



# Defining and measuring health poverty

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## ABSTRACT

Unlike other aspects of welfare (e.g. income), health has been relatively neglected when it comes to defining and measuring aspects of poverty. The aim of the paper is twofold: first we elaborate how the concept of 'health poverty' can be defined and measured, and second we apply the methodology to study health poverty in a variety of cases. The measurement of health poverty allows us to gain insights into different sorts of health deprivation in society as a whole, and in specific subgroups. We measure poverty by means of the widely adopted Foster-Greer-Thorbecke (FGT) class of indicators and apply this to three different health variables: cardiovascular risk, health status and life expectancy. Moreover, the FGT class is additively decomposable, making it possible to gauge the contribution of specific subgroups to overall poverty. We provide two applications of these methods. Firstly, we examine changes in the risk of cardiovascular disease (CVD) in the United States using two waves of the NHANES survey from 2005-06 and 2013-14 ( $n = 3,014$  and  $4,001$  respectively) and use a threshold of 20% 10 year CVD risk to define health poverty. Overall our results indicate a slight decline in the proportion at high CVD risk between these periods. Secondly, we apply poverty measures to health status as measured by the SF-6D index and to empirically derived predictions of life expectancy and estimated using 24,820 individuals from the first 15 waves of the Australian HILDA survey. Trends in poverty over time are compared using several thresholds and decomposed by a variety of sub-groups. Measures of health poverty can be an important instrument for focusing the attention on those with the worst health, or highest risk, in a society and should be used more widely.

## 1. Introduction

The aim of the paper is twofold: first we elaborate how the concept of 'health poverty' can be defined and measured, and second we apply the methodology to three different cases. Although not entirely new, the notion of health poverty is seldom used – in contrast to the notion of income poverty. For instance, in a recent survey on poverty and inequality it is mentioned only once (Rohwerder, 2016: 3). It does have its place in the literature on multidimensional poverty (e.g., Ravallion, 2011; Weziak-Bialowolska, 2016). While we acknowledge the importance of a comprehensive understanding of deprivation and of the appropriate measurement of the health component of deprivation (e.g., Mitchell et al., 2015), we believe that focusing on health poverty as such is useful and relevant, especially for public health policy. This resonates with a large body of work on the health disadvantages faced by specific groups, such as indigenous people, in comparison to the rest of society. For example, in Australia the substantial lower life expectancy of Indigenous Australians – at least ten years less than that of

the non-Indigenous population – has been a key measure of this group's disadvantage in comparison to the rest of society (Close the Gap Campaign Steering Committee, 2017). The measurement of health poverty obliges us to reflect on what we consider to be reasonable minimum standards of health and on how we define and measure deprivation in the domain of health.

The common approach in medicine is to define minimum standards of health by defining critical threshold values. With regard to obesity, for instance, there is a fair amount of agreement on classifications of BMI values (e.g. obesity is classified as BMI 30 units or higher), and it is therefore not surprising that specific poverty measures have been employed recently by Madden (2017) and by Bilger et al. (2017) to measure obesity poverty. However, there has been little recognition that for a wide variety of clinical measures thresholds are often used in guidelines to target interventions or therapies (e.g. strategies to prevent cardiovascular disease are based on risk thresholds, see Jackson et al., 2005). The application of more general health poverty measures provides a way of monitoring what proportion of a population falls above

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or below these thresholds and should be of direct relevance to clinicians and health policy makers.

When it comes to assessing shortfalls in overall health, a major complication is that health, in comparison to income, is multifaceted and therefore much harder to measure accurately. Perhaps this has been the main reason why there are relatively few studies on health poverty. If one is to develop new measures of health poverty, the first choice to be made is that of the health variable which will be taken into consideration. The nature of the health variable affects the way health poverty should be measured, as it does in the case of (socioeconomic) inequality of health (Erreygers and Van Ourti, 2011). Sometimes health is measured by means of a categorical or qualitative variable. A good example is provided by self-assessed health: in many household surveys people are asked to assess their health status by selecting one state from a fixed list of possible health states. This sort of qualitative information entails the use of ordinal poverty measures (Allison and Foster, 2004; Madden, 2015), such as the class of ordinal poverty indicators proposed by Bennett and Hatzimasoura (2011). These measures have already been applied by Brzezinski (2015) to estimate the trends in health poverty in Britain over the period 1991–2008, and by Pascual et al. (2017) to do the same for Spain in the period 2008–2016. While considerable efforts have been made by health economists and health outcomes researchers to map different aspects of health status into a single measure such as Quality Adjusted Life Year (QALY), we can find few attempts (an exception being Simões et al., 2016) to develop a concept of health poverty for these measures (i.e. a level of health that would be considered unacceptably low).

Our approach is based on the assumption that the health variable has ratio-scale properties. In comparison to the case of an ordinal health variable, the advantage is that we can attach meaning to the distance of everyone's health achievement from a given threshold level, which we call the 'health gap' for those who do not reach the threshold. In addition, we allow for the possibility that the relevant threshold levels may differ from one individual to the other, e.g. according to whether a person is young or old. We then measure health poverty by means of the now widely adopted Foster-Greer-Thorbecke (FGT) class of poverty indicators (Foster et al., 1984). By assuming different values of the poverty aversion parameter, we are able to assess three aspects of poverty (incidence, intensity and inequality, known as the three I's of poverty measurement).

We begin by a more detailed explanation of our health poverty measurement procedure (section 2). We apply our methodology to three different situations, involving the risk of contracting cardiovascular diseases (CVD), preference-based measures of health status, and life expectancy. To assess CVD-related health poverty we use American data (section 3). For the two other types of health poverty we use Australian data (section 4). We end with a few concluding remarks and propose an agenda for future research (section 5).

## 2. The measurement of health poverty

In this section we explain the mechanics of health poverty measurement, assuming that health is a variable with ratio-scale properties. The guiding idea is to consider as health poverty the condition of being in 'poor health', i.e. of having a health status worse than what is considered to be minimally acceptable. There are basically two ways of defining health poverty standards: either we adopt a uniform threshold, or we allow for variation, say different thresholds for men and women. The second approach relies on the assumption that what constitutes the level of minimally acceptable health may depend on the average health achievement of an individual's specific reference group.

