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Mortality: A physics perspective

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ABSTRACT

One of the many interests of the late Dietrich Stauffer was the modelling of mortality. Here we review the features of mortality data for various biological species. Age specific mortality (death rate) leads to a discussion of possible models of the death rate, including that of Gompertz, raising the interesting question: is our lifetime finite or could we contemplate living for ever? The answer judging from many different data sources is that without radical changes in our biology it seems death above age 120 is extremely unlikely. We then show how a toy model, linking mortality to the immune system, can predict the general variation of the death rate with time, spanning both infant and adult phases. The outcome provides underpinning support for the many nutritionists and medical experts who increasingly advocate the benefits to mortality of a healthy lifestyle. Age specific mortality within social networks is also shown to be significantly affected by both psychological and physical shocks. The review concludes with the description of novel experiments using soap films for the study of failure. These allow for a reproduction of high infant mortality, the so-called bath-tub curve of mortality, and the Gompertz law.

1. Introduction

> John 5:24: *"He who hears my word, and believes him that sent me, has eternal life, and comes not into judgment, but has passed out of death into life".*
>
> *"As death, when we come to consider it closely, is the true goal of our existence, I have formed during the last few years such close relations with this best and truest friend of mankind, that his image is not only no longer terrifying to me, but is indeed very soothing and consoling! And I thank my God for graciously granting me the opportunity … of learning that death is the key which unlocks the door to our true happiness".* Mozart in letter to his father [1]

During the 20th century, attitudes towards death changed from it being seen as a common occurrence, accepted and even romanticized. Western culture began to view death as a fearful, forbidden occurrence. Dylan Thomas reflected this fear when he wrote, "Do not go gentle into that good night, Old age should burn and rave at close of day; Rage, rage, against the dying of the light" [2]. This fear of death was exacerbated by reduced confidence in religious doctrines

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regarding death and an increase in medical science's intervention in the dying process. Scientific advances also increased the secularization of social and intellectual thought, supplanting religious doctrines. This caused many people to lose touch with longstanding religious and cultural beliefs and practices, which has left a void in many people's ability to deal with death.

Science and technology have influenced how many people approach death. The process of dying has been affected by advances in pharmacology, medical practices, and medical treatment facilities. In premodern Western society, dying usually took less time because of primitive medical practices and the absence of the availability of pain reduction drugs. Dying also often took place in the home in the care of family members. In modern Western society, however, many humans have lost touch with death. Advances in medication and medical practices have reduced much of the physical pain associated with dying. This depersonalization of death is evidenced in the care of the dying person often being removed from the family and placed in the hands of health care professionals. Modern medicine has attempted to tame death by prolonging life. Increasingly this prolongation of life is now recognized, at times, to have been at the expense of the quality of life. The US surgeon Gawande [3] has written extensively about this aspect of mortality, advocating what is in essence an approach to patient care and treatment reminiscent of that taken in medieval times, when treatment was person centred, with care and nourishment offered often in a spiritual setting.

2. Dietrich Stauffer's work on mortality – the Penna model

Dietrich Stauffer was a distinguished theoretical physicist with a broad spread of interests which he applied with great effect to problems in the economic and social sciences. He collaborated easily with colleagues and students across the world with whom he explored a wide range of topics: time evolution of languages, demographic change, opinion dynamics, medical resource allocation, finance and taxation, to name a few. One topic which occupied him over a number of years was mortality [4]. It was this topic about which he spoke during a wonderful visit to our group in Trinity College in 2003. Extensive and engaging discussion demonstrated Dietrich's wide interests. In addition, we enjoyed extra-curricular activity on learning that he wanted to listen to 'real' Irish music. A few enjoyable evenings followed which allowed him to combine his love of beer with love of music in the Dublin hostelries.

Much of Stauffer's work on ageing focussed on theoretical innovations based on the Penna model [5]. The approach is based on ideas proposed long ago by Medawar who sought to explain aging through accumulation of inherited bad mutations over many generations [6]. If such a hereditary disease kills a person before sexual maturity, that person has not produced any children, and this mutation will fail and not spread in the population. However, mutations which are able to act later in old age can spread widely. In the Penna model, which is designed with computer simulations in mind, mutations are modelled within a bit string. In this way, a number of natural phenomena, including the Gompertz law, i.e. the observed exponential increase in mortality in later life, can be reproduced.

Martins and Stauffer [7] generalized the standard Penna model, by considering inheritable mutations in a changing environment, such as occurs during climate change, mass immigration, or a severe pandemic. The model showed a maximal population avoiding extinction at an intermediate mutation rate of the individuals. The authors concluded that death is necessary in order to allow the fittest to survive in this changing environment. Specific effects of mixing different populations were explored using the sexual Penna model in [8].

In other work Stauffer et al. discuss the menopause and show how it can emerge from the sexual Penna model without the need for any influence of human culture [9]. It emerges from admitting the need of a mother to care for children, together with increased risk with age of giving birth. This does beg the question as to whether in an age where many women choose not to have children, does menopause occur later in life or could it even not occur at all? Later work using further modifications to the Penna model explored ideas of self-organized death by inherited mutations, inbreeding and outbreeding. Introducing extra bit strings into the Penna idea also allowed him to explore ideas of love and the interplay with monogamy. Many of these manuscripts are readily found on the arXiv preprint server.

As Dietrich Stauffer himself notes, there are many explanations for aging besides mutation accumulation. It's just that many of them are not amenable to computer simulation.

3. Outline of this manuscript

In Section 4 we show examples of mortality data (also called the death rate) for humans, mammals and other biological species and review recent approaches for their interpretations [10–13]. The death rate $\mu(t)$ is the number of deaths (either in general or it due to a specific cause) in a population per unit time. Usually expressed as the number of deaths per 1000 individuals per year, in mathematical terms it is the negative time derivative of the logarithm of the survival probability $S(t)$,

$$\mu(t) = -\frac{d\ln S(t)}{dt} = -\frac{1}{S(t)}\frac{dS(t)}{dt}. \tag{1}$$

Section 5 is dedicated to mathematical models of ageing, leading to the interesting question: is our lifetime finite or could we contemplate living for ever? Gompertz [14] was the first to propose an empirical law for the adult phase. This model certainly does not allow immortality. An interesting development for humans and perhaps mammals was proposed by

Shklovskii [15]. We review this together with a new innovation which allows the idea to be extended to the whole of life, spanning both infancy (wear in phase) and old age (wear out phase).

