WHEN DOES A COHORT'S MORTALITY DIFFER FROM WHAT WE MIGHT EXPECT? *

For the Ancients, death could be attributed to at least three causes: ageing, temporary vicissitudes and the consequences of an orderly or dissolute life. The Moderns do not diverge much from this view, but can measure these causes with greater precision: senescence is studied at different ages, period effects by year of death, and lifetime experiences by the cohort approach. The next step is to distinguish the action of each of these three causes on the probabilities of death at a given age, at a given time and within a given cohort. This is easier said than done, since the three effects are not independent: any two of them will determine the third.

John WILMOTH**, Jacques VALLIN*** *and* Graziella CASELLI**** *have developed a statistical procedure which makes it possible to estimate the three effects jointly, thus throwing light on the third – the cohort effect – which is much less known and much more surprising than are age and period effects.*

Introduction

The life table, generally calculated to characterize mortality conditions at one point in time, measures above all variations in the risk of death with age. Once passed the delicate period of birth and the youngest ages, this risk increases in a nearly exponential fashion, reflecting to a certain extent the increasing fragility of the human organism as a result of biological aging.

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* An earlier version of this paper was presented, in French, at a conference on *Demographic Modeling*, organized by the Association for Applied Economics in Verona in February 1988, and, in English, at the Annual Meeting of the Population Association of America, held in New Orleans in April 1988. The present version has benefited from the comments of various critics on these two occasions, for which we are most grateful.

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However, the conditions of this biological aging are inscribed within a particular health context which itself evolves over time. For the past two centuries in the developed world, this context has enjoyed considerable improvements, while mortality has seldom slowed its rapid decline. Thus, the age-curve of mortality, while still preserving more or less the same shape, has shown a profound drop in level.

For this reason, a period life table will never correspond to the true life history of a cohort. Cohort mortality combines the age factor (biological aging) and the time factor (health progress) in such a way that, for some generations, mortality may actually decline over large age ranges. The mortality history of a cohort is not the result of a simple combination of the first two factors (age and period), but depends as well on a third dimension belonging to each individual cohort. The vulnerability of an individual at a given age and time is due not only to some theoretical life-potential at that age combined with health conditions at that time, but also to the deterioration (or improvement) of this potential resulting from his past life history. This third component, usually referred to as the "cohort effect", has often excited the demographer's curiosity, a curiosity which, however, has remained largely unsatisfied.

Bringing out this third dimension is indeed not an easy task [15]. To do it correctly, we would need to split cohorts into two groups, perhaps at birth: one (experimental) which we would subject to specific external circumstances, and the other (control) which we would protect from all environmental hazards such that it experiences only theoretical biological aging. After this period of intervention, the two groups would be combined and subjected equivalently to present-day health conditions, and their subsequent mortality experience would be compared. Such a hypothetical comparison is clearly impossible, even in detailed follow-up studies. At best within the present statistical context, we can only hope to compare different cohorts, as opposed to groups within the same cohort, by examining matrices of observed mortality rates or probabilities cross-classified by age and year of birth or observation. Yet even this situation is not extremely frequent. The data necessary for the preparation of such a matrix have commonly been available in published form only for a few countries and since the 1950s. Large arrays of mortality data, constructed from published sources and large-scale historical reconstructions, are now available for a few countries, including Italy [6,24], France [28,30], Japan [39], Belgium [37], the Netherlands [9], and Austria [39]. Based on these data sets, numerous models have been developed which attempt to separate the cohort component from the age and period components.

Given this imperfect data base, we should in fact content ourselves with a detailed description of the structure of the data matrix from three perspectives (row, column, and diagonal) and capitalize on the "rough spots" within this structure in order to formulate or confirm hypotheses on the mechanisms which

link the three components of mortality (age, period, and cohort). In particular, we will not be able to isolate the cohort component except through a consideration of significant mortality differences which we may manage to demonstrate between neighboring cohorts. It will hence be possible to take into consideration but a small fraction of the total weight of the cohort life history, since we will measure only differential effects for cohorts with the most peculiar experience.

Common in past analyses of these data has been a certain fuzziness in the distinction between the three-dimensional structure of the data matrix (what we call "row, column, and diagonal effects") and the three dimensions of mortality (what has often been called "age, period, and cohort effects"). This uncertainty has often been reinforced by the complexity of the model or its fitting. Furthermore, the search for causal explanations at times has gone beyond the limits imposed by the inherent scarcity of information contained in the data being analyzed.

We employ here an appreciably different model which has already been described elsewhere [39,40]. It possesses the desirable characteristics of simplicity and flexibility and was designed with the express purpose of providing a systematic description of a matrix of mortality data. Through its detailed description of the structure of the matrix, it offers the possibility of confirming certain hypotheses or of formulating new ones about the mortality experience of certain peculiar cohorts. The descriptive power of the method will be seen here in application to the French mortality experience over the period 1899–1981.

I. The existence of peculiar cohorts

A) The raw data A simple representation of a matrix of death probabilities can be made in the form of a contour map [10,35]. Previous works have already demonstrated the usefulness of such an approach for documenting the existence of certain peculiar features on a discrete three-dimensional mortality surface [7,8]. A contour map analysis of such a surface is analogous to, although clearly less detailed than, our study of the structure of the data matrix.

Figure 1 presents a contour map of observed French male death probabilities for the period 1899-1981 over the age range 0–99. Such a representation offers a clear description of the general shape of the surface: the increase in mortality with age (except below age 10) and the persistent decline over time. Some particular features of this surface which are evident include the elevated mortality experienced during the two World Wars.

We shall see, however, that this surface may possess other features which are not easily brought out by a contour map analysis. For example, the mortality

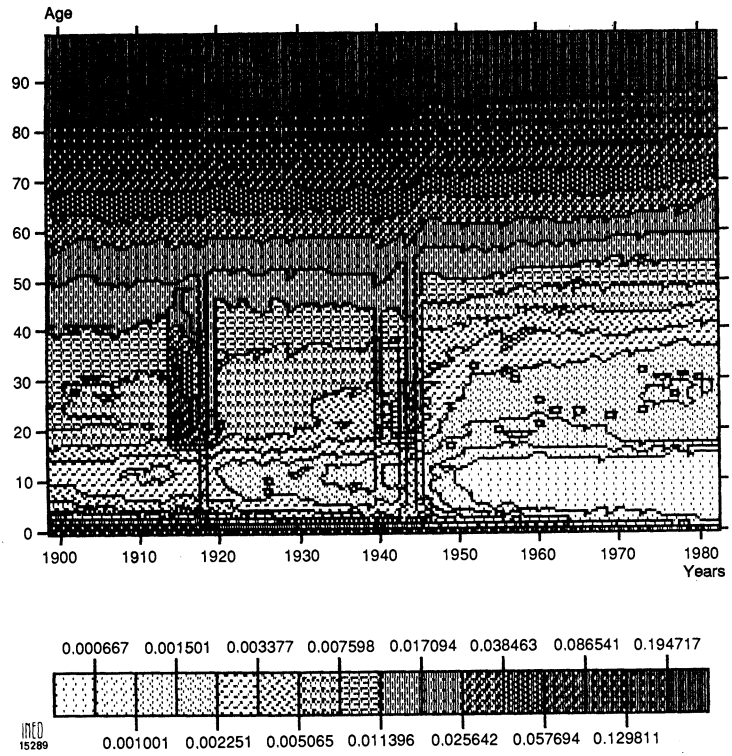

Figure 1. – Contour map of age-specific probabilities of death, q_{ij}, for ages 0–99, years 1899–1981. French males

experience of certain cohorts will be shown to be unusually high or low, so as to produce a small diagonal ridge or valley on the observed mortality surface. Such peculiar features do not show up in Figure 1, since they are masked by larger changes in the level of mortality as we move across the surface either vertically or horizontally. The theoretical interest presented by cohorts with atypical mortality patterns has led us therefore to consider a parametric model which gives a more detailed description of the observed data.

B) Overview of the method As a first result, the model employed provides a synthetic description of the evolution of mortality at the various ages and of its age pattern during the different periods. After a rather classical (logarithmic) transformation of the death probabilities, this description concentrates on the rectangular structure of the matrix: the value in each cell is characterized by its membership within a row (age) and a column (period), represented by i and j respectively. Such a description of the data matrix can

often be improved, however, by a consideration of systematic patterns observed in the residuals of a given diagonal (cohort).

We have chosen to consider an essentially additive model with a logarithmic transformation of the form

$$f_{ij} = \log\left(\frac{q_{ij}}{1 - \frac{q_{ij}}{2}}\right). \qquad 1$$

A standard actuarial estimate of the force of mortality in cell (i, j) is

$$\mu_{ij} = \frac{q_{ij}}{1 - \frac{q_{ij}}{2}}, \qquad 2$$

so that our transformation also has the interpretation $f_{ij} = \log(\mu_{ij})$[1].

The family of models which then interests us has the form:

$$f_{ij} = \alpha_i + \beta_j + \sum_{m=1}^{\rho} \phi_m \gamma_{im} \delta_{jm} + \theta_k + \epsilon_{ij}. \qquad 3$$

where $k = j - i$. The first two terms $(\alpha_i + \beta_j)$ are the additive part of the model, which consists of row effects, αs , and column effects, βs. The next terms are multiplicative adjustments for rectangular non-additivity; their number, ρ, depends on the explicative power and the improvement in fit offered by each successive term. The diagonal effects, θs, are adjustments for non-additivity along diagonals of the matrix, leaving only random errors, ϵs. The model must be fit iteratively, as is described in the Appendix to [41][2].

