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**Working Paper**

## Inequity in the Face of Death

Tinbergen Institute Discussion Paper, No. 12-084/3

**Provided in Cooperation with:**

Tinbergen Institute, Amsterdam and Rotterdam

*Suggested Citation:* Garcia-Gomez, Pilar; Schokkaert, Erik; Ourti, Tom Van; d'Uva, Teresa Bago (2012) : Inequity in the Face of Death, Tinbergen Institute Discussion Paper, No. 12-084/3, Tinbergen Institute, Amsterdam and Rotterdam, <https://nbn-resolving.de/urn:nbn:nl:ui:15-1765/37311>

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TI 2012-084/3

Tinbergen Institute Discussion Paper



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May 21, 2012

## Abstract

We apply the theory of inequality in opportunity to measure inequity in mortality. Our empirical work is based on a rich dataset for the Netherlands (1998-2007), linking information about mortality, health events and lifestyles. We show that distinguishing between different channels via which mortality is affected is necessary to test the sensitivity of the results with respect to different normative positions. Moreover, our model allows for a comparison of the inequity in simulated counterfactual situations, including an evaluation of policy measures. We explicitly make a distinction between inequity in mortality risks and inequity in mortality outcomes. The treatment of this difference - “luck” - has a crucial influence on the results.

*JEL Classification codes:* D63; I12; I14.

*Keywords:* equity, equality of opportunities, mortality, lifestyles.

# 1 Introduction

Economists have always been concerned about the measurement of inequality in outcomes as an important determinant of overall social welfare. Recently, however, there has been an increased interest in inequality of opportunity rather than inequality of outcomes. The theoretical fundamentals of this approach have been explained in important monographs by Roemer (1998) and Fleurbaey (2008). The number of empirical applications is growing rapidly – not only in the domain of income distribution (Roemer et al., 2003; Bourguignon et al., 2007; Devooght, 2008; Lefranc et al., 2008, 2009; Checchi and Peragine, 2010; Aaberge et al., 2011; Almas et al., 2011), but also in other domains such as health (Rosa Dias, 2009; Trannoy et al., 2010; Jusot et al., 2010).

The main intuition of the “inequality of opportunity” approach is that economic agents should be held responsible for at least part of the observed outcome differences. Social concern is restricted to inequalities in outcomes that are not the responsibility of the individual. Where to draw the line between factors for which individuals should be held responsible and factors for which they should not, is a normative question about which opinions in society differ. Economists have therefore proposed a unifying formal framework, which can accommodate different philosophical perspectives. Suppose the outcome of interest  $y$  is linked to a vector of explanatory variables  $z$  through the function  $y = Y(z)$ . The first step is to partition the vector  $z$  into variables for which individuals are not held responsible (often called “circumstances”  $c$ ) and variables for which they are (often called “effort”  $e$ ). This partitioning reflects a normative choice and can be taken as the starting point for the second step: the derivation of inequality measures which capture the inequality in  $y$  in so far (and only in so far) as it is linked to the  $c$ -variables. Of course, the results of this second step will depend on the chosen partitioning, but the formal approach can be applied to any partitioning. Note that the traditional focus on pure outcome (e.g. income) inequality is not less normative than the other approaches: it is just the special case where considerations of individual responsibility are set aside.

The empirical application of this formal framework raises some difficult issues. First, while the theoretical rationale for partitioning the vector  $z$  is clear, it is often difficult to relate the specific empirical variables in  $z$  to common opinions about justice or to more sophisticated philosophical theories. Indeed, many variables seem to be of a “mixed” nature, partly reflecting responsibility and choice, partly reflecting circumstances. Lifestyles are a typical example. Second, in real-world applications the available information will always be incomplete. Estimation of the function  $Y(z)$  will therefore yield unexplained variation, a residual, capturing specification error, the effect of omitted variables and pure random factors. How to treat this residual? Roemer (1998, 2002) has put forward an innovative solution to both problems. He proposed to restrict circumstances to a limited set of variables – and he defines individuals to be of the same “type” if they are in identical circumstances, defined in this restrictive way. All the other variables, including the residual, are then interpreted as effort. More specifically, he defines the level of effort in terms of the percentile occupied by individuals in the outcome distribution of their “type”. This reflects the normative idea that all variables (whether observed or not) that are correlated with type should also be interpreted as circumstance. The Roemer-approach gives coherent theoretical foundations for a pragmatic approach to measuring inequality of opportunity. Yet, the narrow definition of “types” gives only a lower bound for a measure of inequality of opportunity, which is sometimes hard to interpret. Moreover, the pragmatic stance of Roemer does not take up the challenge of linking the empirical measurement exercise to the rich philosophical debate on the different dimensions of equality of opportunity.

In this paper we explore the potential of a more ambitious approach. First, we claim that the difficulty of partitioning  $z$  is linked to the fact that variables exert an influence through different channels, which get mixed up when focusing on the reduced form  $Y(z)$ . We argue it is essential to estimate a model that distinguishes between these different channels and makes it possible to assess their relative importance (see also Fleurbaey and Schokkaert, 2009).<sup>1</sup> To

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<sup>1</sup>Deaton (2011) writes: “It is possible that an inequality that might seem to be prima facie unjust might actually be the consequence of a deeper mechanism that is in part benevolent, or that is unjust in a different

this end, we use a recursive modeling strategy that exploits timing between events and exclusion restrictions. Second, we refrain from the practice of treating the whole residual as “effort”. We introduce specific measures of effort in our model and interpret the residual in terms of “luck”. This richer framework allows us to do a sensitivity analysis with respect to different normative views concerning the location of the responsibility cut (Fleurbaey, 2008) and concerning the treatment of luck (Lefranc et al., 2009; Fleurbaey, 2010).

Our empirical application is to health, and more specifically mortality. Evaluation of inequalities in the face of death certainly are one of the most ethically challenging issues. We exploit rich diagnostic information from hospital admissions and cause of death registries in the Netherlands to estimate a recursive model. In the health domain it is natural to accept that “effort” and individual responsibility are linked to the choice of lifestyles. At least since Grossman (1972), the role of lifestyles has been acknowledged in theoretical models of health production and there exist good examples of rich models of lifestyle and mortality (e.g. Balia and Jones, 2008, 2011). The identification of effort variables is more difficult in the case of income distribution, since it is hard to simply interpret effort as the number of hours worked. The health setting is therefore an interesting one to test the interpretative potential of rich models of inequality of opportunity (Fleurbaey and Schokkaert, 2011). However, while our application is to health, the methodological questions we address (interpretation of a model that distinguishes between different channels, implementation of different philosophical theories, treatment of the residual) are relevant for all applications of equality of opportunity, also in other domains.

Our model reveals strong educational gradients in healthy lifestyles, and healthy lifestyles have a protective effect on mortality in the Netherlands. We use our model to illustrate how different normative views influence the measured degree of inequity, and show that the location of the responsibility cut is of vital importance. We observe, for example, that the traditional measures of inequity (such as socioeconomic and regional inequalities) only capture part of more way”. We interpret this quote as an appeal to base equity judgments on the insights of a model that distinguishes between the different channels.

comprehensive notions of unfairness, and that the usual practice of standardizing for age and gender can have large effects on measured inequity. We further illustrate that the use of a flexible empirical approach which accommodates the likely situation where the mortality impact of “circumstances” depends on “effort” levels, and vice-versa, can dramatically complicate the measurement of inequity. Fortunately, the proposed unifying formal framework seems to offer a way out. Finally, we show that the treatment of the residuals is important in this context, not just from a statistical point of view, but also because it entails normative choices in itself. We interpret the residuals of our mortality model as “option” luck, the kind of luck that can be avoided by prudent people if they take precautions, and “brute” luck, the luck that even reasonable and prudent people remain vulnerable to (Dworkin, 1981). We show that “brute” luck in mortality completely dominates measured inequity. We also test and reject the assumption of “even-handedness” of luck invoked by Lefranc et al. (2009).

The remainder of the paper is organized as follows. Section 2 introduces our formal framework for measuring inequity. Section 3 describes the data, the econometric model and the estimation results. Section 4 contains our results concerning inequity in mortality risks, illustrates the importance of the location of the responsibility cut and compares our approach with the Roemer-approach, which does not distinguish between different channels through which mortality is determined. Section 5 discusses the treatment of luck. Finally, Section 6 concludes.

## 2 Equality of opportunity and fair allocations

We will introduce our basic measurement concepts for the simplest possible deterministic reduced form and then describe how the approach can be extended to a recursive model with random variables. The outcome of interest is mortality  $M$  that is related to a set of variables  $z$  through the function  $M(z)$ . To implement the idea of inequality in opportunity<sup>2</sup>, we partition the vector  $z$  in a subvector of “circumstances”  $c$  for which individuals cannot be held responsible (and that therefore lead to unequal opportunities) and a subvector of “effort” variables  $e$  for which

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<sup>2</sup>We will use the terms “inequality in opportunity” and “inequity” interchangeably.



individuals should be held responsible. The mortality of individual  $i$  can then be written as:

$$M_i = M(c_i, e_i) \tag{1}$$

The traditional economic approach, focusing on socioeconomic differences in mortality (see, e.g. Attanasio and Emmerson, 2003), implicitly takes socioeconomic status (SES) as the circumstance variable – and all other variables as effort. Interpreting lifestyle as the effort variable, and all other variables as circumstances is another example. These are only two possible approaches, however, and we will describe a whole range of alternative views on the responsibility cut in section 4. The formal analysis in this section can be applied to any partition  $(c_i, e_i)$ .

Given a partitioning, the question then is how to measure “illegitimate” inequalities of opportunities, i.e. how to purge a simple inequality measure of the effects for which individuals should be held responsible. Fleurbaey and Schokkaert (2009) propose two methods. The first is called “direct unfairness” – it consists of putting the effort variables at reference values and then measuring inequality in the adjusted advantage measures  $a_i = M(c_i, \tilde{e})$ . It is clear that inequality in  $a_i$  can only be due to differences in  $c_i$ , since the effort variables are fixed. The second starts from the definition of what is considered to be an “equitable situation” – in which all inequalities are linked to effort variables, by putting all circumstance variables at reference values – and calculates individual “fairness gaps” ( $fg_i$ ) as the difference between the actual situation and this equitable point of reference, i.e.

$$fg_i = M(c_i, e_i) - M(\tilde{c}, e_i) \tag{2}$$

Inequity is then defined as inequality in these fairness gaps.