In Section 6 we present examples how human mortality is affected by changes in the environment and social interactions.

While most ageing models focus on the Gompertz Law, few consider the infant state, where the mortality decreases in a hyperbolic manner, reaching a minimum at the end of teenage years, before the Gompertz region takes over. This is the motivation for Section 7 where we introduce recent experimental work for the lifetime statistics of soap films, which shows high initial failure rates due to well-identified defects. Applied to biological systems, the defects could for example be congenital defects, such as aortic valve stenosis in the human embryo.

4. Age specific death rates

Fig. 1 shows age specific death rates for humans in the USA [16]. The axes in Fig. 1a are log linear and we see immediately after birth there is a sharp rise in the death rate which subsequently, over the first 100 days of life, decreases. This is mainly due to the inability of many foetuses to survive following the shock of emerging from the watery environment of the womb via the birth canal into the atmosphere. A number of foetuses have defects, such as a hole in the heart, cleft palates, aortic valve anomalies, breathing or other difficulties, which make survival extremely difficult, if not impossible. From our perspective as physicists we could say that the birth process acts as a filter which admits into life only a sufficiently 'fit' foetus.

In Fig. 1b both axes are logarithmic: the death rate for infant mortality or the 'wear in' phase is seen to decrease as a power law not just for the first year of life but over almost the first two decades of life! So, the effect of any congenital defect remains for a substantial part of early life. Beyond the age of minimum death rate, during the 20–30 age period, the death rate then rises extremely rapidly.

In spite of this reduction of the death rate over the infant period, the death rate for 1960 in the adult or wear out phase quickly reaches the same level as that in 1923. It is as if the trend is essentially unchanged as end of life approaches.

There would thus seem to be two fixed points for the human death rate; one at the beginning of life and the other at the end of life. Both seemingly are resistant to change. Although at this point, we add a note of caution. Total numbers of deaths in any cohort as we approach the extreme end of life, typically age 95 and beyond, are small, so any statistical analysis towards the end of life is subject to error. We return to this point in Section 5.1.

Berrut et al. [16] also examined the shape of the death rate curve for mammals and zoo animals and, intriguingly, found that it was essentially similar to that for humans, see Fig. 2. Fig. 3 shows more detailed data for the hyperbolic form of the infant death rate for primates. Such a variation, similar to that in humans, has also been confirmed for piglets and lambs. Overcrowding in pens proved to be one important reason which caused 25% of deaths during the first week of life.

Fig. 4 shows that even the common fruit fly has a death rate variation which is similar to that of humans. However, the figure implies that the death rate levels off for advanced ages.

5. Mathematical models for the death rate variation with time

5.1. The Gompertz law

Benjamin Gompertz was born in London in 1779 to a family which had been successful diamond merchants. As a Jew he was in those days forbidden to attend university. He therefore taught himself mathematics through reading the works of Isaac Newton, Colin Maclaurin and other leading mathematicians. He entered the London Stock Exchange, became a member of the Mathematical Society of Spitalfields, and was serving as its President when it merged with the Astronomical Society. In 1819 he was elected a Fellow of the Royal Society and became a member of the council in 1832. In 1824 he was appointed actuary to the Alliance Assurance Company founded by his brother in law, Nathan Mayer Rothschild. In this position he became a government advisor and also assisted the Army Medical Board with his calculations. In 1825, as a result of his work on mortality tables for the Royal Society, he proposed his 'law of human mortality'.

This law asserts that as we age (during the wear out phase) the 'force of mortality' or death rate, $\mu(t)$, increases exponentially:

$$\mu(t) = a \exp(ct), \tag{2}$$

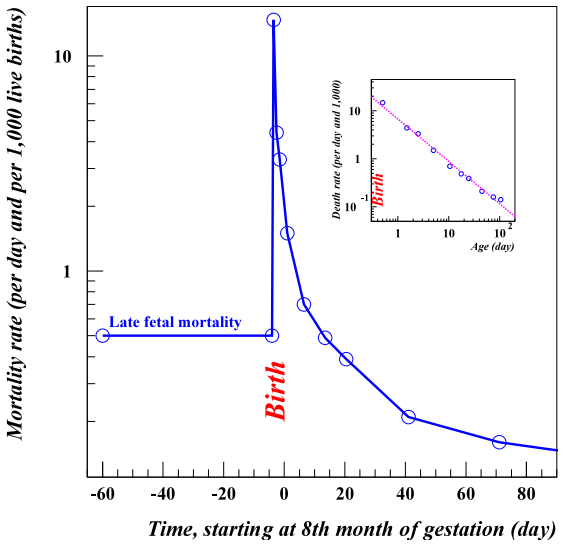
where a and c are positive constants

From Eq. (1), the survival probability S(t) (also called survival or reliability function) of a cohort is given by the integral

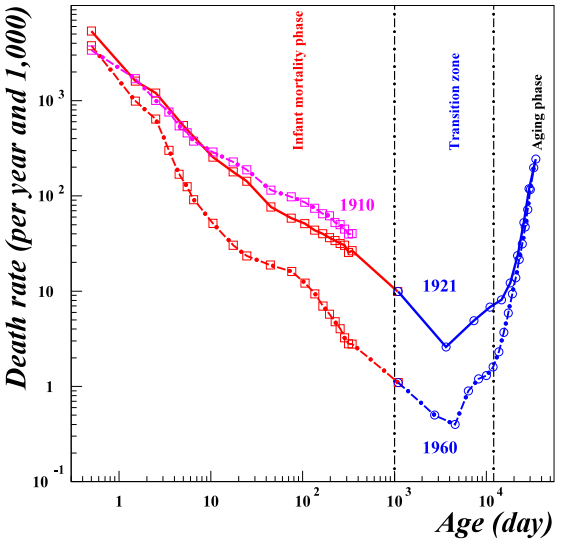
$$S(t) = \exp\left[-\int_0^t \mu(t)\, dt\right] \tag{3}$$

Inserting for the death rate, Eq. (2), yields

$$S(t) = \exp\left[-\frac{a}{c}(\exp(ct) - 1)\right]. \tag{4}$$



(a)



(b)

