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Deciphering infant mortality

Sylvie Berrut [a], Violette Pouillard [b,c], Peter Richmond [d], Bertrand M. Roehner [e,*]

[a] Swiss Federal Office of Statistics, Neuchâtel, Switzerland
[b] Université Libre de Bruxelles (ULB), Brussels, Belgium
[c] University of Oxford (Wiener Anspach Foundation Postdoctoral Fellowship), United Kingdom
[d] School of Physics, Trinity College Dublin, Ireland
[e] Institute for Theoretical and High Energy Physics (LPTHE), University Pierre and Marie Curie, Paris, France

HIGHLIGHTS

- Infant mortality is considered in a systems science perspective.
- The "Transient Shock" conjecture predicts death rate spikes.
- The predictions are confirmed by a whole range of observations.

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ABSTRACT

This paper is about infant mortality. In line with reliability theory, "infant" refers to the time interval following birth during which the mortality (or failure) rate decreases. This definition provides a systems science perspective in which birth constitutes a sudden transition falling within the field of application of the *Transient Shock* (TS) conjecture put forward in Richmond and Roehner (2016c). This conjecture provides predictions about the timing and shape of the death rate peak. It says that there will be a death rate spike whenever external conditions change abruptly and drastically and also predicts that after a steep rise there will be a much longer hyperbolic relaxation process.

These predictions can be tested by considering living organisms for which the transient shock occurs several days after birth. Thus, for fish there are three stages: egg, yolk-sac and young adult phases. The TS conjecture predicts a mortality spike at the end of the yolk-sac phase and this timing is indeed confirmed by observation.

Secondly, the hyperbolic nature of the relaxation process can be tested using very accurate Swiss statistics for postnatal death rates spanning the period from one hour immediately after birth through to age 10 years. It turns out that since the 19th century despite a significant and large reduction in infant mortality, the shape of the age-specific death rate has remained basically unchanged. Moreover the hyperbolic pattern observed for humans is also found for small primates as recorded in the archives of zoological gardens.

Our overall objective is to identify a series of cases which start from simple systems and move step by step to more complex organisms. The cases discussed here we believe represent initial landmarks in this quest.

* Corresponding author.
E-mail addresses: Sylvie.Berrut@bfs.admin.ch (S. Berrut), Violette.Pouillard@ulb.ac.be (V. Pouillard), peter_richmond@ymail.com (P. Richmond), roehner@lpthe.jussieu.fr (B.M. Roehner).

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1. Introduction

Whereas the process of aging has been and still is much studied, the rapid fall of the infant death rate in the hours, days, weeks and months after birth has received relatively little attention.

1.1. Origin of the questioning

The present study originated from the conjunction of two separate (and, at first sight, fairly disconnected) observations.

- The first observation is the rapid (hyperbolic shaped as will be seen later) fall of the postnatal death rate. It is usual in medicine to consider infant mortality up to 12 months or one year after birth. However, we shall see that for humans, the hyperbolic decrease of the death rate extends over a period 10 times longer. Then, after the age of 10, the decrease stops and is replaced by a phase that increases through to end of life (see Fig. 1b). This naturally leads to the idea that there are two different regimes: the postnatal regime and the senescence regime. Whereas the senescence regime is fairly well understood, the postnatal regime appears much more mysterious. Why is the fall of the death rate hyperbolic? Why is the duration for humans approximately equal to 10 years?
- From plants to fish, to birds, to mammals there is a bewildering diversity of living organisms. Yet, if one leaves aside the arthropods,[1] there is a deep similarity in the mechanism through which a new organism starts its life. It begins as a tiny one-cell embryo which divides and grows. In the case of a plant the energy required for the growth process is generated from the food reserve contained in the grain (or in the egg in the case of fish and birds) and the oxygen which diffuses through the grain's (or egg's) envelope. For a mammal the nourishment and the oxygen come through the umbilical cord. After germination, hatching or birth the new organism must become autonomous in the sense of relying only on the resources (oxygen, carbon dioxide, light, food) available in its environment.

  The similarity of these mechanisms naturally leads to the question of whether or not postnatal death rates follow a general pattern.

There is a common saying according to which "science starts with the discovery of a pattern". In the present paper, we identify two patterns of infant mortality, namely the yolk-sac death spike pattern and the hyperbolic decay pattern. The first pattern is fairly well understood, however the second raises questions for which we have no complete answer so far. We believe that, taken together, these patterns give us a new insight into the mechanisms of infant mortality.

1.2. Previous studies

There are fewer studies about infant mortality than aging and senescence.[2]

But we note a connection with a series of papers by Canadian researchers [1–4]. These develop the notion of *frailty* of individuals and its relationship with the likelihood of death. After defining a frailty index made up of several dozen "deficit" indicators ranging from fairly light deficits such as hearing problems to more severe deficits such as cancer or heart disease, they demonstrate that it has a strong correlation (over 0.98) with the death rate.

Two phenomena are analyzed in the present paper: (a) the yolk sac effect for fish and (b) the preterm effect in humans for which the notion of frailty provides a clue. Termination of the yolk sac phase requires fish to switch from one form of feeding to another. This elevates their frailty level with the result that changes in the environment which otherwise would have gone unnoticed bring about a death spike. Similarly, preterm babies have an excess degree of frailty which, as we will see, results in a huge amplification of the postnatal death spike.

In their studies the Canadian researchers restrict their attention to the frailty which comes with aging. The two previous cases illustrate situations of *temporary* frailty, a useful extension of the concept. Incidentally, the notion of frailty can also be extended to the social sciences as was masterfully illustrated by another Canadian researcher, Naomi Klein [5], in a book entitled "The shock doctrine". This is a generalization in the sense that drastic socio-political changes which would not be accepted by a nation in normal times may be imposed and tolerated in times of a natural disaster or social crisis.

1.3. Previous studies in a "physics perspective"

We know of no study of infant mortality that relies on a *physics perspective*. By this we mean simply a comparative cross-species approach in which one investigates a specific phenomenon across a number of different species and where the

[1] Because they have a rigid exoskeleton their development involves widely different instar stages. This mechanism leads to a different postnatal death rate pattern. An example will be shown later. Note that the arthropods are a very large group which includes all insects and crustaceans. Altogether it includes about 80% of all described living animal species.

[2] This situation was confirmed in a message received from an eminent bio-demographer. In an email to one of us (BR) dated 31 January 2016 Prof. Tom Kirkwood wrote:

> "There has been relatively little attention given to the patterns of early mortality. I am not aware of experiments that were done especially for exploring early mortality".

phenomenon is manifestly sufficiently similar to suggest a *core mechanism* is operating. This approach was convincingly illustrated in the book by Viswanathan et al. [6] entitled "The Physics of Foraging". The term "foraging" (i.e. to wander about looking for food) is generally used for insects but this activity is clearly of necessity for all animals. As the forms of foraging will not be the same on ground, in water or in the air, the challenge is to extract the core mechanism from such diverse manifestations. This was achieved by the authors of "The Physics of Foraging". In line with this expression we could have entitled our paper "The physics of infant mortality".

1.4. Infant mortality seen as a transition death spike

In a previous paper [7][3] we proposed the "*Transient Shock*" conjecture which states that:

Any abrupt change in living conditions generates a mortality spike which acts as a kind of selection process.

Although formulated in the context of living organisms, this conjecture may be extended to the failure rate of any system, a point which will be developed later. In the same paper the mechanism described in the conjecture was shown to be at work in the weeks following birth, in the months following marriage and widowhood and also when elderly persons are relocated in nursing homes. In all these transitions between the two different socio-environmental states, a transient mortality spike is observed during which the death rate is temporarily multiplied by a factor of 2 or 3 and even more in the case of the birth transition.

In mammals birth is a much more drastic transition than in many other living organisms. Just as an illustration, note that after hatching small alevins can still get their supply of oxygen by a diffusion process through their skin (this is due to their small size). As their size increases, gill respiration gradually replaces skin respiration. For mammals, respiration via the lungs must start in a matter of minutes following birth.

As explained in Ref. [7], the observed death spikes have a simple interpretation as a selection process. The systems which were adapted for state 1 but not for state 2 are eliminated; this results in a death rate increase. Although this explanation is satisfactory at a qualitative level, at a quantitative level a number of questions remain, for example:

- Is it possible to establish a connection between the characteristics of the transition and the amplitude of the mortality spike?
- The infant mortality phase is characterized by a death rate which decreases as a power law. Is this decrease species dependent or does it follow a general rule? We will see that contrary to the increase of death rates in old age which are very species dependent, the decrease of infant death rates is fairly uniform: $\mu_b \sim 1/t^{\gamma}$, where the exponent γ is of the order of one.

Most of the time the expression "power law decrease" refers to the slow decrease observed when $t \to \infty$. Here, on the contrary, the most conspicuous part of the mortality spike (Fig. 1a) is the sharp decrease immediately following $t = 0$. Whereas a power law decrease is often meant as a fall that is slower than an exponential, in the vicinity of $t = 0$ the fall is much faster than any exponential. In order to emphasize this difference in what follows we will use the expression "hyperbolic power law". This terminology is in agreement with standard usage in some technical fields (see for instance Ref. [10]).

1.5. Shape of the postnatal mortality spike

Fig. 1a shows human postnatal mortality in semi-log scale. It gives a good idea of the shape of the spike. As is common practice, postnatal mortality rates $\mu_b(t)$ were computed with a denominator which is the number of live births, that is to say:

$$\mu_b(t) = (1/S_0)\,(D(t, \Delta t)/\Delta t)$$

where $D(t, \Delta t)$ denotes the number of deaths in the time interval $(t, t + \Delta t)$. Thus, the curve of $\mu_b(t)$ with respect to the age t is meant to represent the speed of decrease of a cohort in the course of time.

This contrasts with the definition of the death rate used for adults (in the Appendix this rate is referred to as the "standard death rate") in which the denominator is the population at the beginning of the period under consideration. The origin and implications of this dual definition are discussed in the Appendix. Needless to say, numerically the two definitions are not very different as long as the cumulative number of deaths remains low, say under 10%.

However, in a semi-log representation with a linear time scale one cannot identify the curve as a power law and even more importantly one cannot explore a broad time interval. The inset of Fig. 1a shows how the data points become aligned in a log–log plot. This graph, however, is still for a short postnatal time interval of only 3 months. In contrast, Fig. 1b covers a whole life span from half a day to 32,000 days corresponding to an age of 90 years!

[3] In a broader way, the present paper should be seen as paper number 4 in a series of socio- and bio-demography investigations started in Ref. [8] and continued in Refs. [9,7].

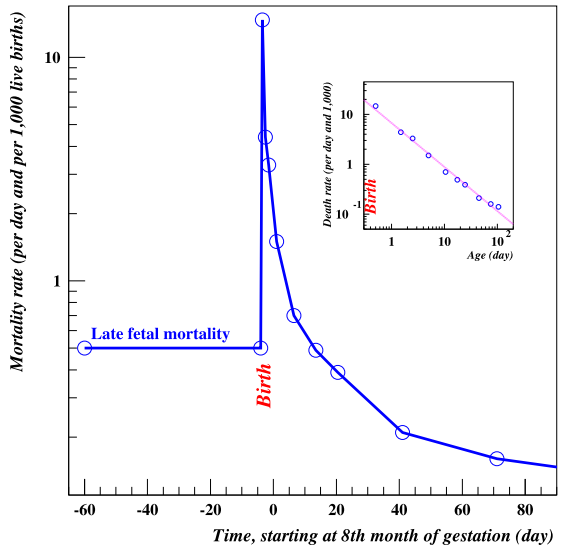

Fig. 1a. Transition from gestation to birth, USA 1923. The graph shows the death rate spike which occurs after birth. The level section on the left-hand side schematically indicates the (time-averaged) rate of late fetal mortality. Then, following birth, "defects" which were not of great consequence 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 day 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 [11, p. 574–575].

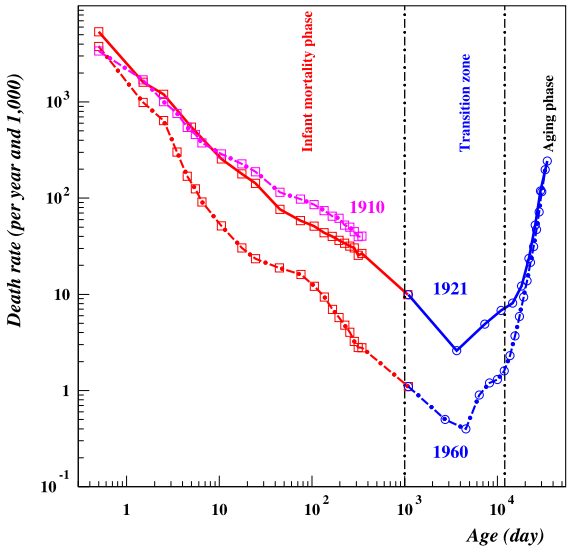

Fig. 1b. US death rates in log–log plot. There are two phases (infant and aging) separated by a transition. During the infant phase the death rate decreases in a hyperbolic way. It can be noted that despite medical progress the sections of the curves on each side which are closest to birth and death have not changed much. 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 $\gamma = 1.01 \pm 0.08$.
Source: 1910: Mortality Statistics of 1910, Bulletin 109 [12]; 1921: Mortality Statistics (1921); 1960: Grove and Hetzel [13, p. 210–211].

