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# Two of a kind? Short-term shocks and the demographic transition in the European demographic history

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# Two of a kind? Short-term shocks and the demographic transition in the European demographic history

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## Abstract

Two types of interaction between mortality and fertility have thus far been identified: short-term (e.g. after a mortality crisis) and long-term (in the demographic transition). This paper suggests that the underlying connection between the two phenomena is instead unique, and that the differences between the various cases (countries and centuries) lie, rather, in the evolution of the independent variable (death rates).

Modern statistical tools (analysis of time series; identification of structural breaks) applied to ancient, aggregate data (Chesnais 1992) to twelve European countries help shed a new light on the demographic mechanisms that guide the dynamics of human population.

## 1. Introduction

Mortality and fertility interact in several ways. Two types of interaction, in particular, have attracted the attention of historical demographers. One is the theory of the demographic transition: a steady decline in mortality brings about a marked and persistent decline in fertility. The other is the "theory" of demographic recovery: when a mortality crisis alters the traditional equilibrium of a population, a series of adjustments follows (higher nuptiality and, later, higher fertility), so that equilibrium is eventually restored.

To the best of our knowledge, these two approaches have thus far been considered separately. Indeed, the former (the demographic transition) applies to the long run, and the latter (recovery) to the short run; the former deals with permanent changes, and the latter with transient ones; the former marks the passage to modern times, while the latter only works in ancient demographic

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regimes. What we contend, instead, and will try to show in this paper, is that the two theories describe basically the same mechanism: an exogenously determined variation in mortality (crisis or decline) affects the subsequent path of fertility.

While the existence of this "causal" link (in a sense that will be better specified later) may lie in the back of most demographers' mind[4], nobody could actually prove it, to the point that some authors questioned it, especially in the eighties: e.g. Coale and Watkins (1986); Cleland and Wilson (1987); Szreter (1993)[5].

In the nineties, however, the idea of a relationship between mortality and fertility re-emerges (e.g. Hirshman 1994; Bongart and Watkins 1996; Wilson 1999; Cleland 2001). Galloway, Lee and Hammel (1998), in their critical review of the literature on the relationship between mortality and fertility in Europe, confirm that the two (mortality and fertility) seem to be scarcely related in terms of *levels*, but their connections are stronger in terms of variations, instead. This is consistent with our view that the core of the homeostatic regulation of population dynamic lies in a lagged relationship between mortality and fertility: the present values of fertility depend on the past values of mortality. This lag may explain why regressing fertility on mortality at the same epoch generally results in a weak association.

The novelty of our approach lies in the use of the techniques of modern time series analysis that allow researchers to detect the existence of lagged relationships between variables: SVAR models (Structural Vector Auto-Regressive) and the method of Bai and Perron (2004) for the identification of structural breaks in time series.

We apply these methods to simple series of birth and death rates in 12 European countries, and we try to describe the type of relationship that seems to emerge between mortality and fertility.

[4] "It is perhaps surprising that while mortality decline is usually cited as the *raison d'être* for fertility decline, it is not often accorded a primary place as a cause of fertility decline. This is understandable, since efforts to establish a direct close connection have had mixed results. Whilst definitive proof of this connection may not be possible, there exist cogent reasons for supposing that it exists" (Kirk, 1996).

[5] "A fall in mortality (especially infant mortality), it was suggested, should stimulate and, therefore, precede the drop in marital fertility. The demonstration that no such straightforward link existed in the European transition is one of the most striking of recent findings. In England and Belgium, for example, in most areas the decline in fertility preceded that of infant mortality. In few countries did change consistently follow the expected pattern. Moreover, within countries, only weak correlations between infant mortality levels and fertility were detectable at best, and in most cases no statistical significant relation existed. Nor was it necessary for infant mortality to fall to any particular level before the fertility decline began" (Cleland e Wilson, 1986, p. 18).

Our main conclusion is that mortality today impacts on what happens tomorrow, in terms of both fertility and mortality, while the impact of today's fertility on future vital rates is negligible.

We take our data from two sources: up to 1985 we use Chesnais (1992), whose data practically coincide with Mitchell's (2003); from 1985 on, we use Eurostat data. The reason why we prefer rough measures (crude birth and death rates) to more refined indicators (e.g. life expectancy, or total fertility rates) is that we need our series to go as far back into the past as possible. Notice, also, that we are not interested in the absolute value of these indicators: instead, we focus on variations over time. This type of exploitation tends to reduce the bias due to differences in the age structures of our populations (Dyson and Murphy 1985: 433). In any case, the crude birth and death rates series are highly correlated with the TFT and the $e_0$ series.

For reasons of homogeneity, we need our data to refer consistently to one calendar year: this is why the English dataset starts only from the beginning of the 19th century: indicators are available also for earlier periods, but not with the yearly detail that we need.

In our series, a few data (years) may be missing: when this omission is quantitatively limited, and refers to years that we know to have been "normal", we simply interpolated; in other cases, however (Greece, for example), we preferred to discard the whole country from our analysis. Please bear in mind that, in the course of this paper, "mortality" consistently means "crude death rate", and "fertility" invariably stands for "crude birth rate".

Beyond this introduction, there are five sections in this paper: sections 2 and 3 discuss the stationarity of our series of birth and death rates. This is not merely a technical point: it is important, because the SVAR method works properly only in the stationary case. The method itself is (briefly) presented in Section 4. Results for the 12 countries under scrutiny come in Section 5 (short term) and 6 (long term).

## 2. Stationarity or non stationarity? (This is *the* question)

Stationarity makes life simpler when one wants to test whether two or more time series influence each other. Unfortunately, the birth and death rates we are dealing with are not stationary: this is why we need to properly transform them before we can proceed.

A time series is said to be (weakly) stationary when its average and its covariance function do not vary with time, or, in simpler words, when the series moves more or less at random around its average. There are basically two cases for stationarity:

a) no statistical dependency exists between the value at time *t* and the preceding values, at times *t-1*, *t-2*,... (*white noise* process), or

b) there is *mean-reverting*, that is the average acts as an attractor (as, for example, in ARMA processes). In this case, shocks may occur, but then some "force" brings the series back to its original path. After a relatively short while, the shock is, so to say, "forgotten", and leaves no permanent trace.

We are basically interested in case (b) in this paper: mortality shocks did occur in the past, but did they leave a durable mark? Let us first try a qualitative answer.

Before the demographic transition, death rates were normally somewhere between 30 and 40 per thousand. Crises were not infrequent, and they would raise death rates up to, let us say, 150 per thousand. After the shock, however, death rates went back to their "normal" levels: no permanent effect, then[6].

Things obviously change during the demographic transition. This, however, can still be analyzed with the same logic if one admits that the series of vital rates now have a trend: oscillations continue more or less as before, but now around a decreasing average ("trend-stationary series"). And even during this phase, the exogenous shocks that occur (e.g. World War I, the Spanish influence, World War II) leave no permanent consequence.

In short, what we contend is that the series of the vital rates are basically stationary: at high levels before the transition, at low levels after the transition, and with a trend during the transition. This is a case known as *structural break* (Perron 1989, 1994), and it can explain why the classical tests (ADF, Phillips-Perron ecc.) frequently reject the stationarity hypothesis: it is not stationarity in itself, but rather the assumption of a unique underlying trend (or, even worse, of a unique average level) during the whole period under examination that proves untenable.

In the next section, we will try to substantiate our claim: we first identify the structural breaks, i.e. the points in time when the trend changes, and then, by detrending, we get new series. These prove stationary, which allows us to analyze the interrelations between birth and death rates.

