Is cancer a disease that can be cured?  
An answer based on a new classification of diseases

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Abstract  Is cancer a disease that can be cured or a degenerative disease which comes predominantly with old age? We give an answer based on a two-dimensional representation of diseases that uses two parameters to characterize age-dependent changes in death rates. These parameters are defined in the following way. In mortality curves (extending from the day after birth to beyond 90 years of age) there is an age, namely $a_c \sim 10$ years, which plays a crucial role in the sense that the mortality decreases in the interval $I_1 = (a < a_c)$ and increases in the interval $I_2 = (a > a_c)$. The respective trends in $I_1$ and $I_2$ are the two parameters used in our classification of diseases. Within the framework of reliability analysis, $I_1$ and $I_2$ would be referred to as the “burn-in” and “wear-out” phases. This leads to define three broad groups of diseases with respect to $a_c$. (AS1) Asymmetry with prevalence of $I_1$; (AS2) Asymmetry with prevalence of $I_2$ and (S) Symmetry, with $I_1$ and $I_2$ both playing roles of comparable importance. Not surprisingly, among AS1-cases one finds all diseases due to congenital malformations and chromosomal abnormalities. In the AS2-class one finds degenerative diseases, e.g. Alzheimer’s disease and other dementias. Among S-cases one finds most diseases due to external pathogens or to a wear-out process. Cancer is one of those mixed cases and in our representation it turns out to be closer to (AS2) than to (AS1).

This representation also provides insight into what we call an overkill effect in old age. This effect tells us that even a highly effective cancer therapy would have no influence whatsoever on the extent of human life unless all other diseases are cured simultaneously.

We conclude that whilst one might develop a cure for many of the known diseases of old age, new diseases will arise and the quest for the ultimate cure will be never ending. Death before the age of 120 seems firmly written in our genetic code and the best we shall be able to do is to help people cope with the inevitability of death as and when it arises.
Introduction

In medical statistics the expression “infant mortality” refers to the first year after birth and during this first year infant death rates (e.g. early, late and post neonatal) are defined with respect to live births and not with respect to the living population at the beginning of the respective time intervals. The 0-1 year definition has its origin in the recording process of postnatal mortality data. However, it is not a logical definition in the sense that the one-year age interval has no real biological basis. A more objective definition of “infant mortality” is suggested by reliability analysis. For technical devices, infant mortality refers to the entire age interval during which the mortality rate decreases. For humans this corresponds to the period 0-10 years. It is this definition of infant mortality which will be used throughout this paper.

For technical devices the successive phases correspond to the well-known stages of the so-called “bathtub” curve.

1. Decreasing failure rate: “burn-in” stage during which defective items fail and are eliminated.
2. Constant failure rate: normal service stage characterized by a small number of random failures.
3. Increasing failure rate: “wear-out” stage during which failures become more frequent.

Fig. 1 suggests that human life comprises similar stages with however the qualification that the constant failure phase covers only a short interval from about 8 to 12 years. For that reason it will be omitted in the subsequent discussion.

**Fig. 1a,b** Death rate from all causes, USA, 1999-2014. In graph (a) the two \( x \) scales are linear whereas the \( y \)-scales are logarithmic. In graph (b) the \( x \) scale of (0,10) is logarithmic, whereas the \( x \) scale of (10,90) is linear. The two straight lines mean that for (0,10) the death rate is hyperbolic of the form \( y \sim 1/x^\gamma \), whereas for (10,90) it is an exponential function in accordance with Gompertz’s law \( y \sim e^{at} \). It can be noted that due to the huge range of variation of the death rate, using a linear scale for \( y \) would produce a completely useless graph in the sense that for (0,10) it would be a vertical line whereas for adult deaths it would be a line superimposed on the \( x \) axis. Thus, a \( y \) log-scale is not just an option, it is a necessity. Source: CDC “Wonder” database 1999-2014 [http://wonder.cdc.gov/ucd-icd10.html]. All data are average annual death rates over the 16-year long interval 1999-2014.
The burn-in and wear-out phases are characterized by two symmetrical processes: elimination of defects in the burn-in phase and accumulation of defects in the wear-out phase.