It shows three phases: (i) The hyperbolic power law decrease. (ii) A transition zone comprised between the two vertical lines. (iii) An aging phase where the death rate increases sharply. The first and third phases are certainly present in all living organisms. The first phase is a selection process through which the items with "manufacturing defects" are eliminated. The increase that occurs in the last stage is of course necessary if the death rate is to reach the level of 1000 per 1000 which signifies total extinction of the cohort. Needless to say, the respective length of each phase is species dependent. As an illustration one can mention the case of naked mole rates. We are told that these small mammals live until the age of 25 and that they remain in good health until the very end of their life [14]. This does not imply that the aging phase does not exist but rather that it is short and therefore that the final increase of the death rate must be very steep.

1.6. Models

Physics relies on both experiments and theory. The exploration of a new field usually starts with a number of insightful questions along with the experiments through which they can be answered. This is what we try to do here. Therefore, it should not come as a surprise that, at this point, we do not propose a full fledged model.

As far as modeling is concerned there are three challenges.

- The first is to identify the systems which will feature death rate spikes and to be able to predict their timing.
- The second is to predict the shape of the spike and its amplitude.
- When there is a power law decrease (i.e. for most organisms which do not go through several development stages) the third challenge is to predict its exponent.

The "Transient Shock" conjecture answers the first point. Although only semi-quantitative, it proved quite effective in the sense that all cases considered here as well as those already analyzed in Ref. [7] were based on TS predictions. The second point is the main unsolved issue. Regarding the third point, it will be seen below that (at least for human populations) there is a close connection between the exponent and the death rate at the age of 10 years.[4]

Although three models have been proposed in Appendix B of the arXiv version of this paper,[5] they are not yet satisfactory mainly because they have no real predictive power. Of course, by their very design, they are able to generate power law death rates but they rely on parameters which (so far at least) cannot be measured directly. As only predictions on *new cases* are meaningful, this means that the models cannot really be tested.

1.7. Gompertz's law versus infant mortality

Benjamin Gompertz was involved with life insurance and the law of aging he discovered in 1825 was first used to help with calculation of life insurance premiums.

Gompertz's law essential states that in any human population, after the age of 35, the death rate increases exponentially with a doubling time of the order of 10 years. For instance, in the United States in 1970, the death rate in the age group 35–44 was 3.1 per 1000 population of both sexes, 7.3 for 45–54, 17 for 55–64, 36 for 65–74 and so on (Historical Statistics of the United States 1975, p. 60).

Strictly speaking, infant mortality refers to the mortality between birth and one year of age. However, we will use this term in a broader sense for the whole period of time following birth during which the death rate decreases before it levels off and starts to climb. For humans, this phase extends from birth to the age of approximately 10 years. In the present paper this phase will be referred to as the *infant phase*. For humans (as well as for several other species) it roughly coincides with the period before sexual maturity.

In reliability studies, the time interval marked by a fall of the failure rate is also called infant phase.[6] The subsequent phase marked by an increasing failure rate is called "wear out" phase.

1.8. Birth transitions defined by the functions that must be switched on

In order to survive an animal or a plant must be able to use the oxygen contained in the air or in the water for generating energy. It must also be able to find food and to digest it. For animals finding food implies several challenges. (i) Identification of the food (ii) Moving to where it is located. (iii) Swallowing and digesting. In addition mammals and birds need to regulate their body temperature. Table 1 provides a summary of such functions in several types of species. The table focuses on a number of important functions but does not intent to provide a complete list. As a matter of fact, one can never be sure to have captured *all* functions. What makes the figures in the last column significant is the fact that the table lists factors which are crucial for development. These numbers can be seen as lower bounds of the real (yet unknown) parameters.

For all the functions that need to be implemented in state 2 there is a non-zero likelihood of failure which will result in an inflated death rate. In other words, one expects a mortality spike each time a new function needs to be implemented. This may happen at the time of birth but in some cases it may also happen after birth. This is illustrated in the next section.

1.9. Outline

We now proceed as follows.

(1) In the next section we focus on examples of infant mortality which are quite revealing because they do not occur at birth but at the end of the yolk-sac phase.

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4 This aspect will be fully developed in a forthcoming paper.

5 http://arxiv.org/abs/1603.04007. The first model proposes a connection with the distribution of defects whereas the second and third establish links between Gaussian or exponential variables on the one hand and power-law death rates on the other hand.

6 Other commonly used expressions are "burn in phase" or "early failure period".

Table 1
Classification of transitions according to the functions which are involved.

Type	State 1		State 2	Oxygen	Food digesting	Food finding	Temperature regulation	Number of +
Mammal	fetus	→	newborn	+	+	no	+	3
Bird	egg	→	newborn	no	+	no	+	1.5
Fish	egg	→	larva + yolk sac	no	no	no	no	0
Fish	larva + yolk sac	→	larva (no yolk)	no	no	+	no	1
C. elegans	egg	→	L1 larva	+	+	+	no	2
Plant	seed	→	seedling	+	+	+	no	2

Notes: "no" means that the corresponding function either was already ensured in state 1 or is unnecessary in state 2; e.g. temperature regulation is only necessary in homeothermic species. On the contrary, the + sign means that in order to survive the newborn must be able to implement the corresponding function. The + sign indicates that the function was already ensured in state 1 but not exactly in the way necessary in state 2. For instance, bird embryos may be able to digest the yolk contained in the egg but unable to digest the food brought to them by their parents. The table suggests that the birth of mammals involves more drastic changes than for the other organisms mentioned in the table. Hence, one expects a particularly high mortality spike. Many other functions are of vital importance (for fishes one can mention inflation of the swim bladder) but most of them are in fact included in the challenge of finding food because this task requires to see (or smell), move, catch, swallow and digest. Another parameter which is certainly of importance is the "complexity" of the systems. For an organism such as C. elegans which has only some 1000 cells one would expect less defects and therefore less infant mortality than for fish or mammals. The key-question is of course "How much less?"; to get an answer will require well designed dedicated experiments.

(2) In the following section, we discuss the case of a simple technical device, namely incandescent light bulbs. This will give us the opportunity to explain in a concrete way what is meant by the expression "lethal defect". Moreover, the discussion of a specific defect will allow us to explain the connection between the characteristics of the defect and the age-specific failure rate. We will see that even a very simple type of defect may lead to a failure rate which decreases with age as a power law.
(3) Then, we present human infant mortality data. The statistical evidence suggests that the death rate in the hours following birth is largely independent of medical care. In order to get a better insight into the selection process at work during the infant mortality phase we distinguish death rates corresponding to various causes of death. It will be seen that some follow a power law behavior whereas others do not.
(4) Next, in the hope of establishing a chain of cases extending from "simple " to "complex" systems, we present and discuss data for other species, e.g. farm animals, primates, insects, plants.

2. The yolk-sac effect

In the previous section we have seen that for the case of human births the simultaneous activation of 3 functions brings about a huge spike. This naturally leads to question what happens when only one or two functions are activated simultaneously. If this activation occurs at a time t_1 after birth one would expect a spike to occur around t_1. Does observation confirm this prediction?

What kind of organisms would be most appropriate for such a test? An idea which comes to mind is to use organisms whose development goes through several stages. This is the case of most insects. Worms giving rise to flies or caterpillars giving rise to butterflies are well known cases. From egg to adult the development of insects involves several stages, not to speak of the successive instars and moults. However, for our purpose those stages are not really appropriate because they are too different from one another. Feeding, for instance, is not at all the same problem for a caterpillar and for a butterfly.

Fish larvae provide much better cases.

2.1. Yolk-sac mortality spikes for fish larvae

When fish emerge from their egg they carry with them a yolk-sac which provides them with food until it is exhausted. The inset of Fig. 2a shows such a yolk-sac for salmons, a species in which it is particularly big; it can nourish salmon alevins for 60 days.

Once the sac is depleted, the fish must find their food themselves, a task which requires a whole chain of functionalities: seeing, catching, swallowing and digesting the food. If any of these functions fails the fish will die from starvation within a few days. Even for the fish which have no functional defect, the transition results in increased fragility to external factors (e.g. level of oxygen dissolved in the water, excess-fish density and so on). Thus, based on genetic defects and excess fragility, one expects a mortality spike in the days following the end of the yolk-sac phase.

In other words, knowledge of the yolk sac duration allows us to predict the timing of the mortality spike for almost all species of fish. As the duration of the yolk sac covers a broad interval from 3 days for anchovies to 60 days for salmon alevins, we are in a favorable situation for testing these predictions.

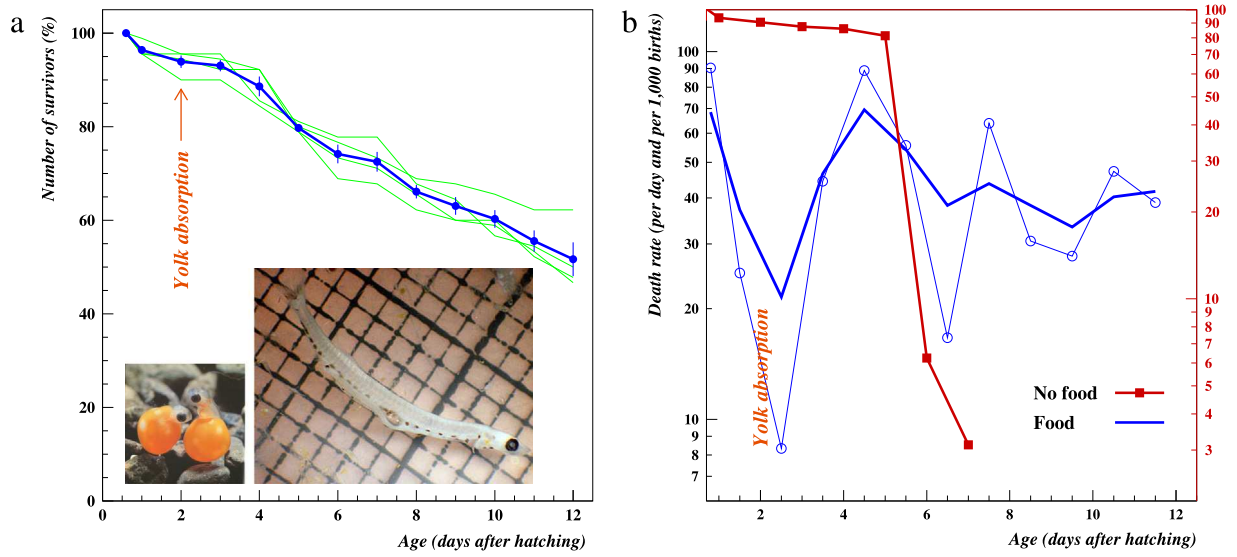

Fig. 2. (a, b) Yolk sac mortality spike for larvae of California anchovy. Left: Survival curve of larvae of California anchovy (*Engraulis mordax*). The larvae were reared in four 10 liter containers containing seawater and 100 eggs. The thin (green) lines are the survival curves for the four containers. The thick (blue) line is the average and the error bars show the standard deviation of the average. The first inset shows two larvae (not of E. mordax but of salmons which have a larger amount of yolk) shortly after birth with their yolk sacs, while the second shows a larva of *E. mordax* whose length is 9 mm which corresponds to an age of 18 days. Right: For the same data the graph shows the death rate i.e. daily deaths divided by the number of hatched eggs. The thin (blue) line displays the daily death rates. The thick (blue) line is a 3-point centered moving average. The peak between days 3 and 8 follows the depletion of the food reserve contained in the yolk sac. The (red) line with the squares corresponds to an experiment in which no food was given; it shows that there is a time lag of about 3 days between yolk-sac depletion and ensuing mortality. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
Source: Laskar [15].

It is interesting to observe that whereas this excess mortality is commonly observed by researchers, it is often attributed to various special reasons (cannibalism, unexpected changes in tank conditions) without any real awareness of the underlying yolk sac factor.[7]

The following subsections describe experimental tests for several species starting with small fish which have usually a short yolk sac phase and ending with large fish which have longer yolk sac phases. Table 2 provides a recapitulation of the tests; it shows good agreement between yolk-sac based predictions and observed death spikes.

2.2. Anchovies and sardines

In Fig. 2a the slope of the survival curve displays slight oscillations. However, given the scale of the statistical fluctuations (shown by the thin lines) it would be tempting to discard them as being non significant. Yet, once death rates are computed a peak appears in Fig. 2b which coincides with the end of the yolk sac phase as indeed shown by the starvation curve.

The comparison of Fig. 2a and b suggests that although commonly used (it is the only curve given in the paper from which these data are drawn) the survival curve is fairly opaque. The death rate curve provides a much better insight. Of course, as the death rate is the derivative of the survival function, it will be reliable and significant only if the size of the sample is large enough to dampen the statistical fluctuations.