Our health poverty measures are based on the normalized health poverty gaps of individuals. The health poverty gap is basically the distance of an individual's health attainment  $h_i$  from the relevant health poverty threshold; it is 'normalized' if it is expressed as a fraction of the threshold. In the case of a uniform health poverty standard  $z$ , the

normalized health gap  $g_i$  is defined as:

$$g_i \equiv \begin{cases} \frac{z - h_i}{z} & \text{if } h_i < z \\ 0 & \text{if } h_i \geq z \end{cases} \quad (1)$$

In the case of differentiated health poverty standards, the gap must be defined with respect to an individual's specific health poverty standard. More formally, suppose there are  $n$  individuals in society, designated by the set  $N = \{1, 2, \dots, n\}$ , and let  $J \subseteq N$  be a given reference group consisting of  $n_J$  individuals. Assume every individual belongs to one and only one reference group, and let the health poverty threshold for reference group  $J$  be  $z_J$ . Then for any individual  $i \in J$  we can define the normalized health gap as:

$$g_i \equiv \begin{cases} \frac{z_J - h_i}{z_J} & \text{if } h_i < z_J \\ 0 & \text{if } h_i \geq z_J \end{cases} \quad (2)$$

How many reference groups there should be, is open to debate. For instance, in health inequality measurement it is usual to standardize for characteristics such as sex and age. The degree to which we should standardize for other characteristics depends on whether these are seen as legitimate or fair grounds for distinction (see Fleurbaey and Schokkaert, 2009, for further discussion). Similarly, there is more than one way to define the uniform or group-specific health poverty thresholds.

Once the threshold values have been determined, we can say that all those whose health achievement falls below the relevant threshold value are health poor, and that the extent of their health poverty is captured by their health gaps. Our aggregate health poverty measures are defined as functions of the normalized health gaps  $g_i$ . More specifically, the FGT index measures health poverty as follows:

$$P_\alpha = \frac{1}{n} \sum_{i \in Q} (g_i)^\alpha \quad (3)$$

where  $\alpha$  is a non-negative poverty aversion parameter and  $Q$  is the subset of the poor (i.e., those for which  $g_i > 0$ ). We will consider different values of this parameter, viz.  $\alpha = 0, 1, 2$ . In the literature  $P_0$  is known as the headcount ratio,  $P_1$  as the poverty gap ratio and  $P_2$  as the squared gap measure (Foster et al., 2010). These capture the incidence, intensity and inequality of health poverty.

One of the advantages of using the FGT index to measure health poverty is its flexibility with regard to decomposition. Thanks to its simple additive structure, the health poverty index (3) is subgroup decomposable. Let the population be partitioned into  $K$  subgroups with sizes  $n_1, n_2, \dots, n_K$ ; these groups may, but need not, coincide with the reference groups. By analogy with (3), we define the poverty level in subgroup  $J$  by:

$$P_{\alpha,J} = \frac{1}{n_J} \sum_{i \in Q_J} (g_i)^\alpha \quad (4)$$

where  $Q_J$  is the subset of the poor in group  $J$ . It is straightforward to see that the level of health poverty in society can be expressed as a weighted average of the levels of health poverty in the  $K$  subgroups, with the weights equal to the population shares  $s_1, s_2, \dots, s_K$ , where  $s_J \equiv n_J/n$ :

$$P_\alpha = \sum_{J=1}^K s_J P_{\alpha,J} \quad (5)$$

We use these decomposition formulas to illustrate the difference in contributions to health between different sub-groups, such as men and women and smokers and non-smokers.

From a policy perspective, it may be important to know whether health poverty occurs more among the (income) poor than among the (income) rich. Subgroup decompositions based on income criteria may provide useful information on the relationship between health poverty



and income. One way to link health poverty to the more traditional analysis of socioeconomic inequalities consists of measuring to what extent health poverty is correlated with income. Bivariate indicators of inequality, such as the Concentration index, allow us to estimate this type of correlation (with regard to obesity, this has been done by Bilger et al., 2017). The health poverty dimension is taken into account by treating the normalized health gaps, or more precisely the normalized health gaps raised to the power of  $\alpha$ , as individual measures of health poverty. As far as income is concerned, we can look both at income ranks and at income levels. While the first choice characterizes rank-dependent indicators, of which the Concentration index is the most prominent example, the second choice leads to level-dependent indicators (Erreygers and Kessels, 2017; Erreygers et al., 2018). Since the normalized health gaps are bounded variables, we have to use the bounded versions of these income-related indicators of inequality. If health poverty occurs more frequently among those with low incomes than among those with high incomes, the values of these indices are negative. Larger absolute values of the indicators reflect higher levels of inequality.

It should be observed that these indicators are similar, but not identical, to the more usual income-related inequality indicators measuring the association between a health variable  $h$  and the income ranks or the income levels. The normalized health poverty gaps  $g^\alpha$  are, in fact, truncated variables: for all individuals who are not health poor, the value of the health poverty gap is equal to zero. Moreover, health gaps are like ill-health variables: larger values indicate worse outcomes.

Estimates of uncertainty such as confidence intervals can be obtained through bootstrapping methods. In the examples which follow we report 95% confidence intervals based on the percentile method using a 1000 replications of the data.

### 3. CVD-related health poverty

The modern approach to the prevention of cardiovascular disease involves assessing a patient's absolute risk of a cardiovascular event occurring over a defined period (e.g., 10 years). For example, the 2013 American Heart Association guidelines for the use of the cholesterol medications statins recommend an algorithm to determine whether a patient needs to be treated with statins, a cholesterol lowering treatment. Traditionally this determination has been based on the level of cholesterol (Stone et al., 2014), but the 2013 guidelines moved to an algorithm that involved the assessment of absolute cardiovascular risk.

