

# The ethics, economics, and demographics of delaying aging

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**Abstract:** Efforts to slow aging are likely to be under-resourced due to a variety of misconceptions and cognitive errors, as well as social discounting. We argue that the social benefits of delaying aging would be enormous across a wide range of ethical frameworks, because more and better life-years would be lived by the same number of people. For any policymakers or philanthropists undeterred by the uncertain time horizons of such an endeavor, research into potential treatments to slow aging is a valuable and neglected investment.

**Keywords:** Anti-aging, population decline, economic growth, population ethics.

Senescence—that is, biological deterioration due to aging—causes most of the world’s death and chronic disease, even in the least developed regions.<sup>1</sup> It also causes enormous economic harm. Given this, should we aim to speed up the discovery of treatments that delay senescence?

In this paper, we make three main claims:

1. Efforts to delay senescence are particularly likely to be under-resourced, due to misconceptions, a variety of cognitive errors, and steep social discount functions.
2. Delaying senescence would lead to enormous benefits in health, productivity, and quality of life, benefits that vastly outweigh common objections against them.
3. Moreover, the intrinsic value of adding life-years by delaying senescence is robustly valuable across ethical frameworks.

Much of what we say here is not new. Our primary aim is to bring together some key details—biological, demographic, economic, and ethical—to help readers consider the potential value of research aimed at delaying senescence.

## 1. Sources of apathy

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<sup>1</sup> We use “senescence” in the broad sense, applying to the whole organism rather than individual cells. Older age is by far the greatest risk factor for nearly all cardiovascular diseases, cancers, metabolic diseases, and neurodegenerative diseases. It is also a major risk factor for death from communicable diseases (especially lower respiratory infections), as well as accidents. Thus, most people who die every year globally are among the 12% of the population older than 70. For some useful visualizations see [thelancet.com/lancet/visualisations/gbd-compare](http://thelancet.com/lancet/visualisations/gbd-compare) and [ourworldindata.org/grapher/number-of-deaths-by-age-group](http://ourworldindata.org/grapher/number-of-deaths-by-age-group).

Senescence is the overwhelming cause of the specific diseases of aging that end up killing the vast majority of people: cardiovascular diseases, cancers, metabolic diseases, neurodegenerative diseases, and immune system dysfunction. Despite this staggering death toll, very little medical research is aimed at understanding the basic mechanisms of aging or how to influence them. For example, that area gets less than 1% of US federal spending on medical research.<sup>2</sup> And, relative to the stakes, there has likewise been very little philanthropic funding or private investment.

Four factors help explain why social resources are vastly under-invested in this problem: (i) many people wrongly assume that senescence is immutable; (ii) people's reasoning about senescence is clouded by an array of cognitive biases; (iii) both public and private investment is discouraged by steep temporal discounting; and (iv) the economic and demographic implications are misunderstood. In this section, we will consider the first three factors in turn; section 2 covers the fourth in more detail.

### 1.1 Misunderstanding senescence

It's very natural to think of biological aging as a single long process that leads first to mature development and then to decline. But in fact, there are two entirely different processes at work—one creative and one destructive. *Maturation* is what happens when healthy cells carry out their functions coded for in their DNA, while *senescence* is what happens when damage to cells accumulates beyond the body's ability to repair it.

Both of these processes are at work even in young animals, as normal metabolic functioning generates several kinds of cellular “wear and tear” including mitochondrial dysfunction, epigenetic dysregulation, genomic instability, telomere attrition, stem cell depletion, and protein aggregation.<sup>3</sup> The body does have defense and repair mechanisms for all of this damage, but those mechanisms don't quite keep up with the rate at which the damage accumulates. In humans, it starts to interfere noticeably with normal functioning in middle age, and eventually impairs every physical system to some degree.

In all animals with the relevant anatomy, the final stage of deterioration looks similar: a combination of atherosclerosis, dementia, cancer, arthritis, muscle atrophy, brittle bones, fragile skin, and so on. The span of healthy life before this stage is called the *healthspan* of a species. But animal bodies

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<sup>2</sup> Of the \$45b NIH budget in 2022, only \$390m went to the Division of Aging Biology of the National Institute on Aging, to support “research to determine the basic biochemical and genetic mechanisms underlying the processes of aging at the cell, tissue, and organ levels” ([nia.nih.gov/about/budget/fiscal-year-2022-budget](https://nia.nih.gov/about/budget/fiscal-year-2022-budget)). This represents .025%, or 1 part in 4000, of federal discretionary spending, to try to understand the thing that ultimately kills the vast majority of Americans. For contrast, the \$8 trillion spent on the “war on terror” equals *two hundred years' worth* of federal spending on health research.

<sup>3</sup> See e.g. López-Otín et. al. (2023), Guo et. al. (2022), Kennedy et. al. (2014), Hou et. al. (2019).

accumulate the damage of aging at very different rates, resulting in more than 100-fold differences in normal healthspans even among mammals.<sup>4</sup> All over the evolutionary tree there are closely related species with dramatic differences in the rate at which they age: for example, naked mole rats can live in perfect health ten times longer than most other rodents, while Greenland sharks can live for hundreds of years, far longer than most other sharks.

The leading explanation for this variance is that, for species with high rates of pre-senescent death, there is little evolutionary pressure to invest in cellular defense and repair mechanisms.<sup>5</sup> But when, due to adaptations and changing conditions, fewer animals die young, it becomes more worthwhile to ward off the accumulation of cellular damage. For example, when an isolated population of opossums enjoyed a reduced rate of predation, their rate of aging had slowed within a few thousand years (Austad 1993). Delayed senescence can also rapidly be achieved through selective breeding (Nagai et. al. 1995; Rose 2004).

More direct interventions have also slowed aging in many animal models, using a variety of dietary, pharmaceutical, genetic and epigenetic techniques.<sup>6</sup> A noteworthy example is rapamycin, a molecule that increases healthspan in every animal model on which it has been tested (e.g., by 25% in mice).<sup>7</sup> Of course, humans have endogenous defense and repair mechanisms that are already far more effective than those of mice. But there is no reason in principle that they couldn't be further enhanced. Indeed, there is already evidence for delayed senescence in some human interventions, and the FDA has recently approved its first ever drug trial to target aging itself.<sup>8</sup>

Still, research on potential treatments is so incipient that we are not even in a good position to assess how difficult it will be to find them.<sup>9</sup> Our challenge is not unlike the one humanity faced in the mid-19th century with respect to communicable disease. At the time, such diseases caused the vast majority of deaths even in the richest countries, and there was no way to know whether we could do much to stop them. As it happens, research on antibiotics and on harnessing the body's endogenous

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<sup>4</sup> See e.g. Kolora 2021, Kirkwood & Austad (2000). Moreover, some strange animals like the freshwater hydra likely do not age at all (Tomczyk et. al. 2015).

<sup>5</sup> Thus, as Kirkwood & Austad (2000) point out, “adaptations that reduce extrinsic mortality (for example, wings, protective shells or large brain) are generally linked with increased longevity (in bats, birds, turtles and humans).” See also MacRae et al. (2015), Tian et. al. (2019), Seluanov et. al. (2018).

<sup>6</sup> For a review of some recent work see Zhang et. al. (2022). For a recent epigenetic approach see Yang et. al. (2021).

<sup>7</sup> Weichhart (2018), Bjedov and Rallis (2020), Bitto et. al. (2016), Selvarani et al. (2021), Wilkinson et. al. (2012).

<sup>8</sup> The approved study is for the drug metformin (Barzilai 2016). See also Kulkarni (2020), Glossman and Lutz (2019), Li et.al. (2014), Johnson et. al. (2013), Mannick et. al. (2014).

<sup>9</sup> For a survey of opinions among experts in the biology of aging, see Cohen (2020). For example, fewer than half of those surveyed “moderately or strongly” agreed with the statement “We have a relatively good understanding of the basic biological mechanisms of aging”, although a slim majority was at least slightly inclined to agree with it. About the same number agreed that “it should be possible to intervene in aging, and evaluate interventions, even in the absence of a clear consensus or mechanistic understanding of what aging is.”

defense system against viruses would become by far the most cost-effective research ever undertaken, fully doubling average expectancy in just over a century. We can thank our ancestors for refusing to be deterred by uncertainty.

## 1.2 Cognitive pitfalls

Aside from biological misconceptions, another reason for the neglect of senescence research is that the very issue triggers a combination of systematic reasoning errors, leading to public apathy and even opposition.<sup>10</sup> (This is apart from substantive objections, which we'll consider in the next section.) Here are five such errors:

*i. Affective salience.* The strength of our emotional reaction to potential harms is largely a function of how dramatic, sudden, unfamiliar, and unpredictable they are.<sup>11</sup> This is why terrorist attacks create fear far out of proportion with their risk.<sup>12</sup> Lacking every single one of these features, senescence is at the other end of this spectrum, as though tailored to minimize our affective response. Of course, it is unexpected and dramatic when people die *unusually early* from e.g. heart attack or stroke, but although such events are also usually manifestations of senescence, few think of them as such.

