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## The role of epidemiologists in eradicability of poverty

Sir—Kenneth Rothman and colleagues (Sept 5, p 810)<sup>1</sup> have responded to criticism that modern epidemiology, with its advanced methodology and orientation towards molecular, genetic, and metabolic sciences, has lost sight of the social-historical causes of disease in populations. They argue that the causal chain is long and that there is merit in studying all of it, from distal to proximal causes. They say that epidemiologists are best equipped to study proximal causation—which often leads to specific, feasible, and effective interventions. Their response, framed in relation to the underlying role of poverty in disease, is inadequate on three counts.

First, the social-environmental backdrop against which a population's disease profile changes over time is not static. Further, although poverty is a long-running feature of human society, many other influences are more changeable. An important point of the criticism that contemporary epidemiology neglects the social-historical context of disease is not just that there are other basic circumstances that also warrant study, but that contextual (social, economic, cultural, technical, and environmental) circumstances keep changing.

Epidemiologists therefore need to maintain research surveillance at the contextual level, while also studying the individual factors. We might thus find that the rising incidence of asthma is explained by ecological population-level changes in early childhood such as microbial exposure or vaccination regimens, the balance of energy intake and expenditure in daily urban life might increase the prevalence of obesity, the liberalisation of trade and travel might enhance the dissemination of infectious diseases, and global environmental changes might pose risks to the health of the population.

Second, we should not expect to be able to account for the occurrence of all diseases in terms of individual-level risk factors. To seek to explain the roller-coaster graphs of mortality in excommunist countries of Europe mainly in terms of personal behaviours risks overlooking the role of population

changes in civic infrastructure, social-economic relations, and community morale.

Third, professional faint-heartedness is inappropriate. Eradication of poverty is a task beyond the capacity of epidemiology, but nor does any other single discipline or professional group have that capacity. The suggestion that this task should be left to economists is naive. Epidemiologists have a professional opportunity and responsibility to help to elucidate the consequences of poverty, which include risks to health, and thus to contribute to a collective, pluralist discussion about social solutions. The choice for epidemiology is not, as implied by Rothman and colleagues, between studying the health effects of tobacco consumption and lobbying against tobacco production and distribution. Rather, epidemiologists should take part in the investigation of all causal influences on risk behaviours and their health outcomes.

Much epidemiological research should be done in collaboration with other research disciplines. As epidemiology matures it will forge stronger interdisciplinary links, both in molecular and social research. Thus useful contributions will be made to immediate and long-term improvements in public health.

A J McMichael

Department of Epidemiology and Population Health, London School of Hygiene and Tropical Medicine, London WC1E 7HT, UK (e-mail t.mcmichael@LSHTM.ac.uk)

- 1 Rothman KJ, Adami H-O, Trichopoulos D. Should the mission of epidemiology include the eradication of poverty? *Lancet* 1998; 352: 810–13.

Sir—Kenneth Rothman and colleagues<sup>1</sup> suggest that epidemiologists who focus on downstream, molecular and biological, pathways are beleaguered by critics who argue that epidemiology has lost its way and should move toward a focus on upstream, social and economic, determinants of population health. One has only to compare the funding of the two kinds of epidemiological research to know that the Goliath of downstream research has little to fear. This contribution frames important issues in a divisive way that will do little to bring about the synthesis of upstream and downstream approaches that is so needed.<sup>2,3</sup>

It would be hard to disagree that the primary task of epidemiologists should be “to acquire insight into the causal chain, starting from root causes and continuing up through the beginning of the disease itself”. To illustrate this approach they use only examples of

biological and molecular pathways. By implication, knowledge of proximal pathways has epistemological priority over knowledge of upstream factors.

Their suggestion that epidemiologists interested in upstream approaches to the eradication of poverty-associated diseases are condemned to being entwined in a Gordian knot of disagreement between economists, abdicates to economists the intellectual and moral responsibilities for studying the greatest contributor to poor health and function in populations. Given the disinterest of economists about the health consequence of economic policy, this position seems unjustifiable. This is particularly so as the evidence increases that factors that influence economic status leave an indelible mark on health status.<sup>4</sup>

What should be the role for epidemiologists in studying the socioeconomic foundations of disease in populations? Advances in the biological and molecular determinants of disease are unlikely to reduce the population disease burden of socioeconomic position. Knowledge of molecular pathways will not lead to increased understanding of the unprecedented loss of life expectancy over the last decade or so in eastern and central Europe? Research into the biological and molecular mechanisms of disease should be seen as complementary to, and not as a substitute for, a rigorous effort to understand the behavioural, social, community, and policy determinants of population health.<sup>5</sup> This research is where opportunities for interventions to reduce socioeconomic inequalities in the health of populations are primarily to be found.

Epidemiological expertise can be a critical component in establishing the health impact of social and economic policy. When epidemiologists abdicate their role in the provision of solid evidence of the impact of social and economic factors on the health of populations, they are left with incomplete knowledge of the chains and webs of causality that Rothman and colleagues' value. Potential opportunities to reduce the major cause of disease in populations are lost by an approach focused solely on downstream approaches. As the discussion of the proper role of epidemiologists matures, more will be gained by efforts to bridge the gaps between downstream and upstream approaches than by building moats between them.

