Chapter 3

Descriptive studies

Introduction

Often an important starting point for many epidemiological investigations is the description of the distribution of disease in populations (descriptive studies). The principal advantages of descriptive studies are that they are cheap and quick to complete and they give a useful initial overview of a problem that may point to the next step in its investigation.

Usually, descriptive studies make use of routinely collected health data, for example death certification data, hospital admission statistics, collated data from computerized general practices or infectious disease notifications. The main sources of routine health data are set out in Chapter 8. Some social and other variables in relation to which disease data may be examined are also available from a wide variety of routine sources. The actual source used for a particular investigation depends on the data that are required. With the exception of census material, routine sources of social data are not discussed in detail in this book.

Often the data required to describe disease distribution in a population and related variables are not readily available or are unsatisfactory for epidemiological purposes. In these circumstances it is necessary to collect the raw material for a descriptive study by special surveys. These surveys are usually cross-sectional in type (see Chapter 4).

Use of descriptive studies

Aetiological

The results of descriptive studies usually only give general guidance as to possible causes or determinants of disease, for example where broad geographical differences in prevalence are shown. Sometimes they may be quite precise, for example where a particular disease is very much more frequent within an occupational group or only occurs in a particular exposure group (e.g. asbestosis). Analysis of the data may indicate that certain attributes or exposures are more commonly found amongst people who have the disease than in those who do not. The converse may also be demonstrated, namely that certain attributes are more commonly found amongst people who do not have the disease than in those who do. This may be an equally valuable finding. It is rarely possible to prove that an agent causes a disease from a descriptive study, but investigations of this type will often generate or support hypotheses of aetiology and justify further investigations.

Clinical

Clinical impressions of the frequency of different conditions and their natural history are often misleading. The clinical impression is influenced by the special interests of individual doctors, by

events that make a particular impression and by the chance clustering of cases. To obtain a balanced view of the relative importance of different conditions, their natural history and the factors that affect outcome requires data from a total population or an unbiased (random) sample. Knowledge of the relative frequency of different diseases is helpful to the clinician when deciding on the most likely diagnosis in individual patients. The probabilities of different diseases vary at different times and in different situations.

Service planning

Health service planning in the past has been largely based on historical levels of provision and responses to demands for medical care. In order to plan services to meet needs rather than demand, and to allocate resources appropriately, accurate descriptive data are required on the relative importance and magnitude of different health problems in various segments of the community. They are also essential in order to evaluate the effectiveness of services and to monitor changes in disease incidence which may indicate a need for control action or the reallocation of resources and adjustments to service provision.

Analysis of descriptive data

Data derived from routine mortality and morbidity statistics (and from cross-sectional surveys) are usually analysed within three main categories of variable:

- time (when?)
- place (where?)
- personal characteristics (who?).

Time

Three broad patterns of variation of disease incidence with time are recognizable. These are shown below.

Variation of disease with time

- Long-term (secular) trends
- Periodic changes (including seasonality)
- Epidemics

Long-term (secular) trends

These are changes in the incidence of disease over a number of years that do not conform to an identifiable cyclical pattern. For example, the secular trend in mortality from tuberculosis in England and Wales has showed a steady fall over many years (Fig. 3.1) but recently the annual number of cases has started to rise. The observation of this trend on its own does not give any indication of its cause. However, it is sufficiently striking to justify specific studies aimed at trying to identify the reasons for the change. The inclusion in Fig. 3.1 of the times at which various discoveries were made or specific measures were introduced gives some enlightenment. The overall trend seems to have been hardly affected by the identification of the causal organism, or by the introduction of chemotherapy and bacille Calmette–Guérin (BCG) vaccination. This suggests that these played little part in the decline in mortality. However, the presentation of these data on an arithmetic scale (as in Fig. 3.1) disguises an important feature of the trend, i.e. a change in the rate at which the decline occurred. When the

