Deciphering infant mortality. Part 1: empirical evidence

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Abstract  This paper is not (or at least not only) about human infant mortality. In line with reliability theory, “infant” will refer here 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 which falls within the field of application of the Transient Shock (TS) conjecture put forward in Richmond et al. (2016c). This conjecture provides predictions about the timing and shape of the death rate peak. (i) It says that there will be a death rate spike whenever external conditions change abruptly and drastically. (ii) It predicts that after a steep rising there will be a much longer hyperbolic relaxation process.

These predictions can be tested by considering living organisms for which birth is a multi-step process. Thus, for fish there are three states: egg, yolk-sac phase, young adult. 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 high accuracy Swiss statistics which give postnatal death rates from one hour after birth up to the age of 10 years. It turns out that since the 19th century despite a great overall reduction in infant mortality, the shape of the age-specific death rate has remained basically unchanged. This hyperbolic pattern is not specific to humans. It can also be found in small primates as recorded in the archives of zoological gardens.

Our ultimate objective is to set up a chain of cases which starts from simple systems and then moves up step by step to more complex organisms. The cases discussed here can be seen as initial landmarks.

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Introduction

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 pattern and the power-law death spike pattern. Whereas the first pattern is fairly well understood, the second raises questions for which we have no complete answer so far. However, we believe that, taken together, these patterns give us new insight into the mechanisms of infant mortality.

Infant mortality seen as a transition death spike

In a previous paper (Richmond et al. 2016c) it was suggested that any major change in the conditions under which a system operates will bring about an increased failure rate. In the same paper this mechanism 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 a state 1 and a state 2, one observes a transient mortality spike during which the death rate is temporarily multiplied by a factor of 2 or 3 and even much more in the case of the birth transition.

As explained in Richmond et al. (2016c), the observed death spikes have a simple interpretation as being a selection process. The systems which were adapted to state 1 but are not adapted to state 2 are eliminated; this results in a death rate increase. Although this explanation is satisfactory at a qualitative level, at a quantitative level there remain questions such as the following.

- Can one establish a connection between the characteristics of the transition on the one hand and and the amplitude of the mortality spike on the other hand?
- 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 old-age death rates which are very species dependent, the decrease of infant death rates is fairly uniform: $\mu = 1/t^\gamma$, where the exponent $\gamma$ is of the order of one.

Most of the time expression 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”. 

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1 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 Richmond et al. (2016a) and continued in Richmond et al. (2016b,c).
Fig. 1a shows postnatal mortality in semi-log scale. It gives a good idea of the shape of the spike.

![Graph showing mortality rate over time](image)

**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 (1947 p. 574-575).*

However, in a semi-log representation one cannot identify the curve as a power law and even more importantly one cannot explore a broad time interval. This is done in Fig. 1b.

It shows three phases: (i) The hyperbolic power law decrease. (ii) A transition zone where the death rate is fluctuating without any definite trend. (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 1,000 per 1,000 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 (Bußenstein 2008). This does not imply that the aging phase does not exist but rather that it is short and therefore that the increase of the death rate is very steep.

**Outline**
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. At present the phenomenon of infant mortality is a black box in the sense that we do not know its mechanisms. As a first step, we need to identify the key variables. Once we have got a better understanding it will be time to express it in mathematical terms. The paper will proceed as follows.

1) In the next section we focus on an example of infant mortality which is quite revealing because it does not occur at birth (in this case hatching) but at the end of the yolk-sac phase.

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 should be meant by the expression “lethal defect”. It will be seen that in this case one can expect a deterministic one-to-one correspondence between an initial defect that is present in a sample of items and the age-specific failure rate.

(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.

**Gompertz’s law versus infant mortality**

Gompertz’s law consists in the fact 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 1,000 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 approximately to the age of 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. The subsequent phase marked by an increasing failure rate is called “wear out” phase.

**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 (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.

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

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3Other 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           | +      | +              | -            | +                      | 3            |
| Bird        | egg         | → newborn           | -      | +              | -            | -                      | 1.5          |
| Fish        | egg         | → larva + yolk sac  | -      | -              | +            | -                      | 0            |
| Fish        | larva + yolk sac | → larva (no yolk)  | -      | -              | +            | -                      | 1            |
| C. elegans  | egg         | → larva             | +      | +              | +            | -                      | 2            |
| Plant       | seed        | → seedling          | +      | +              | +            | -                      | 2.5          |

Notes: The – sign 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.

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.

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 a better case.

Yolk-sac mortality spike 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 insert of Fig. 2a shows such a yolk-sac for salmons, a case in which it is particularly big. 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 functionalities fail the fish will die from starvation within a few days. Thus, one expects a mortality spike in the days following the end of the yolk-sac phase.

It is interesting to observe that whereas this excess mortality has been commonly observed it was often attributed to a number of special reasons (cannibalism, unexpected changes in tank conditions) without real awareness of the underlying reason.

**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 which coincides with the end of the yolk sac phase as indeed shown by the starvation curve.

A similar effect can be found in the data published by a team led by Susana Garrido (2015) 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 decrease between \( t = 0 \) and 5 days, followed by 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.

**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 would be the larvae of salmons for in this case the yolk sac stage lasts about 60 days. At that late age the death spike should be particularly visible and clear. For the present investigation one needs daily data for populations reared in laboratory conditions. We can offer the prediction that whenever appropriate data become available the death rate of salmons should show a peak around the age of 60 days. Let us hope the present paper may encourage the publication of such data.

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

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4 As illustrations one can mention the papers by Lasker et al. (1970) and Garrido et al. (2015).
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. (2001) 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. (2000) of particular interest is the fact that they rely on samples of 2,500 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 of 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 author 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.

