Why study socioeconomic factors and cancer?

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The occurrence of cancer within a population can be studied at many different levels, including forms of social organization, the individual, a particular organ system, or a particular molecule. The causes of cancer can also be studied at these different levels, including socioeconomic factors, lifestyle, the organ burden of a carcinogen, or DNA adducts. Clearly, there are advantages in understanding disease causation at all of the different levels at which it operates. Although cancer risk factors such as tobacco smoke may appear to operate at the individual level, exposure may occur due to a wide range of political, economic and social factors; conversely, tobacco smoke ultimately also has effects at the cellular and molecular levels, including the production of mutations in DNA. Of course, it is important to gain information, and take action, at all possible levels, but the history of public health shows that changes at the population level are usually more fundamental and effective than changes at the individual level, even when a single risk factor accounts for most cases of disease. In this sense, a risk factor such as smoking can be regarded as a secondary symptom of deeper underlying features of the social and economic structure of society. Thus, just as a variety of health effects in various organ systems (for example, various types of cancer) may have a common contributing cause (for example, tobacco smoking) at the level of the individual, a variety of individual exposures (for example, smoking and diet) may have common socioeconomic causes at the population level. In many instances there is clear evidence that cancer is related to socioeconomic factors, but this does not appear to be fully explained by known risk factors. More importantly, there is little evidence as to which socioeconomic factors are of most importance, or whether it is the overall ‘package’ of social inequality that is responsible for the differences in cancer risk. The aim of this book is therefore to summarize what is already known, and to identify gaps in our knowledge.

Socioeconomic factors include education, income, assets, housing and occupation. ‘Social class’ can be used as a convenient summary term for various socioeconomic factors, but can also be used to denote more profound divisions within society.

The primary goal of public health is the prevention of disease in human populations, and socioeconomic factors are of major importance in this context. Epidemiology is the field of scientific investigation that attempts to discover the major causes of disease in the population so that public health action can be taken, although a great deal of epidemiological knowledge has been gained by researchers in related fields, particularly in the social sciences. For example, traditional definitions of epidemiology commonly refer to ‘the study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to control of health problems’ (Last, 1988), although some recent definitions of epidemiology ignore the population perspective (for example, Miettinen, 1985; Rothman, 1986).

Of course, most cancer epidemiologists know about the importance of social class and socioeconomic factors. In the first week of their epidemiological training they learn about the work of Virchow, Chadwick, Engels and others who exposed the appalling social conditions during the industrial revolution, and the work of Farr (1860) and others who developed methods of social class classification and revealed major socioeconomic differences in death rates in the nineteenth century. They also learn about the subsequent dramatic decline in infectious diseases that occurred before the development of modern pharmaceuticals and has been attributed to improvements in nutrition, sanitation and general living conditions (McKeown, 1979), although specific public health
interventions on factors such as urban congestion probably also played a major role (Szreter, 1988). They may also learn that there are now major social class differences in incidence and mortality from cancer and other chronic diseases, and that social class differences in mortality are increasing and are greater now (in relative terms) than they were in the nineteenth century (Marmot & McDowell, 1986; Pappas et al., 1993). After this, they usually forget about socioeconomic factors during the rest of their careers, except perhaps to occasionally adjust for social class in multivariate analyses of risk factors such as tobacco smoking, diet and other ‘lifestyle’ factors.

Nowadays, cancer epidemiologists typically study factors that have very low relative risks (for example, studies of new occupational carcinogens, or dietary studies of low levels of intake of micronutrients, usually reveal relative risks of less than 1.5) and that account for a small proportion of cancer cases. In contrast, in most industrialized countries, studies have repeatedly found strong associations between social class and cancer (Logan, 1982), with a nearly twofold relative risk for cancer when comparing the most disadvantaged group with the most advantaged group (although there are some specific cancer types for which the differential is in the opposite direction). However, few cancer epidemiologists study social class as an issue of major importance in itself, and it did not feature (except for a brief mention as a confounder) in the most comprehensive and authoritative review of the causes of cancer in the United States of America (Doll & Peto, 1981).

So why is social class so often just a footnote in cancer epidemiology studies?

