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Short- and Long-Term Dynamics of Cause-specific Mortality Rates Using Cointegration Analysis

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ABSTRACT

This paper applies cointegration analysis and vector error correction models to model the short- and long-run relationships between cause-specific mortality rates. We work with the data from five developed countries (USA, Japan, France, England and Wales, and Australia) and split the mortality rates into five main causes of death (Infectious&Parasitic, Cancer, Circulatory diseases, Respiratory diseases, and External causes). We successively adopt the short- and long-term perspective, and analyze how each cause-specific mortality rate impacts and reacts to the shocks received from the rest of the causes. We observe that the cause-specific mortality rates are closely linked to each other, apart from the External causes that show an entirely independent behavior, and hence, could be considered as truly exogenous. We summarize our findings with the aim to help practitioners set more informed assumptions concerning the future development of mortality.

KEYWORDS

Causes of death, dependence, cointegration, VECM, impulse-response analysis

1 Introduction

It is commonly known that the mortality rates have been decreasing for many decades now. Although a joyful development *per se*, these changes pose serious problems for insurance companies, pension funds, and social security schemes, as they need to know if the observed decline will continue, slow down or, on the contrary, speed up. In this work, we will not venture to forecast the prospective evolution of mortality rates, but provide new insights on the past developments. We believe that once we understand better the past, we will be able to make better prognoses about the future.

Numerous parametric models have been developed in order to take into account such characteristics of mortality rates development as age, year of birth, and rate of improvement. For a review thereof we direct the interested reader to Booth and Tickle (2008), Cairns (2013) and Debón et al. (2006) including their references. For our part, we want to gain additional insight into the past development of mortality rates by concentrating on a more detailed breakdown of mortality data, namely by causes of death. Indeed, just from an eye inspection of the cause-specific mortality rates, it becomes clear that these rates showed strikingly divergent trends over the last 50 to 60 years. These phenomena have already been extensively studied and described (e.g., Himes, 1994; Horiuchi and Wilmoth, 1997; Costa, 2005; Cutler et al., 2006).

However, it is much more difficult to integrate cause-specific mortality rates into a model, as they are dependent, and this dependence is, strictly speaking, not observable. Indeed, given a death event at a young age from an accident, for example, it is impossible to say what the chances of this person would be to die later from cancer or any other cause, had he or she remained alive. Among theories and methods trying to take into account the dependency structure between the cause-specific mortality rates one can cite models incorporating individual risk factors (e.g., Manton and Poss, 1979; Manton et al., 1991), models employing multiple cause-of-death data (e.g., Mackenbach et al., 1999; Manton and Myers, 1987); and more recently, copulas (e.g., Lo and Wilke, 2010; Dimitrova et al., 2013).

Although possible theoretically, models that take into account the dependency between the causes of death are problematic to use in practice, as they require a significant amount of additional data that are not readily available. For this reason, the most widely used approach is still based on the assumption of independence between the causes of death that was developed more than 50 years ago (Chiang, 1968). In this study, we want to look at the connections between the causes from a different angle. Without trying to describe exactly the dependency structure between the rates of death, we propose an approach based on cointegration analysis that complements the methods and practices mentioned above. In a nutshell, two non-stationary time series are said to be cointegrated if there exists such a linear combination of them that is stationary. Consequently, these time series are linked to each other in the long run and are subject to common stochastic trends. Cointegration analysis thus provides new insights on how cause-specific mortality rates depend from each other and interact in the long run.

Cointegration analysis was first introduced in the seminal paper of Engle and Granger (1987) and received a lot of attention from researchers in the years that followed. Numerous tests allowing one to check for the existence of cointegrated relations between the time variables were developed, those conceived by Søren Johansen (1988) being among the most widely used. Cointegration analysis and the Vector Error-Correction Models (VECMs) based on it quickly became popular in the field of econometrics as they permitted establishing the long-run relationships between such variables as interest rates, consumption, income etc. (e.g., Baillie and Selover, 1987; Clarida, 1992; Johansen and Juselius, 1992).

To the best of our knowledge, cointegration analysis was first applied to the cause-specific mortality rates in Arnold and Sherris (2013, 2015, 2016). We want to go further and extend the analysis by applying a wider range of cointegration and VECM tools to the cause-of-death mortality rates. We aim to identify new relationships and development

patterns which were not covered by the pre-cited authors.