Cardiovascular disease risk assessment involves the use of risk equations that provide a probability that a patient will have a CVD event (e.g., a heart attack or stroke) over a defined period. The most widely used way of calculating risk is commonly known as the *Framingham Coronary Heart Disease Risk Score* which is based on data from a long-term cohort of people living in the town of Framingham, MA (USA). The Framingham study involved the collection of clinical measures and cardiovascular outcomes since the late 1950s (Syed et al., 2014). As part of this study risk prediction equations have been published that calculate the probability of a CVD event over a 10-year period based on patients' age, sex and commonly collected clinical data including systolic blood pressure, the ratio of HDL to total cholesterol, diabetes, smoking and blood pressure treatment status (D'Agostino et al., 2008).

The guidelines for the use of preventative therapies such as statins are based on thresholds that have progressively become lower over time. For simplicity, we employ poverty measures to quantify changes in only one threshold (i.e. 10 year risk of 20%) (World Health Organization, 2007), but the analysis presented could easily be extended to other thresholds.

One reason for developing measures of health poverty is that they can be used to both monitor and evaluate policies to reduce CVD. In particular there is evidence from numerous clinical trials that statins are effective in reducing the risk of CVD. A meta-analysis of 22 trials of

statin versus control shows on average a 21% reduction in the risk of major vascular events for each 1.0 mmol/L reduction in LDL cholesterol (Cholesterol Treatment Trialists' (CTT) Collaboration, 2012). In the following illustrative example, we assume statins have this treatment effect and moreover that they have the same relative effect on all individuals regardless of their absolute risk of CVD.

To explore how poverty measures can be used to evaluate policies regarding treatment with statins we use data from two waves of the United States National Health and Nutrition Examination Survey (NHANES): 2005–2006 and 2013–2014. NHANES is a publicly available dataset approved by the National Center for Health Statistics institutional review board, and all participants provide written informed consent. The NHANES study (see <https://wwwn.cdc.gov/nchs/nhanes/> for more information) is a continuous, annual survey of the non-institutionalized civilian resident population of the United States by the Centers for Disease Control and Prevention (CDC). The data contain results of clinical examinations (including the taking of blood samples) as well as reported information on medication use and other characteristics, such as age, sex and income.

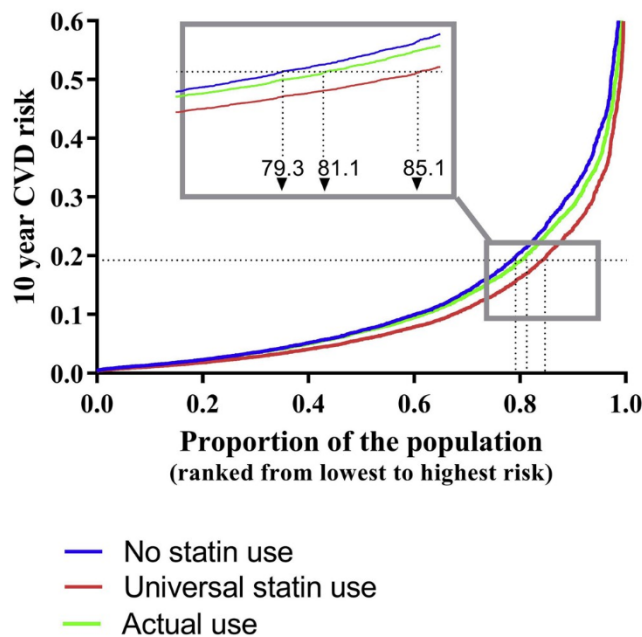
In order to apply poverty measures we need to transform our measure of health so that higher values reflect better health. In the case of CVD risk this can be easily achieved by quantifying the proportion of the population deemed not to be at risk (which we term 'resilience'). More formally, if the risk of a disease such as CVD occurring for individual  $i$  is equal to  $r_i$ , a scalar which varies between 0 and 1, the resilience of individual  $i$  is defined as  $1 - r_i$ . We take resilience as our health variable ( $h_i = 1 - r_i$ ).

For this health variable we assume there are no grounds to treat individuals differently, and therefore we adopt a uniform threshold level  $z$ . If we define the critical CVD risk level as equal to 20%, which historically was a level where treatment is initiated (Lloyd-Jones, 2010), then the corresponding CVD resilience poverty threshold is equal to  $z = 80\%$ . Such a threshold is clinically meaningful as the prescribing of therapies to prevent cardiovascular disease is now based on absolute risk thresholds (Jackson et al., 2005). Since we have a uniform threshold, the normalized CVD resilience gaps of individuals are defined according to formula (1).

Table A1 in the Appendix (which is in the supplementary material) presents descriptive information for the two waves of the NHANES survey considered in this paper. We have restricted the age range to between 30 and 75 years (which is the range used in many clinical guidelines for cholesterol lowering). Between the two surveys the proportion of the sample population using cholesterol lowering medications has increased by 4% ( $P < 0.0001$ ), indicating expanded use of drugs to prevent cardiovascular disease in the United States over the period.

In what follows we have calculated the absolute 10-year risk of a CVD event based on the 2008 Framingham equation estimated by means of the risk factors as measured in the NHANES survey. We denote this data series as the actual risk associated with *current statin use*. The NHANES survey also collects information on whether each respondent is taking cholesterol medications. We use this information to calculate estimated CVD risk under two additional illustrative scenarios and follow an approach which has previously been used to examine the effects of statin use at population level (see, Ueda et al., 2018). The first scenario, which we call *no use of statin medications*, involves adjusting upward the risk of those individuals currently taking statin medications by a factor of 1.26. This is equal to the ratio of 1 to 0.79, where 0.79 reflects the 21% risk reduction conferred by statins. The adjusted risk is an estimate of the risk individuals currently taking statin medications would face if they were not taking any cholesterol medications. The second scenario is *universal use of statin medications* and involves adjusting downward the risk of all individuals currently not taking statins by a factor of 0.79. This is again based on the assumption that the use of statins reduces risk by 21%.

