TI 2023-017/V
Tinbergen Institute Discussion Paper

Distributionally Sensitive Measurement and Valuation of Population Health

Shaun Da Costa[1]
Owen O'Donnell[2,3]
Raf Van Gestel[2]

1 Paris School of Economics and University of Antwerp

2 Erasmus University Rotterdam

3 Tinbergen Institute

Distributionally Sensitive Measurement and Valuation of Population Health

Shaun Da Costa Owen O'Donnell Raf Van Gestel *

March 24, 2023

Abstract

We introduce a measure of population health that is sensitive to dispersion in both age-specific health and lifespan. The measure generalises health-adjusted life expectancy without requiring more data. A transformation of change in the measure gives a distributionally sensitive monetary valuation of change in population health and disease burden. Application to Sub-Saharan Africa between 1990 and 2019 reveals that the change in population health is sensitive to allowing for lifespan dispersion but is less sensitive to age-specific health dispersion. Distributional sensitivity changes relative burdens of diseases, reduces convergence between the burdens of communicable and non-communicable diseases, and so could influence disease prioritisation. It increases the value of health improvements relative to GDP.

Keywords: Health, Lifespan, Life Expectancy, Inequality, Global Burden of Disease, Sub-Saharan Africa

JEL Codes: I14, I15, J11, J17, O15

*Da Costa: Paris School of Economics and University of Antwerp, shaun.dacosta@uantwerpen.be; O'Donnell: Erasmus University Rotterdam, Tinbergen Institute, odonnell@ese.eur.nl; Van Gestel: Erasmus University Rotterdam, vangestel@eshpm.eur.nl. We thank Matthew Robson, Pieter van Baal, and Tom Van Ourti for comments. Da Costa's research was partly funded by the Research Foundation – Flanders (FWO), grant G056918N. O'Donnell's research was partly funded by Swiss National Science Foundation Swiss Agency for Development and Cooperation grant 400640_160374.

1 Introduction

Population health is usually measured with averages, such as life expectancy and health-adjusted life expectancy. These measures ignore dispersion in lifespan and health, which is inconsistent with stated preferences for extending a shorter (health-adjusted) life rather than a longer one by a given amount (Dolan et al., 2005; Dolan & Tsuchiya, 2012; Robson et al., 2023). If there is aversion to dispersion in lifespan and health, then an increase in (health-adjusted) life expectancy will understate (overstate) improvement in population health when mortality falls most at younger (older) ages (van Raalte et al., 2018). The incomplete impression of population health given by averages may affect resource allocation to and within a health system.

We introduce a measure of population health that is sensitive to dispersion in both age-specific health and health-adjusted lifespan. We use nested equity equivalents (Berger & Emmerling, 2020) to allow for aversion to dispersion in both the health and lifespan dimensions. The measure — equivalent health-adjusted lifespan ($EHAL$) — is in the life years metric and nests health-adjusted life expectancy ($HALE$). To ensure maximum applicability, we deliberately constrain the measure to require no more data than $HALE$: a health-extended period life table (Sullivan, 1971) and disease weights (Global Burden of Disease Collaborative Network [GBDCN], 2021). Consequently, $EHAL$ is both a generalisation of $HALE$ (and life expectancy) and always a feasible alternative to it. As far as we know, our population health measure is the only one that is sensitive to dispersion in the distributions of both health and lifespan and does not require estimation or simulation of their joint distribution.

With a period life table, individual lifetime health profiles are not observed. Conditional on sex, there are no differences in ex ante health or lifespan. Our task is to aggregate over simulated distributions of health at each age and ages at death. We do this by first calculating the equally distributed equivalent (EDE) health at each age and then taking a

concave aggregation of the EDE health over ages at death. This requires specification of the degree of aversion to dispersion in each of age-specific health and health-adjusted lifespan.

A life years metric lacks comparability with costs of investments in population health and does not monetize the social value of health returns on such investments. To address these limitations, we derive societal willingness to pay for improvement in the distribution of population health that is sensitive to dispersion in both age-specific health and health-adjusted lifespan. This distributionally sensitive valuation of change in population health is a function of the relative change in $EHAL$ and parameters that derive from a budget constraint and willingness to sacrifice consumption for health and lifespan.

We extend Silber's (1983) equivalent length of life (ELL) measure that adjusts life expectancy for lifespan dispersion. This has been applied within and between countries (Goerlich, 2020; Le Grand, 1987; Muszyńska & Janssen, 2016; Shkolnikov et al., 2003) and is used to produce the inequality-adjusted Human Development Index (Alkire & Foster, 2010; Foster et al., 2005; Hicks, 1997; United Nations Development Programme [UNDP], 2020). Our measure adds adjustments for mean health and health dispersion to the ELL adjustment for lifespan dispersion.

The contribution of our money metric to previous approaches to the valuation of population health gains (Hall & Jones, 2007; Murphy & Topel, 2006) is through the incorporation of aversion to dispersion in both health and lifespan. Murphy and Topel (2006) extend estimation of the value of life and lifespan (Rosen, 1988; Schelling, 1968; Usher, 1973) to partially include the value of health. However, they only capture the indirect effect of health on lifetime utility through the optimal consumption path and do not allow for the value of reduced health dispersion. Edwards (2013) and Córdoba and Ripoll (2017) extend the value of a statistical life (VSL) framework to allow for aversion to lifespan dispersion but do not incorporate the value of health and aversion to health dispersion.[1] Our measure

[1]Córdoba and Ripoll (2017) claim their approach could be extended to allow for different types of risk (dispersion) at each age. However, Bommier et al. (2022) show that their model is inadequate for assessing the value of longevity gains when the intertemporal elasticity of substitution is less than one, which is the

captures the value of both health and longevity with allowance for aversion to dispersion in each dimension. This recognises that the value of progress against disease lies not only in the consequent increase in health-adjusted life expectancy but also in reduced exposure to variation of health and lifespan.

Adler et al. (2021) use a prioritarian social welfare function (Atkinson, 1970) to derive a value of mortality risk reduction that respects the fair innings principle: the social value of an extra year of life is greater when it extends a life that is otherwise shorter (Bognar, 2015; Harris, 2006). We adopt a social welfare function in the same family and so ensure that our measure respects the prioritarian ethic that any given benefit contributes most to social welfare when it goes to the worst off (Parfit, 2000). While the general normative foundation for our approach is consistent with that adopted by Adler et al. (2021), our objective is different and our contribution differs in two main respects. First, we allow welfare to depend on health and derive a measure that is sensitive to the distribution of health (and lifespan), not only to change in mortality risk. Second, we obtain a money metric valuation of changes in the distributions of health and lifespan. We derive the latter measure from willingness to sacrifice consumption for health and lifespan, which brings us somewhat closer to the VSL approach.

Healthcare interventions are often evaluated with respect to effects on quantity and quality of life that are observed, or more often simulated, at the individual level. Hougaard et al. (2013) identify restrictions on social preferences that are required for a population health evaluation function ($PHEF$) to capture in a single number the two-dimensional health of individuals in a population. For example, reliance on aggregate quality-adjusted life years ($QALYs$) requires an assumption, among others, of indifference to dispersion in quantity of life across individuals with equal quality of life. The authors identify distributionally sensitive $PHEFs$ that, like our $EHAL$, relax this assumption.[2] Our objective is different. It is to

most empirically relevant case.

[2]Moreno-Ternero et al. (2023) characterise a more general class of distributionally insensitive $PHEFs$ defined over reference health, in addition to quantity and quality of life, observed for each individual. This permits identification of axioms that link and distinguish $QALYs$ and disability-adjusted life years ($DALYs$).

add distributional sensitivity to measures that summarise population health captured by a health-extended period life table. Hence, we do not define each individual by a fixed number of remaining life years and an age invariant health level, and then assume individual-level observation of each dimension of health. Rather, we start with a hypothetical cohort of individuals, each facing a risk of death and a distribution of health in each year of life. We allow for aversion to cohort dispersion in both lifespan and health at each age.

We illustrate our distributionally sensitive measurement and valuation of population health with an application to Sub-Saharan Africa (SSA) from 1990 to 2019 using data from the Global Burden of Disease (GBD) (GBDCN, 2021). Sensitivity to dispersion in age-specific health has relatively little impact on the change in population health over the period. This is reassuring for the use of $HALE$. Sensitivity to dispersion in health-adjusted lifespan has a large impact. Even allowing for only moderate aversion to dispersion in health and lifespan, $HALE$ increased by around 28% over the period, while $EHAL$ increased by around 70% due to steeper reductions in mortality at younger ages.

We measure the contribution of each of 293 diseases to the overall disease burden in SSA by eliminating morbidity and mortality caused by that disease from the health-extended life table, recomputing $HALE$ and $EHAL$ under this counterfactual, and subtracting the respective measure obtained from the complete life table. Switching from $HALE$ to $EHAL$ substantially increases the burdens of communicable, maternal, neonatal, and nutritional diseases (CMNNs), which are more prevalent at younger ages, relative to the burdens of non-communicable diseases (NCDs), which are more prevalent at older ages. Allowing for distributional sensitivity greatly reduces the extent to which the NCD burden is converging on the CMNN burden and negates a previous finding that the NCD burden had overtaken the CMNN burden in SSA (Gouda et al., 2019).

Allowing for distributional sensitivity causes steep increases in the estimated welfare cost of CMNNs. For example in 2019, adjustment for effects on health and lifespan dispersion increases the monetary equivalent of the burden of each of lower respiratory infections (LRI),

diarrheal diseases, and malaria by at least eight percentage points of GDP. Without distributional sensitivity, we estimate that LRI imposed a burden equivalent to around 6.5% of GDP. With distributional sensitivity, the estimate increases to 16.1%. In contrast, distributional sensitivity reduces the welfare costs of some NCDs with particularly high prevalence at older ages. For example, the monetary equivalent of the burden of ischemic heart disease/stroke falls from 4.5% of GDP without adjusting for effects on health and lifespan dispersion to 3.9% with such adjustment. Such adjustment could affect the prioritization of disease programmes.

Allowing for distributional sensitivity increases the estimated welfare gain from improvements in population health in SSA between 1990 and 2019 from around 46.5% of baseline Gross Domestic Product (GDP) to 67%.

The paper proceeds as follows. In section 2, we first derive our distributionally sensitive population health measure and then use it to obtain a money equivalent of health change and disease burden that takes account of how the impact of a disease on health and lifespan dispersion affects willingness to pay to eliminate it. Section 3 describes the GBD data and explains how we use them to simulate health and age-at-death distributions that, in turn, are used to calculate the population health measures. Section 4 gives the results of the application to population health and disease burden in SSA. The final section concludes by discussing limitations and potential applications.

2 Theory

2.1 Distributionally sensitive measurement

2.1.1 Building blocks

Consider a hypothetical birth-year cohort of same-sex individuals who face the age-specific mortality rates prevailing in their year of birth. The mean age at death of this period life-table

cohort is life expectancy at birth (LE). Lack of attention to morbidity is addressed by health-adjusted life expectancy ($HALE$): expected years lived in full health for a health-extended life-table cohort exposed to age-specific disease incidence and mortality rates prevailing at birth (Sullivan, 1971),

$$HALE = \sum_{x>0}^{T} r(x) \sum_{i=0}^{x-1} h(i) = \sum_{x>0}^{T} r(x) y^m(x) \,, \tag{1}$$

where T is the maximum postulated lifespan, $r(x)$ is the proportion of the cohort that dies in the age interval $[x, x+1)$ and $h(i) = \sum_s p_s(i) h_s$ is expected health at age i, with $p_s(i) \in [0,1]$ the probability of being in health state s at that age and $h_s \in [0,1]$ the respective level of health.[3] For a state equivalent to death, $h_s = 0$, while $h_s = 1$ corresponds to full health.[4] Health states, $s = 1, 2, .., S$, are ordered, such that $h_s \leq h_{s+1}$ $\forall s$. The cumulative sum $y^m(x) = \sum_{i=0}^{x-1} h(i)$ is the (mean) health-adjusted lifespan, which is equivalent to the number of years lived in full health up to age x. If no adjustment is made for mean health, such that $h(i) = 1$ $\forall i$, then eq.(1) reduces to $LE = \sum_{x=0}^{T} r(x) x$.

LE and $HALE$ are statistical expectations that capture an array of information on mortality rates, disease incidence rates, and health state values that enter a health-extended life table. Aggregation over this information to evaluate population health involves the imposition of normative judgment. Restricting attention to the first moment of the age-at-death distribution, as is done with LE, implies indifference to any change comprising a reduction in younger-age mortality that is fully offset by a larger increase in older-age mortality, leaving LE unchanged.[5] Use of $HALE$ imposes the further judgement that, at any given age, only the mean health counts. Change in disease incidence rates that reduced the variation in potential health outcomes without affecting the mean would not register as

[3] To save on notation, we derive the measures in this section assuming a one-year age interval. We relax this assumption in section 3 and the application.

[4] Because health is normalised to zero at death, the age at which death occurs is omitted from the summation over i in (1) and the first sum is over positive values of x.

[5] LE would change as a result of same-sized changes in younger-age and older-age mortality rates. However, LE gives the same weight to each additional life year irrespective of the length of the life that is extended.

an improvement in population health.[6]

We relax these restrictions by invoking a social decision maker (SDM) who may be averse to dispersion in the distributions of age-specific health and lifespan generated by health-extended period life tables. With no such aversion, the SDM prefers the distribution that generates the highest $HALE$. Otherwise, the SDM is prepared to sacrifice $HALE$ for less dispersion in health-adjusted lifespan. We refer to dispersion rather than inequality to avoid giving the impression that our objective is to adjust measures for unfairness arising from social determination of health and lifespan.

Taking account of differences in both age-specific health and health-adjusted lifespan is an example of the general problem of welfare evaluation through aggregation over multiple dimensions. Berger and Emmerling (2020) show how to do this using nested equity equivalents that have been used to produce indices of multidimensional well-being that are sensitive to inequalities in different dimensions (Bosmans et al., 2015; Foster et al., 2005). We first allow for aversion to dispersion in age-specific health distributions. Then, we introduce distributional sensitivity to the aggregation of health-adjusted lifespans.[7]

2.1.2 Sensitivity to health distributions

At each age, there is a cumulative distribution of health, $F\left(h_s(i)\right) = \sum_{t \leq s} p_t(i)$, over the hypothetical cohort members who survive to that age. Each distribution is assumed to

[6] $HALE$ is sensitive to dispersion in lifespan. A permutation in the distribution of deaths that increases lifespan by one year at a younger age and decreases lifespan by one year at an older age, leaving LE constant, would increase $HALE$ if average health were monotonically decreasing in age because the weight on the additional year at a younger age would exceed that on the loss of one year at an older age (see eq.(1)). In addition to having this implicit sensitivity to the age distribution of health, our measure is explicitly sensitive to health dispersion at each age and to dispersion in health-adjusted lifespan.

[7] This is somewhat analogous to an approach to healthcare prioritisation proposed by Echazu and Nocetti (2013) that allows for aversion to both individual-level exposure to health risk and inequality over individuals in ex ante utility of health. We use a period life-table, and so there are no differences in ex ante health or lifespan to consider.

generate welfare according to an Atkinson (1970) social welfare function (SWF),

$$w\left(F\left(h_s(i)\right)\right) = \begin{cases} \sum_s p_s(i) \frac{{h_s}^{1-\varepsilon}}{1-\varepsilon} & \text{for} \quad \varepsilon \neq 1, \\ \sum_s p_s(i) ln\, h_s & \text{for} \quad \varepsilon = 1, \end{cases} \tag{2}$$

where $\varepsilon \geq 0$ is the SDM's degree of aversion to health dispersion across individuals of the same age and $ln\, h_s$ is the logarithm of h_s.[8] Larger values of ε give more weight to worse health outcomes. In the extreme, we get the preferences of a Rawlsian SDM who ranks distributions by the worst health outcome only: $w\left(F\left(h_s(i)\right)\right) \rightarrow min\{h_s(i)\}$ as $\varepsilon \rightarrow \infty$. When $\varepsilon = 0$, eq.(2) collapses to mean health at age i. The iso-elastic function ensures that the ranking of health distributions generated by the SWF is invariant to a proportionate rescaling of the health measure (Atkinson, 1970).

