All's Well That Ages Well: The Economic Value of Targeting Aging*

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Abstract

Human lifespans are increasing with advances in medicine, but the economic value of these gains are poorly understood. Based on U.S. data, we show a compression of morbidity that improves health is more valuable than further increases in life expectancy. However, economic gains to better health diminish unless longevity also improves. Treatments that target aging are hence particularly valuable, as they produce both healthier and longer lives. We calculate a slowdown in aging that increases life expectancy by one year is worth $38 trillion, and for ten years $367 trillion. Evaluating the impact of metformin shows targeting aging offers potentially larger economic gains than eradicating individual diseases. Complementarities between health, longevity and age lead to a virtuous circle that means improvements in aging increase the value of further gains. Aligned with trends in demographics and disease, this implies the gains from age targeting treatments will increase further in the decades ahead.

1 Introduction

Life expectancy has increased dramatically over the last 150 years ([26]), although not all the extra years gained have been healthy. The Global Burden of Disease dataset ([16]) estimates that the proportion of life in good health has remained broadly constant between 1990 and 2019, implying an increasing number of years spent in poor health. Furthermore, the disease burden is shifting towards chronic non-communicable diseases, which are estimated to have caused 72.3% of U.S. deaths in 2016. The result, according to [27], is 'a substantial part of life, and certainly most deaths, now occur in a period in the lifespan when the risk for frailty and disability increases exponentially.' As a consequence, there is a growing emphasis on 'healthy aging' and an emerging body of research focusing on the biology of aging (see [32]) and [4]). According to [6], 'this era marks an inflection point, not only in aging research but also for all biological research that affects the human healthspan.'

These developments pose a number of important questions: Is it preferable to make lives healthier by compressing morbidity or longer by extending life? What are the gains from targeting aging itself, with its potential to make lives both healthier and longer? How does the value of treating aging compare to eradicating specific diseases? How will these gains evolve over time and be affected by demographic trends?

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We take an economic rather than biological perspective to answer these questions. Specifically, we use the Value of Statistical Life (VSL) approach to place a monetary value on the economic gains from longer life, better health, and changes in the rate at which we age ([36], [2], [25]).[1]

VSL models have two distinct advantages for our purposes. Firstly, they are already used by a variety of government agencies to evaluate different policy measures and treatments, e.g., [30], [11]. Secondly, by modeling how economic decisions interact with changes in health and longevity, we can analyze not just the current gains to targeting aging but how these gains will evolve in the future. The results reveal a distinctive feature of age-targeting treatments. Interactions between health, longevity, economic decisions and demographics create a virtuous circle, such that the more successful society is in improving how we age the greater the economic value of further improvements.

2 Results

The economic model for our analysis is based on [25] and calibrated to current U.S. economic, health and demographic data. In the model, individuals make choices about consumption, hours worked and leisure based on wage rates, interest rates, retirement age, and knowledge of remaining life expectancy and likely future health.[2] We use the model to estimate an individual's Willingness to Pay (WTP) for improvements in health and longevity. WTP is measured in U.S. dollars and is derived from the increase in the Value of Statistical Life (VSL) implied by improvements in health and longevity. VSL itself is the sum of the value of each remaining year of life, discounted to the present day and weighted by the survival rate. Because the value of each year of life depends on health, consumption and leisure, the VSL incorporates both the quantity and quality of expected life remaining. Importantly, this means that the VSL is higher than an individual's lifetime income – life is valuable in its own right because individuals value time, health and leisure.

The demographic data underpinning our analysis are: i) a survival function from [18], whereby mortality risk increases exponentially with age; ii) a health deficit function from [24], also increasing exponentially with age, and iii) the 2017 population structure and birth rates from the U.S. Census Bureau. In our baseline calculations, life expectancy (LE) and healthy life expectancy (HLE) at birth are 78.9 and 68.5 years respectively, in line with current U.S. data. Following [19], we set the average VSL of an adult aged between 25 and 65 years to $11.5 million. Whilst our dollar WTP values are sensitive to the precise calibration of our model, the relative importance of different treatments for aging is not.

2.1 Life Extension – The Struldbrugg Case

We first focus on improving life expectancy (LE) which, with reference to "*Gulliver's Travels*" [35], we label the 'Struldbrugg' case. Struldbruggs are born immortal but age normally and so live in continuously worsening health. In our simulations, we achieve this by reducing the rate at which mortality declines with age while holding the rate at which health declines unchanged. The result is an expansion of morbidity, whereby a greater proportion of life is spent in poor health. Whilst both LE and HLE improve, the ratio of HLE to LE deteriorates.

[1] A complementary approach based on a microsimulation model is taken by [17], whose results we discuss in the final section.
[2] Appendices A-D describe the model and its calibration in detail.

The Willingness to Pay (WTP) for increases in LE depends on which years benefit from lower mortality. To provide consistency across simulations, we assume that mortality is subject to a compensating effect [33] whereby it reaches a rate M at age T. Under this specification, there are two ways to extend LE. The first is via a rectangularization of the survival function such that M and T are kept constant but mortality is reduced at all ages less than T so that it rises more rapidly at T.[3] Rectangularization is shown by the red survival function in Figure 1. The second involves an improvement in lifespan, such that mortality reaches M at higher values of T. In this case, survival rates decline more slowly and there is an increased probability of living beyond T, shown by the yellow survival function in Figure 1.

