Distributionally Sensitive Measurement and Valuation of Population Health with an Application to Disease Prioritization in Sub-Saharan Africa *

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Abstract

We introduce a measure of population health that can be calculated from a health-extended period life table and yet is sensitive to inequality in both age-specific health and health-adjusted lifespan. Willingness to pay for a change in this measure provides a distributionally sensitive valuation of a health improvement. We use the measure and its valuation with Global Burden of Disease data to evaluate trends in population health and disease priorities in Sub-Saharan Africa (SSA) between 1990 and 2017. Aversion to inequality in age-specific health has relatively little impact on the measure. Aversion to inequality in health-adjusted lifespan has a large impact. While the distributionally insensitive health-adjusted life expectancy increased by around 20% over the period, our measure increased by 30% due to greater reductions in mortality at younger ages. Allowing for distributional sensitivity greatly reduces convergence in the burdens of communicable diseases, which strike at younger ages, and noncommunicable diseases, and substantially increases prioritisation of the former.

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1 Introduction

Individuals attach a subtantial value to living longer and healthier lives. To study population health, and to set disease priorities, (health adjusted) life expectancy (LE) measures are routinely applied.[1] Substantial progress has taken place over the past century. Global life expectancy at birth has more than doubled between 1900 (32 years) and 2015 (71.7 years)[2], and recent studies show that the increase in life expectancy has been accompanied by a rise in lifespan equality (Colchero et al., 2016; Aburto et al., 2020). The interest in both the overall level and distribution of health, or more accurately length of life, motivates us to develop a measure of population health that accounts for both aspects of health. We first propose the equivalent health-adjusted lifespan($EHAL$). This measure uses nested equity equivalents to incorporate inequality aversion towards health *and* longevity in the health-adjusted life expectancy ($HALE$) measure. We then proceed to value changes in population health, by obtaining the willingness to pay for a change in EHAL due to the elimination of disease causes. This strategy allows to provide a monetary value for disease improvement for a disaggregated set of disease causes. The main motivation for this is, similar to other studies on the value of health, that the value of disease improvements informs on the social returns to health investments and innovations (Murphy and Topel 2006).

Several adjustments to LE for the measurement of population health have been proposed. $HALE$ adjusts LE for mean health in each year of life. But it is still an average. While LE is the average age at which life is lost, $HALE$ is the average age at which full health is lost. While LE is the average age at death, $HALE$ is a weighted average age at death, with weights equal to age-specific mean levels of health. While LE is insensitive to dispersion in lifespan, $HALE$ is insensitive to dispersion in health-adjusted lifespan.[3] It is also insensitive

[1] In the Lancet alone, 174 articles (26/04/2021) have been published with content from the Global Burden of Disease (GBD) - the most comprehensive source of data on burden of disease - of which many describe trends on (health adjusted) life expectancy and expenditures on particular disease causes. https://www.thelancet.com/gbd/collection

[2] https://ourworldindata.org/life-expectancy

[3] $HALE$ is sensitive to dispersion in lifespan. Consider 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

to dispersion in health both within and across ages.

Silber (1983) proposed adjusting LE for lifespan dispersion using Atkinson (1970) and Kolm (1976a,b) inequality indices to produce the equally distributed *equivalent length of life* (ELL): the lifespan that, if it were enjoyed by everyone in the life-table cohort, would yield the same social welfare as the distribution of lifespans arising from passing the cohort through the life table. ELL is a weighted average age at death, with greater weight placed on deaths that occur at younger ages.[4] (Norheim 2013) suggested that the same conceptual apparatus could be applied to health-adjusted (or disability-adjusted) life years but did not explain how to allow for aversion to inequality in both health and lifespan.

We adjust $HALE$ for dispersion both in healthy lifespans and in health at each age. We set out to do this while restricting ourselves to using widely available data that is similar to the required data to calculate $HALE$. That is, the Global Burden of Disease data makes sex- and age-specific disease prevalence and mortality rates available at regional, national and sub-national levels since 1990. In addition, they provide a measure of health achieved in each disease state at each age. Applying equally distributed equivalents to the health and length of life dimension of these health-extended life tables allows us to derive a summary measure of population health that incorporates distributional concerns for health and longevity – equivalent health adjusted lifespan ($EHAL$). The advantage of our measure is that it is measured in the same units as $HALE$ – health-adjusted life years ($HALYs$). The ($EHAL$) further nests ($HALE$) when parameters are set to values that imply no distributional sensitivity. This makes it easy to observe the consequences of allowing for distributional sensitivity. There are at least two disadvantages of this measure.

life expectancy constant. If average health were monotonically decreasing in age, then the additional year of life at a younger age would get more weight in 1 than that given to the loss of one year of life at an older age. Consequently, $HALE$ would increase.The measures we propose also have this implicit sensitivity to the distribution of health over ages. In addition, they are explicitly sensitive to the dispersion in health at each age and to dispersion in health-adjusted lifespan.

[4]The Atkinson index has been used to assess inequalities in lifespans within and across countries (Goerlich 2020; Le Grand 1987; Shkolnikov, Andreev, and Begun 2003) and to produce the (lifespan) inequality-adjusted Human Development Index (UNDP 2010; Hicks (1997)). Goerlich (2020) proves that ELL satisfies the axioms of *normalisation*, *symmetry*, *replication invariance*, *monotonicity*, the *Pigou Dalton transfer principle*, and, depending on the specification, *homogeneity* in lifespan.

First, we cannot take account of correlation between health states at different ages, nor can we allow for correlation between health and lifespan. Clearly, these are limitations.[5] But they are limitations that carry over from $HALE$ and the data used to calculate it. Second, because the unit of measurement is $HALYs$ and not money the measure cannot be easily used to compare the social value of alternative investments that would impact on population health. To correct the second limitation, we propose a measure for willingness to pay (WTP) for improvements in ($EHAL$). By sequentially eradicating each disease from the life table, we construct a ranking of diseases based on the resulting impact on ($EHAL$). In addition this allows to quantify (distributional) improvements in health. While previous literature has incorporated distributional concerns within life expectancy measurement, this is the first paper, to our knowledge, to adjust for inequality aversion with respect to health quality *and* longevity (Goerlich 2020; Norheim 2013; Silber 1983).

Our approach is rooted in, at least, two branches of the economics literature. Firstly, following Silber (1983) and Norheim (2013), we make use of a *health-related* social welfare function (SWF). This is defined over a measure of health rather than utilities and is 'extra-welfarist' in the sense that its goes beyond the ranking of social states based on individual utilities while placing weights on different points of the distribution. 'Extra-welfarists' and others (e.g. Hausman 2012) argue that there are several strong normative and practical reasons to measure social welfare in terms of health rather than utilities within the context of health policy.[6] In this case, utility may not be an appropriate maximand and distribuendum because individuals may adapt to their poor health conditions over time. When people adapt, their physical health remains unchanged but their level of utility improves. Thus, evaluating diseases based on their consequences for utility (or well-being) could lead to the

[5]If a cohort of individuals could be followed from birth until death, then dispersion in lifetime health profiles could be observed and aversion to this dispersion specified. Without direct observation of lifetime health profiles, one might try to simulate them. But this would require information on the correlation between health and mortality risks, and on the length of illness associated with each disease. This information is not available from the Global Burden of Disease - the most comprehensive source of health and mortality data from which population health measures can be calculated for many countries.

[6]See Brouwer et al. (2008) for an overview of the extra-welfarist case.

negation of seriously debilitating conditions (Hausman 2012, 2015). In addition, while there are challenges to obtaining a scalar measure of health, it seems conceptually easier to measure and make interpersonal comparisons using health rather than utility (Hausman 2012; Olsen 1997).[7] Secondly, our approach links closely to the use of 'nested' equity equivalents in several strands of economics literature.[8] Within the multidimensional well-being literature (e.g. Bosmans, Decancq, and Ooghe 2015; Foster, Lopez-Calva, and Szekely 2005), for instance, equity equivalents have been employed in nested forms to construct indexes that are sensitive to inequalities at different levels, such as within and between dimensions. We follow in this line by first employing equity equivalents to the distribution of health each age and then to the resulting distribution of health adjusted life spans. This allows us to construct a distributionally sensitive generalisation of the standard HALE measure. Our research further links with recent works advocating for a distributional concern within the valuation of health and longevity gains (Adler, Ferranna, et al. 2021; Adler, Hammitt, and Treich 2014; Lakdawalla and Phelps 2019). More broadly, our framework is closely related to the literature on the value of health (e.g. Murphy and Topel 2006). We contribute to this literature by explicitly incorporating aversion towards inequality in health states, both theoretically and empirically. While disease progress improves mean population health, it may also decrease the variation of health outcomes within a population. Empirical evidence suggests that individuals are more averse to inequalities in health than other dimensions of welfare, such as income (Costa-Font and Cowell 2019; Hurley, Mentzakis, and Walli-Attaei 2020). Progress against disease could therefore have substantial value for inequality averse societies, which is not captured in standard measures of population health, such as the health adjusted life expectancy, and current approaches to value health.

The measures introduced in our paper are subsequently applied to inform the debate on the double burden of disease in Sub-Saharan Africa (SSA). Throughout the course of

[7] As Hausman (2012) points out, an individual's utility is at least as multidimensional as their health.

[8] See Berger and Emmerling (2020) for a review of similar approaches in different stands of the economics literature.

human history, infectious diseases have levied a significant burden on societies through increased rates of mortality and morbidity (Deaton 2013). Globally, infectious diseases that are prevalent in the SSA region have long been important in health priority setting. Malaria eradication, for instance, has been on the global health and policy agenda for over half a century and there has been a resurgence in efforts to meet this goal by the middle of the 21st century (Feachem et al. 2019). The Global Polio Eradication Initiative has achieved significant progress towards eradication since its foundation more than 30 years ago. It is estimated that the decreased incidence of polio has saved 13 million children from paralysis[9]. The SSA region is currently undergoing a rapid epidemiological transition defined by a shift in the burden of disease from communicable to non-communicable illnesses. This reflects significant progress against early age diseases and HIV/AIDS, which has allowed an increasing number of individuals to reach older ages at which non-communicable diseases (NCDs) are more prevalent. There is a growing concern that NCDs will pose significant challenges for health systems in SSA and may therefore merit additional weight in priority setting (Lemoine et al. 2012). For instance, Gouda et al. (2019) estimate that the total disability adjusted life years (DALYs) attributable to NCDs increased by 67% between 1990 and 2017, which means that NCDs accounted for almost a third the total SSA burden of disease by the end of the period. Migration, changing lifestyles, coexistence of diseases, genetic predisposition, and the fetal origins - or thrifty phenotype - hypothesis are among the possible explanations for this trend (Peer, 2015; Nyirenda, 2016). In addition, access to treatment in the region seems more readily available for infectious diseases than for NCDs. In Ghana, around 80% of health facilities had Malaria drugs available, while this was only true for 35% in the case of drugs for Diabetes (Kushitor and Boatemaa, 2018). This is the true even while diabetes was found to be more stressful than Malaria in Tanzanian focus groups (Metta et al., 2017). Still, when applied at the population level, measures such as the DALY and *HALE* overlook the distributional burden of diseases. For societies averse to

[9]https://www.gatesfoundation.org/what-we-do/global-development/polio (last accessed on 15 May 2020)

inequalities in health, relatively more weight may be placed on communicable diseases (CDs) which cut lives short at early ages due to "fair innings" considerations. That is, societies may believe that all individuals should be given the opportunity to live a reasonable length of life, i.e. a "fair innings" (Harris 2006). It is therefore important to consider the burden of disease in SSA using a normative framework, which incorporates distributional concerns within measures of population health.

To preview our results, we find that progress against any specific disease is valued at most 21% of GDP per capita for females in SSA (HIV/AIDS in 2004). Overall, (inequality and) Health Adjusted Life Expectancy has improved since 1990, and consequently the value of progress against disease has declined. The decline mainly took place among CDs, causing the overall value of progress against NCDs to outweigh the value for CDs.

The remainder of this paper proceeds as follows. In section 2, we provide the theoretical framework used to estimate the value of disease eradication. Section 3 explains the data, and an overview of results is provided in section 4. We conclude in section 5.