At first sight such a characterization may seem too crude to be applied to living organisms. But two observations will help us realize that it can capture essential features of living systems.

- Table 1 shows that deaths linked to perinatal problems and congenital malformations dominate death rates in the first month after birth. In subsequent months the share of these two causes decreases. Overall, for the first year they represent 70% of the deaths, 16% in the second year and 6.6% for the $9 - 10^{-}$ age group.

| Days after birth | $0 - 1^{-}$ | $1^{+} - 7^{-}$ | $7^{+} - 28^{-}$ | $28^{+} - 365^{-}$ |
|------------------|-------------|-----------------|------------------|-------------------|
| Complications of pregnancy and delivery (P00-96) | 79% | 65% | 55% | 7.0% |
| Congenital malformations and abnormalities (Q00-99) | 18% | 26% | 25% | 17% |
| Total (P00-96 + Q00-99) in percent of “All causes” | 97% | 91% | 80% | 24% |

Notes: Deaths related to perinatal problems and congenital malformations are completely predominant in the first month after birth. Here $1^{-}$ denotes the end of the first day (i.e. 23h after birth) whereas $1^{+}$ denotes the beginning of the second day (i.e. 25h after birth). The code numbers following the causes of death are from the ICD-10 classification, that is to say the 10th revision of the “International Classification of Diseases”; in most countries it was introduced between 1995 and 2000.

Source: CDC (Centers for Disease Control): “Wonder” database, 1999-2014.

- Many in both the medical community and population at large seem to overlook the fact that in old age hiding behind any disease that is more or less curable, there is another one which probably will be more difficult to treat for the simple reason that it occurs further along the ‘wear-out’ process. For instance, cancer may be followed by Alzheimer’s disease or another degenerative disease. Fig. 2 from Courchesne et al. (2000) shows that the volume of gray matter (i.e. the external layer of the brain) decreases steadily after the age of 10. It is true that the volume of white matter decreases more slowly but because good functionality requires a combination of both white and gray matter, a loss of capabilities seems inevitable. Moreover, the inset shows that instead of being an “orderly” shrinkage, it is a wear and tear process which distorts the shape of the brain. Short of freezing the whole dynamic of aging, it is difficult to imagine how it can be stopped or reversed. Within such a picture, the diseases of aging should be seen as recurrent manifestations of the global aging mechanism.

\[1\] One needs only remember that white matter consists of more than 100,000 kilometers of nerve fibers which connect parts of the gray matter with each other.
Fig. 2 Decrease of the volume of gray matter. The graph describes quantitatively the shrinkage that the inset shows qualitatively. The data are for 116 healthy volunteers whose mean age was 21.4 years. From the data points it is apparent that the sample comprised only few elderly people; thus, there was nobody between 50 and 65 or over 80. Sources: Adapted from Courchesne et al. (2000) and [http://www.health.harvard.edu/brain-imaging](http://www.health.harvard.edu/brain-imaging) (inset).

### Classification of diseases

Before we define the classification, it should be noted that in principle one needs not only death data but also incidence data. By relying on deaths we actually mix up two phenomena which should be studied separately, that is (i) The emergence of a disease and (ii) How well a disease can be treated. Diseases which are 100% curable will not show up in our data and those which are almost completely curable will appear in reduced form. Fortunately two facts contribute to make death data relevant nevertheless.

- For most diseases curability is rather limited for the youngest (< 1 day) and oldest (> 90 year) age groups. This point is related to the so-called rectangularization of the death rate curve (see Meslé and Vallin 2002 and also Berrut et al. 2016, p. 403, 412).

- We are not interested in absolute death rate values but only in relative death levels and the latter are more or less proportional to incidence levels. When a disease can be completely cured it tends to disappear (e.g. smallpox, cholera or tuberculosis).

