UNDERSTANDING THE RISING TIDE OF DIABETIC CVD

THE STRONG HEART STUDY

Barbara V. Howard, PhD
MedStar Research Institute
Kelly West, MD (1925-1980)

- George Lynn Cross Research Professor of Biostatistics and Epidemiology, University of Oklahoma Health Sciences Center
- Chair, Board of Regents, National Library of Medicine
- Chair, IDF International Epidemiology Group
- WHO Expert Panel on Diabetes 1979
Kelly West, MD (1925-1980)

• 1972 - Initiated the Lawton Area Indian Project to study and control diabetes in American Indians
• ‘Diabetes in American Indians and Other Native Populations in the New World’ *Diabetes*, October, 1974
• Late 70s - congressional appropriation for diabetes treatment programs for American Indians
The Strong Heart Study

A study of cardiovascular disease in American Indians, supported by the National Heart, Lung, and Blood Institute and the Indian Health Service
Outline

• The Strong Heart Study

• The epidemic of CVD in diabetes

• Risk factors - conventional and new
Variations in CHD in American Indians by IHS Area - 1975
*ICD-9 Codes 390–448*

Rate/100,000

[Bar chart showing variations in CHD rates by IHS area, with rates ranging from 0 to 500 per 100,000 population. The chart includes areas such as DAK, ALA, BEM, BIL, NAS, NAV, OK, PHX, POR, TUC, and USA.]
## Hospital Admissions

*Phoenix PHS Indian Hospital, 1957-1966*

<table>
<thead>
<tr>
<th>Tribe</th>
<th>Myocardial infarction (n = 61)</th>
<th>All hospital admissions (N = 24,121)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>%</td>
</tr>
<tr>
<td>Pima</td>
<td>28</td>
<td>45.9</td>
</tr>
<tr>
<td>Apache</td>
<td>7</td>
<td>11.5</td>
</tr>
<tr>
<td>Navajo</td>
<td>1</td>
<td>1.6</td>
</tr>
<tr>
<td>Papago</td>
<td>5</td>
<td>8.2</td>
</tr>
<tr>
<td>Hopi</td>
<td>7</td>
<td>11.5</td>
</tr>
<tr>
<td>Other SW Indians</td>
<td>13</td>
<td>21.3</td>
</tr>
<tr>
<td>Total</td>
<td>61</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Strong Heart Study Objectives

- Measure rates of CVD
- Measure levels of risk factors
- Determine effect of diabetes on CVD
- Follow up to evaluate determinants of CVD
- Follow up to evaluate changes in risk factors
Strong Heart Study Population

4549 American Indians ages 45-74 years

**Arizona**: Pima/Maricopa/Papago in the Gila River, Salt River, and Ak-Chin Indian communities

**Oklahoma**: Apache, Caddo, Comanche, Delaware, Fort Sill Apache, Kiowa, and Wichita

**South/North Dakota**: Oglala Sioux and Cheyenne River Sioux (SD) and the Spirit Lake Tribe in the Fort Totten area (ND)
Strong Heart Study  
1988-2003

• Community Mortality Study
  35–74-year-old men and women
  Deaths between 1984 and 1994
  Medical record review of all possible CVD deaths

• Physical Examinations of Cohort
  ECG, Cardiac and Carotid ECHO
  Anthropometry, BP, ABI, PFT
  Blood, urine and DNA samples
  Medical and medication history
  Diet, PA, QOL, smoking, alcohol
Strong Heart Study
1988-2003

• **Cohort Surveillance**
  Yearly contact
  Medical record review for all cause and CVD
  Mortality and nonfatal CVD

• **Family Study**
  120 families of 30 members each
  Examination similar to that of cohort
  10cM genetic map
Community Involvement

- Study design and implementation
- American Indian investigators and staff
- Medical care to participants
- Data used for community health initiatives
- Education of community youth
- Participation in community health initiatives
The Strong Heart Study