A similar effect can be found in the data published by a team led by Susana and Garrido [16] who reared 820 larvae of European sardines (*Sardina pilchardus*) in laboratory conditions and recorded the number of deaths every day from hatching of the eggs to 60 days later. For sardines the yolk sac phase lasts until they are 3 days old. On the curve of the death rate as a function of age (not shown here) there is a sudden surge in the interval $(5, 8)$ in which the death rate is multiplied by 2. Then, the fall is resumed and continues until day 60. In short, the mortality spike follows the depletion of the yolk sac with a time lag of about 2 days.

2.3. Longer yolk-sac phases: redfish, sturgeons and salmons

As the yolk-sac mechanism is common to all fishes which lay eggs, many data should be available. Of particular interest are the larvae of salmons for in this case the yolk sac stage lasts about 60 days. For the present investigation one needs daily data for populations reared in controlled conditions. Recently, we received some salmon data from a Norwegian

[7] As illustrations note the papers by Laskar [15] and Garrido et al. [16].

Table 2
Testing predictions about larvae mortality spikes.

	Species	Number of eggs n	Hatching rate (%)	End of yolk phase (day)	Predicted interval of death rate spike (day/hour)	Test of prediction through observation	Reference of data
1	California anchovy	400	?	2	4–7 d	C	Laskar [15]
2	European sardine	820	?	4	5–8 d	C	Garrido et al. [16]
3	Black Sea turbot	77,000	74%	3	4–6 d	C, low accuracy	Sahin [20]
4	Redfish	800	74%	10	12–20 d	C, low accuracy	Laurel et al. [18]
5	Siberian sturgeon	∼2000	?	10	12–16 d	C	Gisbert et al. [19]
6	Zebra fish	12,000	90%	8	10–15 d	C	Cousin et al. [21]
7	Salmon	159,200	?	60	65–70 d	C	Seim [17]
8	C. elegans larvae		98%	No yolk	0–3 h	Not yet done	
9	Drosophila larvae		98%	No yolk	0–12 h	Not yet done	

Notes: "C" means that the prediction was confirmed. The prediction is based on the identification of the depletion time of the yolk sac. The death spike starts 2 or 3 days later and lasts between 3 and 7 days. The data given in the paper which are referenced were collected for various objectives (e.g. influence of type of food, ecotoxicology) which most often were quite different from our present purpose. Cases 1–7 correspond to fish whereas 8 and 9 refer to 1 mm-long worm-like larvae. C. elegans has a much shorter life span than the fish: 20 days versus 15 years for sardines (and even longer for turbots or sturgeons) which means that days have to be replaced by 5 mn- or 10 mn-long time intervals. The scientific names are as follows: 1 = *Engraulis mordax*, 2 = *Sardina pilchardus*, 3 = *Scophthalmus maximus*, 4 = *Sebastes mentella* and *Sebastes fasciatus*, 5 = *Acipenser baeri*, 6 = *Danio rerio*, 7 = *Salmo salar*, 8 = *Caenorhabditis elegans*, 9 = *Drosophila melanogaster*.

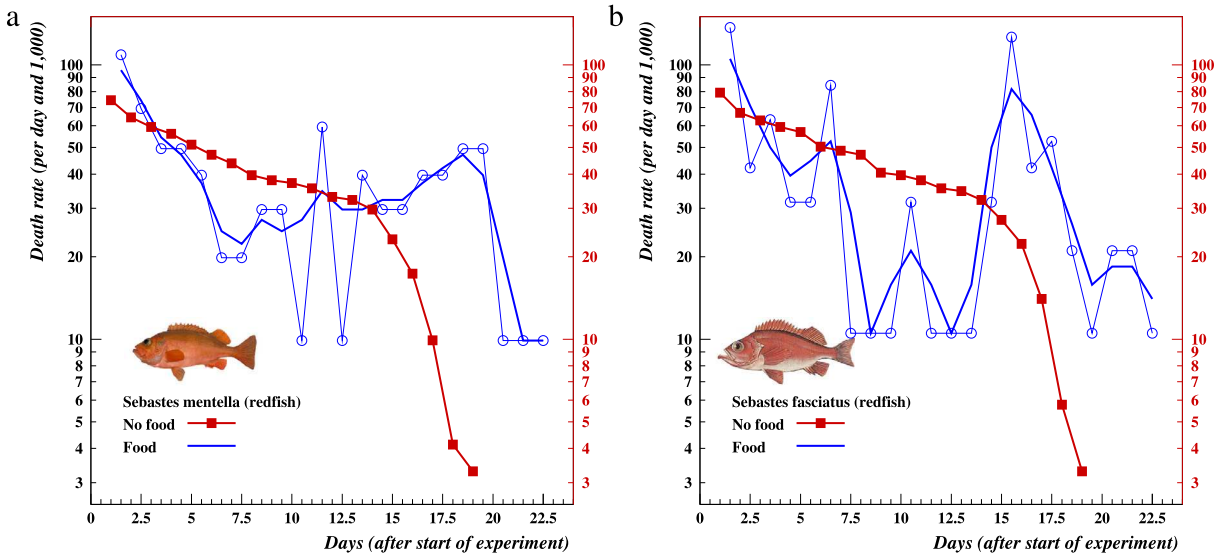

Fig. 3. (a, b) Yolk mortality spike for two species of redfish larvae. Left: *Sebastes mentella*; Right: *Sebastes fasciatus*. Each curve corresponds to a sample of 200 larvae. The red curves with the squares show the percentages of survivors when no food is given to them even after their yolk-sac is depleted. In both graphs, in accordance with expectation, the mortality peak of nourished fish occurs in synchronicity with the collapse of a population that is not nourished. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
Source: Laurel et al. [18, p. 889].

colleague [17]. They confirmed the prediction of a death spike in the interval (60 d, 70 d). Its amplitude is about 10 higher than the background noise. As these data are still provisional they will be analyzed more fully subsequently.

In this subsection we present data for redfish (Fig. 3a, b) and sturgeons (Fig. 4a, b). In both cases the yolk-sac phase lasts about 12 days. Redfish have the additional interest that, in contrast with the majority of fish, the fertilization occurs internally and the female spawns swimming larvae rather than eggs. However, from our perspective this makes little difference because the larvae carry a yolk-sac just as when hatching occurs externally.

The data provided by Laurel et al. [18] have a good side but also two drawbacks. The good side is the fact that they include data for an unfed group which allows us to know fairly exactly the moment when the depletion of the yolk-sac becomes effective. However, one drawback is the fact that the small number of individuals in each sample (namely 200 larvae) leads to fairly large statistical fluctuations. The second drawback is even more serious. It consists in the fact that the larvae were collected offshore and were "stripped from ripe females" collected by a fishing ship. As not all females were exactly at the same stage this collection method created large time lags. This in turn led to fairly broad peaks extending over nearly 10 days. This effect is particularly obvious for *Sebastes mentella.*

What makes the data of Gisbert et al. [19] of particular interest is the fact that they rely on samples of 2500 larvae, i.e. ten times more than in the previous experiment. However, as this experiment (as well as all others) was not designed for the purpose for which we are now using it, there is also a downside, namely the fact that there is no unfed sub-sample. This means that one cannot identify exactly the end of the yolk-sac phase.

For sturgeon the yolk sac phase lasts about 10–12 days which, although much shorter than the 60 days for salmon, is substantially longer than in the cases of anchovy or sardines. As shown in Fig. 4a the transition from endogenous to exogenous feeding is marked by a major death peak. The fact that the transition is distributed over 2 days results in a kind of moving average; in other words, if all larvae were synchronized the peak would likely be much sharper. This can be verified (Fig. 4b) by superposing dispersed spikes and noting that the global curve is fairly close to the observed mortality rate. Incidentally, in the same paper the authors demonstrate that there is a correlation of 0.63 between the diameter of the eggs and the time until first exogenous feeding. That property can possibly be used to improve synchronicity in future experiments.

2.4. Summary of noteworthy cases

Table 2 summarizes the characteristics of cases already explored in which the predictions based on the *Transient Shock* conjecture were confirmed by observation.

3. Problems raised by the measurement of infant mortality

Ever since Gompertz's law was discovered in 1825 the study of aging has attracted considerable attention. In contrast, as we noted earlier, infant mortality has been neglected. A testimony to this neglect can be found in a paper by Raymond

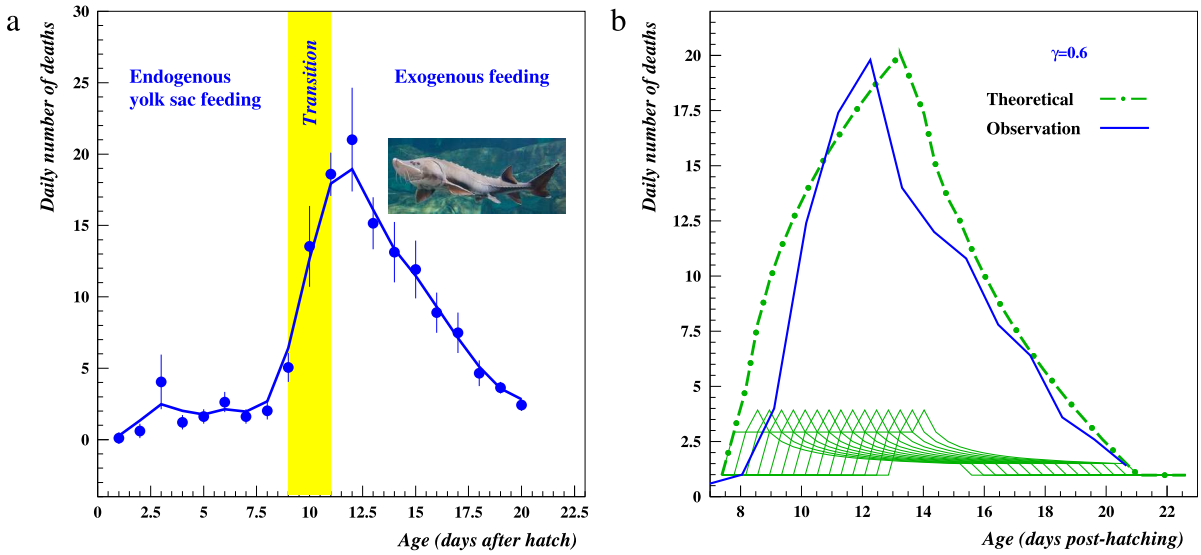

Fig. 4. (a, b) Yolk mortality spike for Siberian sturgeon larvae. Left: Each experiment involved 2700 eggs. The duration between spawning and hatching of the eggs was 7 days, After hatching the fish lived for 9–11 days on the food reserve of their yolk sac. The transition to exogenous feeding was marked by a major death peak. Right: The fact that the observed death rate does not look like the spike of Fig. 1a can be due to a dispersion in the age of reaching the stage of exogenous feeding. The curves at the bottom (thin green lines) are hyperbolic death spikes of the form $1/(t-d)^{\gamma}$, $\gamma = 0.6$. The (green) dot–dash line is their sum. The (blue) solid line is the same curve as in (a). The graph shows that the superposition of the spikes produces a global curve that is fairly close to the observed curve. For the sake of simplicity we assumed a uniform dispersion of the time-lag d; the real (albeit unknown) distribution of d is probably not uniform. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
Source: Gisbert et al. [19, p. 89].

Pearl and his collaborators (1941) where in the conclusion it is stated that the life curves of the beetle *Tribolium confusum* "resemble in their fundamental pattern human life curves more closely than those of any other organism for which life tables have been computed". Yet, as will be seen shortly, the *infant mortality* curve of *Tribolium* differs completely from the human curve.

The lack of interest in infant mortality curves may be attributed principally to three circumstances.

(1) Whereas age-specific death rates over the age of 35 form a straight line in a semi-log (x, $\log y$) plot, infant death rates form a straight line in a log–log plot. This means the age-specific infant death rate is a hyperbolic power law whose determination requires data points as close as possible to the moment of birth. We will see below that hour-by-hour postnatal death rate data for humans has only recently become available.

(2) Whereas old-age mortality has not been substantially affected by medical progress, infant mortality has been dramatically reduced over the past century. Around 1900 infant mortality during the first year of life was still of the order of 150 per 1000 ([22], p. 142–145, [23] Vol. 2, Chapter 6). Today, in most industrialized countries, it is around 3 per 1000! At first sight it might seem that a variable that is so strongly dependent upon external factors does not have much intrinsic biological interest. However, as will be seen below, in spite of the huge reduction in magnitude, the *shape* of the death rate has remained the same. If we write the death rate as $\mu_b(t) = A/t^{\gamma}$, the numerator A has been divided by a large factor but the exponent γ has changed only slowly. Moreover, as shown in Fig. 1b, the death rate shortly after birth has changed very little.

(3) Infant mortality is defined as being mortality after birth whereas mortality occurring between conception and birth is called fetal mortality. Because for the first months after conception fetal mortality data are very uncertain, this variable is not considered as very significant. Nevertheless, in ecological studies, mortality estimates usually cover the whole period after the production of eggs. This is for instance the methodological option used by Itô [24]. Unfortunately, with such an option survivorship curves lose almost all significance. The reason is easy to understand. Many organisms, particularly insects and fishes, produce a large number of eggs of which many die within a short time.[8] Under such conditions all data points for later life will be confined in a narrow range even if one uses a logarithmic scale.

