Chapter 5

Descriptive Studies

Descriptive epidemiologic studies reveal the patterns of disease occurrence in human populations. They provide general observations concerning the relationship of disease to basic characteristics. These characteristics include such personal items as age, sex, race, occupation, and social class. Also of great importance are geographic location and time of occurrence of disease. Thus, the major characteristics of interest in descriptive epidemiology may be summarized under the categories: person, place, and time.

At first glance, the goal of describing disease occurrence in this way may seem trivial and not worthy of the efforts of medical scientists. However, such studies are of fundamental importance and serve a variety of purposes—chiefly:

1 Alerting the medical community as to what types of persons (e.g., young or old, male or female, "white collar" workers or "blue collar" workers) are most likely to be affected by a disease, where the disease will occur, and when. This information is of great value to the physician in making a diagnosis, even though he may not be aware that he is using it.
2 Assisting in the rational planning of health- and medical-care facilities (e.g., number of coronary-care-unit beds needed for the cases of myocardial infarction in a particular community).
3 Providing clues to disease etiology and questions or hypotheses for further fruitful study (e.g., low prevalence of tooth decay in certain areas in the United States suggested further studies concerning the value of fluoride in drinking water).

PERSON

Basic demographic and social characteristics of persons constitute the attributes of greatest concern. Among these characteristics are age, sex, race or ethnic group, marital status, social class or socioeconomic status, religion, and occupation.

Age

Age is one of the most important factors in disease occurrence. Some diseases occur almost exclusively in one particular age group, such as hypertrophic pyloric stenosis in young infants or carcinoma of the prostate in the elderly. Other diseases occur over a much wider age span, but tend to be more prevalent at certain ages than others.

The time of life at which an infectious disease predominates is influenced by such factors as the degree of exposure to the agent at various ages, variations in susceptibility with age, and the duration of the immunity developed after infection. The influence of age-related exposure and duration of immunity is illustrated by the contrast between the single occurrence of chicken pox almost exclusively in young children and the repeated occurrence of gonorrhea, predominantly in adolescents and young adults. Chicken pox is readily transmitted among children playing together or gathering in classrooms, and it produces a lifelong immunity. Gonorrhea is transmitted by sexual contact and results in no immunity.

Many chronic or degenerative diseases such as coronary heart disease and osteoarthritis show a progressive increase in prevalence with increasing age. It is tempting to regard a disease with this age pattern as being due merely to aging itself. It should be remembered, however, that increasing age also marks the passage of time, during which the body is accumulating exposure to harmful environmental influences. For example, the wrinkling and loss of elasticity in skin that we associate with aging can be brought about or accelerated by chronic exposure to sunlight.

Instead of adopting the fatalistic view that a disease is an inevitable consequence of aging, a search for other causative factors should be undertaken. One of the great contributions of epidemiology in the past few decades has been to show that atherosclerosis and its consequences are not due merely to aging, as was previously thought, but that a person's habits and manner of living may contribute importantly to this disease process.

To see how age patterns of disease occurrence lead to clues and hypotheses, note the age trend, reported by Lilienfeld (1956), in the incidence of breast cancer among single and married women in New York State (Fig. 5-1). The steady geometric (note the logarithmic scale) increase in incidence with age diminishes sharply in the forties with a lesser continuing increase in the older years. The reduction in the forties of the rate of increase with age suggested the hypothesis that the hormonal changes of the menopause tend to decrease susceptibility to breast cancer. This hypothesis continues to be of great interest to scientists studying the causes of breast cancer.

Current and Cohort Age Tabulations The tabulation of disease rates in relation to age at one particular time, as in Fig. 5-1, is known as a *current*, or *cross-sectional*, presentation. This shows disease rates as they are occurring simultaneously in different age groups; thus, different people are involved in each age group. The other way to tabulate and analyze age relationships is in terms of *cohorts.* A cohort is a specific group of people, identified at one period of time and followed up as they pass through different ages during part or all of their life-span.

The results of cross-sectional and cohort age analysis can differ

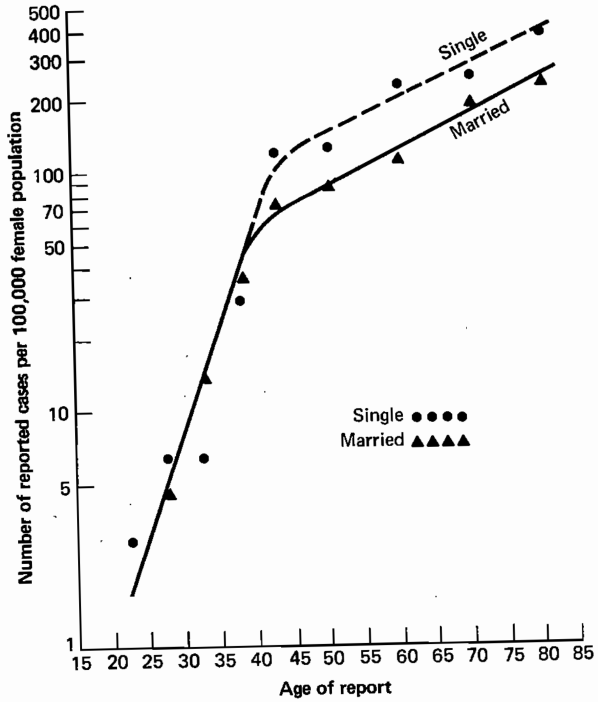


Figure 5-1 Annual age-specific incidence rates of reported cases of female breast cancer, by marital status, New York State exclusive of New York City, 1949. *(Reproduced, by permission, from Lilienfeld, 1956.)*

to a surprising degree, and either approach may be more appropriate for a particular problem. In a classic study, Frost (1939) compared cross-sectional and cohort age analyses of tuberculosis death rates in Massachusetts. Fig. 5-2 shows the cross-sectional curves for males in the years 1880, 1910, and 1930. First of all, note that at all ages the mortality rates decreased between 1880 and 1930. Also, observe that the shapes of the age curves were changing. The 1930 curve was of particular concern to public health workers in the 1930's because it showed tuberculosis mortality rates rising with age

