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Sick Individuals and Sick Populations

The Determinants of Individual Cases

In teaching epidemiology to medical students, I have often encouraged them to consider a question which I first heard enunciated by Roy Acheson: "Why did *this* patient get *this* disease at *this* time?" It is an excellent starting point, because students and doctors feel a natural concern for the problems of the individual. Indeed, the central ethos of medicine is seen as an acceptance of responsibility for sick individuals.

It is an integral part of good doctoring to ask not only, "What is the diagnosis, and what is the treatment?" but also, "Why did this happen, and could it have been prevented?" Such thinking shapes the approach to nearly all clinical and laboratory research into the causes and mechanisms of illness. Hypertension research, for example, is almost wholly preoccupied with the characteristics which distinguish individuals at the hypertensive and normotensive ends of the blood pressure distribution. Research into diabetes looks for genetic, nutritional, and metabolic reasons to explain why some people get diabetes and others do not. The constant aim in such work is to answer Acheson's question, "Why did *this* patient get this disease at this time?"

The same concern has continued to shape the thinking of all of us who came to epidemiology from a background in clinical practice. The whole basis of the case control method is to discover how sick and healthy individuals differ. Equally, the basis of many cohort studies is the search for "risk factors," which identify certain individuals as being more susceptible to disease; and from this we proceed to test whether these risk factors are also causes, capable of explaining why some individuals get sick while others remain healthy, and applicable as a guide to prevention.

To confine attention in this way to within-population comparisons has caused much confusion (particularly in the clinical world) in the definition of normality. Laboratory "ranges of normal" are based on what is common within the local population. Individuals with "normal blood pressure" are those who do not stand out from their local contemporaries, and so on. What is common is all right, we presume.

Applied to etiology, the individual-centered approach leads to the use of relative risk as the basic

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representation of etiologic force, that is, "the risk in exposed individuals relative to risk in nonexposed individuals." Indeed, the concept of relative risk has almost excluded any other approach to quantifying causal importance. It may generally be the best measure of etiologic force, but it is no measure at all of etiologic outcome or of public health importance.

Unfortunately, this approach to the search for causes, and the measuring of their potency, has to assume a heterogeneity of exposure within the study population. If everyone smoked 20 cigarettes a day, then clinical, case control, and cohort studies alike would lead us to conclude that lung cancer was a genetic disease, and in one sense that would be true, since if everyone is exposed to the necessary agent, then the distribution of cases is wholly determined by individual susceptibility.

Within Scotland and other mountainous parts of Britain (Figure 1, left section) *(1)*, there is no discernible relation between local cardiovascular death rates and the softness of the public water supply. The reason is apparent if one extends the inquiry to the whole of the U.K. In Scotland, everyone's water is soft, and the possibly adverse effect becomes recognizable only when study is extended to other regions that have a much wider range of exposure ($r = -0.67$). Even more clearly, a case control study of this question within Scotland would have been futile. Everyone is exposed, and other factors operate to determine the varying risk.

Epidemiology is often defined in terms of study of the determinants of the distribution of the disease, but we should not forget that the more widespread is a particular cause, the less it explains the distribution of cases. The hardest cause to identify is the one that is universally present, for then it has no influence on the distribution of disease.

The Determinants of Population Incidence Rate

I find it increasingly helpful to distinguish two kinds of etiologic question. The first seeks the causes of cases and the second seeks the causes of incidence. "Why do some individuals have hypertension?" is a quite different question from "Why do some populations have much hypertension, whilst in others it is rare?" The questions require different kinds of study and they have different answers.

Figure 2 shows the systolic blood pressure distributions of middle-aged men in two populations—Kenyan nomads *(2)* and London civil servants *(3)*. The familiar question, "Why do some individuals have higher blood pressure than others?" could be equally well asked in either of these settings, since in each the individual blood pressures vary (proportionately) to about the same extent, and the answers might well be much the

Figure 1. Relation between water quality and cardiovascular mortality in towns of the U.K. *(1)*.

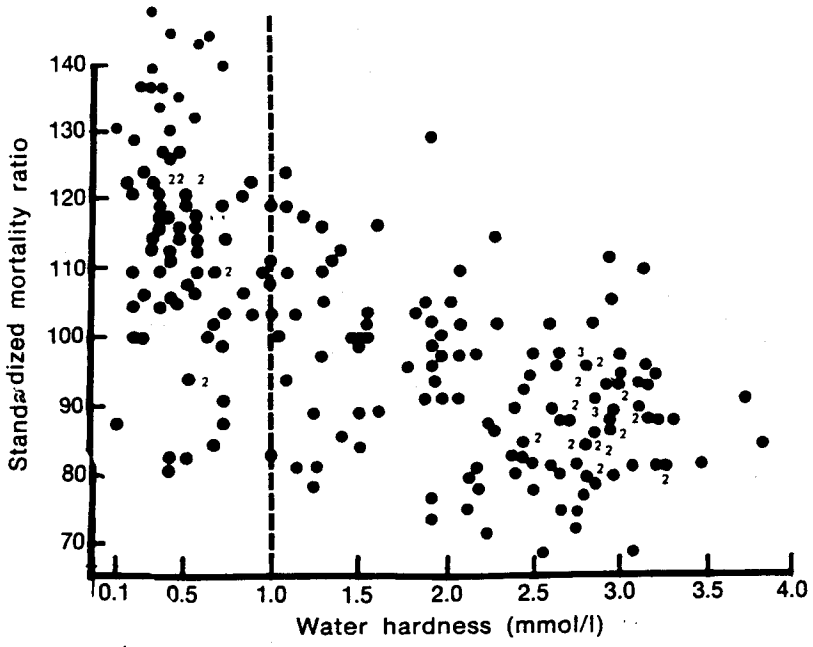
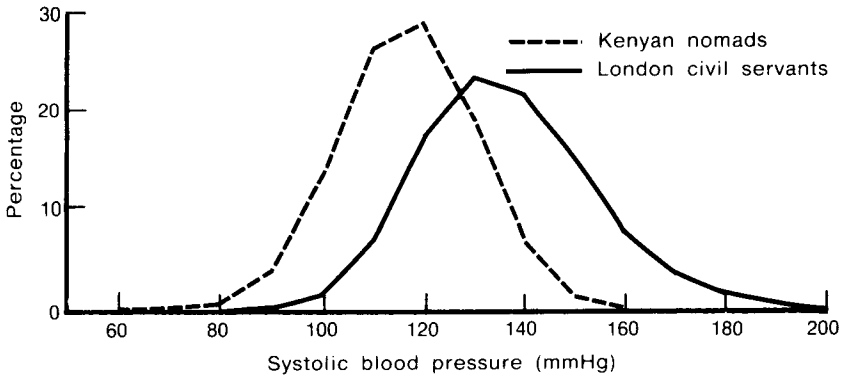