Namely, we want to understand the way the cause-specific mortality rates interact between each other. Using the additional tools offered by the VECMs, we study the short- and long-term impacts that a change in a particular death rate produces in other cause-specific mortality rates. As we do not have prior knowledge about the precise way the cause-specific mortality rates interact, our study is exploratory in nature and gains new insight by observing the historical data from the perspective of cointegration analysis. At the same time, once a certain pattern is revealed in one country, it is impossible to say if this pattern is a reflection of that country's particularities or corresponds to some more fundamental processes and hence, can be generalized to other countries and datasets. For this reason, we start with the gender-specific statistics of deaths-by-cause from five highly populated countries with similar socioeconomic characteristics and available observation periods (USA, Japan, France, England and Wales, and Australia). Thanks to this approach, general common patterns are revealed in regard to the interaction existing between the causes of death. At a later point, our analysis could be extended to include other countries as well.

We see multiple ways of how our findings could be used in practice. First, the general patterns revealed by our approach can serve as a theoretical point of comparison for epidemiological studies on the joint development of cause-specific mortality rates due to particular factors, e.g., air pollution impacting not only respiratory, but also circulatory mortality rates (Zmirou et al., 1998); sedentary behavior impacting both circulatory and cancer mortality rates (Matthews et al., 2012); body mass index providing contrasting effects on circulatory and respiratory mortality rates (Breeze et al., 2006); influenza vaccinations reducing all cause-specific mortality rates (Wang et al., 2007); heat waves impacting several cause-specific mortality rates at once (Basagaña et al., 2011; Rey et al., 2007) etc. In a similar way, results of such comprehensive assessments of cause-specific mortality rates, as the Global Burden of Disease Study (GBDS, 2013), can be confronted with those delivered by our model.

As previously mentioned, copula-based models are capable of taking into account the dependence between the cause-specific mortality rates. In the same time, copulas are, strictly speaking, not identifiable (Tsiatis, 1975). For this reason, research articles usually present several copulas and play with different parameter values, as these choices can have a tremendous impact on the projection results (Dimitrova et al., 2013; Li and Lu, 2019). Efforts are made to narrow the set of possible parameters (Li and Lu, 2019) and the question of how to estimate the correlations between the causes of death remains open (Dimitrova et al., 2013). Our study provides a new basis that can be used to calibrate copula-based models as it shows explicitly the extent to which cause-specific mortality rates depend on each other.

Additionally, we contribute to the current discussion regarding whether a cause of death should be considered as endo- or exogenous. In Arnold and Sherris (2016) the authors observed that the results of the cointegration analysis paralleled the classification used by biologists and demographers between the exogenous and endogenous causes of death. Although this classification is not univocal, under the exogenous causes of death most researchers understand diverse external or environmental factors that produce death, while the endogenous causes of death correspond to biological forces that lead to death (Carnes et al., 2006; Arnold and Sherris, 2016). As different views exist

on this topic (Carnes and Olshansky, 1997), we bring the discussion forward by showing that only the External causes can be classified as entirely exogenous, whereas this is not the case for the infectious and parasitic diseases.

We summarize our findings in a comprehensive form with the objective to help practitioners set more informed assumptions when designing scenarios of the possible future evolution of mortality by cause.

The paper is organized as follows: in Section 2 we briefly present the data preparation process along with some theoretical notions of the cointegration analysis. Results from the impulse-response analysis, short- and long-term dynamics of the cause-specific mortality rates are then presented in Section 3. Section 4 concludes.

2 Data and the cointegration framework

2.1 Preparing the data

We obtained the data for the present study from the WHO Mortality Database (World Health Organization, 2016) that contains the midyear population and the death numbers by country, year, sex, age group and cause of death as far back as 1950. Five developed countries were chosen for the analysis: USA, Japan, France, England and Wales, and Australia (further shortened to US, JP, FR, E&W, and AU respectively).

To ensure consistency between the countries, the WHO defines the causes of death according to the International Classification of Diseases (ICD). This classification changed three times since the inception of the database, switching from ICD-7 to ICD-10 in order to account for advances in medical science and to refine the classification. We split the causes of death under each classification into five main groups: infectious and parasitic diseases (I&P), cancer, diseases of the circulatory system, diseases of the respiratory system and external causes (Table 1). These groups account for approximately 70-80% of deaths in recent years and made up approximately 50%-70% of deaths at the onset of the observations.

Table 1: FIVE MAIN GROUPS OF CAUSES OF DEATHS ACCORDING TO THE VERSIONS OF INTERNATIONAL CLASSIFICATION OF DISEASES

Causes of death	ICD 7	ICD 8	ICD 9	ICD 10
I&P	A001-A043	A001-A044	B01-B07	A00-B99
Cancer	A044-A060	A045-A061	B08-B17	C00-D48
Circulatory	A079-A086	A080-A088	B25-B30	I00-I99
Respiratory	A087-A097	A089-A096	B31-B32	J00-J98
External	A138-A150	A138-A150	B47-B56	V00-Y89

The data are divided into the following age groups: "deaths at 0 years", "at 1", "at 2", "at 3", "at 4", then into five-year age groups "5-9 years", ..., "90-94 years", and finally "deaths at 95 years and above". Having created two new age groups by grouping together the ages from 1 to 4 as well as 85 and above, we obtained the cause-specific mortality rates by following transformations:

1. Grouping the death numbers according to the five causal categories.
2. Distributing the number of deaths at unspecified age proportionally among known age groups.