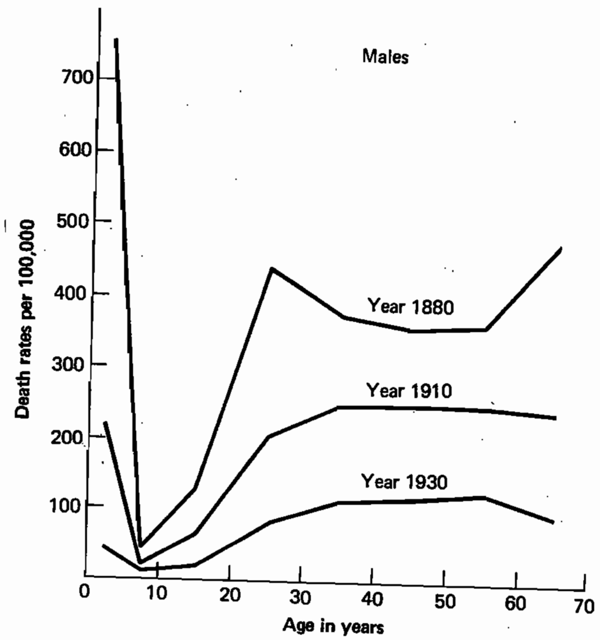


Figure 5-2 Massachusetts death rates from tuberculosis—all forms—by age, 1880, 1910, 1930. *(Reproduced, by permission, from Frost, 1939.)*

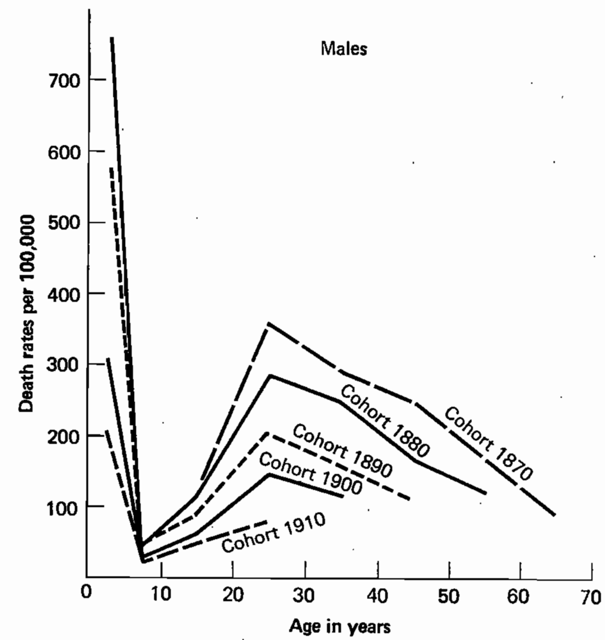
in adult life, reaching a maximum between age 50 and 60. This new age pattern of susceptibility to death from tuberculosis was thought possibly to be due to the failure of many individuals to become infected and acquire immunity during youth.

The matter was clarified, however, by a cohort analysis, shown in Fig. 5-3. Each curve represents the cohort of persons born in the 10-year period leading up to and including the year shown above the curve (e.g., 1870 cohort included individuals born from 1861–1870). In this cohort analysis it is apparent that each group experienced its maximum adult risk of tuberculosis death during the age decade of the twenties with subsequent decline in risk with age. Thus, for any particular group of adults, there was no increase in susceptibility with age, after all. The reason that, in 1930, persons in their fifties

had higher death rates than younger persons was because they belonged to the 1880 cohort, which experienced a greater exposure to tuberculosis than any of the succeeding cohorts.

Note that the frequently quoted calculations of average life expectancy are determined essentially on a cross-sectional rather than a cohort basis, using what are known as "life tables" (Hill, 1971). The presently observed annual mortality rates for each year of age are applied successively to a hypothetical population beginning either at birth or at some other starting point. It is assumed that as this hypothetical population ages, year by year, it will experience the same mortality rates for each year of life as are now observed in the current population at each age. Actually, the cohort of persons born now may be exposed to different risks of death as they go through

Figure 5-3 Massachusetts death rates from tuberculosis—all forms—by age, in successive 10-year cohorts. *(Reproduced, by permission, from Frost, 1939.)*



various age periods of life than are experienced by persons who are *now* in those age periods.

Sex

Some diseases occur more frequently in males, others, more frequently in females. If sex-linked inheritance can be excluded, a sex difference in disease incidence initially brings to mind the possibility of hormonal or reproductive factors that either predispose or protect. For example the greater occurrence of coronary heart disease in young men than in young women cannot be explained entirely by sex differences in the so-called coronary risk factors such as blood lipid concentrations, blood pressure, cigarette smoking, diabetes mellitus, and obesity. There may be some important hormonal factor that contributes to the male-female difference—perhaps protection of the female by estrogens before menopause. Similarly, the greater prevalence of gallstones in women than in men is probably attributable, in part, to the effects of repeated pregnancies and, in addition, to hormonal effects on bile composition.

But men and women differ in many other ways, including habits, social relationships, environmental exposures, and other aspects of day-to-day living. The higher male prevalence of cirrhosis of the liver and chronic bronchitis are at least partly related to the fact that, on the average, men currently drink more alcohol and smoke more cigarettes than women.

Sex differences in disease occurrence are important descriptive findings and often suggest avenues for further research. No disease can be considered to have a well-understood etiology if it manifests a male or female predominance which is not explained.

