A classification system should first and foremost be internally consistent, and beyond this, it should also serve the needs of those using it. Whereas basic clinical researchers will probably be best served by using cause-based classification system, an effect based classification system is likely to be of greater utility to health system planners. Conditions requiring long term care, whatever their aetiology, place similar demands on healthcare systems, patients, their families, and communities. We argue strongly that the term “chronic” will be more readily understood and of greater utility if it refers to conditions requiring long term management by health systems.

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Authors’ reply
The letter by Unwin et al in response to our paper makes some important points about the attributes of disease classifications. We agree that, unfortunately, it is commonplace for classifications based on cause and those based on effect, to be interchanged by the public health community. This not only causes confusion in the minds of public health policy makers, but promotes bureaucratic apathy towards resourcing prevention programmes. We are glad that Unwin et al support our thinking to promote the use of the term “chronic” to denote conditions requiring long term care, which is more readily understood by the public health community.

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Epidemiology of SARS: the missing pathogen?
This is indeed a strange disease. The epidemiology suggests it to be of relatively low infectivity, but high severity. This in itself is odd, especially if the causative agent is a virus and the principal mode of spread by coughing/droplet. Also odd is the undoubted existence of “superspreaders”, who can infect very many of their contacts—I can’t think of any parallels to this in respiratory virology. Perhaps the SARS virus obeys the usual rules of droplet transmitted respiratory infections, and is of high infectivity. However, because of shared antigens, a proportion of the population has an acquired resistance to the new virus, having already been exposed to another, relatively innocuous, virus that provides immune protection. It is possible that the proportion of humanity immune or partially immune to SARS could be as high as, say, 95% if the second virus were a very common one, for example, one of the coronaviruses that causes coryza. This would explain the seemingly low, unexpectedly so, infectivity of the SARS agent.
Perhaps this also explains “superspreaders”. Picture humanity divided into two categories:

1. Those who have met a common related coronavirus, and consequently have a degree of immunity to SARS, say for the sake of argument ± 95% of the population.

2. Those who have not met it, and have no immunity. If the defences of the first group are overwhelmed by exposure to a huge SARS virus inoculum, perhaps they would contract a modified form of the disease, quickly recruit their immune systems to neutralise the virus, and be less likely to be a source of infection to a recognised susceptible individual, be likely to recover, not shed large amounts of virus, not be all that infectious. The second group would get the disease in an exuberant form, excrete quantities of virus, and be likely to succumb before their immune system could meet the challenge—the superspreaders—always allowing that other factors may be implicated in superspreader events, such as a pre-existing condition in the initiator, or variation in the likely number of contacts, this greatly increased by hospitalisation before the risk is known.

The invocation of a high (but presumably variable and incomplete) level of acquired immunity to the SARS agent in affected populations explains other apparent anomalies of the 2003 epidemic—Why did some regions (Russia, Japan, Indonesia, Thailand, Malaysia, etc) not have any cases, despite a high intensity of air traffic with the epicentre of the infection? Above all, how was a pandemic contained to a level where both SARS and SARS-like infections were averted when in the earlier stages of the disease the possibility of widespread transmission was present? This material, be likely to succumb before their immune system could meet the challenge—the superspreaders—always allowing that other factors may be implicated in superspreader events, such as a pre-existing condition in the initiator, or variation in the likely number of contacts, this greatly increased by hospitalisation before the risk is known.

Is the inverse care law no longer operating?

The inverse care law, proposed by Julian Tudor Hart in 1971, states that ‘the availability of good medical care tends to vary inversely with the need for it in the population served.’ A number of authors have now claimed to have found instances of the inverse care law operating in practice.1,2,3,4,5

Given the evidence that this ‘law’ has gained in the healthcare literature over the past 30 years, we were surprised to note that Jordan et al failed to make reference to it in their recent article on the relation between access to services and health.6

In this report, access to services was measured as both straight line distances and car travel time to the nearest GP surgery and hospital as well as the access domain of the index of multiple deprivation 2000, which combines measures of straight line distances to the nearest primary care provider, pharmacy, school, food shop, and post office. Among urban wards, the authors report a consistent inverse association between distance to services and both mortality and limiting long term illness (LLTI) in people aged 0–64 years—although this association was negligible in terms of the relations between LLTI and distance to hospitals.

