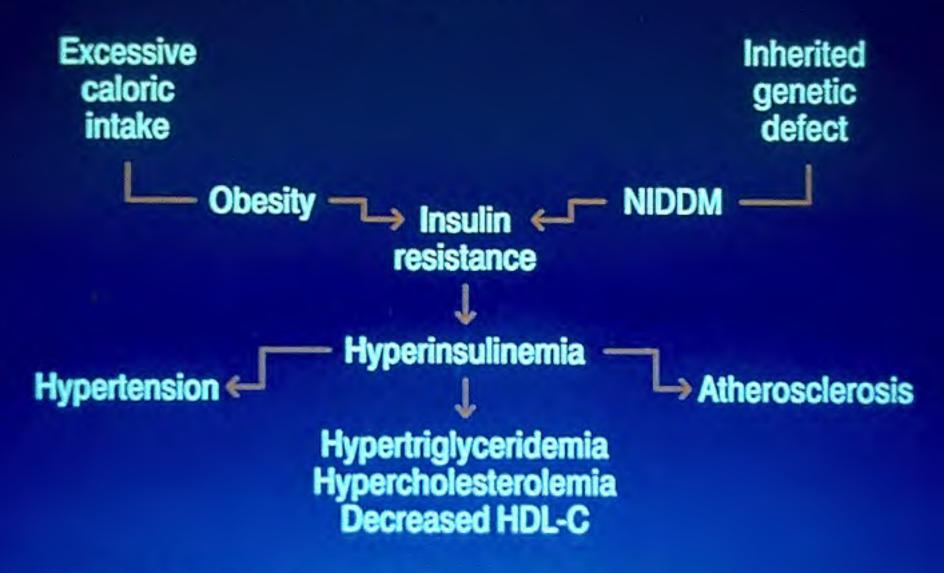
Adapted from Julius et al. JAMA 1990;264:354-358. © Am Med Assn.

### BLOOD PRESSURE TRENDS IN TECUMSEH, MI





### Insulin Resistance Syndrome



#### TREATMENT

#### Lifestyle

- Know your caloric needs to achieve and maintain a healthy weight.
- Know the calorie content of the foods and beverages you consume.
- Track your weight, physical activity, and calorie intake.
- Prepare and eat smaller portions.
- Track and, when possible, decrease screen time (eg, watching television, surfing the Web, playing computer games).
- Incorporate physical movement into habitual activities.
- Do not smoke or use tobacco products.
- If you consume alcohol, do so in moderation (equivalent of no more than 1 drink in women or 2 drinks in men per day).

#### **Impact of Surgery For Obesity**

- Weight had increased by 1.6% in the control group and decreased by 16.1% in the surgical group
- Calorie intake was lower and physical activity was higher in the surgery group than in the control group
- Recovery from high blood pressure, diabetes, high triglyceride levels, and a low HDL ('good') cholesterol level was more frequent in the surgical group than in the control group, both at 2 and 10 years
- After 10 years diabetes had developed in 24% of those in the non-surgery group and 7% in the surgery group

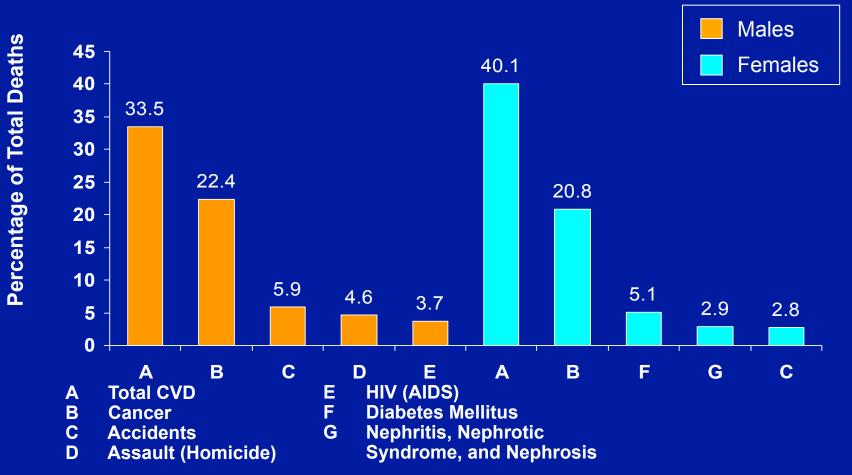
### **Potential Drugs for Metabolic Syndrome**

