

Chapter 6

Social Risk Factors

Klaus Krickeberg and David Klemperer

6.1 Introduction

Among risk factors for infectious diseases, *social* ones present a particular challenge due to the increasing importance of old and new infections on the one hand, and the complexity of social reality on the other. This is not a new phenomenon. Social risk factors for infectious diseases were a subject of much thought throughout, roughly, the early nineteenth century up to the First World War although rigorous epidemiologic methods were of course still rudimentary. Modern public health originated during that period even before the discovery of specific micro-organisms as infective agents (Rosen 1993). In addition to organizing public health services, the main preoccupation of health authorities was *hygiene*, and hygiene then amounted, in modern epidemiologic terms, to the fight against the principal biological risk factor, which is the exposition to the known or suspected infective agent (see Chapter 5). However, social factors like poverty, malnutrition, deficient water supply, crowded housing, and dangerous occupations played an equally important role.

As it is well known, the interest in infectious diseases in general, and in their social risk factors in particular, waned in the sequel, especially after the Second World War, partly as a consequence of over-reliance on antibiotics in spite of early evidence of resistance phenomena (see Chapter 3). Hygienic standards even declined in many places. Then, in the last one or two decades, several phenomena contributed to a resurgence of activities around infectious diseases. More and more people, both in the medical professions and outside, became aware of the dangers of resistance of infective agents or their hosts to drugs or insecticides. Nosocomial infections became more frequent (see Chapter 22). Finally, several *new* infectious diseases emerged (see Chapter 3). Still, this renewed interest in infectious diseases has not yet led to a large coherent body of studies on their social risk factors.

There are several ways of looking at the influence of social risk factors on the distribution and spread of an infectious disease in a given population. What is indeed

K. Krickeberg (✉)
Formerly, Université de Paris V, France; Bielefeld, Germany
e-mail: krik@ideenwelt.de

the meaning of a statement like “Poor people tend to suffer more than rich ones from tuberculosis”? The classical epidemiologic interpretation (see Chapter 11) makes use of input (exposition) and outcome variables that are defined for *persons*. Thus, for every individual person in the population at hand the input, or risk, variable would describe the degree of poverty of that person, and the outcome variable would represent some feature of his or her state of health connected with tuberculosis. An epidemiologic study of relations between these two variables would be *person based*.

However, already in the early times of social epidemiology it appeared sometimes to be necessary or more natural to deal with *communities* in the sense of *groups*, or *sets*, of people, and not with individual persons as the underlying *units* of concepts and studies. One would, for example, describe the degree of poverty of a family or a block of dwellings or an entire geographical area by a certain indicator, and similarly speak of the situation of tuberculosis in such an entity. Sometimes, a community-based study will be conducted if only information concerning the underlying communities as a whole can be obtained, but not data about their individual members. As a rule such a study will provide a rougher picture of the action of the factor “poverty” than a person-based investigation. In other settings, the unit “community” will be in the centre of interest from the start. Moreover, as we will see later, there are social risk factors that can only be defined for groups of people and have no meaning for individual persons. Finally, in a “multilevel” approach, both person-based and community-based concepts on various levels are being investigated.

The challenge with which we are now confronted is to study the effect of social factors more systematically, using modern epidemiologic concepts, and at the same time to keep in mind the relevance and usefulness of the results for public health policy. In the following, after having reviewed the main concepts (Sections 6.2, 6.3, and 6.4), we will try to convey an idea about the kind of past and present work in this area by presenting typical examples, arranged by sources of information and methods of study (Sections 6.5, 6.6, 6.7, 6.8, and 6.9). We then summarize the methodological issues and point out some desirable future directions of research (Section 6.10). Finally, in Section 6.11, we will mention some implications for health policy, knowing fully well that many or most of them will appear unrealistic at this point in time, or controversial, or both.

6.2 Pathways

The exposition to an infective agent is, by definition, a necessary condition for the corresponding infectious disease to manifest itself in an individual. With a few exceptions, it is not a sufficient one, although in the beginning of modern medical bacteriology the third Henle–Koch postulate (Susser 2001) seemed to claim that. Infectious diseases are indeed, like non-infectious ones, mostly multi-factorial. Given the type of agent and the intensity of the exposition of a specific person, both the first manifestation and the course of the disease still depend on additional factors

such as genetic disposition, acquired immunity, and the general state of physical and mental health of the subject including stress, fatigue, and concomitant infections.

Factors of this kind act more or less *directly* on the disease, but they are not easy to define rigorously or to measure. For simplicity of reference, we will call them the *biological* variables, subsuming mental factors in this category. They are influenced, in turn, by factors that *can* be quantified and measured and that normally act indirectly through the biological ones. We can classify these factors with indirect action by the following scheme, which applies to infectious as well as non-infectious diseases and in essence goes back to antiquity:

- environmental factors
- social factors
- lifestyle-related factors
- iatrogenic factors

where of course the dividing lines still need to be made precise. It is *these* factors that enter into public health policy. Hence, from the viewpoint of public health, it is *their* epidemiologic action in which we are interested even when we cannot completely elucidate the underlying mechanisms.