Consider some equally distributed equivalent (EDE) level of health $h_e(i)$, defined such that if everyone at age i were to experience it, then social welfare would be the same as that generated by the unequal age-specific health distribution: $w\left(h_e(i)\right) = w\left(F\left(h_s(i)\right)\right)$. Solving gives [9]

$$h_e(i) = \left[\sum_s p_s(i)\, {h_s}^{1-\varepsilon}\right]^{\frac{1}{1-\varepsilon}}. \tag{3}$$

It is not necessary to invoke a SWF to derive this expression. It is simply the generalised mean of health. For example, setting $\varepsilon = 0$, $\varepsilon = 1$, and $\varepsilon = 2$ gives the arithmetic, geometric, and harmonic means, respectively. $HALE$ restricts attention to the arithmetic mean. We generalise to make the measure sensitive to the distribution of health at each age in a way that is analogous to the inequality-adjusted Human Development Index (Alkire & Foster, 2010; Foster et al., 2005; UNDP, 2020).

Within a health-extended period life-table cohort, independence is imposed between

[8] In principle, our approach could allow aversion to age-specific health inequality to vary with age. In the application, we do not allow this because there is no evidence on which to base specification of parameter heterogeneity.

[9] We calculate this metric over those alive at each age and so $h_s > 0$. For $\varepsilon = 1$, $h_e(i) = exp\left(\sum_s p_s(i)\, ln\, h_s\right)$.

health at age i and both health and mortality at subsequent ages. Therefore, the lifetime equivalent health generated by cohort members who die at age x is $y^d(x) = \sum_{i=0}^{x-1} h_e(i)$. This is a measure of lifespan penalized for the distribution of health – both the mean and dispersion – at each age. If there were no aversion to variation in (health distribution-adjusted) lifespans, then averaging would give a population health measure that we label restricted equivalent health-adjusted lifespan ($REHAL$),

$$REHAL = \sum_{x>0}^{T} r(x) y^d(x). \tag{4}$$

This is $HALE$ with a penalty for age-specific health dispersion that increases with aversion to that dispersion.[10] An increase in age-specific health dispersion, or in aversion to it, increases the shortfall of $h_e(i)$ from $h(i)$, $y^d(x)$ from $y^m(x)$, and $REHAL$ from $HALE$.

2.1.3 Sensitivity to lifespan distribution

Having adjusted lifespans for the age-specific distributions of health, the second stage of our approach is to specify welfare as a nonlinear aggregation over these adjusted lifespans,

$$W\left(F\left(h_s(x)\right), G(x)\right) = \begin{cases} \sum_{x>0}^{T} r(x) \frac{y^d(x)^{1-\eta}}{1-\eta} & \text{for} \quad \eta \neq 1, \\ \sum_{x>0}^{T} r(x) ln\, y^d(x) & \text{for} \quad \eta = 1, \end{cases} \tag{5}$$

where $G(x) = \sum_{i=0}^{x} r(i)$ is the cumulative age-at-death distribution and $\eta \geq 0$ reflects the SDM's aversion to dispersion in adjusted lifespans.

$HALE$, in common with some attempts to value population health while taking account of both its morbidity and lifespan dimensions (Murphy & Topel, 2006), limits attention to the special case that adjusts lifespan with the arithmetic mean of age-specific health ($\varepsilon = 0 \Rightarrow y^d(x) = y^m(x)$). In our more general approach, increasing the parameter η gives more weight to an additional life year – adjusted for the age-specific distributions of health

[10] This can be confirmed by substituting $h_e(i)$ for $h(i)$ in eq.(1) to arrive at eq.(4). When there is no aversion to age-specific health dispersion ($\varepsilon = 0$), $REHAL = HALE$.

– when it extends a shorter life. With $\eta > 0$, a permutation of the distribution of deaths that adds an adjusted life year at age x and subtracts an adjusted life year at age $x' > x$ would leave $REHAL$ constant but increase the welfare measure W.

At any age x, there is no variation in $y^d(x)$. The aggregation in eq.(5) is not over individuals with different lifetime health profiles, which are not observed in a health-extended period life table. It is an aggregation over hypothetical individuals with different lifespans that are adjusted for the distribution of health at each age. Consequently, the welfare measure is sensitive to change in the distribution of health both at each age and over ages.[11]

Consider a health distribution-adjusted lifespan, y_e^d, that is defined such that if all individuals in the cohort were to experience it, social welfare would be the same as that generated by the unequal adjusted lifespans that emerge from the health-extended life table: $W\left(y_e^d\right) = W\left(F\left(h_s(x)\right), G(x)\right)$. Solving for this EDE produces a more general equivalent health-adjusted lifespan,[12]

$$EHAL = \left[\sum_{x>0}^{T} r(x) y^d(x)^{1-\eta}\right]^{\frac{1}{1-\eta}} \tag{6}$$

This measure adjusts life expectancy for a) mean health at each age, as $HALE$, b) health dispersion at each age, as $REHAL$, and c) dispersion in health-adjusted lifespans. It collapses to the standard $HALE$ when there is no aversion to health dispersion at each age and to dispersion in adjusted lifespans, $\varepsilon = \eta = 0$. With no aversion to lifespan dispersion ($\eta = 0$), it reduces to $REHAL$. With no aversion to age-specific health dispersion ($\varepsilon = 0$) but with aversion to dispersion in (mean) health-adjusted life years, it corresponds to a dis-

[11] Consider ages x and $x + k$, $k > 0$, with an equal proportion of deaths at these ages, $r(x) = r(x + k)$. Holding the distribution of deaths constant, let there be a rise in mean health at $x - 1$ and fall of sufficiently greater magnitude in mean health at $x + k - 1$ such that the health distribution-adjusted lifespans change by the same absolute amount, $\triangle y^d(x) = -\triangle y^d(x+k)$. There would be no change in $HALE$ or in $REHAL$. But, provided $\eta > 0$, $W\left(F\left(h_s(x)\right), G(x)\right)$ would increase, reflecting aversion to differences in health distribution-adjusted lifespans. The increase would occur irrespective of whether such differences arose through the length of life or through the age profile of the quality of life (health). Similarly, a fall in the variance of health at a younger age and a sufficiently greater rise in the variance at an older age (such that, $\triangle y^d(x) = -\triangle y^d(x+k)$) would increase welfare without any change in mean health at each age or in the distribution of deaths over ages.

[12] For $\eta = 1$, $EHAL = exp\left(\sum_{x>0}^{T} r(x)\, ln\, y^d(x)\right)$.

tributionally sensitive measure of population health that Norheim (2013) suggested but did not derive or estimate.

2.1.4 Properties

$EHAL$ is monotonically increasing in both unadjusted lifespan – as LE – and in health distribution-adjusted lifespan.[13] The measure will increase if mean health increases at least at one age and does not decrease at any age provided health dispersion is constant at all ages. It will also increase if dispersion falls at one or more ages, and does not rise at any age, provided $\varepsilon > 0$.

Aversion to age-specific health dispersion is one channel through which $EHAL$ prioritizes gains to the worst off. In this case, the worst off corresponds to the least healthy state at a given age. The second channel through which $EHAL$ captures prioritarian (Parfit, 2000) concerns for the worst off in health-lifespan space is the concave transformation (for $\eta > 0$) of health distribution-adjusted lifespans. In the calculation of $HALE$, an additional year of life in good health enjoyed at a younger age would be exactly offset by one less year of life in good health at an older age, leaving that measure unchanged. The same permutation would increase $EHAL$. In addition to reflecting prioritarian ethics, the measure captures the fair innings principle (Harris, 2006) of favouring health and longevity gains to the young over same sized gains to the old.[14]

Since there is greater variation in lifespans than there is in age-specific health, if η is set equal to ε, then $EHAL$ will be more sensitive to the distribution of lifespans than it is to the distributions of health.

[13] $\frac{\partial EHAL}{\partial y^d(x)} = EHAL^{\frac{-\eta}{1-\eta}} r(x) y^d(x)^{-\eta} > 0 \ \forall x$.

[14] The measure does not allow for a discrete change in prioritisation of younger lives on reaching a threshold health-adjusted lifespan, which is one version of the fair innings principle.

2.2 Distributionally sensitive valuation

We now derive a money metric valuation of potential or realised improvements in population health that can also be used to value the burden of disease. The metric is the willingness to pay (WTP) for change in $EHAL$. It inherits sensitivity to the distributions of age-specific health and lifespan from that measure.

Let $U\left(c(x), F\left(h_s(x)\right)\right)$ be the SDM's evaluation of the lifetime welfare generated by each life-table individual who lives to age x while enjoying a lifetime stream of consumption $c(x)$ and being exposed to age-specific health distributions $F\left(h_s(x)\right)$. For empirical tractability, we assume a constant flow of consumption irrespective of age and health: $c(x) = c \ \forall x$ (Bleichrodt & Quiggin, 1999). The cost of this assumption is that we do not capture WTP for any indirect benefits of health improvements that raise labour market productivity and so consumption. In the application, we set c to equal GDP per capita.

The SDM's lifetime welfare evaluation function $U()$ is assumed to be multiplicatively separable into an age-invariant function of consumption, $u(c)$, and welfare generated by the distributions of health up to the age of death, with the latter given by health distribution-adjusted lifespan: $U(c, F\left(h_s(x)\right)) = u(c)\sum_{i=0}^{x-1} h_e(i) = u(c)y^d(x)$. Multiplicative separability into welfare from consumption and health is a common restriction in derivations of WTP for health (Hammitt, 2013).[15]

We again use an Atkinson SWF, now to capture aversion to dispersion in lifetime welfare,

$$\begin{aligned} W\left(c, F\left(h_s(x)\right), G(x)\right) &= \sum_{x=0}^{T} r(x) \frac{U\left(c(x), F\left(h_s(x)\right)\right)^{1-\psi}}{1-\psi} \\ &= \frac{u(c)^{1-\psi}}{1-\psi} \sum_{x=0}^{T} r(x) y^d(x)^{1-\psi} \qquad \psi \neq 1, \end{aligned} \tag{7}$$

where $\psi \geq 0$ represents the degree of aversion to that dispersion.[16] Given that there is no

[15] The restriction is necessary for cost-effectiveness analysis (equivalently, the quality-adjusted life years model) to be consistent with cost-benefit analysis founded on willingness to pay (Bleichrodt & Quiggin, 1999).

[16] $W\left(c, F\left(h_s(x)\right), G(x)\right) = \sum_{x=0}^{T} r(x)\, ln\, U\left(c(x), F\left(h_s(x)\right)\right)$ for $\psi = 1$.

variation in consumption, it is reasonable to assume that aversion to dispersion in lifetime welfare is equal to aversion to dispersion in health distribution-adjusted lifespans, $\psi = \eta$.

Consider a change in population health comprising shifts in age-specific health distributions from $F\left(h_s(x)\right)$ to $F^*\left(h_s(x)\right)$ and a shift in the age-at-death distribution from $G(x)$ to $G^*(x)$. The WTP for such a combination of changes is defined implicitly by $W\left(c - WTP, F^*\left(h_s(x)\right), G^*(x)\right) = W\left(c, F\left(h_s(x)\right), G(x)\right)$.

To obtain a closed form solution, we assume that the SDM uses an iso-elastic function to evaluate the utility from consumption: $u(c)) = \left(c^{1-\gamma} - \underline{c}^{1-\gamma}\right) / (1-\gamma)$, where $\gamma \geq 0$ ($\gamma \neq 1$) (Murphy & Topel, 2006).[17] The parameter $\underline{c}$ is a subsistence level of consumption at which there is indifference between life in full health and death (Hall & Jones, 2007; Rosen, 1988). Larger values of $\underline{c}$ imply a lower value of being alive relative to dead. This parameter arises from the normalisation of utility when dead to zero. At a higher level of consumption, and so lower marginal utility of consumption (with $\gamma > 0$), a marginal extension to lifespan is worth more because it increases lifetime utility by more than can be achieved through an increase in consumption (Hall & Jones, 2007). This effect is stronger at a larger value of γ since the marginal utility of consumption declines more steeply with rising consumption.[18]

Assuming $\psi = \eta$, we obtain

$$WTP = c - \left[\frac{EHAL}{EHAL^*}\left(c^{1-\gamma} - \underline{c}^{1-\gamma}\right) + \underline{c}^{1-\gamma}\right]^{\frac{1}{1-\gamma}} \tag{8}$$

where $EHAL$ and $EHAL^*$ are obtained from eq.(6) applied to the distributions $F\left(h_s(x)\right)$ and $G(x)$ and to $F^*\left(h_s(x)\right)$ and $G^*(x)$, respectively (see Appendix A for derivation).

WTP for a change in population health captured by the ratio of $EHAL$ measures depends on three preference parameters that reflect aversion to dispersion in age-specific health (ε), aversion to dispersion in lifespan (η), and the curvature of consumption utility (γ), as well

[17] $u(c) = ln\, c$ for $\gamma = 1$.

[18] Through this mechanism, an anticipated rise in future consumption due to economic growth would raise the value of any extension to lifespan (Ponthiere, 2011). Since we hold the level of consumption constant, we will miss this effect and so underestimate the welfare gain from increased longevity.

as on the level of consumption in relation to subsistence (c and $\underline{c}$).

Aversion to lifespan dispersion ($\eta > 0$) ensures that WTP is positive for any change in the distribution of deaths that would extend lifespan before death at a younger age and reduce lifespan by the same amount before death at an older age, leaving average lifespan constant. A larger value of η will raise the WTP for any such reduction in the dispersion of lifespan. Similarly, with $\varepsilon > 0$, there is positive WTP for a reduction in health dispersion at any age with all else held constant. And for any given reduction in age-specific health dispersion, WTP is rising with the value of ε.

If there were no aversion to dispersion in both health and lifespan ($\varepsilon = \eta = 0$), then eq.(8) would reduce to the WTP for a change in health-adjusted life expectancy:

$$WTP = c - \left[\left(\frac{HALE}{HALE^*}\right)\left(c^{1-\gamma} - \underline{c}^{1-\gamma}\right) + \underline{c}^{1-\gamma}\right]^{\frac{1}{1-\gamma}} \tag{9}$$

where $HALE$ and $HALE^*$ are obtained from (1) applied to the respective health and age-at-death distributions. This solution is similar to the valuation of changes in lifespan proposed by Becker et al. (2005).

Positive dependence of WTP on the level of consumption arises through three channels. First, with concavity of the consumption component of utility ($\gamma > 0$), the opportunity cost of a marginal dollar of consumption that is forgone to improve (distributions of) health and lifespan is lower at a higher level of consumption. At any given level of consumption, higher γ implies greater WTP because the opportunity cost of investing in health and lifespan is also lower. Second, diminishing marginal utility of consumption also means that at a higher level of consumption the gain in lifetime utility that can be achieved through living longer rises relative to the respective gain obtainable through increased consumption. Third, multiplicative separability of welfare in consumption and health implies that the marginal welfare gain from health is increasing with consumption. So, the marginal benefit of paying more for health is higher, while the marginal (opportunity) cost is lower.

