Cornoni

Department of Epidemiology
School of Public Health
University of North Carolina

# EPID 160, PRINCIPLES OF EPIDEMIOLOGY

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Fall Semester 1970

This manual contains the supportive material for the lecture and laboratory meetings for the first eight weeks of the course.

Two hours credit:

One-hour lecture per week
Two-hour laboratory per week

# TABLES OF CONTENTS

# READING LIST

GENERAL: MacMahon, Brian; Pugh, Thomas; and Ipsen, Johannes. Epidemiologic Methods. Boston: Little Brown & Co., 1960.

Morris, J. N. Uses of Epidemiology. 2nd ed. Baltimore, Maryland: The Williams and Wilkins Company, 1964.

This is a very useful reference book providing a wide series of provocative illustrations of the uses to which epidemiological principles and methods can be put. The book is not a "text" in itself but provides insights into a number of problems and a very full set of references as a guide to further reading. It does not deal extensively with epidemiological method and will thus need to be supplemented by selected readings in this area.

MacMahon, Brian and Clark, Duncan. Preventive Medicine. Boston: Little Brown & Co., 1967.

Lerner, Monroe and Anderson, Odin. Health Progress in the United States, 1900-1960. Chicago: The University of Chicago Press, 1965.

Dubos, Rene. "Man Meets His Environment." Health and Nutrition, VI, 1-11.

EPIDEMIOLOGY AS THE BASIS FOR SCIENTIFIC PUBLIC HEALTH PRACTICE:

Terris, Milton. "The Scope and Methods of Epidemiology." American Journal of Public Health, 52(September 1962), 1371-1376.

Editorial. "The Scope and Methods of Epidemiology." American Journal of Public Health, 52(September 1962), 1502-1504.

Saiger, Geo. L. "Ten Uses of Epidemiology." Canadian Medical Association Journal, 85(October 1961), 992-995.

Mattison, Berwyn F. "Epidemiological Techniques and Data in Planning Public Health Programs." Public Health Reports, 70(1955), 625-632.

THE EPIDEMIOLOGICAL METHOD AND DATA REQUIRED IN EPIDEMIOLOGICAL STUDY:

Recent Studies in Epidemiology. Edited by Pemberton and Willard. Oxford: Blackwell Scientific Publications, 1958.

Comparability in International Epidemiology. Edited by Roy M. Acheson. Milbank Memorial Fund, 1965.

Epidemiological Approaches to the Study of Cancer and Other Chronic Diseases. Edited by William Haenzel. National Cancer Institute Monograph 19, January 1966.

Lilienfeld, Abraham; Pedersen, Elinar; and Dowd, J. E. Cancer Epidemiology: Methods of Study. Baltimore: The Johns Hopkins Press, 1967.

Paul, J. R. Clinical Epidemiology. Chicago: University of Chicago Press, 1958.

Taylor, Ian and Knowelden, John. Principles of Epidemiology. Boston: Little, Brown & Co., 1957.

Papers of Wade Hampton Frost. Edited by K. F. Maxey. New York: The Commonwealth Fund, 1941.

Witt, L. J. Medical Surveys and Clinical Trials. Oxford University Press, 1959.

Gordon, John. "Ecological Investigation of Disease." Research in Public Health, Milbank Memorial Fund, 1952, 49-76.

Gordon, John. "Epidemiology in Modern Perspective." Proceedings of the Royal Society of Medicine, 47(July 1954), 564-570.

Gordon, John. "Epidemiology: The Diagnostic Discipline of Public Health." Royal Sanitary Institute Journal, 74(July 1954), 445-454.

Gordon, John. "Medical Ecology and the Public Health." American Journal of Medical Science, 235(March 1958), 337-358.

Gilliam, Alexander G. "Epidemiology in Non-Communicable Disease." Public Health Reports, 69(October 1954), 907-913.

Clark, Virginia A. and Hopkins, Carl E. "Time is of the Essence." Editorial in the Journal of Chronic Diseases, 20(1967), 565-569.

Editorial. "Modern Concepts of Epidemiology." Journal of Chronic Disease, 2(November 1955), 593-596.

Mainland, Donald. "Notes on the Planning and Evaluation of Research with Examples from Cardiovascular Research." American Heart Journal, 55(1958), 644-655, 824-837, 838-850.

Mainland, Donald. "The Use and Misuse of Statistics in Medical Publications." Clinical Pharmacology and Therapeutics, 4(1960), 411-422.

Mainland, Donald. "The Significance of Nonsignificance." Clinical Pharmacology and Therapeutics, 4(1963), 580-586.

Cassel, John; Patrick, Ralph; and Jenkins, David. "Epidemiological Analysis of the Health Implications of Culture Change: A Conceptual Model." Annals of the New York Academy of Science, 84(December 1960), 938-949.

Cassel, John. "Social Science Theory as a Source of Hypotheses in Epidemiological Research." American Journal of Public Health, 54(September 1964), 1482-1488.

EPIDEMIOLOGICAL STRATEGY AND METHOD
AREAS TO BE COVERED

1. Differences between observational and experimental sciences.

2. Methods of study.

   Case history (retrospective)
   Cohort (prospective, incidence)
   Cross-sectional (prevalence)

   Ref: Epidemiologic Methods, MacMahon, Brian; Thomas Pugh, and Johannes Ipsen, Little Brown & Co., 1960, Chapters 2, 13, 14.
   Preventive Medicine, MacMahon, Brian; Duncan Clark, Little Brown & Co., 1967, Chapter 7.
   Medical Surveys and Clinical Trials, Witt, L. J., Oxford University Press, 1959, Chapter 4.

3. Attributable and relative risk.

   Ref: Epidemiologic Methods, MacMahon, Brian; Thomas Pugh, and Johannes Ipsen, Little Brown & Co., 1960, p. 229 and 260.

4. Interpretations from prevalence (point and period) incidence, mortality, case fatality.

   Ref: Medical Surveys and Clinical Trials, Witt, L. J., Oxford University Press, 1959, Chapter 3.
   Epidemiologic Methods, MacMahon, Brian; Thomas Pugh, and Johannes Ipsen, Little Brown & Co., 1960, Chapter 5.

5. Reliability and validity.

   Ref: Medical Surveys and Clinical Trials, Witt, L. J., Oxford University Press, 1959, pp. 30-40.

6. Association versus cause.

   6.1 Non-causal associations
      6.1.1 chance
      6.1.2 artifact
      6.1.3 secondary

   6.2 Causal associations
      6.2.1 indirect
      6.2.2 direct
      6.2.3 configurational

   Ref: Epidemiologic Methods, MacMahon, Brian; Thomas Pugh, and Johannes Ipsen, Little Brown & Co., 1960, Chapter 2.

7. Bias and selection.

8. Control tables.

9. Calculation of "expected" values.

10. Ecological fallacy.

11. Analytical approaches to continuous and discrete data.

    11.1 Limitation of mean
    11.2 Bimodality
    11.3 Cohort effect

    Ref: Epidemiologic Methods, MacMahon, Brian; Thomas Pugh, and Johannes Ipsen, Little Brown & Co., 1960, Chapter 7.

LECTURE SCHEDULE

Wednesday 12-1, School of Public Health Auditorium

I. Epidemiology as a Foundation Science for Public Health Practice

Lecture # 1, September 23, Current Status of Public Health Practice and Health Care

Lecture # 2, September 30, Role of Epidemiology in Scientifically Based Practice: Epidemiologic Surveillance and Community Diagnosis

Lecture # 3, October 2*, Role of Epidemiology in Scientifically Based Practice: Program Planning and Evaluation

Lecture # 3, October 6*, Role of Epidemiology in Scientifically Based Practice: Program Planning and Evaluation

II. Strategy of Epidemiology

Lecture # 4, October 7, Association vs. Cause in Observational Science, Case History, Cohort and Cross Sectional Approaches

Lecture # 5, October 14, Gathering and Recording Data: Reliability and Validity

Lecture # 6, October 21, Data Processing and Reduction

Lecture # 7, October 28, Analysis and Interpretation

III. Lecture # 8, November 4, Analysis and Interpretation (continued)

Lecture # 9, November 11, Biological Characteristics

November 18, MID-TERM EXAMINATION

Lecture #10, November 25, Social Characteristics

Lecture #11, December 2, Personality Characteristics

Lecture #12, December 9, Behavioral Characteristics

Lecture #13, December 16, The Physical Environment

Lecture #14, January 6, History of Epidemiology and the Development of New Conceptual Models

Lecture #15, January 13, History of Epidemiology and the Development of New Conceptual Models

*Lecture replaces track laboratory.

## OBJECTIVES AND GENERAL COURSE OUTLINE

Epidemiology may be viewed both as a specific body of knowledge concerning various states of health and as a method of study. Thus it is appropriate to talk of "the epidemiology of" typhoid fever or lung cancer, for example (i.e., the specific body of epidemiological knowledge concerning those two diseases) and also to talk of "epidemiological investigation" to determine the factors responsible for any disease or disorder. This course is concerned mainly with the principles underlying epidemiology as a method of study and the scope, potentialities and limitations of this approach.

In the minds of many, the objectives of epidemiological investigation are restricted to discovering the factors responsible for an outbreak or epidemic of some infectious disease. Modern epidemiologists regard this as only one contribution of epidemiology. The scope and uses of epidemiological study have been considerably broadened. This point will be amply documented in this course.

Stated formally the objectives of this course are:

1. To develop a conceptual model of epidemiological enquiry as the basis for scientific public health practice.
2. To illustrate the scope and uses of epidemiological enquiry.
3. To familiarize students with the basic principles of the observational sciences (of which epidemiological enquiry is one).
4. To teach a number of the more important aspects of epidemiological method.

To accomplish these objectives the course will be divided into a lecture and a laboratory/seminar series. The lecture series will be con-

cerned with the philosophy, principles and methods of epidemiology. The laboratory/seminar series will review and illustrate these principles using various areas of application.

# THE NEED FOR SCIENTIFIC PUBLIC HEALTH PRACTICE

1. Failure of many modern programs to follow some of the well defined scientific principles of pioneer programs - These original programs often included:

   a. Clearly defined objectives as to the state of health to be improved.
   b. Objectives stated in operational terms with acceptable indices utilized.
   c. A knowledge (or estimate) of the extent of the existing problem.
   d. Information as to the characteristics of the population at highest risk.
   e. Some clear hypotheses about the circumstances needing to be changed to accomplish the objective.
   f. Some estimates, after a period of time as to whether these circumstances had been changed.
   g. Some estimates as to whether these changes had been accompanied by an improvement in the health problem.

2. The need for a scientific approach to public health practice has been accentuated by

   a. The changing nature of health problems.
   b. The changes in the nature of the groups at most risk in our population.
   c. The changes in the goals of public health programs.
   d. The changes in some of the modes of living in our society.

Suggested reading:

J. N. Morris Uses of Epidemiology. The Williams and Wilkins Co., 1964, pp. 1-33.

# EPIDEMIOLOGIC SURVEILLANCE AND COMMUNITY DIAGNOSIS

A. Categories of Data to be obtained

1. Patterns of Utilization of Existing Health Services
   a. Extent of current utilization
   b. Determinants of utilization and non-utilization
   c. Degree of coordination of existing services
   d. Existing methods of financing services

2. Need for Additional Services and Facilities
   a. As seen by practitioners
   b. As seen by potential utilizers
   c. As seen by professional consultants

3. Impact of Ill Health on the Community
   a. Degree of disability
   b. Cost of services and economic loss due to illness
   c. Social and personal problems created by or associated with ill health

4. Content and Quality of existing practices
   a. Degree and adequacy of communication between professionals and patients
   b. Degree of continuity of care for the individual
   c. Degree of continuity of care for the family
   d. Degree to which preventive, curative and rehabilitative services are integrated
   e. Existing role performances and degree of satisfaction with these. Possibilities for expanding or altering various existing professional roles.
   f. Service loads - numbers of patients and visits, types of conditions, types of actions taken

B. Methods of Data Gathering

1. Community Surveys - using random or stratified random samples of the population

2. Surveys of existing practices and practitioners

3. Combined practice - community surveys (using the practice to develop a reporting system with follow through of those reported by home interview) - to obtain professional and patient opinion over unmet needs, economic and emotional costs of illness, etc.

C. Instruments, Techniques, etc., to be Developed

1. Sampling frames and sampling units

2. Development of questionnaires and survey instruments

3. Development of record forms, record linkage, storage and retrieval systems

# EPIDEMIOLOGIC APPROACH TO SCIENTIFIC PUBLIC HEALTH PRACTICE

| Steps in General Epidemiologic Enquiries | Applied to Scientific Public Health Practice |
|---|---|
| 1. The perception of a meaningful problem and its statement in precise operational terms | Statement of the objective of the program in operational terms. (Differentiate statement of objective from statement of procedures). Requires answers to question:<br><br>What do we wish to accomplish and how will we know whether we have attained this goal?<br><br>Necessitates knowledge of:<br><br>a. Extent of problem and indices to measure this by<br>b. Sorts of people at highest risk |
| 2. Nature of the Conceptual Frame (Underlying Hypotheses) | a. What do we believe has to be changed in order to accomplish our objective?<br>b. What procedures or techniques can we employ to accomplish these changes? |
| 3. Determination of variables to be studied | Leads provided by answers to above questions |
| 4. Collection tabulation and analysis of data | Initiation and development of the program. Methodical recording of data in a standardized fashion. |
| 5. Interpretation of results | a. Have the changes postulated by the conceptual scheme as being necessary for our objective been brought about? (An indicator of the efficiency of our techniques)<br>b. If yes, has our original objective been attained? (An indicator of the validity of the conceptual scheme.)<br>c. Are any of the changes that have occurred a result of our efforts? (Need for control areas of "natural experiments"). |

# STRATEGY OF EPIDEMIOLOGY

Basic strategy: The comparison of two or more groups.