Race

Racial differences in disease prevalence have often been noted. In the case of some diseases (e.g., black-white differences in sickle-cell anemia and skin cancer), the differences are genetically determined. With other diseases, the explanation may not be so simple, especially when racial differences are accompanied by differences in socioeconomic status.

A case in point is the higher prevalence of hypertension and its complications in blacks than in whites in the United States. Suggested explanations have included (1) increased genetic susceptibility in blacks, (2) increased emotional stress in blacks due to racial discrimination, (3) lower average socioeconomic levels in blacks (since in whites the prevalence of hypertension is higher in lower socioeconomic groups), and (4) less access for blacks to good medical care (Howard and Holman, 1970). It may eventually be shown that some or all of these mechanisms are involved.

Marital Status

Marital status is another important descriptive variable. Married persons have lower mortality rates than single persons, including both overall mortality and mortality from most specific diseases. Whether the married state provides health benefits or whether characteristics favoring long life also predispose to marriage has not been decided.

Of great interest in studies of cancer etiology has been the contrast between cancer of the breast and cancer of the uterine cervix in their relation to marital status. Breast cancer is more apt to develop in single women or women who marry late in life, while cervical cancer is associated with early marriage. Further studies stemming, in part, from these observations suggest that cervical cancer is associated with coital activity at an early age and that having a first pregnancy at an early age may help protect a woman from breast cancer.

The data regarding the relationship of breast cancer incidence to age (see Fig. 5-1) also revealed a higher incidence in single women than in married women in their forties and later age decades. Lilienfeld suggested the hypothesis that early artificial or surgical menopause, occurring more often in married than in single women, might be protective against breast cancer. This hypothesis received some confirmation in an analytic case-control study in which it was found that (1) women with breast cancer less often gave a history of artificial menopause than did control subjects, and (2) married women more often gave a history of artificial menopause than did single women.

Socioeconomic Status

Socioeconomic status or social class is a somewhat nebulous concept, but it can be measured fairly conveniently by the occupation or income of the family head, by his or her educational level, or by residence, in terms of the value and amenities of the home or dwelling unit. The British have used occupation to define five social classes—I. Professional, II. Intermediate, III. Skilled, IV. Partly Skilled, and V. Unskilled. Using this classification system, the Registrar General for England and Wales has provided descriptive data relating social class to a variety of conditions.

As mentioned in our discussion of hypertension, many diseases show a distinct social class gradient, with higher rates in the lower socioeconomic classes. Included are rheumatic heart disease, chronic bronchitis, tuberculosis, stomach ulcer, stomach cancer, and nutritional-deficiency diseases.

On the other hand, low socioeconomic status appears to confer protection against some diseases. In the series of annual poliomyelitis epidemics that began in 1947, the higher social classes were the most severely affected. It is believed that in economically disadvantaged groups, poor sanitary conditions had resulted in widespread subclinical infection in the first few years of life, resulting in immunity. When "higher" living standards prevent this early infection, acquiring poliomyelitis later in childhood is more likely to cause paralytic disease.

A marked socioeconomic gradient in infant mortality has long been noted. Table 5-1 shows social class and rates of infant mortality (at age under one year) per 1,000 live births in England and Wales during two time periods, 1930–1932 and 1949–1953. Note that even though there was a marked improvement by the later time period, Social Class V still had over twice the infant mortality rate observed in Social Class I. Infant mortality rates have often been used as an index both of living standards and of availability of medical services in comparing nations or areas within a nation.

PLACE

Where disease occurs is a matter of great importance. Comparison of disease rates in different places may provide obvious clues to

Table 5-1 Infant Mortality Rates in England and Wales as Related to Social Class during Two Time Periods, 1930–1932 and 1949–1953

Social class	Infant mortality rates* 1930–1932	Infant mortality rates* 1949–1953
I	32.7	18.7
II	45.0	21.6
III	57.6	28.6
IV	66.8	33.8
V	77.1	40.8
All classes	61.6	29.5

*Deaths of infants under one year old per 1,000 live births. Registrar General's data, Taylor and Knowelden (1964).

etiology or serve as a stimulus to further fruitful investigation. The places of concern may be as large as a continent or as small as part of a room. As illustrative examples, descriptive findings will be presented from international comparisons, comparisons of regions within the United States and Canada, and comparisons of areas in a city.

International Comparisons

Because of the problems regarding the validity of mortality statistics, described in Chap. 3, it is difficult to take seriously small differences among nations in mortality rates for specific diseases. However, it is also difficult to explain away very large differences as due to artifact—that is, where the death rate for a disease in one country is two or three times as large as the death rate in another. Large differences are particularly impressive when both countries are known to have reasonably good vital statistics systems.

The Unique Position of Japan Ranking the disease-specific mortality rates of various nations has revealed Japan to be among the highest nations for some diseases and among the lowest for others. Table 5-2 shows some age-adjusted or age-specific mortality rates for stomach cancer, colon cancer, breast cancer, cerebrovascular disease (primarily strokes), and coronary heart disease. Note that among the nations studied, Japan is the highest ranking country for stomach cancer and cerebrovascular disease and the lowest ranking for breast cancer, colon cancer, and coronary heart disease.

Because of Japan's unique position among nations regarding these diseases, consideration of the mode of life in Japan has suggested a number of questions and hypotheses for further study. The relatively frequent practice and long duration of breast feeding of infants in Japan has raised the question of whether lactation may diminish the risk of developing breast cancer. The traditional Japanese diet has come under considerable scrutiny in hopes of finding predisposing factors for stomach cancer and protective factors for colon cancer. Also, the low fat intake in Japan has been thought responsible for the low average serum-cholesterol levels observed there and the low incidence of coronary heart disease. Although some portion of the high cerebrovascular death rate may be due to a known tendency of Japanese to attribute any sudden death to a cerebral hemorrhage, the high salt intake of Japanese has come under suspicion as a possible predisposing factor for hypertension and stroke.