[6] This is basically the behavior of vital rates in pre-transitional England (1541-1871), according to Nicolini (2006), who also notes that : " ... until now, surprisingly little has been said about the degree of integration of the series [of vital rates]" (Nicolini, 2006, p. 8).

## 3. The demographic transition as a structural break

In its simplest version, the theory of the demographic transition postulates that in a pretransitional phase ($P_1$), high birth and death rates lead to a demographic growth rate close to zero; subsequently ($P_2$ - the transition itself), both fertility and mortality decline, but the growth rate is positive; in the third and final phase ($P_3$ - post-transitional), low mortality and fertility bring the growth rate back to about zero. Therefore, growth rates can be used to identify structural breaks. A simplified version of our method[7] is described below.

The series of the growth rates can be represented as follows:

$$1) \qquad r_t = \sum_{j=1}^{n} P_{j,t} \cdot \mu_j + \varepsilon_t$$

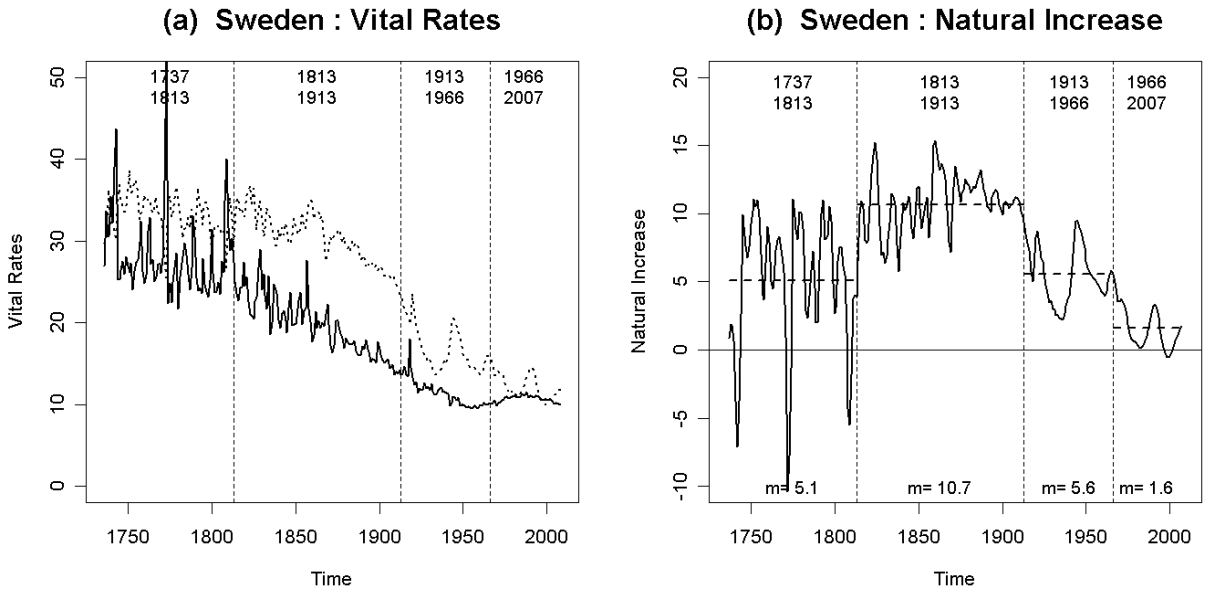
where $r_t$ is the rate of population growth in year *t*; the $P_j$'s are dummies that indicate the current phase of the demographic transition (e.g. $P_{1,t}=1$ if year *t* is in the pretransitional phase, $P_{1,t}=0$ otherwise); $\mu_j$ represents the average growth rate in each of the three phases; and $\varepsilon_t$ is the distance (gap) between the growth rate in year *t* and the average for that phase.

The problem here is to identify our dummies $P_j$ (how many of them[8], and when they change), in such a way that a) the model is parsimonious and b) the predicted values get as close as possible to the empirical ones. We solve this problem by applying the algorithm proposed by Bai and Perron (2003), which reduces considerably the complexity (and the computational time) of the question.

[7] See Bai and Perron (2003) for more details on this method. We implemented it with the *R*-software, *package* "strucchange", function "breakpoints".

[8] The number of phases is not necessarily three. Firstly, the theory may over simplify a more complex reality, with ups and downs, unexpected stops, shocks, etc: these "exceptions" increase the apparent number of phases. Secondly, the series may not be long enough, and the pretransitional phase may not be adequately represented in the data. Finally, the transition may have started late: in this case, it is the post-transitional phase that is missing.

**Figure 1. Demographic phases of the Swedish population.**



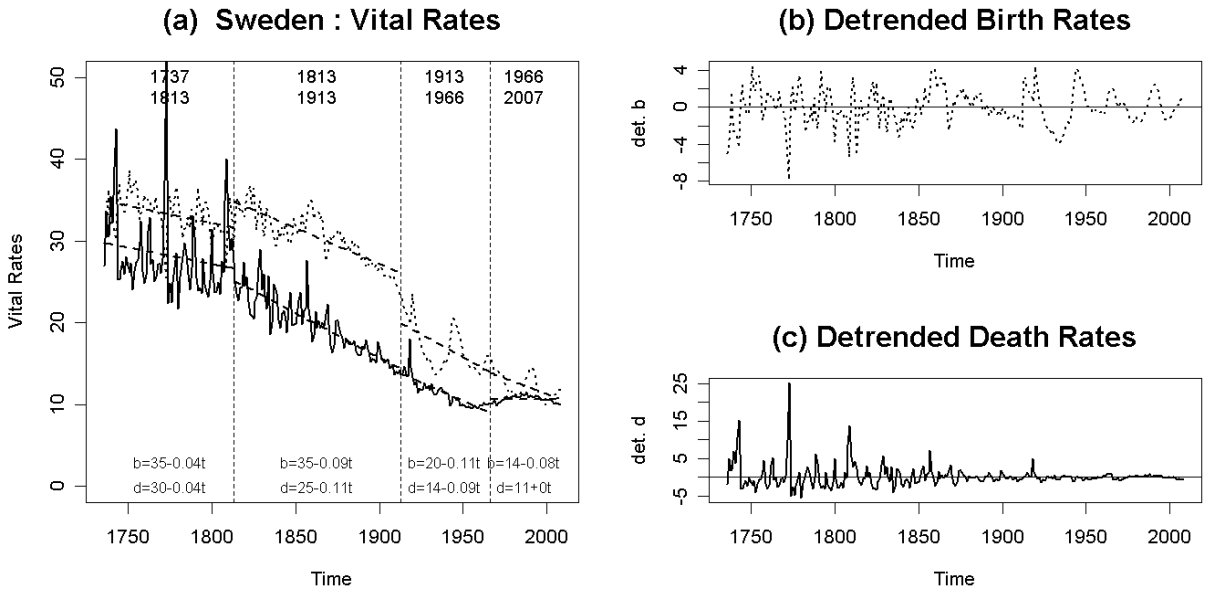

Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

Figure 1 shows an example of what we get. In the case of Sweden, from 1737 to 2008, four phases emerge: a) a pre-transitional phase, from 1737 to 1813, when the average growth rate is about 5‰; b) the first part of the transition, from 1813 to 1913, when r≈10‰; c) the second part of the transition (1913-1968), when r≈5‰; d) the post-transitional phase, when r is just above zero. Table 1 gives the final result of this passage for the 12 countries considered.