Both premature mortality and LLTI are markers of need for health services in themselves. In summary, they are both strongly associated with deprivation in the UK,6,7 and therefore a much broader marker of need for health services. The results of Jordan et al suggest that areas with greater need for health services are nearer to and have greater access to, or concentration of, both health and wider social services. This is in conflict with the inverse care law, which would predict that distance to services should be greater, and therefore access poorer, in areas with higher levels of need.

Are the results of Jordan et al evidence that the inverse care law is no longer operating in the UK? Is it possible that over the past 30 years, we have managed to redistribute primary care services, in particular, so equitably that instead of deprivation, poor health and greater need for services being associated with poor access to services, it is now associated with greater access to services? Alternatively, is it possible that the inverse care law has merely operated in practice in the UK in recent times and that “evidence” for it misinterpreted the original formulation of the law and concentrated on use of services, rather than provision of them?

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References

Do socioeconomic conditions reflect a high exposure to air pollution or more sensitive health conditions?

We read with great interest the recent paper by Martins et al.8 concerning the interaction of socioeconomic conditions on air pollution adverse health effects in elderly people in Sao Paulo, Brazil. These results are very interesting and may promote understandings of which social category of people are most sensitive to air pollution. The authors suggest that socioeconomic deprivation represents an effect modifier of the association between air pollution and respiratory deaths in elderly people for an increase of 10 μg/m³. They conclude that poverty represents an important effect modifier that should be taken into account when determining the health consequences of environmental contamination. We agree with these conclusions. Nevertheless, the question is to know if poor people died because they are more exposed to air pollution, or because of inaccessibility (geographical and socioeconomic considerations) to the healthcare system, or because they were more exposed to air pollution?

We know that people with lower socioeconomic status are more sensitive to a large number of risk factors, affected more often by life habits, or to addictive behaviours, such as smoking habits.7 When air pollution is considered, socioeconomic characteristics as an effect modifier can take two aspects. Firstly, people with low socioeconomic status may be more sensitive in terms of health effect because they have associated diseases and people with certain diseases had a greater risk of dying during an episode of elevated air pollution than healthy persons of the general population.8 Furthermore, people living in underprivileged sectors would have both more limited access to health care and greater exposure to air pollution. Exposure to pollution is the second aspect of the interpretation of the effect modifier. Jerrett et al argue, low socioeconomic conditions may be associated with manufacturing and so with a higher workplace exposure, but also with a lower traffic exposure. In addition, persons with lower socioeconomic characteristics may be exposed to a complex mix of pollution from indoor sources, as well as outdoor pollution because of traffic, industry, and waste burning in developing countries. To explore the link between individual exposure and socioeconomic characteristics because these two factors are strongly correlated.

More studies are needed to investigate this effect modifier and particularly the signification of this effect. To understand this effect we will need individual data on risk factor but also data on individual exposure to have a good interpretation of the results and to have policy implications.

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The comments of Filleul and Harrabi on our paper reflect the major concerns about the role of socioeconomic conditions in the association between air pollution and health effects and keep the topic on discussion. Low socioeconomic status evolves different and complementary aspects that can act synergistically to aggravate health conditions. For instance, being more vulnerable to diseases and having less access to health (geographically and economic considerations) are factors that, in general, are concurrent among poor people and can contribute to death. In addition, if they also are more exposed to air pollution, and probably not only air pollution (e.g. outdoor) but also water pollution, we have the whole picture of what it is to be in the lowest socioeconomic levels of any society. If the discussion focuses only on levels of exposure it reflects the concept of linear dose-response relation between PM10 and respiratory diseases that is well known and accepted. 1

than with the different levels of PM10 correlated with socioeconomic conditions and health measures.

Davey Smith. These risks, however, were also screened between 1970 and 1973 a strong stroke risk

Siblings and adult mortality and stroke risk

In men born between 1906 and 1938 and screened between 1970 and 1973 a strong relation between greater number of siblings and mortality risks was found by Hart and Davey Smith. These risks, however, were also related to adverse behavioural socioeconomic and health outcomes.1

A great number of children, particularly in lower socioeconomic classes and in the earlier decades of the previous century, implies reproduction at earlier and more advanced maternal age, and shorter interpregnancy intervals. These aspects have been neglected in studies on number of siblings in relation to constitut-
Rethinking the terms non-communicable disease and chronic disease

Nigel Unwin, JoAnne Epping Jordan and Ruth Bonita

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