**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. It gives also two examples for which appropriate observations have not yet been made (at least to our best knowledge).
Gompertz’s law was discovered in 1825. Benjamin Gompertz was involved in the business of life insurance. That comes hardly as a surprise because Gompertz’s law has an obvious usefulness for various forms of life insurances. Ever since it was discovered in 1825 the study of Gompertz’s law has attracted considerable attention. In contrast, the study of infant mortality was fairly neglected. A testimony of this neglect can be found in a paper by Raymond Pearl and his collaborators (1941). 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 curves of *Tribolium* differ completely from human curves.

The little interest for infant mortality curves can be attributed mainly 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 that 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 for human populations hour-by-hour postnatal death rate data are
Table 2 Testing predictions about larvae mortality peaks.

| Species                  | Number of organisms $n$ | Hatching rate [%] | End of yolk phase [day] | Predicted interval of death rate spike [day/hour] | Test of prediction through observation | Reference of data |
|--------------------------|-------------------------|-------------------|-------------------------|-----------------------------------------------|---------------------------------------|------------------|
| California anchovy       | 400                     | ?                 | 2                       | 4 – 7                                         | C                                     | Lasker et al. 1970 |
| European sardine         | 820                     | ?                 | 4                       | 5 – 8                                         | C                                     | Garrido et al. 2015 |
| Black Sea turbot         | 77,000                  | 74%               | 3                       | 4 – 6                                         | C, low accuracy                       | Sahin 2001       |
| Redfish                  | 800                     | 74%               | 10                      | 12 – 20                                      | C, low accuracy                       | Laurel et al. 2001 |
| Siberian sturgeon        | ~ 2,000                 | ?                 | 10                      | 9 – 12                                        | C                                     | Gisbert et al. 2000 |
| Zebra fish               | 12,000                  | 90%               | 8                       | 9 – 15                                        | C                                     | Cousin et al. 2016 |
| C. elegans               |                         | 98%               | No yolk                 | 0 – 1h                                        | Not yet done                          |                  |

Notes: “C” means that the prediction was confirmed. Incubation refers to the time between spawning and hatching. The times in the columns “End of” and “Predicted” are expressed in post-hatch days. The data given in the references were collected for various objectives (e.g. influence of type of food, ecotoxicology) which were quite different from the present purpose. Cases 1-6 correspond to fish whereas 7 refers to a 1mm-long worm. 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 by 5mn- or 10mn-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=Caenorhabditis elegans.

now available but such data are relatively recent.

(2) Whereas old-age mortality has not been substantially affected by medical progress, infant mortality has been drastically reduced over the past century. Around 1900 infant mortality during the first year of life was still of the order of 150 per 1,000; nowadays in most industrialized countries it has been reduced to around 3 per 1,000. 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. In other words, if we write the death rate as $A/t^\gamma$, the numerator $A$ has been divided by a large factor, the initial point has remained almost the same, and the exponent $\gamma$ has changed only slowly.

(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 (1980). Unfortunately, with such an option survivorship curves loose almost all significance. The reason is easy to underr-
stand. Many organisms, particularly insects and fishes, produce a large number of eggs of which many die within a short time. Under such conditions all data points for later life will be confined in a narrow range even if one uses a logarithmic scale.

**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 albeit with a different exponent. However, the rule is not valid for insects whose 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.

**From technical devices to biological systems**

**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 $\sigma_1$ of the filament is smaller than the average

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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.
section $\bar{\sigma}$. 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 the resistance per unit of length of the filament ($\rho$ is the resistivity of tungsten). A reduction in $\sigma$ will bring about a local increase in resistance, this in turn will produce a higher heat release and push up local 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 $\sigma_1$ 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, $\delta$, and the time to failure $\theta(\delta)$ of the device.

**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 $S_0 = 1,000$ 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 Itô 1980) 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 $\mu$ as a function of age. The death rate is defined as:

$$\mu(t) = \frac{1}{S_t} \left( \frac{S_{t+\Delta t} - S_t}{\Delta t} \right)$$

where $\Delta t$ denotes a given age interval.

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

$$\mu_b(t) = \frac{1}{S_0} \left( \frac{S_{t+\Delta t} - S_t}{\Delta t} \right)$$
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 Appendix A).

**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 $\sigma_1$ at one point. As a result they will have the same time to failure $\theta_1$ 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 $\sigma_1$ 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 et al. (2006) degrees of redundancy are assumed which are achieved by identical subsystems working in parallel. In other papers (e.g. Peleg et al. 1998) it is assumed that there is a whole range of failure effects, each one characterized by a specific distribution curve.

The light bulb example 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, it results 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 are also an exploration tool.

**Human infant mortality**

**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. 5a, b Postnatal death rates from 1 day to the age of 15 years. Left: Switzerland: Comparison of the two curves for the first 16 days of life suggests that the section of the curve for times shorter than 5 hours 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 4,000 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. Sources: Switzerland: The following website of the “Federal Office of Statistics”: [http://www.bfs.admin.ch/bfs/portal/fr/index/infothek/lexikon/lex2.html](http://www.bfs.admin.ch/bfs/portal/fr/index/infothek/lexikon/lex2.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. 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 that we discussed in the context of old age in Richmond et al. (2016a). Here, the exponent of the power law will become higher when the death rate at age 10-15 decreases. Historically, this is what happened in western countries during the 20th century. In the perspective of transversal
analysis the same effect tells us that $\gamma$ will be smaller in developing countries than in developed countries. Testing these predictions may become the purpose of a subsequent paper.

**How shortly after birth does the hyperbolic law start?**

Mathematically, an 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 hour.

