

Health, inequality, and economic development

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ABSTRACT

I explore the connection between income inequality and health in both poor and rich countries. I discuss a range of mechanisms, including nonlinear income effects, credit restrictions, nutritional traps, public goods provision, and relative deprivation. I review the evidence on the effects of income inequality on the rate of decline of mortality over time, on geographical patterns of mortality, and on individual-level mortality. Much of the literature needs to be treated skeptically, if only because of the low quality of much of the data on income inequality. Although there are many puzzles that remain, I conclude that there is no direct link from income inequality to ill-health; individuals are no more likely to die if they live in more unequal places. The raw correlations that are sometimes found are likely the result of factors other than income inequality, some of which are intimately linked to broader notions of inequality and unfairness. That income inequality itself is not a health risk does not deny the importance for health of other inequalities, nor of the social environment. Whether income redistribution can improve population health does not depend on a direct effect of income inequality and remains an open question.

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1. Introduction

Any introduction to the topic of health and economic development is likely to begin with the celebrated Preston curve, Preston (1975), which shows the cross-country relationship between life-expectancy and income per head. Among the poorest countries, increases in average income are strongly associated with increases in life expectancy but, as income per head rises, the relationship flattens out, and is weaker or even absent among the richest countries. As Preston noted, if such a nonlinear relationship holds within countries, countries with a more equal distribution of income will have a higher average life-expectancy. The health of the rich is not much affected by their income, so that transfers of income from rich to poor will improve the average health of the nation. In recent work, this relationship between income inequality and health has moved from being a supporting player in the story to center stage. Not only does income equality promote health because income does more for the health of the poor, but it also serves as a marker for other desirable features of society. According to this account, equal societies have more social cohesion, more solidarity, and less stress, they offer their citizens more social support and more social capital, and they satisfy humans' evolved preference for fairness. Equal societies are healthier, an argument that is the main topic of Wilkinson (1992, 1996, 2000), as well as the collection of papers edited by Kawachi, Kennedy, and Wilkinson (1999).

This paper explores the theoretical and empirical basis for a connection between inequality and health. I shall be concerned with poor as well as rich countries, and with the links between health and income inequality at different levels of economic development. The proposition that income inequality is a health risk was first proposed for the wealthy countries on the flatter upper

part of the Preston curve, where chronic diseases have replaced infectious diseases as the main cause of mortality. But as we shall see, many of the arguments that income inequality is a health risk are as plausible for poor as for rich countries and in some cases, more so.

I devote a substantial fraction of the paper to theory, albeit illustrated with at least some of the evidence. With a few exceptions, the literature does not specify the mechanisms through which income inequality is supposed to affect health. In consequence, there is little guidance on exactly what evidence we should be examining, or whether the propositions are refutable at all. Section 2 lays out some of the possible stories, starting with the simple case in which health is affected by income, and there is no direct effect of inequality. This is sometimes referred to as the “absolute income hypothesis,” to emphasize that it is income that matters for health, not income relative to other peoples’ incomes, nor income inequality. A name that would be at least as good is the “poverty” hypothesis, that ill-health is a consequence of low income, in the sense that more income improves health by more among those with low incomes than among those with high incomes. It is important to start by establishing the full range of implications for this simple case, and in particular what role is played by income inequality. I also discuss what happens when we make health depend, not on absolute income, but relative income, and what the relative income hypothesis implies for the relationship between health and inequality.

Other theoretical accounts concern the possible effects of income on investments in health and education, the two-way link between nutrition and earnings at low levels of income, and the possible negative effects of inequality on the ability of the political process to deliver public goods. I also consider arguments that our evolutionary history predisposes us towards fairness, and sickens us when live in unequal environments. Such an account can be made consistent with

a story in which relative deprivation is a cause of ill health, and in which inequality is important across groups, but not within them. I also briefly consider the link between inequality and crime. Finally, I consider the important case in which income inequality is in part a consequence of ill-health, so that policies that reduce the likelihood of sickness, shorten its duration, or ameliorate its effects on earnings, can also narrow income inequalities. Some income inequality is a consequence of the fact that earnings cannot be completely insured against ill-health, so that better health insurance is likely to help reduce inequalities in income.

Section 3 turns to the evidence, most of which comes from developed economies. I review cross-country studies on adult mortality for rich countries and on child mortality for both rich and poor countries. A major question is whether the quality of the international data on income distribution is of sufficiently high quality to support the inferences that are being made. Better data, or at least more consistent data, are available within countries, and some of the most interesting evidence on inequality and health comes from studies looking across areas within developed countries, such as Britain, Canada, and the United States, and in a few poor countries. I also review the evidence from studies that link individual mortality and morbidity to the ambient level of income inequality.

My tentative conclusion is that there is no direct link from income inequality to ill-health, in the sense that individuals are no more likely to be sick or to die if they live in more unequal places. The raw correlations that exist in (some of the) data are most likely the result of factors other than income inequality, some of which are intimately linked to broader notions of inequality or unfairness. The fact that income inequality itself is not a health risk does not mean that inequalities more generally are not important, let alone that the social environment in which

people live is irrelevant for their health. Indeed, I shall argue precisely the opposite. But we must narrow and focus our search if we are to make the leap from correlation to policy.

In an attempt to address a relatively broad audience, I have kept the discussion as non technical as possible, relying on citations (and an Appendix) to provide formal demonstrations whenever possible. In the few occasions where there are equations in the main text, I have attempted to provide ample verbal explanation. The literature on income inequality and health is already extensive, and I have tried to tell a coherent story rather than covering everything or producing an annotated bibliography of the literature

2. Theoretical accounts of income inequality and health

2.1 Individual, group, and national health

2.1.1 Health, income and poverty

Most people find it intuitively plausible that the effects of income on health, if they exist at all, should be greater among poor people than among the rich. Although there is a great deal of evidence that the effect is not confined *only* to the poor, the intuition is supported by the evidence. Figures 1 and 2 were calculated using data from the National Longitudinal Mortality Survey in the US. This is a national follow-up study of about 1.3 million people (with about three-quarters of a million people in the public release data) who were interviewed in Current Population Surveys or in a census related sample around 1980, and whose deaths over a follow-up period of 3,288 days were ascertained by matching to the National Death Index, see Rogot et al (1992) for details. The survey collects each person's family income (within one of seven ranges) so that it is possible to link the probability of death during the follow-up to family

income and other variables, the most important of which are sex and age. For adults between the ages of 18 and 85, the log-odds of mortality is approximately linear in age, so a convenient way to summarize the data is to estimate a logit model for the probability of death in which age is entered linearly, together with a series of dummy variables for the age categories. The results are shown in Figure 1, separately for white men and white women aged between 15 and 85 at the time of the interview. This graph shows that, for both men and women, the log-odds of mortality is approximately linear in the logarithm of family income. As a consequence, when we use the results to plot the age-adjusted probability of death against income in Figure 2, we get the non-linear curves as shown. That the curve for women appears less curved than the curve for men is an insignificant consequence of the choice of scale.

The curves in Figure 2 illustrate the point first made by Preston (1975), though Preston's data were on countries, not people. The effect of income on reducing the probability of death at the bottom of the income distribution—and the bottom circle is at about the 5th percentile of family income—is much greater than its effect at the top of the distribution. As a result, if income is redistributed from the rich, whose health is not much affected, to the poor, whose health is more responsive to income, average health will improve. Other things being equal, including average income, nations (or other groups) with a more equal distribution of income will have better average group health. The same is true internationally; anything that raises the GDP of poor countries relative to that of rich countries will improve average health across the world. Within poor countries, infant and child mortality is likely to be particularly sensitive to changes in income near the bottom of the distribution so that, once again, redistribution towards the poor will reduce child mortality even without raising average incomes.

It is often useful to think of this story in terms of poverty. If a country with a high average income has a great deal of income inequality, then there are a relatively large number of people with low income whose health is poor. Although there is no poverty line in Figure 2, below which income matters, and above which it does not, it is at the bottom of the income distribution that the relationship between income and health really matters. And if a rich country has a lot of poor people, it will have low average health relative to its per capita income.

An important application of these ideas is provided by the history of mortality in Britain and the US in the 20th Century. Wilkinson (1989) looks at mortality differences by social class in Britain from 1921 to 1981, and argues that mortality fell most rapidly at times when income differentials were narrowed, particularly at times when incomes of the poor rose more rapidly than those of the rich, such as during World War II. Sen (1999, Figures 2.2 and 2.3) shows how life expectancy in England and Wales from 1901 to 1960 grew most rapidly in the decades 1911–21 (by 6.5 years) and 1940–51 (by 6.8 years), and more slowly at other times, 4 years in 1901–11, 2.4 years in 1921–31, 1.4 years in 1931–41, and 2.8 years in 1951–60. Sen shows that the decadal rate of growth of GDP per capita is strongly *negatively* correlated with decadal increases in life expectancy, and like Wilkinson, focuses on the degree of sharing during the two wars, as well as on the direct nutritional and health interventions that took place during and immediately after the second war. Both wars brought well-paying employment opportunities to many people in Britain for the first-time, including many women. Hammond (1950) discusses how wartime food policy in the 1940s brought fresh milk and vitamins to working people, even to the extent that their nutritional status actually improved during the hostilities. Reductions in income inequality during the wars, if they existed, marked an improvement in the conditions of

the working people, among whom better incomes and better nutrition would have had the largest effects on mortality.

As I shall argue in Section 3.5, it is not possible to link recent increases in income inequality in the US and Britain to mortality changes. It should be noted, however, that recent increases in income inequality, though large enough by postwar standards, are probably not large relative to earlier compressions, particularly those associated with the World Wars, see Lindert (2000) and Goldin and Katz (2001).

The inequality inducing effect of nonlinearity in the relationship between individual income and individual health has come to be known in the literature as a “statistical artefact,” Gravelle et al (1998), in order to distinguish it from mechanisms in which income inequality has a direct effect on individual health. The term is unfortunate to the extent that it suggests there is no real link between income inequality and health, and seems to carry the implication that redistributive policy cannot improve average population health. By contrast, the effect of nonlinearity is so plausible that it would be surprising if income redistribution did not improve average health, and if there were no link between income inequality and average health across population aggregates.

2.1.2 Poverty, health and the effects of inequality at different income levels

The absolute income (or poverty) hypothesis tells us a good deal about how we can expect average income and income inequality to affect population health at different levels of development. At the most obvious, the effects of per capita income mirror those of individual income, and become less important the richer is the country. Eventually, we would expect income inequality to lose its effect too, but it is not enough that *average* income be high enough,

we also need *everyone's* income to be high enough. The bottom tail of the income distribution has to be pulled up beyond the point at which income has much effect on health. Before that, there will still be poverty even in rich countries, so that income inequality will still matter as well as average income. In consequence, the absolute income or poverty hypothesis implies that, among the poorest countries, average income is what matters for population health, and income inequality is relatively less important. Among rich countries, average income is less important, and income inequality relatively more important. Eventually, neither will matter much for population health but, under plausible assumptions, the effect of income inequality relative to that of average income continues to grow as countries become richer. These implications of the absolute income hypothesis are important because it is often claimed that the differential effects of average income and income inequality on health are as described, and that the observation helps establish the case for a direct effect of inequality on health. While the finding it is certainly consistent with such an effect, it is also consistent with a simple model in which income has a larger effect on health among the poor.

The rest of this subsection is devoted to demonstrating the claim in the last paragraph in the context of a simple illustrative model. A more realistic case is developed in the Appendix. I assume that health is a quadratic function of income. Suppose that individual i lives in country s , and that her health h_{is} is a quadratic function of her family income y_{is} so that if, for convenience, I write everything relative to worldwide means, signified by overbars, we have

$$h_{is} - \bar{h} = \alpha + \beta (y_{is} - \bar{y}) - \gamma (y_{is} - \bar{y})^2. \quad (1)$$

(Note that equation (1) could be rewritten as a linear relationship between health, income and income squared, though the parameters would have to be redefined.) Income promotes health, but

by less for the rich, so that both β and γ are positive. Equation (1) is assumed to hold true everywhere, for all individuals, whether or not they live in poor or rich countries, and wherever they are in the epidemiological transition. To find out what happens to the country means, we have to average equation (1) across all i in each country s . When the last term on the right-hand side is averaged, it gives a term in the squared deviation of average income from the global average, plus the variance within each country. Looking across countries, (1) then becomes

$$h_s - \bar{h} = \alpha + \beta (y_s - \bar{y}) - \gamma (y_s - \bar{y})^2 - \gamma v_s \quad (2)$$

where variables subscripted by s are population means, and v_s is the variance of income in country s , which, in this context, is the natural inequality measure. Equations (1) and (2) demonstrate the essential point that, provided the individual equation is nonlinear, which in this case means that the parameter γ is not zero, the inequality of income matters in the aggregate relationship (2), even though it plays no part in the individual relationship (1).

These equations also allow us to be precise about the links between health, average income, and income inequality, and to see how those links change with income. The effect of average income on average health is the derivative of (2) with respect to y_s , which is $\beta - 2\gamma(y_s - \bar{y})$ which, since γ is positive, gets smaller as average income increases. The effect of inequality on population health is (minus) the parameter γ (inequality makes average health worse) which is constant. As a result, as a country becomes richer and average income rises, the effect on population health of income inequality becomes more important relative to the effect on population health of average income. Even when income is large enough for the effect of average income to be zero, the effect of inequality remains constant.