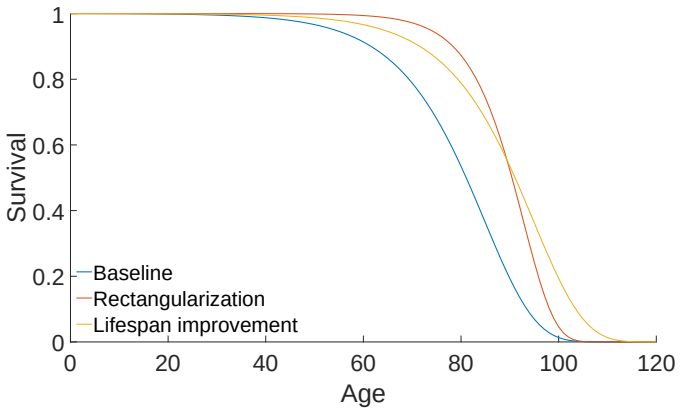

Figure 1: Survival functions under rectangularization and improvement in lifespan

Table 1 shows the WTP for one year increases in remaining LE. The major columns report the WTP at age 0, 20, 40, 60 and 80, with R the WTP for a one year increase via rectangularization and L the WTP for one year increases through improvements in lifespan. The first row (labeled '1^{st}+1 year') is the WTP for the initial one year increase from our baseline calibration. For example, the WTP at birth for the initial one year increase in remaining LE from 78.9 to 79.9 years via rectangularization is \$118,100, and the WTP at age 60 for the first one year increase in remaining LE from 21.7 to 22.7 years through lifespan improvement is \$257,700. The subsequent rows (labeled '2^{nd}+1 year', '3^{rd}+1 year' and so on) show the WTP for additional one year increases, so the row '10^{th}+1 year' in column '20' shows the WTP at age 20 for an increase in remaining LE from 68.0 to 69.0 years, since by the tenth increment the remaining LE at 20 has already risen to 68.0 years.

Three results stand out: i) the WTP for additional gains in LE diminishes as LE rises, ii) the WTP for additional years of LE is greatest for the oldest, and iii) rectangularization is preferred to improvement in lifespan. The WTP is diminishing because as LE rises the gains to further increases accrue progressively more in the future (and hence are discounted more) and in years of poor health (which are less valuable). The fact that WTP increases with age is also partly due to discounting, since the old experience the benefits of extra LE sooner than the young, but mainly because as an individual ages they have a higher probability of reaching even older ages. The likelihood that someone aged 80 will reach 81 is greater than the likelihood of someone aged 20 reaching the same age, hence an older individual values gains in LE in later years more.

[3]The slope of the mortality curve becomes steeper at T under rectangularization, so mortality rates are lower before but *higher* after T. We restrict rectangularization to only operate before T so its benefits are unambiguously positive.

Table 1: Struldbrugg: Willingness to Pay (WTP) for one year increases in remaining life expectancy ($ thousand)

	Age at which WTP calculated									
	0		20		40		60		80	
	R	L	R	L	R	L	R	L	R	L
1^{st}+1 year	118.1	96.5	171.0	141.5	232.0	199.4	285.6	257.7	312.2	288.1
2^{nd}+1 year	114.1	93.4	165.5	137.1	226.1	193.2	279.7	250.0	304.4	278.1
3^{rd}+1 year	110.0	90.4	160.1	132.7	220.0	187.2	273.8	242.1	298.0	268.5
4^{th}+1 year	105.9	87.5	154.5	128.4	213.8	181.3	267.7	234.6	292.0	259.4
5^{th}+1 year	101.8	84.6	148.9	124.3	207.4	175.6	261.5	227.4	286.0	250.7
10^{th}+1 year	81.8	71.5	120.8	105.0	173.0	149.1	226.2	193.8	225.3	212.2
20^{th}+1 year		50.3		74.0		105.9		139.3		152.7
30^{th}+1 year		35.0		51.6		74.3		98.8		109.5

Notes: R denotes rectangularization, L improvements in lifespan T. The rows show the Willingness to Pay (WTP) for the 1^{st}, 2^{nd}, 3^{rd} ... 30^{th} one year increase in remaining life expectancy (LE) at ages 0, 20, 40, 60, 80. The LE remaining at these ages in the baseline simulation is 78.9, 59.0, 39.5, 21.7 and 8.4 years. Some values are missing because there is an upper limit to how much LE can be extended through rectangularization.

Rectangularization dominates improvements in lifespan because it concentrates increases in LE in years when health is better.

2.2 Compressing Morbidity – The Dorian Gray Case

We now hold LE fixed but improve the relationship between health and age. Under this scenario, HLE rises as a proportion of LE, leading to a '*compression of morbidity*' [15]. We refer to this as the 'Dorian Gray' case ([38]). In the novel, Dorian Gray has a portrait painted and whilst the picture ages, Gray himself doesn't, retaining his health and looks until he dies. Following [1], we assume morbidity is also subject to a compensating effect. Our baseline health function declines to H^* at age T^*, meaning that we again have two ways to improve HLE. Under rectangularization, gains are reflected in better health prior to T^* but a faster deterioration around T^*, whereas improvements in healthspan stretch the health function so it declines to H^* at higher values of T^*.