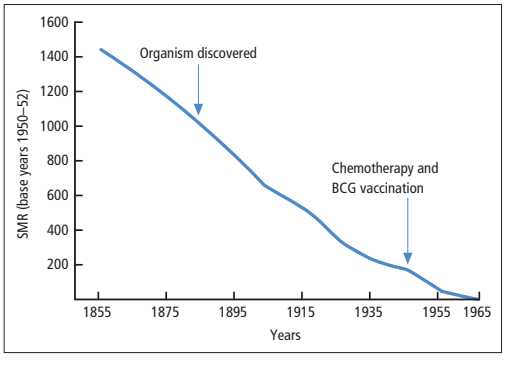

Figure 3.1 Tuberculosis mortality in England and Wales, 1855–1965 (arithmetic scale).

data are plotted on a logarithmic scale (Fig. 3.2) it becomes clear that the introduction of specific measures for the control and treatment of tuberculosis was associated with an acceleration in the established decline in mortality. It is now thought that the decline in mortality from tuberculosis was due to a complex series of changes. Until the 1950s, these were mainly an increase in the resistance of the population to infection and environmental changes that reduced the chances of acquiring infection. After the early 1950s, the rate of decline in mortality was accelerated by the newly available methods of management.

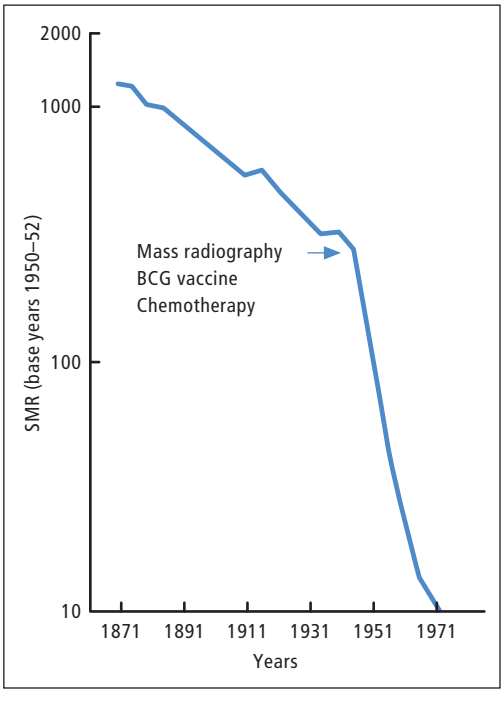

Figure 3.2 Tuberculosis mortality in England and Wales, 1871–1971 (logarithmic scale). (Reproduced with permission from *Prevention and Health: Everybody's Business*, HMSO, 1976.)

It is frequently necessary to examine secular trends both as changes in rates (arithmetic scale) and as rates of change (logarithmic scale) if the nature of a trend is to be fully appreciated.

The secular trend in mortality from carcinoma of the bronchus shows the opposite picture to that for tuberculosis (Fig. 3.3). Until quite recently it had been increasing relentlessly amongst males but the rate of increase has now declined. By contrast, the increase in mortality rates amongst women continues. The powerful correlation between mortality and changes in the national consumption of cigarettes gave rise to the hypothesis that cigarette smoking could be the causal agent, although it did not prove causality. The hypothesis has since been explored through large numbers of epidemiological studies.

Periodic changes

These are more or less regular or cyclic changes in incidence. The most common examples are seen in infectious diseases. For example, until a vaccine was introduced, measles showed a regular biennial cycle in incidence in England and Wales (Fig. 3.4). The cycles were probably the result of changes in

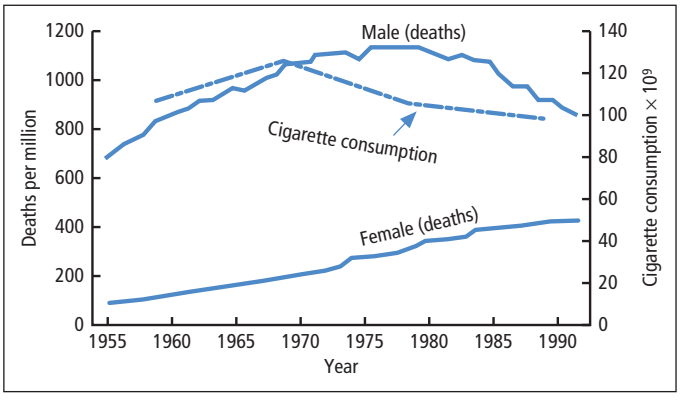

Figure 3.3 Carcinoma of lung, bronchus and trachea. Deaths per million population in England and Wales, 1955–92, and cigarette consumption per year. (Reproduced with permission of the Office of National Statistics).