3. Calculating simple mortality rates as the number of deaths by age, sex and cause divided by the mid-year population by age and sex:

$$m_{x,t,d,s,c} = d_{x,t,d,s,c}/l_{x,t,s,c},$$

with

$d_{x,t,d,s,c}$ = number of deaths at age x, in year t, for cause of death d, gender s and country c;

$l_{x,t,s,c}$ = mid-year population at age x, in year t, gender s and country c;

$m_{x,t,d,s,c}$ = central death rate at age x, in year t, for cause of death d, gender s and country c.

4. Applying the comparability ratios to ensure that the observations under the different versions of the ICD are comparable. A comparability ratio is defined in such a way that the average of the mortality rates over the last two years of a classification coincides with the average of the mortality rates over the first two years of the next classification. For the whole period under the observation, the mortality rates in a new classification are divided by the comparability ratios linking this classification with the previous one(s). In this way, we can smooth the mortality rates across the classifications and remove the discontinuities.
5. Calculating the age-standardized central death rates, the standard population being equal to 1) the US male population in 2007; 2) the Japanese female population in 2009. In this manner, we ensure that the age structure of the population is the same for all countries and does not change over time. By using one relatively young (USA) and one relatively old (Japan) reference population, we can analyze if the population age structure has an impact on the behavior of the cause-specific mortality rates. In total, we obtain 20 datasets: 5 countries, 2 genders, and 2 population structures.

   The age-standardized death rate $m^{US}_{t,d,s,c}$ in year t for cause d, gender s and country c, assuming that the population age structure is constant over the whole observation period and is equal to the age structure of the US males population in 2007 is calculated as follows:

$$m^{US}_{t,d,s,c} = d^{US}_{t,d,s,c}/l_{2007,males,USA}$$
$$d^{US}_{t,d,s,c} = \sum_x m_{x,t,d,s,c} \times l_{x,2007,males,USA}$$

   The age-standardized death rate $m^{JP}_{t,d,s,c}$ in year t for cause d, gender s and country c, assuming that the population age structure is constant over the whole observation period and is equal to the age structure of the JP females population in 2009 is calculated as follows:

$$m^{JP}_{t,d,s,c} = d^{JP}_{t,d,s,c}/l_{2009,females,JP}$$
$$d^{JP}_{t,d,s,c} = \sum_x m_{x,t,d,s,c} \times l_{x,2009,females,JP}$$

6. Taking the natural logarithm of the death rates. Hereafter we will work with the vector of time series $\mathbf{y}_t$ for each gender s, country c, and population age structure $p \in (US, JP)$:

$$\mathbf{y}^p_{t,s,c} = \begin{pmatrix} log(m^p_{t,I\&P,s,c}) \\ log(m^p_{t,Cancer,s,c}) \\ log(m^p_{t,Circulatory,s,c}) \\ log(m^p_{t,Respiratory,s,c}) \\ log(m^p_{t,External,s,c}) \end{pmatrix}$$

To ease the notation, we will sometimes omit the indexes c, s and p, and work with a vector of mortality rates $\mathbf{y}_t = (y_{1t}, y_{2t}, y_{3t}, y_{4t}, y_{5t})^T$, keeping in mind that a separate VECM equation is formulated for each country, sex, and population age structure.

We thus use the same database as in Arnold and Sherris (2016) except for the additional years of observations that we added whenever this was possible (Table 2).

Table 2: OBSERVATION PERIODS BY COUNTRY

Country	Arnold and Sherris (2016)	Current study
USA	1950 - 2007	1950 - 2007
Japan	1950 - 2009	1950 - 2013
France	1952 - 2008	1952 - 2011
England and Wales	1950 - 2009	1950 - 2013
Australia	1950 - 2004	1950 - 2004

When we started the current study, the WHO database provided the information on the mid-year population for the USA only until 2007, and for unknown reasons, the data on Australian numbers of deaths for 2005 were also missing. As a consequence, we were obliged to limit the time series for these two countries to years 2007 and 2004 respectively.

As we will see in the following sections, the conclusions stated in Arnold and Sherris (2016) were reconfirmed using the longer time series for Japan, France, and England and Wales.