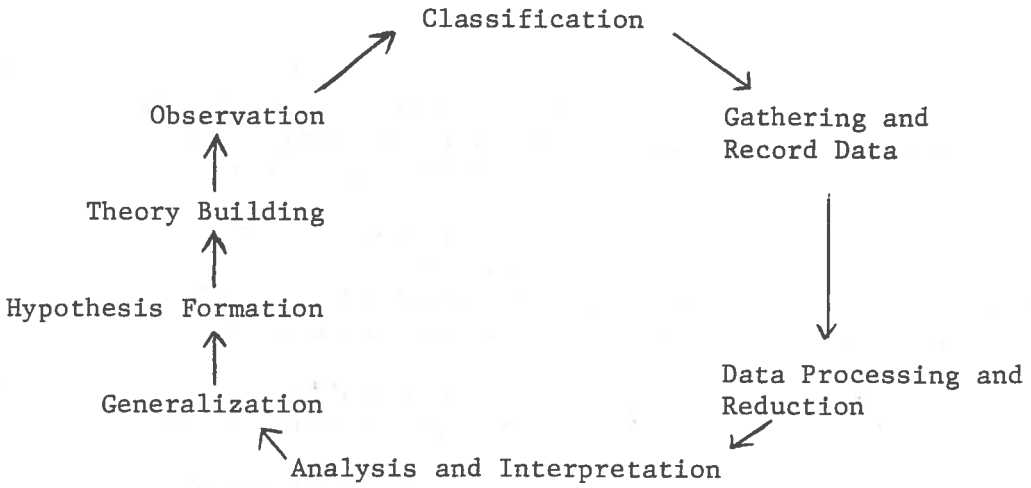
Unit of study: A group or aggregate.

Techniques: Usually those of an observational rather than an experimental science.

Essential differences between observational and experimental approaches:

| | Experimental | Observational |
|---|---|---|
| 1. | Factor(s) of interest to investigator under control of investigator and can be manipulated by him. | Factor(s) of interest to investigator not under his control and cannot be manipulated. |
| 2. | All other factors can be held constant or assigned at random to experimental and control group. | All other factors cannot be held constant. Randomization possible only to a limited extent. |
| 3. | Antecedent - consequent relationships obvious. | Antecedent - consequent relationships may not be obvious. |
| 4. | Evidence for cause clear under conditions in which all other factors are constant | Evidence for cause less clear (based on association), but applicable to "real life" where all other factors not constant. Association may be:<br>a. Non-causal<br>b. Causal |

STEPS IN OBSERVATIONAL SCIENCE




Observation and Classification

Classification on basis of health status (case history approach).

Classification on basis of attributes (cohort approach).

Advantages and disadvantages of each approach.

Relative and attributable risk.

# GATHERING AND RECORDING DATA

Two important concepts:
Reliability of data (reproducibility)
Validity: The degree to which a particular test or index measures what it purports to measure

Reliability: Between observers
Within observer over time
Measured by degree of concordance (or agreement; best assessed case by case rather than by group means)

Validity: Requires an external validating criterion
Two measures of validity

Sensitivity = $\frac{T.P.}{T.P.+F.N.}$ The ability of the test to correctly detect true cases.

Specificity = $\frac{T.N.}{T.N.+F.P.}$ The ability of the test to identify non cases (i.e. not to label them as cases)

Validating Criteria

| | | Diabetes | Not Diabetes | Total |
|---|---|---|---|---|
| Test to be Validated | 120 and over | T.P. | F.P. | |
| Level of Blood Sugar | 119 and less | F.N. | T.N. | |
| (mgm %) | Total | TP+FN | TN+FP | |

## Reliability (Example)

| Subject | Height in Inches | |
|---|---|---|
| | Observer 1 | Observer 2 |
| A | 62 | 62 |
| B | 63 | 65 |
| C | 60 | 61 |
| D | 66 | 66 |
| E | 68 | 63 |
| F | 70 | 70 |
| G | 60 | 60 |
| H | 62 | 62 |
| I | 65 | 68 |
| J | 70 | 70 |
| K | 65 | 65 |
| L | 66 | 66 |
| M | 69 | 67 |
| N | 66 | 66 |
| O | 61 | 60 |
| P | 64 | 64 |
| Q | 67 | 70 |
| R | 70 | 70 |
| S | 69 | 69 |
| T | 70 | 69 |

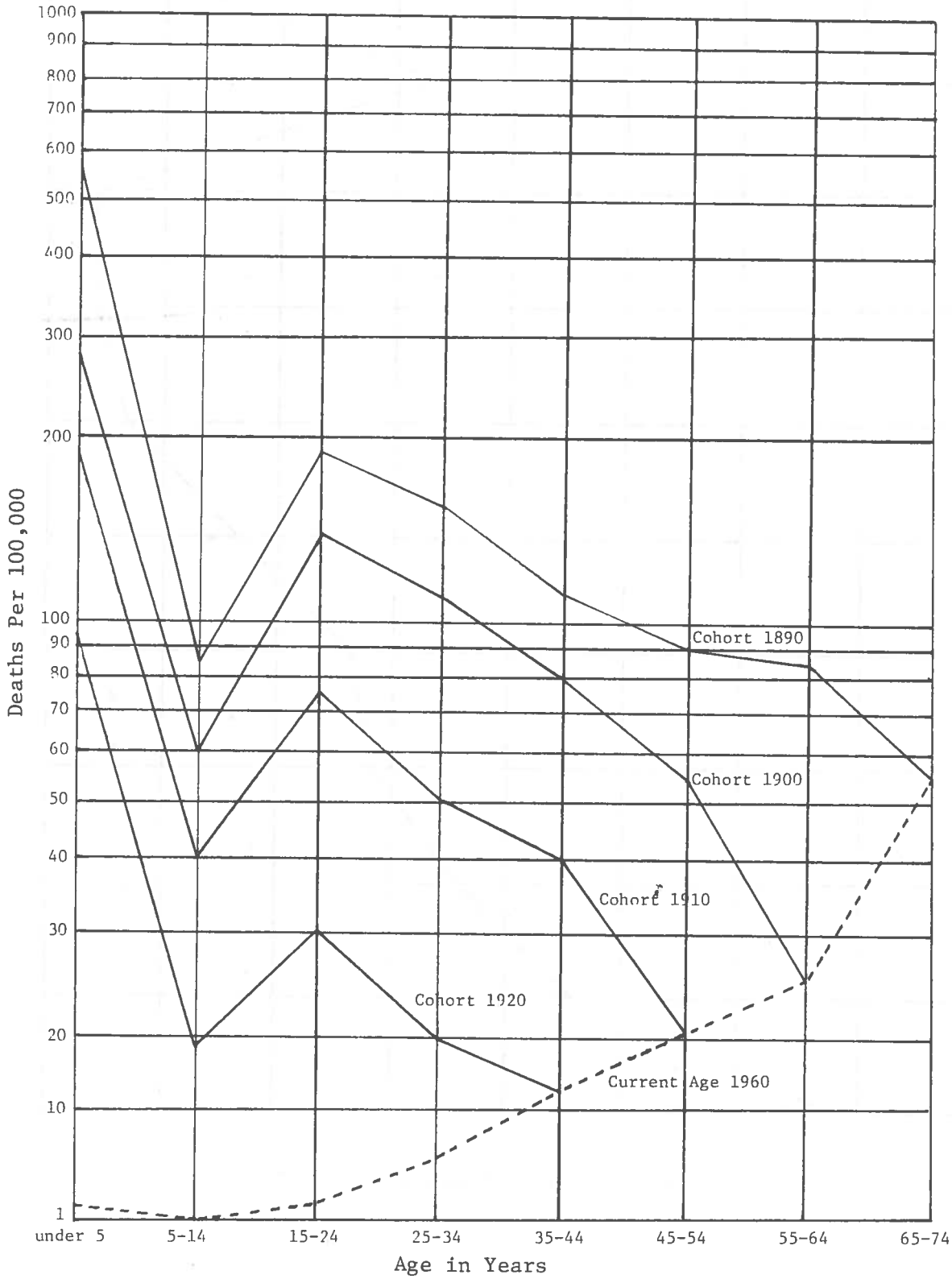
Observer 1

| Observer 2 | 60 | 61 | 62 | 63 | 64 | 65 | 66 | 67 | 68 | 69 | 70 | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 60 | 1 | 1 | | | | | | | | | | 2 |
| 61 | 1 | 0 | | | | | | | | | | 1 |
| 62 | | | 11 | | | | | | | | | 2 |
| 63 | | | | 0 | | | | | 1 | | | 1 |
| 64 | | | | | 1 | | | | | | | 1 |
| 65 | | | | 1 | | 1 | | | | | | 2 |
| 66 | | | | | | | 111 | | | | | 3 |
| 67 | | | | | | | | 0 | | 1 | | 1 |
| 68 | | | | | | 1 | | | 0 | | | 1 |
| 69 | | | | | | | | | | 1 | 1 | 2 |
| 70 | | | | | | | | | | | 111 | 4 |
| Total | 2 | 1 | 2 | 1 | 1 | 2 | 3 | 1 | 1 | 2 | 4 | 20 |

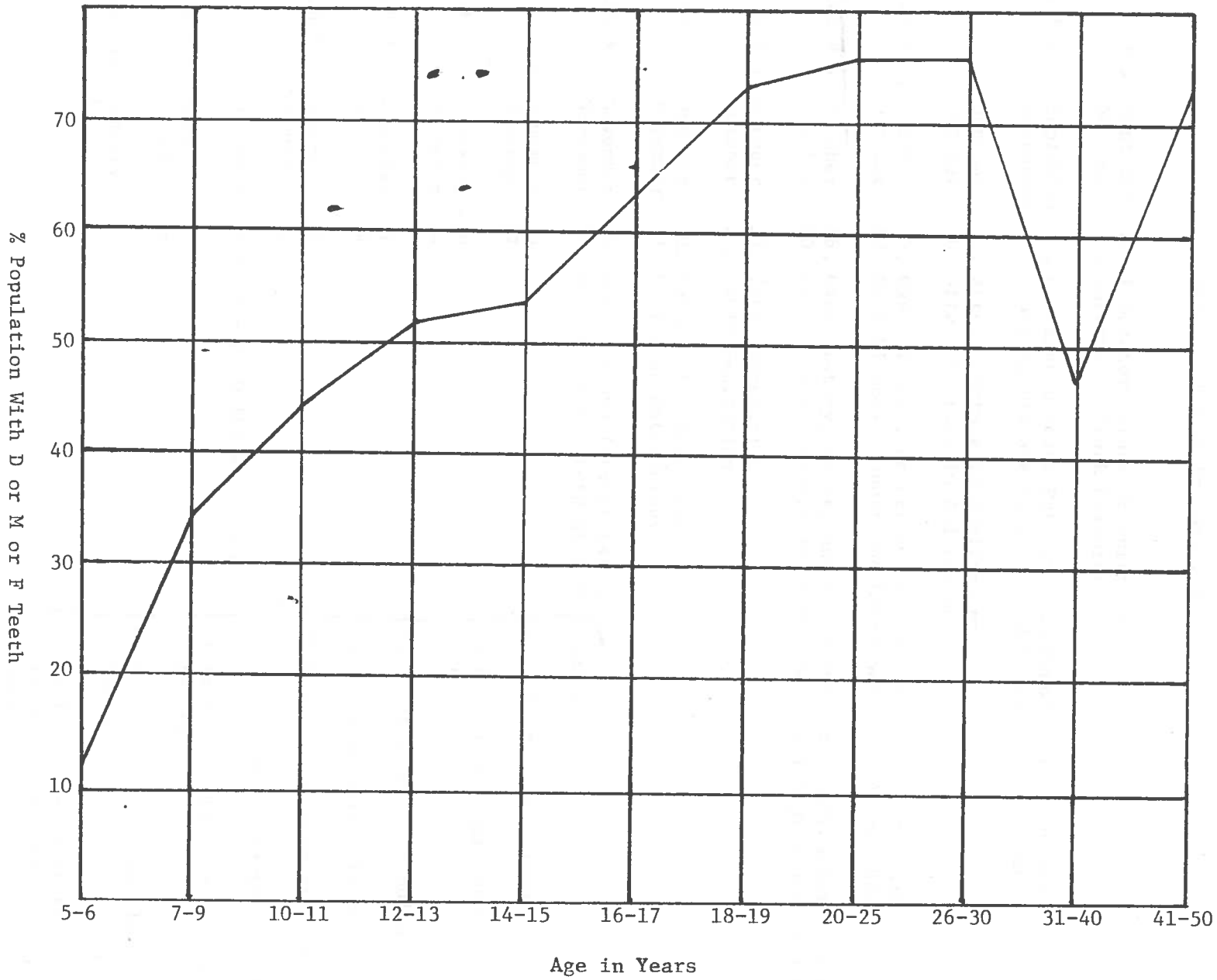
Death Rates from Tuberculosis
1880-1960: Illustrating Cohort Effect:

| AGE | 1880 | 1890 | 1900 | 1910 | 1920 | 1930 | 1940 | 1950 | 1960 |
|---|---|---|---|---|---|---|---|---|---|
| Under 5 | 760 | 570 | 280 | 190 | 95 | 35 | 8 | 3 | 1.7 |
| 5-14 (10) | 120 | 110 | 85 | 60 | 40 | 18 | 5 | 1 | 0.2 |
| 15-24 (20) | 430 | 350 | 270 | 192 | 140 | 75 | 30 | 6 | 0.8 |
| 25-34 (30) | 370 | 360 | 290 | 245 | 150 | 110 | 50 | 20 | 5.5 |
| 35-44 (40) | 360 | 340 | 250 | 245 | 160 | 113 | 80 | 40 | 16.6 |
| 45-54 (50) | 380 | 320 | 260 | 240 | 155 | 120 | 90 | 65 | 20.5 |
| 55-64 (60) | 470 | 380 | 300 | 242 | 160 | 125 | 100 | 85 | 25.7 |
| 65-74 (70) | 510 | 420 | 380 | 270 | 190 | 145 | 120 | 95 | 58.3 |

Tuberculosis Mortality Data
Illustrating Cohort Effect



Per Cent Population With Decayed or Missing or Filled Teeth by Age, 1961:
Illustration of Cohort Effect



LABORATORY SCHEDULE

Tuesdays 3-5 or Fridays 2-4

Lab # 1, September 18, Laboratory Track Orientation
September 22, Laboratory Track Orientation

Lab # 2, September 25, Changing Disease Patterns and Population Structure
September 29, Changing Disease Patterns and Population Structure

October 2, Attend Lecture in Auditorium
October 6, Attend Lecture in Auditorium

Lab # 3, October 9, Case History, Cohort and Cross Sectional Approaches
October 13, Case History, Cohort and Cross Sectional Approaches

Lab # 4, October 16, Case History, Cohort and Cross Sectional Approaches (contd.)
October 20, Case History, Cohort and Cross Sectional Approaches (contd.)