2 Theory

2.1 Distributional sensitivity in the life-years metric

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 the distribution of morbidity is partially 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):

$$HALE = \sum_{x=0}^{T} d(x) \sum_{i=0}^{x-1} h(i) = \sum_{x=0}^{T} d(x) l(x) \,, \tag{1}$$

where T is the maximum postulated lifespan, $d(x)$ is the proportion of the cohort that dies in the age interval $[x, x+1)$ and $h(i) = \sum_s p_s(i)h_s(i)$ is expected health at age i, with $p_s(i)$ being the proportion of the cohort alive at age i that is in health state s at that age and $h_s(i) \in [0, 1]$ is the level of health in that state at age i. For a state equivalent to death, $h_s(i) = 0$, while $h_s(i) = 1$ corresponds to perfect health.[10] Health states, $s = 1, 2, .., S$, are ordered, such that $h_s(i) \leq h_{s+1}(i)\ \forall s$. The cumulative sum $l(x) = \sum_{i=0}^{x-1} h(i)$ is the health-adjusted lifespan — the equivalent number of years lived in full health up to age x. If no adjustment is made for health, such that $h(i) = 1\ \forall i$, then (1) reduces to $LE = \sum_{x=0}^{T} d(x)x$.

2.1.2 Sensitivity to health inequality

LE and $HALE$ are statistical expectations that do not correspond to the lifespan and health-adjusted lifespan, respectively, that any newly born individual could expect to experience. Nonetheless, these summary measures are useful because they 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 involves the imposition of normative judgment when the result is used to evaluate whether population health has improved. Restricting attention to the first moment of the age-at-death distribution, as is done with LE, implies indifference to a change consisting of a reduction in mortality at some younger age that is fully offset by a larger increase in mortality at an older age leaving LE unchanged. 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 an improvement in population health.

We relax these restrictions by invoking a social decision maker (SDM) who chooses between hypothetical distributions of age-specific health and of lifespan generated by alternative health-extended period life tables and who is possibly averse to variation displayed by

[10] Because health is normalised to zero at death, the age at which death occurs is omitted from the summation over i in (1).

these distributions. With no such aversion, the SDM chooses the distribution consistent with the highest $HALE$. Otherwise, the SDM is prepared to sacrifice $HALE$ for less variation in the health-adjusted lifespan. We first derive a measure that is sensitive to age-specific health inequality before incorporating sensitivity to inequality in (health-adjusted) lifespan. We use *inequality* as a synonym for *variation* without implying inequity or any socioeconomic dimension to differences in health and lifespan.

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

$$w\left(F\left(h_s(i)\right)\right) = \sum_s p_s(i) \frac{h_s(i)^{1-\varepsilon}}{1-\varepsilon}, \quad \varepsilon \neq 1, \tag{2}$$

where $\varepsilon \geq 0$ is the SDM's degree of aversion to health inequality across individuals of the same age.[11] 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: $w\left(F\left(h_s(i)\right)\right) \to min\{h_s(i)\} = h_1(i)$ as $\varepsilon \to \infty$. When $\varepsilon = 0$, (2) collapses to mean health at age i. The iso-elastic functional form 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 $h_{EDE}(i)$, 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: $w\left(h_{EDE}(i)\right) = w\left(F\left(h_s(i)\right)\right)$. Solving gives [12]

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

Within the health-extended life-table cohort, independence is imposed between health at

[11] For $\varepsilon = 1$, $w\left(F\left(h_s(i)\right)\right) = \sum_s p_s(i) ln\left(h_s(i)\right)$.

[12] We calculate this metric over those alive at each age and so $h_s(i) > 0$. It is not necessary to invoke the SWF concept to derive this expression. It is simply the generalised mean of health. $\varepsilon = 0$, $\varepsilon = 1$, and $\varepsilon = 2$ give the arithmetic, geometric, and harmonic means, respectively.

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 $\tilde{l}(x) = \sum_{i=0}^{x-1} h_{EDE}(i)$. This is a measure of lifespan penalized for both mean health and health inequality at each age. If there were no aversion to differences in these health *distribution*-adjusted lifespans, then averaging them over the life-table cohort would give a population health measure that we label *restricted equivalent health-adjusted lifespan* ($REHAL$),

$$REHAL = \sum_{x=0}^{T} d(x)\tilde{l}(x). \tag{4}$$

This is $HALE$ with a penalty for age-specific health inequality that increases with aversion to that inequality.[13] An increase in age-specific health inequality, or in aversion to that inequality, increases the shortfall of $h_{EDE}(i)$ from $h(i)$, $\tilde{l}(x)$ from $l(x)$, and $REHAL$ from $HALE$.

2.1.3 Sensitivity to healthy lifespan inequality

$REHAL$ is sensitive to inequality in age-specific health but is insensitive to inequality in lifespans. To capture distributional sensitivity in both dimensions, we specify welfare as a nonlinear aggregation over health distribution-adjusted lifespans,

$$W\left(F\left(h_s(x)\right), D(x)\right) = \sum_{x=0}^{T} d(x)\frac{\tilde{l}(x)^{1-\eta}}{1-\eta}, \quad \eta \neq 1, \tag{5}$$

where $\eta \geq 0$ reflects the SDM's aversion to inequality in adjusted lifespans and $D(x) = \sum_{i=0}^{x} d(i)$.[14]

The function (5) is not a SWF defined over individuals' realised lifetime health profiles – health enjoyed at each age over a lifespan. To construct a distributionally sensitive measure of population health that is both feasible with data from a health-extended period life table and directly comparable (in the life-years metric) with LE and $HALE$, we take a two-stage

[13] This can be confirmed by substituting $h_{EDE}(i)$ for $h(i)$ in (1) to arrive at (4). When there is no aversion to age-specific health inequality ($\varepsilon = 0$), $REHAL = HALE$.

[14] With $\eta = 1$, $W\left(F\left(h_s(x)\right), D(x)\right) = \sum_{x=0}^{T} d(x) ln\left(\tilde{l}(x)\right)$.

approach. First, adjusting lifespans for the age-specific distributions of health by taking a generalised mean of health at each age (3). Then, aggregating over the distribution of these adjusted lifespans.[15] $HALE$ and some attempts to value population health taking account of both its morbidity and lifespan dimensions (Murphy and Topel 2006) limit attention to the special case that adjusts lifespan with the arithmetic mean of age-specific health ($\varepsilon = 0 \Rightarrow \tilde{l} = l$). 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 – that is enjoyed by those who die at a younger age relative to the same additional adjusted life year enjoyed by those who die at an older age. 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 $x + k, k > 0$ – leaving $REHAL$ constant – will increase the welfare measure W. Note that at any age x, there is no variation in $\tilde{l}(x)$. The aggregation in (5) is over (hypothetical) individuals with different lifespans. It is not over individuals with different lifetime health profiles. However, the aggregation is over lifespans adjusted for the level and distribution of health at each age. Consequently, the welfare measure is sensitive to changes in the age-specific distributions of health and to age differences in the distribution of health.[16]

Consider a health distribution-adjusted lifespan, $\tilde{l}_{EDE}$, that is defined such that if all

[15]Berger and Emmerling (2020) examine the general problem of welfare evaluation through aggregation over multiple dimensions, show how to do this using EDEs (or equity equivalents), and discuss when the order of aggregation matters. Closer to the current context, Echazu and Nocetti (2013) propose an approach to healthcare prioritisation with aversion to both individual exposure to health risk and inequality in ex ante utility (of health) over individuals. This involves first calculating the EDE health of individuals and then taking a concave aggregation of the EDE health over individuals. Precisely in the current context, with data from a health-extended life table, there is no inequality in ex ante health or lifespan over individuals. Our task is to aggregate over individuals at each age and over (health-adjusted) ages at death. We do this by first calculating the EDE health at each age and then taking a concave aggregation of the EDE health over ages.

[16]Consider ages x and $x + k$, $k > 0$, with an equal proportion of deaths at these ages, $d(x) = d(x + k)$. Holding the distribution of deaths constant, let there be a rise in mean health at x and fall of greater magnitude in mean health at $x + k$, such health distribution-adjusted lifespan changes by the same absolute amount, $\triangle\tilde{l}(x) = -\triangle\tilde{l}(x + k)$. There would be no change in $HALE$ or in $REHAL$. But, provided $\eta > 0$, $W\left(F\left(h_s(x)\right), D(x)\right)$ 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 greater rise in the variance at an older age could increase welfare without any change in mean health at each age or in the distribution of deaths over ages.

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: $W\left(\tilde{l}_{EDE}\right) = W\left(F\left(h_s(x)\right), D(x)\right)$. Solving for this EDE produces a more general equivalent health-adjusted lifespan,

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

This measure adjusts life expectancy for a) mean health at each age, as $HALE$ (1), b) health inequality at each age, as $REHAL$ (4), and c) inequality in health-adjusted lifespans. It collapses to the standard $HALE$ when there is no aversion to health inequality at each age and to inequality in adjusted lifespans, $\varepsilon = \eta = 0$. With no aversion to lifespan inequality ($\eta = 0$), it reduces to $REHAL$. With no aversion to age-specific health inequality ($\varepsilon = 0$) but with aversion to inequality in (mean) health-adjusted life years, it corresponds to a distributionally sensitive measure of population health that Norheim (2013) suggested but did not calculate.

2.1.4 Properties

$EHAL$ is monotonically increasing in both unadjusted life years and in life years adjusted for age-specific distributions of health, $\tilde{l}(x)$. The former monotonicity carries over from life expectancy: a shift in the distribution of deaths from younger to older ages increases the measure irrespective of (non-negative) aversion to lifespan variation. To understand monotonicity in adjusted life years,[17] note that $\tilde{l}(x)$ can increase due to a rise in mean health or a fall in health inequality at one or more ages up to x. If mean health increases at least at one age, and does not decrease at any age, and if health inequality is constant at all ages, then $EHAL$, like $HALE$, will increase, irrespective of aversion to health inequality. If health inequality falls at one or more ages, and does not rise at any age, then, provided there is aversion to health inequality ($\varepsilon > 0$), $EHAL$ will increase, while $HALE$ will not.

Aversion to age-specific health inequality is one channel through which $EHAL$ prioritizes

[17] $\frac{\partial EHAL}{\partial \tilde{l}(x)} = EHAL^{\frac{-\eta}{1-\eta}} d(x)\tilde{l}(x)^{-\eta} > 0 \ \forall x$.

gains to the worst off. In this case, the worst off corresponds to the least healthy state at a given age, ($h_1(x)$). 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, $\tilde{l}(x)$. 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, to an extent, the *fair innings* principle (Harris 2006) of favouring health and longevity gains to the young over same sized gains to the old.[18]

2.2 Distributional sensitivity in the money metric

To facilitate comparison with opportunity costs of investments in population health, we derive a money metric that is sensitive to the distributions of age-specific health and lifespan: willingness to pay (WTP) for $EHAL$.

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 and Quiggin 1999). The cost of this assumption is that we do not capture WTP for any indirect consumption benefits of health improvements that raise labour market productivity. In the application, we set the level of c at Gross Domestic Product (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: $U(c, F\left(h_s(x)\right)) = u(c)\sum_{i=0}^{x-1} h_{EDE}(i) = u(c)\tilde{l}(x)$. Multiplicative separa-

[18] $EHAL$ is not entirely faithful to this principle because it does not imply a discrete change in the priority afforded to an individual on reaching some threshold health-adjusted lifespan.

bility into welfare from consumption and health is a common restriction in derivations of WTP for health (see Hammitt 2013).[19]

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

$$W\left(c, F\left(h_s(x)\right), D(x)\right) = \sum_{x=0}^{T} d(x) \frac{U\left(c(x), F\left(h_s(x)\right)\right)^{1-\psi}}{1-\psi}$$
$$= \frac{u(c)^{1-\psi}}{1-\psi} \sum_{x=0}^{T} d(x) \tilde{l}(x)^{1-\psi} \qquad \psi \neq 1, \tag{7}$$

where $\psi \geq 0$ represents the degree of inequality aversion.

A monotonic transformation of (7) preserves the SDM's preference ordering of health and age-at-death distributions. We apply $V = (1 - 2 \cdot \mathbb{1}(\psi > 1))((1-\psi)W)^{\frac{1}{1-\psi}}$, where $\mathbb{1}()$ is the indicator function, to get

$$V\left(c, F\left(h_s(x)\right), D(x)\right) = u(c) \left[\sum_{x=0}^{T} d(x) \tilde{l}(x)^{1-\psi}\right]^{\frac{1}{1-\psi}}. \tag{8}$$

If we assume that the degree of aversion to inequality in lifetime welfare is equal to the degree of aversion to inequality in health distribution-adjusted lifespans, $\psi = \eta$, then (8) gives $V\left(c, F\left(h_s(x)\right), D(x)\right) = u(c) EHAL$. That is, social welfare is equal the number of $EHALs$ generated by the inequality-penalized distributions of age-specific health and ages at death scaled by the welfare from consumption.