Positive dependence of WTP on the level of consumption does not mean that improve-

ments in the health of populations of poorer countries are less valuable from a global perspective. Rather, it reflects the high opportunity cost of resources spent on health in those countries. If external sources of health financing were available, then c could be set above GDP per capita and this would increase the WTP for any change in population health. Setting a higher level of subsistence consumption (relative to the mean) would reduce WTP since there would then be less available to spend on health after reaching the level of consumption at which life (even in full health) is considered to be no better than death.

3 Data and method

3.1 Global Burden of Disease data

We use the measures introduced in the previous section to quantify population health and the burden of disease in Sub-Saharan Africa (SSA) in 1990, 2004, and 2019. All data are from the 2019 Global Burden of Disease (GBD) that provides estimates for 293 disease causes disaggregated by sex and age (GBDCN, 2021; Murray et al., 1996). There are data on all 46 countries in the SSA region, including those established between 1990 and 2019. We use aggregated data for SSA in order to assess trends in population health across the region as a whole. For each sex, age, and disease partition, we observe disease prevalence and mortality rates, and disability adjusted life years (DALYs). We stratify all analyses and measures by sex.

3.2 Health distributions

Calculation of the measures given by equations (1), (4), and (6) requires a distribution of health at each age. From the disease-level GBD data, we generate an individual-level dataset in which diseases are assigned to simulated individuals on the basis of age-specific prevalence rates. Within each of 21 age partitions, we simulate 100,000 individuals and use

prevalence rates for 293 causes of disease to randomly assign diseases to simulants. Within each partition, we follow the GBD in assuming independence between diseases — being assigned disease k does not change the probability of being assigned disease j. There is some allowance for comorbidities by defining disease combinations, e.g. HIV and tuberculosis, as a separate disease with its own prevalence. Further, the probability of comorbidities arising by chance varies with disease prevalence rates across age partitions. But given the absence of more explicit allowance for comorbidities in the GBD, our approach should be considered a first order approximation of the true distribution of health outcomes at each age.

We use the data on disease prevalence and DALYs to recover disease-specific disability weights ($z_k \in [0, 1]$) (see Appendix B). Each weight is intended to represent the proportionate health loss (severity) associated with a disease. It is not a preference for avoiding that disease compared with any other (Hausman, 2012). A weight of 0 indicates full health, while 1 is equivalent to death. We reverse the scale to get a measure of health for each disease.

The health of a simulant that is assigned a set K of diseases is $h_s = \prod_{k \in K}(1 - z_k)$, So, a health state is defined by a set of diseases, not a particular disease. At each age, a proportion p_s of the 100,000 individuals we simulate shares the same combination of diseases. Together, h_s and p_s $\forall s$ define the distribution of health at each age, $F(h_s(x))$, that is used in the analysis.[19]

To measure the burden of disease k, we construct counterfactual distributions of health that would emerge from elimination of that disease by setting $z_k = 0$ while holding constant z_j $\forall j \neq k$. We denote these counterfactual distributions as $F^*(h_s(x))$.[20]

[19] Appendix B Figure B1 shows that the simulated health distribution has a lower mean and is more dispersed for older compared with younger age groups.

[20] One could construct a counterfactual for the elimination of a group of diseases, which require assuming independence only between groups, and not within them.

3.3 Age-at-death distributions

For each sex, we construct an age distribution of deaths in a life-table cohort of size ($l_0 =$) 100,000. We use age-specific all-cause mortality rates (${}_nM_x$) provided by the GBD for 21 age intervals, $[x, x+n)$. We first estimate the probability of death in each age interval conditional on survival to the exact age that defines its start. Iterative application of these conditional probabilities to the life table cohort gives the number deaths in each age interval and so an estimate of the age-at-death distribution, $G(x)$, that is used in the analysis (see Appendix C for details).

To measure the burden of a disease, we simulate a counterfactual age-at-death distribution if it were eliminated. The GBD all-cause mortality rate is a sum of disease specific mortality rates, and so the counterfactual age-specific mortality rate after elimination of disease k is ${}_nM_x^{-k}={}_nM_x-{}_nM_x^k$. We estimate these counterfactual mortality rates from GBD estimates of disease-specific mortality rates for each age interval. We then transform them into corresponding counterfactual conditional probabilities of death in each interval and, again, apply these iteratively to get the counterfactual number of deaths in each interval (see Appendix C). This gives a counterfactual age-at-death distribution, which we refer to generically as $G^*(x)$.

We use these counterfactual age-at-death distributions and the respective counterfactual age-specific health distributions to calculate the impact that elimination of a disease would have on the population health measures. And we estimate the willingness to pay to eliminate that disease. In common with other studies that simulate contributions of diseases to population health, we assume that if one set of cause-specific mortality rates were set to zero, the corresponding rates from other causes would remain unchanged. This could bias estimates downward if a disease increases mortality from other causes. On the other hand, if deaths from a disease that is eliminated would occur at the same age due to some other cause in any case, then our estimates will be biased in the other direction. Given these two

offsetting potential biases, the approach can be considered a first order approximation of the true distribution of deaths that would be observed if a disease were eliminated.

3.4 Parameter values

Most of the evidence on aversion to dispersion in health and lifespan is from experiments in which participants choose between distributions of life years or health-adjusted life years (McNamara et al., 2020). This evidence is most informative for choice of the value of η. There is less evidence on aversion to dispersion in age-specific health to guide the setting of ε.[21]

Some studies that elicit aversion to dispersion in health-adjusted life years find extremely high estimates of η, reaching an implausible 28 (Dolan & Tsuchiya, 2011; McNamara et al., 2020; Robson et al., 2017). However, these studies are designed to estimate aversion to socioeconomic differences in life years, which tends to be stronger than aversion to all differences in life years (Hardardottir et al., 2021; Hurley et al., 2020; McNamara et al., 2021). Attempts to elicit the latter type of aversion can still produce incredibly high estimates of η in the range of 5.8-7.6 (Edlin et al., 2012; McNamara et al., 2020; McNamara et al., 2021). Using a representative sample in Ontario, Hurley et al. (2020) elicit aversion to univariate dispersion in health-adjusted life years and find a median value of $\hat{\eta}$ of around 1.0-1.5, with substantial heterogeneity: $\hat{\eta} < 1$ for around a half of the sample and $\hat{\eta} > 3$ for most of the other half. A study using a representative sample of the UK population obtains a median estimate of $\hat{\eta} = 3.2$ and a pooled estimate of $\hat{\eta} = 1.4$ (Robson et al., 2023).[22]

The United Nations uses $\eta = 1$ to capture sensitivity to lifespan dispersion in its inequality-adjusted Human Development Index (UNDP, 2020), which lends this parame-

[21] Attema et al. (2015) elicit social preferences over distributions of health (quality of life) at each of a number of ages but do not estimate ε.

[22] Studies that elicit personal preferences over individual health or lifespan risk under the assumption of iso-elastic utility (Delprat et al., 2016; Herrera-Araujo et al., 2020) tend to find aversion parameter estimates that are smaller than the analogous estimates of (iso-elastic) social preferences over population distributions of health or lifespan, which are relevant here.

ter value some legitimacy as a benchmark. For our main estimates, we set each of η and ε to 1. We test sensitivity to setting each parameter to 2.

To calculate WTP, we assume that aversion to dispersion in lifetime utility and in lifespan are equal, $\psi = \eta$. We are not aware of any preference elicitation studies that distinguish between these two parameters. The curvature of consumption utility parameter, γ, can be inferred from either the inter-temporal elasticity of substitution (IES) or constant relative risk aversion (CRRA) for consumption. Most estimates of IES range from around 0.5 to just above 1 (Browning et al., 1999; Hall, 1988; Havranek et al., 2015), implying values of γ roughly between 1 and 2. There is some evidence that IES is smaller (γ larger) in low-income countries (Atkeson & Ogaki, 1996; Havranek et al., 2015; Ogaki et al., 1996). Direct estimates of CRRA are more variable. Several studies also find values ranging from 1 to 2 (D. Meyer and J. Meyer 2005), while some report estimates as large as 10. Indirect estimates obtained from the wage elasticity of labour supply are below 2 (Chetty, 2006). We follow Murphy and Topel (2006) in setting $\gamma = 1.25$.

We set the level of consumption (c) to GDP per capita for SSA in the respective year (World Bank, 2022). We are not aware of any direct evidence on a level of subsistence consumption that would leave someone indifferent between life in full health and death ($\underline{c}$). While the value of this parameter is often inferred from estimates of the value of a statistical life (VSL) (Hall & Jones, 2007; Jones & Klenow, 2016; Murphy & Topel, 2006), there are few reliable VSL estimates for SSA. Some studies set $\underline{c}$ to zero (Crafts & Haacker, n.d.; Murphy & Topel, 2003; Usher, 1973) and so assume that any life is worth living regardless of the level of consumption achieved. Other studies opt for a value close to the international extreme poverty line (Becker et al., 2005; Soares, 2007) which implies that survival at consumption below $2.15 per day amounts to a life that is not worth living (Cookson et al., 2021). Following Murphy and Topel (2006) again, we set $\underline{c}$ at 10% of GDP per capita.

4 Results

4.1 Population health

Table 1 shows population health measures for SSA in 1990, 2004, and 2019. Between 1990 and 2004, when the HIV epidemic ravaged the region, life expectancy (LE) was stagnant for females and increased only marginally for males. Between 2004 and 2019, when there was rapid expansion of antiretroviral therapy coverage, LE increased by about ten years for both sexes. The $HALE$ estimates indicate that the average female (male) born in SSA in 2019 could expect to live for the equivalent of 62.3 (58.5) years in full health. In the 2004-2019 period, absolute and relative increases in $HALE$ were even steeper than those in LE. Improvements in quality of life added to increases in length of life. If instead of adjusting LE for the arithmetic mean of health at each age to get $HALE$ we adjust for the respective geometric means ($\varepsilon = 1$), then the resulting $REHAL$ estimates are 0.23-0.34 years in full health below the respective $HALE$ values. The relative improvements between 1990 and 2019 in population health measured by $REHAL$ are similar to those obtained with the distributionally insensitive $HALE$ measure.

Table 1: Population health measures, Sub-Saharan Africa

	1990	**2004**	**2019**	**2019-1990**	
				Δ	$\%\Delta$
Female					
LE	55.76	55.89	66.66	10.90	19.6%
HALE	48.57	48.76	62.28	13.70	28.2%
REHAL	48.25	48.42	62.05	13.80	28.6%
EHAL	27.16	30.30	45.63	18.47	68.0%
Male					
LE	51.41	52.86	62.13	10.71	20.8%
HALE	45.82	47.14	58.47	12.65	27.6%
REHAL	45.54	46.84	58.27	12.73	28.0%
EHAL	23.86	27.80	40.85	16.99	71.2%

Note: *LE*=Life Expectancy, *HALE*=Health-Adjusted Life Expectancy, eq.(1), *REHAL*=Restricted Equivalent Health-Adjusted Lifespan, eq.(4) with $\varepsilon = 1$, *EHAL*=Equivalent Health-Adjusted Lifespan, eq.(6) with $\varepsilon = \eta = 1$. See Appendix D, Table D1 for measures at other parameter values.

Adjustment for dispersion in lifespan has a much greater impact on the changes in population health. With aversion to lifespan and age-specific health dispersion both set to one ($\eta = \varepsilon = 1$), the distributionally sensitive $EHAL$ measure increased by more than two thirds between 1990 and 2019 for both sexes. This is substantially larger than the increases in the distributionally insensitive measures because gains in LE and $HALE$ were largely driven by reductions in both infant mortality and, in the latter period, HIV-related mortality among younger adults, which reduced dispersion in the age-at-death distribution.

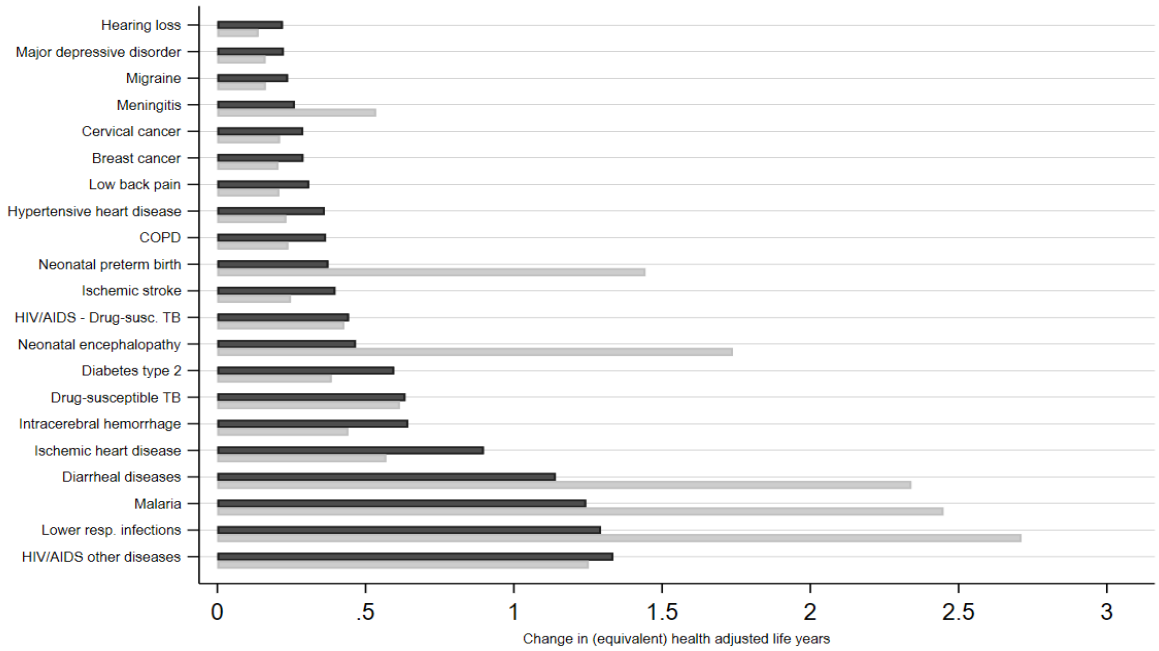
Raising the degree of aversion to dispersion in age-specific health has relatively little impact on the measures, while raising aversion to lifespan dispersion dramatically reduces the magnitude of $EHAL$ because more weight is placed on infant deaths (Appendix D Table D1). Introducing aversion to lifespan dispersion before aversion to age-specific health dispersion does not change the finding that the measure is more sensitive to the former (Appendix D Table D1).