Figure 2. Distributions of systolic blood pressure in middle-aged men in two populations *(2,3)*.



same in each instance (that is, mainly genetic variation, with a lesser component from environmental and behavioral differences). We might achieve a complete understanding of why individuals vary, and yet quite miss the most important public health question, namely, "Why is hypertension absent in the Kenyans and common in London?" The answer to that question has to do with the determinants of the population mean, for what distinguishes the two groups has nothing to do with the characteristics of individuals; it is rather a shift of the whole distribution—a mass influence acting on the population as a whole. To find the determinants of prevalence and incidence rates, we need to study characteristics of populations, not characteristics of individuals.

A more extreme example is provided by the population distributions of serum cholesterol levels *(4)* in East Finland, where coronary heart disease is very common, and Japan, where the incidence rate is low; the two distributions barely overlap. Each country has men with relative hypercholesterolemia (although their definitions of the range of "normal" would no doubt disagree), and one could research into the genetic and other causes of these unusual individuals, but if we want to discover why Finland has such a high incidence of coronary heart disease, we need to look for those characteristics of the national diet which have so elevated the whole cholesterol distribution. Within populations it has proved almost impossible to demonstrate any relation between an individual's diet and his serum cholesterol level, and the same applies to the relation of individual diet to blood pressure and to overweight. But at the level of populations it is a different story; it has proved easy to show strong associations between population mean values for saturated fat intake *versus* serum cholesterol level and coronary heart disease incidence, sodium intake *versus* blood pressure, or energy intake *versus* overweight. The determinants of incidence are not necessarily the same as the causes of cases.

How Do the Causes of Cases Relate to the Causes of Incidence?

This is largely a matter of whether exposure varies similarly within a population and between populations (or over a period of time within the same population). Softness of water supply may be a determinant of cardiovascular mortality, but it is unlikely to be identifiable as a risk factor for individuals, because exposure tends to be locally uniform. Dietary fat is, I believe, the main determinant of a population's incidence rate for coronary heart disease, but it quite fails to identify high-risk individuals.

In the case of cigarettes and lung cancer, it so happened that the study populations contained about equal numbers of smokers and nonsmokers, and in such a situation case control and cohort studies were able to identify what was also the main determinant of population differences and time trends.

There is a broad tendency for genetic factors to dominate individual susceptibility, but to explain rather little of population differences in incidence. Genetic heterogeneity, it seems, is mostly much greater within than between populations. This is the contrary situation to that seen for environmental factors. Thus migrants, whatever the color of their skin, tend to acquire the disease rates of their country of adoption.

Most noninfectious diseases are still of largely un-

known cause. If you take a textbook of medicine and look at the list of contents you will still find, despite all our etiologic research, that most are still of basically unknown etiology. We know quite a lot about the personal characteristics of individuals who are susceptible to them, but for a remarkably large number of our major noninfectious diseases, we still do not know the determinants of the incidence rate.

Over a period of time we find that most diseases are in a state of flux. For example, duodenal ulcer in Britain at the turn of the century was an uncommon condition affecting mainly young women. During the first half of the century, the incidence rate rose steadily and it became very common, but now the disease seems to be disappearing, and yet we have no clues to the determinants of these striking changes in incidence rates. One could repeat that story for many conditions.

There is hardly a disease whose incidence rate does not vary widely, either over time or between populations at the same time. This means that these causes of incidence rate, unknown though they are, are not inevitable. It is possible to live without them, and if we knew what they were, it might be possible to control them. But to identify the causal agent by the traditional case control and cohort methods will be unsuccessful if there are not sufficient differences in exposure within the study population at the time of the study. In those circumstances all that these traditional methods do is to find markers of individual susceptibility. The clues must be sought from differences between populations or from changes within populations over time.

Prevention

These two approaches to etiology—the individual and the population-based—have their counterparts in prevention. In the first, preventive strategy seeks to identify high-risk susceptible individuals and to offer them some individual protection. In contrast, the "population strategy" seeks to control the determinants of incidence in the population as a whole.

The "High-Risk" Strategy

This is the traditional and natural medical approach to prevention. If a doctor accepts that he is responsible for an individual who is sick today, then it is a short step to accept responsibility also for the individual who may well be sick tomorrow. Thus, screening is used to detect certain individuals who hitherto thought they were well but who must now understand that they are in effect patients. This is the process, for example, in the detection and treatment of symptomless hypertension, the transition from healthy subject to patient being ratified by the giving and receiving of tablets. (Anyone who takes medicines is by definition a patient.)

What the "high-risk" strategy seeks to achieve is something like a truncation of the risk distribution. This general concept applies to all special preventive action in high-risk individuals—in at-risk pregnancies, in small babies, or in any other particularly susceptible group. It is a strategy with some clear and important advantages (Table 1).

Its first advantage is that it leads to intervention that is appropriate to the individual. A smoker who has a cough or who is found to have impaired ventilatory function has a special reason for stopping smoking. The doctor will see it as making sense to advise salt restriction in a hypertensive. In such instances the intervention makes sense because that individual already has a problem which that particular measure may possibly ameliorate. If we consider screening a population to discover those with high serum cholesterol levels and advising them on dietary change, then that intervention is appropriate to those people in particular; they have a diet-related metabolic problem.