Regarding social factors, they do not only act directly on biological variables, but they interact with each other, too. They may also have an influence on environmental, lifestyle, or iatrogenic factors, which in turn affect biological variables. Finally, they influence the *transmission* of the infection within a population as it is sometimes being modelled mathematically (see Chapter 12). Thus the overall picture of the action of social risk factors is, finally, fairly complicated and modern studies try to tackle the problem by following this action along the various *paths* it may take.

One also attempts to uncover *causal* effects in some sense. Causation research may use, among others, the full machinery of the mathematical–statistical analysis of dependencies as presented, e.g. in Cox and Wermuth (1996). It is widespread in the study of social risk factors for non-infectious diseases (Chandola and Marmot 2005), but less so for infectious diseases because of the preponderance of the factor “infection” which has been diverting the attention from others. The papers by Krieger (1994, 2001) discuss basic aspects of the epidemiologic reasoning involved and in particular of the search for causes; they contain extensive bibliographies (see also Section 6.6).

6.3 Outcome Variables

Let us first try to gain an overview of the variables we are going to deal with. The particular outcome variables to be studied are determined by the fact that we are treating *infectious* disease epidemiology. In any person-based study concerning a given infectious disease we will naturally be interested in one or several variables of any of the following types:

- The presence of antibodies.
- A clinically diagnosed infection.

- An infection defined and diagnosed by biological, chemical or physical procedures, e.g. blood, sputum, or urine examination in the laboratory or X-rays.
- Various aspects of the evolution of the disease like hospitalization, time to recovery, fatal issue, etc.
- Direct health consequences of infections such as dental caries, periodontal problems or virus-induced cancers.

A community-based investigation will use corresponding outcome variables that describe the state of health in a population, e.g. an incidence, prevalence or mortality in a city or country. Such a variable is usually obtained by averaging the values of an underlying person-defined variable over such and such population, but dealing directly with this original individual-level variable may be either not feasible or not the focus of attention.

6.4 Social Risk Factors

There will probably be less agreement about the exposure variables or determinants, to be studied, i.e. about the concept of a *social risk factor*. Let us start again with person-based concepts. In the older literature one encounters the expression “demographic variables”, which stands for age, sex, marital status, place of residence, religion, race (ethnicity), education, occupation, etc. Many of these variables appear in the descriptive epidemiology of classical health statistics. In modern social epidemiology we find a more differentiated view of risk factors (Bourdieu 1979). Variables like age, sex, marital status, number of children or nationality are sometimes called *horizontal* social factors because a priori they do not imply the idea of a hierarchy, i.e. of “upper” and “lower” classes. In contrast, there are *vertical* variables tied to the idea of social status such as education, profession, income, housing, and to some extent also previous migration. Variables like gender or ethnicity or place of residence may belong to one or the other category depending on the context. Vertical variables are the social risk factors in the narrower sense, and it is them that we are investigating when we are looking at social inequality and injustice and their influence on infectious diseases.

Similar remarks apply to social risk factors defined for communities. They focus on exposure patterns within groups of people and not on individual-level factors and may be of manifold nature. Certain social risk factors such as *segregation* or the existence of particular *social networks* and of *social capital* cannot even be reasonably defined for single persons; they are relations *between* members of a group of people.

6.5 Routine Health Statistics

Descriptive epidemiology is often identified with classical medical statistics. This amounts to the regular, e.g. monthly or yearly, cross-sectional study of some of the “demographic” social risk factors listed above such as age, sex, place of residence

and others. By studying the geographical factor on the scale of entire nations or regions, we gain a global view of the distribution of the incidence or prevalence of infectious diseases over the world, of their relative weight and of their “burden”. This is the subject of Chapter 1. A somewhat more detailed description is provided by WHO publications (Murray et al. 2004), which, however, largely reflect the reports received from Ministries of Health and other national administrations. These reports, in turn, are based on national health information systems (Krickeberg 2007) that are often unreliable. They derive their information mainly from hospital and health centre records (see Chapter 8). Systematic statistical control and correction mechanisms for these systems are rare both at the national and the WHO level.

For particular infectious diseases like malaria, tuberculosis or leprosy, specialized information systems exist in some countries; see, e.g. the recent description of the Tuberculosis–Leprosy Management System in Malaysia where the social factor “migration” is taken into account (Dony et al. 2004). Another part of descriptive routine information on infectious diseases comes from epidemiologic surveillance; see Chapter 9 for more details and examples. Particular features of the evaluation of information containing a geographical component are presented in Chapter 11.

