LEADING ARTICLE

Epidemiology for Prevention

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This paper illustrates the basis of, expectations for and evaluation of prevention from an epidemiological perspective. Specifically, the extent to which epidemiologists could and should be involved in designing and evaluating public health interventions is addressed. Changes in the view on disease causation and epidemiology's role for the future of public health are discussed. Examples from cardiovascular epidemiology are used to illustrate the ever more complex, but still incomplete, knowledge on which prevention is based. A few current examples illustrate paradoxes where health information must balance academic discord. Methodological problems in the evaluation of intervention studies often fail to live up to the expectations of prevention. Outcome indicators of preventive projects must be developed and traditional appraisals of effects be supplemented with process analyses using both quantitative and qualitative methods. Social conditions for prevention are discussed and areas for further research are suggested.

FROM DESCRIPTION AND OBSERVATION TO ACTION AND EVALUATION

Epidemiology includes routine surveillance and research strategies to test hypotheses of the causes of diseases, measurements of risks to health, and evaluation of preventive, diagnostic and therapeutic programmes or technologies. Epidemiology is created with and by people and its results affect people.

Lilienfeld has described how the definition of epidemiology changed during the early part of this century. He emphasized that the subject had matured to a discipline but that it was still understood by many to be the science of epidemics. A common feature in definitions was the emphasis on "the distribution of", meaning essentially the statistical distribution, not the more emotive social view of distribution that is used in modern social epidemiology. From having been closely associated with working for change, epidemiology has, during the 1980s, also been "modernized" with the common aim of measuring the risks of disease. Susser suggests that this change in emphasis is unfortunate and has occurred at the price of social understanding, with the risk that any knowledge brought to prevention will be fragmentary and mechanical.

Epidemiology gathers its methodology from a number of fields both within and outside the traditional medical field, but it appears historically to be nearer to social science. As epidemiology focuses on the distribution of ill-health as well as social determinants it is not purely an observational discipline but an actor as well. The epidemiologist also has a specific responsibility to inform, even outside the scientific community.

Epidemiological strategy is to observe and compare, rarely to experiment. Initially, the epidemiologist seeks to evaluate the statistical association between "exposure" and "disease". This requires a time course, from today's exposure to future disease, from the appearance of disease to previous exposure or, historically, from yesterday's exposure to today's disease. In the next phase, the biological and social consequences of these relationships must be analysed. Data do not contain their own knowledge. Technology without theory makes the epidemiological landscape more a caricature than a portrait.

Wegman has discussed epidemiology's place in prevention and he predicts a division between the discipline of epidemiology and public health work; a generation gap that has become apparent in that those who gather data for preventive programmes are now rarely the same people who are responsible for these programmes. Epidemiology's central role in public health work has also been questioned because of its strongly positivistic view of causality, which has produced too deterministic explanatory models, invalidating the analysis of dynamic occurrences and human behaviour.

During the era of infectious diseases the dogma of "one illness, one cause" prevailed and a shift in the balance between agent, host and environment was considered to be able to trigger disease. When Henle and Koch formulated their postulates for the identification
of ‘the cause’ of infectious diseases in 1882 they required that the suspected ‘parasite’ must be present in every case of the disease, but should be absent in other diseases. This doctrine did not explain why certain individuals were more susceptible to disease or analyse the causes of starvation, undernourishment or occupational disease during industrialization.

As infectious diseases began to be replaced in industrialized societies by chronic diseases and society became more urbanized during the early part of this century, people’s ‘behaviour’ became more disease-producing and it became increasingly more obvious that diseases could have ‘several causes’. The completely industrialized society recognized the ‘same cause’ as producing several diseases and an intensive discussion ensued about the terms ‘sick’ and ‘well’ and what effects health care has really had on public health. The circle has now closed: while poverty-related diseases still dominate the global health scene, infectious disease epidemiology has revived in the wake of the HIV pandemic. Never previously has the research society mustered its resources as has occurred to meet this threat, possibly allied to the fact that the threat endangers both rich and poor.