#### Class AS1: diseases in which burn-in predominates

Fig. 3 shows a typical case in which burn-in plays a predominant role. Some congenital malformations may lead to death in the hours after birth whereas others may bring about death a long time after birth. As a matter of fact, it is quite remarkable that even 50 years after birth the cause of death can be traced back to a congenital malformation.
Fig. 3 Death rate due to burn-in. On the left, between birth and 10 years, the death rate is divided by $10^4$ whereas on the right it is multiplied by a much smaller factor, namely only 7. This shows that there is a very active burn-in process which contrasts with a rather sluggish wear-out process. Source: Same as in Fig. 1

Similar values of the exponent $\gamma$ would be obtained for cases of the same kind. Thus infant mortality due to “Chromosomal abnormalities” (Q90-99) has $\gamma = 1.18$ and “Complications of pregnancy and delivery” (P00-96) has $\gamma = 1.59$.

Class AS2: diseases in which wear-out predominates

Fig. 4 Death rate due to wear-out. The first deaths due to Alzheimer’s disease occurred in the $10^+ - 15^-$ age group; thus the green broken line should be seen as symbolically representing the level zero for younger age groups. Source: Same as in Fig. 1

For the case of Alzheimer’s disease shown in Fig. 4 there is no $I_1$ phase in the sense that for this disease the first death occurs in the $10^+ - 15^-$ age group. A similar shape is observed for similar diseases. Thus for all dementia the contribution of $I_1$ is also very small and in the $I_2$ phase the doubling time is 6.8 years.

Intermediate cases
By “intermediate cases” we mean diseases whose rate decreases substantially in the infant phase and raises significantly in the aging phase.

**Common features of diseases due to pathogens**

‘Bacterial diseases’ such as the one shown in Fig. 5 are typically of this kind.

![Graph showing death rate per million and per year vs. age (year)](image)

**Fig. 5 A case in which there is both burn-in and wear-out.** Based on the ranges of the two graphs one may say that the wear-out process is more considerable than the burn-in process. **Source: Same as in Fig. 1**

This time, in contrast with Fig. 4, the decline in the infant phase cannot be interpreted as being due to the existence of pre-natal defects. How then should one interpret such a graph? A natural interpretation is to say that the decline is due to a strengthening of the immune system. If correct, this interpretation would lead us to say that there is a weakening of the immune system starting right away after the age of 10.

Incidentally, we point out here that the peak which occurs between the age of one month and one year is not a statistical fluctuation for it is found in many countries and various time periods. We might speculate that it arises in a similar manner to that observed for fish as they leave the yolk sac and finally forage independently. Thus here perhaps the peak is related to the fact that between 6 and 12 months, babies shift from the anti-bodies given by the mother to their own immune system.

This interpretation of the dynamics of the immune system can be tested by examining other cases in which one would also expect the immune system to play a role. For instance, in table 2 we examine the case of viral infections,

**Difference in adult profiles between bacterial and viral diseases**

For viral diseases the increase in the adult phase is much slower than for bacterial diseases. In addition, as shown in Table 2, the secondary peak is weaker and occurs earlier.

**Distribution of diseases in the \((\gamma, \alpha)\) plane**

The values of the parameters \(\gamma, \alpha\) estimated previously for various diseases can now
### Table 2  Similarities and differences between diseases due to bacteria or viruses.

| Disease                        | Position of secondary peak [year] | Exponent of infant decline | Doubling time of adult phase [year] |
|-------------------------------|----------------------------------|-----------------------------|-----------------------------------|
| Bacterial diseases (A00-09+A30-49) | 0.53                            | 0.31 ± 0.29                 | 7.6 ± 0.16                        |
| Influenza and pneumonia (J09-18) | 0.53                            | 0.31 ± 0.24                 | 7.3 ± 0.70                        |
| Viral infections (A80-B34)     | 0.05                            | 0.52 ± 0.21                 | 15.4 ± 10.5                       |

Notes: It should be noted that the class J09-18 is dominated by bacterial pneumonia (separate data for viral versus bacterial pneumonia are shown in Fig. 6a). Although it follows broadly the same pattern, the viral case differs from the bacterial case in two respects (i) the secondary peak occurs earlier (ii) the mortality rate levels off after the age of 50 instead of increasing steadily as in Fig. 5. These features remain unexplained so far.  
Source: CDC (Centers for Diseases Control): “Wonder” database, 1999-2014.

be used to position each disease in the $\gamma, \alpha$ plane (Fig. 6).