- Sgufk Ibthag Muschuma (O’odham)
- Natsu witu Pihi (Comanche)
- Tawahe Cante Suta Woospe (Lakota)
The Rising Tide of CVD
CVD and Total Mortality Rates
SD/ND vs Strong Heart Study, Men, 1986

per 10,000

45-54 55-64 65-74

S/ND  SHS  '  S/ND  SHS  '  S/ND  SHS

CVD Mortality  Total Mortality
CVD and Total Mortality Rates

Arizona vs Strong Heart Study, Men, 1986

per 10,000

45-54  55-64  65-74

AZ  SHS  '  AZ  SHS  '  AZ  SHS

CVD Mortality  Total Mortality
CHD Incidence in American Indians Compared to ARIC Population

CHD includes fatal and nonfatal events plus revascularization
Prevalence of Atherosclerotic Plaque in SHS and ARIC/CHS

![Graph showing prevalence of atherosclerotic plaque across different age groups for SHS and ARIC/CHS. The x-axis represents age groups (45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, >79 years) and the y-axis represents prevalence (%). The SHS data is shown in purple bars, and the ARIC/CHS data is shown in yellow bars. Significant differences are indicated by P<0.01.]
Trends in CVD Mortality

*The Strong Heart Study*

![Graph showing trends in CVD mortality for Arizona, Oklahoma, and Dakota for women and men over 3-year periods from 1984-1986 and 1991-1993.](image)
Prevalence of Diabetes
Strong Heart Study, by Gender and Center

Women

Men

%}

AZ OK ND/SD AZ OK ND/SD

Diabetes

IGT
<table>
<thead>
<tr>
<th></th>
<th>Percent/8.2 year - Follow up</th>
<th>Number/1000 per year</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nondiabetic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>17.0</td>
<td>21</td>
</tr>
<tr>
<td>Women</td>
<td>8.5</td>
<td>10</td>
</tr>
<tr>
<td><strong>Diabetic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>23.5</td>
<td>30</td>
</tr>
<tr>
<td>Women</td>
<td>20.8</td>
<td>25</td>
</tr>
</tbody>
</table>

**CVD by Gender and Diabetic Status**
### Contribution of Diabetes to CHD

*Strong Heart Study*

<table>
<thead>
<tr>
<th></th>
<th>Women</th>
<th>Men</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hazard Ratio</td>
<td>6.3</td>
<td>3.1</td>
</tr>
<tr>
<td>Prevalence</td>
<td>60%</td>
<td>50%</td>
</tr>
<tr>
<td>PAR</td>
<td>76%</td>
<td>51%</td>
</tr>
</tbody>
</table>
• CVD rates in American Indians are now higher than in the general U.S. population

• The rates are increasing

• The majority of CVD occurs in people with diabetes
Effect of Epidemic of Diabetes on Duration-Related Complications

- Cumulative DM cases
- Prevalence of complications (%) among those with DM
- % with DM>15-year duration

16 new cases/yr. Death occurs after 30-year DM; Complication incidence 50% after 15-year DM. After 30–years, no. of new cases equals no. of deaths.
Change in U.S. CHD Death Rate

1950-2010 projection

NIH, NHLBI. Morbidity & Mortality 1996 Chartbook on Cardiovascular, Lung, and Blood Diseases, US DHHS, 1996
• **Conventional Risk Factors**

• **New Risk Factors**
CVD Risk Factors

(*)HR

<table>
<thead>
<tr>
<th>Factor</th>
<th>No Diabetes</th>
<th>Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>1.04 (p &lt; 0.0001)</td>
<td>1.02 (p &lt; 0.0001)</td>
</tr>
<tr>
<td>Gender</td>
<td>1.39 (p = 0.01)</td>
<td>1.13 (p = 0.15)</td>
</tr>
<tr>
<td>Smoke</td>
<td>1.12 (p &lt; 0.0001)</td>
<td>1.12 (p &lt; 0.0001)</td>
</tr>
<tr>
<td>SBP (10mm)</td>
<td>1.12 (p = 0.01)</td>
<td>1.34 (p &lt; 0.01)</td>
</tr>
<tr>
<td>LDL (30mg)</td>
<td>0.94 (p = 0.01)</td>
<td>0.92 (p &lt; 0.001)</td>
</tr>
<tr>
<td>HDL (5mg)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