3.1. Two classes of explanations

Later we show that the decrease of infant mortality has the same shape as in humans for various animal species: monkeys, lambs, birds, even crocodilians[9] albeit with a different exponent. However, the rule is *not* valid for insects whose

[8] For instance, as a fairly extreme case, the Atlantic mackerel, (*Scomber scombrus*), lays about one million eggs of which only a few survive until 70 days after laying.

[9] The crocodilian group includes true crocodiles, alligators and caimans.

development proceeds through successive life stages. For the sake of brevity, species which follow the power law decrease will be referred to as infant mortality power law (IMPOL) species.

For IMPOL species, this raises the question of the origin of this similarity. Two possible mechanisms come to mind.

- It may be that IMPOL species share similar initial biological "defects" which are then "filtered out" through the infant mortality process. This will be called explanation *A*.
- Alternatively, it may be that there is a great variety of lethal effects that may differ between individuals and species, but that they have some properties in common which ensure that their global effect in the course of time will take the power law form that we observe. This situation, which will be referred to as explanation *B*, would be similar to the addition of non-identical random variables whose global contribution, according to the central-limit theorem of probability theory, takes the form of a Gaussian variable.

4. From technical devices to biological systems

4.1. Examples of causes of failure

When a collection of technical devices are put into operation at the same time t_0 a fairly high failure rate is usually observed during a length of time that reliability engineers call the *infant mortality* phase. One by one, in the course of time, items which have a defect will fail. It is important to recognize that the length of time that it takes for defects to manifest themselves can be very variable.

Let us give two examples which illustrate this point.

- First, we consider incandescent light bulbs. The light is produced by a wire filament heated to a high temperature by an electric current I passing through it. Suppose that at some point the section σ_1 of the filament is smaller than the average section $\overline{\sigma}$. The amount of heat produced in one second per unit of length of the filament is given by $h = rI^2$ where $r = \rho/\sigma$ is the resistance (expressed in Ohm/meter) per unit of length of the filament and ρ is the resistivity of tungsten.

  A reduction in σ will bring about a local increase in resistance, this in turn will produce a higher heat release and push up local temperature. In addition, the resistivity of tungsten increases with temperature. Around this hot spot the evaporation of tungsten will be faster. Sooner or later, this positive feedback process will lead to the severance of the filament and the failure of the lamp.

  As this mechanism is familiar to all physicists, one may wonder why we explained it in some detail. The reason is very simple. We wished to point out that there is a correspondence between σ_1 and the life-time, $\theta(\sigma_1)$, of the lamp. Before elaborating further we wish to present a second illustration.
- Consider a device which contains one or several spinning wheels and suppose that one of the axes is slightly off center. This will result in vibrations which, sooner or later, will damage the wheel and lead to the failure of the whole device. Here too, there will be a one-to-one correspondence between the magnitude of the defect, δ, and the time to failure $\theta(\delta)$ of the device.

4.2. Mathematical description of the distribution of failure times

Let us consider more closely what the existence of the functions $\theta(\sigma_1)$ or $\theta(\delta)$ implies for a large sample of devices. In reliability studies one is usually interested in the average life-time (also called mean time to failure or MTTF) of a device. In the present paper we wish to go further in the sense that, instead of its mere average, we will study the statistical distribution of the life-times. This means that for a cohort of $S_0 = 1000$ items which start to work at the same moment, we wish to know how many will fail in the first second, first minute, first hour and so on.

Mathematically, the statistical distribution of life-times can be described in a number of ways which are fairly equivalent. One possible description is through the decrease in the course of time of the number of survivors $S = S_t$. Although, this survivorship description is commonly adopted especially in biology (see for instance Ref. [24]) it is not very suggestive because all these survivorship curves are of course decreasing functions. A more suggestive representation is through the evolution of the death rate μ as a function of age. The death rate is defined as:

$$\mu(t) = (1/S_t)\,(\Delta S_t/\Delta t)$$

where Δt denotes a given age interval.

However, in medical statistics the postnatal death rate is defined by replacing in the previous formula S_t by S_0:

$$\mu_b(t) = (1/S_0)\,(\Delta S_t/\Delta t)\,.$$

Usually during the infant phase S_t and S_0 are not very different which means that $\mu(t) \simeq \mu_b(t)$. However when S_t becomes much smaller than S_0, the two formula may lead to fairly different death rate shapes (see the Appendix).

4.3. Black box style explanations of the failure rate pattern

With these definitions in mind, let us come back to the example of the light bulbs. So far, we have considered one lamp, now we consider a sample of lamps. Suppose that all their filaments are *exactly* identical with (as before) a section that is reduced to σ_1 at one point. As a result they will have the same time to failure θ_1 which means that for all lamps their failure rate $\mu_b(t)$ will be zero for $t < \theta_1$ and equal to 1 for $t = \theta_1$.

In reality the filaments cannot be completely identical. If the minima σ_1 are distributed according to a density function $f(\sigma_1)$ it will result in a density function $f(\theta_1)$ which in turn determines the function $\mu_b(t)$. So, there is a one-to-one correspondence between the profile $f(\sigma_1)$ of the filaments and the shape of $\mu_b(t)$.

The data presented in the following sections suggest that $\mu_b(t) \sim 1/t^{\gamma}$. To explain such a fairly "exotic" behavior it might be tempting to assume a fairly complex internal structure of the system. For instance, in the work of Gavrilov and Gavrilova [25] degrees of redundancy are assumed which are achieved by identical subsystems working in parallel. In other papers (e.g. Ref. [26]) it is assumed that there is a whole range of failure effects, each one characterized by a specific distribution curve.

The example of the light bulb suggests that a *single cause* of failure involving one parameter (e.g. the local diameter of the wire) is sufficient to explain any shape of $\mu_b(t)$, no matter how exotic. Naturally, the fact that one cause of failure may be sufficient does not imply that there is indeed only one cause of failure. However, the previous argument tells us that before turning to complex internal structures one should rather try to get a better knowledge of the most likely causes of failure.

Conversely, we conclude from the previous discussion, that the shape of the infant death rate as a function of age gives information on the failure mechanisms at work in the system. In other words, postnatal death rates may also be an exploration tool.

5. Human infant mortality

5.1. Hyperbolic power law fall of postnatal rates: can one predict the exponent?

Human data are far more detailed than for any other living organism.

Fig. 1b and Fig. 5a, b show that the postnatal death rate has kept its power law shape in spite of a considerable reduction in the global level of infant mortality. This is shown for Switzerland from 1885 to 2013 and for Britain from 1921 to 2010.

The factor which determines the exponent of the power law appears fairly clearly on Fig. 1b and Fig. 5a, b. It is not determined by what happens immediately after birth but rather by the death rate at the end of the infant mortality phase. Indeed, it can be seen that in the close vicinity of birth (i.e. for a few hours after birth) all death rate curves converge toward the same point. In order to see this effect most clearly one needs series for the same country which start as soon as possible after birth and which are widely apart in the course of time. The Swiss data shown in Fig. 5a turn out to best fulfill these conditions.

This is another instance of a fixed point model of the kind already discussed in the context of old age in Ref. [8]. Here, the exponent of the power law will become higher when the death rate at age 10–14 decreases. Historically, this is what happened in western countries during the 20th century. Between 1900 and 1980 the death rate in the age-group 10–14 was divided by a factor of the order of 10. (Historical Statistics of the United States p. 60.) Similarly the same argument tells us that γ will be smaller in developing countries (which have comparatively large death rates in the age group 10–14) than in developed countries. Testing these predictions more precisely may be the purpose of a subsequent paper.

5.2. How shortly after birth does the hyperbolic law start?

Mathematically, a hyperbolic law $1/t^{\gamma}$ cannot hold until $t = 0$; there must be a cut-off time t_c prior to which another law sets in. Fig. 6a shows that t_c is of the order of 1 h.

We see that the end of the decrease phase occurs in the age interval 10–14 years. After that age the death rate starts to increase, slowly at first and then after the age of 30 it assumes an exponential growth in accordance with Gompertz's law.

5.3. Infant death rates by cause of death

Since 1995 the Swiss Federal Statistical Office publishes daily, weekly, monthly and yearly infant death rates by cause of death. The list has about 20 entries which fall into two broad classes. (i) Congenital malformations, e.g. of the nervous or circulatory system (ii) Diseases, e.g. infectious diseases, cancer, diseases of the digestive system, neuropathies.

Not surprisingly, the malformation category is largely predominant in the earliest part of life. As a matter of fact, the number of deaths due to diseases is so small that in order to get significant estimates one needs to add up the death numbers for all the 19 years for which data are available.

As an illustration let us compare deaths from cancer, and congenital malformations.

Table 3 shows that the number of deaths for the two causes move in opposite directions; thus, clearly, they cannot be ruled by the same law. In fact, the daily deaths by cancer first start to decline and then level off around the age of one

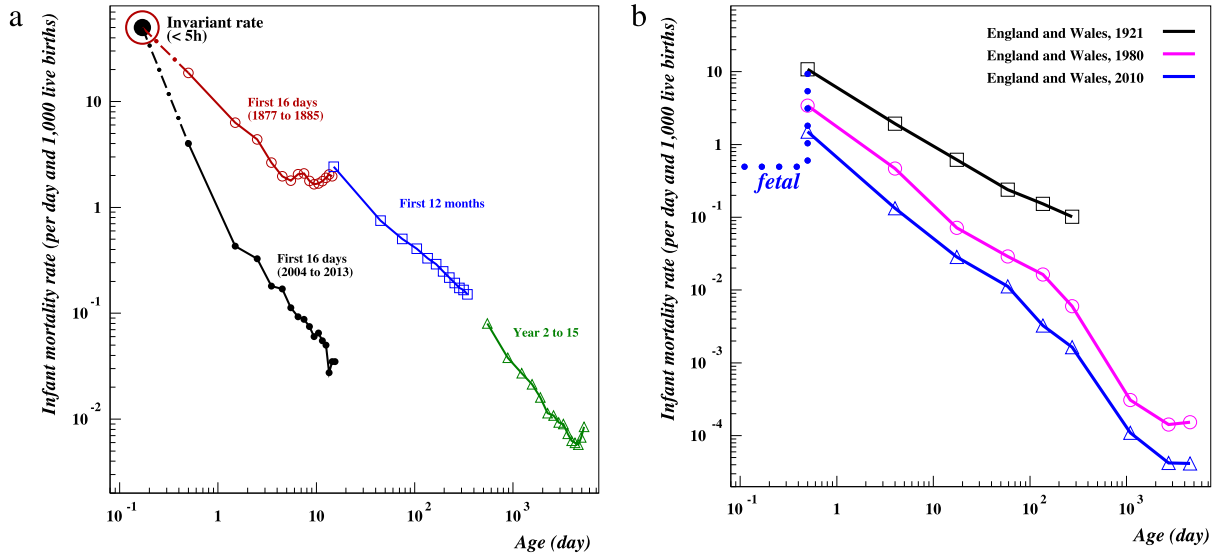

Fig. 5. (a, b) Postnatal death rates from 1 day to the age of 15 years. Left: Switzerland: The upper curve is for 1877–1885 while the lower curve is for 2004–2013. Comparison of the two curves for the first 16 days of life suggests that the section of the curve for times shorter than 5 h after birth has remained unchanged despite medical progress. The slope of the regression line for the first 12 months is $\gamma = 0.85 \pm 0.04$; this slope is almost the same as for the first 4 days; regarding the days 5 to 16, so far we have no explanation for why there is a level section; for the first 15 years the slope is $\gamma = 1.12 \pm 0.14$. Right: England and Wales (1921–2010). Comparison of the curves shows also that in spite of a huge decrease in infant mortality, the age-specific pattern remained basically the same. The end of the decrease around the age of 10 years (i.e. some 4000 days) marks the limit of what in this paper we call the infant phase. As the fetal phase would correspond to negative ages, the magnitude of the late fetal rate is indicated in a fairly schematic way, basically for the purpose of comparison with postnatal rates. From top to bottom the slopes of the regression lines are $\gamma = 0.75 \pm 0.05$, $\gamma = 1.16 \pm 0.12$, and $\gamma = 1.19 \pm 0.1$ respectively.
Source: Switzerland: The following website of the "Federal Office of Statistics": http://www.bfs.admin.ch/bfs/portal/fr/index/infothek/lexikon/lex/2.html provides a compilation of historical series and in particular it contains all the annual issues of "Mouvement de la population de la Suisse" (i.e. Vital statistics of Switzerland) starting in 1877. The data for the first 16 days are from the volume of 1885. The data for the first 12 months are from the "Annuaire Statistique de la Suisse" (Statistical Yearbook of Switzerland) (p. 75). Britain: Child mortality statistics, 2013, Table 1 and Table 17, Office of National Statistics (UK).