Fig. 1. Examples of death rate data for the USA [16]. (a) Death rate from 1923, showing transition from gestation to birth, with a clear death rate spike after birth. The level section on the left-hand side schematically indicates the (time-averaged) rate of late foetal mortality. Then, following birth, "defects" which were not of great consequence during gestation, suddenly lead to a dramatic increase of the failure rate. The highest point corresponds to the first day, the second and third points are for days 2 and 3 respectively. The fourth point is the (daily) average for the age interval (3, 7). In the weeks and months following birth, the death rate decreases as a power law. For the inset log–log plot of the same data the coefficient of linear correlation is 0.996 and the slope (i.e. the exponent of the power law) is 0.88. Source: Linder and Grove [17, p. 574–575]. (b) US death rates in log–log plot. There are two phases (infant and aging) separated by a transition. During the infant phase the death rate decreases in a hyperbolic way. In 1910 the slope of the falling regression line (i.e. the exponent of the power law) was $\gamma = 0.65 \pm 0.04$ whereas in 1960 it was $\gamma = 1.01 \pm 0.08$. This decrease has continued in more recent years. Source: 1910: Mortality Statistics of 1910, Bulletin 109 [18]; 1921: Mortality Statistics (1921); 1960: Grove and Hetzel [19, p. 210–211] and Wang et al. [20]. Note: it has been pointed out, for example by Wang et al. [20], that records of deaths can sometimes be erroneous due to the cause of death been incorrectly diagnosed. This is important when considering deaths conditional on a particular disease. However in this paper we only consider total deaths from all diseases.

The survival function $S(t)$ thus decreases as a double exponential. This is much faster than the decay of nuclear material for which the force of mortality or death rate, $\mu(t)$, remains constant with time.

The evidence for the validity of the law of Gompertz in so far as it applies to humans up to ages around 95 is overwhelming. Fig. 5 (left) shows data published by Richmond and Roehner [10] for a group of countries where the data is reliable and spans ages up to 95. Fig. 5 (right) shows data for a second group of countries where the data is either

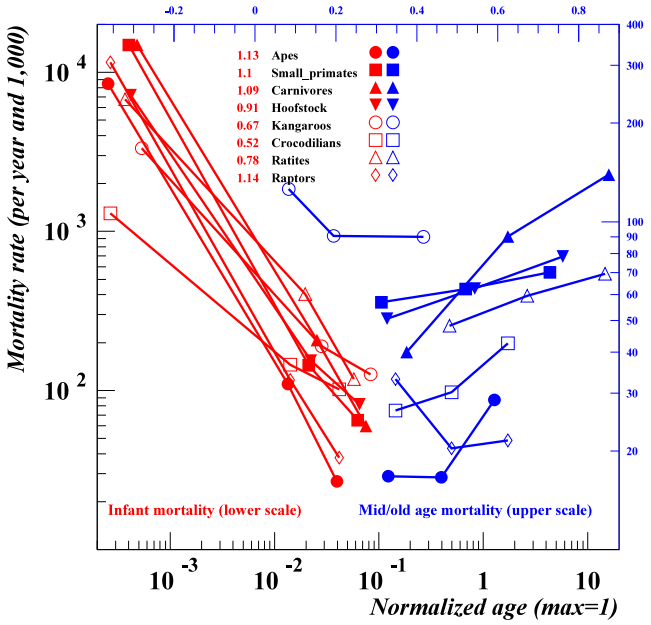


Fig. 2. Infant vs. mid age mortality rates for various animal species. The graph shows that across species infant death rates are more orderly than later age death rates. Note that the scales for later age death rates are on the right-hand side and top horizontal axes. The numbers preceding the names of the sub-groups are exponents of the power law for infant death rates. Ages have been normalized by dividing by the maximum age of a species, so as to lie between zero and one. Even with only three data points, thanks to the good correlations, the error bars at a probability level of 0.95 are only about ±8% on average. (For additional details see [16].)
Source: Kohler et al. [21].

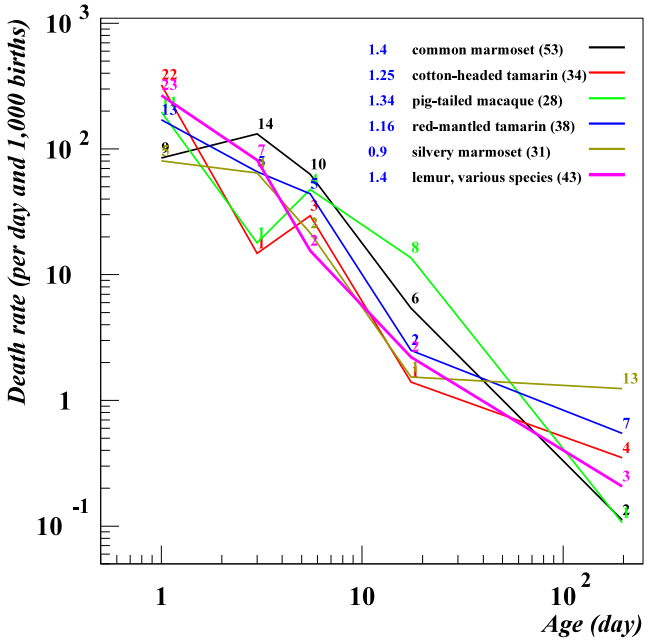


Fig. 3. Infant mortality of primates showing the same hyperbolic form as that for humans. The numbers which follow the names give the size of the subgroup of individuals whose birth and death were recorded in London Zoo during the period 1970–2000; numbers on the curves give deaths in each age interval. Finally, the numbers preceding the names in the table are the power law exponents characterizing the time decay of the curve.
Source: Archives of the Zoological Society of London [16].

incomplete or less reliable and spans a lesser age interval. In Fig. 5 (left) the convergence of data towards an apparent fixed point is clearly seen. For the data in Fig. 5 (right) this convergence is, not surprisingly, less evident.

