It is too early to tell what the effect of our knowledge of the human genome will have. This, of course, no doubt that the environment will be a major determinant of disease incidence and prevalence [9] and may be possible to identify biological risk factors that may benefit individuals. Perhaps the study of those who do not get diseases will become as important as the study of the afflicted.

'Sick individuals and sick populations' is still an important paper and Geoffrey Rose's comments upon the failure of case-control and cohort studies to detect necessary agents which are homogeneous within a population remain entirely valid. However, altering population risks will, for the most part, require changes in lifestyle and environment.

References


Commentary: Causes of incidence and causes of cases—a Durkheimian perspective on Rose

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Geoffry Rose's seminal 1985 article 'Sick individuals and sick populations' and his 1992 book 'The Strategy of Preventive Medicine', have made a huge impact on the fields of epidemiology and public health. A causal Social Science Citation Index's recently published two work of the work. The central lesson that has been learned from the field is that a large number of people at a small risk may give rise to more cases of disease than the small number who are at high risk. This insight, which has profound implications for prevention and public health, has been incorporated into research contexts through an understanding of the difference between the incidence of absences and relative risk. But there is another aspect to Rose's work that has had a more difficult hearing and that runs counter to mainstream epidemiological approaches solidified under the risk factor paradigm. This is Rose's contention that the causes of cases of disease and the causes of disease inciden may be different and require different types of research strategies. In particular, he argues that we need to find the determinants of prevalence and incidence rates, not just the causes of disease. This has become a central theme in the 'epidemiology wars' with factions sympathetic to Rose's position arguing that epidemiology has lost its public health relevance because of a myopic concentration on individual-level risk factors.

Rose's assertion that the key to understanding incidence and prevalence lies in 'characteristics of populations and not individuals' [5] is, as Chaffee notes, a startling claim [9].

Also, all disease ultimately resides in the individual body and is defined at the individual level. Individual bodies get diseased and become sick. Population incidence then is merely the averaging of these individual cases across the population. How is it possible, then, that an understanding of the causes of incidence could be different from an understanding of the causes of cases and, more generally, how does the characteristic of a population enlighten us about disease etiology?

In order to understand Rose's claims it is essential to examine two key underlying concepts in Rose's work: the concept of 'cause' and the relationship between wholes and parts. In what follows we will discuss these concepts and their basis on this foundation, indicate live situations where the causes of incidence and prevalence may derive distinct treatment.

Two central concepts—cause and the relationship between wholes and parts

Rose's notion of cause

The distinction between Rose's view of causation and that of other theories lies in the types of factors that can be defined as 'causes'. In particular, he argues that in clinical medicine, the concept of cause is more complex and involves multiple factors that interact in a non-linear fashion.
priority, based on the greater certainty about the role they play in the disease aetiology of particular individuals. Rose, in contrast, develops a hierarchy based on different criteria: no greater priority to more fatal causes which he feels hold greater potential for prevention strategies. In Rose's view, these direct causes, are often denied at the population level rather than at the individual level, i.e., they are 'unrecognized' as characteristic of groups or populations and not characterized of individuals and are therefore untreatable within the group. These causes will not be detected by studies that focus on comparing characteristics of individuals within a population, as most traditional epidemiological studies do. In Rose's words: 'In these circumstances all these traditional methods do is to find market; of individual susceptibility.' 109 Although Rose does not fully elaborate on exactly what he means by susceptibility factors, he uses it to imply not only genetic susceptibility, but also other individual-level characteristics that lead to disease in the face of the social and physical environment conditions currently present in the population in which the individual is living. For example, having an affordable personality would only tend to lead along in a social context where drugs are available. This personality feature could be conceptualized as a susceptibility factor that would only be of significance under specific social conditions. The inadequacy of these susceptibility factors is limited since the underlying causes can be removed, susceptibility ceases to matter. 109,110 It is not so much that Rose dismisses these 'susceptibility factors' as causes (indeed they do indeed participate in the disease process and are a primary cause of cases), but the priority is given to population-level causes that facilitate the expression of these susceptibility factors (i.e., allowing susceptibility factors to translate into disease) or influence the prevalence or distribution of 'susceptibility for individual level' factors themselves.

