Fundamental causes of inequalities in mortality: an empirical test of the theory in 20 European populations.

“Fundamental causes” of inequalities in mortality: an empirical test of the theory in 20 European populations

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Abstract

The “fundamental causes” theory stipulates that when new opportunities for lowering mortality arise, higher socioeconomic groups will benefit more because of their greater material and non-material resources. We tested this theory using harmonized mortality data by educational level for 22 causes of death and 20 European populations from the period 1980-2010. Across all causes and populations, mortality on average declined by 2.49% (95%CI: 2.04-2.92), 1.83% (1.37-2.30) and 1.34% (0.89-1.78) per annum among the high, mid and low educated, respectively. In 69% of cases of declining mortality, mortality declined faster among the high than among the low educated. However, when mortality increased, less increase among the high educated was found in only 46% of cases. Faster mortality decline among the high educated was more manifest for causes of death amenable to intervention than for non-amenable causes. The difference in mortality decline between education groups was not larger when income inequalities were greater. While our results provide support for the fundamental causes theory, our results suggest that other mechanisms than the theory implies also play a role.

Key words

health inequalities; mortality; cause of death; trends; Europe; fundamental causes theory; multilevel regression

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4 tables

Introduction

Mortality is usually lower among those in more advantaged socioeconomic positions, as indicated by a higher education, occupational class or income level (Commission on Social Determinants of Health, 2008), and this association is very persistent, both across countries (Mackenbach et al., 2008) and over time (Mackenbach et al., 2015c). One remarkable feature is that as mortality in the whole population declines, relative inequalities in mortality tend to increase, because relative declines in mortality are often larger in higher than in lower socioeconomic groups (Mackenbach et al., 2015c, Borrell et al., 1997, Mackenbach et al., 2003, Strand et al., 2010, Shkolnikov et al., 2012).

This is why Link and Phelan have proposed the theory of "fundamental causes". This theory stipulates that socioeconomic status is a fundamental cause of inequalities in mortality, in the sense that it "embodies an array of resources, such as money, knowledge, prestige, power, and beneficial social connections" which can be used "to avoid disease risks or to minimize the consequences of disease once it occurs" so that "as opportunities for avoiding disease expand so health inequalities continue to exist" (Link and Phelan, 1995, Phelan et al., 2010, Phelan et al., 2004).

In essence, the fundamental causes theory implies that health results from purposive action or "health-directed human agency", and that socioeconomic differences in the availability of the means to achieve health goals (behavior change, access to health care, protection from occupational risks, road safety, …) are the crucial factor on which the fundamental

relationship between socioeconomic status and health rests (Phelan and Link, 2005a). If the theory is correct, one should expect to find that as opportunities for lowering mortality arise, declines in mortality will be larger among those with a higher socioeconomic position.

In this paper, we test four specific hypotheses derived from this theory, using harmonized mortality data by educational level for 22 causes of death and 20 European populations from the period 1980-2010.

Theoretical background

Freese and Lutfey have in several papers given a lucid account of the main assumptions underlying the fundamental causes theory (Freese and Lutfey, 2011, Lutfey and Freese, 2005). In their words, the fundamental causes theory proposes a "metamechanism": "an abstract mechanism that explains the generation of multiple concrete mechanisms that reproduce a particular relationship [between socioeconomic status and health] in different places and different times" (Lutfey and Freese, 2005). The specific "metamechanism" central to the fundamental causes theory is that of purposive action with different means: individuals with lower and higher socioeconomic position are assumed to all pursue good health, but differ in their means to achieve this goal (Freese and Lutfey, 2011).

This may well be true in some (or even many) instances, but as Freese and Lutfey have shown, "differences in means among purposive agents do not account for all the […] ways that socioeconomic status causes health". For example, the fact that lower educated car

drivers less often wear their seat belts cannot be explained by differences in access to the resource of seat belts, because all cars have seat belts installed (Freese and Lutfey, 2011). They have therefore proposed several other "metamechanisms" that may account for the durable relation between socioeconomic status and health: (1) *Spillovers within socioeconomic groups*. Even without striving for good health as an individual, a person with a higher socioeconomic position will still benefit from the purposive action of other individuals in the same group, e.g. because they campaign for a safer neighborhood. (2) *Different preferences for health*. People in higher socioeconomic groups "may exhibit a stronger and more consistent preference for future good health than others", e.g. because of different time horizons or as part of their cultural "habitus". (3) *Different treatment by institutions*. Institutions such as schools and medical care facilities may treat people from lower and higher socioeconomic status differently, with different health outcomes, independent from individual health-directed agency, as a result (Freese and Lutfey, 2011).

With a few exceptions (Lutfey and Freese, 2005), most empirical tests of the fundamental causes theory have been of a macrosocial nature, and have therefore not been able to distinguish between different mechanisms. Nevertheless, these studies have provided general support for the fundamental causes theory. For example, Phelan and Link compared trends in mortality by socioeconomic status between causes for which the capacity to prevent death has or has not increased significantly, and found that over time sharp disparities emerged in the United States for the first, but not for the second group (Phelan and Link, 2005b). Educational disparities in the United States widened over time for mortality from heart disease and lung cancer but not for mortality from non-preventable

cancers (Masters et al., 2012). Mortality from diseases for which there has been more progress in prevention or treatment is more strongly associated with education in the United States than mortality from diseases with less technological progress (Glied and Lleras-Muney, 2008). Also, in the 1960s and 1970s, when knowledge that smoking causes lung cancer became available in the United States, a sharp gradient of smoking favoring the higher educated emerged where none had existed in the 1950s (Link, 2008). Similarly, in a comparison of successive birth cohorts in the United States gaps between educational groups in mortality from preventable causes increased rapidly (Masters et al., 2015).