![Graph](image-url)

**Fig. 6a,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 hours 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: $0.97 \pm 0.12$, malformations: $1.12 \pm 0.07$, infectious disease: $0.69 \pm 0.10$. 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.

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.

**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.

| 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.

Source: Swiss Federal Office of Statistics.

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 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:

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

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 $\gamma$ 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.

**Incidence of birthweight**

Previously we have seen that the exponent $\gamma$ was not really constant but was rather declining with age. This materialized in two ways (i) In Fig. 6a we saw a sharp bent after 7 hours; (ii) in Fig. 6b, because of the averaging process, the decline of $\gamma$ was
smaller but nevertheless visible.

Fig. 6a suggests that the change in $\gamma$ should be attributed to prematurity. Indeed, in the case of pre-term birth, not only is the death rate higher (which is hardly surprising) but also the $\gamma$ is higher. The reason is easy to understand. Ultimately, after a few years, all infants, whether preterm or not, will tend to have the same weight and same death rate. Such a convergence is already visible in Fig. 7a in spite of the fact that it covers only one year. On the other hand, the initial death rate level of pre-term babies is in proportion of their degree of prematurity. Thus, inevitably, the slope $\gamma$ must be higher for large initial death rates.

Fig. 7a,b  Incidence of low birthweight on death rates. Left: 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. Right: There is a linear relationship between the prematurity index $p$ and the exponent $\gamma$ which takes the form: $\gamma = -ap + b$, $a = 2.0 \pm 0.2$, $b = 2.90 \pm 0.02$. Source: Swiss Federal Office of Statistics.

Fig. 7b summarizes the relationship between prematurity and the exponent $\gamma$. The prematurity index used for the horizontal axis of Fig. 7b was defined as: $p = \text{gestational age at birth}/(9 \text{ months})$. Then, the regression line follows the equation:

$$\gamma = -ap + b, \quad a = 2.0 \pm 0.2, \quad b = 2.90 \pm 0.02$$

This relationship is defined with high accuracy as shown by the small size of the error bars for $a$ and $b$.

It would be really surprising that a relationship that holds with such accuracy for humans would not hold as well for other mammals. Of course, the coefficients $a$ and $b$ will not be exactly the same but one would expect a linear relationship to hold with good precision.
The next step would be to set up an experiment specially designed to observe this effect and to measure $a, b$ with good accuracy. Until this is done, we must rely on makeshift data extracted from experiments which were conducted for a completely different purpose. As an illustration, one can mention an experiment described in a paper by Price et al. (1972). In this paper the authors measured the death rate of monkeys ($Macaca mulatta, Macaca fascicularis, Macaca arctoides$) at age $0−8$ days and $8−30$ days for different classes of birthweights. As the number of births is not very large (only 91 for the 3 species) the data shown in the paper display large random fluctuations. By lumping together successive birthweight classes, keeping only two, the fluctuations could be reduced substantially. As a result, one gets a relationship similar to (1) with the following coefficients:

$$\gamma = -ap + b, \quad a = 4.3, \quad b = 5.8$$

There are probably substantial error bars for $a, b$ but they could not be estimated because the regression line is defined by two points only.

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.

**Farm mammals: piglets and lambs**

![Graph of postnatal mortality of piglets.](image)

**Fig. 8 Postnatal mortality of piglets.** The slope of the regression line is $-1.6\pm0.4$. 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.

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 death⁶.

![Table 4 Neonatal mortality of piglets and lambs](image)

| 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 thousands births. Sources: Piglets: US Dept of Agriculture 1992, Glastonbury 1976. Lambs: Berger 1997.

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 4.

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.

**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 taxon⁷. 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.

**Postnatal death rates for various species of primates**

As the detailed data that we needed were not available⁸ one of the authors (V.P.) conducted a special investigation using registers from the London Zoo called *Daily Occurrences*. 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.

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⁶For 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.

⁷A taxon is a group of species with some common characteristics. More details can be found in Campbell and Reece 2004, p. 767-769.

⁸Later on we will use some data published in Kohler et al. (2006); 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.
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 $\gamma = 1.24 \pm 0.2$.

**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 $\gamma$ of the power law. 

*Source: Archives of the Zoological Society of London. For more information about these data, please contact Ms. Violette Pouillard.*

**Consistency of separate data sets**

In physics it is the rule that an experiment done by one team is repeated 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 opportunity to carry

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9Yet, 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. 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. (2006), 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. *Sources: Archives of the Zoological Society of London, Kohler et al. (2006).*

out such a comparison for the case of small primates. The two observations 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. (2006) 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. (1975) we are told that “the neonatal mortality rate was 10.8%, and the post-neonatal mortality rate (deaths between 31-183 days) was 6.9%. These data allows us to compute $\gamma$. 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. (1978) gives the following distribution of deaths 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 this population of *Macaca mulata* was “special” in some respect. In order to get a clearer understanding, additional information would be needed about the conditions prevailing at that breeding center.

**Broader view of animals kept in zoos**

The dataset used in the paper by Kohler et al. (2006) 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.

1. The infant death rates are much more regular and uniform than the death rates of mid/old age (we come back to this point below). They are also much higher.

2. With respect to the exponent $\gamma$, 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
Fig. 10 Infant mortality rates versus mid-age rates. The scales for the mid/old age rates are given by the axis on the right-hand side and the top axis. The numbers which precede the names of the subgroups are the exponents $\gamma$ 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 of the data: Kohler et al. (2006).

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.

Insects

Before becoming adult, insects go through several life stages: (i) embryo, (ii) larva, (iii) pupa (or nymph which has already the form of an adult but not yet its size) 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. The nymphal stage should be included in the adult stage because the nymph has already all the properties
of the adult.