2.2 Cointegration analysis in application to the cause-specific mortality rates

As already mentioned above, the causes of death are not independent. Cointegration analysis is then a tool that can help to understand better and model the dependence between the cause-specific mortality rates. As introduced in Engle and Granger (1987), the time series $\mathbf{y}_t$ that consist of the n non-stationary elements $\{y_{it}\}$, for $i = 1, ..., n$, are said to be cointegrated with a cointegrating vector β if a linear combination $\beta'\mathbf{y}_t$ is stationary:

$$\beta_1 y_{1t} + \beta_2 y_{2t} + ... + \beta_n y_{nt} = z_t, \tag{1}$$

where z_t is a stationary variable of stochastic deviations. In other words, while being non-stationary themselves, the cointegrated time series do not drift too far away from each other, i.e., there exists a long-run equilibrium relationship between them. Also, there may be more than one cointegrating vector, so that β becomes a matrix. The variables are

then linked to each other by several cointegration relations, and each relation is linearly independent from the others.

In Arnold and Sherris (2015, 2016) the time series of all cause-specific mortality rates were found to be non-stationary and to have stochastic trends. It was also shown that at least one cointegrating relation existed between the causes of death in each country.

Multivariate dynamic systems of the non-stationary but cointegrated variables can then be modeled using a Vector Error Correction Model (VECM), an extension of the Vector AutoRegression (VAR) models, which includes not only the time dependency between the variables up to a lag $p-1$, but also long-run equilibrium relations between them:

$$\Delta \mathbf{y}_t = \mathbf{c} + \mathbf{d}t + \mathbf{\Gamma}_1 \Delta \mathbf{y}_{t-1} + \mathbf{\Gamma}_2 \Delta \mathbf{y}_{t-2} + \cdots + \mathbf{\Gamma}_{p-1} \Delta \mathbf{y}_{t-p+1} + \mathbf{\Pi} \mathbf{y}_{t-1} + \epsilon_t \quad (2)$$

where $\Delta \mathbf{y}_t = \mathbf{y}_t - \mathbf{y}_{t-1}$ denote the first differences of the data time series, $\mathbf{c}$ and $\mathbf{d}$ are $(n \times 1)$ vectors of constants, $\mathbf{\Gamma}_i$ is a $(n \times n)$ matrix of autoregressive coefficients for $i = 1, 2, ..., p-1$, and $\mathbf{\Pi} \mathbf{y}_{t-1}$ represents the cointegrated term. The latter provides the model with the information on the long-run equilibrium between the variables that would otherwise be lost if a VAR model were applied to the differenced variables. The rank of the matrix $\mathbf{\Pi}$ corresponds to the number of cointegration relations.

The first differences of the cause-specific mortality rates being stationary (as verified in Arnold and Sherris, 2016), the equation (2) will only hold if the term $\mathbf{\Pi} \mathbf{y}_{t-1}$ is also stationary, that is, if the variables are cointegrated. Then the $(n \times 1)$ vector ϵ_t is a vector of white noise terms, with

$$E(\epsilon_t) = \mathbf{0}, \quad (3)$$

$$E(\epsilon_t \epsilon_l) = \begin{cases} \mathbf{\Omega} & \text{for } t = l \\ \mathbf{0} & \text{for } t \neq l, \end{cases} \quad (4)$$

where $\mathbf{\Omega}$ is a symmetric positive definite matrix. More details on the VECM and VAR models can be found in Hamilton (1994) and Lütkepohl (2005).

The number of the cointegrating relations, if any, can then be found using the trace and the maximum eigenvalue tests developed by Johansen (1995). The Johansen approach also allows finding the matrix $\mathbf{\Pi}$ as

$$\mathbf{\Pi} = \alpha \beta', \quad (5)$$

where β is a $(n \times r)$ matrix containing r vectors each representing a cointegration relation and α is a $(n \times r)$ loading matrix that indicates how a particular variable is impacted by the cointegration relation. Under the Johansen approach, we can also test for the form of the deterministic elements. Let $\mu_t = \mathbf{c} + \mathbf{d}t$ denote the deterministic part of the model and let $\mathbf{d} = \alpha\rho + \alpha_\perp \gamma$, where $\alpha \alpha_\perp = 0$. As the mortality rates are known to have a trend, we will consider the following forms of the deterministic elements (Johansen, 1995):

- NT: no trend in the VECM, but a linear trend in the levels of the variables: $\mathbf{c} \neq 0, \rho = 0, \gamma = 0,$ hence $\mathbf{d} = 0$,

[1] The corresponding VAR model has p lags: $\mathbf{y}_t = \mathbf{c} + \mathbf{d}t + \xi_1 \mathbf{y}_{t-1} + \xi_2 \mathbf{y}_{t-2} + \cdots + \xi_p \mathbf{y}_{t-p} + \epsilon_t$