| LIPIDS    | HTN      | DIAB      | OBESE       | coag   |
|-----------|----------|-----------|-------------|--------|
|           |          |           |             |        |
| Stains    | Diuretic | SU        | orlistat    | ASA    |
| ezetimibe | Ace/Arb  | MF        | sibutramine | clopid |
| Bile seq  | a block  | ins       | rimonabant  |        |
| niacin    | Bblock   | gilt      |             |        |
| fibrate   | ССВ      | PPG reg   |             |        |
|           |          | incretins |             |        |
|           |          |           |             |        |

### INCIDENCE OF HYPERTENSION IN BLACK AND WHITE POPULATIONS BY AGE AND SEX



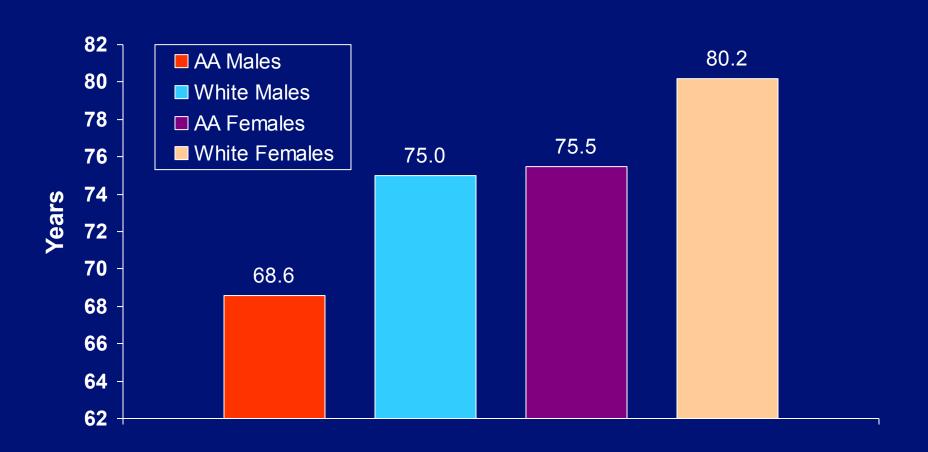
### Leading Causes of Death for African American Males and Females



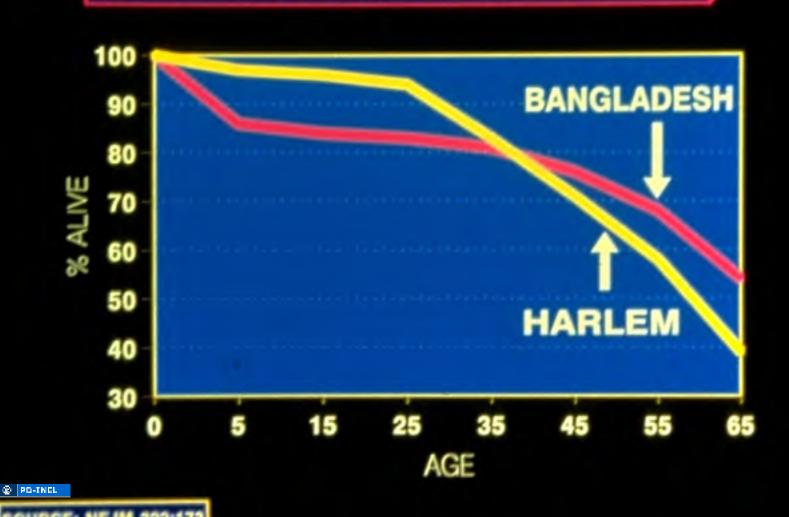
CVD=cardiovascular disease.