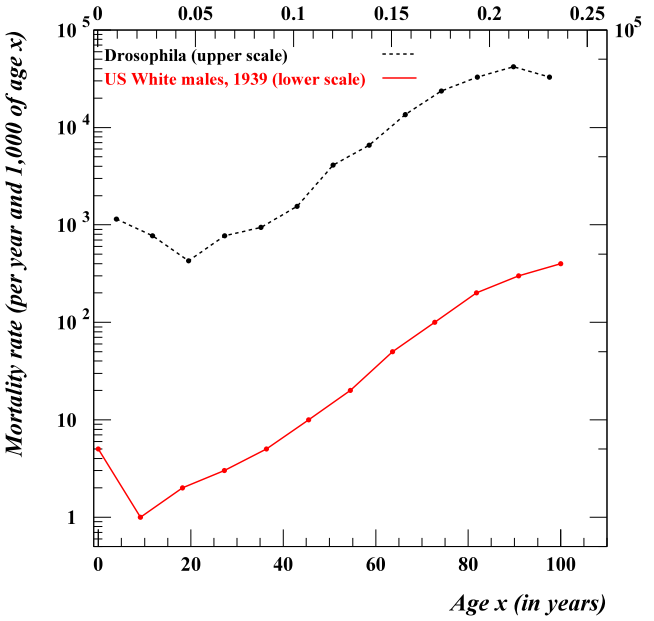


Fig. 4. Age specific mortality rates for humans and drosophila. The Gompertz law holds in both cases. The levelling off for humans should be attributed to statistical fluctuations, for the drosophila data the reason for this is not entirely clear [10].

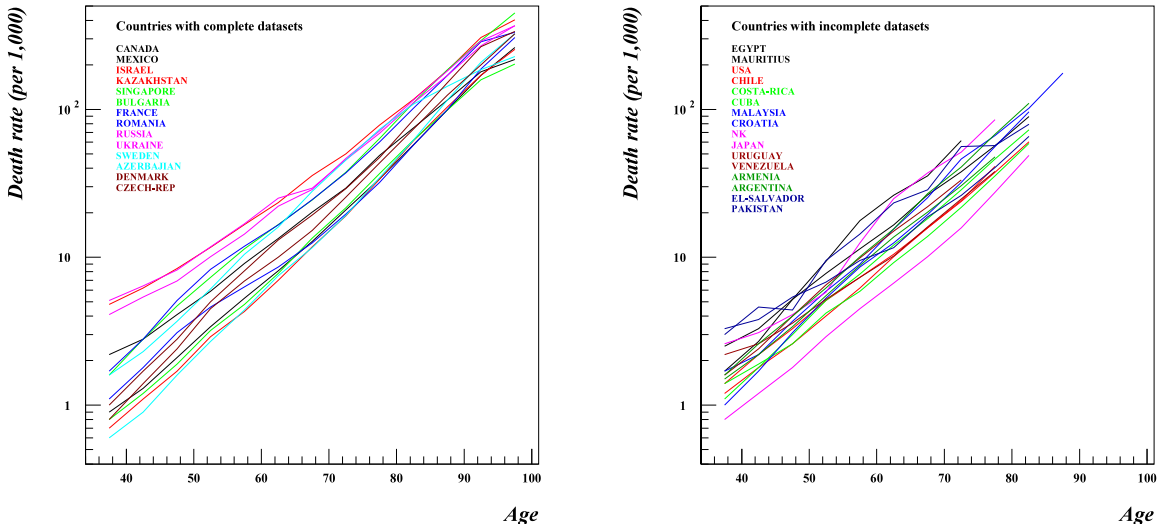


Fig. 5. Demonstration of the Gompertz law for two sets of countries. (Left) The death rate data includes the age group 95–99. All data appears to converge towards an end point of (∼120,1000). (Right) This data obtained for different countries is limited to ages below 85 years.
Source: Figures from Richmond and Roehner [10], data source United Nations Demographic Yearbook, 2011.

Deuteronomy 34:7 says: "Moses was 120 when he died; his eye was not dim, and his natural force abated" and the Jewish people often wish others the toast ביז הונדערט און צוואנציק *or* 'may you live till 120'. What do our graphs say about the value of the fixed point towards which the death rate curves approach?

Fig. 6 shows, for clarity, only three death rate curves, all belonging to France, but spanning a time period of 200 years. Extrapolating each to higher ages determines the fixed point within the red circle centred at approximately 120 years [10]. According to the data, the death rate rises above 10^3 just below this value, so the maximum age limit lies a few years below this value and has increased slightly over the past 200 years from around 110 to 115 years. Without radical change in our biology or environmental conditions it does seem that substantial change is unlikely. The issue however remains an active issue for research [20,22–27].

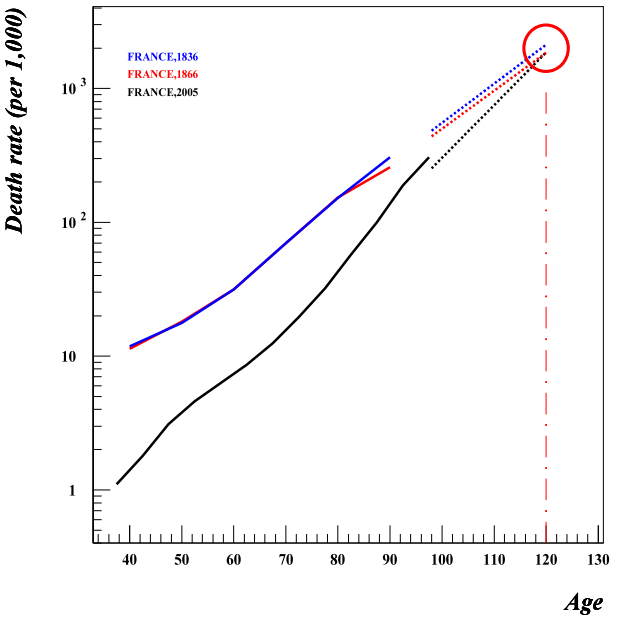


Fig. 6. Gompertz' law in France. The data beyond age 95 are regression lines which appear to converge at age 120. Although we note by this age the probability of death is greater than unity! From the probability axis we might deduce that the maximum life over the period has increased from around 110 to 115. For details see [10].
Source: (2005) and United Nations Demography Yearbook, 2011.

5.2. Toy models linking mortality to the immune system

As we noted in the introduction, a number of different approaches exist for the modelling of Gompertz' law. Here we comment on the approach of Shklovskii [15], as it naturally lends itself to an extension spanning infant mortality [28].

Shklovskii treats the immune system very simplistically akin to a big bag in which active ("good") molecules drift around randomly, in the manner of gas molecules in a balloon. There are numerous types of active molecules, but in the most basic form of the model one assumes only a single type. The immune system defends itself against invaders ("bad cells") which, if they multiply, can cause disease.