Lab # 5, October 23, Table Generation
October 27, Table Generation

Lab # 6, October 30, Review of Methodology
November 3, Review of Methodology

Lab # 7, November 6, Analysis and Interpretation
November 10, Analysis and Interpretation

Lab # 8, November 13
November 17

Lab # 9, November 20
November 24

Lab #10, December 1
December 4

Lab #11, December 8
December 11

December 15 EXTRA--EPID 161 will meet

Lab #12, January 5
January 8

Lab #13, January 12
January 15

Tracks:

#1 Information Systems - Cordle

#2 Health Relevant Behavior - Patrick

#3 Major Diseases - Hulka

#4 Epidemiologic Methods - Slome

#5 Population Dynamics - Omran

#6 Psycho-social Factors - Jenkins

#7 Infectious Disease - Drake, Becker

#8 Psychiatric Epidemiology - Kaplan

#9 Applications of Statistical Techniques to Epidemiology - Cornoni

#10 Etiology of Disease - Spiers

## CHANGES IN POPULATION AND HEALTH PROBLEMS

"It is to the current census and to the local health department and its division of vital statistics that most physicians turn for their information about populations, health conditions, and trends of disease within their local communities and cities. For instance, one can usually determine, in the United States at least, the total number of people living within a given area according to the last census, their ages, sexes and the relative racial percentages. Added to this it is obviously desirable to know the local birth and death rates, and particularly the current and past frequency with which reportable and even some nonreportable diseases, illnesses, or accidents have occurred. Other features desirable to know are: the local seasonal effect upon the rates for diseases and injury; what areas in the community are prone to high rates of this or that illness; and what the impact of local industrial practices or living conditions is upon this picture. It is obvious that, if one is to understand or interpret these data, one must know the people from whom they come, for diseases shift constantly, with growth or decline of populations, changing customs, new fashions, and new ways of living."[1]

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1. Paul, John R., Clinical Epidemiology, The University of Chicago Press, Chicago, 1966, pp. 98-99.

PART I Changes in Population Composition

U. S., 1900-1968

(a) Increase in size of population

Estimated Total Population for the United States
June 1, 1850 to July 1, 1968[2]

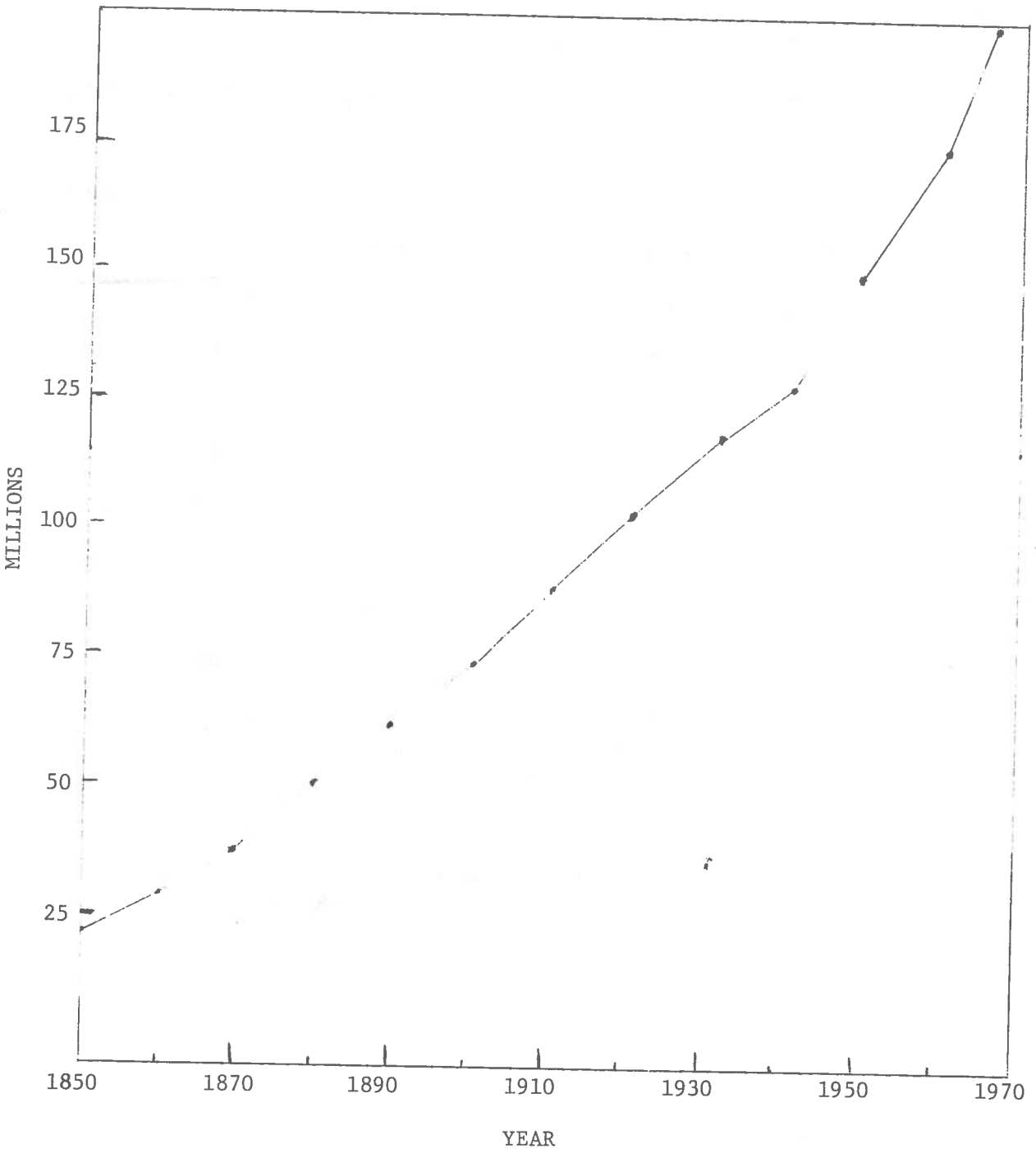
| Census Date | Number of Persons | Increase over preceding census | |
|---|---|---|---|
| | | Number | Percent |
| 1850 | 23,191,876 | 6,122,423 | 35.9 |
| 1860 | 31,443,321 | 8,251,445 | 35.6 |
| 1870 | 39,818,449 | 8,375,128 | 26.6 |
| 1880 | 50,155,783 | 10,337,334 | 26.0 |
| 1890 | 62,947,714 | 12,791,931 | 25.5 |
| 1900 | 75,994,575 | 13,046,861 | 20.7 |
| 1910 | 91,972,266 | 15,977,691 | 21.0 |
| 1920 | 105,710,620 | 13,738,354 | 14.9 |
| 1930 | 122,775,046 | 17,064,426 | 16.1 |
| 1940 | 131,669,275 | 8,894,229 | 7.2 |
| 1950 | 150,697,361 | 19,028,086 | 14.5 |
| 1960 | 178,464,236 | 27,766,875 | 18.4 |
| 1968* | 199,861,000 | 21,396,764 | 12.0 |

2. U. S. Bureau of the Census, Statistical Abstract of the United States, 1964 (Eighty-fifth Edition) Washington, D. C., 1964, p. 5.

*Estimated as of July 1, 1968. Statistical Abstract of the United States 1969. U. S. Department of Commerce, Bureau of the Census, U.S.G.P.O., Washington, D. C.

ESTIMATED TOTAL POPULATION FOR THE UNITED STATES

JUNE 1, 1850 TO JULY 1, 1968



(b) Change in age composition.

Age Distribution of the Population
United States, 1850-1968[3,4]

| YEAR | Percent Distribution | | | | | |
|---|---|---|---|---|---|---|
| | Age | | | | | |
| | Total | Under 5 | 5-19 | 20-44 | 45-64 | 65 and Over |
| 1850 | 100.0 | 15.1 | 37.4 | 35.1 | 9.8 | 2.6 |
| 1860 | 100.0 | 15.4 | 35.8 | 35.7 | 10.4 | 2.7 |
| 1870 | 100.0 | 14.3 | 35.4 | 35.4 | 11.9 | 3.0 |
| 1880 | 100.0 | 13.8 | 34.3 | 35.9 | 12.6 | 3.4 |
| 1890 | 100.0 | 12.2 | 33.9 | 36.9 | 13.1 | 3.9 |
| 1900 | 100.0 | 12.1 | 32.3 | 37.8 | 13.7 | 4.1 |
| 1910 | 100.0 | 11.6 | 30.4 | 39.1 | 14.6 | 4.3 |
| 1920 | 100.0 | 11.0 | 29.8 | 38.4 | 16.1 | 4.7 |
| 1930 | 100.0 | 9.3 | 29.5 | 38.3 | 17.5 | 5.4 |
| 1940 | 100.0 | 8.0 | 26.4 | 38.9 | 19.8 | 6.9 |
| 1950 | 100.0 | 10.7 | 23.2 | 37.7 | 20.3 | 8.1 |
| 1960 | 100.0 | 11.3 | 27.1 | 32.2 | 20.1 | 9.2 |
| 1968* | 100.0 | 9.3 | 29.6 | 31.1 | 20.4 | 9.6 |

3. Spiegelman, Mortimer, Introduction to Demography, The Society of Actuaries, Chicago, p. 234.

4. U. S. Bureau of Census, op. cit., p. 24.

*Estimated as of July 1, 1968.

(c) Change in sex composition

Age-Sex Distribution of the Population

United States, 1850-1968[5,6]

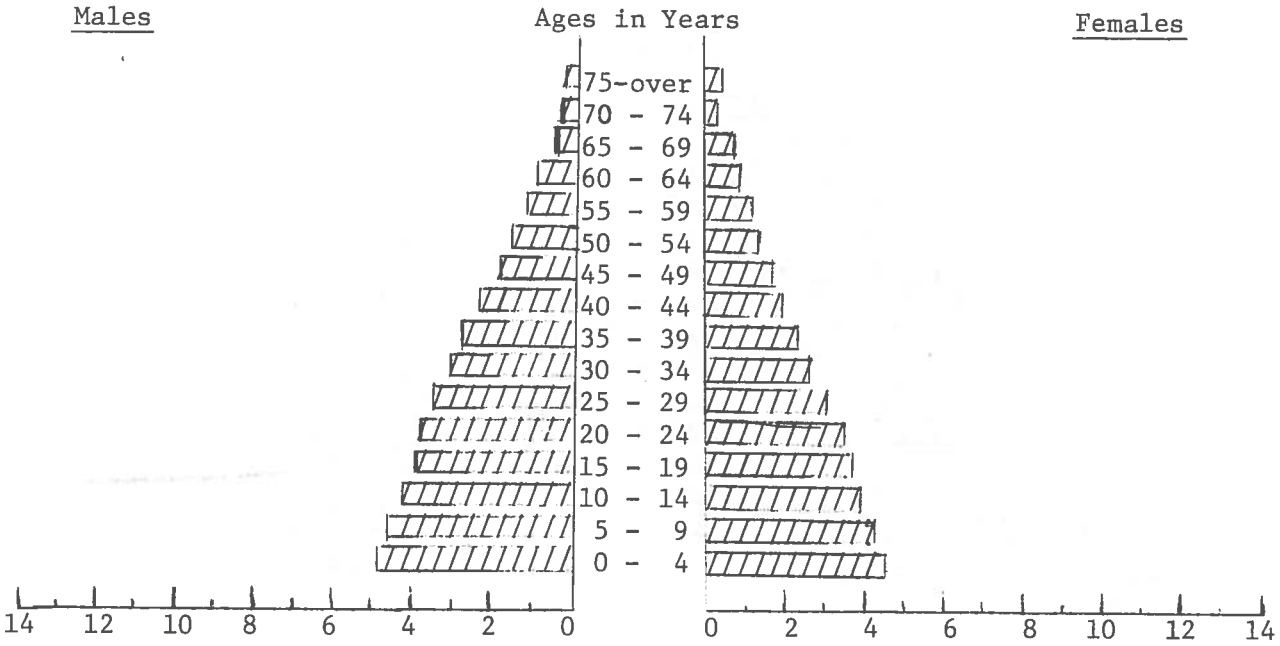
| Year | Males per 100 Females | | | | | |
|---|---|---|---|---|---|---|
| | Total | Under 5 | 5-19 | 20-44 | 45-64 | 65 and Over |
| 1850 | 104.3 | 102.4 | 100.9 | 108.1 | 106.4 | 101.3 |
| 1860 | 104.7 | 102.4 | 101.2 | 107.9 | 111.5 | 98.3 |
| 1870 | 102.2 | 102.9 | 101.2 | 99.2 | 114.5 | 100.5 |
| 1880 | 103.6 | 103.0 | 101.3 | 104.0 | 110.2 | 101.4 |
| 1890 | 105.0 | 103.6 | 101.4 | 107.3 | 108.3 | 104.2 |
| 1900 | 104.4 | 102.1 | 100.9 | 105.8 | 110.7 | 102.0 |
| 1910 | 106.0 | 102.5 | 101.3 | 108.1 | 114.4 | 101.1 |
| 1920 | 104.0 | 102.5 | 100.8 | 102.8 | 115.2 | 101.3 |
| 1930 | 102.5 | 103.0 | 101.4 | 100.5 | 109.1 | 100.5 |
| 1940 | 100.7 | 103.2 | 102.0 | 98.1 | 105.2 | 95.5 |
| 1950 | 99.0 | 103.9 | 102.9 | 97.0 | 100.2 | 89.6 |
| 1960 | 97.1 | 103.4 | 102.7 | 95.6 | 95.7 | 76.3 |
| 1968* | 95.4 | 104.2 | 103.1 | 95.0 | 92.1 | 74.9 |