The "high-risk" strategy produces interventions that are appropriate to the particular individuals advised to take them. Consequently, it has the advantage of enhanced subject motivation. In our randomized controlled trial of smoking cessation in London civil servants, we first screened some 20,000 men and from them selected about 1,500 who were smokers with, in addition, markers of specially high risk for cardiorespiratory disease. They were recalled and a random half received antismoking counseling. The results, in terms of smoking cessation, were excellent because those men knew they had a special reason to stop. They had been picked out from others in their offices

Table 1. Prevention by the "high-risk strategy": advantages.

1. Intervention appropriate to individual
2. Subject motivation
3. Physician motivation
4. Cost-effective use of resources
5. Benefit-risk ratio favorable

because, although everyone knows that smoking is a bad thing, they had a special reason why it was particularly unwise for them.

There is, of course, another and less reputable reason why screening enhances subject motivation, and that is the mystique of a scientific investigation. A ventilatory function test is a powerful enhancer of motivation to stop smoking; an instrument which the subject does not quite understand, that looks rather impressive, has produced evidence that he is a special person with a special problem. The electrocardiogram is an even more powerful motivator, if you are unscrupulous enough to use it in prevention. A man may feel entirely well, but if those little squiggles on the paper tell the doctor that he has got trouble, then he must accept that he has now become a patient. That is a powerful persuader. (I suspect it is also a powerful cause of lying awake in the night and thinking about it.)

For rather similar reasons, the "high-risk" approach also motivates physicians. Doctors, quite rightly, are uncomfortable about intervening in a situation where their help was not asked for. Before imposing advice on somebody who was getting on all right without them, they like to feel that there is a proper and special justification in that particular case.

The "high-risk" approach offers a more cost-effective use of limited resources. One of the things we have learned in health education at the individual level is that once-only advice is a waste of time. To get results we may need a considerable investment of counseling time and follow-up. It is costly in use of time, and effort, and resources, and therefore it is more effective to concentrate limited medical services and time where the need—and therefore also the benefit—is likely to be greatest.

A final advantage of the "high-risk" approach is that it offers a more favorable ratio of benefits to risks. If intervention must carry some adverse effects or costs, and if the risk and cost are much the same for everybody, then the ratio of the costs to the benefits will be more favorable where the benefits are larger.

Unfortunately, the "high-risk" strategy of prevention also has some serious disadvantages and limitations (Table 2).

The first centers around the difficulties and costs of screening. Supposing that we were to embark, as some had advocated, on a policy of screening for high cholesterol levels and giving dietary advice to those individuals at special risk. The disease process we are trying to prevent (atherosclerosis and its complications) begins early in life, so we should have to initiate screening perhaps at the age of ten. However, the abnormality we seek to detect is not a stable lifetime characteristic, so we must advocate repeated screening at suitable intervals.

In all screening one meets problems with uptake, and the tendency for the response to be greater amongst those sections of the population who are often least at risk of the disease. Often there is an even greater problem; screening detects certain individuals who will receive special advice, but at the same time it cannot help also discovering much larger numbers of "borderliners," that is, people whose results mark them as at increased risk but for whom we do not have an appropriate treatment to reduce their risk.

The second disadvantage of the "high-risk" strategy is that it is palliative and temporary, not radical. It does not seek to alter the underlying causes of the disease but to identify individuals who are particularly susceptible to those causes. Presumably in every generation there will be such susceptibles, and if prevention and control efforts were confined to these high-risk individuals, then that approach would need to be sustained year after year and generation after generation. It does not deal with the root of the problem, but seeks to protect those who are vulnerable to it, and they will always be around.

The potential for this approach is limited—sometimes more than we could have expected—both for the individual and for the population. There are two reasons for this. The first is that our power to predict future disease is usually very weak. Most individuals with risk factors will remain well, at least for some years; contrariwise, unexpected illness may happen to someone who has just received an "all clear" report from a screening examination. One of the limitations of the relative risk statistic is that it gives no idea of the absolute level of danger. Thus, the Framingham Study has impressed us all with its powerful discrimination between high- and low-risk groups, but when we see (Figure 3) *(5)* the degree of overlap in serum cholesterol level between future cases and those who

Table 2. Prevention by the "high-risk strategy": disadvantages.

1. Difficulties and costs of screening
2. Palliative and temporary—not radical
3. Limited potential for (a) individual
   (b) population
4. Behaviorally inappropriate

remained healthy, it is not surprising that an individual's future is so often misassessed.

Often the best predictor of future major disease is the presence of existing minor disease. A low ventilatory function today is the best predictor of its future rate of decline. A high blood pressure today is the best predictor of its future rate of rise. Early coronary heart disease is better than all the conventional risk factors as a predictor of future fatal disease. However, even if screening includes such tests for early disease, our experience in the Heart Disease Prevention Project (Table 3) *(6)* still points to a very weak ability to predict the future of any particular individual.

This point came home to me only recently. I have long congratulated myself on my low levels of coronary risk factors, and I joked to my friends that if I were to die suddenly, I should be very surprised. I even speculated on what other disease—perhaps colon cancer—would be the commonest cause of death for a man in the lowest group of cardiovascular risk. The painful truth is that for such an individual in a Western population the commonest cause of death—by far—is coronary heart disease! Everyone, in fact, is a high-risk individual for this uniquely mass disease.

