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Age spectrometry of infant death rates as a probe of immunity: Identification of two peaks due to viral and bacterial diseases respectively

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HIGHLIGHTS

- Changes in infant mortality informs us on immunity.
- Its hyperbolic decrease reveals two transient peaks.
- They reveal weak responses to viral/bacterial diseases.

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ABSTRACT

After birth, setting up an effective immune system is a major challenge for all living organisms. In this paper we show that this process can be explored by using the age-specific infant death rate as a kind of sensor. This is made possible because, as shown by the authors in Berrut et al. (2016), between birth and a critical age t_c, for all mammals the death rate decreases with age as a smooth hyperbolic function. For humans t_c is equal to 10 years. It turns out that for some causes of deaths and specific ages the hyperbolic fall displays temporary spikes which, it is assumed, correspond to specific events in the organism's response to exogenous factors. One of these spikes occurs 10 days after birth and there is another at the age of 300 days. It is shown that the first spike is related to viral infections whereas the second is related to bacterial diseases. By going back to former time periods during which infant mortality was much higher than it is currently, one gets a magnified view of these peaks. They give us useful information about how an organism adapts to new conditions. Apart from the reaction to pathogens, the same methodology can be used to study the response to changes in other external conditions, e.g. temperature or oxygen level.

1. Introduction

The word "spectrometry" used in the title may at first sight appear surprising. However, after a moment of hesitation, we decided to use it because it describes very well the idea which leads the investigation conducted in this paper. This title

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also reveals that our approach relies very much on ideas from physics.[1] How the concept of spectral analysis can be a guide in biological research is further explained in our conclusion.

It can be mentioned that the present investigation is part of a broader study of mortality rates conducted over the past three years [1–4].

1.1. How detailed analysis of death rates can teach us the causes of diseases

The methodology used in this paper requires several steps. Here, to start with, is a simpler example of how to discover the source of a disease through the analysis of appropriate death rates.

Annual mortality data by cause of death do not give any understanding whatsoever of the mechanism of diseases. However, the situation changes if we consider *monthly* mortality data by cause of death. Why? Because each month is characterized by an average temperature and therefore monthly data will tell us how diseases react to temperature changes. It turns out that for influenza there is a huge spike in January–February whereas for enteritis (i.e. inflammation of the small intestine) there is a peak in August. This observation tells us something about the way these diseases appear and are transmitted.

Now, if in addition we can decompose the data according to age-groups we will see that the summer peak of enteritis affects predominantly children under the age of 5. At the beginning of the 20th century this observation led medical authorities to suspect that pathogens contained in cow milk given to children were the main culprits. The simple advice of boiling the milk before feeding young children led to a great reduction in death through diarrhea and enteritis.

This example shows how decomposition according to appropriate parameters can enhance our understanding. In this paper we will use a similar procedure.

1.2. Methodology

Our comparative analysis of death rates is made possible by two circumstances.

- The existence of an "International Classification of Diseases. By the end of the 19th century physicians and statisticians from all developed countries agreed on an "International Classification of Diseases". The adoption of this classification was an essential step because it ensured cross country comparability. In this paper we use both the current classification which is ICD-10 and earlier classifications.
- Hyperbolic pattern. In [1] it was shown that the age-specific postnatal death rate follows a hyperbolic decrease $\mu_b(t) \sim 1/t^{\gamma}$ until a critical age t_c. After t_c it starts to go up and then keeps increasing until death. For humans, $t_c = 10$ years.

This hyperbolic pattern holds, not only for the global (i.e. "all causes") mortality but also for individual diseases, albeit with distinct values of γ. Fig. 1 illustrates this statement for the case of metabolic diseases (e.g. diabetes of type 1, disorders of cholesterol metabolism). One must insist on the fact that it is this background of a smooth hyperbolic fall which makes possible the detection of peaks which in turn reveal specific effects in certain age intervals. With a more noisy background the small peaks identified later on would be invisible. In this sense peak detection and hyperbolic power law are closely connected.

Why did we illustrate the hyperbolic pattern through metabolic disorders instead of a disease that would be closer to the class of infectious diseases which will be considered later on in this paper? We picked up a class of diseases which is not directly related to the immune system in order to emphasize that apart from the immune system there must be another factor responsible for the hyperbolic fall and subsequent exponential increase. This remains an open question.

In the present note we identify and study two deviations with respect to the hyperbolic fall. What is our purpose in so doing?

We wish to see if the infant death rate can be used as a kind of spectrometer from which information can be derived about the complex mechanisms which lead to the regulation of the immune system.

1.3. Steps

To carry out this program the paper takes the following steps.