The results, shown in Table 2, indicate that the WTP for improvements in HLE is diminishing as HLE rises, and that the WTP is again increasing with age. As in the Struldbrugg case, this reflects a combination of discounting and the higher probability of older individuals reaching even older ages. However, an additional force is at work in this case because better health also raises the benefits of consumption and leisure. As health improves in later life, individuals respond by allocating more consumption and leisure to these years and so gains in health at older ages become even more attractive. This shifting of consumption to later years also explains why rectangularization is initially preferred as HLE gains extend. As HLE increases, more of the gains in HLE are coming from later years and improvements in lifespan bring larger proportional gains in later years.

Tables 1 and 2 reveal that the economic value of gains from an extra year of HLE are always larger than those from an extra year of LE. An increase in LE in the Struldbrugg case gives the individual additional years in which to enjoy lifetime consumption and leisure, but declining health makes this less appealing than the increase in health at each age that is the Dorian Gray case. This preference for HLE over LE extends to support a full compression of morbidity. Even though the WTP for additional years of HLE is declining in

Table 2: Dorian Gray: Willingness to Pay (WTP) for one year increases in remaining healthy life expectancy ($ thousand)

	Age at which WTP calculated									
	0		20		40		60		80	
	R	H	R	H	R	H	R	H	R	H
1^{st}+1 year	242.0	216.3	377.0	328.5	472.7	429.7	570.8	536.9	692.5	653.8
2^{nd}+1 year	233.4	210.0	359.0	317.8	452.3	416.8	538.0	519.2	612.0	618.6
3^{rd}+1 year	224.1	203.8	341.0	307.6	432.6	404.6	513.4	503.7	588.7	598.8
4^{th}+1 year	214.2	197.9	322.6	297.7	413.0	393.0	493.0	489.8	531.6	583.9
5^{th}+1 year	203.7	192.1	303.8	288.3	393.1	381.9	474.0	477.1		
10^{th}+1 year	136.6	165.3	197.0	245.7	230.0	331.6				

Notes: R denotes rectangularization, H improvements in healthspan T^*. The rows show the Willingness to Pay (WTP) for the 1^{st}, 2^{nd}, 3^{rd} ... 10^{th} one year increase in remaining healthy life expectancy (HLE) at ages 0, 20, 40, 60, 80. The HLE remaining at these ages in the baseline simulation is 68.5, 48.8, 30.4, 14.9 and 4.8 years. Some values are missing because there is an upper limit to how much HLE can be extended through rectangularization or improvements in lifespan.

Table 2, it never falls below the WTP for the first increase in LE in Table 1. Individuals always prefer an extra year of healthy life expectancy to adding an additional year to current U.S. life expectancy.

2.3 Slowing Aging – The Peter Pan case

We now consider the WTP for slowing down aging itself, which leads to simultaneous improvements in health and mortality. To do so we assume aging occurs through the accumulation of biological damage. Slower aging reduces the rate at which this damage accrues, lessening the pace at which health and mortality deteriorate with age. In the extreme case, where aging is not just slowed but eliminated, mortality and health become independent of age and the individual is 'forever young'. We refer to this as the "*Peter Pan*" case, after the eponymous play and novel [3].[4]

To allow for a slowdown in aging we multiply chronological age a by a constant δ. For $\delta = 1$, biological damage accumulates at its current rate but for $\delta < 1$ each passing year produces less damage and smaller deteriorations in health and morality. The lower is δ the more slowly aging occurs and the greater the gap between biological and chronological age. The 'forever young' case is given by $\delta = 0$.[5]

In contrast to Struldbrugg and Dorian Gray, the WTP now consists of two components, one representing the gains in mortality and one for the gains in health. Table 3 shows the total WTP for slowing down aging where, to ensure comparability with early results, we vary δ so as to achieve one year step increases in LE. Compared to the Struldbrugg case, Peter Pan brings about larger WTP because now both health and LE are increasing. The WTP for further improvements in aging is still declining but it does so at a slower rate due to the complementarities between health and longevity. The higher is life expectancy the greater the WTP for an increase in health, and the better is health the greater the WTP for improvements in life expectancy.

As previously, Table 3 shows that the WTP for improvements in LE increase across age bands. In other words, the gains from slowing down aging are greater for the old. This is consistent with the argument that we are entering a fourth stage of Omran's epidemiological transition - 'the age of delayed degenerative

[4] "Mrs Darling put her hand to her heart and cried "Oh, why you can't remain like this for ever!" This was all that passed between them on the subject, but henceforth Wendy knew that she must grow up. You always know after you are two. Two is the beginning of the end."

[5] Details are in Appendix B.

diseases' ([29], [28]). According to Table 3, the value of improvements in aging will rise as the average age of society increases, leading to a shift in the diseases the medical system should focus on. We return to this dynamic when discussing the aggregate gains to society.