*ii. Naturalness.* The fear of things that are perceived as “unnatural” fuels a common aversion to GMO foods, nuclear power, and even vaccines.<sup>13</sup> And because of their prevalence, deaths from senescence can seem more like the “natural course of events”, while deaths from the less common specific diseases of aging (Alzheimer's, for example) may not.

*iii. Inaction and normality.* We care far more about the bad consequences of interfering with a normal state of affairs than we do about the bad consequences of allowing it to prevail. (This is

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<sup>10</sup> In a Pew Research poll conducted in 2013, 63% agreed with “Medical advances that prolong life are generally good”, while only 41% agreed (and 51% disagreed) with “Medical treatments that slow the aging process and allow the average person to live decades longer, to at least 120 years old, would be a good thing for society” (Pew Research Center 2013). A poll by Public Policy Polling in 2022 framed things very differently: “Leading scientists have identified cellular aging as the root cause of many chronic, deadly diseases including cancer, Alzheimer's or dementia, heart disease, stroke, diabetes, and others. Would you support or oppose medical research that seeks to treat the cellular aging process as a means to prevent or delay the onset of all of these diseases at once?” 69% expressed support and 8% expressed opposition. But even framed this way, only 50% expressed support for the idea that it should be a government priority to increase funding for such research.

<sup>11</sup> See Slovic et al. (2004), Lowenstein et. al. (2001), Fox-Glassman & Weber (2016).

<sup>12</sup> Sunstein (2003). Consider: the attacks of 9/11 killed 3000 and led to \$8 trillion being spent on efforts to fight terrorism. Since federal agencies value a statistical American life at about \$10m, it follows that by their lights this is nearly a million lives' worth of discretionary spending.

<sup>13</sup> See Scott & Rozin (2020), Meier et. al. (2019). In the Pew poll mentioned in fn 10, 58% agreed that the kind of radical life extension mentioned there “would be fundamentally unnatural”.

“omission bias” as typically mediated by normality conditions.)<sup>14</sup> Of course, aging is considered a maximally normal background condition, so we should expect its harms to be downplayed while undue attention is paid to any possible harms of an intervention.<sup>15</sup> Meanwhile, this bias should be attenuated when considering specific diseases of aging: after all, their timing can be unpredictable, and many are not considered “normal” at any age (e.g. Parkinson’s).

*iv. Just world bias.* When things feel like an inevitable part of the structure of the world, we tend to look for reasons why they are good or necessary.<sup>16</sup> Thus, one hears that “death gives life meaning”, a claim at odds with the research on mortality salience, death anxiety, and the coping mechanisms of the terminally ill.<sup>17</sup> Since immortality is not on the table, the relevant question is whether 80-odd years is an optimal span for a meaningful life. We know of no reason to think this, except aversion to the cosmic injustice that our days were numbered by a quirk of evolution.

*v. Scope insensitivity.* Our response to harms is largely insensitive to their scale, for at least two reasons. First, our capacity for empathy is bounded: however much we can feel for the suffering of one person, we cannot feel a thousand times more for the suffering of a thousand people.<sup>18</sup> Given this, we should hardly expect an appropriate affective response to senescence, the world’s greatest cause of death and disease. And second, we tend to focus more on the *proportion* of a problem that

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<sup>14</sup> “Omission bias” is when we consider actions that cause bad outcomes more wrong or regrettable than inactions that allow equally bad outcomes to occur. The strength of this bias largely hinges on judgments of normality, being strongest when the relevant *actions* interfere with normal background conditions, while the relevant *inactions* allow normal background conditions to prevail. Note also the connection to biases in favor of the status quo, default actions, and indirect over direct causes of harm. See Feldman (2020), Feldman et. al. (2020), Fillon et al. (2021), Yeung et. al. (2022), Prentice & Koehler (2003), Baron & Ritov (2004).

<sup>15</sup> Of course, even if omission bias is entirely irrational in non-moral contexts, there may still be a *moral* difference between, for example, killing and letting die. The problem is that no legitimate moral distinction of this type should turn on whether the relevant behaviors happen to be considered *normal*—so we should be wary that such a judgment is influencing our moral intuitions in the case of senescence.

<sup>16</sup> Dozens of studies show subjects shifting their memories, predictions, beliefs, and moral judgments to avoid representing the world as deeply unjust. (This is related to other phenomena including victim-blaming, status-quo bias, and system justification.) For example, when faced with people described as victims of a random tragedy, subjects subconsciously lower their assessment of the victim’s moral character, downplay the badness of the tragedy, and also predict more meaning and enjoyment in the victims’ later lives. See Ellard et. al. (2016), Hafer & Sutton (2016), Bartholomaeus & Strelan (2019), Callan & Ellard (2010). Of particular interest is how this interacts with ageism: the suffering of older people is perceived as less unfair than the suffering of younger people (Callan et. al. 2012).

<sup>17</sup> In fact, the best explanation for the relevant study results appears to be that mortality salience *threatens* one’s sense of meaning—which causes some people to attempt to restore a sense of meaning by more deeply identifying with ideologies or worldviews. Note that this idea has very different experimental predictions from the idea that awareness of death *makes* lives more meaningful. See Routledge et. al. (2010), Burke et. al. (2010).

<sup>18</sup> See e.g., Dickert et. al. (2015); Slovic & Västfjäll (2010). This issue arises even when comparing one person versus eight, and so can’t be entirely due to our difficulty grasping very large numbers—though the latter can certainly compound the problem.

can be solved, than on the absolute amount of good done.<sup>19</sup> Unfortunately, while curing cancer or Alzheimer's would feel like solving a whole problem, delaying senescence will only ever feel like a partial mitigation.

We doubt this is an exhaustive list of the systematic errors hindering our cognitive and affective response to the idea of delaying senescence.<sup>20</sup> But at a minimum, it seems we should take deliberate steps to counteract these errors.

The following thought experiment can help as a kind of cognitive palate cleanser. Suppose we had evolved with a healthspan 20 years longer, so that the senescence we actually suffer from at 70 didn't occur until the age of 90. In such a world, *that* timeline would feel natural and normal, the expected and familiar arc of life. No one would take seriously the idea of taking steps to *advance* senescence by twenty years in order to secure any of its purported benefits (e.g., making life more meaningful). The idea would be dismissed as absurd and horrific. Indeed, if we faced some new risk—a strange virus perhaps—that would cause us to senesce 20 years earlier, we would move mountains to find a cure.

Is there an important difference between this situation and our own that morally or rationally justifies our inaction? Or are we simply lulled into apathy by what feels natural and familiar and expected?

### 1.3 Social discounting

The final source of apathy we want to highlight is discounting. Because the relevant research is in its infancy, it's extremely difficult to estimate how soon we might have treatments that significantly increase healthspan. Certainly, few people expect that they themselves or even close loved ones are likely to benefit from such treatments. And even those inclined to improve the world more broadly tend to place a far higher value on efforts that will do so sooner as opposed to later.

However, if people in the medium and long-term future matter as much as those alive now, the importance of aging research is not diminished by the fact that it may only help the former. Indeed, philanthropists and governments acting in the interest of future generations are uniquely positioned to offset the fact that this research area will be neglected by those who implicitly or explicitly operate with a steep social discount function (that is, nearly everyone).

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<sup>19</sup> In studies, subjects are more motivated to solve the whole of a small problem than only part of a much larger problem—even if the second does far more good in absolute terms. In fact, just the act of conceptualizing someone's suffering as part of a larger problem tends to diminish our willingness to help that person. See e.g., Bartels & Burnett (2011).

<sup>20</sup> For example, *zero-sum thinking* seems to be at work when it's said that the elderly "have had their fair share", as though by living longer they'd be taking something away from others. In fact, the young would also benefit, initially from increased quality of life, and then from longer lifespans themselves. See Johnson et. al. (2022).

Barring near-term human extinction, there will be future generations for whom it no longer feels natural or inevitable for people's bodies and minds to deteriorate at the rate they do now. We suspect they will look back at us with enormous pity for our short health-spans, and (eventually) even for the need to suffer from the ravages of aging at all. And just before those people arrive, there will be people who die right on the cusp of new treatments—people who would have enjoyed more years of healthy life had we tried harder, earlier.<sup>21</sup> Will those people be us, or our children, or our grandchildren, or some more distant generation? We can't say. But for patient, impartial altruists, it shouldn't really matter.

## 2. Longer lives and quality of life

How will the world change with longer healthspans? Aside from a healthier and longer-lived society with a smaller share of dependents—the uncontroversial benefits—a common concern is the resulting population increase. Perhaps surprisingly, we believe this too is a benefit. Global populations are set to peak and then to begin a persistent decline later this century; reducing mortality rates would only slow the speed of this decline. We detail these arguments below along with more speculative effects on scientific progress, governance structures, etc.

