adults, for whom recent trends in mortality have not been favourable,[5] particularly for residents of deprived areas.[20] It is, of course, the people within this age group who die of causes—in particular, accidents and violence—which are not the outcome of long term biological processes and which will plausibly respond rapidly to increasing social disruption. Indeed, homicide is the cause of death found to be most strongly related to both income inequality indices used by Kennedy *et al.*[8] Similarly, more rapid responses to increasing inequality and social polarisation may be expected for psychological distress, general wellbeing, and morbidity than for chronic disease mortality. The finding of a relative deterioration in health status of civil servants anticipating job change and non-employment in comparison with those remaining in stable employment[21] provides an example of this. Inequality may make people miserable long before it kills them.

The apparently overly rapid response of mortality to changes in income distribution may have various explanations. Firstly, relatively small absolute changes in mortality are involved, with increases in life expectancy of about two years being seen in the period covered by the analyses of Wilkinson[5] and Kaplan,[9] while 30 year increases, unrelated to any systematic change in income distribution,[17 18] have been seen over the century. Major determinants of variations in mortality between countries or between areas within countries need not be the same as the major determinants of overall population mortality.

Secondly, those countries that are now experiencing the largest increases in income inequality are precisely those that have systematically underinvested in human resources for many years. The countries and governmental units which are currently those experiencing the greatest increases in inequality will contain the populations whose social and biological assets have been most undermined.

Increases in income inequality go hand in hand with underinvestment, which will reap poor health outcomes in the future. In the United States, poor investment in education and low expenditure on medical care is seen in the states with the most unequal income distribution.[9] Similarly, low birth weight is commoner in the states with the greatest inequalities, with the possible long term detrimental influences on adult health that go with this. Cross nationally, higher levels of both social expenditure and taxation as a proportion of gross domestic product are associated with longer life expectancy, lower maternal mortality, and a smaller proportion of low birthweight deliveries.[22] The relative and even absolute deterioration in social and biological assets that is occurring in increasingly unequal societies can be expected to produce poor health outcomes in the future.

The only coherent argument against redistributive social policies is that they hinder overall economic growth. Here it is supposed that the greater rewards offered to the entrepreneurially successful makes them even more successful and in turn drives overall economic growth, which, through the "trickle down" effect, ultimately benefits the poor. Cross national comparisons, however, show the reverse: if anything, countries with greater income inequalities have shown lower levels of economic growth.[23] The current government, however, continues to pay no heed to the growing evidence[5 18 20 21] that increasing income inequality is bad for the economy, bad for crime rates, bad for people's working lives, bad for infrastructural development, and bad for health—in both the short and long term.

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Potential transmission of BSE via medicinal products

Patients can be reassured that measures are in place to reduce risk

The identification of 10 cases of Creutzfeldt-Jakob disease, which seem to represent a new variant,[1] and the announcement by the Spongiform Encephalopathy Advisory Committee (SEAC) on 20 March that these cases could be linked to exposure to bovine spongiform encephalopathy, have caused great concern. Patients are worried about the risks of developing Creutzfeldt-Jakob disease not only from eating beef but also from medicinal products of bovine origin and are looking to doctors, pharmacists, and pharmaceutical companies for reassurance.

The risk of transmission of bovine spongiform encephalopathy via medicinal products depends on whether the infective agent is a human pathogen and on the level of exposure to the agent. To date, no epidemiological link has been made between any spongiform encephalopathy that is transmissible in animals and human disease, despite exposure of humans to the scrapie agent for at least 200 years. This suggests that the risk of transmission to humans is small.[2]

Measures aimed at minimising exposure to transmissible spongiform encephalopathies via medicinal products were introduced soon after the report of the Southwood Committee in 1988, in guidelines for manufacturers issued by Britain's Committee on Safety of Medicines in 1989, and essentially adopted by the European Committee for Proprietary Medicinal Products in 1992.[3] Materials were to be sourced from cattle aged under 6 months from countries free of bovine

spongiform encephalopathy or from countries where a low number of cases had been reported, provided the disease was notifiable in that country and the carcases of affected animals were destroyed and their progeny not used.

