fees seem to have reduced attendance at rural health facilities in Ghana[9] and treatment for sexually transmitted diseases in Kenya.[10] The World Bank itself acknowledges a problem when user fees reach 1% of annual household income.[11]

The South pays back debt at enormous social cost, and this contributes to the dismantling of its health and educational systems. By 1990 over $50bn a year was pouring from indebted developing nations to their creditors.[12] If the summit is serious in its desire to relieve poverty, says the Group of 77 nations, the collective voice of the South, then African debt must be cancelled and the debts of other poor countries reduced by a target date.[13] The draft declaration of the conference also calls for urgent consideration to be given to the relief of multilateral debt (owed to the World Bank and International Monetary Fund).

UN conferences are often the lowest common denominator for international action. As doctors, how can we promote the summit's undoubted good intentions? Firstly, we can increase public awareness of the urgent need to reduce poverty, both nationally and internationally, emphasising its deleterious effect on health. Health issues will not receive sufficient attention at the conference and need to be highlighted in future. Through universities and research institutions, understanding of the causes of poverty and the effectiveness of antipoverty campaigns can be improved. Finally, integration of the results of social science and health research into economic decision making should be encouraged so that balancing budgets does not unbalance the lives of people.

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1 UN. *The world summit for social development: draft declaration and draft programme of action.* New York: UN, 1994.
2 UN Development Programme. *The human development report.* Oxford: Oxford University Press, 1992:34.
3 Unicef. *The state of the world's children.* Oxford: Oxford University Press, 1995.
4 Khor M. Social summit: forum for North-South economic talks. *South-North Development Monitor* 1994;**No 3410**:43.
5 Robinson N, ed. *Agenda 21, earth's action plan.* Oceana, 1993.
6 Eurostep. *Eurostep. Rich countries block efforts to fight poverty.* Brussels: Eurostep, 1995. (Press release from UN social summit preparatory meeting, New York.)
7 Madeley J, Sullivan D, Woodroffe J. *Who runs the world?* London: Christian Aid, 1994.
8 Lennock J. *Paying for health. Poverty and structural adjustment in Zimbabwe.* Oxford: Oxfam, 1994.
9 Waddington CJ, Enyimayew KA. A price to pay: the impact of user changes in Ashanti-Akim district, Ghana. *International Journal of Health Planning and Management* 1989;4:17-47.
10 Moses S, Manji F, Bradley JE, Nagelkerke NJD, Malisa MA, Plummer FA. Impact of user fees on attendance at a referral centre for sexually transmitted diseases in Kenya. *Lancet* 1992;**340**:463-6.
11 World Bank. *World development report 1993: investing in health.* Washington, DC: World Bank, 1993.
12 Rich B. *Mortgaging the earth.* London: Earthscan, 1994.
13 Steel J. A bunch of good intentions signifying nothing. *Guardian* 1994 Dec 28:18.

Patterns of disease: diabetes mellitus and the rest

We should be investigating the relations between diseases

See pp 555, 560

As early as 400 BC Indian doctors observed that diabetes was a disease of well fed people. In 1895 Bose wrote "Amongst the Zemindars and Talookdars, who consider it a pride and honor to lead an indolent life, diabetes is a common disorder."[1] In 1962 my 76 year old Aunt Nina attributed her diabetes to being old, fat, and inactive.

Much of what I have read about epidemiology of diabetes in the past 20 years has merely refined what my Aunt Nina said about her non-insulin dependent diabetes. We have learnt more about its causes and have more precise estimates of the relative risks of developing it as a function of age, obesity, level of physical activity, and, from Rimm and colleagues' paper in this week's journal, cigarette smoking and alcohol use (p 555).[2] But substantial advances in our understanding of its epidemiology and that of other non-communicable diseases have been rare.

The epidemiology of diabetes has recently seen a change of focus—apparent in the paper by Perry and colleagues in this issue with its examination of the interrelations between diabetes and other risk factors for coronary heart disease (p 560).[3] Although Lilienfeld and Lilienfeld defined epidemiology's concerns as "the patterns of disease occurrence in human populations and the factors that influence these patterns,"[4] this is not how we have investigated diabetes or in fact any non-communicable disease. We do not look at diseases within populations; instead we look at a disease, "our" disease. For 40 years we have been diabetes epidemiologists, cancer epidemiologists, cardiovascular epidemiologists, and AIDS epidemiologists. Few researchers have cut across disease boundaries and examined more than one disease. Fewer still have tried to examine the patterns of diseases in a population.

An important exception was Omran with his development of the concept of the epidemiological transition. His classic paper, which has received relatively little attention since it was published in the early 1970s, showed clearly the existence of both patterns of diseases and processes that produce these patterns.[5] Moreover, the changing patterns of disease are very predictable. Omran charted the rapid fall in infectious diseases with rising socioeconomic status. Increasing life expectancy then unmasked chronic diseases, which occur in a distinctive pattern. Initially, deaths from trauma increase. Next comes a peak of non-insulin dependent diabetes, not initially associated with a rise in coronary heart disease. Coronary heart disease emerges after diabetes, and cancer five years after the others have emerged.

This pattern is almost universally seen and is almost universally ignored. An episystems approach, which investigates the processes and patterns of diseases, could lead to important insights.[6] For example, we need to evaluate the complex interrelations not only between diabetes and cardiovascular disease but also between diabetes and cancer, rheumatic fever, and polio.