The hegemony created by the germ theory meant that bacterial causes were sought for all disease and attempts were made to find the responsible organism causing all diseases whose aetiology was hitherto unknown. Microbiology became the most important discipline in medicine and the prevention of infectious diseases came to be the ‘first epidemiological revolution’ in public health. Social medicine in England in the 1940s marked the beginning of the ‘second epidemiological revolution’ with its patterns of chronic diseases. It also meant a revival of the sociological school that had flourished under Virchow 100 years earlier. Awareness of the causes of these diseases in the community generated an intellectual climate in which community medicine was born together with the insight that chronic diseases could also be prevented. Implementation of the second epidemiological revolution would, according to Terris, necessitate radical changes in the agricultural and industrial sectors, with reductions in the production of tobacco, alcohol, animal fat products and drugs. Effective prevention of injuries also required unpopular invasion of people’s daily living.

It is unlikely that such obvious statistically detectable risks to public health, for instance, smoking and occupational factors, such as asbestos and radon, will be identified in the future. Despite the numerical power of these risks it required a long series of corroboratory results before the research community reached a consensus. Prevention has still not been able to handle the consequences of this agreement. In order to discover much weaker associations between risk factors and disease, future research will need much more sensitive instruments. Molecular epidemiology represents a potential link between epidemiological observational studies and experimental research with greater ‘power of resolution’ than present register epidemiology. A corresponding modulation of the social causality is indicated and social epidemiology’s weapon is theory.

Weak associations, however, are not necessarily less important from a public health point of view. A factor’s importance is a function both of its potential for damage and its prevalence. Despite the fact that an epidemiological association may have considerable importance for illness in a population, it may have only marginal relevance for disease in the individual. The risk of dying of cardiovascular disease over a 10-year period has been assessed to be 4.9% in middle-aged men whose serum cholesterol levels are over 6.2 mmol/l compared with 1.7% in those with a serum cholesterol level less than 5.2 mmol/l—a difference of approximately 3%. However, this difference may fail to convince an otherwise healthy man of the need to alter his dietary habits.

Vandenbroucke divided epidemiology’s developmental phases into two wave movements of vocational epidemiology. The second wave is, in his opinion, currently ebbing out in favour of a transition towards a technical-methodological epidemiology. Social and preventive medicine appear to have been surpassed by molecular biology and the new receptor pharmacology. Vandenbroucke suggests that many of today’s social epidemiologists, like the 19th century miasmatics, work with a ‘black box’ view of epidemiology in which environment in its widest sense produces disease and in which prevention does not presuppose knowledge of pathogenetic mechanisms. These epidemiologists will lose the battle against the molecular biologists in the same way that miasmatics lost to the bacteriologists. However, Loomis and Wing suggest that the basis for this view is unsound. Bacteriologists won because they were right. They had found the cause of the disease. The challenge for the epidemiologists of the next century will be to develop theories and models that view the causes of disease not as discrete characteristics of the individual but rather as both arising within, and expressing complex systems that not only explain observations but even lay the foundations for practical intervention. Krieger in her search for the spider in the web of causation also calls for a comprehensive epidemiological theory, an ecosocial framework to better integrate biological and social understandings, and to combat the view of epidemiology as a collection of
methods. Loomis and Wing would like to see epidemiology change from a reductional working philosophy, a challenge that the public health consequences of the current global environmental, demographic and social changes require to be taken up. The development of sociological theories may be able to contribute more refined measurements of social stratification and models that can better predict disease on the basis of social support, work demands, control and rewards. According to Susser epidemiologists must bridge the gap between the manifestations of disease, on the one hand, and viruses, molecules and genes on the other. They must also bridge the gap between social behaviour, political structure and economic power. Only then will we be able to understand and effectively prevent disease.

CAN PREVENTION WITHSTAND SCIENTIFIC DISSENSION?
There are rarely unambiguous or clear-cut foundations for taking measures; even a lack of action may be interpreted as a decision. Despite the fact that the pathophysiology of cardiovascular disease, for example, is not completely known, it can still be controlled. The fact that epidemiological research, in spite of its widespread general interest, sometimes has only a limited impact is an enigma. Has not epidemiology presented enough information on the negative effects of smoking? Is there still insufficient evidence for the role of saturated fats in the causation of arteriosclerosis? Have epidemiologists only drawn the biological conclusions of their research rather than the social, economical and political consequences? Has research been steered towards easily quantified areas under the illusion of being able to solve multifactorial health problems?

However, society, by its very nature, is a conservative and shock-absorbing system and since the ever more complex epidemiological foundation, more than with most other branches of science, is of immediate interest to the non-scientific community, it seemingly invites several alternative explanations.