Assuming that random encounters between the immune cells and the bad cells are governed by Poisson statistics, the probability of a number M of encounters per time interval δt is given by

$$P(M) = {N_0}^M exp(-N_0)/M! \tag{5}$$

where N_0 is the average number of encounters (a quantity proportional to the number density of immune cells). To survive and grow the bad cell must avoid any encounter ($M = 0$). The probability of growth and death in the time interval t to $t + \delta t$ is:

$$P\,(0) = \mu = \exp(-N_0) \tag{6}$$

For this model to have validity in our context, N_0 should be greater than 1. This can be checked when we note that for humans the age at which mortality begins to be governed by the Gompertz law is about ~25 (see Fig. 4), where μ is $\sim 3.10^4$ which yields N ~ 8.

Now suppose the immune system decays with time. The simplest assumption for ages greater than 25 is $N\,(t) \sim N_{25} - ct$ where c is a constant and N_{25} is the value of N at age 25. Gompertz' law emerges immediately:

$$P\,(t) = \mu(t) = exp(ct - N_{25}) \tag{7}$$

The other interesting point made by Schklovskii at this stage is that other outcomes are possible if one admits other decay laws for the immune system. For example, an eminently reasonable choice might be:

$$N\,(t) \sim N_0 \exp(-ct) \tag{8}$$

which reduces to the simple form above for $N(t)$ on expanding the exponential and neglecting all terms higher than those linear in t. The exponential decay of $N(t)$ admits deviations from Gompertz at higher ages; thus, if c is positive, the death rate curve falls below the linear Gompertz rate, leading to higher life spans.

One sometimes reads of people whose age is greater than 120, but more often than not the date of birth is of questionable validity. Moreover, as we have already remarked, the numbers of centenarians is small and statistical fluctuations dominate any estimation of deviations from Gompertz. Perhaps more experiments with the humble fruit fly mentioned above (Fig. 4) might offer a route to test this concept in more detail. Also experiments with soap films, as described in Section 7, might also offer the possibility to test for deviations.

Is it possible to complete the picture and link the entire death rate curve to the immune function model, spanning both infant and adult regimes? The answer seems to be affirmative. Let us suppose with Shklovskii that the number of molecules in our toy immune system peaks at $t = 25$ when $N = N_{25}$. That being the case, we might expect prior to that time the number density is of the form

$$N(t) = N(25) - \delta|t - t(25)|^{\gamma} \text{ where } \gamma > 0 \tag{9}$$

Substituting this expression into Eq. (6) yields for times $t < 25$

$$\ln[\mu(t)] = \delta|t - t(25)|^{\gamma} + \ln[\mu(25)] \tag{10}$$

The death rate decreases as time approaches 25, as it should.

But within this model what growth rate corresponds to the apparent power law decay of the deathrate in the infant regime? The reader can easily check that for $t < 25$, a growth law

$$N(t) = \alpha ln[t] + N(0) \tag{11}$$

where α is a constant, satisfies the requirement. Differentiating gives a growth rate

$$dN/dt = \alpha/t. \tag{12}$$

This suggests that the immune system responds very strongly immediately after birth and then the growth rate slows during infancy, up to late teenage years. At this time the immune system begins to decline, leading to the 'wear out' regime governed by the Gompertz law. The second point to observe is that the decay in time for infant mortality extends over all populations and time eras. The reduction in infant mortality achieved by medical advances is effectively mediated through the prefactor α.

But what evidence do we have for this result? Without any data for the strength of the immune system we can only speculate at this stage. If more detail about the strength of the immune system becomes available we can anticipate models based around a universal law covering both the infant and adult phases will be possible. The idea behind this model is interesting since nutritionists and other health professionals already believe that it is important to maintain a strong immune system for good health. To the best of our knowledge no one has ever justified these statements even via a heuristic link between the immune system and the death rate or mortality such as that presented here. Outstanding questions remain, for example: can we quantify the strength of an immune system over time? Do deviations from Gompertz at higher ages really exist? Hopefully these ideas will encourage more studies in these directions.

6. Other effects on mortality

6.1. The role of environmental and social interactions

Thus far we have considered mortality of individuals and links to internal physiology. But are there other interactions between humans or the outside environment which also affect health and mortality? Richmond and Roehner [11] conjectured that any abrupt change in living conditions generates a mortality spike which acts as a kind of selection process. They went further to assert it applies to all living and inanimate complex objects.

Obvious examples of the impact of environmental interactions are humans hit by moving vehicles, or shootings which can lead to immediate death. An example of catastrophic failure of a complex inanimate object is that experienced by space shuttle Challenger which exploded just after take-off due to failure of an O-ring joint in one of the solid rocket boosters.

Are there examples affecting humans where the change is seemingly routine, yet where a spike in mortality is clear? Answers are to be found in the three cases shown in Fig. 7 [11]. Fig. 7(a) shows death rates following admission of elderly people to nursing homes. Done for what may be the best of reasons, the mortality spike for such persons in Paris is clear. Fig. 7(b) shows deaths of both males and females (aged 72) shortly after admission to state mental hospitals in Maryland, USA. Finally, 7(c) shows suicides of prison inmates following arrest in the US during the 1980s. Again, the peak in mortality is clearly visible.

An interesting illustration occurs with fish. When fish emerge from the egg they survive for a while by eating the yolk sac. Once this has been consumed, they must survive by foraging for themselves. But not all fish have the skill or strength to do this successfully and this can be seen in the survival statistics. Small fish, such as the Californian anchovy, can consume their yolk in two days and around 4 to 7 days later one sees a spike in the mortality. The phenomenon has been confirmed for sardines and larger fish such as Black sea turbot and the Siberian sturgeon. Very large fish, such as salmon take up to 60 days to eat the contents of the yolk sac. Based on this hypothesis, Berrut et al. [16] predicted that there should be a mortality spike around a week later. Investigations made in a Norwegian fish farm later confirmed this to be the case.