8.6- year follow up
n = 261

8.2- year follow up
n = 420
Distribution of LDL Cholesterol in American Indians

LDL Cholesterol (mg/dl)
Distribution of Systolic Blood Pressure in Diabetic and Nondiabetic American Indians

![Histograms showing distribution of systolic blood pressure in diabetic and nondiabetic American Indians.](image-url)
## RR for Total Cholesterol in Framingham vs Strong Heart Study

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th></th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
<th>Women</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>FHS</td>
<td></td>
<td>SHS</td>
<td></td>
<td>FHS</td>
<td></td>
<td>SHS</td>
</tr>
<tr>
<td>160-199</td>
<td>Ref</td>
<td></td>
<td>Ref</td>
<td></td>
<td>Ref</td>
<td></td>
<td>Ref</td>
</tr>
<tr>
<td>200-239</td>
<td>1.19</td>
<td>1.63</td>
<td>1.23</td>
<td>1.09</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>240-279</td>
<td>1.66</td>
<td>2.31</td>
<td>1.28</td>
<td>1.55</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥ 280</td>
<td>1.93</td>
<td>2.87</td>
<td>1.71</td>
<td>2.57</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Implementation of Findings

• Health Care Providers
  – Increased awareness of CVD because of SHS
  – Development of Indian-specific “Framingham” predictive equation

• Prevention
  – Stop Atherosclerosis in Native Diabetics Study
  – 3-year randomized intervention for LDL and BP
    • usual targets
    • LDL 75mg/dl, BP 115/75
    • endpoint carotid and coronary ECHO
• Conventional Risk Factors

• New Risk Factors
Age- and Center-Adjusted Hazard Ratios for Albuminuria as Predictor of CVD

Micro-albuminuria

Macro-albuminuria

Women, Men
<table>
<thead>
<tr>
<th></th>
<th>No Albuminuria N=685</th>
<th>Micro-Alb N=519</th>
<th>Macro-Alb N=372</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV Mass (gms)</td>
<td>155 ± 36 a</td>
<td>160 ± 39 a</td>
<td>181 ± 50 b</td>
</tr>
<tr>
<td>LV Hypertrophy (%)</td>
<td>23% a</td>
<td>31% b</td>
<td>49% c</td>
</tr>
<tr>
<td>Fractional Shortening (%)</td>
<td>35 ± 6 a</td>
<td>35 ± 6 a</td>
<td>33 ± 8 b</td>
</tr>
<tr>
<td>Ejection Fraction (%)</td>
<td>64 ± 8 a</td>
<td>63 ± 9 a</td>
<td>60 ± 12 b</td>
</tr>
</tbody>
</table>

Values with same superscript are not significantly different.
Prevalence of Abnormal Diastolic Function Based on Albuminuria Status

* Adjusted covariates: Age, gender, BMI, SBP, HgbA1C, coronary artery disease, ejection fraction, LV mass
Distribution of Fasting Insulin Levels

Strong Heart Study
IR and MS as Predictors of Diabetes

- Tertile of HOMA IR:
  - 1st Tertile
  - 2nd Tertile
  - 3rd Tertile

- ATP III Metabolic Syndrome:
  - Absent
  - Present
IR and MS as Predictors of CVD

Tertile of HOMA IR

1st Tertile 2nd tertile 3rd Tertile

Percent (%)

Absent Present

ATP III Metabolic Syndrome

Percent (%)
• The insulin resistance syndrome is not a strong determinant of CVD in non-diabetic American Indians

• Among the CVD risk factors and parameters of CV function, only lipoproteins and PAI-1 change significantly with increasing IR