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.

Now, let us have a look at some data.

Successive instars

The best test is provided by successive instars. It can be recalled that in its nymphal stage, an insect has already its adult shape but because of the rigidity of its outside cuticle, in order to achieve its adult size, it must undergo several molts. The phase between two successive molts is called an instar. Often there are up to 5 or 6 instars. The last instar is the adult. Itô (1980) gives several survivorship curves on which successive instars are plotted. It appears that often the screening between the first and second instar is weaker (in the sense of a smaller $\mu(t)$) than the screening following the hatching which produced the first instar. As an illustration one can mention the case of *Mogannia minuta* (sugar cane cicada) as particularly clear (p. 84). As the survivorship curves drawn by Itô were not designed to study this specific issue, they do not have the required accuracy in the sense that most instar death rates are compressed within a narrow interval. One would need the original data. We leave this question open for further examination.

Beetle

Finally, we consider the case of a beetle, namely *Tribolium confusum* Duval* (Pearl et al. 1941). It can be noted that (as shown 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. The fact that Fig. 11 does not display a power law suggests that a different mechanism is at work.

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
Fig. 11  **Infant death rate of a flour beetle** (*Tribolium confusum*). In the present case, unlike previous cases, $\mu_b$ does not have a steep downward slope after “birth”. Birth, here, in fact means emergence from the pupal stage. This stage follows a previous life stage as a larva in the form of a worm. Thus, there are several successive filtration processes. *Source: Pearl et al. (1941, p. 13-14)*

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.

**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. This parallelism is described in the following lexikon.

**Lexikon of development terms for fish versus plants**

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

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 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 were able to find so far is too low to allow this prediction to be tested.

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10For instance, according to Hanley et al. (2004), 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 10d-15d after germination. Its amplitude may be small (perhaps about 1%) but should be visible on samples of 1,000 seeds or more.
The hyperbolic power law effect

Plants are simpler than insects whose development goes through several successive stages. Moreover, field observations of plants are easier to perform and are more reliable than observations on animals for the obvious reason that unlike animals, plants do not move around\textsuperscript{11}. Yet, surprisingly, 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.

Field observation of palm trees

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? A further discussion can be found in Appendix C of the version of the paper available on arXiv.

![Fig. 12 Infant death rate of a palm tree.](image)

**Fig. 12** Infant death rate of a palm tree. This palm tree (*Enterpe globosa*) lives about 200 years and reaches a height of 20 meters. 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 (1975, p. 263)

Conclusion

\textsuperscript{11}However, for plants a major difficulty is how to define the moment of death. For plants which exhibit positive phototropism, the termination of effect may be used to define death in a more accurate way than just by its aspect.
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 (i.e. $1/t^\gamma$ starting in the vicinity of $t = 0$ and then holding for large $t$) decrease of the death rate. (c) The exponent $\gamma$ 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 predating, 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 $\gamma$ 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 $\gamma$ 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 $\gamma$.

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 (larva, pupa, nymph).

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. 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.

**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 Alfred Espinas (1878). 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 processes.

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12 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.
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 (Bianconi et al. 2013, 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 1,000 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

**Appendix A: 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.

**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. A1a. 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. 1b for an exponential and a power law fall.

| Definition of standard versus postnatal death rates |
|------------------------------------------------------|
| \( t \): age \( (u = \log t) \)                       |
| \( s(t) \): survivors at age \( t \)                  |
| \( \mu(t) \): standard death rate \( \mu = \frac{1}{s(t)} \frac{ds}{dt} \) \( \mu = \log \mu \) |
| \( \mu_b(t) \): postnatal death rate \( \mu_b = \frac{1}{s_0(t)} \frac{ds}{dt} \) \( y_b = \log \mu_b \) |

**Fig. A1a** Standard death rate \( (\mu) \) and postnatal death rate \( (\mu_b) \). The logarithms of the death rates, \( y, y_b \), are used below in log-log plots.

For a power law, the curves of \( \mu \) and \( \mu_b \) have same shape but different exponents. Intuitively, this is quite understandable because for a slow decrease of \( s(t) \) one has:
\( t \geq t_0 = 0 : s(t) = s_0 e^{-\lambda t} \)

\[ \mu_b(t) = \lambda e^{-\lambda t} \quad y_b = \log \lambda - \lambda e^u \]

\[ s(t) = s_0 e^{-\lambda t} \quad \mu(t) = \lambda \quad y = \log \lambda \]

\[ t \geq t_0 > 0 : s(t) = A/t^\rho, \quad A = s_0^\rho \]

\[ \mu_b(t) = (A\rho/s_0)(1/t^{\rho+1}) \quad y_b = \log(A\rho/s_0) - (\rho + 1)u \]

\[ s(t) = A/t^\rho, \quad A = s_0^\rho \]

\[ \mu(t) = (A\rho)(1/t) \quad y = \log(A\rho) - u \]

**Fig. A1b** 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 \(s_0\).

\[ \mu_b \approx \mu; \text{ in most cases examined in this paper } \mu_b \sim 1/t \text{ which means that } \rho \text{ is close to zero and } s(t) \text{ almost level during the infant mortality phase.} \]

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

**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 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.

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.

Possible data sources

For reasons which are easy to understand the process of aging gets considerable attention. Within the broader discipline of biodemography, gerontology has become a
field in itself with its own journals (e.g. “Experimental Gerontology” or “Biogerontology”). Yet, as observed by Levitis et al. (2013), “gerontologists focus on aging and usually take neither data on, or interest in, the periods before adulthood”. Apart from the gerontologists, life tables are occasionally set up by ecologists. As an example, one can cite the work of Itô (1980). We already mentioned the fact that almost none of the life tables contained in his book can be used for studying infant mortality. In this respect the key-requirement is to start the observations as shortly as possible after birth or germination. If the first data point refers to a few weeks after birth (as is seen in many survivorship curves) the main part of the information will be lost.