Table 3: Peter Pan: Willingness to pay (WTP) for one year increases in remaining life expectancy ($ thousand)

	Age at which WTP calculated				
	0	20	40	60	80
1^{st}+1 year	178.7	262.6	333.9	378.5	380.2
2^{nd}+1 year	175.1	257.4	328.5	373.8	377.7
3^{rd}+1 year	171.5	252.2	323.1	369.2	375.0
4^{th}+1 year	168.0	247.0	317.8	364.6	372.0
5^{th}+1 year	164.5	241.9	312.4	360.0	368.9
10^{th}+1 year	147.5	217.3	286.3	337.6	352.6
20^{th}+1 year	116.9	172.6	236.1	293.1	319.1
30^{th}+1 year	91.1	134.7	189.6	247.3	281.6

Notes: The rows show the Willingness to Pay (WTP) for the 1^{st}, 2^{nd}, 3^{rd} ... 30^{th} one year increase in remaining life expectancy (LE) at ages 0, 20, 40, 60, 80. The life expectancy remaining at these ages in the baseline simulation is 78.9, 59.0, 39.5, 21.7 and 8.4 years.

2.4 Reversing Aging – The Wolverine case

An alternative to the Peter Pan scenario is a reversal of aging whereby biological damage is repaired rather than its accumulation slowed. For our literary reference we turn to the Marvel character Wolverine ([8]), who possesses a healing factor enabling body tissue to be regenerated. Recent advances have shown that such regeneration is possible in mice and humans ([13], [22]).

We capture this by assuming a one-time intervention at age 65 that rewinds an individual's biological clock back to a specific age Z, e.g., health and mortality rates are reset at the level previously associated with age Z.[6] Table 4 reports the WTP for such a reversal of aging, where gains are measured as before in increments of one-year increases in life expectancy at various ages. For example, row '1^{st}+1 year' in column '0' shows a total WTP of $103,500 for a first reversal in aging at age 65 that increases life expectancy at birth from 78.9 to 79.9 years.

Whilst reversing aging sounds more dramatic than a slowing down of age, the differences in our model are subtle. This is because we assume aging slows down over the entire adult life while a reversal occurs only at age 65. For this reason, the WTP for Peter Pan in Table 3 is greater than that for Wolverine in Table 4 at younger ages. As a result, the WTP for reversal rises faster with age than under Peter Pan. This effect is heightened under Wolverine because reversal leads to a relative improvement of health at older compared to younger ages, and so these years become more valuable as relatively more consumption is allocated to them.

[6] Details are in Appendix B.

Table 4: Wolverine: Willingness to Pay (WTP) for reversing aging at age 65 ($ thousand)

	Age at which WTP calculated				
	0	20	40	60	80
1^{st}+1 year	103.5	153.8	228.6	339.6	372.9
2^{nd}+1 year	103.0	153.1	227.5	338.2	373.4
3^{rd}+1 year	102.5	152.3	226.4	336.7	373.4
4^{th}+1 year	102.0	151.5	225.2	335.1	373.0
5^{th}+1 year	101.4	150.7	224.0	333.4	372.4
10^{th}+1 year	98.2	145.9	216.9	323.9	366.0
20^{th}+1 year	90.1	133.9	199.4	299.9	343.1
30^{th}+1 year	80.7	120.0	179.0	272.3	313.6

Notes: The rows show the Willingness to Pay (WTP) for a reversal of aging at 65 that leads to the 1^{st}, 2^{nd}, 3^{rd} ... 30^{th} one year increase in remaining life expectancy (LE) at ages 0, 20, 40, 60, 80. The LE remaining at these ages in the baseline simulation is 78.9, 59.0, 39.5, 21.7 and 8.4 years.

2.5 Targeting Aging vs Single Diseases

The results in the Peter Pan and Wolverine cases suggest that the gains to slowing or reversing aging are substantial. This raises two further questions: i) how much can aging be realistically slowed? and ii) how does the WTP for slowing down aging compare to that for the reduction or eradication of specific diseases? In this section, we explore these questions with reference to metformin, a drug prescribed for Type 2 diabetes that is considered to produce 'protective effects against several age-related diseases' [5]. Our focus on metformin is not intended to advocate its specific use compared to other treatments. Instead, it is motivated by the availability of estimates of its impact on the incidence of various age-related co-morbidities in a study [37] of 41,204 diabetic men with an average age of 75.

The study by [37] estimates a set of factors $0 \leq \lambda_{a,i} \leq 1$ that measure the reduction in the incidence of disease i after a years of treatment. Denoting the incidence of disease in the absence of metformin by $\pi_{a,i}$ and the same incidence when taking metformin by $\pi^*_{a,i}$, the factors satisfy $\pi^*_{a,i} = \lambda_{a,i}\pi_{a,i}$. If $\lambda_{a,i} = 1$ then metformin has no effect on the incidence of the disease; if $\lambda_{a,i} = 0$ the disease is completely eradicated. The factors after five years of treatment are 0.52 for dementia, 0.33 for cardiovascular diseases, 0.32 for cancer, 0.29 for depression, and 0.58 for frailty-related diseases, representing substantial reductions in the incidence of these five disease categories of between 42% and 71%. We use the Global Burden of Disease dataset ([16]) to identify the number of U.S. deaths and years lost to illness due to each of the age-related morbidities, and adjust them downwards when taking metformin by the $\lambda_{a,i}$ factors. As with Peter Pan and Wolverine, the WTP for metformin consists of two components representing the gains to mortality as well as health arising from reductions in the incidence of these five diseases.