The guidelines included a classification of various tissues and body fluids according to potential risk of infectivity, based on experimental data from studies of scrapie in sheep and goats.[2 3] Brain and spinal cord were ranked most highly infective, lymphoreticular tissue less so, and most other tissues and body fluids as of low or no detectable infectivity. More recent studies of cattle with proved bovine spongiform encephalopathy have to date detected infectivity only in the brain and spinal cord with none detectable in other tissues or fluids, including gut and lung (from which some heparins are sourced); pancreas (the source for bovine insulin); bone, bone marrow, skin, and cartilage (the raw materials for gelatin); milk (from which lactose and lactulose are derived); and serum (used in vaccine production).[2 4]

The guidelines also recommended purification procedures known to remove or inactivate agents causing transmissible spongiform encephalopathies, autoclaving or treatment with sodium hydroxide or sodium hypochlorite being considered more effective than extraction by organic solvents, protein removal, or filtration, although no procedure guarantees complete inactivation of these agents.[3 5] Bioassay can now be used to test the efficacy of purification methods in removing scrapie or bovine spongiform encephalopathy agent.

In response to the recent crisis in Britain, pharmaceutical companies have made available information on the sourcing and processing of their products, together with risk assessments based on this information. In statements issued by the manufacturers of bovine insulins available in Britain, the risk to patients is assessed as negligible. With regard to gelatin, the Spongiform Encephalopathy Advisory Committee concluded in its statement of 24 March that it was safe for use in pharmaceutical and medical devices. The Association of the British Pharmaceutical Industry has provided assurance that there is no threat from medicines that have been manufactured in Britain since 1989 to the same standards as became obligatory elsewhere in the European Union in 1992.

Whether or not patients exposed to products of bovine origin before the respective measures were implemented could be incubating disease will depend in the first place on whether or not bovine spongiform encephalopathy proves to be transmissible to humans, as well as the sources and purification processes used at the time and the extent of exposure to the products in question. For some products it can be demonstrated in the laboratory that the purification or extraction procedures in use since well before the advent of bovine spongiform encephalopathy were sufficient to eliminate disease activity. The route of dosing would also be a factor, a higher dose being required to cause infection (in animal models) orally than parenterally, and subcutaneously than intravenously.[2] In reality, with the current state of knowledge, the risks in some cases are as unquantifiable as those of having eaten beef in the mid-1980s.

For patients currently receiving medication of bovine origin, which will have been sourced and manufactured according to the guidelines, there is a need to keep the perceived risks of continuing such medication in perspective. Doctors and patients will need to weigh these unknown and possibly non-existent risks against the known risks of discontinuing or changing medication; for example, restabilising diabetic patients on porcine or human insulins may prove difficult because they have a different action profile from bovine insulin. In discussing the potential risks with patients, doctors can refer to the measures described above, which have now been in place in Britain for some seven years (longer than elsewhere in Europe), and to the fact that the Spongiform Encephalopathy Advisory Committee believed that they were "sufficient with current knowledge to satisfactorily protect...human health."[2]

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Conflict of interest: Medical adviser to CP Pharmaceuticals Limited, a manufacturer of bovine insulin.

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Shortage of organs for transplantation

Crisis measures must include better detection and maintainence of donors

Of all the problems foreseen in the pioneering days of organ transplantation, a shortage of donor organs was not even remotely considered as a barrier to progress. Such has been the success of transplantation over the past two decades that organ shortage is now considered the major limitation. This week sees the publication of an extensive study by the British Transplantation Society's working party on organ donation.[1] Chaired by Professor John Fabre, the working party examined a variety of issues influencing rates of organ donation in Britain.

Clearly, the fact that fewer young people now die because of road traffic accidents or intracranial haemorrhage is a cause of donor loss that must be welcomed. However, the report highlights the fact that many medical and financial practices still mitigate against the efficient identification and recruitment of organ donors. In particular, the lack of intensive care beds means that many potential donors are not being ventilated, with the decision depending on locally devised prognostic criteria. As a result, waiting lists for renal transplantation continue to rise, putting increasing pressure on dialysis budgets. While it would be inappropriate to increase budgets for intensive care purely to reduce dialysis costs, most authorities agree that the number of intensive care beds in Britain is inadequate in comparison with other western European health services.[2 3]

Given the inadequacy of intensive care facilities, the working party recommends several initiatives to address the current shortage of donors, including interventional (elective) ventilation, greater use of non-heart beating donors, better training of staff, and better transplant coordination, all of which would require better funding.

Interventional ventilation—ventilating, solely for the purpose of organ donation, a comatose patient who is close to death from severe brain damage—runs up against legal and ethical impediments; it is imposed on an individual not for his or her own good but specifically to benefit others, and as such