For simplicity, we refer to a model which contains an additive and some indeterminate number of multiplicative terms (but no diagonal effects) as an AM model. We may specify the number of multiplicative terms included by writing, for example, AM(2) in the case where $\rho = 2$. Likewise, when diagonal effects are included, we refer to the AMD model, in general, or to an AM(ρ)D model, for a specific ρ.

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[1]The alternative choice of a multiplicative model of the untransformed death probabilities was rejected since the variance of the error term in such a model would be far from constant across the matrix. As discussed in [3], the variance of μ_{ij} is approximately $\frac{\mu_{ij}}{N_{ij}}$, where N_{ij} is the population exposed to risk in cell (i, j). This ratio would show extreme variability across the age range (at older ages, for example, μ_{ij} would be relatively large, and N_{ij} would be small), while the variance of $\log(\mu_{ij})$ $(\approx \frac{1}{\mu_{ij} N_{ij}})$ would be considerably more stable.

[2]A methodological appendix was presented in the French version of this paper published in *Population*, 2, 1989. For lack of space, it could not be included here, but will be found in English in [41].

By design, the model gives preferential treatment to rectangular structure, while considering diagonal structure as a secondary element. In short, we attempt to describe the structure of the data matrix using simple additive and multiplicative terms over the rows and columns of the array (rectangular structure), and then attempt to improve the description by introducing a diagonal effect (diagonal structure). This "improvement" may be understood in two senses. On the one hand, in the case where the AM model was fit with a small number of multiplicative terms (usually 2 or 3, for the matrices of mortality rates which we have examined), the diagonal term tends to pick up residual patterns of mortality excess or deficit for certain cohorts, and hence the description is improved by its greater *completeness*. On the other hand, when the AM model was fit with a large number of multiplicative terms (usually 3 or more), the inclusion of a diagonal term tends to reduce the number of multiplicative terms necessary for a complete description, and hence the improvement is one of greater *simplicity*.

In general, we have found that the additive term, consisting of simple row and column effects, is consistently capable of describing an extremely large portion of the total variance in the matrix. Nevertheless, a certain amount of non-additive structure seems to be common. In introducing both multiplicative and diagonal terms, we have chosen to distinguish between two types of non-additivity: the multiplicative terms describe the slow transformation in the shape of the age curve of mortality, while the diagonal term isolates cohorts whose mortality experience is consistently higher or lower than usual. It would of course be possible to replace the diagonal term by several additional multiplicative terms (see [39] for a discussion), but such a strategy would possess clear disadvantages. First, it would be less parsimonious, since several multiplicative terms would generally be needed to replace the single diagonal term. Second, and more importantly, we would fail in this way to make a distinction between two types of non-additive patterns which have differing causal interpretations. The gradual transformation of the shape of the age curve of mortality, as depicted by the multiplicative terms, may reflect changes in either period- or cohort-based factors. Conversely, unusually high or low levels of mortality for certain cohorts in comparison with neighboring cohorts, as brought out by the diagonal term, may be interpreted much less ambiguously as the result of more or less abrupt changes in the life history of successive cohorts.

We distinguish between two fitting procedures, called *exploratory* or *complete*, which may be used for different analytic purposes. A complete fit employs the full iterative procedure described in the Appendix to [41], while the exploratory fit stops automatically after one iteration. The exploratory fit is hence equivalent to fitting an AM model and then deriving the diagonal effects, θ_k, from the residuals of that model. While such an approach is computationally simple, it contains an implicit, but inappropriate, assumption of independence between the various terms of the model. Empirically, it will be observed that the crucial difference between the complete and exploratory fits is that the latter

tends to underestimate the magnitude, but not in general the direction, of the diagonal effects, since some portion of the diagonal structure is absorbed previously by the terms of the AM model. Nevertheless, we successfully employ the exploratory fit in some preliminary stages of analysis, both to lighten the required computations and to simplify the initial presentation of the method.

The current model distinguishes itself from previous examples of "age-period-cohort (APC) analysis" [4,14,15,20] in two important ways. First, it offers greater flexibility in the choice of the appropriate model from a family of models. Second, and more importantly, it differs philosophically in arguing for a thoroughly descriptive approach as a means to improved theoretical understanding. Our method is essentially narrative or historical: we first concentrate on a detailed description of the development of mortality, and then attempt to interpret that description.

It is true that the current model requires several identifiability constraints (see Appendix to [41]), as do other APC models. There should be no misunderstanding, however, about the interpretation of these restrictions. They do not in any way presume *a priori* to reflect underlying relationships between causal factors related to age, period, and cohort. They were chosen, rather, to highlight the predominant additive structure of the data matrix, as well as the clear presence of two distinct non-additive patterns. We have purposefully avoided the appellation "age, period, and cohort effects". Instead, we refer to the parameters of the model first using a coldly data-analytic jargon ("row and column effects", "rectangular non-additivity" or "multiplicative terms", "diagonal effects", etc.). We then attempt to offer valid substantive labels: for example, we interpret the αs as "the average age pattern of mortality over all periods", and the θs as "excess (or deficit) cohort mortality". As mentioned in the introduction, theoretical models which incorporate age, period, and cohort variables are faced with the inescapable problem of data inadequacy (due to an absence of observations on control groups). It is this shortcoming which has created the identification problem in traditional APC modeling and has encouraged the use of identifying restrictions which assume prior knowledge of existing causal relationships connected with age, period, or cohort. A descriptive formulation of the problem, such as the one we are proposing, avoids the ambiguities inherent in the use of such questionable restrictions and hence facilitates the task of model interpretation.

C) Preliminary results An exploratory application of this model [40] was made using French male mortality data for the period 1899–1981 and single-year ages 0–89 [30]. The observed probabilities of death are age- and cohort-specific and correspond to the French "Tables de générations". That is, q_{ij} gives the observed conditional probability of death (or proportion dying) between ages i and $i+1$ for the cohort $j-i$, whose members attain age i some time during the calendar year j. In our case, we

have $i = 0, ..., 89$ and $j = 1899, ..., 1981$. The result of using age-cohort data in the analysis is that we know with precision the age interval and the cohort membership corresponding to a probability, which is itself measured over two adjacent periods: when we refer to year j, the death may have occurred either in year j or in $j + 1$.

1. Additive structure

The top curve in Figure 2 hence shows the additive row effects, which represent the average age pattern of mortality over the entire period 1899–1981. Similarly, Figure 3 depicts the additive column effects, which demonstrate the average evolution of mortality from 1899 to 1981.

Figure 3 illustrates clearly the strong perturbations due to the two World Wars[3]. As much as these perturbations may be interesting for the differences

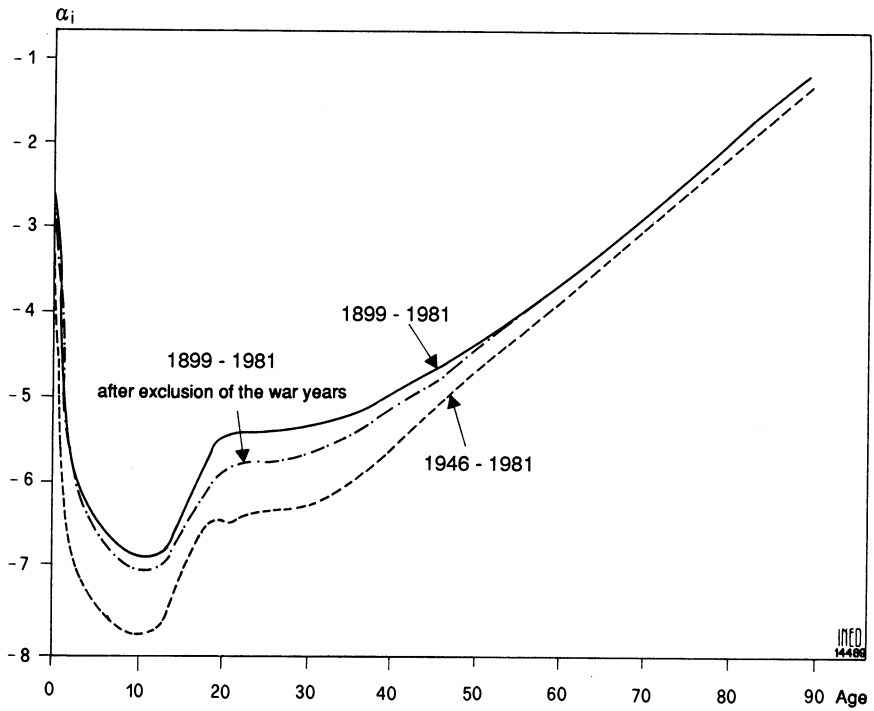

Figure 2. – Row effects, $\hat{\alpha}_i$, for various applications of the simple additive model. French males

[3]Here, 1913-1919 and 1939-1945, owing to the fact that age-cohort data are employed.