To give a quantitative sense for the value of these effects, we will study a hypothetical intervention that delays age-related decline by 20 years. We assume that once it sets in, the length of biological decline is unchanged. In other words, this 20-year increase in healthspans also increases lifespans by 20 years. Because we expect such treatments to come *eventually*, it is more accurate to conceptualize the proposed intervention as bringing forward the discovery of an anti-senescence treatment.<sup>22</sup>

We will argue that, under conservative but plausible assumptions, a 20-year increase in healthspans would result in a 50+% increase in annual income per capita, for each extra year the treatment is available. Most of this comes from increased productivity, which is the focus of this section:

- 25% from reducing the share of dependents;
- 25% from increasing returns to scale (the effect of populations on productivity);
- additional spillover effects.

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<sup>21</sup>See e.g., Bostrom (2005).

<sup>22</sup>To the extent that the marginal investments we consider determine whether or not major gains in life-expectancy are *ever* achieved serves to make the investments even more valuable than we conclude here (since this is equivalent to bringing it forward from 'never' to some earlier time).

In section 3 we will address the additional value coming from the intrinsic benefit of living longer. As a preview of that benefit: we estimate it to be worth, in monetary terms, an additional 30+% of income per capita value on top of the 50+% increase documented in this section.

## 2.1 A smaller share of dependents

The least controversial economic benefit of extending healthspans would be to permanently reduce the share of dependents. That is, a larger fraction of the population at any time will be contributing goods and services for the whole of the population to enjoy. In countries with social pension systems—like Social Security in the United States—this amounts to increasing the ratio of contributors to dependents, and thus decreases the payments per worker to sustain a high living standard for the non-working. Analogous benefits arise in countries with less formal transfers to their dependents.

Basic economic accounting identities can inform the social value of increasing the proportion of working-age individuals in the general population, which will be reflected in higher per capita consumption. Consider a standard production function representation of the macroeconomy. Denote  $Y$  total economic production (GDP),  $A$  economic productivity,  $K$  economic capital,  $L$  the labor force, and  $N$  the population size. Then we can define per capita consumption,  $y$ , as:

$$y = \frac{Y}{N} = \frac{AF(K,L)}{N},$$

where  $F$  is a function that combines capital and labor into economic output. A standard assumption that we will employ is that the function  $F$  has *constant returns to scale*.<sup>23</sup> This means that for a fixed level of productivity,  $A$ , if all inputs are doubled, the resulting output is doubled. For example, to double the production of a factory, one approach is to build an exact replica (double  $K$ ) and fill it with identical labor (double  $L$ ).

An intervention that increased life-years by 25% (20 years on a base expectancy of 80) would cause an increase in the population size by about that same 25%, relative to non-treatment scenarios. The exact percentage difference will depend on the age distribution, but an elasticity of one between population size and lifespans is a reasonable approximation.<sup>24</sup>

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<sup>23</sup> For those unfamiliar with this terminology and notation, this is written just to be a generalized version of the common Cobb-Douglas formulation, where the function is multiplicative between  $K$  and  $L$ , with exponents on each that sum to one.

<sup>24</sup> Imagine a population with a perfectly uniform age distribution (i.e., the same number of 5 year olds, 55 year olds, and 75 year olds, etc., but no one lives past 80). If lifespans increase from 80 to 100, there are 25% more age groups that will be populated by the same number of people (after, of course, the 20 years it takes for the initial individuals who avoided death to age to 100).



If  $L$  is conceptualized as the number of individuals in their prime years, this term increases proportionately more. Standard working lives are about 40 years, so a 20-year increase in prime-years represents a 50% increase in the working population. Theories of savings and investment imply that  $K$  ought to scale with  $L$ . So, let's assume that  $K$  and  $L$  both increase by 50%. The numerator (total production) would then increase by 50% under our constant returns to scale production function. The denominator, population, has only increased by 25%. This implies a 25% increase in per capita income/consumption,  $y$ .

This is a conservative estimate. Research on human capital demonstrates that workers are more productive later in their careers, when experience has accumulated but physical and mental health remains strong (Mincer 1974). If an anti-senescence intervention allows people in some of their most productive years to continue working in perfectly good health, at the ages of 60-80 in this scenario, that would be more valuable than a uniform increase in the labor force.

Beyond this, retirees do not only produce zero goods and services, but they consume a majority of health resources. With a smaller share of the population needing medical treatment, those resources could be directed towards making our lives better along other dimensions. We estimate this benefit to be in the lower single-digits as a percent of GDP per year, a large effect but not nearly as large as the labor force participation gains.

To put this in context, consider that standard projections of all damages due to climate change, when monetized, are valued at annual per capita income losses of less than 10%. (And even pessimistic estimates only reach about 30%; Diaz and Moore 2017.) Even this rote, mechanical effect of reducing the share of dependents makes this anti-senescence intervention comparable to preventing all future greenhouse gas emissions (and undoing all historical emissions!) in terms of human living standards. And there are good reasons to believe the benefits are much larger, still.

## **2.2 Implications of the coming depopulation**

Let's turn to the most salient second-order effect of increasing healthspan—that of a larger population. This is a consequence that people are instinctively concerned about, but we think is actually an important point in favor of delaying aging.

As noted earlier, an increase in lifespans by  $x\%$  increases the size of the population by about that same value, after a transition period. This would be in a context of unprecedented and persistent global population decline, according to consensus demographic projections (United Nations 2022). In particular, the UN expects a lower number of births every year into the indefinite future, so that by 2060 each generation fails to replace itself, and peak population is reached by 2086. No known

demographic or social force is expected to arrest this decline, and even an increase in healthspan of the sort considered here would only delay it.<sup>25</sup>

While population decline may have its benefits in terms of reduced harm to the environment—a point we will return to at length below—there are sound macroeconomic reasons why many astute observers are now more concerned about future scenarios with too *few* people (e.g., Jones 2022, MacAskill 2022). To see why, a bit of background is useful.<sup>26</sup>

*i. Fixed costs and non-rival goods.* Theories of economic growth tell us that the larger the global economy is, the more efficiently it produces the things that make life good (this is referred to as *increasing returns to scale*). If this is correct, larger populations better promote flourishing lives. The specific economic ideas underpinning this surprising result garnered respective Nobel Prizes—to Paul Krugman in 2008 and Paul Romer in 2018—and are now embedded within standard theories of growth, development and trade. Romer and Stanford economist Chad Jones summarized the relevant ideas as follows (Jones and Romer 2010: 231).

*In practice, urbanization, increased trade, globalization in all its forms, and the positive trend in per capita income all point in the same direction. In the long run, the benefits of a larger population that come from an increase in the stock of available ideas decisively dominates the negative effects of resource scarcity. In such a world, any form of interaction that lets someone interact with many others like her and share in the ideas they discover is beneficial, and the benefit need not be exhausted at any finite population size.*

The line of reasoning recognized in Krugman’s Nobel Prize relies on the existence of *fixed costs*, those that do not depend on the level of production or population. As an example, consider the design and construction of a road that connects a village to a nearby city. A large share of the costs to build and maintain this infrastructure do not depend on how much future travel will be done, or how big either population is. Therefore, the larger the population is, the lower these costs are per person. Krugman’s insight was that costs of this sort are pervasive, and their existence explains the geography of economic production (Krugman 1980). It follows that larger populations can fund and support a richer variety of products and public goods.

The line of reasoning recognized in Romer’s Nobel Prize is the *non-rivalry* of certain goods, the most important of which are ideas. Non-rivalrous goods are those that are not diluted (or “used up”) when employed by some individuals. Take the now-canonical example of oral rehydration therapy: a simple combination of salt, sugar and water can prevent deaths from dehydration. This discovery

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<sup>25</sup> Universal uptake of the hypothetical 20-year life-expectancy increase we consider in 2050, for example, would shift the population peak to around 2100.

<sup>26</sup> See also Geruso and Spears (this volume) for a detailed summary of the long-term consequences of low birth rates and depopulation.

has saved countless lives. The physical resources necessary are abundant, but they are useless without the *idea*. Once the idea is known, it is even more “abundant” than the physical resources: all caregivers can use this recipe simultaneously. Since people are the source of ideas, a larger population *creates* more ideas without also diluting their value by having more people apply them.<sup>27</sup>

*ii. Quantifying the benefits of scale.* The theory of increasing returns is mathematically rigorous in a way that allows us to estimate magnitudes from parametric assumptions. But first, let’s consider some of the historical data and (quasi-)experiments used in statistical research.

First, note that global population sizes and per capita economic growth have moved together, according to historical facts presented by Michael Kremer (1993). The main point is obvious, but profound: many hundreds of years ago the population was small and economic growth was slow; as populations grew economic growth accelerated. This pattern could be explained by other channels (or could be causally reversed), but it is useful to know that the broad historical facts are consistent with increasing returns. Indeed, Kremer finds further support for the causal interpretation by showing that—for historically technologically separated regions—larger initial populations were followed by higher living standards thousands of years later, when world regions were reintegrated by European exploration.