- First, we must identify the age intervals where the death rate deviates from its hyperbolic fall.
- Once intervals with excess mortality have been detected, one must make sure, through comparative analysis, that these death rate peaks have a broad validity and are not just seen in one country.

[1] An earlier and in some respects more detailed version of this paper can be found on the arXiv website: arXiv:1610.02198.

- The next step is to identify the factors through which these peaks can be explained. It will be seen that a first peak (around the age of 10 days) is related to viral infections while a second (around 6–12 months) is related to bacterial infections.[2] There are good reasons to think that the second peak is due to the transition from protection provided by maternal antibodies to an individual endogenous immune system. In its shape this bump is very similar to the one observed in fish during the transition from yolk sac feeding to feeding on exogenous sources of food. It seems that in both cases the transition brings about a fragility which results in a transient death rate peak (more details can be found in [7]).

1.4. Macroscopic biology

Before closing this introduction, a last word is in order to explain why a paper on the formation of the immune system is published in a physics journal by a team of researchers comprising two physicists. The reason is very simple. It turns out that physics journals are more open to inter-disciplinary explorations than are biological or medical journals. Broadly speaking, the new field that physicists began to explore in the early 2010s can be referred to as macroscopic biology. Among the pioneering works in this field one can mention [8–10].

Macroscopic biology differs from molecular biology in the same way as macroscopic physics differs from microscopic (i.e. atomic) physics. Rightly or wrongly, we believe and conjecture that there are some simple laws at the level of macroscopic biology in the same way as there are simple macroscopic laws in physics, e.g. the Ohm law or the Snell–Descartes law.[3] The key-point here is that despite their simple mathematical form, these laws depend upon intricate underlying microscopic phenomena. In short, we believe that explorations at macroscopic level can usefully complement the microscopic perspective provided by molecular biology.

2. Local peaks superposed to the hyperbolic law

2.1. Identification of the age intervals of the peaks

It is well known that over the past century in all developed countries infant death rates have been divided by a factor of one hundred.[4] Therefore our exploration of death rate peaks will be easier and more successful if we use data from the first half of the 20th century.

In Fig. 1 it can be seen that between 0 and 10 years the infant death rate decreases by several orders of magnitude. Without a vertical log-scale, the whole curve would be compressed against the x-axis. The log-scale makes the curves readable but Fig. 2(a) still does not reveal any clear signal. In such a situation a common technique is to take the ratio of successive curves in order to filter out the huge variations. This is done in Fig. 2(b) for the case of Switzerland (several ratios) and the United States (one ratio).

The ratios compare the death in given time intervals I_k starting in 1906 to a reference time interval which is I_r = 1975–1978. Because of the expectation of steady medical progress one expects any disease to appear in the form of a "bump" which becomes higher as one moves to earlier I_k. This is indeed what is observed.

The death ratios reveal two peaks, one which starts around 10 days and the other around 300 days after birth. These peaks are common to the two countries. In addition the curves for Switzerland show a steady decrease in the peaks' amplitudes. The fact that there are only few data points to cover the whole age interval means that the locations of 10 and 300 days are defined with substantial error bars. In what follows we will try to improve the localization.

We can conclude this first experiment by saying that there are two indentations on the curves of the age-specific mortality from "all causes" which are present both in Switzerland and in the US. While not easy to identify on the rates themselves, they become clearly visible on the ratios of the death rates for successive decades. The amplitudes of these indentations become larger as one moves back in time.

2.2. Identification of the underlying diseases

Although Fig. 2(b) suggests that the peaks were higher in the first half of the 20th century they may still exist (albeit in reduced form) in present-day data. If this assumption is correct it will make their identification much easier because we will be able to use the "CDC WONDER" database [6] which covers the period 1999–2014 and provides unparalleled accuracy about causes of death. The fact that the database gives cumulative figures for a 16-year interval and for a large country is important because even if the effect that we wish to detect is much smaller than in the early 20th century we may nevertheless have a chance to detect it.

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[2] For the purpose of illustration examples of viral diseases are: hepatitis, herpes, influenza, measles and smallpox; examples of bacterial diseases are: bacterial pneumonia, diphtheria, enteritis, legionella and tuberculosis.

[3] Named after the Dutch astronomer Willebrord Snellius who discovered it in 1621 but did not publish it and the French physicist René [11], it should in fact be called "Sahl's law" after the Iraqi scientist Ibn Sahl who discovered *and* published it in 984 (Wikipedia article entitled "Snell's law".

[4] For instance, in the US on average over the time interval 1999–2015, there have been only 210 deaths annually due to viral diseases in the 0–10 age interval CDC Wonder database [6].