The literature has thus far mainly opted (implicitly or explicitly) to calculate direct unfairness. This may indeed seem the most natural of the two approaches. This choice is not innocuous, however. A natural condition to be imposed on an inequity measure is what Fleurbaey and Schokkaert (2009) call “compensation”: inequity should only be zero if two individuals with exactly the same value for the effort (responsibility) variables also have the same mortality. Only in that case do we fully include in our measure of inequity all effects of differences in

circumstances. It is immediately clear that the fairness gap satisfies this condition, while the measure of direct unfairness does not. Consider two individuals  $i$  and  $j$  with  $e_i = e_j = e$ . It is very well possible that  $M(c_i, \tilde{e}) = M(c_j, \tilde{e})$ , while at the same time  $M(c_i, e) \neq M(c_j, e)$ . On the other hand,  $fg_i = fg_j$  if and only if  $M(c_i, e) = M(c_j, e)$ .

The problem with direct unfairness is linked to the existence of what Gravelle (2003) in a similar setting has called “essential nonlinearities”, i.e. a situation where the value of  $e$  influences the marginal effect of  $c$ . More generally, the idea of “essential nonlinearities” also helps to understand the differences between the results for direct unfairness and for the fairness gap. Fixing effort (respectively circumstances) at their reference value in  $M(c_i, \tilde{e})$  (resp.  $M(\tilde{c}, e_i)$ ) in a certain sense “removes” the impact of the values of  $e_i$  (resp.  $c_i$ ) on the marginal effect of  $c_i$  (resp.  $e_i$ ) on mortality. These “essential nonlinearities” will therefore not be taken into account in the calculation of direct unfairness, which is simply based on  $M(c_i, \tilde{e})$ . However, they do reappear in the fairness gap through the observed mortality  $M(c_i, e_i)$ . If the marginal effect of effort on mortality depends positively (negatively) on the value of the circumstances (or vice versa), we may expect a positive (negative) effect on inequity as measured by the fairness gap. For example, and anticipating our results in section 3 and 4, we find that mortality is higher among older individuals and among individuals with low education, but also that the effect of education is larger for older individuals. Under the ethical stance that takes age as the effort variable and education as the circumstance variable (commonly known as age-standardisation), the difference between direct unfairness and the fairness gap will crucially depend on the magnitude and sign of the interaction between education and age. Since we find that the combination of low education and old age reinforces the mortality risk, one would expect larger inequities as measured by the fairness gap compared to direct unfairness, which is exactly what we find in our empirical analysis. We will further illustrate in section 4 that these “essential nonlinearities” indeed play a crucial role.<sup>3</sup>

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<sup>3</sup>The literature (e.g. Fleurbaey, 2008) shows that different approaches to inequality of opportunity not only differ in their degree of respect for the compensation principle, but also embody different ideas about “reward”, i.e. about what is the optimal distribution in the hypothetical situation where all individuals are of the same

In many cases it will be difficult to classify a given variable  $z$  unambiguously in  $c$  or in  $e$ . The problem can be illustrated – and is at the same time partly solved – by the use of a recursive model that underlies the reduced form  $M(z_i)$  and that identifies different channels through which variables affect mortality. Let us specify:

$$M_i = m(H_i, L_i, x_i) \tag{3}$$

$$H_i = h(L_i, x_i) \tag{4}$$

$$L_i = l(x_i, \pi_i) \tag{5}$$

where  $M$  stands for mortality,  $H$  for health “events (or shocks)”,  $L$  for lifestyles,  $x$  is a vector of exogenous variables that affect lifestyles, health and mortality (such as education or age) and  $\pi$  a vector of (exogenous) preference shifters (only influencing  $L$ ). For expositional purposes it is convenient to summarize eqs. (3)-(5) in the quasi-reduced form

$$M_i = m [h(l(x_i, \pi_i), x_i), l(x_i, \pi_i), x_i] \tag{6}$$

which can be compared with the reduced form

$$M_i = M(x_i, \pi_i) \tag{7}$$

Consider now the effect of one specific variable in  $x_i$ . Take for the sake of illustration age. Eq. (6) shows that age may influence mortality through three different channels: (a) there may be an effect of age on mortality, conditional on health shocks and lifestyle; (b) age may affect health shocks (and hence mortality), conditional on lifestyle; (c) age may have an influence on lifestyle. A priori there is no reason why these effects should have the same ethical status, e.g. people with lower age could be held responsible for their lifestyle, but not for the effects of age on health shocks and on mortality. When working with the reduced form these effects cannot be distinguished. The finer distinctions can be introduced in the quasi-reduced form (6), however. This seems a decisive advantage. We will show relevant examples in section 4.

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type. The reward principles underlying direct unfairness and the fairness gap are discussed in Fleurbaey and Schokkaert (2011): they are closely related to the principle of liberal reward.

Let us finally relax the assumption that the model is fully deterministic. Introducing a stochastic term  $\varepsilon$  in each of the three equations (3)-(5) yields (in obvious notation) the quasi-reduced form

$$M_i = m [h(l(x_i, \pi_i, \varepsilon_i^L), x_i, \varepsilon_i^H), l(x_i, \pi_i, \varepsilon_i^L), x_i, \varepsilon_i^M)]. \quad (8)$$

In any empirical application such as our own, one has to estimate the functions (3)-(5). One can then obtain estimates for the expected value of mortality  $\widehat{E}(M_i|x_i, \pi_i)$  and apply the inequity measures to this. This is the most common approach in applied work. It corresponds to ignoring everything that cannot be explained by the empirical model. If all this could be considered to be “luck”, then this would mean that this approach simply ignores “luck” in the measurement of inequity in mortality. We return to this issue below. A second approach is to focus on actual outcomes after the realization of “luck”. In the context of our empirical model, this corresponds to considering not only the predicted expected value of  $M_i$  but also the residuals of the model. In this broader approach, these can be classified, just as all other variables, as either circumstances  $c$  or effort  $e$ . The measures of direct unfairness and fairness gap can then be applied to this “extended” set of variables. A third approach has been proposed by Lefranc et al. (2009). They treat “luck” as a separate variable with its own characteristics, which cannot be reduced to either circumstances or effort. According to them, “luck” does not create any inequity if it is “even-handed”, i.e. if it hits in the same way individuals which differ in circumstances. These three approaches have a straightforward interpretation when the residuals of the model indeed capture “luck”. In practice, however, they will also capture the effects of specification errors and of omitted variables – some of which may be seen as legitimate, others as illegitimate sources of inequality. In other words, the residuals capture everything which is not observed by the social planner (or the analyst). This makes the interpretation more difficult. In section 4, we will follow the common approach, neglect the residuals and focus on expected values. In section 5 we explore some implications of the other two approaches to the interpretation of luck.

### 3 Explaining differences in mortality: data and model

We will impose the model in eqs. (3)-(5) on data for the Netherlands. In this section, we describe the data, discuss the empirical modeling strategy and the resulting estimates.

#### 3.1 Description of the data

We use data from a representative sample of non-institutionalized Dutch individuals taken from the health module of three cross-sectional surveys on living conditions (HSLC hereafter). The HSLC's contain information on lifestyles and on the variables  $x$  and  $\pi$ . They were linked by Statistics Netherlands to two administrative datasets: the national medical registry (NMR hereafter) and the cause-of-death registry (CD hereafter) which contain, respectively, all hospitalizations between 1998 and 2005, and all deaths between 1998 and 2007 in the Netherlands. These linkages provide us with survival information for each individual in the HSLC and with the opportunity to exploit exceptionally rich and objective diagnostic information linked to each hospital admission. As far as we know, such information has not been used before to analyze disease-specific impacts of lifestyles on mortality. Since the linkage between HSLC and NMR is only available from 1998 onwards and since we want to ensure a sufficiently long mortality follow-up, we use the HSLC's for 1998, 1999 and 2000.<sup>4</sup> We dropped individuals younger than 40 when surveyed by HSLC as they represent only about 5% of those who died by 2007.

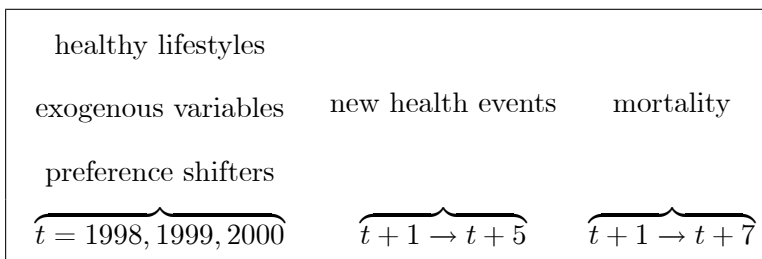


Figure 1: Timeline of HSLC and follow-up in NMR and CD

<sup>4</sup>In addition, a change to the HSLC questionnaire in 2001 renders some variables incomparable before and after this date.

We model lifestyles, health events and mortality in three stages in chronological order (see the timeline in Figure 1). Indicators of lifestyles, exogenous variables, and preference shifters (respectively, contained in vectors  $L_i$ ,  $x_i$  and  $\pi_i$ ) are taken from HSLC ( $t = 1998, 1999$  or  $2000$ ). Health events  $H_i$  are indicators of whether individuals were hospitalized due to the respective health event during the following 5 calendar years ( $t+1$  and  $t+5$ ) but not in  $t$ , and are modelled as functions of lifestyles and exogenous variables at time  $t$ . Finally, an indicator of whether the individual died between  $t+1$  and  $t+7$  is assumed to depend on lifestyles and exogenous variables at time  $t$  and health events from  $t+1$  to  $t+5$ . The timespan for health events and mortality was determined by data availability (as said above, respectively until 2005 and 2007) and, conditional on this, by our objective to ensure equal time at risk for all individuals (even if for individuals in HSLC 1998 and 1999 a longer follow up was possible). We have a total number of 12,484 observations. There seems to be sufficient variation in mortality, since about 11 percent of the individuals have died by 2005, 2006 or 2007 (see appendix).

We obtain indicators of the occurrence of *new* health problems from diagnostic information (ICD-9-CM codes) in the NMR, and refrain from using existing health events to minimize biases due to reverse causation. We selected those codes that are likely to correspond to a new health problem if the individual did not go to the hospital for the same code during the previous year. This excludes diagnoses for which individuals are usually first treated as outpatients or which relate to chronic conditions (for example, all mental problems were excluded as these diagnoses are normally related to chronic conditions and are usually preceded by outpatient visits). We considered six groups of new adverse health events: the incidence of i) cancer, ii) circulatory diseases, iii) stroke, iv) respiratory problems, v) digestive problems and vi) genitourinary problems.<sup>5</sup>

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<sup>5</sup>We are grateful to Isabelle Soerjomataram for her guidance in the identification of new health events from NMR data.