For a cleaner estimate on the direction and size of causality, Peters (2022) studies a specific case of quasi-random population change—that is, change not caused by economic conditions—allowing him to isolate the effect of population on economic productivity. To summarize his setting: after WWII the Allies controlled the expulsion of German refugees from neighboring countries by near-random assignment. Peters verifies that people who were placed near more people ended up much wealthier (more than offsetting the negative effects from sharing farmland). An increase of per capita incomes of nearly 0.5-0.7% was associated with each 1% increase in population. Eden and Kuruc (2023) take a different approach and calibrate the key parameters from the Romer-Jones theories of population-ideas relationships directly to empirical evidence from research and development processes. They estimate a 0.3-0.5% increase in income per capita in response to a long-run population increase of 1% through this channel.

*iii. Climate change and resource scarcity.* How do these benefits compare with the negative environmental effects generated by larger populations? As suggested by the Jones and Romer passage above: favorably, according to macroeconomists. Consider how, despite concerns like those of Paul Ehrlich, who predicted in 1968 that “the battle to feed all of humanity is already lost” due to unprecedented population growth (Ehrlich 1968), global rates of hunger and extreme poverty have

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<sup>27</sup> Consider the argument in reverse: imagine a world history with 20% fewer people. Which 20 of your favorite 100 inventors, writers, or political leaders would you be willing to delete from that history? Would it be worth the natural resources that would have been saved?

instead fallen as populations continue to grow. Against Ehrlich's predictions, even prices of minerals in fixed supply continued to fall. And none of this is an accident. The historical record is one where human ingenuity, when pressed into action, has alleviated resource constraints in ways that are predictable at a high level. More people means more ingenuity and also greater benefits from covering fixed costs.<sup>28</sup> Indeed, while there are some environmental factors that are worsening, others have seen major reversals indicating that population sizes alone do not account for environmental degradation. For example, local levels of pollution have fallen rapidly in many locations despite population growth, while global agricultural land use has peaked and begun falling in recent decades, and even some wild populations have begun recovering.<sup>29</sup>

More formally, Eden and Kuruc (2023)<sup>30</sup> show that in a standard macroeconomic representation of natural resource constraints, the profits earned by owners of these resources summarize how severely these resources constrain per capita incomes. If the amount of agricultural land, for example, were an important constraint on economic well-being, its price would be bid up. Instead, the current situation is one where these resources do not command high prices, indicating that (on the whole) they are not a major concern for a world of 8 billion, nor should they be for the smaller populations of the coming centuries.

A related concern is that a larger population increases the level of atmospheric greenhouse gases. Perhaps surprisingly, timing makes this a non-issue in our setting. If there were broad uptake of the envisaged anti-aging treatment by 2050, the effect on population size would be small at first, with the full effect not materializing for several decades after that.<sup>31</sup> At the earliest, large population changes would occur by 2070-2080. At that point, almost all historical greenhouse gasses will have been emitted, and the world will likely be near carbon neutrality.<sup>32</sup> Kuruc et al. (2023) make this point in detail using leading models of climate change that are modified to account for the benefits of scale described above; these models decisively prefer larger populations to smaller ones.

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<sup>28</sup> Ester Boserup (1965) and Julian Simon (1981) were onto this, even before Romer had mathematically formalized these theories of economic growth. These two were contemporaries of Ehrlich's but saw the benefits of population. Indeed, Simon and Ehrlich had a famous wager about how the prices of certain natural resources would evolve, Ehrlich believing that minerals in fixed supply would become so binding on human well-being that their (inflation-adjusted) price would dramatically increase. Ehrlich lost as those prices fell, food became more affordable, and even many measures of pollution fell.

<sup>29</sup> For respective examples/citations consider: (i) local US air pollution since the Clean Air Act; (ii) <https://ourworldindata.org/peak-agriculture-land>, (iii) humpback whale recovery post-conservation efforts (Bedjar et al, 2016).

<sup>30</sup> Building on the work of Weil and Wilde (2009).

<sup>31</sup> The population pyramid in 2050 will still have fewer people in their 70s and 80s than in the decades of middle age (partly because of higher death rates in those decades and partly because the total number of births peaked in the 2010s), meaning that the full effects of the life extension technology wouldn't be seen until the widest part of that pyramid reaches the end of their extended lifespan, just after 2100.

<sup>32</sup> See e.g., Ou et al., (2021).

Thus, we expect that by the time population size is affected by anti-aging technology, the volume of greenhouse gas in the atmosphere will be largely fixed, and the task at hand will be to develop and employ *removal* technologies in order to keep the planet from continuing to warm. Both the absolute and per-capita increases in productivity that we project—especially the gains from innovation—should accelerate this effort. To better see this, imagine that for some reason the population contracts dramatically just as we reach net zero emissions. In that case, we will have left our descendants with much the same problem of warming, but fewer and less productive hands available to solve it.

Overall, environmental economic models that account for the benefits of population sizes find that larger populations ought to be good, on net, for per capita living standards. If we use a number on the conservative side of the Peters (2022) estimates—say 0.5% increase in GDP per capita for every 1% increase in (working age) population—that results in another 25% increase to income per capita (coming from the 50% increase in working age population and gross production hypothesized above).

What about the more distant future? At some point, likely many years after the first anti-aging treatments are discovered, our descendants may gain near-complete control over senescence. In such a scenario, if fertility rates stop falling and people broadly elect never to senesce, there would be continued population growth of a kind that is linear and relatively slow. Assuming this creates a tradeoff where population growth comes at a social/environmental cost, various interim solutions are possible, such as requiring those who elect not to senesce to offset these negative externalities.

### **2.3 Other spillover effects**

In this subsection, we will briefly consider other second-order effects of longer healthspans. These are potentially large, but more speculative. On the whole, they offer further reasons to support anti-senescence research.

*i. Expanded horizons.* Without needing to resort to claims about the importance of the far future, common sense and simple economic theory suggest that increasing the time horizon of human decision-making will lead to better (non-discounted) aggregate outcomes. There is less to say here quantitatively, but the qualitative point is important. If our lifespans were much longer, the democratic body's concern for sea level rise and other effects of climate change would be more widespread, and we would plausibly take more action. Likewise for infrastructure, research and development, and other long-term social investments. Econometric evidence supports this at the individual level: when and where life expectancies are longer, individuals respond by sacrificing more in the present for their future (e.g., Jayachandran and Lleras-Muney 2009; De Nardi, French and Jones 2009; Oster et al. 2013). It is hard to predict exactly how this would operationalize in voting behaviors and/or philanthropic preferences if lifespans were dramatically increased. But as

important research about the far future has taught us (e.g., Bostrom 2003; Ord 2020), even small shifts in resources towards patient endeavors have the potential to generate tremendous (expected) value.

Along with our time horizons, we expect that longer lives would eventually encourage our spatial horizons to expand as well: a shrinking population of people with short lives is unlikely to attempt to spread through the galaxy. Larger populations bring the incentives, the innovators, and the ability to cover the fixed infrastructure costs and population transplants necessary for such projects. While this consideration is certainly speculative, insofar as space colonization is a reducer of existential risks and/or improves our lives in other important ways, increasing the probability that this happens could be an important channel through which longer lives are welfare-increasing in the long run.

*ii. Scientific progress.* One kind of objection to increasing healthspans stems from the idea that “science advances one funeral at a time”. The concern here is that older scientists tend to be less innovative while controlling a fixed number of senior research positions, and that newcomers are reluctant to challenge them (Azouly et al. 2019). Therefore, some believe that progress requires allowing such figures to retire and filling their positions with younger researchers. We believe this objection misses some key points, and that extending healthspans will, on net, have a very positive effect on scientific progress.

First, the objection assumes a fixed number of senior research positions, but our projected gains both to population and per capita GDP describe a world where a far higher absolute number of individuals is engaged in research.

Second, at least some of the cognitive inflexibility of senior researchers is surely due to the damage of aging itself.<sup>33</sup> Aging atrophies the brain, and even the brains of young adults have begun accumulating damage. For a while, the cost of this damage to raw processing power and cognitive flexibility is more than offset by additional knowledge gained and skills acquired. However, at some point the scales tip in the opposite direction, and our hypothetical treatment delays the point at which that would occur by 20 years.

It is hard to overstate the potential gains from extending the careers of top researchers at their cognitive peak. Research scientists train for so long that by the time they can maximize their potential, they are not very far from cognitive decline. Far from simply extending the length of their career by 50%, our hypothetical treatment more than doubles the period where they are at what we now consider the peak of their powers—fully educated, skilled, funded, and networked, but also still fully cognitively vibrant.

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<sup>33</sup> See e.g., Kupis et al. (2021).