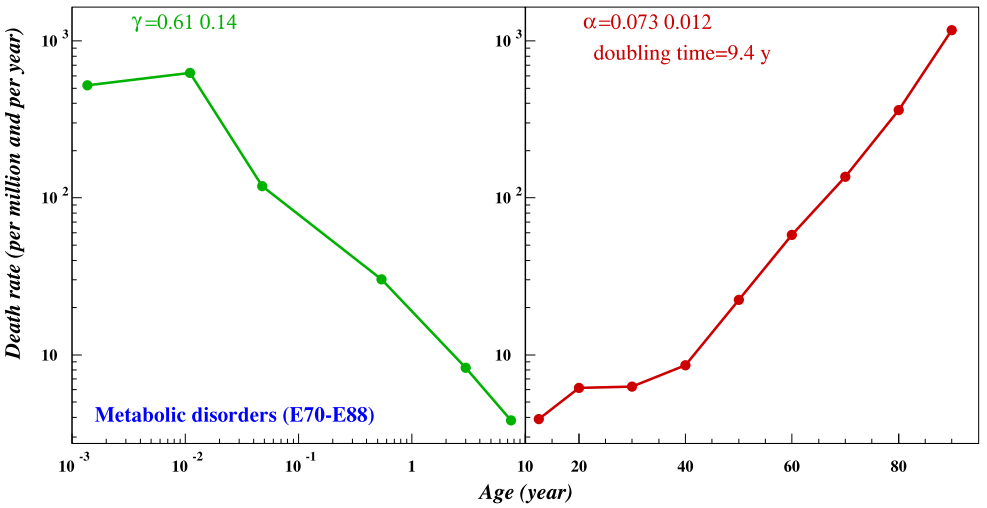

Fig. 1. Death rate due to metabolic disorders, USA 1999–2014. Included in this class of disorders are for instance intolerance to lactose and defects in the metabolism of carbohydrate. These down- and up-going curves ($\mu_b(t) \sim 1/t^{\gamma}$, $\mu(t) \sim \exp(-\alpha t)$ respectively) are similar to those for "all causes" death rates except that the exponents are not the same. For γ it is 0.99 ($\pm$0.09) instead of 0.61 ($\pm$0.14) here. For the doubling time in the adult phase it is 8.6 ($\pm$0.8) years instead of 9.4 ($\pm$1.6) years here. γ and the doubling time can be seen as signatures of a specific class of diseases. It should be noted that the x axis has two different scales: logarithmic from 10^{-3} year to 10 years and linear from 10 years to 95 years. This is necessary for with a linear scale the decreasing curve would be squeezed in a narrow interval. In the case of "all causes" of death the right-hand graph corresponds to the Gompertz law (more details can be found in [5]).
Source: CDC "Wonder" database [6] 1999–2014 (http://wonder.cdc.gov/ucd-icd10.html). The data are average annual death rates over the 16-year long interval 1999–2014.

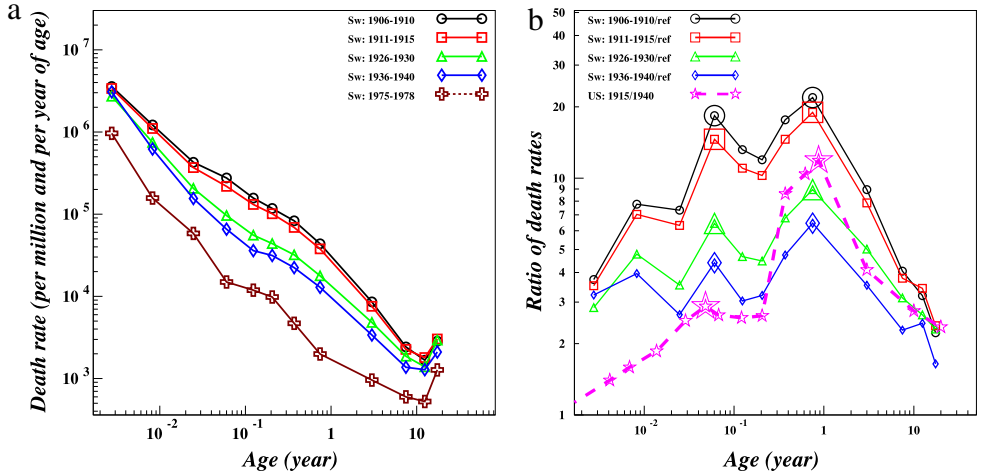

Fig. 2. (a), (b) Death rates for all causes in Switzerland and the United States. (a) Death rates in Switzerland in successive decades. Death rates for the US were also computed and are similar to those for Switzerland. In Fig. 2(b) we show the more significant death rate ratios for both Switzerland and the United States. The "ref" label corresponds to the rates for 1975–1978.
Source: Switzerland: Encyclopédie statistique de la Suisse [12]. Statistique historique. Santé. Mortalité et causes de décès [Statistical Encyclopedia of Switzerland. Historical Statistics; section: Health, Mortality by causes of death.]; available on Internet at the following address: https://www.bfs.admin.ch/bfs/fr/home/ofs/histoire.html USA: [13, p. 574].