4.2 Disease burdens

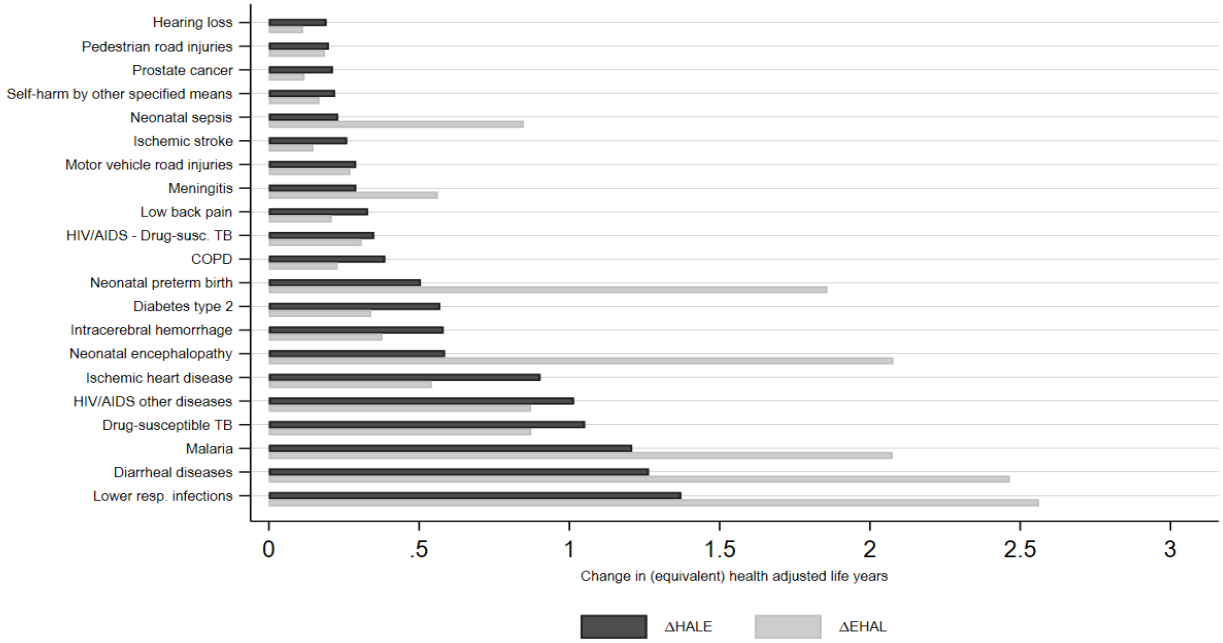
We now examine the impact of distributional sensitivity on measures of disease burden by comparing the simulated increase in $HALE$ that would occur if a disease were eliminated with the respective simulated increase in $EHAL$. That is, we compare $\Delta HALE = HALE^* - HALE$ with $\Delta EHAL = EHAL^* - EHAL$, where $HALE^*$ and $EHAL^*$ are calculated from equations (1) and (6), respectively, using the counterfactual distributions of health, $F^*(h_s(x))$, and age-at-death, $G^*(x)$, that would be achieved if a disease were eliminated. Figure 1 shows 2019 estimates for the 20 diseases with the largest burdens measured by $\Delta HALE$. The lighter shaded bars show the respective disease burdens measured by $\Delta EHAL$ that are estimated with both dispersion aversion parameters set to 1.[23]

For both females and males, the largest differences between the measures of disease burden are for diseases that primarily affect neonates, infants, and young children: lower

[23]See Appendix D Figures D1 and D2 for the disease burdens in 1990 and 2004, respectively.



(a) Female



(b) Male

Figure 1: Top 20 disease burdens with and without distributional sensitivity, 2019

Notes: $\Delta HALE = HALE^* - HALE$, where each variable is obtained from eq. (1) applied to the observed distributions $F(h_s(x))$ and $G(x)$ for $HALE$ and the counterfactual distributions $F^*(h_s(x))$ and $G^*(x)$ obtained if a disease were eliminated for $HALE^*$. $\Delta EHAL = EHAL^* - EHAL$, where each variable is obtained from eq. (6) with $\varepsilon = \eta = 1$ and application is to the observed or counterfactual distribution. 20 diseases/conditions with largest burdens measured by $\Delta HALE$.

respiratory infections (LRI), diarrhea, malaria, neonatal encephalopathy, neonatal preterm birth, and protein energy malnutrition. If these conditions were eliminated, it would increase expected lifespan in good health and reduce lifespan dispersion. Consequently, these conditions impact the distributionally sensitive $EHAL$ by substantially more than they impact $HALE$.

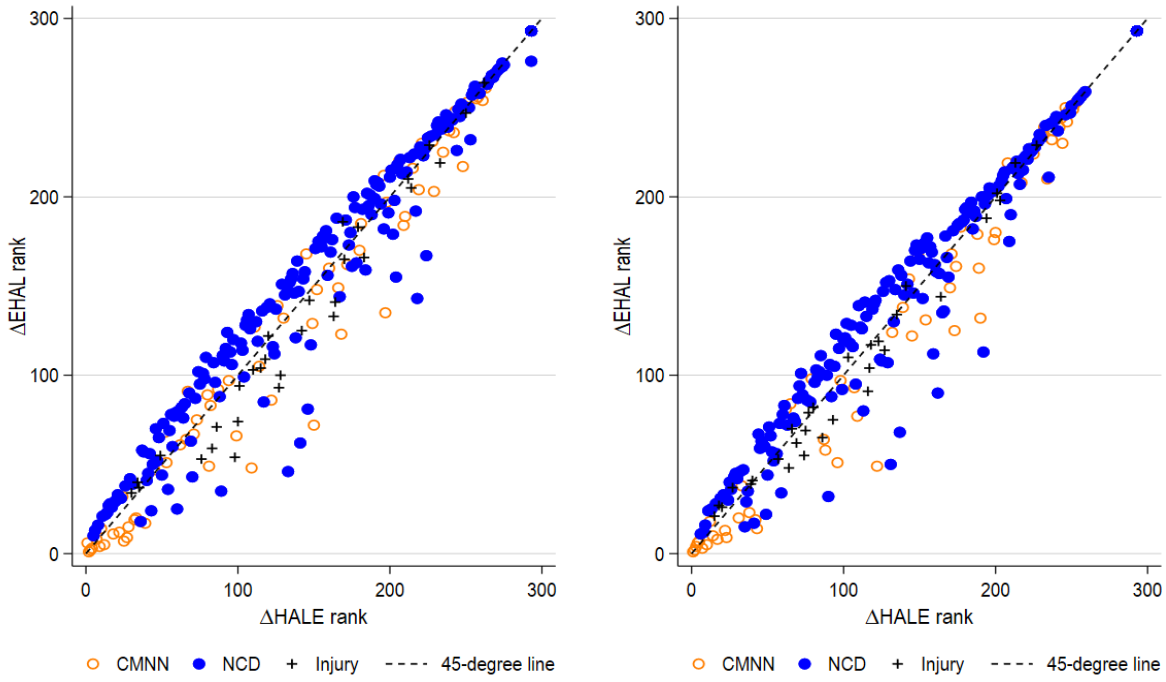
Distributional sensitivity has the opposite effect on the estimated disease burdens of non-communicable diseases (NCDs) and chronic conditions that have greater prevalence at older ages. For example, elimination of each of breast cancer, alzheimer's disease, hearing loss, ischemic stroke and heart disease, depression, chronic obstructive pulmonary disease (COPD), back pain, and diabetes, would increase $EHAL$ by less than it would increase $HALE$. This is because the consequent improvements in health and extensions of life at older ages would increase dispersion in health-adjusted lifespans.

Figure 2 extends the analysis to all 293 diseases. The x-axis shows disease ranks by burden measured with $\Delta HALE$. A rank of 1 indicates the disease with the largest burden. The y-axis shows ranks by $\Delta EHAL$, with $\varepsilon = \eta = 1$. Each mark represents a disease. Colours/symbols distinguish between communicable, maternal, neonatal, and nutritional diseases (CMNNs), NCDs, and injuries. Many CMNNs lie below the 45 degree line, indicating that their burdens are relatively larger using the distributionally sensitive $EHAL$ measure.[24] This is because these conditions disproportionately impact at younger ages. Adjusting for impacts on health and lifespan dispersion moves many NCDs in the opposite direction. Their disease burdens rank lower (further from 1) with $EHAL$ because this measures penalizes the increase in disparity in health-adjusted lifespan that would result from eliminating diseases that are more prevalent at older ages.[25] Discrepancy between the disease burden ranks produced by the two measures increases substantially when the dispersion aversion parameters are increased to 2 (Appendix D, Figure D3). For females, Kendall's rank correlation coef-

[24]For females, out of 77 CMNNs, 43, 25, and 9 are below, above, and on the diagonal respectively. For males, the respective numbers are 40, 16, and 25.

[25]For females, out of 186 NCDs, 40, 128, and 18 are below, above, and on the diagonal. For males, the respective numbers are 39, 119, and 28.

ficient for disease burdens measured by $\Delta HALE$ and $\Delta EHAL$ falls from 0.88 (p-value $<$ 0.01) with $\varepsilon = \eta = 1$ to 0.59 (p-value $<$ 0.01) with $\varepsilon = \eta = 2$. For males, there is a similar decrease (0.89 to 0.65).



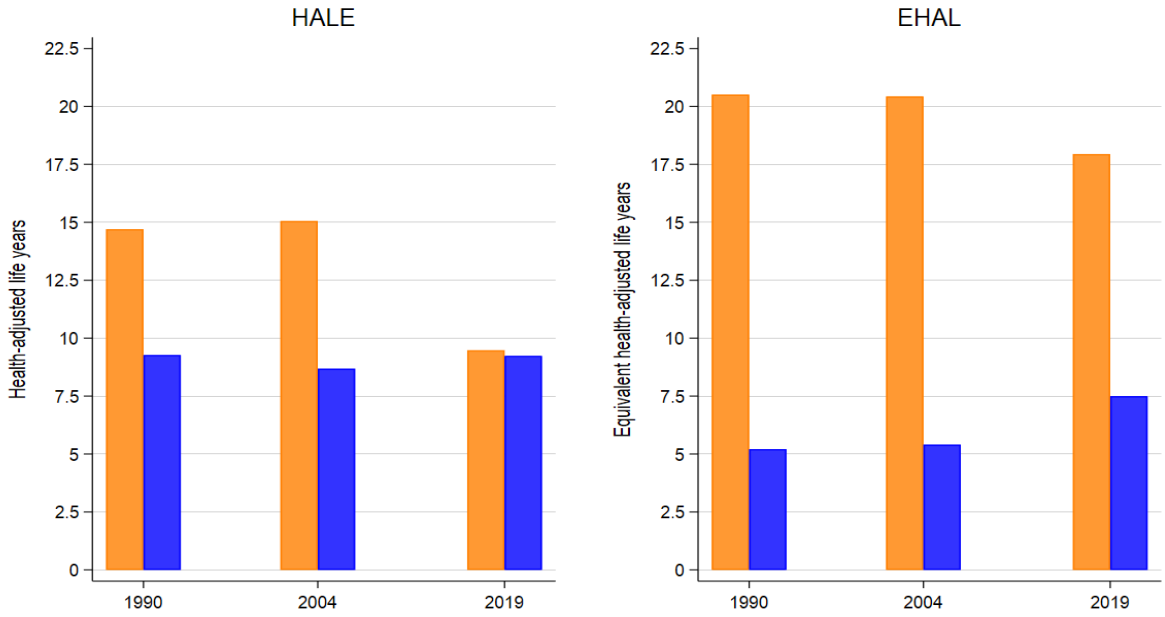
a) Female b) Male

Figure 2: Disease burden ranks with distributionally sensitive and insensitive measures, 2019

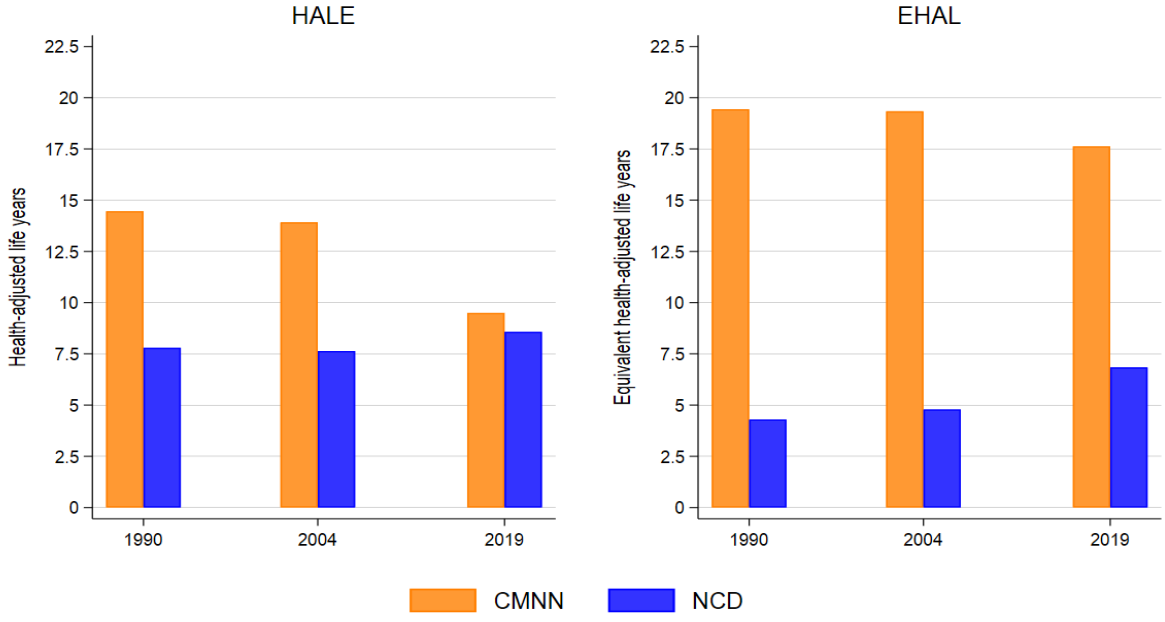
Notes: x-axis and y-axis show disease burden ranks by $\Delta HALE$ and $\Delta EHAL$, respectively. See notes to Figure 1 for definitions. Lower number indicates higher disease burden rank.

The left column of Figure 3 shows total burdens of all CMNNs and all NCDs measured by $\Delta HALE$. In 1990, females and males in SSA lived in full health for about 14 years less because of CMNNs. The respective burden of NCDs in 1990 was around 8-9 health-adjusted life years. By 2019, the the CMNN burden had fallen substantially for both sexes, with most of the decrease occurring between 2004 and 2019. Over the 1990-2019 period, the NCD burden remained roughly constant for females and increased for males. As a result, there was little difference between the CMNN and NCD burdens in 2019.

The right column of the figure shows CMNN and NCD burdens measured by $\Delta EHAL$ with $(\varepsilon = \eta = 1)$. For both sexes, allowing for distributional sensitivity increases the



(a) Female



(b) Male

Figure 3: Communicable and non-communicable disease burdens with and without distributional sensitivity

Notes: Top panel shows $\Delta HALE$ for counterfactual in which all communicable, maternal, neonatal, and nutritional diseases (CMNNs) or all non-communicable diseases (NCDs) are eliminated. Bottom panel shows $\Delta EHAL$, with $\varepsilon = 1, \eta = 1$, for same counterfactuals.

magnitude of the CMNN burden and slows its decline between 2004 and 2019 substantially. The NCD burdens are smaller with distributional sensitivity because these diseases constrain dispersion in health-adjusted lifespans, as well as reducing the mean. While for both sexes the distributionally sensitive NCD burden increases, mainly between 2004 and 2019, it remains a long way short of the respective CMNN burden in 2019. The previously claimed shifting burden of disease from CMNNs to NCDs in SSA (Gouda et al., 2019) is much less evident when the impact of each disease group on the dispersion of health-adjusted life years is taken into account.

