

and equipped inspectors and laboratory support both in the private and public sector are needed.

The dynamic nature of the food trade and the increasing national awareness of actual and potential problems, uncovered as countries improve their surveillance systems, serve to highlight the need for comprehensive food safety legislation. Such legislation should be cap-

able of rapid modification to meet new trade activities, or newly recognized hazards in existing operations.

(Source: Caribbean Epidemiological Center, Epidemiology Unit, Health Programs Development, PAHO.)

Infectious and Chronic Disease Epidemiology: Separate and Unequal?

Definitions

As applied to disease or illness, the word "chronic" means slow progression and long duration. It is the opposite of "acute," a term which implies a swift onset and short course. Despite the simplicity of definition, no one has satisfactorily classified all diseases on the basis of duration. Indeed, most diseases on any list are sometimes acute and sometimes chronic. A cerebrovascular accident may be immediately fatal or produce sequelae which persist for months or years. Heart disease, usually classified as chronic, is acute for those myocardial infarct victims who die before reaching the hospital. The tendency to consider infection as synonymous with acute is equally misleading. Many infections or their sequelae are chronic: sinusitis, cystitis, syphilis, tuberculosis, paralytic poliomyelitis, congenital rubella, and rheumatic heart disease, to name a few.

Acuteness or chronicity are often not permanent attributes of a disease. An acute disease may be redefined when scientific advances permit identification of the preclinical phase. A chronic condition may be transformed into an acute illness when early treatment aborts sequelae. In the Baltimore study of chronic diseases (1), one in 10 "substantial conditions" would have had complete recovery with appropriate care.

Latency

A long interval between exposure to the putative risk factor(s) and disease onset is believed to characterize most chronic illnesses. But many infections appear after latent periods as long as those proposed for chronic diseases. Thus, infection with a tubercle bacil-

lus acquired in childhood is often first manifest in late adult life. Herpes zoster represents reactivation of childhood chickenpox in many, if not all, cases. A large proportion of infections in the compromised host undoubtedly reflects activation of dormant infection. Indeed, the incubation period for the majority of infections afflicting adults today is either delayed or poorly defined.

Transmissibility

Many infectious diseases are propagated from person to person. However, this is by no means true of all infectious agents: blood poisoning caused by preformed toxins, Legionnaires' disease, and coccidioidomycosis are not transmitted from person to person. Some chronic diseases of as yet unknown etiology may turn out to be transmissible. Clusters of leukemia and lymphoma suggest a transmissible agent as do the recent studies of residents of households in which victims of multiple sclerosis reside (2). It would be premature to divide epidemiologists into those who deal with transmissible or nontransmissible conditions. If leukemia, cervical cancer, multiple sclerosis, arthritis, and diabetes prove to be caused by a transmissible agent—as many now suspect—persons now classified as chronic disease experts may find themselves to be infectious disease epidemiologists.

Etiology

At the turn of the century, infectious disease was the major area of research in medicine. The discoveries of specific agents which produced specific diseases were straightforward and satisfying, and led to one of the

basic tenets of medicine: a single disease process has a single causation. Clinical observation, bacteriologic investigation, and the development of the antimicrobials in the early 1940s led to the preeminence of infectious disease as a medical problem whose etiology and management were established. In contrast, chronic disease epidemiology has attended the study of diseases of unknown cause, conditions which are increasingly recognized as multifactorial in origin. Thus, the dichotomy became cause-known/unifactorial vs. cause-unknown/multifactorial.

Although it is true that the necessary cause of most acute diseases is a known agent, and the necessary cause of most chronic diseases remains unknown, this is surely more a function of the state of the art than the nature of disease. All diseases have multiple causes. The necessary microbial agent is not the sole determinant of outcome. As Stewart (3) has written, "If two susceptible subjects are exposed to equal doses of the same germ, and one develops infection while the other does not, the factor governing the development of the infection clearly lies outside the germ."

For most diseases, the frequency of exposure exceeds the frequency of illness. Only the availability of the necessary agent has provided the reagents required to demonstrate that most of those infected with the tubercle bacillus or the poliomyelitis virus are not sick. We are just at the threshold of understanding why most of those who smoke cigarettes do not develop lung cancer (4). Heritable and environmental determinants of chronic diseases may well precede comparable discoveries in the arena of infection.

Behavioral Considerations

Evidence accumulated in the United States during the last 20 years indicates that the most important chronic diseases are caused by a variety of personal and social habits, such as improper diet, excessive drinking and smoking, lack of exercise, and unsafe driving and working practices. Behavioral considerations also determine the distribution of many infectious diseases. For example, venereal disease, the most important epidemic infection in the United States today, does not occur among the chaste, and active tuberculosis is disproportionately frequent among those who abuse alcohol.

In neither acute, infectious, nor chronic disease is a complete understanding of cause required for prevention. Smallpox was prevented before isolation of the virus; lung cancer can be prevented before identification of the specific carcinogen in cigarette smoke. When an infectious disease is transmitted or maintained because of attitudes, behavior, or surroundings, a purely germ-oriented approach is unlikely to provide effective control.