As one goes through various causes of death, e.g. cancer, heart diseases, malformations, metabolic diseases, enteritis, bacterial diseases, viral diseases, it becomes quickly clear that only the last two in this list show visible peaks. These are shown in Fig. 3.

Can we identify them with the indentations displayed in Fig. 2(b)? Two reasons make this identification plausible.

- The peaks displayed for viral infections on the one hand and bacterial diseases on the other hand are roughly positioned as suggested in Fig. 2(b). However, this reason is not completely compelling because of the lack of accuracy due to the small number of data points. Over the first year after birth the data given by WONDER are limited to the standard postnatal intervals: 1st day, 1–7d (early neonatal), 7–28d (neonatal), 28–365d (late neonatal).

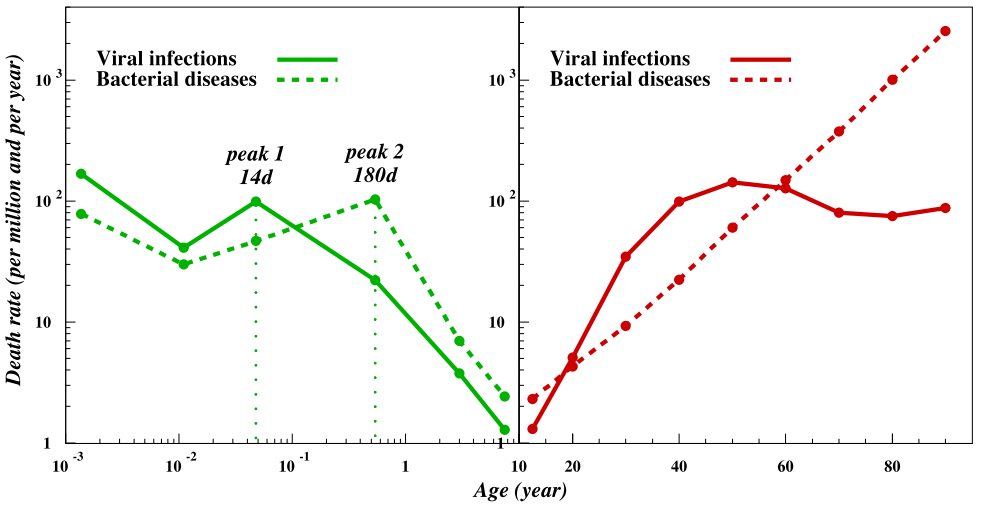

Fig. 3. Death rates for viral and bacterial diseases, USA, 1999–2014. The left-hand graph shows the death rate per calendar year, day of age and million live births. The ICD10 code numbers for viral infections and bacterial diseases are $(A80 - B34)$ and $(A00 - 09 + A30 - 49)$ respectively. The two peaks are found approximately at the locations where they were expected. The right-hand side of the graph shows that in contrast with bacterial diseases (as well as most other diseases) the age-specific death rate of viral infections deviates strongly from the expected Gompertz-like exponential growth. The reason remains an open question.
Source: CDC WONDER database, 1999–2014 [6].

- Are the amplitudes of the peaks of Fig. 3 consistent with what we expect? The steady decrease of the amplitude of the indentations seen in Fig. 2(b) makes us expect fairly small deviations in the period 1999–2014. This is indeed the case. For the 16 years of the period under consideration the first four data points[5] of the curve for viral infections correspond to a total number of deaths of 30, 44, 371 and 1,337 respectively, that is to say averages of 1.8, 2.7, 23 and 83 deaths annually. These are very small numbers.[6] The deaths for the interval i_4 are largest because this interval is about 50 times broader than the one-week intervals i_2, i_3.

Incidentally, we see that more age intervals would lead to even smaller numbers (hence more statistical fluctuations) and therefore would not be very useful. In addition, it can be observed that the contribution of these deaths to the total mortality is very small. The 371 deaths represent only 0.65% of the total number of 56,616 deaths in the age interval i_3. For that reason it is undetectable in the curve for "all causes".