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 $D(x)$ to $D^*(x)$. The WTP for such a change is defined implicitly by $V\left(c, F\left(h_s(x)\right), D(x)\right) = V\left(c - WTP, F^*\left(h_s(x)\right), D^*(x)\right)$. 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 and Topel 2006).[20] The parameter $\underline{c}$ is a subsistence level of consumption at which there is indifference between life in full health and death

[19] 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 and Quiggin 1999).

[20] $u(c) = lnc$ for $\gamma = 1$

(Rosen 1988). It arises from the normalisation of utility when dead to zero. Larger values of $\underline{c}$ imply a lower value of being alive relative to dead. 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 and Jones 2007). This effect is stronger at a larger value of γ since the marginal utility of consumption declines more steeply with rising consumption.[21]

Solving, we get

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

where $EHAL$ and $EHAL^*$ are obtained from (6) applied to the distributions $F\left(h_s(x)\right)$ and $D(x)$ and to $F^*\left(h_s(x)\right)$ and $D^*(x)$, respectively.

WTP for a proportionate change in the $EHAL$ measure of population health depends on three social preference parameters that reflect aversion to inequality in age-specific health (ε), aversion to inequality in lifespan (η), and the curvature of consumption utility (γ), as well as on the level of consumption in relation to subsistence ($c \,\&\, \underline{c}$).

Aversion to lifespan inequality ($\eta > 0$) 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 inequality at any age with all else held constant. And for any given reduction in age-specific health inequality, WTP is rising with the value of ε.

If there were no aversion to both health and lifespan inequality ($\varepsilon = \eta = 0$), then (9) would reduce to the WTP for a change in health-adjusted life expectancy:

$$WTP = c - \left[\left(\frac{HALE}{HALE^*}\right)\left(c^{1-\gamma} - \underline{c}^{1-\gamma}\right) + \underline{c}^{1-\gamma}\right]^{\frac{1}{1-\gamma}} \tag{10}$$

[21] Through this mechanism, an anticipated rise in future consumption that accompanies 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.

where $HALE$ and $HALE^*$ are obtained from (1) applied to the distributions $F\left(h_s(x)\right)$ and $D(x)$ and to $F^*\left(h_s(x)\right)$ and $D^*(x)$, respectively. This solution is similar to the valuation of infra-marginal changes in lifespan proposed by others (Becker, Philipson, and Soares 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 \$ 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 improvements 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. However, it would be unreasonable to expect that a low-income country would be willing to pay in excess of its income, and so accumulate debt, in order to achieve gains in population health (Sunstein 2014). 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.

2.3 Aggregation issues

$EHAL$ and WTP are summary measures of the prevailing distributions of morbidity and mortality that are captured by a health-extended period life table. They pay no attention to the morbidity and mortality that currently living individuals were exposed to when they were younger. And they ignore the health of future generations. These are limitations. But they are not limitations compared with the distributionally insensitive measures of population health — LE and $HALE$ — that we seek to extend. Aggregating over cohorts — as opposed to confining attention to a hypothetical period life-table cohort — would raise methodological and ethical issues. For example, 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 change in the size of a population in response to health improvement and sidestep Parfit's (1984) repugnant conclusion — favouring a larger population in very poor health over a smaller population in good health.

3 Data and method

3.1 Global Burden of Disease data

We use the measures introduced in the previous section to quantify contributions of diseases to levels and trends of population health in Sub-Saharan Africa (SSA). All data are from the 2017 Global Burden of Disease (GBD) that provides estimates for 293 disease causes disaggregated by age and sex covers for each year from 1990 to 2017 (GBD 2018; Murray et al. 1996). There are data on all 46 countries in the SSA region (including new states, such as South Sudan) for the entire period. We use aggregated SSA-level data for each age-sex

partition. We construct all measures separately for males and females.[22]

For each disease and age-sex partition, we observe rates of prevalence and mortality rate, and disability adjusted life years (DALYs). We use the data on prevalence and DALYs to recover the underlying disease-specific disability weights that are on a 0-1 scale, with 1 representing death and 0 indicating no impairment (full health). We reverse the scale to get a measure of health in each disease state for each age (and sex). We assign diseases to simulated individuals on the basis of the sex- and age-specific disease prevalence rates. Attaching the measure of health to the assigned diseases gives a simulated distribution of health for each age (and sex). We repeat the procedure with the disability weight associated with a particular disease set to zero in order to simulate the impact of eliminating that disease on the distribution of health at every age.

Application of disease-, sex- and age-specific mortality rates to the life-table cohort generates a distribution of ages at death. We then set mortality from a particular disease to zero at all ages and so generate a counterfactual age-at-death distribution if mortality from that disease were eliminated. Using the counterfactual distributions of health and ages at death, we calculate $HALE$, $EHAL(\varepsilon)$, and $EHAL(\varepsilon, \eta)$ that would be obtained if the disease were eliminated, and we calculate the WTP to eliminate the disease. We now discuss each of these steps in more detail.

3.2 Health distributions

Calculation of the measures given by (1), (**??**), and (**??**) requires a distribution of health at each age. From the disease-level GBD data, we simulate an individual-level dataset in which diseases are assigned to simulated individuals on the basis of age-sex specific prevalence rates. Within each of 42 sex- and age-specific partitions, we simulate 100,000 individuals, and use prevalence rates for 293 causes of disease to randomly assign diseases to simulants.

[22]Stratifying by sex allows us to remain agnostic about weights placed on sex-specific diseases, such as prostate and ovarian cancers. Without sex stratification, measures would reflect the sex composition of the cohort and how it changes with age.

Within each partition, we follow the GBD in assuming independence between diseases: the probability of being assigned disease k does not change if disease j is already assigned. 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 the sex and age partitions. But given the absence of more explicit allowance for comorbidities, our approach, like that of the GBD, should be considered a first order approximation of the true distribution of health outcomes at each age.

From the data on disease-specific DALYs, for each disease, we extract the respective disability weight that indicates the proportionate health loss associated with that disease (holding years lived constant). As mentioned in section **??**, the GBD disability weights are intended to measure health impairment, not preferences (Salomon et al. 2003).[23] The health of a simulant that is assigned a set K of diseases at age i is

$$h_s(i) = \prod_{k \in K} (1 - z_k(i)), \tag{11}$$

where $z_k(i)$ is the disability weight for disease k at age i.

Note that we define a health state ($h_s(i)$) by a set of diseases, not a particular disease. If a simulant is unique in the combination of diseases it is assigned, then $p_s(i) = 1/100,000$ given that we simulate 100,000 individuals at each age. Otherwise, $p_s(i)$ is the proportion that shares the same combination of diseases. Together, $h_s(i)$ and $p_s(i)$ $\forall s$ define the distribution of health at each age. We identify the effect of eliminating disease k on the health distribution by setting $z_k(i) = 0$ $\forall i$, holding constant $z_j(i)$ $\forall j \neq k$.

Figure 1 shows simulated health distributions for six sex-age partitions. For both sexes,

[23] 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 (**salomon2012common**). 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 zero value for death by asking some respondents to say which of two programmes would general 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.

the health distribution shifts to the left and becomes more dispersed in moving from younger to older groups. Older people face lower average health and higher health risk.

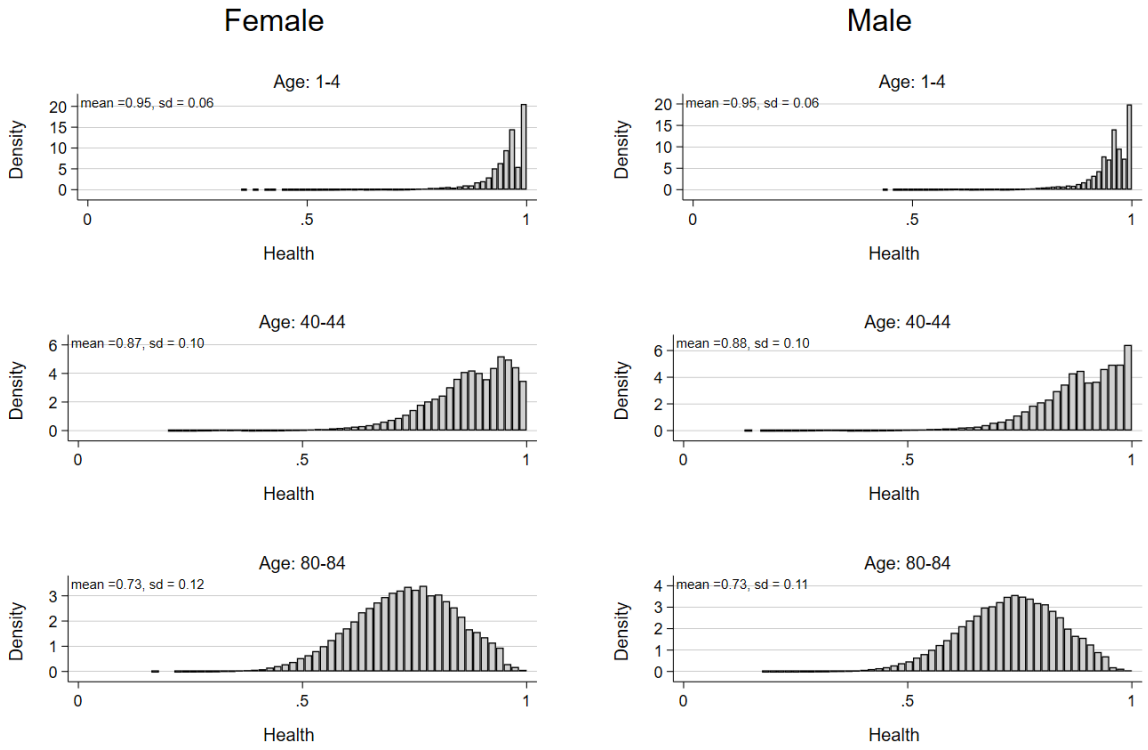

Figure 1: Simulated health distributions by sex and age

Notes: Simulated distribution of health $f\left(h_s(i)\right)$ obtained from 100k simulants within each sex-age partition assigned diseases according to sex-age specific prevalence.

3.3 Age-at-death distributions

For each sex, we construct a distribution of deaths by age from sex- and age-specific mortality rates in the GBD. The data give the all-cause mortality rate at each age x: $M(x) = \frac{D(x)}{P(x)}$, where $D(x)$ is the number of deaths observed in the population at age x and $P(x)$ is the size of the population in the middle of the reference year. For a cohort, the probability of dying in the age interval $[x, x+1)$, conditional on survival to x, is $\mu(x) = \frac{D(x)}{N(x)}$, where

$N(x)$ is the number in the cohort that survives to x.[24]. This number is given by $N(x) = P(x) + (1 - a(x))D(x)$, where $a(x)$ is the average proportion of the age interval survived by those who die within it (Chiang 1968). Substitution gives $mu(x) = \frac{M(x)}{1+(1-a(x))M(x)}$, which we calculate using the GBD values of $M(x)$ and age-sex specific values of $a(x)$ for SSA from the UN World Population Prospects (Nations 2019). Successively applying these conditional probabilities of death at each age yields the distribution of deaths across ages and the proportion of the cohort dying at each age:[25]

$$d(x) = \mu(x) \prod_{i=0}^{x-1} (1 - \mu(i)) \tag{12}$$

Calculation of a counterfactual age-at-death distribution after the elimination of a disease makes use of the fact that the GBD all-cause mortality rate is an additive sum of disease specific mortality rates: $M(x) = \sum_k \frac{D_k(x)}{P(x)} = \sum_k M_k(x) =$, where $D_k(x)$ is the number of deaths due to disease k at age x. Then, the counterfactual mortality rate after elimination of mortality caused by disease k is $M^{-k}(x) = M(x) - M_k(x)$. These counterfactual mortality rates are transformed into corresponding counterfactual conditional probabilities of death $\mu^*(x)$ (Chiang 1968) and the proportion of cohort deaths at each age: $d^*(x) = \mu^*(x) \prod_{i=0}^{x-1} (1 - \mu^*(i))$.[26]

We use the counterfactual age-at-death distributions and the respective counterfactual age-specific health distributions to calculate the impact that elimination of any disease would have on LE, $HALE$, and $EHAL$, as well as the WTP to eliminate each disease. In common with other studies that use disease specific mortality rates and health-extended life tables to simulate the contributions of diseases to measures of population health, our approach only considers the first order effects of eliminating a disease. It assumes that if cause-specific

[24]To keep things simple, we explain the method here for an age interval of one year, although the analysis is done using wider age groups. See Appendix B for the method with an age intervals of size n and for more details on calculation of actual and counterfactual age-at-death distributions

[25]More precisely, this is the proportion dying in the interval $x + a(x)$.