**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?

Because of their relative simplicity, it is tempting to use unicellular organisms. In some cases such as *Euglena gracilis*, a unicellular swimming organism about 50 micrometers in length, reproduction leads to clones of the parent organism. In other cases, such as *Paramecium caudatum* a swimming organism about 100 micrometers in length, the reproduction by division can lead to offsprings which are not identical to the parent (through the process of an exchange of genetic material between two organisms prior to division). However, no matter whether the children are clones or not, there is the challenge of distinguishing those which died from those alive. This, in different form, is the difficulty already discussed for plants. Unfortunately, even for phototropic organisms (such as *Euglena gracilis*) there is always a small percentage who do not respond to the stimulus which means that this method can hardly be used.

*Caenorhabditis elegans*, a little worm about 1mm 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 hours following hatching is about 4% per day; it falls to about 1.5% per day in the following 16 hours (Smith 2011 p. 24, Tew 2008 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 covering a wide range of cases a clearer understanding will emerge.
Appendix B. Indications about possible models

As already noted, at this point of our investigation it is too early to propose a full fledged model. However, it may be useful to give some hints in order to start the discussion.

The models delineated below are able to generate a power law under some more or less plausible assumptions. However, to be honest, one must recognize that there are probably many alternative models which can also generate power laws under “plausible” assumptions, especially since plausibility is a fairly subjective notion. A better criterion is to ask whether or not the models have a predictive power.

Models based on interdependent defects

We assume that at birth a living organism \( i \) has a number of defects, \( F_i \) which determines its probability of dying. For instance, one may say: “An individual with 20 defects will die in one day, whereas one with only 5 defects will die in one week”. For a population cohort of \( n \) individuals the average number of defects will be: \( \overline{F}(t) = (1/n) \sum_i^N F_i(t) \) and the assumption made at individual level will translate into a relationship between \( F \) and the age-specific frequency of death in the population, that is to say the (normalized) age-specific death rate \( \mu_b(t) \). Through this assumption the survival problem is reduced to a discussion regarding defect numbers. A simple form of the functional relationship would be proportionality: \( \mu_b(t) = k\overline{F}(t) \); this is what will be assumed thereafter but any increasing function would be acceptable as well.

The assumption that the death rate is proportional to the average number of defects is of course fairly natural, but now we must also explain why the death rate decreases fastest when it is highest (namely immediately after birth) and then slower as individuals become older.

There are three mechanisms through which the average number of defects may decrease in the course of time.

1. Through the death of individual \( i \) the term \( F_i \) will be reduced by zero. As the first individuals to die will be those with the largest numbers of defects the decrease of the average will be fastest immediately after birth. The individuals who die after a while may have only one or two defects so their deaths will reduce \( \overline{F} \) very little.

2. There will also be a self-correction mechanism. For instance, a major source of defects is birth prematurity but for those newborns who remain alive, their defects will correct themselves as they become older just because of natural maturation.

3. An additional effect may concentrate the deaths shortly after birth, namely that fact that the defects are certainly not independent. Incidentally, the interdependence of defects may be experienced when executing computer code. A single mistake, e.g.
a variable that was not defined, has a cascading effect and results in an avalanche or error messages. Once this mistake is corrected, everything falls in order. The same effect may happen in living organisms. For instance, suppose that for some reason an organ $O$ which produces an hormone $H$ is underdeveloped because enzyme $E$ is in low supply. So, at this stage, there are 3 interdependent defects: low level of $E$, underdeveloped organ $O$ and low level of $H$. Then, by correcting the core defect $E$ the two other defects will fall in line: $O$ will reach the right size and the concentration of $H$ will reach the right level.

It is reasonable to assume that defect interdependence is highest when $F_i$ is largest. Thus, self-correction will lead to a very fast decrease of $\overline{F}$ at the beginning as long as individuals with many defects are present.

An argument which gives credence to such a mechanism is the close correlation that is observed between low weight and premature birth. Instead of speaking of defect numbers, this mechanism could be rephrased in terms of unfinished growth. If the premature baby is kept in an incubator and fed appropriately, it will be able to terminate its growth process. For instance, the lungs, one of the last organs to be completed, will eventually function properly with the consequence that the appropriate level of oxygen will be delivered in the blood flow. This will *ipso facto* result in the improved working of many organs across the whole body. Naturally, this argument does not apply to malformations which cannot be self-repaired.

Before closing the discussion of this model one can propose a plausible conjecture. If the system has many initial causes of failure corresponding to various mechanisms it seems reasonable to expect that the dispersion in their time constants will increase along with their number. The infant mortality phase comes to an end when all initial defects have emerged and have been eliminated either through a self-repairing process or by elimination of the faulty items. Thus, one would expect the length of the infant mortality phase to increase with the number and diversity of the defects, that is to say basically with the “complexity” of the organism.

Note that in order to apply this argument to living organisms one must renormalize the time scale with respect to size because, roughly speaking, the life span of living organisms increases with their size.

We now turn to a class of models which relies on a global parameter, for instance the birthweight of the organism or more generally any parameter of significance for the death rate.

