Basic Public Health and Epidemiology

Objectives:

1. Learn basic concepts of public health / epidemiology
2. Begin to think quantitatively
3. Understand how causes of disease are identified
4. Learn examples of how prevention works
5. Consider the application of prevention science to Africa
Key Concepts in Public Health

1. Natural populations are the target.

2. Surveillance systems provide information.

3. Public health infrastructure links institutions that gather, disseminate and act on surveillance information.

4. Communities are mobilized.

5. Health and disease are the result of social processes.
Rudolf Virchow said . . . . (1845)

Mass disease means society is out of joint.
Ottawa Charter
WHO

The fundamental conditions and resources for health are:

Peace
Shelter
Education
Food
Income
A stable eco-system
Sustainable resources
Social justice
Equity
Public health is the science of protecting and improving the health of communities through education, promotion of healthy lifestyles, and research for disease and injury prevention.

Public health involves the application of many different disciplines including:

- biology, sociology, mathematics, anthropology, public policy, medicine, education, psychology, computer science, business, engineering, and much, much more . . . .

- Public health is concerned with protecting the health of entire populations.

- These populations can be as small as a local neighborhood, or as big as an entire country.

- Public health professionals try to prevent problems from happening or re-occurring through implementing educational programs, developing policies, administering services, and conducting research, in contrast to clinical professionals, such as doctors and nurses, who focus primarily on treating individuals after they become sick or injured.
What is Epidemiology?

Classic definition: Study of the distribution and determinants of disease in populations.

Also: Study of outcomes and effectiveness of medical interventions. In its technical application epidemiology relies heavily on statistics.

“Science and medicine have different ends. The intellectual foundation for medicine lies not in basic science but in epidemiology. The epidemiological setting provides the proper ground on which debates about the applicability of research evidence to practice should take place.”

Lancet, 1995
Key Concepts in Epidemiology

Epidemiology has Two Branches:

1. Observational Studies-
   Including - cross-sectional, case series, case-control, prospective (cohort) studies

2. Interventional Studies-
   Randomized controlled trials; individuals or communities

Some Basic Vocabulary:

Measures of occurrence: Prevalence Incidence

Measures of risk: Relative risk Odds Ratio Attributable Risk

Age adjustment
Learning to think quantitatively . . . . .

Mathematics is the language of science.
John Snow’s Investigation of Cholera Deaths, London, 1854

<table>
<thead>
<tr>
<th>Water Supply</th>
<th>Number of Houses</th>
<th>Deaths</th>
<th>Deaths per 10,000 Houses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Southwark &amp; Vauxhall Co.</td>
<td>40,046</td>
<td>1,263</td>
<td>315</td>
</tr>
<tr>
<td>Lambeth Co.</td>
<td>26,107</td>
<td>98</td>
<td>37</td>
</tr>
<tr>
<td>Rest of London</td>
<td>256,423</td>
<td>1,422</td>
<td>59</td>
</tr>
</tbody>
</table>
John Snow’s Cholera Map, London, 1854
## Vital Statistics Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Numerator (Events)</th>
<th>Denominator (Pop. at risk)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth rate</td>
<td>Live Births</td>
<td>Midyear pop.</td>
</tr>
<tr>
<td>Crude death rate</td>
<td>Deaths</td>
<td>Midyear pop.</td>
</tr>
<tr>
<td>Infant mortality rate</td>
<td>Deaths before age 1</td>
<td>Live Births in the year</td>
</tr>
<tr>
<td>Age-specific mortality</td>
<td>Deaths for a specific age group</td>
<td>Pop. in age group</td>
</tr>
<tr>
<td>Age-adjusted mortality</td>
<td></td>
<td>Crude rate adjusted to a standard pop.</td>
</tr>
</tbody>
</table>
Leading Causes of Death in US, 2000
Centers for Disease Control & Prevention

- Heart Disease
- Cancer
- Stroke
- Respiratory Disease
- Injury
- Diabetes
- Pneumonia/Flu
- Alzheimer's Disease
- Kidney Disease

Percentage (of all deaths)
Actual Causes of Death in the US, 1990 & 2000

www.cdc.gov, 2004
Age-Adjusted Prevalence of Overweight or Obesity in US Adults

from National Center for Health Statistics website
www.cdc.gov/nchs
Blood Lead Measurements 1975-1981

Trends in Serum Cholesterol, Men, Selected Countries
Serum Cholesterol and Risk of CHD, MRFIT

CHD and Saturated Fat Intake in 40 Countries

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**Serum Cholesterol and Risk of CHD, MRFIT**

- A scatter plot showing the relationship between serum total cholesterol (mg/dl) and age-adjusted CHD death rates per 10,000 men per 6 years.