During the 1980s there was a marked increase in synthesis methods, as a possible effect of the fact that the sheer amount of research had become impossible for users to survey. The classical literature review was replaced by consensus conferences, 'state-of-the-art' meetings, delphi methods and meta-analyses. One may wonder, however, if these methods do not oversimplify more than they synthesize, and whether the emphasis on consensus can result in contradictory results or anomalies that may all be rejected all too easily as being exceptions to the rule. To what degree does this reflect a desire to give legitimacy to established truth and practice?

An epidemiological association does not necessarily imply a causal association. Also, ecological associations are not necessarily consistent with associations at the individual level. These limitations are illustrated by the French paradox: despite high dietary intake of saturated fats, and the consequently relatively high serum cholesterol levels, cardiovascular mortality in France more closely resembles that in China and Japan, than that in northern Europe. The blood pressure levels, smoking habits and body weight of Frenchmen do not differ from those of people in other western European countries. The moderate but regular amounts of wine drunk by the French have been suggested to reduce the risk of heart disease by 40%. This epidemiological association has, however, also been claimed to be an artifact caused by those already ill stopping drinking alcohol. The scientific nucleus of the debate is based on the 'total consumption model's' credibility, that is, whether the level of the accumulated alcohol consumption is (causally) related to alcohol-related injuries, both medical and social, in society. The controversy is fuelled by the demonstration of a U-shaped curve of the association between alcohol consumption and cardiovascular mortality, showing that both teetotallers and alcoholics have an increased risk. The problem thus illustrates the conflict between population-based preventive strategy and a high-risk strategy, as well as the question of whether solidarity is a part of prevention. How should health policy balance a risk factor with both positive and negative effects? And can community-oriented prevention be directed to certain social groups without causing stigmatization? Is speculation about a known risk factor's possible positive effects 'permissible'? An inverse relationship has been shown by studies that suggest that low cholesterol levels are associated with an increased mortality from other causes than cardiovascular diseases. This sort of relationship, if it is causal, invalidates population-based attempts to reduce cholesterol levels in the population. The fact that increased cholesterol levels do not appear to be a risk factor in women is similarly controversial and has resulted in discussions about whether conclusions from epidemiological studies in men can be applied to women.

Medical technology is primarily assessed with respect to its efficacy and risk of causing harm. Recently, even the costs of the use of technology as well as the ethical and social consequences of its use have begun to be considered. However, few established technologies have been tested using all these criteria. There is indeed
no general agreement as to which criteria of usefulness and injury should be applied. Aims may thus be to prolong life, to reduce suffering or to improve the quality of life. These aims may also conflict with one another and differ depending on who has specified them, patients or professional assessors.

What burden of proof should be required of prevention programmes that are aimed at the whole population, such as health examinations with measurement of serum cholesterol, or blood pressure screening? Should standards be raised as these measures are aimed at healthy and asymptomatic individuals? How should the usefulness of these interventions be balanced against possible negative effects, and whose criteria should be applied? How should the costs of interventions made in the name of improved health be measured, and do they justify the means? Can we afford them and have we adequate competence to act on the consequences?

Intervention studies are needed to be certain that a change in the level of a risk factor affects the number of cases of disease in a population. The debate on cholesterol’s importance shows that not even studies such as these are interpreted similarly. Holme analysed 19 randomized clinical trials and estimated that for each 1% reduction in cholesterol level there was a reduction in the incidence of myocardial infarction of 2.5%. However, the positive effects found in the majority of these studies have been balanced by an increased risk of death from other causes (including accidents and suicide). Some studies have even reported an increased mortality and low cholesterol levels were suggested to predispose to aggressive behaviour, although this was later disproved. Ravnskov has suggested that a cholesterol myth has been created in the medical literature by the fact that studies showing a positive effect of reduction in serum cholesterol levels are more readily published than negative studies, and they are cited more often as well.

There is a considerable hiatus between the epidemiological observations of relationships between risk factors and cardiovascular disease, on the one hand, and the scanty documentation of the effects of intervention on the other. Even when the association with many risk factors has been confirmed, their importance in the population may not be as evident, either because only a limited proportion of the population carries the risk factor, or because its prevalence in the population is not known with certainty.