A last comment is in order regarding the right-hand graph which covers the adult age interval 10–90. Strictly speaking, this graph is not necessary for the present investigation. However, it is of interest because of the sharp difference in shape of the two curves. In old age, the bacterial death rate is 200 times larger than the viral death rate. No doubt, such a huge difference has a biological significance.

3. Magnification of the signatures of viral/bacterial diseases

In the previous sections we were able to locate the peaks and to identify the diseases that they describe. In addition we saw that, unsurprisingly, these peaks had been markedly reduced in amplitude over the past century. Thanks to this understanding, we can now get a more accurate picture of these signatures. For that purpose we need to introduce two improvements.

- We need to view these diseases in the past when their amplitude was larger. Moreover, we must use a country whose population is as large as possible which is why we mostly limit ourselves to the United States.
- We need to find data sets which provide as many data points as possible over the age interval (0, 10 years).

As we already observed, these conditions go hand in hand in the sense that the second would be useless if the number of deaths is too small for then most of the age intervals would have small numbers of deaths which would give rise to large statistical fluctuations.

When one wants to go back in time one must be careful about two things.

5 They are for the following age intervals: $i_1 = (0, 1^-)$, $i_2 = (1^+, 7^-)$, $i_3 = (7^+, 28^-)$, $i_4 = (28^+, 365^-)$.

6 That is why we cannot give similar data for Switzerland. As its population is 37 times smaller than the US population one would not see a single death.

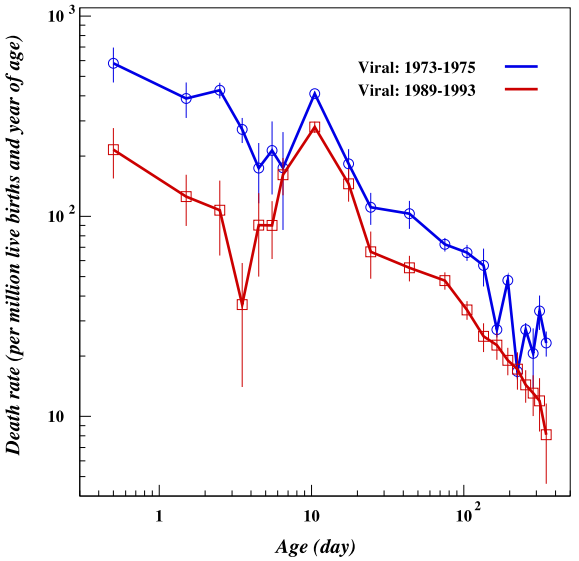

Fig. 4. Infant death rates for viral diseases, USA, 1973–1975 and 1989–1993. The peak value occurs at the age of 10.5 days after birth. The error bars are ±standard deviation. This graph is very similar to graphs describing the yolk sac effect for fish (see for instance the graph for sturgeons in [1], graph 4a). This is not surprising because like breast milk yolk is known to have an anti-body content [7]; this gives the yolk-sac effect an immunity facet in addition to the nutrition facet.
Source: Vital Statistics of the United States for the corresponding years.

- There is a major difference between the data provided by the CDC WONDER database [6] and those given in the digitized paper volumes of the "Vital Statistics of the United States" in the sense that the VSUS volumes give the death numbers for a *selection* of causes whereas WONDER gives them for *all* causes.
- In addition, the list of death causes (and also their definition) has changed with successive revisions of the "International Classification of Diseases".

For our present purpose, this has two direct implications.
(1) In the VSUS volumes the cause "Viral diseases" starts to appear in 1968; thus we cannot go back in time earlier than 1968. Moreover in the first years after being introduced this entry has fairly irregular data. That is why we considered the time interval 1973–1975.
(2) The entry "Bacterial diseases" does not exist in the VSUS volumes and as the entries are only a selection it would be hazardous to set it up by combining several sub-entries. For this reason we prefer to focus on one specific bacterial disease, namely tuberculosis. As is well known, tuberculosis was the major cause of death in the United States in the early 20th century.

3.1. Viral diseases

Fig. 4 shows the peak of viral diseases. It extends from 5 to 22 days after birth with a peak value at 10 days. This peak is not only present in the US for all years for which data are available, we have also seen at he beginning that it exists as well in Switzerland.[7] What questions do these curves raise? One can mention the following points; probably medical doctors would raise many others.

- One may wonder why exactly there was an overall decrease over the 16 years between the two time intervals. As far as we know, between 1973 and 1993 there was no vaccination before the 10th day after birth. In 2006 in the US influenza vaccination was not done earlier than 6 months of age. Thus, the decrease should probably be attributed either to a decrease in incidence or an improvement in post-natal care especially for premature births.
- Even if one accepts the overall reduction as a fact, it can be observed that the reduction was much smaller around the peak value than elsewhere; this raises another question.