The art of the possible
A related argument is that socioeconomic factors are ‘not easily modifiable’. Public health, like politics, can be viewed as ‘the art of the possible’ and socioeconomic factors are often placed into the ‘too hard’ basket. However, governments have repeatedly shown that social and economic differences are not ‘God-given’ but, for better or worse, are directly affected by government policies, often in unexpected ways (Black, 1993; Hewlett, 1993). Even when governments can have little effect on the overall gross national product (GNP), they can have major effects on how it is distributed by changes in the money supply, the level of inflation, the level of employment, the minimum wage and the average wage, taxation, and the level and availability of social services and social security benefits. There is some preliminary evidence that inequitable distribution of the GNP can have a more significant impact on overall national mortality rates than the actual level of GNP (Wilkinson, 1992, 1994). For example, in some countries, a large increase in GNP has been accompanied by little benefit in terms of health, whereas some relatively poor countries (for example, China, Jamaica and Costa Rica) have made major improvements in health care and life expectancy (Sen, 1980). Thus, the way in which the GNP is ‘shared’ is as important as its absolute level. Public health measures that aim to address the health problems of poverty may
ultimately find themselves in conflict with government policies (or may even have the ultimate policy impact of changing the government), but this does not make the role of socioeconomic factors any less important, or less worthy of study.

Single risk factors

However, perhaps the main reason why socioeconomic factors have received little attention in cancer epidemiology is that they do not appear to be ‘real causes’, or at least are not as straightforward as factors such as tobacco smoke. Modern epidemiology became widely recognized with the discovery of tobacco smoking as a cause of lung cancer in the early 1950s (for example, Doll & Hill, 1952) and subsequent decades have seen major discoveries relating to other causes of cancer such as asbestos, ionizing radiation, hepatitis B, and dietary factors. These epidemiological successes have in some cases led to successful preventive interventions without the need for major social or political change. For example, occupational carcinogens such as asbestos can (with some difficulty) be controlled through regulatory measures, and exposures to known occupational carcinogens have been reduced in industrialized countries in recent decades. Another example is the successful World Health Organization campaign for the elimination of smallpox (Tesh, 1988). More recently, some countries have passed legislation to restrict advertising of tobacco and smoking in public places and have adopted health promotion programmes aimed at changes in ‘lifestyle’.

These successes of ‘risk factor’ epidemiology have been striking and have undoubtedly prevented many cases of cancer. However, in recent years epidemiologists have struggled to find new major risk factors for cancer, and studies have increasingly focused on rare exposures or weak risk factors. As a result, epidemiologists have increasingly resorted to high-technology solutions (including new molecular markers of exposure) in an attempt to measure the risks associated with these weak risk factors (Pearce et al., 1995).

A more fundamental problem is that the success of ‘risk factor’ epidemiology has been more temporary and more limited than might have been expected (Loomis & Wing, 1991; Wing, 1994). It is one thing to discover that tobacco smoke is the major cause of lung cancer, but redressing this situation is a different problem entirely. For example, Graham (1989) suggests that smoking can be viewed as a strategy enabling women to cope with stress, while at the same time undermining their health and that of their children (Power et al., 1991). Why do manual workers smoke more than non-manual workers (and find it more difficult to give up)? Why have most physicians taken notice of the epidemiological evidence and given up smoking whereas nurses continue to smoke in great numbers?

Moreover, it can be argued that the fundamental problem of tobacco lies in its production rather than in its consumption (Tesh, 1988). As long as tobacco is produced (and governments provide subsidies and incentives to tobacco farmers) then someone somewhere is going to smoke it. In my own country (New Zealand/Aotearoa), tobacco was unknown before the arrival of European explorers in the eighteenth century. Tobacco was one of the ‘gifts’ given by the English representatives before the signing of the Treaty of Waitangi, which was followed by extensive settlement (and colonization) of New Zealand by Europeans. Nowadays, Maori women have some of the highest smoking rates and the highest lung cancer rates in the world. More generally, the (limited) success of legislative measures in industrialized countries has led the tobacco industry to shift its promotional activities to developing countries so that more people are exposed to tobacco smoke than ever before (Barry, 1991; Tominaga, 1986).

Levels of causality

Thus, the link between tobacco and lung cancer is as much a social, economic and political problem as it is a problem of individual ‘lifestyle’. In the context of this book, smoking and socioeconomic factors are not alternative explanations for disease; rather smoking is one mechanism by which socioeconomic factors cause disease, and smoking is therefore an intermediate factor in the causal pathway leading from socioeconomic factors to disease.