**CHD and Saturated Fat Intake in 40 Countries**

- A scatter plot showing the relationship between cholesterol saturated fat index (per 1000 kcal/day) and death rate per 100,000 male population (age 55-64) due to coronary heart disease.

- The correlation coefficient $r = 0.78$.
Causal Process in Atherosclerosis

Diet High in Animal Fat → Elevated Cholesterol → Hypertension → Obesity / Diabetes → Atherosclerosis

- Smoking
- Physical Inactivity
The Development Process of CVD

Pathway

Social and Environmental Conditions \rightarrow Adverse Behavioral Patterns \rightarrow Major Risk Factors \rightarrow First Event/Sudden Death \rightarrow Disability/Risk of Recurrence \rightarrow Late Death

Target Population

Whole Population \rightarrow Whole Population \rightarrow Persons with Risk Factors \rightarrow Cases with First Fatal or Non-Fatal Events \rightarrow Survivors \rightarrow Late Deaths

Interventions

Policy and Environmental Change \rightarrow Behavior Change \rightarrow Risk Factor Detection and Control \rightarrow Emergency Care/Acute Case Management \rightarrow Rehabilitation/Long-Term Care \rightarrow End-Of-Life Care
Death Rates from CHD and Stroke, US, 1950-2002

Rate Per 100,000

CHD

Stroke


70%

70%
Figure 3 Trends in age standardised (world population) death certification rates from cerebrovascular diseases in men in all age groups from the European Union, eastern European countries (Bulgaria, Czech Republic, Hungary, Poland, Romania, and Slovakia), the USA, and Japan, 1965 to 1997.
Goal of adjustment

• Age Adjustment
  - Age influences rate of most disease
  - Comparison of rates must account for age differences in populations

• To reduce distortions and incomparability of rates when making comparison over time and among populations

• So we are not comparing apples to oranges
### Crude Mortality Rates in Florida & Alaska 1988

<table>
<thead>
<tr>
<th></th>
<th>Florida</th>
<th>Alaska</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Deaths</td>
<td>131,044</td>
<td>2,064</td>
</tr>
<tr>
<td>Total Pop.</td>
<td>12,335,000</td>
<td>524,000</td>
</tr>
<tr>
<td>Rate (/100,000)</td>
<td>1,062</td>
<td>394</td>
</tr>
</tbody>
</table>
## Age-Adjusted Mortality for Florida & Alaska, 1988

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Age - Specific Death Rate</th>
<th>U. S. Pop. Structure (%)</th>
<th>Standardized Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Florida</td>
<td>Alaska</td>
<td></td>
</tr>
<tr>
<td>&lt;5</td>
<td>284</td>
<td>274</td>
<td>7.4</td>
</tr>
<tr>
<td>5-19</td>
<td>57</td>
<td>65</td>
<td>21.5</td>
</tr>
<tr>
<td>20-44</td>
<td>198</td>
<td>188</td>
<td>39.9</td>
</tr>
<tr>
<td>45-64</td>
<td>815</td>
<td>629</td>
<td>18.7</td>
</tr>
<tr>
<td>&gt;65</td>
<td>4425</td>
<td>4350</td>
<td>12.5</td>
</tr>
</tbody>
</table>

100%

Age - adjusted rate = 817.8  808.5

[ Crude rate = 1,062  394]
Cross-Sectional Community Survey

- Population based
- Requires sampling frame to be representative
- Provides prevalence estimates
- Can be a baseline for a Prospective Study
Prevalence and Incidence

Prevalence = \frac{\text{Number of Cases in Population}}{\text{Population}}

Incidence = \frac{\text{Number of New Cases}}{\text{Total Number at Risk}}
Prospective or Cohort Study