- TC: linear trend in the cointegration relation combined with a linear trend in the levels of the variables (i.e., no linear trend in the differenced variables): $\mathbf{c} \neq 0, \rho \neq 0, \gamma = 0,$ hence $\mathbf{d} = \alpha\rho$,
- QT: linear trend in the differenced variables, thus the quadratic trend in the levels of the variables (i.e., the VAR model) : $\mathbf{c} \neq 0, \rho \neq 0, \gamma \neq 0,$ hence $\mathbf{d} = \alpha\rho + \alpha_\perp\gamma$.

In the tables that follow we will refer to the abbreviations NT, TC and QT when describing the form of the deterministic elements chosen for a particular dataset.

Once the coefficients of the VECM model (equation 2) are defined, they allow us to assess the short- and long-term dynamics of the system. Indeed, the coefficients of the $\mathbf{\Gamma}_i$ matrices indicate if and to what extent the cause-specific mortality rates interact in the short run. On the other hand, the analysis of the coefficients of the matrices α and β provide us with the information on the long-term relationships in the system.

In particular, the Johansen approach can be used to test if every coefficient in the cointegration relation (i.e., in the matrix β) is significantly different from zero. If this is not the case, we can conclude that a particular variable does not participate in the long-run equilibrium. In Arnold and Sherris (2016) it was found that in all countries and at least for one of the sexes the pair of mortality rates corresponding to the Infectious&Parasitic diseases and the External causes did not appear significantly in the long-run equilibrium. The cointegration analysis hence showed that the long-term equilibrium relationship existed only between the mortality rates that could be classified as endogenous causes of death (Cancer, Circulatory, and Respiratory diseases), exogenous causes (Infectious&Parasitic diseases, External causes) being excluded from it. Interestingly, this result coincides with the distinction used by biologists and demographers between the exogenous and endogenous causes of death. In this paper, we will complement this study by analyzing first, the short-term component and second, the matrix α, that is, the impact that the cointegration relation performs on a particular mortality rate.

2.3 Introducing the lag of 2 to the VECM setup

As a first step when working with a VECM setup, one has to define the lag order to be used in the VECM or the corresponding VAR model. In Arnold and Sherris (2016) the VAR models with the lag order of one were indicated as optimal using Akaike's Information Criterion, Hannan-Quinn Criterion, Schwarz Criterion, and Final Prediction Error for the majority of the datasets. These criteria are based on the natural logarithm of the determinant of the estimate of the residual covariance matrix $\hat{\Sigma}_\epsilon = \frac{1}{T}\sum_{t=1}^{T}\hat{\epsilon}_t\hat{\epsilon}_t'$, where T is the number of observations in the time series, to which a penalty for the number of parameters is added. While in the general case it is essential to pay particular attention to the parsimony of the model, in our case we need a VAR model with a lag order of at least two in order to have a full range of parameters in the corresponding VECM. Indeed, for the VAR with the lag order of one, the corresponding VECM equation has no lagged values and consists only of the cointegration relation, errors and the eventual deterministic terms:

$$\Delta\mathbf{y}_t = \mathbf{c} + \mathbf{dt} + \mathbf{\Pi}\mathbf{y}_{t-1} + \epsilon_t. \tag{6}$$

Such an equation implies that there is no connection between the first differences of the cause-specific mortality rates in the short run, which seems to be too strong an assumption to be adopted at the onset of the analysis. If as a preliminary step we allow for the presence of the $\mathbf{\Gamma}_1 \Delta \mathbf{y}_{t-1}$ term on the right-hand side of the equation, we can later study its relative importance as well as the significance of the coefficients of the corresponding parameter matrix $\mathbf{\Gamma}_1$. In case some of the matrix $\mathbf{\Gamma}_1$ coefficients turn out to be significantly different from 0, we can then analyze the short-run adjustments of the cause-specific mortality rates.

The models that were chosen as best describing the datasets in Arnold and Sherris (2016) comprised already the VAR(2) models for some of the countries. To be able to make the full analysis of the short-run adjustments, we check if for every dataset we can find models with the lag order of two that would suitably describe the data.

First, we apply the Johansen approach to define the number of cointegration relations and the form of the deterministic elements, then we test the residuals of the fitted VECM. The models suggested by the Johansen approach are shown in the Tables A1-A4 of Appendix (second column). These are the models that will be used in the subsequent analysis of the short- and long-term dynamics of the cause-specific mortality rates. Further columns contain the results of the tests on the residuals of the fitted VECM. The overall fit is similar to that of the models proposed in Arnold and Sherris (2016) except for the lower fit for the Japanese datasets. Also, it was not possible to find any suitable VAR(2) model for the E&W females with the JP females population structure, so in the following sections, we will use 19 datasets instead of 20.