While these are important studies, they only cover a single population (with one exception (Willson, 2009)), and so we do not know whether their results apply more generally and also to other high-income countries – as would be necessary under a theory pretending to provide a generic explanation for the existence and persistence of health inequalities. We therefore set out to test a set of hypotheses derived from the theory in a dataset covering 20 European populations.

This dataset does not only allow us to simply compare mortality trends between low and high socioeconomic status groups, but also to test four specific hypotheses, and thereby to shed light on some of the specific mechanisms underlying the relation between socioeconomic status and mortality. The *first hypothesis*, following directly from the fundamental causes theory, is that when mortality in the whole population declines, mortality generally declines faster among the higher than among the lower educated. We will also assess whether these inequalities in mortality decline are larger when mortality

declines faster. We expect the strongest associations for truly preventable causes of death (Phelan et al., 2004, Mackenbach et al., 2015b, Masters et al., 2015, Glied and Lleras-Muney, 2008), and if the speed of mortality decline is an indicator for the degree to which that cause of death has become amenable to prevention, one would expect inequalities in mortality decline between educational groups to be larger when mortality declines faster.

Our *second hypothesis* deals with increasing mortality. Although we expect mortality from most conditions to decrease over time, we also anticipate to find some causes of death for which mortality has increased, as in the case of the recent rise of alcohol-related mortality seen in many European countries (Mackenbach et al., 2015a) and in the case of some Central & Eastern European countries where a mortality crisis occurred in the 1990s (Mackenbach et al., 2013). Our hypothesis, which was not part of the original fundamental cause theory but has been proposed by others before us (Miech et al., 2011), is that when mortality goes up, the increase will be less in higher educational groups, because they will be better to protect themselves against these new dangers.

Our *third hypothesis* exploits the distinction between "preventable" and "non-preventable" causes of death, following the reasoning applied in several previous studies (Phelan and Link, 2005b, Phelan et al., 2004, Mackenbach et al., 2015b). Although many causes of death have over the past decade become partly or largely preventable, e.g., by behavior change or improved medical care, some have not or less so, which may or may not be captured by variable speeds of actual mortality decline as postulated in the first hypothesis. We hypothesize that inequalities in mortality decline are larger for preventable than for non-

preventable causes of death. We will also assess for which category of preventability (i.e., behavior change, medical care, or injury prevention) differences in mortality decline are most evident. This may help us to discern different mechanisms; for example, if inequalities in mortality decline are largest for causes preventable by behavior change, that would point to differences in (barriers for) individual-level purposive action, whereas larger inequalities for causes preventable by medical care would suggest a more important role for differences in institutional treatment.

Our *fourth hypothesis* is that inequalities in mortality decline between educational groups are larger in countries where inequalities in material resources are larger. If the fundamental causes theory holds, one would expect the link between education and mortality from preventable causes to be stronger in countries with a more unequal distribution across educational groups of "general resources, like knowledge, money, power, prestige, and social connections" (Link and Phelan, 1995)(page 88). Because income is an important resource for healthy living (Morris et al., 2000), we will use income inequalities for testing this fourth hypothesis.

Data and methods

Data

Our analysis used mortality data from the period 1980-2010 from 20 populations in 17 European countries: Finland, Sweden, Norway, Denmark ('North'); United Kingdom (England & Wales and Scotland), Belgium, France, Switzerland, Austria ('West'); Spain (Barcelona, Basque Country and Madrid) and Italy (Turin) ('South'); and Slovenia, Czech Republic, Hungary, Poland, Lithuania and Estonia ('East'). Most data covered complete national or regional populations with the exceptions of England & Wales, Scotland, and France (1%, 5.3% and 1% representative samples of the population, respectively).

Most of these data were originally collected in the framework of a longitudinal mortality follow-up of a population census, in which socioeconomic information of the population-at-risk and of the deceased came from the same source, i.e. the census. However, data for Barcelona, Hungary, Czech Republic, Poland and Estonia derived from cross-sectional unlinked studies, in which socioeconomic information on the population-at-risk came from the census, and socioeconomic information on the deceased came from the death certificate. The analyses were restricted to the ages between 35 and 79 years. An overview of data sources is given in web appendix table S1, and more details can be found elsewhere (www.demetriq.eu).

Socioeconomic status was indicated by highest level of completed education. We focused on educational inequalities in mortality (instead of, e.g., occupational inequalities in mortality) primarily because comparable data on educational attainment are available for both men

and women in all European populations under study. Education is also the most stable measure of socioeconomic position because it is normally completed early in adulthood, which avoids most problems of reverse causation (i.e., health outcomes at older ages cannot change a person's level of education) (Daly et al., 2002). In addition to these technical advantages, there are also good theoretical reasons to use education as a marker of socioeconomic position, because education indicates investment in human capital, including communication, problem solving and complex thinking, fostering a sense of personal control which encourages and enables a healthy lifestyle (Mirowsky and Ross, 1998). Education was classified according to the International Standard Classification of Education (ISCED-97) (Unesco, 1997). The categories used in this analysis were 'no, primary or lower secondary education' (ISCED 0-2; 'low'), 'upper secondary and post-secondary non-tertiary education' (ISCED 3-4; 'mid') and 'tertiary education' (ISCED 5-6; 'high').

Our dataset allowed us to study 22 specific causes of death, plus four large groupings of causes of death (cardiovascular disease, cancer, other diseases and external causes). ICD-code numbers are given in web appendix table S2. In order to reduce random variation, for each population we only included results for causes of death for which the observed number of deaths in the total population over the whole observation period for that population exceeded 200 (males and females assessed separately). Web appendix table S3 presents summary information on the mortality data included in the analyses. The total number of deaths was more than 7.5 million.