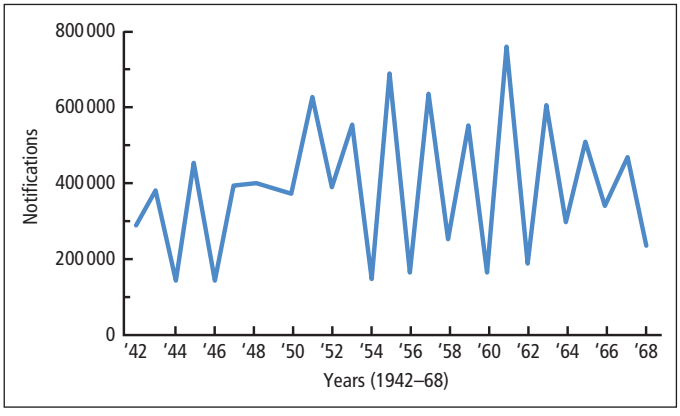

Figure 3.4 Notifications of measles in England and Wales showing periodic variation (prior to introduction of measles vaccination). (Reproduced with permission of the Office of Population Censuses and Surveys (Crown copyright).)

the levels of child population (herd) immunity (see p. 105). Other infectious diseases such as whooping cough, rubella and infectious hepatitis show less regular, but nevertheless distinct, cycles with longer intervals between peaks.

Seasonality

This is a special example of periodic change. The environmental conditions that favour the presence of an agent, and the likelihood of its successful transmission, change with the seasons of the year. Respiratory infections, which spread directly from person to person by the air-borne route, are more common in winter months when people live in much closer contact with each other than in the summer. By contrast, gastrointestinal infections, which spread by the faecal–oral route, often through contamination of food, are more common in summer months when the ambient temperatures favour the multiplication of bacteria in food. The regular seasonality of gastrointestinal infections is shown in Fig. 3.5 in which the number of notifications of food poisoning for each quarter in 1974–89 are plotted. A particularly interesting feature of food poisoning incidence is that the marked seasonality is combined with a noticeable secular trend. The number of cases notified from late 1988 and early 1989 was much higher than in previous years. This could be due to contamination in the food chain, a decline in standards of food storage, distribution or preparation, or the result of an increase in notification rates following publicity given to the problem of food poisoning.

Some non-infectious conditions, for example allergic rhinitis, deaths from drowning and road accidents, also display distinct seasonality. For

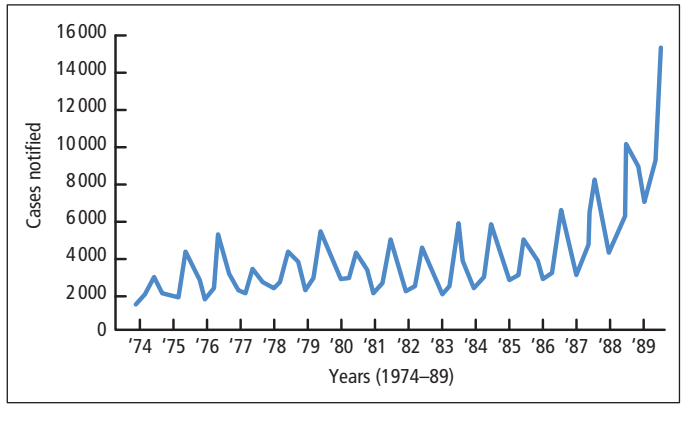

Figure 3.5 Quarterly notifications of food poisoning in England and Wales, 1974–89.

Table 3.1 Seasonality of birth of schizophrenic and neurotic people compared with that of the general population (expected) showing an increased frequency of births of schizophrenic people in the first part of the year but no seasonality amongst neurotic people. (Adapted from Hare E, Price J, Slater E. *Br J Psychiatry* 1974; **124**: 81–86.)