Once we have the periods, we *assume* that trends are linear within each of them: we can therefore estimate this linear trend and subtract it from the actual values[9]. These differences should in principle form a stationary series (average=0, no trend), but is this really the case?

[9] This is better than using moving averages, which can create serial autocorrelation, even when it does not exist "in nature", and this would be particularly unfortunate here, because the presence of autocorrelation is precisely one of the things that we want to investigate.

**Figure 2. Vital rates in Sweden - before and after the proposed transformation.**




Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

Table 2 reports the results of two tests of non-stationarity (more precisely: of "unit root" test) on the original, non transformed series. Conclusions are mixed: the ADF test generally suggests non-stationarity for births, but with two clear exceptions (Italy and Spain), and two more countries that cannot be properly classified (Norway and France). With the PP test, instead, stationarity characterizes five countries (France, Finland, Italy, Spain and Sweden), but not the remaining seven. Death rates series are frequently stationary (especially with PP), but not always.

All doubts disappear, however, with the transformed series: they are stationary, in all countries, at all times, and according to all possible tests (Table 3). This circumstance, incidentally, suggests that the original (non transformed) series are stationary with trend, or, sometimes, with multiple trends.

**Table 1. Basic features of our series and of the demographic phases we identify**

| | Country | No of years | Period | No. of phases | Phases |
|---|---|---|---|---|---|
| North | Denmark | 209 | 1800 to 2008 | 4 | 1800-1847-1922-1970-2008 |
| | Finland | 258 | 1751 to 2008 | 5 | 1751-1804-1867-1912-1966-2008 |
| | England and Wales | 161 | 1838 to 1998 | 5 | 1838-1859-1886-1912-1969-1998 |
| | Norway | 274 | 1735 to 2008 | 4 | 1735-1812-1921-1965-2008 |
| | Sweden | 273 | 1736 to 2008 | 4 | 1736-1812-1912-1965-2008 |
| Center | Austria | 189 | 1820 to 2008 | 4 | 1820-1883-1911-1968-2008 |
| | Belgium | 179 | 1830 to 2008 | 4 | 1830-1870-1911-1965-2008 |
| | France | 269 | 1740 to 2008 | 5 | 1740-1793-1844-1886-1953-2008 |
| | Germany | 192 | 1817 to 2008 | 4 | 1817-1870-1911-1967-2008 |
| | Netherlands | 169 | 1840 to 2008 | 3 | 1840-1870-1968-2008 |
| South | Italy | 147 | 1862 to 2008 | 4 | 1862-1881-1912-1973-2008 |
| | Spain | 131 | 1878 to 2008 | 4 | 1878-1898-1941-1979-2008 |

**Table 2. Test of stationarity on the original (non-transformed) series of death (d) and birth (b) rates**

| | Country | ADF b | | PP b | | ADF d | | PP d | |
|---|---|---|---|---|---|---|---|---|---|
| | | Lag | Statistics (p.value) | Lag | Statistics (p.value) | Lag | Statistics (p.value) | Lag | Statistics (p.value) |
| North | Denmark | 5 | -2 (0.57) | 4 | -13.53 (0.35) | 5 | -2.55 (0.34) | 4 | -37.71 (<0.01) |
| | Finland | 6 | -2.49 (0.37) | 5 | -38.43 (<0.01) | 6 | -4.79 (<0.01) | 5 | -131.28 (<0.01) |
| | England and Wales | 5 | -1.9 (0.58) | 4 | -9.4 (0.58) | 5 | -1.23 (0.9) | 4 | -18.49 (0.09) |
| | Norway | 6 | -3.05 (0.14) | 5 | -17.64 (0.12) | 6 | -5.82 (<0.01) | 5 | -151.49 (<0.01) |
| | Sweden | 6 | -2.7 (0.28) | 5 | -21.47 (0.05) | 6 | -5.21 (<0.01) | 5 | -143.66 (<0.01) |
| Center | Austria | 5 | -2.07 (0.55) | 4 | -11.06 (0.48) | 5 | -3.42 (0.05) | 4 | -44.56 (<0.01) |
| | Belgium | 5 | -2.19 (0.50) | 4 | -15.8 (0.21) | 5 | -2.27 (0.46) | 4 | -64.8 (<0.01) |
| | France | 6 | -3.01 (0.15) | 5 | -37.41 (<0.01) | 6 | -3.33 (0.07) | 5 | -114.45 (<0.01) |
| | Germany | 5 | -2.01 (0.57) | 4 | -11.77 (0.44) | 5 | -2.35 (0.43) | 4 | -16.86 (0.15) |
| | Netherlands | 5 | -2.42 (0.40) | 4 | -15.67 (0.22) | 5 | -1.17 (0.91) | 4 | -18.03 (<0.1) |
| South | Italy | 5 | -3.5 (0.04) | 4 | -37.56 (<0.01) | 5 | -1.48 (0.79) | 4 | -22.32 (<0.02) |
| | Spain | 5 | -3.48 (0.05) | 4 | -23.45 (0.03) | 5 | -0.84 (0.96) | 4 | -15.97 (0.19) |

Note: Both series - of death (d) and birth (b) rates - have been tested for non-stationarity (unit root test) twice: with the Augmented Dickey-Fuller (ADF) and with the Phillips-Perron test (PP). Each cell reports the result of the test and (in brackets) the asoociated p.value. If *p* is low (e.g., below 0.1 - grey cells), the series is likely to be stationary.
Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

Table 3. Unit root test on the transformed (de-trended) series of death (d) and birth (b) rates

| | Country | ADF (det.b) | | PP (det.b) | | ADF (det .d) | | PP )det.d) | |
|---|---|---|---|---|---|---|---|---|---|
| | | Lag | Statistics (p.value) | Lag | Statistics (p.value) | Lag | Statistics (p.value) | Lag | Statistics (p.value) |
| North | Denmark | 5 | -4.58 (<0.01) | 4 | -78.1 (<0.01) | 5 | -4.9 (<0.01) | 4 | -88.7 (<0.01) |
| | Finland | 6 | -5.57 (<0.01) | 5 | -137.45 (<0.01) | 6 | -6.46 (<0.01) | 5 | -143.98 (<0.01) |
| | Engalnd Wales | 5 | -3.24 (0.08) | 4 | -45.00 (<0.01) | 5 | -4.95 (<0.01) | 4 | -108.95 (<0.01) |
| | Norway | 6 | -5.48 (<0.01) | 5 | -78.20 (<0.01) | 6 | -7.42 (<0.01) | 5 | -153.37 (<0.01) |
| | Sweden | 6 | -5.31 (<0.01) | 5 | -91.76 (<0.01) | 6 | -8.4 (<0.01) | 5 | 156.3 (<0.01) |
| Center | Austria | 5 | -4.9 (<0.01) | 4 | -67.55 (<0.01) | 5 | -5.19 (<0.01) | 4 | -92.84 (<0.01) |
| | Belgium | 5 | -4.14 (<0.01) | 4 | -49.6 (<0.01) | 5 | -5.87 (<0.01) | 4 | -120.7 (<0.01) |
| | Germany | 5 | -5.38 (<0.01) | 4 | -67.35 (<0.01) | 5 | -5.2 (<0.01) | 4 | -72.92 (<0.01) |
| | France | 6 | -5.43 (<0.01) | 5 | -93.83 (<0.01) | 6 | -5.63 (<0.01) | 5 | -148.24 (<0.01) |
| | Netherlands | 5 | -3.26 (0.08) | 4 | -43.86 (<0.01) | 5 | -3.66 (0.03) | 4 | -73.22 (<0.01) |
| South | Italy | 5 | -4.48 (<0.01) | 4 | -53.14 (<0.01) | 5 | -4.17 (0.02) | 4 | -76.37 (<0.01) |
| | Spain | 5 | -4.82 (<0.01) | 4 | -63.44 (<0.01) | 5 | -4.33 (<0.01) | 4 | -105.57 (<0.01) |