5. Spiegelman, Mortimer, op. cit., p. 234.

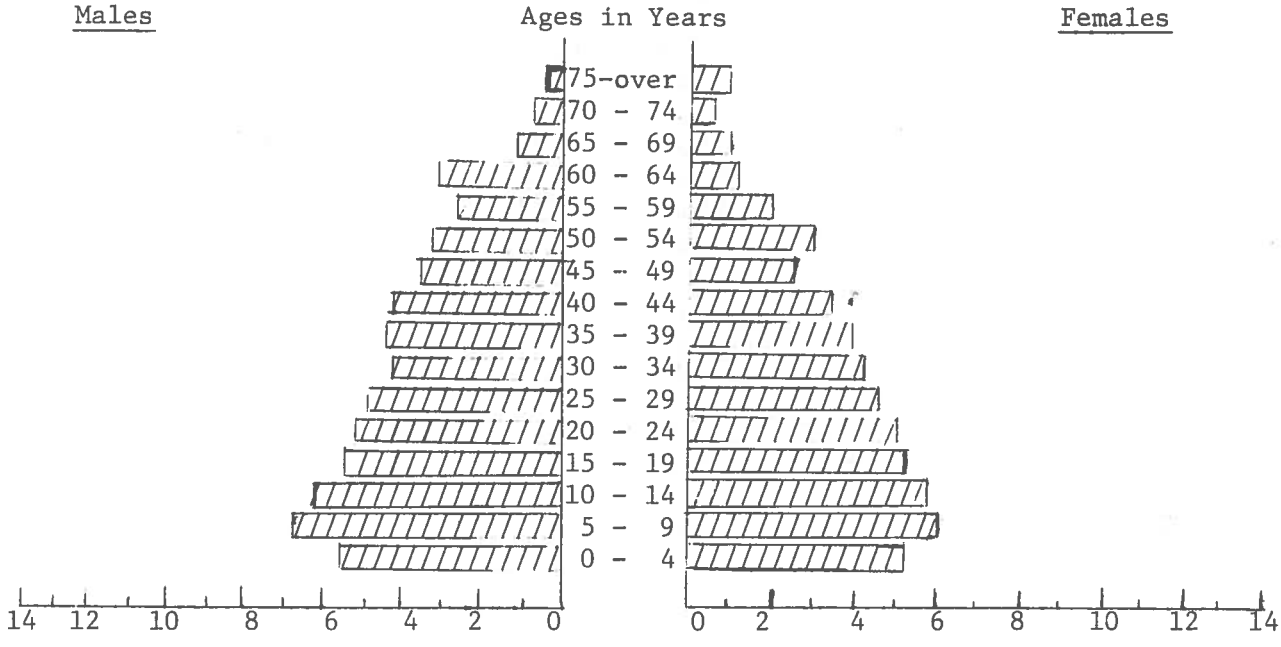
6. U. S. Bureau of Census, op. cit., p. 24.

*Estimated as of July 1, 1968.

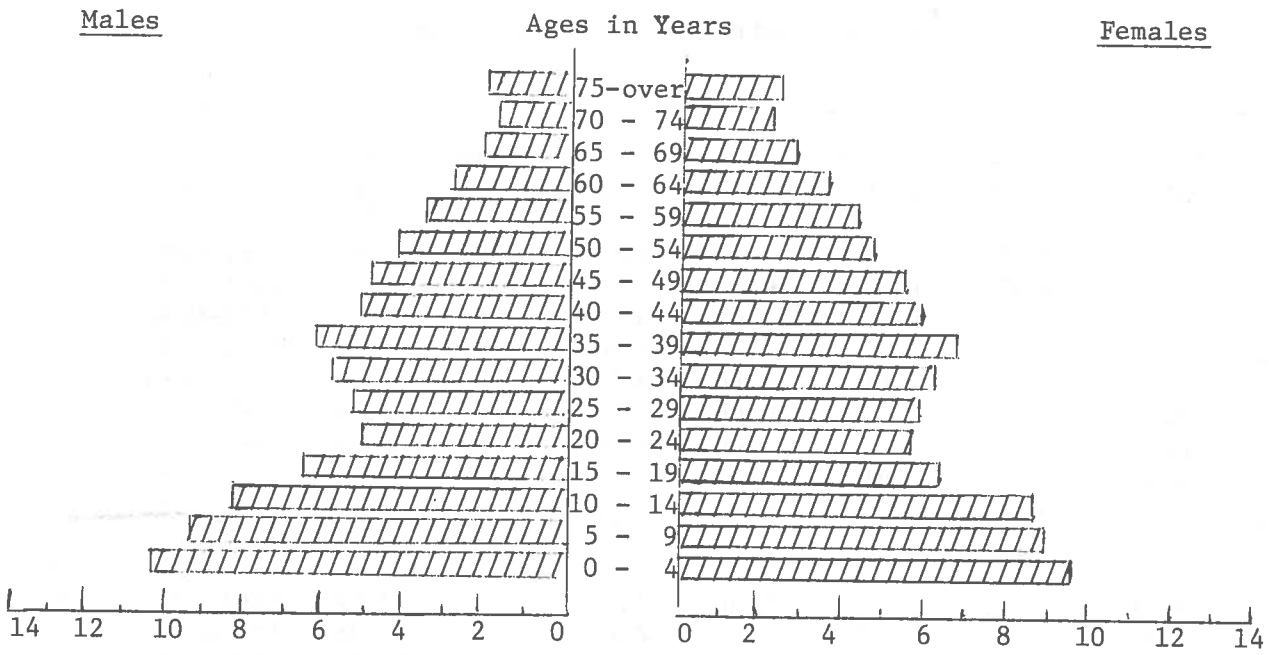
UNITED STATES - 1900




UNITED STATES - 1930

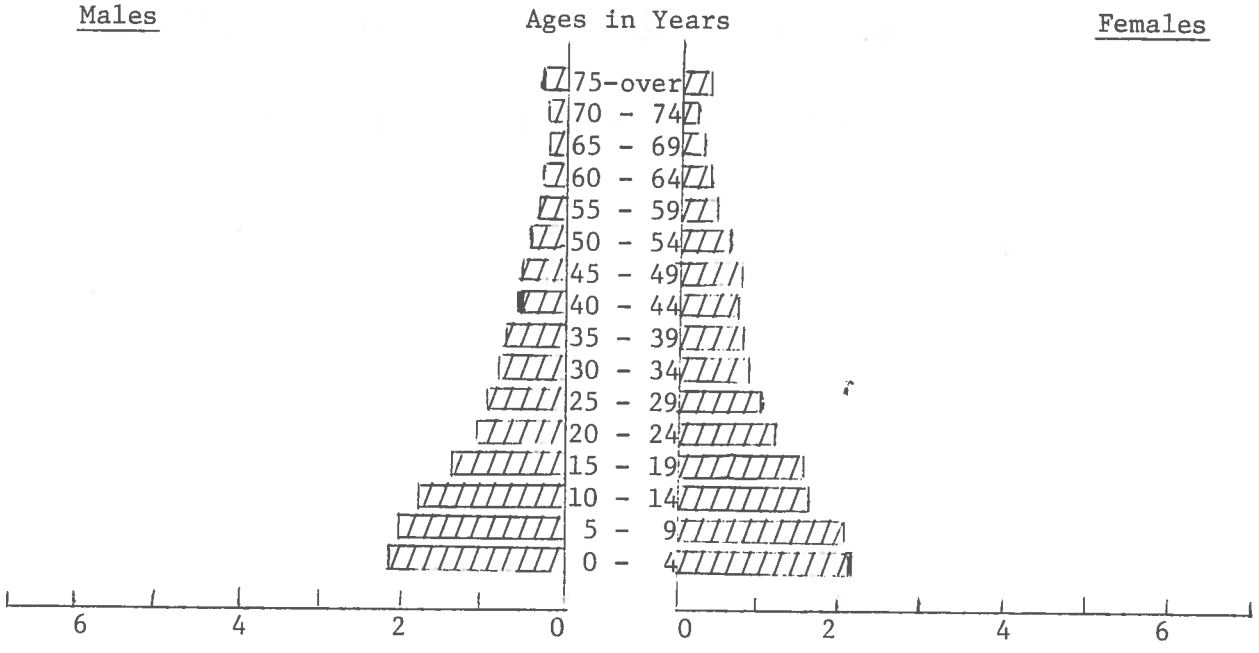


UNITED STATES - 1960




Source: U. S. Census of Population 1960 General Population Characteristics

PHILIPPINES - 1963



(e) Change in life expectancy

Average Expectation of Life in Years[7]