An analysis of existing health statistics can only yield very crude and limited insights into the action of social factors on infectious diseases. The units of study are necessarily fairly large communities like provinces of a given country. Very few social risk factors are being taken into account. Let us look at a typical “*Health Statistics Yearbook*”, e.g. the one edited by the Vietnamese Ministry of Health for the year 2003. There, incidences or prevalences of infectious diseases are mostly given for the eight regions of the country. For tuberculosis, malaria, leprosy and AIDS they are also provided by province, and the resulting inter-provincial comparisons are presented in the form of maps. There is only one socio-economic factor in addition to the geographic one; it concerns malnutrition of children. Hence a study of a factor like income would necessitate the concomitant use of tables of per capita income in every province in 2003.

A good example of such a simultaneous use of several “official” sources of indicators (WHO, UNDP and others) is the study of Hobdell et al. (2003) about the relation of oral diseases and socio-economic status. Its aim is not *intra-* but *inter-*country comparisons; thus the underlying units are countries. The outcomes are measured, in addition to (non-infectious) oral cancer prevalence, by the prevalence of dental caries and periodontal diseases. Socio-economic risk factors appear in the form of country indicators such as the Human Development Index, Mean Years of Schooling, Gross National Product per capita, or the Gini Index, which describes unequal distribution of income in a country. It turns out that many of these risk factors are strongly associated with dental caries and even more with destructive periodontal disease (but very little with oral cancer). A community study of this kind leaves of course open the question of pathways of action: to which extent do the socio-economic factors studied act on factors known to favour, e.g. periodontal disease, such as access to water, toothbrushes and toothpaste, tobacco, and psychosocial stress? Do they also act in some other way? To answer such questions, finer studies are required.

6.6 Classical Studies

The influence of social conditions, in particular of vertical risk factors, on infectious diseases has been suspected, observed and mentioned for a long time. For example, in the Middle Ages leprosy was known to be endemic particularly among the poor (Rosen 1993, p. 39). Typhoid fever was called “jail fever” in England in the sixteenth and seventeenth centuries, because it seemed to be an inevitable consequence of going to prison (Rosen 1993, p. 65). In the nineteenth century, merely stating hypotheses and describing observations gradually made way for more and more rigorous studies based on the quintessence of all epidemiologic research, namely the comparison of groups defined by different levels of exposure. Virchow’s classical report (Virchow 1848) on a typhoid fever epidemic in Upper Silesia (which was in fact louse-borne typhus as we know by now) marks a transition. He carefully describes factors such as topography, history, ethnicity and culture, dwellings, hygiene and nutrition as well as outcomes, but the paucity of health statistics and the short duration of his mission do not yet allow a comparative study.

The subsequent development and the state of knowledge and consciousness on the eve of the First World War are very well presented in the three chapters of the classical book by Mosse and Tugendreich (1913) devoted to, respectively, sexually transmissible diseases, general infectious diseases and tuberculosis. We are going to review the chapter on tuberculosis written by M. Mosse. It is the most substantial and interesting of the three, which is not surprising since tuberculosis had then been the most studied infectious disease in relation to social factors – as it probably still is.

In 1913, the necessity of comparing populations seems to have been widely recognized. Most of the papers summarized in this chapter describe investigations that might be looked upon, from a modern point of view, as record-based retrospective cohort studies, that is, they draw on official health statistics or hospital records and present, for various levels of the risk factors, corresponding data on morbidity or mortality. Questions around what we would now call “hospital bias” are being discussed in this context. Statistical material from health insurances in the United States and Germany is also already being exploited. A few other studies give data on risk factors for various levels of an outcome variable, which amounts to a rudimentary case–control study, but they are methodologically deficient and do not provide any insight. There are both person- and community-based studies.

Modern concepts of epidemiology appear. For example, the word “factor” is used from the beginning in the sense of “social risk factor”, and the chapter is organized accordingly, with horizontal risk factors first and vertical ones afterwards. Pathways are already being considered and so is the relation of specific social factors with others.

The fundamental idea of a *confounder* plays both implicitly and explicitly an important role although the term itself is not yet used. Mosse talks about “veiling” (*Verschleierung*) and later points out the role of the factor “age” as a possible confounder, or criticizes the lack of stratifications by age in certain studies. In a

pioneering paper published in 1906, Newsholme deals with the decrease of general mortality in England and tries to identify those social factors that contribute *specifically* to diminishing TB mortality. He concludes that these specific factors are “housing” and “isolation of patients in sanatoria”. His method consists of comparing the temporal evolution of mortalities in several countries. A study by Dörner starting with data from 1852 shows that for a fixed income level, the factor “housing” has an important influence on TB incidence. An interesting discussion in the same realm of confounding factors tries to explain why alcoholics seem to be less prone to TB morbidity and mortality than the general population. Schmid in 1899 eliminates the possible confounder “working in agriculture or not” in a study of the action of the geographical factor “altitude” by stratification.