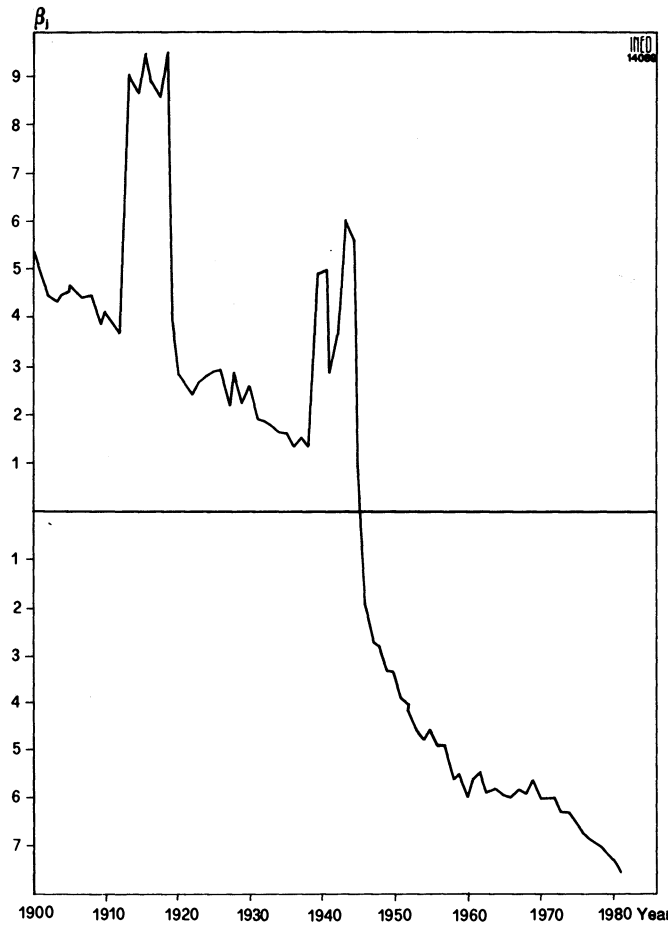

Figure 3. – Column effects, $\hat{\beta}_i$, for complete matrix (1899–1981). French males

which they introduce into the life histories of the cohorts studied, we will see that they may also disturb the analysis of the diagonal effects in the periods where they occur. For this reason, we have found it useful to restrict a large part of the analyses which follow to a period free of large fluctuations, i.e., the postwar era (1946–1981). The bottom curve in Figure 2 gives the average age pattern of mortality for this reduced period. The latter curve differs in level from the preceding one due primarily to mortality's general decline over time. The two curves also differ in shape, however, since this decline was particularly sharp at the youngest ages and since the adult ages in the period 1946–1981 are

exempt of all excess war mortality (in the same Figure can be seen, as well, the average age pattern of mortality for the period 1899–1981 after the exclusion of the war years).

2. Multiplicative terms

A portion of the variance which remains unexplained by the simple additive model is taken into consideration by the inclusion of one or more multiplicative terms. As concerns the entire period 1899–1981, it seems necessary to retain three multiplicative terms ($\rho = 3$) in order to describe the non-additive rectangular structure of the matrix, whereas two appear to suffice ($\rho = 2$) when the analysis is limited to the period 1946–1981. (The criteria for these choices are detailed in the Appendix to [41].)

It is somewhat difficult to illustrate the role played by these multiplicative terms. Figure 4 gives a contour map representation of the combined multiplicative effects [35,40]. The axes of the map consist of the column (x-axis) and row (y-axis) indices. The level of the map at each point is determined by the values

$$\hat{\phi}_1\hat{\gamma}_{i1}\hat{\delta}_{j1} + \hat{\phi}_2\hat{\gamma}_{i2}\hat{\delta}_{j2} + \hat{\phi}_3\hat{\gamma}_{i3}\hat{\delta}_{j3} \; . \qquad 4$$

The two darkest levels represent the positive values for the combined multiplicative effects; the two lightest levels, the negative values. A positive value for a given age and period indicates that the addition of multiplicative terms augments the predicted level of mortality from that foreseen by the simple additive model; conversely, a negative value indicates a decrease in the predicted level due to the inclusion of multiplicative terms.

Figure 4 can thus be read either "by rows" or "by columns". For a given age, within the general context of mortality decline, passing from a darker to a lighter region indicates that the decrease in mortality at that age has been faster than the average; on the contrary, the passage from a lighter to a darker region indicates a slower decrease, or even the absence of a decline. From this point of view, Figure 4 is notable for its division into four opposing sectors which contrast the ages above and below 40 years and the periods before and after the Second World War. Below 40 years of age, the decline has been more rapid than the average (and this is particularly true below the age of 10), while the opposite is true above age 40.

Similarly for a given period, as we advance in age, the passage from a darker to a lighter region indicates that mortality rises less quickly which age than in the average life table; and inversely, if we go from a lighter to a darker region. Again in this case, we may observe the division of Figure 4 into four sectors, illustrating the progressive transformation of the shape of the age curve of mortality.

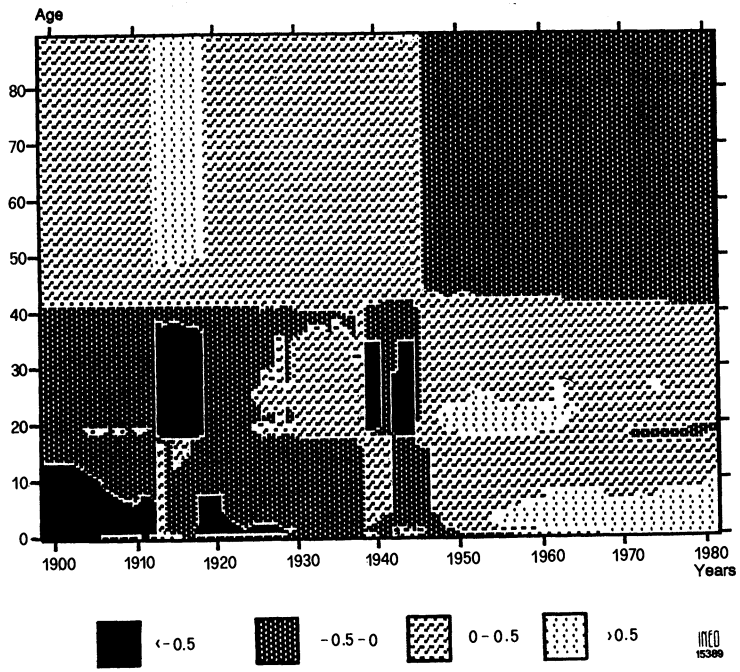

Figure 4. – Contour map representation of the combined multiplicative terms (i.e., $\sum_{m=1}^{3} \hat{\phi}_m \hat{\gamma}_{im} \hat{\delta}_{jm}$) for complete matrix (1899–1981). Non-additive rectangular structure, French males

Beyond these large-scale modifications with respect to the additive model, a careful reading of Figure 4 also allows us to identify certain particularities of the matrix which are brought out be the AM(3) model. We note above all, reading within rows, the atypical evolution at the ages which paid the heaviest price during the two wars. At these ages, excess war mortality produces multiplicative terms which are starkly positive. However, in the years which follow the wars, the multiplicative terms turn negative, due to the simple fact that the sum of the residuals from the additive model over a single row (or column) are, by definition, zero. We note also the peculiar case of youths aged 18-20. Strongly affected in recent years by a rise in mortality due to road deaths, these ages demonstrate an increase in the values of the multiplicative terms during the 1970s.

In the same manner, reading by columns, we may note the unusual case of World War I, during which excess mortality was so marked between ages 20 and

40 that the ages above 50 years are affected by strongly negative multiplicative terms. We see thus that the multiplicative terms offer a non-negligible possibility for correction of the additive model, going even so far as controlling for certain fluctuations which are highly period-specific.

3. Residual diagonal structure

We can thus be rather sure that the non-random portion of the residuals left after the fitting of the AM model may be attributed to residual diagonal structure in the data matrix, whose effect is represented by the terms θ_k for each cohort. Figure 5 illustrates the estimated diagonal effect, $\hat{\theta}_k$, in the case where we apply the model to the entire period 1899–1981 (the original estimates are shown along with smoothed version, obtained using robust smoothing procedures [36]). The cohorts[4] affected run from c.1810 to c.1981, hence those who cross the age range 0–89 between 1899 and 1981. Clearly, the results can hardly be considered significant at the extremes of the graph, since in those cases the cohorts are observed for very few years of time. It is best to ignore the cohorts from before c.1830 or after c.1960, for which the results appear to be subject to strong random fluctuations. For cc.1830-1890, there would appear to be no important non-zero diagonal effects; however, beginning with the cohorts born near the end of the last century, the estimated values, $\hat{\theta}_k$, demonstrate notable oscillations[5].

An initial group of cohorts, cc.1894-1904, with positive diagonal terms is followed by another group, cc.1905-1926, showing negative effects (with the exception of the positive values for the cohorts cc.1917-1919). Then, a second oscillation begins, with positive terms from c.1927 to c.1938, then negative ones from c.1939 to c.1949. Finally, a third oscillation seems to be asserting itself with a series of positive terms observed for the cohorts born in the 1950s.

We know that the value of 100 x $\hat{\theta}_k$ approximates the average percentage by which the observed mortality of a cohort differs from the reference level estimated by the appropriate AM model (see Appendix to [41]). When this diagonal effect is noticeably different from zero, we say that the cohort, or its mortality experience, is "peculiar". When the value of $\hat{\theta}_k$ is negative, we will speak of a "mortality advantage" or an "advantaged cohort"; when positive, of a "mortality disadvantage" or a "disadvantaged cohort". We may thus say that cc.1894-1904, cc.1927-1938, and cc.1950-1959 appear to be disadvantaged,

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[4] Following Ryder [26], we write "c." plus a year to refer to the birth cohort from that calendar year. Likewise, "cc." refers to a group of cohorts.

[5] In [39], we have argued that it is very unlikely that such oscillations would occur by chance variation alone. We will not, however, pursue the issue of statistical significance in the present work.