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### **Estimated Life Expectancy: 2001**



### SURVIVAL OF MEN: HARLEM (U.S.) & BANGLADESH



### Is There a Unique Etiology for Hypertension in African Americans?

#### Hepeated Observations on Hacial Differences in the Pathophysiology of Hypertension

|                                   | Black vs. White  |   | Family History |       | Genetic Influence |       |
|-----------------------------------|--|---|----------------|-------|-------------------|-------|
| Variable of Interest              | Normotensive   | Hypertensive  | Black          | White | Black             | White |
| Kidney:                           |  | 200   |                | - 6   |                   |       |
| Creatinine clearance              | B=W  | B <w< td=""><td>?</td><td>?</td><td>?</td><td>Yes</td></w<>           | ?              | ?     | ?                 | Yes   |
| Renal blood flow                  | ?  | B <w< td=""><td>?</td><td>?</td><td>?</td><td>?</td></w<>             | ?              | ?     | ?                 | ?     |
| Excretion of Na <sup>+</sup> load | B <w< td=""><td>B<w< td=""><td>?</td><td>+&lt;-</td><td>?</td><td>Yes</td></w<></td></w<>      | B <w< td=""><td>?</td><td>+&lt;-</td><td>?</td><td>Yes</td></w<>      | ?              | +<-   | ?                 | Yes   |
| Increase BP with Na+ load         | B>W  | ?   | ?              | ?     | ?                 | ?     |
| Decrease BP with low Na+          | B>W  | B>W   | ?              | ?     | ?                 | Yes   |
| Fractional Excret, Li+            | B=W  | B=W   | ?              | ?     | ?                 | ?     |
| Plasma Renin Activity             | B <w< td=""><td>B<w< td=""><td>+&gt;-</td><td>+&gt;-</td><td>?</td><td>Yes</td></w<></td></w<> | B <w< td=""><td>+&gt;-</td><td>+&gt;-</td><td>?</td><td>Yes</td></w<> | +>-            | +>-   | ?                 | Yes   |
| Aldosterone                       | B=W  | B=W   | ?              | ?     | ?                 | ?     |
| Sympathetic (UNE/PNE)             | B=W  | B=W   | +=-            | +<-   | ?                 | Yes   |
| Response to Stressors             | B=W  | B>W   | +>-            | +>-   | ?                 | Yes   |
| Dopamine 8-Hydroxylase            | B <w< td=""><td>B<w< td=""><td>?</td><td>?</td><td>?</td><td>Yes</td></w<></td></w<>           | B <w< td=""><td>?</td><td>?</td><td>?</td><td>Yes</td></w<>           | ?              | ?     | ?                 | Yes   |
| Kallikrein                        | B <w< td=""><td>B<w< td=""><td>+&gt;-</td><td>+&gt;-</td><td>?</td><td>?</td></w<></td></w<>   | B <w< td=""><td>+&gt;-</td><td>+&gt;-</td><td>?</td><td>?</td></w<>   | +>-            | +>-   | ?                 | ?     |
| Red Cell Transport                | B <w< td=""><td>B<w< td=""><td>?</td><td>+&lt;-</td><td>?</td><td>Yes</td></w<></td></w<>      | B <w< td=""><td>?</td><td>+&lt;-</td><td>?</td><td>Yes</td></w<>      | ?              | +<-   | ?                 | Yes   |
| Atrial Natriuretic Factor         | ?  | B>W   | ?              | ?     | 2                 | 2     |

Natriuretic Hormone + = family history positive for hypertension; - = family history negative; = = groups are similar; BP = blood pressure; UNE = urinary norepinephrine; PNE = plasma norepinephrine; ? = unknown Updated from Grim, et al and Savage, et al. Saunders: Cardiovascular Disease in Blacks, F.A.