In this sense, the apparently competing explanations for disease causation (for example, ‘tobacco smoking’ or ‘socioeconomic factors’) can be reconciled by recognizing that these explanations operate at different levels of analysis (Pearce, 1996). The occurrence of cancer (and other diseases) within a population can be studied at many
different levels (Susser, 1973), including forms of social organization, 'the individual', a particular organ system, or a particular molecule. The causes of cancer can also be studied at these different levels, including socioeconomic factors, 'lifestyle', the organ burden of a carcinogen, or DNA adducts. For example, Potter (1992) argues that:

'A question relevant to the etiology of cancer that is seldom asked is: What gets cancer – the genes, the cell, the organism, or perhaps even the population? The potential answers are not necessarily exclusive, even given reductionist tendencies and the genuine and justified excitement over discoveries in the molecular biology of cancer. Rather these are levels of explanation that may be more or less coherent within themselves but provide even more information when they exist in a framework provided by all of the explanatory modes.'

Clearly, there are advantages in understanding disease causation at all of the different levels at which it operates. Although cancer risk factors such as tobacco smoke may appear to operate at the individual level, exposure may occur due to a wide range of political, economic and social factors; conversely, tobacco smoke ultimately also has effects at the cellular and molecular levels including the production of mutations in DNA.

So what is the most appropriate level at which to commence the study of the causes of cancer? Most researchers will immediately answer that their own discipline has it right, and all of the others have got it wrong. Usually this is presumed to be so obvious that no supporting arguments are necessary. Molecular biologists will focus on the carcinogenic process at the molecular level, with the belief that this will ultimately explain the major causes of cancer. In recent years, much of public health activity (including epidemiological research and some social science research) has focused on aspects of individual 'lifestyle' (perhaps mirroring economic and political trends, which have placed greater emphasis on individual responsibility in recent years) and the targeting of specific 'risk factors'. In contrast, some social scientists and epidemiologists emphasize that the major improvements in health status have come from social and economic changes and their influence on factors such as housing, income and nutrition (McKeown, 1979; Szreter, 1988).

'Top-down' and 'bottom-up'

These various pathways to understanding the disease process fall into two main approaches, which mirror wider scientific debates in recent centuries.

The 'bottom-up' approach [variants of which include reductionism, positivism, or the downstream approach (McKinlay, 1993)] focuses on understanding the individual components of a process at the lowest possible level and using this information as the 'building blocks' to gain knowledge about higher levels of organization. One current example is molecular epidemiology, which attempts to understand disease at the molecular level and then (ultimately) to use this knowledge in public health policy (for example, by screening populations for susceptibility to specific carcinogens). This approach stems from the clinical tradition and is typified by an emphasis on specific risk factors and the use of the randomized clinical trial as a paradigm. It certainly yields useful information about the level under study (for example, the molecular level), but it is debatable whether it is an effective and efficient long-term strategy for gaining knowledge or preventing disease at the population level. As Smith (1985) notes, this approach lacks distinctive theory regarding the occurrence of disease at the population level, and its products can be likened to 'a vast stockpile of almost surgically clean data untouched by human thought' (Anonymous, 1994). Although it has an air of scientific purity, this approach is in fact rarely used in other sciences or related disciplines; for example, nobody would attempt to predict the weather or the motion of the planets from measurements of individual molecules. Such an approach is not only impossible in practice (because of the infinitely large amount of information required), but recent work in chaos theory has shown that such an approach is also impossible in theory because small inaccuracies can produce huge effects in non-linear systems (Firth, 1991).

In contrast, the 'top-down' approach [variants of which include the structural approach (Tesh, 1988), the dialectical approach (Levins & Lewontin, 1985), and the upstream approach (McKinlay, 1993)] starts at the population level so as to ascertain the main factors that influence health status within the
population. Studying disease at the population level usually requires a greater emphasis on observational (epidemiological) studies rather than experimental studies, and may also involve a greater use of ‘ecological’ studies of ‘sick populations’ rather than ‘analytical’ epidemiological studies of ‘sick individuals’ (Rose, 1992). Thus, the ‘top-down’ approach stems from the demographic/social science tradition (rather than the clinical trial paradigm). The study of socioeconomic differences in cancer primarily belongs to this tradition, which has been supported in a recent editorial in The Lancet (1994) that argued for the ‘need to move away from the almost exclusive focus of research on individual risk, toward the social structures and processes within which ill-health originates, and which will be more amenable to modification’ (McKinlay, 1993).

**Links between levels**

It should be emphasized that, even though it is important to start at (and return to) the population level, it is also important to conduct studies at other levels so as to explain the mechanisms by which these population factors operate. In particular, it is of interest to ascertain to what extent the observed effects at the population level are explained by known risk factors. For example, in the Whitehall study, Marmot et al. (1984) found extensive social class differences in coronary heart disease, which were only partially explained by known coronary risk. Syme and Berkman (1979) and Cassel (1976) proposed a more general explanation in which psychosocial factors (stress) influence susceptibility to various specific risk factors.