Time →

Past  Present  Future
Determine Exposure  Determine Outcome
Cohort Study

- Exposed or subjects
- Unexposed or controls

Onset of study

Direction of inquiry

With Outcome
Without Outcome
With Outcome
Without Outcome
Prospective Study

Hepatitis B Antibody Status and Liver Cancer

<table>
<thead>
<tr>
<th>Hep B Ab Status</th>
<th>Number of People at Baseline, 1975</th>
<th>Number of Cases 1975 - 1986</th>
<th>Incidence Rate, per 100,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pos.</td>
<td>3,454</td>
<td>152</td>
<td>495</td>
</tr>
<tr>
<td>Neg.</td>
<td>19,253</td>
<td>9</td>
<td>5</td>
</tr>
</tbody>
</table>

Relative Risk = $\frac{495}{5} = 99$
## Prospective or Cohort Study

### Study Design

**Exposure:**

<table>
<thead>
<tr>
<th></th>
<th>Number with Disease</th>
<th></th>
<th>Number without Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>A</td>
<td></td>
<td>B</td>
</tr>
<tr>
<td>No</td>
<td>C</td>
<td></td>
<td>D</td>
</tr>
</tbody>
</table>

**Statistic of Interest:**  

Relative Risk = \[
\frac{A}{A+B} \cdot \frac{C}{C+D}
\]
## Prospective or Cohort Study

### Smoking and Mortality

Vital Status at 20 Years

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Dead</th>
<th>Alive</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smokers</td>
<td>333</td>
<td>1336</td>
<td>1498</td>
</tr>
<tr>
<td>Non-Smokers</td>
<td>132</td>
<td>486</td>
<td>819</td>
</tr>
</tbody>
</table>
Prospective or Cohort Study

Smoking and Mortality

Vital Status at 20 Years

Incidence in Exposed \[ = \frac{A}{A+B} = \frac{333}{1498} = 22.2 \text{ per 100} \]

Incidence in Unexposed \[ = \frac{C}{C+D} = \frac{132}{819} = 16.1 \text{ per 100} \]

Relative Risk \[ = \frac{22.2}{16.1} = 1.4 \]
Randomized Controlled Trial

Study Design
Demonstration by Pasteur of Anthrax Vaccination, 1881

- 60 Sheep
  - 10 Controls
  - 25 Vaccinated
    - May 5, 1st Vaccination
      - May 17, 2nd Vaccination
        - May 31, Injection of Anthrax Culture
          - 24 Survivors
            - 1 pregnant ewe dying
          - 10 Survivors
          - 23 Dead
            - 2 Moribund
  - 25 Unvaccinated
Randomized Controlled Trial

Experimental subjects

Subjects meeting entry criteria

Controls

Onset of study

Intervention

Time

With Outcome

Without Outcome

With Outcome

Without Outcome
### Randomized Controlled Trial

#### Study Design

<table>
<thead>
<tr>
<th>‘Exposure - ie, treatment-status’</th>
<th>Disease Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>A</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>No</td>
<td>C</td>
<td>C</td>
</tr>
</tbody>
</table>

Statistic of Interest: Relative Risk = \[
\frac{A}{A+B} \div \frac{C}{C+D}
\]
### Streptomycin in the Treatment of Tuberculosis

#### Appearance of chest x-ray at six months

<table>
<thead>
<tr>
<th>X-ray Appearance</th>
<th>Streptomycin</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Considerable Improvement</td>
<td>28</td>
<td>4</td>
</tr>
<tr>
<td>Moderate Improvement</td>
<td>10</td>
<td>13</td>
</tr>
<tr>
<td>No Change</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Moderate Deterioration</td>
<td>5</td>
<td>12</td>
</tr>
<tr>
<td>Considerable Deterioration</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Death</td>
<td>4</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td><strong>55</strong></td>
<td><strong>52</strong></td>
</tr>
</tbody>
</table>

Brit Med J, 1948
### Randomized Controlled Trial

Lipid Research Clinics CHD Primary Prevention Trial

#### Disease Outcome

<table>
<thead>
<tr>
<th>Treatment</th>
<th>CHD</th>
<th>Non-CHD</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>155</td>
<td>1751</td>
<td>1906</td>
</tr>
<tr>
<td>No</td>
<td>187</td>
<td>1713</td>
<td>1900</td>
</tr>
</tbody>
</table>
Randomized Controlled Trial
Lipid Research Clinics CHD Primary Prevention Trial

