Causes of Inequality in Health: Who You Are? Where You Live? Or Who Your Parents Were?

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Abstract

Amongst 33-year olds in the UK National Child Development Study (NCDS), ill health (as measured by cardinalised responses to a question on self-assessed health) is concentrated amongst the worse off. We seek to decompose these inequalities in ill health into their socioeconomic causes. In this decomposition, inequalities in health status depend on inequalities in each of the underlying determinants of health and on the elasticities of health status with respect to each of these determinants. We estimate these elasticities using regression models that allow for unobserved heterogeneity at the community level. We find that only 6% of health inequality is accounted for inequalities in unobserved area-level influences, and only 4% by inequalities in parental education and social class. The bulk of health inequality is accounted for by inequalities in income and housing tenure, though inequalities in educational attainment and in maths scores at age 7 also play a part.

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I. Introduction

In both the industrialised and the developing worlds, there is a good deal of interest in—and commitment on the part of policy-makers to reducing—socioeconomic inequalities in health. The gaps in health status between the poor and the better-off can be remarkably large, especially in the developing world. In Bolivia, for example, there is a fourfold difference between the infant mortality rate (IMR) amongst the poorest fifth of the population and the rate amongst the richest fifth (Gwatkin et al. 2000). In England and Wales, where the average IMR is less than one-tenth of that in Bolivia, the IMR amongst children born into households headed by an unskilled manual worker is 1.7 times that amongst children born into households headed by a professional (Drever and Whitehead 1997). Domestically, and in their international development work, many governments have shown a commitment to closing the gap in health outcomes between the poor and better-off. The British government, for example, has committed itself to reducing health inequalities in the UK and to focusing its development aid on improving the health of the world's poorest people (UK Department for International Development 1999). International organisations—including the World Health Organisation (World Health Organization 1999) as well as multilateral aid agencies such as the World Bank (World Bank 1997)—have also put the improvement of the health of the world's poor as a priority goal.

A major impediment to the realisation of these policy aspirations is the relatively weak knowledge base on which to formulate policies. A good deal is, of course, known about the broad determinants of health (Evans et al. 1994) and about the multiple deprivations of the poor (World Bank 2000). Many sensible policy proposals have been proffered on the basis of this general knowledge. What is lacking, however, is sufficiently detailed knowledge that allows priorities to be meaningfully set. As a result, many policy documents (Acheson 1998) end up looking like laundry lists of policies and programmes, whose costs are often only partially estimated and whose impacts are largely unknown.

Our aim in this paper is to shed light on this issue by "unpacking" the causes of socioeconomic inequalities in adult health. We employ a framework in which health is determined proximately by people's usage of health services, their diet, consumption of cigarettes and alcohol, physical surroundings, and so on, but which is in turn determined by the factors that influence people's health-related behaviour broadly-defined. The underlying determinants of health are therefore factors such as income, education, access to health services, etc., and it is the systematic variation in these underlying factors across socioeconomic groups that generates socioeconomic inequalities in health. The framework we use is innovative in that it makes clear how socioeconomic inequalities in health depend *both* on the impact that the various underlying determinants of health have on health outcomes *and* on the distribution of these underlying health determinants across socioeconomic groups. Health may be especially sensitive to one particular underlying determinant—access to health services, for example. But if this determinant is not especially unequally distributed across socioeconomic groups, it cannot be an important underlying cause of socioeconomic inequalities in health. Our approach provides an insight into both of these components of the health inequalities story, and provides us with a way of partitioning socioeconomic inequalities in health into their underlying causes.

In our analysis, we put a good deal of emphasis on two sets of determinants of health and their roles in generating socioeconomic inequalities in health. Both have been the subject of a good deal of debate in the literature on the determinants of health and in the literature on socioeconomic health inequalities. The first is geography. The role of geography in shaping child and adult mortality outcomes in the developing world has received much attention in the last few years, both in terms of the impact of physical geography (location, climate, etc.) (Bloom and Sachs 1998) and the impact of manmade community-level influences on health (roads, infrastructure, health service quality, etc.) (Lavy et al. 1996; Thomas et al. 1996). But the idea that where people live may get reflected in their health has also received attention in industrialised countries (Sloggett and Joshi 1994; Curtis and Rees-Jones 1998; Wiggins et al. 1998; Mitchell et al. 2000). One especially interesting hypothesis is that affluent communities have a variety of characteristics that are, in effect, public goods—low crime, good public amenities, low pollution, and so on—and which impact on the health of all people living in an area, whatever their socioeconomic circumstances. Thus the effects of living in an affluent area compound the effects of favourable individual circumstances and mitigate against the effects of unfavourable individual socioeconomic circumstances (Wilkinson 1996;

Kawachi and Kennedy 1997; Kawachi et al. 1997; Kennedy et al. 1998). The same logic applies to living in a deprived area. This raises the issue of how far socioeconomic inequalities in health are due to inequalities in the socioeconomic circumstances of *areas* rather than inequalities in the socioeconomic circumstances of *individuals*. To put it another way (Macintyre et al. 1993; Curtis and Rees-Jones 1998), how far should policy be focused on *places* rather than on the *people* living in them?