In fact, we suspect that the funerals of researchers—and the decline that precipitates them—come at enormous cost to the progress of science. But we only notice the full tragedy when scientists die “early”. For example, von Neumann’s death in his early fifties is tragic in part because it was unexpected, but also because he would otherwise have continued his work for another 20 years or so. However, that’s exactly as long as the healthspan extension we are considering would allow researchers to continue with physical and cognitive vigor.

In short, by assuming a zero-sum world and ignoring the effects of aging on cognition, the “funerals objection” likely gets things exactly wrong. Instead, the world is poised to enjoy enormous research benefits from a larger and more cognitively vibrant population.

*iii. Long-lived dictatorships.* Another concern about increasing lifespans is that if autocrats and dictators were to live longer, this would extend their regimes and have an overall negative effect on the world. (Giving Stalin 20 more prime years, for example, would have almost certainly been bad for the USSR and the world.) But this concern seems misguided to us for two reasons.

First, the notion that the natural death of a dictator will promote political liberalization seems to contravene historical fact. Recent reviews show that the natural deaths of dictators are almost never followed by democratic shifts, and rarely even bring down that dictator’s particular regime.<sup>34</sup> Indeed, these studies find virtually no correlation between the death of a dictator in office and a country’s more long-term prospects for better government.

A far better case can be made that per capita GDP growth has a positive influence on political liberalization. (Most studies find a mutually reinforcing effect, with each positively influenced in the long run by the other.)<sup>35</sup> We would therefore expect the predicted 50% per capita GDP growth from healthspan extension to have significant positive effects on political liberalization, on net, and for this effect to far outweigh any cost from delaying the natural deaths of dictators.

*iv. Inequality.* Another common concern about anti-aging treatments is that they would increase inequality. After all, they would almost certainly be rolled out to the (globally) wealthy at first.<sup>36</sup> Aside from considerations of fairness, this may seem to imply that we are limiting our focus to gains for the already well off, some of which will be worth less because of diminishing marginal utility from consumption. Again, this objection does not hold weight under scrutiny.

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<sup>34</sup> See Kendall-Taylor (2016), Hummel (2020). Indeed, the one positive correlation between leader death and long-term liberalization found in these studies held only in economically developed countries, underscoring the importance of the following paragraph.

<sup>35</sup> See for example Acemoglu and Robinson (2019), wherein wealthier societies have more power to promote their freedoms, and a wider ‘corridor’ exists for state and social power to (positively) offset one another.

<sup>36</sup> In wealthier countries, where treatments for senescence are likely to be available first, there would be an enormous incentive for insurance companies and national healthcare programs to subsidize them, given the savings they would reap as the diseases of aging are staved off.

First, all health technologies are distributed unevenly, a fact that usually does not make us resist their discovery. Even sanitation—the simplest and most important health-related technology—is unequally distributed, and therefore in some sense “exacerbates inequality”. It would be ludicrous not to avail ourselves of sanitation because of this; instead, we must work hard so that everyone has access. (Also, recall that the question we’ve been asking is whether we want to *speed up* the arrival of a technology whose initial uptake will likely be unequal regardless of when it arrives. The effect of bringing this about earlier would make it available to both rich and poor people *earlier* than it otherwise would have been.)

Second, functional deterioration due to aging causes its own form of inequality, especially within the least developed countries. The world’s poorest—largely reliant on manual labor for income while lacking healthcare and social safety nets—are hardest hit by the harms of aging, just as they are hardest hit by nearly every ubiquitous problem. A treatment that ultimately offered them additional years in full health would greatly mitigate this source of inequality.

Third, historically the global poor appear to receive a greater share of the benefits from growth in non-rivalrous goods, as compared to growth in rivalrous goods, precisely because rich-country consumption does not crowd out use by the rest of the world. As a particularly relevant example, health improvements and life-expectancy gains in the global economy have spread to the global poor more rapidly than income gains. The benefits of life-extension technology may follow the same pattern.

A final point applies not only to concerns about inequality, but also to the other concerns we have considered. Imagine someone arguing that we should not discover better treatments for cancer or arthritis or dementia, for example, because the cures would be unequally distributed at first. (Or because this would keep older scientists or dictators alive, or...) If such arguments sound specious when applied to the specific diseases of aging, it’s worth asking whether they are actually driving one’s opposition to treatments for senescence itself.

## **2.4 Summary of quality-of-life benefits**

We have tried to give a sense for the improvements in the day-to-day experience of living in a world with longer lives and larger, more innovative (and perhaps more patient) populations. We argue that a technology that adds 20 prime years of life to each individual would add more than 50% to per capita incomes. Some of this gain would come through additional consumption of the things we have; some would be through the value of new products and technologies.

Across even just the US population, bringing such a technology forward by even one year would therefore be worth 10 trillion dollars (half of the current size of the US economy). The benefits to



the entire global population would of course be worth far more. And this is even before we consider the intrinsic value of more happy and healthy years per lifetime, the issue we turn to next.

### 3. The intrinsic value of life-years

So far, we have focused on the quality of life in a world with longer healthspans, due to productivity gains as well as less age-related suffering per life-year. But in addition to a higher quality of life, people would also have *more* life. In this section, we'll consider how to compare the value of these gains in quantity of life to the gains we've already discussed. As we'll see, there is a great deal of intrinsic value to these extra life-years, across a wide range of ethical frameworks.

#### 3.1 A standard measure

As a first pass, consider that people value their life-years, especially those they spend in good health. A standard measure used to value different types of health improvements is the quality-adjusted life-year (QALY). Conditions that affect quality of life are assigned a numerical fraction of a healthy life year (defined to be worth 1.0) based on reports from individuals with those conditions. If living with chronic back pain is estimated to be worth 0.9 QALYs, curing an individual's pain for 10 years of their life would be equivalent to extending their life by 1 year.<sup>37</sup> As a numerical example relevant to our case: the average 18-40 year old American diagnosed with diabetes loses 11 QALYs between lower life expectancy and loss of quality of life,<sup>38</sup> so giving  $z$  people access to a 20 year healthspan increase (trivially worth 20 QALYs) would be QALY-equivalent to preventing about  $2z$  people from ever receiving a diabetes diagnosis.

Health economists have also estimated dollar values for QALYs by observing how much people are willing to sacrifice monetarily to avoid losses in QALYs—e.g., wage premiums for dangerous jobs, willingness to pay for safer vehicles, etc. This kind of estimate is used by governments to assess whether specific treatments/preventative measures are worthwhile, based on the public's assessment of money-longevity trade-offs. For example, in the US, a value of approximately \$100,000-\$150,000 is assigned to a year of healthy life.<sup>39</sup> Our envisaged anti-aging treatment would thus be worth about

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<sup>37</sup> An increase from 0.9 to 1.0 for 9 years = 0.9 QALYs = one additional year living at 0.9.

<sup>38</sup> See: <https://nccd.cdc.gov/Toolkit/DiabetesBurden/YLL/QALY>

<sup>39</sup> See [www.bloomberg.com/graphics/2017-value-of-life/](http://www.bloomberg.com/graphics/2017-value-of-life/) for US governmental values of approximately \$10M per life saved; see [www.openphilanthropy.org/research/technical-updates-to-our-global-health-and-wellbeing-cause-prioritization-framework/](http://www.openphilanthropy.org/research/technical-updates-to-our-global-health-and-wellbeing-cause-prioritization-framework/) for an example of a large philanthropist documenting their internal valuation of \$100,000 per life year saved. To state the obvious, the monetary value of averting deaths is a contentious and controversial topic where individual behavior in smaller stakes settings (i.e., the wage premium associated with slightly more dangerous jobs) is used to extrapolate to the implied monetary value of averting deaths with certainty. We take no novel stances here and intend only to follow existing conventions to valuing additional life years.

\$2-3M per person who receives it, or increasing their annual consumption by \$20,000-30,000 (30-50%) per year. One attempt to directly value individuals' willingness-to-pay for anti-senescence treatments found a much higher value per life-year saved than this (Scott, Ellison and Sinclair 2021).

Given this, the full economic value of the envisioned treatment would combine the productivity gains discussed in the previous section with these intrinsic value gains, with the result that the treatment is approximately welfare-equivalent to doubling annual per capita income.

### 3.2 Life-years and population ethics

Assume that adding more good years *ipso facto* increases the total well-being in people's lives—that is, even aside from any gains in quality of life. From this, it doesn't immediately follow that having more good years is a better outcome, ethically speaking. To see this, let's consider some competing approaches to the relationship between well-being and the goodness of outcomes.<sup>40</sup> We won't compare the merits of these views here, but the issues are sufficiently difficult that a degree of moral uncertainty about them seems warranted.

*i. Totals.* Perhaps the simplest view ranks outcomes by the total amount of well-being they contain—call this *simple totalism*. On this view, adding good life-years constitutes a clear intrinsic benefit even without any gains in quality of life. This is the case whether those life-years come in the form of extended lives, or in the form of new people added to the population (say, by increasing fertility). So, of course, simple totalism will find much to like about anti-senescence technologies.