Davis Company, 1991. O PO-TNEL

### Physiologic Differences Between Blacks and Whites

- Plasma Renin Activity
- Renal Function
- Vascular Reactivity
- Sodium Sensitivity
- Expanded Plasma Volume

# Ethnicity and Plasma Volume in Hypertension

- 172 consecutive cases were examined
- Arbitrary cut point for plasma volume were established
- Subject with normal plasma volume were excluded for the analysis

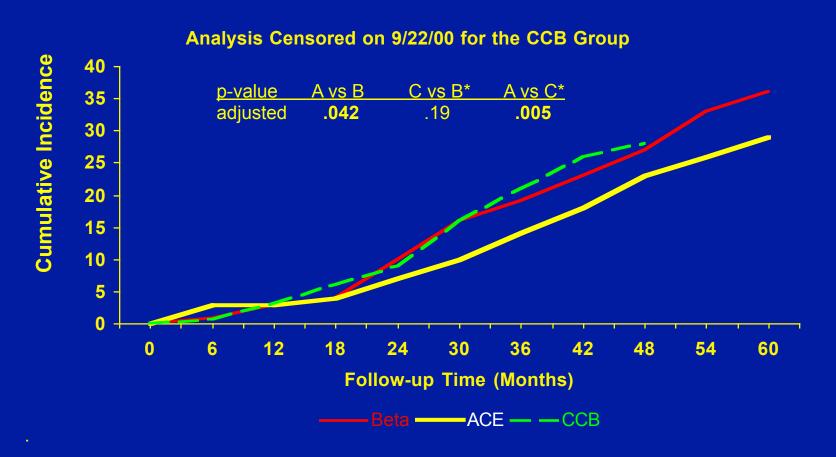
### African American Study of Kidney Disease and Hypertension

### ACHIEVED BLOOD PRESSURE IN AASK

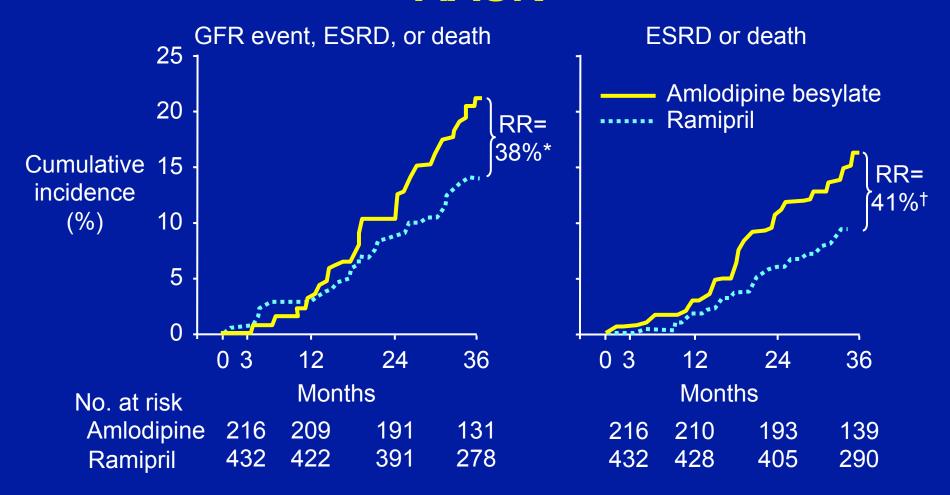
|                 | ACE   | CCB   | ВВ    | LOW   | USUAL |
|-----------------|-------|-------|-------|-------|-------|
| SBP             | 133.6 | 131.4 | 134.2 | 126.9 | 140.0 |
| DBP             | 81.1  | 80.7  | 80.9  | 76.6  | 85.2  |
| NEED FOR STEP 5 | 28%   | 24%   | 32%   | 35%   | 23%   |

PD-INEL K. Jamerson

# Cumulative Incidence of Confirmed Declining GFR Event, Dialysis or Death by Drug group (Data as of 10/19/01)



### **Incidence of Renal Events and Death: AASK**



GFR, glomerular filtration rate; ESRD, end-stage renal disease; RR, adjusted risk reduction. \*P=0.005 (95% CI, 13-56%); †P=0.007 (95% CI, 14-60%).