The second set of health determinants whose role in generating socioeconomic health inequalities we explore are childhood and parental factors. It is increasingly acknowledged that health in adulthood is linked to health-related experiences—if not health status itself—at previous stages in the "lifecycle" (Grossman 1972; Wadsworth 1991; Kuh and Ben-Shlomo 1997; Kuh et al. 1997; Wadsworth and Kuh 1997; Power et al. 1998; Claeson and Waldman 2000; Claeson et al. 2001), including the pre-natal period (Cheung et al. 2000; Eriksson et al. 2000; Lackland et al. 2000). What is less well known is how important such factors are in explaining *socioeconomic inequalities* in adult health. And yet, such evidence seems important. If much of the health inequalities we observe are the product of inequalities in health and nutrition prior to birth, stronger countervailing policies may be required to redress the effects of these inequalities. Likewise, if inequalities in adult health largely reflect the inequalities in human capital investments made by parents during childhood, reducing such inequalities—by, for example, increasing access amongst the poor to publicly-funded early child development programmes—might prove to be a useful weapon in tackling socioeconomic inequalities in adult health.

We employ a dataset that is unusually well suited to exploring the underlying causes of socioeconomic inequalities in health, both in general terms and the specific roles played by geography and childhood and parental factors. The data we use are taken from the British National Child Development Study (NCDS), a cohort study that has followed all children born in Great Britain in the first week of March 1958 (Ferri 1993). In addition to containing good data on health status in early adulthood, the NCDS contains detailed information on contemporaneous underlying determinants of health as well as underlying determinants at prior dates. By linking the individual- and household-level NCDS data to data on location of residence, we are able to explore the effects on health inequalities of geographic inequalities.

The plan of the paper is as follows. Section II of the paper establishes the extent of socioeconomic health inequalities in the NCDS sample. Section III sets out the method we use to decompose the underlying causes of health inequalities. This involves combining data on socioeconomic inequalities in the various underlying determinants of health with estimates of the elasticity of health with respect to these determinants. We obtain these estimates from a regression analysis of the NCDS data. Section IV outlines the data used for the regression analysis and for computing inequality indices of the various socioeconomic determinants. Section V presents the results of the regression analyses, and Section VI presents the results of the decomposition exercise. The final section—section VII—presents the conclusions of the paper.

II. Health Inequalities in the NCDS Sample

Our data are taken from the fifth and previous follow-ups of the NCDS. At the fifth follow-up, cohort member were 33 years of age. Our sample—after deletion of cases with missing information on any of the variables of interest (at birth or any subsequent contact)—consists of just under 6,000 individuals, with slightly fewer men than women. This is about half of the 11,400 cases in contact at age 33.

Measuring health

We measure health at age 33 through a question on self-assessed health (SAH). The question posed is "How would you describe your health *generally*: excellent, good, fair or poor?" (italics in original). This question is widely used in health interview and multi-purpose surveys, and has been used in a variety of comparative studies of health inequalities (Kunst et al. 1995; Van Doorslaer et al. 1997; Van Doorslaer and Koolman 2000). It has also been found in a large number of settings to be a good predictor of mortality and of the onset of disability (Idler and Benyamini 1997). Its categorical character presents problems, however, from the point of view of measuring health inequalities. The temptation is to dichotomise it, by setting a cut-off point above which people are said to be in good health. Though widely used, this practice is not to be recommended, since the choice of cut-off point can completely change the ranking of countries or periods (Wagstaff and Van Doorslaer 1994).

The obvious alternative is to assign to the categories a score. The problem is knowing which score to assign to each category. It is not obvious that one should assume that the gaps between the categories is constant. There is, in fact, evidence suggesting that such an assumption would be unwarranted. Several surveys have been undertaken that contain both the self-assessed health question and questions underlying a health utility index (HUI). The mean values for the various self-assessed health categories suggest that moving from "fair" to "poor" is perceived as a larger deterioration in health than the movement from "excellent" to "good" (Humphries and van Doorslaer 2000). One possibility would be to use the mean HUI scores from these surveys as scores for the various SAH categories. Unfortunately, this is not possible in the present case, since the number of SAH categories in the NCDS (4) is less than that in these other surveys (5). Instead, we use another approach which also "stretches out" the SAH scale. This involves assuming that underlying the SAH responses is a latent ill health variable with a standard lognormal distribution (Wagstaff and Van Doorslaer 1994). This appears to stretch out the scale rather too far (compared to the HUI indices) (Humphries and van Doorslaer 2000) and will result in us over-estimating the "true" degree of inequalities in ill health. But it seems to be fairly robust with respect to ranking of countries and time periods, and does not seem in other data to result in an index of inequality that is significantly different from that which results if the HUI scores are used. Applying the lognormal approach to our data produces scores for the latent ill health variable indicated in Table 1.