For Rose, causal priority is based on the certainty of the causal attribution or universality of the effect. Rather than cause, the hierarchy is based on the efficiency with which (i.e., how serious) the cause predisposes to the occurrence or increase the incidence of the disease—its morbidity for efficient preventative strategies. Typically, the causes about which we have the greatest concern are the causes in the level of reality, entities at which the disease is defined and more temporarily predominant to the extent of the disease. To see an extreme example for heuristic purposes: a myocardial infarction is caused by a lack of blood-circulation to an area of the heart. This is a cause that can be verified with some certainty and is a necessary and universal cause of this disease. On that basis, it is a high priority cause of myocardial infarction. However, from the perspective of prevention, it is mostly useless as a cause. The cause is so temporally remote to the damage that the window of opportunity to intervene on this cause is very limited. The closer in the causal chain the cause is to the onset of the disease the less opportunity there is for prevention. Even when prevention is possible, it must be prioritized at the level of individuals, an inefficient strategy when many individuals are at risk. Of course if a risk factor only appears to be a cause but turns out not to be one, it will have no preventive effects. Causal certainty is clearly of great importance. However, despite the greater uncertainty that might attach to causes that are more distal from the disease both in terms of level of generality as well as reversibility, Rose gives priority to these distal causes because his hierarchy is based on potential preventive efficiency rather than degree of scientific certainty.

Relationship between whole and parts
The notion of a hierarchy among causes, however, does not fully explain the distinction that Rose makes between the causes of disease and the causes of incidence. Regardless of his prioritization of distal causes, should not the causes of incidence merely be the more distal causes of the cases of disease? If so, the causes of incidence and cases would not be distinct.

To answer this question it is useful to examine Rose's understanding of the relationship between whole and parts, and between groups and populations. The idea that these populations comprised. Rose envisions a Dutch-Doman perspective when he contends that although populations are comprised of individuals, the population has characteristics that are distinct from the mere summation of the characteristics of the individuals in the population. The characteristics of the population may be influenced by characteristics of the individuals but the characteristics and behaviors of the individuals are also shaped by the characteristics of the population.

This relationship is clear seen in Durkheim's explication of social facts. Durkheim defines a social fact as 'everything of which life is made up or is lived upon or is acted out, fixed or not, capable of existing as an individual external to us; or as a way of acting which is general throughout a given society, while at the same time existing in its own right independent of the individual manifestations.' Social facts include all of the spoken and unspoken rules of society into which individuals within that society are born and educated. The rules have a history that was prior to the history of the individuals affected by them and that is sustained even though the individuals who compose the group change. They render some things normal and others abnormal, some in the realm of easy choice and some not of reach. One can accept or rebel against these norms but in either case they produce constraints on individual behaviours. Each person is born into a set of social conditions over which they have limited control. In a similar way the physical environment into which individuals are born exists external to and provides constraints on the individual. It is part of the environment, the individual shapes the environment. These social and environmental acts of interest in a dynamic way with individual-level factors to influence health. While of course these social facts are neutral in the behaviour of individuals, they are distinct from those behaviours and can be understood causally and manipulated as a level of organization outside of the level of the individual. An example, it has often been seen as the cases that are changed prior to the change in behaviour of individuals conforming to them.

Durkheim's social facts provide a framework for understanding Rose's contention that the causes of incidence and the causes of disease are distinct. It is rooted in the observation that whole and parts have different characteristics and that therefore, the causes of incidence (the whole) can be distinct from the within-population causes of cases, the parts of which the whole are comprised. But what lies at the root of this distinction? If incidence is sustained by aggregating numbers of cases over time in a population, how is it that the 'cases of cause' and the 'causes of incidence' may differ?

When we consider the cases of cause, we usually receive our interest to causes that act within an assumed context—a
tact causes field—that we accept as a constant background. In examining the causes of physical distance we typically accept as the "tact cause field" human biological characteristics that are universal. For example, in trying to ascertain the cause of stroke we usually do not consider as a causal factor the human brain's functional need for blood flow. This is assumed as the background in which causes of disease function. Given that all humans require blood to flow through their veins that need to be oxygenated, what is the cause of this organ's malfunctioning?