![Fig. 6 Location of diseases in the ($\gamma, \alpha$) plane](image)

**Fig. 6  Location of diseases in the ($\gamma, \alpha$) plane.** $\gamma$ is the power of the hyperbolic fall $\mu_\gamma \sim 1/t^\gamma$ of the infant phase while $\alpha$ is the exponent of the exponential increase in the adult phase: $\mu \sim e^{\alpha t}$. Along the $\gamma$ axis are all diseases related to congenital defects including those resulting from preterm birth. Along the $\alpha$ axis are all degenerative diseases. In between the remaining diseases may be closer to the first or second group denoting a stronger congenital or degenerative component respectively.

**What is the status of cancer?**
we now discuss the status of cancer in the present framework.
There is a long standing debate about the status of cancer. Is it a disease that is
susceptible of being cured (like tuberculosis, say) or is it a degenerative disease (like Alzheimer’s disease, say)? Can our framework give us some clues?

For heart diseases the wear-out explanation seems fairly natural. Nobody would claim that it is possible to cure completely all heart diseases. If one accepts the idea that the heart is a pump the fact that it is limited to a certain number of beats seems reasonable.\footnote{Cross species comparisons suggest that this number is somewhere between one and two billion beats.}

**Public attitude toward cancer.** For cancer the stated objective of many programs is to find “a cure for cancer”, to “beat cancer” or to “win the war on cancer”\footnote{Just as an illustration, here are three typical sentences.}

- “Those who took part in the overnight relay will not rest until a cure for cancer has been found”.
- “Twelve hours of fun, friendship and fundraising to beat cancer”.
- In his speech after signing the ‘National Cancer Act’ of 1971, President Richard Nixon used the expression “the conquest of cancer”, perhaps in a parallel with the conquest of the Moon.

Thus, the goal is not just to find a cure for specific forms of cancer but to eradicate cancer as a major cause of death, just as tuberculosis was eliminated as a major disease.

**Status of cancer in the present classification.** If cancer is due to a few mechanisms that may be knocked out, a global remedy may indeed be conceivable. However, if it is due to a (programmed) weakening of the immune system that hope does not seem realistic. One faces the same problem for technical devices in the sense that it is not always obvious whether an increasing failure rate is due to one isolated hidden defect or to the simultaneous attrition and wear of many components. In the first case the defect may be eliminated, however in the second case one would need to replace almost every part of the device.

Fig. 7 shows that in the infant phase cancer looks very similar to the case of bacterial diseases of Fig. 5 except that there is no secondary peak. In their adult phases
the two cases look also very similar both in shape and doubling-time. The case of bacterial diseases was (tentatively) explained by successive changes in the strength of the immune system. Surprisingly for a disease such as tuberculosis for which a treatment is available, its adult phase mortality has a doubling time of about 9.1 years which is not very different from the doubling time of cancer. The same observation holds for other bacterial diseases for which antibiotics are supposed to be highly effective. These features are consistent with a conception in which one assumes that any therapy, no matter how effective, can only help the immune system but cannot replace it. Thus, even for a well-treated disease such as tuberculosis, the therapy barely influences the global death rate at ages beyond 100.

**Can the human life span be extended?**

Over the past century the life expectancy at birth in developed countries has been multiplied at least by a factor of two but this was achieved through a fall in infant and middle-age adult mortality. The ultimate boundary of human life span is still around 120 years (for more detail in this respect see Richmond et al. 2016). Thus, a natural question is whether the life span can be extended and what are the conditions for that.