Many traditional practices in Japan are now changing, and it will be of considerable interest to learn whether disease rates will change in ways that are consistent with the above hypotheses. Already, the migration of Japanese to places where they adopt new eating and living habits has permitted comparative studies aimed at identifying environmental factors that predispose to disease. Gordon (1957) compared mortality rates for Japanese in Japan, Hawaii, and the United States mainland and found contrasting trends for cerebrovascular disease and for coronary heart disease. Cerebrovascular disease mortality rates in both sexes decreased, and coronary mortality rates in men increased from Japan to Hawaii to the United States mainland. This suggested that as Japanese were adopting "the American way of life," their susceptibility to the two diseases in question was moving in the direction of that found in other Americans. (The assumption that migrants are genetically similar to those who remain in their native land should always be viewed with caution.) In order to explore in detail the reasons for the

Table 5-2 Ranking of Death Rates of Various Countries, from Highest to Lowest*

Rank	Stomach cancer, males, 1964–1965	Colon cancer (except rectum), males, 1964–1965	Breast cancer, females, 1964–1965	Cerebrovascular disease, males, ages 65–74, 1964	Coronary heart disease, males, ages 45–54, 1964
1	Japan (69)	Scotland (16)	Netherlands (26)	Japan (1,680)	Finland (442)
2	Chile (58)	Denmark (14)	England & Wales (24)	Scotland (901)	Scotland (358)
3	Austria (42)	U.S.A.—white (14)	Denmark (24)	Finland (751)	U.S.A. (354)
4	Finland (40)	Canada (13)	Scotland (24)	West Germany (750)	Australia (324)
5	West Germany (37)	New Zealand (13)	Canada (23)	Italy (708)	Northern Ireland (324)
6	Italy (34)	Northern Ireland (13)	New Zealand (23)	Hungary (706)	Canada (311)
7	Portugal (33)	Ireland (13)	South Africa (23)	Austria (663)	New Zealand (293)
8	Netherlands (28)	Australia (12)	Northern Ireland (22)	Northern Ireland (656)	England & Wales (245)
9	Belgium (27)	Belgium (12)	U.S.A.—white (22)	Israel (614)	Israel (214)
10	Switzerland (26)	England & Wales (12)	Switzerland (22)	England & Wales (612)	West Germany (183)
11	Norway (26)	France (12)	Ireland (22)	Australia (611)	Denmark (181)
12	Scotland (25)	Switzerland (11)	Belgium (21)	Czechoslovakia (560)	Norway (164)
13	South Africa (25)	U.S.A.—nonwhite (11)	Israel (21)	Switzerland (530)	Netherlands (162)
14	Ireland (24)	Netherlands (11)	U.S.A.—nonwhite (20)	France (528)	Austria (159)
15	England & Wales (23)	South Africa (11)	Australia (19)	U.S.A. (495)	Belgium (159)
16	Sweden (22)	Austria (10)	Sweden (19)	Norway (492)	Czechoslovakia (151)
17	Northern Ireland (22)	West Germany (10)	West Germany (18)	Denmark (478)	Hungary (147)
18	Denmark (22)	Sweden (10)	Austria (17)	New Zealand (475)	Switzerland (134)
19	France (21)	Norway (8)	Norway (17)	Netherlands (416)	Italy (133)
20	Israel (18)	Italy (8)	France (16)	Canada (414)	Venezuela (131)
21	U.S.A.—nonwhite (18)	Portugal (8)	Italy (16)	Sweden (394)	Sweden (125)
22	Canada (18)	Israel (7)	Finland (14)	Belgium (334)	France (74)
23	New Zealand (17)	Finland (5)	Portugal (13)	Venezuela (281)	Japan (51)
24	Australia (15)	Chile (4)	Chile (9)		
25	U.S.A.—white (9)	Japan (3)	Japan (4)		

*Death rates per 100,000 shown in parentheses (rounded from the original).

Sources: Stomach, colon, and breast cancer: Segi et al. (1969); cerebrovascular and coronary heart disease: World Health Organization (1967).

above geographic trends in cerebrovascular and coronary disease, parallel data collection methods have been established in three on-going epidemiologic studies of Japanese, located in Hiroshima, Japan; Honolulu, Hawaii; and in the San Francisco Bay area (Belsky et al., 1971).

Comparisons of Regions within Countries

The availability of mortality statistics for states and finer geographic subdivisions in the United States and other nations has permitted the discovery of interesting place-to-place variations in disease occurrence. Differences in mortality rates between urban and rural areas are a common finding. The higher mortality from lung cancer in cities than in farming areas is consistent with an etiologic role of either cigarette smoking or air pollution, since both are more common in cities.

The North-to-South Gradient of Multiple Sclerosis Geographic variation within nations may take the form of a distinct north-to-south gradient, which suggests that climate or other factors related to latitude may be involved. An example is the finding in the United States and Canada of generally higher mortality rates for multiple sclerosis the farther north one looks (Fig. 5-4). Confirmation of the north-to-south trend also comes from other nations and from prevalence rates found in several cities (Fig. 5-5). While hypotheses abound, to date no one has convincingly explained this geographic distribution of multiple sclerosis (Alter, 1968).

Areas within a City

When studying disease occurrence within a city, it is often desirable to plot the occurrence of disease in each census tract, since information about other characteristics of persons in each tract is available.