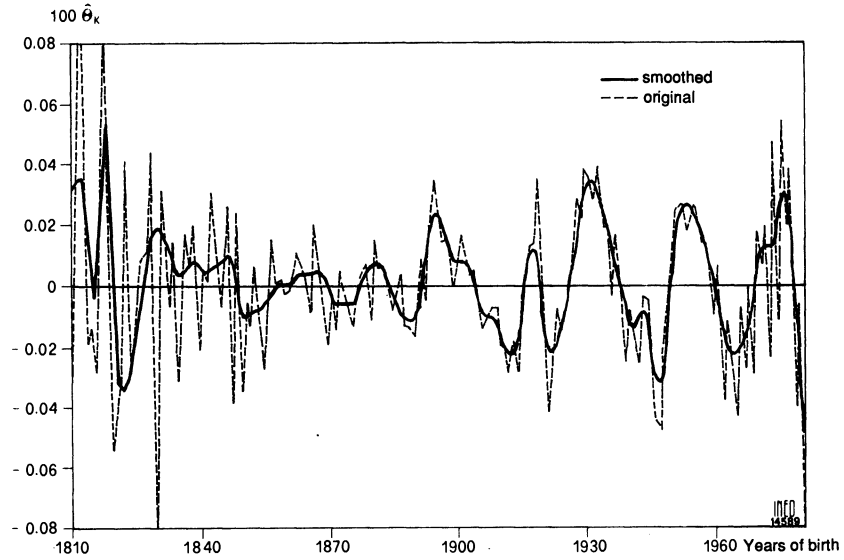


Figure 5a. – Estimated diagonal effects, 100 x $\hat{\theta}_k$, based on *complete matrix (1899–1981)* and *exploratory fit.* Residual diagonal structure, French males

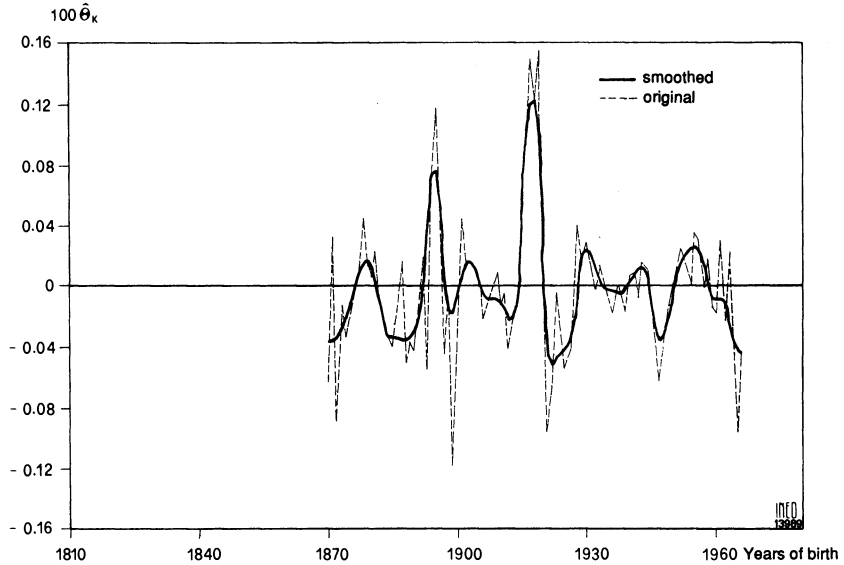


Figure 5b. – Estimated diagonal effects, 100 x $\hat{\theta}_k(15, 29)$, based on complete matrix (1899–1981) and exploratory fit, restricted to *ages 15-29.* French males

being characterized by a vulnerability which is higher than that which one might expect based on a simple consideration of the age pattern of mortality and its evolution over time. For the first two groups of cohorts, we are seeing most certainly some of the long-term consequences of the two World Wars, some of which have already been noted by other authors [1,3,4,7,8,16,25,28]. The case of the 1950s cohorts, however, raises new and interesting questions.

Since, by definition, the estimated diagonal effects are constrained to have a sum of zero[6], the cohorts demonstrating negative terms and hence appearing to be advantaged are most likely advantaged, in fact, only by comparison with the mortality disadvantage of the most vulnerable cohorts. The reference mortality of the AM model is necessarily the result of the average experience of the advantaged and disadvantaged cohorts, and hence any discussion of mortality advantage or disadvantage must always be understood in a relative sense.

In this paper we have chosen to concentrate our study on the disadvantaged cohorts and have built an interpretation of the observed cohort peculiarities around the assumption that the mortality advantage of the other cohorts is little or nothing more than a relative effect. Application of the same method to all-cause mortality data for several countries has given some support to an opposing hypothesis, namely that the advantaged cohorts are a select group of individuals who survived participation in the two World Wars. It is unclear at this moment which interpretation is the most consistent across countries, sexes, and causes of death, but we develop the former one for the time being since it seems *a priori* the more plausible, and since it is more consistent with previous discussions in both the demographic [16] and medical literatures [13].

The relation suggested by Figure 5a between the diagonal effects and the life history of certain generations during the two wars remains, however, rather unclear. The effect of the first war appears to be of a smaller magnitude than that of the second, whereas in general one would expect the opposite to be true. Furthermore, the particular case of the cohorts c.1917, c.1918, and c.1919 seems to require a separate explanation.

It is however possible, as we have already suggested, that the choice to analyze the matrix for the entire period 1899–1981, hence including the war years themselves, has perturbed the results in some unknown way. Although the multiplicative terms take into account a good portion of these period-based fluctuations, there may well remain traces of the most exaggerated deviations, which may have the effect of confusing the estimation of the diagonal term. The

[6] Actually, it is a weighted sum which must be zero:

$$\sum_k w_k \theta_k = 0,$$

where w_k is the number of observations in the data matrix for cohort k.

wars resulted in a brutal transformation of the typical age pattern of mortality, with very sharp perturbations and discontinuities apparent for the age groups involved in combat (in 1914, for example, a striking discontinuity can be noted between ages 17 and 18, where the observed probabilities of death pass abruptly from 5 to 82 per thousand). One can verify that the inability of the AM model to account completely for such perturbations contributed, at least in part, to produce locally maximum values of $\hat{\theta}_k$ in Figure 5a for c.1895 and c.1919.

In a more general sense, if our interest is in studying the long-term effects of various events on the future experience of certain cohorts, it would in any case be more logical to restrict our analysis to ages above the ones at which the said events occurred. The situation is rather complex to the extent that the diverse events which seem to modify subsequent cohort mortality experience happen at quite differing ages. A rather simple compromise consists of retaining only the years after the occurrence of the events of interest, which in this case is the period after the Second World War. This choice allows us to eliminate the disturbances due to the two wars and, at the same time, to avoid other difficulties of interpretation which inevitably accompany the analysis of a too-large matrix. In addition, this solution presents the advantage of basing the analysis on the high-quality data of the postwar period.

Figure 6 presents the diagonal effects obtained from an exploratory application of the method to the last 36 years of the same data matrix, hence for the

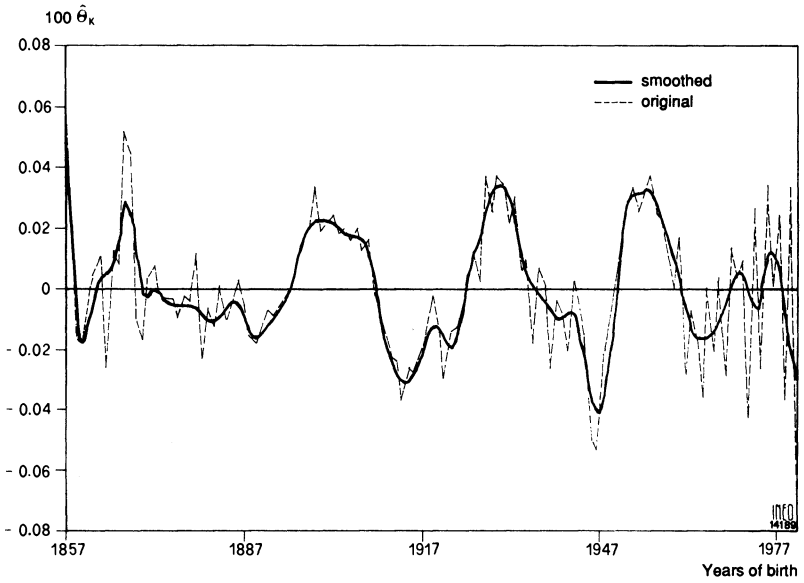


Figure 6. – Estimated diagonal effects, 100 x $\hat{\theta}_k$, based on *postwar matrix (1946–1981)* and *exploratory fit.* French males

period 1946–1981. The three oscillations in the diagonal term appear even more clearly than before. The long-term effects of World War I show here a greater magnitude, but the affected cohorts are slightly different: cc.1896-1909 instead of cc.1894-1904. We note also that the cohorts cc.1917-1919 no longer demonstrate positive effects. However, the negative series concerning cc.1910-1927 continues to be interrupted by three cohorts which, in contrast, show diagonal effects that are clearly less negative. To be precise, we must point out that the cohorts affected have changed slightly: the exceptions within this group are now c.1918, c.1919, and c.1920, instead of c.1917, c.1918, and c.1919.

D) Explanatory hypotheses This reading of Figures 5 and 6a requires a certain number of explanations which may differ from one group of cohorts to the next. In short, we must attempt to understand what has produced the mortality disadvantage of cc.1896-1909, cc.1927-1938, and cc.1950-1959, as well as the particular case of cc.1918-1920.

In the case of the first two large groups of cohorts, the link with the two World Wars seems rather obvious. Several mechanisms may come into play, however, according to whether we consider cohorts who were directly involved in combat, those who went through adolescence during the difficult years, or finally those who were born during the wars. The particular case of cc.1918-1920 may require a consideration of two types of explanations: participation in the Second World War, but also the potential effects of the Spanish flu, especially since the maximum observed effect is for c.1919.