[26]We use the same values of $a(x)$ in the actual and counterfactual scenarios. While this may introduce some bias, it should be small given the highly disaggregated disease level data and the limited impact of these values on overall life expectancy (Preston, Heuveline, and Guillot 2001).

mortality rates were set to zero, the corresponding rates from other causes would remain unchanged — there is mutual exclusivity. This could bias our estimates downward if one disease increases mortality from other causes. On the other hand, if individuals who would have died from a disease that is eliminated would have died in any case from another cause at the same age, 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 have been observed after the elimination of a diseases.

3.4 Parameter values

To calculate the measures, we must choose values for the welfare and utility function parameters: ε, η, γ, ψ, and $\underline{c}$. We first note that ε and η are defined over the same measurement scale, (equivalent) years of life. For this reason, and because we are unaware of studies assessing aversion to inequalities in health status (ε), we restrict $\varepsilon = \eta$.[27] Furthermore, since our assumptions in **??** allow us to define ψ directly over health adjusted lifespans rather than lifetime utilities, we impose the constraint that $\eta = \psi$.[28] Concerning aversion to lifespan inequalities, estimates of the Atkinson inequality aversion parameter (η) vary from as low as 1.2 to as high as 28 (Dolan and Tsuchiya 2011; Hurley, Mentzakis, and Walli-Attaei 2020; O'Donnell, Robson, and Van Ourti 2022; Robson et al. 2017). However, many of these studies capture aversion to health inequalities associated with socio-economic status. Fewer studies consider aversion to "pure" inequalities in lifespan Hurley, Mentzakis, and Walli-Attaei 2020; Pinho and Botelho 2018.). Hurley, Mentzakis, and Walli-Attaei 2020 find that the degree of aversion to this type of inequality may be lower than aversion to inequalities related to socio-economic status. This implies that some of the large estimates elicited in the existing literature may be unsuitable for analysing distributions of "pure" lifespan.

[27]Some studies have used revealed or stated preferences to estimate constant relative risk aversion over health prospects. The limited evidence available suggests a value between 0 and 1, with some estimates above this range (Herrera-Araujo, Hammitt, and Rheinberger 2020).

[28]We are unaware of any studies directly eliciting this parameter. However, there are studies that concerning a related concept: risk aversion over the lifespan. Delprat, Leroux, and Michaud 2016, for instance, elicit a risk aversion parameter that is relatively low among the participants.

An alternative approach is to look at the preferences of decision makers and institutional bodies rather than the public. The United Nations Development Programme (UNDP), for instance, utilises the Atkinson inequality index with $\eta = 1$ (i.e., the geometric mean) to capture inequalities in lifespan within its inequality adjusted human development index (IHDI) (see Alkire and Foster 2010). The IDHI is regularly used to rank countries in the UNDP's annual human development report and therefore could serve as an acceptable benchmark to set within our framework. However, the rationale for the choice of = 1 seems to be based more on the convenient analytical properties of the geometric mean rather than any strong normative foundation. Consequently, given the varying evidence on η, we opt for values of 0.5 and 2 to capture a range of aversion attitudes.

The curvature of consumption utility can be inferred from the inter-temporal elasticity of substitution (IES) (1) or constant relative risk aversion (CRRA) over consumption (). Most estimates of IES range from 0.5 to just above 1 (Browning, Hansen, and Heckman 1999; Hall 1988; Havranek et al. 2015), implying values of roughly between 1 and 2. There is some evidence that IES is smaller (larger) in low-income countries (Atkeson and Ogaki 1996; Havranek et al. 2015; Ogaki, Ostry, and Reinhart 1996), possibly because spending a large fraction of tightly constrained budgets on necessities limits opportunities for inter-temporal substitution. 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. We follow Murphy and Topel 2006 in setting = 1.25.

For the level of consumption (c), we use GDP per capita for SSA. To our knowledge, there is no direct evidence on the level of subsistence consumption that leaves someone indifferent between life (in full health) and death ($\underline{c}$). Its value is often inferred from estimates of the value of a statistical life (VSL) (Hall and Jones 2007; Jones and Klenow 2016; Murphy and Topel 2006). However, there are few reliable estimates of the VSL in SSA. Sometimes $\underline{c}$ is set to zero (**crafts2003welfare**; Murphy and Topel 2003; Usher 1973), which implies that any life is worth living regardless of the consumption achieved. Some studies have opted for

a value close to the international poverty line (Becker, Philipson, and Soares 2005) but lives below this threshold are generally considered to be worth living (see Cookson et al. 2020. We set $\underline{c}$ at 10% of GDP per capita, also in line with Murphy and Topel (2006).

4 Results

4.1 Levels and trends of population health

Table 1 shows estimates for females and males of life expectancy (LE), health-adjusted life expectancy ($HALE$), and our measure of equivalent health-adjusted lifespan ($EHAL$) for four configurations of aversion to inequality in age-specific health and lifespan. We show estimates for 1990, 2004, and 2017 using the respective health-extended period life table. Between 1990 and 2017, life expectancy at birth in SSA increased by 10.7 years for females and 10.2 years for males. Adjusting LE for mean health at each age gives estimates of $HALE$ that imply that the average female born in SSA in 2017 could expect to live for the equivalent of 57.8 years in full health. The respective estimate for males is 54.45 years. Between 1990 and 2017, the relative increase in $HALE$ was approximately the same as the relative increase in LE for both females and males (19-20%). Adjusting for age-specific health inequality using a low degree of inequality aversion ($\varepsilon = 0.5$), while continuing to assume no aversion to lifespan inequality ($\eta = 0$), gives values of $EHAL$ that are 0.14-0.19 years (1.7-2.3 months) in full health below the respective $HALE$ values. Raising the degree of aversion to age-specific health inequality to $\varepsilon = 2$ brings $EHAL$ below $HALE$ by 0.6-0.9 of a year. Adjusting life expectancy to take account of mean health at each age ($LE \rightarrow HALE$) clearly has a much greater impact than adjusting for age-specific health inequality ($HALE \rightarrow EHAL(\varepsilon > 0, \eta = 0)$. This may be surprising given there is substantial variation in health, particularly at older ages (Figure 1). A possible explanation is that only a small proportion of the SSA population is expected to survive to old age.[29] For both

[29]Inconsistent with this explanation, applying mortality rates of (World Bank defined) high-income countries to the SSA data gives a similar result: adjusting for inequality in age-specific health but not adjusting

females and males, the relative improvement in population health between 1990 and 2017 is the same irrespective of whether it is measured using life expectancy (LE), life expectancy adjusted for mean health ($HALE$), or life expectancy adjusted for mean health and health inequality at each age ($EHAL(\varepsilon > 0, \eta = 0)$).

Adjustment for inequality in health-adjusted lifespan has a much greater impact. Setting the respective inequality aversion parameter (η) even to 0.5 produces a difference of more than 4 years between $EHAL(\varepsilon = 0.5, \eta = 0)$ and $EHAL(\varepsilon = 0.5, \eta = 0.5)$ in 2017 for both males and females. In 1990, the respective difference is around 7 years for both sexes, which is similar to the difference between LE and $HALE$ in the same year. That is, adjusting the measure of population health for inequality in lifespan has roughly the same absolute effect as adjusting life expectancy for mean health. For the relative change in population health between 1990 and 2017, the effect of adjusting for inequality in lifespan is even greater than the effect of adjusting life expectancy for mean health. The latter adjustment effectively has no impact: both LE and HALE increased by roughly 20% for males and females over the period. But the increase in ($EHAL(\varepsilon = 0.5, \eta = 0.5)$) is around 31% for males and 29% for females. These increases reflect the that fact that gains in LE have been largely driven by reductions in both infant mortality and HIV-related mortality among younger adults, which have condensed the age-at-death distribution. Raising the lifespan inequality aversion parameter to 2 reduces $EHAL(\varepsilon, \eta)$ dramatically. At this parameter value, substantial weight is placed on infant deaths when aggregating over the age distribution of deaths.

4.2 Disease burdens

We now turn to the burdens of specific disease estimated in both the life-years metric and the money metric. In each case, we compare distributionally sensitive estimates with estimates that are obtained without taking any account of how a disease affects dispersion in health and lifespan. In the life-years metric, we do this by comparing the change in $EHAL(\varepsilon, \eta)$

for inequality in lifespan has relatively little impact, i.e. $HALE - EHAL(\varepsilon > 0, \eta = 0)$ is relatively small (close to 0.2 when $\varepsilon = 0.5$, and close to 1 if $\varepsilon = 2$).

Table 1: Life-years measures of population health

	1990	2004	2017	2017-1990 Δ	2017-1990 %Δ
Female					
LE	55.65	55.87	66.31	10.66	19.2%
HALE	48.34	48.67	57.78	9.44	19.5%
EHAL(ε=0.5,η=0)	48.18	48.51	57.59	9.41	19.5%
EHAL(ε=2,η=0)	47.61	47.92	56.90	9.29	19.5%
EHAL(ε=0.5,η=0.5)	41.31	42.84	53.37	12.06	29.2%
EHAL(ε=2,η=2)	3.11	3.42	3.61	0.5	16.1%
Male					
LE	51.53	53.01	61.72	10.19	19.8%
HALE	45.33	46.70	54.45	9.12	20.1%
EHAL(ε=0.5,η=0)	45.19	46.56	54.28	9.09	20.1%
EHAL(ε=2,η=0)	44.69	46.02	53.67	8.98	20.1%
EHAL(ε=0.5,η=0.5)	37.92	40.53	49.64	11.72	30.9%
EHAL(ε=2,η=2)	2.56	3.01	3.14	0.58	22.7%

Note: LE=Life Expectancy, HALE=Health Adjusted Life Expectancy, EHAL=Equivalent Health Adjusted Lifespan.

that would occur if a disease were eliminated with the respective change in $HALE$. The left panels of Figure 2 shows these estimates, for 2017 and for each sex, for the 20 diseases with the largest burdens measured by the increase in $HALE$ that would occur if each disease were eliminated (lighter shaded bars).[30] The darker shaded bars in the left panels show the respective increases in $EHAL$ with both inequality aversion parameters set to 0.5.

For both females and males, the largest differences between the disease burdens measured by $HALE$ and $EHAL$ are for diseases that primarily affect neonates, infants, and young children: lower respiratory infections (LRI), diarrhea, malaria, neonatal encephalopathy, neonatal preterm birth, and protein energy malnutrition. Elimination of each of these conditions would increase the distributionally sensitive $EHAL$ by substantially more than it would increase $HALE$. This is because eliminating diseases that affect mortality primarily at young ages would not only increase the expected length of life in good health, it would also reduce the dispersion in lifespans as more children survive into adulthood.

[30]See Figures **??** and **??** in Appendix C for the respective changes in disease burdens in 1990 and 2004.