We classified these 22 causes of death in four groups according to their "preventability" (Web appendix table S4), following the reasoning of a previous study (Mackenbach et al., 2015b). In brief, we distinguish between conditions amenable to behavior change, to medical care, and to injury prevention. Our criterion for classifying causes of death as "amenable to behavior change" was that the combined population-attributable fraction (PAF) for smoking, alcohol abuse, overweight, low fruit and vegetable intake, physical inactivity and unsafe sex was >50% in the Global Burden of Disease study 2000 (World Health Organization, 2002). Our criterion for classifying causes of death as "amenable to medical care" was that (a) relative 5-year survival rates around the year 2000 exceeded 70% in Eurocare (Verdecchia et al., 2007) and/or 80% in the United States Surveillance, Epidemiology and End Results (SEER) program (Ries et al., 2008), and/or (b) effective screening programs are available and had been implemented in European countries around the year 2000 (Mackenbach and McKee, 2013), and/or (c) they are among the conditions included in most selections of "conditions amenable to medical intervention" (Nolte and McKee, 2004). We also considered deaths due to injuries (road traffic accidents, accidental fall, suicide) as amenable to prevention through various means (Mann et al., 2005, Peden, 2004, Sethi, 2010, van der Feltz-Cornelis et al., 2011). All other causes of death were consideren non-preventable.

Data are available for all the European countries included in this analysis to show that these countries differ considerably in their extent of income inequality, rate of poverty and risk of social exclusion (OECD, 2011). We used the Standardized World Income Inequality Database (SWIID) to extract estimates of Gini coefficients of inequality in equivalized household

disposable (post-tax, post-transfer) income (Solt, 2009). Although these data do not directly reveal inequalities in average income between education groups, it is likely that countries with larger income inequalities generally also have larger inequalities in average income between education groups, because international comparisons of income returns to education show countries with large income inequalities generally to also have high income returns to education (Autor, 2014). The SWIID data allowed us to create three groups of observations: populations and time-periods with low income inequalities (Gini index < 26), populations and time-periods with medium-sized resource inequalities (Gini index 26-30), and populations and time-periods with large income inequalities (Gini index > 30)(see Web appendix table S5 for data on income inequality by population and time-period).

Analysis

Mortality rates by educational level were age-standardized, for men and women separately, using the European Standard Population (Ahmad et al., 2001).

Our analysis consisted of two parts. In a first part we calculated per cent per annum changes in age-standardized mortality over time for each period between two available observations (measured as the distance in years between the mid-points of each follow-up interval, and taking into account the fact that lengths of time intervals varied between 4 and 11 years (web appendix table S1)). We then compared median per cent per annum changes in mortality among the high, mid and low educated for each cause of death (taking together both sexes, and all populations and periods). We also determined the number of instances (i.e., combinations of sex, population, and cause of death) of declining and increasing mortality in the whole population, and the number of instances in which mortality among

the high educated declined more or increased less than mortality among the low educated, respectively.

In the second part we performed a regression analysis of the age-standardized mortality rates (by education, sex, population, period and 22 specific causes of death) on education, time and the interaction of education and time, in an analytical set-up inspired by that of Miech et al (Miech et al., 2011), controlling for sex, population, and cause of death. The regression equation can be written as follows:

$$\text{Log } M_{e,s,p,d,t} = \alpha + \beta_1 * E_m + \beta_2 * E_l + \beta_3 * T + \beta_4 * E_m * T + \beta_5 * E_l * T$$

in which M = age-standardized mortality rate, E = dummy variables indicating level of education, T = linear variable for calendar_year - 1980, α = constant indicating intercept, β = regression parameters, e = education, s = sex, p = population, d = cause of death, t = calendar_year, l = low education, m = mid education. This was done in a multilevel framework using linear mixed-effects models in which all parameters α and β consist of a fixed factor and a random factor with the interaction term sex*cause*population as clusters (Verbeke and Molenberghs, 2000). We used an autoregressive model of order 1 (AR(1)) to take into account the serial autocorrelation in the observed mortality rates; in our dataset the correlation between adjacent measurements is 0.75. In order to directly test differences between adjacent education groups the education dummy variables were defined as Helmert contrasts (Venables and Ripley, 2013). After obtaining the correct p-values for these differences the model was reparameterized in order to obtain time trends for each of the

education groups. The parameters of interest are β_4 and β_5 which indicate the (pooled) differences between education groups in (linear) changes in mortality over time. The logarithmic transformation of the mortality rate ensures that regression parameters indicate relative, not absolute, effects. For presentation purposes, regression parameters were transformed into estimates of per cent per annum mortality changes in all three education groups. These are reported in table 4 together with p-values for differences between education groups based on the Helmert parametrization. The multilevel regression analyses were conducted in R (Bates et al., 2014).

Results

Table 1 presents the results of an analysis in which we compared changes in mortality by educational group for all causes of death. For all-cause mortality, the total number of observations is 106, including 100 instances of declining mortality, and in 78% of these cases mortality declined more among the high than among the low educated. Median percent annual decline was 1.7% among the low educated, 1.8% among the mid educated, and 2.5% among the high educated. Faster mortality decline among the high educated is seen in a large majority of cases for almost all causes of death. For the 22 specific causes of death the total number of observations of mortality change was 1452, including 1021 instances of declining mortality, and in 69% of these cases mortality declined faster among the high than among the low educated.

Table 1 here

Median mortality decline was not faster among the high than the mid or low educated for hypertensive disease, stomach cancer, prostate cancer, leukemia, pneumonia, and appendicitis. Alcohol-related conditions are the only specific cause of death for which median mortality change was positive, i.e. for which mortality often increased, in all education groups. For most causes of death, however, median mortality decline was stronger among the high than among the mid than among the low educated.

Table 2 presents the results of a similar analysis by country, which shows that in about half of the populations median mortality decline was faster among the high than among the mid than among the low educated, with differences being particularly striking in the Czech Republic, Hungary, Lithuania and Estonia. In some populations, however, such as Denmark, England & Wales and Spain (Barcelona), median mortality change was more favorable among the low than among the high educated.