For these new models, we also need to check the significance of the β matrix coefficients. As we can see in Tables 3 and 4, for 15 out of 19 considered datasets the cause-specific mortality rates corresponding to the causes I&P and External do not appear significantly (at a 1% significance level) in the long-term steady-states, which confirms the conclusion made in Arnold and Sherris (2016).

Table 3: p VALUES FOR THE NULL HYPOTHESIS THAT THE I&P AND THE EXTERNAL CAUSES OF DEATH ARE NOT SIGNIFICANTLY DIFFERENT FROM ZERO, US MALES POPULATION BASE, VAR(2) MODELS

Country	Model	Males	Females
US	VAR(2), QT, 1 CR	0.0655	0.0007
JP	VAR(2), TC, 2 CR	0.4878	0.0810
FR	VAR(2), NT, 1 CR	0.1945	-
	VAR(2), QT, 1 CR	-	0.0062
E&W	VAR(2), QT, 1 CR	0.1607	0.0015
AU	VAR(2), NT, 1 CR	-	0.0438
	VAR(2), QT, 1 CR	0.2570	-

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation. A null hypothesis is accepted at a α% significance level when the p value is higher than α%.

Table 4: p VALUES FOR THE NULL HYPOTHESIS THAT THE I&P AND THE EXTERNAL CAUSES OF DEATH ARE NOT SIGNIFICANTLY DIFFERENT FROM ZERO, JP FEMALES POPULATION BASE, VAR(2) MODELS

Country	Model	Males	Females
US	VAR(2), QT, 1 CR	0.0173	0.0000
JP	VAR(2), QT, 1 CR	0.0530	0.1906
FR	VAR(2), NT, 1 CR	0.0999	0.0696
E&W	VAR(2), QT, 1 CR	0.0788	No model
AU	VAR(2), NT, 1 CR	0.1917	-
	VAR(2), QT, 1 CR	-	0.0691

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation. A null hypothesis is accepted at a α% significance level when the p value is higher than α%.

As already mentioned, the VAR(2) models indicated in the Tables 3 and 4 will be used in the analysis that follows in section 3.

3 Dynamics of the cause-specific mortality rates

In the following sections, we present detailed analysis for the two datasets: US and JP males using the US males population structure. We summarize the most interesting findings and provide the details in the Appendix for the remaining 17 datasets.

3.1 Impulse-response analysis

First, to get a high-level overview of interactions between the cause-specific mortality rates (as described by the VECM equations built in the preceding chapter) we apply the framework of impulse-response analysis (see, e.g., Lütkepohl, 2005). At this point, we do not differentiate between the short- and long-term elements of the VECM, and analyze the system as a whole. More detailed analysis of the short- and long- term components including the statistical significance of the parameters will follow.

Basically, impulse-response analysis means that we first give a single shock to one cause-specific mortality rate and then analyze and compare the responses to this shock from every other cause-specific mortality rate. In this way, we study the impact of an unexpected change in a particular mortality rate on the dynamics of the system of mortality rates as this was observed in the past. The initial value taken by the variable that receives the shock is equal to its own standard deviation.

When analyzing the results, we successively adopt two points of view. First, we compare the impacts that a particular cause induces on other cause-specific mortality rates. Then, we compare the responses of a particular cause to the individual shocks received from the rest of mortality rates. In this way, we are able to determine not only if a particular cause influences the others and to what extent, but also if it is influenced by the rest of the causes and to what extent.

Once a shock is given to a particular cause-specific mortality rate, it propagates in the system and confers new values to the rest of the variables. This development can be suitably exposed on a chart. For example, Figure 1 shows the responses of every cause-specific mortality rate to the shock given to the Circulatory mortality rate for US males with the US males population structure (standard deviation of the differenced Circulatory mortality rate = 0.0235). Overall, the I&P, as well as the Respiratory mortality rates, show the most important reactions to the shock given to the Circulatory mortality rate. The Cancer and the External mortality rates are insignificantly impacted by the shock given to the Circulatory mortality rate. As for the Circulatory mortality rate itself, after having received the initial shock, it maintains the increased value until the end of the simulation period.