We consider three indicators of healthy lifestyles, whether individual: i) is a nonsmoker, ii) exercises (at least 1 hour per week) and iii) is not overweight (i.e., if  $BMI < 25$ ).<sup>6</sup> Since we have no information on diet but do control for exercise, the variable “overweight” is intended to proxy for diet. Our vector of exogenous variables  $x_i$  is composed of dummy variables representing age-sex categories, highest level of education achieved, house ownership, marital status and whether there are children in the household. The preference shifters in vector  $\pi_i$  are indicators of religion, region and urbanization (population density) of the area of residence. More information on all variables used, as well as descriptive statistics can be found in the appendix.

### 3.2 Specification of the model

In this section we explain how we implement empirically the conceptual model in eqs. (3)-(5). We specify a system of probit equations for mortality  $M_i$ , the new health events represented in vector  $H_i = [H_{CAi}, H_{CIi}, H_{Si}, H_{Ri}, H_{Di}, H_{Gi}]$  (cancer, circulatory diseases, stroke, respiratory, digestive and genitourinary disease) and indicators of whether the individual does not smoke, exercises and is not overweight,  $L_i = [L_{NSi}, L_{Ei}, L_{NW_i}]$ .<sup>7</sup> Denote by  $O^*$  the latent variables underlying each observed variable  $O$ ,  $O = M, H_{CA}, H_{CI}, H_S, H_R, H_D, H_G, L_{NS}, L_E, L_{NW}$ . The observed binary outcome  $O_i = 1$  if  $O_i^* > 0$  and  $O_i = 0$  otherwise.

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<sup>6</sup>While the relation between health onsets, mortality and BMI might be non-linear, we did not include an indicator for underweight as only a small proportion of the sample reports to be underweight. For similar reasons we exclude indicators of alcohol consumption.

<sup>7</sup>While more detailed information is available concerning survival between  $t$  and  $t + 7$ , the exact timing of the new health events, and frequency of smoking, exercise and BMI, we opted to restrict our attention to binary indicators. First, preliminary analysis of our data indicated that these thresholds are the most relevant for mortality and/or health events. Second, this choice makes the estimation of our econometric model more tractable (see also Balia and Jones, 2011).

We specify the following model for the latent variables:

$$M_i^* = \sum_{\substack{h=CA,CI, \\ S,R,D,G}} \beta^h H_{hi} + \sum_{l=NS,E,NW} \gamma^l L_{li} + x_i \delta^M + \varepsilon_i^M \quad (9)$$

$$H_{hi}^* = \sum_{l=NS,E,NW} \gamma^{hl} L_{li} + x_i \delta^h + \varepsilon_i^h, \quad h = CA, CI, S, R, D, G \quad (10)$$

$$L_{li}^* = x_i \delta^l + \pi_i \lambda^l + \varepsilon_i^l, \quad l = NS, E, NW \quad (11)$$

where  $x_i$  and  $\pi_i$  are as defined above and  $\beta^h, \gamma^l, \delta^M, \gamma^{hl}, \delta^h, \delta^l, \lambda^l$  (with  $l = NS, E, NW, h = CA, CI, S, R, D, G$ ) are (vectors of) coefficients to be estimated. The error terms are assumed to follow a multivariate normal distribution,  $(\varepsilon_i^M, \varepsilon_i^{CA}, \varepsilon_i^{CI}, \varepsilon_i^S, \varepsilon_i^R, \varepsilon_i^D, \varepsilon_i^G, \varepsilon_i^{NS}, \varepsilon_i^E, \varepsilon_i^{NW}) \sim MVN(0, \Sigma)$  where  $\Sigma$  is a  $10 \times 10$  symmetric matrix with all elements in the diagonal equal to 1 and off-diagonal elements equal to the correlations between the corresponding error terms.

As explained above, we observe mortality, health events and lifestyles at different periods in time. In particular, health events are observed one to five years after lifestyles (and exogenous variables), and mortality one to seven years after the same variables.<sup>8</sup> By accounting for timing in this way, we expect to remove one source of endogeneity, reverse causality, particularly from the relationship between health events and lifestyles. For example, if we measured exercise and the onset of a respiratory disease in the same year, then there could be reverse causality due to some individuals stopping or starting exercising as a consequence of being diagnosed with a respiratory disease. Our specification is able to test for further correlation across unobservables of different equations (for example, across health equations, due to comorbidities). If unobservables in the three stages of our model are correlated, then ignoring such correlations by estimating a series of univariate probit models would lead to inconsistent estimates of the observed effects.

Due to its recursive structure and non-linearity, our model would be identified even without exclusion restrictions. However, identification of effects of lifestyles on health events and mortality equations is aided through such exclusion restrictions. In particular, preference shifters  $\pi_i$  (religion, region and urbanization) only influence lifestyles and not health events (nor mor-

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<sup>8</sup>We do not include time subscripts as we have only one observation per individual.



tality).<sup>9</sup> We are not aware of other studies using religion, region and/or urbanization while jointly modeling lifestyles, health events, and/or mortality, but there is some indirect evidence supporting our exclusion restrictions. Religion has regularly featured as a source of exogenous variation to estimate the impact of lifestyles on economic outcomes (e.g., Auld, 2005), and also region has been used as an instrument for mortality (Balía and Jones 2008).<sup>10</sup>

### 3.3 Estimation results

We estimate the model by maximum simulated likelihood and correct standard errors for clustering at the year and municipality level.<sup>11</sup> The null hypothesis that all correlation coefficients equal zero is rejected ( $p < 0.0001$ ) implying that we cannot resort to a series of univariate probit models.

Let us first look at the estimated correlations between error terms across equations (Table 1). The estimated correlations between mortality/health equations and lifestyles and between mortality and health equations are generally small and insignificant, suggesting that relationships between stages of our model are fully explained by observables. The estimated correlations

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<sup>9</sup>Our data does not include variables that could be used as exclusion restrictions for the health events equation, i.e. no variable enters the lifestyle and health event equations, but not the mortality equation. More generally and beyond our data, it seems plausible that few variables might satisfy the necessary conditions in practice.

<sup>10</sup>While not a formal test of instrument validity, univariate probit models for the mortality equation (9) and the health event equations (10) with religion, region and urbanization as additional regressors showed no evidence of joint and/or individual significance. In addition, the Wald-test of joint significance in the lifestyle equations supports the strength of the instruments ( $p - value < 0.001$ ).

<sup>11</sup>Maximum simulated likelihood is consistent when the number of observations is large and when there is a sufficient number of draws. In practice, a reasonably small number has been found to result in well-behaved estimates. The estimates presented in this paper are based on five draws. Five draws lead to an optimization time of one week and increasing the number further would drastically increase optimization time of our 10-equation model (Hajivassiliou 1997). Therefore, we tested the sensitivity of our estimates to the number of draws by experimenting with sub-models of our 10-equation model which take considerably less time to optimize. We monitored the behavior of the estimates when increasing the number of draws from 5 to 250 and found that our main results remained qualitatively unchanged.

between health events are mostly positive, reflecting comorbidities.<sup>12</sup> Significant and sizeable correlations are found especially between the equations of cancer and other health equations. All correlations between healthy lifestyles are significant. Unobserved factors driving the likelihood of smoking correlate negatively with those related to overweight, while the correlations are positive between exercise on the one hand and not smoking and not being overweight on the other hand.

Table 1: Estimated correlation coefficients between equation of multivariate probit model

	Dead	Cancer	Circulat.	Stroke	Respir.	Digest.	Genito.	Nonsmok.	Exerc.
Cancer	0.035								
Circulatory	0.077	0.025							
Stroke	-0.014	0.082*	0.050						
Respiratory	0.076	0.164***	0.052	0.101*					
Digestive	-0.033	0.091*	0.053	0.011	-0.119*				
Genitourinary	0.068	-0.044	0.128*	0.009	0.078	0.118*			
Nonsmoker	0.012	0.032	-0.006	0.003	-0.018	0.034	0.011		
Exercise	0.000	0.009	0.010	-0.035	0.004	0.029	-0.028	0.151***	
Not overweight	0.044	0.020	-0.007	0.014	0.022	0.046	-0.016	-0.137***	0.069***

\*\*\*, \*\* and \* represent significance at 1%, 5% and 10%, respectively.

We now turn to the estimated effects of observed exogenous and endogenous variables. In order to assess their magnitude, we present average marginal effects on the probability that each of the outcomes equals 1. For each outcome, we obtain probabilities separately from the respective equations in the multivariate probit model. This corresponds to neglecting the correlations between error terms of mortality and each of the health events (lifestyles) and health events and lifestyles, which were shown above to be generally small and insignificant. Additionally, we obtain total effects of lifestyles on the probability of dying. These are obtained by: i) computing, for each individual, the marginal probabilities of having each of the health events, with and without adopting the respective healthy lifestyle, ii) replacing the actual occurrence of each health event by these probabilities, i.e., their expected values and iii) computing the resulting marginal probability of dying. Table 2 presents these average marginal effects, and the second column presents the total effects of lifestyles on the probability of dying.

<sup>12</sup> Among those with at least one new health event, only 12.4% experience two or more new health events.

Table 2: Estimated marginal effects derived from the multivariate probit model

Total effects of lifestyles		Effects in individual equations <sup>b</sup>									
	Dead	Dead	Cancer	Circulat.	Stroke	Respir.	Digest.	Genito.	Nonsmoker	Exercise	Not overweight
<b>Health events</b>											
Cancer		0.137***									
Circulatory		0.010									
Stroke		0.094***									
Respiratory		0.098*									
Digestive		0.074*									
Genito.		0.025									
<b>Lifestyles</b>											
Nonsmoker	-0.036	-0.031***	-0.007	-0.009	-0.016***	-0.003	-0.009***	0.003			
Exercise	-0.037	-0.034***	-0.008	-0.008	-0.006*	-0.004	-0.003	-0.002			
Not overweight	-0.009	-0.006	-0.008	-0.007	-0.005	-0.003	-0.008***	0.001			
<b>Age/gender<sup>a</sup></b>											
Male 40-50		0.000	-0.032***	0.010**	0.002***	-0.001	-0.002	-0.007***	-0.045***	-0.031**	-0.155***
Male 50-60		0.031***	-0.012*	0.033***	0.009	0.003	0.000	-0.006***	-0.003	-0.103***	-0.219***
Female 50-60		0.001	-0.006	0.007*	0.002	-0.002	-0.004	0.010***	0.095***	-0.007	-0.070***
Male 60-70		0.100***	0.039***	0.080***	0.032***	0.015***	0.000	0.003	0.100***	-0.100***	-0.163***
Female 60-70		0.036***	0.007	0.028***	0.011***	0.003	-0.001	0.019***	0.197***	-0.027	-0.135***
Male 70-80		0.273***	0.072***	0.107***	0.064***	0.025***	0.002	0.010**	0.132***	-0.157***	-0.077***
Female 70-80		0.138***	0.024***	0.052***	0.037***	0.015***	0.006	0.020***	0.293***	-0.110***	-0.104***
Male 80+		0.571***	0.016	0.044***	0.021***	0.050***	0.014	-0.002	0.231***	-0.299***	-0.051
Female 80+		0.400***	0.012	0.041***	0.064***	0.026***	0.008	0.006	0.358***	-0.261***	-0.071**
<b>Married</b>		-0.024***	0.002	0.001	0.001	-0.002	-0.001	-0.001	0.078***	0.043***	-0.039***
<b>Has children</b>		-0.029***	-0.012**	-0.001	-0.006	-0.004	-0.004	0.012***	0.018	-0.037***	0.035***