• IR leads to diabetes before it leads to CVD
Inflammatory Markers?
Age- and Center-Adjusted Hazard Ratios for Fibrinogen as Predictor of CVD
## Elevated Fibrinogen and ECHO Abnormalities

<table>
<thead>
<tr>
<th></th>
<th>OR (adj)</th>
<th>95% CI</th>
</tr>
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<tbody>
<tr>
<td><strong>ECHO</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVH</td>
<td>1.38</td>
<td>1.10-1.73</td>
</tr>
<tr>
<td>Art. Stiffness</td>
<td>1.45</td>
<td>1.11-1.88</td>
</tr>
<tr>
<td>Low Contr.</td>
<td>1.05</td>
<td>0.70-1.57</td>
</tr>
<tr>
<td>Any of above</td>
<td>1.51</td>
<td>1.22-1.87</td>
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## CRP - Population Comparisons

<table>
<thead>
<tr>
<th>Study</th>
<th>Median/ Mean*</th>
<th>Pop. based</th>
<th>Comments</th>
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<tbody>
<tr>
<td>SHS</td>
<td>3.8</td>
<td>Yes</td>
<td>Am. Indian</td>
</tr>
<tr>
<td>NHANES</td>
<td>2.1</td>
<td>Yes</td>
<td>US &gt; 40 years</td>
</tr>
<tr>
<td>PRINCE</td>
<td>2.05</td>
<td>Yes</td>
<td>US &gt; 21 years</td>
</tr>
<tr>
<td>CHS</td>
<td>2.67*</td>
<td>Yes</td>
<td>US AV = 77 years</td>
</tr>
<tr>
<td>IRAS</td>
<td>3.8*</td>
<td>No</td>
<td>Diabetes</td>
</tr>
<tr>
<td>NHS</td>
<td>2.8</td>
<td>No</td>
<td>Women</td>
</tr>
<tr>
<td>MRFIT</td>
<td>1.95</td>
<td>No</td>
<td>High-Risk Men</td>
</tr>
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</table>
## CRP and CVD

<table>
<thead>
<tr>
<th>Model</th>
<th>RR for CRP</th>
<th>(95% CI)</th>
<th>Covariates</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.2</td>
<td>(1.05-1.38)</td>
<td>none</td>
</tr>
<tr>
<td>2</td>
<td>1.29</td>
<td>(1.12-1.49)</td>
<td>gender, age, center, BMI, smoke, meds</td>
</tr>
<tr>
<td>3</td>
<td>1.22</td>
<td>(1.05-1.40)</td>
<td>plus HT, DM, albuminuria</td>
</tr>
<tr>
<td>4</td>
<td>1.15</td>
<td>(0.99-1.35)</td>
<td>plus PAI-1, fibrinogen</td>
</tr>
</tbody>
</table>
• CRP high in populations with high rates of obesity and diabetes

• CRP, therefore, is a weaker predictor of CVD

• Fibrinogen’s stronger predictive value may reflect its relation to thrombosis as well as inflammation
Adiponectin (ACRP30/ adipoQ)

- Produced by adipocytes, but plasma levels lower in obesity
- Sequence homologies to collagen and complement factor C1q
- Metabolic actions - related to increased insulin sensitivity
- Relations to inflammation
  - Structural homologies to TNFα
  - Inhibits NF-κB signaling
Adiponectin as Predictor of CHD: Multivariate Analysis

- Model 1: Waist, age, % FAT, SBP, albuminuria
- Model 2: + HDL, LDL
- Model 3: + QUICKI

Odds Ratio CHD [95% CI]
Adiponectin as a Predictor of CHD: 
Subgroups (Model 2)
Adiponectin is:

• Related to BMI (-), fasting insulin (-), HDL
• Protective against later rises in fasting BG
• Positively related to albumin excretion
Markers of Pre-Clinical Disease?
Abnormalities of Cardiac Structure and Function in Diabetic and Non-Diabetic SHS Participants Without Known Coronary Disease