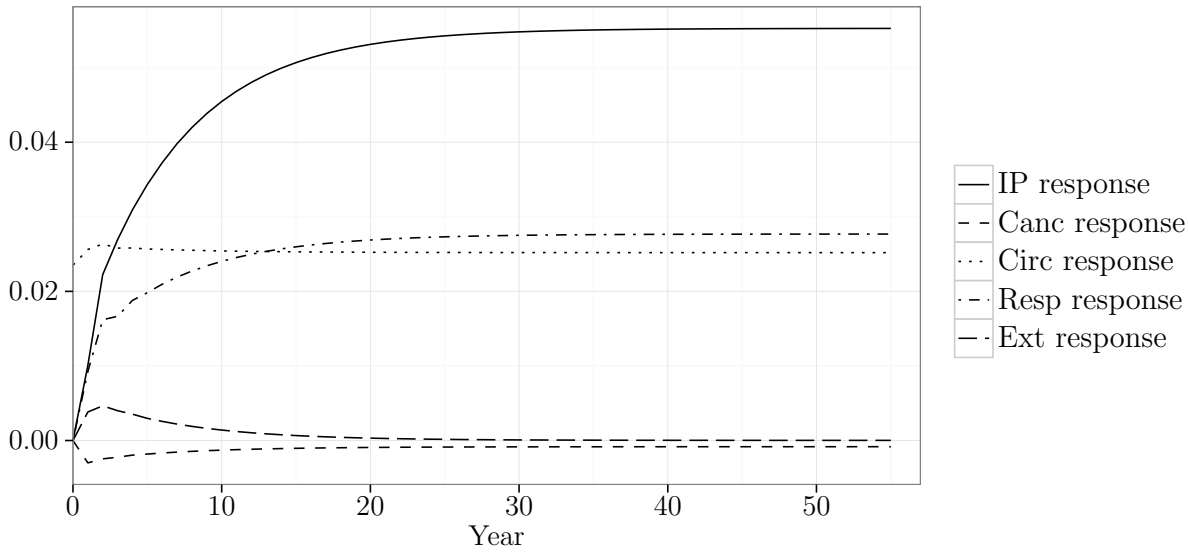

Figure 1: Responses to the shock given to the Circulatory cause,
US males, US males population base

The responses to the shocks from the Circulatory cause observed in the dataset for the JP males with the US males population structure are shown on Figure 2 (standard deviation of the differenced Circulatory mortality rate = 0.0501). Similar to the US males dataset, the Respiratory cause shows the most important response from the shock given to the Circulatory mortality rate. The response of the I&P mortality rate is slightly less important than that of the Respiratory rate. Interestingly, both responses have a negative sign whereas in the US males dataset they also have the same sign, but a positive one. One further observation for the JP males dataset is that the External causes also show a non-negligible response to the shock from the Circulatory cause.

We see that in both cases the system comes rather quickly to a new equilibrium. As the same observation holds for the rest of the datasets, we will compare the responses following individual shocks at time t = 20 years.

On Figures 1 and 2 the responses are shown in absolute values. However, since the cause-specific mortality rates have different standard deviations, each system receives a shock of a different amplitude. As such, the responses are not comparable between the

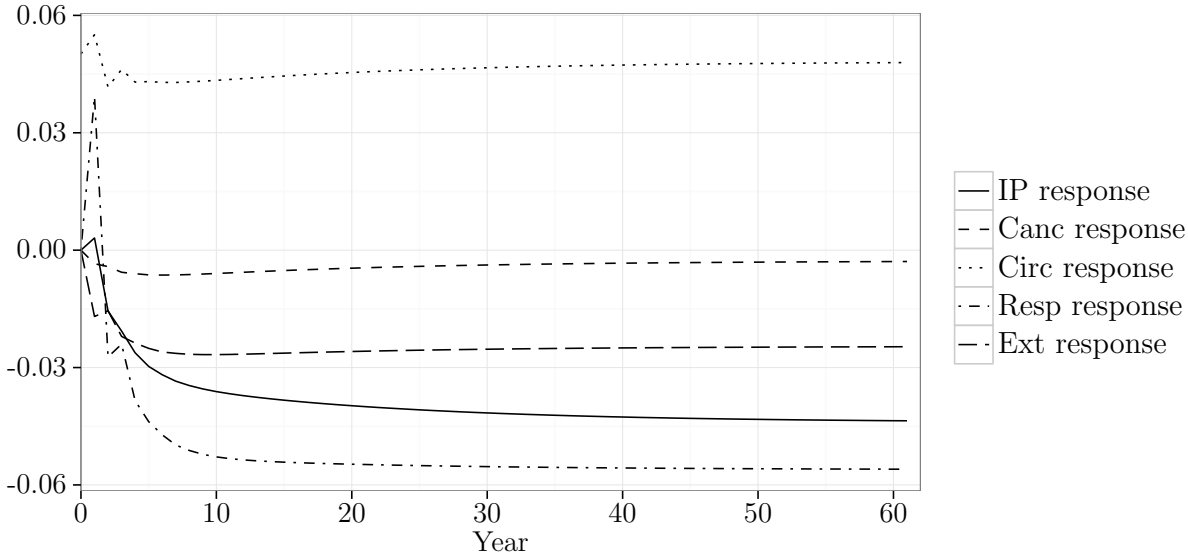

Figure 2: Responses to the shock given to the Circulatory cause, JP males, US males population base