There are two reasons to expect large gains when comparing metformin to single disease treatments. The first is the rising prevalence of age-related co-morbidities, which makes treatments targeting aging valuable as their impact will be felt across multiple diseases (see [17]). The second is synergies *between* diseases: reducing the incidence of any given disease has more impact on life expectancy and health when the incidence of other diseases is also reduced, the competing risks argument in [10].

We make three assumptions regarding the age at which treatment starts: 75 (the average age of participants in the study), 65 (all participants are over 65), and 50. Because [37] only study diabetic men over the age of

65, the $\lambda_{a,i}$ factors may not accurately capture the impact of metformin on women, non-diabetics, or those aged less than 65. Metformin may also have less of an impact at even higher ages [12].[7] However, the WTP calculations we present are broadly linear in the $\lambda_{a,i}$ factors so it is relatively easy to scale the gains up or down. For example, if the impact for non-diabetics is only 10% of that for diabetics then multiplying the WTP by 0.1 gives an appropriate estimate of the gains.

Based on [37] metformin has a sizable effect on life expectancy. At birth it rises by 2.9 years, whether metformin is started at age 75, 65 or 50. The increase in LE extends to all ages; starting metformin at 75 raises remaining LE by 3.0 years at 20, 3.0 years at 40, 3.3 years at 60, and 4.3 years at 80. The additions to remaining HLE at birth vary from 1.7 to 2.5 years.

Table 5: Willingness to pay (WTP) for metformin treatments and disease eradications ($ thousand)

	Age at which WTP calculated				
	0	20	40	60	80
Metformin started at age 75	188.4	281.4	429.2	697.7	1365.7
Metformin started at age 65	365.9	546.3	833.2	1354.6	1835.0
Metformin started at age 50	712.5	1064.0	1622.8	2204.1	2336.7
Eradication of cancer	361.8	528.5	749.4	840.6	436.7
Eradication of dementia	68.4	102.0	155.6	247.2	397.1
Eradication of cardiovascular disease	439.7	651.9	928.7	1139.3	1123.9

Notes: The life expectancy remaining at ages 0, 20, 40, 60, 80 when not taking metformin is 78.9, 59.0, 39.5, 21.7 and 8.4 years. At these ages when taking metformin it is 82.7, 62.9, 43.3, 25.2 and 3.0 years.

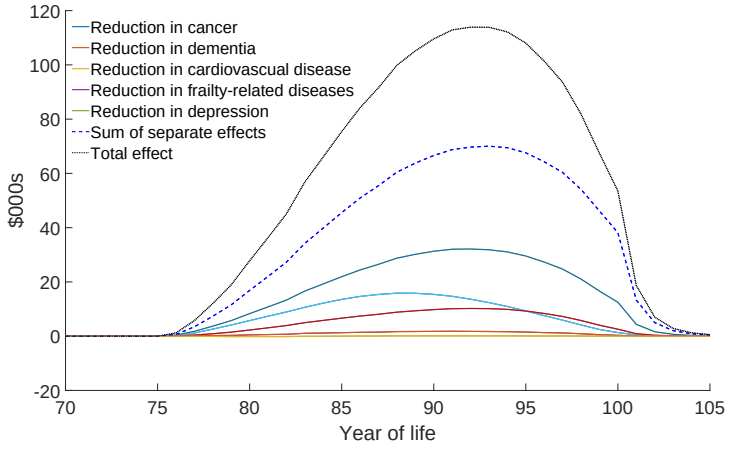

Figure 2: Willingness to Pay (WTP) by year of life for metformin treatment started at the age of 75

As shown in Table 5, the benefits of metformin are substantial, often matching or exceeding those from the complete eradication of cancer, dementia or cardiovascular diseases. Because the benefits accumulate over time, WTP rises with age and the greatest gains come from starting treatment early. Figure 2 decomposes

[7]The study by [37] estimates $\lambda_{a,i}$ for $65 < j < 74$, so we extend the factors to older ages by extrapolating a generalized logistic function that is fitted to the first 9 years. The effects of treatment have mostly leveled off after 9 years so this make little difference.

the WTP for metformin starting at 75 by year of life in which the benefits occur. The solid lines are the WTP for each of the five co-morbidities separately, the dashed line is the sum of these separate effects, and the dotted line is the total WTP for metformin. The total significantly exceeds the sum of the separate effects due to metformin's beneficial impact on competing risks.

2.6 Aggregate Gains

We now sum across the individuals in the population to calculate the aggregate WTP for society from slowing down aging. To fully capture the gains to society we also need to include the benefits to as yet unborn generations, as in [25]. Focusing on the aggregate WTP reveals a powerful additional dynamic affecting the value of targeting aging. Slowing down aging leads to a population which is on average older and larger (as more people live for longer), both of which lead to a higher aggregate WTP for further improvements. There is then a virtuous circle around improvements in aging – the better society ages the more valuable are further gains. We calculate this aggregate WTP by combining the age-specific individual WTPs from the Peter Pan scenarios with the latest U.S. Census Bureau data on the population, its age structure and birth rates. Once again, for consistency, we measure improvements in terms of step increases in life expectancy, adjusting the speed of aging to achieve these incremental gains.