Disease Outcome

Incidence in ‘exposed’ - ie, treated = \( \frac{155}{1906} \) = 8.1 per 100

Incidence in ‘unexposed’ - ie, untreated = \( \frac{187}{1900} \) = 9.8 per 100

Relative Risk = \( \frac{8.1}{9.8} \) = 0.83
### Table 1

**Descriptive summary of sodium-reduction trials in hypertensive subjects**

<table>
<thead>
<tr>
<th>Author and year</th>
<th>Duration</th>
<th>Blinding</th>
<th>Urinary Na change</th>
<th>(No) Changes in confounders</th>
<th>Blood pressure change</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Crossover trials</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parkey et al 1973 (8), (n = 15)</td>
<td>1</td>
<td>NR</td>
<td>-08</td>
<td>(Wt)</td>
<td>-6.7</td>
</tr>
<tr>
<td>MacGregor et al 1982 (9), (n = 19)</td>
<td>1</td>
<td>DB</td>
<td>-76</td>
<td>(Wt), (K)</td>
<td>-10.0^2</td>
</tr>
<tr>
<td>Watt et al 1983 (10), (n = 18)</td>
<td>1</td>
<td>DB</td>
<td>-50</td>
<td>(Wt), (K)</td>
<td>-0.5</td>
</tr>
<tr>
<td>Richards et al 1984 (11), (n = 12)</td>
<td>1-1.5</td>
<td>NR</td>
<td>-105</td>
<td>(Wt), K</td>
<td>-5.2</td>
</tr>
<tr>
<td>Grobbel et al 1987 (12), (n = 40)</td>
<td>1.5</td>
<td>DB</td>
<td>-72</td>
<td>(Wt), (K)</td>
<td>-0.8</td>
</tr>
<tr>
<td>MacGregor et al 1988 (13), (n = 20)</td>
<td>1</td>
<td>DB</td>
<td>-92</td>
<td>(Wt), (K)</td>
<td>-8.0^2</td>
</tr>
<tr>
<td>Dodson et al 1989 (14), (n = 9)</td>
<td>1</td>
<td>DB</td>
<td>-76</td>
<td>(Wt), (K)</td>
<td>-9.7</td>
</tr>
<tr>
<td>ANHMR C 1989 (15), (n = 88)</td>
<td>2</td>
<td>DB</td>
<td>-67</td>
<td>(K)</td>
<td>-2.6</td>
</tr>
<tr>
<td>Benetos et al 1992 (16), (n = 20)</td>
<td>1</td>
<td>DB</td>
<td>-78</td>
<td>(Wt), (K), (Ca)</td>
<td>-6.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Parallel trials</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Morgan et al 1978 (17), (n = 31, 31)</td>
<td>24</td>
<td>BP obs</td>
<td>-27</td>
<td>NR</td>
<td>-1.3</td>
</tr>
<tr>
<td>Morgan and Myers 1981 (18), (n = 6, 6)</td>
<td>2</td>
<td>BP obs</td>
<td>-98</td>
<td>K</td>
<td>-6.0</td>
</tr>
<tr>
<td>Morgan and Myers 1981 (18), (n = 6, 6)</td>
<td>2</td>
<td>BP obs</td>
<td>-78</td>
<td>K</td>
<td>-4.0</td>
</tr>
<tr>
<td>Costa et al 1981 (19), (n = 20, 21)</td>
<td>12</td>
<td>NR</td>
<td>-53</td>
<td>(Wt), (K)</td>
<td>-18.3</td>
</tr>
<tr>
<td>Silman et al 1983 (20), (n = 10, 15)</td>
<td>12</td>
<td>BP obs (RZ)</td>
<td>-117</td>
<td>(Wt), (K), (P:S)</td>
<td>-8.7</td>
</tr>
<tr>
<td>Puska et al 1983 (21), (n = 15, 19)</td>
<td>1.5</td>
<td>BP obs</td>
<td>-89</td>
<td>(Wt), (K), (A)</td>
<td>-13.3</td>
</tr>
<tr>
<td>Fagerberg et al 1984 (22), (n = 15, 15)</td>
<td>2.3</td>
<td>NR</td>
<td>-89</td>
<td>(Wt), (K), (A)</td>
<td>-13.3</td>
</tr>
<tr>
<td>Maxwell et al 1984 (73), (n = 18, 17)</td>
<td>3</td>
<td>NR</td>
<td>-171</td>
<td>Wt</td>
<td>-2.0</td>
</tr>
<tr>
<td>Erweimann et al 1984 (24), (n = 44, 50)</td>
<td>6</td>
<td>BP obs (RZ)</td>
<td>-58</td>
<td>NR</td>
<td>2.7</td>
</tr>
<tr>
<td>Chunners et al 1980 (25), (n = 48, 52)</td>
<td>3</td>
<td>NK</td>
<td>-34</td>
<td>(K)</td>
<td>-3.1</td>
</tr>
<tr>
<td>Logan 1986 (26), (n = 37, 38)</td>
<td>6</td>
<td>BP obs</td>
<td>-32</td>
<td>(Wt), (K)</td>
<td>-1.1</td>
</tr>
<tr>
<td>Dodson et al 1989 (14), (n = 17, 17)</td>
<td>3</td>
<td>BP obs</td>
<td>-50</td>
<td>(Wt), (K)</td>
<td>-13.0</td>
</tr>
<tr>
<td>ANHMR C 1989 (27), (n = 50, 53)</td>
<td>2</td>
<td>DB</td>
<td>-71</td>
<td>(A)</td>
<td>-5.5</td>
</tr>
<tr>
<td>Scharman et al 1992 (28), (n = 46, 45)</td>
<td>2</td>
<td>DB</td>
<td>-84</td>
<td>(Wt), (K)</td>
<td>-8.0</td>
</tr>
<tr>
<td>Parker et al 1992 (29), (n = 16, 15)</td>
<td>1</td>
<td>DB</td>
<td>-80</td>
<td>(Wt), (A), (K), (Ca), (Mg)</td>
<td>2.2</td>
</tr>
<tr>
<td>Parker et al 1992 (30), (norm A) (n = 15, 15)</td>
<td>1</td>
<td>DB</td>
<td>-57</td>
<td>(Wt), (A), (K), (Ca), (Mg)</td>
<td>-0.1</td>
</tr>
</tbody>
</table>