Table 1: Cardinalisation of SAH variable using standard lognormal assumption

	SAH response category			
	1	2	3	4
	Excellent	Good	Fair	Poor
Relative frequency	34.0%	51.4%	11.7%	2.9%
Latent ill health variable	0.3404	1.3070	4.0278	9.8062

Measuring socioeconomic status

The measure of socioeconomic status we use in measuring health inequalities is household income at age 33. Household income is equivalised to take into account differences in household structure—the old adage that "two can live as cheaply as one" is undoubtedly over-optimistic, but serves to illustrate the idea of household economies of scale. The equivalence scale we use is simply the square root of the number of household members, or equivalently the number of household members, raised to a power elasticity equal to 0.5. This can be thought of as an intermediate position between the per capita adjustment (equivalent to a power elasticity of 1.0 and an assumption of no economies of scale in household consumption), and no adjustment (equivalent to a power elasticity of 0.0 and an assumption of unlimited economies of scale in household consumption). Empirically, the median power elasticity implied by the equivalence scales in the OECD countries is around 0.4 (Buhmann et al. 1988).[1]

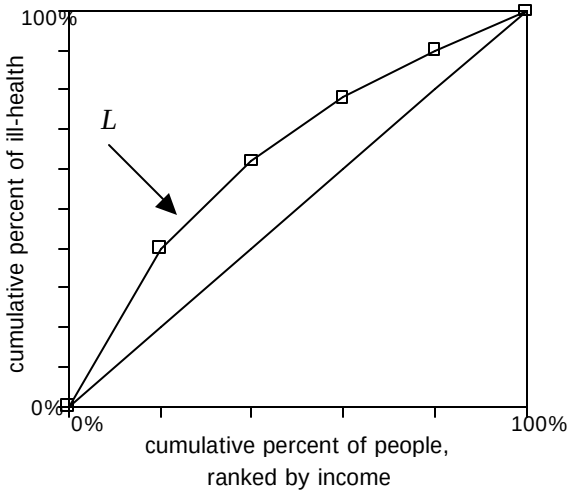
Measuring inequality

We measure inequalities (by income) in ill health—and in the socioeconomic determinants thereof—using a concentration index (Wagstaff et al. 1991; Kakwani et al. 1997). The curve labelled L in Figure 1 is an ill health concentration curve. It plots the cumulative proportion of ill health (on the y-axis) against the cumulative proportion of the sample (on the x-axis), ranked by income, beginning with the most disadvantaged person. If the curve L coincides with the diagonal, all individuals, irrespective of their household income, suffer the same level of ill health. If, as is more likely, L lies above the diagonal, inequalities in ill health favour the better-off; we will call such inequalities *pro-rich*. If L lies *below* the diagonal, we have *pro-poor* inequalities in ill health (inequalities to the disadvantage of the better-off). The further L lies from the diagonal, the greater the degree of inequality in ill health across income groups. The concentration index, denoted below by C, is defined as twice the area between L and the diagonal. C takes a value of zero when L coincides with the diagonal and is negative when L lies above the diagonal and health inequalities are pro-rich. C is positive when L lies below the diagonal and health inequalities are pro-poor. Issues concerning computation and statistical inference have been discussed elsewhere (Kakwani et al. 1997) and need not be repeated here.[2]

[1] This particular equivalence scale doe not, of course, take into account the ages of the household members.

[2] Comparing $L(s)$ to the diagonal presupposes that all inequalities in ill health across income groups can be eliminated. This would be unrealistic if the socioeconomic groups varied in their average age or their gender mix. In the NCDS cohort, the issue of age variation does not arise, and the variation in gender mix was so small that use of the alternative inequality measure, I^*, proposed in Kakwani et al. (1997) made virtually no difference.

Fig 1: Ill-health concentration curve



Inequalities in ill health in the NCDS cohort

The concentration index for SAH is indicated in Table 2. Inequalities in ill health are pro-rich—i.e. ill health is more common amongst the poor. The t-ratio tests the hypothesis that the concentration index is significantly different from zero, so the values of the test statistic in this case indicate that socioeconomic inequalities in ill health are statistically significant in the UK. At –0.092, the concentration index is somewhat smaller in absolute size than the (demographically-adjusted) concentration indices for SAH for the British population as a whole reported in two recent studies (Van Doorslaer et al. 1997; Van Doorslaer and Koolman 2000), though these other studies' use different equivalence scales than that used here (a UK scale in the former case, and an OECD scale in the latter case). In the event, the index reported in Table 2 is not significantly different from the indices reported in these two other studies.[3]

Table 2: Inequalities in health in the NCDS sample

	SAH
Concentration index (*C*)	-0.089
t-statistic for *C*	-10.469

Note: C was computed on microdata using eqn (19) in Kakwani et al. (1997).

[3] The t-ratios for testing equality of the NCDS SAH concentration index and the demographically-adjusted concentration indices in these two other studies are 1.27 and 0.62.

III. Decomposing the Causes of Health Inequalities: Theory

The previous section established that inequalities in health by income are pro-rich in Britain and are not the result of sampling variability. In the rest of the paper we aim to "unpack" or decompose the underlying sources of these inequalities. In this section, we outline the relevant theory.