Let us look at some particularly valuable studies described by Mosse, starting with risk factors related to “income” or “wealth”. Sørensen divided the Danish population in the 1880s into three social groups, stratified by age and sex, and found striking differences of TB mortality. For the same outcome variable, a community study by Neef covering the same period investigates the factor “average income in a district of the city of Breslau”; Bertillon around 1909 picks six districts (*arrondissements*) of Paris from “very rich” to “very poor”; and Marié-Davy defines an exposure variable for all 20 “arrondissements” of Paris by “number of windows per person”, resulting in a very clear inverse relation with TB mortality in the period 1858–1902.

The influence of the factor “profession” on TB morbidity and mortality is the subject of several studies, some of them based on insurance records. A remarkable analysis by Tobler gives TB mortality in Switzerland for the period 1889–1900 in many professions, stratified by age, with farmers displaying the lowest and stonemasons the highest death rates. Very “modern” is also a paper published in 1912 by the Statistical Office of the city of Halle (Germany), which attempts to disentangle the action of the factors “profession” and “social standing”, the first one having a markedly higher influence.

The modernity of the book by Mosse and Tugendreich contrasts with some papers published recently but written in the spirit of the middle of the nineteenth century. We quote Zahraoui-Mehadji et al. (2004) as a deterrent. Its purpose was to measure the risk of infection by HIV, hepatitis B and C and syphilis incurred by traditional barbers in the Casablanca region of Morocco. The four corresponding outcome variables were well defined in serological terms. A sample of 150 male barbers was selected. While HIV serology turned out to be negative for all of them, the other three outcome variables were positive for many. Regarding the risk factor studied here, it was clearly the factor “occupation”. However, since a sample was taken only from the particular occupation “traditional barber”, comparison with other occupations was impossible and the public health recommendations made on the basis of this work are standing on shaky grounds.

We are now going to jump to the present and try to shed some light on the state of the field by reviewing a few selected investigations, starting with the methodologically simplest ones.

6.7 Studies Centring Round a Single Dichotomous Risk Factor

They form the next higher level of epidemiologic sophistication. In a series of papers from Israel, the influence of the social factor “Jewish or Bedouin” on some infectious disease-related outcome variables was investigated by various methods, largely based on hospital records. The article by Levy et al. (1998) is a characteristic example. The target population of this study consists of all children under 15 years of age in the Negev region, and the risk factor determines the two complementary subpopulations to be compared, viz. Jewish and Bedouin children, respectively. The authors remark that these two population groups have equal access to tertiary health care in the only regional hospital, free from financial barriers, and they thus discard a conceivable confounder. The groups differ by lifestyle and religion. The Jewish lifestyle is largely urban and industrialized whereas the Moslem Bedouins are in transition from their traditional nomadic life to settlement. The main outcome variable is “hospitalization for infectious diseases”. The study consists in estimating, in each of the two populations, the frequency of such hospitalizations during the period 1989–1991, expressed as numbers of hospitalizations due to infectious diseases per 10,000 child years. There were 15,947 hospitalizations for any cause during that period, documented by computerized hospital records. The number of children in each population group and in any year of the period in question was obtained from population statistics. Per 10,000 child years there were 250 hospitalizations for infectious diseases of Bedouins and 121 of Jews. For hospitalizations because of diarrhoea, the corresponding figures were 114 and 32, respectively, and for pneumonia, 55 and 19. These differences were even more pronounced in infants, especially for diarrhoea. For infectious diseases again, paediatric intensive care hospitalizations were over 2.5 times more frequent for Bedouin children than for Jewish ones, and in-hospital mortality was near to 6 times higher.

Technically speaking, this is again a *record-based cohort* study. A recent example of several *case-control* studies conducted in parallel treats the outcome variable “new case of avian influenza” in Vietnam (Pham Ngoc Dinh et al. 2006). Each of the social risk factors “the patient *prepared* dead or sick poultry less than 8 days before onset”, “there *was* such poultry in the household in this period”, and “there is no indoor water source in his house”, turned out to have a significant influence, while certain others did not. In addition, a multiple logistic regression was done.