Rheumatic Fever in Baltimore Figure 5-7 from Gordis et al. (1969) shows the distribution in Baltimore census tracts of the homes of hospitalized rheumatic fever cases in 1960–1964. Most of

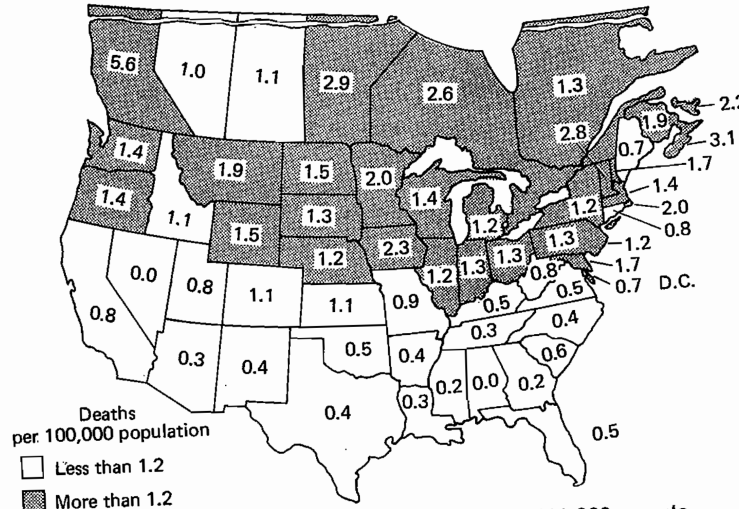
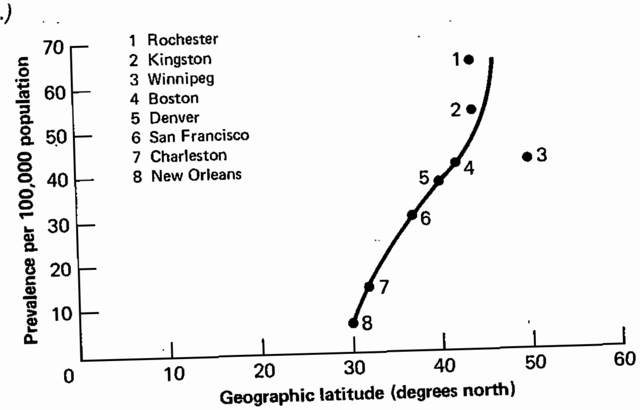


Figure 5-4 Multiple sclerosis mortality rates per 100,000 population in the U.S. states and in Canadian provinces. *(Reproduced, by permission, from Alter, 1968, adapted from Limburg, 1950.)*

Figure 5-5 Multiple sclerosis prevalence rates in cities of the United States and Canada. *(Reproduced, by permission, from Alter, 1968.)*



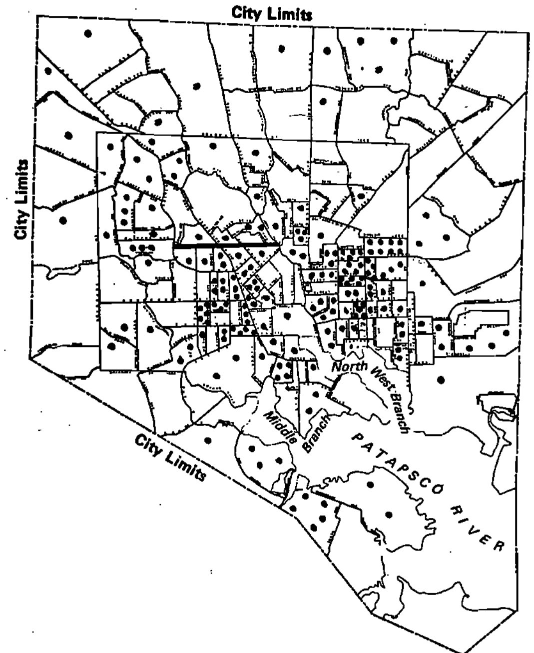


Figure 5-6 Residence distribution of hospitalized rheumatic fever patients, Baltimore 1960–1964. Heavy line shows North Avenue. Each dot represents one patient. *(Reproduced, by permission, from Gordis et al., 1969.)*

the cases occurred in two clusters on either side of the central business district—in the low-income area south of North Avenue, which is shown by a heavy line. Naturally, we cannot form a judgment based only on the "numerator" cases but must relate these to the "denominator" populations in each tract to develop rates.

Thus, annual incidence rates were calculated for groups of census tracts in this area and ranged as high as 40.2 per 100,000. In contrast, the incidence rate for the entire city was only 15.6 per 100,000.

Studying the high and low incidence areas further revealed that nonwhite children suffered a higher incidence of rheumatic fever than did whites. Only among whites was higher socioeconomic

status related to lower incidence of rheumatic fever. When housing characteristics were examined, the degree of crowding was the variable that was most closely related to rheumatic fever occurrence. The authors emphasized the importance of socioeconomic conditions in this disease and showed that the higher incidence in nonwhites might have been due to the crowded living conditions in which most Baltimore nonwhites lived.

TIME

The pattern of disease occurrence in time is often an extremely informative descriptive characteristic. A great variety of time trends may be found in the literature; these involve simple increases or decreases of disease incidence, or more complex combinations of these changes in time. To provide an introduction to this interesting subject, a few examples of short-term, periodic, and long-term trends will be described.

Short-term Increases and Decreases in Disease Incidence

Short-term changes are those increases or decreases in disease incidence that are measured in hours, days, weeks, or months. These are most often observed in the study of epidemics of infectious disease, as will be illustrated below. However, important short-term trends also have been noted in the occurrence of symptoms of, or even deaths due to, chronic noninfectious disease, in relation to both natural phenomena such as heat waves and man-made stresses such as marked increases in air pollution.

Epidemics An *epidemic*, or *outbreak*, is the occurrence of a disease in members of a defined population clearly in excess of the number of cases usually or normally found in *that* population. In investigating epidemics, a careful tabulation of the distribution of disease-onset times of the affected members of the population, in terms either of counts of cases or incidence ("attack") rates, may be very helpful in determining the initiating causes and mechanism of spread.