As explained previously, our health poverty threshold is based on a



\*Notes: Based on risk calculated using the Framingham population using data from the public NHANES 2005–2006 data set.

Fig. 1. 10 year cardiovascular risk of US population (aged 30–75 years)\*.

10-year CVD risk level of 20%. We measure the proportion of the population that would be recommended for treatment with statins under various treatment policies. Fig. 1 illustrates the absolute risk in 2005–2006 under the three scenarios outlined above. We rank the population from lowest to highest risk, similar to Pen's 'Parade of Dwarfs' often used to represent income inequality (Pen, 1971: 48–59). Under the *no use of statin medications* scenario (represented by the blue line) 79.3% of the population would be below (and 20.7% above) the critical CVD risk threshold. The actual use of statins in 2005–2006 (represented by the green line) resulted in a rightward shift in the line representing the distribution of risk. This increases the proportion below the threshold (i.e. 81.1% of the population has a 10-year CVD risk below 20%). Hence the use of statins in 2005–06 has resulted in around 1.8% of the US population moving below the 20% risk threshold. The final scenario, *universal use of statin medications* (represented by the red line), illustrates the hypothetical maximum gain from universal treatment. This would further increase the proportion of the population below the threshold (i.e. around 85.1% of people would have a risk below 20%, or an additional 4% of the population). What this scenario quantifies is the excess risk above 20% that could potentially be removed by statin treatments.

Fig. 2 compares the CVD risk associated with the actual use of statins at two points in time, 2005–2006 and 2013–2014. Fig. 2 illustrates the outward shift of the CVD risk curve between 2005–2006 and 2013–2014. As a result of the changes, an additional 1.8% of the sample population have seen their risk reduced to a level below 20% CVD risk threshold (i.e. the proportion below the 20% CVD risk threshold has increased from 81.1% to 82.9% over the period).

The use of clinical thresholds to determine whether a patient should receive treatment has strong parallels with the measurement of poverty. The intent of clinical guidelines for the treatment of CVD is to target treatment at patients deemed at a high risk. Measures tracking the proportion of the population above a threshold (e.g. 20% 10 year risk) provides information on the overall performance in the prevention of CVD. As explained in section 2, we measure CVD-related health poverty by looking at the CVD resilience gaps of individuals. The proportion of the population that exceeds a certain CVD risk (e.g. those deemed at high risk) is captured by the headcount ratio, i.e. the poverty measure

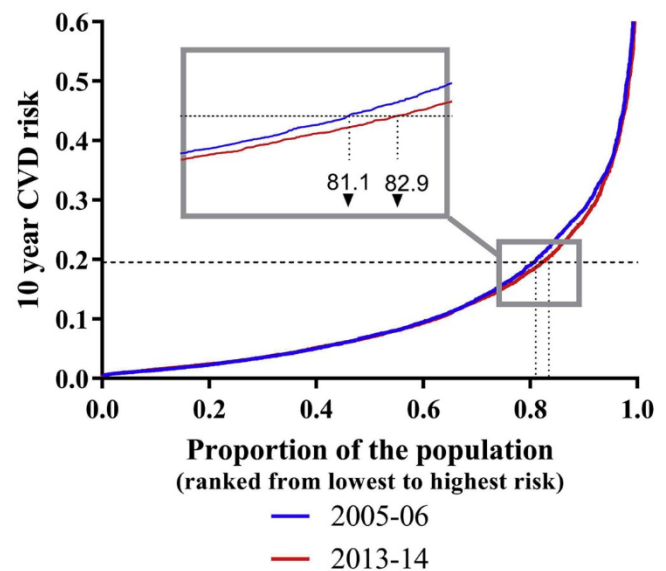


Fig. 2. Comparison of actual CVD risk over time.

$P_0$ . The poverty gap ratio  $P_1$  and the squared gap measure  $P_2$  both provide measures of the degree to which CVD risk exceeds a given threshold.

Table 1 reports the three measures of poverty. The values of the headcount ratio accord with those reported in Figs. 1 and 2 and estimate the fraction of the population deemed at high risk (i.e. > 20%). The poverty gap ratio calculates the average distance to the threshold, where the normalized health gap is taken as a measure of an individual's distance. The squared gap measure has a similar interpretation, but by squaring the gaps greater weight is given to those who suffer relatively high risks. In terms of the headcount ratio, moving from the current treatment to universal treatment would have a higher absolute effect of the level of health poverty (−3.94% and −3.18%) than moving to no treatment at all (1.85% and 2.22%). In terms of the other poverty measures we see similar patterns occurring; however, because of the small absolute values of the indices, the differences appear to be more modest. The changes in the poverty measures are useful in quantifying the medical concept of a 'treatment gap' which is often defined as the number of people with a condition or disease who need treatment for it but who do not get it (Kale, 2002). Here, the treatment gap would equate to the difference between current and universal treatment described above.

#### 4. Health poverty in Australia

We now illustrate how we can gain insight into health poverty in Australia based on metrics commonly used by health economists, i.e. reported health-related quality of life and estimates of life expectancy. While these measures have been employed previously in the measurement of health inequalities (e.g., Gerdtham and Johannesson, 2000), we explore here how they can be used for health poverty analysis. We also decompose health poverty according to different group classifications, based on gender, smoking habits and ethnicity, and we estimate to what extent health poverty is related to income. For the empirical estimates we use data from the *Household, Income and Labour Dynamics in Australia* (HILDA) Survey which is a household-based panel study which collects a wide range of information about economic and personal well-being, labour market dynamics, family life and health status (including use of the Short-Form (SF-36) health status instrument) (<http://melbourneinstitute.unimelb.edu.au/hilda>). The HILDA survey has involved annual interviews since 2001. The HILDA survey was reviewed by the Human Research Ethics Committee of the University of Melbourne; we have used the de-identified dataset that is made