Our measure of inequality, the concentration index, is additively decomposable in the following sense. Suppose we have a linear regression model linking SAH, y, to a set of K determinants, x_k:

$$(1) \qquad y_i = a + \sum_k b_k x_{ki} + e_i,$$

where the b_k are coefficients and e_i is an error term. We assume that everyone in the NCDS sample—irrespective of their income—faces the same coefficient vector, b_k. Interpersonal variations in SAH are thus assumed to derive from systematic variations across income groups in the determinants of SAH, i.e. the x_k. We make use of the following result (Wagstaff et al. 2000) that links the concentration index for y, C, to the inequalities in the underlying determinants of y:

$$(2) \qquad C = \sum_k (b_k \bar{x}_k / m) C_k + GC_e / m = \sum_k h_k C_k + GC_e / m,$$

where m is the mean of y, $\bar{x}_k$ is the mean of x_k, C_k is the concentration index for x_k (defined analogously to C) and GC_e is the generalised concentration index (GCI) for the error e_i, and h_k is elasticity of y with respect to x_k. The GCI is analogous to the slope index of inequality used by epidemiologists (as opposed to the *relative* index of inequality). The value of GC_e is most easily computed as a residual from eqn (2).[4]

Thus inequality in ill health can be decomposed into explained and unexplained inequality. The former is made up of K terms corresponding to the K covariates. Together, these terms are a weighted average of the concentration indices for the covariates, where the weight for the kth covariate is its elasticity. Thus, the larger the elasticity h_k, and the more unequally distributed across income groups x_k is (i.e. the larger is C_k), the greater the importance of inequality in x_k in accounting for inequality in ill health. This reinforces the point made above to the effect that a covariate may be an especially important determinant of health (i.e. its elasticity, h_k, may be large), but if it is not especially unequally distributed across income groups (i.e. C_k is fairly small), it is not going to be a key part of the explanation of socioeconomic inequalities in health.

[4] Or AE_e can be computed directly by running a regression of a variable representing twice the residual multiplied by the variance of the relative rank on a constant and the relative rank.

IV. Modelling Issues

To implement the decomposition, we require two types of data—data on the elasticities (the h_k), and data on the concentration indices (the C_k). The latter are easily computed from the NCDS data using the same methods used to compute the concentration index for ill health.

We use regression methods to obtain estimates of the elasticities. The decomposition approach could be applied using either a "production function" (an equation linking health to its proximate determinants) or a "demand" or "reduced-form" equation (an equation linking health to its socioeconomic or underlying determinants). To avoid—or at least reduce the degree of—endogeneity (Rosenzweig and Schultz 1983), and to increase the policy relevance of our results, we have opted for a reduced-form equation.[5] Thus rather than telling us how much smaller health inequalities would have been in the absence of inequalities in factors such as cigarette smoking, our results will tell us how much smaller health inequalities would have been in the absence inequalities in factors such as education, income, and so on. As we have cardinalised SAH at age 33, standard regression techniques can be employed to establish the relationship between it and the various determinants of health.

One other issue that arises is whether to model the effects of location explicitly (i.e. include area-level variables amongst the x's in the regression equation), or whether to treat the area-level effects as unobservables. We opted for the latter approach, because we were concerned not to understate the contribution of area-level inequalities by omitting relevant area-level variables. So, we include in our regression equation fixed effects at the community level. For each community we are able to recover a fixed effect from the regression process, and we can then compute a concentration index for these fixed effects. We will thus be able to tell how far inequality in area-level fixed effects contributes to health inequalities across individuals, but not what it is at the community level whose inequalities matter most for health inequalities. This is the disadvantage of our approach. The advantage is that our estimate of the contribution of are-level inequalities is not contingent on what data we happen to have available at the community level. The fixed effects are easily estimated using dummy variables and can then be retrieved. Estimation was undertaken using STATA (Deaton 1997).

[5] It is possible that the problem of endogeneity exists even in the reduced-form equation. It is not uncommon, for example, to find household income treated as endogenous in reduced-form health equations in the economics literature—cf. e.g. Lavy et al. (1996).

V. Data

With the "lifecycle" in mind, we can distinguish between underlying determinants of health at different ages. Our variables are defined in Table 3, which also shows the means and concentration indices, the latter indicating how pro-poor or pro-rich the determinant in question is. The variables are listed with the adulthood contemporaneous variables first, moving back around the lifecycle through early adulthood and the teenage years (the educational attainment variables), school-age childhood, early childhood, birth, and the influences active prior to birth.