Evolutionary biology may provide the best model. This discipline has been important for understanding and predicting the rise and fall in species. Species are constantly in transition, with the numbers of one species rising and those of another falling. This symphony of change is orchestrated by the environment, which interacts with the genetic background of plants and animals. Different species change in response to each other as the result of conditions affecting both of them. They also change as the result of environmental conditions tending to "run together," yielding strong interrelations in the ecosystem. Darwin described "how plants and animals

remote in the scale of nature are bound together by a web of complex relations."[7]

Isn't this exactly how diseases "evolve" on a population basis? Diseases are constantly in transition, with one disease rising and a second falling in a systematic relation with each other. We should not talk about the origin of "disease" but the origin of "diseases": within a population diseases are intimately bound together by a web of complex relations, which we should be investigating. We need to examine and model the evolution of patterns of diseases.

We need to break away from our orientation towards single diseases and begin to focus on the big picture. For example, life expectancy has increased enormously almost everywhere during the past 40 years, which has unmasked diabetes as well as coronary heart disease. Yet for any given life expectancy, say 65, the causes of death within populations are almost identical worldwide. As life expectancy is becoming more and more similar worldwide so are the causes of death. In addition the disease patterns in the epidemiological transition are also related to many other features of society, such as socioeconomic status, war, the status of women, and, as Omran has shown, fertility and population growth.

An episystems approach to diabetes and other non-communicable diseases is meant not to replace the existing approaches but rather to complement them and provide new insights. An understanding of what drives the process and the patterns of disease will most certainly be important for forecasting the future diseases in a population. Moreover, if we are able to understand the process then prevention will not be disease specific and preventive approaches may affect a wide variety of different, biologically unrelated diseases.

The next generation of diabetes epidemiologists needs to break away from existing models and begin to think about ways of understanding what drives the evolution of diabetes and other diseases in a population. A prerequisite is much better monitoring of disease as patterns cannot be detected unless the data have been collected.[8]

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1 West KM. *Epidemiology of diabetes and its vascular lesions*. New York: Elsevier, 1978.
2 Rimm EB, Chan J, Stampfer MJ, Colditz GA, Willett WC. Prospective study of cigarette smoking, alcohol use, and the risk of diabetes in men. *BMJ* 1995;310:555-9.
3 Perry IJ, Wannamethee SG, Walker MK, Thomson AG, Whincup PH, Shaper AG. Risk factors for the development of non-insulin dependent diabetes in middle aged British men. *BMJ* 1995;310:560-4.
4 Lilienfeld A, Lilienfeld D. *Foundations of epidemiology*. New York: Oxford University Press, 1980.
5 Omran AR. The epidemiologic transition—a theory of the epidemiology of population change. *Milbank Memorial Fund Quarterly* 1971;49:509-38.
6 LaPorte RE, Sauer F, Marlet E, Gamboa C, Akazawa S, Gooch T. Health and climate change. *Lancet* 1994;343:302-3.
7 Darwin C. *On the origin of species*. New York: Cambridge University Press, 1975.
8 LaPorte RE. How to improve monitoring and forecasting of disease patterns. *BMJ* 1993;307:1573-4.

How can we improve the detection of glaucoma?

Thorough testing and better targeting

Primary open angle glaucoma is an insidious disease that affects 1-2% of people over 40. In Britain it accounts for about one in eight new registrations for blindness, although early detection can prevent much damage to sight.[1] Over 90% of new confirmed cases result from the seven to eight million sight tests for those aged over 40 conducted at opticians' premises each year.[2] Available data suggest, however, that only about half of all cases of primary open angle glaucoma are detected[3]; and one in five people with newly confirmed disease already have advanced visual field loss.[2]

Although a high proportion of the relevant population attends opticians fairly regularly, the standard of testing for glaucoma is uneven.[4-7] A recent large survey has shown that optometrists who routinely used tonometry detected over twice as many glaucomas as did optometrists who relied mainly on ophthalmoscopy.[7,8] Those who, in addition, used perimetry in most patients at high risk of glaucoma detected over three times as many glaucomas, while routine perimetry yielded somewhat more again. Nevertheless, a few years ago only half of patients were being tested by tonometry and a tenth by perimetry,[4] although the availability of new electronic instruments will have improved these proportions. Almost 60 000 patients a year are referred by optometrists for secondary examination because of suspected glaucoma.[8] The most recent survey indicated that 32% of such patients referred had glaucoma, 23% had ocular hypertension, and 16% had other diagnoses, while in 29% no abnormality was found.[2]

To improve the present system of detecting glaucoma the trend to more comprehensive testing by optometrists needs to be encouraged. Ophthalmoscopy is mandatory. Routine tonometry (which takes two minutes per patient with an electronic tonometer[3]) is clearly practicable and important in identifying those patients with high intraocular pressure; these are the patients to whom lowering the pressure can be expected to be most effective. Routine perimetry (which takes three to four minutes per patient with a semiautomated perimeter[3,9]) will often be economically feasible, especially in large practices where equipment can be shared and the task can be carried out by assistants who have not been trained in optometry. Elsewhere, it is reasonable to expect that at least patients at high risk of glaucoma should have their visual fields tested; this group includes people with raised intraocular pressure, optic discs which suggest the possibility of glaucoma, high myopia, diabetes, a family history of glaucoma, or Afro-Caribbean origin, who collectively account for about one fifth of patients over 40.[10] In effect, this calls for the good standards of testing already practised by about a quarter of optometrists[4,7] to be more generally adopted.

Patients should be referred to consultant ophthalmologists with any of the following: an intraocular pressure ≥ 26 mm Hg; intraocular pressure of 22-25 mm Hg, plus optic discs or visual fields with features characteristic of glaucoma; or a visual field defect that persists on repeated testing and that suggests glaucoma, even with no other signs. To help with ranking referrals according to priority, optometrists should be asked to indicate if glaucoma is "almost definite," on which their judgment is generally reliable.[8]

An important problem is how best to handle the many borderline cases in which insufficient reason exists to justify