4.3 Valuation of disease burden and health change

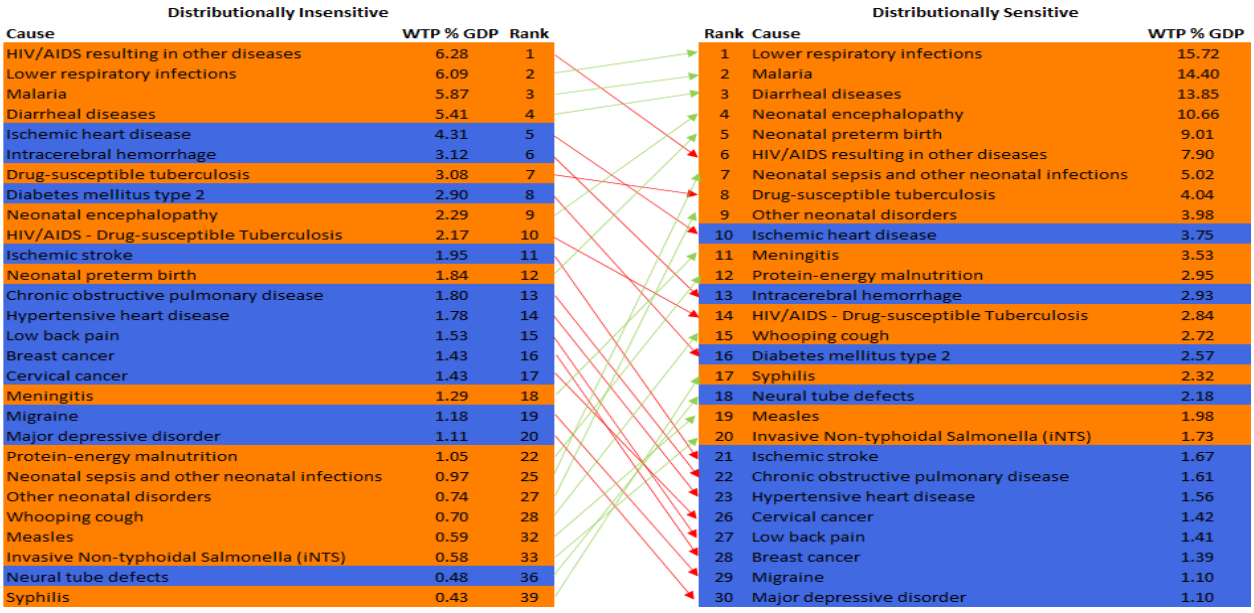
Figure 4 shows, for 2019, the extent to which distributional sensitivity affects disease burdens expressed in monetary values. The left panel shows estimates obtained without aversion to health and lifespan dispersion. Each value is a per person WTP to eliminate a disease as a percentage of GDP per capita. It is calculated using eq.(9), where $HALE^*$ is obtained from the counterfactual health and age-at-death distributions that would emerge if there were no disability or mortality from that disease. This WTP can be interpreted as the monetary equivalent of the welfare cost imposed by a disease. Diseases are ranked from top to bottom in decreasing WTP. We show the 20 diseases that impose the largest welfare cost, plus a few others that are in the top 20 using the distributionally sensitive valuation shown in the right panel.[26] Colours again distinguish between CMNNs, NCDs, and injuries.

For females, we estimate that HIV/AIDS imposed the largest per capita welfare cost, equivalent to 6.3% GDP per capita. LRI is a close second (6.1% GDP). For males, the top two are LRI (6.8% GDP) and diarrheal diseases (6.3% GDP).[27]

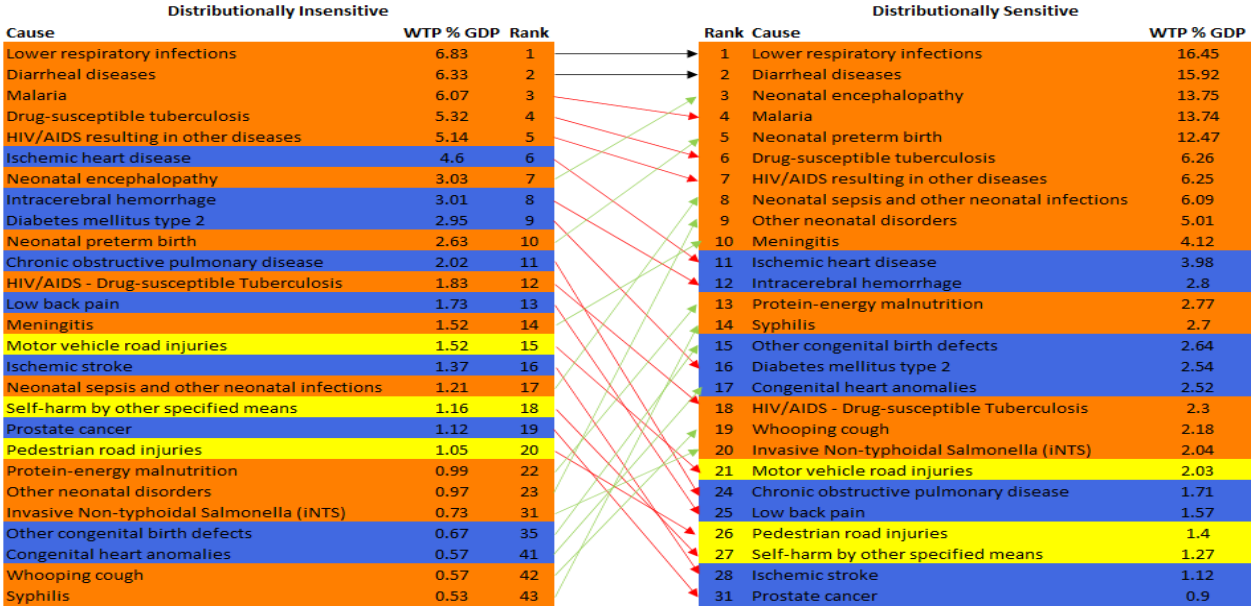
The right panel of Figure 4 shows the value of the welfare cost of each disease with

[26]The top 20 diseases by WTP calculated from eq.(9) are necessarily the same as the top 20 by the $\Delta HALE$ measure.

[27]We estimate WTP per capita as a % of GDP per capita. Hence, the total welfare cost is a weighted average of the female and male estimates, not the sum of the two.



(a) Female



(b) Male

Figure 4: Welfare costs of diseases with and without distributional sensitivity, 2019

Notes: orange = CMNN, blue = NCD, yellow = Injury. Welfare cost is WTP to eliminate disease calculated from equations (9) and (8) ($\varepsilon = \eta = 1$) to give distributionally insensitive and sensitive valuations, respectively. In each case, $\gamma = 1.25$, c =GDP per capita, and $\underline{c} = 10\%$ of GDP per capita. Left panel shows the 20 diseases with the largest welfare costs using the distributionally insensitive valuation, plus those that move into the top 20 using the distributionally sensitive valuation.

allowance for distributional sensitivity. These values are obtained from eq.(8) with $\varepsilon = \eta = 1$.[28] Adjustment for aversion to health and lifespan dispersion generally increases the welfare cost of CMNNs. Among females, the per capita welfare cost of LRI increases from 6.1% to 15.7% of GDP per capita. For each of diarrheal diseases and malaria, distributional sensitivity increases the welfare cost by about 8 percentage points of GDP. There is a more modest impact on the welfare cost of HIV/AIDS because mortality from this disease peaks at an older age than mortality from LRI, diarrhea, and malaria, which are largely childhood illnesses.

Distributional sensitivity has less impact on the monetary values of NCD and injury burdens, and even reduces some of these burdens for conditions that are more prevalent at older ages. For example, among females, the welfare cost of ischemic heart disease falls from 4.3% to 3.8% of GDP because this disease mostly affects those who already enjoy more than the average equivalent life years in full health. WTP to eliminate the disease is reduced because doing so would increase dispersion in the distribution of health-adjusted lifespan. The welfare cost of low back pain is approximately the same (≈1.4-1.5% GDP for females and ≈1.6-1.7% GDP for males) with and without distributional sensitivity.

We use eq.(8) and eq.(9) to obtain distributionally sensitive and insensitive, respectively, valuations of welfare gains generated by changes in population health. For example, to get the distributionally sensitive valuation of the change between 1990 and 2019, we use eq.(8) with $EHAL$ equal to the 1990 value and $EHAL^*$ equal to the 2019 value. Table 2 shows the estimates for all pairwise comparisons of years. Without allowing for distributional sensitivity, the change in female population health between 1990 and 2004 reflected in the $HALE$ measure is valued at the equivalent of only 1.2% of 1990 GDP. The respective valuation of the change that occurred between 2004 and 2019 is an estimated welfare gain of 46.4% of baseline GDP. The steep increase in the welfare gain from improved health is also seen for males. In part, it is due to the progress made in reducing mortality from HIV in the

[28] See Appendix D, Figure D4 for estimates with $\varepsilon = \eta = 2$.

latter period. For both females and males, the value of the welfare gain from improvements in health between 1990 and 2019 is larger by about 20 percentage points of baseline GDP using the distributionally sensitive valuations. This is due to the greater weight placed on the disproportionate reductions in CMNN-related mortality at younger ages.

Table 2: Welfare gains from population health changes, % of baseline GDP

	1990-2004	**2004-2019**	**1990-2019**
Female			
Distributionally insensitive	1.20%	46.44%	46.88%
Distributionally sensitive	26.67%	60.51%	66.56%
Male			
Distributionally insensitive	8.26%	42.99%	46.34%
Distributionally sensitive	34.20%	58.86%	67.43%

Note: Distributionally insensitive estimates from eq.(9) with $HALE$ and $HALE^*$ given by eq.(1) applied to the baseline and endline years, respectively. Distributionally sensitive estimates from eq.(8) with $EHAL$ and $EHAL^*$ given by eq.(6) applied to the baseline and endline years, respectively. Distributionally sensitive estimates use $\varepsilon = \eta = 1$. Valuations expressed at % of GDP in the baseline year.

5 Discussion

Our measure allows monitoring of trends in population health that takes into account changes in dispersion of both age-specific health and health-adjusted lifespan and yet does not require more data than current measures. It offers the opportunity to evaluate and compare disease burdens while paying attention to impacts on health and lifespan dispersion. This potential for distributionally sensitive measurement and valuation of population health can be used to better inform social decision makers seeking to narrow health and lifespan differences, and not only to improve average outcomes.

Application to the burden of disease in Sub-Saharan Africa reveals that an apparent convergence of the burdens of communicable and non-communicable diseases is not robust to distributionally sensitive measurement. While measures of disease burden do not identify potential effects of feasible policies, they do inform deliberations that lead to the setting of healthcare priorities. The measure used could influence the prioritisation of programmes.

We find that while allowing for aversion to dispersion in health-adjusted lifespan has a large effect on population health trends in SSA, there is less sensitivity to age-specific health dispersion. One explanation would be that a relatively small proportion of the SSA population survives to old ages at which health is most dispersed. Inconsistent with this hypothesis, we found similar insensitivity to age-specific health dispersion when we applied mortality rates of high-income countries to the SSA data. An alternative explanation is that there is much less dispersion in age-specific health than there is in lifespan. With iso-elastic social preferences, it may be that a larger parameter value is required to capture aversion to the more limited dispersion in age-specific health.

Interesting potential applications of our measure are not limited to low-income, high-mortality populations. For example, in the years immediately preceding the COVID-19 pandemic, life expectancy was stagnant in the United States as a result of falling older-age mortality offsetting rising mortality among younger and middle-age adults (Acciai & Firebaugh, 2017; Case & Deaton, 2017, 2020; Harper et al., 2021; Woolf & Schoomaker, 2019). By penalizing life expectancy for the increase in lifespan dispersion, our measure would reveal the full extent of deterioration in population health in this period. Since mortality from COVID-19 has been much higher at older ages, it has reduced lifespan dispersion and our measure would show a more muted negative impact than is indicated by the change in (health-adjusted) on life expectancy.

Our method of aggregation is normatively founded on the principle that an additional health-adjusted life year is of greater social value when it extends a shorter (health-adjusted) life. Not everyone will agree with this ethic. Initially, the GBD explicitly age-weighted health-adjusted life years, giving the lowest weights in infancy and the highest weight at age 25 (Murray et al., 1996; World Bank, 1993). This non-monotonic age-weighting was intended to indirectly allow for the instrumental value of health through its consequences for the well-being of dependents (Murray, 1994). It was discontinued after criticism of its logical and ethical justification (Anand & Hanson, 1997; Bognar, 2008; Brock, 2004; Broome, 2002). We

do not explicitly weight on age. Rather, we take a concave aggregation over (simulated) lives that differ in quantity and quality. Concavity ensures that the loss of a health-adjusted life year from a shorter life counts more than the same loss from a longer life. Rather than using age as a proxy for an equity-relevant characteristic (Bognar, 2008), our approach directly captures ethical concerns that may motivate age weighting. One is the prioritarian concern for the worst off with the least health-adjusted life years (Adler et al., 2021; Parfit, 2000). Another is that justice is served by giving first to those who are in greatest need in the sense of having had least of a good (Kamm, 2002) — again, those with fewest health-adjusted life years. Coherent objections to the ethical foundations of our approach can certainly be made (Broome, 2002). But this is true of any population health measure that is interpreted other than strictly descriptively. Aggregation involves assigning different degrees of importance to different aspects of a distribution.

To ensure that our approach is feasible whenever standard population health measures are used, we constrained the measure to require no more data than those in a health-extended period life table. Hence, the measure summarises distributions of morbidity and mortality for a hypothetical cohort. It does not capture the morbidity and mortality currently living individuals were exposed to when younger and will experience when (and if) older. Nor does it take account of correlation between health states at different ages and between health and lifespan. Clearly, these are limitations. But they are inherited from the population health measures — LE, $HALE$, and $DALYs$ — that we extend by adding distributional sensitivity. If a cohort of individuals could be followed from birth until death, then it would be possible to measure and value cohort health allowing for aversion to dispersion in observed lifetime health profiles. One could simulate these profiles if estimates of disease-specific illness duration and correlation between health and lifespan were available. With information on the correlation structure at some aggregate level, copulas might be used to estimate joint distributions of health and lifespan at each age from the marginal distributions (Wu et al., 2014).

Aggregation over cohorts would raise methodological and ethical issues. Taking a weighted average of the health of age groups, with each group weighted by its population size, would conflate demographic and mortality processes. For instance, more weight would be placed on younger age groups in countries with higher fertility rates. Yet fertility, and so the population age structure, is endogenous to longevity (Wolpin, 1997). By limiting attention to a single (albeit hypothetical) cohort of fixed size, we avoid the question of how to value health-induced change in population size and sidestep Parfit's (1984) repugnant conclusion.

Unlike other attempts to value changes in population health (Hall & Jones, 2007; Murphy & Topel, 2006), our approach allows for aversion to dispersion in health and lifespan. But it does not allow for the instrumental value of health in raising human capital and earning capacity. It misses, for example, the negative impact that HIV/AIDS had on education (Baranov & Kohler, 2018) and income (McDonald & Roberts, 2006; Tompsett, 2020) in SSA as well as the positive effects that antiretroviral therapy had on these outcomes (Da Costa, 2023). Jones and Klenow (2016) measure country well-being as a function of levels of life expectancy, consumption, and leisure as well as inequalities in the latter two dimensions. Their approach could possibly be generalised to allow for aversion to inequalities health status and lifespan.

References

Acciai, F., & Firebaugh, G. (2017). Why did life expectancy decline in the United States in 2015? A gender-specific analysis. *Social Science & Medicine*, *190*, 174–180. https://doi.org/https://doi.org/10.1016/j.socscimed.2017.08.004

Adler, M. D., Ferranna, M., Hammitt, J. K., & Treich, N. (2021). Fair innings? The utilitarian and prioritarian value of risk reduction over a whole lifetime. *Journal of Health Economics*, *75*, 102412.