Note: Both series - of detrended death (det.d) and birth (det.b) rates - have been tested for non-stationarity twice: with the Augmented Dickey-Fuller (ADF) and with the Phillips-Perron test (PP). Each cell reports the result of the test and (in brackets) the associated p.value. If *p* is low (e.g., below 0.1 - grey cells), the series is likely to be stationary.
Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

## 4. The VAR (Vector Auto-Regressive) method

Imagine that we know the elements $b_1$, $b_2$, ..., $b_{t-1}$ of a process B, and we want to guess the next values: $b_t$, $b_{t+1}$, $b_{t+2}$, ... If we can improve our forecast (i.e. reduce the error) by exploiting also the available information on another process D ($d_1$, $d_2$, ..., $d_t$), we can say that D causes (or, better, "Granger-causes") B. Analogously, there is instantaneous causation when a given value of D ($d_t$) helps to improve the forecast of the value of B in the same year ($b_t$).

The direction and intensity of this causal relationship can be determined with the VAR methodology, by estimating the so-called "impulse response functions": a variation in the death rate in year $t$ ($d_t$) gives a measurable impulse to the values $b_t$, $b_{t+1}$, $b_{t+2}$, ... of the birth rate (response). The estimation process is complex, however, and is composed of several intermediate steps. Let us quickly go through this procedure, and see what each step means.

**4.1 The reduced form.**

The reduced form is the starting point: the current value of the variables is imagined to depend on all the current and lagged (i.e. previous) values of all variables. Analytically the reduced form is represented by a system of simultaneous equations, (see eq. 2), where $v_{.,t}$ are the vital rates at time t ($v_{1,t}$ = death rates; $v_{2,t}$ = birth rates; b=parameters; $q_1$, $q_2$ = constant terms):

2)
$$\begin{cases} v_{1,t} = q_1 + b_{1,1}^{(1)} v_{1,t-1} + \ldots + b_{1,p}^{(p)} v_{2,t-p} + u_{1,t} \\ v_{2,t} = q_2 + b_{2,1}^{(1)} v_{1,t-1} + \ldots + b_{2,p}^{(p)} v_{2,t-p} + u_{2,t} \end{cases}$$

Here the independent variables are the same for both equations, which is the structure of the so-called SURE models (Seemingly Unrelated Regression Equation). The parameters $b^{(k)}_{i,j}$ can be estimated with a simple linear regression, as if the two equations were independent of each other.

Remember, however, that we have arbitrarily excluded instantaneous causality. But, was this exclusion granted? Notice that if instantaneous causality exists, the residuals will be correlated.[10]

Now, let us imagine that instantaneous causality exists: its strength can be estimated through a Cholesky decomposition. But this is possible only if the direction of the instantaneous causality is assumed *a priori*. In this paper, we will assume that mortality may instantaneously-cause fertility, but not vice versa.

[10] The values outside the principal diagonal of the variance-covariance matrix of the residuals $\Sigma_u$ should not statistically differ from 0 (as measured by a Wald test). If they do, instantaneous causality exists between mortality and fertility.

We now have two distinct estimates: of the delayed causes (the matrices of parameters $\mathbf{B}_1$, $\mathbf{B}_2$,..,$\mathbf{B}_p$) and, on the other hand, of the instantaneous causes (the matrix of parameters $\mathbf{A}_0$). Since we want to see how the two work together, we need to move to the "final form" of the model.

### 4.2 The final form

The model of eq. 2 (reduced form; finite order, i.e. finite number of terms) can be transformed, through a Wold decomposition[11], into the following moving average, infinite order process:

3) $$\mathbf{v}_t = \mathbf{q} + \mathbf{B}_1 \mathbf{v}_{t-1} + ... + \mathbf{B}_p \mathbf{v}_{t-p} + \mathbf{u}_t = \boldsymbol{\mu} + \mathbf{u}_t + \sum_{j=1}^{\infty} \mathbf{C}_j \mathbf{u}_{t-j}$$

With the Wold decomposition, the current state of the variables (vector $\mathbf{v}_t$) depends on a constant term ($\boldsymbol{\mu}$), and on the whole history of shocks ($\mathbf{u}_t$). The matrixes $\mathbf{C}_j$ describe the way these shocks propagate in the system, and they can be computed directly from the matrixes $\mathbf{B}_i$ of the reduced form. Instantaneous causality ($\mathbf{A}_0$) is still absent, here. But we can introduce it, if we now write

4) $$\mathbf{v}_t = \boldsymbol{\mu} + \mathbf{P}\mathbf{w}_t + \sum_{j=1}^{\infty} \mathbf{D}_j \mathbf{w}_{t-j}$$

where $\mathbf{P}=\mathbf{A}_0^{-1}$, $\mathbf{w}_{t-j}=\mathbf{P}^{-1}\mathbf{u}_{t-j}$ and $\mathbf{D}_j = \mathbf{C}_j\mathbf{P}$. Each parameter $d^{(j)}_{h,k}$ of the matrixes $\mathbf{D}_j$ measures the influence on one of the variables (k) in year t, produced by one standard deviation ($\sigma_h$) in the variable h, occurred j years before. Therefore, one can answer questions such as: "in year t, what is the effect on the birth rate of a given increase in the death rate observed 10 years before?"

Finally, we get to the *impulse response function* of variable *k*:

5) $$irf_{h,k} = (p_{h,k}, d^{(1)}_{h,k}, d^{(2)}_{h,k}, ...)$$

which describes the effects of all the past and present shocks of variable *h* on variable *k*. These are the effects in each year, but if we cumulate them, we get the cumulated impulse response function, i.e. the overall effect on variable k of a unitary variation in variable h.

[11] The Wold decomposition works only if the series are stationary.

Beyond the impulse response function, variance decomposition too proves a useful tool: it tells in what proportion the variability of one of the series (e.g. birth rates) can be explained by the variability of the other (e.g. death rates)[12].

## 5. Short term dynamics

In the transformed series that we created in Section 3, by definition, all long-term trends have been removed: what remains is only short term variation, which we will now analyze. What can we expect to find? Basically, the "recuperation" mechanism illustrated by several historical demographers: Livi-Bacci (1978), Galloway (1988), Nicolini (2006), and others. A stylized description of what happens could be is as follows:

1) there is a mortality crisis in year t; in the same year, the birth rate declines (instantaneous cause of mortality on fertility);
2) after the crisis is over, mortality declines to lower-than-normal values, because of selection (only the strongest have survived). Statistically speaking, this translates into a dependency of current on past values: mortality today depends on mortality in the recent past;
3) after the crisis is over, nuptiality rebounds, and, after a while, so does fertility (i.e. past mortality Granger-causes current marriage and birth rates);
4) after the peak of the birth rate, mortality too increases (but, probably, just modestly), because of high infant mortality (past births Granger-cause current mortality);
5) finally, all the series (death, birth and marriage rates - although these last are not considered here) get back to their "normal" values (the series are stationary).

In short, the de-trended series display both instantaneous and lagged causality (possibly with feed-backs) between fertility and mortality[13]. With these expectations in mind, let us now look at our results, in Table 4.

[12] Which we did using the package “vars” in the *R*-software.