2.1.3 Separating the aggregation effect from the direct effect

One use of the curves in Figure 2 is to calculate how income inequality can be expected to affect mortality across aggregates of people, such as cities, states, or whole countries. This allows a direct link to be drawn from the degree of curvature of the income-mortality relationship in the individual data to the role of income inequality in explaining group mortality. Such calculations have been done by Wolfson et al (1998), also using the NLMS data, who then go on to show that the effects predicted by the nonlinearity are insufficient to explain the actual relationship between income inequality and mortality across the US states. But it turns out that it is not strictly necessary to go back to the individual data, and that it is often possible to calculate the effects of the aggregation from the grouped data themselves. This is important, because it gives us a way of telling whether the effects of inequality at the group level can be explained through the nonlinear effects of income alone, or whether we have evidence of a direct effect of inequality at the individual level.

The result is due to Miller (2000), who points out that the shape of the curve in the individual data is carried through to the aggregate data, albeit with additional terms in inequality, so that any additional effects of inequality due to a direct effect on individual health can be observed using only the aggregate data. Again, the quadratic case illustrates the point. Suppose that for individual i in state s , equation (1) holds as before, with the addition of a direct negative effect of inequality on health. We can write this as

$$h_{is} - \bar{h} = \alpha + \beta (y_{is} - \bar{y}) - \gamma (y_{is} - \bar{y})^2 - \theta v_s \quad (3)$$

The presence of v_s in the individual equation represents a pure effect of inequality on individual health, whose size depends on the parameter θ . When we average (3) over all individuals in state

s , we again get equation (2), but with the additional variance term carried through,

$$h_s - \bar{h} = \alpha + \beta (y_s - \bar{y}) - \gamma (y_s - \bar{y})^2 - (\gamma + \theta) v_s \quad (4)$$

In equation (4), the variance term appears from the aggregation, through the parameter γ , but also through the “direct” effect of inequality on health, the parameter θ . But because the quadratic term appears in the state level relationship (4) just as it does in the individual relationships (1) or (3), we can estimate γ from the aggregate data by looking at the quadratic term in (4). Given γ , the parameter θ can be recovered by subtraction, so that the aggregate data are potentially informative about whether the effect of inequality is an aggregation effect, or something else.

2.1.4 Relative income, absolute income, and inequality

Individual health is affected by many things other than individual income, and it is possible that the relationship between health and income itself is spurious, with income standing proxy for some other variable. An obvious candidate for such a variable is education; if conditional on education, income has no effect on health, redistributive taxes that narrow the distribution of income will have no effect on health. In the US, the relationship between income and mortality is not much affected by controlling for education, Elo and Preston (1998), Deaton and Paxson (2001a), but there are few other studies or data sets that would allow us to come to general conclusions about the separate roles of income and education.

Even if income is the crucial variable, it is possible that health is determined, not by absolute income, but by income relative to some aspiration level, or relative to the incomes of others. Easterlin (1975) long ago found evidence that happiness is independent of income in the long-run, and health may follow the same pattern. That health depends on income relative to average

incomes of one or more reference groups is the *relative income* hypothesis. This could happen in a number of different ways. One case is where relative income determines access to material goods, for example when the people who live in a town are the market for local land for housing, with the richest getting the hilltop plots with fine views, and the poorest getting the plots downwind of the smokestacks, see Gaarder (2000) who argues that this is one mechanism that sets who gets exposed to air pollution. The local housing case is an example where it is not money itself that is important, but *rank*, here determined by money. More generally, rank is important in determining how much control people have over their lives, particularly at work and, as demonstrated in the Whitehall studies, the degree of such control is an important contributor to health.

The relative income theory is consistent with an effect of income inequality, although it does not imply it. To see how things might work out, suppose that, as before, individuals are labeled i , but that we use s to index the relevant comparison or reference group. An individual's health might then be above or below the population mean depending on whether her income is above or below the average income in the reference group. Hence, with β positive, we might write

$$h_{is} = \bar{h} + \beta (y_{is} - y_s). \quad (5)$$

For the group as a whole, group average health h_s is just average health \bar{h} , which is the same for all groups, so that neither group income nor group income inequality has any effect on group health. This annihilation of the effect of income on health as we move from the individual to the group will always happen if the reference group is *within* the unit over which we are aggregating. For example, if reference groups are geographically local, average income will be unrelated to average health at the region, province, or state level. Such an account offers one explanation for

why income might be related to health within groups, but not between them.

The relative income story is also consistent with a role for income inequality at the group level, and the mechanism is the same as for the absolute income hypothesis, the nonlinearity of the relationship between income, now *relative* income, and health. Again, an obvious example is provided by the quadratic version of equation (5), in which individual health depends on income relative to group income and the square of income relative to group income:

$$h_{is} = \bar{h} + \beta (y_{is} - y_s) - \gamma (y_{is} - y_s)^2 \quad (6)$$

When we average health over individuals within each group, the first term vanishes, as before, while the second term becomes the variance of income,

$$h_s = \bar{h} - \gamma v_s \quad (7)$$

This provides us with one account of what several researchers have observed, that within states or countries, individual health is related to individual income, while between them, average health is independent of average income but is negatively related to income inequality. In Sub-section 2.5 below, I present another formulation that has similar properties.

2.1.5 The impossibility of identifying reference groups

An immediate problem with the implementation of any model of relative income is the identification of the relevant reference group. In a few cases, such as Whitehall, the reference group (or at least what is likely people's most important reference group) is a ready-made part of the design. More usually, reference groups are not clearly defined, and indeed people will often have multiple such groups, comparing themselves to their neighbors, to their co-workers, to those they meet in social and religious organizations, and to those they see on television or read about in

newspapers. One way of dealing with this is to recognize that reference group incomes cannot be observed, and to work out the effects of the omission on the relationship between the two things that we can observe, health and income. As shown in Deaton (2001), this procedure brings inequality back into the story even when it has no direct role.

Figure 3 illustrates for the simple case where there are two groups, labeled “economists” (on the left) and “doctors” on the right. Income is measured on the horizontal axis, health on the vertical axis. The two ellipses show where economists and doctors are located in terms of their health and incomes. One can think of individuals in each group as scattered within the two elliptical areas. Doctors have higher incomes than economists, and within each group, individual health depends on individual income relative to other members of the group. The two parallel steep lines show the relationships between income and health for each of the two groups. Although doctors have higher incomes than economists, their individual health is no better on average because their absolute income does not matter, only their income relative to other members of their group. Suppose that an epidemiologist analyzes the data on economists’ and doctors’ health, but without knowing which is which. When the data are pooled, the relationship between health and income is the flatter, broken line. By mixing the two groups, omitting the relevant information on group, the relationship is “attenuated” or flattened out.

Inequality comes into this story because the degree of flattening depends on ratio of within-group income inequality to between-group income inequality. This is easy to see in the Figure. If doctors and economists are moved further apart, by moving the two ellipses horizontally away from each other, the broken line will become flatter, i.e. more attenuated. If the within group inequality is increased, holding between group difference fixed, so that the ellipses are stretched

out along their individual income to health lines, the broken line will become steeper, increasing the gradient between income and health. The steepness of the gradient depends positively on the ratio of within to between group inequality. For example, in Whitehall, if there is only one reference group, that of British civil servants, the gradient of health with respect to income (or rank) is likely to be steeper than in a study containing a mixture of indistinguishable reference groups. More generally, if health depends on relative, not absolute income, and there is an increase in income inequality that increases inequality within groups more than it increases it between groups, the slope of the gradient will increase.

In Deaton (2001), I show that if health depends on relative income as in equation (5), so that

$$h_{is} = \bar{h} + \beta (y_{is} - y_s) + \varepsilon_{is} \quad (8)$$

where ε_{is} is a random term that ensures that there is a scatter around the line, then the expectation of health conditional on individual income takes the form:

$$E(h_{is}|y_{is}) = \bar{h} + \frac{\beta \sigma_w^2}{\sigma_w^2 + \sigma_b^2} y_{is} \quad (9)$$

where σ_w^2 and σ_b^2 are the within- and between-group variances of income.

2.2 Inequality, education, health, and economic growth

The literature on economic development has recently re-examined the links between inequality and growth. In the past, economists have often thought that income inequality would be good for growth. One mechanism works through saving. If only the rich save, and if saving finances investment for growth, redistribution towards the rich will be growth enhancing. More generally, the disincentive effects of redistributive taxes are themselves likely to inhibit initiative, entre-

preneurship, and risk-taking, and thus to lower output and discourage growth. The recent literature has explored a number of mechanisms that operate in the other direction, and several of these are relevant to the argument here, or can be adapted to be relevant. I draw in particular on the useful survey articles by Benabou (1996) and Aghion et al (1999); see also Bertola (2000), Bardhan, Bowles, and Gintis (2000) and Galor and Moav (2000).

In an economy where everyone can borrow and lend without restriction, each person's investment should not be restricted by each person's own resources. If someone has an investment opportunity that can be expected to earn a good rate of return, it should be undertaken; the money needed to fund it will be earned by the investment, with profit left over. If the person with the idea, or with the ability to carry out the project lacks the funds to do so, he or she can borrow money, paying it back as the project pays off. If we think of investments as construction projects, building a factory or a bridge, the financial standing of the investor will indeed often not be relevant. But if we are thinking of investments in education (human capital) or health (health capital), matters may be different, and the lack of access to borrowing on reasonable terms may prevent many poor people from making profitable investments in themselves or in their children. The inability to borrow is likely to afflict the poor more than the rich; for one thing, the poor do not have other assets that can be used as collateral. In consequence, redistribution of income—or of assets, such as land—from rich to poor may increase levels of education and health. Education promotes health—Elo and Preston (1996) estimate that, around the world, a year of extra education reduces mortality rates by about 8 percent—so that redistribution will improve average health, both directly and indirectly.

Even when loans are available in principle, many parents may be unwilling to take the risk of

borrowing substantial sums of money for their children's education. Although the investments may be profitable on average, not all will be, and not all children will be either willing or able to help their parents repay the loans taken out in their behalf. Some poor parents will simply not be willing to take the risk. Once again, redistribution may increase investment in health and in schooling.

It is also possible that even some better-off people are unwilling to borrow much to educate their children, or to go deeply into debt for expensive medical care. If so, redistribution of income or of assets from rich to poor may reduce health and educational investment among the rich almost as much as it increases them among the poor. Even so, redistribution may promote growth if the returns to investment are higher among the poor than among the rich. If poor people rarely finish primary school, and if rich people usually finish college, and if the rate of return, either in future earnings or in subsequent health, is higher to an additional year of elementary school, (Schultz, 1991, Strauss and Thomas, 1998) the redistribution from rich to poor will increase the rate of return to education as a whole, increasing both growth and average health. A similar story may hold for girls' education, which may have a higher or lower rate of return in earnings than boys' education, but certainly has a higher rate of return in terms of health.

Figure 4 shows relevant data on education for rural India, see Filmer and Pritchett (1999) for related evidence for a wide range of countries. The 52nd Round of the National Sample Survey collected data in 1995–96 on education enrolment and status of respondents. The graph shows the percentage of boys and girls aged from 7 to 12 (inclusive) that are currently enrolled in school as a function of the logarithm of total household expenditure per head, a measure of overall living standards in the household in which the child resides. The graphs are calculated using non-

parametric regression using 20,307 boys and 17,321 girls aged 7 to 12. Boys are more likely to be in school than girls, both are more likely to be in school when they live in better-off households, and the effects of additional resources are larger for girls than for boys. Although the slopes of the curve vary somewhat with per capita expenditure, they are clearly flatter to the right. In consequence, redistribution from richer to poorer households will increase the total percentage of children in school, and will increase girls' enrolment more than boys'. Because education is an input into both health and production, and because the rate of return to education is higher at lower than at higher levels of education, and higher for girls than for boys, the positive effects of redistribution will be further enhanced

Figure 5 shows some contrasting data on child vaccinations from the same Indian survey. As was the case for school enrolment, the fractions of children under 5 who have all three of the most important vaccinations is lower for poor than for rich households. However, unlike education, the slope is almost as steep among better-off than among poorer households, so that this graph indicated no major effect of redistributing income on raising child vaccination rates. Nor is there any great difference between vaccination rates between boys and girls.

These examples are only suggestive. The curves in Figures 4 and 5 make no attempt to control for other factors—such as parental education, school quality or health service provision—that are likely to be positively correlated with both household income and the outcome, so that the effects of income are almost certainly overstated. Nevertheless, the graphs illustrate that income redistribution may (or may not) increase health and educational investments in children.

2.3 Nutritional wages, destitution, and land-holdings

The nutritional wage model provides an elegant and rigorous account of how inequality affects both health and earnings while explicitly recognizing that health and earnings are simultaneously determined. The ideas go back to Leibenstein (1957), with the fundamental work by Mirrlees (1975) and Stiglitz (1976). An excellent and still useful survey of the field is Bliss and Stern (1978). That nutritional wage models can account for persistent poverty and destitution in poor countries is eloquently argued in Dasgupta and Ray (1986), (1987) and Dasgupta (1993); an excellent textbook summary is provided by Ray (1998, Chapter 13).

An important insight of the nutritional wage story is that poor workers, at risk of under-nourishment, may not be able to earn enough to buy the food that would sustain the work required. If this nutritional trap is to be avoided, two conditions must be satisfied. The first is physiological; the worker must consume enough calories to be able to do undertake whatever work he or she does for wages, as well as to sustain basic metabolism and resting bodily functions. The second condition comes from the market; the wages from earnings must be sufficient to buy the calories consumed. But these two conditions are not necessarily mutually compatible, especially when wage rates are low. At such wages, there may be no combination of work and calories that satisfies both the physiological and market conditions. In this situation, there is a critical wage rate that just enables the worker to work and survive. For any wage rate below it, long-term survival is not possible by working in the labor market.

