

PostScript

LETTERS

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Rethinking the terms non-communicable disease and chronic disease

We welcome Ackland *et al*'s timely discussion on the terms "non-communicable" and "chronic" disease.¹ Their argument is that conditions currently labelled "non-communicable" are in fact "communicable" because the risk behaviours that underlie them are highly transmissible. Thus they argue for a change in label, from "non-communicable diseases" to "chronic diseases".

Their argument, however, confuses one classification system, which is based on *cause* (namely, communicable diseases compared with non-communicable diseases compared with injuries), with a second classification system, which is based on *effect* (namely, acute conditions compared with chronic conditions). Their argument also overlooks the growing consensus that chronic conditions include certain communicable diseases, such as HIV/AIDS.^{2,3} In fact, certain non-communicable conditions are acute in nature, while certain communicable conditions require chronic, ongoing care. For example, HIV/AIDS clearly has an infectious aetiology but requires long term management by the healthcare system. As such, it has a great deal in common with type 2 diabetes. Conversely, acute appendicitis is a "non-communicable disease" that requires an urgent health care response quite similar to bacterial meningitis.

A comparison has been made of the burden of disease in sub Saharan Africa using the current global burden disease study broad classification system (three cause based groups, of which non-communicable diseases is one) and an alternate classification based on the effect based groupings of chronicity or acuteness.⁴ The overall patterns in children aged under 5 are similar with both classification systems, but in people aged over 5 there are noticeable differences. For example, 35% of disability adjusted life years (DALYs) were attributable to non-communicable diseases, but 86% were attributable to chronic conditions, reflecting in particular the impact of HIV/AIDS and TB.

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 a 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 paper¹ 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 for diseases that contribute so significantly to the burden of disease. Why would a jurisdiction dedicate scarce resources towards preventing and controlling diseases that are long term (chronic) and where causation is unclear (non-communicable)? Many jurisdictions will only take on the concept of an epidemic seriously when there is a clear link to the traditional concept of "communicable" or infectious disease.

So it is very important to encourage use of language in describing diseases that is of practical value to both clinical researchers and policy makers. This language should engage everyone in thinking about public health responses to diseases from both the perspectives of disease prevention and disease control. While international classification systems are unlikely to change quickly, we are seeking a shift towards more appropriate use of terminology when describing major disease states.

Our desire is to encourage people to think about the upstream issues of causation of diseases in order to motivate a more pragmatic

focus on public health interventions. Indeed we support use of the term "chronic" to emphasise the long term implications for the health system, but more importantly wish to discourage use of the term "non-communicable" where there is mounting evidence for the non-microbial, communicable nature of many chronic diseases. Qualifying some chronic diseases as being "transmissible" may be helpful in highlighting the role of social, cultural, and societal factors as disease vectors. Herein lie the opportunities for improving public health responses and interventions. 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 \pm 95% of the population.

(2) Those who have not met it, and have no immunity, \pm 5%. 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 produce antibodies to a recognised infectious agent, 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 infectious 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.

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 disease? Why did some affected regions (Guangdong, Hong Kong, Toronto) struggle to contain the disease, when others (Singapore, Vietnam) had no such difficulty? Above all, how was a pandemic averted when in the earlier stages of the spread of SARS this outcome appeared very much a probability?

I believe that a coherent mathematical model of the SARS epidemic could be constructed from the above theory. This of course would not necessarily lend it validity, but it may be worth looking at.

Even if this aperçu should prove anywhere near the mark, it still would fail to answer another question—Will epidemic SARS recur? On the other hand, the possibility of a pandemic might be seen to recede.

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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.”¹ A number of authors have now claimed to have found instances of the inverse care law operating in practice.^{2,3} Given the prominence 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.⁴

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 general practitioner, primary 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 addition, they are both strongly associated with deprivation in the UK,⁵ 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 rarely operated in practice in the UK in recent times and that “evidence” for it has misinterpreted the original formulation of the law and focused on use of services, rather than provision of them?²

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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*¹ concerning the influence 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 $\mu\text{g}/\text{m}^3$. They conclude that poverty represents an important risk factor 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 ill, or because of inaccessibility (geographical and economic 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 according to different life habits, or to addictive behaviours, such as smoking habits.² 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 increased of air pollution than did members of the general population.³ Furthermore, people living in underprivileged sectors would have both more limited access to health care⁴ and greater exposure to air pollution. Exposure condition is the second aspect of the interpretation of the effect modifier. Jerrett *et al*⁵ argue, low socioeconomic conditions may be associated with manufacturing and so with a higher workplace exposures, but also with a lower mobility. 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. It seems necessary 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 significance 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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Authors' reply

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 (geographical 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 (indoor and outdoor) but soil and 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 In our paper we showed that the different effect size of PM₁₀ on respiratory deaths for each studied region, measured as a percentage increase in mortality for each 10 µg/m³ of PM₁₀, is more correlated with socioeconomic conditions than with the different levels of PM₁₀ concentration in each region. However, we agree with Filleul and Harrabi that different study designs can provide information on individual exposure status to clarify the contribution of socioeconomic status on the association between air pollution and health effects. We are developing additional studies focusing on the analysed populations looking for answers to the questions our study brought up. Nevertheless, the results already presented are enough to suppose that "different people" may react differently to the same risk factor.