There is another, related reason why the predictive basis of the "high-risk" strategy of prevention is weak. It is well illustrated by some data from Alberman *(7)*, which relate the occurrence of Down's syndrome births to maternal age (Table 4). Mothers under 30 years are individually at minimal risk, but because they are so numerous, they generate half the cases. High-risk in-

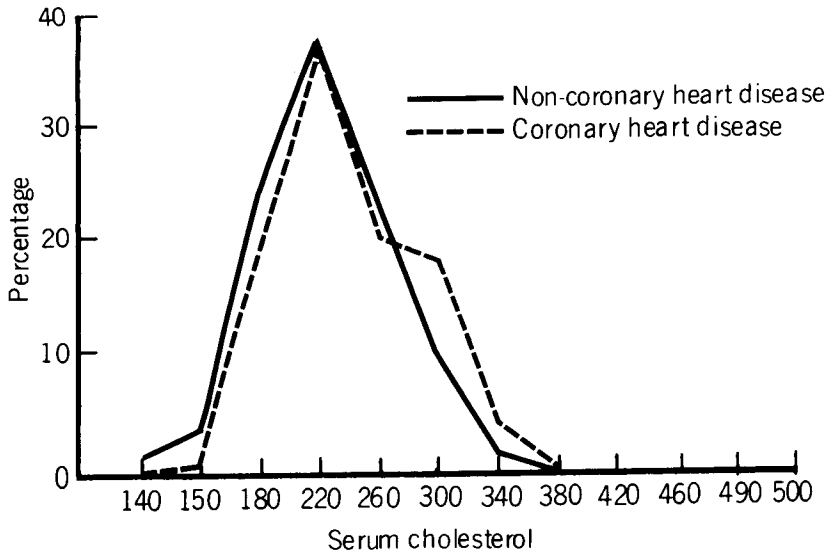
Table 3. Five-year incidence of myocardial infarction [MI] in the U.K. Heart Disease Prevention Project.

Entry characteristic	% of men	% of MI cases	MI incidence rate %
Risk factors alone	15	32	7
"Ischemia"	16	41	11
"Ischemia" + risk factors	2	12	22
All men	100	100	4

Table 4. Incidence of Down's syndrome according to maternal age *(7)*.

Maternal age (years)	Risk of Down's syndrome per 1000 births	Total births in age group (as % of all ages)	% of total Down's syndrome occurring in age group
< 30	0.7	78	51
30-34	1.3	16	20
35-39	3.7	5	16
40-44	13.1	0.95	11
≥ 45	34.6	0.05	2
All ages	1.5	100	100

Figure 3. Percentage distribution of serum cholesterol levels (mg/dl) in men aged 50 to 62 who did or did not subsequently develop coronary heart disease (Framingham Study) *(5)*.



dividuals aged 40 and above generate only 13% of the cases.

The lesson from this example is that *a large number of people at a small risk may give rise to more cases of disease than the small number who are at a high risk*. This situation seems to be common, and it limits the utility of the "high-risk" approach to prevention.

A further disadvantage of the "high-risk" strategy is that it is behaviorally inappropriate. Eating, smoking, exercise, and all our other life-style characteristics are constrained by social norms. If we try to eat differently from our friends, it will not only be inconvenient, but we risk being regarded as cranks or hypochondriacs. If a man's work environment encourages heavy drinking, then advice that he is damaging his liver is unlikely to have any effect. No one who has attempted any sort of health education effort in individuals needs to be told that it is difficult for such people to step out of line with their peers. This is what the "high-risk" preventive strategy requires them to do.

The Population Strategy

This is the attempt to control the determinants of incidence, to lower the mean level of risk factors, to shift the whole distribution of exposure in a favorable direction. In its traditional "public health" form, it has involved mass environmental control methods; in its modern form it is attempting (less successfully) to alter some of society's norms of behavior.

The advantages are powerful (Table 5). The first is that it is radical. It attempts to remove the underlying causes that make the disease common. It has a large potential—often larger than one would have expected—for the population as a whole. From Framingham data one can compute that a 10 mm Hg lowering of the blood pressure distribution as a whole would correspond to about a 30% reduction in the total attributable mortality.

The approach is behaviorally appropriate. If nonsmoking eventually becomes "normal," then it will be much less necessary to keep on persuading individuals. Once a social norm of behavior has become accepted and (as in the case of diet) once the supply industries have adapted themselves to the new pattern, then the maintenance of that situation no longer requires effort from individuals. The health education phase aimed at changing individuals is, we hope, a temporary necessity, pending changes in the norms of what is socially acceptable.

Unfortunately, the population strategy of prevention has also some weighty drawbacks (Table 6). It offers only a small benefit to each individual, since most of them were going to be all right anyway, at least for many years. This leads to the *Prevention Paradox (8)*, "A preventive measure which brings much benefit to the population offers little to each participating individual." This has been the history of public health—of immunization, the wearing of seat belts, and now the attempt to change various life-style characteristics. Of enormous potential importance to the population as a whole, these measures offer very little—particularly in the short term—to each individual, and thus there is poor motivation of the subject. We should not be surprised that health education tends to be relatively ineffective for individuals and in the short term. Mostly people act for substantial and immediate rewards, and the medical motivation for health education is inherently weak. Their health next year is not likely to be much better if they accept our advice or if they reject it. Much more powerful as motivators for health education are the social rewards of enhanced self-esteem and social approval.

There is also in the population approach only poor motivation of physicians. Many medical practitioners who embarked with enthusiasm on antismoking education have become disheartened because their success rate was no more than 5 or 10%; in clinical practice one's expectation of results is higher. Grateful patients are few in preventive medicine, where success is marked by a nonevent. The skills of behavioral advice are different and unfamiliar, and professional esteem is lowered by a lack of skill. Harder to overcome than any of these, however, is the enormous difficulty for

Table 5. Prevention by the "population strategy": advantages.

1. Radical
2. Large potential for population
3. Behaviorally appropriate

Table 6. Prevention by the "population strategy": disadvantages.

1. Small benefit to individual ("Prevention Paradox")
2. Poor motivation of subject
3. Poor motivation of physician
4. Benefit-risk ratio worrisome

medical personnel to see health as a population issue and not merely as a problem for individuals.

In mass prevention each individual has usually only a small expectation of benefit, and this small benefit can easily be outweighed by a small risk *(8)*. This happened in the World Health Organization clofibrate trial *(9)*, where a cholesterol-lowering drug seems to have killed more than it saved, even though the fatal complication rate was only about 1/1,000/year. Such low-order risks, which can be vitally important to the balance sheet of mass preventive plans, may be hard or impossible to detect. This makes it important to distinguish two approaches. The first is the restoration of biological normality by the removal of an abnormal exposure (e.g., stopping smoking, controlling air pollution, moderating some of our recently acquired dietary deviations); here, there can be some presumption of safety. This is not true for the other kind of preventive approach, which leaves intact the underlying causes of incidence and seeks instead to interpose some new, supposedly protective intervention (e.g., immunization, drugs, jogging). Here, the onus is on the activists to produce adequate evidence of safety.