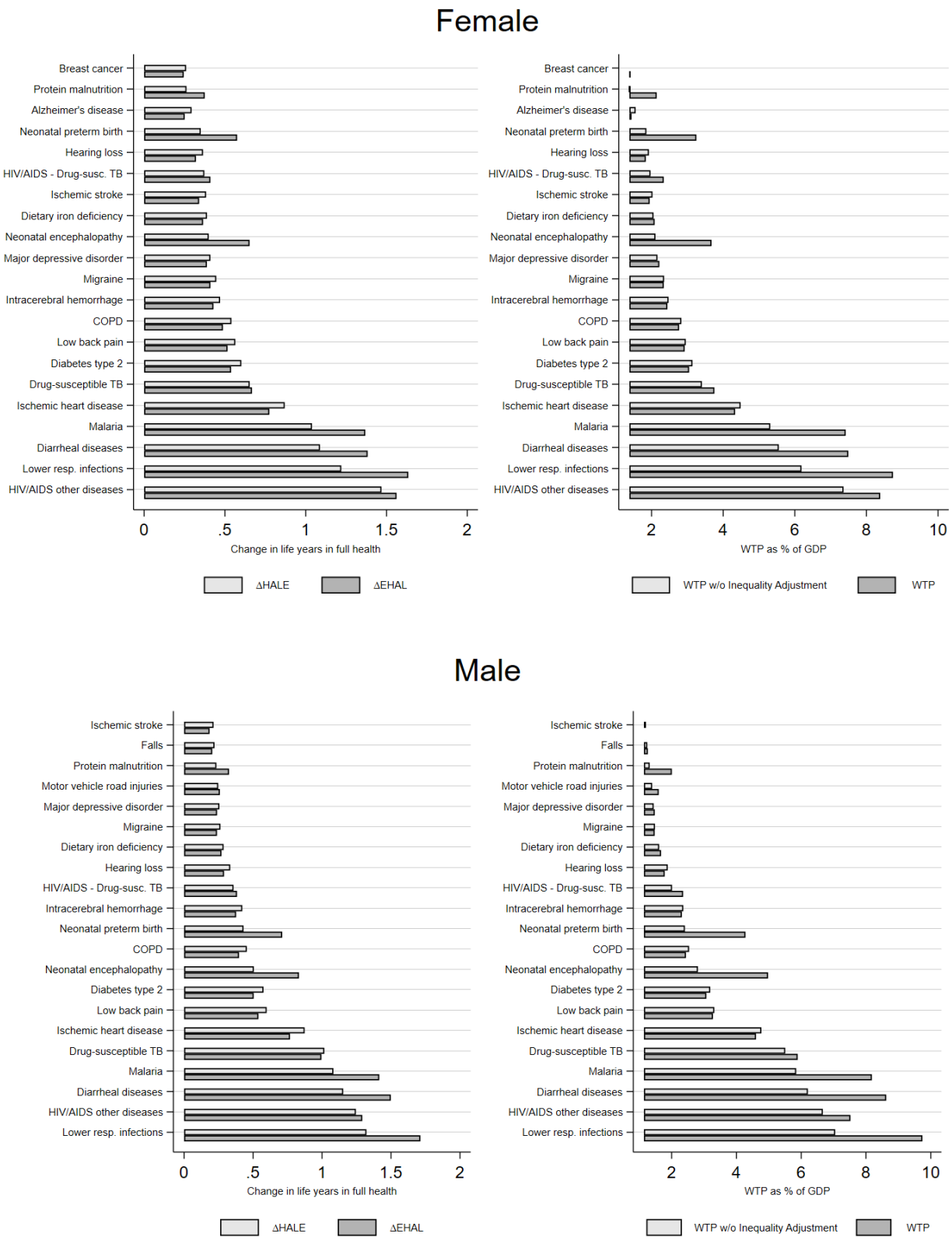

Figure 2: Life-years and money metrics of disease burdens with and without distributional sensitivity, top 20 diseases, 2017

Notes: The left panel shows increases in $HALE$ and $EHAL(\varepsilon = 0.5, \eta = 0.5)$ from elimination of each disease for the 20 diseases with the largest increases in $HALE$. The right panel shows WTP to eliminate each disease calculated from (10) (light shading) and from (9) with $\eta = \psi = 0.5$ (dark shading). For both WTP estimates, $\gamma = 1.25$, c =GDP per capita, and c =10% GDP per capita.

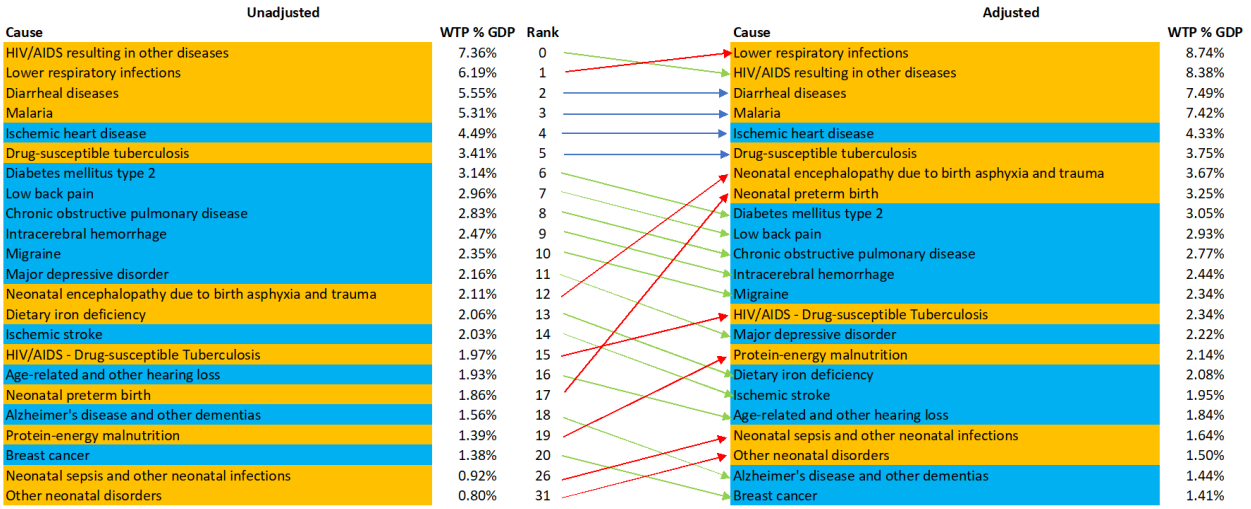

Figure 3: Female top 20 diseases ranked by distributionally insensitive and sensitive WTP, 2017

Notes: Distributionally insensitive WTP from (10). Distributionally sensitive WTP from (9) with $\theta = \psi = 0.5$. In each case, $\gamma = 1.25$, c =GDP per capita, and $\underline{c} = 10\%$ of GDP per capita. Diseases that are in the top 20 either by distributionally insensitive WTP or distributionally sensitive WTP included. Diseases are order from top to bottom by distributionally insensitive WTP.

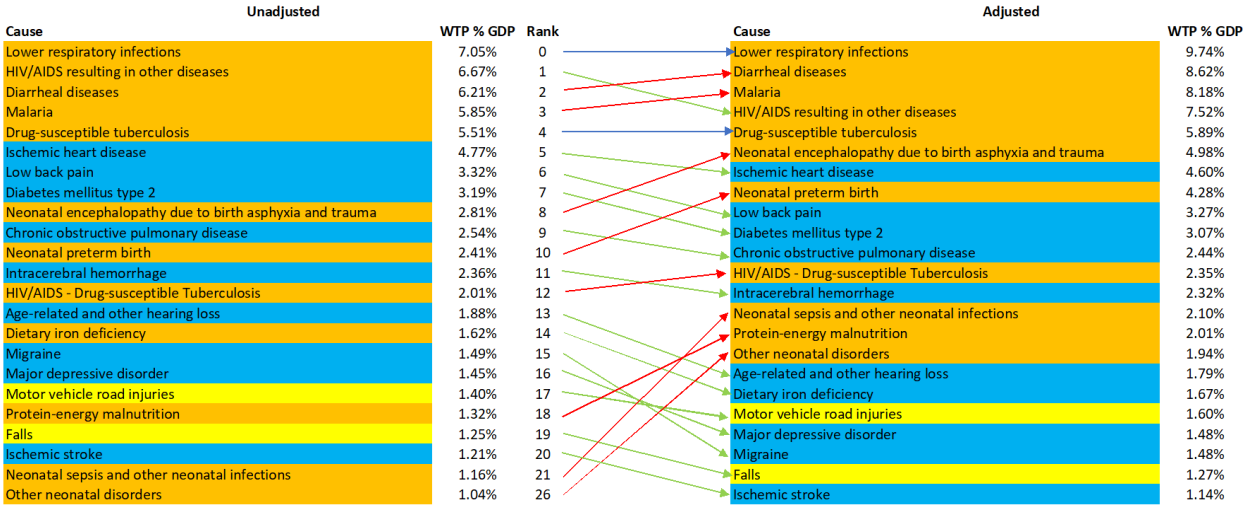

Figure 4: Male top 20 diseases ranked by distributionally insensitive and sensitive WTP, 2017

Notes: Distributionally insensitive WTP from (10). Distributionally sensitive WTP from (9) with $\theta = \psi = 0.5$. In each case, $\gamma = 1.25$, c =GDP per capita, and $\underline{c} = 10\%$ of GDP per capita. Diseases that are in the top 20 either by distributionally insensitive WTP or distributionally sensitive WTP included. Diseases are order from top to bottom by distributionally insensitive WTP.

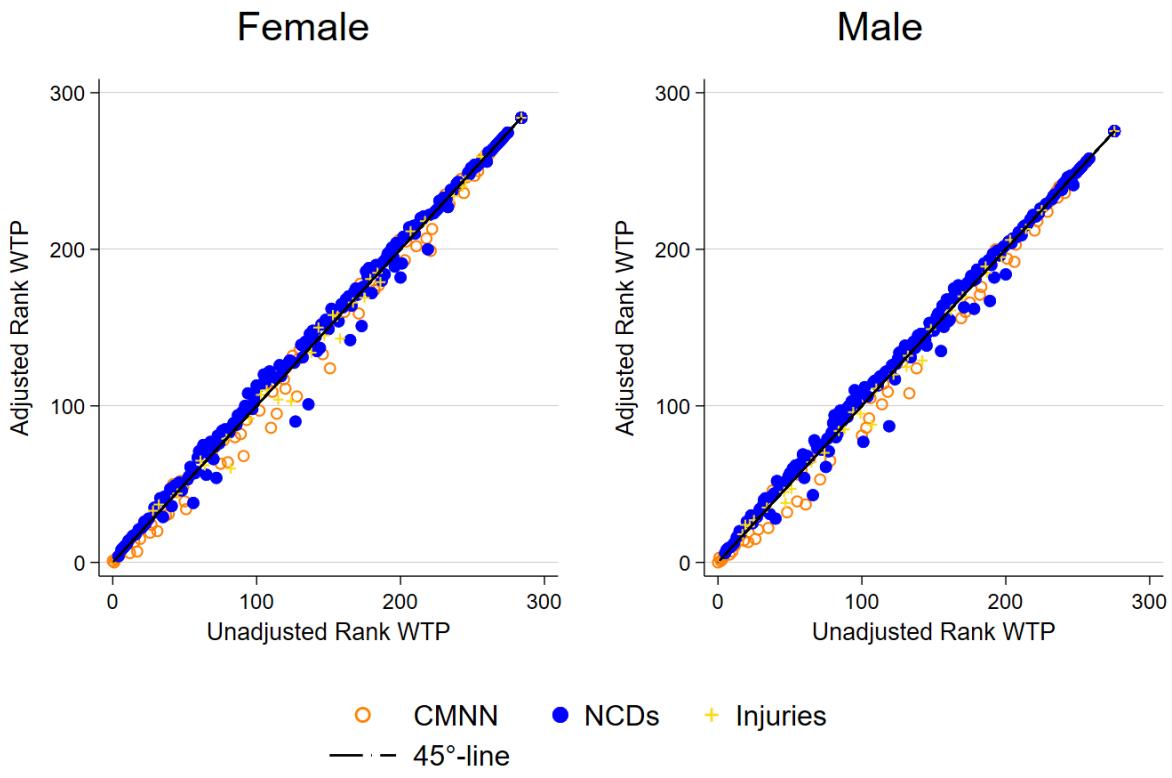

Figure 5: All diseases ranked by distributionally insensitive and sensitive WTP, 2017

Notes: Distributionally insensitive WTP from (10). Distributionally sensitive WTP from (9) with $\eta = \psi = 0.5$. In each case, $\gamma = 1.25$, c =GDP per capita, and $\underline{c} = 10\%$ of GDP per capita. Lower numbers indicate higher ranking (larger WTP).