**Table 1**  
Different measures of CVD-related health poverty.

	Headcount ratio $P_0$ ( $z = 0.8$ )			Poverty gap ratio $P_1$ ( $z = 0.8$ )			Squared gap measure $P_2$ ( $z = 0.8$ )		
	2005–2006	2013–2014	Difference	2005–2006	2013–2014	Difference	2005–2006	2013–2014	Difference
No treatment (NT)	20.71%	19.30%	–1.42%	3.80%	3.53%	–0.27%	1.31%	1.23%	–0.08%
Current treatment (CT)	18.86%	17.08%	–1.78%	3.03%	2.65%	–0.38%	0.90%	0.77%	–0.12%
Universal treatment (UT)	14.92%	13.90%	–1.02%	2.08%	1.92%	–0.16%	0.55%	0.52%	–0.03%
Difference (NT)–(CT)	1.85%	2.22%	0.77%	0.88%	0.88%	0.41%	0.41%	0.45%	–0.03%
Difference (UT)–(CT)	–3.94%	–3.18%	–0.95%	–0.73%	–0.73%	–0.35%	–0.35%	–0.25%	–0.25%

Note: The 95% confidence intervals obtained via bootstrapping (1000 replications) are reported in Table A5 of the supplementary material.

Source: own calculations, based on data from the NHANES surveys of 2005–06 and 2013–14.

**Table 2**  
Regression coefficients of the life expectancy estimation.

	Men ( $n = 11,943$ )			Women ( $n = 12,877$ )		
	Coefficients	Z score	Hazard ratio	Coefficients	Z score	Hazard ratio
12 years or more of education	0.077	0.96	1.081	–0.014	–0.11	0.986
Current smoker	<b>0.530</b>	4.92	1.629	<b>0.483</b>	3.65	1.621
Currently married	–0.446	–5.40	0.640	–0.112	–1.22	0.894
Rural region	–0.208	–1.98	0.812	–0.205	–1.5	0.814
Real equivalent household income	–0.007	–3.38	0.993	0.000	0.21	1.000
Bodily pain	0.038	1.81	1.038	0.044	1.89	1.045
General health	–0.147	–6.11	0.864	–0.099	–3.44	0.906
Mental Health	<b>0.078</b>	2.67	1.081	<b>0.067</b>	1.99	1.070
Physical functioning	–0.066	–3.22	0.936	–0.105	–4.56	0.900
Health transitions	–0.057	–1.61	0.944	0.021	0.40	1.022
Role-emotional	–0.002	–0.18	0.998	0.006	0.42	1.006
Role-physical	–0.007	–0.49	0.993	0.006	0.36	1.006
Social functioning	–0.025	–1.08	0.975	–0.063	–2.44	0.939
Constant	<b>–11.500</b>	–46.58		<b>–12.36</b>	–40.34	
Gamma	<b>0.110</b>	36.04		<b>0.111</b>	30.58	

Note: Coefficients in bold significant at  $P < 0.05$ .

available to researchers.

#### 4.1. Measurement of health status

Our measure of health status consists of SF-6D scores derived from questions in the SF-36 health survey that is a widely used measure of health-related quality of life. The SF-6D is composed of six multi-level dimensions: physical functioning, role limitations, social functioning, pain, mental health and vitality that are measured on an index anchored at 1 for full health and 0 for dead (Brazier et al., 2002). We use the values for the SF-36 health survey that are reported as part of the publicly released HILDA data set. Callander et al. (2012) already suggested to use SF-36 values for the purpose of the measurement of multidimensional poverty in Australia.

More formally we denote the univariate health status variable SF-6D by  $q_i$ . In this case, our health variable ( $h_i = q_i$ ) ranges between 0 (a state of very poor health equivalent to death) and 1 (full health). In contrast to the first application, we now assume that the threshold values are not uniform. The reference groups are defined in terms of age and sex. We opt to derive our threshold value  $z_J$  for reference group  $J$  from the average health achievement  $\bar{q}_J = \frac{1}{n_J} \sum_{i \in J} q_i$  of this group. More specifically, we adopt a relative approach: we assume the poverty threshold is a fraction  $\lambda$  below the average health achievement of the reference group. Hence, the poverty threshold for reference group  $J$  is  $z_J = (1 - \lambda)\bar{q}_J$ . An alternative approach, which we do not explore here, would be to define the poverty threshold in absolute terms, which would give  $z_J = \bar{q}_J - x$ .

#### 4.2. Predictions of life expectancy

Our final application involves estimates of life-expectancy which are

derived empirically by taking advantage of matching the HILDA survey respondents to the Australia National Death Index (Watson and Summerfield, 2014) so that a year of death is recorded in the public data set. To simplify the estimation of life expectancy we use a proportional hazards survival regression model using a Gompertz parametric form, which has been shown to perform well in modelling human survival and in modelling life expectancy of the HILDA participants (Clarke and Leigh, 2011).

We adopt a life-table approach so that individual age is used as the time at risk. This implies that the mortality experience of HILDA respondents at different ages provides estimates of the hazard and survivor functions for individuals over their remaining lifetime. Individual life expectancy was conditioned on a wide range of explanatory variables as reported in the first wave of HILDA including age, sex, year of birth, socioeconomic conditions (marital status, education level, income), lifestyle choices (smoking), and health (general health status, bodily pain, social functioning). The definitions of the variables and summary statistics are in Table A2 of the Appendix. The continuous variables (income and health status measures) were transformed to variables that measure the deviations from the mean value in each age/sex category. Our estimation procedure ensures that for every age and sex group the average predicted life expectancy approximates the observed life expectancy reported for the Australian population (see Table A3 of the Appendix).

Table 2 provides details on the coefficients of the Gompertz proportional hazard survival regressions models for men and women, as several variables (e.g. income) have a differential effect by gender. The estimated coefficients  $\beta$  and  $\gamma$  form the basis of an empirically derived survivor function for each individual  $i$ :

$$S(a_i) = \exp(-\exp(x_i\beta)\gamma^{-1}(\exp(\gamma a_i) - 1)) \quad (8)$$

where  $a_i$  is the years of age and  $x_i$  a vector of explanatory variables. Life expectancy  $e_i$  is then calculated by using standard life table methods (Lee and Wang, 2003). The same estimated equation (reported below) is applied to all survey waves to provide estimates of life expectancy for individuals that are dependent on covariates which can change over time (e.g., changes in health status as measured by domains of the SF-36). Finally, the average age at death was estimated by adding the life expectancy to the age at the time of the survey.