## IMPLICATIONS OF THE AASK STUDY

- Aggressive control of blood pressure can eliminate ethnic differences in ESRD
- Inadequate treatment of hypertension may causes excess risk of target organ disease.
- Cultural rather than genetic differences may underlay the excess risk of hypertensive ESRD

 ARE OTHER ETHNIC GROUPS AT RISK FOR CARDIOVASCULAR DISEASE?

Kenneth A. Jamerson

# Prevalence of MI in Asian Indians Living in the U.S.: Introduction

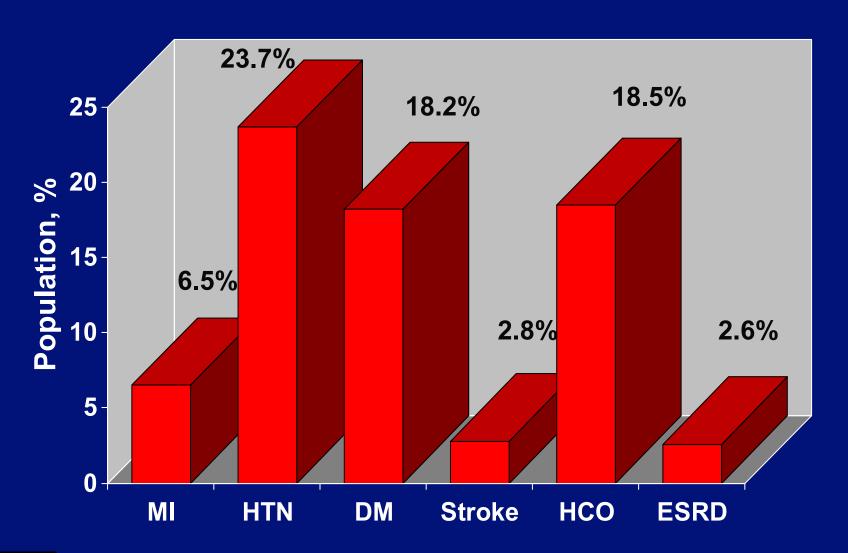
- Approx. 1.9 million Asian Indians currently live in the United States and are one of the fastest growing ethnic minorities in this country
- Data on epidemiology of MI in this community is very limited
- Present study estimates the prevalence of MI and associated risk factors in this group

# Prevalence of MI in Asian Indians Living in the U.S. -- Results

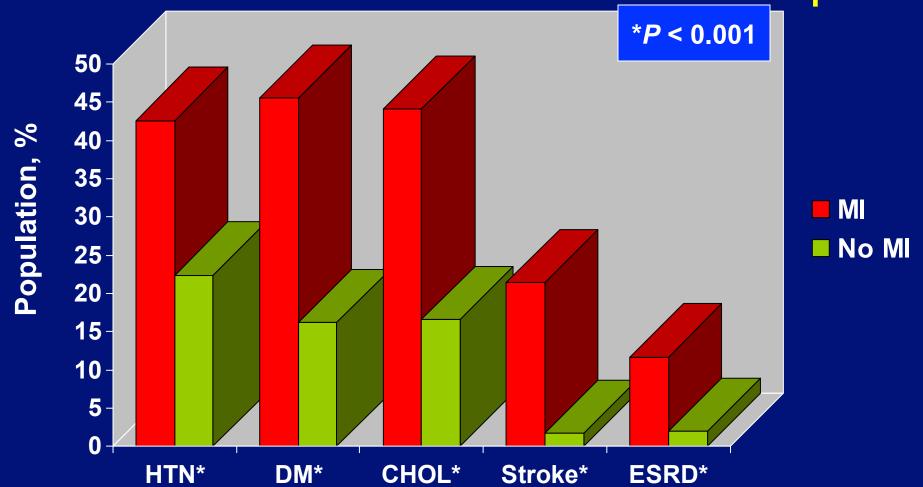
- Total population surveyed—1046 adults\*
- 537 men (51.3%), 509 women (48.7%)
  - sex ratio- 1.06
- Mean age of the population 53.7 yrs (± 11.3 yrs)
  - ages ranged from 17 to 87 yrs
- Mean age for men 53.7 yrs (± 11.3 yrs);
   women 51.9 yrs (± 11.3 yrs)