George A Kaplan

Department of Epidemiology, School of Public Health, University of Michigan, Ann Arbor, MI 48109, USA

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## T-cell-depleted stem-cell transplantation for rheumatoid arthritis

Sir—Patrick Durez and colleagues (Sept 12, p 881)<sup>1</sup> report that stem-cell transplantation may be useful in the treatment of rheumatoid arthritis. The opportunity to undertake such therapies is novel and of major interest. However, reports of patients with inadequate detail add little to our understanding. The use of terms such as intractable are unhelpful without some mention of disease duration and an objective description of disease severity. The only indicator of disability that they used, the health-assessment questionnaire, is difficult to interpret because Durez and colleagues used an unconventional range which includes score 13 whereas the accepted range is 0-3.<sup>2</sup> Rather than using well-defined and accepted criteria for remission in rheumatoid arthritis they simply describe the patient as being free of arthritis after treatment.<sup>3</sup> The finding of a C-reactive protein of 2 mg/dL after treatment suggests a continuing inflammatory response albeit at a much lesser level. There should be some mention of the risks of stem-cell transplantation, both of the short-term morbidity and mortality associated with immunosuppression and any potential long-term side-effects of chemotherapy. The conclusion is optimistic in the context of a chronic disease such as rheumatoid arthritis. Trials of antirheumatic drug therapy show that remission may occur but is unlikely to last into the mid-term or long-term.<sup>4</sup> Until both safety and long-term benefit of stem-cell transplantation for rheumatoid arthritis can be shown, it should only be used in the context of a controlled clinical trial.

\*R Munro, R Madhok

Centre for Rheumatic Diseases, Royal Infirmary, Glasgow G4 0SF, UK

- 1 Durez P, Toungouz M, Schandené L, Lambermont M, Goldman M. Remission and immune reconstitution after T-cell-depleted stem-cell transplantation for rheumatoid arthritis. *Lancet* 1998; **352**: 881.
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### Authors' reply

Sir—R Munro and R Madhok's comments give us the opportunity to expand on the clinical details of our patient. In 1991, this 22-year-old woman developed a symmetric and erosive polyarthritis, which was diagnosed as rheumatoid arthritis according to the American College of Rheumatology criteria.<sup>1</sup> Laboratory investigations showed an erythrocyte sedimentation rate of 100 mm/h, C-reactive protein of 12 mg/dL, and a positive Waaler-Rose test (16 IU/mL). She was treated for several months with sulphasalazine (3 g daily), low-dose prednisone (up to 25 mg daily), and indometacin from September, 1991 to April, 1992. Because this regimen was ineffective, methotrexate was given up to 25 mg per week until December, 1993, without benefit. The patient also received repeated intra-articular injections of corticosteroids and yttrium-97 in addition to a series of intravenous pulses of methylprednisolone (250-1000 mg), which resulted in a transient improvement. Other therapies used without success included azathioprine (2 mg/kg daily), intravenous immunoglobulins, and cyclosporin (5 mg/kg daily). In 1995, she underwent total hip arthroplasty for end-stage arthritis. Despite treatment with methylprednisolone (24 mg per day), methotrexate, and cyclosporin

she required large doses of analgesics, including morphine, for joint pain.

After the transplantation of haemopoietic stem cells (HSC) on Aug 28, 1997, there was a striking improvement which was already initiated by the conditioning regimen (figure). The score on the health-assessment questionnaire calculated as described by Pincus and colleagues<sup>2</sup> decreased from 1.3 before to 0.1 after HSC transplantation. Clinical remission defined by the American College of Rheumatology criteria<sup>3</sup> was maintained at the last follow-up 14 months after transplantation. The patient has not needed any anti-inflammatory or analgesic treatment or disease-modifying antirheumatic drugs for more than 8 months.

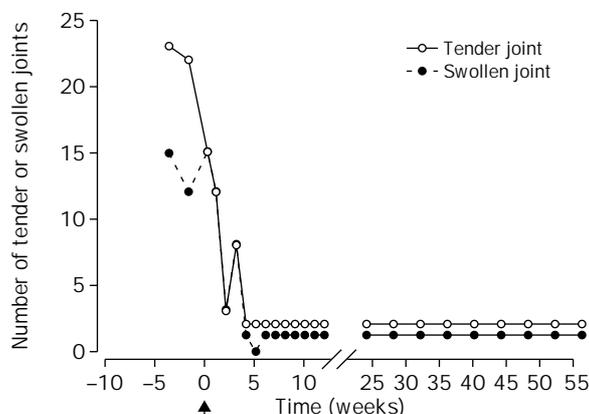
We agree that the morbidity of this procedure should not be underestimated. Indeed, our patient transiently had severe mucositis that necessitated parenteral nutrition and developed *Pneumocystis carinii* pneumonia 4 months after transplantation, which was successfully treated with co-trimoxazole. However, severe rheumatoid arthritis is associated with an excess mortality related to toxicity of disease-modifying antirheumatic drugs.

We agree that more patients and a longer follow-up are needed to establish the place of this procedure in the management of severe rheumatoid arthritis.

\*Patrick Durez, Michel Toungouz, Alain Kentos, Thierry Appelboom, Michel Goldman

Hôpital Erasme, Université Libre de Bruxelles, 1070 Brussels, Belgium

- 1 Arnett FC, Edworthy SM, Bloch DA, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum* 1988; **31**: 315-24.
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Numbers of tender and swollen joints before and after T-cell-depleted HSC transplantation