For a thorough discussion of the propagation of epidemics the reader is referred to Sartwell (1965). A few basic principles should be mentioned, however, before we consider some examples of time patterns. Epidemics only affect susceptible members of the population, of which there may be many or few. Others in the population are immune due to antibodies related to previous disease occurrence, immunization, or passive transfer from mother to infant. Still others may be resistant due to other inherent factors. After a person is exposed to the disease-causing agent, there is an incubation period until the disease first appears. Susceptible persons may also develop inapparent infections, in which no symptoms or signs become evident. The infectious agent may leave the host during the communicable period, which varies in timing and duration from one disease to the next.

Infections are transmitted from one person to another in a variety of ways: by direct personal contact, by touching contaminated objects, or by droplets spraying from one person to another close by, as during talking or sneezing. Evaporation of such droplets may yield "droplet nuclei" which, like certain disease-carrying dusts, may remain airborne for longer periods and travel longer distances. Other modes of transmission include vehicles such as certain foods or water, and *vectors* such as arthropods which carry the infectious agent.

The infection is usually introduced into the population directly or indirectly by one or more persons. If there is a sufficient proportion of susceptibles and the infection spreads rapidly enough, the disease will show a trend of increasing incidence through time to a maximum, followed by a fairly steady diminution until the disease disappears completely or almost completely. The decrease is largely due to the fact that the population begins to run out of susceptible individuals as those who were previously susceptible acquire the disease and become immune. As susceptibles become increasingly scarce, the infectious agent, no matter how well and how rapidly transmitted, finds less and less fertile soil in which to grow, so to speak. The rise through time from a negligible incidence rate to a maximum followed by a fall to low levels again appears graphically as a simple epidemic curve, usually but not always involving short-term trends involving days, weeks, or months.

Fig. 5-7 shows an epidemic of measles (rubeola) that occurred

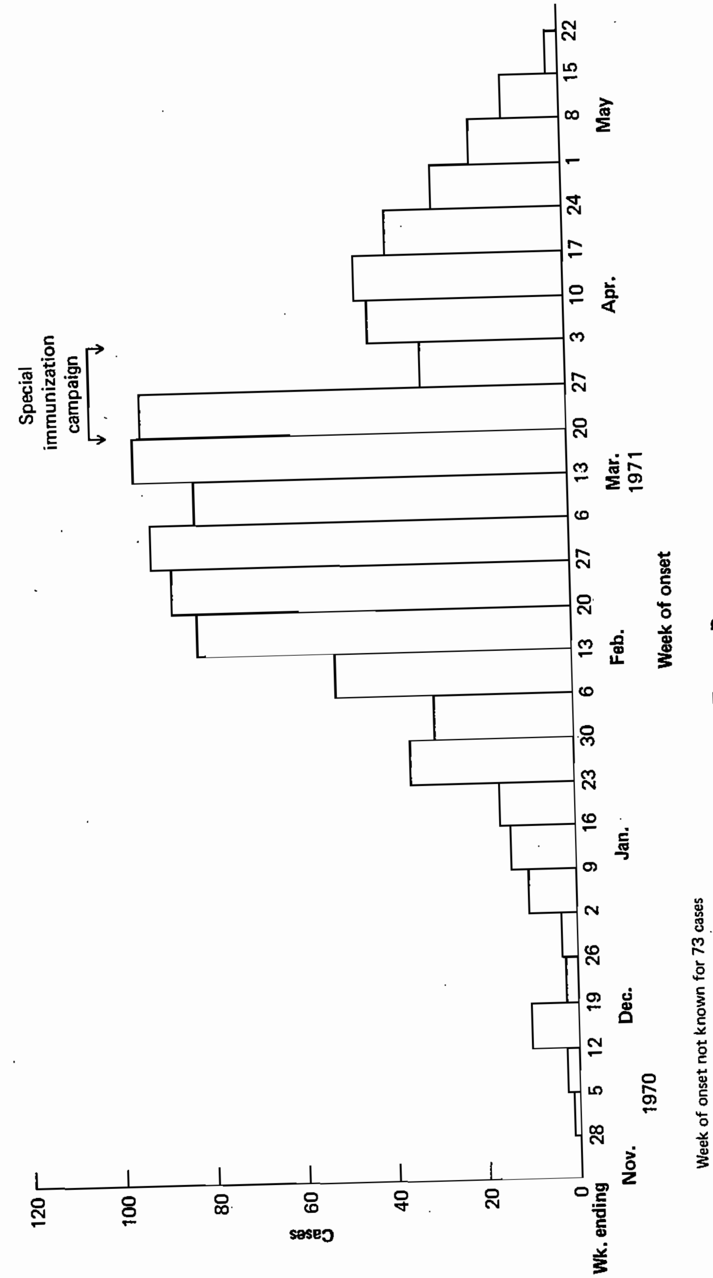


Figure 5-7 Measles cases, by week of onset. Dallas, Texas, December 1, 1970–May 22, 1971. *(Reproduced, by permission, from Luby et al., 1971.)*

among children in Dallas, Texas from late 1970 almost to the middle of 1971 (Luby et al., 1971). By May 1971 there were 1,071 reported cases. The histogram displaying the epidemic's time sequence is of special interest because it shows an abrupt drop after the apparent peak of the epidemic was reached at the end of March. During the 2-week period in which the abrupt fall in case counts began, a special immunization campaign for children was carried out. Although alternative explanations should be considered, it appears that the campaign was helpful in controlling the epidemic by sharply reducing the number of susceptible individuals in the population.