Study Design

No study design is unique to any branch of epidemiology. The epidemiological study of both acute and chronic conditions usually requires a denominator and/or a comparison group, can be done retrospectively or prospectively, and can examine prevalence or incidence. The search for causality in a food poisoning outbreak, examining the attack rates of those with and without exposure to the suspect food, applies the same principles as those used in a comparison of the incidence of uterine cancer among those with and without the suspect hormone. Cross-sectional or case-control comparisons are used to validate or refute clinical tenets of acute and chronic disease. Such studies led to the delayed recognition that most of the symptoms attributed to pinworm are equally frequent in uninfected children (5), that splinter hemorrhages traditionally attributed to bacterial endocarditis are equally common in hospitalized patients without endocarditis (6), and that the symptoms attributed to gallbladder disease are equally prevalent in women without gallbladder disease (7). The same principles of study design that apply to clinical trials of vaccine or prophylactic antimicrobials apply to the study of lipid lowering drugs or anti-hypertensive agents.

A major tool of the chronic disease epidemiologist has been the population survey, a prototype of which has been the Framingham Study (8). In community-based studies, entire populations of persons, including a majority who are presumably well, are examined for a variety of characteristics and diseases. Cross-sectional studies define the usual, if not normal, and prospective studies define putative risk factors. Observations such as those made in Framingham led to the recognition that blood pressure and plasma cholesterol were important predictors of coronary artery disease.

In the past, infectious disease epidemiologists worked from the vantage of sick persons. Epidemics were described in terms of the ill, and the well population was used primarily for age- and sex-specific denominator data. But community-based studies of the distribution of disease and its precursors are by no means the purview of chronic disease epidemiologists alone. The Seattle Virus Watch Study (9), which has added important information to our knowledge of the transmission and frequency of respiratory infections, is a case in point.

Analytic Methodology

One phenomenon which perhaps best distinguished the chronic from the infectious disease epidemiologist is the use of more sophisticated mathematical methods feasible with computer-assisted analysis. Because neither the etiology of chronic disease nor its manage-

ment was as simple or obvious as the situation which appeared to exist in infectious disease, progressively sophisticated mathematics were developed by epidemiologists and biostatisticians, at a time when most research in the field of infectious disease involved clinical observations or experiments conducted in the laboratory. The danger is that goodness of fit sometimes substitutes for common sense or biologic plausibility (10). Chronic disease epidemiologists are often in the awkward position of analysis without hypothesis; in the absence of either an agent or a unique outcome, they must perform hypothesis-seeking exercises. As good statisticians and epidemiologists know, the pitfalls of data dredging greatly exceed those of hypothesis testing. The multiple possible analyses render almost a certainty that some variables will be significantly associated with some diseases.

In the days before linear regression and multiple logistic function, many infectious disease epidemiologists personally gathered and manually tabulated their data. This experience clarified the sometimes remarkable limitations of data—which by virtue of categorization and computerization may gain unwarranted credibility. Experience gained in the shorter time-frame of some infectious processes also provide valuable insights about the hazards of early assumption. Farr (11) demonstrated a remarkable correlation of cholera mortality and altitude in 19th century London but failed to consider water as the variable of interest. A recent report (12) of an excess of hepatitis among young women using oral contraceptives would have profited by a consideration of the probable differences in lifestyle among women who chose oral contraceptives as compared to those without such contraceptive practices.

Many infectious disease epidemiologists come from the ranks of clinicians and laboratorians, and lack the skills traditionally considered in the purview of the chronic disease epidemiologists. These skills are now essential to the discovery of those variables which, in the presence of the necessary agent, determine infection, disease, and outcome. Whereas the infectious agent can usually be isolated and enumerated with precision, the extraneous factors which determine morbidity and mortality are more difficult to quantify. It is the task of epidemiology to find other methods to assess with precision the contribution of these factors to infectious disease. The arbitrary separation of infectious disease from chronic disease epidemiology in teaching and research does disservice to this need.

Conclusion

Some scientific disciplines are best able to answer certain questions in medicine. Much of modern epidemiological effort has been directed toward investigating problems regarding which the rest of science has

few useful leads. Any disease, acute or chronic, which lacks either a logical structure or a plausible hypothesis is difficult to study. But the identification of a necessary agent, microbial or otherwise, does not answer all relevant and important questions any more than demonstration of an associated variable confirms causality or predicts prevention.

Epidemiologically, acute diseases differ from chronic diseases in two major aspects: immediacy of response and uniqueness of observation. The lessons learned in infectious disease, where the agent and outcome were more readily available to test predictions, must be shared with those epidemiologists who—in their haste to assign causality—sometimes abandon biologic wisdom in favor of quantitative ideology. Many of the unanswered questions in acute/infectious disease epidemiology need to be addressed by those techniques currently attributed to and taught with chronic disease epidemiology. Acute and chronic disease epidemiologists have important lessons to offer each other. A sharing of experience and methodologies could avert the unfortunate plethora of truly terrible data analyzed ad nauseam, or good data poorly interpreted. Once these lessons have been learned, we should discard the qualifiers and call an epidemiologist an epidemiologist. Acute and chronic disease epidemiologists are not separate and unrelated species, any more than acute and chronic diseases can be neatly categorized.