The existence of this critical wage seriously disrupts the usual analysis of supply and demand in the labor market. Employers demand labor in the usual way and are prepared to employ more labor the lower is the market wage. Provided the wage rate is high enough, above the critical

point, higher wages elicit more labor from workers in the usual way. However, when wages are lower than the critical level, there is no labor supply, because workers can no longer obtain enough calories to do the work. As a result, if the demand for labor is less than its supply at the critical wage, the resulting unemployment cannot be eliminated by decreasing the wage rate to bring supply and demand into balance. The surplus workers are unemployed, and worse than that, must live on whatever they can beg or come by without working. They are chronically under-nourished, and cannot undercut those in work by offering to work for less because, at any lower wage, they will not be well-enough nourished to be useful to potential employers.

What are the solutions to the destitution produced by such a labor market? One possibility is to find a way of providing nutrition outside of the labor market, so that even at a low wage rate, the worker will have enough food to work. This will happen if basic nutritional needs can be met without having to work for wages, for example by growing food on the worker's own plot of land. Seen this way, the problem of destitution is a problem of landlessness, and can be resolved by redistribution of land so as to give to every family a small plot that is sufficient to guarantee their basic needs, and to enable them to participate gainfully in the labor market. Destitution is therefore a problem of maldistribution and its solution is a more equitable distribution of land.

The nutritional explanation of destitution and chronic malnutrition in India by Dasgupta and Ray is echoed by Fogel's (1994) account of the economic and health history of Europe. He argues that, even at the end of the eighteenth century in England and France, food production was so low that, given its likely distribution over people, perhaps a fifth of the population was capable of no more than a few hours of light work each day. These people were chronically malnourished, short in stature, and in life expectancy. Only the increase in agricultural product-

ivity in the nineteenth century permitted an escape from this nutritional trap, and the beginning of the transition to better health, lower mortality, and lengthening life-expectancy.

The story of nutrition and wages has much to commend it. Unlike most other accounts, it directly confronts the two-way causality between health and earnings. It also provides a general equilibrium explanation of unemployment and poor health that has an obvious relevance to poor countries now as well as to the historical record in now rich countries. It also pinpoints land reform as a policy prescription. And for our immediate purpose, it identifies the inequality of land holding as a cause of malnourishment and poor health.

Yet the theory has many critics who doubt its descriptive realism and fault its implications, see in particular Binswanger and Rosenzweig (1984), Rosenzweig (1991, 720–28) and Strauss and Thomas (1998). Workers who are trapped by their low nutrition and inability to work would devote all their energies to finding food, and would have no energy for consuming anything other than food, for saving, or even for procreation, Gersovitz (1983). Wage rates appear to be flexible downward, which the theory says is impossible. Even in the poorest economies, food is typically too cheap relative to the wage rate to make the trap plausible. For example, Subramanian and Deaton (1994) calculate that in rural Maharashtra in 1983, 2,000 calories (in the form of standard coarse cereals) could be purchased for less than 5 percent of the day wage, a finding that is consistent with the observation that poor agricultural workers in India typically eat their fill of cheap calories at the end of the work day, see also Swamy (1997). With food so cheaply obtainable, nutritional wage traps seem too easy to escape.

More broadly, the model does not draw a clear distinction between *nutrition*, which comes from the food that can be bought for money, and *nutritional status*, which depends on disease as

well as on nutritional inputs. A plentiful supply of food will not nourish someone whose drinking water and food are contaminated, and chronic malnutrition typically needs to be addressed through public health measures as well as by increasing the supply of food. This criticism applies, not only to the nutritional wage theory as an account of destitution in poor countries, but also to McKeown (1976) and Fogel's (1994) arguments about the historical importance of nutrition, see particularly Preston (1996).

2.4 Inequality, politics, and public goods

It is often argued that inequality may make it more difficult for people to agree on the provision of public goods, such as health, water supply, waste disposal, education and police. Such mechanisms have long been recognized in the literature on political economy, and a simple formal account has recently been provided by Alesina, Baqir, and Easterly (1999). The story is one of a local community whose members want to provide a public good, and who must decide how much to spend. A useful way to think of this is to imagine the members of the community evenly spaced out in a circular town, with a clinic to be built in the center. The radius of the town—or the size of the community—is a metaphor for inequality; in equal communities all live close to the center, while in unequal ones, the members are on average much further from the middle. The value of the clinic to each person diminishes with their distance from the center, so that the people at the edge of town value it less than those who live in the middle. The average (or median) value of the clinic is therefore higher the more compact is the community. If the size of the clinic is decided by voting, more specifically by the wishes of the median voter, a larger clinic will be built in the more compact town, because the median value of the clinic to its users

is higher. More generally, when people's preferences are heterogeneous, goods held in common are less valued on average, and fewer of them will be provided.

Although they note the potential application to income inequality, Alesina, Baqir, and Easterly think of their model as applying to racial divisions in the United States, and to ethnic fractionalization more widely, a variable that often appears to have negative consequences in cross-country growth studies. In the context of the cities and counties of the US examined by Alesina et al, ethnic fractionalization is closely related to the fraction of the population that is black, which is *positively* related to total spending, but *negatively* related to the shares in spending of "productive" public goods, such as health, roads, and education. For health, the total effect offsets the share effect, so that the absolute amount of health spending is positively associated with fractionalization. Income inequality is included in the models, although the results are not presented in the paper. However, I understand from private communication with one of the authors that the effects vary from model to model and that there are no robust negative effects of inequality on either total spending or its distribution. It should also be noted that Putnam and his collaborators (1983), in their study of social capital in Italy, also see equality as an important element of the civic community.

While I know of no work in developing countries that links the provision of public health resources to income inequality, Szreter (1988) has provided a fascinating account of politics and sanitation in Britain in the middle of the 19th century, see also Easterlin (1999). The urbanization of population associated with the industrial revolution led to a sharp reduction in public health, with mortality higher in cities than in the countryside, and a *decline* in overall life expectancy. Urban populations often had no access to clean water, and no facilities for disposal of human and

other wastes, which were allowed to accumulate as a perpetual hazard to health. Crowding aided the transmission of infectious diseases, some of which can only be sustained in populations above a critical size. Pollution from smoke and other factory discharges contaminated the atmosphere and the environment. Yet many cities, in Britain and in Europe, were slow to address these problems, even when the necessary policies were well understood. Although the germ theory of disease was generally accepted only after 1870, earlier explanations, such as the “miasma” theory, also emphasized the importance of cleaning up the environment. And while money is always a factor limiting public construction, these were periods of relatively rapid economic growth. Indeed, the coexistence of rapid economic growth and mortality increase (as well as a decrease in stature) during this period is regarded as something of a puzzle by economic historians, see Haines and Kintner (1999), Schofeld and Reher (1991) and Fogel (1996), who are typically so confident of the link between health and incomes that they often use measures of the former—such as stature—as reliable indicators of the latter, Steckel (1994).

Szreter (1988) argues that the key to understanding the mortality transition in England lies in local politics. Although the industrializing cities were in fact well supplied with fresh water, it was used for commercial purposes, not supplied to homes. And the new entrepreneurial class, rich though they were, saw no point in spending each others’ money for public sanitation which had no obvious commercial benefit. It was only after political reform, and particularly the limited political emancipation of working men, that new political coalitions could develop that made sanitation and public health a priority.

This is a story of 19th Century England, not of the world today, and it is about politics, not about income inequality. Yet central to the plot are the Reform Acts and their (limited) extension

of democracy which may or may not have (directly) reduced *income* inequality, but certainly reduced *political* inequality. This increase in equality was not only valuable in its own right, it subsequently helped reduce other inequalities, in health, and perhaps eventually in incomes, in accord with the general thesis of Sen (1999). In a more contemporary context, it is hard not to see political action, or at least the lack of it, as one of the reasons behind the low level of provision of schools and clinics in Indian villages. Chattopadhyay and Duflo's (2001) work in India shows how the mandated representation of women as leaders of village councils (in a randomly selected third of all Indian villages) has led to small but perceptible gains in public goods important to women and children, particularly water, fuel, and roads (whose construction provides employment opportunities for women) though, perhaps surprisingly, male leaders invest more in education.

2.5 Evolution, equality, and relative deprivation.

2.5.1 Evolution, stress, and inequality

It might easily be supposed that hierarchic, unequal societies are an inevitable part of the human condition. Yet for the vast majority of our evolutionary history, humans lived in hunter-gatherer groups that were not only not hierarchic, but aggressively egalitarian, Erdal and Whiten (1991). As has long been argued, perhaps first by McKeown (1976, 1979), human health is maximized when we live under the conditions under which we evolved, pursuing regular exercise (walking 10 to 15 miles a day, as foragers and hunters did), and eating low fat, low-salt, low-meat, sugar-free, high fibre, largely vegetarian diets. By the same token, given that hierarchies and social inequalities were unknown for most of our history, modern inequalities are likely to be a hazard

to our health. This argument is forcefully and eloquently put by Wilkinson (2000).

That foraging groups were egalitarian appears to be widely agreed. Such arrangements could perhaps have come from lack of a technology for storing food. When a kill has been made, and the meat is too much to be consumed at once, sharing and subsequent reciprocity are the only mechanisms that can turn meat today into meat tomorrow. Members of groups that used such mechanisms would therefore have a survival advantage over members of those who did not, so that a preference for sharing, fairness and reciprocation may be evolved attributes. With the invention of settled agriculture, with the associated ability to fill and hold food in granaries, as well as to build herds of animals, egalitarian and reciprocal sharing was less efficient, and could give way to hierarchies within which rich and powerful individuals could dominate others. Although such systems and their industrial successors are vastly more productive than is foraging, the benefits come at the price of a nagging and health-compromising outrage over the loss of equality. And while humans will perhaps evolve to suit this new environment, we have only given up foraging for a very short time, only 50,000 or so years of our perhaps million-year history. Adaptation to the new environment has its benefits, in terms of production, longevity, health, and population size, but it has a lingering cost that prevents our health from reaching its full potential.

Wilkinson and others have begun to weave together a plausible story of the processes that support such an account. Psychosocial stress is the main pathway through which inequality affects health. Wilkinson draws a contrast between societies in which relationships “are structured by low-stress affiliative strategies which foster social solidarity” on the one hand, and societies characterized by “much more stressful strategies of dominance, conflict and submission.

Which social strategy predominates is mainly determined by how equal or unequal a society is.” (Wilkinson, 2000, p. 4). Equality is seen as a precondition for the existence of stress-reducing networks of friendships, while inequality and relative deprivation are seen as compromising individual dignity, and promoting shame and violence. At the same time the biological mechanisms through which chronic stress compromises health are beginning to be understood; excellent surveys can be found in Sapolsky (1998), Brunner and Marmot (1999) and Wilkinson (2000, Chapter 2.)

2.5.2 Income inequality and relative deprivation

The story outlined above is persuasive in many respects. That the social environment in which we live helps determine our health is surely right, and the effects of psychosocial stress are now well-documented in both human (particularly Whitehall civil servant) and animal populations. Yet it is less clear why income inequality is the only, or even the prime villain in the piece. I have made an attempt to explain why it might be so in Deaton (2001). In this account, I treat income relative to other members of a reference group as the key variable, and hypothesize that stress on each individual is proportional to the total amount of income accruing to community members with higher incomes, expressed in units of group mean income. Figure 6 illustrates. It shows the cumulative distribution, $F(x)$, of income x for the reference group, and calculates the mortality risk, on the vertical axis, for a person with income x , on the horizontal axis. This person is hurt by all the incomes of the people above him, to the right of x . Consider one such person, with income y . The burden that the people around y place on x is in proportion to their distance above x , or the distance $y - x$, so that for the group in the strip from y to $y + dy$, the contribution to the

mortality risk of someone with income x is the shaded area, which is the product of the number of people, $dF(y)$, and the income difference $y - x$. The same sort of effect operates for all incomes above x , so that the total risk is the total area above the curve from point A up to the highest income.

This area, divided by the mean of group income, was proposed by Yitzhaki (1979) as a measure of relative deprivation, a term frequently used by Wilkinson in his discussions of social stress. Although the meaning here is more specialized (and precise) than that intended by Wilkinson, the formalization appears to be well-chosen, in that it generates many of the effects that he proposes. If relative deprivation is drawn as a function of income, we get curves like those in Figure 7, drawn for a selection of states in the U.S. The theory can be tested by matching these curves to the relationship between mortality and income for each state.

The measure of relative deprivation defined above and illustrated in Figures 6 and 7 has a number of interesting properties. First, relative deprivation, and thus mortality risk, is lower for people with higher income. Second, the rate at which relative deprivation declines with income is lower at higher incomes. This convex shape is then consistent with the relationship between mortality risk and income that we find in data such as the NLMS, and which is illustrated in Figure 2. Third, the amount of relative deprivation at any given level of income depends on the amount of income inequality in the group. Indeed, the amount of relative deprivation for someone with mean income is itself an inequality index, the relative mean deviation, sometimes called the Pietra or Robin Hood index. More unequal “states” in Figure 7, like the District of Columbia and California, have higher relative deprivation curves than Maine or Florida, where income is more equally distributed. Fourth, if we average relative deprivation over all the members of the

group, so as to get group mortality risk, we get another measure of inequality, the gini coefficient. This conforms almost exactly to Wilkinson's (1998) statement that "income inequality summarizes the health burden of individual relative deprivation."

In summary, the relative deprivation theory of mortality risk has three important implications: (i) within groups, mortality risk is a convex and declining function of income; (ii) conditional on an individual's relative income, inequality matters for individual health; and (iii) for groups, mortality risk is independent of group income, but is directly related to the gini coefficient. The results of testing this theory are postponed to Section 3, where they can be presented in the context of other, related work on mortality differences across the US states.