	Quarter of birth			
	Jan–Mar	**Apr–June**	**July–Sept**	**Oct–Dec**
Schizophrenic people				
Observed	1383.0	1412.0	1178.0	1166.0
Expected	1292.1	1342.8	1293.1	1211.1
Observed as a percentage of expected	107	105	91	96
Neurotic people				
Observed	3085.0	3172.0	2949.0	2882.0
Expected	3024.1	3150.6	3042.0	2844.2
Observed as a percentage of expected	101	101	97	101

most of these, the explanation for the seasonality is not difficult to infer. There are seasonal variations in the incidence of certain other conditions, however, for which there is as yet no rational explanation. For example, schizophrenic people are more likely than the general population to be born in the early months of the year (February and March) (Table 3.1). Many hypotheses have been offered to explain this observation, including the proposition that the disease is caused by an intrauterine infection, that the mothers of schizophrenic people are more likely to miscarry at certain times of the year (thereby resulting in a deficit of births in months other than January to March) and that the mothers are more likely to conceive in April to June than are other women. However, none has yet been proved.

It should be noted that the seasonality in disease patterns related to climatic conditions is reversed in the southern Hemisphere.

Epidemics

These are temporary increases in the incidence of disease in populations. The most obvious epidemics are of infectious diseases such as influenza (Fig. 3.6) but non-infectious epidemics do occur. For example, there was an increase in asthma deaths in the 1960s associated with the increased use of pressurized aerosol bronchodilators (Fig. 3.7).

The word 'epidemic' is also sometimes used to describe an increase in incidence above the level expected from past experience in the same population (or from experience in another population

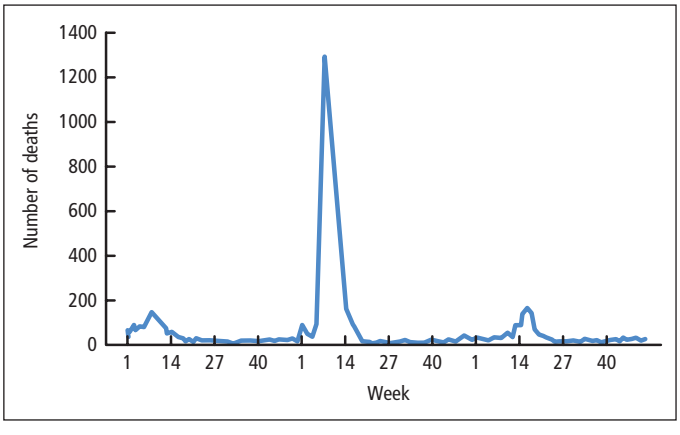

Figure 3.6 Weekly deaths from influenza in England and Wales, 1975–77.