All p<0.001
Relation of LV Mass/Height^{2.7} to Subsequent CVD and Non-CVD Mortality

* Adjusted for covariates
LV Function and CVD Mortality in Diabetes

- **LVEF**
  - EF <40%
  - 40-54%
  - >=55%

- **Abnormal LV Filling**
  - E/A > 1.5

- Cumulative Survival
  - Months
    - 0, 10, 20, 30, 40, 50
  - Cumulative Survival
    - 1.00, 0.98, 0.96, 0.94, 0.92, 0.90

- Time (months)
  - -10, 0, 10, 20, 30, 40, 50, 60, 70
  - Cumulative Survival
    - 1.1, 1.0, 0.9, 0.8
Univariate Relative Risk of Mortality in Diabetes

- Cardiovascular Mortality:
  - ST Depression: 9.54 (p<0.001)
  - PCA Ratio: 3.17 (p=0.001)
  - QTc: 2.07 (p=0.018)

- All-Cause Mortality:
  - ST Depression: 4.68 (p<0.001)
  - PCA Ratio: 2.11 (p=0.001)
  - QTc: 1.95 (p=0.0004)
Multivariate Relative Risk of Mortality in Diabetes*

- ST Depression: p=0.0009, Relative Risk = 3.68
- PCA Ratio: p=0.006, Relative Risk = 2.61
- QTc: p=0.082, Relative Risk = 1.87
- Cardiovascular Mortality: p=0.002, Relative Risk = 2.36
- All-Cause Mortality: p=0.001, Relative Risk = 2.03

* Adjusted for age, sex, BMI, BP, HDL, LDL, TGs, albuminuria, alcohol use, smoking, prevalent CHD, and center
Application to the Clinical ECG

- 50 uV of ST depression = 1/2 mm (half a small box)
- The QTc is the longest interval from onset of QRS to the end of the T-wave
- PCA cannot be measured from ECG alone
Strong Heart Family Study

- Phenotypes include ECG/BP, ECHOs, lipid and thrombotic markers, and glucose tolerance
- Covariates include demographic factors, lifestyle, reproductive history, and medical history
Numbers of Examined Relative Pairs

Strong Heart Family Study

<table>
<thead>
<tr>
<th>Relationship</th>
<th>DA (N=326)</th>
<th>OK (N=310)</th>
<th>AZ (N=345)</th>
<th>SHFS (N = 981)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parent-offspring</td>
<td>336</td>
<td>245</td>
<td>319</td>
<td>900</td>
</tr>
<tr>
<td>Siblings</td>
<td>330</td>
<td>319</td>
<td>350</td>
<td>999</td>
</tr>
<tr>
<td>Half-siblings</td>
<td>86</td>
<td>77</td>
<td>44</td>
<td>207</td>
</tr>
<tr>
<td>Avuncular</td>
<td>954</td>
<td>802</td>
<td>721</td>
<td>2477</td>
</tr>
<tr>
<td>Grandparent-grandchild</td>
<td>129</td>
<td>35</td>
<td>12</td>
<td>291</td>
</tr>
<tr>
<td>Grand avuncular</td>
<td>395</td>
<td>139</td>
<td>261</td>
<td>795</td>
</tr>
<tr>
<td>First cousins</td>
<td>1142</td>
<td>843</td>
<td>866</td>
<td>2851</td>
</tr>
<tr>
<td>First cousins once removed</td>
<td>1091</td>
<td>409</td>
<td>909</td>
<td>2409</td>
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<tr>
<td>Second cousins</td>
<td>487</td>
<td>57</td>
<td>233</td>
<td>777</td>
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<tr>
<td>Other</td>
<td>621</td>
<td>322</td>
<td>145</td>
<td>1088</td>
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<tr>
<td>Total Relative Pairs</td>
<td>5571</td>
<td>3248</td>
<td>3975</td>
<td>12794</td>
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## Heritabilities for CVD Risk Factors