Based on the '1^{st}+1 year' row of Table 3 and current census data, the total WTP for a 2017 slowdown in aging leading to a +1 increase in LE is $37.6 trillion ($29.7 trillion for those alive in 2017 and $7.9 trillion for those not yet born). The corresponding number for a +10 year increase in LE is $366.8 trillion (split $291.9 trillion, $74.8 trillion). Based on a 2% interest rate, the value of this 10-year increase is $7.2 trillion at an annual rate (or 33.6% of 2019 GDP).

The value of a further additional improvement in aging in 2050 is shown in rows four to six of Table 6. These depend on the individual WTPs for a second incremental slowing of aging (e.g., the 2^{nd} row of Table 3) as well as the projected population age structure in 2050. To obtain estimates of the latter, we start from the 2017 population and forecast forward using current birth rates and mortality rates from 2017 adjusted for the assumed initial improvement in aging. We abstract from net immigration by setting it to zero. Using the positive Census Bureau projections for immigration would produce higher estimated aggregate WTPs.

The aggregate WTPs for the 2017 and 2050 improvements in aging are of similar magnitudes. For smaller improvements in LE the WTPs for the second wave are worth slightly less (approximately 1-2%) but for larger improvements slightly more (approximately 1%). To better understand this pattern, Table 6 provides a decomposition (described in Appendix C) of the factors driving the change in the aggregate WTP. One reason the aggregate WTP changes between the two rounds is due to changes in individual age specific WTPs. As shown in Table 3, the WTP for further improvements in aging declines at each age and so aggregate WTP is lower. This effect is shown in the first line of the decomposition, where the population structure and size are held constant between 2017 and 2050 but the lower individual WTPs applied. For the case of +1 year LE this lowers the aggregate WTP by $45.8 billion. The aggregate WTP will also change between the two waves of aging improvements because of changes in population. Independent of the improvements in aging we model, the US population is expected to see an increase in its average age of 4.0 years between 2017 and 2050 and (assuming zero net immigration) a decline of 1.6 million people. The increase in average age boosts the aggregate WTP (improvements in aging are more valuable for the old) whilst a shrinking population lowers it (aggregation occurs over fewer people). The second row of the decomposition shows the

aging effect dominates, so that the combined impact is positive and raises the aggregate WTP between the two sets of improvements (by $113.7 billion in the +1LE case).

Table 6: Society willingness to pay (WTP) for successive slowdowns in aging ($ trillion)

	Increase in remaining life expectancy in years				
	+1	+2	+3	+5	+10
Society WTP for first slowdown in aging in 2017					
- living population	29.7	59.3	88.8	147.4	291.9
- unborn generations	7.9	15.6	23.3	38.5	74.8
- total	37.6	75.0	112.1	185.9	366.8
Society WTP for second slowdown in aging in 2050					
- living population	30.0	60.3	90.7	152.1	307.9
- unborn generations	6.8	13.5	20.1	32.9	62.4
- total change	36.9	73.8	110.8	185.0	370.3
Change in society WTP from 2017 to 2050					
Change in individual WTP between first and second slowdown of aging	-0.05	-0.20	-0.50	-1.63	-8.82
Change in society WTP from increase in population due to slowdown of aging	0.11	0.48	1.13	3.41	15.98
Change in society WTP from independent changes in population	0.26	0.69	1.29	2.92	8.77
Change in society WTP of unborn generations	-1.03	-2.10	-3.23	-5.62	-12.47
= total change	-0.70	-1.14	-1.31	-0.91	3.47

Additional changes in the age structure are also induced by the assumed improvement in aging. This leads to more people alive at older ages and in better health in 2050, so raising the aggregate WTP. The third row of the decomposition shows this effect to be substantial, worth $256.7 billion for the +1 year LE case and $8.8 trillion for +10 years LE. Importantly the size of this channel increases at a faster rate than the gains to LE, e.g., its value for an increase of +10 years is 34 times the size of the +1 year effect. Improvements in LE have a disproportionate impact on the size and age of the older population and so this induced population change increases rapidly in response to improvements in aging. It is this effect that produces the virtuous circle through aggregation. The final reason the aggregate WTP changes between the two sets of improvements is due to changes in the number of future births. Based on current fertility rates and zero immigration, the projection is for a declining number of births. That leads to a lower value of the aggregate WTP for the second wave of improvements.

For small improvements in LE the negative impacts of declining WTP at the individual level and fewer births are larger than the positive effects from changes in population structure. As a consequence, the aggregate value of gains to aging declines. However, for larger increases in LE the induced changes in population rises rapidly and leads to increasing returns to aging at the aggregate level. Closer examination suggests this virtuous circle is likely to extend to the case of even small gains in aging. Two of the four factors driving the change in the WTP are independent of our model (the current projections of an aging society and the birth rate assumptions). Examining the two factors that reflect our analysis (changes in the individual WTP and

induced population changes) reveals their sum to be always positive for any improvement in aging, showing an aggregate virtuous circle for all improvements. Further support for increasing aggregate WTPs would be provided if we used the Census projections of positive net immigration, as this would increase both the population size in 2050 and the number of future births.