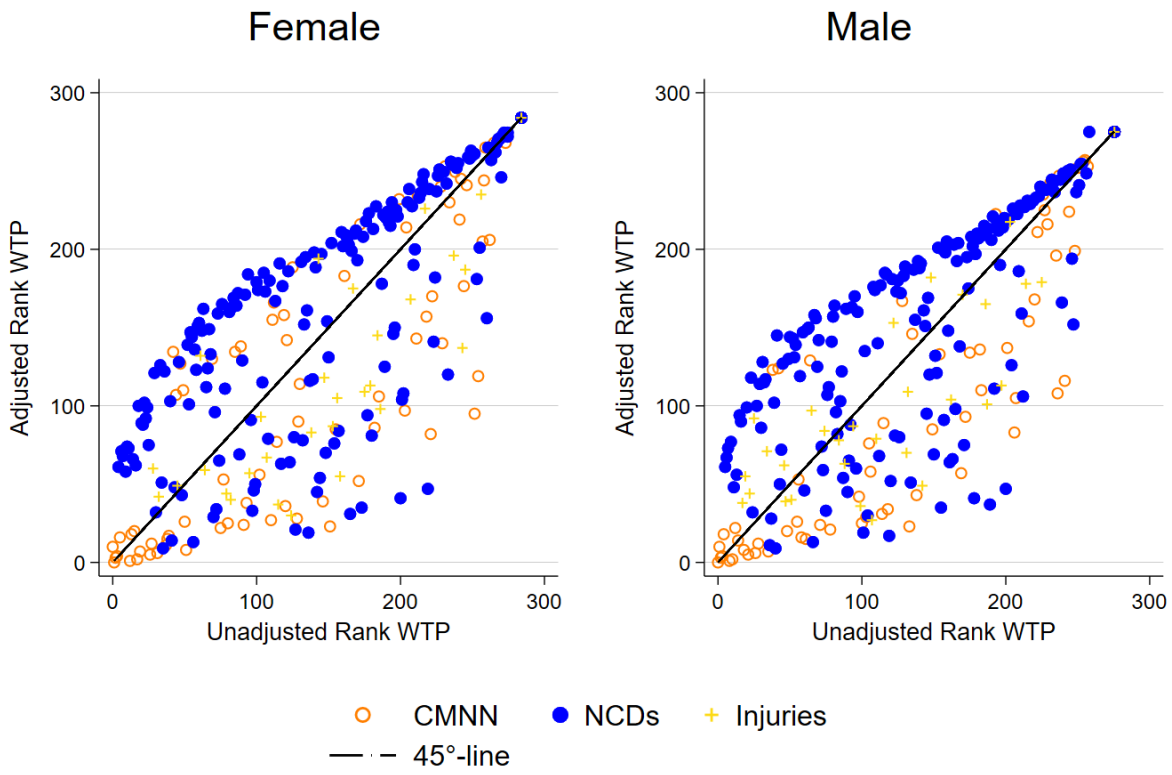

Figure 6: All diseases ranked by distributionally insensitive and sensitive WTP, 2017

Notes: Distributionally insensitive WTP from (10). Distributionally sensitive WTP from (9) with $\eta = \psi = 2$. In each case, $\gamma = 1.25$, c =GDP per capita, and $\underline{c} = 10\%$ of GDP per capita. Lower numbers indicate higher ranking (larger WTP).

The right panels of Figure 2 shows WTP to eliminate diseases in 2017.[31] Each estimate is the percentage of GDP the sub-Saharan African SDM would be willing to pay to eliminate the morbidity and mortality caused by that disease. The lighter shaded bars show WTP without considering the SDM's aversion to health and lifespan dispersion that would result from elimination of the disease. These estimates are calculated using (10), with $\eta = \psi = 0$. The ranking of the 20 diseases (for each sex) that the representative agent would be willing to pay most to eliminate is the same as the ranking of the 20 diseases that would increase $HALE$ by most if they were eliminated. The darker shaded bars in the right panel show WTP inclusive of the value attached to reduced exposure to dispersion in age-specific health and

[31]See Appendix B, **??** and **??** for the respective figures for 1990 and 2004

lifespan as a consequence of the elimination of each disease. These estimates are calculated using (9), with $\eta = \psi = 0.5$.

Without taking account of inequality aversion, we estimate that per capita WTP to eliminate lower respiratory infection would be 6.2% of GDP per capita for females and a little more than this for males (7.1%). Taking account of aversion with respect to age-specific health risk and lifespan risk would raise WTP to 8.7% of GDP for females and around 9.7% for males. Taking account of inequality aversion raises WTP to eliminate diarrheal diseases from 5.6% to 7.5% of GDP for females and from 6.2% to 8.6% for males. We estimate that WTP to eliminate malaria is 5.3% of GDP without allowing for inequality aversion and 7.4% with inequality aversion for females. The respective estimates for males are 5.9% and 8.2%. Taking account of inequality aversion has a more modest impact on WTP to eliminate HIV/AIDS for both sexes, which reflects the fact that mortality from this disease peaks at an older age than the largely childhood illness of LRI, diarrhea, and malaria.

Taking account of inequality aversion has a much smaller impact on WTP for the elimination of NCDs and even decreases WTP for some. For example, WTP to eliminate each of low back pain and diabetes is stable around 3% of GDP with and without taking into account the dispersion of health and lifespan. The reductions in WTP for example ischemic stroke are because the condition is most prevalent in middle- and old-age. Elimination would mostly benefit those who already enjoy more than the average equivalent life-years in full health, and so increase dispersion in health distribution adjusted lifespan. The WTP for low back pain among women is approximately the same when unadjusted for health inequality (3.32%) and when adjusted (3.27%). Low back pain does not affect age-at-deah, but has impact on health at older age. Hence, the minor change is consistent with earlier findings that adjusting for health inequality matters less compared to adjustments for length of life inequality. Taking account of health and lifespan dispersion effects increases the WTP to eliminate many communicable diseases that are most prevalent at younger ages and decreases WTP to eliminate NCDs that are most prevalent at older ages. Consequently, the

relative value placed on the elimination of communicable diseases increases markedly when consideration is given to distributional effects. For example, for males, the ratio of WTP to eliminate LRI to WTP to eliminate diabetes rises from 2.0 to 2.9.

Figures 3 and 4 show the extent to which taking account of distributional effects changes the ranking of disease burdens measured. Changes in ranks are consistent between the $EHAL$ and the money metric. The left panels show the top 20 diseases ranked by WTP with no adjustment for effects of disease elimination on the dispersion of health and lifespan. Shading is used to distinguish between communicable diseases, NCDs, and accidents and injuries. The right panel shows rankings by WTP adjusted to account for distributional sensitivity. Clearly, the distributional adjustment predominantly results in communicable diseases and other conditions most prevalent among infants and children moving up the league table and NCDs moving down. For females, LRI would move from being the second most burdensome disease to the most. For males, LRI remains at the top of the population health impact league table, while both diarrhea and malaria would move above HIV/AIDs. For both sexes, neonatal conditions would leapfrog a number of NCDs, such as COPD, diabetes, and low back pain, and they would draw approximately level with ischemic heart disease. Figure 6 extends this analysis to all 296 diseases. It is seen that elimination of many NCDs would increase $EHAL$ by less than their elimination would increase $HALE$. Many CDs lie below the 45 degree line, illustrating that the rank after inequality adjustment decreases relative to the rank before adjustment. Since lower ranks represent the highest disease burdens, CDs gain in importance. This is because these conditions impact on health and mortality primarily at older ages. Hence, if they were to be eliminated, then inequality in lifespans would rise and this would partially offset the increase in $HALE$ that arises from the increase in mean health and average age at death.

Additional results in Appendix C confirm that adjusting $EHAL$ and WTP for distributional sensitivity matters the most for communicable diseases, primarily those diseases that affect younger individuals. Increasing the parameters capturing inequality aversion to

2 drastically changes the results. Relative to WTP values without adjustment, adjusting for inequality aversion in health and longevity drastically increases WTP values for early age diseases.

4.3 Trends in the burden of disease

Since 1990, the prevalence of communicable diseases (CDs) has been falling, while the prevalence of NCDs has risen. When no attention is paid to the impact on health and lifespan dispersion, the burden of disease in SSA is shifting from CDs to NCDs. This is seen in the left panel of Figure 7, which shows estimates of aggregated disease burdens — the increase in $HALE$ that would result from eliminating CDs and NCDs in each year. In 1990, eliminating CDs would add the equivalent of around 14 years in full health for both males and females. The respective increase from eliminating NCDs is estimated to be 8-9 years. Hence, the CD burden as a ratio of the NCD burden was 1.6 for females and 1.8 for males. By 2017, NCD burden was slightly larger than the CD burden for both sexes. This is consistent with Gouda et al. (2019) who estimate that the percentage of total DALYs due to NCDs increased from 18.6% in 1990 to 29.8% in 2017.

The middle panel of Figure 7 shows estimates of increases in $EHAL(\varepsilon = 0.5, \eta = 0.5)$ from eliminating CDs and NCDs. Once allowance is made for even a modest degree of aversion of inequality in (age-specific) health and lifespan, the decline in the burden of CDs slows and it remains larger than the NCD burden in 2017 for both sexes. If the degree of inequality aversion is raised to 2 for both health inequality and lifespan inequality, then the burden of CDs starkly drops because ilnesses striking at young ages are weighted heavily, but it remains very large relative to the burden of NCDs through the period.

Figure 8 shows similar patterns of results in the money metric. Without taking account of effects on health and lifespan dispersion, elimination of the CD burden is valued at a little more than 90% of GDP for both sexes in 1990. By 2017, this distributionally insensitive valuation of the burden of CDs had fallen to around 50% of GDP for both sexes, and

was less than the value of eliminating NCDs. With a modest degree of inequality aversion ($\eta = \psi = 0.5$), the value of eliminating CDs remains greater than the value of eliminating NCDs in 2017. And with greater inequality aversion ($\eta = \psi = 2$), the monetary value of the burden of CDs is multiples of the burden of NCDs even in 2017.

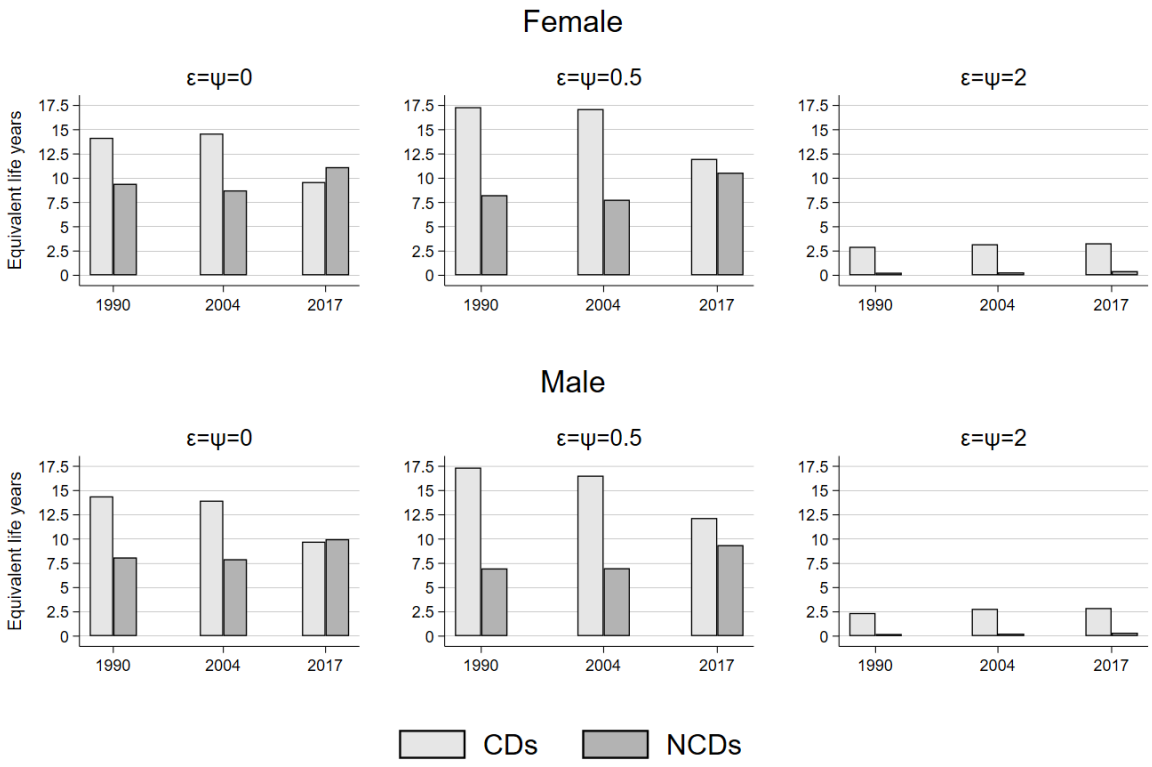

Figure 7: Communicable and non-communicable disease burdens - life-years metric

Notes: Left panel shows increases in $HALE$ from eliminating communicable diseases (CDs) and non-communicable diseases (NCDs) calculated from 1. Equivalently, this panel shows increases in $EHAL(\varepsilon = 0, \eta = 0)$ from eliminating the respective disease groups. Middle and right panels show increases in $EHAL(\varepsilon = 0.5, \eta = 0.5)$ and $EHAL(\varepsilon = 2, \eta = 2)$, respectively, each calculated from (6).