The AIDS epidemic has naturally given rise to a large number of studies, to which we will come back in Section 6.9. In Africa, the factor “migration” has been the focus of much interest, continuing a line of research that started in 1949 for syphilis and gonorrhoea (Kark 1949). Not surprisingly, migration always turned out to be an influential risk factor. The particular “circular” nature of the migration at hand where workers return regularly to their fixed home was more and more taken into account. Earlier investigations centred around HIV infections contracted by the male migrant worker while away, and infections of his female partner were considered as having originated from him after his return. A recent more refined cross-sectional study in a rural district of South Africa (Lurie et al. 2003) succeeded in separating this “inside” pathway from infections outside a regular partnership by sampling 168 couples and

recording for each of them, in addition to the variable “migrant”, *which* of the two partners was positive or negative. Strictly speaking, this was a community study, the “communities” being couples. There were only 9% “positively concordant” couples but 21% “discordant” ones, and in 29% of discordant couples it was the female partner who was infected. Migrant couples were more likely than non-migrant ones to have at least one partner infected and to be discordant.

6.8 Community Studies

It is natural that questions about pathways and causal relations should be treated, and perhaps even answered, via community studies. Here, the key problem that poses itself right in the beginning is how to *define*, in a given setting, the appropriate exposure variables in a rigorous and quantitative manner. We are going to present three recent community studies on risk factors for tuberculosis mortality or incidence that illustrate basic and methodological aspects, and also provide a good introduction to the literature. They differ in the definitions of outcomes and risk factors as well as in the statistical methods used for evaluating the data.

Recall first that the units of community studies are groups of people. The first study (Ferreira Antunes and Alves Waldman 2001) and the second one (Kistemann et al. 2002) define outcomes and exposures in such a group essentially as averages of the corresponding variables for the members of the group whereas the third one (Acevedo-Garcia 2001) deals with a more sophisticated concept derived from the *distribution* of risk factors *within* the group. The first two studies employ classical statistical procedures, whereas the third one takes a Boolean approach.

The units of the study Ferreira Antunes and Alves Waldman (2001) are the 96 districts of the Brazilian Metropolis, São Paulo. The outcome variables are defined as suitably standardized annual mortality rates by tuberculosis for the years 1994–1998. One of the main purposes was to estimate the percentage of tuberculosis deaths attributable to a HIV co-infection; the result was 22%. In addition, the analysis revealed the following associations of tuberculosis mortality with social risk factors: a certain social development index (not to be made precise here): negative association; mean number of dwellers per bedroom: positive; mean number of dwellers per dwelling: no association; population density: no association; mean number of rooms per household: negative; recent immigration from abroad or other Brazilian states: positive. The associations are defined by a simultaneous autoregressive analysis that takes the spatial distribution of the districts into account. The authors also suggest some interpretations of these results in terms of possible pathways. For instance, the positive association of tuberculosis deaths with bedroom overcrowding in contrast to the absence of association with district-level overcrowding may indicate that prolonged contact is needed for disease transmission, and the negative correlation with the household size is consistent with respiratory transmission. Immigrants may contribute to higher tuberculosis mortality both because they bring along relatively more new sources of transmission than there are in the population already in place, and because their economic and social status is lower.

The article by Kistemann et al. (2002) on tuberculosis incidence in the large German city of Cologne pursues similar purposes and uses similar methods. Its list of social risk factors is more detailed, though, and hence there is a more refined discussion of possible pathways and causal relations. Regarding immigration, it is interesting to note that deprivation of immigrants already settled rather than high prevalence at the moment of arrival seems to be the decisive factor. In general settings, material deprivation acts both through poor housing conditions and the associated increased exposure to the infective agent, and by lowering immunocompetence, but in Cologne where crowded housing is not a severe problem the first factor contributes relatively little.

In the study by Acevedo-Garcia (2001) a unit is determined by a common ZIP (postal) code in the state of New Jersey, USA, and an ethnic group (non-Hispanic whites, African-Americans, Hispanics, Asians), which amounts to $591 \times 4 = 2,364$ units. All of them are included in the study; thus formally, as in the two papers discussed before, we have a census-like cross-sectional survey. Only one outcome variable is investigated, its value for a given ZIP code and a given ethnic group being defined as the average annual tuberculosis incidence over the years 1985–1992 in this “unit”.

The essential idea of the concept of risk factors on the level of these units is to take into account the distribution of risks within the ZIP code area in question. To this end, the partition of the ZIP area into (street-) blocks is used. For example, in order to define the exposure to *poverty* in the ZIP area 08544 among Asians, consider, in each block in that ZIP area, the proportion of poor people, suitably determined. By exposure to poverty in that unit the author means, then, the *average*, or *expected*, value of this proportion if we select a block at random from the ZIP area 08544, the probability of selecting a given block being the proportion of Asians living in this block to all Asians in the whole ZIP area 08544. Exposure to dilapidated (i.e. rented) and to overcrowded housing is defined analogously. In the paper (Acevedo-Garcia 2000) it is argued that these factors may have an *indirect* effect on the transmission of tuberculosis. Aspects of *segregation* that may act *directly* are described by factors that are defined along the same lines: isolation (contact with members of the same ethnic group), contact with immigrants and concentration (partial and total density, e.g. per square kilometre).