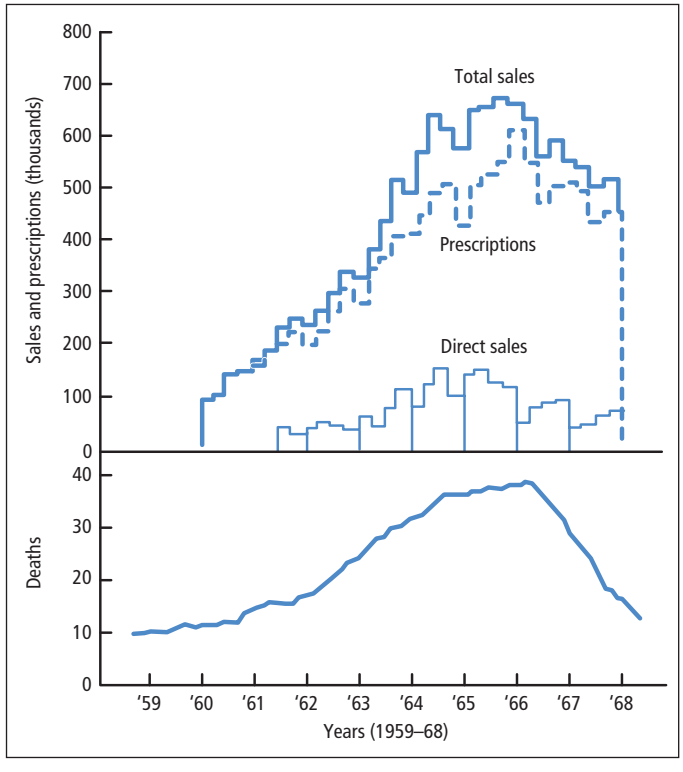

Figure 3.7 Sales and prescriptions of asthma preparations compared with deaths from asthma among people aged 5–34 years, in England and Wales, 1959–68. (After Inman WHW, Adelstein AM. *Lancet* 1969; **ii**: 279.)

with similar demographic and social characteristics). However, if the strict definition of epidemic is used, it is inappropriate to use the term to describe secular trends in diseases such as diabetes or malignant melanoma, since there is no evidence that they are temporary increases in incidence.

Place

Variations in the incidence or prevalence of disease with place can be considered under three headings.

Variation of disease with place

- Broad geographical differences
- Local differences
- Variations within single institutions

Broad geographical differences

Variations in the incidence of disease are sometimes related to factors such as climate, social and cultural habits (including diet), and the presence of vectors or of other naturally occurring hazards. Although the incidence of disease does not respect administrative boundaries between countries or regions, these boundaries often follow broadly natural ecological boundaries and tend to encompass common social and cultural groups. Much valuable information pointing to possible causes of disease has been obtained by comparisons of routinely collected data between countries and other administrative units. For example, various forms of cancer and other conditions show striking geographical difference in incidence (Table 3.2).

Local differences

The distribution of a disease may be limited by the localization of its cause. Thus, if a main water sup-

Table 3.2 Geographical variation in the incidence of disease. Comparison of death rates in England and Wales with those in Japan (1979) for various causes shows considerable discrepancies. Both are highly industrialized countries with well-developed health services, but they have very different cultures and racial origins. (Data from *World Health Statistics Annual*, WHO, Geneva, 1981.)

	Rates per 100 000	
Disease	**England and Wales**	**Japan**
England and Wales high, Japan low		
Cancer of breast (females)	47.9	6.6
Cancer of prostate	20.2	2.9
Cancer of colon	20.9	6.3
England and Wales low, Japan high		
Cancer of stomach	23.0	43.8
Cirrhosis of liver (males)	5.0	21.1
Suicide	8.5	18.0

ply becomes contaminated, the illnesses that result from the contamination will be clustered in people living within the distribution area of the water. 'Spot-maps' on which cases are marked may show local concentrations that suggest possible sources. In interpreting such maps, it is important to relate the spatial distribution of cases to the density of population. The classical study of the 1854 cholera outbreak in the Golden Square area of London by John Snow used such a technique and led him to identify the particular water pump that was the source of the infection. In this instance, cases were clustered in the streets close to the Board Street pump, while comparatively few cases occurred in the vicinity of other pumps in the area.

A special kind of locality difference is that which exists between urban and rural environments. In general, people who live in urban areas are subjected to different hazards from those experienced by people who live in rural areas. These differences alter their risk of certain diseases, sometimes to the advantage of the country person and sometimes to the benefit of the town dweller. In urban areas, there may be better housing and sanitation but more overcrowding and air pollution; more leisure but less exercise, fresh food and sunlight; more industrial hazards but fewer risks of infection from animal contacts and vectors. In industrial societies, however, where commuting is a common practice, the distinction between town and country dwellers is often blurred. Table 3.3 shows some differences in mortality between urban and rural areas in England and Wales.

Variations within single institutions

In institutions such as schools, military barracks, holiday camps and hospitals, variations in attack rates by class, platoon, chalet or ward may focus attention on possible sources or routes of spread. For example, in an outbreak of surgical wound infection, identifying the bed positions of patients, ward duties of staff and theatres used may suggest the identity of a carrier or other source of infection. Similarly, in places of work the danger of developing disease may be shown to be inversely related to distance from source of a chemical hazard.

A high incidence of a disease amongst people who share the same environment does not prove that a factor within the environment was the cause of the disease. It may be that the people have chosen, or have been chosen, to share the same environment because they have an increased susceptibility to that disease or because of pre-existing disease or disability.