Conclusions

Case-centered epidemiology identifies individual susceptibility, but it may fail to identify the underlying causes of incidence. The "high-risk" strategy of prevention is an interim expedient, needed in order to protect susceptible individuals, but only for so long as the underlying causes of incidence remain unknown or uncontrollable; if causes can be removed, susceptibility ceases to matter.

Realistically, many diseases will long continue to call for both approaches, and fortunately competition between them is usually unnecessary. Nevertheless, the priority of concern should always be the discovery and control of the causes of incidence.

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Diseases Subject to the International Health Regulations

Cholera, yellow fever, and plague cases and deaths reported in the Region of the Americas up to 30 June 1985.

Country and administrative subdivision	Cholera Cases	Yellow fever		Plague Cases
		Cases	Deaths	
BOLIVIA	–	*45*	*28*	–
Cochabamba	–	1	–	–
La Paz	–	44	28	–
BRAZIL	–	*4*	*3*	*21*
Bahía	–	–	–	6
Ceará	–	–	–	15
Mato Grosso	–	4	3	–
COLOMBIA	–	*4*	*4*	–
Antioquia	–	1	1	–
Guaviare	–	2	2	–
Meta	–	1	1	–
ECUADOR	–	–	–	*3*
Loja	–	–	–	3
PERU	–	*18*	*12*	*21*
Cajamarca	–	–	–	10
Cuzco	–	7	5	–
Huánuco	–	3	2	–
Junín	–	3	2	–
Madre de Dios	–	2	1	–
Piura	–	–	–	11
San Martín	–	3	2	–
UNITED STATES OF AMERICA	–	–	–	*4*
New Mexico	–	–	–	4

Nosocomial Infection Surveillance, U.S.A., 1983

Introduction

Nosocomial infections are an important cause of morbidity and mortality in hospitalized patients; approximately 5 to 6% of hospitalized patients develop nosocomial infections *(1)*. These infections result in a prolongation of hospitalization and cost over a billion dollars a year *(2)*. The National Nosocomial Infections Study (NNIS) has collected and analyzed data on the frequency of nosocomial infections in United States hospitals since 1970. This report provides descriptive data on nosocomial infections in a sample of U.S. hospitals in 1983.

Materials and Methods

The methods of this study and characteristics of participating hospitals have been described in detail *(3)*. In brief, hospitals participating in NNIS conducted active hospital-wide surveillance using uniform defini-

tions of nosocomial infections. During 1983, 54 hospitals regularly reported data to CDC. For each nosocomial infection detected, the following information was reported: site of infection, date of onset, whether the infection was associated with a surgical procedure, the pathogen(s) isolated, occurrence of secondary bacteremia, antimicrobial susceptibility of bacterial pathogens, service of the patient, and, for those patients who died with a nosocomial infection, the relationship of the infection to death. In addition, the hospitals reported the number of patients discharged each month from the six primary services: medicine, surgery, obstetrics, gynecology, pediatrics, and newborn.

Data are recorded on standardized forms, which are sent to CDC each month. When the data are received at CDC, they are coded, entered into a computer, and edited before being analyzed.

Results

The NNIS Sample. The hospitals participating in NNIS are not a probability sample of U.S. hospitals; however, the 54 hospitals that reported data regularly in 1983 range in size from 80 to over 1,200 beds, are located throughout the United States, and include hospitals owned by state and local governments, as well as by for-profit and nonprofit organizations. The geographic distribution of the 54 hospitals among the four regions of the country (Northeast, North Central, South, West) is roughly the same as for all 6,053 U. S. hospitals included in the American Hospital Association Annual Survey of Hospitals *(4)*. Hospitals affiliated with medical schools, referred to as teaching hospitals, are still greatly overrepresented among the NNIS hospitals; 56% (30/54) of the NNIS hospitals are teaching hospitals, whereas only 14% of the hospitals across the country are affiliated with medical schools. Similarly, the 54 NNIS hospitals tend to be large, with a median size of 407 beds, compared with a median size of only 110 beds for the 6,053 U.S. hospitals.

Despite these limitations, previous analyses have shown that data collected in NNIS can be usefully interpreted by stratifying the 54 reporting hospitals into three categories: 1) nonteaching hospitals, 2) small teaching hospitals (500 or fewer beds), and 3) large teaching hospitals (more than 500 beds) *(3)*.

The infection rates (number of hospital-acquired infections/1,000 patients discharged) were highest in the large teaching hospitals and lowest in the nonteaching hospitals (Table 1), as were the infection rates on each of the six services (Table 2). In all three categories of hospital, the infection rate was highest on the surgery service (SURG), followed generally by medicine (MED), gynecology (GYN), and obstetrics (OB). The one exception was in the small teaching hospitals, where the infection rates on the medicine and gynecology services were similar. Lowest infection rates were reported on the newborn (NEW) and pediatrics (PED) services.

The urinary tract was the most frequent site of infection, followed by surgical wounds and lower respiratory tract in all three hospital categories (Table 3). On each service and for each site of infection, the infection rates were highest in the large teaching hospitals and lowest in the nonteaching hospitals.

In all three hospital categories, infections of the urinary tract, surgical wounds, and lower respiratory tract accounted for over 70% of the infections (Table 4). Primary bacteremia accounted for a higher percentage of infections in the large teaching hospitals than in the other hospitals.

Combined Rates by Service and Site. In general, the site-specific infection rate within each service was highest in the large hospitals and lowest in the nonteaching hospitals (Table 5). The site-specific infection rates by service show that for each hospital category, urinary tract infections occurred predominantly on the surgery, medicine, and gynecology services. Surgical wound infections occurred predominantly on the surgery, obstretrics, and gynecology services. Lower respiratory infections occurred predominantly on the medicine and surgery services. Primary bacteremia occurred primarily on the surgery, medicine, and newborn services. Cutaneous infections occurred primarily on the newborn service.