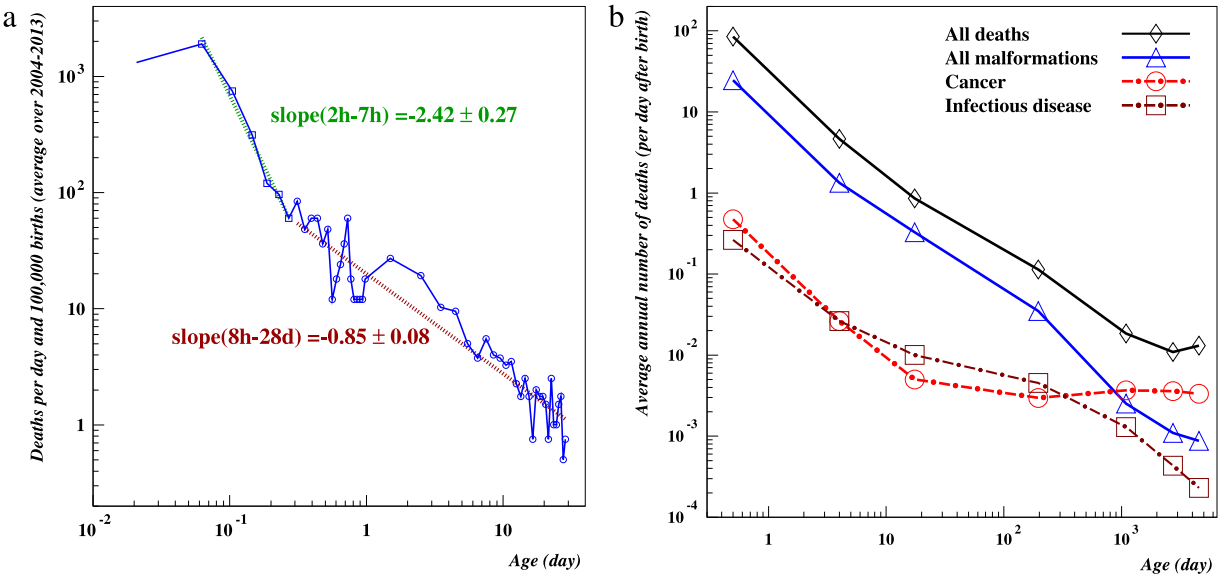

Fig. 6. (a, b) Infant death rate in Switzerland from one hour after birth to the age of 12 years. Left: From one hour after birth to 28 days after birth (neonatal mortality). If one leaves apart the first hour, the following 7 h are characterized by a much steeper slope than later times. This effect can be "explained" in terms of prematurity. Right: With its much broader time scale this graph gives the global picture over the whole interval marked by a decrease of the infant death rate. The slopes of the log–log regression lines for the 3 power law cases are as follows: all death: 0.97 ± 0.12, malformations: 1.12 ± 0.07, infectious disease: 0.69 ± 0.10. On each curve the 6 data points are averages over the following age intervals. Days: (0–0.9), (1–6.9), (7–27.9), (28–364.9); years: (1–4.9), (5–9.9), (10–14.9). The curves refer to males and are averages over the 19 years 1995–2013.
Source: Swiss Federal Office of Statistics.

Table 3
Number of deaths from two causes in Switzerland (average over 1995–2013).
Source: Swiss Federal Office of Statistics.

Cause of death	Day 1	Year 1.0–4.9
Cancer (tumor)	0.42	5.4
Congenital malformations	25	3.7

Notes: It can be seen that the death numbers move in opposite directions: up for cancer, down for malformations. However malformations remain important even several years after birth.

year. So, why does one observe a power law for total death numbers? The reason is suggested by Table 3: during the first day the deaths from malformations are 60 times more frequent than those from cancer. Subsequently the deaths due to malformations decrease but even years after birth, e.g. for the age group (1 year–4.9 years), they remain quite significant and indeed of the same order of magnitude as the deaths from cancer.

Fig. 6b shows that unlike cancer deaths which do *not* follow a power law, deaths from infectious diseases follow a power law. It is true that there are fluctuations but there is no *systematic* deviation. The exponent for diseases is somewhat lower than the exponent of total deaths:

$$\gamma(\text{infection}) = 0.69 \pm 0.10, \qquad \gamma(\text{total}) = 0.97 \pm 0.12.$$

This observation raises a question. What would be the shape of the infant mortality curve during an outbreak of infectious disease of the kind that occurred at the end of the 19th century? One would expect a curve that would be a composition of malformation and infectious deaths. This should give a power law in two parts with γ close to 1 immediately after birth and then around 0.7 for ages between 2 and 10. Once data become available it will be possible to check this prediction.

5.4. Incidence of prematurity on the exponent of the power law

The expressions "preterm birth", "prematurity" and "birthweight" refer to three distinct yet related notions.

- Preterm refers to the duration of pregnancy. Preterm birth means birth before a gestational age of 38 weeks. Preterm birth ranges from 22 to 37 weeks.
- The word "prematurity" has a functional and structural meaning. It refers to the fact that birth occurs at a moment when the organs of the baby are not completely mature. For instance, the lungs are one of the last organs to mature which implies that preterm babies may have lungs that are not yet fully functional. The degree of prematurity depends upon two parameters: the development rate and the delivery time; it must be assessed qualitatively through medical examination.
- The birthweight parameter is simply the weight of the baby at birth. As this variable is well defined, easy to measure and has a substantial overlap with the two other notions, it is often taken as an indicator which can represent them too. However, the introduction of ultrasonography has made direct determination of gestational age much more precise.

Prematurity is similar to the frailty index described in the introduction. Thus, studying the effect of birthweight on mortality gives us the opportunity to measure the effect of frailty in a fairly clean case. This is done in Fig. 7a, b, c.

Before discussing this graph a comment is in order regarding what can appear as a relative data paucity. The three data points in Fig. 7a represent the death rate in three standard time intervals defined in obsetrics:

(1) Early neonatal: day 0–day 7, middle point: day 3.5.
(2) Late neonatal: day 7–day 28, middle point: day 17.5.
(3) Post neonatal: day 28–day 365, middle point: day 196.5.

Medical practice being what it is, one would not expect more fine-grained data to be available elsewhere. Graph 7b exhibits 5 data points instead of 3 only because the children were followed beyond the age of one year. This is only possible in Scandinavian countries because of the existence of national medical and vital statistics data bases. In Norway, for instance, it was possible to link the "Medical Birth Registry of Norway" (MBRN) to the "National Cause of Death Registry".

Of course, three or even five data points would be highly insufficient for proving that the phenomenon follows a power law. However, as these data are merely subsets of the whole data set shown in Fig. 5a, b and Fig. 6a, b, it seems reasonable to admit that they follow a power law too. Moreover, our main objective is to determine only one parameter namely the slope γ of the regression lines. This procedure will lead us to a simple and, we think, interesting conjecture. If (either through a piece of good luck or because medical doctors would like to check the validity of our conjecture), more detailed data become available in the future, it will be possible to see if our conjecture is confirmed or not.

Fig. 7a shows two things:

(1) The death rate increases greatly (by a factor 1000) with the degree of prematurity. It is of course hardly surprising that it increases but the fact that it is multiplied by one thousand is noteworthy.
(2) There is a convergence of the regression lines with increasing age.

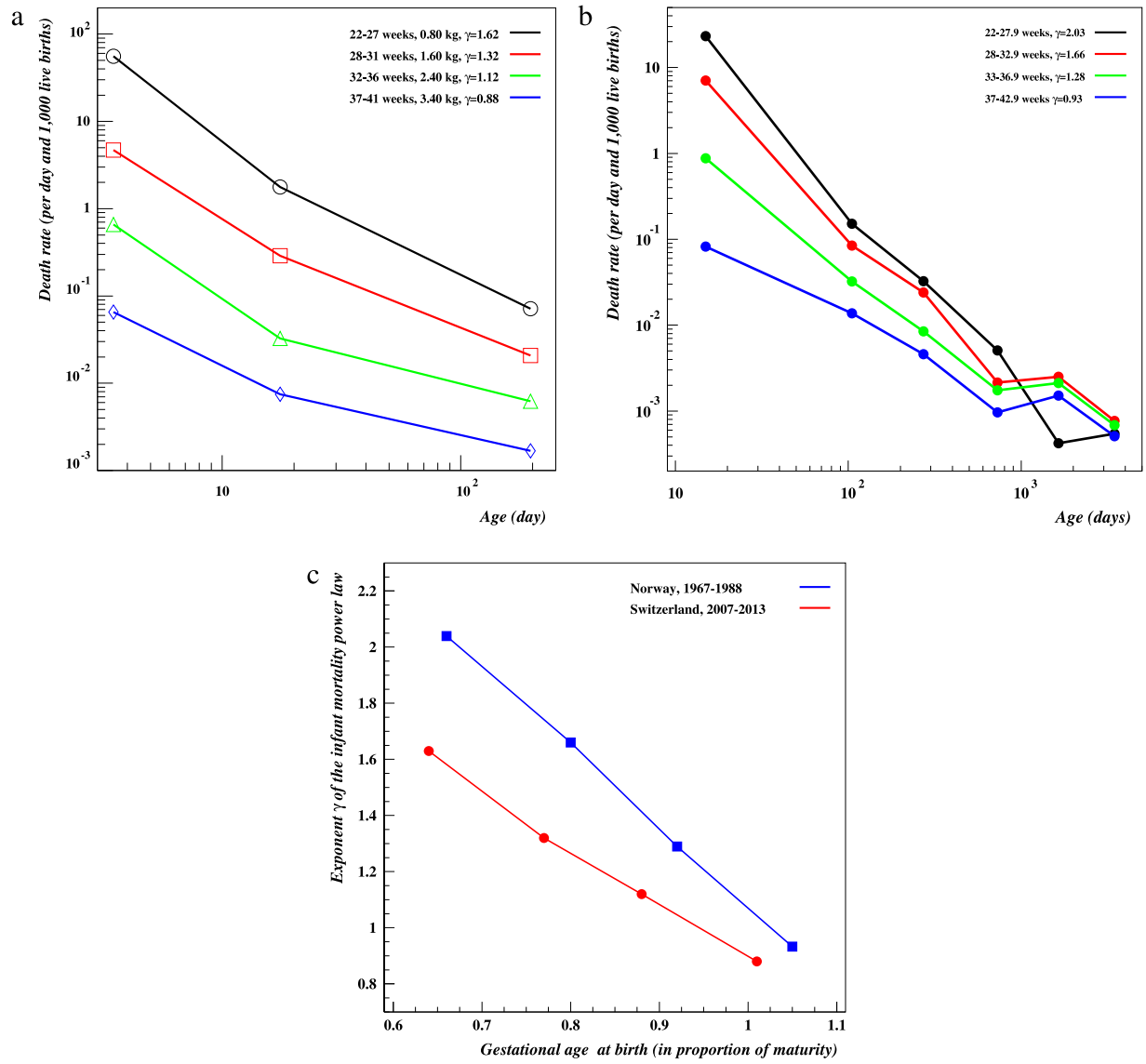

Fig. 7. (a, b) Incidence of low birthweight on death rates. For both graphs the gestational age at birth increases from top to bottom. Left: This graph is for Switzerland and it covers only the first year. The 3 data points are for the following standard age intervals: early neonatal (0–7 d), late neonatal (7–28 d) and post neonatal (28–365 d). Low birthweight (which is usually associated with preterm birth) results not only in higher levels of death rate but also in higher exponents. As explained in the text, the two effects are in fact closely connected. The data are averages over 7 years, namely 2007–2013. Right: This graph is for Norway and it covers the period from birth to early childhood (6–12.9 years). (c) Incidence of the degree of maturity p on the exponent γ of the power law. Squares: Norway, solid circles: Switzerland. p can be seen as an inverse frailty index in the sense that the accumulation of deficits increases when the degree of maturity decreases. Let us recall that γ is defined by the relation: death rate $= 1/\text{age}^{\gamma}$. The linear regressions $\gamma = -ap + b$ take the following form: Norway: $a = 2.8 \pm 0.1$, $b = 3.93 \pm 0.01$, Switzerland: $a = 2.0 \pm 0.2$, $b = 2.90 \pm 0.02$.
Source: Same as for Table 4.

Long-term studies (particularly the one by Swamy et al. [27]) demonstrate that at least one third of extreme preterm children (22–27 weeks) face disabilities which extend into their later life. However, their death rate in late childhood (6–12.9 year old) is less than 10 times higher than the death rate at same age in the general population ([27], Fig. 1). Thus, with respect to term babies, their death rate ratio falls from about 1000 shortly after birth to about 10 a few years later. This convergence of the regression lines is particularly clear in Fig. 7b but is already visible in Fig. 7a.

The consequence of this convergence is that the regression lines become steeper as the degree of prematurity increases. This helps us to understand an observation made in Fig. 6a, namely that the exponent γ becomes higher nearer to birth. This is indeed consistent with the fact that shortly after birth a large percentage of the deaths are due to prematurity which, as seen above, is characterized by high values of γ.

Fig. 7c and Table 4 give the relationship between the index of maturity and the exponent γ for several sets of data. The prematurity index used for the horizontal axis of Fig. 7c was defined as: $p =$ (gestational age at birth)/(9 months). It

Table 4
Relationship between the exponent γ and the gestational age p.
Source: (i) Switzerland: Swiss Federal Office of Statistics, Taux de mortalité périnatale et infantile selon l'âge gestationnel [Perinatal and infant death rates as a function of gestational age]. (ii) Norway: Swamy et al. [27]. (iii) England and Wales: Office of National Statistics (ONS). Child mortality statistics. Table 6: Live births, stillbirth, and linked infant deaths: ONS case groups and birthweights. (iv) European countries: European Perinatal Health Report 2010 [28].