**Keeping the meaning of Gompertz’s law in mind**

The difficulty of the challenge can be assessed through the following well-known (but often forgotten) observation. The number $s(t)$ of radioactive atoms in a sample decreases as an exponential function of their “age”: $s(t) = e^{-\mu t}$. “Beating” an exponential function is already a difficult task; yet, this decrease corresponds to a constant death rate $(1/s)ds/dt = \mu$ whereas Gompertz’s law tells us that in a population of humans the death rate itself increases exponentially with age $\mu(t) \sim e^{\alpha t}$. The graphs presented in the first part of this paper show that the same law also holds at the level of most individual diseases, albeit with different values of $\alpha$. Thus, one needs to “beat a double exponential” which is quite a challenge.

It is true that this fairly standard argument may not sound very compelling because it is purely mathematical; that is probably why it is often overlooked. In what follows we present it in a more concrete way which, at the same time, will reveal the conditions that must be fulfilled to “beat” Gompertz’s law.

First, we introduce a classification of diseases based on how effectively we can treat them.

**Differentiated responses of diseases to therapy**

Diseases respond in different ways to our efforts to cure them. This suggests a dis-
Distinction between the following classes.

1. Diseases which have been eradicated in developed countries, e.g. smallpox, rabies, cholera, leprosy. In terms of age-specific death rates these diseases have a rate close to zero (say below 1 per million) even in old age.

2. Diseases which are more or less curable, e.g. tuberculosis, pneumonia, hepatitis. These diseases are characterized by death rates which are of the order of 1 per million at age 30 but show a Gompertz-like increase pattern which leads to substantial rates (of the order of 1,000 per million) in old age.

3. Diseases which are not yet curable, e.g. cerebrovascular accidents, multiple sclerosis, Alzheimer’s disease. Like those of (2) these diseases have a low rate in young adults but not at all for the same reason. In (2) it is because an effective therapy is available whereas in (3) it is because the wear-out process has only just started.

The diseases in the first two classes are caused by exogenous pathogens whereas those in class (3) are mostly due to endogenous causes. Cancer and heart diseases may lie somewhere between (2) and (3) but closer to (3) than to (2).

Although different in terms of therapy, the classes (2) and (3) can be merged together for the purpose of the forthcoming argument because they both have a substantial rate in old age.

The “overkill” argument

During the Cold War the expression “overkill capability” referred to the fact that both the US and the USSR possessed enough nuclear weapons to destroy one another many times over. Similarly, mortality in old age is ruled by an overkill effect. What we mean by that expression is that even if a complete cure could be found for 99% of the diseases with only one disease remaining unchecked, this single disease would obliterate any sample population, no matter how large, between the ages of 90 and 120. This is due to the fact that the decrease in the number of survivors brought about by any unchecked disease is exponential which implicates that, unless the exponent is very small (which would correspond to diseases that are eradicated), such a decrease will suppress any population within a few years. Just as one atomic bomb may destroy a whole city, similarly a single unchecked disease may reduce a whole population to extinction.

Let us illustrate this argument by the case of cancer.

According to Fig. 8d, presently the death rate from cancer at the age of 90 is \( \mu(90) = 1 \) per 100 and per year. This means that in a sample of 100 people aged 90 the number

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5 It can be remembered that as late as 1892 an outbreak of cholera claimed 8,600 deaths in Hamburg. Throughout the 19th century there had been similar cholera epidemics in London, New York and Paris.
of survivors will decrease as:

\[(1/s)(ds/dt) = \mu \to s(t) = s(90) \exp[-\mu(t - 90)] = 100 \exp[-1 \times (t - 90)]\]

where \(t\) is the age. Thus, one gets:

\[s(100) = 100 \exp(-1 \times 10) = 0.0045, \quad s(120) \simeq 10^{-13}\]

Thus, a single unchecked disease is able to suppress the whole population within a few years.

It can be observed that this is a very conservative calculation in the sense that we assumed that \(\mu(t)\) keeps its \(\mu(90)\) value in subsequent years whereas in fact for almost all diseases \(\mu(t)\) continues to rise after 90 even though possibly at a slower rate than before 90.

In contrast, for an eradicated disease the mortality at 90 is of the order of \(10^{-4}\) per 100. Under such conditions the previous calculation would read:

\[s(100) = 100 \exp(-0.0001 \times 10) = 99.9, \quad s(120) = 99.7, \quad s(200) = 98\]

In this case there is a marked extension of the human life span. However, as stressed above, for this impressive effect to work not a single unchecked disease should remain.