Pathogens. Of the 28,248 infections reported, 66% were caused by single pathogens, and 19% were caused by multiple pathogens (Figure 1). No pathogen was identified in 5% of the infections, and no culture was obtained in 10%. Of the 85% of infections in which pathogens were identified, 86% were caused by aerobic bacteria, 2% by anaerobic bacteria, and 7% by fungi

Table 1. Infection rates by hospital category, 1983.

Hospital category	Infections	Discharges	Rate[a]
Nonteaching	6,845	281,122	24.4
Small teaching	7,875	255,601	30.8
Large teaching	13,528	328,559	41.2
Total	28,248	865,282	32.7

[a]Cases/1,000 discharges.

Table 2. Infection rates[a] by hospital category and service, 1983.

Hospital category	Service					
	Surgery	Medicine	Gynecology	Obstetrics	Newborn	Pediatrics
Nonteaching	32.1	27.8	13.5	10.3	8.9	2.2
Small teaching	42.6	35.0	35.6	15.6	11.0	11.0
Large teaching	57.5	47.5	31.4	16.9	18.4	16.8
Total	44.3	37.1	27.4	14.7	13.4	11.1

[a]Cases/1,000 discharges.

Table 3. Infection rates[a] by hospital category and site of infection, 1983.

Hospital category	Infection					
	UTI[b]	SWI[c]	LRI[d]	CUT[e]	BACT[f]	Other
Nonteaching	11.1	4.0	4.1	1.3	1.3	2.5
Small teaching	13.0	6.3	4.6	1.5	1.7	3.9
Large teaching	15.0	7.0	7.5	2.7	3.8	5.2
Total	13.1	5.8	5.5	1.9	2.4	3.9

[a]Cases/1,000 discharges.
[b]UTI = urinary tract infection.
[c]SWI = surgical wound infection.
[d]LRI = lower respiratory tract infection.
[e]CUT = cutaneous infection.
[f]BACT = primary bacteremia.

Table 4. Percentage of site-specific infections by category, 1983.

Infection	Hospital category			
	Nonteaching	Small teaching	Large teaching	Total
UTI[a]	45.8	42.2	36.3	40.2
SWI[b]	16.1	20.4	16.8	17.7
LRI[c]	16.5	14.8	18.4	16.9
BACT[d]	5.8	5.3	9.1	7.3
CUT[e]	5.7	4.8	6.6	5.9
Other	10.1	12.5	12.7	12.0

[a]UTI = urinary tract infection.
[b]SWI = surgical wound infection.
[c]LRI = lower respiratory tract infection.
[d]BACT = primary bacteremia.
[e]CUT = cutaneous infection.

Figure 1. Distribution of infections, by etiology, 1983.

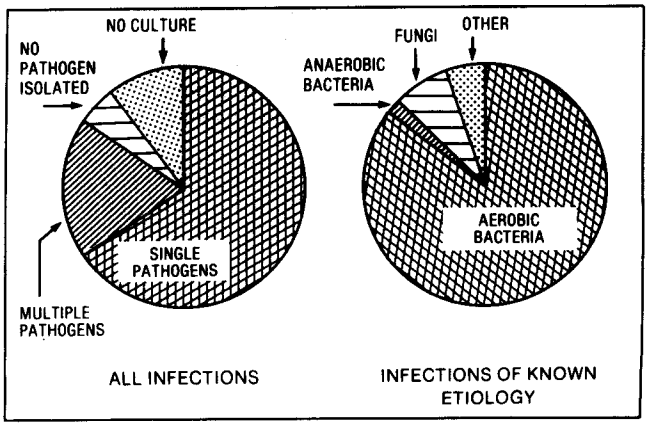

Table 5. Site-specific infection rates[a] by service, 1983.

1. Nonteaching hospitals

Service	Infection						
	UTI[b]	SWI[c]	LRI[d]	BACT[e]	CUT[f]	Other	All sites
Surgery	13.3	8.9	5.1	1.3	1.2	2.3	32.1
Medicine	15.3	0.4	5.6	2.2	1.3	3.1	27.8
Gynecology	8.2	3.8	0.4	0.2	0.2	0.7	13.5
Obstetrics	2.8	4.1	0.2	0.2	0.3	2.7	10.3
Pediatrics	0.0	0.5	0.5	0.1	0.3	0.8	2.2
Newborn	0.3	0.1	1.4	1.3	3.8	2.2	8.9
Total	11.1	4.0	4.1	1.3	1.4	2.5	24.4

2. Small teaching hospitals

Service	Infection						
	UTI	SWI	LRI	BACT	CUT	Other	All sites
Surgery	16.0	13.5	6.6	1.6	1.0	3.9	42.6
Medicine	19.1	0.7	6.7	2.6	1.5	4.5	35.0
Gynecology	20.6	11.8	0.8	0.4	0.3	1.6	35.6
Obstetrics	3.4	8.0	0.7	0.3	0.4	2.7	15.6
Pediatrics	1.4	0.6	1.3	1.6	1.1	5.0	11.0
Newborn	0.4	0.2	0.8	1.3	4.8	3.4	11.0
Total	13.0	6.3	4.6	1.7	1.5	3.9	30.8

3. Large teaching hospitals

Service	Infection						
	UTI	SWI	LRI	BACT	CUT	Other	All sites
Surgery	19.2	15.5	10.1	4.3	2.9	5.5	57.5
Medicine	21.1	1.3	10.2	5.5	2.9	6.4	47.5
Gynecology	16.2	7.7	3.0	1.1	0.7	2.2	31.4
Obstetrics	4.7	7.5	0.4	0.7	0.6	3.0	16.9
Pediatrics	2.2	2.4	3.2	2.4	2.3	4.4	16.8
Newborn	0.7	0.4	4.0	2.6	5.5	5.2	18.4
Total	15.0	7.0	7.5	3.8	2.7	5.2	41.2

[a]Cases/1,000 discharges.
[b]UTI = urinary tract infection.
[c]SWI = surgical wound infection.
[d]LRI = lower respiratory tract infection.
[e]BACT = primary bacteremia.
[f]CUT = cutaneous infection.