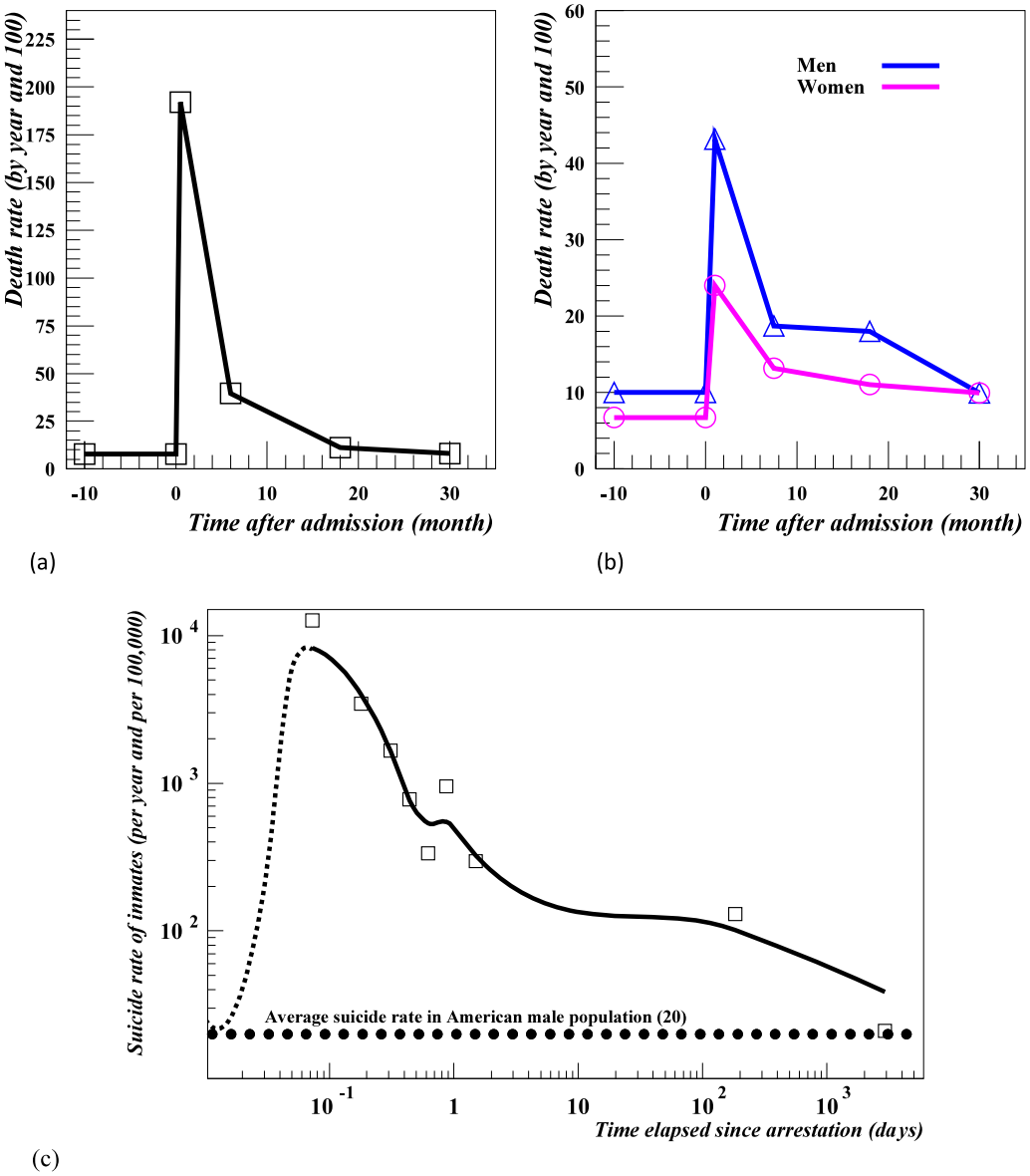


Fig. 7. Effect of changes of the environment to the mortality rates. (a) Deaths of elderly people following admission to nursing homes in Paris. (b) Deaths of both male and female inmates (aged 72) admitted to state mental hospitals in Maryland USA. (c) Suicides of prison inmates following arrest in the USA during the 1980s. (For details see [11].) Note the abscissa scale is in months for figures a and b but in days for figure c.

6.2. The impact of social interactions on mortality

William Farr in England and Louis Bertillon together with his son Jacques in France, using data available in the 19th century concluded that for all age groups, the death rate of married people is less that the death rate of non-married be they single widowed or divorced [29,30]. However, this conclusion was questioned by many researchers over the years, mainly because of doubts concerning the reliability of the data then available. Using more recent data Richmond and Roehner [12] performed similar calculations. The conclusions remained valid and held for all age groups and all diseases. The death-rate by heart attack was shown to be more than twice as high for non-married persons. Moreover, for young widowers the death-rate within a year or so of the loss of a spouse was up to 20 times higher than the background rate. This was validated not just for European countries, such as France or the US, but also for China (see Fig. 8). China is interesting for three reasons. It is an important non-western country with different cultural and family links. The country has a large population so we can expect more accurate results than in the case of countries with smaller populations;

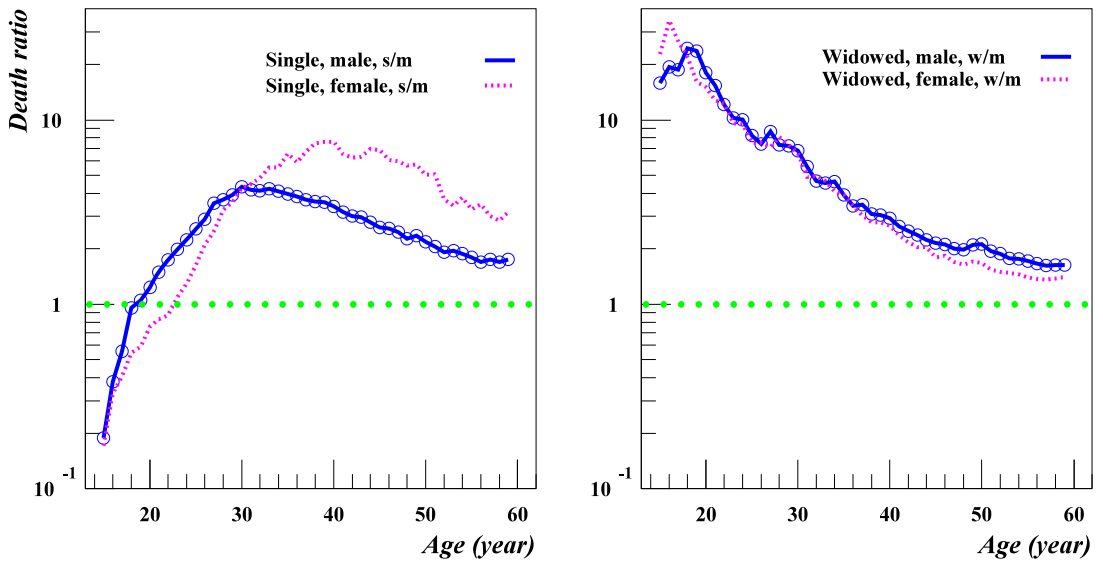


Fig. 8. The Farr Bertillon effect in China. Defining the death ratio as the deathrate for persons relative to the overall deathrate for such persons, the graph showing death ratio of persons relative to the background level, based on data for 1990. The data for male and females are closer than for Western countries and the smoothness suggests fluctuations are small.
Source: Primary sources were used from http://bbs.pinggu.org/thred-1530030-1-1.html.

instead of using 5- or 10-year age groups, we can use 1-year age groups. Several provinces (e.g. Fujian, Jiangsi, Sichuan, Xinjiang) have a tradition of early marriage which also helps magnify the young widower effect.