The second reason why this virtuous circle exists for even small improvements in aging is connected to whether the WTP for gains to LE are really declining at the individual level. Throughout we have focused on measuring improvements in health, longevity and aging by focusing on step increases in LE or HLE. However, one reason the individual WTPs for Peter Pan decline in response to further improvements in aging is that each +1 increase in LE represents a smaller percentage increase in LE. If instead we focus on percentage improvements in aging (e.g., a 1% slowdown in biological aging rather than a slowdown generating a +1 year increase in LE) then at an individual level we have increasing WTP. In other words, measuring aging biologically rather than chronologically leads to increasing returns to aging at the individual level, which feeds into even larger increasing returns at the aggregate level.

2.7 Discussion

The economic value of gains from targeting aging are large because improvements in aging lead to complementarities between health and longevity, affect a large number of diseases due to the rising prevalence of age related comorbidities, and create synergies arising from competing risks. Crucially, improvements in aging lead to a virtuous circle where slowing aging begets demand for further slowing in aging. This virtuous circle arises because society's gains from improvements in aging rise with the average age of society, increase with the quality of life in old age, and depend on the number of older people. Improvements in aging lead to increases in all of these, such that society has an increasing willingness to pay for further slowdowns in aging. This provides a distinctive dynamic to targeting aging compared to treatments aimed at specific diseases, where gains diminish once successful treatments are discovered.

Our estimates are larger than those in [17], who calculate a slowdown in aging producing a 2.2 year increase in LE as worth $7.1 trillion to those age over 51. This case is closest to our +2 year example in Table 6. Adjusting for differences in chosen discount rates and VSLs, and restricting our gains to just the over 50s, leads to an estimate of the aggregate gains as worth $21 trillion. The remaining differences are attributable to [17] assuming a phased rather than immediate improvement in aging. Whilst differences remain, the most important insight is that their different approach (using an empirical microsimulation model based on U.S. individual data) arrives at similar very large estimates for the value of improvements in aging.

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A Economic Model

At the heart of our model is lifetime expected utility from the perspective of age a, given by

$$\int_a^\infty H(t)u(c(t),l(t))S^*(t,a)e^{-\rho(t-a)}dt$$

where $H(t)$ denotes health at age t, $u(c(t),l(t))$ the utility function (which depends on consumption $c(t)$ and leisure $l(t)$), $S^*(t,a)$ is the survival rate from age a to t, and ρ is the subjective discount rate determining the weight individuals give to the future. As shown in [25], assuming an optimizing agent gives the value of a life year at age t as $v(t) = w(t)(T - l(t)) - c(t) + u(c(t),l(t))/u'_c$ where $w(t)$ is the wage rate, $T - l(t)$ is working hours and u'_c is the marginal utility of consumption, $\partial u(.)/\partial c$.

The value of a life year therefore depends on two items – a term reflecting the value of utility gained that period from consumption and leisure, $u(c(t),l(t))/u'_c$, and a term reflecting savings. Years where savings are positive are given a higher value as they provide financing for consumption at other points in life. An important feature of this model is that the value of life is substantially higher than the value of income earned over a life. That is because leisure itself has a value and the wage at any age provides a way to value this, even if an individual is not working.

Using this approach the value of life at age a is $V(a) = \int_a^\infty v(t)e^{-r(t-a)}S^*(t,a)dt$, where r is the real return the individual earns on their assets. Based on this formula, [25] show that WTP at age a for improvements in longevity in response to changes in medical knowledge (ζ) is

$$\int_a^\infty v(t)S(t,a)\frac{\partial \log S(t,a)}{\partial \zeta}dt$$

whilst the WTP for improvements in health is

$$\int_a^\infty \frac{H'_\zeta(t)}{H(t)}\frac{u(c(t),l(t))}{u_c}S(t,a)dt$$

where $S(t,a) = S^*(t,a)e^{r(a-t)}$, the discounted survival function.

Following [25], we assume utility depends on a composite z of consumption and leisure such that $z = [\phi c^{1-\frac{1}{\eta}} + (1-\phi)l^{1-\frac{1}{\eta}}]^{\frac{\eta}{\eta-1}}$, where η denotes the elasticity of substitution between consumption and leisure, the willingness of the individual to trade off consumption iagainst leisure. The utility function is

$$u(z) = \frac{z^{1-1/\sigma} - z_0^{1-1/\sigma}}{1-1/\sigma}$$

where z_0 (as in [31]) is a normalization capturing an individual's attitude towards life versus non-existence. The parameter σ is the intertemporal elasticity of substitution (IES) that plays a key role in the model as it captures the willingness of the individual to reallocate consumption across time periods. The higher the IES the more an individual is concerned about total life consumption and the lower it is the more they are concerned about per period consumption.