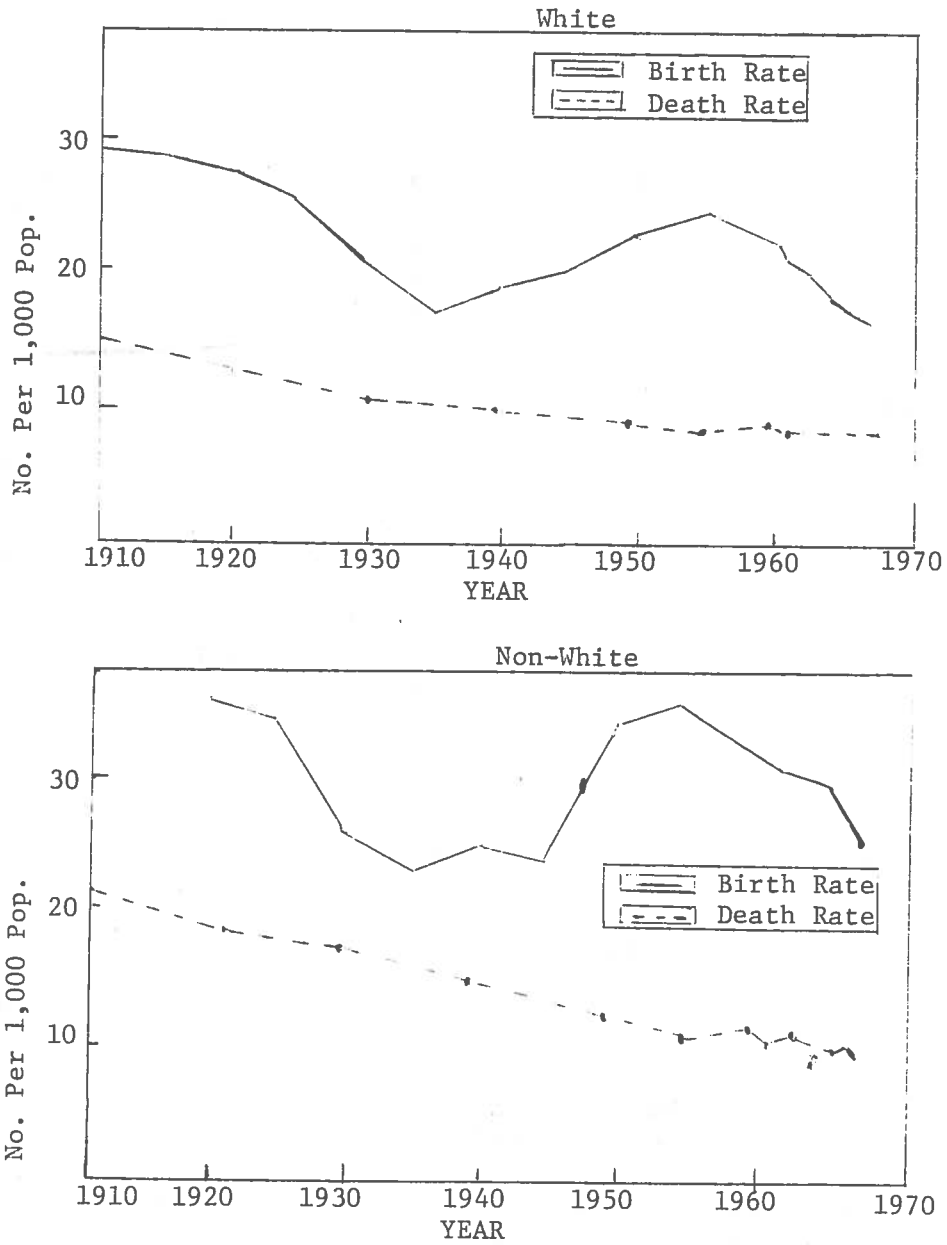
| | Year | At Birth | | Age 20 | | Age 40 | | Age 65 | |
|---|---|---|---|---|---|---|---|---|---|
| | | Male | Female | Male | Female | Male | Female | Male | Female |
| White | | | | | | | | | |
| | 1900-1902 | 48.2 | 51.1 | 42.2 | 43.8 | 27.7 | 29.2 | 11.5 | 12.2 |
| | 1909-1911 | 50.2 | 53.6 | 42.7 | 44.9 | 27.4 | 29.3 | 11.2 | 12.0 |
| | 1919-1921 | 56.3 | 58.5 | 45.6 | 46.5 | 29.9 | 30.9 | 12.2 | 12.8 |
| | 1929-1931 | 59.1 | 62.7 | 46.0 | 48.5 | 29.2 | 31.5 | 11.8 | 12.8 |
| | 1939-1941 | 62.8 | 67.3 | 47.8 | 51.4 | 30.0 | 33.2 | 12.1 | 13.6 |
| | 1949-1951 | 66.3 | 72.0 | 49.5 | 54.6 | 31.2 | 35.6 | 12.8 | 15.0 |
| | 1959-1961 | 67.6 | 74.3 | 50.3 | 56.4 | 31.8 | 37.2 | 13.0 | 16.0 |
| | 1962 | 67.6 | 74.4 | 50.2 | 56.4 | 31.7 | 37.3 | 12.9 | 16.0 |
| | 1967 | 67.8 | 75.1 | 50.2 | 56.9 | 31.8 | 37.8 | 13.0 | 16.5 |
| Non-White | 1900-1902 | 32.5 | 35.0 | 35.1 | 36.9 | 23.1 | 24.4 | 10.4 | 11.4 |
| | 1909-1911 | 34.0 | 37.7 | 33.5 | 36.1 | 21.6 | 23.3 | 9.7 | 10.8 |
| | 1919-1921 | 47.1 | 46.9 | 38.4 | 37.2 | 26.5 | 25.6 | 12.1 | 12.4 |
| | 1929-1931 | 47.6 | 49.5 | 36.0 | 37.2 | 23.4 | 24.3 | 10.9 | 12.2 |
| | 1939-1941 | 52.3 | 55.5 | 39.7 | 42.1 | 25.2 | 27.3 | 12.2 | 14.0 |
| | 1949-1951 | 58.9 | 62.7 | 43.7 | 46.8 | 27.3 | 29.8 | 12.8 | 14.5 |
| | 1959-1961 | 61.5 | 66.6 | 45.8 | 50.2 | 28.7 | 32.4 | 13.0 | 15.4 |
| | 1962 | 61.5 | 66.8 | 45.6 | 50.2 | 28.6 | 32.4 | 12.7 | 15.2 |
| | 1967 | 61.1 | 68.2 | 44.8 | 51.3 | 28.3 | 33.4 | 12.7 | 15.8 |

7. U. S. Bureau of Census, op. cit., p. 56.

PART II Some Dynamics of Population Change

a) Changing Birth Rate and Total Death Rate (1900-1960) - United States

Crude Birth and Death Rates per 1,000 Population
United States 1910 - 1967



Annual Mortality Rates per 1,000 Persons at Specific Ages
United States Death Registration States[8,9]

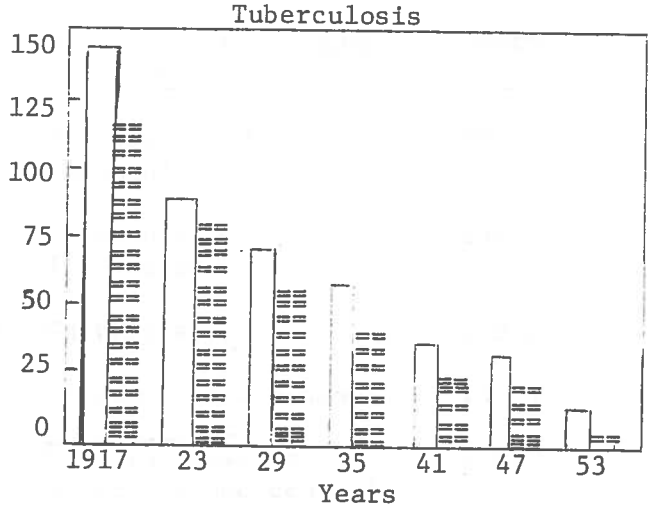
| | At Ages | | | | |
|---|---|---|---|---|---|
| Males | Under 1 Year | 1-4 | 15-24 | 45-54 | 55-64 |
| 1900 | 179.1 | 20.5 | 5.9 | 15.7 | 28.7 |
| 1910 | 145.5 | 14.6 | 4.8 | 15.2 | 28.7 |
| 1920 | 103.6 | 10.3 | 4.8 | 12.6 | 24.6 |
| 1930 | 77.0 | 6.0 | 3.5 | 13.6 | 26.6 |
| 1940 | 61.9 | 3.1 | 2.3 | 12.5 | 26.2 |
| 1950 | 37.3 | 1.5 | 1.7 | 10.7 | 24.1 |
| 1960 | 30.6 | 1.2 | 1.5 | 9.9 | 23.1 |
| 1964 | 27.8 | 1.0 | 1.6 | 9.6 | 22.9 |
| Percent Decrease 1900 to 1964 | 84.47 | 95.12 | 72.88 | 38.85 | 20.20 |
| Females | | | | | |
| 1900 | 145.4 | 19.1 | 5.8 | 14.2 | 25.8 |
| 1910 | 117.6 | 13.4 | 4.2 | 12.1 | 23.7 |
| 1920 | 80.7 | 9.5 | 5.0 | 11.7 | 22.4 |
| 1930 | 60.7 | 5.2 | 3.2 | 10.6 | 21.2 |
| 1940 | 47.7 | 2.7 | 1.8 | 8.6 | 18.1 |
| 1950 | 28.3 | 1.3 | 0.9 | 6.4 | 14.1 |
| 1960 | 23.2 | 1.0 | 0.6 | 5.3 | 12.0 |
| 1964 | 21.5 | 0.9 | 0.6 | 5.2 | 11.4 |
| Percent Decrease 1900 to 1964 | 85.21 | 95.28 | 89.65 | 63.38 | 55.81 |

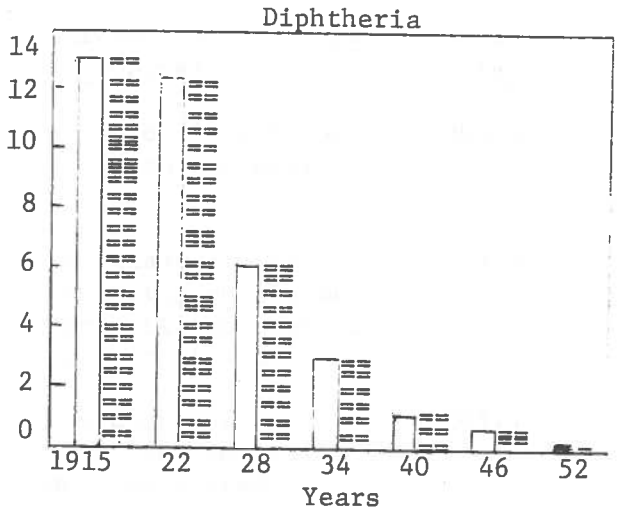
8. Vital Statistics Rates in the United States 1900 - 1940, United States Department of Commerce Bureau of the Census.

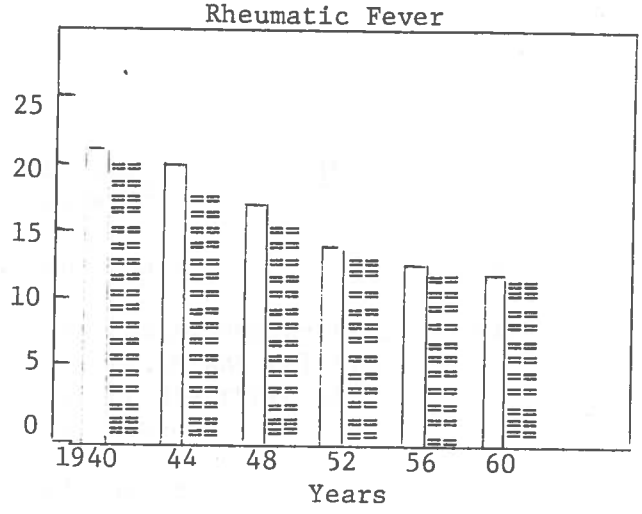
9. Vital Statistics of the United States 1950, 1960, 1964, United States Department of Health Education and Welfare, Public Health Service.

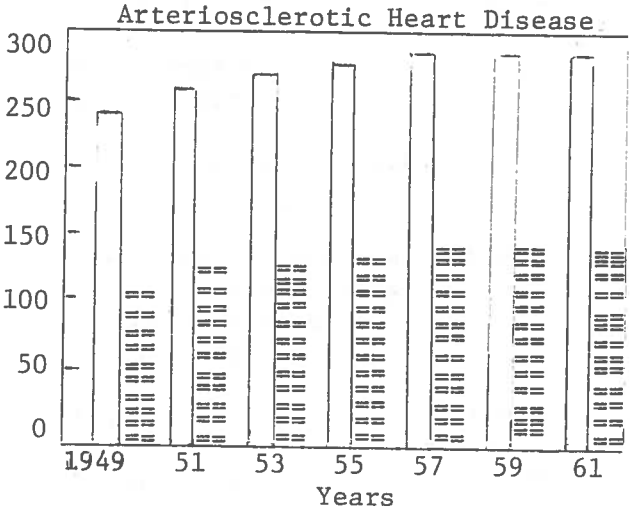
PART III: Changes in Disease Patterns

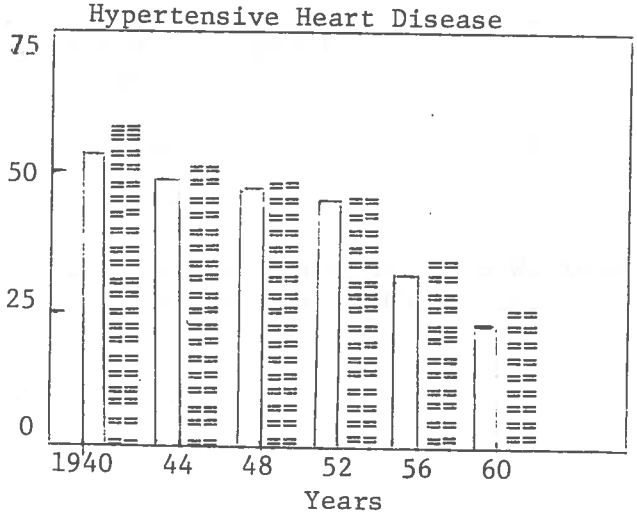
Changing Mortality Rates per 100,000 for Whites
By Sex (Age-Adjusted) for the United States

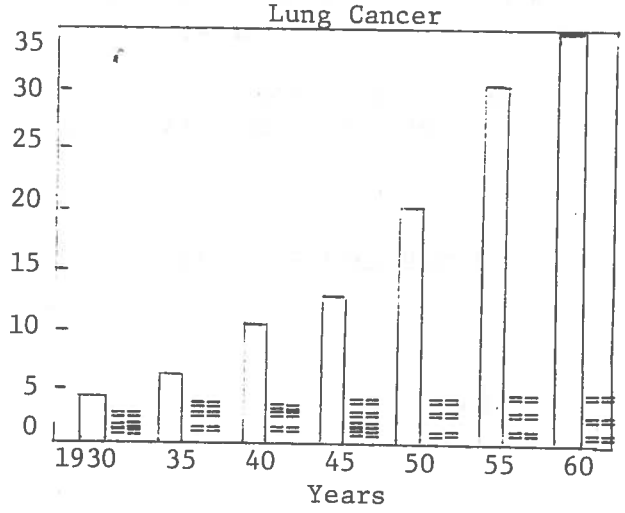


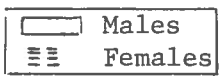



b) Five Leading Causes of Death for the United States and Selected Countries. (Death Rates per 100,000)

| | United States 1900 Cause | Death Rate[10] |
|---|---|---|
| 1. | Influenza and pneumonia | 202.2 |
| 2. | Tuberculosis (all forms) | 194.4 |
| 3. | Gastritis | 142.7 |
| 4. | Diseases of the heart | 137.4 |
| 5. | Vascular lesions affecting the central nervous system | 106.9 |

| | United States 1961 Cause[11] | Death Rate |
|---|---|---|
| 1. | Arteriosclerotic and degenerative heart disease | 304.3 |
| 2. | Malignant neoplasms, including neoplasms of lymphatic and haematopoietic tissues | 149.4 |
| 3. | Vascular lesions affecting central nervous system | 105.4 |
| 4. | All accidents | 42.9 |
| 5. | Hypertension with heart disease | 34.6 |

| | Ceylon 1961 Cause[11] | |
|---|---|---|
| 1. | Pneumonia | 49.2 |
| 2. | Gastritis, duodenitis, enteritis and colitis except diarrhoea of the newborn | 41.5 |
| 3. | All accidents | 27.1 |
| 4. | Arteriosclerotic and degenerative heart disease | 25.5 |
| 5. | Anaemias | 24.9 |

| | U.A.R. (Egypt) 1961 Cause[11] | |
|---|---|---|
| 1. | Gastritis, duodenitis, enteritis and colitis except diarrhoea of the newborn | 632.8 |
| 2. | Bronchitis | 150.7 |
| 3. | All accidents | 59.9 |
| 4. | Hypertension with heart disease | 44.6 |
| 5. | Arteriosclerotic and generative heart disease | 40.4 |