Alkire, S., & Foster, J. E. (2010). *Designing the inequality-adjusted Human Development Index* (Working Paper No. 2010/28). United Nations Development Programme. https://hdr.undp.org/system/files/documents/hdrp201028pdf.pdf

Anand, S., & Hanson, K. (1997). Disability-adjusted life years: A critical review. *Journal of Health Economics*, *16*(6), 685–702.

Atkeson, A., & Ogaki, M. (1996). Wealth-varying intertemporal elasticities of substitution: Evidence from panel and aggregate data. *Journal of Monetary Economics*, *38*(3), 507–534.

Atkinson, A. B. (1970). On the measurement of inequality. *Journal of Economic Theory*, *2*(3), 244–263.

Attema, A. E., Brouwer, W. B., l'Haridon, O., & Pinto, J. L. (2015). Estimating sign-dependent societal preferences for quality of life. *Journal of Health Economics*, *43*, 229–243. https://doi.org/https://doi.org/10.1016/j.jhealeco.2015.07.006

Baranov, V., & Kohler, H.-P. (2018). The impact of aids treatment on savings and human capital investment in Malawi. *American Economic Journal: Applied Economics*, *10*(1), 266–306.

Becker, G. S., Philipson, T. J., & Soares, R. R. (2005). The quantity and quality of life and the evolution of world inequality. *American Economic Review*, *95*(1), 277–291.

Berger, L., & Emmerling, J. (2020). Welfare as equity equivalents. *Journal of Economic Surveys*, *34*(4), 727–752.

Bleichrodt, H., & Quiggin, J. (1999). Life-cycle preferences over consumption and health: When is cost-effectiveness analysis equivalent to cost–benefit analysis? *Journal of Health Economics*, *18*(6), 681–708. https://doi.org/https://doi.org/10.1016/S0167-6296(99)00014-4

Bognar, G. (2008). Age-weighting. *Economics and Philosophy*, *24*, 167–189.

Bognar, G. (2015). Fair innings. *Bioethics*, *29*(4), 251–261. https://doi.org/https://doi.org/10.1111/bioe.12101

Bommier, A., Le Grand, F., & Harenberg, D. (2022). *Recursive preferences and the value of life. Theory and application to epidemics* (tech. rep.). ETH Zurich. https://francois-le-grand.com/docs/research/BLH_Recursive_Prefs_Epidemics.pdf

Bosmans, K., Decancq, K., & Ooghe, E. (2015). What do normative indices of multidimensional inequality really measure? *Journal of Public Economics*, *130*, 94–104.

Brock, D. W. (2004). Ethical issues in the use of cost effectiveness analysis for prioritisation of health care resources. In S. Anand, F. Peter, & A. Sen (Eds.), *Public health, ethics, and equity* (pp. 201–224). Oxford University Press.

Broome, J. (2002). Measuring the burden of disease by aggregating well-being. In C. J. L. Murray, J. A. Salomon, C. D. Mathers, & A. D. Lopez (Eds.), *Summary measures of population health: Concepts, ethics, measurement, and applications* (pp. 91–113). World Health Organization.

Browning, M., Hansen, L. P., & Heckman, J. J. (1999). Micro data and general equilibrium models. In J. B. Taylor & M. Woodford (Eds.), *Handbook of Macroeconomics vol.1A* (pp. 543–633). Elsevier.

Case, A., & Deaton, A. (2017). Mortality and morbidity in the 21st century. *Brookings Papers on Economic Activity*, *Spring*, 397–476.

Case, A., & Deaton, A. (2020). *Deaths of despair and the future of capitalism*. Princeton University Press.

Chetty, R. (2006). A new method of estimating risk aversion. *American Economic Review*, *96*(5), 1821–1834. http://www.jstor.org/stable/30034997

Chiang, C. L. (1968). *An introduction to stochastic processes in biostatistics*. New York: Wiley.

Cookson, R., Skarda, I., Cotton-Barratt, O., Adler, M., Asaria, M., & Ord, T. (2021). Quality adjusted life years based on health and consumption: A summary wellbeing measure for cross-sectoral economic evaluation. *Health Economics*, *30*, 70–85.

Córdoba, J. C., & Ripoll, M. (2017). Risk aversion and the value of life. *The Review of Economic Studies*, *84*(4), 1472–1509.

Crafts, N. F., & Haacker, M. (n.d.). *Welfare implications of HIV/AIDS*. International Monetary Fund.

Da Costa, S. (2023). Estimating the welfare gains from anti-retroviral therapy in Sub-Saharan Africa. *mimeo University of Antwerp*.

Delprat, G., Leroux, M.-L., & Michaud, P.-C. (2016). Evidence on individual preferences for longevity risk. *Journal of Pension Economics & Finance*, *15*(2), 160–179.

Dolan, P., Shaw, R., Tsuchiya, A., & Williams, A. (2005). Qaly maximisation and people's preferences: A methodological review of the literature. *Health Economics*, *14*(2), 197–208.

Dolan, P., & Tsuchiya, A. (2011). Determining the parameters in a social welfare function using stated preference data: An application to health. *Applied Economics*, *43*(18), 2241–2250.

Dolan, P., & Tsuchiya, A. (2012). It is the lifetime that matters: Public preferences over maximising health and reducing inequalities in health. *Journal of Medical Ethics*, *38*(9), 571–573. https://doi.org/10.1136/medethics-2011-100228

Echazu, L., & Nocetti, D. (2013). Priority setting in health care: Disentangling risk aversion from inequality aversion. *Health Economics*, *22*(6), 730–740.

Edlin, R., Tsuchiya, A., & Dolan, P. (2012). Public preferences for responsibility versus public preferences for reducing inequalities. *Health Economics*, *21*(12), 1416–1426. https://doi.org/https://doi.org/10.1002/hec.1799

Edwards, R. D. (2013). The cost of uncertain life span. *Journal of Population Economics*, *26*(4), 1485–1522.

Foster, J. E., Lopez-Calva, L. F., & Szekely, M. (2005). Measuring the distribution of human development: Methodology and an application to mexico. *Journal of Human Development*, *6*(1), 5–25. https://doi.org/10.1080/1464988052000342220

Global Burden of Disease Collaborative Network. (2021). Global burden of disease study 2019 reference life table. https://doi.org/10.6069/1D4Y-YQ37

Goerlich, F. (2020). Distributionally adjusted life expectancy as a life table function. *Demographic Research*, *43*, 365–400.

Gouda, H. N., Charlson, F., Sorsdahl, K., Ahmadzada, S., Ferrari, A. J., Erskine, H., Leung, J., Santamauro, D., Lund, C., Aminde, L. N., et al. (2019). Burden of non-communicable diseases in sub-saharan africa, 1990–2017: Results from the global burden of disease study 2017. *The Lancet Global Health*, *7*(10), e1375–e1387.

Hall, R. E. (1988). Intertemporal substitution in consumption. *Journal of Political Economy*, *96*(2), 339–357.

Hall, R. E., & Jones, C. I. (2007). The value of life and the rise in health spending. *The Quarterly Journal of Economics*, *122*(1), 39–72.

Hammitt, J. K. (2013). Admissible utility functions for health, longevity, and wealth: Integrating monetary and life-year measures. *Journal of Risk and Uncertainty*, *47*(3), 311–325.

Hardardottir, H., Gerdtham, U.-G., & Wengström, E. (2021). Parameterizing standard measures of income and health inequality using choice experiments. *Health Economics*, *30*(10), 2531–2546. https://doi.org/10.1002/hec.4395

Harper, S., Riddell, C. A., & King, N. B. (2021). Declining life expectancy in the united states: Missing the trees for the forest. *Annual Review of Public Health*, *42*(1), 381–403. https://doi.org/10.1146/annurev-publhealth-082619-104231

Harris, J. (2006). *The value of life: An introduction to medical ethics*. Routledge.

Hausman, D. M. (2012). Health, well-being, and measuring the burden of disease. *Population Health Metrics*, *10*(1), 1–7.

Havranek, T., Horvath, R., Irsova, Z., & Rusnak, M. (2015). Cross-country heterogeneity in intertemporal substitution. *Journal of International Economics*, *96*(1), 100–118.

Herrera-Araujo, D., Hammitt, J. K., & Rheinberger, C. M. (2020). Theoretical bounds on the value of improved health. *Journal of Health Economics*, *72*, 102341.

Hicks, D. A. (1997). The inequality-adjusted human development index: A constructive proposal. *World development*, *25*(8), 1283–1298.

Hougaard, J. L., Moreno-Ternero, J. D., & Østerdal, L. P. (2013). A new axiomatic approach to the evaluation of population health. *Journal of Health Economics*, *32*(3), 515–523. https://doi.org/https://doi.org/10.1016/j.jhealeco.2013.01.006

Hurley, J., Mentzakis, E., & Walli-Attaei, M. (2020). Inequality aversion in income, health, and income-related health. *Journal of Health Economics*, *70*, 102276.

Jones, C. I., & Klenow, P. J. (2016). Beyond gdp? welfare across countries and time. *American Economic Review*, *106*(9), 2426–57.

Kamm, F. M. (2002). Health and equity. In C. J. L. Murray, J. A. Salomon, C. D. Mathers, & A. D. Lopez (Eds.), *Summary measures of population health: Concepts, ethics, measurement and applications* (pp. 685–706). World Health Organization.

Le Grand, J. (1987). Inequalities in health: Some international comparisons. *European Economic Review*, *31*(1-2), 182–191.

McDonald, S., & Roberts, J. (2006). Aids and economic growth: A human capital approach. *Journal of Development Economics*, *80*(1), 228–250.

McNamara, S., Holmes, J., Stevely, A. K., & Tsuchiya, A. (2020). How averse are the uk general public to inequalities in health between socioeconomic groups? a systematic review. *European Journal of Health Economics*, *21*, 275–285. https://doi.org/https://doi.org/10.1007/s10198-019-01126-2

McNamara, S., Tsuchiya, A., & Holmes, J. (2021). Does the uk-public's aversion to inequalities in health differ by group-labelling and health-gain type? a choice-experiment.

Social Science & Medicine, *269*, 113573. https://doi.org/https://doi.org/10.1016/j.socscimed.2020.113573

Moreno-Ternero, J. D., Tornøe Platz, T., & Østerdal, L. P. (2023). QALYs, DALYs, and HALYs: A unifying framework for the evaluation of population health. *Journal of Health Economics*, *87*, 102714. https://doi.org/https://doi.org/10.1016/j.jhealeco.2022.102714

Murphy, K. M., & Topel, R. H. (2003). The economic value of medical research. In K. M. Murphy & R. H. Topel (Eds.), *Measuring the gains from medical research* (pp. 125–146). Chicago University Press.

Murphy, K. M., & Topel, R. H. (2006). The value of health and longevity. *Journal of Political Economy*, *114*(5), 871–904.

Murray, C. J. L. (1994). Quantifying the burden of disease: The technical basis for disability-adjusted life years. *Bulletin of the World Health Organization*, *72*, 429–445.

Murray, C. J. L., Lopez, A. D., World Health Organization, World Bank, & Harvard School of Public Health. (1996). *The Global Burden of Disease : A comprehensive assessment of mortality and disability from diseases, injuries, and risk factors in 1990 and projected to 2020*. Harvard School of Public Health on behalf of the World Health Organization; the World Bank.

Muszyńska, M., & Janssen, F. (2016). The concept of the equivalent length of life for quantifying differences in age-at-death distributions across countries. *Genus*, *72*(6), 2035–5556. https://doi.org/10.1186/s41118-016-0011-1

Norheim, O. F. (2013). Atkinson's measure of inequality: Can measures of economic inequality help us understand trade-offs in health care priority setting? In N. Eyal, S. A. Hurst, O. F. Norheim, & D. Wikler (Eds.), *Inequalities in health: Concepts, measures and ethics*. Oxford University Press.

Ogaki, M., Ostry, J. D., & Reinhart, C. M. (1996). Saving behavior in low-and middle-income developing countries: A comparison. *IMF Staff Papers*, *43*(1), 38–71.

Parfit, D. (1984). *Reasons and persons*. Oxford University Press.

Parfit, D. (2000). Equality or priority? In M. Clayton & A. Williams (Eds.), *The ideal of equality* (pp. 81–125). Palgrave MacMillian.

Ponthiere, G. (2011). Measuring variations in lifetime welfare ex ante and ex post: Some exploratory calculations. *Bulletin of Economic Research*, *63*(3), 255–291.

Preston, S., Heuveline, P., & Guillot, M. (2001). *Demography - measuring and modelling population processes*. Blackwell Publishing, Oxford.

Robson, M., Asaria, M., Cookson, R., Tsuchiya, A., & Ali, S. (2017). Eliciting the level of health inequality aversion in england. *Health Economics*, *26*(10), 1328–1334.

Robson, M., O'Donnell, O., & Van Ourti, T. (2023). Aversion to health inequality – pure, income-related, and income-caused. *mimeo Erasmus University Rotterdam*.

Rosen, S. (1988). The value of changes in life expectancy. *Journal of Risk and Uncertainty*, *1*(3), 285–304.

Salomon, J. A., Vos, T., Hogan, D. R., Gagnon, M., Naghavi, M., Mokdad, A., Begum, N., Shah, R., Karyana, M., Kosen, S., Farje, M. R., Moncada, G., Dutta, A., Sazawal, S., Dyer, A., Seiler, J., Aboyans, V., Baker, L., Baxter, A., ... Murray, C. J. (2012). Common values in assessing health outcomes from disease and injury: Disability weights measurement study for the global burden of disease study 2010. *The Lancet*, *380*(9859), 2129–2143.

Schelling, T. C. (1968). The life you save may be your own. In S. B. Chase (Ed.), *Problems in public expenditure analysis. studies in government finance* (pp. 127–162). Brookings Institution.

Shkolnikov, V. M., Andreev, E. E., & Begun, A. Z. (2003). Gini coefficient as a life table function: Computation from discrete data, decomposition of differences and empirical examples. *Demographic Research*, *8*, 305–358.

Silber, J. (1983). ELL (the equivalent length of life) or another attempt at measuring development. *World Development*, *11*(1), 21–29.

Soares, R. R. (2007). Health and the evolution of welfare across brazilian municipalities. *Journal of Development Economics*, *84*(2), 590–608.

Sullivan, D. F. (1971). A single index of mortality and morbidity. *HSMHA health reports*, *86*(4), 347.

Tompsett, A. (2020). The lazarus drug: The impact of antiretroviral therapy on economic growth. *Journal of Development Economics*, *143*, 102409.

United Nations. (2019). World population prospects 2019, online edition. rev. 1. [Accessed: 2021-06-21]. https://population.un.org/wpp/Download/Standard/Mortality/

United Nations Development Programme. (2020). *Human Development Report 2020*. United Nations. http://report.hdr.undp.org

Usher, D. (1973). An imputation to the measure of economic growth for changes in life expectancy. In M. Moss (Ed.), *The measurement of economic and social performance* (pp. 193–232). NBER.

van Raalte, A. A., Sasson, I., & Martikainen, P. (2018). The case for monitoring life-span inequality. *Science*, 1002–1004. https://doi.org/10.1126/science.aau5811

Wolpin, K. I. (1997). Determinants and consequences of the mortality and health of infants and children. In M. Rosenzweig & O. Stark (Eds.), *Handbook of Population and Family Economics* (pp. 483–557). North Holland.

Woolf, S. H., & Schoomaker, H. (2019). Life expectancy and mortality rates in the united states, 1959-2017. *JAMA*, *322*(20), 1996–2016. https://doi.org/10.1001/jama.2019.16932

World Bank. (1993). *World Development Report 1993: Investing in Health*. Oxford University Press.