EXPECTATIONS OF PREVENTION
The importance of different factors is usually assessed in terms of relative risks as incidence ratios. If one wishes to reflect risk factors as a public health problem, then both their potential for causing damage and their prevalence must be considered. This is often expressed in terms of attributable risks (aetiological fractions), which represent that proportion of the total number of cases of a disease in the population that can be ‘explained’ by a certain risk factor. This thus also suggests what proportion of ill-health that (theoretically) can be prevented if the risk factor (the cause) can be eliminated. Analogous to, for instance, a vaccine trial we can estimate the ‘efficacy’ of a preventive programme. Unrealistic expectations are an enemy of such programmes. Measuring their potential impact should therefore be part of planning for interventions. Aetiological fractions may also be useful in the interpretation of changes that are developing in risk factor patterns and mortality. Analysis of the decline in cardiovascular mortality in the Minnesota Heart Survey, reported by Sprafka et al. for example, showed that about 20% of the observed decline in mortality between 1973 and 1986 could be ascribed to changes in known risk factors calculated from Framingham data.

Collins et al. suggest that the importance of known risk factors is probably underestimated. They reviewed the methods used in the nine larger prospective cardiovascular epidemiological trials including a total of 420 000 subjects and showed, using the risk factor blood pressure as an example, that 1) there does not appear to be a threshold level below which the risk level is constant, and 2) that many prospective studies are marred by a ‘regression-dilution’ bias. This arises because the initial risk factor measurements (usually single values) have a considerable variation in random and measurement errors. Thus there is a disproportionate number of subjects among those with the lowest initial risk factor levels who ‘happened’ to have lower values than usual, as well as a disproportionate number of subjects among those with high levels who initially had ‘excessively’ high levels. This causes a systematic underestimation of the regression effect (slope) and therefore of the risk factor’s importance. By the use of repeated blood pressure measurements from the Framingham study, they found that blood pressure’s importance as a risk factor was underestimated by approximately 60%. They suggest that this also applies to cholesterol and smoking.

The three traditional risk factors, hypertension, smoking and hypercholesterolaemia together explain only 20% of the variation in death from myocardial infarction in men between the populations that are included in WHO’s MONICA project. They explain 27% of the variation in women. Thus other factors that are of importance for the risk of developing cardiovascular
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1981, 246 possible 'risk factors' for cardiovascular dis-

dease had been put forward, and McCormick and

Skrabanek claimed that a multifactorial aetiology gen-

erally meant that the aetiology was 'unknown' and they

suggested the term 'risk marker' in order to avoid
giving the impression of unrealistic prospects of prevention. 28

The three major risk factors may, however, explain a

much larger proportion of the differences between

individuals in the risk of developing, for example, a

myocardial infarct. It is not unusual for a single sub-

tject to have more than one risk factor. Data from the

MONICA study in northern Sweden show that just over

one-third of the population aged 25–64 years have none

of the three major risk factors, while approximately

one-quarter have two or three. When the fact that risk

factors are thought to act synergistically is consid-

ered, 29 about 60% of the myocardial infarcts may be

explained by these three risk factors together. The

majority of this aetiological fraction originates from

these factors' synergistic effects. Analysis of inter-

actions is therefore necessary to understand better their

biological modus operandi as well as to be able to make

intervention more effective. Preventive programmes have

the greatest chance of success if several risk factors are

influenced simultaneously.

A further problem in interpreting the results of inter-

vention studies arises when the study group is selected

according to the variable that is simultaneously the out-

come measure, e.g. when evaluating change in a high-

risk group. A subject who visits his doctor when he is

not feeling well, commonly feels much better a week or

so later, but he does not necessarily owe it to the doctor.

Regression to the mean occurs when results are ana-

lysed, in particular when a group of subjects with ex-

reme values of, say, serum cholesterol, that include a

considerable random variation, are followed-up at a

later date. This problem highlights the need for a con-

rol group, or when this is impossible, that a series of

initial values rather than a single measurement should

be used as the selection criteria. 19

The calculation of aetiological fractions may also

be useful in the planning and analysis of how realistic

expectations of results are likely to be (often too op-

timistic) in various trials of intervention. Gunning-

Schepers, for example, has simulated the effect of

various hypothetical attempts at intervention, and taken

the varying lag periods in the appearance of effects,

the natural trend in the patterns of risk factors and

the multifactorial influences into consideration. 30, 31

They suggest that it is extremely difficult to detect a

reduction in mortality that occurs in populations less

than 65 years of age and that the total effect will be

underestimated if premature death is used as the out-

come measure. As the lag period for the influence on

other diseases than cardiovascular diseases may be

longer, an underestimation of the total mortality will

result if the evaluation is performed too soon.