2.6 Inequality and crime

Crime is often treated as a public health issue. This is clearly appropriate in the case of homicide or other crimes against persons, but there is also a great deal of violence and stress associated with other crimes, such as theft or drug dealing. Much of the work on crime and inequality is unabashedly empirical, but there is some theory to provide guidance. The seminal paper on the economics of crime is Becker (1968), who proposed that criminal activity should be seen as the outcome of an optimal choice in which the expected benefits of crime, money from theft, or the satisfaction of murdering an enemy, are weighed against the expected costs, including the costs of legitimate activity foregone, as well as the costs of punishment weighted by the probability of apprehension. Criminals are no different from everyone else so that we would all be criminals given the right incentives. Becker's theory was used by Ehrlich (1973) to examine crime rates across the states of the US, who then needed an operational measure of the benefits of crime

relative to the opportunity cost in terms of legitimate activity in the labor market. Ehrlich argued that the benefit of theft is likely to be related to average (median) community income or wealth. Because only those with low incomes, and thus low opportunity cost of legitimate activity, were likely to find the benefits of crime greater than the costs, Ehrlich included in his regression the fraction of the population whose incomes were below half median state income, calling this a measure of income inequality. (It would more usually be thought of as a poverty measure.)

In general, there is no reason to suppose that only the poor will commit crimes. Instead, we might model the net benefit of a contemplated crime for person A against person B as proportional to the income difference between them. If so, the incentive for person A to commit a crime is proportional to the sum of all such income differences above him, which takes us back to the measure of relative deprivation proposed in the previous subsection. Within any community, the average of such incentives over all people is simply the gini coefficient of income, a result that might have been approached directly by noting that the gini coefficient is the ratio to the mean of (half the) the average of all income differences between people. If the rate of apprehension and mean income is the same in all communities, it would therefore be plausible that higher inequality communities would show higher crime rates.

The probability of apprehension will generally depend on the level of crime, which may obscure any simple relationship with income inequality. Indeed, as Wittenberg (2000) shows, the interaction of crime and crime prevention can generate multiple equilibria, including outcomes where there is a great deal of inequality, but very little crime. When the potential victims are rich enough to make attractive targets, they may also be rich enough to afford extensive security measures. Whites in apartheid South Africa were well protected against the crime that might

have been expected given the extraordinary levels of income inequality between them and the Black majority population. At the other end of the spectrum, murder is common among hunter-gatherer bands precisely because, in these egalitarian structures there is no central control, no hierarchic structure, and no police.

One might also challenge the basic premise of Becker's analysis, that criminals are no different from the rest of us, but simply face different costs and benefits. That everyone will turn to crime given sufficient incentives may be doubted, as in Sen's vehement denial that inequality leads to crime, "crime needs some assertion and confidence and you can't do it when you are down on your knees. It has been seen that even during famines, which are periods of massive inequality, there is no increase in crime. I have seen the Bengal famine, and even there, people would die outside sweet shops, but not a glass would be broken," Business Standard (2001).

Nevertheless, as we shall see in Section 3, the data often show a quite robust correlation between homicide and inequality.

2.7 Income inequality as a consequence of ill-health

It is clear that not only does economic status influence health status, but also that health status affects economic status. Unfortunately, and with the notable exception of the nutritional wage model, the literature has tended to concentrate on one or other direction of causality, without paying enough attention to the interaction between the two, something that deserves serious study in its own right. At low levels of income and nourishment, the effects of disease and food (meaning income) should not be thought about separately. Inadequate nourishment compromises the immune system and makes it less able to resist infection; ensuring adequate income is likely to

be important even for a strategy that focuses mainly on public health. At the same time, although income may be sufficient to guarantee adequate nutrition, nutrition will not improve nutritional status if the body is unable to absorb the food because of chronic diarrhea or intestinal infection. The provision of public health measures, clean water and waste disposal, is necessary even for a strategy that focuses mainly on growth and income. Even more obviously, and in rich countries as well as poor, the ability to work is compromised by ill-health. People with low incomes may be more likely to contract a disease, they are less able to spend money to mitigate its consequences, and they may find it difficult to comply with complex and time-intensive medical regimes or to seek medical attention in the first place.

When interactions are important, the distribution of income will depend on the distribution of health. Any measure that reduces the spread of health conditions across the population will narrow the distribution of income. In particular, anything that helps people recover more rapidly from an illness will reduce the persistence of ill-health, which reduces the long-term variance of health across the population. Better insurance arrangements, or better and more widespread clinics are obvious candidates to reduce such persistence, and so will not only improve population health (if they work at all), but also improve the distribution of income. Clean water, whose lack affects the poor more than the rich, will improve the incomes of the poor relative to the rich, and reduce income inequality. Malaria eradication campaigns and vaccination drives will have the same effect, not only improving population health, but also narrowing the distribution of income. This would be true even in a Lewis world in which there is an unlimited supply of labor at the subsistence wage. Better health may not improve the wage rate, and cannot do so under the Lewis assumption, but it can enable more people to work at that wage.

Income differences *between* countries are also affected by their differences in population health. In consequence, the speed at which new health technology is transmitted from the industrialized to developing countries narrows their relative population healths as well as their relative incomes, see Sachs (2001). Current debates about international pricing of drugs and patent protection are important in this context. Whether the faster transmission of drugs and vaccines will improve the distribution of income *within* poor countries depends on the prevalence of disease within the income distribution. For AIDS, prevalence in poor countries is (still) higher among the rich, while for malaria or tuberculosis, prevalence is higher among the poor. In the past, health innovations have often *widened* health inequalities when first introduced; examples are better sanitation a century or more ago, Preston and Haines (1991), tobacco and lung cancer more recently, and child health improvements in contemporary Brazil, Victora et al (2000). Faster transmission of best-practice healthcare may therefore widen the income distribution within the receiving countries, at least in the first instance.

3. Empirical evidence on inequality and health

3.1 Measuring income inequality

Income inequality is not an easy thing to measure so that, before looking at the evidence on the effects of inequality, it is worth starting with a discussion of what we know about inequality itself. There are both conceptual and practical issues. Comprehensive treatments of the theory of inequality measurement were developed in the 1970s by Atkinson (1970) and Sen (1973), the latter updated in Foster and Sen (1999). Although there are a number of axioms on the nature of inequality that are broadly accepted, these will not always be sufficient to permit us to make

unambiguous inequality rankings between *any* two distributions of income. Instead, the axioms induce a “partial ordering” whereby we can sometimes rank one distribution as more unequal while, in other cases, we can judge the inequality of distributions only by choosing a specific inequality measure, with different measures giving different results. In particular, different inequality measures give a different emphasis to different parts of the distribution. For example, the gini coefficient is more sensitive to inequality (or to measurement error) at the top of the income distribution, whereas measures that work with the logarithms of income, such as the Theil measures, or the variance of logarithms, are quite sensitive to inequality at the bottom. Although neither the conceptual issues nor the choice of inequality indicator are probably the most important issues in the health literature, it should be noted that many of the indexes that are used in the public health literature do not satisfy even the generally accepted axioms. For example, the Robin Hood index (more usually known as the relative mean deviation), Kennedy et al (1996), is unaffected by transfers between individuals on the same side of the mean. If a transfer program were to transfer incomes from those just below the median to those near the bottom, the Robin Hood index would not change, even though there would have been a real reduction in inequality (and very likely a decrease in mortality risk too.) Perhaps most of the public health work uses as its inequality measure the share of income accruing to the bottom x (often 50) percent of the population. Once again, transfers within the bottom x percent, or within the top $1 - x$ percent, will leave the measure unaffected, even though such transfers are capable of having a substantial effect on income inequality more broadly.

There has also been a good deal of discussion about the appropriate definition of household income, and in particular the treatment of household size. A standard procedure in the economics

literature is to “equivalize” household income by dividing income by some measure of household size, either household size itself, or the number of equivalent adults, for example the number of adults plus half the number of children, or the square root of the total number of people in the household. Such per equivalent measures attempt to capture the resources available to each person in the household, and recognize that, at the same level of income, members of a larger household are worse off than members of a smaller household. (Even so, it should be noted that there is some evidence, Elo and Preston, 1996, that, conditional on family income, larger family size may not increase mortality.) When income inequality is calculated, it is also important that equivalized income be assigned to individuals, and that inequality be calculated over persons, not households. These apparently technical details can sometimes have serious effects on the measurement of income inequality, and their treatment in the public health literature has often been cavalier.

Conceptual problems are dwarfed by measurement problems. Income itself is hard to measure, and the difficulties multiply when we try to measure income inequality. Measurement error in income, even if it has little effect on the measurement of mean income, will inflate the measured variance and measured income inequality. The measurement of income is sensitive to survey design, particularly to the choice of the reference periods for income (longer reference periods give lower measured inequality), and to exactly how the income question is asked. The degree of disaggregation of income categories is important, as is whether incomes are reported as a number, or in a set of predefined ranges such as less than \$10,000, \$10,000 to \$20,000, and so on, up to some open ended top category, such as more than \$50,000. The choice of cutoff points for the ranges is important, particularly the top band which effectively limits the highest income

that can be reported. Some surveys permit people to report negative incomes (losses from business activities) and some do not. Some surveys collect data on income, and some on consumption; the latter is almost always less unequally distributed than the former. The response rate from surveys varies over space and time, and richer households are typically less likely to agree to participate, in many cases because they live in communities where the enumerators cannot reach them, see Groves and Couper (1998). In rich countries, and in some not so rich countries, response rates have been falling over time, perhaps in response to the increasing competition from market researchers. There are also differences across countries in the degree to which people are prepared to cooperate with government surveys. Response rates in the US are typically much higher in the US than in Western Europe.

Because different countries use different survey instruments, and have different survey protocols, useful cross-country comparisons of income inequality require detailed knowledge of the specific surveys. Similar issues sometimes arise with comparisons over time within one country, even where the statistical service is of the highest quality. Specifically, the US Census Bureau, in the summer of 2000, decided that the large increase in household income inequality between 1992 and 1993, which it has previously presented as real, was in some unknowable part due to changes in survey methodology, particularly changes in the highest level of income permitted in the questionnaires, as well as the introduction of computer-aided interview technology, Jones and Weinberg (2000). In consequence, the US no longer has a consistent continuous time series of household income inequality. That the US is worse (as opposed to more transparent) than other countries seems unlikely.

International data on income inequality come from a number of standard sources. Perhaps the

best is the Luxembourg Income Study (LIS) which contains information on the distributions of disposable income for 25 (wealthy) countries over a period of 20 years, although not all countries have data for all years, see Gottschalk and Smeeding (2000, pp. 273–4). The LIS permits access to the micro data from broadly comparable income data for the covered countries. Note that the underlying surveys do *not* use the same questionnaire, so that the comparability is not perfect, nor are response rates the same for all countries. Nevertheless, the data are well-documented and have been widely analyzed, so that their properties are well understood. Some authors have taken income inequality data for industrialized countries from other, non-LIS sources, such as Sawyer (1976); these are now superceded by the LIS.

Matters are a good deal more difficult for income distribution data from the large numbers of developing countries that are not covered by the LIS. For many years, popular sources of income distribution data were Jain (1975) and Paukert (1973), which are essentially compendia of inequality estimates then available in the World Bank and International Labor Office, respectively. In more recent years, research on international patterns of income inequality has been transformed by the availability on the World Bank website of the inequality data assembled by Deininger and Squire (DS) (1996). These data, which have seen widespread use, contain more than 2,600 observations on gini coefficients (and many quintile shares) for more than 100 developed and developing countries for dates between 1947 and 1994. To be included in the DS data set, estimates have to come from an identifiable source, be national in coverage, and be based on either consumption or income. (Which comes from which is identified.) A subset of the observations are labeled “high-quality” and these have been widely (and mostly uncritically) used in a large number of papers, including papers on income inequality and health. Much of the high-

quality data comes from industrialized countries, so that many researchers interested in development have used at least some of the “non-recommended” data.

While DS’s data and documentation are a great improvement over what was previously available, they do not support the uncritical use that has been made of them, as shown in an important study by Atkinson and Brandolini (1999). Atkinson and Brandolini focus their attention on the subset of the DS data for the OECD countries, for which there are good, well-documented surveys (including the LIS) which can be used for comparison. DS’s “high-quality” estimates do not do well in this comparison, either across countries, or in some cases over time within countries. For example, DS shows Sweden as one of the more unequal countries in the OECD, with more income inequality than the UK, whereas in the LIS (as reported in Gottschalk and Smeeding, 2000), Sweden has the lowest income inequality and the UK the highest apart from the US. In some cases, such as Germany, the DS time series of inequality is quite different from that computed directly from the surveys. Although the DS data may be more reliable for poor countries than for rich, it is unlikely, especially since the poor countries contain a much larger fraction of the data that are not endorsed by DS themselves.

We are currently in the position of not having any consistently reliable set of data on income inequality outside the countries covered by the LIS. This is in spite of the existence of the 50 or so surveys that have been collected under the aegis of the World Bank’s Living Standard Measurement Survey (LSMS) which was set up in 1980 with the original purpose of generating comparable data on income distribution for a wide range of countries. While the LSMS surveys are broadly comparable, the questionnaires are not identical across surveys, and some have differed a great deal. Nevertheless, a research program within the World Bank could be set up to

use the LSMS surveys, together with the other unit-record data sets available, to generate a series of inequality measures for which the quality guarantee could be based on a detailed analysis of both survey protocols and the individual data.

Research on inequality and health has also used data on measures of inequality for areas within countries. In principle, such measures are less problematic, if only because they are usually calculated from national surveys using a uniform survey instrument so that, even if there are errors, the patterns across areas may not be much affected. One problem is sample size, especially for small areas. Inequality measures are usually less precisely estimated than means so that, for example, the US Bureau of the Census publishes estimates of mean income by state using the Current Population Survey, which has a sample size of around 50,000 households each year. However, it publishes inequality measures by state only for three year moving averages. Several developing countries also have regular, national household surveys that are large enough to support considerable disaggregation; India, Indonesia, and Pakistan are examples. The Indian National Sample Survey (NSS) collects detailed consumption data from more than 120,000 households every five years or so, and these surveys are designed to be representative for more than 70 regions of the country. Yet even this survey does not support the measurement of income inequality for districts, the level at which the Indian census publishes much of its data on child mortality.