The overkill argument can be checked by observation. Over the past century the eradication of several diseases together with better treatment for a number of others led to a marked increase in the number of centenarians and supercentenarians (i.e. older than 110). Yet, the upper bound of the human life span did not change. In 1997 Jeanne Calment died at the age of 122 years and 164 days, a record which remains unmatched ever since. Moreover she was, so to say, an outlier in the sense that the second oldest person reached an age of only 119 years and 97 days. Even if in the future the record of 122 years is broken most likely it will be surpassed by only a short margin of less than one year.

**How would an effective cancer therapy work in old age?**

In 1900 pneumonia and tuberculosis were the leading causes of death in the United States with annual rates of the order of 2,000 per million. However, by 1950 death rates had decreased by a factor of 10 and further reductions occurred in subsequent

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6 The fact that most of the oldest persons are living in developed countries does not mean that there are no supercentenarians in developing countries. It is rather due to the fact that in the early 20th century in many developing countries birth dates were not recorded in a systematic and reliable way. The same reason explains that there are no reliable data for supercentenarians in the 18th or 19th centuries. Of course, reliable birth data would exist for persons belonging to the aristocracy but such persons represented but a small proportion of the total population.

7 At that time cancer came only in 8th position.
years. Thus, such therapies can be said as having been really effective. What was their effect in old ages? In other words how did this therapy affect the shape of the age-specific curves. Before performing the test we were expecting that, in accordance with Gompertz’s law, the exponential shape would remain unchanged. This is indeed what we observed albeit with a notable increase in the exponent. In short, even such highly effective therapies had a much reduced effect on old age mortality. The same outcome is likely for cancer.

### Tuberculosis

![Tuberculosis](image1)

### Pneumonia

![Pneumonia](image2)

**Fig. 8a,b Pattern of change of death rates over one century in the United States.** For tuberculosis and pneumonia there is a well-defined pattern in the sense that in the adult phase the slope (that is to say the exponent $\alpha$) increases as the global mortality decreases. The fact that for Switzerland almost identical curves are observed suggests that this pattern is shared also by other developed countries. The ICD10 code definition of tuberculosis and pneumonia are A16-19 and J09-18 respectively. Sources: Linder and Grove (1947, p. 248-254); Grove and Hetzel (1968, p. 378-469); “Wonder” database of the “Center for Diseases Control” (CDC).

The case of pneumonia suggests that the curves for different years converge toward a common limit around 110 years. This is another instance of a fixed point mechanism of the kind that we already studied for global mortality (Richmond et al. 2016). Adopting this viewpoint, the increase in slope is an obvious consequence since if the mortality at age 10 falls yet the mortality at age 110 remains stable, the slope of the straight line joining these two points must increase. For tuberculosis the convergence to a fixed point (assuming one exists) is much slower. The fact that the curves for 1900 and 1940 are almost flat suggests that the immune system barely changed in effectiveness over time. This might seem at variance with the suggestion that its effectiveness declines with age. But note that the death rate in 1900 is already very high at age around 20 years and comparable with that in old age for adults today. This then suggests that over time lifestyles, nutrition and medical care have actually improved the effectiveness of the immune system in young adults today such that the death rate on the log scale is reduced by 4 orders of magnitude.

**Noteworthy observation about the status of Gompertz’s law**
In order to prevent any confusion we wish to emphasize two points regarding the role played by Gompertz’s law in the arguments developed in the present paper.

- Our arguments require only what may be called a weak form of the law of Gompertz, basically that the death rate increases with age but with a slope which is allowed to fluctuate with age. For instance, whether or not the slope decreases in old age is irrelevant for our reasoning as can clearly be seen in the subsection about the overkill effect. In fact, this effect will exist (along with the resulting life span wall around age 120) even if the death rate is assumed to be constant beyond the age of 90. It could even be allowed to decrease slightly (although this is never observed) without the conclusion of the argument being changed.