World Bank. (2022). *World Development Indicators*. The World Bank. https://datatopics.worldbank.org/world-development-indicators/

Wu, X., Savvides, A., & Stengos, T. (2014). The global joint distribution of income and health. In J. Ma & M. Wohar (Eds.), *Recent advances in estimating nonlinear models* (pp. 249–279). Springer.

APPENDICES

Appendix A Derivation of WTP for health change

Define the willingness to pay (WTP) for change in age-specific health distributions from $F\left(h_s(x)\right)$ to $F^*\left(h_s(x)\right)$ and the age-at-death distribution from $G(x)$ to $G^*(x)$ by $W(c - WTP, F^*(h_s(x)), G^*(x)) = W(c, F(h_s(x)), G(x))$, where $W()$ is given by eq.(7).

Assuming aversion to dispersion in lifetime welfare (ψ) is equal to aversion to dispersion in health distribution-adjusted lifespan (η), we get

$$WTP = c - u^{-1}\left[u(c)\frac{\left[\sum_{x=0}^{T} r(x)y^d(x)^{1-\eta}\right]^{\frac{1}{1-\eta}}}{\left[\sum_{x=0}^{T} r^*(x)y^{d^*}(x)^{1-\eta}\right]^{\frac{1}{1-\eta}}}\right]. \tag{A1}$$

Using eq.(6), we can write eq.(A1) as,

$$WTP = c - u^{-1}\left[u(c)\frac{EHAL}{EHAL^*}\right]. \tag{A2}$$

Specifying $u(c)) = \left(c^{1-\gamma} - \underline{c}^{1-\gamma}\right) / (1-\gamma)$ and solving eq.(A2) gives eq.(8).

Appendix B Disability weights and health distributions

DALYs are the sum of years of life lost (YLL) due to premature mortality and the years of life in full health that are lost due to living with disability (YLD). For disease k,

$$DALY_k = YLL_k + YLD_k, \tag{B1}$$

where $YLL_k = \sum_x D_{k,x} \cdot L_x$, $D_{k,x}$ is the number of deaths due to disease k at age x, and L_x is the number of years by which that age falls short of life expectancy at birth. The latter term is derived from a reference life table constructed from the lowest age-specific mortality rates across all locations worldwide with populations greater than 5 million.

$YLD_k = P_k \cdot z_k$, where P_k is the number of disease cases prevalent in the population across all ages and $z_k \in [0, 1]$ ($\equiv DW_k$ in GBD notation) is the disease's disability weight. We derive the weights from GBD data on YLD_k and P_k. A weight of 0 corresponds to full health, while a weight of 1 is equivalent to death. To estimate the weights, survey respondents in five countries (including one in SSA) were asked to make pairwise comparisons of health vignettes, each of which described impairments associated with a disease state. They had to identify the vignette representing better health overall (Salomon et al., 2012). The relative severity of a disease state is derived from the relative frequency of respondents who judged the corresponding vignette to represent worse health than the comparison. The weights are anchored on a value of 1 for death by asking some respondents to say which of two programmes would generate more total health – one that prevented 1000 immediate deaths or another that prevented a larger number of people succumbing to a non-fatal disease that caused impairments described by a corresponding vignette.

Figure B1 shows the simulated distribution of health $f\left(h_s(i)\right)$ obtained from 100,000 simulants within each of six sex-age partitions that are assigned diseases according to sex-age specific prevalence rates. Given the set of diseases, K, assigned to each simulant, health is obtained from $h_s = \prod_{k \in K}(1 - z_k)$.

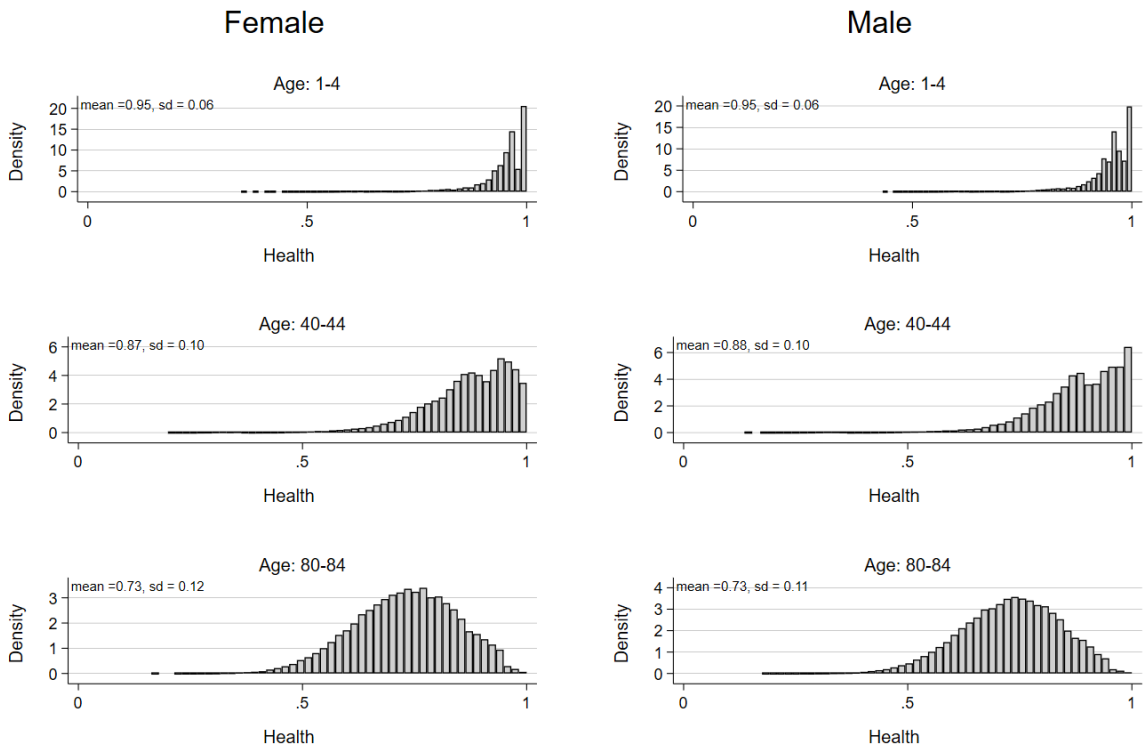


Figure B1: Simulated health distributions by sex and age

Appendix C Derivation of age-at-death distributions

We stratify all analyses by sex. Here, we describe the derivation of age-at-death distributions for one sex.

To construct a life table and an age-at-death distribution ($G(x)$) for the life-table cohort, we need the conditional probability of death in each age interval, $[x, x+n)$: ${}_nq_x = \frac{{}_nd_x}{l_x}$, where ${}_nd_x$ is the number of cohort deaths in the interval and l_x is the number of cohort survivors to exact age x. We estimate these conditional probabilities from

$$ {}_nq_x = \frac{n \cdot {}_nM_x}{1 + (n - {}_na_x) \cdot {}_nM_x}, \tag{C1} $$

where ${}_nM_x$ is the age-interval mortality rate and ${}_na_x$ is the average number of years lived in the interval for those who die within it (Chiang, 1968). We use GBD estimates of all-cause mortality rates for 21 age partitions: $[0, 1)$, $[1, 4)$, $[5, 9)$, ..., $[90, 94)$, $[95, \infty)$(GBDCN, 2021). We use values of ${}_na_x$ for SSA provided by the UN World Population Prospects (United Nations, 2019).

Starting with a radix of ($l_0 =$) 100,000 births, we obtain the number of survivors to each exact age by iterative application of the conditional probabilities, $l_{x+n} = l_x(1 - {}_nq_x)$. The number of cohort deaths in each age interval (more precisely, at age $x + {}_na_x$) is then ${}_nd_x = l_x - l_{x+n}$. We use the proportion of cohort deaths at each age to define $G(x)$ that is used in the analysis.

To calculate cause-deleted life tables and counterfactual age-at-death distributions, we use the fact that the GBD all-cause mortality rate is an additive sum of disease specific mortality rates: ${}_nM_x = \sum_k \frac{{}_nD_x^k}{{}_nP_x} = \sum_k {}_nM_x^k$, where ${}_nD_x^k$ is the number of deaths due to disease k in the age interval and ${}_nP_x$ is the respective mid-year population size. Then, the counterfactual mortality rate after elimination of mortality caused by disease k is ${}_nM_x^{-k} = {}_nM_x - {}_nM_x^k$. We

obtain these counterfactual mortality rates — denoted generically by ${}_nM_x^*$ — from GBD estimates of cause-specific mortality rates and transform them into corresponding counterfactual conditional probabilities of death within each interval using

$$ {}_nq_x^* = \frac{n \cdot {}_nM_x^*}{1 + (n - {}_na_x^*) \cdot {}_nM_x^*}, \tag{C2} $$

where ${}_na_x^*$ is the counterfactual average number of years lived in the age interval for those who die within it. To our knowledge, there are no disease specific SSA data for this variable. We therefore set ${}_na_x^* = {}_na_x$ in all calculations. While this may introduce some bias, it should be small given the highly disaggregated disease level data and the limited scope for the values of ${}_na_x$ to impact on the population health measures (Preston et al., 2001).

We estimate counterfactual values for the number of survivors to each age and the number of deaths within each age interval by applying the formulas given above to the counterfactual conditional probabilities. The proportions of deaths within each and every interval under the counterfactual that a disease is eliminated gives an estimate of $G^*(x)$ that is used in the analysis.

Appendix D Additional results

Table D1: Measures of population health, Sub-Saharan Africa

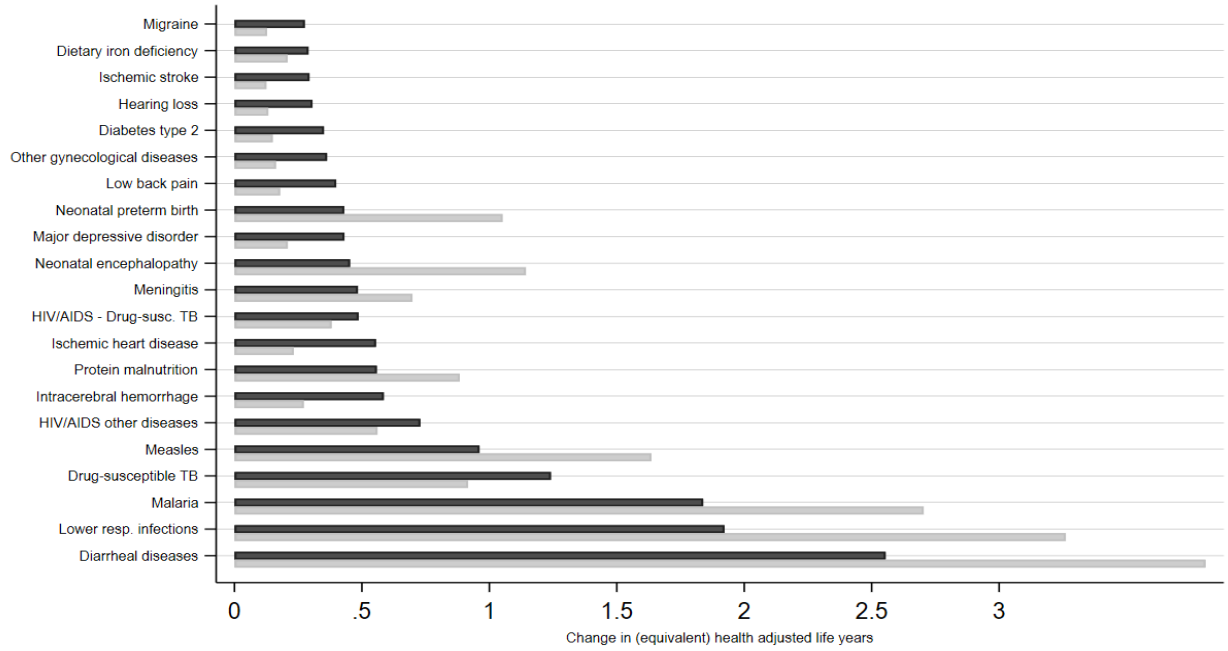
	1990	**2004**	**2019**	**2019-1990**	
				Δ	$\%\Delta$
Female					
$REHAL\,(\varepsilon = 2)$	47.74	48.01	61.77	13.92	29.1%
$EHAL\,(\varepsilon = 0, \eta = 1)$	27.32	30.48	45.78	18.47	67.6%
$EHAL\,(\varepsilon = 0, \eta = 2)$	3.12	3.39	3.84	0.72	23.0%
$EHAL\,(\varepsilon = 2, \eta = 2)$	3.11	3.38	3.83	0.72	23.3%
Male					
$REHAL\,(\varepsilon = 2)$	45.19	46.46	58.02	12.84	28.4%
$EHAL\,(\varepsilon = 0, \eta = 1)$	23.98	27.95	40.98	17.00	70.9%
$EHAL\,(\varepsilon = 0, \eta = 2)$	2.57	3.01	3.36	0.79	30.7%
$EHAL\,(\varepsilon = 2, \eta = 2)$	2.56	2.99	3.35	0.79	31.0%

Note: $REHAL$=Restricted Equivalent Health-Adjusted Lifespan, eq.(4), $EHAL$=Equivalent Health-Adjusted Lifespan, eq.(6).

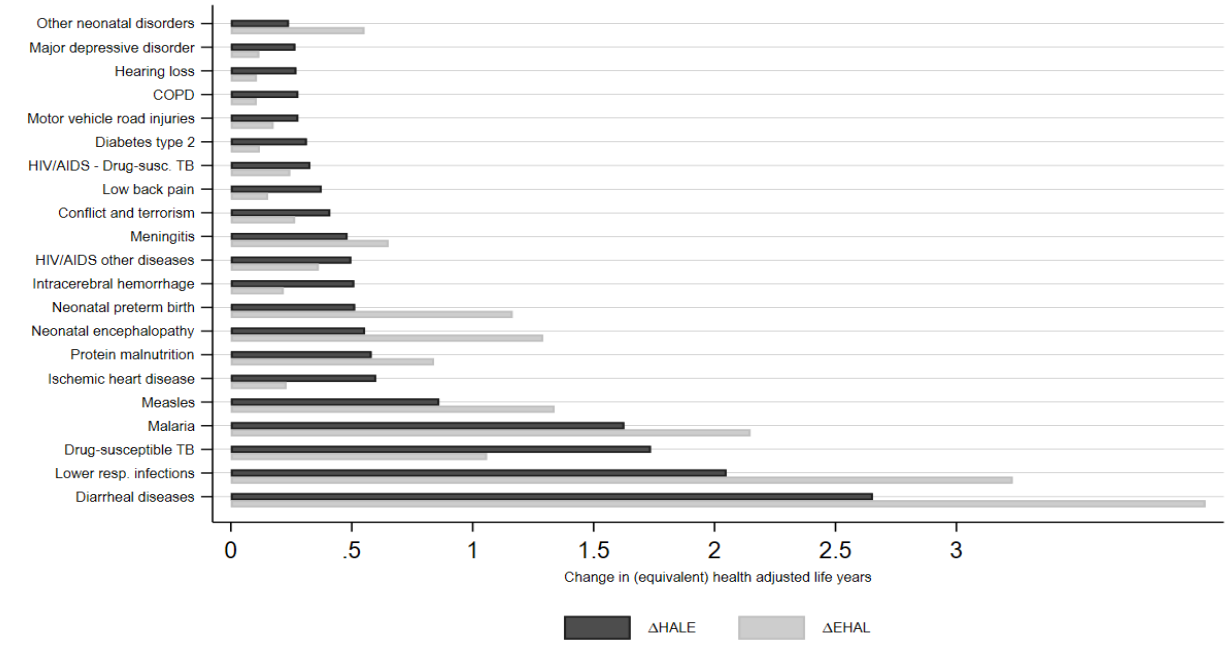
Table D2: Welfare gains from population health changes, $ per capita

	1990-2004	**2004-2019**	**1990-2019**
Female			
Distributionally insensitive	8.84	434.71	344.07
Distributionally sensitive	195.79	566.37	488.56
Male			
Distributionally insensitive	60.64	402.35	340.13
Distributionally sensitive	251.02	550.93	494.92

Note: Distributionally insensitive estimates from eq.(9) with $HALE$ and $HALE^*$ given by eq.(1) applied to the baseline and endline years, respectively. Distributionally sensitive estimates from eq.(8) with $EHAL$ and $EHAL^*$ given by eq.(6) applied to the baseline and endline years, respectively. Distributionally sensitive estimates use $\varepsilon = \eta = 1$. Valuations are per capita expressed in 1990 $ amounts.