Table 3: Variable definitions and descriptions statisticx

Variable	Definition	mean	CI
female	female	0.487	-0.046
partner	has partner	0.864	0.114
parent	is parent	0.734	0.067
fem_kids	female w/ kids	0.776	-0.025
logeqinc	log equivalent income	6.291	0.066
own0	owns own home	0.756	0.139
own1	local government housing	0.105	-0.392
sphied	spouse has university degree	0.151	0.412
spmeded	spouse has A-levels	0.171	0.198
qgrad	graduate	0.123	0.398
qteach	teaching qualification	0.138	0.192
qalev	A-levels	0.143	0.035
maths	maths ability at age 7	5.311	0.058
lanurs	attended local government nursery	0.091	0.002
privprsk	attended private preschool	0.046	0.200
lowbw	low birthweight baby	0.054	-0.100
motheduc	mother was educated	0.261	0.157
sc1birth	father was social class 1	0.041	0.270
sc2birth	father was social class 2	0.140	0.127
sc3abirt	father was social class 3	0.102	0.120

Note: Social class 1 is professional, social class 2 is intermediate non-manual, and social class 3 is junior non-manual.

The contemporaneous adult influences are the variables "female" through to "spmeded". Roughly 50% of the sample is female. The slight negative concentration index indicates that there is a slight tendency for females to be poorer than males. Roughly 90% of the sample has a partner, and people with partners tend to be have higher incomes than people who do not. Almost three quarters of the sample are parents with dependent children—those who are not tend to be worse-off than those who are. We also include a female and parent interaction variable. Income is, as indicated, measured by equivalent income. In the regressions, we use the natural log of this. The concentration index is therefore not the Gini coefficient, but would have been had we used income rather than the log of income. Three quarters of the NCDS sample owned their own home

at age 33, and home ownership is concentrated amongst the better-off. Around 10% live in social housing, and these tend to be the poorer NCDS cohort members. Around 32% of the sample had partners with more than low education, being split fairly equally between the medium and high education categories. Persons in the NCDS sample with university-educated partners tend to live in better-off households.

The early adulthood and teenage years variables are the three variables capturing the educational attainment of the individual in question. A little over 12% of the sample have a degree as their highest qualification. Unsurprisingly, these tend to be the better-off. This cohort went through the old teacher training system in which non-graduates were able to obtain a teaching qualification. This is classified along with other non-degree qualifications, such as nursing qualifications, in the category "teaching qualification". A little under 14% of the sample had this as their highest qualification. They tended to be better-off, on average, than the sample as a whole, but less well-off than graduates (the concentration is positive but smaller). Just under 15% of the sample had "A-levels" as their highest qualification (the school-leaving examinations providing entry to higher education), and these were very slightly better-off than the sample as a whole.

From school-age childhood our non-proximate influence on adult health is the individual's test score in mathematics at age 7. Those doing best at age 7 tended to be have higher incomes at age 33, though the inequality is not large. These maths test scores are correlated with other cognitive scores and are treated as a measure of both ability and early attainment. We capture early childhood influences on adult health by pre-school attendance, which was, for this cohort, something of a rarity, of erratic quality and not recorded in great detail. Nearly 10% of the sample attended a local government nursery or nursery school during childhood, but those who did were no better off or worse off at age 33 than the sample as a whole. Local authority nurseries would tend to select for social disadvantage, but public nursery schooling would have been more of a geographic lottery. A more marked pro-rich inequality is evident amongst the 5% of the sample who attended a private pre-school during childhood. Moving back further round the lifecycle, we include low birth weight. Around 5% of the sample was classified as low birth weight, and those who were tended to be amongst the lower income groups at age 33. Finally, moving still further back around the lifecycle, we include mother's education and father's social class, both of which could clearly have impacts on a person's health even prior to birth. Roughly one quarter of the sample had an educated mother, and around 30% had a father in social class 3 or higher.

In addition to the variables listed in Table 3, we include area-level fixed effects, defined at the level of the ONS ward cluster. There were 43 of these in the classification (Wallace et al. 1995), based upon the socioeconomic profile of wards at the 1991 census. In our sample, their coverage ranged from 10 individuals per group of similar wards to 845, and averaging 230. The wards in these clusters are not in general contiguous. We did not attempt to estimate fixed effects for individual wards, as many of these only had one or two observations.

VI. Regression Results

In this section, we present the regression estimates of the regression parameters in eqn (1) that are then used in the decomposition in eqn (2). Table 4 presents the results, starting with just the adult contemporaneous variables, and then gradually adding the additional (sets of) variables corresponding to successively earlier stages in the lifecycle.

The gender effect on the reporting of ill health at age 33 is conditional on whether or not the individual has any children. Women report significantly worse health than men if they have children, but not otherwise. These effects are fairly robust with respect to the inclusion of influences from other stages of the lifecycle. Having a partner slightly worsens health, but the effect is not significant, and bearing in mind the interaction it is clear that being a parent contributes to worse health only in the case of women. These effects are also fairly robust. The effect of current income, by contrast, whilst strong throughout is reduced by the addition to the model of the area-level fixed effects, and even further by the addition of the variables capturing influences at other stages of the lifecycle. Home owners have lower levels of ill health (though never significantly so), while local authority tenants have significantly *higher* levels of ill health. The fact that the local government housing tenure is significant even though current income is included in the equation suggests strongly that it is not just current income that matters for health but also long-term wealth. Of interest also is the fact that the impact of local authority tenancy falls as area-level fixed effects are added to the model but very little thereafter. By contrast, the impact of home ownership *rises* in absolute size as area-level fixed effects are added. Omission of the area-level influences seems to bias the estimates of the household's own wealth as reflected in the housing variables, reflecting presumably the tendency for housing types to cluster geographically. Having a medium-high educated spouse goes with less ill health, but the estimated effect is much reduced—and gradually loses significance—by the addition of the individual's own education and other influences.