Table 2: (continued)

Effects in individual equations <sup>b</sup>										
	Dead	Cancer	Circulat.	Stroke	Respir.	Digest.	Genito.	Nonsmoker	Exercise	Not overweight
<b>House owner</b>	-0.019***	-0.005	-0.004	-0.001	-0.003	-0.002	-0.003*	0.075***	0.096***	0.051***
<b>Education<sup>a</sup></b>										
Lower voc.	-0.015**	-0.002	-0.002	-0.002	0.003	0.000	-0.001	0.048***	0.077***	0.021*
Lower gen.	-0.026**	0.004	-0.001	-0.002	0.003	0.002	0.004	0.057***	0.125***	0.090***
Higher sec.	-0.017**	0.002	-0.006	-0.001	-0.003	0.002	0.008***	0.072***	0.162***	0.086***
Higher educ.	-0.021**	-0.002	-0.014**	-0.004	0.003	-0.001	-0.002	0.132***	0.275***	0.162***
<b>Religion<sup>a</sup></b>										
Catholic								0.014	0.022*	-0.019
Dutch reformed								0.034***	-0.043***	-0.040***
Presbyterian								0.069***	-0.033*	0.033*
Muslim								0.115***	-0.190***	-0.198***
Other/none								0.069***	-0.062***	0.014
<b>Region<sup>a</sup></b>										
North								-0.033**	-0.036**	-0.047***
East								-0.025**	-0.012	-0.001
South								-0.032***	-0.015	0.001
<b>Urbanization<sup>a</sup></b>										
Very high								-0.047***	-0.004	0.039**
High								-0.027***	-0.004	0.012
Average								-0.023*	0.003	0.027*
Low								-0.013	0.009	0.020

<sup>a</sup>Reference categories described in the appendix.

<sup>b</sup>\*\*\*, \*\* and \* represent significance of the corresponding estimated coefficient in the multivariate probit model at 1%, 5% and 10%, respectively.

With respect to the mortality equation, we find a positive and significant impact of the health events upon mortality, with the exception of genitourinary and circulatory problems. The highest effect on mortality is that of cancer which increases the probability of dying within 7 years by about 14 percentage points, while digestive problems, stroke and respiratory problems increase that probability between 7 and 10 percentage points. Surprisingly, we find no effect of circulatory diseases. Conditional on the observed health events and exogenous variables, we still find a protective effect of healthy lifestyles for mortality. Not smoking or exercising decreases the probability of dying by about 3 percentage points, conditional on the health problems considered here, while the further decrease caused by not being overweight is small and insignificant. The other exogenous variables have a significant, but smaller protective effect on mortality, with the exception that elderly (and especially elderly males) have a much higher probability of dying.

The average marginal effects for the health event equations show that lifestyles influence mortality through prevention of observed health events, namely stroke (not smoking and exercising) and digestive problems (not being overweight and not smoking). Thus overweight affects mortality only via the onset of health events, while the other lifestyles also have a direct impact on mortality. We find no effect of lifestyles on the other health events which was unexpected for circulatory diseases and cancer.<sup>13</sup> Contrary to the mortality equation, we find that the exogenous variables are rather unimportant for the onset of new health events. We find a few significant effects, but the magnitudes of these effects are small. This means that these variables (for example homeownership) mainly matter for the new health events via lifestyles, but that there is hardly any direct effect. The only exception is age and gender which play a more prominent role, capturing disease-specific age-gender patterns.

Finally, the exogenous variables are more important in the lifestyle equations, showing for example a strong educational gradient in healthy lifestyles. Preference variables (religion, region

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<sup>13</sup>We tried to check whether there was a positive effect for lifestyles-related cancers (e.g. a relationship between smoking and lung cancer). Unfortunately, the number of observations with lifestyle-related cancers was too small to derive statistically sound conclusions.

and urbanization) also contribute significantly to the explanation of lifestyle differences.

### 3.4 Generalized residuals

For the treatment of “luck” we need to obtain residuals of our estimated multivariate probit model. In a linear probability model, the residual is easily computed as the difference between actual and predicted outcomes. In probit models, generalized residuals have been proposed that equal the expected value of the error term, conditional on the estimated parameters, the explanatory variables, and observed outcome (Gourieroux et al., 1987). We implement this in our multivariate probit model obtaining, for example for the lifestyle equations:  $gr_i^l = E(\varepsilon_i^l | L_{li} = 1; x_i, \pi_i) = \frac{\phi(x_i \hat{\delta}^l + \pi_i \hat{\lambda}^l)}{\Phi(x_i \hat{\delta}^l + \pi_i \hat{\lambda}^l)}$  and  $gr_i^l = E(\varepsilon_i^l | L_{li} = 0; x_i, \pi_i) = \frac{-\phi(x_i \hat{\delta}^l + \pi_i \hat{\lambda}^l)}{1 - \Phi(x_i \hat{\delta}^l + \pi_i \hat{\lambda}^l)}$ . In section 5, we will interpret these generalized residuals as a deterministic value – the “realization of luck” – in our comparison of different ways to handle luck in the measurement of inequality of opportunity. In the next section, we will neglect them and use simply the estimated expected value of mortality.

## 4 Inequity in mortality risks: the importance of the responsibility cut

In this section we focus on inequity in mortality *risks*. We will (i) show how different  $(c, e)$ -partitionings – reflecting different normative views – influence the measurement of inequity; (ii) discuss the results of a set of policy-relevant counterfactual simulations and iii) compare our results with those of the Roemer-approach, which does not allow for the different channels through which variables might influence mortality.

Using the complete recursive model, we simulate for each individual his/her predicted probability of dying, conditional on the actual values of all variables. Call this  $M_i^B$ . To measure “direct unfairness”, we simulate probabilities  $M^S(c_i, \tilde{e})$  conditional on actual values of circum-

stance variables and reference values of effort variables. The fairness gap is computed as

$$fg_i = M_i^B - M^S(\tilde{c}, e_i) \quad (12)$$

where  $M^S(\tilde{c}, e_i)$  is the simulated probability of dying conditional on actual values of effort variables and reference values of circumstances. All these calculations neglect the actual mortality experience of individuals and the estimated correlations between the error terms of the multivariate probit.

In order to calculate the different measures, we have to choose reference values  $\tilde{c}$  and  $\tilde{e}$ . For the fairness gap it makes sense to pick as reference values for  $\tilde{c}$  the characteristics of the type that can be assumed to be in the “best” situation, i.e. for which mortality is lowest conditional on the values of  $e$ . This fits in the interpretation of  $M(\tilde{c}, e_i)$  as an equitable reference point.<sup>14</sup> For consistency, we then make a similar choice for  $\tilde{e}$ . The reference values corresponding to the “best” situation are obtained as the categories for each of the exogenous variables  $x_i$  and the preference shifters  $\pi_i$  that have the lowest probability of dying, conditional on the remaining variables, as predicted from our multivariate probit model. Hence, we use as reference values: i) those living in a less densely populated area, ii) in the West of the Netherlands, iii) married individuals, iv) with children; v) homeowners; vi) those who have completed at least an undergraduate (non-) university degree, and vii) Presbyterians. Only for the age-gender categories, have we deviated from this procedure by choosing the youngest females as the reference category, rather than those between 50 and 60 which have a slightly lower estimated probability of dying.

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<sup>14</sup>It is also in line with the broader analysis in Fleurbaey and Schokkaert (2011). They introduce health care explicitly in the model and show that the choice of  $\tilde{c}$  is related to the position taken with respect to vertical equity. If different groups are treated differently by the health care system,  $\tilde{c}$  should refer to the type that receives the “best” health care.

#### 4.1 An overview of different normative choices

To structure our discussion, we write the quasi-reduced form (6) explicitly in terms of the variables that have been introduced in the previous section,

$$M_i = m [h(l(ed_i, ho_i, d_i, ag_i, reg_i, b_i), ed_i, ho_i, d_i, ag_i), l(ed_i, ho_i, d_i, ag_i, reg_i, b_i), ed_i, ho_i, d_i, ag_i] \quad (13)$$

where  $ed_i$  stands for education,  $ho_i$  for homeownership,  $ag_i$  for age and gender,  $d_i$  is the vector of other demographic variables (being married and having children),  $reg_i$  includes region and urbanization and  $b_i$  stands for religious beliefs. In the notation used earlier, the preference shifters are  $\pi_i = (reg_i, b_i)$ . Different normative perspectives can now easily be accommodated within this framework. Table 3 shows different partitionings of the variables into legitimate (effort) and illegitimate (circumstance) sources of inequality.



Table 3: Partitioning between legitimate and illegitimate variables (effort and circumstances) for different ethical positions

Ethical position	Model	Legitimate	Illegitimate
All Illegitimate (ALLILLEG)	Full		Age-gender, education, house ownership, married, children
Control (CONTROL)	Full	Married, children, religion, region, urbanization, house ownership	Age-gender, education
Preference (PREF)	No lifestyles equations	Lifestyles	Age-gender, education, house ownership, married, children for health events and mortality
Authentic preference (PREFA)	Full	Age-gender, house ownership, married, children for lifestyles	Education for lifestyles, health events and mortality; age-gender, house ownership, married, children for health events and mortality
Standardization (STAND)	Full	Age-gender	Education, married, children, religion, region, urbanization, house ownership
Standardization 2 (STANDC)	Full	Age-gender for health events and mortality	Education, married, children, religion, region, urbanization, house ownership; age-gender for lifestyles
SES Inequality (SES)	Full	Age-gender, marital status, children, religion, region, urbanization	Education, house ownership
Regional inequality (REG)	Full	Age-gender, education, married, children, religion, house ownership	Region, urbanization

Table 4 shows the corresponding results for inequity with the fairness gap, and Table 5 those with direct unfairness. We have also calculated confidence intervals for differences in inequity estimates within and between Tables 4 and 5 using bootstrap methods.<sup>15</sup> This statistical inference shows that differences in estimates discussed later in this section are relevant from a statistical point of view. As a measure of inequality we use the variance.<sup>16</sup> The evaluation of the actual situation is in the second column of both tables (“baseline”). The other columns refer to the results of counterfactual simulations and will be discussed in the next subsection. The last row in the tables gives the mean predicted probability of dying – this is the average of  $M_i^B$  (i.e. the first terms in the fairness gap).