(Figure 1). Together viruses, protozoa, and parasites accounted for 5% of the infections of known etiology. *Escherichia coli, Staphylococcus aureus,* enterococci, and *Pseudomonas aeruginosa* were the most frequently reported pathogens. *E. coli* was the most frequently reported pathogen on the medicine, surgery, obstretrics, and gynecology services; *S. aureus* was the most frequently reported pathogen on the pediatrics and newborn services. *P. aeruginosa* was the second most frequently identified pathogen on both the medicine and surgery services and was less frequent on the other four services; enterococci were the second most frequently identified pathogens on the obstetrics and gynecology services and third most frequently identified on the medicine and surgery services; coagulase-negative staphylococci were the second most frequently identified pathogens on the pediatrics and newborn services and fourth most frequently identified on the obstetrics service.

E. coli was the pathogen most frequently associated with urinary tract infections, followed by enterococci, *P. aeruginosa, Klebsiella* spp., and *Proteus* spp. *S. aureus* was the pathogen most frequently associated with surgical wound infections, followed by enterococci, *E. coli*, coagulase-negative staphylococci, and *P. aeruginosa*. *P. aeruginosa* was the pathogen most frequently associated with lower respiratory tract infections, followed by *S. aureus, Klebsiella* spp., *Enterobacter* spp., and *Escherichia coli*. Coagulase-negative staphylococci were the pathogens most frequently associated with primary bacteremia, followed by *S. aureus, E. coli, Klebsiella* spp., and enterococci.

Mortality. Of the 54 NNIS hospitals, 50 (representing >50% of hospitalized patients with fatal nosocomial infections) assessed and reported the relationship of infection to death. These 50 hospitals reported mortality data on a total of 26,096 infections. Approximately 1% of infections were reported to have caused death, and 3.6% were reported to have contributed to death (Table 6). Infections were more often reported to cause or contribute to death in the teaching hospitals.

Table 6. Infections reported as having caused or contributed to death of patient, 1983.

Hospital category	Number of infections	Percentage that caused death	Percentage that contributed to death
Nonteaching	6,728	0.5	3.7
Small teaching	7,140	1.3	4.0
Large teaching	12,228	0.8	3.4
Total	26,096	0.9	3.6

Discussion

Nosocomial infections remain a significant cause of morbidity and mortality in hospitals in the United States. The National Nosocomial Infections Study is the only source of data collected prospectively on nosocomial infections from a group of U.S. hospitals. The nosocomial infection rate in NNIS hospitals during 1983 was 3.3 infections/100 discharges, which is similar to the rate of infection reported for the three-year period 1980 through 1982 *(3)*. These data probably understimate the true incidence of nosocomial infections in these hospitals; the Study on the Efficacy of Nosocomial Infection Control programs (SENIC Project) found that between 5 and 6% of hospitalized patients develop nosocomial infections *(1)*. Many factors contribute to this underestimating, including variability in the intensity of surveillance and availability of laboratory support. Despite the use of standard definitions of nosocomial infections by infection control personnel, the intensity of the surveillance conducted in these hospitals varies. In addition, the intensity of surveillance varies by service and pathogen. Furthermore, detection of viral infections depends more on the availability of virology laboratory support than on surveillance; therefore, hospitals without such support will not detect most viral infections.

Infection rates consistently increased from the nonteaching to the small teaching to the large teaching hospitals for all services and sites of infection, suggesting that this stratification of hospitals by hospital category effectively defines groups of patients who have different levels of risk for the development of nosocomial infections. This differential risk of infection is undoubtedly a by-product of severity of illness and the frequency of invasive diagnostic and therapeutic modalities. Data currently are not available specifically for intensive care units, but the rates of infection were highest on the surgery and medicine services, which have more high-risk patients, and lowest on the pediatrics and newborn services.

On the other hand, Valenti *et al.* have shown that viral nosocomial infections are more common in children than in adults *(5)*. In addition, Welliver and McLaughlin have shown that viruses account for approximately 14% of nosocomial infections in the pediatric population in a hospital where viral cultures are done routinely *(6)*. Since only a small proportion

of NNIS hospitals have diagnostic virology laboratories, many viral infections probably go undetected. The failure to detect these infections may partially explain the lower nosocomial infection rates reported by NNIS hospitals on the pediatrics and newborn services. In addition, other factors, such as the short duration of stay by many pediatric patients and the frequent use of isolation precautions on the pediatric and newborn services, may reduce the incidence of nosocomial infections on these services.

Infection rates on different service and at different sites of infection within the three hospital categories varied little from those reported for 1982 *(3)*. However, the increase in the primary bacteremia rates at nonteaching and large teaching hospitals identified from 1980 through 1982 continued through 1983, and a slight increase in the lower respiratory tract infection rate at nonteaching and large teaching hospitals emerged. Further study will be needed to identify the factors responsible for these increases.

Specimens for microbiologic testing were obtained from 90% of the patients reported to have nosocomial infections. For 85% of the nosocomial infections reported, an etiologic agent was identified; approximately 86% of these were aerobic bacteria. Fungi, parasites, and viruses were infrequently reported, reflecting in part the frequency with which these pathogens are looked for.

E. coli, S. aureus, enterococci, and *P. aeruginosa* were the four most common nosocomial pathogens. *E. coli* was the most frequently identified pathogen on the four adult services, reflecting the fact that this organism was the primary cause of urinary tract infections, which in turn were the most common type of infection on these services. *S. aureus* was the pathogen most frequently identified on the pediatrics and newborn services. Coagulase-negative staphylococci were the second most frequent cause of nosocomial infection on the pediatrics and newborn services and were an important cause of bacteremia on all services except gynecology. Further study will be required to assess the contribution of coagulase-negative staphylococci to the increasing rate of primary bacteremia, though recent studies suggest that the increasing use of long-line catheters may be responsible for this trend *(7)*.