^1 NR, not reported; Wt, body weight; DB, double blind; K, potassium intake/excretion; ANHMR C, Australian National Health and Medical Research Council; Ca, calcium intake/excretion; BP obs, observers blinded; RZ, random zero manometer; A, alcohol intake; P:S, ratio of polyunsaturated to saturated fatty acid; Mg, magnesium excretion.

^2 P < 0.05.

^3 n values given for each study are not the number of subjects in the sodium-reduction treatment and control groups, respectively.

^4 -23% intracellular Na.
Systolic BP Change in the DASH Trial

Control diet  
-5.9  
(-8.0 to -3.7)‡

DASH diet  
-2.1  
(-3.4 to -0.8)‡

-5.0  
(-7.6 to -2.5)‡

-1.3  
(-2.6 to 0.0)*

-2.2  
(-4.4 to -0.1)*

-1.7  
(-3.0 to -0.4)†

Sodium Level

Systolic Blood Pressure (mm Hg)
Prevalence of Obesity in Populations of African Origin

Cappuccio et al, in press
Prevention of Type 2 Diabetes, Finnish Lifestyle Prevention Study

Per capita Daily Energy Intake in Cuba, 1980-2005

Franco, Cooper et al  Am J Epidemiology, 2007
Prevalence of Obesity in Cienfuegos and Havana, Cuba, 1990-2000
Trends in Mortality from Diabetes, CHD, Cancer and All-Causes, Cuba, 1980 - 2005

Diabetes

CHD

Cancer

All-Causes

Mortality Rate per 100,000

Year
Measures to improve public health, relating as they do to such obvious and mundane matters as housing, smoking, and food, may lack the glamour of high-technology medicine, but what they lack in excitement they gain in their potential impact on health, precisely because they deal with the major causes of common disease and disabilities.