Table 2 here

Table 3 shows that when mortality in the whole population goes up, the high educated sometimes have more, sometimes less favorable mortality trends. For the 22 specific causes of death the total number of observations of increasing mortality was 418 (12 for rheumatic heart disease, plus 31 for hypertensive disease, etc.), and in 192 of these cases (46%) mortality increased less among the high educated. Examples of causes of death with increasing mortality for which median mortality change was less favorable among the high than among the low educated are rheumatic heart disease, hypertensive disease, colorectal cancer, leukemia, and appendicitis. Clearly less increasing mortality among the high educated is only found for lung cancer.

Table 3 here

A summary of the results of the multilevel regression analyses is presented in table 4. We see that in the dataset as a whole, for all 22 specific causes of death and 20 populations

combined, there is a substantial mortality decline among the high educated, of around 2.49% per annum per year (95% Confidence Interval: 2.04-2.92). The decline is less strong among the mid educated (1.83%; 95% CI: 1.37-2.30) and particularly the low educated (1.34%; 95% CI: 0.89-1.78). This confirms what we saw in table 2. The difference between mid and high educated is statistically significant (p=0.045), but the difference between the mid and low educated is not.

Table 4 here

When we stratify the regression analysis by speed of mortality decline in the whole population, we find somewhat larger differences in the magnitude of educational inequalities for conditions with faster than for conditions with slower mortality decline. When mortality declines faster than the median, the estimated annual mortality decline among the low, mid and high educated is 4.60%, 5.58% and 6.48%, respectively. When mortality declines slower than the median, the estimated annual mortality decline among the low, mid and high educated is 1.00%, 1.56%, and 2.33%, respectively. In the first case, the difference between low and high educated is around 1.9%-point, whereas in the second case it is 1.3%-point.

When mortality increases, mortality increases about as much among the low, mid and high educated (3.27%, 3.49% and 3.69% per annum, respectively), so contrary to our expectations it increases a bit more among the high educated although the differences are not statistically

significant (table 4). This confirms what we have seen in table 3, where we found many rising causes of death for which trends were least favorable among the high educated.

When a distinction is made between causes of death based on their preventability, either by behavior change, medical care or injury prevention, we find the largest differences in mortality decline between low and high educated for the group of causes amenable to behavior change: mortality increases by 0.80% per annum among the low educated, but declines by 0.93% per annum among the high educated, a difference of 1.73%-points. Differences between low and high educated are also substantial for conditions amenable to injury prevention, and as expected are smallest for non-preventable conditions (table 4).

When we stratify the analysis by level of income inequality, we paradoxically find the most consistent differences in mortality decline between the low, mid and high educated at low levels of income inequality (0.71%, 1.59%, and 2.68% per annum, respectively; differences between low and mid, and between mid and high educated are both statistically significant with p-values of 0.054 and 0.017, respectively). Inconsistent differences are found in situations of intermediate or high income inequalities.

Discussion

Summary of main findings

Faster mortality decline among the high educated was found for most causes of death and in most populations, and inequalities in mortality decline were larger when mortality declined faster. Whereas this confirms our first hypothesis, the results for the other three hypotheses were mixed. When mortality increased, less increase among the high educated was found in only 46% of cases. Faster mortality decline among the high educated was more manifest for causes of death amenable to intervention and less so for non-amenable causes. The difference in mortality decline between education groups was not larger when income inequalities were greater.

Limitations

This is probably the most comprehensive analysis of trends in inequalities in cause-specific mortality ever conducted, but its broad international scope inevitably raises issues of data comparability. Despite extensive harmonization efforts, our comparisons between countries may be biased by differences in data collection, for example with regard to study design, population coverage, time-periods covered, and data classification (web appendix table S1). For example, in Hungary, the Czech Republic, Poland and Estonia mortality data were collected in a cross-sectional unlinked design, which may produce over- or underestimation of mortality inequalities as compared to studies using a longitudinal design (Shkolnikov et al., 2007). However, as our analysis focused on changes over time, and study-designs remained the same, it is unlikely that our results are biased as a result of these differences in data design.

The data for Italy and Spain come from studies conducted in largely urban settings, which are not necessarily representative of the national population in these countries. However, the magnitude and patterns of inequalities in mortality in these Italian and Spanish populations (Mackenbach et al., 2008, Mackenbach et al., 1997) have been confirmed by studies covering the complete Italian and Spanish national populations (Regidor et al., 2012, Marinacci et al., 2013). Also, whether or not our results can be generalized to the national populations of these countries is not really an issue in this hypothesis-testing study, with the exception of our testing of the fourth hypothesis for which we had to assume that national estimates of income inequality for Italy and Spain apply to these regional populations.

In all populations covered by our analysis, the relative size of the lower educated group has diminished over time, while that of the higher educated group has increased over time. It is likely that this has also led to changes in the composition of these groups, e.g. that the lower educated group has become more homogeneous and/or more extreme in terms of socioeconomic disadvantage (Mackenbach, 2012). While this may have contributed to the less favorable development of mortality among the low educated, it is important to note that the reverse applies to the high educated, who as a group have become larger and more heterogeneous in terms of, e.g., the socioeconomic status of their family of origin. Apparently, this increased heterogeneity has not prevented them from achieving strong mortality declines.

Probably the most important limitation of our study is that we have used only one indicator of socioeconomic position. Although educational level is a widely used socioeconomic indicator in mortality studies, for the reasons mentioned in the Data and methods section, one cannot assume that results of analyses as reported in this paper will also apply to mortality by occupational class or income (Geyer et al., 2006). We therefore recommend to further test our hypotheses with other indicators of socioeconomic status. In the European context, this means replication with occupational class as a socioeconomic indicator (Toch-Marquardt et al., 2014), because mortality data can generally not be classified by income level.