Our model follows a three stage life of childhood/education, work and then retirement. We assume that adulthood begins at age 20 and consumption during this is financed by parents. We assume an initial wage that is constant between 20 and 25 and then starts to rise with age such that $w(a)/w(20) = \gamma \log(a)$ until retirement at $a = R$, with γ reflecting the degree to which wages rise with experience. For $a > R$ we set wages equal to $w(a) = \Psi(a)w(R)$. In the case where $\Psi(a) = 1$ the wage post-retirement is equal to its retirement value and doesn't decline (consistent with [7]). The case of $\Psi(a) < 1$, $a > R$ is consistent with [20] and [21] and we allow for this with the interpretation offered by [7] that the discount reflects a shift to part time work paying a lower salary. The post-retirement wage falls in line with health with elasticity ξ.

B Health and Mortality

We use a Gompertz equation for mortality, where imposing a compensating effect of mortality gives the restricted expression $\mu(a) = Me^{\beta(a-T)}$. We set $T = 97.6$ and $M = 0.3319$ based on the cross-country evidence of [23], and then calibrate $\beta = 0.0966$ so that life expectancy at birth matches that in the U.S for 2018 (78.9 years).

For health we follow [24], [14] and assume at age a that an individual has disabilities given by $D(a) = E + B^{-\mu a}$ and health at age a is $H(a) = [D(0)/D(a)]^{\alpha}$. We impose a compensating effect of morbidity using the restriction $B = D^*e^{-\mu T}$ so that $D(a) = E + D^*e^{\mu(a-T)}$. For calibration purposes we use the results of [1] and [34]: $E = 0.0821$, $B =\exp(-0.504)$, $\alpha = 0.34$. We choose μ to match U.S. healthy life expectancy in 2018 of 68.5 years (World Bank data), where HLE is defined by $\int_0^{\infty} H(t)S^*(t,a)dt$.

For Peter Pan we assume aging is captured by a frailty index $F(a) = \theta e^{\delta a}$ and impose a compensating effect such that $\theta = F^*e^{\delta T}$ so that $F(a) = F^*e^{\delta(a-T)}$ We assume the disability index is given by $D(a) = E+BF(a)^{\psi}$ and mortality by $\mu(a) = M^*F(a)^{\lambda}$, pinning down a relationship between our earlier parameterization and this common factor. For our Peter Pan simulations we vary δ, μ and T in order to simultaneously elongate both the health and survival functions.

For our Wolverine simulations we introduce a repair function $R(x) = I(a)e^{-\delta Z}$ such that $I(a) = 1$ for $a \geq x$ and 0 otherwise. Multiplying our frailty index $F(a)$ by $R(x)$ gives the function $\theta e^{\delta(a-Z)}$ for $a \geq x$ and $\theta e^{\delta a}$ otherwise. Therefore, the effect of the repair is to reset a person's biological clock by Z years.

C Aggregation

The aggregate WTP based on the age distribution of the population in 2017 is

$$\int_0^{\infty} WTP(a)_{2017}N(a,2017)da + WTP(0)_{2017}\int_0^{\infty} B(2017+t)e^{-rt}dt$$

where $WTP(a)_{2017}$ is the WTP at age a for the initial improvement in aging, $N(a,2017)$ is the number of people of age a in 2017 and $B(2017+t)$ is the number of births in the year 2017+t. A similar expression is used to calculate the aggregate WTP in year 2050 for a second improvement in aging.

The difference between the two aggregate WTPs is given by the following decomposition

$$\begin{aligned}
&\int_0^{\infty} WTP(a)_{2050}N^*(a,2050)da + WTP(0)_{2050}\int_0^{\infty} B(2050+t)e^{-rt}dt \\
&- \int_0^{\infty} WTP(a)_{2017}N(a,2017)da + WTP(0)_{2017}\int_0^{\infty} B(2017+t)e^{-rt}dt \\
&= \int_0^{\infty} \left(WTP(a)_{2050} - WTP(a)_{2017})\right) N(a,2017)da \\
&+ \int_0^{\infty} WTP(a)_{2050}\left(N^*(a,2050) - N(a,2050)\right)da \\
&+ \int_0^{\infty} WTP(a)_{2050}\left(N(a,2050) - N(a,2017)\right)da \\
&+ WTP(0)_{2050}\int_0^{\infty} B(2050+t)e^{-rt}dt - WTP(0)_{2017}\int_0^{\infty} B(2017+t)e^{-rt}dt
\end{aligned}$$

where $N^*(a,2050)$ is the number of people of age a in 2050 allowing for the impact of the initial improvement in aging and $N(a,2050)$ is the number of people of age a in 2050 in the baseline projection without the improvement in aging.

D Calibration

Parameter	Value	Source
$1/\sigma$	1.5	[9] Table 3
η	1.509	[9] Table 4
ϕ	0.224	[9] Table 4
$w(20)$	6.98	VSL at birth is \$11.5 million [19]
R	65	Retirement age
γ	1.35	[7] Figure 1
$\Psi(R)$	0.68	[7] Table 1
ξ	1.75	[7] Figure 1
$r = \rho$	0.02	[25]
M	0.3319	[23] Table 2 average across male and female
T	97.6	[23] Table 2 average across male and female
β	0.0966	Life expectancy (LE) at birth is 78.9 years
E	0.0821	[1] Table 3 average across male and female
B	exp(-0.504)	[1] Table 5
α	0.34	[14], [34]
μ	0.0516	Healthy life expectancy (HLE) at birth is 68.5 years
z_0/z	0.1	[25]