\*Members of Bochasanwasi Shri Akshar Purushottam Swaminarayan Sanstha, a prominent Hindu sect

### Asian Indians Living in the U.S. --Prevalence of MI and Risk Factors



Prevalence of MI in Asian Indians
Living in the U.S. -- Distribution of Risk
Factors in MI and Control Group



# Prevalence of MI: Data from India

- In two large studies from New Delhi, India, the prevalence of MI between ages 25–64 was 1.05%
- Prevalence among Asian Indian immigrants to the U.S. was 5.31% in this age group

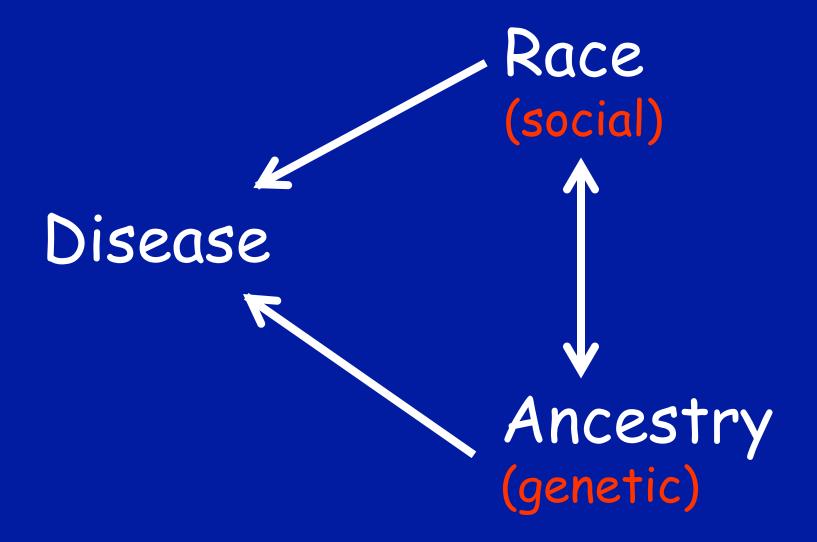
### Conclusions

- Prevalence of MI among Asian Indians in the United States is higher than in India
- It approaches the same level as whites and slightly higher than Hispanics and blacks in the United States
- HTN, HCO, DM, ESRD, stroke and FH of MI were independent risk factors for MI in this group

Source: Nanda NC et al. Unpublished.

# Models to explain Health Disparities

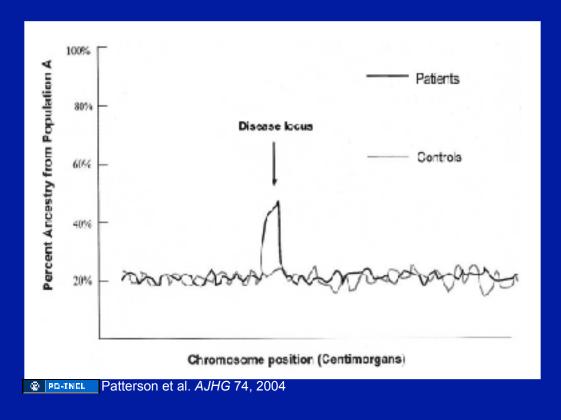
- Racial Genetic Model
  - Cause of HD: population differences in the distribution of genetic variants
- Health-behavior Model
  - ➤ Cause of HD: differences between R/E groups in the distribution of *individual behaviors* related to health such as diet, exercise, and tobacco use
- > SES Model
  - Cause of HD: over-representation of some R/E groups within lower SES
- Psychosocial Stress Model
  - Cause of HD: stresses associated with minority group status, especially the experience of racism and discrimination



Although much genetic variation (85-90%) is shared among all human populations, about 5% of SNPs have high levels of allele frequency differential ( $\delta$ >50%). We call these markers Ancestry Informative Markers (AIMs).