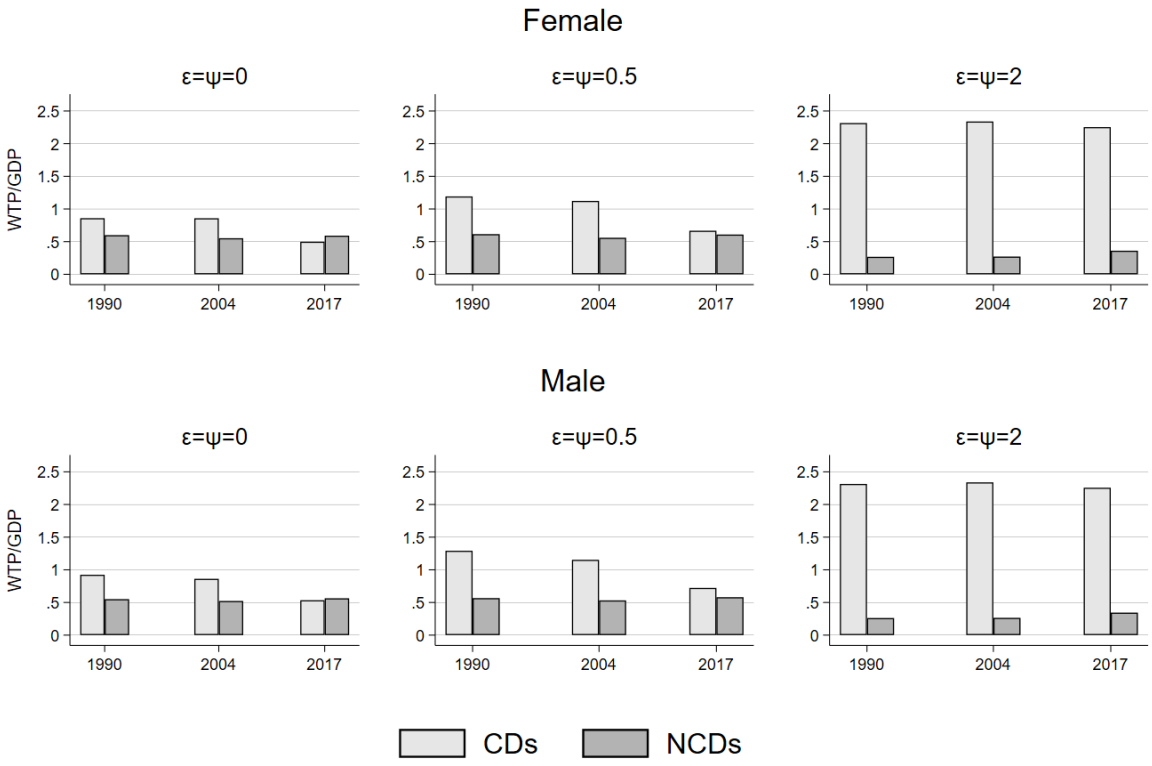

Figure 8: Willingness to pay to eliminate communicable diseases (CDs) and non-communicable diseases (NCDs)

Notes: Left panel, WTP from (10). Middle and right panels, WTP from (9) with $\eta = \psi = 0.5$ and $\eta = \psi = 2$, respectively. In each case, $\gamma = 1.25$, c =GDP per capita, and $\underline{c} = 10\%$ of GDP per capita.

5 Conclusion

We provide a measure of population health that is sensitive to inequality in age-specific health and health adjusted lifespan. The application of this measure and its valuation to the double burden of disease in Sub-Saharan Africa illustrates the potential importance of accounting for inequalities in the measurement of population health. While previous studies have found a decreasing gap in prevalence and disease burden between communicable and non-communicable diseases, our results suggest that the gap remains when using distributionally sensitive measures. This may have important implications for healthcare priority setting in developing countries.

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APPENDIX

A. Disability adjusted life years

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,

$$DALY_k = YLL_k + YLD_k,$$

where

$$YLL_k = \sum_x D_{k,x} \cdot L_x,$$

with $D_{k,x}$ the number of deaths due to disease k at age x and the L_x is the number of years by which that age falls short of life expectancy at birth. The latter is derived from a reference life table, which is constructed from the lowest age-specific mortality rates across all locations with populations greater than 5 million. The years lost to disability are obtained as the product of the number of disease cases prevalent in the population across all ages, P_k, and the disease's disability weight, DW_k,

$$YLD_k = DW_k \cdot P_k, \tag{13}$$

Each disability weight represents the magnitude of the health loss associated with that disease and is measured on a scale between 0 (perfect health) and 1 (equivalent to death).

B. Cause-deleted life tables

The GBD mortality data is provided in age intervals. Using standard life table notation, we define x as the starting age of the interval and n as the length of the interval. More formally,

the interval is defined as $[x, x+n)$. The last age interval (95+ years) is open ended. The primary input from which period life tables are constructed is age-sex specific mortality rate for each age interval:

$$ {}_nM_x = \frac{{}_nD_x}{{}_nP_x} \tag{14} $$

where ${}_nD_x$ is the number of individuals dying between exact ages x and $x+n$ during the year for which calculations are being made and ${}_nP_x$ is the mid-year population aged between exact ages x and $x+n$. The age-sex specific mortality rates are additive in the GBD data, so that the all-cause mortality rate is the sum of disease specific mortality rates for each age-sex partition.

The first step in the construction of the life table is the calculation of the probability of dying in an interval:

$$ {}_nq_x = \frac{{}_nd_x}{l_x} \tag{15} $$

where${}_nd_x$ is the number of individuals dying in a cohort between exact ages x and $x+n$, and l_x is the number of people alive at age x. Note that ${}_nd_x$ relates directly to term $d(x)$ described in Section 2.1. In that section $d(x)$ is defined at single ages (instead of intervals) for notational simplicity but also differs in that it reflects the the proportion of the initial population cohort l_0, i.e., $d(x) =_n d_x/l_0$. The calculation of ${}_nq_x$ requires some assumption on the age distribution of deaths within the interval. Given a set of age-sex-specific mortality rates ${}_nM_x$, the conditional probability of dying in each interval can be calculated as (Chiang 1968):

$$ {}_nq_x = \frac{n \cdot {}_nM_x}{1 + (n -_n a_x)_nM_x} \tag{16} $$

where ${}_na_x$ is the average number of years lived in the interval $[x, x+n)$ those who die in the interval. These values are not provided by the GBD. Instead we use the values provided by the UN World Population Prospects for males and females for the Sub-Saharan African region. Starting with a radix population of 100,000 individuals (i.e., $l_0 = 100,000$) the l_x values can by iteratively applying the ${}_nq_x$ values at each age as follows:

$$ l_{x+n} = l_x(1 - {}_nq_x),\ x = 0, 1, 5, 10, ..., 95 $$

The number of individuals dying in a cohort between exact ages x and $x+n$ is then given as:

$$ {}_nd_x = l_x - l_{x+n} \tag{17}$$

which with our assumptions above defines the number of individuals dying at age $x +_n a_x$. These values be easily translated into the proportion of individuals dying at each age $x +_n a_x$ required for our main analysis. Other life table functions (e.g. life expectancy at birth) can then easily be derived from these values using standard life table functions.

The next step of our approach is to calculate cause-deleted life tables. Let us denote the mortality rate without a specific disease k as:

$$ {}_nm_x^* = \frac{{}_nD_x - {}_nD_x^{-k}}{{}_nP_x} \tag{18}$$

where ${}_nD_x^{-k}$ is the number of age-sex specific deaths due to the disease k in the interval. This counterfactual mortality rate can then be used as before to generate a probabilities of death excluding mortality from the disease:

$$ {}_nq_x^* = \frac{n \cdot {}_nM_x^*}{1 + (n -_n a_x^*)_nM_x^*} \tag{19}$$

where ${}_na_x^*$ is the counterfactual average number of years lived in the interval $[x, x + n)$ those who die in the interval. To our knowledge, there are no disease specific data for ${}_na_x^*$ available for the Sub-Saharan African population. We therefore set ${}_na_x^* =_n a_x$ in all calculations and calculate counterfactual ${}_nd_x^*$ values using the same formulas as above.

C. Additional results

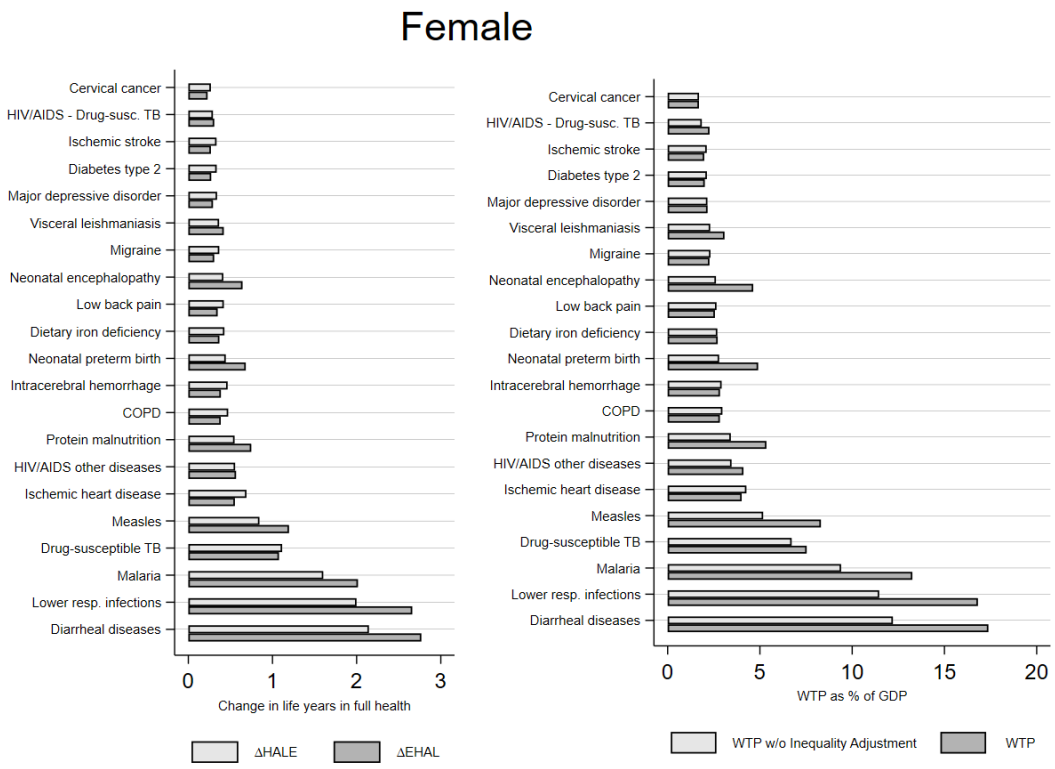

Figure 9: Disease impacts on HALE and WTP with and without distributional sensitivity 1990

Notes: Inequality aversion parameters ε and η are both set to 0.5.

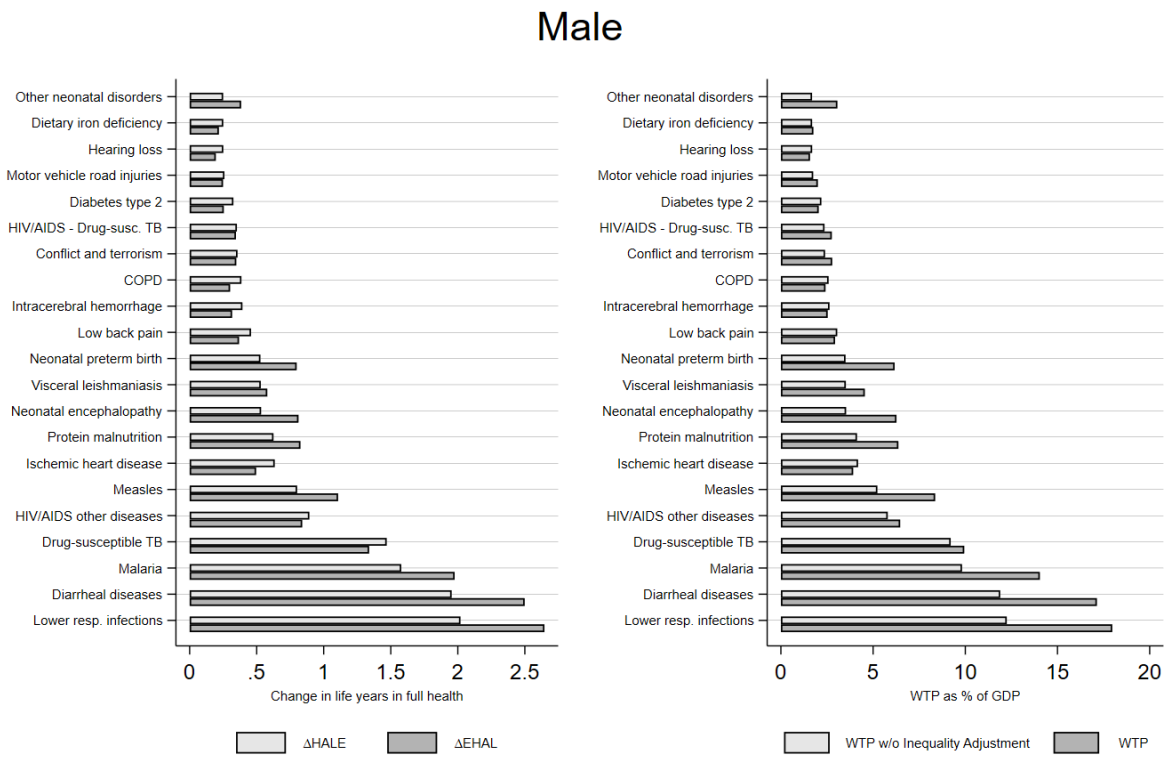

Figure 10: Disease impacts on HALE and WTP with and without distributional sensitivity 1990

Notes: Inequality aversion parameters ε and η are both set to 0.5.