Prevention is usually aimed at large groups of sub-

jects and requires many to take part to the advantage of

relatively few. Just as it is important to inform indi-

vidual patients of a treatment's expected benefits and
dangers, ethics should be taken seriously when health

checks to discover disease in otherwise healthy people

are performed. This will help to maintain credibility.

Invitation to mammography examinations has been

shown to reduce the risk of death from breast cancer by

30%, despite the fact that women most at risk do not

take part. In the Swedish WE-study one death was

prevented by screening 4000 women, in whom 13.5

biopsies were performed and 7.4 cases of breast cancer

were detected. Similarly, the prevention of one case of

cervical cancer has been estimated to require 400 000

tests and 200 biopsies. It is therefore essential that

subjects are informed of the value of such examinations

in absolute, rather than relative terms. 32

DOES HEALTH EQUALITY CALL FOR
INEQUALITY IN PREVENTION?

Clarifying social- and gender-related stratification is

one of public health's pivotal tasks. In the field of car-

diovascular diseases, for example, a transition has oc-

curred in recent decades in which, from being diseases

that predominantly affect white collar workers they

now mainly affect blue collar workers. 33–35 In the

USA, 35 Finland 36 and Sweden 33 increasing stratifica-
tion has occurred parallel to the declining trend in

mortality from cardiovascular disease. As the epidemic

of cardiovascular disease in the western world de-

veloped, it first affected the economically more privileged.

The better off were also the first to benefit from the

decline in mortality. The reduction in mortality was

seen earlier and more obviously in socially more priv-

ileged areas in 507 administrative units in the US

between 1968 and 1982. 37

Blane used 'lost years' to illustrate social differences

in an English material. 38 In 1971, in 1000 men aged

15–64, ischaemic heart disease caused 14 lost years in

the highest social class compared to 21 in the lowest.

Ten years later, the corresponding figures were 12 and

29 years. The total mortality in lost years declined over

the 10-year period from 48 to 37 years in the highest

social class, while it increased from 101 to 103 years
in the lowest social class. However, this development was not seen in women. Violence and accidents caused more lost years in the lowest social class than both cardiovascular disease and cancer. Prevention aimed at altering lifestyle in order to prevent chronic disease may therefore be more successful in the well-educated. Another English study similarly shows that, despite the fact that people in higher social classes live longer than working class people, they did not have more symptoms of disease during their last year of life, but rather a superior quality of life as well as better access to health care.39

Carr-Hill recently analysed the sources of error in studies of social inequality.40 He suggests, first, that many studies are based on occupation at the time of death. A longitudinal starting point needs to consider social mobility and selection. There has been a considerable increase in the number of professional occupations in people in higher social classes, while a number of manual occupations have disappeared. This has complicated the interpretation of time trends. Baker and Illsley studied life span and the pattern of causes of death during 1964 to 1984 and found that the social aetiology had changed from economic to behavioural as poverty-related diseases were replaced by consumption-related diseases, and that these first affected middle-aged men.41

Blaxter has also reported interesting associations between perceived health, social environment and lifestyle factors.42 She concluded that social circumstances, including social support, have greater relative importance for health than health behaviour such as smoking, exercise and dietary habits. However, the association between these risk behaviours appears stronger in better environments than in the socially vulnerable. Smoking, for example, does more extra damage, and not smoking has a greater advantage in those already privileged. Unhealthy behaviour apparently does not reinforce the damage as much as a healthy behaviour has extra benefit.

Similar synergies have obvious relevance to the design of preventive programmes and are also relevant when the increased social stratification of ill-health is being analysed. The centuries old gender difference in survival, with shorter life span in women, has altered to an apparently constant 6-year longer life from birth in women than in men. Several studies, however, suggest that these extra years that benefit women are generally years with some kind of handicap.43 The difference in life span between men and women is surprisingly constant in various countries in western Europe. After having been through the classical demographic and epidemiological phases the difference in life span between different industrialized countries has been considerably reduced while gender differences have simultaneously increased.44 Newer Swedish data suggest, however, that the differences between men and women have decreased. Whether this represents a temporary fluctuation or is an expression of the changing exposure and social conditions of women is a central question in public health. It may be that this is not completely unrelated to the 'paradox' that women report more ill-health and symptoms and perceive their health as being worse than men.44,45