Interpretation

A previous cross-sectional study ended with a recommendation to look at differences between socioeconomic groups in mortality trend (Mackenbach et al., 2015b), like some previous studies have done (Miech et al., 2011, Phelan and Link, 2005b, Masters et al., 2012). The present study, based on mortality trend data from a large number of European populations, generally confirms previous findings from the United States that relative declines in mortality are larger among the high than among the low educated. For the fundamental causes theory to be valid, it is not necessary that this is always the case – we suggest that in the stochastic world of inaccurately measured social phenomena the 69% we found provides sufficient support for our first hypothesis.

In further support of our first hypothesis we also found that inequalities in mortality decline between educational groups are larger when mortality decline in the whole population is faster. We assumed that the speed of mortality decline indicates the degree to which that

cause of death has become preventable, which is likely to be true although not necessarily by individual-level purposive action, as our results show. As can be seen in table 1, mortality declines from stomach cancer, a cause of death that cannot be regarded as "preventable" by behavior change, medical care or injury prevention (Web appendix table S4), are faster than the average for all causes of death together. This may be an example of a disease for which mortality decline does not depend on individual-level purposive action, but on improvements in the environment (e.g., methods of food conservation) which depend on collective action (Howson et al., 1986). Interestingly, we found no inequalities in speed of mortality decline for this cause of death.

Our results for the other three hypotheses were mixed. We found no support for our second hypothesis, i.e. that when mortality increases, the high educated are able to better protect themselves. Our results were in accordance with this hypothesis in the case of all-cause mortality, but in an analysis using all specific causes of death, we found more favorable trends for the high educated in only 46% of all cases of increasing mortality. Table 3 actually shows several causes of death for which mortality increase, if it occurred, was larger among the high than the mid or low educated, such as hypertensive disease, colorectal cancer and suicide. Somewhat stronger increases among the high educated were also found in the multilevel regression analysis (table 4). A possible explanation is that in early stages of the epidemic rise of a disease the rates are higher in higher socioeconomic groups, because the latter are the first to adopt new risk behaviors and products, as has happened in the case of smoking and delayed childbearing (Cavelaars et al., 2000, Menvielle et al., 2005). This would imply the presence of "countervailing mechanisms" (Lutfey and Freese, 2005) which may

arise from general processes of “diffusion of innovation” (Glied and Lleras-Muney, 2008, Chang and Lauderdale, 2009), but also from “status pursuit”: adopting behavior that is bad for health but helps to maintain or increase social status (Lutfey and Freese, 2005).

We did find support for our third hypothesis, i.e. that inequalities in mortality decline are larger for “preventable” than for “non-preventable” causes of death (table 4). The fact that the largest inequalities in mortality decline were found for conditions amenable to behavior change also provides support for the specific “metamechanism” postulated in the fundamental causes theory, i.e., individual-level purposive action with different means (Freese and Lutfey, 2011). However, if that would be the main mechanism, one would expect to not also find larger inequalities in mortality decline from conditions amenable to medical care. Of course, it is possible that lower educated patients with these conditions less actively seek care than higher educated patients, but an equally plausible explanation is that lower educated patients do not gain access to the health care system, or that the health care system is less effective in treating lower educated than higher educated patients. Previous European studies also found large inequalities in mortality from conditions amenable to medical care (Stirbu et al., 2010), but these could not be explained by simple measures of inequalities in health care utilization (Plug et al., 2012), suggesting differences in how patients are treated instead of differences in utilization of care.

Finally, we found no support for our fourth hypothesis, i.e. that inequalities in mortality decline are systematically larger when income inequalities are larger. Because we used a measure of net income, income inequalities will be larger if a country has larger inequalities

in market income (e.g., because of larger financial returns to education) or when a country has a less redistributive system of taxes and social transfers. For example, Spain has similar inequalities in market income as the Czech Republic, but because of greater redistribution in the Czech Republic the latter has smaller inequalities in net income (OECD, 2011). We found that countries in the same class of income inequalities (Web appendix table S5) are quite heterogeneous with regard to the magnitude and even direction of differences between high, mid and low educated in mortality decline (table 2). For example, Spain has rather large income inequalities, but mortality decline among the low educated is not less than that among the mid or high educated. On the other hand, the Czech Republic has small income inequalities but differences in mortality decline between high and low educated are large. This suggests that inequalities in access to material resources are not the main factor underlying the advantage of high over low educated in benefiting from opportunities for mortality decline, perhaps because of the extensive welfare arrangements that have been created in most European countries. Inequalities in access to other resources, such as knowledge, social connections or cultural capital may be more important. We therefore recommend further studies to test whether countries with larger inequalities in non-material resources do have larger inequalities in mortality decline.

Conclusions

Our results provide further support for the fundamental causes theory, but in this broad European setting the empirical reality appears to be more heterogeneous than the theory predicts. Our results suggest the presence of “countervailing mechanisms” as well as of other “metamechanisms” than the theory implies. Furthermore, our findings make it less

likely that, in a modern European setting, the main barrier for achieving good health is a lack of material resources.