Table 4: Inequity in mortality according to different ethical positions using the fairness gap

Ethical position	Baseline	Counterfactual situations			
		Educ1	Educ2	Exercise	Gender
ALLILLEG	0.0239	0.0183	0.0215	0.0167	0.0158
CONTROL	0.0229	0.0174	0.0206	0.0161	0.0149
PREF	0.0239	0.0182	0.0215	0.0168	0.0157
PREFA	0.0239	0.0182	0.0215	0.0167	0.0157
STAND	0.0061	0.0031	0.0047	0.0041	0.0047
STANDC	0.0066	0.0035	0.0052	0.0039	0.0050
SES	0.0020	0.0005	0.0012	0.0011	0.0015
REG	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
$\overline{M}_i^B$	0.1039	0.0850	0.0971	0.0814	0.0761

<sup>15</sup>Results available from the authors.

<sup>16</sup>Predicted probabilities of dying are bounded by 0 and 1. This boundedness has consequences for the choice of inequality measure. Erreygers (2009) has proposed the idea that inequality in attainments (here: predicted probability of dying) should be equal to inequality in shortfalls (here: predicted probability of surviving). This so-called “complementarity”-property was further explored by Lambert and Zheng (2011), who show that the variance is the only measure satisfying this requirement that is also subgroup decomposable.

Table 5: Inequity in mortality according to different ethical positions using direct unfairness

Ethical position	Baseline	Counterfactual situations			
		Educ1	Educ2	Exercise	Gender
ALLILLEG	0.0239	0.0183	0.0215	0.0167	0.0158
CONTROL	0.0102	0.0072	0.0089	0.0064	0.0058
PREF	0.0146	0.0121	0.0131	0.0146	0.0094
PREFA	0.0218	0.0158	0.0192	0.0170	0.0148
STAND	0.0002	0.0001	0.0001	0.0001	0.0002
STANDC	0.0002	0.0001	0.0001	0.0001	0.0002
SES	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
REG	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
$\overline{M}_i^B$	0.1039	0.0850	0.0971	0.0814	0.0761

A useful benchmark is that of “pure” inequality in mortality risks  $M_i^B$ , which can be interpreted as the case where all differences are considered to be illegitimate. In that case direct unfairness and the fairness gap coincide – see the first row in the tables.<sup>17</sup> As soon as we accept that individuals are held responsible for some variables, inequity (or inequality of opportunity) does no longer coincide with pure inequality. How to think about this individual responsibility? Two broad streams can be distinguished in the literature on responsibility-sensitive egalitarianism (Fleurbaey, 2008; and Fleurbaey and Schokkaert, 2011, for a discussion in the context of health).

The original philosophical inspiration of that literature (Rawls, 1971, Dworkin, 1981) was that persons as autonomous moral agents should assume responsibility for their goals and their conception of the good life, i.e. that they should be held responsible for their preferences. This

<sup>17</sup>Inequity as measured by the fairness gap and by direct unfairness will only be equal if we use a translation-invariant measure. This is related to defining the fairness gap as an absolute deviation, and is an additional reason for our choice of the variance.

“preference approach” was attacked by authors such as Arneson (1989), Cohen (1989), and Roemer (1998). They claimed that preferences are often the product of social influences, for which individuals cannot be held responsible, and they advocated the common-sense view that individuals should be held responsible only for what they have genuinely chosen, as opposed to what they have inherited from circumstances. This “control” (or choice) approach has dominated the empirical literature until now, perhaps because it indeed captures common sense. Yet, it is not so easy to implement as it may seem. Indeed, from a broader ethical perspective, “genuine control” requires correcting for interindividual differences in the environment and also for differences in the decision-making abilities of the individuals. But this brings us on a slippery slope: if, as scientists, we reason within a deterministic model, what room is left for control? Where do we have to stop in our quest for underlying causes that are not under the control of the individual? Does free choice really exist?<sup>18</sup>

Although these considerations may look very abstract, they have to be faced when operationalizing the control-approach. Indeed, for each of the variables in eq. (13), one has to decide if they are chosen or not. Age and gender certainly are not under individual control, but what about educational attainment? This is partly a matter of choice, partly a matter of innate (uncontrolled) capacities. For the purposes of this exercise we assume that educational attainment is not a matter of choice. All the other variables are seen as under the control of the individuals.<sup>19</sup> This means that the advantage measure for direct unfairness in the control

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<sup>18</sup>For economists working within the paradigm of rational choice, the problem of determining the boundaries of control is even more difficult. Indeed, in this paradigm individual decisions result from a mechanical optimization exercise with a given objective (preferences) and a given set of options (determined by the budget set and possibly additional constraints). All endogenous variables are causally influenced by other variables of the model and therefore cannot embody free will (Fleurbaey 2008, p. 87).

<sup>19</sup>We did some sensitivity analysis. Moving religion, region and urbanisation to the category of “non-controlled” variables hardly changes the results.

approach becomes

$$a_i^{CONTROL} = m \left[ h(l(ed_i, \widetilde{ho}, \widetilde{d}, ag_i, \widetilde{reg}, \widetilde{b}), ed_i, \widetilde{ho}, \widetilde{d}, ag_i), l(ed_i, \widetilde{ho}, \widetilde{d}, ag_i, \widetilde{reg}, \widetilde{b}), ed_i, \widetilde{ho}, \widetilde{d}, ag_i \right], \quad (14)$$

while the fairness gaps are given by

$$fg_i^{CONTROL} = M_i^B - m \left[ h(l(\widetilde{ed}, ho_i, d_i, \widetilde{ag}, reg_i, b), \widetilde{ed}, ho_i, d_i, \widetilde{ag}), l(\widetilde{ed}, ho_i, d_i, \widetilde{ag}, reg_i, b), \widetilde{ed}, ho_i, d_i, \widetilde{ag} \right] \quad (15)$$

The “preference approach” holds individuals responsible for their preferences, i.e., their conceptions of a good life, even if these preferences are not chosen and are not under their control. At first sight, this is easier to implement in our setting, as we can simply say that individuals are responsible for their lifestyle (but obviously not for the additional factors influencing health shocks or mortality). This therefore gives

$$a_i^{PREF} = m \left[ h(l(\widetilde{ed}, \widetilde{ho}, \widetilde{d}, \widetilde{ag}, \widetilde{reg}, \widetilde{b}), ed_i, ho_i, d_i, ag_i), l(\widetilde{ed}, \widetilde{ho}, \widetilde{d}, \widetilde{ag}, \widetilde{reg}, \widetilde{b}), ed_i, ho_i, d_i, ag_i \right] \quad (16)$$

$$fg_i^{PREF} = M_i^B - m \left[ h(l(ed_i, ho_i, d_i, ag_i, reg_i, b_i), \widetilde{ed}, \widetilde{ho}, \widetilde{d}, \widetilde{ag}), l(ed_i, ho_i, d_i, ag_i, reg_i, b_i), \widetilde{ed}, \widetilde{ho}, \widetilde{d}, \widetilde{ag} \right] \quad (17)$$

Holding individuals fully responsible for their lifestyle is perhaps a bit too easy, however. First, chosen lifestyles reflect both preferences and environmental factors (e.g. the budget constraint). A theoretically more coherent implementation of the “preference”-approach would therefore be to assume constrained utility maximization, specify a functional form for lifestyle preferences and identify its parameters through the estimation of the structural model. While our data did not allow this more ambitious approach<sup>20</sup>, it still seems worthwhile to correct to some extent for economic constraints on lifestyle choices. Educational attainment is a good proxy for these constraints. Second, the philosophical argument for holding individuals responsible for their preferences is that these reflect their *authentic* views of the good life. Involuntary

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<sup>20</sup>Moreover, specifying a functional form for preferences would necessarily require us to restrict the range of possible heterogeneity in individual preferences.

addictions and biased information should in this richer view be corrected for. Again, particularly interesting questions arise with respect to the effect of education – it has been argued by Cutler and Lleras-Muney (2010) that, correcting for income, the effect of education on lifestyles is mainly related to differences in cognitive abilities. Could we then not draw the conclusion that these do not reflect differences in genuine preferences? Here again, we arrive on a slippery slope: where does correcting revealed preferences lead to unacceptable paternalism? Yet, to illustrate the implications of both concerns (the economic and the philosophical one), we define a third ethical position (“authentic preferences”), where the effect of education on lifestyles is taken to be illegitimate. This obviously yields

$$a_i^{PREFA} = m \left[ h(l(ed_i, \widetilde{ho}, \widetilde{d}, \widetilde{ag}, \widetilde{reg}, \widetilde{b}), ed_i, ho_i, d_i, ag_i), l(ed_i, \widetilde{ho}, \widetilde{d}, \widetilde{ag}, \widetilde{reg}, \widetilde{b}), ed_i, ho_i, d_i, ag_i \right] \quad (18)$$

$$fg_i^{PREFA} = M_i^B - m \left[ h(l(\widetilde{ed}, ho_i, d_i, ag_i, reg_i, b_i), \widetilde{ed}, \widetilde{ho}, \widetilde{d}, \widetilde{ag}), l(\widetilde{ed}, ho_i, d_i, ag_i, reg_i, b_i), \widetilde{ed}, \widetilde{ho}, \widetilde{d}, \widetilde{ag} \right] \quad (19)$$

Note that we need our recursive model to implement the (“authentic”) preference approach, as we have to distinguish between the role of the variables in the different equations of the model.

The results for these different approaches are given in the second (“baseline”) column of Tables 4 and 5. In the case of direct unfairness, the differences are substantial. Note the much smaller value for control, and the substantial effect of purging preferences of the effect of education. The differences are much smaller for the fairness gap. Indeed, holding individuals responsible for variables under control or for lifestyles hardly decreases this inequity measure, compared to the case where all variables are illegitimate. The distinction between direct unfairness and the fairness gap turns out to be vital: “essential nonlinearities” are crucially important. We explained already in section 2 why we should indeed expect a larger value for inequity based on the fairness gap in the case where the marginal effect of the circumstance/effort variables depend positively on the value of the effort/circumstance variables, i.e. strengthen their respective effects on mortality.

The “control” and “preference” approaches have strong philosophical underpinnings. The economic literature, however, has until now focused on more pragmatic and partial approaches such as socioeconomic or regional inequalities (Lee 1991, Smith 1999, Wagstaff and van Doorslaer 2000, Cutler et al. 2006, Bengtsson and van Poppel, 2011). Moreover, standardization for age and gender is quite common. This follows from the idea that differences are only inequitable, if they are caused by institutions – and that inequalities linked to age and gender reflect irremediable biological differences (Wagstaff and van Doorslaer 2000).