*Strong Heart Family Study*

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Proportion of Variance Due to Genes</th>
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<tbody>
<tr>
<td></td>
<td>%</td>
</tr>
<tr>
<td>BMI</td>
<td>44</td>
</tr>
<tr>
<td>WHR</td>
<td>54</td>
</tr>
<tr>
<td>LDL-C</td>
<td>39</td>
</tr>
<tr>
<td>HDL-C</td>
<td>50</td>
</tr>
<tr>
<td>InTG</td>
<td>40</td>
</tr>
<tr>
<td>SBP</td>
<td>23</td>
</tr>
<tr>
<td>F Glu</td>
<td>29</td>
</tr>
<tr>
<td>ln Ins</td>
<td>44</td>
</tr>
<tr>
<td>ln Fibrinogen</td>
<td>23</td>
</tr>
</tbody>
</table>
Heritability (h²) of LV Mass and Geometry in SHS-FS

* Heritability for QTL needed for power = 80% in SHS
## Genotype by Diabetes Status Interaction for CVD Risk Factors

<table>
<thead>
<tr>
<th>Trait</th>
<th>$\sigma_g$, D</th>
<th>$\sigma_g$, ND</th>
<th>$\rho_g$ (D, ND)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>5.23 (.81)</td>
<td>5.81 (.55)</td>
<td>.34 (p=.0002)</td>
</tr>
<tr>
<td>% Fat</td>
<td>5.59 (.69)</td>
<td>6.14 (.57)</td>
<td>.52 (p=.0005)</td>
</tr>
<tr>
<td>WHR</td>
<td>0.41 (.07)</td>
<td>0.58 (.06)</td>
<td>.69 (p=.0353)</td>
</tr>
<tr>
<td>HDL-C</td>
<td>8.97 (1.34)</td>
<td>9.24 (1.08)</td>
<td>.62 (p=.0307)</td>
</tr>
<tr>
<td>TG</td>
<td>3.73 (.94)</td>
<td>3.90 (.48)</td>
<td>.02 (p=.0096)</td>
</tr>
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# Genetic and Environmental Correlations Between Diabetes and CVD Risk Factors

<table>
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<tr>
<th>Risk Factor</th>
<th>Genetic</th>
<th>Environmental</th>
</tr>
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<tbody>
<tr>
<td>BMI</td>
<td>.55 (.14)***</td>
<td>.10 (.13)</td>
</tr>
<tr>
<td>% Fat</td>
<td>.38 (.14)*</td>
<td>.11 (14)</td>
</tr>
<tr>
<td>WHR</td>
<td>.58 (.15)***</td>
<td>.13 (14)</td>
</tr>
<tr>
<td>HDL-C</td>
<td>-.37 (.27)**</td>
<td>-.32 (.18)**</td>
</tr>
<tr>
<td>TG</td>
<td>.65 (.21)***</td>
<td>.33 (13)**</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>.40 (.17)**</td>
<td>.20 (.11)***</td>
</tr>
<tr>
<td>PAI – 1</td>
<td>.67 (.17)***</td>
<td>.18 (.10)</td>
</tr>
<tr>
<td>SBP</td>
<td>.57 (.21)**</td>
<td>.11 (.11)</td>
</tr>
</tbody>
</table>
SUMMARY

• There is a rising tide of CVD in diabetes
  – LDL and blood pressure are strong risk factors
  – Albuminuria, fibrinogen and ECHO/ECG abnormalities are also strong risk factors

• Current strategy for prevention
  – Aggressive control of LDL and BP
SUMMARY, cont

• Future directions for understanding the impact of diabetes on CVD
  – Understand mechanism of cardiac abnormalities
  – Explore further the inflammatory/thrombotic axis
  – Study adipocyte mediators

• Future direction - Genetics
  – Localize and identify genes contributing to CVD risk and measure their effects
  – Identify genes that modulate therapeutic responses
The Strong Heart Study
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The Strong Heart Study
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