(Source: Reprinted from Elizabeth Barrett-Connor, "Infectious and Chronic Disease Epidemiology: Separate and Unequal?". *Am J Epidemiol* 109(3):245-249, 1979.)

References

- (1) Commission on Chronic Illness. Chronic illness in a large city—The Baltimore study. In *Chronic Illness in the United States*. Vol. IV. Cambridge, Harvard University Press, 1957.
- (2) Schocket, A. L. and H. L. Weiner. Lymphocytotoxic antibodies in family members of patients with multiple sclerosis. *Lancet* 1:571-573, 1978.
- (3) Stewart, G. T. Limitations of the germ theory. *Lancet* 1:1977-2081, 1968.
- (4) Emery, A. E. H., R. Anand, N. Danford, et al. Arylhydrocarbonhydroxylase inducibility in patients with cancer. *Lancet* 1:470-471, 1978.
- (5) Weller, T. H. and C. W. Sorensen. Enterobiasis: its incidence and symptomatology in a group of 505 children. *N Engl J Med* 224:143-146, 1941.

(6) Kilpatrick, Z. M., P. A. Greenber, and J. P. Sanford. Splinter hemorrhages—their clinical significance. *Arch Intern Med* 115:730-735, 1965.

(7) Price, W. H. Gallbladder dyspepsia. *Br Med J* 2:138-141, 1963.

(8) Dawber, T. R., W. B. Kann and L. P. Lyell. An approach to longitudinal studies in a community: the Framingham Study. *Ann NY Acad Sci* 107:539-556, 1963.

(9) Fox, J. P., C. E. Hall, and M. R. Councy. The Seattle Virus Watch. II. Objectives, study population and its observation, data processing and summary of illnesses. *Am J Epidemiol* 96:270-285, 1972.

(10) Feinstein, A. R. *Clinical Biostatistics*. St. Louis, C.V. Mosby, 1977.

(11) Langmuir, A. D. Epidemiology of airborne infection. *Bacteriol Rev* 25:173-181, 1961.

(12) Morrison, A. S., H. K. Jick, and H. W. Ory. Oral contraceptives and hepatitis. A report from the Boston Colla-

borative Drug Surveillance Program, Boston University Medical Center. *Lancet* 1:1142-1143, 1977.

Editorial Comment

Dr. Barrett-Connor's article has been summarized for the *Epidemiological Bulletin* because it makes a substantial contribution to the application of epidemiology in the field of disease prevention and control. It addresses a subject of current controversy and discussion in many Latin American and Caribbean countries, and is important for the organization of services, teaching of epidemiology, and research in the countries of the Region.

National Registry of Tumor Pathology in Brazil

During the VI International Cancer Congress, held in São Paulo, in 1954, a discussion was held on the need for an international coding system for neoplasms. As a result, the Nomenclature and Statistics Committee of the International Union against Cancer accepted the *Manual of Tumor Nomenclature and Coding* (MOTNAC), published by the American Cancer Society in 1951, as the basis for an international coding system. MOTNAC was revised in 1968. In 1976 it was succeeded by the *International Classification of Diseases for Oncology* (ICD-O), published by the World Health Organization.

Recognizing the importance of a uniform coding system for neoplasms, the Pan American Health Organization published a Portuguese language version of MOTNAC in 1972 and a Spanish version in 1974. These translations were widely distributed in Latin America. Spanish and Portuguese language versions of ICD-O were published by PAHO in 1977 and 1978, respectively.

Because of a lack of national statistics on cancer, the existence of only a small number of tumor registries (hospital and population-based), and the need for data on the incidence of cancer in Brazil, the National Division of Chronic Degenerative Diseases in 1975 developed a program for oncological coding.

In 1975 and 1976, 50 courses were held in 49 cities,

describing the methodology and value of the National Registry of Pathology. These courses were attended by 2,912 participants: 283 pathologists, 516 physicians from other specialities, 1,200 medical students, and 913 paramedics. Subsequently, didactic kits for coding diagnostic information were provided to the 109 laboratories which participated in the initial phases of the program. The results of this initial phase, with all the histopathological data collected in 1975, were published in the "Registro Nacional de Tumores" (Brazilian Ministry of Health, 1978).

The success of this endeavor by the National Division of Chronic Degenerative Diseases and the interest of pathologists in the program stimulated continued work. As a result, the registry expanded into a national program designed to obtain information on the incidence of cancer throughout the entire country; this program is known as the National Registry of Tumor Pathology (NRTP).

In 1978 an Agreement was signed by the Ministry of Health and PAHO's Latin American Center for Health Sciences Information (BIREME) in São Paulo, for the development of a computerized registry which facilitates storage and rapid analysis of large amounts of data.

As the coordinating and executive center, in 1978 BIREME contacted a large number of laboratories, in