Personal characteristics

The chances of an individual developing a disease may be affected by personal characteristics. The

Table 3.3 Differences in mortality amongst males between urban and rural districts in England and Wales 1969–73 (SMRs).

	Urban with populations				
	Conurbations	**Over 100 000**	**50 000–100 000**	**Under 50 000**	**Rural**
Malignant neoplasms					
Bronchus, trachea and lung	118	109	98	90	79
Bladder	112	109	99	96	82
Chronic rheumatic heart disease	114	110	88	94	85
Ischaemic heart disease	99	106	107	101	95
Influenza	84	98	90	116	111
Bronchitis	117	109	98	96	76
Motor vehicle accidents	87	95	98	99	124
Accidental poisoning	126	110	100	89	67
Homicide	151	99	95	71	56

analysis of data on the incidence of disease in relation to the personal characteristics of victims provides useful indicators of possible causes. The personal characteristics can be classified as shown below.

Variation of disease due to personal characteristics

Intrinsic factors (affect susceptibility if exposed to causal agents)
- Age
- Gender
- Ethnic group

Personal habits or lifestyle (affect exposure)
- Family
- Occupation and socioeconomic group

Intrinsic factors

Age

Most diseases vary in both frequency and severity with age. In general, children are more susceptible to infectious diseases, young adults are more accident prone and older adults tend to suffer the results of long exposure to occupational and other environmental hazards. In infancy, immaturity and genetic defects affect susceptibility to disease. In later life, physiological changes, degenerative processes and an increased liability to malignant tumours are the dominant determinants of the patterns of illness.

The fact that the incidence of most diseases varies with age can complicate the comparison of morbidity and mortality between populations with dissimilar age structures. For example, the age structure of a population of military personnel is likely to be substantially different from that of a group of practising physicians. Therefore, it is to be expected that the two groups will differ in their incidence of many diseases. In order to make a valid comparison between these populations it is essential to adjust the data to take account of differences in their age structure. This procedure is called standardization (see Chapter 9).

Age differences in the incidence of disease may also be accounted for by a so-called 'cohort effect'. This occurs when individuals born in a particular year, or living at a particular point in time, are exposed to the same noxious agent. They then carry an enhanced risk of the disease caused by that noxious agent for a long period, sometimes for the rest of their lives. For example, the children who were exposed to radiation in Hiroshima and Nagasaki in 1945 when the atomic bombs were detonated have had higher than expected incidence of leukaemia throughout their lives.

Gender

There is evidence that males are intrinsically more

Table 3.4 Death rates at different ages for males and females in England and Wales, 2003 (deaths per 1000).

Age	Males	Females
Stillbirths	5.7	4.9
Under 1 year	5.9	4.5
1–4 years	0.25	0.20
5–9 years	0.12	0.10
10–14 years	0.16	0.11
15–19 years	0.49	0.24
20–24 years	0.78	0.27
25–34 years	0.94	0.44
35–44 years	1.58	0.94
45–54 years	3.85	2.54
55–64 years	9.7	6.0
65–74 years	27.2	17.0
75–84 years	73.6	50.5
85 and over	188.1	159.8

vulnerable to disease and death than are females. This is first apparent in the differential rates of stillbirth and early neonatal mortality, and remains throughout life (Table 3.4). Indeed, during later life, with the exception of disorders that are specific to the female, there are few diseases which have a greater incidence in women than in men. In most societies, men are exposed to a greater number and variety of hazards than are females often because of differences in their leisure and work activities. Even when the two sexes are exposed to the same hazards for the same period of time, there is evidence that women are less likely to develop disease and that they survive better than men. Some diseases appear to vary in incidence between the sexes only because they are more readily diagnosed in one sex than the other, for example gonorrhoea in men, or because they are more likely to come to medical attention, for example in mothers of young children.