Pure inequality *after standardization for age and gender* can almost be seen as a second benchmark of comparison. Direct unfairness then measures the inequality in

$$a_i^{STAND} = m [h(l(ed_i, ho_i, d_i, \widetilde{ag}, reg_i, b_i), ed_i, ho_i, d_i, \widetilde{ag}), l(ed_i, ho_i, d_i, \widetilde{ag}, reg_i, b_i), ed_i, ho_i, d_i, \widetilde{ag}], \quad (20)$$

while the fairness gaps are given by

$$fg_i^{STAND} = M_i^B - m [h(l(\widetilde{ed}, \widetilde{ho}, \widetilde{d}, ag_i, \widetilde{reg}, \widetilde{b}), \widetilde{ed}, \widetilde{ho}, \widetilde{d}, ag_i), l(\widetilde{ed}, \widetilde{ho}, \widetilde{d}, ag_i, \widetilde{reg}, \widetilde{b}), \widetilde{ed}, \widetilde{ho}, \widetilde{d}, ag_i] \quad (21)$$

The results in Tables 4 and 5 show that this standardization has a tremendous effect on measured inequity, which falls considerably – more so for direct unfairness than for the fairness gap, where it remains substantial (0.0061). Note, however, that the normative status of this standardization exercise is far from clear. Age and gender are not under the control of the individuals – and they may influence preferences. The reference to “irremedial” inequality versus that caused by “institutions” is not very convincing either: surely the effect of age and gender on health is not invariant over time and space and does depend on policy. Even if one fully accepts the logic of standardization for health variables, it is much less obvious to also standardize in the lifestyle equations.<sup>21</sup> Our model allows us to refine the approach and include the latter as illegitimate sources. This then gives

$$a_i^{STANDC} = m [h(l(ed_i, ho_i, d_i, ag_i), ed_i, ho_i, d_i, \widetilde{ag}), l(ed_i, ho_i, d_i, ag_i), ed_i, ho_i, d_i, \widetilde{ag}]$$

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<sup>21</sup>Lifestyle differences may be important for explaining gender differences in mortality - see, e.g., Pampel (2003).

and

$$fg_i^{STANDC} = M_i^B - m \left[ h(l(\tilde{ed}, \tilde{ho}, \tilde{d}, \tilde{ag}, \tilde{reg}, \tilde{b}), \tilde{ed}, \tilde{ho}, \tilde{d}, ag_i), l(\tilde{ed}, \tilde{ho}, \tilde{d}, \tilde{ag}, \tilde{reg}, \tilde{b}), \tilde{ed}, \tilde{ho}, \tilde{d}, ag_i) \right].$$

The results in Tables 4 and 5 show a slight change in inequity compared to the usual age-gender standardization.

The partial approaches that are prominent in the health economic literature accept that standardization is desirable and focus on the inequality due to socioeconomic status and region. It is easy to translate them in our framework. For the former we take  $c = (ed, ho)^{22}$ , for the latter we take  $c = reg$  (i.e. region and urbanization). The corresponding expressions for  $a_i$  ( $a_i^{SES}$  and  $a_i^{REG}$ ) and for  $fg_i$  ( $fg_i^{SES}$  and  $fg_i^{REG}$ ) can easily be derived following the same logic as before. The resulting measures of inequity turn out to be very small. The differences with the control-approach, which gives a much larger inequity-value, are particularly striking. This is not so surprising for region, as this only has significant effects in the lifestyle equations. It is less straightforward for socioeconomic status, however, as education plays an important role in the explanatory model. We come back to the effects of education in the next subsection.

The results in Tables 4 and 5 show that the decision to classify age and gender either as legitimate or illegitimate sources of inequality is crucial. This makes sense since age and gender are the most important determinants of mortality (consult the average marginal effects in Table 2). To see the importance of this effect, we re-evaluated the “preference” and “control” approaches with age as a legitimate variable.<sup>23</sup> We find that inequities become very small for direct unfairness. The inequities decline but remain much larger than those for socioeconomic inequality for the fairness gap which is in line with our earlier explanation of “essential nonlinearities” implying here that the marginal effect of education is stronger for older individuals. It is important to emphasize that these findings should *not* be interpreted as a weakness of

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<sup>22</sup>We treat homeownership as an indicator of wealth, and therefore consider it to be closely linked to socioeconomic status. This choice does not have an essential impact on our results, however. A sensitivity analysis with socio-economic status only measured by education yields very similar results.

<sup>23</sup>These results are available from the authors on request.



our approach. Quite the contrary, there are good reasons for the differential treatment of age and gender in the different normative approaches, and our findings simply point to the crucial importance of the decisions taken in this regard.

## 4.2 Simulating counterfactual situations

Additional insights into the interplay between the different causes of inequity can be obtained by considering the results of some counterfactual simulations. This will also illustrate how different normative perspectives impact on policy conclusions. We consider in that order the effects of educational policy, of changing lifestyles and of removing gender differences. Results are shown in columns 3 to 6 of Tables 4 and 5. Our discussion will focus on the equity effects, but it is obvious that for a complete evaluation of policy measures their efficiency should also be taken into account. An indication about this is given by the mean predicted probability of dying  $M_i^B$  in the counterfactual situations, as given in the last row of Tables 4 and 5.

### 4.2.1 Educational policy and inequity

Educational differences are one of the driving forces of inequity in health and educational policy is often seen as an important component of any attempt to improve the health situation of the population. We therefore simulate two policies. The first (educ1) consists in equalizing education for all at the highest level. This is not a realistic policy goal, but the results give us an idea on the upper bound of equity and efficiency that can be reached by educational policy. The second policy (educ2) is perhaps more realistic. It raises the educational attainment of the lowest educated group to the second lowest education level.<sup>24</sup>

Policy educ2 has only a small effect on the mean predicted probability of dying; educ1 is

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<sup>24</sup>Our recursive model addresses potential endogeneity in the relationship between health events and lifestyles, but assumes that education is an exogenous variable. This is sufficient to illustrate the usefulness of our approach to “inequality of opportunity” - that distinguishes between different channels - but might be insufficient to simulate counterfactual educational policies when education is an endogenous variable. The findings in this section should be interpreted with this limitation in mind.

(not surprisingly) more effective. The equity effects are substantial, both for direct unfairness and for the fairness gap – and this *whatever the ethical stance* that is taken. (Of course, one has to take into account that regional and socioeconomic inequity was already small in the actual situation and therefore cannot improve much in absolute terms). This result throws a new light on the results concerning socioeconomic inequality (where SES is operationalized by education and house ownership) in the previous subsection. It would be very misleading to conclude from the latter results that education is after all not so important from the point of view of equality of opportunity. Quite the contrary, the counterfactual simulations show that education is an important driving force of inequity in the face of death. The reason again has to be found in the “essential nonlinearities”: changing the education level influences the effects of other important variables, such as age and gender.

It is interesting to compare the results for PREF and for PREFA. Remember that in the partitioning PREF, lifestyle differences are considered to lead to legitimate inequalities, even if they are explained by educational differences. With the partitioning PREFA this is not the case. One would expect that equalizing education has a much stronger effect on inequity in the latter case. This is exactly what is found with direct unfairness. It is not true for the fairness gap, however. As mentioned before, this is because the fairness gap includes all indirect effects of circumstances in the measure of inequity.

### 4.2.2 Changing lifestyles<sup>25</sup>

Suppose now that society succeeds in letting all individuals exercise at least one hour a week. The results are given in column 5 ('exercise'). This change has a surprisingly large effect on average probabilities of dying, even larger than the ambitious educ1-policy in which all individuals are assigned the highest possible educational level.

The equity effects of this policy are interesting, because responsibility for lifestyles was one of the main factors differentiating the "preference" from the "control" approach. This has strong effects in the case of direct unfairness. If we hold people fully responsible for their level of exercise (in approach PREF) inequity is *not* affected by this policy – for obvious reasons. In the base situation direct unfairness is calculated as the variance in  $M^S(c_i, \tilde{e})$  - where  $\tilde{e} = e^*$ , the "best" possible lifestyle. The policy 'exercise' simulates the predicted probabilities for  $e_i = e^*$ . In the control approach, however, equalizing the level of exercise has a large effect on measured inequity, as we assume that many of the factors influencing the level of exercise are beyond individual control. Note, however, that there is also a strong effect in the case of PREFA: in this approach we do not hold individuals responsible for differences in exercise that are explained by differences in educational level. This leads to a larger perceived inequity in the base situation, but this difference between PREF and PREFA becomes smaller if exercise is equalized.

Changing lifestyles has also a strong effect in the fairness gap approach, but here the effects are surprisingly similar for the various ethical positions – for the reasons explained earlier, this is most surprising for PREF. Again, the explanation lies in the importance of the "essential nonlinearities".

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<sup>25</sup>We did several other lifestyle simulations, but do not show the results because they had similar (and somewhat smaller) efficiency and equity impacts. These simulations include situations: i) in which there are no smokers; ii) where overweight is eradicated; iii) where smoking behavior and overweight of the late 1980s would apply (which was obtained by running the same lifestyle equations on HSLC's for the late eighties - the full model cannot be estimated as the link with NMR and CD is only available since 1998). In addition, we have also simulated the case where regional differences and differences in urbanisation are removed. All results of these additional simulations are obtainable from the authors upon request.

### 4.2.3 Removing gender differences

Let us finally consider a simulation in which we remove the gender gap, i.e. we equalize the lifestyles, the occurrence of health shocks and the direct effect on mortality for men and women (column 6 – ‘gender’). This is not a realistic short run policy, but there are indications in the literature that the gender gap is recently becoming smaller – and that gender differences are (at least to some extent) influenced by social factors (Rogers et al., 2011, Quah, 2011). Surely the different treatment of men and women in society will have an impact on the gender gap. The counterfactual where the gender gap disappears may be illustrative for the potential importance of this effect, or, at least, gives an idea about its upper bound.

Of all policy simulations, removing gender differences has the strongest effect on the average probability of dying. It also has a strong effect on equity (both for the case of the fairness gap and direct unfairness) for all the ethical approaches that do not hold individuals “responsible” for their gender – i.e. for the “philosophically inspired” preference and control-approaches. As soon as one accepts the need for standardization, however, the effect on inequity is much smaller (see the results for STAND). Since we know that gender differences are not fully biologically determined, there seems to be a real issue here. The common practice of quasi-automatic demographic standardization may yield a biased picture of inequity.