The observed time pattern of an epidemic may provide a strong indication of the mode of initiation and spread. Figs. 5-8 and 5-9 show two different outbreaks of the same disease, infectious hepatitis. Fig. 5-8 depicts the number of cases, by *week* of onset, in an epidemic occurring in Barren County, Kentucky, lasting from June 1970 through April 1971. Fig. 5-9 is drawn on a different time scale and shows the number of cases, by *day* of onset, in an epidemic that occurred in Orange County, California, between August 21, 1971 and September 13, 1971.

The essential distinction between the two epidemics is their duration, particularly in relation to the known incubation period of this disease, which ranges from 15 to 50 days and is commonly about 25 days.

Although the communicable period for this disease has not been clearly defined, the clustering of the Orange County cases within such a narrow time interval—the great bulk appearing within 9 days and all within 24 days—suggested that the outbreak resulted from a "point source," that is, a single common exposure to the virus. With an incubation period measured in weeks, person-to-person spread among the group of cases could not have been a significant factor in an epidemic that ended so soon after it started.

In contrast, the bulk of the Barren County cases occurred over an interval of 4 months, and the total epidemic lasted for 10 months, so there was ample time for direct person-to-person spread. This mode of transmission, however, would not be the only possible mechanism consistent with a hepatitis epidemic of this duration. Prolonged exposure of a population to a contaminated food or water supply could also result in a long-lasting epidemic. However, no such mechanism could be incriminated in Barren County.

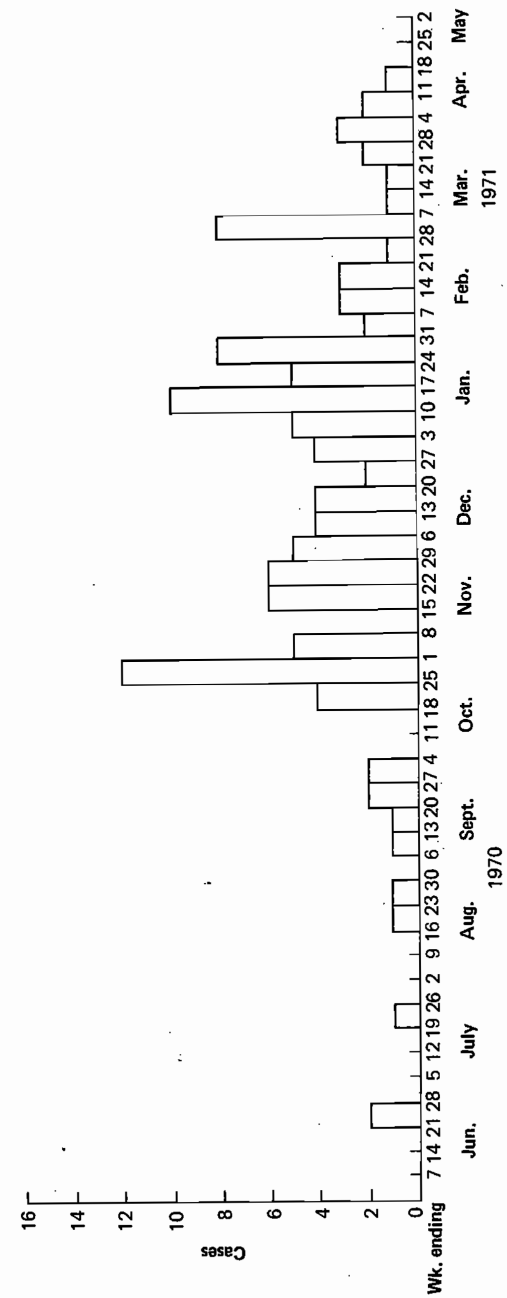


Figure 5-8 Infectious hepatitis cases, by week of onset, Barren County, Kentucky, June, 1970–April, 1971. *(Reproduced, by permission, from Carman et al., 1971.)*

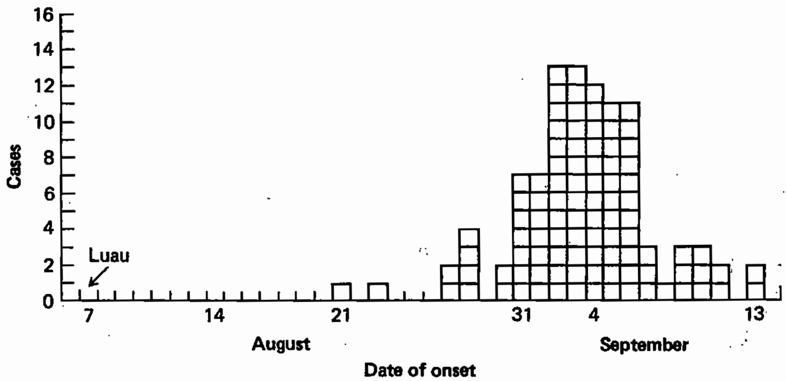


Figure 5-9 Cases of infectious hepatitis in individuals attending luau, by day of onset, Orange County, California, 1971. (Date of onset for one case undetermined.) *(Reproduced, by permission, from Philp et al., 1972).*

Further investigation of each epidemic revealed quite interesting information. In Barren County, most of the 118 cases occurred in children attending two elementary schools. The first observed case in June occurred in a boy whose parents frequently baby-sat for other children in the area. Among the children exposed to his parents was a seven-year-old boy who was the first hepatitis case at one of the schools. The seven-year-old's illness began on September 26. All but one of the cases involving children in that school and their families could be traced directly or indirectly to contact with the seven-year-old. Similarly, at the other school the first illness occurred in a nine-year-old girl on September 21. It was not determined how she became infected but she spread the disease to a total of 34 persons via 5 children with whom she had contact (Carman et al., 1971).

In Orange County almost all the 99 cases were members of a private sailing club. The only time all the cases were together during the prior year was at a club luau at a remote island off the California coast on August 7. One clue to the vehicle of infection was found in the age distribution of incidence. The attack rate was 6 percent in persons under age 15 and was 62 percent in persons age 15 and over.