[7] Unfortunately the Swiss Office of Statistics does not seem to have data by age for this ICD entry. Thus, one cannot make a direct comparison.

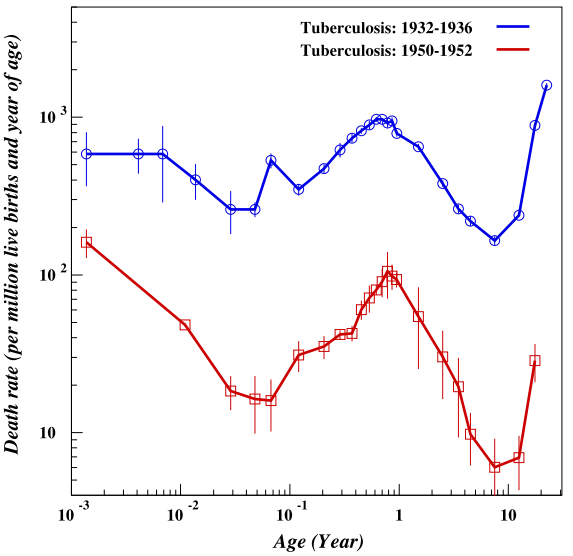

Fig. 5. Infant death rates for tuberculosis, USA, 1932–1936 and 1950–1952. The peak value occurs at the age of 290 days after birth. The error bars are ±standard deviation of the rates in successive years.
Source: Vital Statistics of the United States for the corresponding years.

3.2. Bacterial diseases: tuberculosis

Fig. 5 shows the peak for tuberculosis considered as representative of the broader class of bacterial diseases. It is much (130 times) wider than the viral peak and extends from 25 days after birth to the age of 6 years. This peak is not only present in the US it is also present in Switzerland. Because the disease was very serious at the end of the 19th century fairly detailed Swiss data are available for 1877–1881. The Swiss curve (not shown here) is parallel to (and of course higher than) the US curve of 1932–1936. The bacterial peak has a larger amplitude than the viral peak: the top-to-bottom ratio is 3 for the viral peak and 6 for the tuberculosis peak.

3.3. Prediction, test and confirmation

If the previous identification and interpretation of the two infant mortality peaks is correct, it can be used for the purpose of making predictions. Then, if such predictions can be tested successfully it will give more confidence in the validity of our explanation.

In the previous subsection we tried to magnify the peak effect by moving back to former decades, at least as far as permitted by data availability. Alternatively, one can focus on specific years characterized by high mortality rates due to a pandemic. One possibility which comes to mind immediately is the influenza outbreak of October 1918.

In fact, the death rate was abnormally high between September 1918 and January 1919 (there was even a new outbreak in early 1920) but the peak of the epidemic was reached in October. In this month there were 117,000 deaths due to influenza in the Registration Area of the United States; this represented 52% of the total for the 4 months from September to December. As one knows, influenza is caused by a virus but there was also an associated outbreak of pneumonia and bronchopneumonia (221,000 deaths together). In contrast to influenza, pneumonia may be caused by both viruses and bacteria.

Thus, based on the former analysis one would expect two peaks: a virus peak around the age of 10 days and a bacteria bump between a few months and one year. Fig. 6 shows that such peaks are indeed present; as a matter of fact, they are somewhat broader than expected, probably due to the magnitude of the epidemic.

4. Conclusion

4.1. Age-specific analysis of infant death rates

In spectral analysis one draws radiation intensity as a function of wavelength in order to detect peaks and troughs which in turn identify emission or absorption wavelength characteristic of specific atoms (see below). Here, we analyzed infant death rates as a function of age in order to characterize internal responses to exogenous factors.

A key was to use the ratios of the death rates which filter out what is common to them and thereby reveals their differences even if they are small. Here we have used this tool to compare the death rates in different time periods but such a differential comparison could also be done for two regions or countries. Then, in the second part, we have shown how to identify the