[13] Reference to the calendar year may not be neutral, here. Imagine a severe mortality crisis beginning in the winter of year *t* and ending in spring of year t+1: in our data it will probably appear as a relatively mild crisis, covering two successive years. Instantaneous causality between death and birth rates is affected, too: when the mortality crisis begins, conceptions typically decline, so that births will be below average already in year t+1. Apparently, there is instantaneous causality (low mortality and low fertility in year t+1); in reality there is only a poorly measured short lag. With mortality crises with other characteristics (for instance longer, or starting in summer, etc.) results may differ.

The first part of Table 4 (Lag selection) tells us about the so-called "order of the model": how far back in time dependency extends. This lag is typically estimated to be of 2 years, with rare exceptions at 1 and 3. This is consistent with what is otherwise known from the literature: we will therefore stick to the value 2 for our lags.

With this estimated lag, we can proceed to the reduced form of the model (eq. 2). The second part of Table 4 (causality) shows the results of the tests we used (F and $\chi^2$, respectively) to verify our hypotheses of Granger-causality (from D to B and vice versa) and instantaneous causality between the two variables. As before, a low p.value (in brackets) suggests that that particular type of causality (with a lag of 2 years in the case of Granger causality) is probably operating[14].

[14] More precisely, we reject the hypothesis that either Granger- instantaneous causality is absent.

**Table 4. Results of the VAR model: order of the model, causality and t-test on parameter stability.**

| Country | | (1) Lag Selection | | (2) Causality | | | (3) Fluctuation test |
|---|---|---|---|---|---|---|---|
| | | HQ | SC | D→B (F-stat) | B→D (F-stat) | D--B ($\chi^2$-stat) | OLS-CUSUM (stability) |
| North | Denmark | 1 | 1 | 1.13 (0.32) | 0.41 (0.66) | 0.27 (0.61) | Yes |
| | England Wales | 2 | 2 | 10.95 (0.01) | 1.10 (0.34) | 0.02 (0.88) | Yes |
| | Finland | 2 | 1 | 6.92 (0.01) | 2.14 (0.12) | 64.80 (0.01) | Only b |
| | Norway | 1 | 1 | 4.56 (0.01) | 1.05 (0.35) | 18.65 (0.01) | Only b |
| | Sweden | 3 | 2 | 10.08 (0.01) | 0.32 (0.73) | 45.89 (0.01) | Yes |
| Center | Austria | 2 | 1 | 5.34 (0.01) | 1.76 (0.17) | 8.08 (0.01) | Only b |
| | Belgium | 2 | 1 | 8.71 (0.01) | 2.72 (0.07) | 4.82 (0.03) | Yes |
| | France | 2 | 1 | 9.17 (0.01) | 1.61 (0.2) | 0.01 (0.93) | Yes |
| | Germany | 2 | 2 | 2.76 (0.06) | 1.44 (0.24) | 4.46 (0.03) | Yes |
| | Netherlands | 2 | 2 | 28.09 (0.01) | 0.66 (0.52) | 7.19 (0.01) | Yes |
| South | Spain | 2 | 1 | 1.7 (0.19) | 4.27 (0.02) | 8.13 (0.1) | Yes |
| | Italy | 3 | 2 | 17.35 (0.01) | 3.34 (0.04) | 29.38 (0.01) | Yes |

Notes: (1) Lag Selection: the order of the model is based on criteria HQ and SC. (2) Causality: from death to birth rates (D->B); from birth to death rates (B->D); and instantaneous causality between the two (D↔B). When the p.value (in brackets) is low (e.g. below 0.1) the causal link is deemed probable (and the cell is grey). (3) Fluctuation test: the test OLS-CUSUM is used to asses the stability of parameters over time (in the reduced form of the VAR model).
Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

In short, what emerges from our analysis is:

1) mortality almost always Granger-causes fertility. The only exceptions seem to be Denmark and Spain;

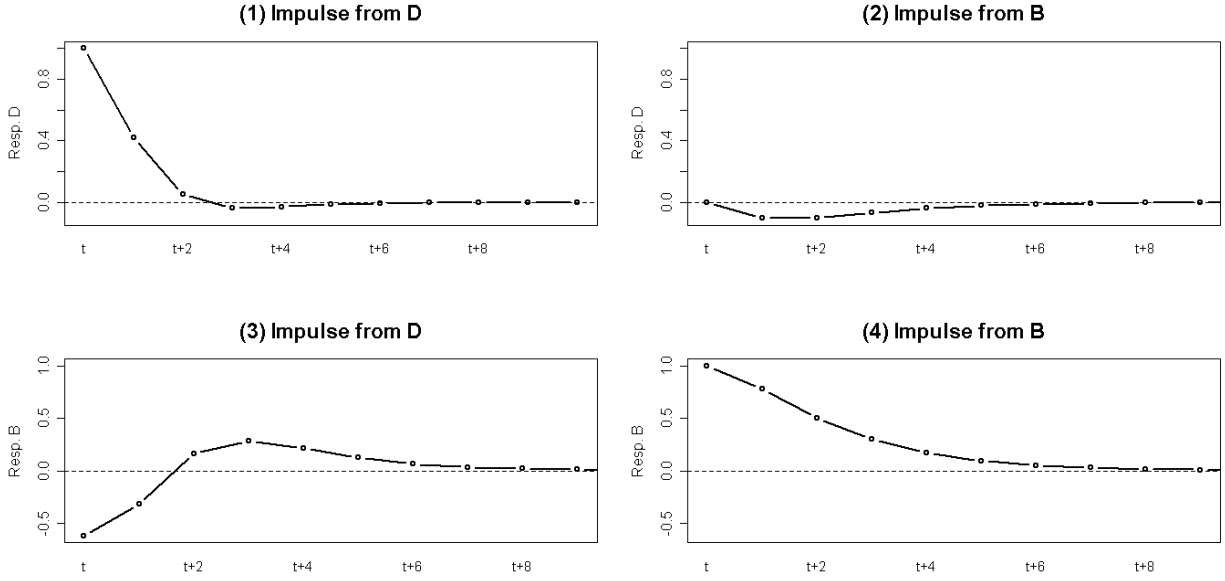
2) the birth rate normally does not Granger-cause mortality; when it does, its effect is small (e.g. in Italy, Belgium and Spain);
3) instantaneous causation is generally present: exceptions are England and Wales; Denmark and France.

The third part of table 4 tests whether the parameters of the model (measuring the sense and strength of the causal relations we have just discussed) do or do not change significantly over time. This is done through the OLS-CUSUM, and the results of this test are summarized with a "Yes" (parameter stability holds for both series), "No" (it holds for neither), "only b" or "only d" (parameter stability holds only for birth or death rates, respectively). Instability affects Austria, Finland and Norway but only in that part of the model that refers to the dynamic of mortality.

Of particular interest to us is the fact that the parameters of the birth rate series are stable: the Granger-influence of mortality on fertility remains relatively constant in all the countries, for the whole period considered, which spans, in the longest case, 250 years. (Remember that these are only short terms variations: long term connections will be considered in the next section.)

After estimating the parameters of the VAR model, we can derive the impulse response functions of the system. There are four of them for each country: let us consider Sweden as an illustrative case (Figure 3). What we see is, on average, how intense the response of a given variable is (on the Y axis) after a unit impulse (i.e. a shock of one standard deviation) coming from either the death (D) or the birth (B) rate series. We also see the average response 0, 1, 2,..., n years after the impulse has occurred.

**Figure 3. Impulse response functions of the series (D=death rate; B=birth rate). Sweden (1749-2008).**




Source: Own elaborations on Chesnais (1992) and Eurostat.