**Model based on the incidence of birthweight**

In this subsection we will ask ourselves how a power law distribution might arise from a Gaussian distribution due to a simple heterogeneity in an important physical
parameter.
In humans low birthweight is a crucial determinant of infant mortality. Here, however we are not interested in global infant mortality but in the age-specific pattern of infant mortality. So far, in order to describe this pattern we have used the infant mortality rate \( \mu_b(t) \); here, however, we wish to describe it by a density probability function. For instance the density function of the the life span \( T \) (seen as a random variable) would be:

\[
f_T(t)dt = \frac{\text{Number of individuals who die in the age interval } (t, t + dt)}{\text{Total number of individuals}}
\]

The right-hand side is nothing else than \( \mu_b(t)dt \) (except for a possible normalization factor which is unimportant here); thus: \( f_T(t) \sim \mu_b(t) \).

The next step is to establish a connection between the distribution of birthweights and the distribution of life spans. For instance, one may say: “For a birthweight \( W \) in the interval (1.5kg,1.6 kg) the life duration \( T \) will be about one week (on average), whereas for a birthweight in the range (2.0kg,2.1kg) it will be 4 months”. More generally, for the two random variables \( W \) and \( T \) one may posit a relationship of the form: \( T = g(W) \). Clearly, \( g(w) \) should be an increasing function, and in addition one should have \( g(0) = 0 \). A possible example would be a parabolic relationship: \( T = W^2 \).

Now, how can we get a power law density function for \( T \)?
The distribution of \( W \) is known to be Gaussian\(^{13}\), which means it has the following density function\(^{14}\):

\[
f_W(w) = \frac{1}{\sigma \sqrt{2\pi}} \exp \left[ -(w - m)^2 / 2\sigma^2 \right] \quad (B.1)
\]

What will be the distribution of \( T \)?
According to a well-known result, if the equation \( t = g(w) \) has only one solution \( w_1 = g^{-1}(t) \) the density function of \( T = g(W) \) will be (Papoulis 1965, p. 126):

\[
f_T(t) = \frac{f_W(w_1(t))}{g'(w_1(t))} \quad (B.2)
\]

where \( g'(w) \) denotes the derivative of \( g(w) \).

As an illustration, we consider the case: \( T = g(w) = w^\alpha \). It leads to: \( g'(w) = \alpha w^{\alpha - 1} \) and \( w_1(t) = t^{1/\alpha} \). Clearly the power law behavior can only come from the

---

\(^{13}\)Yet the tails are somewhat more heavy than would be expected from a standard Gaussian. That is why it was suggested by some authors to represent it by a mixture of two Gaussian distributions: one for the central values and a second one, much flatter (that is to say with a large standard deviation), which would account for the tails.

\(^{14}\)Of course, \( W \) takes only positive values which means that \( f_W(w) \) should be zero for \( w \leq 0 \).
denominator, not from the numerator. Thus, we must have:

\[(t^{1/\alpha})^{\alpha-1} \sim t^{\gamma}\]

which leads to: \(\alpha = 1/(1 - \gamma)\) that is to say \(\gamma = 1 - 1/\alpha\).

The resulting power law behavior is little affected by the function which stands at the numerator provided that \((w_1 - m)/\sigma\) is not too large; otherwise the exponential becomes so small that everything else is suppressed.

Fig. B1a,b shows the case \(\alpha = 4, \gamma = 0.75\). The graph on the left-hand side shows the functions (“g” means “Gaussian” and “h” means “hyperbolic”)

\[f_T(t) \sim y_g y_h \quad y_g = \exp \left[ -\left( \frac{t^{1/4} - m}{2\sigma} \right)^2 \right], \quad y_h = 1/t^{\gamma}\]

The graph on the right-hand side shows the results of a simulation with the same parameters.

**Remark** Why has the transformation \(T = W^4\) (the exponent 4 is just for the purpose of illustration, the argument is the same for any exponent larger than 1) the dramatic effect of making \(f_T(t)\) diverge at \(t = 0\)? At first sight, this transformation seems fairly smooth. Yet, it has one dramatic feature: the derivative of the inverse function \(g^{-1}(t) = t^{1/4}\) diverges at \(t = 0\). The reason why this leads to the divergence of \(f_T(t)\) is easy to explain intuitively.

- Firstly, the fact that the initial distribution is Gaussian is unimportant. For the sake of simplicity we can as well assume that \(f_W\) is a uniform distribution that is to say has a rectangular density function.
- The probability assigned to the interval \(t \in I_1 = (0.01, 0.101)\) will be the same as the probability assigned to the interval \(w \in J_1 = (0.01^{1/4}, 0.11^{1/4}) = (0.32, 0.56)\). Similarly, the probability assigned to \(t \in I_2 = (15.0, 15.1)\) will be given by the probability of \(w \in J_2 = (15.0^{1/4}, 15.1^{1/4}) = (1.968, 1.971)\). Thus, for \(I_1, I_2\) of the same length, \(J_1\) is 80 times larger than \(J_2\). As the distribution of \(W\) is supposed uniform, we will have \(P\{t \in I_1\} = 80P\{t \in I_2\}\).

The same argument is illustrated in the insert graph of Fig. B1 b. The intervals \(I_1, I_2\) were selected for the sake of making the graph best readable: \(I_1 = (0.2, 2.5), I_2 = (15.17, 3)\). With these intervals \(J_1\) is 7.5 times larger than \(J_2\).