The nationwide nosocomial infection surveillance system is expanding in four directions. First, despite the usefulness of the three-category stratification of hospitals used in this report, an infection-risk index identifying patients with different levels of risk of nosocomial infection is needed to permit more meaningful comparisons between hospitals. Infection rates at different institutions could then be compared within levels of patient risk or standardized for differences of distribution of patient risk. Second, microcomputer software to support an integrated nosocomial infection information management system in each hospital is being developed to improve the quality and timeliness of information collected in NNIS. Third, data on antimicrobial usage are needed to permit an assessment of the impact of antimicrobial usage on patterns of resistance in nosocomial pathogens. Finally, additional hospitals will be included in the surveillance system to provide data from a more representative sample of all acute-care hospitals in the United States.

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(Source: Adapted from W. R. Jarvis *et al.* Nosocomial infection surveillance, *CDC Surveillance Summaries* 33(2SS):9SS–21SS, 1983.)

Editorial Comment

This article summarizes surveillance data collected and analyzed by the U.S. National Nosocomial Infection Study (NNIS) coordinated by the Hospital Infections Program of the Centers for Disease Control. As the authors note, the sample of U.S. hospitals participating in the study is not a representative sample. Nevertheless, the sample is sufficient to provide useful data on the general magnitude of the nosocomial infection problem in three important categories of hospitals.

This article also draws attention to some of the difficulties involved in determining the magnitude of the nosocomial infection rate. As indicated in the article, determining actual patient risk may be difficult. In this study general hospital discharges are used as the denominator for calculating infection rates. Yet not all patients who are discharged from the hospital have been placed at significant risk for acquiring a nosocomial infection during their hospital stay. This problem was partially solved in this study by dividing hospital discharge totals according to the hospital type and the medical service within the hospital. Still, not all patients of the particular services were exposed to intravenous manipulations, surgery, urinary tract catheterizations, and other special procedures that would place them at increased risk. The extent to which the denominator includes patients not exposed to high risk procedures determines how much the nosocomial infection rate is underestimated. At present the CDC is exploring methods for defining the denominator so that it includes only those patients who are actually placed at significant risk during their hospital stay.

This study emphasizes active hospital-wide surveillance using uniform definitions of nosocomial infections. It does not rely on passive case reporting by physicians and nurses on individual hospital services. The extent to which surveillance identifies cases, of course, influences the accuracy of the measurement of the problem. A recurrent problem is the very-short-stay patient (such as those hospitalized for routine deliveries, one-day surgical procedures, or emergency room observations of 24 to 48 hours) who may be placed at risk and develop a nosocomial infection sometime after hospital discharge. Many surveillance systems are not designed to detect these cases. In some situations, short-stay patients are not even counted as hospital discharges, and may not enter in the numerator *or* the denominator.

In spite of these considerations, this article demonstrates the value of active hospital surveillance in determining the magnitude of the problem and its trends. A wide variety of useful data can be obtained through carefully designed surveillance systems. Active searching in the hospital for patients who are at risk of acquiring or have acquired a nosocomial infection is essential. Active searching is the proper role for hospital infection control personnel. Passive surveillance is too incomplete to provide an accurate picture of the extent of the problem for hospital infection control committees and has no role in a hospital infection control program.

Limited information from a variety of special studies, small groups of interested professionals, and a few major national infection control programs indicates that nosocomial infections are significant problems in PAHO's Member Countries. The resulting human suffering and the additional economic costs associated with expensive treatment and prolonged hospital stays are considerable. Many countries, Barbados, Brazil, Chile, Colombia, Costa Rica, Cuba, Mexico, and Panama, have taken steps to develop nosocomial infection control programs that incorporate most of the program elements recommended by PAHO. These include active surveillance and hospital infection control committees, and national and local norms and guidelines for hospital asepsis, medical procedures, isolation of patients, and control of antibiotic usage. Few include, however, hospital employee safety and health programs and too many continue to depend on routine bacteriological monitoring of the hospital physical environment. Nevertheless, there is an encouraging trend towards an active surveillance approach.

Calendar of Courses and Meetings

Master's Program in Entomology

The Faculties of Medicine, Agronomy, and Natural and Exact Sciences of the University of Panama are offering a Master's Program in Entomology which will begin on 30 September 1985.

The Program offers three options, general entomology, medical entomology, and agricultural entomology, and consists of 33 to 39 credits distributed between curriculum course work and seminars, to be completed over a period of two years. Training is to be supplemented with research, the results of which are to be presented by students as the dissertation for the master's degree in science with specialization in general, medical, or agricultural entomology.

To be admitted as a regular student, a candidate must possess a university degree and complete the University's application form.

Further information may be obtained from: Coordinación del Programa de Maestría en Entomología, Facultad de Medicina, Universidad de Panamá, Estafeta Universitaria, Panamá, Republic of Panama.

II National Congress on Hygiene and Epidemiology

This Congress will be held from 14 to 17 October 1985, in Havana, Cuba. Among the topics to be discussed are the following: epidemiology, community hygiene, school hygiene, radiation hygiene, radiological protection, psychology, engineering, veterinary medicine, and other specialized topics in related science areas.

Those interested may write for further information to: Consejo Nacional de Sociedades Científicas Médicas, Calle 4, No. 407 entre 17 y 19, Vedado, Havana, Cuba.

I World Congress on Pneumology and Chest Diseases

This Congress will be held from 30 November to 5 December 1985 in the Civic Center of the Peruvian Social Security Institute in Lima, Peru. The following events will be held at the same time: I Latin American Health and Social Security Meeting on the Problem of Tuberculosis; Annual Meeting of the American College of Chest Physicians, Peruvian Chapter; and the VI National Tuberculosis Seminar. These events will be held under the auspices of the Peruvian Society of Phthisiology, Pneumology, and Chest Diseases; the Peruvian Ministry of Health; the Medical College of Peru; and the Schools of Medicine of the University of Peru.

The plenary sessions will include the following topics: asthma, immunity in respiratory diseases, respiratory infections and their treatment, and interstitial pulmonary pathology. There will also be several seminars and forums.

Further information may be obtained from: Sociedad Peruana de Tisiología, Neumología y Enfermedades del Tórax, Av. General Garzón 2022, Lima II, Peru.

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