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Table 1. Comparison of mortality change between high, mid and low educated, by cause of death (based on pooling of data over countries and time)

		Median per cent annual change			Declining mortality	
	Number of observ-ations	Low	Mid	High	Number of observ-ations	% Faster among high
Total mortality	106	-1.7%	-1.8%	-2.5%	100	78
Cardiovascular diseases	106	-3.5%	-4.1%	-4.4%	104	74
Rheumatic heart disease	59	-6.0%	-7.1%	-9.6%	47	68
Hypertensive disease	80	-1.3%	-1.2%	-0.9%	49	57
Ischaemic heart disease	100	-3.7%	-4.7%	-5.2%	97	78
Other heart disease	80	-1.5%	-1.6%	-2.0%	55	56
Cerebrovascular disease	94	-3.6%	-3.7%	-4.6%	91	64
Cancer	106	-0.7%	-1.0%	-1.6%	90	79
Lung cancer	93	0.5%	-0.2%	-1.4%	52	77
Breast cancer	53	-0.8%	-2.4%	-2.0%	41	66
Colorectal cancer	82	-0.5%	-1.0%	-1.9%	63	78
Stomach cancer	82	-3.4%	-3.4%	-3.5%	72	50
Prostate cancer	41	-0.8%	-2.1%	-2.0%	29	76
Hodgkin's disease	18	-4.9%	-5.0%	-7.7%	15	73
Leukemia	76	-1.3%	-0.8%	-1.6%	54	65
Other diseases	106	-0.1%	-0.5%	-1.2%	65	80
Tuberculosis	31	-4.9%	-7.1%	-7.5%	25	68
Other infectious diseases	64	1.9%	1.7%	-0.4%	22	82
Pneumonia	82	-1.4%	-1.1%	-2.6%	52	79
Asthma	39	-4.8%	-6.8%	-9.0%	35	77
Appendicitis etc.	47	-3.7%	-2.4%	-2.2%	31	77
Peptic ulcer	63	-2.1%	-3.0%	-4.1%	42	74
Alcohol-related causes	89	3.4%	3.0%	2.9%	20	65
External causes	104	-0.8%	-1.1%	-2.4%	72	72
Road traffic accidents	80	-3.6%	-3.4%	-4.5%	67	69
Suicide	73	-0.9%	-1.2%	-2.5%	44	77
Homicide	26	-0.7%	-1.5%	-1.9%	18	67

In grey: median mortality decline stronger among high than among mid than among low educated.
Number of observations = number of combinations of population, period, and sex for which information on mortality change was available.

Table 2. Comparison of mortality change between high, mid and low educated, by population (based on pooling of data over causes of death and time)

			Median % annual change		
		Number of observ-ations	Low educated	Mid educated	High educated
North	Finland	210	-2.0%	-2.0%	-3.1%
	Sweden	33	-0.9%	-1.7%	-1.4%
	Norway	150	-0.9%	-1.6%	-2.4%
	Denmark	70	-2.0%	0.2%	-1.3%
West	Scotland	21	-3.8%	-4.8%	-2.8%
	England & Wales	125	-2.8%	N/A	-2.1%
	Belgium	27	-1.9%	-3.7%	-4.3%
	France	24	-2.7%	-2.9%	-0.7%
	Switzerland	123	-3.1%	-3.3%	-3.5%
	Austria	57	-3.1%	-3.7%	-3.8%
South	Spain (Barcelona)	98	-2.4%	-1.8%	-1.6%
	Spain (Basque)	29	-1.6%	0.5%	-0.7%
	Spain (Madrid)	22	-3.2%	-3.0%	-4.0%
	Italy (Turin)	154	-3.0%	-3.3%	-3.8%
East	Slovenia	16	-1.1%	-1.1%	-2.0%
	Czech Republic	39	-1.5%	-5.0%	-5.2%
	Hungary	76	-0.2%	-1.7%	-3.9%
	Poland	42	-1.5%	-2.3%	-1.6%
	Lithuania	58	2.8%	1.7%	-1.1%
	Estonia	31	1.7%	-0.6%	-2.0%

In grey: median mortality decline stronger among high than among mid than among low educated
Number of observations = number of combinations of period, cause of death and sex for which information on mortality change was available.

Table 3. Comparison of mortality change between high, mid and low educated, by cause of death, increasing mortality in whole population only (based on pooling of data over countries and time)

		Median % annual change			Less mortality increase among high educated	
	Number of observ-ations	Low educated	Mid educated	High educated	Number of observ-ations	% of all observ-ations
Total mortality	**6**	**2.3%**	**1.5%**	**-0.7%**	**5**	**83**
Cardiovascular diseases	**-**	**-**	**-**	**-**	**-**	**-**
Rheumatic heart disease	12	-3.3%	1.8%	12.5%	3	25
Hypertensive disease	31	3.7%	5.2%	7.3%	14	45
Ischaemic heart disease	-	-	-	-	-	-
Other heart disease	25	2.2%	1.8%	2.9%	13	52
Cerebrovascular disease	-	-	-	-	-	-
Cancer	***16***	***0.9%***	***1.2%***	***0.3%***	***10***	***63***
Lung cancer	41	3.2%	2.1%	1.0%	31	76
Breast cancer	12	1.2%	0.6%	1.1%	7	58
Colorectal cancer	19	0.1%	0.7%	2.3%	5	26
Stomach cancer	10	-1.2%	2.2%	8.1%	2	20
Prostate cancer	12	1.0%	3.3%	1.8%	7	58
Hodgkin's disease	-	-	-	-	-	-
Leukemia	22	-0.8%	2.5%	2.2%	7	32
Other diseases	***41***	***1.2%***	***1.1%***	***1.1%***	***22***	***54***
Tuberculosis	6	7.4%	3.1%	9.3%	4	67
Other infectious diseases	42	3.5%	4.6%	4.6%	22	52
Pneumonia	30	1.7%	3.9%	4.1%	12	40
Asthma	-	-	-	-	-	-
Appendicitis etc.	16	1.9%	2.3%	7.6%	2	13
Peptic ulcer	21	1.0%	2.8%	4.8%	7	33
Alcohol-related causes	69	4.4%	4.1%	4.5%	36	52
External causes	***32***	***1.4%***	***1.4%***	***1.7%***	***17***	***53***
Road traffic accidents	13	-0.8%	2.2%	2.9%	3	23
Suicide	29	1.3%	1.2%	2.4%	14	48
Homicide	8	1.0%	4.9%	1.7%	3	38

In grey: median mortality change more favorable among high than among mid than among low educated. Number of observations = number of combinations of population, period, and sex for which information on mortality change was available.