We therefore look for causes of disease in terms of variations within an accepted (and usually unarticulated) causal field. When Rose discusses "cases" he appears to mean people within a population who become sick. When we look for the causes of "cases" we refer to those causes that distinguish people within a population and the period who become sick from those within that population who do not. Ubiquitous population characteristics serve as the "null equal field." Technically we therefore do not look for all causes of disease but rather those causes that vary between diseased and non-diseased people within that particular population and time period. When we examine individuals (cases) within a population one access as the context given background the social and environmental groups to which these individuals belong. The social and environmental forces of the group are held constant—they provide the tacit field. Since they are held constant they have no detectable influence on the causes of "cases" (i.e., the causes or differences in disease status between individuals within that population).

Moreover social facts and population and environmental exposures more generally interact with individual characteristics leading to varied effects. For example, the effects of social facts may differ depending on the characteristics of the individual, their bodies, and other aspects of the social and physical context—causes referred to by Rose as "susceptibilities." Asterisks (i.e., signs that were not expressed as a"susceptibilities" of causes of disease) often contribute to disease in the presence of certain social facts or population exposures. Thus although it may be the population-level exposures that result in the expression of these individual "susceptibilities" at causes of disease, only the effects of the individual-level factors are apparent, the effects of the population-level exposures themselves are hidden. One can see only the susceptibilities (i.e., the characteristics of people for whom the social facts lead to "cases") and the particular idiosyncratic ways in which the social facts entered into and interacted with the individual. Only by looking across groups can the influence of the social facts become apparent.

When one shifts levels of organization and looks at the difference in the rate of disease between populations of over time, the causes change from those of the population to those between individuals within a given population are likely to pale in comparison to the social facts that now vary significantly. The tacit causal field is shifted. Differences in laws, customs, physical environments, the shape and extent of social networks, etc., may provide the best explanations for the differences in incidence between groups. The incidence itself, the amount of disease in the population, is a characteristic of the population. It changes from the interaction among the characteristics of the social and physical environment and the susceptibilities of the individuals within them. Thus, this view clarifies the distinction Rose makes between "causes of cases and causes of incidence." He does to the fact that the causes of within-population variability (some of cases in Rose's terminology) may be very different from the causes of between-population variability (causes of incidence). Which are often population-level or social factors. Studies that factor in causes of within-population variability may thus miss important disease determinants. 2 It is particularly important to note that the effects of population-level characteristics cannot be simply reduced to the effects of similarly termed mechanisms at the individual level. For example, the effect of living in an area with a high unemployment rate is to affect what is in many ways other than increasing the probability that an individual would be unemployed. The exposed and unemployed are different at two levels of organization. At the individual level, the unemployed, unlike those without a job at all, are exposed while those with a job are unexposed. At the group-level, however, both individuals with and without a job are exposed to the health consequences of living in an area with a high unemployment rate (e.g., areas of poor infrastructure, dilapidated housing, interacting with unemployed people, etc.).

We must also be careful not to rely on these population-level factors, social facts or "causes of incidence." We must view the individual body to cause disease. They need to affect individuals and thus ultimately be treated as causes of individual cases of disease in the same generic sense but not "causes of cases" in Rose's sense, i.e. they cannot be treated in within-population comparisons. In addition, although population-level factors ultimately cause disease by affecting individuals, they do not necessarily enter the body in a simple, clear and distinct manner that can be reduced to some particular individual-level factor. Rather, the pathways through which characteristics of populations enter the body are likely to be numerous and interactive. Social and environmental factors, for example, determine proximity to industrial areas, contact with immunes, and help shape health-behavioural. Social factors interact with the specific biological and social history of the individual to shape the physical health manifestation. They influence individual-level factors without being reducible to them. In this way, the disease occurrence in an individual is not just a manifestation of the individual's characteristics but an interaction between the characteristics of the individual and the environment, both physical and social, to which he/she is exposed and which he/she helps create. Thus, his disease of any individual is incorporated causes at a level of organization above (and below) the individual.

These aspects of Rose's work—his hierarchy of the importance of status and his understanding of the relationship between status and risk—are apparent on several different situations that have particular impact for the distinction between the causes of incidence and the causes of cases. We discuss these briefly from the least to the most controversial.