$\gamma = -ap + b$	a	b
Switzerland, 2007–2013	2.0 ± 0.2	2.90 ± 0.02
Norway, 1967–1988	2.8 ± 0.1	3.93 ± 0.01
England and Wales, 1999–2013	1.7 ± 0.5	2.40 ± 0.05
European countries, 2010	1.9 ± 0.6	2.01 ± 0.06
Average	2.1 ± 0.17	2.81 ± 0.02

Notes: γ refers to the exponent in the relationship: death rate $\sim 1/\text{age}^{\gamma}$. Once a is known to be close to 2, a value of b close to 3 is expected because for term births: $p = 1 \Rightarrow b = \gamma + a \sim 1 + 2$. The line labeled "European countries" includes all European countries for which data are available and whose annual number of births exceeds 50,000; the sample comprises the following countries: Austria, Czech Republic, Denmark, England and Wales, Finland, Norway, Poland, Romania, Switzerland; it can be observed that unfortunately such data are not available for several major European countries such as France, Germany, Italy or Spain.

can be seen that the various relationships are fairly close; this is all the more remarkable on account of the fact that there are big discrepancies in the underlying death rates. For instance, for babies born at 22–27 weeks, the neonatal (0–28 d) mortality rate is 234 in Norway but 430 in Switzerland and the postneonatal mortality (28–365 d) is 98 in Romania but 22 in Switzerland.

5.5. Extension of the relationship between prematurity and γ

In the previous subsection we have seen that there is a connection between the accumulation of deficits and the selection rate measured by the parameter γ. The phenomenon of prematurity is not confined to the human species.

> *As a conjecture we propose that the validity of the relationship $\gamma = -ap + b$ extends to other mammalian species; the parameters a and b are expected to be close to 2 and 3 respectively.*

Through observations made on farm animals it should be possible to determine if this conjecture is correct. It can be observed that artificial insemination which is common practice in animal breeding makes the determination of the gestational age quite easy and precise.

In a general way, for living organisms the postnatal selection process has two facets: (i) A pure selection process through which fatal defects are eliminated through the death of the corresponding individuals. (ii) An adaptive process through which non-fatal defects (e.g. non-mature organs) may get corrected. For a better understanding one would of course wish to separate these effects. This may be done fairly easily for technical devices. Let us illustrate this point through the example of the light bubbles discussed earlier.

In order to manufacture thin tungsten wires one needs to heat and stretch the metal. In such a process annealing plays a key-role. In annealing, that is to say slow cooling, atoms migrate to their appropriate positions in the crystal lattice and therefore the number of dislocations decreases which results in a wire with a more uniform effective section. In other words, the duration of annealing plays the same role as the length of pregnancy. The shorter is annealing, the higher is the density of defects and the more rapid will be the lamp's failure. Will there be an adaptive process? Probably not, except if the lamp has a special device (found in some expensive light bulbs) through which it is started and turned off progressively. In this way there will be an annealing process every time the lamp is turned off.

The following sections are devoted to non-human living organisms. We will proceed from the cases that are closest to humans such as farm mammals and primates to more remote cases such as plants and trees.

6. Farm mammals: piglets and lambs

For farmers early deaths of farm animals represent a substantial economic loss. In the United States and Australia about 16% of the new born piglets died before reaching the age of one month. This led to studies done by the Departments of Agriculture whose main aim was to understand the causes of death.[10]

As a by-product these studies gave death rates by age which allowed us to check whether they follow a power law or not. The power law shape was confirmed (Fig. 8) with exponents summarized in Table 5.

Apart from the studies concerning farm animals there are few sources from which one can get accurate data about infant mortality for animals. One other important source consists in the records from zoological gardens. It is this source that will be used in the two following subsections.

[10] For instance the study about lamb mortality revealed that overcrowded pens lead to a high mortality due to the ewes laying on their lambs. This cause of death represented 25% of the deaths which occurred from day 1 to day 7.

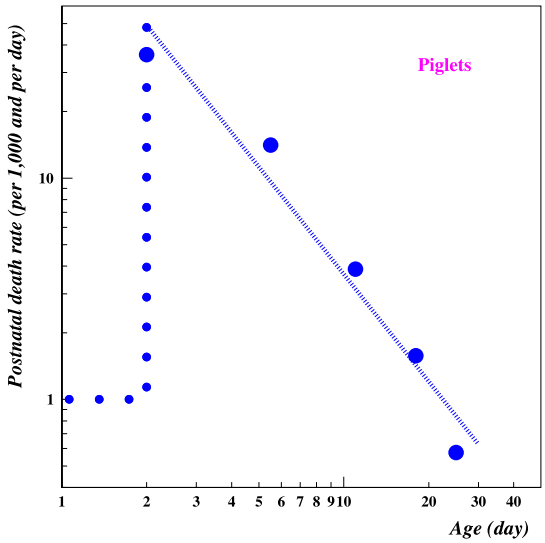

Fig. 8. Postnatal mortality of piglets. The slope of the regression line is -1.6 ± 0.4. The data are from a study of the US "National Animal Health Monitoring System" (NAHMS) for 1990. The dotted part of the curve is a schematic representation of the spike (age 0 and negative ages can of course not be displayed on a log-scale). As fetal death rates of piglets are not well documented the level was set to a value similar to late fetal rate in humans.
Source: US Dept of Agriculture 1992 [29].

Table 5
Neonatal mortality of piglets and lambs.
Source: Piglets: US Dept of Agriculture 1992 [29], Glastonbury [30]. Lambs: Berger [31].

Case	Number of age intervals	Exponent of power law	Correlation (log–log)
Piglets			
USA, 1992	5	1.60 ± 0.40	0.98
Australia, 1976, including still-births	8	1.20 ± 0.25	0.97
Australia, 1976, excluding still-births	8	0.98 ± 0.34	0.92
Lambs			
USA, 1997	10	1.00 ± 0.30	0.92

Notes: Neonatal and pre-weaning correspond approximately to the same period of time after birth, namely about 30 days. All three surveys are large scale studies involving several thousand births.

7. Primates

Primates are one of the main attractions of zoos and it is not surprising therefore that major zoological gardens have large populations of species of this taxon.[11] Primates have the additional interest of being a kind of stepping-stone between humans and other, more distant, mammals. For all these reasons primates warrant a close look.

7.1. Postnatal death rates for various species of primates

As the detailed data that we needed were not available[12] one of the authors (V.P.) conducted a special investigation [34] using registers from the London Zoo called *Daily Occurrences* (the complete title is: Zoological Society of London Archives: Daily Occurrences [35]). These records are available at the Archives of the Zoological Society of London. These data record the arrivals and departures of animals on a daily basis. It can be added that in a general way primates get particularly close attention.

Fig. 9a shows that all small primates investigated follow similar power laws with an exponent which, on average is equal to $\gamma = 1.24 \pm 0.2$ (see Fig. 9b).

11 A taxon is a group of species with some common characteristics. More details can be found in Ref. [32, p. 767–769].

12 Later on we will use some data published in Ref. [33]; these data have the advantage of being available for a broad range of species but they have a poor time resolution in the sense that they give infant mortality data only for the ages of one week and one year.

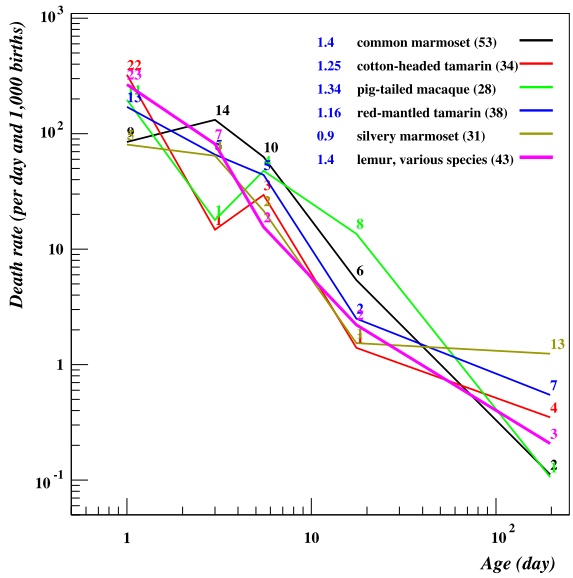

Fig. 9a. Infant mortality rates of primates. The numbers which follow the names of the species give the size of the subgroup of individuals whose birth and death dates were recorded in the *Daily Occurrences* volumes of the London Zoo in the period 1970–2000. The numbers printed on the curves give the deaths in each age interval. Finally the numbers (in blue) which precede the species names are the exponents γ of the power law. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
Source: Archives of the Zoological Society of London. For more information about these data, please contact Ms. Violette Pouillard.

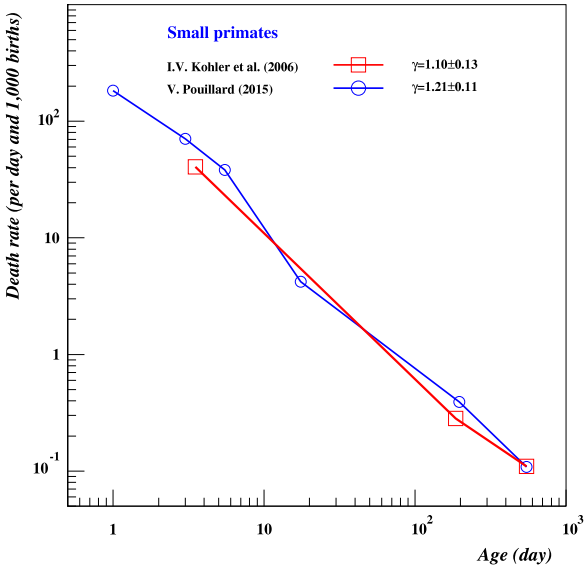

Fig. 9b. Infant mortality rates of primates. For the whole group of small primates the graph provides a comparison between two separate data sets. It is reassuring to see that they show a good agreement. The comparison provides a welcome confirmation of the data of Kohler et al. [33], a check that is all the more useful due to the fact that they consist in only 3 data points of which only 2 can be considered as pertaining to infant mortality.
Source: Archives of the Zoological Society of London, Kohler et al. [33].

7.2. Consistency of separate data sets

In physics it is the rule that an experiment done by one team is repeated[13] and checked by one or several other researchers. In biology this is fairly rare and in the social sciences it is exceedingly rare. Here, however, we have the

[13] Yet, never *exactly* in the same way in the sense that the devices and measurement methods are not the same. Taken together, these experiments will show what are the necessary and sufficient conditions for observing the effect under consideration. In other words, these semi-repetitions define the envelop of parameters within which the effect occurs.

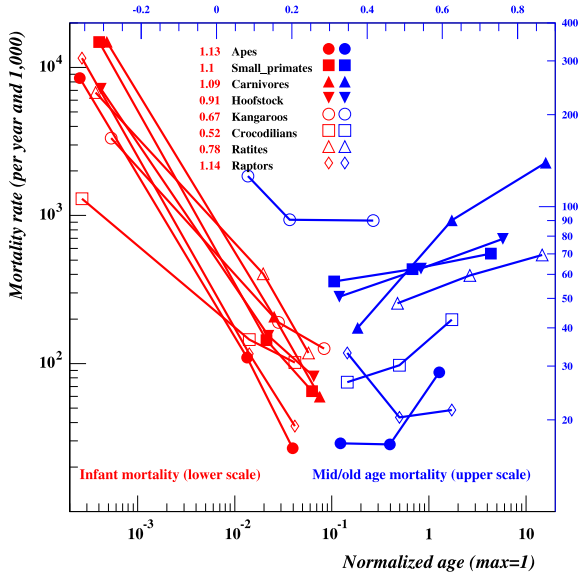

Fig. 10. Infant mortality rates versus mid-age rates. By showing that across various species the infant death rates are much more orderly than the late-age death rates, this graph gives a strong incentive for studying the former. The scales for the late age rates are given by the axis on the right-hand side and the top horizontal axis. The numbers which precede the names of the subgroups are the exponents γ of the power law for the infant death rates. Despite the fact that there are only 3 data points, thanks to the good correlations, the error bars (at probability level 0.95) are only about $\pm 8\%$ on average.
Source: Source of the data: Kohler et al. [33].

opportunity to carry out such a comparison for the case of small primates. The two observations (shown in Fig. 9b) were performed independently and rely on data that differ in several respects.

- The time periods are not the same: 1970–2000 versus 1998–2003. This has an incidence on the manner of recording the data. During the 1970–2000 period the data were mostly recorded by hand whereas 1998–2003 already belongs to the digital era.
- The zoos are not the same: London zoo versus various zoos in North America and Europe. Not only are the animals not the same but even the species are not exactly the same although there is of course a broad overlap. The species belonging to the "small primate" category considered by Kohler et al. are listed on p. 416 of their paper.
- Finally, the sources of the data are not the same: *Daily Occurrences* of the London zoo versus "International Species Information System" (ISIS). We will say more about ISIS in a moment.

In short, the quasi-coincidence of the two data sets is a good testimony of the robustness of this kind of observations. In particular it shows that despite limited data points the observations collected compiled by Kohler et al. [33] reflect fairly well the overall shape of the infant mortality rates as a function of age.