In Sweden, mortality is still high in the year following a crisis (panel 3.1). But 2 years later, on average, mortality is back to its "normal" level, and in the next two years (t+3 and t+4) it is even, very slightly, below average.

Panel 3.3 describes what interests us most, here: how mortality affects fertility. The short term effect (in year t itself and in year t+1) is a sharp reduction of the birth rate, which goes down by about half a standard deviation. From year t+2, on average, recuperation begins, and for the following 4-5 years the birth rate remains above its average.

Panel 3.2 confirms that a shock in fertility has only small effects on the evolution of mortality rates.

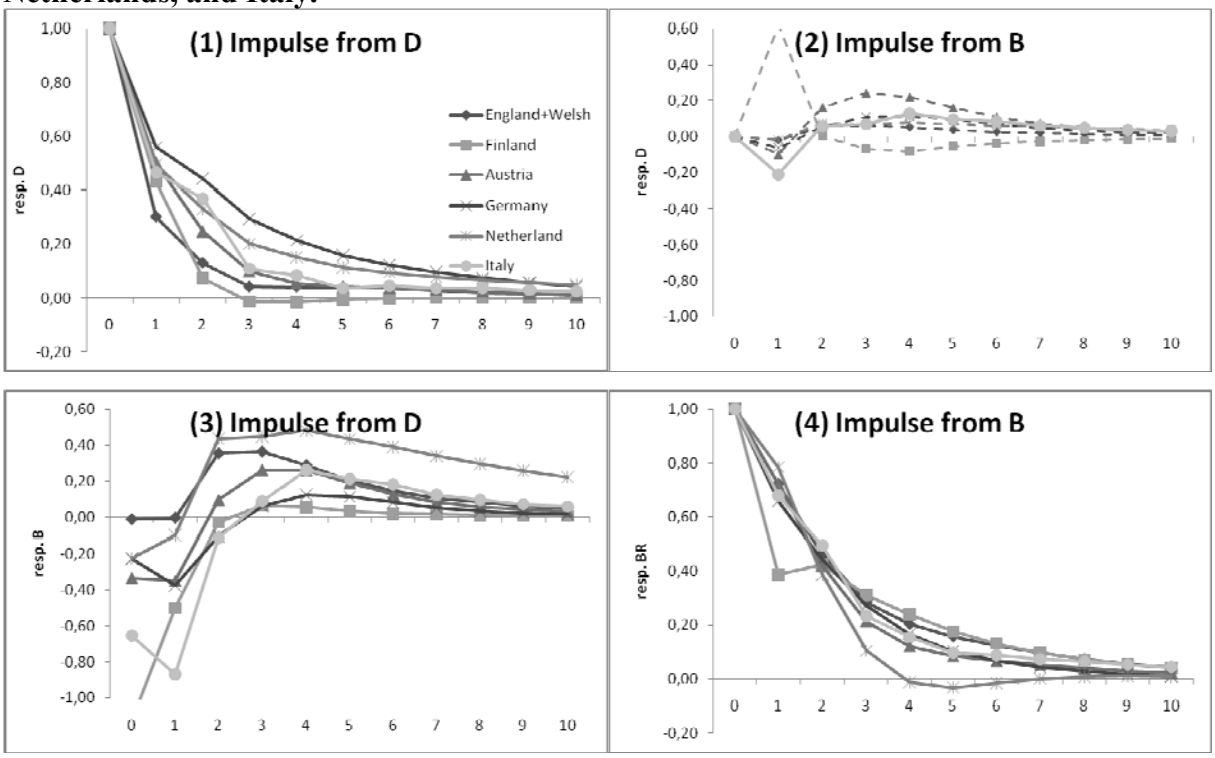
Finally, Panel 3.4 shows that the birth rate, if it is above (below) average in year t, will likely be above (below) average also for the next five years or so.

The pattern we have just considered for Sweden is not exceptional: something similar can be seen for all the 12 European countries under examination. Figure 4 shows the impulse response function for England and Wales, Austria, Germany, the Netherlands, and Italy; Figure 5 shows the same for Denmark, Norway, Sweden, Belgium, France, and Spain. We formed the two

groups on the basis of the effect that the birth rate seems to exert on the death rate (see panel 4.2 and 5.2).

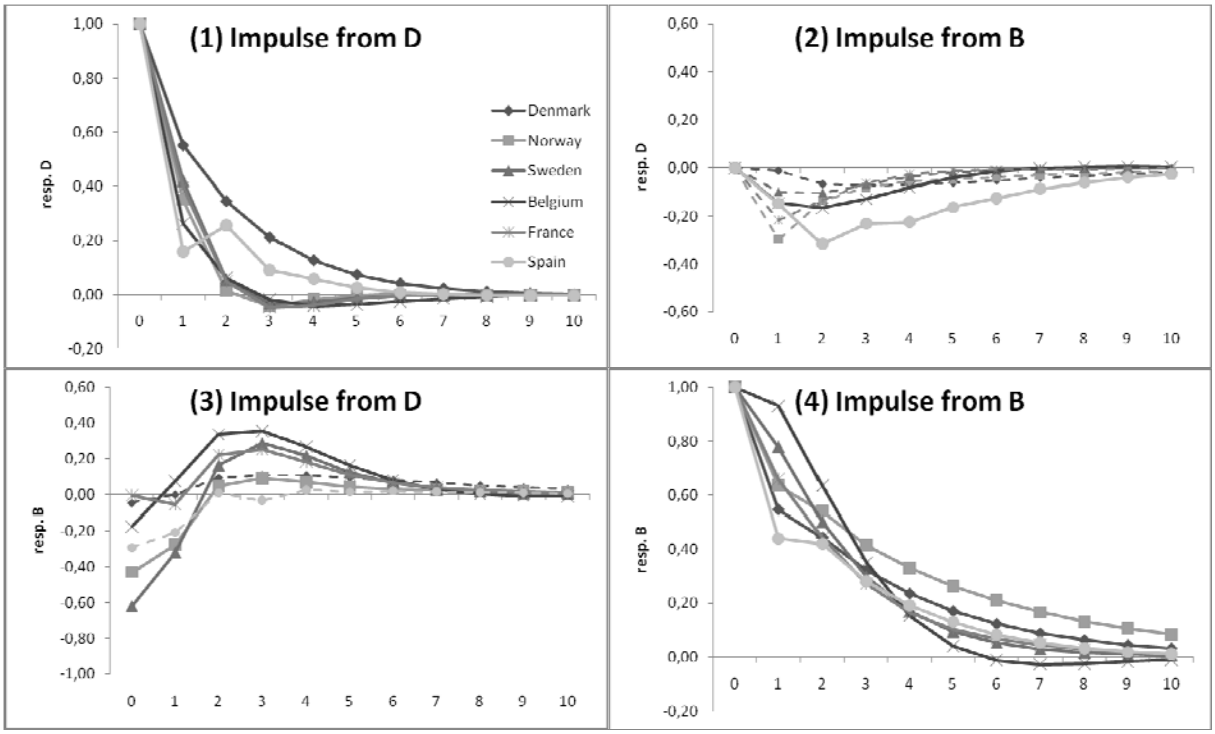
In the former group (Figure 4.2), when the birth rate is high in a given year, the death rate will also be high, in the next few years. In the latter (Figure 5.2), the reverse seems to be true, although the impulse response functions are not always significant for this type of relation, and the response is, in all cases, weak.