Ethnic group

This term tends to be used very loosely to describe a number of personal characteristics, including some that are strictly genetically determined, for example skin colour, and some that have nothing to do with genetics, for example country of birth and religion. It is often difficult to disentangle these ethnic characteristics from a number of other factors which affect the incidence of disease, for example dietary habits, religious practices, occupation and socioeconomic status. The effect of ethnicity on the incidence of disease is best studied in communities where people of different groups are living side by side and in similar circumstances. For example, studies in the UK have shown a higher prevalence of type 2 diabetes in Asians compared with the white population. This is probably due to genetic differences. On the other hand, in New Zealand the differences in the cot death rate between Maoris and Europeans is related principally to the lower socioeconomic status of most Maoris and lifestyle factors such as maternal smoking.

Personal habits or lifestyle

Family

Some diseases are especially frequent in certain families because of a common genetic inheritance, which is an intrinsic characteristic of the individuals. The risk of disease among members of the same family may also be increased because the members share a common environment and culture. Culture affects a wide range of disease-related factors such as type of housing, dietary habits and the way in which food is prepared, as well as the individual's reaction to illness.

Occupation and socioeconomic group

Some people are exposed to special risks in the course of their occupation. These include exposures to dust (particularly coal dust, silica and asbestos), toxic substances and gases used in industrial processes, and the risks of accident. Some occupations influence habits such as the amount of tobacco smoked and of alcohol consumed or the regularity of meals, which in turn affect disease incidence.

When interpreting any observed correlation between occupation and disease it is necessary to take account of the factors which determine a person's choice of occupation. Some may affect the person's susceptibility to disease; for example, tall and powerful people may choose physically demanding

Table 3.5 SMRs for ages 15–64 years (England and Wales) showing trends by social class for specific causes of death.

Cause of death (ICD number)	I	II	IIIN	IIIM	IV	V
Malignant neoplasm of stomach (151)	50	66	79	118	125	147
Malignant neoplasm of trachea, bronchus and lung (162)	53	68	84	118	123	143
IHD (410–414)	88	91	114	107	108	111
Cerebrovascular disease (430–438)	80	86	98	106	111	136
Bronchitis, emphysema and asthma (490–493)	36	51	82	113	128	188

occupations whilst others may chose 'sheltered' occupations because they already suffer mentally or physically disabling diseases. Some, because of chronic disease, may be unable to keep demanding jobs in the higher socioeconomic groups; they tend to move down the social scale (social class migration).

Social class is derived from occupation and status within an occupational group (i.e. manager, foreman, unskilled). The concept of social class encompasses income group, education and social status, as well as occupation. Most diseases show a positive social class gradient, with a higher incidence in manual workers than in professional groups (Table 3.5).

Interactions of time, place and personal characteristics

Frequently, two or more factors correlate with the incidence of a disease and also with each other. It may be that only one factor is a causal agent or determinant and that the correlation with a second factor is fortuitous. Sometimes, however, two separate causes of disease interact with each other in such a way that the effect of the two acting together in the same individuals is greater than that of either acting alone. For example, while people who work with asbestos and who do not smoke have a higher incidence of bronchial carcinoma than other non-smokers, those who smoke have a much higher incidence than would be expected in people with similar smoking habits in the general population. Interactions such as this are often very complex and the analysis of observed distributions can do no more than indicate possible determinants which merit more detailed and carefully controlled enquiry. Time, place and personal interactions can be separated if circumstances arise in which one of the variables can be kept constant while the others change. For example, comparison of disease frequency in migrant populations with the frequency in their place of origin is often informative, particularly where migrants move from an area with a high incidence of disease to one with a low incidence, or vice versa. When they migrate, they take with them their original hereditary susceptibilities but they change their risk of exposure to harmful agents. For example, the incidence of cancer of the stomach is higher in Japanese living in Japan than those living in the USA, while for cancer of the large bowel the reverse is true. In time, when migrants are assimilated into the host culture, they may be exposed to new risks in that culture. Thus, studies of migrant groups can also be used to measure the latent period between exposure and onset of disease. For example, the incidence of multiple sclerosis is higher in Europeans who migrated to South Africa before the age of 15 than in those born in South Africa.

It must be stressed that caution is needed in studies of migrants because they are self-selected from the original population and their risks of disease may have been different from those who did not migrate.