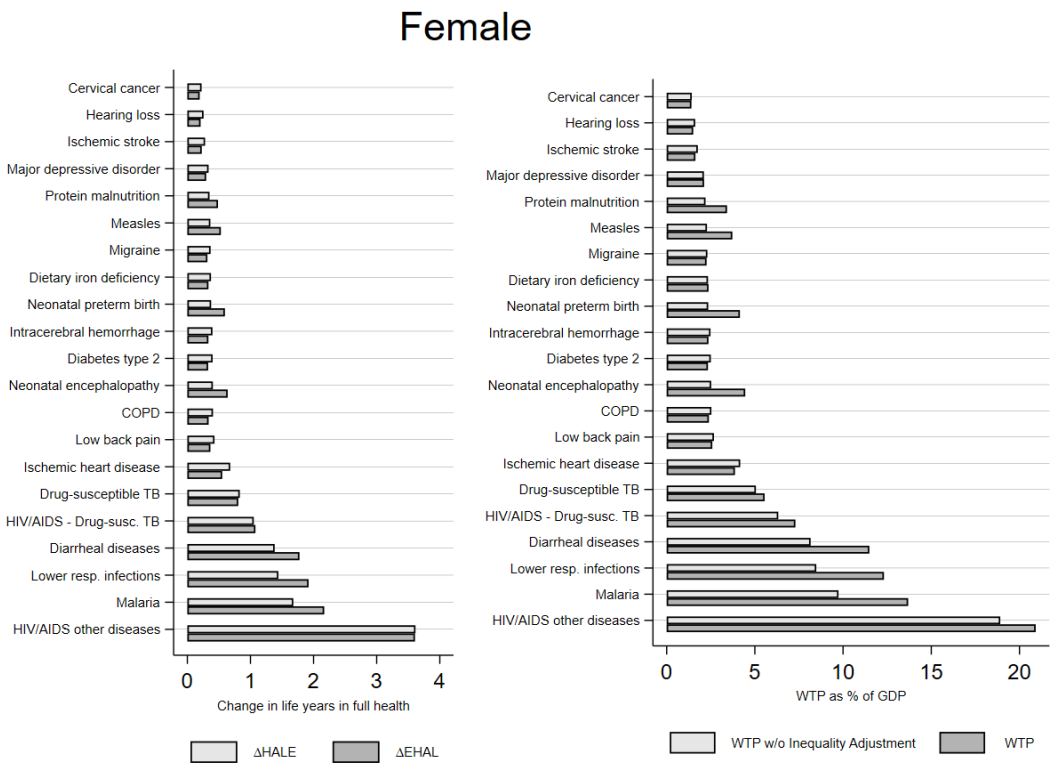

Figure 11: Disease impacts on HALE and WTP with and without distributional sensitivity 2004

Notes: Inequality aversion parameters ε and η are both set to 0.5.

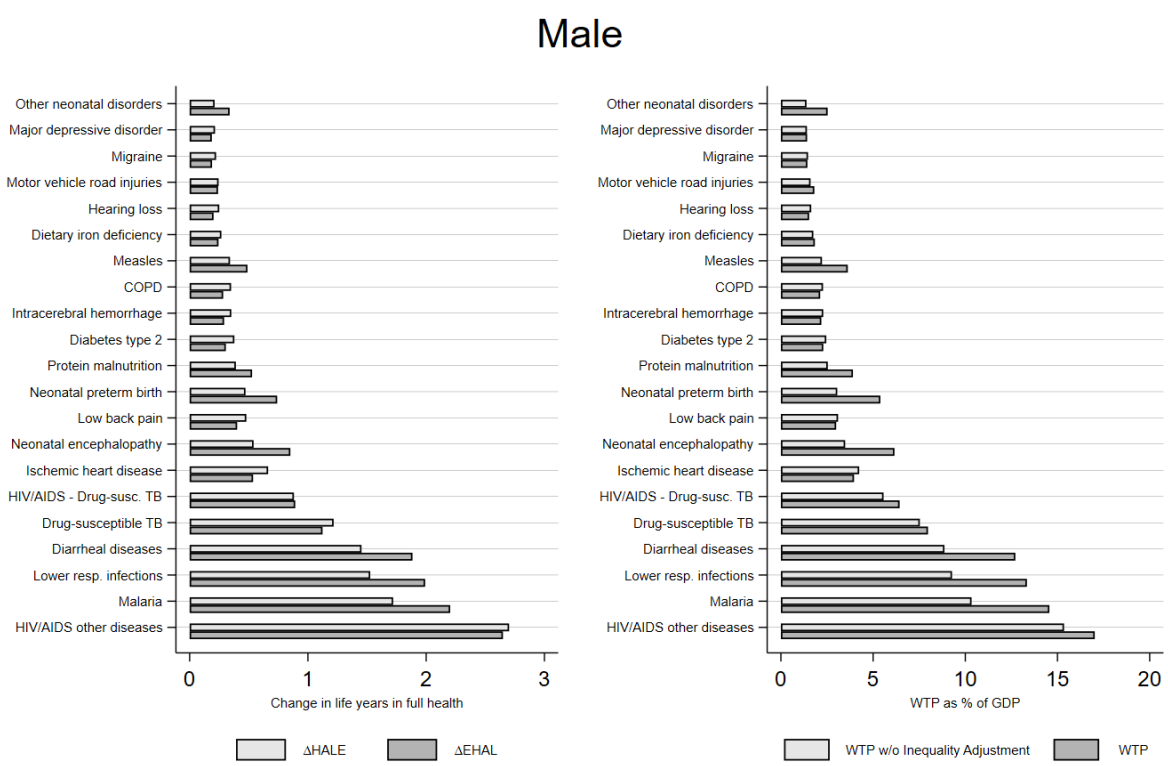

Figure 12: Disease impacts on HALE and WTP with and without distributional sensitivity 2004

Notes: Inequality aversion parameters ε and η are both set to 0.5.

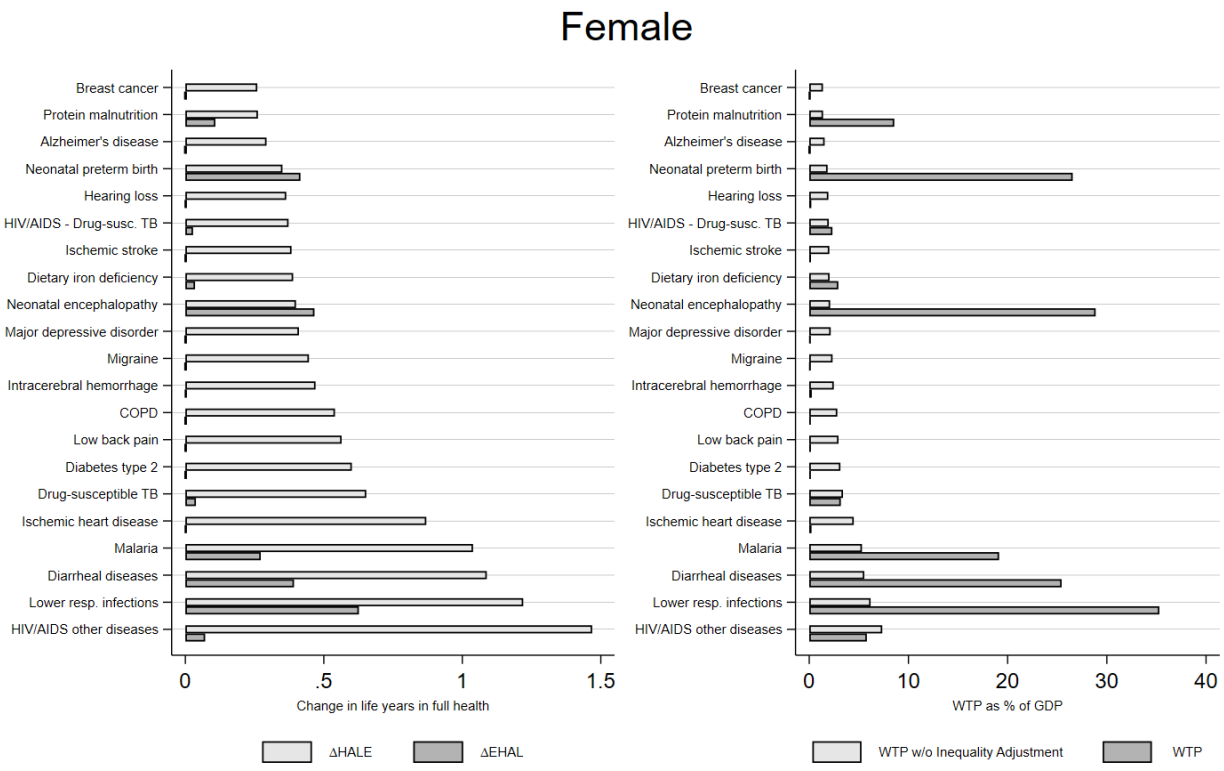

Figure 13: Disease impacts on HALE and WTP with and without distributional sensitivity 2017

Notes: Inequality aversion parameters ε and η are both set to 2.

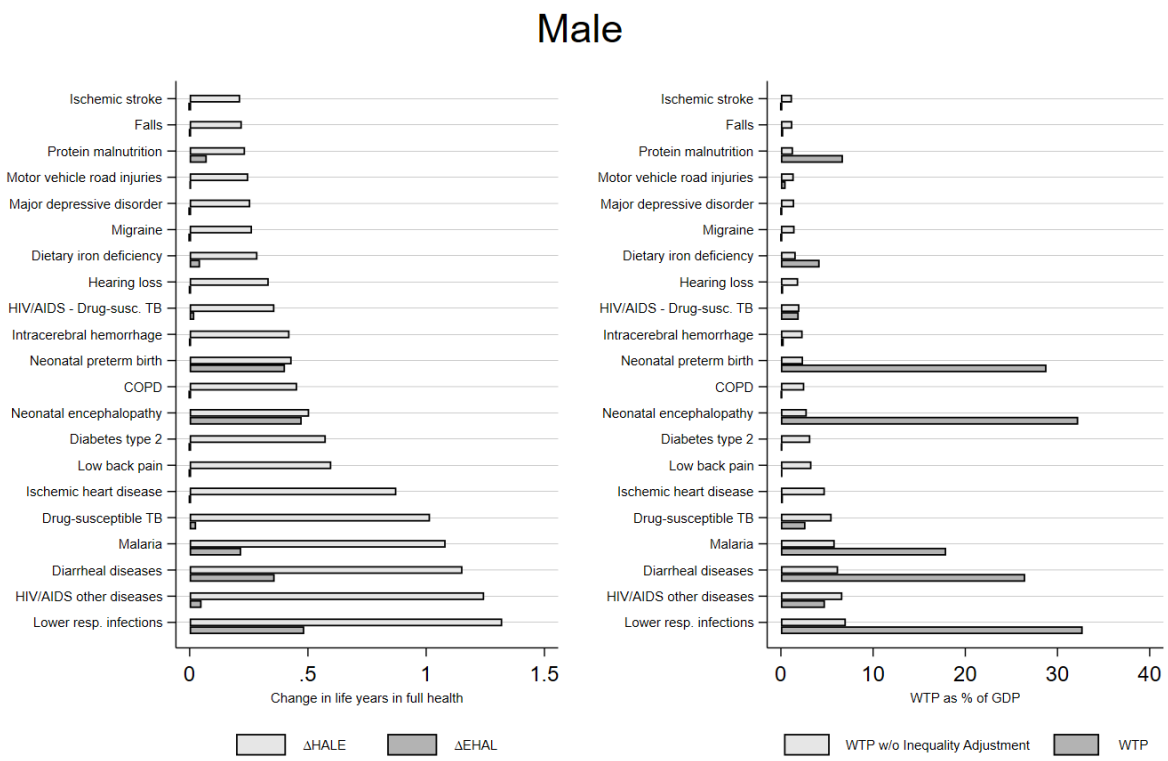

Figure 14: Disease impacts on HALE and WTP with and without distributional sensitivity 2017

Notes: Inequality aversion parameters ε and η are both set to 2.