- The global “all causes” death rate is an aggregate variable whose properties depend upon the underlying components. As the underlying diseases do not behave in the same way their addition will produce a mixed bag which will basically be controlled by a few predominant components. We have already observed that the relative importance of diseases has greatly changed over the past century. Therefore one should not be surprised to see the shape of the global death rate change in the course of time. Depending on whether tuberculosis or cancer is the predominant cause of death the shape will not be the same and this will be all the more visible in old age when the the age groups become smaller and the fluctuations therefore larger. There may even be transversal differences from country to country due to differing relative weights of diseases. In other words, from a global perspective, any discussion about the detailed shape of Gompertz’s function is irrelevant and fairly

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8There has been a debate about this point in the past two decades.
useless.

Conclusion

This paper could have been entitled “Is cancer a disease that can be cured? And, besides, does it really matter?”. To the first question we could only give an answer in terms of likelihood. Although we decided against including the second question into the title, it is in fact this second interrogation that we were able to answer in a definite way.

We conclude that whilst one might develop a cure for many of the known diseases of old age, new diseases will arise and the quest for the ultimate cure will be never ending. Death is inevitable and the best we shall be able to do is to help people cope with the inevitability of death as and when it arises.

Naturally, our reasoning would be invalidated if major changes to human DNA are allowed. But that would open uncharted territory which has major ethical implications for society.

Appendix A: Span of life versus life expectancy

In the previous section we focused on maximum life span rather than on life expectancy for the simple reason that the 120 year limit is a certainty whereas life expectancy is a statistical average which gives only a likelihood.

Yet, because life expectancy is a standard demographic indicator it may be of interest to know how many years a successful cancer therapy would add to it.

We will give two answers.

- The first answer is taken from papers by Jay Olshansky and associates (1990, 2001). Their conclusion can be summarized as follows.
  Eliminating all forms of cancer would increase life expectancy at birth by only three years.

  The problem with these papers is that they rely almost purely on statistical projections. This gives readers precise information but leaves them without real understanding. Moreover, these projection techniques are pushed too far sometimes. Just as an illustration, the paper of 2001 gives projections that extend to 2577; one wonders how reliable such long-term projections can be.

- To explain the smallness of the 3 year extension, the key-point is that it is much more effective to reduce infant and childhood mortality than mid- or old-age mortality. This is a simple statistical effect that is illustrated in Table A1.

  As in developed countries infant mortality is currently at a level of 3 per 1,000 (compared to 150 per 1,000 some 150 years ago) one cannot gain much on this side. Thus, further reduction must come from therapies for old age diseases. Not only
### Table A1 How life expectancy is affected by a mortality reduction in different age groups

| Hypothesis                  | Age group 1 | Age group 2 | Age group 3 | Life expectancy | $H_1 - H_0$ |
|-----------------------------|-------------|-------------|-------------|-----------------|-------------|
| $H_0$: Initial death rates  | 30          | 30          | 940         | 79.20           |             |
| $H_1$: Reduced death rate in 1 | 10          | 30          | 960         | 80.80           | 1.60        |
| $H_2$: Reduced death rate in 2 | 30          | 10          | 960         | 79.80           | 0.60        |

Notes: All rates are per 1,000. In order to better show the principle of the calculation the number of age groups was limited to 3. Under hypothesis $H_1$ short lives of less than 5 years are replaced by lives lasting beyond 80 whereas under hypothesis $H_2$ medium lives in the interval 50-55 are similarly replaced by lives of age group 3. Thus, there is no surprise in the fact that the increase in life expectancy (i.e. average duration of life) is higher under $H_1$ than under $H_2$. This effect would be even stronger if group 2 would be an older age group. For instance, the same calculation with 70-75 as age group 2 gives a difference $H_1 - H_2$ equal to 1.40 year instead of 1.00.

This calculation shows that an effective cancer therapy would affect life expectancy at birth much less than the huge infant and early childhood mortality reduction that occurred during the 20th century.

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is this much more difficult to achieve from a medical perspective because these are degenerative diseases, but in addition, even if successful, it will bring about only modest extensions of life expectancy.

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