Most household surveys have a two-stage stratified design in which, at the first stage, primary sampling units (PSUs) are randomly selected with probability proportional to population size. These PSUs are typically small geographical units, such as villages or census tracts. Within each PSU, the same number of households are selected so that, over the two stages together, each

household in the population has an equal chance of being selected into the survey. In some studies, investigators have calculated measures of local income inequality based on the households in each PSU. This procedure is obviously dangerous when there are only a few households in each PSU and, even when this is not the case, respondents within PSUs are sometimes not randomly selected, so that the relationship between the sample estimate and its population counterpart cannot be assessed. Note too that PSUs are selected for *statistical* convenience, not analytical meaning, and frequently do not correspond to any sensible definition of a community.

3.2 Cross-country studies of income inequality and health

Cross-country studies have played an important part in the literature on income inequality and health. Preston's (1975) seminal analysis looked at international patterns of GDP and life expectancy, and it was on the basis of his findings that Preston suggested that there should be a negative relationship between income inequality and health. Rodgers (1979) and Flegg (1982) were early studies that followed Preston's lead, explicitly looking for (and finding) effects of income inequality on mortality. Rodgers used the Paukert (1973) data for 56 (unnamed) countries and, controlling for income and other variables, found hazardous effects of inequality on life-expectancy at birth, life-expectancy at age 5, and on the rate of infant mortality, with the last only significant in the developed countries in the sample. Flegg (1982) looked only at child mortality, and found significant effects of income inequality on child mortality in developing countries using the Jain (1975) data. These authors, like Preston, thought of the nonlinearity in the relationship between income and health as the basis for their results, and did not propose any direct effect of income inequality at the microeconomic level. Such a direct effect was found in

by Waldmann (1992) who used UN and World Bank sources, supplemented by income inequality data from Jain (1975), to investigate infant mortality on a cross section of up to 57 developing and developed countries. As expected, he found that, conditional on mean income, the share of income going to the poorest 20 percent of the population decreased infant mortality, and more surprisingly, that the share of income going to the top 5 percent *increased* infant mortality. This is a direct effect of inequality; the infant mortality rates among the poor increase when the rich get richer, even when their own incomes do not suffer.

Perhaps the single most cited finding in the literature is Wilkinson's (1992, 1994, 1996) demonstration of a relationship between income inequality and life expectancy across a number of industrialized countries, not only in levels but, more impressively, in changes over time. Countries, such as France and Greece, that narrowed their income distributions by reducing relative poverty, increased their life-expectancies, while those, such as the UK and Ireland, whose income distributions widened, fell behind, Wilkinson (1996, Figure 5.4). Wilkinson interprets these results as showing that, as countries become wealthier and move through the epidemiological transition, the leading cause of differences in mortality moves from material deprivation to social disadvantage. Material deprivation provokes poverty and infectious disease, while social disadvantage provokes stress and chronic disease.

Later research has cast considerable doubt on the robustness and reliability of many of these findings. As expected, one of the main difficulties lies in the unreliability of the data on income inequality. For example, using the Deininger and Squire data, Gravelle, Wildman and Sutton (2000) fail to replicate Rodgers (1979) results for developed and developing countries, while Mellor and Milyo (2001) use a sample of 47 developing and developed countries in 1990 and

find that the positive correlation between the gini coefficient and infant mortality vanishes once secondary school enrolment is controlled for, while the negative correlation between income inequality and life expectancy is eliminated by controlling for income per head. Mellor and Milyo also fail to replicate Wilkinson's results for developed economies. Although the DS data have their own problems, the original results are clearly not robust. The same is true of Waldmann's findings; Baumbusch (1995) replicated Waldmann's analysis using data of the same vintage, but found that income accruing to the top 5 percent *reduced* infant mortality once the data were updated from the 1993 edition of the World Bank's *World Development Report*.

The single most important and careful study of the LIS countries is by Judge, Mulligan and Benzeval (1997), who emphasize the poor quality of the data in previous work, and use the LIS data in their own examination of life expectancy and infant mortality in Australia, Belgium, Canada, Finland, France, Germany, Ireland, Italy, Luxembourg, The Netherlands, Norway, Sweden, Switzerland, the U.K., and the U.S. In these data, which are the best international data currently available, the correlation between the gini coefficient and life expectancy is -0.17 , insignificantly different from zero, and neither the gini nor other measures of income inequality are close to significance in any of the regressions explaining life expectancy. The situation is somewhat different for infant mortality rates, where there is a significant positive (i.e. harmful) effect of the ratio of the 90th to the 10th percentile. This measure of inequality exerts a significant effect in several of the regressions, though it becomes insignificant when controls are added for the negative effects on mortality of female labor force participation. In these data, the raw correlation between infant mortality and inequality is driven largely by the US, which is very unequal and has relatively high infant mortality.

If these results are not entirely definitive, it is because the LIS data, although better than any other, are neither fully comparable nor fully accurate. The debate between Wilkinson (1998) and Judge, Mulligan and Benzeval (1998) has focused on differences in response rates across the LIS surveys, and their possible effect on the results. It is also possible that, as with the difference between infant mortality and life-expectancy, there will be links between specific causes at specific ages and the plausibly associated measures of inequality, see for example McIsaac and Wilkinson (1997). Yet, it is surely time to agree that there is currently no evidence that income inequality drives life expectancy and general adult mortality within the industrialized countries. It remains to be seen whether this means there is no relationship, or whether there is a relationship that is being obscured by still inadequate data. Judgement on that depends a good deal on whether there exists a relationship between income inequality and health in other contexts, on which more below.

That, conditional on income, there should be a cross-country relationship between infant mortality and income inequality, at least in poor countries, is both theoretically plausible, and rather better supported by the (admittedly inadequate) data that are available. The plausibility comes from recent work from the World Bank which, following the methodology pioneered by Filmer and Pritchett (1999), has used Demographic and Health Surveys around the world to construct a synthetic measure of wealth, which is then used to explain infant mortality rates, see Gwatkin (2000) for an overview. The measure of wealth is an index based on the ownership of various durable goods, and while the measure is undoubtedly correlated both with actual wealth and income, we have no way of calibrating the transformation, and thus of using the results to relate *income* to child health. Nevertheless, the results show very strong gradients in child health,

with infant mortality rates heavily concentrated at the bottom of the distribution. Wagstaff (2000) uses nine (mostly) LSMS surveys from developing countries to calculate child mortality rates by quintile of equivalent consumption, and shows that child and infant mortality rates typically decline most rapidly between the bottom and second quintile. Whether these results imply that infant mortality rates are convex in income depends on the degree of convexity of the relationship between income and the asset index, and on the density function of equivalent consumption, but that infant mortality is concentrated at the bottom of the income distribution seems likely. Yet there is also some evidence on the other side. In particular, Murthi, Guio, and Drèze (1995) find very little effect of poverty on child mortality across districts in India once they control for other factors, most importantly female literacy and urbanization.

To the extent that the DS data are accepted, there is a good deal of empirical evidence from developing countries linking infant and child mortality to the DS measures of income inequality conditional on the level of GDP per head and a range of other variables, for example in Pritchett and Summers (1996), Filmer and Pritchett (1999) and Hales et al (1999). Whether this evidence extends to adult mortality and life expectancy is difficult to know, not only because of the data difficulties with income inequality, but because of the quality of the data on adult mortality. Few poor countries have complete registration systems for deaths, so that good evidence on adult mortality (or life expectancy at age 5, for example) is hard to come by for most developing countries. In a few cases, such as India, there are sample registration surveys, and some data on adult deaths can be gleaned from the Demographic and Health Surveys. But for most countries, data on life expectancy are extrapolated from the data on infant mortality rates, and contain little additional information. An exception to this generalization comes from the countries of Eastern

Europe and the former Soviet Union, where life expectancy has been falling as income inequality has increased (see Marmot and Bobak, 2000, Fig. 3, which shows a 12 countries correlation coefficient of -0.63 .) As is widely recognized, the Eastern European experience is difficult to interpret because so much else has been going on, so that it is hard to isolate the effect of income inequality.

Finally, there are a number of cross-country studies that link other health outcomes to income inequality. Steckel (1995) finds a relationship between human stature (a measure of cumulative nutritional status) and income inequality on a sample of developed and developing countries using the income distribution from Jain (1975). Over (1999) looks across cities in the developing world and finds that the US Census Bureau's estimates of HIV infection rates are positively related to the DS measures of countrywide income inequality. He interprets his findings in terms of upper income men demanding the services supplied by lower income women, and these results perhaps come closest to providing substance to Farmer's (1999) contention that disease occurs along the "fault lines" in the income distribution. Gaarder (2001) argues that income inequality is likely to worsen the health consequences of pollution because the poor have lower baseline health and are therefore more susceptible. She includes the gini coefficient in a meta-analysis of previous estimated effects of particulate concentration on mortality at various sites around the world and finds significant positive effects. Fajnzylber, Lederman, and Loayza (2000) find a significant relationship between DS gini coefficients and both homicide and robbery rates for a group of 45 (for homicides) and 34 (for robberies) developed and developing countries. A good deal of this is driven by Latin American countries, where both crime and inequality are very high. Using data on 17 countries in the Americas, and gini coefficients from DS, Mujica et al (2000)

confirm the positive correlation (0.55) between homicide and income inequality, but find a negative correlation (−0.78) between suicide rates and income inequality.

3.3 Within country area studies of income inequality and health

As skepticism has grown about the international relationship between income inequality and health, attention has switched to studies within countries, particularly of mortality and income inequality across the states of the US. Two studies, by Kaplan et al (1996), and by Kennedy, Kawachi, and Prothrow-Stith (1996a, b), both published in the *British Medical Journal*, and inspired by Wilkinson's (1992) cross-country work, found a relationship across the states between various measures of income inequality and age-adjusted all cause mortality, as well as a number of other measures, including infant mortality rates, deaths from cancer, coronary heart disease, homicide, as well as disability, low birth weight, and crime. Kawachi and Kennedy (1997) established that the results were robust to the choice of inequality indicator, while Lynch, Kaplan and Pamuk (1998) extended the results to 282 Metropolitan Statistical Areas (MSAs) in 1990, finding that the loss of life from income inequality "is comparable to the combined loss of life from lung cancer, diabetes, motor vehicle crashes, HIV infection, suicide, and homicide in 1995." Kawachi, Kennedy and Prothrow-Stith (1997) argue that income inequality works by reducing social capital, in particular the degree of trust between people, a (very poor) state-level measure of which is constructed from the General Social Survey. Such an account is very much in the spirit of stories of psychosocial stress within unequal social structures. In support of this explanation, Wolfson et al (1999) estimate the degree of nonlinearity in the income to mortality curve using the NLMS (as in Figure 2) and show that the effects of income inequality on state

mortality rates are too large to be explained by the nonlinearity argument alone so that there must be some direct effect of income inequality on individual mortality. The implications of these results for economic policy have not gone unnoticed, see for example Kaplan and Lynch's (2001) editorial in the *American Journal of Public Health* entitled "Is economic policy health policy?"

These within nation results do not suffer from the same data problems as do the international comparisons. Income inequality is usually measured from incomes collected in the census, which is administered in the same form to all households in all states. Nor is there any question about the existence of the correlation. Figure 8, taken from Deaton (2001) shows a typical scatter plot between the log odds of age-adjusted mortality (the log of the ratio of the fraction dying to the fraction not dying) on the vertical axis, and the variance of the logarithm of household income per equivalent, with equivalents defined as 1 for adults and 0.5 for children aged 18 and less. The District of Columbia is included as the 51st state and, although it is an outlier in the sense of having higher income inequality and higher mortality than any state, it lies along the regression line defined by the other observations.

Nevertheless, there are serious questions about whether the correlation between income inequality and mortality is robust through time, and whether it comes from the effects of income inequality or some other factor that is correlated with it. Mellor and Milyo (2001) use data for the 48 continental states from five census years, 1950, 1960, 1970, 1980, and 1990, and reproduce the strong hazardous effect of the gini coefficient on all cause mortality when only year dummies, the age composition of the state, and median income are included as controls. The inclusion of controls for the average level of education in each state eliminates the significance of the gini coefficient and, once the authors include controls for the fractions of people in each state who are

urbanized and who are black, the gini coefficient attracts a negative sign, though one that is not significantly different from zero. Similar reversals are found for the fraction of births that are low-birthweight while, over the five decades, there is no relationship across states between deaths from cardiovascular disease, from malignant neoplasms, or from liver disease. Indeed, for the first two, income inequality has a negative and significant relationship with deaths once controls are entered for income, education, race, and urbanization. Only for homicides and, to a lesser extent, infant mortality and deaths from accidents, is the gini coefficient a risk factor conditional on the other controls. Mellor and Milyo also subject the hypothesis to a much more stringent test, looking at the relationship between 10 and 20 year changes in mortality and the corresponding changes in income inequality. This is perhaps *too* severe a test because it places a great deal of weight on the timing of the link between income inequality and mortality. Even so, it is worth noting that, with one exception, none of the income inequality to mortality relationships survives the test. The exception is homicide, where the relationship with income inequality is well-determined and holds over time as well as in the cross-section.

Related robustness issues are reported in my own work, Deaton (2001). A particular concern is the pooling of data across racial or ethnic groups with different incomes and different mortality rates. In the US, blacks have higher mortality rates than whites, and lower incomes, so that states with a high fraction black tend to have higher mortality rates as well as higher income inequality. As can be seen from Figure 8, such states tend to be predominately in the South where many other special factors are likely to operate. If data are pooled for 1980 and 1990, the log odds of age-adjusted mortality responds to the gini coefficient with a coefficient of 1.7 for males, and 1.1 for females. In the same regression, the mean of the logarithm of equivalized income reduces

mortality for men, but barely significantly, and not at all for women. To illustrate the size of the effect of inequality, the 1990 gini coefficients for Louisiana and New Hampshire were 0.47 and 0.40 respectively, which would account for a 12 percent difference in mortality rates, more than half of the difference shown in Figure 8.