10. Monroe Lerner and Odin W. Anderson, Health Progress in the United States, 1900-1960, p. 16.

| | Guatemala 1961 Cause[11] | Death Rate |
|---|---|---|
| 1. | Gastritis, duodenitis, enteritis and colitis except diarrhoea of the newborn | 220.5 |
| 2. | Influenza | 115.8 |
| 3. | Pneumonia | 113.0 |
| 4. | Whooping cough | 74.7 |
| 5. | Measles | 61.2 |

| | Philippines 1961 Cause[11] | Death Rate |
|---|---|---|
| 1. | Tuberculosis, all forms | 87.1 |
| 2. | Pneumonia | 82.6 |
| 3. | Gastritis, duodenitis, enteritis and colitis except diarrhoea of the newborn | 57.5 |
| 4. | Bronchitis | 39.7 |
| 5. | Malignant neoplasms including neoplasms of lymphatic and haematopoietic tissue | 20.2 |

11. Annual Epidemiological and Vital Statistics, 1961, World Health Organization, Geneva, 1964.

The following non-specific categories have been excluded:

1. All other diseases classified as infective and parasitic (B17)
2. Other diseases of heart (B27)
3. Other diseases peculiar to early infancy, and immaturity unqualified (B44)
4. Senility without mention of psychosis, ill-defined and unknown causes (B45)
5. All other diseases (B46)

Also excluded:

1. Congenital malformations (B41)
2. Birth injuries, postnatal asphyxia and atelectasis (B42)
3. Infection of the newborn (B43)

All forms of tuberculosis have been grouped into one category (B1 and B2).

All forms of accidents have been grouped into one category (BE47 and BE48).

c) Infant Mortality Rates per 1,000 Live Births in Selected Countries, 1901-1960

| Year | U. S. | England and Wales | Netherlands | Sweden | New Zealand |
|---|---|---|---|---|---|
| 1901 | - | 151.3 | 149.3 | 102.9 | 71.4 |
| 1905 | - | 128.2 | 130.9 | 88.3 | 67.5 |
| 1910 | - | 105.4 | 107.9 | 75.1 | 67.7 |
| 1915 | 99.9 | 109.7 | 86.8 | 75.8 | 50.1 |
| 1920 | 85.8 | 79.9 | 82.5 | 63.3 | 50.6 |
| 1925 | 71.7 | 75.0 | 58.4 | 55.7 | 40.0 |
| 1930 | 64.6 | 60.0 | 50.9 | 54.7 | 34.5 |
| 1935 | 55.7 | 56.9 | 40.0 | 45.9 | 32.3 |
| 1940 | 47.0 | 57.4 | 39.1 | 39.2 | 30.2 |
| 1945 | 38.3 | 47.0 | 79.7 | 29.9 | 28.0 |
| 1950 | 29.2 | 29.9 | 25.2 | 21.0 | 22.7 |
| 1955 | 26.4 | 24.9 | 20.1 | 17.4 | 20.1 |
| 1960 | 25.2 | 22.4 | 15.8 | 15.0 | 19.5 |

12. Swaroop, Satya, Introduction to Health Statistics, E. & S. Livingstone, LTD., London, p. 272-273.

U. S. INFANT MORTALITY RATES BY RACE: BIRTH-REGISTRATION STATES, 1915-1960
(Exclusive of fetal deaths. Deaths under 1 year per 1,000 live births in each specified group)

| YEAR | All Races | White | Nonwhite |
|---|---|---|---|
| 1915 | 99.9 | 98.6 | 181.2 |
| 1916 | 101.0 | 99.0 | 184.9 |
| 1917 | 93.8 | 90.5 | 150.7 |
| 1918 | 100.9 | 97.4 | 161.2 |
| 1919 | 86.6 | 83.0 | 130.5 |
| 1920 | 85.8 | 82.1 | 131.7 |
| 1921 | 75.6 | 72.5 | 108.5 |
| 1922 | 76.2 | 73.2 | 110.0 |
| 1923 | 77.1 | 73.5 | 117.4 |
| 1924 | 70.8 | 66.8 | 112.9 |
| 1925 | 71.7 | 68.3 | 110.8 |
| 1926 | 73.3 | 70.0 | 111.8 |
| 1927 | 64.6 | 60.6 | 100.1 |
| 1928 | 68.7 | 64.0 | 106.2 |
| 1929 | 67.6 | 63.2 | 102.2 |
| 1930 | 64.6 | 60.1 | 99.9 |
| 1931 | 61.6 | 57.4 | 93.1 |
| 1932 | 57.6 | 53.3 | 86.2 |
| 1933 | 58.1 | 52.8 | 91.3 |
| 1934 | 60.1 | 54.5 | 94.4 |
| 1935 | 55.7 | 51.9 | 83.2 |
| 1936 | 57.1 | 52.9 | 87.6 |
| 1937 | 54.4 | 50.3 | 83.2 |
| 1938 | 51.0 | 47.1 | 79.1 |
| 1939 | 48.0 | 44.3 | 74.2 |
| 1940 | 47.0 | 43.2 | 73.8 |
| 1941 | 45.3 | 41.2 | 74.8 |
| 1942 | 40.4 | 37.3 | 64.6 |
| 1943 | 40.4 | 37.5 | 62.5 |
| 1944 | 39.8 | 36.9 | 60.3 |
| 1945 | 38.3 | 35.6 | 57.0 |
| 1946 | 33.8 | 31.8 | 49.5 |
| 1947 | 32.2 | 30.1 | 48.5 |
| 1948 | 32.0 | 29.9 | 46.5 |
| 1949 | 31.3 | 28.9 | 47.3 |
| 1950 | 29.2 | 26.8 | 44.5 |
| 1951 | 28.4 | 25.8 | 44.8 |
| 1952 | 28.4 | 25.5 | 47.0 |
| 1953 | 27.8 | 25.0 | 44.7 |
| 1954 | 26.6 | 23.9 | 42.9 |
| 1955 | 26.4 | 23.6 | 42.8 |
| 1956 | 26.0 | 23.2 | 42.1 |
| 1957 | 26.3 | 23.3 | 43.7 |
| 1958 | 27.1 | 23.8 | 45.7 |
| 1959 | 26.4 | 23.2 | 44.0 |
| 1960 | 26.0 | 22.9 | 43.2 |

d) Age and Sex Specific Death Rates for All Causes for United States and Selected Countries, 1961[14]

| Age | United States | | Austria | | Netherlands | | Sweden | | England and Wales | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Male | Female | Male | Female | Male | Female | Male | Female | Male | Female |
| 0 | 28.4 | 22.0 | 37.0 | 28.1 | 17.8 | 12.9 | 17.7 | 13.7 | 23.9 | 18.8 |
| 1-4 | 1.1 | 0.9 | 1.5 | 1.1 | 1.2 | 0.8 | 0.9 | 0.7 | 1.0 | 0.8 |
| 5-9 | 0.5 | 0.4 | 0.6 | 0.4 | 0.6 | 0.3 | 0.5 | 0.4 | 0.5 | 0.3 |
| 10-14 | 0.5 | 0.3 | 0.5 | 0.3 | 0.4 | 0.3 | 0.4 | 0.7 | 0.4 | 0.2 |
| 15-19 | 1.2 | 0.5 | 1.3 | 0.5 | 0.7 | 0.3 | 0.9 | 0.3 | 0.9 | 0.4 |
| 20-24 | 1.8 | 0.7 | 1.8 | 0.6 | 1.1 | 0.4 | 1.0 | 0.5 | 1.1 | 0.5 |
| 25-29 | 1.7 | 0.9 | 2.0 | 0.8 | 1.0 | 0.5 | 1.1 | 0.6 | 1.1 | 0.6 |
| 30-34 | 2.0 | 1.2 | 2.1 | 1.1 | 1.0 | 0.6 | 1.4 | 0.7 | 1.3 | 0.9 |
| 35-39 | 2.8 | 1.8 | 2.8 | 1.5 | 1.5 | 1.1 | 1.7 | 1.0 | 1.9 | 1.3 |
| 40-44 | 4.5 | 2.7 | 3.7 | 2.4 | 2.5 | 1.7 | 2.3 | 1.6 | 3.0 | 2.2 |
| 45-49 | 7.3 | 4.1 | 5.4 | 3.3 | 4.1 | 2.6 | 3.7 | 3.0 | 5.2 | 3.5 |
| 50-54 | 12.1 | 6.3 | 9.8 | 5.4 | 7.3 | 4.3 | 6.5 | 4.4 | 9.3 | 5.4 |
| 55-59 | 17.9 | 8.9 | 16.9 | 8.5 | 12.0 | 6.4 | 10.8 | 6.7 | 16.8 | 8.1 |
| 60-64 | 27.9 | 14.5 | 27.3 | 14.2 | 19.3 | 10.6 | 18.2 | 11.1 | 28.6 | 13.7 |
| 65-69 | 41.2 | 22.2 | 43.9 | 23.1 | 29.8 | 19.1 | 29.6 | 19.5 | 44.0 | 23.0 |
| 70-74 | 57.1 | 56.5 | 60.1 | 42.0 | 45.9 | 34.0 | 48.6 | 36.8 | 68.5 | 40.7 |
| 75-79 | 83.2 | 57.9 | | | 74.3 | 64.0 | 78.5 | 66.5 | 104.1 | 69.1 |
| 80-84 | 127.6 | 99.8 | 133.8 | 113.3 | 123.3 | 110.3 | 131.2 | 117.2 | 163.5 | 118.2 |
| 85 + | 209.1 | 187.8 | | | 216.3 | 200.9 | 231.5 | 211.4 | 257.4 | 227.1 |
| All Ages | 10.7 | 7.9 | 13.2 | 11.1 | 8.3 | 6.8 | 10.3 | 9.2 | 12.6 | 11.4 |

14. World Health Organization, op. cit., pp. 288-293.

ASSIGNMENT

Pages 1.2-1.9

1. What are the predominant features of the population changes in U.S. since 1850? And what are the implications for health and health care?

2. What are the similarities and differences between the population pyramids for U. S. and Phillipines?

3. Describe the major changes in life expectancy shown on page 1.9 for different time periods for each race and sex and age of the U. S.

Page 1.10

4. Describe the trend in birth and death rates in the U. S. for whites and non-whites.

5. What possible forces may explain the changes noted?

Page 1.11

6. What facts emerge in respect of the changing mortality rates for the different sex-age groups shown?

Pages 1.12-1.14

7. In respect of the 6 diseases shown what are the changes seen in mortality rates for each sex?

8. What changes have occurred in the leading causes of death in the U. S.?

9. How do they compare with the causes of death in Ceylon, U.A.R., Guatemala and the Phillipines?

Pages 1.15-1.16

10. Infant mortality rates have changed and are different in different countries. What have been the changes, and how do the countries shown compare?

11. What are possible explanations for the changes and the differences?

Page 1.17

12. What differences are shown in the age and sex specific death rates for the countries shown?

Summary

Briefly summarise the major changes in the U. S. population since 1910 and the extent to which births, deaths and life expectancy changes could have contributed to them.

# CASE HISTORY, CROSS-SECTIONAL AND COHORT STUDIES

PART I

A case history study was undertaken to determine whether patients with lung cancer differed from other persons in respect to their smoking habits. Patients initially diagnosed as having cancer of the lung and subjects without cancer of the lung were interviewed to find out the number of cigarettes smoked. It was a "blind" study, meaning that the interviewer did not know whether the respondent was a lung cancer patient or a control. The following table gives the percent of subjects, with and without lung cancer, according to the quantity of cigarettes smoked.

Table 1: Most Recent Amount Smoked by Subjects With and Without Cancer of the Lung (Response of Subjects at Interview)

| | Number of Cigarettes Smoked Daily | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | | 1-4 | | 5-14 | | 15-24 | | 25+ | | Total | |
| | No. | % | No. | % | No. | % | No. | % | No. | % | No. | % |
| Subjects with cancer | 56 | 6.1 | 65 | 7.1 | 352 | 38.3 | 255 | 27.8 | 190 | 20.7 | 918 | 100.0 |
| Subjects without cancer | 342 | 22.0 | 160 | 10.3 | 580 | 37.3 | 321 | 20.7 | 150 | 9.7 | 1553 | 100.0 |

What associations are shown in Table 1?

Could any of the biases inherent in case history studies have influenced the results of this study?

What additional data would enable you to test for this bias?

Remember that Table 1 was drawn from initial diagnosis. A more thorough diagnostic procedure on patients having cancer revealed that some persons who were first classified as having cancer did not actually have a malignancy.

Now knowing that some of the patients who thought they had cancer at the time they were interviewed about their smoking habits turned out to be incorrectly diagnosed, can you think of any way of using this information to check on the presence of any bias that you have suspected?