Incidentally, the requirement of the conservation of the probability mass that we used above also quickly leads to the result (2). Indeed:

\[f_T(t)dt = f_W(w)dw \Rightarrow f_T(t) = f_W(w) \frac{dw}{dt} = f_W(w) \frac{dg^{-1}}{dt} = f_W(w) \frac{1}{g'(w_1)}\]

Are such high exponents consistent with what is known about the relationship \(T = g(W)\)? There are three answers to this question.
Fig. B1a,b  Density function of life duration: left: calculated from the theoretical expressions, right: generated through a random number simulation. The Gaussian distribution describing the weight distribution has the parameters \( m = 2.5, \sigma = 1 \). The simulation relied on 10,000 random drawings. The insert gives an intuitive explanation of why the transformation \( T = W^4 \) leads to a density function \( f_T \) which has a very high probability in the vicinity of \( T = 0 \).

(1) So far we did not find any data giving the function \( T = g(W) \). In medical statistics it is another variable which is commonly used namely the “birthweight specific infant mortality” which means infant mortality as a function of birthweight. In this expression, “infant mortality” has the standard medical meaning of “under one year of age” rather than the broad meaning used in the present paper.

(2) As a function of birthweight, infant mortality increases sharply when \( W \) decreases. For \( W = 2.0 \) kg, the infant mortality is 20%, then \( W = 1.5 \) kg gives a mortality of 40% and \( W = 1 \) kg of 80%. In a broad sense, such a rapid change is consistent with exponents \( \alpha \) that reach high values.

(3) At first sight it might seem that in the present model we cannot get \( \gamma > 1 \). This is not really true however as can be seen in Fig. B1a. It shows that the slope of \( f_T(t) = y_g y_h \) is strictly equal to \( \gamma \) only in the vicinity of the maximum of the function \( y_g \). On the left-hand side of the maximum the slope will be smaller than \( \gamma \) whereas on the right-hand side it will be higher than \( \gamma \).

(4) In the previous argument we considered \( T \) as a function of birthweight but this is only one possible case. \( T \) can be connected as well to other variables, for instance to the effectiveness of the immune system at birth. The main reason for selecting the birthweight was data availability. The effectiveness of the immune system may be a “better” variable but, to our best knowledge, no data are available.
**How can one generate a power law death rate by superposition?**

The transition from a specific death rate (e.g. exponential or Gaussian) to a power law death rate can be presented in a more general framework.

Let the death rate be conditional on a parameter $a$, that is to say: $\mu \equiv \mu(t|a)$ and suppose that the parameter $a$ is determined by a distribution $f(a)$. Under these assumptions the unconditional death rate resulting from the superposition of the processes $\mu(t|a)$ is:

$$\mu(t) = \int_0^\infty \mu(t|a) f(a) da$$

(B.3)

The expression (B.3) is very general. It gives the mortality rate that results from the superposition of a set of “elementary” processes with weights $f(a)$. As an illustration, let us consider the following case:

$$\mu(t|a) = Ae^{-at}, \quad f(a) = \left[ \frac{1}{b \Gamma(d)} \right] \left( \frac{a}{b} \right)^{d-1} e^{a/b}$$

(B.4)

The function $f(a)$ is the density function of a Chi-squared distribution with $d$ degrees of freedom. With this choice we obtain:

$$\mu(t) = \frac{A}{(1 + bt)^d}$$

(B.5)

In order to connect this expression with observation we write it in log-log form by defining $y = \log \mu$ and $u = \log t$:

$$y = -d \log(1 + be^u) + \log A$$

(B.6)

For large values of age\textsuperscript{15}, typically $t \gg 1/b$, one gets: $y = -du + \log(A/b^d)$ which leads to the identification: $d = \gamma$. For $t \ll 1/b$, one gets: $y = -dbe^u + \log A$ which shows a behavior similar to what was observed for beetles (Fig. 11).

For this model it would be an important step forward to be able to identify the parameter $a$ with a physical variable for this will provide a more direct contact with observation.

\textsuperscript{15}In the case of human mortality, “large” would mean a few years.
Comparison of the two classes of models

The adaptive mechanism on which the first model relies establishes a connection between what we observe (i.e. the age-specific infant mortality) and what is going on inside the system. This could give an insight and understanding of the phenomenon of infant mortality. However, as already pointed out, because of its many free parameters the model has no predictive power. This obstacle may be overcome by doing additional observations focused on simple subsystems.

Unlike the first, the second kind of models does not relate what is observed to what is happening inside. Yet, it has the merit of offering testable predictions. The successive steps can be summarized as follows:

\[
\text{Measurement of } \gamma \rightarrow \alpha \rightarrow T = g(W) \rightarrow \text{Comparison of } g(W) \text{ to observation}
\]

If the predicted form of \( T = g(W) \) agrees with observation, we will get more confidence in the model, yet we will not learn anything new. On the contrary, if the predicted form does not agree with observation, this will raise a question. If \( T = g(W) \) displays a well defined pattern but which differs from the predicted function, then one must understand why.

Appendix C: Additional data about plant mortality

Is the high exponent found for palm trees also observed for other big trees? As already said, there are few studies available which is why their discussion is made in the present appendix rather than in the paper itself. One study that is cited in Valen (1975), namely Hett and Loucks (1971), concerns a species of maple tree. Although less high than \textit{Euterpa}, it is also a big tree which can live up to 250 years. Observations were conducted in Wisconsin for trees ranging from germination time to the age of 16 years and they led to \( \gamma = 1.35 \pm 0.16 \). This value shows that the \( \gamma \) value of \textit{Euterpa} may be exceptional.\(^{16}\)

Before closing this subsection about plants a word of caution must be added to observe that the methodology used for trees relies on the assumption that the age structure of the population under observation is in a stationary state. Why? The reason is simple. Valen’s observations cover an age interval of 150 years. Clearly such an observation was not made by following a cohort of seedlings in the course of time.