The problem posed by the cohorts born during the 1950s is more delicate, as they seemingly have never suffered the effects of any wide-ranging crisis. In this regard, it would seem most inappropriate to point to the wars in Indochina (1950-1954) or Algeria (1954-1962), whose effects on French mortality were completely negligible in comparison to the two World Wars. These cohorts did, however, live through an important period in the history of French health care. It was essentially during the 1950s that childbirth in a hospital setting suddenly became the general rule. This change was accompanied by developments in obstetrical interventions which without a doubt allowed the saving of a large number of children who would otherwise have died at birth. It is entirely possible that, on the one hand, the first cohorts thus saved may have remained unusually frail afterwards and that, on the other hand, these interventions may have provoked a certain amount of obstetrical trauma. Indeed, one may note that mortality due to obstetrical lesions rose during this period [33]. At a later time, on the contrary, with the perfection of techniques of obstetrical intervention, childbirth in a hospital setting was able to produce in full its beneficial effects.

Clearly, these preliminary hypotheses, and most notably the last one, remain to be verified. It is not possible for us to provide a definitive confirmation at this time, but we will try all the same to determine, in the next section, if the

explanations proposed are consistent with the differences observed between the two sexes and with the distribution of the diagonal effects across different age groups. To this end, we have subdivided the 1946-1981 matrix into four large age groups (ages 0-14, 15-29, 30-64, and 65-89) and have analyzed the diagonal effects within each of these subdivisions. In a final section of the paper, we examine whether a consideration of cause-specific mortality can help us further to refine these explanations.

II. The consequences of differing life histories

In the preceding pages, we presented some preliminary results pertaining to the observed mortality advantage or disadvantage of certain male cohorts in France, both for the full period, 1899–1981, and a restricted one, 1946–1981. It was useful in this initial analysis to employ the simpler exploratory fit of the model described previously. In the discussion which follows, however, it will be necessary to employ the complete fitting procedure which, although more difficult to calculate, has the advantage of minimizing the effect of the multiplicative terms on the diagonal effects and hence of recovering the full magnitude of the latter. The use of the complete fit is most important when we wish to compare the diagonal patterns from different matrices, since each may require differing numbers of multiplicative terms (according to the criteria given in the Appendix to [41]). The remainder of this paper will effect such a comparison between male and female mortality and among various causes of death, in all instances for the postwar years in France.

A) Long-term effects of the two World Wars

1. Cohorts adult during the wars

A large number of cohorts took part in the First World War: for all cohorts born between 1869 and 1899, more than 60% of the members surviving in 1914 were eventually mobilized. For cc.1876-1898, this figure even rises to more than 80% [28]. Nevertheless, among these cohorts some were more massively involved in combat than others. Judging by the number of battlefield deaths, the cohort having suffered most directly the effects of the war was c.1894, which lost 27% of its members, followed closely by the other cohorts from c.1892 to c.1895, all of which were reduced by more than a fourth. From this point of view, the other cohorts were considerably less affected, although among

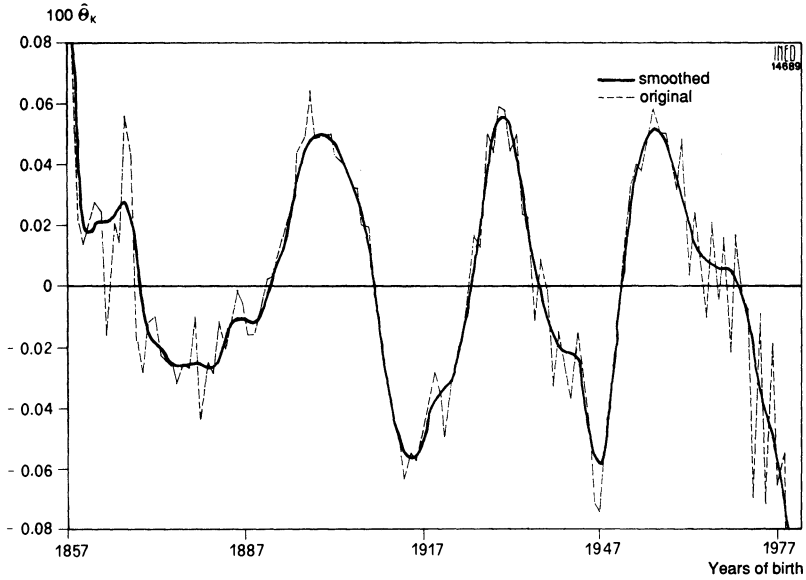
them are sure to be found an enormous number of wounded survivors and even many who were exposed to toxic gas warfare. Still more important than war wounds for the mark which they left on survivors, however, would have been the deplorable living conditions at the front and especially the problem of nutritional deprivation. From this perspective, it was probably the youngest soldiers who suffered the most, to the extent that their physiological and psychological needs were the greatest.

We would expect, therefore, that the maximum long-term effects will be found among cc.1892–1899, and indeed, in Figure 5a (analysis of the complete matrix), we may note a maximum diagonal effect for cc.1894–1895, framed by near-zero values for cc.1890–1891 on the one side and cc.1899–1900 on the other. In the same way, the cohorts having contributed the most heavily to the Second World War are cc.1915–1919 and in particular cc.1917–1918, of which more than 60% were taken as prisoners of war [28]. If we may again reason that the youngest cohorts should have been the most vulnerable, we may expect to find the heaviest long-term mark for cc.1917–1919, and this is indeed what we have already noted in an initial reading of Figure 5a.

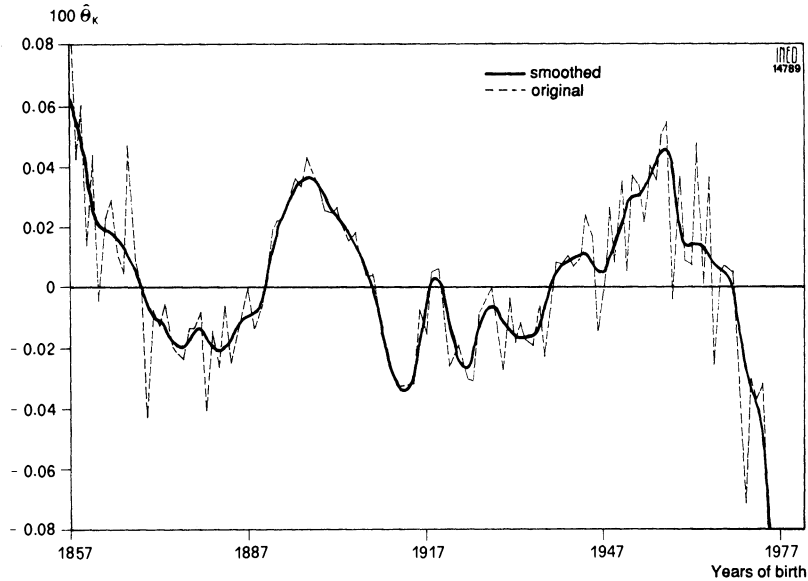
However, as already noted, Figure 5a is distorted by inadequacies in the AM fit as concerns the two World Wars and hence carries a risk of confusion between direct and long-term effects of these crucial events. It is possible to illustrate this point by analyzing the diagonal structure within each of the four chosen age groups of the matrix. For the age group 15–29 (Figure 5b), we find an exceptionally high diagonal effect (of the order of 7 to 15%) for cc.1894–1896 and for cc.1915–1919, an effect which can only be explained by the war losses themselves. On the contrary, this diagonal effect is much more modest at 30–64 years (of the order of 1 to 3% for cc.1894–1896 and negative for cc.1915–1919) and practically null at 65–89 years.

In fact, looking at Figures 6 (exploratory) or 7a (complete) and hence restricting the analysis to the postwar data (1946–1981), the cohort most heavily marked by World War I is c.1899, while for World War II, c.1919 appears the most affected at long-term, at least among those involved in combat. Said otherwise, in one case as in the other it is a question of the youngest cohort having been mobilized and not of those cohorts who were most directly involved in combat.

Since it is a question of the long-term effects of military operations, we may naturally think that women should not have been affected. It may thus be interesting to compare at this time the situation of men to that of women. Surprisingly, Figure 7b shows a movement for women which appears analogous to that of Figure 7a for men. For the First World War this movement is, however, of a somewhat smaller magnitude and does not concern exactly the same cohorts. Among females, cc.1891–1897 appear to be the most heavily affected, a group which is notably older than the analogous group of males (cc.1896–1899).



a) French males



b) French females

Figure 7. – Estimated diagonal effects, 100 x $\hat{\theta}_k$, based on *postwar matrix (1946–1981)* and *complete fit*

The relative disadvantage of these female cohorts can only be explained by a general deterioration of living conditions during the war. Although all women were undoubtedly touched to some extent by these adverse conditions, it was apparently pregnant women and young mothers who were the most seriously affected, since the cohorts showing the greatest mortality disadvantage later in life were, for the most part, between 20 and 25 years old between 1914 and 1918.

For females, the case of the Second World War is somewhat more complex since we observe distinctly positive values for cc.1918–1920. It would appear that such an observed mortality disadvantage can only be explained with reference to the Spanish flu, as we will see later on.

2. *Cohorts adolescent during the wars*

Among other authors on the subject of the long-term effects of the World Wars on survivors, Shiro Horiuchi [16] in particular has insisted upon the physiological weakening of cohorts having lived through adolescence during one or the other of the two wars. In our analysis as well, we observe quite clearly this phenomenon: whether for male cc.1900–1908 or cc.1928–1933, we find clearly positive diagonal effects (Figure 7a), and this proves to be true whatever the age group considered (above age 15). Horiuchi was able to document such a mortality disadvantage only in the case of males. Yet the male specificity of the phenomenon remains rather astonishing, since it seems difficult to imagine that young girls could have been so completely spared the effects of deteriorating living conditions during the wars. In fact, Figure 7b demonstrates that a similar weakening effect did indeed affect women as well. It is still a mystery as to why this effect is so notably moderated for women in comparison to men. Horiuchi put forth the hypothesis (originally proposed by Okubo [25]) that certain nutritional deficiencies during adolescence could provoke, more acutely in males than in females, certain "detrimental effects on the growth of the blood vessel structures", which would produce a situation favorable to the development of cardiovascular disease later in life.