Denoting individual empirically derived life-expectancies by  $e_i$ , the expected age at death is the sum of the age and the predicted remaining life years of a person can easily be calculated. If  $a_i$  is the age of person  $i$ , then  $d_i \equiv a_i + e_i$  is this person's expected age at death. Let  $\bar{e}_j$  be the average life expectancy of the members of reference group  $J$ , i.e.  $\bar{e}_j = \left( \sum_{i \in J} e_i \right) / n_j$ . Assuming that all members of group  $J$  have the same age  $a_j$ , the average expected age at death of any person belonging to this group is equal to  $\bar{d}_j = a_j + \bar{e}_j$ . If for person  $i \in J$  the expected age at death lies above this average expected age at death, i.e. if we have  $d_i > \bar{d}_j$  or equivalently  $e_i > \bar{e}_j$ , then this person is relatively well-off in health terms. Health poverty concerns those who have a predicted age at death significantly below that of their reference group. In this application, therefore, the health variable we are focusing on is expected age at death ( $h_i = d_i$ ), and as reference values for the determination of the poverty thresholds we take the group-specific average expected age at death ( $\bar{d}_j$ ). Using the survival equations reported in Table 2 to estimate  $e_i$  the overall average  $d_i$  of the HILDA cohort over all the waves is 81.1 years for men and 86.6 for women, which are close to the official estimates of life expectancy for Australia (Australian Bureau of Statistics, 2010).

As with SF-6D, we follow a relative approach to define poverty thresholds. The thresholds are defined by deducting a fixed fraction  $\lambda$  from the group-specific average expected ages of death. The threshold levels are then equal to  $z_j = (1 - \lambda)\bar{d}_j$ . We assume that any member of group  $J$  whose predicted age at death is lower than the threshold value  $z_j$  is health poor.

#### 4.3. Results

To illustrate measures of health poverty we adopt a threshold equal to 95% of each group's reference level ( $\lambda = 0.05$ ) for both measures of health status and life expectancy. At the mean age this equates to an SF-6D health status score that is on average 0.04 units lower and for life expectancy around 4 years shorter than the average life expectancy for the reference group. (Table A4 in the Appendix contains comparable results for different thresholds and shows similar trends as those we discuss below.)

We report the results for the FGT poverty indices  $P_0$ ,  $P_1$  and  $P_2$  for SF-6D and life expectancy in Table 3 and in Figs. A1 and A2 of the Appendix. As far as SF-6D is concerned, the headcount ratio for the population as a whole tended to fluctuate around a value of 33%. Two

phases can be distinguished. Between 2001 and 2009 health poverty decreased significantly, with the headcount ratio falling from 35.36% to 31.09% ( $\Delta = -4.27\%$ ). However, between 2009 and 2015 the decrease has come to a halt and health poverty rates have increased to their original levels ( $\Delta = 4.55\%$ ). The poverty gap ratio and the squared gap measure indicate similar trends. It seems that 2009 was a turning point, possibly related to the global economic recession. Health poverty in terms of life expectancy is lower, with the headcount ratio averaging around 22%. The three poverty rates also show a decreasing trend up until 2009. Since then there is some evidence of a rise in poverty rates, but to a lesser extent than for SF-6D poverty.

Interesting results can be obtained by various decompositions of poverty for both quality of life and life expectancy. Since the results are very similar for the two health variables and the three poverty measures, we report only the results for the life expectancy headcount ratio. In Table 4 we report the values of this ratio for men and women, smokers and non-smokers, and indigenous and non-indigenous people. The headcount ratios for men are systematically above those for the population as a whole, while the ratios for women are systematically below. For both men and women the poverty headcount ratios reached a minimum in 2009, and tended to increase again after that. The gap between women and men, i.e. the absolute difference in their poverty headcount ratios, remains substantial but has varied over time (the gap was 9.94% in 2002 and 5.74% in 2013). There appears to be a persistent but weakening difference between men and women when it comes to life expectancy-related health poverty. This gap is not due to the fact that women tend to live longer than men, since we correct for that using age and sex specific poverty thresholds. One of the main reasons for the difference lies in the prevalence of smoking. As Table 4 shows, there are huge differences in health poverty rates between smokers and non-smokers. While roughly half of the smoking population lives in health poverty, only one seventh of the non-smoking population does. Moreover, especially since 2009, the poverty headcount ratios for smokers have risen substantially and in 2014 and 2015 were above 60%. As a result, the gap between smokers and non-smokers has widened from 36.38% in 2001 to 46.47% in 2015.

For the indigenous population, too, we find health poverty rates which are much higher than those for the rest of the Australian population. Between 40% and 50% of the indigenous population lives in health poverty, roughly twice the rate of the non-indigenous population. According to our calculations, the gap between the indigenous and non-indigenous population has widened, from 18.07% in 2001 to 28.18% in 2015.

We also looked at the association between health poverty and income. The HILDA dataset provides information on the household income of every individual in every wave of the survey. A nice visual representation of the association can be obtained by grouping individuals into quintiles according to the level of their equalized household income. Fig. 3 represents the life expectancy-related poverty headcount ratios for each of the five quintiles, with the first quintile being the poorest and the fifth the richest. The first remarkable

**Table 3**

Poverty rates (%) in terms of health status (SF-6D index scores) and life expectancy for Australia, 2001–2015,  $\lambda = 5\%$ .