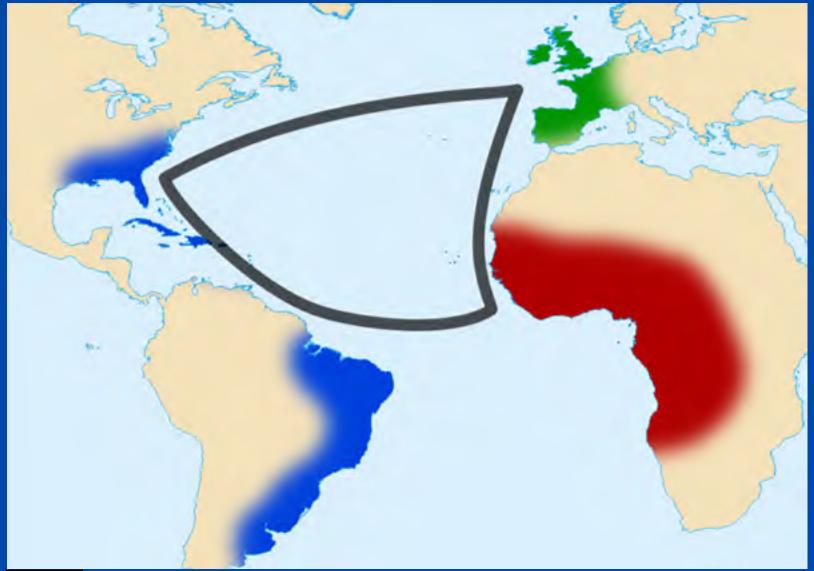
#### Admixture mapping for disease genes

Disease gene identification may be facilitated if we know which parts of the genome the cases and controls have inherited at a disproportionate rate from one of the parental populations.



### **Era of Genomic Ancestry and challenges** related to Health.

- 1. Group definition and membership.
- 2. Can we accurately assess genomic ancestry?
- 3. How does genomic ancestry relate to skin color and possibly SES?
- 4. How useful is genomic ancestry for informing us about disease risk?
- 5. Health Disparities: are they due to biological differences?
- 6. How do we prevent repeating the negative past abuses of "race".



☑ Semhur adapted from Francois Nancy (wikipedia)

### The Future

Whole genome Association

Population Genomics

**HuGENet** 

Gene Expression

Model Systems

Pharmacogenomics

**Proteomics** 

#### **Additional Source Information**

for more information see: http://open.umich.edu/wiki/CitationPolicy

Slide 4: Chobanian AV et al. JAMA. 2003;289:2560-2572; Heart Disease and Stroke Statistics- 2005 Update, AHA.; ALLHAT Investigators. JAMA. 2002;288:2981-2997

Slide 5: Source Undetermined

Slide 6: S. Julius, et al: JAMA 264:354-358, 1990

Slide 7: S. Julius, et al: JAMA 264:354-358, 1990

Slide 8: DeFronzo RA, Ferrannini E, Diabetes Care, 1991, 14:173-194. American Diabetes Association

Slide 11: K. Jamerson

Slide 12: Source Undetermined

Slide 13: Adapted from Heart Disease and Stroke Statistics—2004 Update. American Heart Association; 2003:6.

Slide 14: National Vital Statistics Reports. 2004;52:33–34.

Slide 15: NEJM 322:173

Slide 17: Updated from Grim, et al and Savage, et al. Saunders: <u>Cardiovascular Disease in Blacks</u>, F.A. Davis Company, 1991.

Slide 19: Source: Chysant 1979

Slide 21: K. Jamerson

Slide 22: Source Undetermined

Slide 23: Agodoa LY et al. *JAMA*. 2001;285:2719-2728.

Slide 26: Nanda NC et al. Unpublished.

Slide 27: Nanda NC et al. Unpublished.

Slide 28: Nanda NC et al. Unpublished.

Slide 29: Nanda NC et al. Unpublished.

Slide 30: Gopinath N et al. J Assoc Physicians India. 1995;43:30-33.; Chadha SL et al. Indian J Med Res. 1990;92:424-430.

Slide 31: Source: Nanda NC et al. Unpublished.

Slide 33: K. Jamerson

Slide 35: Patterson et al. AJHG 74, 2004

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