A second confirmation can be mentioned. In a paper by Hird et al. [36] we are told that "the neonatal mortality rate was 10.8%, and the post-neonatal mortality rate (deaths between 31 and 183 days) was 6.9%. These data allows us to compute γ. One gets: $\gamma = 1.06$ which is quite consistent with the previous results.

Finally, it must be mentioned that we also came across a paper whose results are inconsistent with the previous ones. The study by Shaughnessy et al. [37] gives the following distribution of deaths of *Macaca mulata* over the 4 first weeks after birth: $w_1 = 24\%$, $w_2 = 59\%$, $w_3 = 17\%$, $w_4 = 9.6\%$. The fact that $w_1 < w_2$ is in contradiction with all observations that we have reported so far. Secondly, although w_2, w_3, w_4 follow a power law, the corresponding exponent is equal to 2.6 which is much higher than the exponents seen so far. These data relied on births which occurred between 1966 and 1972 at the breeding colony of the "Litton Bionetics" company in Maryland. One is tempted to think that for some reason a number of early deaths went unrecorded.

8. Broader view of animals kept in zoos

The data set used in the paper by Kohler et al. [33] was not designed to study infant mortality. This is shown clearly by the fact already mentioned that there are only 3 (very distant) data points: 1 week, 1 year and 2 years. Without the control test performed in the previous subsection it would have been hazardous to use them to study postnatal mortality. However, encouraged by the consistency seen for small primates we have considered other subgroups. The results are summarized in Fig. 10.

One can learn two things from this graph.

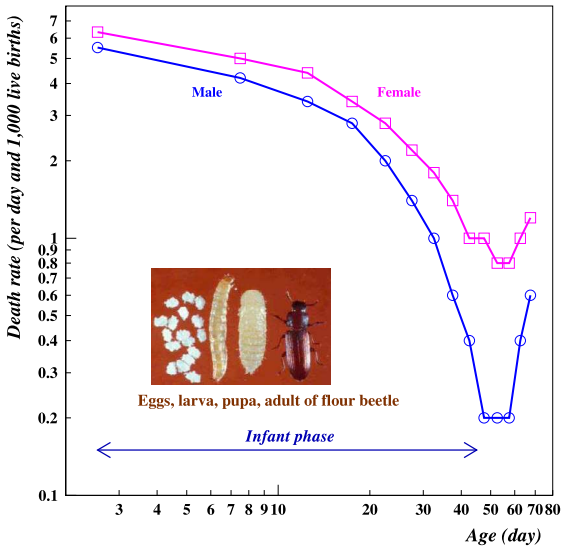

Fig. 11. Infant death rate of a flour beetle (*Tribolium confusum*). Unlike previous cases, this curve shows a very mild selection process in the 20 days following "birth". Actually, birth, here, means emergence from the pupal stage. This stage follows a previous life stage as a larva as illustrated in the inset. These prior selection processes may explain the slow decrease of the present death rate curve.
Source: Pearl et al. [38, p. 13–14].

(1) The infant death rates are much more regular and uniform than the late-age death rates (we come back to this point below). They are also much higher.
(2) With respect to the exponent γ, of the 8 subgroups there are two which emerge as quite different, namely kangaroos and crocodilians. The average of the 6 others is (with probability level 0.95): $\gamma_m = 1.02 \pm 0.12$. The case of the crocodilians is particularly spectacular because they have not only a lower slope but also a much lower overall death rate.

Why do the mid/old age rates show such a high dispersion? Two answers come to mind.

It may be due to the well known fact that aging processes are very diverse, as shown by the classification of survivorship curves into three types I, II, III, not to mention all kinds of intermediate types.

There may be a second reason. A look at the ISIS records shows that most of the animals do not spend their whole life in the same zoo; instead they are repeatedly loaned (or sold) by one zoo to another. This may be disturbing for the animals but it raises also a book keeping problem. In the paper by Kohler et al. (p. 429) it is reported that even highly visible animals such as gorillas "are assigned multiple identification numbers in various regions around the world". What occurs for gorillas is also likely to occur for other species. The only difference may be that for low profile species the inaccuracies in the records remain unnoticed.

9. Insects

Before becoming adult, insects go through several life stages: (i) embryo, (ii) larva, (iii) pupa and (iv) finally adult. According to our definition of the infant mortality we will consider only the adult stage, i.e. the stage which leads to sexual maturity. In each such transformations the insects are exposed to changing external situations. From caterpillar (larval form) to butterfly there is clearly a drastic change in environmental conditions. In other words, there are several filtration processes which may be independent or inter-dependent.

9.1. Why is there only a slight screening?

We consider the case of a beetle, more specifically *Tribolium confusum* whose age-specific death rate is shown in Fig. 11. Compared with human mortality (e.g. Fig. 5a), this graph shows a very mild screening process. Between day 0 and 10 the death rate is divided by 1.5, whereas in Fig. 5a it is divided by 20 for the 19th century curve (and much more for the 2004–2013 curve). Needless to say, this figure underestimates the real ratio because, taking into account the respective life spans, one day of a beetle represents about 20 days of a human. So the ratio is actually a factor 260. How can one explain such a huge difference?

A likely explanation is that here time zero does not represent a real birth but rather the transition from the pupa to the adult stage. This transition was preceded by two others: from egg to larva and from larva to pupa. In the case of *Tribolium confusum* the larva, pupa and adult live in the same environment, namely places where flour or similar kinds of food are stored. Thus, for most of its functions such as breathing, swallowing, digesting the selection processes at work for larva have already paved the way for the survival of the adult. In other words, this transition requires few new functionalities. It is true

that in terms of shape the pupa phase involves major transformations some of which may be defective but this may result in the failure of the adult to emerge, an event which is not taken into account in postnatal mortality.

9.2. Testable predictions

How can this explanation possibly be tested? It suggests three predictions, each of which can be checked.

(1) One should see a similar age-specific curve in related species, for instance *Tribolium castaneum*.
(2) One should see a more drastic selection process in the transition from eggs to larvae. At this point it is difficult to predict if it will be a power law with respect to age but it should be more severe than above. The larva stage lasts about 16 days (at a temperature of 35 °C) and within this time interval one would expect a division of the death rate by a factor of the order of 5 or more.
(3) For species for which there is a marked difference between the larva and the adult, e.g. Caterpillar versus butterfly, one would expect a stronger selection process than for *Tribolium confusum*.

9.3. Infant versus late age mortality

Incidentally, it can be noted that (as documented in Pearl's paper) the survival curves of males and females in late age are very different. The 50% proportion is reached for 170 versus 210 days respectively whereas the 10% proportion is reached for 410 versus 375 days respectively. In contrast during their infant phase, male and female rates display parallel changes. This, once again, illustrates the fact that infant mortality is "simpler" (in the sense of being less affected by exogenous factors) than aging.

10. Plants and trees

In its principle, the process which leads from a tiny embryo to a seedling does not much differ from what we see in the growth of fish or mammal embryos. It is marked by similar steps of division and differentiation. For many (yet not all) plants the growth of the embryo continues for a given time interval after the formation of the seed. This means that germination cannot occur immediately. If, for some reason, this delay is not respected the seedling will face a prematurity problem just as in human premature births.

10.1. Yolk sac effect in plants

In the time interval between germination and formation of roots and leaves, the nutrients contained in the seed play the same role as the yolk sac for fish larvae.[14] This parallelism is described in the following lexicon.

Lexicon of development terms for fish versus plants

Fish:	embryo	egg	hatching	yolk sac	larva	young adult
Plant:	embryo	seed	germination	endosperm cotyledon	seedling	juvenile

Notes: Despite the different vocabulary there is a strong parallelism between the successive phases. The two terms given for the yolk sac phase reflect the difference between plants whose seed germinate under or over the surface of the soil (they are called hypogeal or epigeal species).

Therefore one would not expect the main death spike to occur right at germination but rather when the nutrients contained in the seeds are exhausted. The accuracy of the data that we could find so far is too low to allow this prediction to be tested.

In many respects plants are living organisms that are "simpler" than animals. However, because their movements are more limited a major difficulty is to define the moment of death. One possibility is to define it through the termination of growth. Thus, if the small radicle which emerges from a seed stops growing one may consider that development has been interrupted and take this is as equivalent to death.

Just as an illustration of what can go wrong one can mention the following observation. After the stem has emerged from the seed, the plant must be able to distinguish upward versus downward directions. This reaction to gravity is known as gravitotrophism. It turns out that for a small fraction (of the order of 1%) of the seeds this mechanism does not work with the result that their stems grow downward into the ground instead of upward. As such a development forbids photosynthesis it leads eventually to growth termination.

[14] For instance, according to Hanley et al. [39], this phase lasts about 11 days for sunflowers (*Helianthus annuus*) and 12 days for pea (*Pisum sativum*). Thus, one should observe a mortality spike in the interval 10–15 d after germination. Its amplitude may be small (perhaps about 1%) but should be visible on samples of 1000 seeds or more.

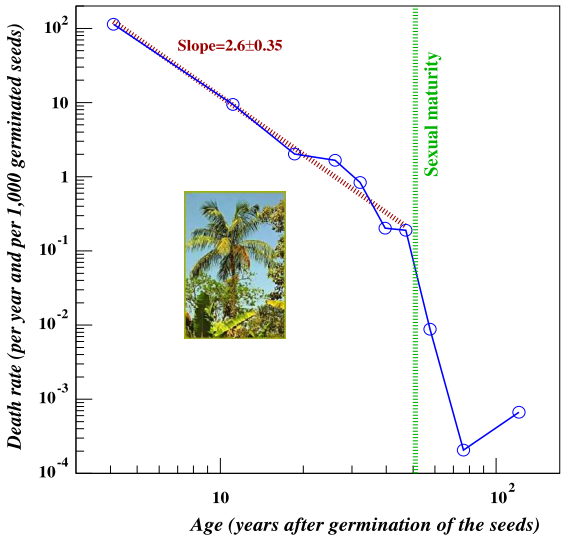

Fig. 12. Infant death rate of a palm tree. This palm tree (*Euterpe globosa*) lives about 200 years and reaches a height of 20 m. The observations were made in Puerto Rico. Instead of being close to one as for most other cases, it is close to 2.5. At this point we do not know why.
Source: Valen [40, p. 263].

10.2. Field observation of palm trees

Very few life tables have been set up for plants. In 1975, Valen wrote "Although the study of survivorship [curves] has been an important part of animal ecology for 30 or 40 years, there are few studies on plants". The same observation still basically holds in 2015.

The results for the palm trees *Enterpe globosa* studied by Valen are summarized in the graph of Fig. 12. Two features are of particular interest.

- As for humans, the age interval during which the death rate decreases roughly coincides with the period before sexual maturity.
- The death rate follows a power law fairly accurately. The $(\log t, \log \mu_b)$ correlation is 0.998. Yet, with an exponent equal to $\gamma = 2.6 \pm 0.4$ the decrease is almost three times steeper than in previous cases. Is this property shared by other big trees? Needless to say, in order to answer this question one would need experiments made in greenhouses under controlled conditions rather than field observations. A further discussion can be found in Appendix C of the version of the paper available on arXiv.

11. Conclusion

This paper is a contribution to a fairly new field in relation with biodemography and biosociology. The following landmark papers give an idea of its scope: Altshuler et al. [41], Anonymous [42,43], Li et al. [44], Wang et al. [45]. Here we studied how a group of living organisms react to an abrupt and drastic change in living conditions.

11.1. Main results

What we learned in this paper can be summarized in the following observations.

(1) **General** (a) Infant mortality (in the sense of being a phase during which the death rate *decreases*) is an ubiquitous phenomenon in living organisms. It was shown to exist in mammals, fish and plants. (b) In all cases for which appropriate data could be found, we have seen that there is a hyperbolic power law decrease (i.e. $1/t^\gamma$ starting in the vicinity of $t = 0$) of the death rate. (c) The exponent γ is usually close to one. The plant instances in which we found exponents as high as 3 need to be confirmed by observations under controlled conditions (i.e. no predators, appropriate supply of water, and so on). (d) For most animal species it appears that there is much more regularity in infant death patterns than in old-age death patterns.

(2) **Yolk sac larvae** The *Transient Shock* conjecture offered predictions regarding the existence and timing of death rate spikes and these predictions were confirmed by observation.

(3) **Humans** (a) What we called the hyperbolic power law starts to hold in the second hour following birth. (b) The fact that in the hours following birth γ is of the order of 2 rather than 1 can be explained by the effect of premature birth. (c) There is a negative correlation between γ and the mortality rate in the 10–14 age group. (d) If one makes a distinction between different causes of death, most of them follow a power law, yet not necessarily with the same exponent. For instance, death due to infectious disease is characterized by an exponent of 0.69 instead of 0.97 for the all-causes curve. (e) There is a linear relationship between the index of prematurity and the exponent γ.
(4) **Primates** For primates (and more generally for any mammals for which data are available) one observes the same infant mortality pattern than for humans.
(5) **Multi-stage organisms** For insects in their adult life stage the mortality rate does also decrease with age but it does not follow a power law. This may be due to the fact that this stage was preceded by several selection processes.

11.2. Origin of the hyperbolic power law of mortality decrease

There is one question for which we have no answer so far, namely how can one explain the origin of the power law decrease observed for the infant mortality rate, a feature that is common to so many species. Two types of explanation come to mind.