Situations in which the cause of incidence and the causes of cases warrant separate consideration

Causes of incidence as antecedents of particular identified causes of cases

As Rose notes, although many individual-level risk factors have been identified as the causes of rate increase
over time or place, the key is prevention lies in the social facts related to this change in individual behaviours. In this case, the identification of social facts that may be antecedents to the particular causes of cases may be effective. For example, it is clear that the increase in lung cancer during the second half of the twentieth century in the US was due to an increase in smoking, an individual-level behaviour. One possible approach to disease prevention is to encourage individual patients to stop smoking. However, a more efficient approach may be to examine the cause of the increase in individual's smoking behaviour—the social antecedents to this individual behaviour. When the antecedents of many individuals within a group change over time, it is likely that social facts play a role. The question of why the proportion of individuals smoking increased was one point to potential group-level characteristics (e.g., advertising, role models, promotion of smoking reduction) that influence individual smoking behaviours. Individuals may not be aware of these social antecedents so that asking people why they smoke may not reveal them. In addition, the smoking behaviours of individuals can be detected as a cause of cancer in epidemiology studies. In the US, the number of smoking cessation programs has been increased, and the number of programs offering assistance for smoking cessation has increased. Understanding these social antecedents to particular individual risk factors can provide potent and efficient tools for intervention strategies but these antecedents cannot be identified in studies focusing on within-population comparisons.

Inability to detect individual-level factors due to relative ubiquity within a population

There are a number of individual-level behaviours that may provide potential causes for the decrease in lung cancer incidence (e.g., why the mean level of a disease relative to high in a particular community). In these cases, individual-level factors may not be detectable within a population due to insufficient variability. For example, the use of cigarette within the US has increased. It is possible that individual smoking is responsible for the high rates of coronary artery disease within the US. However, studies of individuals within the US may not be able to detect this factor because the variation is the same within the US. All smokers above the threshold for an effect on this disease. Therefore, an increase in a causal factor can only be detected through a comparison of fat causes in the US and other countries. As Rose notes, which a behaviour is unidirectional within a group (e.g., if a high-fat diet in that factor does not distinguish one case from another within the same causal field of the society. It may, however, be very important in explaining between-population differences in coronary rates. Any factor is ubiquitous, it is likely that potential social facts are at work. If everyone (or nearly everyone) has a high fat diet it is likely that there are social norms and other structural factors influencing this behavior. These ubiquitous individual-level factors and the social facts behind them, cannot be detected in within-population comparisons.

Contextual effects

Group-level factors may interact with the causes of cases (individual-level) factors that distinguish differences between non-differentiated individuals within a group or may be related to disease indicators of common causes of cases. For example, social network characteristics (e.g., number and density of social networks) may have a large impact on the disease rate controlling for those factors that distinguish differences between non-differentiated individuals within the population. A compromised immune system, for example, may be an important cause of why some person develops or infection and another does not within a population, and the size and density of the social networks within the population may interact with this cause of the cases (compromised immune system) to lead to the particular occurrence in the population.

From another perspective, of course, this group-level factor could also be viewed as an antecedent to a different individual-level risk factor (exposure to the virus agent). However, this group-level variable is not reducible to the individual-level variable without any sense of information. Knowledge of social network characteristics may allow a better prediction of human incidence in the group than attempting to do so by determining each one's individual future risk of exposure to the agent. There are other contextual effects that cannot be readily reduced to antecedents of any particular cause of a case at all. This is because the contextual effect may lead to a myriad of different causal pathways through which the disease may enter the body, rather than just being the antecedent of a particular risk factor. Neighborhood or socioeconomic environment (which may be related to disease after controlling for the sociocultural characteristics of individuals) is an example of such a contextual effect for any particular disease. Neighborhood or socioeconomic environment may affect the body through increasing exposure to harmful environmental agents, increasing susceptibility to harmful agents, influencing social network characteristics, genetic vulnerabilities, etc., which in turn interact with a myriad of different individual-level variables. Contextual effects can only be detected by statistics that involve both within-population and between-population comparisons (or both individual-level and population-level factors).