In our opinion, this explanation is probably too specific. We should rather attempt to explain the lesser weakening of females in terms of a much more general phenomenon. Since the Second World War, improvements in health conditions have been both more rapid and more extensive for women than for men. The latter have additionally borne more heavily the weight of postwar industrialization [29,31]. For these reasons, the contrasts between male cohorts may have been reinforced, whereas those differentiating female cohorts may have been attenuated.

3. Cohorts born during the wars

Given the particular fragility of new-borns, we may well wonder whether war infants do not experience, in adulthood, the long-term consequences of having been born during a period of unusually adverse conditions. This is indeed what has been observed in the case of Italians of both sexes born during the First World War [3,4,7,8], although a comparison of French and Italian data has shown equally that this phenomenon is not generalizable and was linked most likely to a more acute deterioration of the Italian health environment during World War I [7]. In any case, we do not find in this study any notable weakening of French cohorts born during the wars, with the exception of c.1917 and c.1918, for which we have already put forth other explanations.

B) Long-term effects of the Spanish flu The winter of 1918–19 has gone down in history for having known the most severe epidemic of influenza in modern epidemiological history, the Spanish flu, which was followed by a recurrence of lesser severity in 1920. We have already advanced the idea that the mortality disadvantage of cc.1918–1920, clearer for females than for males, may be at least in part one of the long-term consequences of this epidemic. This proposed link may seem contrary to common sense, in that we know that individuals who survive a viral infection have the advantage of indefinite immunity. We know also, however, that this immunization, being limited in the case of influenza to a specific form of a virus which may mutate quite frequently, is actually of little value. On the contrary, it would seem that the Spanish flu may have weakened certain generations in two different ways. First, new-borns, being extremely vulnerable, may have developed various complications, especially of the respiratory system, which left their after-effects. Second, as with all viral illness, the Spanish flu must have been the source of numerous congenital deformations for the children of women struck ill during early pregnancy. The Spanish flu having been rampant above all at the end of 1918 and somewhat more moderately at the beginning of 1919, the cohorts which could have retained the heaviest long-term scar are those born from 1918 to 1920, hence being those who were less than a year old at the time of the epidemic or who were conceived in the three preceding months. In fact, these are the cohorts who, whether for males of females, demonstrate the most notable mortality disadvantage in Figure 7. Furthermore, if we treat separately the various age groups within the complete matrix (periods 1899–1981), we realize that this weakening effect was already particularly apparent between 0 and 15 years.

It is true as well that c.1919, among those cohorts directly involved in combat in World War II, was the one which seemed to suffer the greatest mortality disadvantage later in life. This cohort has thus accumulated various adverse

effects throughout life: first those of the Spanish flu epidemic, to which were added those of the war. In comparing Figures 7a and 7b, however, it is clear that the weakened condition of females born in this year comes out more clearly than that of males, in part due to the effect of smoothing. We can only emphasize that the two graphs are not, from this point of view, completely comparable. Among men the peculiarity of cc.1918–1920 can be observed only within a larger and more important oscillation, an oscillation due, as noted, to the long-term effects of the two World Wars. The result is that the particular case of the male cohorts from these years appears relatively less important. When considered relative to the larger movement, however, we see that this is an erroneous impression. On the other hand, since among women the Spanish flu constitutes the principal factor of cohort differentiation, the peculiarity of these cohorts comes out quite clearly.

C) Cohorts born in the 1950s We have already put forth the hypothesis that the vulnerability of the 1950s cohorts observed in Figure 7 could be due to sudden changes in obstetrical practices, whereby nearly all children were suddenly being born in a hospital setting and many survived thanks only to the recent development of various obstetrical interventions. This explanation will remain rather fragile as long as we have available only a very limited data series on these cohorts (the last cohort, c.1959, was only 22 years old in 1981).

Neither must we forget the possibility that this result might be artifactual, due in effect to an inadequate description of the rectangular structure of the data matrix by the AM(2) model. It is well known that the cohorts of the 1950s were particularly affected by a rise in roadway mortality among young people observed up until the middle of the 1970s. Figure 4 has shown, however, that the multiplicative terms managed to control rather well for this phenomenon. At any rate, there is less risk in this case than in the case of the two wars, since the changes here are much more progressive and are not followed by a sudden reversal. Furthermore, when we analyze the diagonal effects separately for each age group, the mortality disadvantage of the 1950s cohorts appears already in the first age group (0–14 years), and it is not particularly stronger in the second group (15–29 years) where accident mortality is the most important.

This phenomenon is evident among women as well as men. Figure 7b demonstrates, for the female cohorts of the 1950s, positive values of $\hat{\theta}_k$ which stand in constrast to the estimates for the surrounding cohorts, even though the latter are not strongly negative.

The generalization of hospital-based childbearing, while perhaps being the principal factor producing the mortality excess for these generations, may not be the sole cause. Use of the drug thalidomide, for example, has been linked to various congenital accidents towards the end of the 1950s. Use of this drug,

which has been banned in France since 1961, may have helped to reinforce the relative disadvantage of these cohorts.

III. Does an analysis by cause of death lend further insight?

The long-term, debilitating effects of these traumatic events seem to have been of a rather general nature for the cohorts concerned. We can wonder nevertheless whether some causes of death have proven to be more sensitive to this weakening process than others, especially in terms of disease etiology. We employ for this purpose an etio-anatomic classification system developed at INED [33,34], from which we retain only seven of nine large etiological categories[7] and three particular etio-anatomic cross-classifications:

1. infectious and parasitic disease,
2. malnutrition, intoxication, allergy, etc.,
   a. nervous system,
   b. liver and biliary ducts,
3. accidents and homicides,
4. neoplasms,
   a. respiratory system,
5. hereditary and congenital conditions,
6. degenerative disease,
7. suicide.

The data employed are the product of a reconstruction of cause-specific mortality data for the period 1925 to 1978 carried out at INED [33]. This reconstruction was not able, however, to reproduce the precision available in the case of total mortality. On the one hand the cause-of-death data on which the rates are based were available only by five-year age groupings. The cause-specific probabilities by single-year age groups were estimated by splitting the all-cause probabilities according to the proportion of total deaths due to the given cause within the appropriate five-year age group. Furthermore, we are dealing in this case with age-period death probabilities (representing exactly the calendar year of the event but straddling two cohorts), rather than age-cohort data as before. In this case, the cohort k is in fact a combination of cohorts k and $k-1$. This circumstance presents no difficulty in most cases, but does affect the results quite seriously for cohorts born during years in which a war produced an abrupt change in the monthly birth rate. As a result, the diagonal effects for c.1915 and c.1920 are consistently over-estimated, while for c.1916 and c.1919 they are under-estimated. We must therefore accept in advance that an analysis by cause of death will regrettably lend no further insight into the nature of the debilitation affecting cc.1918–1920.

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[7] We hence exclude the categories referring to diabetes and ill- defined conditions.

A) A rather general peculiarity The cohorts for which we have already noted the existence of a relative debilitation effect in the case of all-cause mortality, seem in fact to demonstrate a peculiar mortality experience for the majority of the various causes of death. This is particularly true for males, for whom cohort peculiarities are clearly the most important.

Table 1 presents the average value of $\hat{\theta}_k$ for four groups of male cohorts over the period 1946–1978, as well as the number of multiplicative terms, ρ, included in the model in each case. The cohorts chosen are all those for whom we have noted a mortality disadvantage, with the exception of cc.1918–1920. The value of $\hat{\theta}_k$ gives the approximate percentage of excess mortality relative to the level estimated by the AM model. For the first three groups of cohorts, each of the large etiological categories analyzed shows clearly positive values, with the exception of accidents and suicides.

TABLE 1. – AVERAGE DIAGONAL EFFECT (100 x $\hat{\theta}_k$) FOR FOUR GROUPS OF DEBILITATED COHORTS. FRENCH MALES, 1946–1978.
(The values displayed below represent the approximate average percentage of observed excess mortality, cf. Appendix to [41])

Etio-anatomic categories	Group of peculiar cohorts (1) 1895-99	1900-08	1928-33	1951-59	ρ
All causes	4.2	3.7	5.1	4.0	2
1. Infection	1.1	3.7	0.3	13.1	2
2. Malnut., intox., etc. (2)	6.3	10.1	5.1	...	1
a. nervous system (2)	−0.1	2.7	15.3	...	2
b. liver et b. s. (2)	3.0	−1.5	13.4	...	3
3. Accidents	−2.8	1.3	8.0	−1.4	2
4. Cancers	4.4	3.6	5.7	−4.2	2
a. respiratory sys. (2)	12.5	8.0	11.1	...	1
5. Hered. or cong. dis. (3)	...	...	...	−2.1	2
6. Degenerative disease	3.3	3.8	3.2	2.8	2
7. Suicide	−3.9	3.7	3.1	...	1

(1) Excluding cc.1918–1920, for which cause-specific data are inadequate.
(2) Analysis restricted to ages 25–89 to avoid perturbations due to highly erratic data below age 25.
(3) Analysis restricted to ages 0–14.

The situation of the 1950s cohorts is somewhat less clear, even though they do demonstrate strongly positive values for infectious disease, accidents, degenerative disease, and suicide. It should not be surprising that this same peculiarity does not show up in the case of cancer, since even the oldest cohort in this group was only 28 at the end of the period being studied. It is *a priori* more astonishing to note the same absence of a peculiarity in the case of hereditary and congenital conditions, when we have put forth the hypothesis of a weakening linked to congenital malformations or obstetrical accidents. Under such circumstances, however, it seems unclear whether the declared cause of death would correctly reflect the antecedents of the disease. On the other hand, it may appear rather curious that degenerative disease should play an important differentiating role for such young generations. It seems quite likely, therefore, that the after-effects of congenital maladies may have often been assimilated into the category of degenerative disease.