As the author remarks, an epidemiologic study can be analysed in two ways. The first, usual, route is regression, which means starting from the exposure variables and finding their relative contribution to the outcomes. The second method, which she employs here, focuses on the outcomes first and tries to identify *configurations* of risk factors that favour a given outcome. Outcomes are categorized as *no*, *low*, *high* and *very high* tuberculosis incidence. Risk factors are dichotomized in order to define configurations as in Boolean algebra. For each outcome category and each ethnic group, the frequency of ZIP areas that display a certain configuration among all ZIP areas in the same outcome category and ethnic group is given for several important configurations. This allows comparisons between outcome categories and reveals in some cases significant differences, too lengthy to be described here.

In a similar vein, the cross-sectional study by (Tellez et al. 2006) involves a blend of individual-level and community-level risk factors. The outcome is caries severity. From the start, only African-American families in Detroit, MI, USA who live in neighbourhood clusters of essentially the same low-income status are considered. The study thus focuses on social factors other than poverty. After accounting for individual characteristics of persons, there is still significant variation between clusters. In particular, caries severity increases with a higher number of grocery stores and decreases with a higher number of churches.

We close this section with an example of a community study of a very different kind, namely by mathematical modelling (see Chapter 12). For a change, this work showed that a particular behaviour of an outcome variable was *not* the result of the influence of a certain social factor contrary to what had been suspected. Mosse and Tugendreich (1913) quote a study from Denmark that described a periodicity of the incidence of syphilis with a period of around 16 years and suggest that this might be due to a corresponding periodicity of socio-economic factors. Similar periodicities were observed later in other countries too. This led to a long controversy until Grassly et al. (2005) showed that the underlying cause was in fact the dynamics of building up immunities within the population.

6.9 Surveys on the Literature

In the year 2004, two expository articles appeared that might guide the reader further through the meanders in our field. The first one (Myer et al. 2004) although restricted to South Africa, is particularly interesting and useful by presenting the history, by referring to concrete settings and by critically analysing the basic issues. It concerns non-infectious as well as infectious diseases; however, given the context of South Africa, the latter play indeed the main role.

The survey by Poundstone et al. (2004) covers only HIV infections but mentions most facets of modern social epidemiology of infectious diseases in general. In particular, it reviews attempts to establish categories of social determinants. Thus, one might distinguish *individual*, *social* and *structural* level factors. Social-level factors fall into one of the categories: *cultural context*, *social networks*, *neighbourhood effects* and *social capital*. However, it is not always clear for which units of study they are defined.

In contrast to these two surveys, the purpose of the article by Justin et al. (2007) is not to provide an overview, nor to evaluate or analyse. It counts and compares the number of papers or citations devoted to such and such subject during such and such period between 1966 and 2005 as found by various search strategies. There is even a *p*-value for a hypothesis of the type “the number of citations has increased”!

6.10 Discussion

The idea that social factors and in particular social injustices influence the state of health is, for infectious diseases, very old and particularly prominent. However, it

is precisely for infectious diseases that it cannot easily be formulated in exact terms and supported by correct studies. The reason may be found in the complex dual aetiology of infectious diseases. On the one hand, there is the main, and necessary, factor, which is the infection by the pathogen. Within and between given populations, it may follow complicated ways of transmission. On the other hand, there are many more factors including social ones, both on a person and a population level, and they interact with each other in addition to influencing the process of infecting individual persons and the circulation of pathogens in populations. Social factors are tied to environmental, lifestyle and iatrogenic ones. Moreover, behaviour is to some extent conditioned by genetic structures as we are discerning now in a more and more precise manner. Some of these many determinants may also act directly on certain infectious diseases. Thus it is not easy to isolate the action of a given social factor and to eliminate numerous possible confounders.

We have seen that on the eve of the First World War the study of social risk factors for infectious diseases using simple person-based concepts and models had already made much progress. It placed the individual into the centre of interest; typical risk factors were income, profession and housing. Population-based notions appeared only in very elementary forms, and mostly derived from person-based ones.

A fresh impetus came only much later, partly under the influence of work on non-infectious diseases (Cassel 1976; Marmot et al. 1991; Marmot and Wilkinson 1999; Chandola and Marmot 2005). In some countries, research in this area had also been hampered by political pressure when a field like social epidemiology was suspicious and notions like social class and social injustice amounted to heresy; see Leibfried and Tennstedt (1980) for Germany during the Nazi regime and Krieger (1994) for the United States during the McCarthy era.