Table 2: Most Recent Amount Smoked by Subjects Incorrectly Diagnosed (Response of Subjects at Interview)

| | Number of Cigarettes Smoked Daily | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 0 | | 1-4 | | 5-14 | | 15-24 | | 25+ | | Total | |
| | No. | % | No. | % | No. | % | No. | % | No. | % | No. | % |
| Subjects incorrectly thought to have lung cancer | 35 | 16.7 | 25 | 12.0 | 83 | 39.7 | 50 | 23.9 | 16 | 7.7 | 209 | 100.0 |

Can you use this new information to test your hypotheses about bias?

How would you interpret these results?

PART II

Some recent studies have investigated the possible relationships of psychological variables, "personality types" or "behavior patterns," and coronary heart disease. In the study used in this exercise, Type A behavior pattern is defined as a person manifesting an intense, sustained drive for achievement and as being continually involved in competition and deadlines. Type B is the more relaxed person, not showing this intense drive and involvement in competition.

A study was done in which a representative sample of a given population was examined at one point in time and at this time persons were classified for behavior type and coronary heart disease.

Table 3: Comparative Prevalence of CHD with Type A and Type B Behavioral Patterns: Cross Sectional Study

| | 40-49 years | | 50-59 years | | Total Subjects | |
|---|---|---|---|---|---|---|
| Behavior Pattern Basis | CHD Present | CHD Absent | CHD Present | CHD Absent | CHD Present | CHD Absent |
| Type A | 41 | 1196 | 39 | 577 | 80 | 1773 |
| Type B | 19 | 1220 | 14 | 418 | 33 | 1638 |
| Total | 60 | 2416 | 53 | 995 | 113 | 3411 |

What association between behavior type and coronary heart disease is shown in Table 3?

How would you state these relationships quantitatively (in terms of rates)?

For purposes of your calculations, explain why you choose the figures you used for the numerator and denominator?

What are the possible limitations of cross-sectional studies?

For the further exploration of behavior type and the risk of coronary heart disease, what additional data do you need?

(Do not turn the page until you have worked through this problem)

A next step in this research involved the use of a cohort study design. In other words, a group of subjects without coronary heart disease but already classified in regard to behavior type were followed over a period of time to see how many developed coronary heart disease.

Table 4: Coronary Heart Disease by Age and Behavior Pattern. Cohort Study

| | 40-49 years | | 50-59 years | | Total subjects | |
|---|---|---|---|---|---|---|
| Behavior Pattern Basis | CHD Present | CHD Absent | CHD Present | CHD Absent | CHD Present | CHD Absent |
| Type A | 45 | 1072 | 49 | 530 | 94 | 1602 |
| Type B | 18 | 1186 | 21 | 394 | 39 | 1580 |
| Total | 63 | 2258 | 70 | 924 | 133 | 3182 |

What association between behavior type and coronary heart disease are shown in Table 4?

How would you state these relationships quantitatively?

Why did you choose the figures you used for the numerator and denominator?

Does this kind of study help you solve the antecedent--consequence problem (or the "cart before the horse" problem)?

What kinds of statements about the risk of coronary heart disease can you legitimately make from this type of study?

Calculate the relative risk of coronary heart disease for Type A persons and Type B persons for

a) ages 40-49
b) ages 50-59
c) Total subjects

If it were possible for us to change behavior pattern, how much coronary heart disease could be prevented in each age band and for the total sample?

PART III

For the purpose of studying coronary heart disease among Blacks and Whites in Evans County, Georgia, a sample of all persons aged 40-74 and 50 percent of the persons 15-39 was selected from the population. Medical histories, physical examinations and laboratory tests were performed on these persons. In addition, the social class was determined for each person based on their occupation, source of income and educational attainment.

Figure I

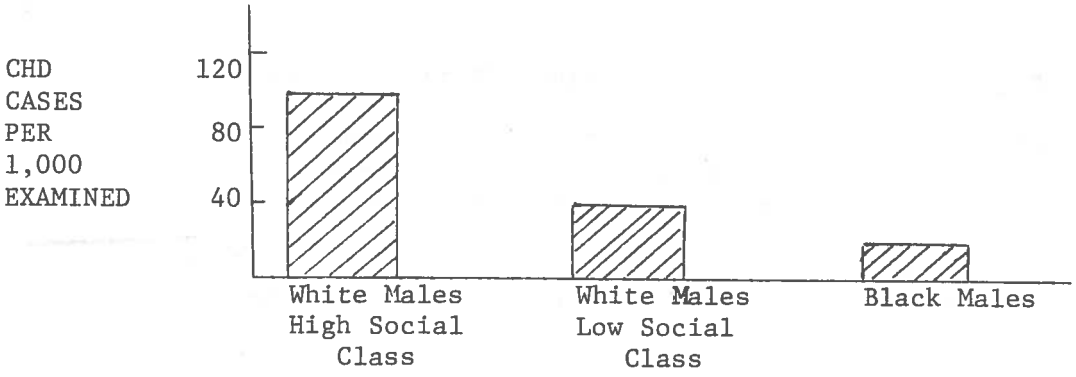



Figure I shows the prevalence of coronary heart disease by social class for White and Black males aged 40-74 years. The rates have been age-adjusted.

Because of the small number of cases of coronary heart disease, the White Males were classified into only upper and lower social class and the Black were not divided.

What associations are shown in Figure I?

What interpretations are possible?

What method could be used to determine if such a bias exists?

PART IV

The association between the occurrence of rubella (German measles) during pregnancy and the birth of a malformed infant was first described by N. McAlister Gregg.[1] He reported a study of 78 cases of congenital cataract. Thirteen of these were from his own patients and the others were from his colleagues. In all but two cases, the mother was asked if she had had German measles during her pregnancy. He described the frequency of rubella as follows:

| Total Number of Congenital Cataracts | 78 |
|---|---|
| German measles infection | 68 |
| History of kidney disease | 1 |
| No history of measles or unknown | 9 |

On the basis of these findings Dr. Gregg was convinced that there was a causal relationship between rubella and congenital cataracts.

1. a. Would you agree with this conclusion?

   b. What might have been some alternative explanations for his findings?

Gregg's findings led to an extensive series of studies by other investigators.

In Australia, Charles Swan conducted a series of investigations in different parts of the country. The principal method was (1) to have all notified cases of congenital anomalies examined to ensure accuracy of diagnosis, (2) to question the mother as to exposure to rubella during pregnancy and (3) if exposed, to determine at what stage of pregnancy the exposure occurred.

As a result of these investigations, Swan concluded: "On the available evidence, a woman who contracts rubella at some stage during the first 4 months of pregnancy has a 3 to 1 chance of giving birth subsequently to a

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1. Gregg, N. McAlister, "Congenital Cataract Following German Measles in the Mother," Transactions of the Opthalmological Society of Australia, 3 (1941), pp. 35-46.

congenitally defective infant. After the fourth month, the risk of congenital malformations is minimal. The main anomalies comprise cataract, deaf-mutism, cardiac disease and microcephaly. Termination of pregnancy is considered to be justifiable if a mother contracts German measles during the 'critical period,' i.e. the first 4 months of gestation."[2]

The data upon which these conclusions were based comprised 435 cases of congenital anomalies whose mothers admitted to having had rubella. The cases were distributed as follows:

| | Month of Pregnancy When Rubella was Contracted | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | Unkn. | Total |
| No. of congenitally malformed children | 90 | 150 | 105 | 44 | 7 | 8 | 7 | 3 | 1 | 20 | 435 |

2. Do these data support Swan's conclusions?

Subsequent to Swan's report, a number of further investigations using a different methodology were undertaken in various parts of the world. An example is the study conducted by Lundstrom[3] in Sweden in 1951 when a widespread epidemic of rubella occurred. The staff of all maternity hospitals in the country were requested to question all women who came for delivery or who were treated for spontaneous abortion concerning rubella during pregnancy. Information, then, was collected on the children born of all mothers who reported having had rubella (1029) and also from a random sample of mothers who had not contracted rubella (2226).

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2. Charles Swan, "Rubella in Pregnancy as an Aetiological Factor in Congenital Malformation, Stillbirth, Miscarriage, and Abortion," J. Obstet. and Gynae. Br. Emp., 56, 1949, 602.

3. Rolf Lundstrom, "Rubella During Pregnancy," Acta Ped., 41, 1952, 583-594.

The results were as follows:

| | Mothers Who Had Rubella | | Mothers Who Did Not Have Rubella |
|---|---|---|---|
| | 1-4 months of pregnancy | 5-9 months | |
| Total deliveries | 579 | 450 | 2226 |
| No. of deaths (still-births + neonatal deaths) | 34 | 9 | 40 |
| No. of deaths and/or malformations | 60 | 25 | 71 |
| Total no. of deaths, malformations, and prematures | 96 | 32 ~~32~~ ~~34~~ | 129 |

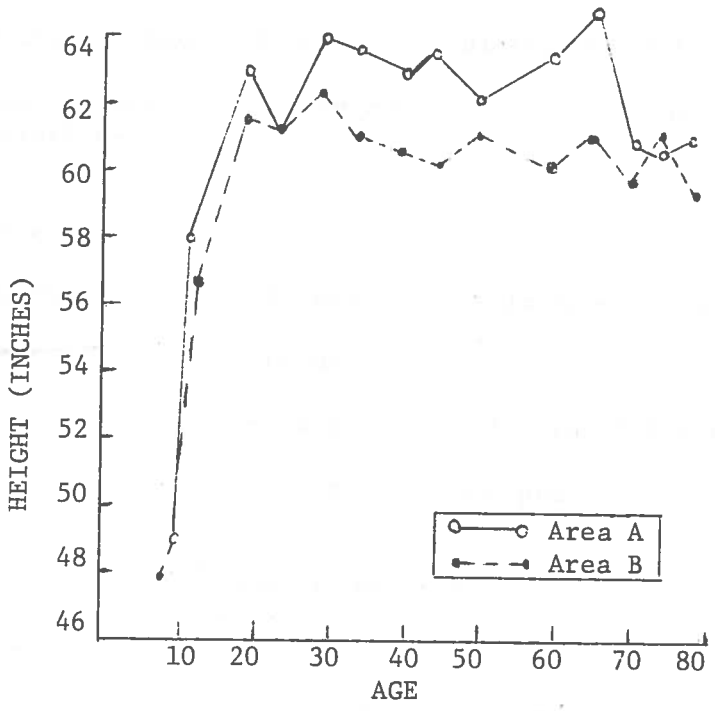
3. From those data what is the risk of the fetus either dying, being malformed, or being premature if the mother contracts rubella during the first 4 months of pregnancy? What is the risk if she contracts it in a subsequent month of pregnancy?

What explanations can you advance to explain the differences between the risks calculated from Lundstrom's data and those concluded by Swan?

PART V Interpretation of Incidence and Prevalence Data

The following three figures illustrate data drawn from community surveys. They are prevalence or cross-sectional surveys.

Figure 1: The Average Height of Females in Inches at Various Ages in Two Areas




Figures 2 and 3: The Percentage of Males and Females at Various Ages Diagnosed as Having Rheumatoid Arthritis in Two Areas (1953-1955)

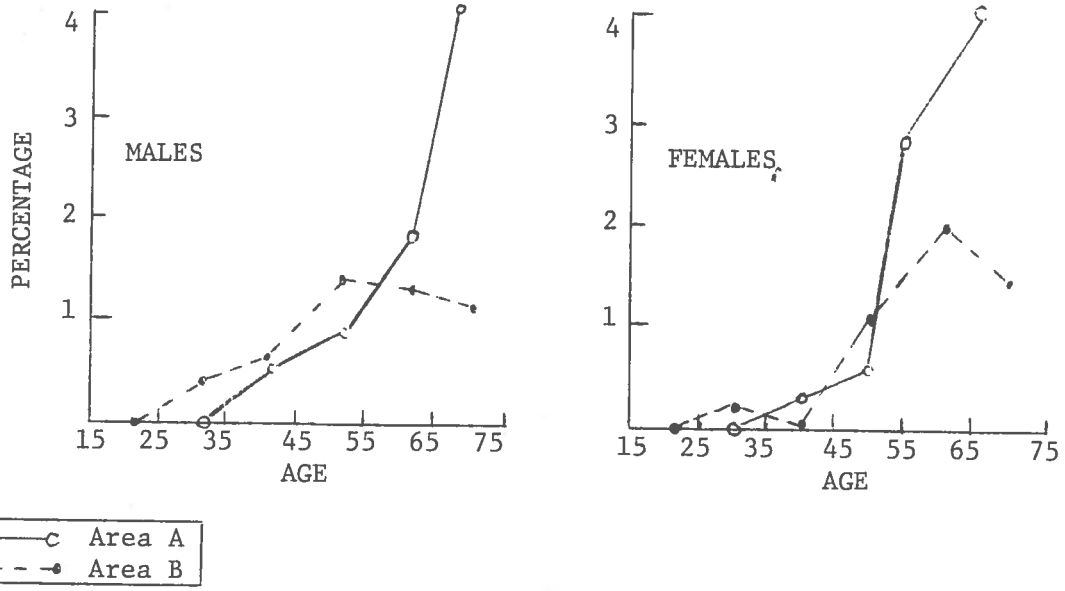


Figure 1

Describe the distribution of height by age in the two areas.

Is there a difference in the two areas?

What are the possible interpretations?

Could migration have influenced the distributions? If so, how?

What type of surveys would be necessary to provide data to correctly interpret these relationships?

Figure 2 and 3

Describe the prevalence by age and sex in each community.

How do the rates differ by areas?

What are the possible interpretations for the differences between the areas?

Could any of the following factors have produced these differences? And if so, in what way?