There are also social differences in self-reported ill-health. Eighteen per cent of adult Swedish working class women report that they suffer from cardiovascular disease compared with only 10% of civil servants. The corresponding figures for men are 14% and 11%, respectively. Despite the fact that 40% of the adult Swedish population reports that it suffers from chronic ill-health or symptoms of ill-health, three-quarters report that their health is 'good'. Perceived health may thus reflect quality of life, but has simultaneously shown itself to be able to predict both mortality and handicap.46

One of public health's main concerns is thus coming to terms with the social stratification of many common diseases. MacIntyre suggests that a future challenge in public health will be to study the interaction between structural and cultural/individual explanations, as a unilateral focus on one or the other will cause a misdirection in prevention.47 Little is still known about how people on different social levels are able to take advantage of and assimilate health information in society, such as that which occurs via population-based preventive programmes that aim at altering lifestyle.

The official Swedish enquiry into the exercise of power in society (maktutredningen) emphasizes that it is mainly the well-educated and middle class which use and assimilate society's various offers. Whether or not this also applies to public health measures is not known. Increasing inequality could result if the effects of prevention depend on social conditions, and this would be an unfortunate effect of health politics. This special treatment of different social groups should not be construed as consciously discriminating against various educational or occupational groups but is a consequence of structural differences in treatment. This leads into a discussion of the premises for peoples' actions and the importance of 'cultural capital' for social stratification. Cultural capital includes a superior ability to gather information, i.e. an ability to keep oneself well informed about where in social interactions profits can be gathered. Social strata with little cultural capital are often too late in their efforts and miss their
opportunities. There is also an opposite theory that a general improvement in the level of education leads to a strengthening of the role of the citizen and the official Swedish enquiry into the exercise of power in society suggests that influencing public opinion is characterized by the best educated people initially distancing themselves, but that as time passes the differences between social classes narrow.48

EVALUATION PERSPECTIVES
Evaluation of larger, better-known programmes, such as those in cardiovascular disease, have traditionally been 'before and after' studies, sometimes with 'control populations' as a reference and with the clinical trial as their model. Mortality is often used as a measure of outcome. It seems, however, as pointed out by Fries et al.49 unreasonable to expect that differences in mortality would be seen in the short term. Several international studies also suggest that the reduced mortality from cardiovascular disease results from a reduction in the age-specific incidence rather than from an improved survival after diagnosis.50 Swedish data show that less than 20% of deaths from cardiovascular disease are preceded by clinically verified heart disease.51 Fries suggests that these findings indicate that primary prevention should be assessed according to measures of disease rather than mortality and illustrates this with data from four large intervention programmes. There was a total of 595 deaths in the intervention groups compared with 600 in the control groups. This should be compared with 2556 and 3116 serious conditions, respectively, in the two groups.52 Collins et al. have recently also pointed out that not even large meta-analyses have the statistical power to detect an effect on longevity, particularly in the studies of primary prevention. Prevention may influence the cause of death but will only marginally delay death.53 Preventive strategies, however, have in general been aimed at diseases that threaten life.46

The belief that alterations in lifestyle prolong life is probably a belief in the impossible, as ageing itself competes with other risks of disease. Tsevat et al. have estimated that in a population of 35-year-old American men, hypertension, smoking, raised serum cholesterol levels, and being overweight are responsible for 1.1, 0.8, 0.7 and 0.6 lost years.54 The corresponding figures for women are 0.4, 0.7, 0.8 and 0.4 years, respectively. A 35-year-old man or woman is estimated to win 2.3 and 2.8 years, respectively, by stopping smoking. If the mortality from cardiovascular disease could be eliminated, a 35-year-old American would be expected to add a further 3.2 years to his life span.54

In a review of the measures of intervention, Erben et al. in the spirit of Antonovsky, stress the importance of positive indicators of health, subjective measures of health and measures of functional ability.55 Just as the aims of prevention should preferably be formulated in these terms, so should the consequences of preventive programmes be evaluated according to the same yardstick.

Evaluation research, in contrast to local development projects, aims to formulate and attempt to answer more general questions, e.g. in connection with performance and assessment of preventive programmes, their benefits and consequences. The study of events in a local community and understanding patterns of influence require not only studies of medical effects: even studies with process analytical basis in which all aspects of different prospective programmes are examined and in which quantitative and qualitative data complement each other.48 It is these means rather than the ends that process analysis focuses on.