The fundamental idea underlying all of modern epidemiology of investigating the influence of factors by comparing the outcomes for different levels, or values, of these factors is still valid. However, the emphasis has now shifted to social factors of a much more complex structure, belonging, for example, to the categories *social networks*, *social environments* and *social capital*; see Badura (2006) for a survey. They are mostly defined for populations and hard to quantify. It is not easy to translate ideas on the pathways along which they act alongside the transmission mechanism into hypotheses that can be tested. Mathematical modelling of the flow of infections in relation to social factors is also difficult. In this area, most of the work is still to be done; the studies that we have presented are just first attempts.

6.11 Implications for Public Health Policy

The general conclusions to be drawn from observations on social determinants of diseases, and in particular infectious ones, are old, and may nowadays appear almost obvious. They amount to changing the social environment, eliminating absolute and reducing relative poverty and promoting education for the less well-to-do. In Great Britain, the Chadwick report of 1834 led to the “Poor Law Amendment

Act” (Rosen 1993, p. 176). An even more outspoken report of the Poor Law Commission appeared in 1842 (Rosen 1993, p. 187). In Germany, Virchow’s conclusions (Virchow 1848) were quite explicit and insisted in particular on the disastrous effects of the lack of instruction, which he attributed in part to the Catholic Church. In the United States, the City Inspector of the New York Board of Health, John C. Griscom, published a study in 1845 on the sanitary conditions of the “labouring population” as factors that should be changed in order to improve the health situation (Rosen 1993, p. 213).

However, alleviating poverty and raising the level of education depend on political willingness and is everywhere a slow process with many ups and downs even where it is explicitly declared a goal to be attained. Thus, in the first place, progress in fighting infectious diseases has come from public hygiene, in particular sewage disposal, clean water supplies, and food control, and from vaccination programmes. The social factor “instruction” played an indirect role in the form of health education, which led to better personal hygiene. To some extent, the studies mentioned above also contributed to exerting pressure for eliminating the worst excesses in crowded and unhealthy living quarters. Later on, the general improvement of living standards such as housing, nutrition and working conditions certainly played a role in reducing morbidity and mortality by infectious diseases although it is hardly possible to quantify this effect.

Implications of insights into the action of more *specific* social factors are a matter of the recent past and sparse. They concern, for example, *screening* and *prevention*, and here some studies have led to actions that had not been obvious from the beginning. This is particularly true for AIDS. Thus, the study by Lurie et al. (2003) described in Section 6.7 gave rise to the recommendation to extend preventive measures to the wives of migrant workers. The report on trends of AIDS incidence and prevalence in Uganda (Green et al. 2006) discusses and compares general preventive strategies that had been influenced by the knowledge of the action of social factors. The studies on social risk factors for tuberculosis described in Section 6.8 also lend themselves to applications both to surveillance and prevention. Thus Ferreira Antunes and Alves Waldman (2001) deduce from their results a reduced effectiveness of certain existing control programmes.

References

- Acevedo-Garcia, D. (2000) Residential segregation and the epidemiology of infectious diseases. *Soc. Sci. Med.* 51, 1143–1161
- Acevedo-Garcia, D. (2001) Zip code-level risk factors for tuberculosis: Neighborhood environment and residential segregation in New Jersey, 1985–1992. *Am. J. Public Health* 91, 734–741
- Badura, B. (2006) Social capital, social inequality, and the healthy organization. In: Noack, H. and Kahr-Gottlieb, D. (eds.), *Promoting the public’s health. The EUPHA 2005 conference book*, Verlag für Gesundheitsförderung, Gamburg, 53–60
- Bourdieu, P. (1979) *La distinction*. Editions de Minuit, Paris
- Cassel, J. (1976) The contribution of the social environment to host resistance. *Am. J. Epidemiol.* 104, 107–123