If we now confine the calculations to white mortality alone, so that we no longer have the mechanical effects described above, the coefficient on the gini drops to 1.1 for men and to 0.6 for women, about a third lower than for all-race mortality. Nevertheless, these effects remain significantly different from zero, and still show that inequality is a health hazard for the white population. If we recalculate the gini coefficients so as to measure only inequality among whites, the effects are further reduced, to 0.6 for men, and 0.4 for women, and only the former is (marginally) significantly different from zero. This result means that the effect of inequality on whites comes, not from the inequality of white incomes, but from the inequality between whites and blacks, raising the suspicion that the effect has more to do with race than with income inequality. Such a suspicion is borne out by controlling for the fraction of the population that is black in each state. It turns out that a high fraction black raises mortality rates among both males and females (note that these are *whites*) and that conditional on race, income inequality has no effect on mortality. At this stage, it is unclear *why* the fraction black should exert such a strong effect on white mortality (black mortality is also higher in states where there are relatively many blacks), though it might be argued that it is itself some sort of marker for the inequality that characterizes race relations in the US. Even so, the effect is *not* one that works through *income* inequality; once the fraction black is included in the regression, the gini coefficient has no effect.

There is an obvious concern here that I have simply replaced one invalid variable, income

inequality, with another, racial composition, and that both stand proxy for something else. This is particularly the case with the state data, where there are at most 51 observations (or 102 observations if we pool data from 1980 and 1990), and where it would be easy to confound racial composition (or income inequality) with geographical factors, especially given the peculiar role of the South. Nevertheless, Deaton and Lubotsky (2001) show that the results carry through to the 287 MSAs that can be consistently identified between 1980 and 1990. These data can be used to replicate the findings of Lynch, Kaplan, and Pamuk (1998), and to show that, once again, the inclusion of racial composition eliminates (and sometimes even reverses) the effect of income inequality. And because there are so many more MSAs than states, it is possible to work within regions, and to show that whether we look at cities in the South, or cities in other regions, and conditioning on city average income, white mortality is higher in cities where the fraction of blacks is higher.

Once the fraction black is controlled for, the cross-state and cross-city mortality results help elucidate another puzzle, which is why there is such a strong relationship between income and mortality in the individual data, and so little at the state or city data. Controlling for the fraction black, the state or city mean logarithm of equivalized income has a significant negative effect on mortality rates, particularly for men. Although the effects are not as large as in the individual data, the results suggest that the differences might well be eliminated by controlling for a fuller range of other factors.

Controlling for racial composition also makes the results consistent with the findings of Ross et al (2000) who find that, in contrast to the US, there is no relation between income inequality and mortality for the 10 provinces and 53 metropolitan areas of Canada, where race is not the

salient issue that it is in the US. Yet that there should be no relationship across the states between mortality and income inequality, either in the US or Canada, is surprising in light of the arguments about nonlinearity. For Britain, there appears to be no area study on income inequality and health, though Ben-Shlomo, White and Marmot (1996) find that mortality in the 8,464 wards of England is affected not only by an index of deprivation based on household characteristics, but also by the within-area dispersion of the deprivation index. Again, this is what is to be expected if the deprivation measure is more closely linked to mortality among high deprivation people. Chiang (1999) looks at mortality rates in the 21 counties and cities of Taiwan in 1976, 1985, and 1995 using household survey data to calculate measures of income and income inequality. He finds strong protective effects of income in 1976 and 1985, and little effect of income inequality, but finds that the situation is reversed in 1995, at which date income inequality is a hazard, and income has no effect. Chiang interprets his findings as support for Wilkinson's idea that income is important at low levels of income, and income inequality at high levels of income which, as we have seen, is also consistent with a nonlinear effect of income, and no direct influence of inequality. Regidor et al (1997) find no relationship between (a nonstandard) measure of income inequality and the prevalence of long-term disability across the 17 regions of Spain.

It is widely believed that there is a link between income inequality and crime (including homicide) in the U.S. I have already noted Mellor and Milyo's (2001) finding that homicide was the only negative health outcome that was robustly linked to income inequality in their tests, and such findings have consistently appeared in the literature since Ehrlich (1973), see Hsieh and Pugh (1992) for an oft-quoted review and meta-analysis.

There appear to be few relevant studies from developing countries, even where it would be

possible to do so, for example in the work on fertility and child mortality in India by Murthi, Guio, and Drèze (1995) and Drèze and Murthi (2001). However, Drèze and Kheera (2000) find that homicide rates across India are *unrelated* to measures of consumption inequality, but are positively associated with the fraction of “missing” women. Although the authors do not make the point, a link between homicide among men and the shortage of women invites a socio-biological explanation in terms of mating behavior. Pena, Wall and Person (2000) find that infant mortality risks are higher among the poor, and higher still when the poor live in relatively wealthy neighborhoods, which is consistent with a negative role for inequality.

3.4 Studies of income inequality and health using individual data

Studies using individual level data face different data problems from either the national or the area studies. Yet they have the advantage of being able to look for a direct effect of income inequality without having to handle the effects of inequality that work through aggregation. But there are compensating difficulties. Because mortality is a rare event, large sample sizes are required to give enough deaths to reliably estimate mortality rates. At the same time, those few health related surveys that follow people from interview to death are typically very poorly endowed with economic information, including incomes. Nevertheless, there are several surveys in the US that have been used to look at the determinants of mortality at the individual level. The National Longitudinal Mortality Study starts from data collected in the Current Population Survey, mostly around 1980, and then uses the National Death Index to check whether members of responding households are dead by each follow up date. Currently, around 1.3 million people have been tracked for up to a decade. In principle, the CPS provides excellent and detailed

economic information, but many of the rounds used for the NLMS were not the March surveys, when income data are collected, and so contain only rudimentary information on household incomes.

There are two other US health surveys with later merges of death certificate data, the National Health Interview Survey (NHIS), which interviews around 50,000 households every year, and the National Health and Nutritional Examination Survey (NHANES) the first round of which surveyed more than 14,000 people between 1971 and 1975, for whom information on deaths has been merged up to 1987. A final source of mortality data is the Panel Study of Income Dynamics (PSID), which has followed around 5,000 households (and their children and split-offs) since 1968. Because this is a panel survey, returning regularly to each household, deaths are reported by surviving family members. All four of these surveys have been used to look at the relationship between mortality, income, and income mortality.

Sweden also has a data set that works in same way as the NLMS in the US, though with even more comprehensive data. Since 1975, Statistics Sweden has interviewed around 7,000 individuals each year in its Survey of Living Conditions, and these people have been linked, not only to the national death statistics, but to income information from the national income tax statistics, see Gerdtham and Johannesson (2001).

A second line of work has used, not mortality, but self-reported measures of health status. The questions are included in a large number of surveys, if only because they are asked easily and quickly. They ask respondents to rate their health on a five point scale from “poor,” to “fair”, to “good” to “very good” and “excellent.” Many investigators convert this to a binary indicator of poor health, corresponding to the “poor” and “fair” categories; such an indicator has been

validated as a powerful predictor of subsequent morbidity and mortality, even conditional on a physician's examination, Idler and Benyamini (1997). These questions are included in the NHIS, in the Behavioral and Risk Factor Surveillance Study (BRFSS) and, from 1995 onwards, in the Current Population Survey.

The interpretation of the individual studies, and of the extent to which they support a link from inequality to health depends a great deal on who is doing the interpreting. Nevertheless, there is general agreement that the results from these studies are weaker and more ambiguous than the area studies. For example, Lochner et al (2001), using the NHIS and merged mortality data, find only a small effect of state income inequality on mortality (relative risk of living in the top five most unequal states compared with the 10 most equal states of 1.12). This effect, which is estimated with controls for family income and the state level poverty rate, is only statistically significant for near-poor whites. Fiscella and Franks (1997) find no effect of PSU level inequality on the probability of dying in the NHANES follow-up, but there are real questions about whether their measure of inequality—the share of income accruing to the bottom 50 percent of the population—can be adequately measured from the PSU data in the NHANES itself, within which respondents are not randomly drawn. Daly et al (1998) find no effect of state level income inequality on individual five year mortality rates using data from the PSID. As I shall explain in more detail below, state level income inequality also has no detectable effect on individual mortality in the NLMS.

Studies by Soobader and LeClere (1999) who use the NHIS with merged census county level inequality data, Kennedy et al (1998) using the BRFSS merged with state level inequality data, Fiscella and Franks (2000) using the NHIS, and Mellor and Milyo (1999) using the CPS with

inequality data from the CPS at various geographical levels, all find some effects of income inequality on self-reported morbidity. But the estimated effects are typically modest, and Mellor and Milyo show that their effects are removed once controls are introduced for income and its square, as well as for fixed state effects. (Note again that this last is a severe test; Mellor and Milyo only have three years of CPS data, so they are effectively demanding a link between changes in morbidity and changes in income distribution between 1995 and 1997). But LeClere and Soobader (2001) also demonstrate considerable fragility in the results, showing that the effects seem to work only for whites aged 18–44 in high inequality counties, and middle-aged whites in very high inequality counties. There are no effects for other whites, nor for non-Hispanic blacks.

I have already noted the paper by Wolfson et al (1999) showing that the degree of curvature in the NLMS (Figure 2) is insufficient to explain the large effects of income inequality on mortality at the state level. In Deaton (2001), I address more directly the role of state income inequality on mortality in the NLMS. The NLMS distinguishes seven income groups, so that at the first stage of the analysis, I use a logit model to estimate the log odds of dying during the 10 year follow up as a linear function of age including dummy variables for each of the seven income groups. These logits are estimated for white males and females separately, using data only for those aged 18 to 75 at the time of first interview. (The log odds of mortality is approximately linear in age over this range.) In order to conduct a state level analysis, each of these models is fitted to data for a single state, thus allowing inequality—or any other state level effect—an unrestricted effect on the relationship between mortality and income. The first-stage produces numbers for each state like the points shown as circles in Figure 1 so that, at the second

stage, it is possible to examine whether these points are higher in states where income inequality is higher. Note that this two-stage procedure is as general as a single stage model in which individual mortality is linked to state level data on income inequality.

My original concern was to test the model of relative deprivation presented in Section 2.5.2 above. This was done by comparing the effects of each income group in each state with the predicted values from computing a relative deprivation curve as illustrated in Figure 7. Within states, the relative deprivation story does well, outperforming a simple model in which income itself accounts for the differences across income groups. However, the relative deprivation model accounts for essentially none of the variation in mortality *across* states which, given the theory, means that the gini coefficient does not predict interstate mortality differences in the NLMS data. This finding is supported (even without controls for income) using the 1.3 million observations in the full NLMS. Table 13 in Rogot et al (1992) shows no correlation across the states between age adjusted mortality and income inequality, a finding that is in direct contradiction with Figure 9 and with the findings listed at the outset of Section 3.4 above. This contradiction is resolved (at least in part) by the demonstration that, in these individual level data as in the aggregate state-level data, the fraction black in each state is a powerful predictor of white mortality.

Once again, there is no direct effect of income inequality. Because the racial composition of states is such a strong predictor of mortality in the aggregate state-level and MSA data, as well as in the individual data from the NLMS, it would be interesting to discover whether the Lochner et al (2001) findings on mortality in the NHIS follow-up can also be attributed to racial composition; given their partition of states into inequality groups, it seems likely.

Taking income and mortality together, the Swedish data used by Gerdtham and Johanesson

(2001) are probably of higher quality than anything currently available in the US. Gerdtham and Johansson use the 284 municipalities of Sweden as their communities, and examine individual mortality for 41,006 individuals aged between 20 and 84 who were interviewed between 1980 and 1986 and whose mortality was followed-up until the end of 1996. Mortality is assessed relative to individual income, community income, and community income inequality, with the latter two measured from the survey data itself, a procedure which is subject to the reservations raised above. As in all similar studies, individual income is strongly protective, even allowing for education and a host of other variables, including initial health status, but neither inequality nor mean community income appeared to have any effect. The last result is evidence against the relative income hypothesis so that, once again, we are led back to the original model in which health is an increasing nonlinear function of absolute income.

3.5 Inequality and mortality decline in the US and Britain

A final source of evidence comes from examining whether the increase in income inequality in the 1980s in both Britain and the U.S. can be linked to mortality. Wilkinson (1996) argues that for Britain, mortality rates for infants and for young adults fell less rapidly after 1985 than would have been the case had income inequality remained constant. Figure 5.10 of Wilkinson (1996, page 97) plots a time series of mortality, not only of infants, but also of children and young adults, and shows that the sum of age-adjusted mortality rates fell less rapidly after 1985 than it did in the decade from 1975 to 1985. These findings, together with the corresponding evidence for the U.S. have recently been examined in Deaton and Paxson (2001b). Their results are as follows.

There were large increases in income inequality in both Britain and the U.S. in the 1970s and 1980s. In both countries, inequality in family and household income increased from the early or mid-1970s until around 1990, with (arguably) little increase but certainly no decline since. By the early to mid 1980s, inequality had risen to new postwar highs and continued to increase, at least until 1990. As pointed out by Wilkinson, the rate of decline of infant mortality was particularly rapid in the decade from 1975 to 1985, and less rapid thereafter. The same is true in the US, though the period of rapid decline starts somewhat earlier, in the late 1960s, and finishes earlier, around 1980. In both countries, the rate of decline of mortality rates among young adults has slowed steadily, and by 1985 mortality rates are either flat or actually rising. For infants and young adults taken together, the rate of mortality decline has therefore been a good deal slower in recent years than in the period before the increase in income inequality.