A common reason for rejecting this view is the intuition that *making people happy* matters more than *making happy people*.<sup>41</sup> Such an intuition suggests that if we could add a fixed amount of well-being to the world, it would be better to do so by increasing the well-being of already-existing people than by adding more people with good lives. Some theorists go so far as to deny that there is any benefit to increasing the number of people with positive well-being. For example, on their view, adding happy life-years to the world by increasing fertility wouldn't improve the world at all, at least not intrinsically.<sup>42</sup> Likewise for most of the expected gains in total well-being achieved by reducing existential risk.

Importantly, delaying senescence is different. It adds happy life-years—and combats population decline—without adding to the total stock of people who will ever live. And as we'll see, although

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<sup>40</sup>For the sake of simplicity, we'll set aside sources of value other than well-being. We also take no stand on the moral import of axiological comparisons: only strict consequentialism treats them as decisive when it comes to the rightness of an action.

<sup>41</sup>For an overview of some of the relevant issues and competing views see Greaves (2017).

<sup>42</sup>Raising fertility would also likely increase overall quality of life by the mechanism of increasing returns to scale, though it wouldn't obviously improve the labor force ratio, reduce medical costs, or expand horizons; and the benefit to scientific progress would not be as dramatic as that achieved by delaying senescence.

many views discount the value of adding extra happy people, they still tend to assign full value to more happy years lived by already-existing people. This means that the intrinsic value of these years is surprisingly robust across a range of ethical views.

*ii. Averages.* According to *averagism*, we should measure the goodness of an outcome by its *average* amount of well-being—that is, the total amount of well-being divided by the number of people who exist. On this view, adding happy people doesn’t intrinsically make the world better unless their level of well-being is higher than average. But adding happy life-years *does* intrinsically make the world better: it increases the numerator (the total amount of well-being) without increasing the denominator (the total number of lives). In short, on this view, a world with *longer* net-positive lives is *pro tanto* better, but a world with *more* net-positive lives is not.

*iii. Critical levels.* Another idea is to rank outcomes by total well-being but apply an absolute value tax on each individual’s contribution to the total. Thus, a given person’s well-being only counts positively towards the total if it’s higher than some “critical level”, so the world would be made no better by simply adding lives that are barely worth living.<sup>43</sup> Clearly, on this view, there is intrinsic value to adding life-years above the critical level. But there is once again an extra structural benefit to doing so without adding more *people*, because only by adding more people do we incur any additional tax. In short, on this view, delaying senescence achieves more good per life-year than if those same life-years came along with new people.

*iv. Value asymmetries.* Would it make the world better, if by causing unimaginable suffering to some already-existing people, a lot of new people with decent lives could be added? Many think not. One way to make good on this intuition is to modify totalism by giving extra weight to lives whose overall well-being is net-negative.<sup>44</sup>

As a variant of totalism, this view grants the intrinsic value of adding good life-years, even if this is done by adding new people. But it grants a structural advantage to adding good life-years without adding new people. To see why, imagine we are summing the numerical values of playing cards, where diamonds are negative and the other three suits are positive. Because of the law of large numbers, a hand with only a few cards is far more likely to be net-negative than a hand with many cards. Likewise, in a world where most experiences are good, shorter lives are more likely to be net-negative, while living longer gives people a better chance at an overall good life. (Moreover, note that

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<sup>43</sup> The level is *positive* in the sense that life can be worth living for people whose well-being is below that level; it’s just that adding such a life would not make the *world* better.

<sup>44</sup> Asymmetrical views aggregate *positive* well-being normally: my having a positive experience is intrinsically good to the degree that it makes my life better. But when it comes to negative well-being, there is a disconnect between the degree to which it makes a life worse, and the degree to which it is intrinsically bad.

value-asymmetric views will also amplify the benefit of improving quality of life, assuming this helps some people go from having net-negative to net-positive lives.)

v. *Slice-variants*. All three views just sketched could be revised so that, rather than using *entire lives* as the relevant unit—for averaging, or taxing, or assessing as net-negative—they use *time-slices of lives* instead. In that case, the structural benefits we’ve been discussing would no longer apply. For example, if we measure outcomes by dividing total well-being by some enumeration of life-moments, then adding happy years increases both the relevant numerator *and* the relevant denominator. Similarly, if we apply critical levels or value-asymmetries to time-slices or moments, there’s no structural benefit to adding life-years without adding people. Still, as far as we can tell, proponents of the views above intend to use whole lives and not slices as the relevant units of evaluation—perhaps because using slices has some strange consequences.<sup>45</sup>

vi. *Identities*. The idea behind so-called “person-affecting” views is that, when we consider possible outcomes of an action A, we should consider the well-being of people who *would have existed anyway* as in some way more important than the well-being of people who only exist as a result of the action—or “A-contingent people”.<sup>46</sup> This is yet another way of trying to cash out the making-people-happy intuition.

A major problem with this idea is that, if identities depend on particular gamete pairs, they are so precarious that any important discovery or policy change will pretty quickly alter the identities of almost everyone causally downstream. Some theorists avoid this result by replacing identity with a generous counterpart relation, so that only *extra* people count as A-contingent—or, to put it more carefully, there are only as many A-contingent people as the difference in population between the relevant outcomes (Meacham 2012).

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<sup>45</sup>Setting aside hard choices about thinness and overlap of slices, many of the things we value in life just don’t seem to fit into time-slices, in the sense that their value is not merely the additive value of the experience at every smaller slice. There are also counterintuitive results for specific variants. For example, slice-averagism tells us that a world of people who pop into existence fully formed only to live happily for a few hours/days would be as good as having those people live for a hundred years at the same level of happiness. For a slice-based critical level view, extending someone’s life in a way that is good for that person and doesn’t affect anyone else’s well-being might still make the world worse. (On the other hand, the slice-variant does keep “muzak-and-potatoes” lives of Parfit 1986 from exceeding the critical level just by getting longer) For slice-asymmetry, there’s a bias against high variance in well-being, to the point that it can make the world worse even if it makes one’s own life a bit better. It seems wrong that we could improve the world by smoothing out this variance, even at the cost of individual well-being.

<sup>46</sup>Note that such views deviate from a natural structural constraint on any axiology, viz. that whether a world A is better than a world B doesn’t depend on which world happens to be actual, or what the decision-situation is. As a result, they do not offer a theory of what makes some ways the world could be better, in an absolute way, than others. Or rather, since such views violate basic intuitive constraints on an axiology, we should really say that these identities matter to which of various possible outcomes is better-relative-to-our-decision-point.

Clearly, for person-affecting views of all stripes, the best way to add good life-years to the world is to do so without adding more people. At the outset, an anti-aging treatment would be enjoyed by people who would have existed whether or not it was discovered. And for counterpart-theoretic variants that avoid the problem of proliferating non-identities, the value of added years won't be discounted, because the outcomes don't differ in the total number of people they contain.

*vii. Side constraints.* So far we've been considering views that reflect the "making people happy" intuition in their axiologies—their theories of what sorts of things make the world as a whole better or worse. An alternative way of making good on that intuition is to embed it in a theory of right action, independently of our axiology. One might accept simple totalism (or reject axiologies altogether)<sup>47</sup> while positing special obligations towards already-existing people, or people who would-exist-anyway, or some counterpart-theoretic variant of that idea. (There could even be a special obligation to lift people over a critical level, or minimize net-negative lives.) In each case, there will be an analogous structural benefit to adding years without adding people, this time emerging from our theory of right action rather than our axiology.

To sum up this section: there are important structural benefits, across a wide range of views in population ethics, to adding years of life without adding more people—even setting aside the large increases in average quality of life established in earlier sections. This is important not just for those who accept one of these views in population ethics, but for anyone who affords them some weight due to moral uncertainty.<sup>48</sup>

## 4. Conclusion

At some point in the not-too-distant future, we expect that humanity will dramatically delay the onset of senescence. We have argued that such a development will have extraordinarily large benefits, especially against a backdrop of otherwise declining human populations. Moreover, these benefits are robust across ethical views, both through an increase in average quality of life, and through the intrinsic value of additional life-years.

Despite all this, investments in bringing this future forward have so far been small, mostly for reasons that have nothing to do with the genuine merits of the cause. The value of this research therefore deserves a thorough reconsideration by those who (i) appreciate uncertain but potentially high-reward research, (ii) care about benefits that may only accrue to our descendants, and (iii) can see past the cognitive biases that afflict the general public on this issue.

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<sup>47</sup> Indeed, this is arguably what "person-affecting" views already do: see the previous fn.

<sup>48</sup> See the related discussion in Greaves & Ord (2017).