- Chandola, T. and Marmot, M. (2005) Social epidemiology. In: Ahrens, W. and Pigeot, I. (eds.), *Handbook of Epidemiology*. Springer, Berlin Heidelberg, 893–916
- Cox, D. and Wermuth, N. (1996) *Multivariate Dependencies: Models, Analysis and Interpretation*. Chapman & Hall, London
- Dony, J.F., Ahmad, J. and Khen Tiong, Y. (2004) Epidemiology of tuberculosis and leprosy, Sabah, Malaysia. *Tuberc. (Edinb.)* 84 (1–2), 8–18
- Ferreira Antunes, J.L. and Alves Waldman, E. (2001) The impact of AIDS, immigration and housing overcrowding on tuberculosis deaths in Sao Paulo, Brazil, 1994–1998. *Soc. Sci. Med.* 52, 1071–1080
- Grassly, N.C., Fraser, F. and Garnett, G.P. (2005) Host immunity and synchronized epidemics of syphilis across the United States. *Nature* 433, 417–421
- Green, E.C., Halperin, D.T., Nantulya V. and Hogle, J.A. (2006) Uganda's HIV prevention success: The role of sexual behavior change and the national response. *AIDS Behav.* 10 (4), 335–346
- Hobdell, M.H., Oliveira, E.R., Bautista, R., Myburgh, N.G., Lalloo, R., Narendran, S. and Johnson, N.W. (2003) Oral diseases and socio-economic status (SES). *Br. Dent. J.* 194 (2), 91–96
- Justin, M. C., Wilson, M.L. and Allison, E.A. (2007) Analysis of social epidemiology research on infectious diseases: historical patterns and future opportunities. *J. Epidemiol Community Health* 61, 1021–1027
- Kark, S.A. (1949) The social pathology of syphilis in Africans. *S. Afr. Med. J.* 23, 77–84
- Kistemann, T.h., Munzinger, A. and Dangendorf, F. (2002) Spatial patterns of tuberculosis incidence in Cologne (Germany). *Soc. Sci. Med.* 55, 7–19
- Krickeberg, K. (2007) Principles of health information systems in developing countries. *Health Inf. Manag. J.* 36 (3), 8–20
- Krieger, N. (1994) Epidemiology and the web of causation: Has anyone seen the spider? *Soc. Sci. Med.* 39, 887–903
- Krieger, N. (2001) Theories for social epidemiology in the 21st century: an ecosocial perspective. *Internat. J. Epidemiol.* 30, 668–677
- Leibfried, S. and Tennstedt, F. (1980). *Berufsverbote und Sozialpolitik 1933. Die Auswirkungen der nationalsozialistischen Machtergreifung auf die Krankenkassenverwaltung und die Kassenärzte*. 2nd. Ed. Series: Arbeitspapiere des Forschungsschwerpunktes Reproduktionsrisiken, soziale Bewegungen und Sozialpolitik, University of Bremen, Bremen
- Levy, A., Fraser, D., Vardi, H. and Dagan, R. (1998) Hospitalizations for infectious diseases in Jewish and Bedouin children in southern Israel. *Eur J Epidemiol* 14 (2), 179–186
- Lurie, M., Williams, B.G., Zuma, K.D.K., Mkaya-Mwamburi, D. et al. (2003) Who infects whom? HIV-1 concordance and discordance among migrant and non-migrant couples in South Africa. *AIDS* 17 (15), 2245–2252
- Marmot, M.G., Smith, G.D., Stansfeld, S., Patel, C., North, F., Head, J., et al. (1991) Health inequalities among British civil servants: the Whitehall II study. *Lancet* 337(8754), 1387–1393
- Marmot, M.G. and Wilkinson, R.G. (1999) *Social Determinants of Health*. Oxford University Press, Oxford
- Mosse, M. and Tugendreich, G. (eds.), (1913) *Krankheit und soziale Lage (Disease and social condition)*. Lehmanns, München. Reprinted 1981 *WiSo Med*, Göttingen, 3rd Ed.
- Murray, C.J.L., Lopez, A.D., and Mathers, C.D. (2004) *The global epidemiology of infectious diseases*. WHO global burden of disease and injury series 1, WHO, Geneva
- Myer, L., Ehrlich, R.I. and Susser, E.S. (2004) Social epidemiology in South Africa. *Epidemiol. Rev.* 26, 112–123
- Dinh P.N., Long H.T., Tien N.T.K., Hien N.T., Mai Le T.Q., Phong Le H., Tuan Le V., Van Tan H., Nguyen N.B., Van Tu P. and Phuong N.T.M. (2006) Risk factors for human infection with avian influenza A H5N1, Vietnam, 2004. *Emerg. Infect. Dis.* 12 (12), 1841–1847
- Poundstone, K.E., Strathdee, S.A. and Celentano, D.D. (2004) The social epidemiology of human immunodeficiency virus/Acquired immune deficiency syndrome. *Epidemiol. Rev.* 26, 22–35

- Rosen, G. (1993) A history of public health. Expanded Edition. The John Hopkins Press, London
- Susser, M. (2001) Glossary: causality in public health science. *J. Epidemiol. Community Health* 55, 376–378
- Tellez, M., Sohn, W., Burt, B.A. and Ismail, A.I. (2006) Assessment of the relationship between neighborhood characteristics and dental caries severity among low-income African-Americans: a multilevel approach. *J. Public Health Dent.* 66 (1), 30–36
- Virchow, R. (1848) Mittheilungen über die in Oberschlesien herrschende Typhusepidemie (Informations on the present typhoid fever epidemic in Upper Silesia). *Archiv für pathologische Anatomie und Physiologie und für klinische Medizin* II (1–2)
- Zahraoui-Mehadji, M., Baakrim, M.Z., Laraqui, S., Laraqui, O., El Kabouss, Y., Verger, C., Caubet, A. and Laraqui, C.H. (2004) Infectious risks associated with blood exposure for traditional barbers and their customers in Morocco [in French]. *Santé* 14 (4), 211–216