Even so, it is unlikely that income inequality has much to do with these mortality trends. First, the episodes of rapid decline in the infant mortality rate are episodes, not trends. Prior to the periods of rapid decline in each country, progress was slower, with a rate of decline comparable to that after the end of the episode. Yet income inequality was not high prior to the onset of the rapid decline, so that in neither country since 1950 has there been a consistent relationship between income inequality and the rate of decline of infant mortality. Second, we know a good deal about the causes of the decline in infant mortality, much of which can be attributed to declines in perinatal mortality through new techniques for preventing the deaths of low-birth weight babies. These techniques diffused more quickly in the US than in the UK, so that the rapid decline in mortality started first in the US and its possibilities were more rapidly exhausted there. There seems no reason other than coincidence to link the timing of this exhaustion to the

rise in income inequality. Third, among young adults, much of the increase in mortality is attributable to HIV/AIDS, for which the rise in income inequality in the mid-1980s is not the cause. Finally, if we look at mortality rates of adults aged 45 and above, there is a period of unusually rapid mortality decline (particularly although not exclusively for men) that began around 1970 (again a little earlier in the US), and continues to the present. So if income inequality is hazardous for the young, it is protective for their elders! Once again, a more convincing explanation lies in the increased use, first in the US and later in Britain, of life-saving technologies for dealing with cardiovascular disease, angioplasty, coronary artery bypass grafts, and the use of clotbusting drugs and even aspirin.

4. Summary and conclusions

The stories about income inequality affecting health are stronger than the evidence. Judging by the explosion of interest and of citations, there is a strong appeal to the idea that, before the epidemiological transition, income determines mortality while, after it, income inequality determines mortality. That in poor countries, income protects against poor sanitation, unhealthy working and living environments, poor nutrition, and a plethora of infectious diseases. That in rich countries, where these evils are but distant memories, income inequality is an indicator of the quality of social arrangements, of stress, and of mortality. Yet, as we have seen, even if it is true that, at higher income levels, income inequality becomes more important as a cause of death, there is no need to assume that the relationship between income and mortality changes with economic development. If it is poverty, not inequality, that drives mortality, so that income has a much bigger effect on health at low than high incomes, average income will eventually cease to

be associated with poor health, while the effects of inequality will endure for much longer because, even in rich economies, there are some who are not so rich. Income inequality will continue to affect mortality until everyone ceases to be poor, which happens long after *average* income has risen out of the range of poverty.

But it is *not* true that income inequality is a major determinant of population health. There is no robust correlation between life-expectancy and income inequality among the rich countries, and the correlation across the states and cities of the US is almost certainly the result of something that is correlated with income inequality, but that is not income inequality itself. The rapid increases in income inequality in the 1980s have not been associated with any slowdown in the rate of mortality decline. Studies of individual mortality and income inequality show no link, except for one survey where the estimated effects are small and are confined to one population group. Infant and child mortality in developing countries is primary a consequence of poverty so that, conditional on average income, income inequality is important only because, given average income, inequality is effectively a measure of poverty. But it is low incomes that are important, not inequality, and there is no evidence that making the rich richer, however undesirable that may be on other grounds, has any effect on the health of the poor or their children, provided that their own incomes are maintained. The only exception to these generalizations is perhaps the case of homicide, where income inequality itself appears to play a genuine role.

These conclusions are not different from those of earlier commentators, particularly Judge (1995), Judge, Mulligan and Benzeval (1997) and Doorslaer and Wagstaff (1999). Yet they must not be misinterpreted. They do not imply that the social environment is not important for individual health, let alone that individual health is determined by individual characteristics and the

provision of personal medical care. We know from Whitehall and from other studies that positions in hierarchies matter, perhaps through an ability to control one's life, but in any case through some mechanism that works through relationships with other human beings. My own empirical results have drawn attention to another social factor, the effects of racial composition on mortality, something that remains to be fully investigated. And I have emphasized several other cases where reductions in deprivation in one dimension, whether it be land ownership, democratic rights, women's agency, or income, will bring benefits not only in and of themselves, but also to the relief from other deprivations, in this case particularly the deprivation of ill-health. This is of course Sen's (1999) theme in *Development and Freedom*, that relief from any one of a number of interlinked deprivations, each of which is an important unfreedom in its own right, helps promote relief from the others. This is quite different from a story in which income inequality is the principal actor and main villain.

My conclusions carry a number of implications for the direction of future research. The most obvious is that attention should be directed away from further attention to income inequality per se. I have already emphasized the puzzling role of racial composition on mortality in the US, about which nothing is understood. More generally, the urgent need is to refocus research to investigate the role that income plays in promoting health. We need to know much more than we do about whether the effects of income come from income itself, or from correlates, such as education, wealth, control, or rank. We need to know why income is so important in the individual level studies, and so apparently unimportant at the aggregate level. If income is indeed directly protective, we need to know whether the effect is really nonlinear and by how much, because it is this, and not any direct effect of income inequality on health, that determines

whether and by how much income redistribution can improve population health.

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Appendix: Income, income inequality and mortality in economic development

In Section 2.1.1, I used a simple quadratic version of the absolute income hypothesis to illustrate how income and income inequality were likely to have different effects on population health at different levels of average income. The quadratic model is useful, but not very plausible, if only because increases in income will eventually reduce health. A more realistic model links individual health to a latent variable, itself a function of income, and assumes that death takes place when this latent variable falls below some critical value. Under suitable assumptions, such a formulation yields explicit predictions for the probability of death for individuals, as well as for communities, and the resulting equations provide a better way of linking population health to population income and income inequality.

As before, I write h_{is} for the health of individual i in population s , and assume that health is linear in the logarithm of individual income,

$$h_{is} = \alpha + \beta \ln y_{is} + \varepsilon_{is} \quad (10)$$

where the random term ε_{is} is assumed to be normally distributed with mean zero and variance σ^2 . The individual dies when h_{is} falls below the critical level c . The probability that this happens, or the probability of death, is written p_{is} which is

$$p_{is} = p(\text{death} | y_{is}) = \Phi\left(\frac{c - \alpha - \beta y_{is}}{\sigma}\right) \quad (11)$$

where Φ is the distribution function of the standard normal. Suppose that, within each country s , the logarithm of income is normally distributed with mean μ_s and variance v_s . We can then rewrite (10) as

$$h_{is} = \alpha + \beta\mu_s + \beta(\ln y_{is} - \mu_s) + \varepsilon_{is} \quad (12)$$

so that p_s , the fraction of people dying in the state, or the probability of death conditional on the state mean of log income can be written

$$p_s = p(\text{death} | \mu_s) = \Phi\left(\frac{c - \alpha - \beta\mu_s}{\sigma\sqrt{1 + \beta^2 v_s / \sigma^2}}\right) \quad (13)$$

Equation (13) links population mortality rates to the population mean of log income, μ_s , and the variance of log income, v_s .

If we differentiate, first with respect to μ_s , and then with respect to v_s , we obtain

$$\frac{\partial p_s}{\partial \mu_s} = -\beta\varphi\left(\frac{c - \alpha - \beta\mu_s}{\sigma\sqrt{1 + \beta^2 v_s / \sigma^2}}\right) \quad (14)$$

and

$$\frac{\partial p_s}{\partial v_s} = \left(\frac{\beta^2}{\sigma^2}\right)\left(\frac{c - \alpha - \beta\mu_s}{\sigma(1 + \beta^2 v_s / \sigma^2)}\right)\varphi\left(\frac{c - \alpha - \beta\mu_s}{\sigma\sqrt{1 + \beta^2 v_s / \sigma^2}}\right) \quad (15)$$

where φ is the pdf of the standard normal. As mean log income μ_s goes to infinity, the pdf goes to zero, so that the derivative in (14) goes to zero when income is sufficiently large. The same is true for the derivative with respect to income inequality in (15) because $\varphi(x)x$ goes to zero as x goes to infinity. As countries become sufficiently rich, neither population mean income nor income inequality have any effect on population mortality rates. However, if we take the ratio of (15) to (14), the pdf functions cancel, and the ratio grows linearly and indefinitely with mean log

income. Hence, as claimed, the effect of income inequality relative to the effect of income is larger among richer countries.

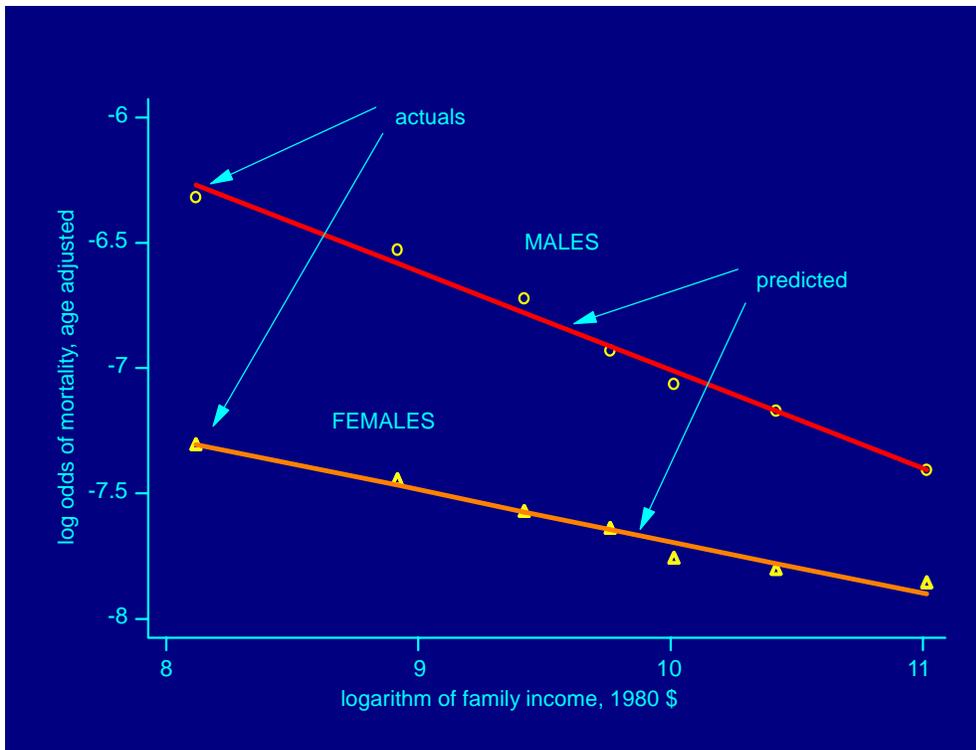


Figure 1: Age adjusted log odds of mortality and the logarithm of family income, National Longitudinal Mortality Study

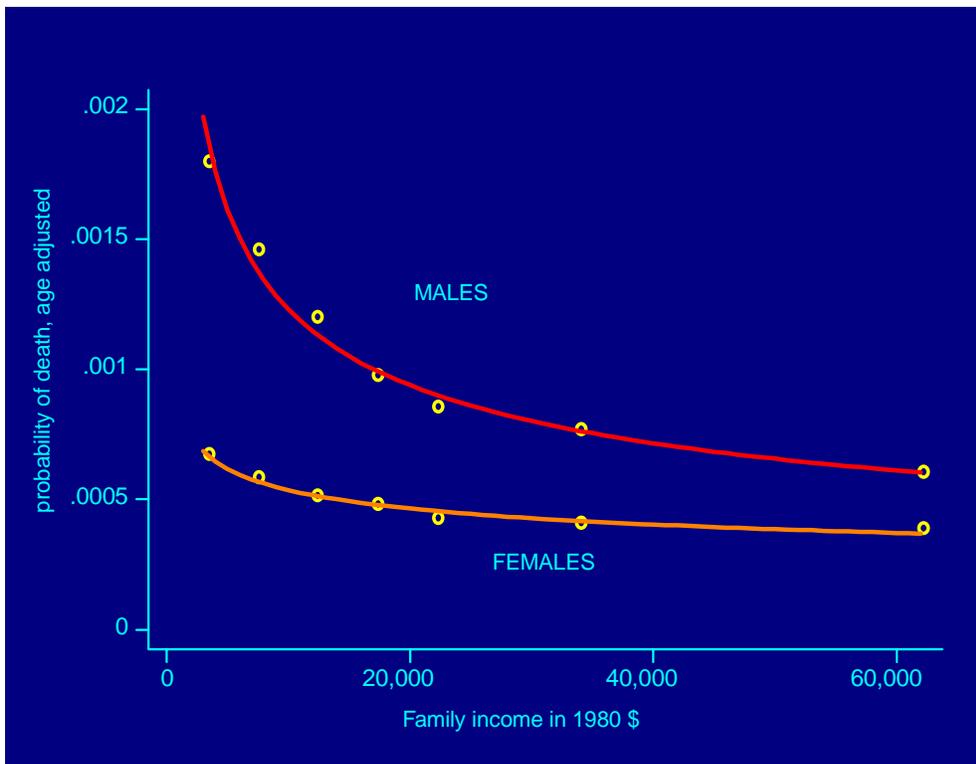


Figure 2: Age adjusted probability of death and family income, National Longitudinal Mortality Study