## 4.3 The Roemer-approach

The previous sections have illustrated the advantages of working with the full model. It allows to differentiate the channels through which variables impact mortality. Moreover it can be used to gain additional insights by running counterfactual simulations. At the same time it requires a lot of information, however, which is not always available. It is therefore interesting to compare our approach with the more popular, pragmatic approach proposed by Roemer (1998). As described before, it consists in defining “types” as individuals with the same values for the circumstance variables and then focusing on the outcomes of these types. Effort variables are deliberately *not* included, since individuals are seen as exerting the same effort when they lie at the same rank

in the distribution of mortality for their circumstance type. The entire residual is assumed to capture effort.<sup>26</sup> All variables which are correlated with circumstances are interpreted as being part of circumstances, as they are not freely chosen by the individuals.

We compare this method with our control-approach in which  $c_i = (ag_i, ed_i)$ . In its most basic version (see e.g. Rosa Dias 2009), the Roemer-method is empirically implemented by regressing mortality upon the type characteristics, i.e. estimating  $M_i = \tilde{m}(ag_i, ed_i)$ , without including any other explanatory variables. This is a deliberately misspecified model. The “misspecification” is justified as a way to take up the effect of effort variables that are correlated with circumstances in the estimated coefficients of the circumstance variables. One then computes direct unfairness as inequality in the simulated values of  $\tilde{m}(ag_i, ed_i)$ , again omitting the stochastic part.

We have implemented this approach upon our data by estimating a single “reduced” probit equation for mortality with age-gender and education as the only explanatory variables. The misspecification of the model changes the coefficient estimates drastically (see average marginal effects in the appendix). This leads to a direct unfairness estimate of 0.0228, much larger than the estimate of 0.0102 for direct unfairness within the control approach based on our recursive model.<sup>27</sup> Given the results described before, it is not surprising that integrating the correlation between legitimate and illegitimate variables into the measure of inequity leads to a higher value for the inequity measure. In fact, the Roemer-approach gives a value which is very close to our results with the fairness gap. In this sense, it seems to be a useful approximation in the case where effort variables are not available.

The Roemer-approach raises some normative issues, however. By construction it picks up in the circumstances all correlations with effort variables. This makes it impossible to accommodate normative positions where this correlation does not necessarily lead to illegitimate inequity. The

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<sup>26</sup>This is not relevant for the comparison in this section, since we focus on (ex ante) mortality risks. We come back to the treatment of the residuals in the following section.

<sup>27</sup>We also compared the cases where home ownership is included (and age-gender excluded) in the set of illegitimate variables. The results confirmed the findings in the text.

most prominent example is the preference approach: obviously preferences can be correlated with circumstances (e.g. with socioeconomic status), but this would not mean that individuals should not be held responsible for them (or, formulated differently, one can still argue that preferences should be respected, whatever their origin). If one wants to implement such normative positions, in which some sources of correlation matter to define inequality of opportunity while others do not, the pragmatic solution of Roemer will not be sufficient and one cannot do without the estimation of a more refined model.

## 5 Luck and responsibility

Until now, we focused on the predicted probabilities of dying – and not on whether the individual actually dies or not. This essentially means that we made two normative choices. First, predictions of these probabilities are subject to error, since the model does not perfectly capture reality. We have ignored this in the evaluation of inequity. Second, we assumed that there is no inequity if predicted probabilities of dying are equal for all individuals with the same observed “effort”. Of course, in reality some will die and others will not. One could argue that not the ex ante probabilities but the final outcomes ex post should ultimately matter for social evaluation. This reflects the classical divide in social choice under uncertainty between the ex ante approach that applies a standard social welfare criterion to individual expected utilities and the ex post approach that looks at the possible final distributions of welfare (Fleurbaey, 2010).<sup>28</sup>

Neither of these options is obvious. The residuals partly take up the effect of omitted variables. In so far as these are considered to be “illegitimate” sources of inequality, neglecting them by focusing on predicted probabilities will tend to lead to an underestimation of overall inequity. Second, it is not obvious that “luck” can be neglected. In this respect, Dworkin (1981) drew the distinction between “option luck”, the kind of luck that can be avoided by prudent

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<sup>28</sup>We have refrained from using the ex ante-ex post terminology in this paper, as it is also used in the literature on inequality of opportunity with a different (but related) meaning, in which “ex post” basically refers to the compensation criterion introduced in section 2 (see, e.g., Fleurbaey and Peragine, 2009).

people if they take precautions, and “brute luck”, the luck that even reasonable and prudent people remain vulnerable to. In his view, individuals can be held responsible for their option luck, but not for their brute luck. Although this distinction is not beyond criticism (see, e.g., Fleurbaey and Schokkaert, 2011, for a brief sketch of the discussion) it is nevertheless a useful reference point for the discussion. While it might be possible to defend that the stochastic factors in the lifestyle equation reflect option luck, it is very hard to argue that there is no brute luck involved in health shocks (such as getting cancer or a stroke) or in unexpected death. Surely, people cannot be held responsible for this brute luck?

Let us try to gather some additional insights from our model. By way of introduction, note that the variance of predicted probabilities  $M_i^B$  is 0.0239 – while the variance of observed mortality is 0.0980. Not surprisingly, the residual is far from negligible. To proceed, we will refer to this unexplained part as “luck” – and subsume the effects of omitted variables and specification errors also under this denominator. This is a pragmatic stance. It can also be given some theoretical underpinning, however. In a deterministic view of the world, “pure” luck does not exist as all events can (ex post) be linked to some causal factors. Luck is then everything which could not be predicted (ex ante) on the basis of the available information. In this view of the world, therefore, the effects of unobserved and therefore omitted variables can be seen as luck.

We noted already that there are two possible approaches to incorporating luck in the measurement of inequality of opportunity. The first is to treat the residuals just like all other variables. In our empirical application we calculate for each individual observation and each equation of the multivariate probit the generalized residuals  $gr_i$  and we classify them either as circumstances or as effort. This classification will again depend on the normative position that is taken. We argued already that it is natural to interpret the stochastic factors in the lifestyle equations as option luck (and hence akin to effort for which individuals can be held responsible) but those in the mortality and health shock equations as brute luck (and therefore as part of circumstances). We will implement this assumption in our “control” and “preference” approaches.

In the STAND (standardization for age and sex) scenario we classify all the generalized residuals as circumstances. Of course, in the restricted ethical stances focusing on “socioeconomic status” or on “region”, all other factors, including luck, are seen as legitimate causes of inequality, i.e. as effort.<sup>29</sup>

We calculate inequity as direct unfairness and as the fairness gap in predicted outcomes for the different ethical approaches. This focus on predicted outcomes is natural as introducing luck (and hence removing uncertainty due to luck) should make one consider outcomes rather than probabilities. It is also consistent with our statistical model. The reference values for the generalized residuals are chosen to be equal to zero which corresponds to a situation with absence of luck.<sup>30</sup> The results are shown in Table 6.<sup>31</sup> They are not surprising, but still very striking. Somewhat provocatively stated, considering brute luck as circumstance or effort completely dominates the results and swamps the findings with respect to inequity in mortality risks.

Table 6: Inequity in mortality outcomes for various ethical positions, considering luck as circumstance

Ethical position	Direct unfairness	Fairness gap
CONTROL	0.0980	0.0980
PREF	0.0980	0.0980
STAND	0.0881	0.0980
SES	<0.0001	<0.0001
REG	<0.0001	<0.0001

<sup>29</sup>To avoid an overload of results, we only present results for the basic scenarios “preference” and “standardisation for age and sex” and not for the alternative versions “authentic preferences” and “alternative standardisation”.

<sup>30</sup>The predicted outcomes for direct unfairness and the fairness gap are obtained as follows: we predict that someone dies  $\widehat{M}_i = 1$  when the sum of the linear index and the generalised residual in eq. (9) is larger than zero, i.e.  $\sum \widehat{\beta}^h \widehat{H}_{hi} + \sum \widehat{\gamma}^l \widehat{L}_{li} + x_i \widehat{\delta}^M + \widehat{g}r_i^M > 0$ , and we predict  $\widehat{M}_i = 0$  otherwise. Note that in order to predict  $\widehat{M}_i$ , we also need to predict in a similar way (including the generalized residuals) whether someone experiences a new health event ( $\widehat{H}_{hi}$ ) and/or whether someone adopts a healthy lifestyle ( $\widehat{L}_{li}$ ) using eqs. (10)-(11). Obviously the subdivision of the explanatory variables and the generalized residuals in circumstance and effort is maintained while recursively predicting outcomes for both direct unfairness and the fairness gap.

<sup>31</sup>In case of predicted outcomes - which are binary variables - the variance delivers the same information as the mean. We stick to the variance for consistency with the earlier sections that are based on predicted probabilities.



An alternative approach to luck and equality of opportunity is proposed by Lefranc et al. (2009). They do *not* classify luck as either circumstances and effort, but put it in a third box. Loosely speaking, they claim that in an equitable situation luck should be “even-handed”, i.e. the distribution of luck should not differ significantly across groups of individuals with different values for the circumstance variables. Indeed, under this condition, the different types will be “hit” by brute luck to the same extent. In our empirical example, we assume that we do observe effort and hence luck; and therefore we can test directly the even-handedness of the generalized residuals for mortality.<sup>32</sup> We do this in two ways. First, we test whether the distribution of luck is similar across groups of individuals with identical circumstances, conditional on their effort. Second, we test whether the distribution of luck is similar across groups of individuals with identical circumstances without controlling for differences in effort. The first approach is the one advocated by Lefranc et al. (2009) and has the implied ethical value judgment that luck is allowed to be correlated with effort, insofar as luck remains uncorrelated to circumstances. The second approach requires that the distribution of luck is the same for different types, even if these types have different effort levels.

For the empirical application we focus on the control-approach but we simplify the age and the education variables so that we obtain eight types as the possible combinations of three binary variables: male/female, young/old (+ 60), high/low (less than secondary) education. We apply both tests of “even-handedness” to the generalized residuals of the mortality equation in order i) to keep the analysis as simple as possible and ii) to stick as close as possible to the concept of ‘brute luck’. For the first method we give the results for “all lifestyles healthy” and for “all lifestyles unhealthy”, for the second method we simply pool all the observations for each type. Non-parametric Kruskal-Wallis tests of equality of the distributions of generalized residuals between each pair of types decisively reject the null in all cases (results not shown,

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<sup>32</sup>Lefranc et al. (2009) also consider the specific (Roemer) case in which only circumstance variables are observed (and effort variables and luck are not) and they focus on that case in their empirical application. However, if one accepts that effort is observable – as we do – their suggestion that luck should be even-handed can be tested directly.

available from the authors). By way of illustration we show some kernel density plots in Figures 2 and 3: Figure 2 compares the results for old males with either high or low education (conditional on effort). Figure 3 shows the kernel density plots for old males and females with either high or low education.

The results of the tests are clear: luck is *not* distributed in an even-handed way over the types. Focusing on actual outcomes rather than on mortality risks therefore adds an additional dimension of inequity. For our specific empirical issue (inequity in the face of death) the treatment of the residuals is of essential importance.