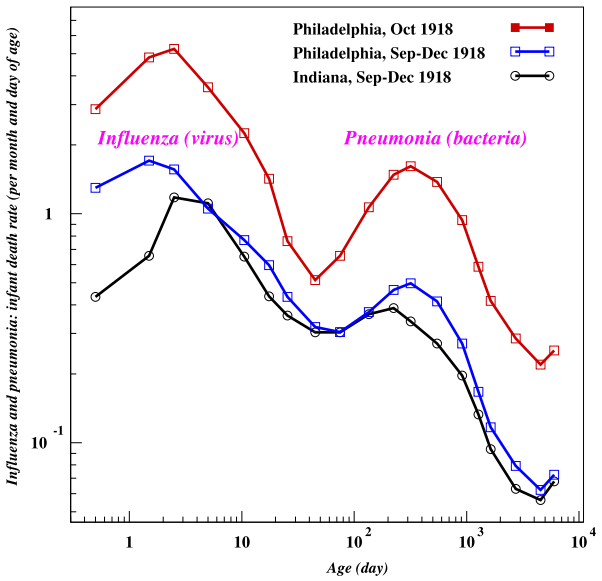

Fig. 6. Age-specific infant death rates due to influenza and pneumonia in the United States in the fall of 1918. The two peaks occur in the age intervals where they were expected: around 6–10 days for the viral peak and around 300 days for the bacterial peak.
Source: [14].

diseases responsible for the anomalies. Finally, by going back to earlier decades we could give a fairly accurate description of the viral and bacterial peaks.

4.2. A major interrogation

Apart from the specific topic considered in this paper, a major interrogation is how to explain the pattern seen in Fig. 1. As already observed, it cannot be explained solely by changes in the immune system.

4.3. Transition stages of the immune system

As this paper is written not by physicians but by physicists its main objective was to present the evidence as clearly as possible. The question of how to interpret the peaks shown in Figs. 4 and 5 will be discussed only briefly.

The broad bacterial peak is probably in relation with the transition from immunity based on maternal antibodies to an autonomous immune system. As a proof of the effectiveness of these antibodies, [15] mention the fact that babies with agammaglobulinemia (a deficiency in the production of antibodies) are nevertheless well protected against bacterial infection up to the age of 6 months. Then, maternal antibodies wane over the period of 6 to 12 months. Moreover, maternal antibodies in all species have been reported to inhibit antibody generation after vaccination. This makes vaccination before 6 months fairly ineffective.

[16] mentions that the inability of the immune system of the late neonate to fully respond to an antigenic stimulus has also been observed in several other mammals, e.g. in pigs, cows, horses, mouses, rats. The problem here is to find appropriate data.

- The population must be large enough for otherwise small variations of the infant death rate will be hidden by the background fluctuations.
- The death rate must be recorded with a good time resolution for otherwise narrow death spikes will not be visible.

4.4. A parallel between spectral analysis in physics and biology

This subsection (as well as the corresponding figures) is more particularly destined to those of our readers who are biologists. However the broad perspective in which we consider spectral analysis can also be of interest to physicists and particularly to econophysicists for in physics we see spectral analysis as a major technical tool but we may not realize that it belongs to a vast class of methods in which one separates a composite into individual components labeled by a new variable.[8] It is a way to extract more information from a sample through a kind of transversal analysis.

[8] Even for light wavelength decomposition is not the only possible. Although much less frequently used, decomposition of light according to polarization is also possible.