1. Different attack rates in each area.

2. Migration.

3. A more severe form of rheumatoid arthritis in one area than in the other.

Why are you unable to identify the reason for the difference?

What additional data is needed to identify the reason for the difference?

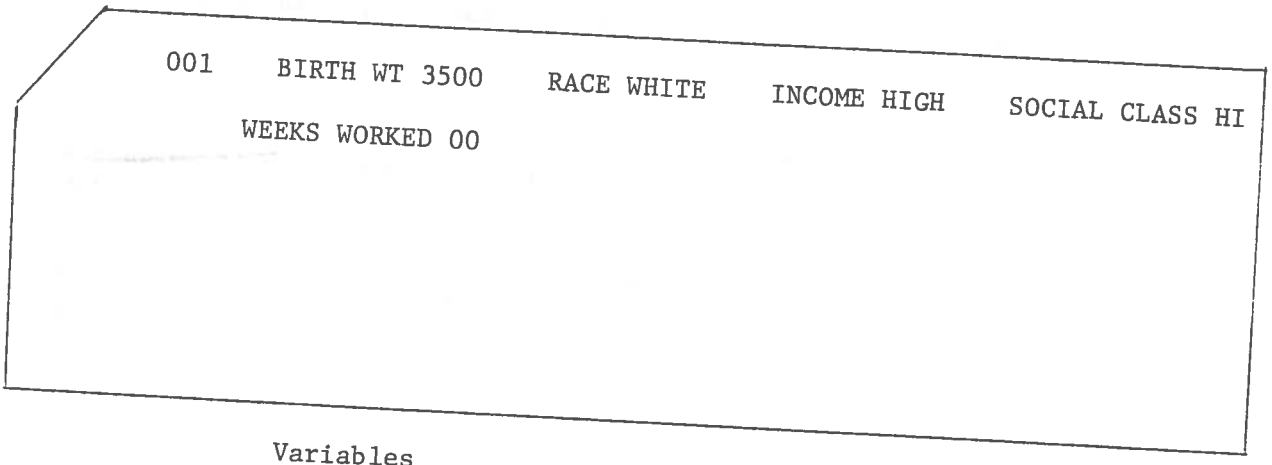
# TABLE GENERATION

Instructions:

Shuffle deck of cards to destroy any order that may have been imposed by previous workers.

Each card is the summarization of observations on an individual with the identification number for that particular individual.

Example - one individual




| Variables | Examples |
|---|---|
| State of Health (Dependent Variable or Condition): | |
| Birth Weight | 3500 (grams) |
| Four Group Characteristics (Independent Variable); | |
| Race | White |
| Income | High |
| Social Class | Hi |
| Amount of Time Worked (Weeks) | 00 |

Object:

To determine the association between the four group characteristics (race, income, social class, and amount of time worked) and the state of health (birth weight).

Prematurity is defined as birth weight of 2500 grams or less.

Specifically:

Prepare the appropriate tables to show:

1. The association between birth weight and race.

2. The association between birth weight and income.

3. The association between birth weight, race and income.

4. The association between prematurity and social class.

5. The association between prematurity and amount of time worked.

6. The association between prematurity, social class and amount of time worked.

NOTE: For Tables 1, 2, and 3, birth weight is used as a continuous variable; it is desirable to use a summarization statistic, e.g. mean weight.

For Tables 4, 5, and 6, the data is categorical, in the sense that a birth weight of 2500 grams or less defines prematurity while over 2500 grams defines maturity.

# ANALYSIS AND INTERPRETATION

OBJECTIVE: To Illustrate Some Principles of the Scientific Method: The Interrelationship of Variables

## I. Associations Between Two Variables:

For purposes of this course an association is said to exist between two variables when changes in one are accompanied by changes in the other.

Example (1)

Impairments by Age: (U. S., 1959)

| Age (in years) | Impairments: Rate per 1,000 Population |
|---|---|
| Under 25 | 52.9 |
| 25-44 | 130.6 |
| 45-64 | 212.4 |
| 65-74 | 376.6 |
| 75 and over | 615.0 |

Source: U. S. National Health Survey, 1959.

In this example the variables age and impairments are associated in that increasing age is accompanied by an increasing rate of impairment.

Example (2)

Impairments by Sex: (U. S., 1959)

| Sex | Impairments: Rate per 1,000 Population |
|---|---|
| Both Sexes | 141.4 |
| Male | 160.8 |
| Female | 123.1 |

Source: U. S. National Health Survey, 1959.

In this example impairments are associated with sex in that the impairment rate varies when the sex varies.

Exercise 1.

Number of Persons Injured per 1,000 Persons
Per Year by Age (U. S., July-December 1957)

| Age (in years) | Number Injured per 1,000 Persons per Year |
|---|---|
| Under 5 | 244 |
| 5-14 | 370 |
| 15-24 | 387 |
| 25-44 | 255 |
| 45-64 | 274 |
| 65+ | 265 |
| All Ages | 298 |

Source: U. S. National Health Survey, May, 1958.

What is the nature of the association demonstrated in the above table?

Exercise 2.

Infant Mortality from Bronchitis and Pneumonia by Family Size

| Number of Children Mother Has Borne | Number of Post Neonatal Deaths (28 days-1 year) Per 1,000 Live Births per Year |
|---|---|
| 1 | 3.2 |
| 2 | 4.6 |
| 3 | 7.6 |
| 4 | 9.4 |
| 5 and over | 13.8 |

Source: Morris, J. N., Uses of Epidemiology, E. & S. Livingstone, London, 1957, p. 79.

What is the nature of the association demonstrated in the above table?

Exercise 3.

Number of Duodenal Ulcers per 100,000 Population per Year by Age for Each Sex. (City of York 1952-1957)

| Age (in years) | MEN<br>Number of Duodenal Ulcers per 100,000 Population per Year |
|---|---|
| 15-24 | 143 |
| 25-34 | 268 |
| 35-44 | 237 |
| 45-54 | 220 |
| 55-64 | 247 |
| 65 and over | 148 |
| All Ages | 215 |

| Age (in years) | WOMEN<br>Number of Duodenal Ulcers per 100,000 Population per Year |
|---|---|
| 15-24 | 37 |
| 25-34 | 55 |
| 35-44 | 87 |
| 45-54 | 71 |
| 55-64 | 40 |
| 65 and over | 38 |
| All Ages | 57 |

What is the nature of the associations demonstrated in the above tables?

II. Interrelationships of More Than Two Variables:

In epidemiological studies some of the most valuable clues are obtained from analysis of the relationships between 3 or more variables.

The following examples are presented to illustrate the various types of interrelationships that may exist between 3 variables.

Exercise 4.

Some Variables Associated with Perinatal Mortality

Table 4A: Perinatal Mortality by Ethnic Group

| | No. of Deliveries | No. of Perinatal Deaths | Perinatal Mortality Rate Per 1,000 Deliveries |
|---|---|---|---|
| White | 8402 | 146 | 17.4 |
| Black | 6946 | 178 | 25.6 |
| Total | 15348 | 324 | 21.1 |

What conclusions can be drawn from the data presented in Table 4A?

Table 4B: Perinatal Mortality by Economic Status

| | No. of Deliveries | No. of Perinatal Deaths | Perinatal Mortality Rate Per 1,000 Deliveries |
|---|---|---|---|
| * Private Patients | 8844 | 149 | 16.8 |
| **Staff Patients | 6504 | 175 | 26.9 |
| Total | 15348 | 324 | 21.1 |

* = High economic status
** = Low economic status

What conclusions can be drawn from the data presented in Table 4B?

Taking the information from Tables 4A and B together we can now say that both ethnic group and income are associated with perinatal mortality. Specifically, the association is that Blacks and low income groups have high perinatal mortality rates. Whites and high income groups have lower infant mortality rates.

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Reference for Table 4A, B, and C:

Hendricks, Charles, M.D. "Delivery Patterns and Reproductive Efficiency Among Groups of Differing Socioeconomic Status and Ethnic Origins." American Journal of Obstetrics and Gynecology, 97(1967), 609.

The question that now must be asked is whether ethnicity is associated with perinatal mortality because of some purely racial factor or whether ethnicity is associated with perinatal mortality only because there is an undue concentration of one of the income groups in one ethnic group.

To put this in other words: Do Blacks tend to have high perinatal mortality rates because of some racial (genetic) factor common to Blacks, or is it because most Blacks in this sample are in the low income groups, and it is the low income that forms the crucial association with high perinatal mortality.

Exactly the same question could be asked about the association of low income groups and high perinatal mortality. Is this association brought about by virtue of some of the things in the way of life of low income groups that differs from the way of life of high income groups, or does this association exist because most of the low income groups from which these figures were drawn happen to be Blacks?

The answer to these questions are obtained by controlling each variable. This means comparing the perinatal mortality of Blacks in low income groups with the perinatal mortality rate of Whites in low income groups, then repeating the analysis for high income groups. In this way the income group variable has been held constant or controlled. The same data can be examined to find out the effect of holding ethnic group constant.

Table 4C: Perinatal Mortality by Ethnic Group and Economic Status

| | Private | Staff | Total |
|---|---|---|---|
| White | 137/7908 (17.3) | 9/494 (18.2) | 146/8402 (17.4) |
| Black | 12/936 (12.8) | 166/6010 (27.6) | 178/6946 (25.6) |
| Total | 149/8844 (16.8) | 175/6504 (26.9) | 324/15348 (21.1) |

You will notice in Table 4C that 3 variables - Ethnic Group, Income Group, and Perinatal Mortality Rate - all are included.

What conclusions can be drawn from Table 4C?

Exercise 5.

Some Variables Associated with Prematurity

Table 5A: Incidence of Prematurity in Relation to Work During Pregnancy

| | Number | Percentage of Births Which Were Premature |
|---|---|---|
| Mothers of single first births | 1318 | 6.8 |
| Mothers not gainfully employed during pregnancy | 780 | 4.7 |
| Mothers gainfully employed for less than 28 weeks of pregnancy | 285 | 8.4 |
| Mothers gainfully employed for more than 28 weeks of pregnancy | 253 | 11.1 |

Source: Stewart, A., "A Note on the Obstetric Effects of Work During Pregnancy." Br. J. Prev. and Soc. Med., 9: 159, July 1955.

What association is demonstrated by Table 5A?

Table 5B: Incidence of Prematurity According to Mothers' Social Class

| Social Class | Percentage of Live Births Born Prematurely |
|---|---|
| Highest social class | 5.5 |
| Lowest social class | 7.4 |

Source: Rider, Rowland V., et al., "Associations Between Premature Births and Socio-Economic Status." Am. J. P. H., 45:1022, 1955.

What association is demonstrated by Table 5B?

As in the previous exercise (Tables 4A, B, and C), we now have inter-relationships between 3 variables. Both work during pregnancy and social class are seen to be associated with prematurity. Specifically, mothers who work during pregnancy and low social class mothers have the highest prematurity rates.

Once again the questions must be asked as to whether mothers who work during pregnancy have a higher rate of premature births than do housewives because of some aspect of working, or because working mothers are more likely to be of low social class. We therefore have to control for the variables social class and working mothers.

Table 5C: Incidence of Prematurity According to Mothers' Social Class for Working and Non-Working Mothers

| | Percentage Premature Births | | |
|---|---|---|---|
| Social Class | Mothers not gainfully employed | Mothers employed for less than 28 weeks of pregnancy | Mothers employed for more than 28 weeks of pregnancy |
| Highest | 2.6 | 3.3 | 5.9 |
| Middle | 4.0 | 8.5 | 10.4 |
| Lowest | 7.8 | 10.4 | 13.7 |

Source: Stewart, A., "A Note on the Obstetric Effects of Work During Pregnancy." Br. J. Prev. Med., 9:159, 1955.

What conclusions can be drawn from Table 5C?

How do the relationships between the 3 variables - Working Mothers, Social Class, and Prematurity - differ from the relationships between the three variables - Ethnic Group, Economic Status, and Perinatal Mortality Rate (from Exercise 4)?

Exercise 6.

Some Variables Associated with Rheumatoid Arthritis

Table 6A: Prevalence of Rheumatoid Arthritis by Education

| Educational Level | Rheumatoid Arthritis per 100 Population |
|---|---|
| Less than 5th grade | 15.4 |
| 5th - 8th grade | 6.3 |
| 9th grade and over | 4.1 |

Describe the association demonstrated in Table 6A.

Table 6B: Prevalence of Rheumatoid Arthritis by Income Level

| Income Level | Rheumatoid Arthritis per 100 Population |
|---|---|
| Less than $3000 per year | 7.4 |
| $3000 - $4499 | 5.1 |
| $4500 and over | 3.2 |

Describe the association demonstrated in Table 6B.

From the accumulated data of Tables 6A and B, what is the next question that must be asked? How can this question be answered?

Table 6C: Prevalence of Rheumatoid Arthritis by Education and Income (Men Only)

| Income | Education | | |
|---|---|---|---|
| | Less than 5th grade | 5th - 8th grade | 9th grade & over |
| Less than $3000 | 6.5 | 4.5 | 10.0 |
| More than $3000 | 28.6 | 4.6 | 2.0 |

Source: (Tables 6A, B, and C) King, Stanley H., and Sidney Cobb. "Psychosocial Factors in the Epidemiology of Rheumatoid Arthritis." J. Chronic Dis., 7:466, 1958.

What are conclusions that can be drawn from Table 6C?

How do the relationships between the three variables - Education, Income, and Rheumatoid Arthritis - differ from the relationships between the three variables of Exercise 4 and the three variables of Exercise 5?