\(^{16}\)Although Valen cites the paper by Hett and Loucks, he does not discuss their results and why they are so different; in fact, he does not even give their results. This is just a confirmation of the fact that, unlike physics, life sciences and social sciences develop a kind of “mosaic knowledge”. By this term we mean that basically these fields are made up of a multitude of disconnected results. This can be illustrated by the title of a randomly selected paper: “The demography of the short-lived perennial halophyte \textit{Spergularia maritima} in a sea-shore meadow in south-western Sweden” by A. Telenius, Journal of Ecology 81,1,61-73 (1993). The study concerns one species in one place, in one country without any attempt to broaden it to other places or other species.
Instead Valen conducted a transversal study of the population. Although the study by Hett and Loucks covers only 16 years of age, they used the same method. One should keep in mind that, due to possible fluctuations in the age structure, transversal studies may not correctly reflect and describe the survivorship curve. In other words, the values of $\gamma$ need to be confirmed by plant studies based on longitudinal analysis.

One study of this kind can be found in Silvertown and Dickie (1980). They followed the growth of several perennial plants (that is to say plants which live several years) after their germination over a period of one year. On average the authors made a field visit every 42 days. For several of their species, their initial cohort comprised less than one hundred plants. We will limit ourselves to one species (namely Anthyllis vulneraria, a plant about 30 cm high with green leaves and yellow flowers) whose initial cohort numbered over 300. The values of the infant mortality death rates derived from the survivorship curve plotted in the paper lead to the following exponent: $\gamma = 3.4 \pm 1.9$. This high exponent suggests a high initial death rate and the latter is quite consistent with the qualitative observations reported by the authors. They tell us that “no individuals reached the flowering stage in any of the population under observation and most cohorts experienced over 80% mortality in their first year”. They add that such high levels of pre-reproductive mortality agree well with the observations made by other researchers. In the particular case of Anthyllis vulneraria, a study conducted in the Netherlands on 1,117 plants showed that only 27% ever reached the flowering stage (Sterk 1975). Moreover, the author notes (p. 333) that “younger plants have a smaller survival chance owing to the high seedling mortality”.

**Observations in greenhouse under controlled conditions**

The exponents $\gamma$ observed so far were equal to 2.6, 1.3, 3.4 respectively which gives an average of 2.43 that is to say two or three times higher than the exponents found for animal species. As these were field observations should these high values not be attributed to the fact that in the field the trees are exposed to various adverse conditions? In other words can these observations be confirmed by controlled observations performed in greenhouses?

We will mention one observation of that kind. Lidia Cruz and her collaborators (2014) report an experiment in which the seedlings of 6 cacti species were grown in a greenhouse during a period of 16 months after germination. For the species E. reichenbachii the exponent was $\gamma = 2.6$ and the average for the 6 species was 1.98. This shows that high values of $\gamma$ are not limited to field observations of trees. However, not all trees have a $\gamma$ higher than 1. An experiment for acacia trees performed in India gave values of $\gamma$ around 0.7 (Shaukat et al. 1999).
Acknowledgments We would like to thank Prof. Thomas Kirkwood for his encouragements. His paper on the Gompertz law (Kirkwood 2015) was a source of inspiration and a guide in writing the present paper.

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The objective of the comments within square brackets is to indicate the implications of the work under consideration for the present investigation. They may be removed in the final version.

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[This study was written in a time marked by the emergence of sociology as a new field. It is interesting to observe that at that time it seemed natural for the author to discuss human and animal societies within the same conceptual framework. Nevertheless, Alfred Espinas is quite aware of the fact that in order to be meaningful such broad comparisons must focus on a small number of basic and well selected features. The author explains his epistemological positions in the following terms (p. 5),]
followed by my translation:

"Nul être vivant n’est seul. La vie en commun n’est pas dans le règne animal un fait accidentel. Elle n’est point, comme on le croit souvent, le privilège de quelques espèces isolées dans l’échelle zoologique, castors, abeilles et fourmis; elle est au contraire un fait normal, constant, universel.

C’est une tentative aussi vaine que fréquemment renouvelée que celle de découvrir les lois de la vie sociale dans l’homme indépendamment de toute comparaison avec les autres manifestations de la vie sociale dans le reste de la nature. Mais il faut reconnaître qu’un simple rapprochement ne suffit pas”.

No living organism is completely isolated. The act of living together is not limited to a small number of social species such as beavers, bees or ants. Quite on the contrary it is a universal feature of animal life. It would be a vain and worthless attempt to try to unravel the laws of social life independently of the study of social behavior in animals. Yet, one must recognize that in order to be fruitful such a comparison must be well focused.

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[The authors describe several mathematical structures which may play a role in the process of aging, for instance systems working in series (i.e. fast global failure) or in parallel (i.e. delayed global failure), systems beset with initial damages, avalanche-like situations where new defects appear in proportion to the number of defects already existing. Based on these effects (or their combination) it is possible to generate Gompertz’s law of aging in many ways. However, unless confirmed by the observation the internal structure, such models remain merely a mathematical game. Better than an avalanche of speculative models largely based on thin air, modeling should be done hand in hand with appropriate experiments One should not think that such a situation where several theoretical frameworks are in competition is special to the life sciences. It also occurs in physics. For instance, the solubility of gases in water can be explained by several effects.

- Equality at equilibrium of the flows in both directions. As the velocities are known from the equipartition principle, it is possible to derive the concentrations.
- Number of molecules of water in the solvation shell, i.e. the shell surrounding a molecule of gas (Sharlin et al. 1998)
- Concentration gradient in the film at the interface between the gas and the liquid
Respective interaction strengths between molecules in the gas on the one hand and in the liquid on the other hand. Obviously, if the molecules of the gas and of the liquid have a strong attraction solubility will be higher.

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