## References

- Acemoglu, D., & Robinson, J. A. (2019), *The Narrow Corridor: States, societies, and the fate of liberty* (Penguin).
- Arrhenius, G., Ryberg, J. and Tännsjö, T. (2010), 'The Repugnant Conclusion', in *The Stanford Encyclopedia of Philosophy*.
- Austad, S. N. (1993), 'Retarded Senescence in an Insular Population of Opossums', *Journal of Zoology* 229: 695-708.
- Azoulay, P., Fons-Rosen, C., and Graff Zivin, J. S. (2019), 'Does Science Advance One Funeral at a Time?', *American Economic Review* 109/8: 2889-2920.
- Baron, J., and Ritov, I. (2004), 'Omission Bias, Individual Differences, and Normality', *Organizational Behavior and Human Decision Processes* 94/2: 74-85.
- Bartels, D. M., and Burnett, R. C. (2011), 'A Group Construal Account of Drop-in-the-Bucket Thinking in Policy Preference and Moral Judgment', *Journal of Experimental Social Psychology* 47/1: 50-57.
- Bartholomaeus, J., and Strelan, P. (2019), 'The Adaptive, Approach-Oriented Correlates of Belief in a Just World for the Self: A Review of the Research', *Personality and Individual Differences* 151: 109485.
- Barzilai, N., Crandall, J. P., Kritchevsky, S. B., and Espeland, M. A. (2016). 'Metformin as a Tool to Target Aging', *Cell Metabolism* 23/6: 1060-1065.
- Bejder, M., Johnston, D.W., Smith, J., Friedlaender, A. and Bejder, L. (2016), 'Embracing conservation success of recovering humpback whale populations: evaluating the case for downlisting their conservation status in Australia', *Marine Policy* 66: 137-141.
- Bitto, A., Ito, T. K., Pineda, V. V., LeTexier, N. J., Huang, H. Z., Sutlief, E., and Kaerberlein, M. (2016), 'Transient Rapamycin Treatment Can Increase Lifespan and Healthspan in Middle-Aged Mice', *eLife* 5: e16351.
- Bjedov, I., and Rallis, C. (2020), 'The Target of Rapamycin Signalling Pathway in Ageing and Lifespan Regulation', *Genes* 11/9: 1043.
- Boserup, E. (1965), *The Conditions of Agricultural Growth: The Economics of Agrarian Change Under Population Pressure* (Routledge).

Bostrom, N. (2003), 'Astronomical Waste: The Opportunity Cost of Delayed Technological Development', *Utilitas* 15/3: 308-314.

Bostrom, N. (2005), 'The Fable of the Dragon-Tyrant', *Journal of Medical Ethics* 31/5: 273-277.

Burke, B. L., Martens, A., and Faucher, E. H. (2010), 'Two Decades of Terror Management Theory: A Meta-Analysis of Mortality Salience Research', *Personality and Social Psychology Review* 14/2: 155-195.

Callan, M. J., Dawtry, R. J., and Olson, J. M. (2012), 'Justice Motive Effects in Ageism: The Effects of a Victim's Age on Observer Perceptions of Injustice and Punishment Judgments', *Journal of Experimental Social Psychology* 48/6: 1343-1349.

Campbell, J.M., Bellman, S.M., Stephenson, M.D., and Lisy, K. (2017), 'Metformin Reduces All-Cause Mortality and Diseases of Ageing Independent of Its Effect on Diabetes Control: A Systematic Review and Meta-Analysis', *Ageing Research Reviews* 40: 31-44.

Cohen, A.A., Kennedy, B.K., Anglas, U., Bronikowski, A.M., Deelen, J., Dufour, F., ... and Fülöp, T. (2020), 'Lack of Consensus on an Aging Biology Paradigm? A Global Survey Reveals an Agreement to Disagree, and the Need for an Interdisciplinary Framework', *Mechanisms of Ageing and Development* 191: 111316.

De Nardi, M., French, E., and Jones, J.B. (2009), 'Life Expectancy and Old Age Savings', *American Economic Review* 99/2: 110-115.

Diaz, D., Moore, F. (2017), 'Quantifying the economic risks of climate change', *Nature Climate Change* 7/11: 774-782.

Dickert, S., Västfjäll, D., Kleber, J., and Slovic, P. (2015), 'Scope Insensitivity: The Limits of Intuitive Valuation of Human Lives in Public Policy', *Journal of Applied Research in Memory and Cognition* 4/3: 248-255.

Eden, M., and Kuruc, K. (2023), 'The Marginal Benefits of Population: Evidence from a Malthusian Semi-Endogenous Growth Model', *Population Wellbeing Initiative Working Paper* 2305. Available at: [https://sites.utexas.edu/pwi/files/2023/01/Eden\\_Kuruc\\_Malthusian.pdf](https://sites.utexas.edu/pwi/files/2023/01/Eden_Kuruc_Malthusian.pdf).

Ehrlich, P. (1968), *The Population Bomb* (Ballantine Books).

Ellard, J.H., Harvey, A., and Callan, M.J. (2016), 'The Justice Motive: History, Theory, and Research', *Handbook of Social Justice Theory and Research*: 127-143.

Feldman, G. (2020), 'What is Normal? Dimensions of Action-Inaction Normality and Their Impact on Regret in the Action-Effect', *Cognition and Emotion*, 34/4: 728-742.

- Feldman, G., Kutscher, L., and Yay, T. (2020), 'Omission and Commission in Judgment and Decision Making: Understanding and Linking Action-Inaction Effects Using the Concept of Normality', *Social and Personality Psychology Compass* 14/8: e12557.
- Fillon, A., Kutscher, L., and Feldman, G. (2021), 'Impact of Past Behaviour Normality: Meta-Analysis of Exceptionality Effect', *Cognition and Emotion* 35/1: 129-149.
- Fox-Glassman, K. T., and Weber, E. U. (2016), 'What Makes Risk Acceptable? Revisiting the 1978 Psychological Dimensions of Perceptions of Technological Risks', *Journal of Mathematical Psychology* 75: 157-169.
- Geruso, M., and Spears, D. (2023), 'With a Whimper: Depopulation and Longtermism', *Essays on Longtermism* (Oxford University Press).
- Glossmann, H. H., and Lutz, O. M. (2019), 'Metformin and Aging: A Review', *Gerontology* 65/6: 581-590.
- Greaves, H. (2017), 'Population Axiology', *Philosophy Compass* 12/11: e12442.
- Greaves, H., and Ord, T. (2017), 'Moral Uncertainty About Population Axiology', *Journal of Ethics & Social Philosophy* 12: 135-167.
- Guo, J., Huang, X., Dou, L., Yan, M., Shen, T., Tang, W., and Li, J. (2022), 'Aging and Aging-Related Diseases: From Molecular Mechanisms to Interventions and Treatments', *Signal Transduction and Targeted Therapy* 7/391:1-40.
- Hafer, C. L., and Sutton, R. (2016), 'Belief in a Just World', in *Handbook of Social Justice Theory and Research*: 145-160.
- Hall, R.E. and Jones, C.I. (2007), 'The Value of Life and the Rise in Health Spending', *The Quarterly Journal of Economics* 122/1: 39-72.
- Hou, Y., Dan, X., Babbar, M., Wei, Y., Hasselbalch, S. G., Croteau, D. L., and Bohr, V. A. (2019), 'Ageing as a Risk Factor for Neurodegenerative Disease', *Nature Reviews Neurology* 15/10: 565-581.
- Hummel, S. (2020), 'Leader Age, Death, and Political Liberalization in Dictatorships', *The Journal of Politics* 82/3: 981-995.
- Jayachandran, S. and Lleras-Muney, A. (2009), 'Life Expectancy and Human Capital Investments: Evidence from Maternal Mortality Declines', *The Quarterly Journal of Economics* 124/1: 349-397.
- Johnson, S. C., Rabinovitch, P. S., and Kaeberlein, M. (2013), 'mTOR is a Key Modulator of Aging and Age-Related Disease', *Nature* 493/7432: 338-345.