In a development of this approach, Richmond and Roehner [13] used disability data as a proxy for mortality shown in Fig. 9. In this way the authors were able to calculate the relative strength of the bonds between an individual and both their spouse and children as a function of age. They showed for example that age specific disabilities parallel the strength of family ties. The ties between a husband and wife were weakened by a large age gap between the couple and the ties between parents and children were strongest when the child was under five years old. In this way it is possible to begin to quantify interactions between social beings just as a natural scientist might quantify interaction energies and forces between molecules.

7. A new approach for studying failure statistics using soap films

Our discussion so far has centered on data for biological systems. The understanding of mortality curves is of relevance also to man-made products, where the term mortality rate is replaced by failure rate. While vast amounts of data undoubtedly exist, these are generally not available to consumers, but kept by the producers, possibly to avoid litigation. A notable exception is the data bank for the lifetime of computer hard-drives which is maintained by the cloud-storage provider Backblaze.[1]

Having access to a simple physical system as a source of failure data would allow for a set of controlled experiments to accompany and probe theories of failure. Since such experiments are by definition of a statistical nature, the datasets need to be sufficiently large; failure times should not be too long.

Haffner et al. recently suggested that soap films, produced under controlled conditions and confined in cylindrical tube, may serve as such a system [32]. Here we use the word soap film to describe a thin aqueous film (thickness less than 1 to 100 μm), stabilized by surfactant molecules, which assemble mainly along the two liquid-air interfaces [33]. The longevity of such liquid films depends crucially on their (local) thickness. This generally reduces with time, as the films drain due to capillary forces and gravity, with a further loss of liquid due to evaporation.

The end state of these processes is what is called a black film, with a thickness of 30 nm, or even 5 nm (Newton Black film). (The films appear black in reflection due to destructive interference of light [34]). It is not guaranteed though, that an individual film reaches this state, as it is prone to rupture beforehand, ultimately due to statistical (thermal) fluctuations [35]. Nevertheless, average lifetimes can be controlled, for example via liquid viscosity which slows down drainage, the humidity of the environment (control of evaporation) [36,37], or a dust-free environment.

Having had access to lifetime data for over 2500 soap films from previous experiments [38], and conducting additional experiments, Haffner et al. showed that the failure rate of ensembles of such films are best described by the Gompertz law, i.e. an exponential increase of the failure rate with time [32].

[1] https://www.backblaze.com/blog/how-long-do-disk-drives-last/.

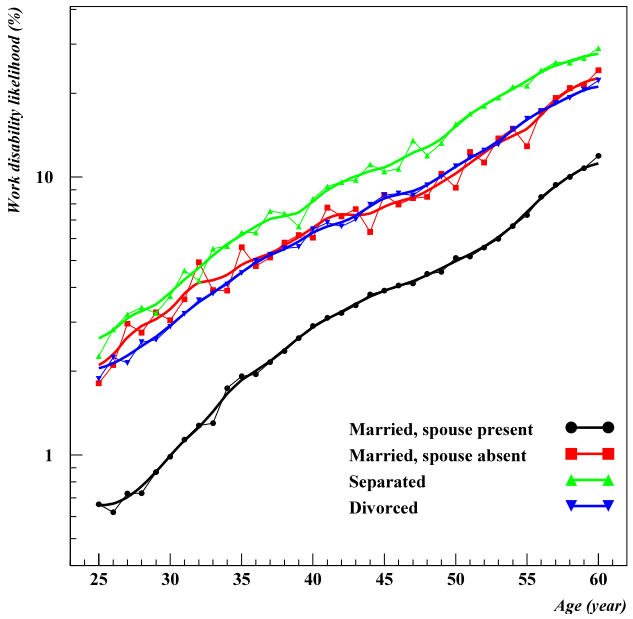


Fig. 9. Likelihood of disability in various marital situations for males and females in 1990. The curves fall into two classes: married with partner present, married with partner absent, separated and divorced. Clearly it is the real presence of a partner which matters rather than the social status of being married.
Source: 5% sample of the US census of 1990 downloaded from the IPUMS database (Ruggles et al. [31]).

Follow-up experiments by Bois et al. [39] focused on understanding the burn-in phase (or infant mortality). Lifetimes were determined for individual soap films, contained in fully sealed and vertically aligned tubes, and also in tubes which were sealed only at the bottom end. Exposure to increased evaporation reduced the average lifetime in the second data set down to about a fifth of the average lifetime of films contained in the fully sealed tubes.

In Fig. 10a we show as a function of time the fractions of films contained in the 144 tubes with two stoppers, and in the 144 tubes with only one stopper, respectively. After slightly over three minutes the film population in the one-stopper tubes has vanished entirely. Also shown is the survival or reliability function $S(t)$ of the combined data set, which steadily decreases from its initial value of 1. After about 15 min only 10% of the initial 288 films remain intact, all of which are in the two-stopper tubes.

From the survival function $S(t)$ we can compute the corresponding failure rate, $\mu(t)$, using Eq. (1). Fig. 10b shows that following a steep initial rise due to the rupture of films in the one-stopper tubes, $\mu(t)$ decays again in the time interval from 2 to 5 min, as this short-living film population gradually vanishes. This behavior is reminiscent of the death rate spike after birth shown in Fig. 1a (high burn-in rate or infant mortality). From about 5 min onwards, $\mu(t)$ begins to rise again, as now rupture also becomes prevalent in the films contained in the two-stopper tubes. This functional variation of $S(t)$ is what is called the bath-tub curve of failure, a curve shape we have already encountered in Figs. 1b and 4.