Detailed analysis of suspect foods revealed only one item regarding which *all* persons *not* consuming it remained free of the disease. This was a mai-tai punch. Sizable proportions of persons *not* eating each of the other foods did become ill, which tends to exonerate these other food items. The description of the food-handling situation at the luau by Philp and his associates (1972) clearly shows how the mai-tai could have served as the vehicle for the fecal-oral transmission of the hepatitis virus.

> All food and beverages were brought to the island by boat. Commercial bottled water for drinking was imported. Little attention, however, was paid to food-handling practices. The only water for handwashing was a single water tap located 85 yards beyond the luau site and 35 yards beyond the two privies available to the group. Handwashing was a rare event. Foods were cooked in an earthen pit for 6 hours to the point of disintegration. Unwashed papaya, pineapple, and oranges were peeled and cut for a fruit mix. Mai-tai punch was prepared from the cut fruits, unwashed fresh strawberries, instant tea, canned pineapple juice, canned lemon-lime drink, bottled water, club soda, and rum. All were mixed in a new plastic garbage can. The punch was served from a 3-tiered fountain made with fiberglass bowls suspended over the garbage can reservoir. Punch was pumped from the can to the top of the fountain, where it flowed over the fiberglass tiers back down into the garbage can. Persons who drank punch filled their cups under one of the streams as it cascaded from one bowl to the next. Many reported punch running over their hands and back into the punch reservoir. Due to the presence of pieces of fruit, particularly strawberries, the pump which forced the punch to the top of the fountain plugged frequently and was unplugged by one or more persons reaching into the bottom of the garbage can and pulling fruit pieces out of the pump. Persons who unclogged the pump also reported sand at the bottom of the can.

Recurrent or Periodic Time Trends

The incidence of certain diseases shows regular recurring increases and decreases. This regular pattern may exhibit cycles which last several years. Many cycles occur annually and represent seasonal variation in disease occurrence. Seasonal variation is a well-known characteristic for many infectious diseases and is usually based on characteristics of the infectious agent itself, the life pattern of its vector or other animal hosts, or changes in the likelihood of person-to-person spread. For example, waterborne gastrointestinal infections often exhibit a peak occurrence in the later summer months when recreational swimming and other factors facilitate their transmission. On the other hand upper respiratory infections frequently show a seasonal rise in or near winter, aided by the concentration of people indoors where virus-containing airborne droplets are readily exchanged.

Shorter-term periodic variations have also been observed. For example, death rates from automobile accidents show weekly cycles with the highest rates occurring on weekends, particularly Saturdays. To date there are no available statistics on the number of passenger-miles driven on each day of the week. Thus it is not possible to state whether the weekend increase in deaths is due merely to an increased exposure of the population to the moving automobile or whether the risk of death per passenger-mile actually increases, possibly due to such factors as more reckless driving or more alcohol consumption on weekends.

Long-term or Secular Trends

Some diseases exhibit a progressive increase or decrease in occurrence that is manifested over years or decades. These long-term time trends are often referred to as *secular* trends.

Figure 5-10 shows the mortality rates in United States males of several leading types of cancer, from 1930 to 1968. A marked secular increase in mortality from lung cancer has occurred, representing about a fifteenfold increase in 1967 as compared to 1930. This increase is believed to be due largely to an increase in the proportion of men who smoked cigarettes. Fig. 5-10 also shows a marked downward trend in stomach cancer mortality. This improvement has not been explained but remains gratifying, nonetheless.

As has been discussed, when a marked increase in incidence occurs in a short period of time, it is quite apparent to the medical community and is referred to as an epidemic, an emotionally loaded

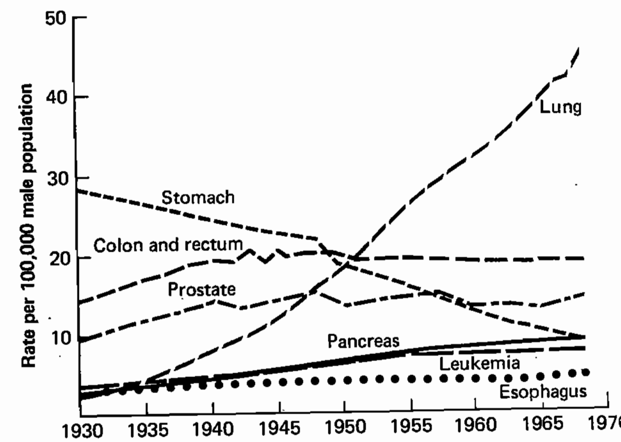


Figure 5-10 Age-adjusted male cancer death rates, by site, United States, 1930–1968. *(Reproduced, by permission, from Silverberg and Holleb, 1972. Data from the U.S. National Vital Statistics Division and Bureau of the Census.)*

term that spurs prompt action. Long-term trends are barely perceptible and might go unnoticed were it not for the study of vital statistics. It would perhaps be well to label as "epidemics," the long-term increases such as that noted for lung cancer. If this term were applied, more action might be taken to investigate the causes and to institute control measures.

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Chapter 6

Prevalence Studies

In going beyond descriptive observations to delve more deeply into disease etiology, there are, as defined in Chap. 4, three basic types of observational investigations:

1. Prevalence or cross-sectional studies
2. Case-control studies
3. Incidence or cohort studies

These will be discussed in greater detail here and in the next two chapters. As will be seen, prevalence studies are, conceptually, quite straightforward, and provide a good basis for subsequent consideration and comparison of the other two study types.

How Prevalence Studies Are Carried Out

Initial Steps The question(s) for study must be clearly defined in terms of the relationship between some possible predisposing factor(s) and the disease under investigation. Then a suitable study