Both types of higher education reduce ill health, but the effect and level of significance thereof is reduced by the inclusion of the person's maths ability at age 7, which has a significant negative effect on the reporting of ill health at age 33, irrespective of the other variables included in the model. This suggests that it is not just educational attainment and investment that matters for health but also a person’s underlying abilities. The coefficients on the two pre-school variables suggest that attendance at a local authority facility is associated with worse adult health whilst attendance at a private pre-school is associated with better health. Neither effect, however, comes even close to achieving statistical significance. Low birth weight leads, other things equal, to worse reported health at age 33 but the effect is not significant. Having an educated mother and a father from non-manual social classes is associated with better reported health at age 33, but the impact of mother's education is not significant, and the impact of father's social class is only close to achieving significance in the case of fathers from social class 3.

In the final analysis, relatively few factors continue to have a significant impact when the fixed effects and the other variables are all included. These are: parenthood in the case of women; income; local government housing tenure; maths ability at age 7; and father's social class. In the final model, the area-level fixed effects are jointly significant at the 5% level but not the 1% level, the test statistic having a value of 1.58 with an F distribution and (42,5794) degrees of freedom.

Table 4: Parameter estimates of SAH regression mode

	model 1		model 2		model 3		model 4		model 5		model 6		model 7	
Variable	**Coef.**	**t**	**Coef.**	**t**	**Coef.**	**t**	**Coef.**	**t**	**Coef.**	**t**	**Coef.**	**t**	**Coef.**	**t**
female	-0.058	-0.893	-0.058	-0.888	-0.055	-0.844	-0.054	-0.828	-0.053	-0.803	-0.054	-0.817	-0.048	-0.728
partner	0.105	1.270	0.105	1.271	0.084	1.015	0.081	0.971	0.079	0.949	0.081	0.972	0.076	0.919
parent	0.013	0.220	0.018	0.303	0.010	0.159	0.009	0.155	0.009	0.153	0.009	0.154	0.010	0.158
fem_kids	0.073	2.274	0.072	2.237	0.067	2.069	0.066	2.064	0.066	2.059	0.066	2.046	0.063	1.964
logeqinc	-0.228	-6.713	-0.219	-6.341	-0.202	-5.774	-0.193	-5.512	-0.193	-5.506	-0.193	-5.504	-0.190	-5.413
own0	-0.027	-0.368	-0.044	-0.593	-0.038	-0.513	-0.032	-0.426	-0.032	-0.433	-0.033	-0.447	-0.034	-0.453
own1	0.445	4.675	0.390	4.012	0.383	3.931	0.382	3.923	0.380	3.906	0.378	3.882	0.370	3.799
sphied	-0.243	-3.764	-0.216	-3.274	-0.141	-1.927	-0.132	-1.807	-0.129	-1.768	-0.129	-1.762	-0.117	-1.582
spmeded	-0.157	-2.597	-0.128	-2.101	-0.109	-1.782	-0.108	-1.761	-0.106	-1.724	-0.105	-1.710	-0.095	-1.544
qgrad					-0.182	-2.286	-0.135	-1.662	-0.132	-1.617	-0.131	-1.613	-0.108	-1.303
qteach					-0.150	-2.258	-0.126	-1.878	-0.125	-1.856	-0.124	-1.843	-0.114	-1.699
qalev					-0.038	-0.586	-0.019	-0.297	-0.018	-0.272	-0.017	-0.252	-0.013	-0.196
maths							-0.026	-2.796	-0.026	-2.768	-0.025	-2.705	-0.024	-2.553
lanurs									0.053	0.705	0.052	0.695	0.050	0.672
privprsk									-0.070	-0.671	-0.071	-0.678	-0.036	-0.338
lowbw											0.104	1.083	0.103	1.076
motheduc													-0.037	-0.676
sc1birth													-0.090	-0.779
sc2birth													-0.050	-0.731
sc3abirt													-0.141	-1.916
constant	2.811	14.754	2.900	12.514	2.839	12.199	2.900	12.413	2.892	12.373	2.884	12.330	2.880	12.310
FEs	No		Yes		Yes		Yes		Yes		Yes		Yes	