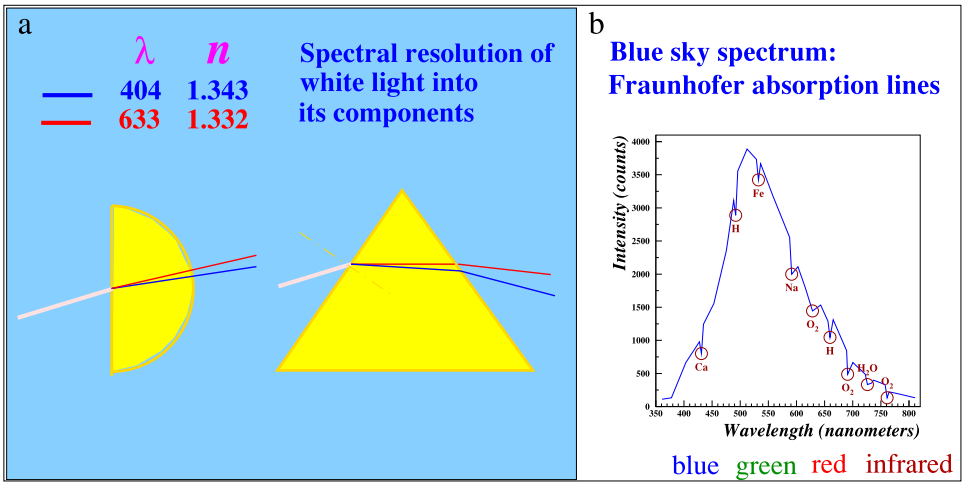

Fig. 7. (a), (b) Spectral decomposition (also called dispersion) based on wavelength of the response of light when it interacts with a material. This figure (which is more particularly destined to those of our readers who are not physicists) explains how a wealth of information can be obtained by decomposing a mixture (white light) into elementary components (separate wavelengths). (a) In the half-cylinder and prism (supposed filled with water) the deflection of light is wavelength dependent. λ (given in nanometers) denotes the wavelength and n is the corresponding refractive index. Note that for clarity the difference in the deflection angles of blue and red rays has been much exaggerated; actually the difference is only 0.83%, which for an angle of 30° would result in a difference of 0.25°. (b) When this decomposition is used to analyze the light coming from the Sun it reveals a great number of indentations (dips) which correspond to black lines in the spectrum. They are due to the absorption of light at specific wavelengths by the atoms and molecules located between emission and reception. Some of the elements mentioned in the graph, e.g. hydrogen, belong to the high atmosphere of the Sun whereas others, e.g. water, are found in the atmosphere of the Earth. Similarly, spectral analysis can also be carried out for the light coming from distant stars. This makes it a powerful tool for exploring the universe. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
Source: The blue sky spectrum data are from the Wikipedia article entitled "Fraunhofer lines".

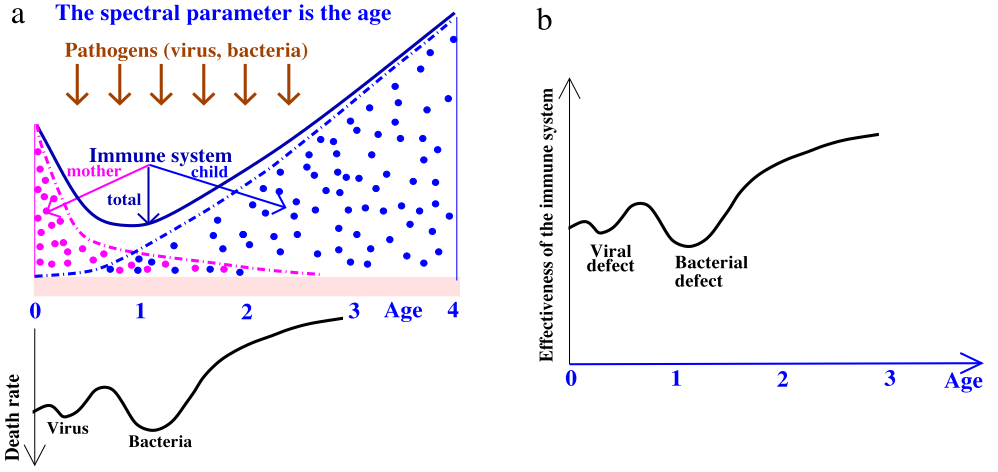

Fig. 8. (a), (b) Decomposition based on age of the response of the immune system. The figure draws a parallel between spectral analysis in astrophysics as shown in Fig. 7(a), (b) and age spectral analysis of death rates. (a) The figure schematically represents the two sources of the immune system: (i) the antibodies transmitted by the mother and (ii) the autonomous immune system of the child which builds up progressively. (b) In the effectiveness of the immune system there are two gaps; the first is respective to viruses whereas the second is respective to bacteria.

Figs. 7(a), (b) and 8(a), (b) parallels spectral analysis of light and age spectral analysis as carried out in this paper. We hope that such a parallel will encourage econophysicists to use this idea more broadly.

The fact that the refractive index changes with the wavelength may seem fairly simple but one should realize that its microscopic explanation is very complicated. The basic mechanism which makes the velocity of light smaller in a medium than in vacuum can be described by an analogy with a game in which a person at one end in a line of people whispers a message to the ear of the next person who similarly transmits it to the next person and so on until the message reaches the other end of the line. Similarly, a photon may be absorbed by an atom which will remain in this excited state during about 10^{-15} s before emitting a new photon and returning to its ground state. The new photon will travel a short distance before

being absorbed and re-emitted again. Between two molecules there is vacuum and therefore the photons always travel at the standard speed of light in vacuum. This mechanism explains why the speed of light in a material is slower than in vacuum but to explain why in glass or water red light travels faster than blue light is much more difficult[9] .

4.5. Generalization

This paper focused on reactions to pathogens but it is also conceivable to analyze the response to a sudden change in temperature (see [17]) or a change in oxygen level. The age-specific decomposition of the deaths that occurred in Switzerland as a result of the heat wave of 2015 does not reveal any clear pattern. One would need to know if the exposition to heat of different age-groups was the same. It is possible that elderly people remained inside air-conditioned apartments or nursing homes.

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