CONCLUDING COMMENTS
This review has primarily attempted to discuss the conditions for prevention from the point of view of epidemiological evaluation and decision-making. Epidemiology has developed more from the perspective of disease than from that of public health. This is probably related to the fact that our experience of 'health' is episodes of disease affecting individuals rather than the state of health of the population. What the doctor is to the patient, the epidemiologist is to society and epidemiology is thus one of the foundations of public health sciences. Biological, as well as methodological knowledge is required, and these are seldom found in a single individual. Formulating problems, selecting methods, analysing and documenting data is a group process. While time consuming, it provides the necessary insight that public health research requires—input
from several sources. There is thus an epidemiological potential in prevention and there is a challenge in bringing the development of epidemiological theory and methods nearer to practical public health efforts. False expectations are however a major obstacle to prevention. With this as the starting point, a number of problem areas can be identified affecting an epidemiological way of reasoning:

- to afford greater importance to ‘structural’ epidemiology that analyses the interaction between society’s economic, political and ideological changes on the one hand and morbidity and mortality on the other.

This is a truly interdisciplinary undertaking in which the arsenal of epidemiological methods will need to be supplemented by the societal perspective of social research. A number of welfare political measures and procedures thus await evaluation: and epidemiology should take greater advantage of the abilities of natural experiments and avoid the retrospective discovery that the work was done too late.

In particular to analyse the consequences of community-oriented health work, its benefits and possible harm for public health.

The improvement of health and prevention of diseases are traditional social tasks and medicine has considerable experience of describing risks of disease. However, society’s well meaning health information has not been examined scientifically or critically. Similarly, as experiences from general educational programmes have shown themselves to benefit those already well-educated, it would appear that analogue consequences develop within the health area and that signs of increased social differences in ill-health may even be interpreted as expressions of the general structure of public health efforts.

To make better and more current analysis of the development of health in different geographical and social strata as a central role for epidemiological surveillance.

It should be obvious that the social dimension be included in epidemiological and health care research’s basic registers. The rapid development of geographical information systems may, if transferred to the health area, contribute to a more relevant epidemiological description of health problems and care requirements than can be provided by descriptive epidemiology based on administrative units.

to research the area of communication between those who generate public health knowledge and those who will use it.

Prevention in the form of well-intentioned health information may easily be appreciated by people in general as being over-protective and be felt to be manipulative. When complicated situations and uncertain causal mechanisms are simplified at the same time as conflicting scientific conclusions are broadcast in the mass media, confidence in public health will be affected. Therefore testing whether the general public can take more scientific bickering and professional self criticism should be an important task for research. In this connection, mass media’s examination and intermediary role should be analysed together with the forms for reporting on public health.

To develop theoretical and methodological measures of outcome in prevention that even consider the programme’s possible negative, ethical and health economical effects, as well as people’s assessment of various risks to health.

The criteria for success of preventive programmes must also be defined. Which measures of outcome are relevant for the various actors, politicians, the medical profession and the people? Which norms and values ought to be used? Can net effects sanction increases in differentiation between social groups as a result of prevention? How may advantages be related to disadvantages and may prevention’s cost effectiveness be put into operation?

To develop and evaluate models for the calculation of expected effects of various intervention strategies.

Before preventive programmes are planned it is important that they are based on realistic expectations. The expected effects vary considerably depending on the measures of outcome used. Computerized epidemiological models constructed on calculations of aetiological fractions have been used to simultaneously take competing causes of death, lag periods in effects and general time trends in development of disease into consideration. These models may provide an important tool in the dialogue between the researcher and the decision-maker.

To develop methods for evaluation of processes in preventive programmes.

Studies of medical effects are insufficient to explain events and to understand patterns of influence in the
local community. A process analytical starting point is needed which can examine the programme from all angles. The modulation of the synthesis of measures of outcome with democratic, structural and social effects requires the development of means to complement current quantitative epidemiology with qualitative methods.

As health is not a self-evident general aim of all sectors of society, community-based prevention will of necessity become political and perhaps public health endeavours in the future may even become election issues. Prevention is not solely a health campaign, a one-off health examination, a brochure or a television advertisement. It requires popular participation as well as informed consent. This mobilization is a condition for prevention. The boundaries of public health will thus be pushed back and preventing the final epidemic, the destruction of our planet by nuclear weapons, has been called the 'third epidemiological revolution'. Contemporary concerns, however, focus more on the overloading of Earth's natural system and the life-supporting ecological fabric, than on the possibilities of nuclear war.

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