VII. Decomposition Results

Table 5 shows the results of the decomposition in eqn (2) for the full model. We have included all variables, irrespective of whether the coefficient in question is significantly different from zero. The column headed "*Elasticity*" is the elasticity of SAH with respect to the *k*th covariate, h_k, and indicates the weight attached to the concentration index for the covariate in question, C_k. The column headed "*Contr. to C*" is the product of h_k and C_k, and gives the contribution to C of inequality in x_k. Thus, for example, in the case of the maths score, h_k is -0.087 (a 10 percent rise in the maths score reduces ill health by just under one percent), C_k, is 0.058 (higher maths scores are somewhat more common amongst the better off), and the contribution to C of this inequality in maths scores is –0.005. This is the same sign as C (ill health is concentrated amongst the poor), so inequality in maths scores contributes positively to inequality in ill health, in the sense that inequality in health would have been smaller in the absence of inequality in maths scores, or with a smaller amount of inequality. For some variables, "*Contr. to C*" is positive, indicating that inequality in health would have been larger in the absence of inequality in the variable in question. This is true of the "partner" variable, which is estimated to have a positive (albeit insignificant) effect on the incidence of ill health and is concentrated amongst the better off. To facilitate interpretation, Table 6

sums the contributions of grouped variables (e.g. the demographic variables) and vectors of dummy variables (e.g. the two variables capturing housing status). The contributions are also expressed as percentages of *C* to indicate the percentage of inequality in ill health explained by inequality in the variable in question.

The most striking thing about Table 6 is the importance of inequalities in the contemporaneous adulthood variables in explaining inequalities in ill health. A full *three quarters* of inequalities in ill health at age 33 is explained by inequalities in the demographic variables, income, housing status and spouse's education. Of these, income inequality is by far the most important contributory factor, accounting for a full sixty percentage points of inequality in ill health. Of the remaining one-quarter not explained by inequalities in the contemporaneous adulthood variables, the biggest contributory factors are inequalities in the individual's own education and inequalities in the maths score at age 7, which each account for six percentage points. Inequalities in early childhood (i.e. preschool attendance), birth (i.e. low birth weight), and in parental influences (i.e. parental education and social class) account for a mere four additional percentage points of the total inequality in ill health at age 33. Furthermore, inequalities in area-level influences on ill health—captured by the area-level fixed effects—account for only six percentage points of the total inequality in ill health, once inequalities at the individual and household levels have been controlled for. Interestingly, results not shown in Table 6 indicate that this percentage remains the same whatever the model specification.

Table 5: Decomposition results for SAH

Variable	Coef.	Mean	Elasticity	CI	Contr to C
female	-0.048	0.487	-0.016	-0.046	0.001
partner	0.076	0.864	0.045	0.114	0.005
parent	0.010	0.734	0.005	0.067	0.000
fem_kids	0.063	0.776	0.033	-0.025	-0.001
logeqinc	-0.190	6.291	-0.814	0.066	-0.053
own0	-0.034	0.756	-0.017	0.139	-0.002
own1	0.370	0.105	0.026	-0.392	-0.010
sphied	-0.117	0.151	-0.012	0.412	-0.005
spmeded	-0.095	0.171	-0.011	0.198	-0.002
qgrad	-0.108	0.123	-0.009	0.398	-0.004
qteach	-0.114	0.138	-0.011	0.192	-0.002
qalev	-0.013	0.143	-0.001	0.035	0.000
maths	-0.024	5.311	-0.087	0.058	-0.005
lanurs	0.050	0.091	0.003	0.002	0.000
privprsk	-0.036	0.046	-0.001	0.200	0.000
lowbw	0.103	0.054	0.004	-0.100	0.000
motheduc	-0.037	0.261	-0.007	0.157	-0.001
sc1birth	-0.090	0.041	-0.003	0.270	-0.001
sc2birth	-0.050	0.140	-0.005	0.127	-0.001
sc3abirt	-0.141	0.102	-0.010	0.120	-0.001
FEs	1.000	2.771	1.887	-0.003	-0.005
Sum					-0.088

Table 6: Consolidated decomposition results for SAH

	Contr to CI	% total CI
demographics	0.005	-6%
income	-0.053	60%
housing status	-0.013	14%
spouse education	-0.007	8%
own education	-0.006	6%
maths ability at age 7	-0.005	6%
preschool	0.000	0%
low birthweight	0.000	0%
mother's educ & father's SC	-0.003	4%
location (FEs)	-0.005	6%
Sum of explained portion	-0.088	99%
CI for dep vbl	-0.089	
Residual	-0.001	1%

VIII. Conclusions

We set out to unpack the causes of inequalities in health in contemporary Britain, using inequalities in ill health amongst 33-year old NCDS cohort members as the case study. Our particular interest was in the relative contributions of inequalities in two sets of factors. The first was inequalities in geography. In effect, we set out to answer the question: How far are health inequalities between poor and better-off people due to poor people living in unhealthy areas? The second was inequalities at earlier stages in the lifecycle. We set out to answer the question: How far are inequalities in health in adulthood due to inequalities in childhood and human capital investments and to inequalities in parental status and human capital?

The decomposition method we used makes it clear that two factors have to be borne in mind when assessing the contribution to health inequality of a particular variable or set of variables. The first is the elasticity of health with respect to the variable in question—by what percentage would health rise or fall in response to a ten percent increase in the variable in question? The second is the degree of inequality in the variable in question—how unequally distributed is the variable in question across the income distribution? *Both* these numbers have to be large for the variable in question to be a major part of the story of why health inequalities exist. Variable *X* might have a very large elasticity, but if inequality in it is very small, it cannot contribute in any major way to explaining health inequality. Likewise, variable *Y* may be highly unequally distributed, but if its elasticity is very small, it cannot be a part of the explanation of health inequalities.