What appears most striking in Table 1, then, is the similarity of diagonal structure observed for males in each of the large etiological categories. This result was perhaps not expected, but is nevertheless quite reasonable. It is easy to understand that cohorts which have been weakened in one way or another are less resistant to the onset of infection, cancer, or degenerative disease, which clearly pose a lesser threat to an organism which is in a perfect state of health. The same is surely true for the grouping malnutrition, intoxication, etc., but we may be at a loss to explain why in this case the diagonal effect is much stronger than in the case of total mortality. It is likely that these cohorts are even more susceptible to diseases related to malnutrition or intoxication due not only to their weakened state, but also to an elevated exposure as a result of either behavioral peculiarities or less favorable living conditions. Similarly, in the cases of accidents and especially suicides, we may ask whether the observed excess mortality is linked merely to a lower chance of surviving a violent event or also in part to a greater exposure to risk. Individuals having undergone grave and traumatic experiences in the past and perhaps living permanently with a physical or psychological scar as a result of those experiences, may show an unusually high propensity towards suicide.

As concerns the question of behavior, it is necessary to mention also the phenomenon of smoking. Within the fourth category, we have purposefully isolated neoplasms of the respiratory system, which are known to be directly linked to the consumption of tobacco. The diagonal effect proves to be more pronounced for these cancers than for the others, most notably for the cohorts who participated in the First World War. It was essentially among World War I recruits that the consumption of tobacco in France became a widespread phenomenon. It is also possible that, as in the case of suicide, the propensity to smoke is greater among individuals who retain the memory, or indeed the physical effect, of unpleasant and traumatic experiences from the past.

B) Certain particular cases all the same This relative uniformity among the principal causes of death in the analysis of cohort peculiarities is not however without nuance. On the one hand, within the grouping malnutrition, intoxication, etc., alcoholism plays an unusual role which proves to be different from one group of cohorts to the next. On the other hand, what we have noted for men does not always prove to be the case for women. Finally, a new group of peculiar cohorts appears in the analysis by cause of death which had not been noted in the study of all-cause mortality.

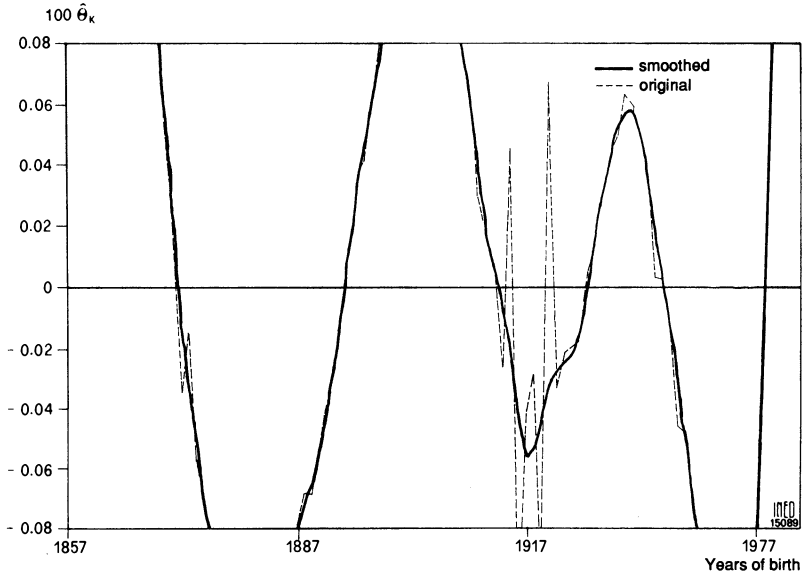
1. Malnutrition and alcoholism

Figure 8a depicts the values of $\hat{\theta}_k$ for males within the etiological category malnutrition, intoxication, etc. (restricting the analysis to ages 25–89, as noted in Table 1). The long-term mortality disadvantage of the cohorts most heavily affected by the two World Wars appears quite distinctly. We may note however that the range of affected cohorts is broader than that which we have considered up to the present: the values of $\hat{\theta}_k$ are systematically positive from c.1894 to c.1914. Additionally, another group of cohorts (cc.1863–1873), which we have never before mentioned, appears here to be characterized by a mortality disadvantage in the case of malnutrition. Even if these cohorts begin to be close to the ends of the graph, hence increasing the likelihood of random fluctuations due to a scarcity of observations, the contrast with the surrounding cohorts is so large that we may not dismiss the results as statistically insignificant.

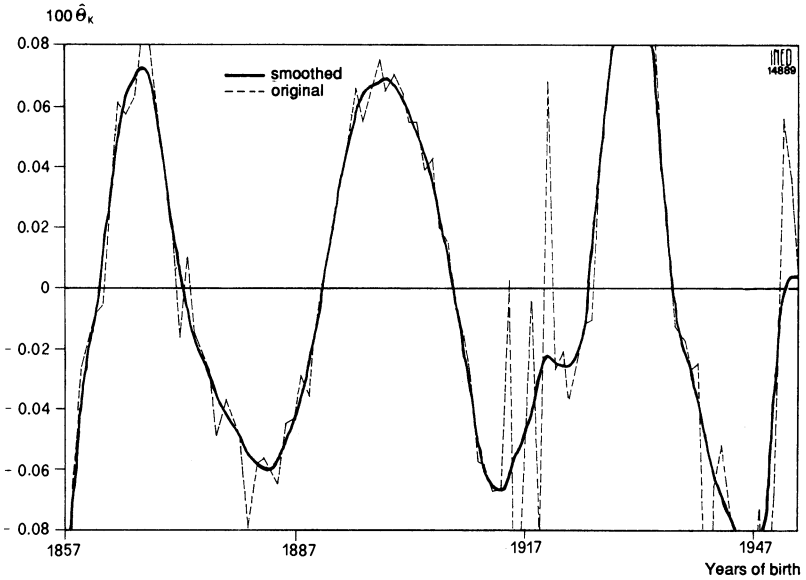
It is known that the grouping malnutrition, intoxication, etc., is rather heterogeneous[8]. It includes essentially two types of causes of death: various endocrine and metabolic disorders related to malnutrition, and all conditions related to alcohol abuse. The portion attributable to each of these two pathologies has evolved considerably over time. Previously, it was endocrine and metabolic disorders which were dominant. Today, it is alcoholism.

This circumstance allows us to understand why, in Table 1, the diagonal effect is quite strong for each of the first three cohort groupings when we consider the entire etiological category, whereas it appears progressively and shows its full effect only for the third group of cohorts when we isolate conditions of the nervous system (essentially alcoholic psychosis) and of the liver (alcoholic cirrhosis). Mortality linked to alcohol grew remarkably in France in the fifteen years following the Second World War and, although reaching a maximum in the 1960s, has remained very high ever since [31,33]. The cohorts weakened

[8]The logic behind this category is that it regroups conditions which constitute the organism's response to contact with or absorption of foreign substances.



a) French males



b) French females

Figure 8. – Estimated diagonal effects, $100 \times \hat{\theta}_k$, for the etiological category *malnutrition, intoxication, etc.* Postwar data (1946–1978)

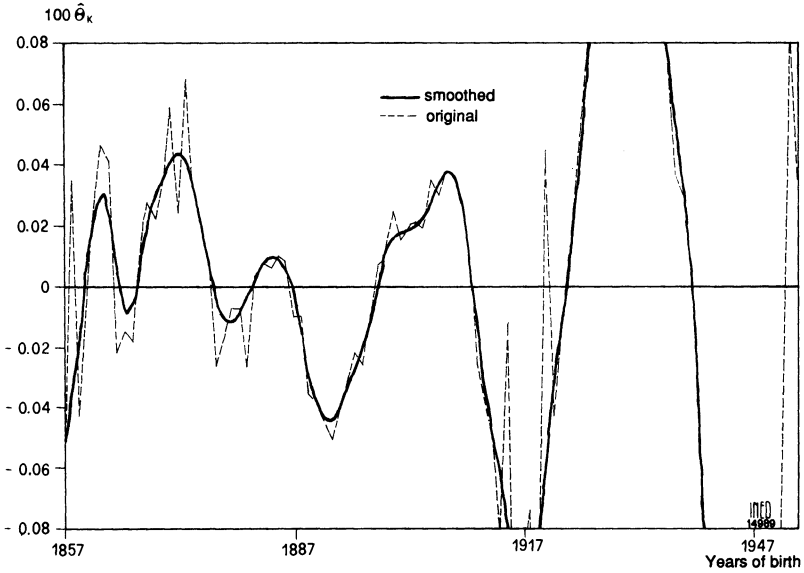
by the First World War experienced this rise in alcoholism rather late in life, whereas the younger cohorts (cc.1928–1933) have suffered its full effect.

Concerning alcohol itself, we note an important difference between conditions of the nervous system and those of the liver. In both cases (Figures 9a and 9b), the long-term effect of the Second World War is quite evident. On the contrary, the effect of the First World War is absent (or slightly negative) for cirrhosis of the liver, whereas it appears rather clearly for alcoholic psychosis. In the latter case, however, it is above all a question of cc.1905–1910, and thus of a group which is notably younger than the one to which we have normally referred. The development of cirrhosis of the liver generally requires an early and ample exposure to alcoholic consumption, whereas death related to alcoholic psychosis is most often the consequence of a more rapidly developed alcoholic condition (leading ultimately to *delirium tremens*). We may thus understand how the excess mortality observed for cirrhosis of the liver affects only the generations weakened by the Second World War, whereas the mortality disadvantaged in the case of alcoholic psychosis touches also the youngest of the cohorts weakened by First World War (the older cohorts from this group remaining exempt from this post-1945 phenomenon).