- Johnson, S. G., Zhang, J., and Keil, F. C. (2022), 'Win-Win Denial: The Psychological Underpinnings of Zero-Sum Thinking', *Journal of Experimental Psychology: General* 151/2: 455-474.
- Jones, C.I. (2022), 'The End of Economic Growth? Unintended Consequences of a Declining Population', *American Economic Review* 112/11: 3489-3527.
- Jones, C.I. and Romer, P.M. (2010), 'The New Kaldor Facts: Ideas, Institutions, Population, and Human Capital', *American Economic Journal: Macroeconomics* 2/1: 224-45.
- Kendall-Taylor, A., and Frantz, E. (2016), 'When Dictators Die', *Journal of Democracy* 27/4: 159-171.
- Kennedy, B.K., Berger, S.L., Brunet, A., Campisi, J., Cuervo, A.M., Epel, E.S., Franceschi, C., Lithgow, G.J., Morimoto, R.I., Pessin, J.E., Rando, T.A., Richardson, A., Schadt, E.E., Wyss-Coray, T. and Sierra, F. (2014), 'Geroscience: Linking Aging to Chronic Disease', *Cell* 159/4: 709-713.
- Kirkwood, T. B., and Austad, S. N. (2000), 'Why Do We Age?', *Nature* 408/6809: 233-238.
- Kolora, S.R.R., Owens, G.L., Vazquez, J.M., Stubbs, A., Chatla, K., Jainese, C., ... and Sudmant, P.H. (2021), 'Origins and Evolution of Extreme Life Span in Pacific Ocean Rockfishes', *Science* 374/6569: 842-847.
- Kremer, M. (1993), 'Population Growth and Technological Change: One Million BC to 1990', *The Quarterly Journal of Economics* 108/3: 681-716.
- Kulkarni, A.S., Gubbi, S., and Barzilai, N. (2020), 'Benefits of Metformin in Attenuating the Hallmarks of Aging', *Cell Metabolism* 32/1: 15-30.
- Kupis, L., Goodman, Z.T., Kornfeld, S., Hoang, S., Romero, C., Dirks, B., Dehoney J., et al. (2021), 'Brain Dynamics Underlying Cognitive Flexibility Across the Lifespan', *Cerebral Cortex* 31/11: 5263-5274.
- Kuruc, K., Vyas, S., Budolfson, M., Geruso, M. and Spears, D. (2023), 'A Larger World Population Raises Average Living Standards, Net of Climate Damages', *Population Wellbeing Initiative Working Paper* w2302. Available at: [https://sites.utexas.edu/pwi/files/2023/01/Stabilization\\_Climate.pdf](https://sites.utexas.edu/pwi/files/2023/01/Stabilization_Climate.pdf).
- Lam, D. (2011), 'Presidential Address to The Population Association of America'.
- Li, J., Kim, S.G., and Blenis, J. (2014), 'Rapamycin: One Drug, Many Effects', *Cell Metabolism* 19/3: 373-379.

- Loewenstein, G. F., Weber, E. U., Hsee, C. K., and Welch, N. (2001), 'Risk as Feelings', *Psychological Bulletin* 127/2: 267-286.
- López-Otín, C., Blasco, M. A., Partridge, L., Serrano, M., and Kroemer, G. (2023), 'Hallmarks of Aging: An Expanding Universe', *Cell* 186/2: 243-278.
- MacRae, S. L., Croken, M. M., Calder, R. B., Aliper, A., Milholland, B., White, R. R., ... and Vijg, J. (2015), 'DNA Repair in Species with Extreme Lifespan Differences', *Aging* 7/12: 1171.
- McAskill, W. (2022), *What We Owe the Future* (Hachette Book Group).
- Mannick, J. B., Del Giudice, G., Lattanzi, M., Valiante, N. M., Praestgaard, J., Huang, B., ... and Klickstein, L. B. (2014), 'mTOR Inhibition Improves Immune Function in the Elderly', *Science Translational Medicine* 6/268: 268ra179.
- Martin-Montalvo, A., Mercken, E. M., Mitchell, S. J., Palacios, H. H., Mote, P. L., Scheibye-Knudsen, M., ... and De Cabo, R. (2013), 'Metformin Improves Healthspan and Lifespan in Mice', *Nature Communications* 4/1: 1-9.
- Meacham, C. J. (2012), 'Person-Affecting Views and Saturating Counterpart Relations', *Philosophical Studies* 158: 257-287.
- Meier, B. P., Dillard, A. J., and Lappas, C. M. (2019), 'Naturally Better? A Review of the Natural-is-Better Bias', *Social and Personality Psychology Compass* 13/8: e12494.
- Mincer, J. (1974), *Schooling, Experience, and Earnings* (Columbia University Press).
- Nagai, J., Lin, C. Y., and Sabour, M. P. (1995), 'Lines of Mice Selected for Reproductive Longevity', *Growth, Development, and Aging: GDA* 59/3: 79-91.
- Ord, T. (2020), *The Precipice: Existential Risk and the Future of Humanity* (Hachette Book Group).
- Oster, E., Shoulson, I., and Dorsey, E. (2013), 'Limited Life Expectancy, Human Capital, and Health Investments', *American Economic Review* 103/5: 1977-2002.
- Ou, Y., Iyer, G., Clarke, L., Edmonds, J., Fawcett, A. A., Hultman, N., ... and McJeon, H. (2021), 'Can updated climate pledges limit warming well below 2° C?', *Science* 374/6568: 693-695.
- Parfit, D. (1986), 'Overpopulation and the Quality of Life', in P. Singer (ed.), *Applied Ethics* (Oxford University Press), 145-164.
- Peters, M. (2022), 'Market Size and Spatial Growth—Evidence from Germany's Post-war Population Expulsions', *Econometrica* 90/5: 2357-2396.

Pew Research Center (2013), 'Living to 120 and Beyond: Americans' Views on Aging, Medical Advances and Radical Life Extension'.

Prentice, R. A., and Koehler, J. J. (2002), 'A Normality Bias in Legal Decision Making', *Cornell Legal Review* 88/3: 583-650.

Public Policy Polling, (2023), 'National Survey on Behalf of the Alliance for Longevity Initiatives' [https://a4li.org/wp-content/uploads/2022/01/NationalPollResults\\_1.22.pdf](https://a4li.org/wp-content/uploads/2022/01/NationalPollResults_1.22.pdf)

Rose, M. R., Passananti, H. B., and Matos, M. (eds.) (2004), *Methuselah Flies: A Case Study in the Evolution of Aging* (World Scientific).

Routledge, C., Ostafin, B., Juhl, J., Sedikides, C., Cathey, C., and Liao, J. (2010), 'Adjusting to Death: The Effects of Mortality Salience and Self-Esteem on Psychological Well-Being, Growth Motivation, and Maladaptive Behavior', *Journal of Personality and Social Psychology* 99/6: 897.

Scott, A. J., Ellison, M., and Sinclair, D. A. (2021), 'The Economic Value of Targeting Aging', *Nature Aging* 1/7: 616-23.

Scott, S. E., and Rozin, P. (2020), 'Actually, Natural is Neutral', *Nature Human Behaviour* 4/10: 989-990.

Seluanov, A., Gladyshev, V. N., Vijg, J., and Gorbunova, V. (2018), 'Mechanisms of Cancer Resistance in Long-Lived Mammals', *Nature Reviews Cancer* 18/7: 433-41.

Selvarani, R., Mohammed, S., and Richardson, A. (2021), 'Effect of Rapamycin on Aging and Age-Related Diseases—Past and Future', *Geroscience* 43: 1135-1158.

Simon, J. (1981), *The Ultimate Resource* (Princeton University Press).

Slovic, P., Finucane, M. L., Peters, E., and MacGregor, D.G. (2013), 'Risk as Analysis and Risk as Feelings: Some Thoughts about Affect, Reason, Risk, and Rationality', in *The Feeling of Risk* (Routledge), 21-36.

Slovic, P., and Västfjäll, D. (2010), 'Affect, Moral Intuition, and Risk', *Psychological Inquiry* 21/4: 387-398.

Sunstein, C.R. (2003), 'Terrorism and Probability Neglect', *Journal of Risk and Uncertainty* 26: 121-136.

Tian, X., Firsanov, D., Zhang, Z., Cheng, Y., Luo, L., Tomblin, G., and Gorbunova, V. (2019), 'SIRT6 is Responsible for More Efficient DNA Double-Strand Break Repair in Long-Lived Species', *Cell* 177/3: 622-638.

Tomczyk, S., Fischer, K., Austad, S., and Galliot, B. (2015), 'Hydra, a Powerful Model for Aging Studies', *Invertebrate Reproduction & Development* 59(sup1): 11-16.

United Nations, Department of Economic and Social Affairs, Population Division, (2022), *World Population Prospects 2022: Methodology of the United Nations Population Estimates and Projections*, UN DESA/POP/2022/TR/NO. 4.

Weichhart, T. (2018), 'mTOR as Regulator of Lifespan, Aging, and Cellular Senescence: A Mini-Review', *Gerontology* 64/2: 127-134.

Wilkinson, J. E., Burmeister, L., Brooks, S. V., Chan, C. C., Friedline, S., Harrison, D. E., and Miller, R.A. (2012), 'Rapamycin Slows Aging in Mice', *Aging Cell* 11/4: 675-682.

Yang, J. H., Hayano, M., Griffin, P. T., Amorim, J. A., Bonkowski, M. S., Apostolides, J. K., ... and Sinclair, D. A. (2023), 'Loss of Epigenetic Information as a Cause of Mammalian Aging', *Cell* 186/2: 305-326.

Yeung, S. K., Yay, T., and Feldman, G. (2022), 'Action and Inaction in Moral Judgments and Decisions: Meta-Analysis of Omission Bias Omission-Commission Asymmetries', *Personality and Social Psychology Bulletin* 48/10: 1499-1515.

Zhang, B., Trapp, A., Kerepesi, C., and Gladyshev, V.N. (2022), 'Emerging Rejuvenation Strategies—Reducing the Biological Age', *Aging Cell* 21/1: e13538.