In our empirical analysis, we focused on self-assessed health at age 33. We cardinalised this categorical variable, by assuming the responses to the question came from a standard lognormmal distribution, and making the latent variable we use increasing in *ill* health. The values so assigned to the responses in the SAH question appear from other research to accord reasonably well with values implied by health utility indices. We estimated elasticities using regression analysis, and worked with a "demand" or "reduced-form" equation rather than a production function—our variables are the "underlying" determinants of health, rather than the "proximate" determinants. We captured geographic influences using area-level fixed effects. These do not allow us to pinpoint what it is about areas that contribute positively or negatively to ill health, but they do allow us to capture the effects on health of area-specific influences without having to worry about whether we are omitting any relevant influences. We captured contemporaneous adult influences on health at age 33 by a vector of demographic and household composition variables, household income and spouse's education. Teenage and early adulthood factors are captured by the individual's own educational attainment, school-age childhood influences (and inherited ability) by a maths test score at age 7, and pre-school influences by attendance at pre-school, stratified by type of institution. Moving further back around the lifecycle, we also include low birth weight, and mother's education and father's social class.

Our results provide a clear-cut and thought-provoking answer to the question posed in the title of the paper. NCDS cohort members who were well-off at age 33 tended to have mothers who were better educated than poorer cohort members and to have fathers from the higher social classes. But because the estimated elasticity of SAH with respect to these variables is very small once contemporaneous variables are included, the total contribution to health inequalities of these parental inequalities is a mere 4%. Area-level inequalities are also estimated to play only a very small role alongside individual influences in shaping health inequalities at age 33 amongst NCDS cohort members. There is relatively little cross-sample variation in the estimated area-level fixed effects and they are not systematically associated with a person's rank in the income distribution (C_k is very small). Only 6% of overall inequality in SAH can be explained by inequality in area-level fixed effects. The bulk of health inequality amongst NCDS cohort members at age 33 stems not from where they live, or who their parents were, but rather who they are. A full three quarters of inequality in ill health is explained by inequality in contemporaneous adult variables—demographics and household composition, income, housing status (capturing long-term wealth undoubtedly), and spouse's education. Income inequality is by far the most important of these (accounting for 60% of the total), but inequality in housing status is also important (14%). Inequalities during the early adult and teenage years also matter (inequalities in educational attainment account for 6% of the total inequality in health), as do inequalities during the school-age years (maths ability at age 7 accounts for a further 6%). By contrast, inequalities during the pre-school years (in pre-school attendance) and at birth (low birth weight) may have influenced their resources as they grow older, but do not have any perceptible independent impact on inequality in SAH at age 33.

Several points should be borne in mind in connection with these results. First, our focus has been on cohort members at age 33. It is quite possible that the results would be rather different if the exercise were repeated on the cohort members at the next sweep at age 42. Second, our focus has been on self-assessed health (SAH). Whilst this question has been seen to be useful as a predictor of the onset of disability and of mortality, it may well be that a "harder" variable, such as an activities-of-daily-living (ADL) index or mortality itself, might yield different results. Third, what is true of Britain is not necessarily true of other countries. It may well be that policies aimed at reducing geographic inequalities—in access to health services through the Ness's resource-allocation formulae, but also in income, deprivation and employment opportunities—have indeed reduced both the extent of geographic inequalities and the effects these influences have on health outcomes. There are undoubtedly areas where geographic inequalities could be narrowed further, and there are undoubtedly areas where the link between one's health and where one lives could be weakened further. But it seems highly likely that the extent of these inequalities—and the impacts of these factors on health—may well be much larger in other countries, especially those in the developing world and in the transition economies. The same may be true of inequalities in health influences across the lifecycle and the impact they have on adult health. Policies in the UK may well have had an impact both on the degree of inequality in—and the effect on adult health of—the incidence of low birth weight, pre-school attendance, maths test scores at age 7, and educational attainment at school and university. In other countries, the extent of inequalities in these factors and their impact on health may well be higher than in the UK. Further research along these lines would seem warranted to shed light both on how the UK fares in an international context, and how far the results are different when people are older and where other "harder" measures of health are employed. Finally, our results may underestimate the importance of early life factors and geography insofar as they influence current individual circumstances. Policies aimed at reducing inequalities in these influences *may* therefore have a payoff in terms of reducing inequalities in adult health. Empirical work showing the strength of the various pathways and their inequalities would need to be done to establish this. In the meantime, what our results suggest is that since health inequalities at age 33 appear to be so heavily influenced by inequalities in individual circumstances at that age, policies aimed at reducing inequalities in health determinants in adulthood will *definitely* have a payoff. Inequalities in adult health are *not* the inevitable product of geography and the lottery of birth.

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