Table 4. Results of regression analysis

		Low educated: % p.a. change			Mid educated: % p.a. change			High educated: % p.a. change			P-value signif. test	
	Number of observ-ations	Estimate	95% CI		Estimate	95% CI		Estimate	95% CI		Low vs mid	Mid vs high
All observations	5955	**-1.34**	-1.78	-0.89	**-1.83**	-2.30	-1.37	**-2.49**	-2.92	-2.04	0.131	0.045
Decreasing mortality (> median)	2223	**-4.60**	-5.12	-4.06	**-5.58**	-6.13	-5.02	**-6.48**	-7.00	-5.96	0.012	0.020
Decreasing mortality (< median)	2226	**-1.00**	-1.55	-0.45	**-1.56**	-2.13	-0.99	**-2.33**	-2.87	-1.79	0.167	0.054
Increasing mortality	1506	**3.27**	2.56	3.99	**3.49**	2.75	4.24	**3.69**	2.97	4.40	0.675	0.717
Amenable to behavior change	1137	**0.80**	-0.21	1.82	**0.17**	-0.89	1.24	**-0.93**	-1.92	0.08	0.400	0.141
Amenable to medical care	2811	**-2.42**	-3.04	-1.80	**-3.07**	-3.71	-2.43	**-3.44**	-4.05	-2.82	0.154	0.424
Amenable to injury prevention	735	**-1.54**	-2.77	-0.29	**-1.69**	-2.96	-0.41	**-3.04**	-4.26	-1.81	0.867	0.134
Non-preventable	1272	**-0.76**	-1.70	0.18	**-0.99**	-1.96	0.00	**-1.47**	-2.39	-0.53	0.748	0.486
Low income inequalities	2619	**-0.71**	-1.34	-0.07	**-1.59**	-2.22	-0.96	**-2.68**	-3.30	-2.05	0.054	0.017
Intermediate income inequalities	1950	**-2.63**	-3.28	-1.98	**-2.86**	-3.55	-2.16	**-2.51**	-3.16	-1.86	0.647	0.480
High income inequalities	1386	**-1.16**	-2.35	0.06	**-0.47**	-1.93	1.02	**-1.66**	-2.86	-0.46	0.478	0.216

Results of multilevel regression analyses of log ASMR on education, year and education*year, controlling for sex, population and cause of death. For details about the autoregressive regression model, see Data and methods section. Analyses included 22 specific causes of death. Median value of mortality decline used for stratification by speed of mortality decline = -3.02% per annum. For classification of causes of death by preventability, see Web appendix table S4. Low income inequalities = Gini < 26. Intermediate income inequalities = 26 < Gini < 32. High income inequalities = Gini > 32. Number of observations = number of combinations of population, cause of death, year, sex and educational group for which information on mortality rates was available.

Supplementary materials

Table S1. Data sources

	Overview of data sources									
Region	Country	Design	Inclusion	Age-range	Periods covered					
North	Finland	Longitudinal	All	35–79	1980-85	1985-90	1990-95	1995-00	2000-05	2005-10
	Sweden	Longitudinal	All	35–79			1990-94	1995-99	2000-04	2005-08
	Norway	Longitudinal	All	40–79	1980-85	1985-90	1990-95	1995-01	2001-06	2006-09
	Denmark	Longitudinal	All	35-79			1991-95	1996-00	2001-05	
West	UK (Scotland)	Longitudinal	All	35–79			1991-95	1996-00	2001-05	2006-10
	UK (England&Wales)	Longitudinal	1% sample	35–79	1981-86	1986-91	1991-96	1996-01	2001-06	2006-09
	Belgium	Longitudinal	All	35–79			1991-96	2004-05		
	France	Longitudinal	1% sample	35–79	1980-82*	1987-90	1990-95	1995-99	1999-04	2004-07
	Switzerland	Longitudinal	Swiss nationals	35–79			1990-95	1995-00	2000-05	2005-08
	Austria	Longitudinal	All	35–79	1981-82		1991-92		2001-02	
South	Spain (Barcelona)	Cross-sectional	All (city)	35–79			1992-96	1997-01	2002-06	2007-10
	Spain (Basque Country)	Longitudinal	All (region)	35–79				1996-01	2001-06	
	Spain (Madrid)	Longitudinal	All (region)	35–79				1996-97	2001-03	
	Italy (Turin)	Longitudinal	All (city)	35–79	1981-86	1986-91	1991-96	1996-01	2001-02	2006-10
East	Slovenia	Longitudinal	All	35–79			1991-95		2002-06	
	Czech Republic	Cross-sectional	All	35–79	1982-85			1998-03		
	Hungary	Cross-sectional	All	35–79	1978-81		1988-91		1999-02	
	Poland	Cross-sectional	All	35–64			1991-93		2001-03	
	Estonia	Cross-sectional	All	35–79			1987-91		1998-02	
	Lithuania	Longitudinal**	All	35–69			1988-90		2001-05	2006-09
	* Data also available for 1982-87									
	** Cross-sectional in 1988-02, adjusted to longitudinal									

Table S2. ICD-10 codes for the causes of death included in the analysis

	ICD-10 codes
Total mortality, all causes	A00-Y98
Cadiovascular diseases	I00-I99
Rheumatic heart disease	I00-I09
Hypertensive disease	I10-I15
Ischaemic heart disease	I20-I25
Other heart disease	I26-I52; I98
Cerebrovascular disease	I60-I69
Cancer	C00-D48
Cancer of trachea, bronchus and lung	C33-C34
Breast cancer	C50
Colorectal cancer	C18-C21
Stomach cancer	C16
Prostate cancer	C61
Hodgkin lymphoma	C81
Leukemia	C91-C95
Other diseases	Rest (A00-U85)
Tuberculosis	A15-19, B90
Other infectious diseases	Rest A00-B99
Pneumonia	J10-J18
Asthma	J45-J46
Appendicitis, hernia, cholecystitis and lithiasis	K35-K38; K40-K46; K56; K80-K83
Peptic ulcer	K25-K28
Alcohol-related causes*	F10; I42.6; K70; X45
External	V01-Y98
Road traffic accidents	V01-V89, Y85
Suicide	X60-X84, Y87.0
Homicide	X85-Y09, Y87.1