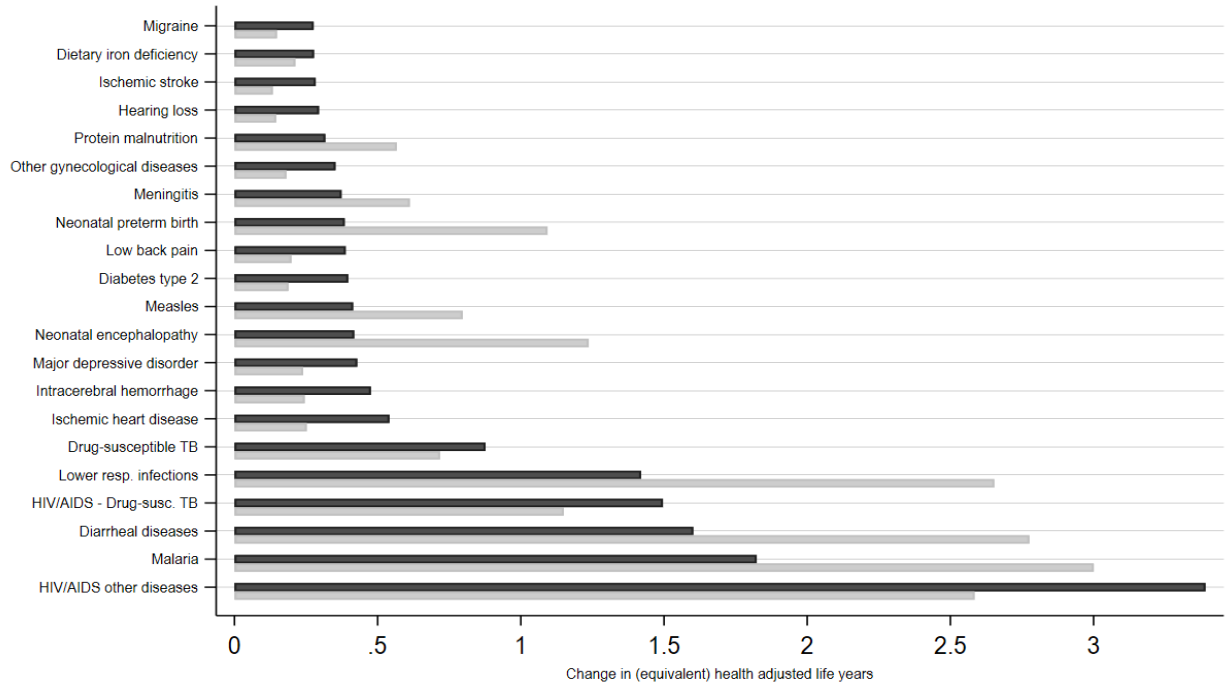
(a) Female



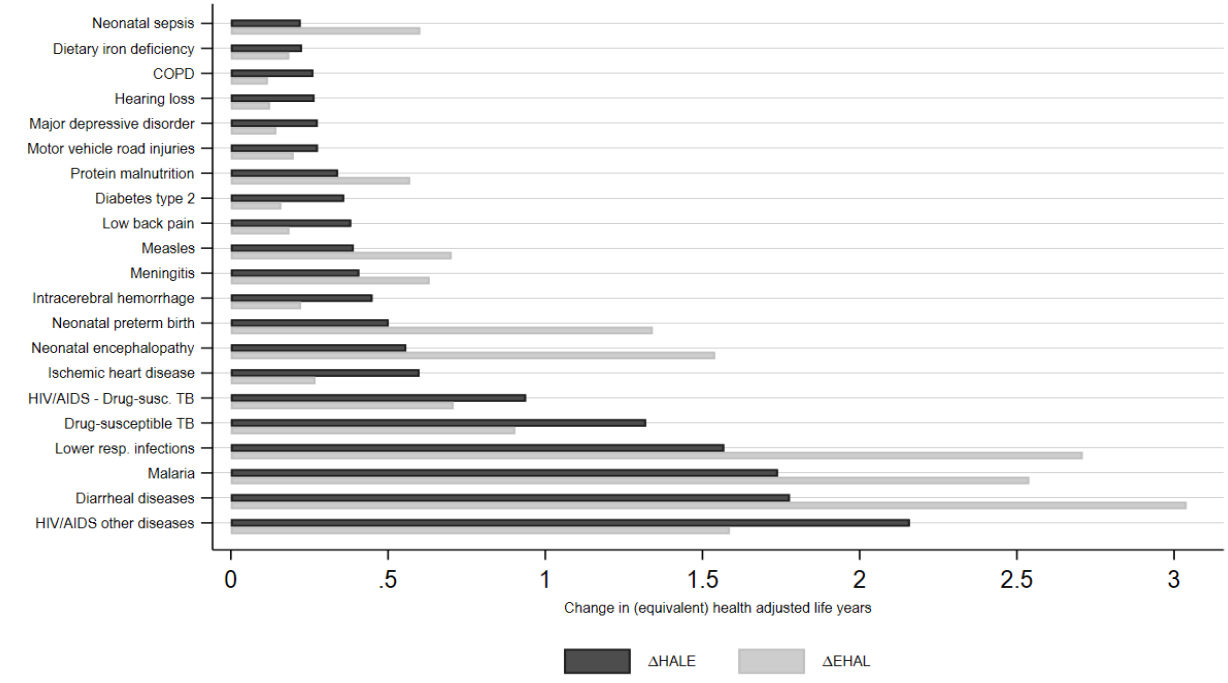
(b) Male

Figure D1: Top 20 disease burdens with and without distributional sensitivity, 1990

Notes: Increases in $HALE$ and $EHAL(\varepsilon = 1, \eta = 1)$ from elimination of each of the 20 diseases/conditions with largest burdens measured by increase in $HALE$.



(a) Female



(b) Male

Figure D2: Top 20 disease burdens with and without distributional sensitivity, 2004

Notes: Increases in $HALE$ and $EHAL(\varepsilon = 1, \eta = 1)$ from elimination of each of the 20 diseases/conditions with largest burdens measured by increase in $HALE$.

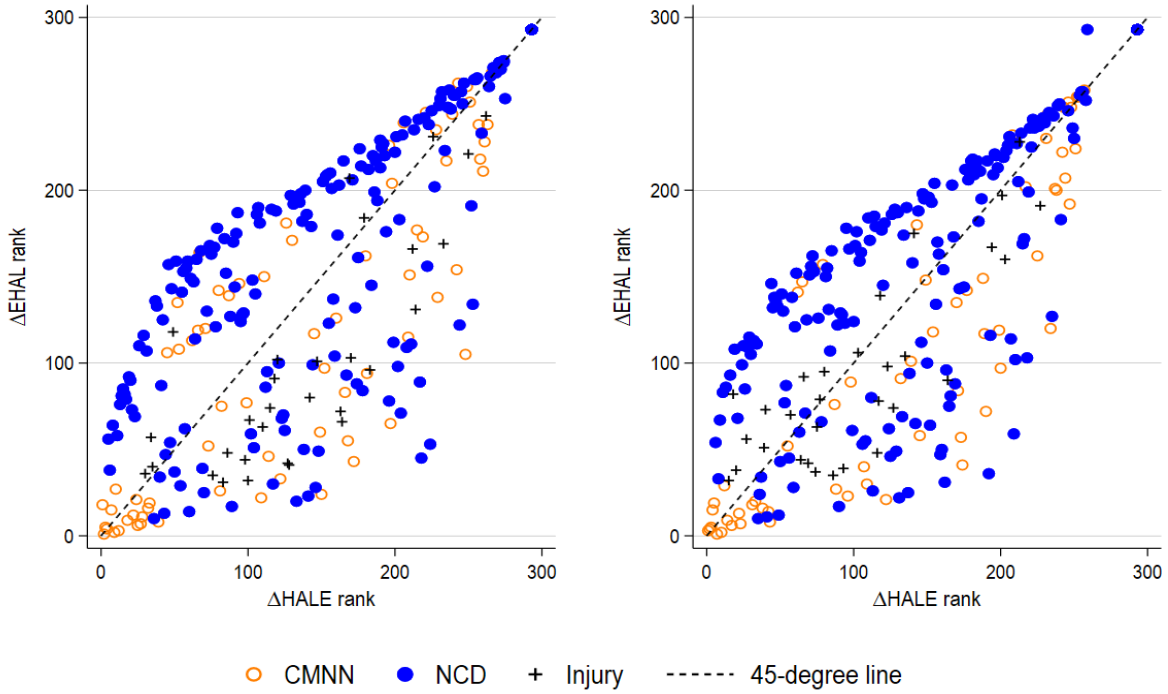
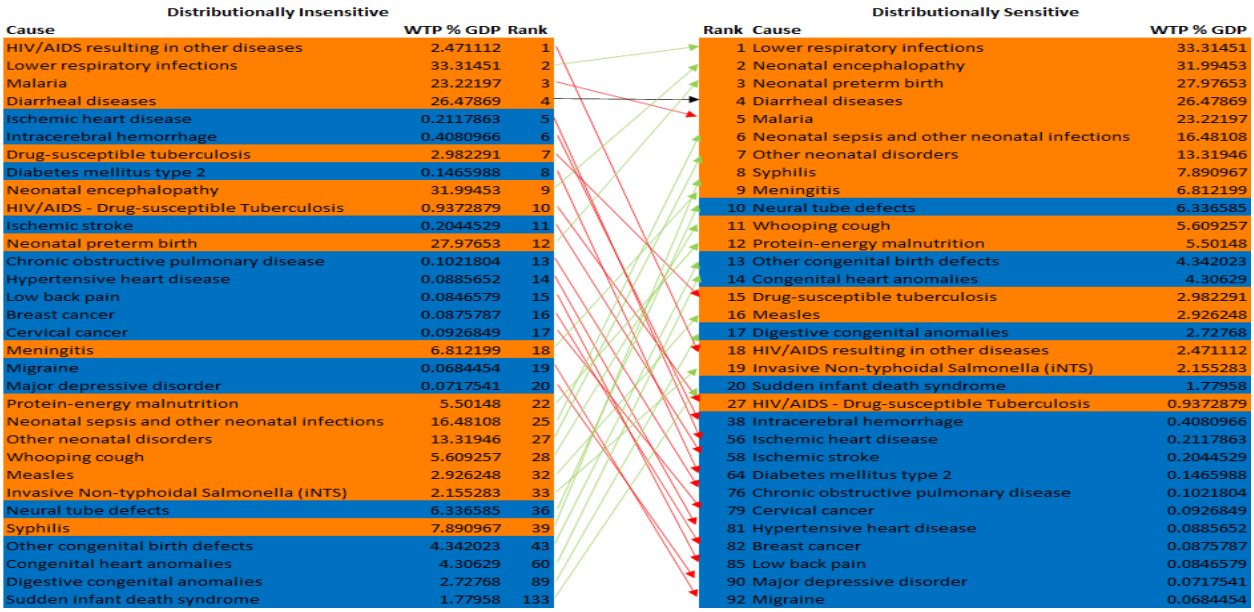
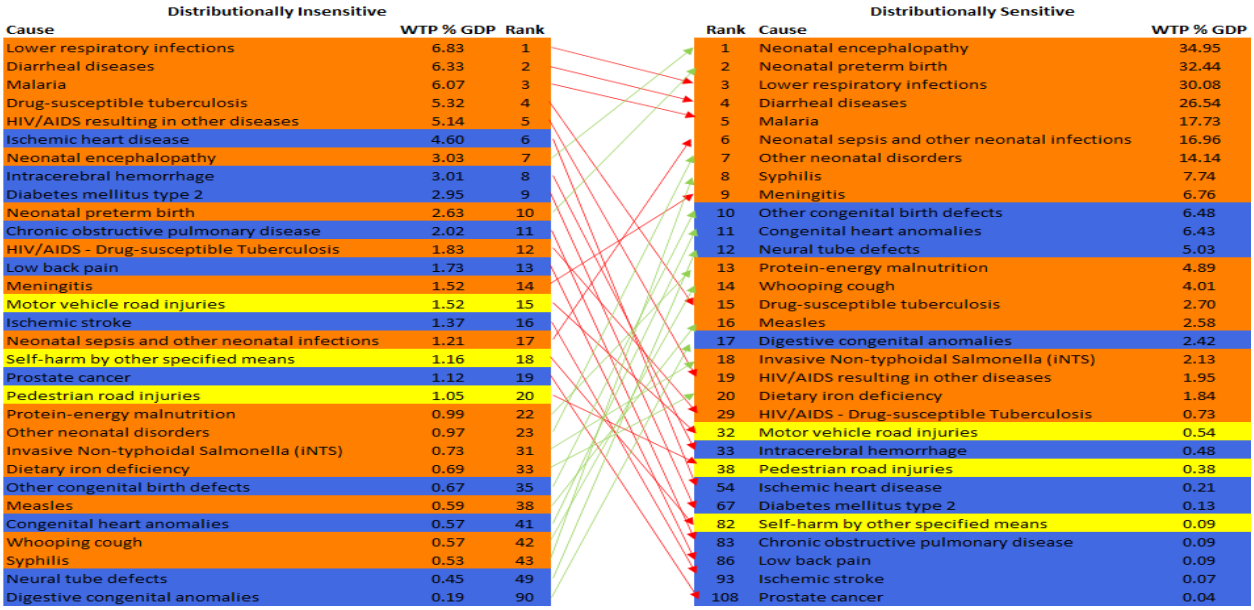


Figure D3: Disease burden ranks with distributionally sensitive and insensitive measures, 2019 ($\varepsilon = \eta = 2$)

Notes: x-axis shows disease ranks by $HALE^*$ - $HALE$, where $HALE$ is from eq. (1) applied to the observed distributions and $HALE^*$ is from the counterfactual distributions after elimination of the respective disease. y-axis shows ranks by $EHAL^*$ - $EHAL$, where each measure is obtained from eq. (6) with $\varepsilon = \eta = 2$. Lower number indicates higher disease burden rank. Dashed line is at 45-degree.



(a) Female



(b) Male

Figure D4: Top 20 disease burdens ranked by distributionally insensitive and sensitive willingness to pay, 2019

Notes: CMNN=orange, NCD=blue, Injuries=yellow. Distributionally insensitive WTP from (9). Distributionally sensitive WTP from (8) with $\varepsilon = \eta = 2$. In each case, $\gamma = 1.25$, c =GDP per capita, and $\underline{c} = 10\%$ of GDP per capita. The figure shows the top 20 ranked diseases using the distributionally insensitive or sensitive WTP measures. Diseases are ordered from top to bottom by distributionally insensitive WTP.