Influence of the mean level (incidence or prevalence) of the disease on risky behavior

One type of contextual effect, deserves particular consideration —the effect of the mean itself (e.g., incidence or prevalence of disease) on individual likelihood of acquiring disease (and consequently on disease incidence). This is one expression of "dependence happening" where the occurrence of disease in any individual is influenced by the occurrence of disease in other individuals. This can be seen for both risk factors and diseases. Infectious disease provides the most obvious example where the mean number of infected individuals in a group has an important impact on the number of individuals who will come into contact with the disease agent and thereby the number of people who will acquire the disease. If few people in the population are infected, this is unlikely that anyone individual is likely to become infected unless the vaccination rate is high. Thus, since the disease incidence is calculated from the number of infected people at a
particular moment in time, this average has an important influence on the disease incidence in the future. A classic example of this type of process is herd immunity, where the individual risk of acquiring infection depends on the prevalence of immunity in the community. Indeed, the mean level of disease at one moment in time becomes incorporated into the fate of individual at a later moment in time. Thus, the dynamics in interaction between the group and the individual is embedded in the individual-level expression of the disease. Thus, incidence at one point in time is derived from past past incidence.

Similarly, a tendency for behavioral components to move towards the state of the outcomes can have an effect on future incidence. The incidence of alcoholism in any society at any particular point in time is influenced not only by the number of individuals with identified individual-level risk factors such as psychiatric vulnerability or unacceptable personality traits. It is also influenced by societal norms regarding alcohol use and the availability of alcohol in the community. The stronger the norms and the greater the availability the higher the probability that a susceptible individual will become an alcoholic, alcohol norms and availability are, in turn, influenced by the mean level of alcoholism in the community. In looking at incidence at a given point in time the effect of past incidence of alcoholism may not be apparent. The past incidence provides the current norms in which the individual-level factors now operate. Therefore, it becomes apparent that this means has an effect on future incidence is being predicted, thus, individual cases and incidence at one point in time result in part from the general effects of incidence on prevalence of the state outcome in the past. The number of individuals participating in an activity similarly influences the norms about risk behaviors which in turn influences the number of and type of people who will engage in those, for example, if smoking is normative, many people will engage in this behavior despite the evidence of risk. Therefore, when population-level factors may help shape the number of people who engage in such behaviors as well as the relative risk who in the population is likely to do so. The effects of past incidence on prevalence and norms on risk of disease (or risk of acquiring a risk factor) is another example where the investigation of population-level factors is crucial to understanding the causes of disease and, within population investigation of the causes of disease focusing on individual-level causes of differences between individuals may be irrelevant.

Definition of health and illness
Characteristics of populations in the incidence of disease and their determinants are complex. Socioeconomic factors play a significant role in the incidence of disease in the broader sense, by determining what we consider normal to be influenced by what is prevalent. What is common is all right, we presume. [5]-[7] One implication of this is that social factors may also influence disease incidence in the broader sense, by determining what we consider to be a disease. Social factors influence our expectations of how many ages and pains are normal, how long we expect to live and what we expect our bodies to look like and our minds to accomplish. Thus, definitions and biological variance can be used as disease or redefined as normal, obesity, inherited conditions, anxiety, acne, post-menstrual stress and gender identity disorder are just a few examples. These expectations change over time and place based on the number of ill people, average life expectancy and other types of rates and norms. These types of influences cannot be understood simply by examining differences between who is ill and who is not ill within a population. Rose's contribution cannot be overestimated. The insight that characteristics of populations cannot be reduced to individual characteristics and that both may have important impacts on health surpasses new realms for health prevention and disease prevention. But Rose's conceptualization requires a shift in thinking, particularly in contexts where individual autonomy and choice is a great priority. Social facts imply that individual autonomy and choice is constrained by social position and physical environment. One cannot, as an individual, simply choose to be healthy or to behave in a way that increases one's health. There are social limitations to the choices faced by individuals. Rose's conceptualization suggests that although the particular combination of individual-level risk factors of each person with the disease may vary, there are general social and physical factors that may increase the number of individuals who will become diseased. Population-level factors can change the rate of disease in a society even when the prevalence of recognized individual-level risk factors remains unchanged. Which realm is chosen for the prevention of any particular disease is a decision based on the assessed relative efficiency and the potential for unintended consequences of our choice. This in turn requires that the complexity in developing research strategies that can examine causes at levels of aggregation other than the individual level. The population level may not be ruled by the best choice for examining etiological or for intervention. Inequality, in many cases, may be well and it deserves recognition and consideration.

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