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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 measures.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 constitu-

tional diseases of complex origin. These casualties seem to be connected with non-optimal maturation of ovarian follicles and in fact, the maturing oocytes they contain. Conceptions at the extremes of maternal reproductive age or during the critical stages of postpartum restoration of the ovulatory pattern (or durante lactatione) are constrained by protraction of the preovulatory phase of the menstrual cycle that entrains preovulatory overripeness of the oocyte (PrOO). This results—in analogy with experiments in animals and observations in humans—in a continuum of pregnancy wastage, perinatal and postnatal mortality, or morbidity.^{2–4} In addition, mothers with low socioeconomic status are known to suffer disproportionately more from low standards of nutrition, abnormal body mass index, and inherent menstrual disorders. They also use less safe methods of contraception resulting in gradually more unplanned and unwanted pregnancies³ and, in turn, in shorter interpregnancy intervals and intratubal aging of the oocyte (waiting to be fertilised)—that is, postovulatory overripeness of the oocyte (PoOO).^{2–4}

The driving force behind decreasing rates of newborns with neural tube defects or Down's syndrome was evident long before the introduction of prenatal diagnosis, selective abortion and folate supplementation. The true reason for these decreases has never been revealed and the biased scientific preoccupation with recent changes in lifestyle and risk factors for explaining the enigmatic decrease of cardiovascular diseases has also been blamed. A same scenario, therefore, may exist here and ovopathy might be the common causal pathway for developmental anomalies and "innate" constitutional entities of complex origin. The relentless decrease of conceptopathology, running parallel with increasing socioeconomic levels and improving healthcare provision, would be responsible for their decrease.^{3,4} Intrauterine mortality, stillbirth, and infant mortality began to decrease at the end of the 19th century, while in fact, cardiovascular diseases only after a lifetime delay since the 1960s. Many other unexplained correlates with cardiovascular diseases (and other chronic diseases) are elucidated by this causal pathway, for example, comorbidity, intergenerational matrilineal transmission and strong social patterning, discordance in monozygotic twins, poor fetal growth and low birth weight, seasonality of conception correlated with geographically latitudinal gradient, and finally, male gender biases.⁴

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BOOK REVIEW

Global public goods for health; health economic public perspectives

Edited by Richard Smith, Robert Beaglehole, David Woodward, and Nick Drager. Published by Oxford University Press, Oxford, 2003, pp 287. ISBN 0-19-852544-3 (hardback). 0-19-852798-5 (paperback)

This volume explores the applicability of the concept of global public goods to health and health related issues as well as the question of the added value—for example, in terms of new analytical insights or a better understanding of various policy approaches and instruments—of looking at challenges through the lens of global public goods. As the stage setting chapter 1 by Woodward and Smith notes, "...as globalization progresses...matters which were once confined to national policy are now issues of global impact and concern; yet no one nation necessarily has the ability, or the incentive, to address the[se] problems" (page 3). So cross border cooperation is important for a global public good, let us say, polio eradication, to emerge and to be available for the consumption—or enjoyment—of local communities or countries.

The volume's chapter analyses are written by a multi-disciplinary team of authors and cover three main sets of issues: (1) the global public goods properties of the control or eradication of select communicable conditions (including polio, tuberculosis, anti-microbial drug resistance), and the health consequences of a number of global environmental "bads" (such as the global climate change or the depletion of the ozone layer); (2) the importance of knowledge (including medical knowledge, genomics knowledge, and public health infrastructure and knowledge) as a critical input to people's improved health status and enhanced public health conditions; and (3) how to enable global public goods for health, such as international law and health regulations. However, running through the individual chapter analyses also are common themes. Among them are such issues as the prioritisation of global public goods and the politics of their provision, their "production" and financing.

The discussions on these themes is analytically rigorous yet clear and focused, leading to practical and pragmatic—yet in part, also innovative—policy conclusions and recommendations. Thus, the book should be of interest to researchers and students as well as policymakers and practitioners alike.

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