Bois et al. argue that the role of defects (in the case shown in Fig. 10, referring to tubes with only one stopper) for high initial failure rates is also seen in human mortality data; this is exemplified by death-rate for humans due to congenital aortic valve stenosis, i.e. aortic valve narrowing [39].

In the same paper Bois et al. also demonstrate how using mortality data it is possible to deduce the spectrum of defects within an assembly of similar artefacts, but prepared under different environmental conditions. In an extensive suite of experiments Bois et al. find that the average lifetime of a soap film increases roughly linearly with the length of the tube in which it is confined (a reduced humidity gradient along the tube leads to reduced evaporation). Given lifetime data for films contained in tubes of different lengths, it is thus possible to extract information about the underlying distribution of tube-lengths [39].

8. Conclusions

Mortality is of importance to everyone, especially as they get older! Empirical studies across many countries and time eras reveal regularities which can be captured via mathematical laws, and intuitive physical models, which give new insights, are possible. The data together suggests our life on this earth will be of finite span with $\sim$120 years or so being the maximum age for the majority of us. Are the few who claim to live beyond this age statistical fluctuations, using inaccurate birth data or genuine instances of deviations from the law of Gompertz? Without much more accurate data for centenarians it is, at present, impossible to say. However, simple physical models linking mortality to the immune

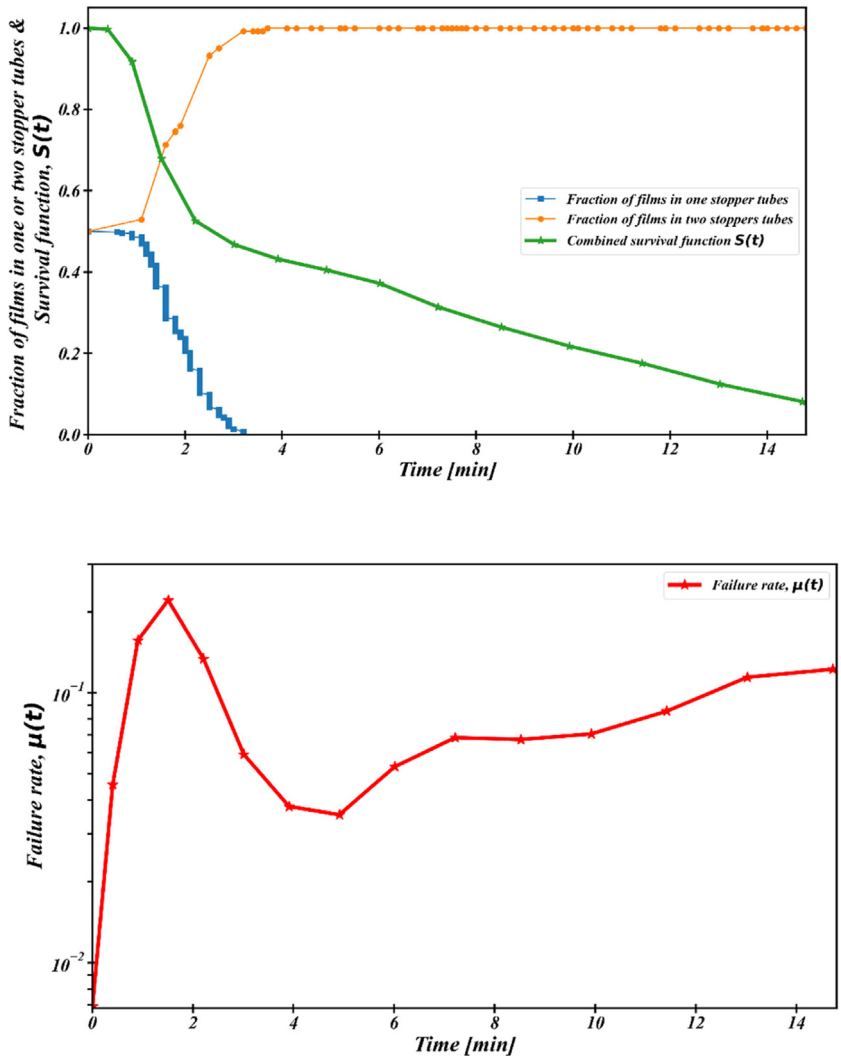


Fig. 10. Experimental data for a total of 288 soap films contained in glass tubes (length 3 cm, diameter 1.6 cm), 144 of which were sealed with one, and 144 sealed with two stoppers. Data is shown prior to when only 10% of the initial number of films are left in order to avoid fluctuations in the computation of the failure rate, $\mu(t)$, due to the small number of films remaining. (a) The films in the one-stopper tubes all rupture within about three minutes; as their fraction of the total number of films drops to zero, the fraction of films contained in the two-stopper tubes rises to one. Also shown is the survival function, $S(t)$, of the combined data set. Over the 15 min shown it decays from 1 to about 0.1. (b) Variation of the corresponding failure rate $\mu(t)$, computed using Eq. (1). At about 2.5 min $\mu(t)$, starts to decrease, as the rupturing of the films in the one-stopper tubes slows down. The failure rate rises again after about 5 min, due to the increased occurrence of film rupture in the two-stopper tube.

system do not exclude the option of deviations from Gompertz' law. Of immediate relevance is that these models linked to the immune system offer support to those health professionals who increasingly advocate healthy life-styles for all. Even the infirm, we now know, can improve their mortality via changes to diet and their exercise regime. Old wives saying 'if you stop moving (externally and internally via the intestine) you seize up' really do seem to have an element of validity!

Environmental shocks might be expected to affect the mortality, and data presented here for elderly and prisoners illustrates this clearly. Much less is known about the impact of atmospheric pollution, novel medication and the recent craze for vaping. Meaningful data in these areas can take considerable time to collect, as is clear from the studies of the effect of smoking on health which took many decades of painstaking effort. Drug use is an increasing problem and it will be interesting to monitor carefully over time the impact on mental health of marijuana consumption in those US states and other countries around the world where it is now legalized. Government finances may become healthy from taxation but whether the citizens will be equally healthy is an open question at the present time.

We hope our review of empirical data, mathematical models, and a novel approach using soap films for mortality studies stimulates more interest from the physics community. This could also offer insights that add value to the religious, political and medical professionals such as Gawande [3], who are thinking more deeply about quality of life for humans.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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