Even when social class differences for a particular cancer site are explained by the operation of known risk factors this does not mean that socioeconomic factors are not of importance. Of course, it is important to gain information, and take action, at all possible levels, but the history of public health shows that changes at the population level are usually more fundamental and effective than changes at the individual level, even when a single risk factor accounts for most cases of disease. In this sense, a risk factor such as smoking can be regarded as a secondary symptom of deeper underlying features of the social and economic structure of society (Townsend & Davidson, 1982). Thus, just as a variety of health effects in various organ systems (for example, various types of cancer) may have a common contributing cause (for example, tobacco smoking) at the level of the individual, a variety of individual exposures (for example, smoking and diet) may have common socioeconomic causes at the population level.

Furthermore, the ‘populations’ that epidemiologists study are not just collections of individuals conveniently grouped for the purposes of study, but are instead historical entities. Every population has its own history, culture, organization, and economic and social divisions, which influence how and why people are exposed to particular factors. For example, Terris (1979) argues that:

> ‘The causes of cholera in India today go back hundreds of years in India’s history, to the British invasion and destruction of once-flourishing textile industries; the maintenance of archaic systems of land ownership and tillage; the persistence of the caste system and the unbelievable poverty, hunger, and crowding; the consequent inability to afford the development of safe water supplies and sewage disposal systems; and, almost incidentally, the presence of cholera vibrios.’

As a result of such historical considerations, the strength, and even the direction, of socioeconomic disease gradients will vary between populations and over time; for example, coronary heart disease was at one time a disease of the affluent, but has become a disease of the poor as smoking and eating habits have changed over time (Wing, 1988). Furthermore, although specific cancer risk factors will play an important role in any population, their contribution to disease risk will be modified by the baseline disease risk and the presence of various co-carcinogens and cancer promoters, making it impossible to assume a universal dose–response relationship (Wing, 1994). Thus, generalization of study findings is much more difficult in the population sciences than in the physical and biological sciences, and appropriate interventions will differ widely between populations.

**Socioeconomic factors and cancer**

This book on socioeconomic factors and cancer has been prepared with these issues in mind. Just as mortality from most infectious diseases primarily
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decreased due to general improvements in housing, income and nutrition (rather than treatment or prevention aimed at specific viruses or bacteria), it is likely that the greatest advances in cancer prevention will come from social and economic changes that in turn affect 'lifestyle' and exposure to specific risk factors.

In many instances there is clear evidence that cancer is related to socioeconomic factors, but this does not appear to be fully explained by known risk factors. More importantly, there is little evidence as to which socioeconomic factors are of most importance, or whether it is the overall 'package' of social inequality that is responsible for the differences in cancer risk. The aim of this book is therefore to summarize what is already known, and to identify gaps in our knowledge.

The book is intended to cover the major groups of risk factors that may contribute to socioeconomic differences in cancer, but it is not intended to be exhaustive. In particular, we do not address genetic factors; these undoubtedly play a role in most cancers, but are unlikely to play a major role in social class differences in cancer risk. It should be noted, however, that genetic factors may make a minor contribution by causing early disease (for example, Down's syndrome) that may itself affect both social class selection and subsequent cancer risk, or by acting as determinants (or susceptibility factors) for cancer in combination with various exposures arising in polluted environments.

In most countries there are major ethnic differences in socioeconomic status, and ethnic differences in cancer risk are therefore undoubtedly affected by the various socioeconomic factors considered in this book. However, a comprehensive review of ethnic differences in cancer (including cancer in indigenous peoples, migrants, and other ethnic groupings) is beyond the scope of this book. We therefore do not directly consider ethnic differences in cancer risk except for countries (for example, the United States) where data on ethnic differences may be available as a surrogate for data on socioeconomic differences in cancer risk.

In the following three introductory chapters, we present an overview of issues of poverty and health and methods of measuring social inequality including income, education, housing, assets, occupation and employment status (we do not consider regional, gender or ethnic differences in health, which are important in their own right but are beyond the scope of this book).

In Part I of the main body of the book, we then summarize current knowledge regarding socioeconomic differences in cancer incidence, survival and mortality. In Part II we discuss general explanations for social inequalities in cancer, and then consider to what extent the socioeconomic differences in cancer risk may be explained by specific risk factors and aspects of health care. Finally, in the concluding chapter we attempt to draw conclusions regarding what is already known, and to make recommendations for further research.

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References


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