* Alcohol-related causes include: alcoholic psychosis, dependence and abuse; alcoholic cardiomyopathy; alcoholic cirrhosis of the liver; accidental poisoning by alcohol

Table S3. Summary information on the mortality data included in the analysis

	Number of deaths	Average ASMR
Total mortality	**7,622,030**	**880.7**
Cardiovascular diseases	***2,841,530***	***319.0***
Rheumatic heart disease	29,539	4.8
Ischaemic heart disease	1,391,511	169.0
Hypertensive disease	94,298	11.1
Other heart disease	224,964	33.5
Cerebrovascular disease	679,428	75.9
Cancer	***2,245,754***	***311.1***
Lung cancer	507,581	68.2
Breast cancer	172,752	52.5
Colorectal cancer	224,810	35.8
Stomach cancer	124,718	18.5
Prostate cancer	88,547	30.4
Hodgkin's disease	3,916	1.2
Leukemia	51,407	8.5
Other diseases	***1,494,204***	***185.9***
Tuberculosis	19,423	5.9
Other infectious diseases	30,539	6.2
Pneumonia	101,995	14.1
Asthma	14,448	3.1
Appendicitis etc.	28,207	4.2
Peptic ulcer	51,782	5.0
Alcohol-related causes	176,086	25.4
External	***510,502***	***66.0***
Road traffic accidents	80,890	11.1
Suicide	147,511	23.9
Homicide	9,534	2.2

ASMR = age-standardized mortality rate. Average ASMR = arithmetic average of population-specific ASMRs.

Table S4. Classification of causes of death by preventability

	Amenable to			
	Behavior change	Medical care	Injury prevention	Total preventable
Rheumatic heart disease	no	yes	no	yes
Hypertensive disease	no	yes	no	yes
Ischaemic heart disease	yes	no*	no	yes
Other heart disease	no	no	no	no
Cerebrovascular disease	no	yes	no	yes
Lung cancer	yes	no	no	yes
Breast cancer	no	yes	no	yes
Colorectal cancer	no	no	no	no
Stomach cancer	no	no	no	no
Prostate cancer	no	yes	no	yes
Hodgkin's disease	no	yes	no	yes
Leukemia	no	yes	no	yes
Tuberculosis	no	yes	no	yes
Other infectious diseases	no	no	no	no
Pneumonia	no	yes	no	yes
Asthma	no	yes	no	yes
Appendicitis etc.	no	yes	no	yes
Peptic ulcer	no	yes	no	yes
Alcohol-related causes	yes	no	no	yes
Road traffic accidents	no**	no**	yes	yes
Suicide	no**	no**	yes	yes
Homicide	no**	no**	yes	yes

Notes:
* Ischemic heart disease mortality can also be avoided by appropriate medical care, but behavior change probably played a larger role in many European countries
** Although mortality from injuries can also partly be prevented by behavior change and/or medical care, injuries have been classified as a separate group

Table S5. Income inequality in the populations included in the study, by time period

country	year	Gini_net		country	year	Gini_net
Austria	1982	26.5		Lithuania	1989	22.1
Austria	1991	26.1		Lithuania	2003	33.6
Austria	2001	26.1		Lithuania	2007	34.9
Belgium	1993	23.7		Norway	1983	22.1
Belgium	2004	26.0		Norway	1988	23.0
Czech Republic	1983	20.8		Norway	1993	23.8
Czech Republic	2001	23.7		Norway	1998	23.4
Denmark	1993	22.7		Norway	2004	25.7
Denmark	1998	23.2		Norway	2008	24.4
Denmark	2003	21.4		Poland	1992	26.2
England/W	1983	27.6		Poland	2002	28.5
England/W	1988	31.6		Scotland	1993	33.9
England/W	1993	33.9		Scotland	1998	34.3
England/W	1998	34.3		Scotland	2003	34.2
England/W	2003	34.2		Scotland	2008	35.8
England/W	2008	35.8		Slovenia	1993	21.5
Estonia	1989	24.3		Slovenia	2004	23.1
Estonia	2000	36.1		Spain (Barcelona)	1994	35.2
Finland	1983	20.3		Spain (Barcelona)	1999	33.8
Finland	1988	20.9		Spain (Barcelona)	2004	31.6
Finland	1993	20.8		Spain (Barcelona)	2008	31.7
Finland	1998	23.6		Spain (Basque Country)	1998	34.1
Finland	2003	25.2		Spain (Basque Country)	2004	31.6
Finland	2008	26.3		Spain (Madrid)	1997	34.9
France	1981	29.9		Spain (Madrid)	2002	32.6
France	1984	33.8		Sweden	1992	22.9
France	1988	29.4		Sweden	1997	21.5
France	1992	28.7		Sweden	2002	22.9
France	1996	29.0		Sweden	2007	24.6
France	2001	27.5		Switzerland	1993	29.8
France	2005	28.0		Switzerland	1998	25.4
Hungary	1980	21.0		Switzerland	2003	27.6
Hungary	1990	26.8		Switzerland	2007	31.5
Hungary	2001	27.3				
Italy (Turin)	1984	31.1				
Italy (Turin)	1989	30.5				
Italy (Turin)	1994	33.5				
Italy (Turin)	1999	33.7				
Italy (Turin)	2004	34.0				
Italy (Turin)	2008	32.4				

Gini_net = Gini coefficient of equivalized household disposable (post-tax, post-transfer) income.