**Figure 4. Impulse response functions in England, Finland, Austria, Germany, the Netherlands, and Italy.**




Note: a dotted line indicates that the impulse response function is non significant (cf. Table 4 – Causality).
Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

**Figura 5. Impulse response function of Denmark, Norway, Sweden, Belgium, France, Spain**




Note: a dotted line indicates that the impulse response function is non significant (cf. Table 4 – Causality).
Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

As for the rest, the 12 European countries form a fairly homogeneous group: the type of response is always the same, and it is only the intensity that changes. Notice that, in all cases, if the death increases (mortality crisis), the birth rate declines in that year (t) and, normally, also in the next (t+1), but then recovers and stays above average for about 4 years.

Let us now rapidly analyze the variance of our series (Table 5), in order to evaluate how much the variability of one of them (e.g. the birth rate) reflects the variability of the other (e.g. the death rate). Take Sweden, for instance: after 10 years, we read 18.2% in the column "Variance of B explained by the variance of D" (the death rates does impact on the birth rate), but only 0.3% in the parallel column "Variance of D explained by the variance of B" (the birth rates has practically no influence on the death rate).

**Table 5. Variance decomposition of the birth and death rates in 12 European countries**

| Area | Country | Variance of D (death rates) | | | | | | Variance of B (birth rates) | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | t=0 | | t=5 | | t=10 | | t=0 | | t=5 | | t=10 | |
| | | D | B | D | B | D | B | D | B | D | B | D | B |
| North | Denmark | 100 | 0 | 99.4 | 0.6 | 99 | 1 | 0.1 | 99.9 | 1.3 | 98.7 | 2.2 | 97.8 |
| | Finland | 100 | 0 | 98.8 | 1.2 | 98.8 | 0.12 | 33.9 | 66.1 | 29.4 | 70.6 | 28.5 | 71.5 |
| | England Wales | 100 | 0 | 98.7 | 1.3 | 98.4 | 1.6 | 100 | 0 | 15.5 | 84.5 | 18.2 | 81.8 |
| | Norway | 100 | 0 | 99.2 | 0.8 | 99.2 | 0.8 | 7.4 | 92.6 | 5.7 | 94.3 | 5.3 | 94.7 |
| | Sweden | 100 | 0 | 99.7 | 0.3 | 99,7 | 0.3 | 20,4 | 79.6 | 17.8 | 82.2 | 18.2 | 81.8 |
| Center | Austria | 100 | 0 | 98 | 2 | 97.4 | 2.6 | 4.5 | 95.5 | 8.4 | 91.6 | 9.6 | 90.4 |
| | Belgium | 100 | 0 | 96.2 | 3.8 | 96 | 4 | 2.8 | 97.2 | 11.7 | 88.3 | 12.8 | 87.2 |
| | Germany | 100 | 0 | 98.7 | 1.3 | 98.1 | 1.9 | 2.4 | 97.6 | 5.6 | 94.4 | 6.1 | 93.9 |
| | France | 100 | 0 | 98.7 | 1.3 | 98.7 | 1.3 | 100 | 0 | 6.8 | 93.2 | 7.5 | 92.5 |
| | Netherlands | 100 | 0 | 99.5 | 0.5 | 99.1 | 0.9 | 4.5 | 95.5 | 29.0 | 71.0 | 41.7 | 58.3 |
| South | Spain | 100 | 0 | 91.0 | 9.0 | 89.0 | 11.0 | 6.7 | 93.3 | 6.9 | 93.1 | 6.8 | 93.2 |
| | Italy | 100 | 0 | 98.1 | 1.9 | 97.5 | 2.5 | 25.4 | 74.6 | 36.2 | 63.8 | 37.7 | 62.3 |
| All | average | 100.0 | 0.0 | 98.0 | 2.0 | 97.4 | 2.3 | 26.2 | 74.3 | 14.5 | 85.5 | 16.2 | 83.8 |
| | median | 100.0 | 0.0 | 98.7 | 1.3 | 98.4 | 1.5 | 6.7 | 93.0 | 10.1 | 90.0 | 11.2 | 88.8 |
| | st. dev. | 0.0 | 0.0 | 2.4 | 2.4 | 2.9 | 2.9 | 38.0 | 36.2 | 11.3 | 11.3 | 13.2 | 13.2 |

Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

Let us now consider the decomposition of the variance in the birth rates. After 10 years, the variance of the death rates "explains" between 2.2% (in Denmark) and 41.7% (in Finland) of the variance of the birth rates. The average is about 16%, but with marked variability (the standard deviation is about 13%).

Let us now sum things up: in the 12 countries considered and in the short run, mortality Granger-causes fertility, but not vice versa. The influence is limited, however (only about 16% of the variability in the series of the birth rates can be explained in this way), and the strength of the link varies considerably from country to country.

## 6. The long term and the demographic transition

The homeostatic system that operated in the short term in pre-transitional regimes may also help to explain at least part of the demographic transition. Let us see how.

In the demographic transition, decline occurs first in mortality, and later in fertility. Our variance decomposition led us to the conclusion that, on average, only 16% of the variability in the series of birth rates depends on the variability of mortality in the 10 preceding years. This means that, once mortality starts to decline, the depressing effect on fertility is small: birth rates do not react immediately, and, as we see from panel 3 of Figures 3 to 5, they may at first even go in the "wrong" direction, i.e. increase slightly.

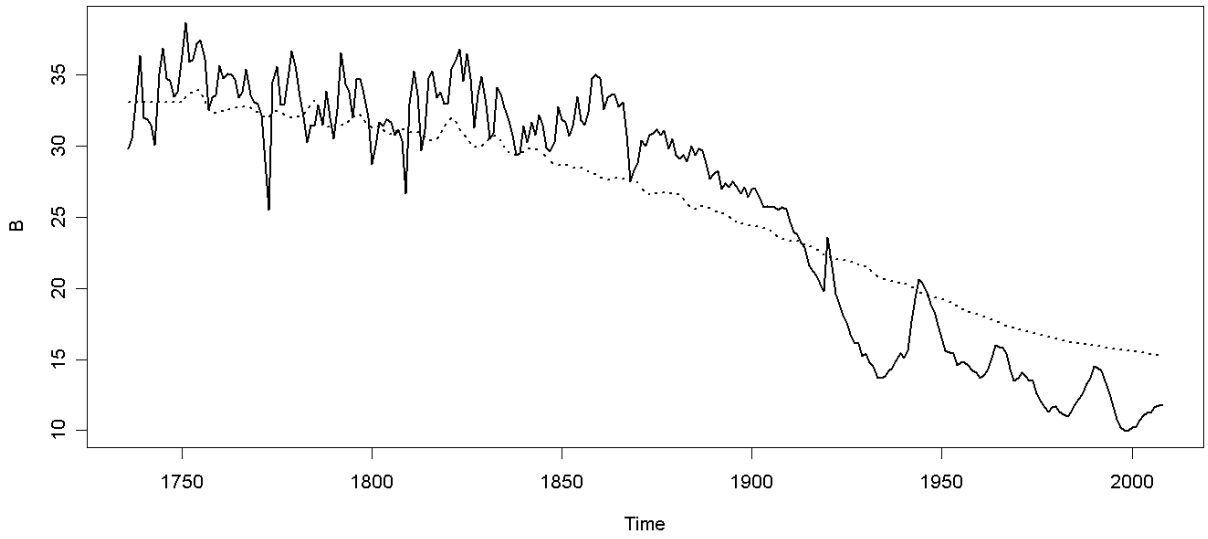
It is only after a while, when the mortality decline has gained momentum (or when it is abrupt, as in the developing countries of the second half of the 20th century), that the depressing effect on fertility becomes discernible, which, because of autocorrelation, further depresses fertility in the subsequent years.