	Year	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015
	N	12210	11290	11051	10900	10957	11173	10767	10682	10927	11625	14848	14929	14929	15181	15053
SF-6D Index	P0	35.36	33.53	33.84	33.63	34.45	32.16	32.13	31.71	31.09	33.45	32.95	32.69	33.54	34.64	35.63
	P1	5.09	4.72	4.89	4.76	4.82	4.59	4.55	4.46	4.35	4.75	4.73	4.65	4.77	5.15	5.17
	P2	1.10	0.99	1.04	0.99	1.00	0.98	0.96	0.93	0.90	1.00	1.02	0.98	1.00	1.12	1.12
Life expectancy	P0	22.43	22.70	23.11	23.02	22.55	21.91	21.79	21.00	20.94	22.00	21.24	21.10	21.33	22.30	21.84
	P1	1.20	1.23	1.22	1.22	1.19	1.16	1.14	1.07	1.05	1.17	1.10	1.08	1.12	1.14	1.16
	P2	0.11	0.12	0.11	0.11	0.11	0.11	0.10	0.09	0.09	0.11	0.10	0.10	0.10	0.10	0.11

Note: Figs. A1 and A2 of the Appendix provide the 95% confidence intervals around these estimates.

Source: Own calculations, based on data from the HILDA survey.

**Table 4**

Life expectancy poverty headcount ratios in Australia, 2001–2015, by subgroups.

Year	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015
Men	27.02%	27.94%	28.07%	27.10%	26.33%	26.15%	25.78%	24.79%	24.40%	25.80%	24.68%	24.49%	24.39%	26.23%	25.74%
Women	18.32%	18.00%	18.70%	19.43%	19.25%	18.20%	18.34%	17.69%	17.89%	18.64%	18.18%	18.15%	18.65%	18.85%	18.39%
Gap	8.70%	9.94%	9.36%	7.67%	7.08%	7.94%	7.44%	7.11%	6.51%	7.16%	6.50%	6.34%	5.74%	7.38%	7.36%
Smokers	50.30%	51.16%	52.85%	53.89%	52.30%	53.51%	52.79%	52.11%	51.53%	54.95%	55.77%	55.96%	58.56%	60.33%	61.03%
Non-smokers	13.92%	14.61%	14.91%	14.75%	14.81%	14.25%	14.37%	13.89%	14.13%	14.77%	14.12%	14.34%	14.27%	14.96%	14.56%
Gap	36.38%	36.55%	37.93%	39.14%	37.49%	39.26%	38.42%	38.22%	37.40%	40.17%	41.66%	41.62%	44.30%	45.38%	46.47%
Indigenous	40.19%	41.36%	40.61%	40.22%	42.49%	42.99%	39.82%	38.39%	39.74%	47.08%	49.54%	46.09%	44.53%	48.98%	49.26%
Non-indigenous	22.12%	22.38%	22.79%	22.73%	22.19%	21.50%	21.41%	20.65%	20.53%	21.44%	20.61%	20.49%	20.74%	21.59%	21.08%
Gap	18.07%	18.98%	17.82%	17.49%	20.29%	21.49%	18.41%	17.74%	19.22%	25.64%	28.93%	25.60%	23.80%	27.39%	28.18%

Note: At baseline women represented 52.7%; smokers 23.3% and Indigenous 1.7% of the sample.

Source: Own calculations, based on data from the HILDA survey.

observation is that in every year there is a negative social gradient: the higher the income quintile, the lower the poverty headcount ratio. As a result, there exists a wide gap in health poverty between the first and the fifth quintile. Moreover, this gap is widening over time: while the poverty headcount ratio of the fifth quintile has fallen from 13.64% in 2001 to 9.34% in 2015 ( $\Delta = -4.31\%$ ), that of the first quintile has risen from 32.64% to 36.77% ( $\Delta = 4.03\%$ ), with much of the rise occurring after 2009. As a result, the gap between the fifth and the first quintiles has broadened from 18.82% in 2001 to 27.43% in 2015. The first and second quintiles are the only ones for which the poverty headcount ratio has increased over the period 2001–2015, indicating that while life expectancy-related health poverty rates have improved for the population as a whole, this did not happen among those who are income poor. We find similar patterns when looking at health status (Fig. 4). Another way of looking at the association between income and health poverty is to calculate aggregate measures of income-related inequality of health poverty. Fig. 5 represents the evolution of the bounded Concentration index. (The results are similar for the level-dependent index and are not reported here.) As expected, the index values are negative for every wave of the survey, confirming the social gradient visible in the poverty headcount ratios of the income quintiles. Moreover, the index tends to increase in absolute value over time, albeit not uniformly so, which suggests that socioeconomic inequality of health poverty has worsened over the period 2001–2015. These findings are in line with previous research on the relation between income levels and health outcomes in Australia. Based on a simple cross-tabulation of health and poverty status, Buddelmeyer and Cai (2009: 11) have drawn attention to the “clear positive association between ill-

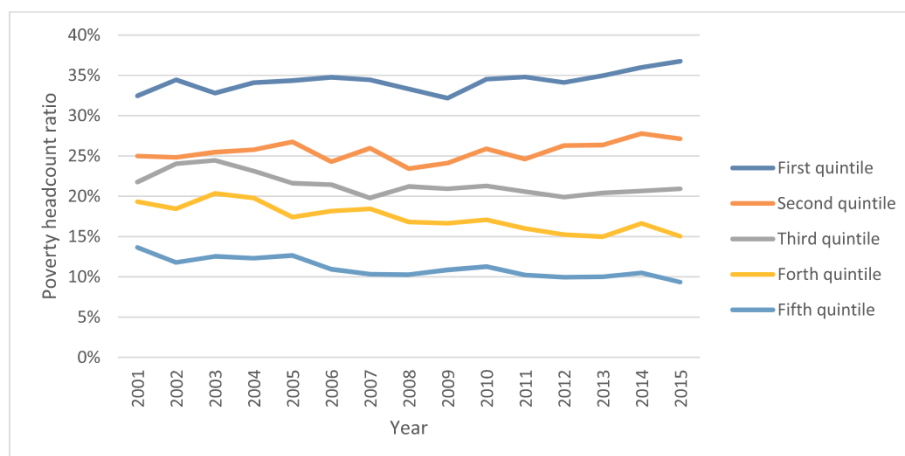
health and poverty”. Our framework allows a more precise measurement and detailed analysis of the correlation between income and ill-health.