- First, there is the filtering effect of defective individuals that we have already discussed (explanation of type A). This can be referred to as a static explanation in the sense that it does not assume any transformation taking place in individual organisms. A tentative model is outlined in the arXiv version of the present paper.
- Then, there is a dynamic explanation in which one assumes an improvement of the immune system in the course of time. It is the immune system which ensures survival in later life; in addition we know that vaccination actually works; therefore it seems natural to assume that the effectiveness of the immune system improves as individuals interact with the outside world and experience in small doses various "nasty" microorganisms which trigger the emergence of antibodies. We also know that this process takes time for at birth the immune system is largely under-developed and newborn individuals have to rely on what is called passive immunity, namely maternal antibodies transmitted to them.

In order to substantiate the second explanation one would need age-specific data for vaccination effectiveness.[15] An alternative metric would be to measure the antibody concentration in the blood of animals (which were not subject to vaccination) as a function of their age.

11.3. Agenda for future research

We wish to stress the fact that for various organisms such as plants, bacteria, microorganisms, insects it would be relatively easy to set up infant mortality experiments because, in contrast with the study of aging, the observation time can be much shorter. Our plan is to build a chain of systems which starts from the simplest (e.g. technical devices, plants, primitive animals such as *C. elegans*) and progressively embraces more complicated systems. Such a program was already considered at the end of the 19th century by Espinas [46]. As a matter of fact, at that time it seemed to be a fairly natural idea to complement sociological investigations with studies about other living organisms. Nowadays such an approach has become fairly uncommon.

As an example of their interest, such experiments may shed new light on the hierarchical complexity structure of living organisms. Here again a parallel with technical systems may help to explain this idea.

A modern airliner is made up of many functional components (wings, engine, computer and so on). These components comprise large numbers of smaller elements (screws, electronic chips and so on). In the last step of the building process the functional components are put together on the assembly line. Finally, tests are performed with the purpose of detecting possible defects.

In principle deficiencies may occur at the three levels: small elements, components, assembly line. However, observation shows that the small elements have a very low defect rate. This is fairly understandable because they are produced through standard manufacturing processes and are fairly easy to control. Similarly cell division seems to be a very reliable generation process. There are some $N = 3 \times 10^{13}$ cells in the human body ([47], note that a large proportion of them are red blood cells). On average 1% of them must be replaced every day. This raises the question of what defect rate is acceptable. The answer is certainly highly organ dependent. A defect rate of 1 per 1000 may be quite acceptable for red blood cells but may not allow an organ such as an eye to work properly. Needless to say, a component may be defective even though all its elements are good. Similarly, if mistakes are made at the assembly line level the aircraft may be defective even though all its components are flawless.

A careful analysis of postnatal death rates across various species may give useful information about this multi-level organization.

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[15] It is defined as $E = 1 - I_v/I_{nv}$ where I_v is the incidence of the disease under consideration (e.g. influenza) in the vaccinated population and I_{nv} is the incidence in the population that is not vaccinated. If not a single vaccinated person gets the disease, E will be equal to 1. On the contrary, if $I_n = I_{nv}$ which means that the vaccine has no effect, one gets $E = 0$.

Definition of standard versus postnatal death rates

t : age ($u = \log t$)
$s(t)$: survivors at age t
$\mu(t)$: standard death rate $\quad \mu = |(1/s)(ds/dt)| \quad y = \log \mu$
$\mu_b(t)$: postnatal death rate $\quad \mu_b = |(1/s_0)(ds/dt)| \quad y_b = \log \mu_b$

Fig. A.1. Standard death rate (μ) and postnatal death rate (μ_b). The logarithms of the death rates, y, y_b, are used below in log–log plots.

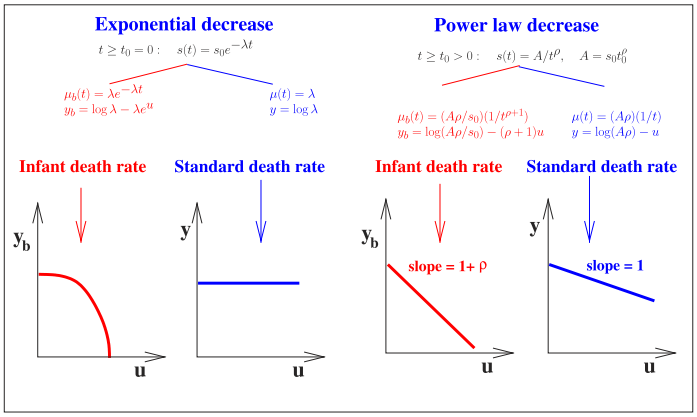

Fig. A.2. Log of infant death rate ($y_b = \log \mu_b$) and of standard death rate ($y = \log \mu$) as a function of $u = \log t$. The shapes are shown in two typical cases: exponential and power law decrease of $s(t)$. As expected, $\mu(t)$ falls off slower than $\mu_b(t)$ for the simple reason that for any $t > 0$, the denominator $s(t)$ is smaller than s_0.

Acknowledgments

We would like to thank Prof. Thomas Kirkwood for his encouragement. His paper on the Gompertz law [48] was a source of inspiration and a guide in conducting the present study. We are grateful to Mr. Rudi Ripman Seim for sending us mortality data for salmon alevins. Finally, we wish to mention that the vigilant reading of a reviewer helped us to significantly improve the paper.

Appendix. Infant mortality data

In this Appendix we first discuss the definitions of mortality rates. Then, we explain the conditions under which infant mortality should be measured. Finally, we give some indications about possible data sources.

A.1. Definitions

In statistical sources death rates are computed in two different ways depending on whether they concern infant mortality (defined in the broad way of post-natal mortality used in this paper) or not. The definitions are recalled in Fig. A.1. This can create a good deal of confusion because a given survival function will lead to different mortality patterns depending on which definition one uses. This is illustrated in Fig. A.2 for an exponential and a power law fall.

For a power law, the curves of μ and μ_b have same shape but different exponents. Intuitively, this is quite understandable because for a slow decrease of $s(t)$ one has: $\mu_b \simeq \mu$; in most cases examined in this paper $\mu_b \sim 1/t$ which means that ρ is close to zero and $s(t)$ almost level during the infant mortality phase.

In the next subsection we explain that this dual definition is a consequence of how the data are recorded.

A.2. Reason of the dual definition of mortality rates

The infant mortality definition is usually used for newborns under one year. We have seen that the infant mortality phase (i.e. falling mortality) in fact extends until 10 years. So, why was a one year threshold selected?

The (standard) age-specific death rate is a ratio of two numbers: the numerator is the number of deaths which occurred in one year in a given age-group (e.g. 15–19) whereas the denominator is the number of (living) people in this age-group. The first number is provided by the death certificates while the second is provided by the census.

Now, let us try to compute the death rate of newborn babies between 1 and 2 months of age with the previous definition. There is no problem for the numerator because death certificates give the date and time of birth and death which defines

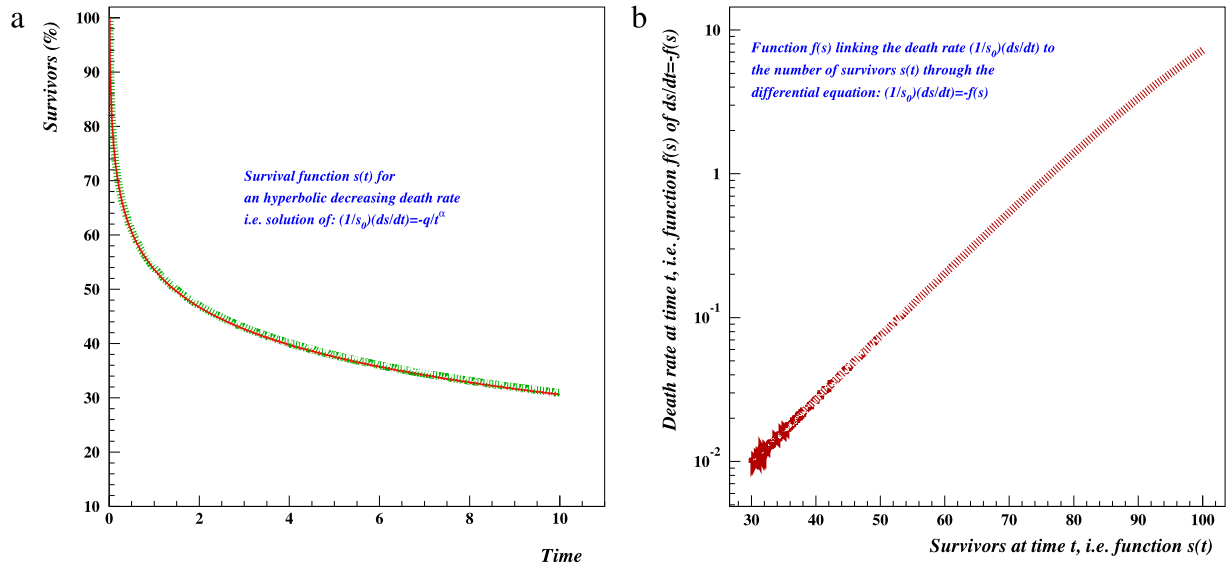

Fig. A.3. (a, b) Shape of survival curve and differential equation. Left: Solution $s(t)$ of $(1/s_0)(\mathrm{d}s/\mathrm{d}t) = -q/t^\alpha$ for the initial condition $(t = t_1 > 0, s = s_0)$ $(q = 0.1,\ t_1 = 0.5,\ s_0 = 10)$. Here $\alpha = 1$ (the shape is of course similar for other values of α around 1) in which case the analytical solution is: $s(t) = s_0 - s_0 q \ln(t/t_1)$ (dotted line in green); the thin solid line (in red and almost superposed on the green line) was computed by numerical integration. Right: Horizontal axis: s, vertical axis: μ_b. The semi-log graph shows that the function $f(s)$ in the homogeneous differential equation $(1/s_0)(\mathrm{d}s/\mathrm{d}t) = -f(s)$ is basically an exponential (if one excepts the slight saturation effect visible in the upper right corner); this can also be seen analytically but is cumbersome to write and not very transparent. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

the age at death with an accuracy of up to one hour. For the denominator one would need the number of living newborns of that age. However, this number will have strong monthly fluctuations because of the seasonal variability of the birth rate. In other words, this definition cannot be used and must therefore be replaced by another. In order to get a rate the simplest way is to divide the deaths by the total number of live births in the same year.

Over one year of age one can in principle use age-group census data but it should be observed that, specially for young ages, the definition of an age group requires a number of conventions. For instance, in order to compute ages one must define (somewhat arbitrarily) a specific census date even though census operations may take several months. This shows that the infant death rate is a simpler and clearer concept than the standard death rate.

These definitions have another noteworthy implication. In the computation of an infant death rate one needs to take into account the length of the age interval under consideration. Thus, to get the rate per day for the age 1–2 months one will divide the number of deaths not only by the live births but also by the length of the interval expressed in days (i.e. 30 days). On the contrary, in computing the (standard) death rate for the 14–19 age group the length of the interval (i.e. 5 years) is irrelevant because the denominator refers to the same age group.

A.3. Homogeneous differential equation giving hyperbolic death rates

One may wonder what is the form of the differential equation $(1/s_0)(\mathrm{d}s/\mathrm{d}t) = -f(s)$ which leads to the kind of death rates seen in this paper, that is to say decreasing hyperbolically. Fig. A.3a, b shows that, basically, $f(s)$ must be an increasing exponential function.

A.4. In search of "natural" infant mortality rates

For humans as well as other living organisms infant death rates may be inflated by temporary diseases or epidemics. In addition, for species reared in laboratories or zoos death rates may be amplified because the living conditions are not good enough. For animals kept in zoos this can just be the result of a lack of space, whereas for small species (e.g. fish larvae) it may be due to the fact that we do not well know how to feed them.

On the other hand, especially in past decades, human infant mortality has been drastically reduced thanks to medical progress. This led to the survival of babies afflicted by malformations who would not have been viable otherwise.

This leads to the following practical rules for data selection.

- We discard all data collected in the field that is to say in conditions (e.g. bad weather, predators) which cannot be controlled.
- We discard the data recorded in populations in which the spread of a disease or other inappropriate conditions have led to high mortality rates. For instance, there are recurrent disease outbreaks in hatcheries, fisheries or zoos.

A.5. Suggested experiments

Although the data reported in the present paper cover a wide range of species, they are all complex multicellular organisms. One would need empirical evidence for "simpler" organisms. What organisms would be particularly appropriate?

Caenorhabditis elegans, a little worm about 1 mm long which has only about one thousand cells, would seem to be a good candidate. The adults lay eggs which hatch after a few hours. The death rate in the 12 h following hatching is about 4% per day; it falls to about 1.5% per day in the following 16 h [49, p. 24], [50, p. 26]. At sexual maturity it is at a minimum level of 1% from which it then increases exponentially over a period of 30 days at the end of which it reaches 100%.

We hope that the publication of the present paper will lead other researchers to perform accurate measurements of infant mortality rates. We are confident that once more observations become available for a wide range of cases a clearer understanding will emerge.

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