Can our model satisfactorily mimic the demographic transition? In order to answer this question, we tried to estimate the series of the birth rates starting from that of the death rates (our independent variable). This estimate is obtained by applying recursively the birth rate equation - second equation in (2), with the estimated parameters - to the observed death rates[15]. Then we continue, using this forecasted birth rate plus the observed death rates to produce a new forecast for the birth rate two years later, and so on: each forecasted value for the birth rate becomes the starting point of a new forecast. Finally, the estimated birth rates series is compared to the empirical one, in order to appreciate how much of the long-term dynamic of fertility can be explained by the homeostatic system described in the previous section[16].

[15] Only at the very beginning of the procedure did we also have to use the initial (observed) values for the birth rates. But we verified what is intuitively evident: the initial ("exogenous") values of $b$ are quickly forgotten and become irrelevant in forecasting the series. The only things that matter are the death rates (exogenous variable) and the estimated parameters.

[16] For our forecast, we use the estimates of the model in its reduced form, with no simultaneous effect of mortality on fertility. However, we also tried the alternative method (i.e., including simultaneous effects) and the results are virtually the same. Our interpretation is that simultaneous effects play some part in the short run, but they are almost negligible in the long run.

**Figure 6. Sweden: empirical and model (forecasted) birth rates.**

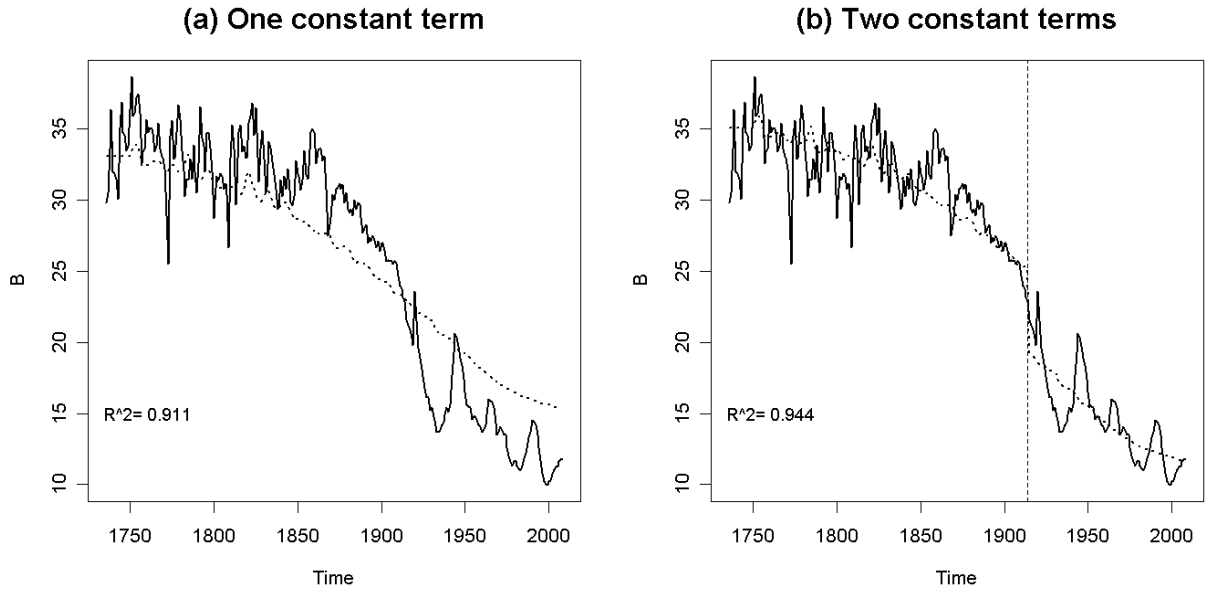



Source: Own elaborations on data taken from Chesnais (1992) and Eurostat.

Figure 6 shows that, in the case of Sweden, there is a good fit between the empirical and the estimated data. But we can do better than this: Figure 6 has been built on the assumption that there is only one constant term for the whole period, with no discontinuities. But we can drop this assumption and admit the possibility of one "jump" in the series, i.e. a moment when the constant of the system changes from an initial to a final value[17].

[17] Indeed, if it varies from $q_2$ to $q^*_2$, the constant term is no longer constant: but we will ignore this contradiction, here, and stick to the original terminology. The constant term can be easily found with least squares, when it is unique; but we need Bai and Perron's (2003) method when there are two. Of course, one could also consider more complex cases, of with three or more constant terms (piecewise constants), but we will not do it here.

**Figure 7. Sweden: actual and model birth rates, in two scenarios (a) system stability (one constant term); (b) discontinuity (two constant terms).**




Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

The constant term represents the long-term difference between the birth and the death rate: basically the parameter that, given mortality, drives fertility in the long run. Now, using the 2-constant model we see that this parameter seems to change around 1914, shifting the curve downwards, and therefore causing a smaller rate of natural increase: this sounds reasonable to us.

Table 6 gives an overview of what happens to our forecasted birth rates in the 12 countries under examination. With just one constant term, we can explain about 90% of the variance of the birth rates in the 12 countries, from 79% in Norway to 95% in Italy. Assuming discontinuity, of course, the goodness of fit improves, up to about 94%, on average. The breaks in the series seem to occur somewhere between 1908 and 1933.

**Table 6**. **Goodness of fit between model and actual birth rates in 12 European countries: with one and two constant terms.**

| Area | Country | One constant | Two constants | |
|---|---|---|---|---|
| | | R2 | Breakdate | R2 |
| North | Denmark | 0.92 | 1921 | 0.93 |
| | Finland | 0.92 | 1914 | 0.91 |
| | England Wales | 0.94 | 1909 | 0.95 |
| | Norway | 0.79 | 1923 | 0.87 |
| | Sweden | 0.91 | 1914 | 0.94 |
| Center | Austria | 0.83 | 1913 | 0.96 |
| | Belgium | 0.90 | 1908 | 0.94 |
| | France | 0.91 | 1879 | 0.95 |
| | Germany | 0.88 | 1912 | 0.95 |
| | Netherlands | 0.90 | 1972 | 0.96 |
| South | Italy | 0.95 | 1915 | 0.95 |
| | Spain | 0.92 | 1933 | 0.92 |
| All | average | 0.90 | \ | 0.94 |
| | st. dev. | 0.05 | \ | 0.03 |

Source: Own elaborations on data taken from Chesnais (1992) and Eurostat

## 7. Conclusions

Demographic recovery after a crisis focuses on the short term; the demographic transition deals with the long term. Yet, this article shows that the same mechanism that drives the former may also explain the latter, once we admit the possibility of an exogenous (and, in the case of the demographic transition, persistent) decline in the death rates. It is precisely the fact that birth rates react with a lag to a decline in mortality that has contributed to shape the demographic transition. But, apparently, we do not need a special theory to "explain" the decline in the birth rates: this may be interpreted as a simple byproduct of the decline in the death rates, within a homeostatic system that existed long before the demographic transition took place.

The underlying logic of the system may not have changed, but at least two things did change over time: one is the equilibrium level of mortality; the other is the interaction between mortality and fertility (in the two-constant model). The latter simply means that the growth rate tends to be lower now than it was in pre-transitional times.

Nuptiality and migration are still left out of the picture, despite the attention that several demographers devoted to them (e.g. Chesnais 1986, 1992). This omission, together with the neglect of what has happened outside Europe, will have to be remedied in some future work.

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