## 5. Conclusions and future directions

The purpose of this study has been to explore how poverty measures can be used to capture inequalities in health outcomes by revealing to what extent people's health falls below minimally acceptable thresholds. We show that health poverty measures accord closely with widely used practices in medicine to target treatments based on thresholds, such as critical cardiovascular risk levels. Poverty measures also provide a way of systematically quantifying the degree to which a population's health could be improved by treating all persons eligible for treatment (i.e., who are below a threshold).

The dominant approach to measuring health inequalities has been to quantify socio-economic related health inequalities (e.g., the gradient of health outcomes by levels of income) using measures such as the Concentration index. Unlike clinicians that often target treatment using thresholds (e.g., CVD risk), health economists have tended not to focus on the bottom of the health distribution. Our feeling is that poverty measures provide a useful additional set of indices which are likely to have an intuitive appeal among clinicians and policy makers, and which allow economists to draw upon useful properties such as subgroup decomposition. Measuring health poverty is a way of paying more attention to those who are worst off when it comes to health.

As far as the usefulness of health poverty estimates is concerned, there is an obvious connection to the research on the measurement of

**Fig. 3.** Life expectancy poverty headcount ratios in Australia, 2001–2015, by income quintiles.

Source: Own calculations, based on data from the HILDA survey.



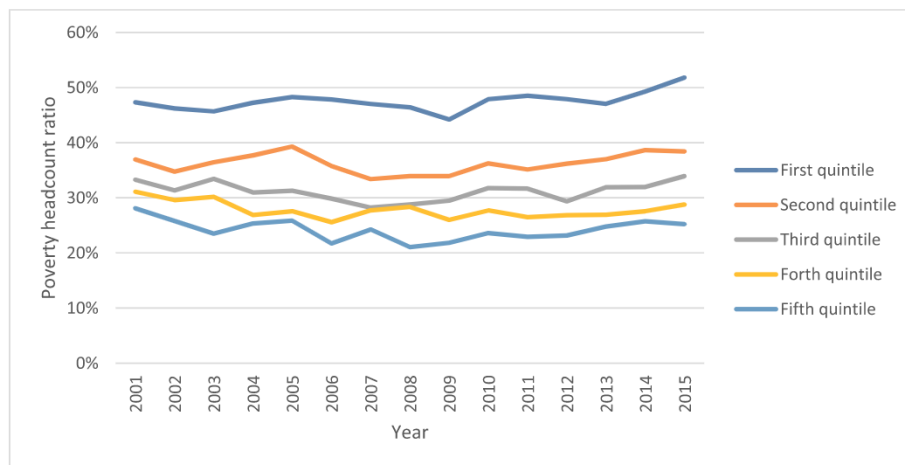


Fig. 4. Quality of life poverty headcount ratios in Australia, 2001–2015, by income quintiles.  
Source: Own calculations, based on data from the HILDA survey.

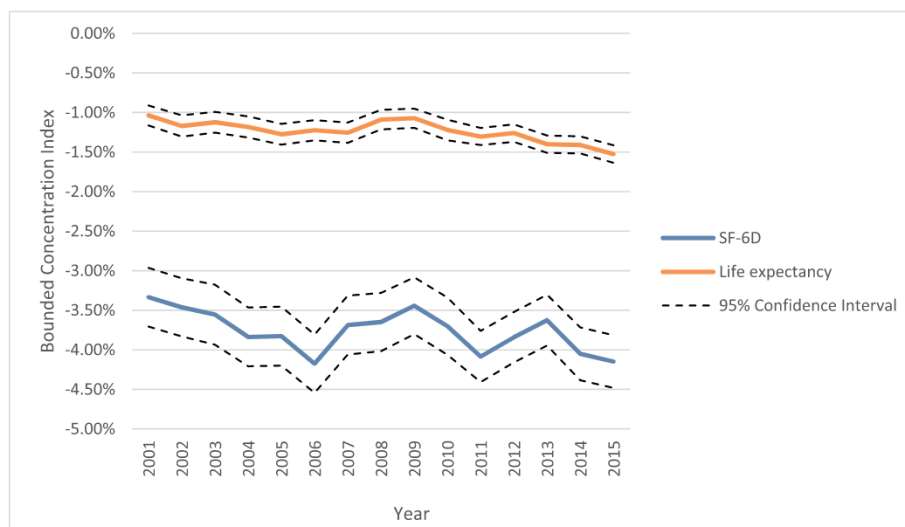


Fig. 5. Bounded Concentration indices of health poverty in Australia, 2001–2015.  
Source: Own calculations, based on data from the HILDA survey.

multidimensional poverty (e.g., [Alkire and Santos, 2014](#)). Policy makers may find health poverty calculations useful in order to identify which groups are most disadvantaged in terms of health. However, we do not claim there must be an automatic link between high health poverty rates for certain groups and health policy priorities. Policy makers should also take into account to what extent high levels of health poverty are fair or unfair (cf. the distinction of fair and unfair inequalities in health by [Fleurbay and Schokkaert, 2009](#)).

The systematic measurement of health poverty will require considerable further research. There is scope to apply the health poverty measures developed here much more broadly. Many national health surveys provide information on health status using a generic instrument such as the SF-6D scores of individuals and some provide clinical measures that enable CVD risk to be estimated. A key issue to advance the measurement of health poverty will be to determine thresholds for health status measures such as SF-6D which would define those who are in what would be considered “poor” health. Potentially the income poverty literature (e.g. [Ravallion, 1998](#)) can provide a roadmap for this research. Recent research involving the use of citizens' workshops to define a sufficient level of capabilities could also be worth exploring in future work ([Kinghorn, 2019](#)). Such standard measures of health or illness facilitate comparing poverty rates across countries and over time. To the extent that it is possible to estimate individual life

expectancies, there is also room for comparisons of these types of health poverty between countries and ultimately to develop measures based on more holistic health measures such as Quality Adjusted Life Years.

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## Appendix A. Supplementary data

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