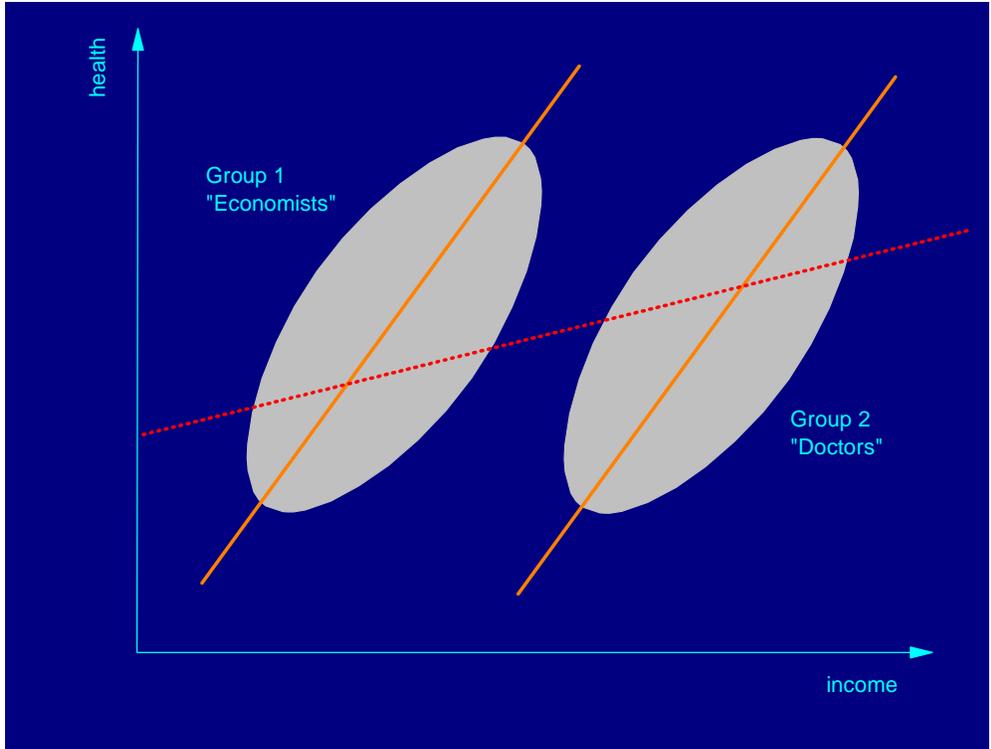


Figure 3: Health and income in two reference groups: the effects of within- and between-group inequality on the gradient

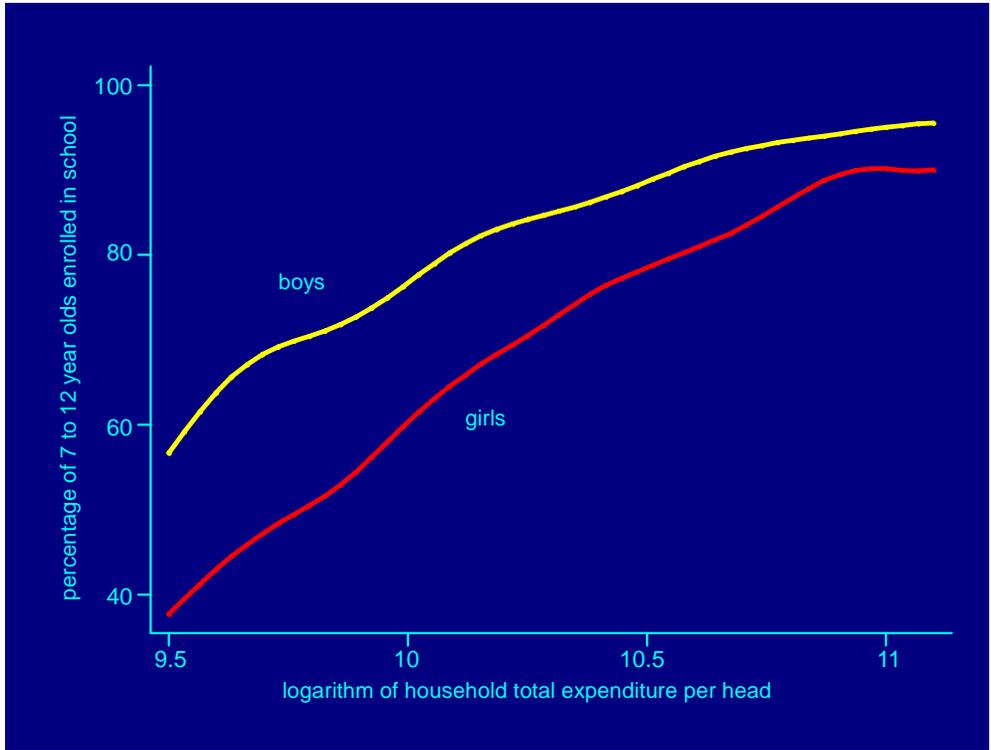


Figure 4: Percentages of children aged 7 to 12 who are enrolled in school, rural India, National Sample Survey, 1995–96

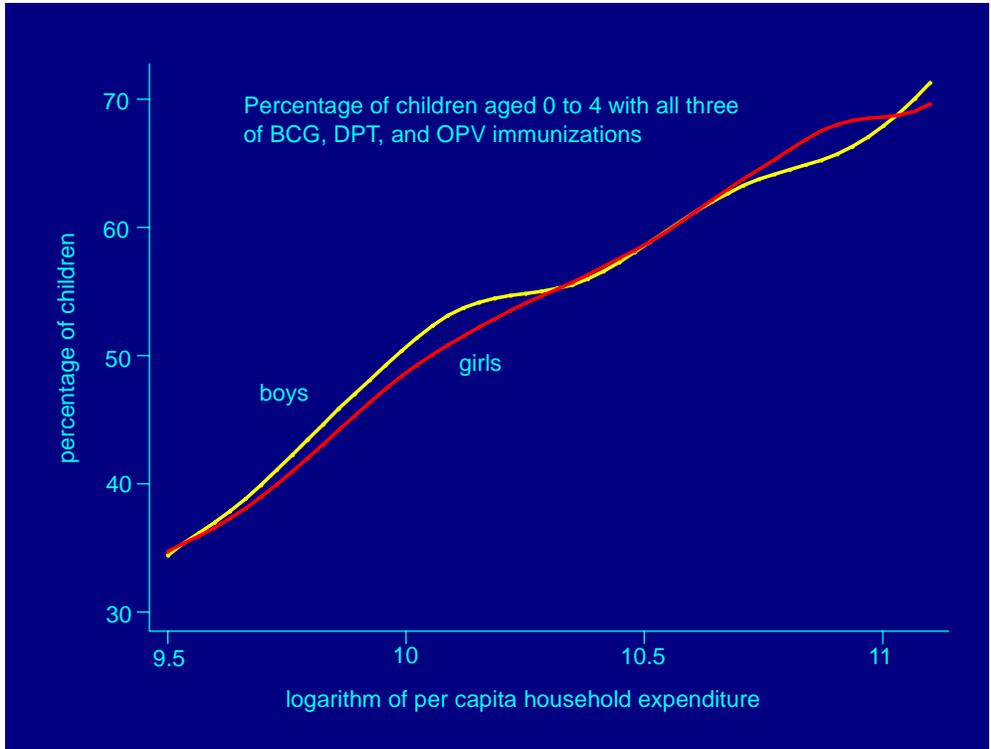


Figure 5: Percentages of children under 5 who are vaccinated, rural India, National Sample Survey, 1995–96.

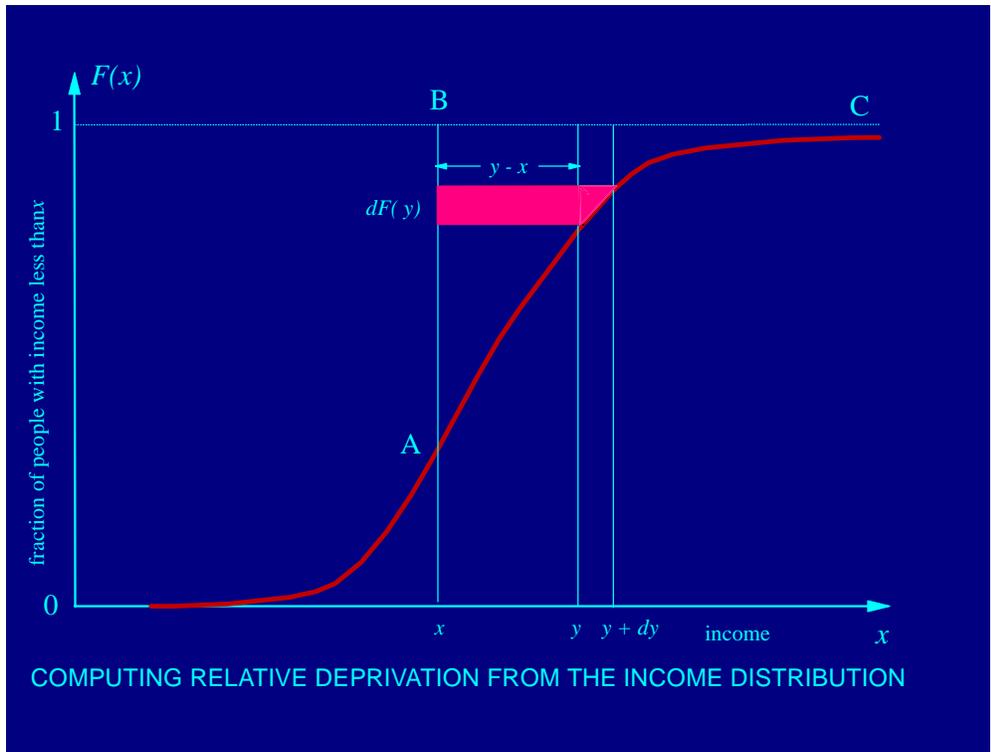


Figure 6: The definition of relative deprivation from the cumulative distribution of income

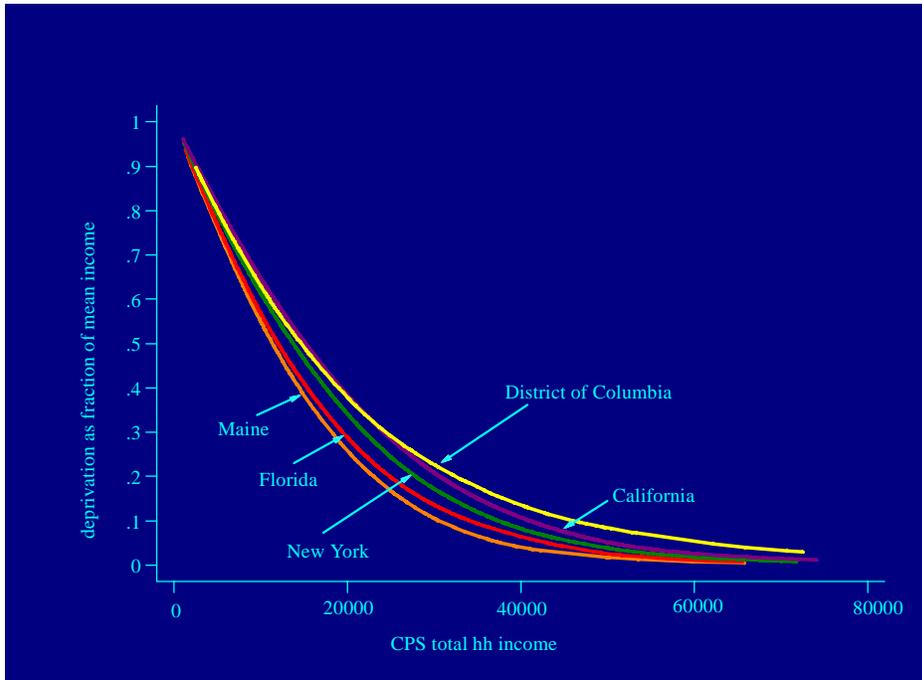


Figure 7: Relative deprivation curves for selected US states, Current Population Survey 1991.

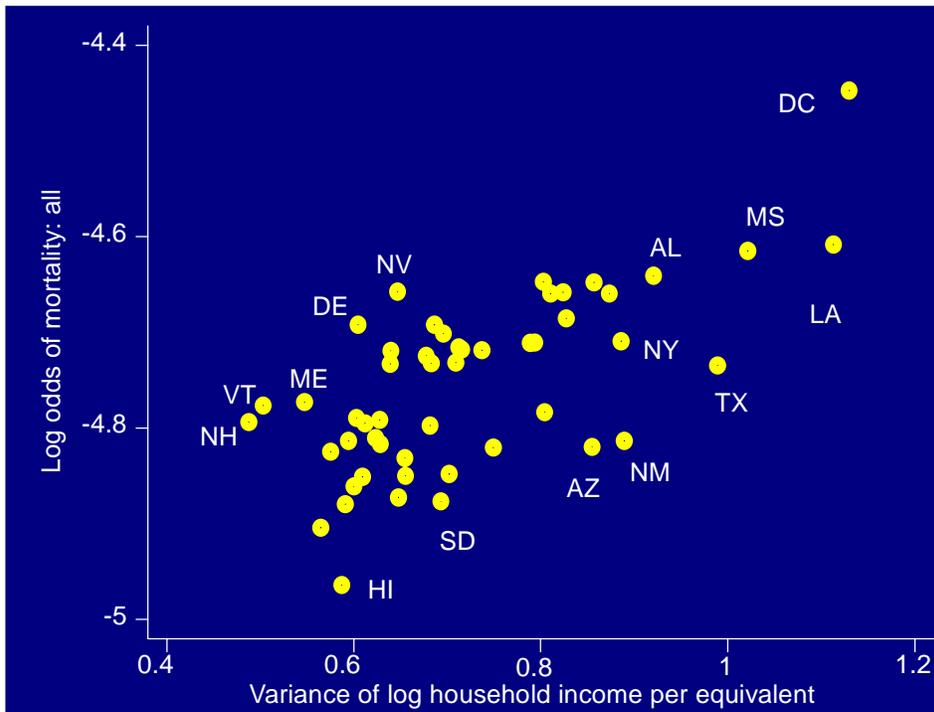


Figure 8: Inequality and the log odds of mortality, vital statistics and census data, 1990