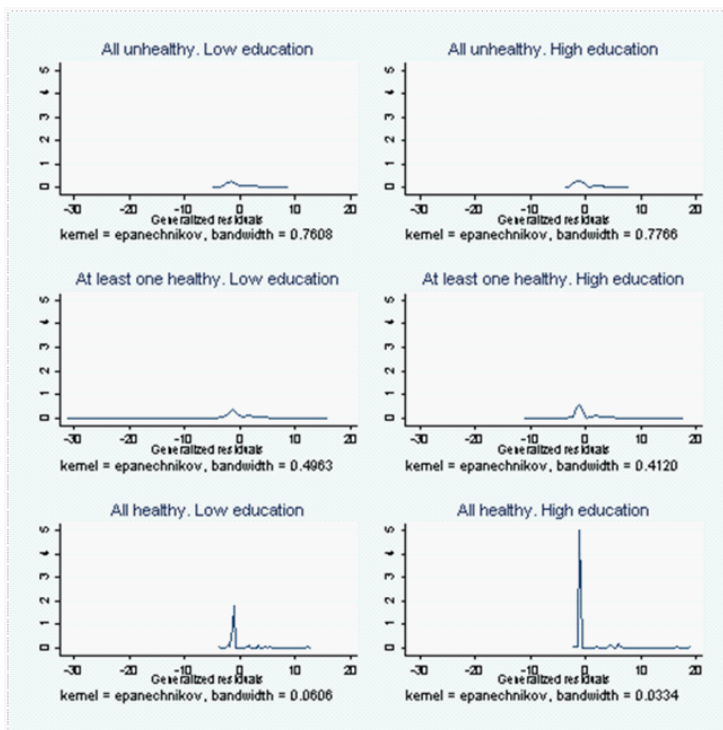


Figure 2: Distributions of luck for old males with low and high education (conditional on effort)

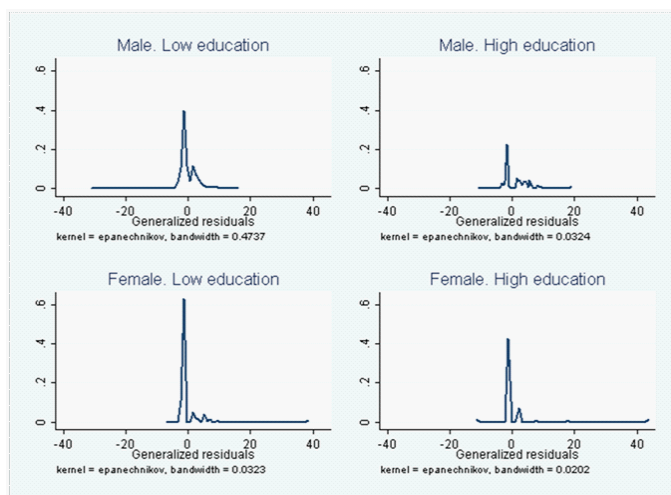


Figure 3: Distributions of luck for old males and females with low and high education

## 6 Conclusion

In this paper we focused on inequity in mortality. We estimated - on a rich dataset - a model that identifies different channels through which variables affect mortality. We have used this model to implement different approaches and measures from the theory of inequality of opportunity. Rather than summarizing the findings of our empirical work, we draw attention to some methodological issues that point to useful directions for future research and that go beyond the specific issue of inequity in mortality.

(a) The traditional measures of inequity that are most popular among economists (such as socioeconomic or regional inequalities) should not be interpreted as measuring a comprehensive notion of unfairness. They only capture a part of inequality in opportunity. The recent theories of equality of opportunity have introduced a formal framework which can be used to accommodate richer normative views. This makes it possible to link the empirical literature more closely to the cut between legitimate and illegitimate sources of inequity that has been suggested by different philosophical theories. The flexible nature of the recent economic approach of equality

of opportunity allows for meaningful sensitivity analyses to compare the implications of these different normative perspectives.

(b) The pragmatic approach proposed by Roemer (1998) is very useful in many cases, but is not sufficiently flexible to integrate relevant approaches such as, e.g., the preference approach to inequality of opportunity. If the available data are sufficiently rich, it is worthwhile to go beyond the estimation of a reduced form. The latter is needed to differentiate between different normative approaches, since the same reduced form variable may work through different channels that have different normative implications. Moreover, nonlinearities in the model may be essential. The difference between direct unfairness and the fairness gap is therefore of crucial importance.

(c) Counterfactual simulations are useful to get a better insight into the relative importance of different explanatory factors. Moreover, they show how the evaluation of the equity of policy measures does depend on the normative position that is taken.

(d) Care is needed in the treatment of unexplained variation. The common approach to simply disregard it may lead to misleading conclusions, unless one is willing to take the extreme normative position that ex ante risks are all that matters and that actual outcomes are no cause of additional social concern. In the face of death, brute luck seems to swamp all other factors, but it is to be expected that luck also plays an essential role in other contexts. More work is needed to better understand the implications of different approaches to luck, e.g. concerning the question whether luck is just a variable as all the others (and should be classified as either circumstance or effort) or whether, on the contrary, it should be put in a different box. In empirical applications, at the very least, a sensitivity analysis with respect to the different approaches is useful – or even necessary.

(e) The usual practice of standardizing for age and gender in health economic applications should be reconsidered. It has a tremendous effect on measured inequity. In so far as demographic (mainly gender) differences are codetermined by social and behavioral factors and are not only linked to biological differences, they should be considered explicitly in any analysis of

inequality of opportunity.

(f) It should be the ambition to collect better data for estimating richer structural models.

Our recursive model is a first step in the direction of a full structural model, but when one accepts the preference approach to inequality of opportunity, the aim should be to identify the preference parameters from a full-fledged model of utility maximization.

## **Acknowledgements**

Teresa Bago d’Uva, Pilar García-Gómez, and Tom Van Ourti are supported by the NETSPAR project “Health and income, work and care across the life cycle II”. Pilar García-Gómez also acknowledges support from a Marie Curie fellowship, and Tom Van Ourti from the National Institute on Ageing, under grant R01AG03739801. Teresa Bago d’Uva and Pilar García-Gómez are Postdoctoral Fellows of the Netherlands Organisation for Scientific Research – Innovational Research Incentives Scheme – Veni. We thank the Netherlands Central Bureau of Statistics for access to the linked data sets used for this research (POLS Gezondheid 1998-2000, LMR 1998-2005, DO 1999-2007), and Isabelle Soerjomataram for valuable help with identifying new health events from NMR data. Further, we thank André Decoster, Nicolas Hérault, Guyonne Kalb, Marion Leturcq, Owen O’Donnell, Hans van Kippersluis, Eddy van Doorslaer and seminar participants at the University of Oxford and Barcelona, and conference participants at the 7th iHEA World Congress in Beijing, the 8th iHEA World Congress in Toronto, the XXX Jornadas AES, the Equity in Health workshop at CORE (UCL) and the workshop on Equality of Opportunity in Ghent for useful comments. The usual caveats apply and all remaining errors are our responsibility.

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# Appendix

## Description of the data

Table 7: Variable description

Variable	Mean
<b>Endogenous variables</b>	
<b>Died between t+1 and t+7</b>	0.110
<b>Health problems diagnosed between t+1 and t+5</b>	
Cancer	0.055
Circulatory disease	0.044
Stroke	0.023
Respiratory disease	0.013
Digestive disease	0.011
Genitourinary disease	0.010
<b>Healthy lifestyles</b>	
Not overweight (BMI<25)	0.483
Nonsmoker currently	0.705
Exercise (more than 1 hour a week)	0.456
<b>Exogenous variables</b>	
<b>Age-gender</b>	
Male between 40 and 50	0.160
Female between 40 and 50 (reference category)	0.179
Male between 50 and 60	0.137
Female between 50 and 60	0.142
Male between 60 and 70	0.098
Female between 60 and 70	0.099
Male between 70 and 80	0.063
Female between 70 and 80	0.072
Male 80+	0.019
Female 80+	0.030
<b>Married</b>	0.751
<b>Has children</b>	0.251
<b>Highest degree of education attained</b>	
Primary education (reference category)	0.256
Lower vocational education	0.196
Lower general or scientific secondary education	0.090
Higher vocational, general or scientific secondary education	0.271
Higher vocational education or a university degree	0.188
<b>House owner</b>	0.631

Table 7: (continued)

<b>Variable</b>	<b>Mean</b>
<b>Preference shifters</b>	
<b>Religion</b>	
Catholic	0.359
Protestant - Dutch reformed	0.174
Protestant - Presbyterian	0.074
Other protestant (reference category)	0.327
Muslim	0.009
Other religion, or not religious	0.057
<b>Region</b>	
North	0.120
East	0.231
West (reference category)	0.404
South	0.245
<b>Urbanization</b>	
Very low population density (reference category)	0.178
Low population density	0.240
Average population density	0.221
High population density	0.242
Very high population density	0.120

The 6 groups of adverse health events were obtained as follows. First, we aggregated all diagnoses that could indicate new adverse health events in the following groups: i) infectious (infectious and parasitic diseases – some codes within 001-139); ii) cancer (neoplasms – some codes within 140-239); iii) endocrine (endocrine, nutritional and metabolic diseases and immunity disorders – some codes within 240-279); iv) nervous (diseases of the nervous system and sense organs – some codes within 320-389); v) circulatory (some codes within 390-422 within diseases of the circulatory system); vi) stroke (some codes within 430-459 within diseases of the circulatory system); vii) respiratory (diseases of the respiratory system – some codes within 460-519); viii) digestive (diseases of the digestive system – some codes within 520-579); ix) genitourinary (diseases of the genitourinary system – some codes within 580-629); x) skin (diseases of the skin and subcutaneous tissue – some codes within 680-709); xi) musculoskeletal (diseases of the musculoskeletal system and connective tissue – some codes within 710-739); xii) injury

(injury and poisoning – some codes within 800-999). Second, we estimated a univariate mortality model and excluded those groups showing no evidence of influence, i.e. groups iii), iv), x) and xii). Third, we removed all groups with an incidence below 1%, i.e. groups i) and xi) to avoid too small cell sizes. More information can be obtained from the authors upon request.

### Some results Roemer model

Table 8: Marginal effects in the "reduced" Roemer model

<b>Inequity</b>	0.023
<b>Marginal effects</b>	
Male between 40 and 50	0.000
Male between 50 and 60	0.039
Female between 50 and 60	0.006
Male between 60 and 70	0.130
Female between 60 and 70	0.047
Male between 70 and 80	0.346
Female between 70 and 80	0.181
Male 80+	0.499
Female 80+	0.653
Lower vocational	-0.025
Low general	-0.035
Higher secondary	-0.033
Higher education	-0.048