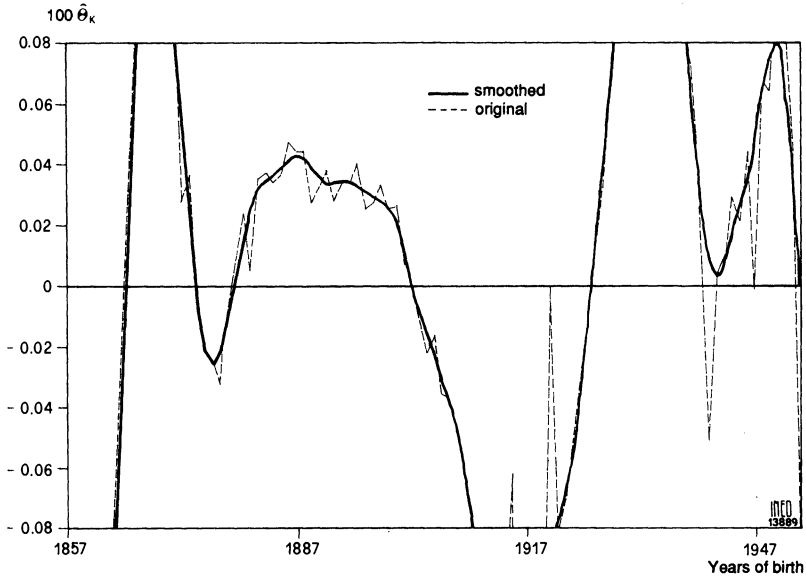
The mortality disadvantage in the case of the etiological category malnutrition, etc., for cc.1894–1904 seems to be linked very weakly to alcoholism and must thus be due to endocrine and metabolic disorders. The same may be said of cc.1863–1873. On the other hand, for cc.1928–1933 the average values of $\hat{\theta}_k$ for alcoholic conditions are clearly higher than for the entire etiological category (see Table 1). This result underlines the prominant role of alcohol in the excess mortality of these cohorts.

2. *The case of women*

The disparities observed between female cohorts, while being less apparent at the level of all-cause mortality, are also less consistent from one cause to another. What is rather striking in an examination of Table 2 is that there exist groups of female cohorts for whom the overall level of debilitation seems negligible, but who nevertheless demonstrate a noteworthy mortality disadvantage for certain causes of death. The two most notable examples include first the etiological category malnutrition, intoxication, etc., for which we observe clearly positive values of $\hat{\theta}_k$ in the three cohort groups affected by the wars, and second the cohorts born in the 1950s for whom an already significant mortality disadvantage overall appears even more clearly for infection, accidents, and degenerative disease.



a) Disorders of the *nervous system*



b) Disorders of the *liver and biliary ducts*

Figure 9. – Estimated diagonal effects, $100 \times \hat{\theta}_k$, for etiological category *malnutrition, intoxication, etc.*, by anatomical location. French males, postwar period (1946–1978)

TABLE 2. – AVERAGE DIAGONAL EFFECT (100 x $\hat{\theta}_k$) FOR FOUR GROUPS OF DEBILITATED COHORTS. FRENCH FEMALES, 1946–1978.
(The values displayed below represent the approximate average percentage of observed excess mortality, cf. Appendix to [41])

Etiological categories	Group of peculiar cohorts (1) 1891-99	1900-08	1928-33	1951-59	ρ
All causes	3.6	1.6	−1.3	3.0	2
1. Infection	−10.2	−5.3	2.8	7.1	2
2. Malnut., intox., etc. (2)	6.5	4.0	9.1	...	2
3. Accidents	−3.7	−4.9	2.8	3.9	2
4. Cancers	−2.0	−2.1	−0.9	−3.2	2
5. Hered. or cong. dis. (3)	...	...	...	−0.4	2
6. Degenerative disease	7.0	4.6	−4.6	7.0	2
7. Suicide	−1.8	1.1	−1.0	...	1

(1) Excluding cc.1918–1920, for which cause-specific data are inadequate.
(2) Analysis restricted to ages 25–89 to avoid perturbations due to highly erratic data below age 25.
(3) Analysis restricted to ages 0–14.

Figure 8b, showing for women the values of $\hat{\theta}_k$ for the etiological category malnutrition, intoxication, etc., offers in effect a pattern which appears very similar to Figure 8a for men. Although situated at a much lower level for women than for men, alcohol-related mortality has followed exactly the same evolution for the two sexes, and thus all which we have said for men concerning the respective roles of endocrine and metabolic disorders and alcoholic psychosis applies also in all likelihood for women. In addition, the particular case of cc.1863–1873 already noted for males shows up again for females. It is still not understood why the diagonal contours for females come out much more distinctly for this etiological category than for the others, a result which remains rather mysterious.

Concerning the female cohorts born in the 1950s, a consideration of causes of death proves to be particularly interesting, in that it confirms the existence of a significant mortality disadvantage for certain causes precisely where we were hesitating to proclaim the result significant in the case of overall mortality. Furthermore, among the causes implicated as the sources of the observed excess mortality for these female cohorts, we find the same ones already underlined for males: infection, accidents, and degenerative disease.

3. The case of cohorts 1863–1873

Figure 8 brought out a non-negligible mortality disadvantage affecting cc.1863–1873, for men as well as women. A careful examination of Figure 7 would have already allowed us to note an elevated value of $\hat{\theta}_k$ for c.1867 and c.1868, again for both sexes. At the time, however, it did not seem advisable to put much weight on such a result, given the location of these cohorts in a somewhat isolated corner of the data matrix. Thus once again, an analysis by cause of death has permitted us to verify the existence of situations whose peculiarity was not completely apparent at the level of overall mortality. Indeed, we observe a mortality disadvantage for the cohorts born around 1867–1868 in the cases of infectious disease and, although less clearly, of degenerative disease. Might we imagine that the cholera epidemic of 1867–1868 could have left a long-term scar on cohorts who were infants at that time? It is a hypothesis which may merit looking into.

By way of conclusion

Although the interpretation of the descriptive results presented in this paper must be considered tentative, we may attempt nevertheless to put forth a few conclusions with regard to the contribution of this new method for describing the data, to the validity of the diagonal mortality contours thus documented, and to the fundamental hypothesis retained for their interpretation.

The systematic exploration of the structure of the matrix of death probabilities has allowed us not only to confirm several unusual characteristics already outlined by other authors, but also to bring out previously unknown rough spots affecting other peculiar cohorts. Thus have we been able to document the relative mortality disadvantage of the cohorts born or conceived during the Spanish flu of 1918–1919 or during the 1950s, as well as to note the particular case of the cohorts born around 1867–1868. It seems to us, therefore, that the method employed in this work has at least the advantage of providing a more comprehensive description of the available data. Additionally, the extension to an analysis by cause of death made it possible to observe that, beyond the large-scale occurrences which seem to have an impact on all causes and thus show up clearly at the level of overall mortality, there exist as well more specific phenomena which, although limiting their effects to certain causes, do not prove to be less significant.

It is nevertheless possible to wonder whether this description is indeed an accurate representation of reality. From this point of view, we must raise questions as much about the data themselves as about the method. As regards the data, a first reassurance is that, when the same method is applied to various countries having known quite similar histories, the results resemble strongly those presented in this work for France [39]. Secondly, we may add that the

French data employed seem to us to be particularly well suited to this type of analysis, in that they are based upon an excellent death registration system by both age and year of birth and upon an abundant series of censuses undertaken at rather irregular dates[9]. Concerning the method, we may first have confidence in its validity due to the fact that several findings already outlined by other authors with different methods have been confirmed by the present one. Secondly, application of the method [39], both to an original matrix composed of appropriately chosen model life tables and to the same matrix after having introduced artificial diagonal contours and simulated random noise, has helped to remove residual doubts concerning the accuracy of the description provided: in no instance did an unexpected diagonal effect appear in the analysis of either the original or modified matrix, whereas the simulated diagonal effects were brought out with reasonable accuracy by the analysis.

It remains to be noted that we have deliberately favored the fundamental hypothesis that the peculiar cohorts characterized in our analysis by positive values of $\hat{\theta}_k$ have undergone a process of debilitation, as opposed to the alternative hypothesis that other cohorts, characterized by negative values of $\hat{\theta}_k$, are showing the after-effects of a selection process. We have thus preferred, for example, to consider that the cohorts having lived through adolescence during the war years were weakened, rather than to uphold the notion of a selection effect due to excess infant mortality among cohorts born during the wars or to excess adult mortality among cohorts involved in combat. As noted earlier, the hypothesis of a selection effect has found some support in comparisons of cohort mortality patterns among several countries involved in the two World Wars. At this time, however, neither interpretation appear to be completely adequate, which indicates the need for further comparative work across countries, sexes, and causes of death in order to determine the most plausible explanation for the contours of cohort mortality outlined in the present work.

Allow us to insist, one last time, on the fact that the cohort peculiarities which we have documented and have attempted to place in their proper historical perspective, may be said to represent merely the emerging tip of the iceberg of all conceivable cohort effects in the broadest sense of the term. Nevertheless, the approach of analyzing intercohort heterogeneity has allowed a greater appreciation for the importance of this third component, and has fostered an increased understanding of at least a portion of the influence which it wields in the structure and evolution of mortality.

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[9]During the 37 years of the postwar era on which we have concentrated our study, six censuses were carried out: 1946, 1954, 1962, 1968, 1975, 1982. Being based on intercensal intervals which are hence quite irregular, the reconstruction of age-specific population totals on January 1 of each year hardly runs the risk of a systematic inaccuracy, even at advanced ages, due to any tendency towards age-heaping.

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