datasets, that is, a response that would be considered high in one dataset can be considered as medium or low in another dataset. To bring the results to the same comparable basis, we will divide the absolute responses by the standard deviation of the cause-specific mortality rate that receives the shock. Then the response of the Respiratory cause to the shock from the Circulatory cause, i.e., the value of the cause-specific Respiratory mortality rate at time t = 20 will be expressed as a proportion of the shock received by the system, i.e., of the standard deviation of the cause-specific Circulatory mortality rate.

The results for every dataset are shown in Tables A5 and A6 of the Appendix. For the sake of readability, along with a numerical value we provide a label that indicates if the response can be considered as low, medium, or high: low if $|response| < 3/8$, medium if $3/8 \leq |response| < 7/8$, and high if $7/8 \leq |response|$. These labels are indicative only and were chosen to provide a roughly equal number of responses "medium" and "high" (40% of all responses), the rest being attributed to the category "low" (60% of all responses). The tables are organized as follows: Each row contains responses of all causes to the shock given to the cause X, each column contains responses of the cause Y to the shocks from all causes. In this way, we can judge simultaneously if a particular cause impacts each of the remaining causes and if it reacts to the shocks received from other causes and to what extent.

A synopsis of the observations summarized across the 19 datasets is presented in Table 5.

Table 5: IMPULSE-RESPONSE ANALYSIS: RESPONSE OF THE MORTALITY RATE Y TO THE SHOCK GIVEN TO THE CAUSE X, HIGH-LEVEL SUMMARY ACROSS ALL COUNTRIES, SEXES AND POPULATION STRUCTURES

X \ Y	I&P	Cancer	Circulatory	Respiratory	External
I&P		Low	Low	Low	Low
Cancer	High		Med	High	High
Circulatory	High	Low		High	Med
Respiratory	Med	Low	Low		Low
External	High	Low	Low	High	

In a nutshell:

- The I&P and the Respiratory causes have virtually no impact on all other causes, but show important responses to the shocks received from them.
- The Cancer and the Circulatory mortality rates have an important impact on other causes, especially on the I&P and the Respiratory mortality rates, but show little response to the shocks from other causes.
- The External causes have an equivocal behavior. On the one hand, they have almost no impact on the Cancer and the Circulatory causes, but importantly impact the I&P and the Respiratory causes. On the other hand, they are not impacted by the I&P and the Respiratory causes, but show important responses to the shocks from the Cancer and the Circulatory causes.

This first analysis shows that the cause-specific mortality rates show different behaviors. In the same time, when a system is analyzed as a whole, many effects are necessarily blended. Therefore, we need to decompose our analysis by separately assessing the short- and long-term dynamics of the system of the mortality rates in order to understand better how the causes of death are related to each other. In the following subsections, we will see what drivers in particular lie behind the observed development of the cause-specific mortality rates.

3.2 Short-term dynamics

Once the VECM equations are estimated for each dataset, we can use them to separate the short-term adjustments from the long-term dynamics for each cause-specific mortality rate. Indeed, if a particular coefficient γ_{ij} of matrix $\mathbf{\Gamma}_1$ is significant, then the cause i is influenced by the cause j in the short run. We calculate the standard deviations and the corresponding t-ratios of the estimates as shown in Lütkepohl (2005).

We start with the dataset for US males with the US males population structure. In the preceding chapter the following model was chosen as best describing this dataset:

$$\Delta \mathbf{y}_t = \mathbf{c} + \mathbf{dt} + \mathbf{\Gamma_1} \Delta \mathbf{y}_{t-1} + \mathbf{\Pi} \mathbf{y}_{t-1} + \epsilon_t =$$

$$= \begin{bmatrix} \mathbf{-2.7716} \\ -0.1500 \\ -0.2428 \\ \mathbf{-2.0573} \\ 0.1558 \end{bmatrix} + \begin{bmatrix} \mathbf{0.0096} \\ 0.0000 \\ 0.0000 \\ \mathbf{0.0050} \\ 0.0000 \end{bmatrix} \mathbf{t} + \begin{bmatrix} -0.1212 & -0.7074 & -0.1879 & 0.1743 & 0.3232 \\ -0.0036 & 0.0150 & \mathbf{-0.1659} & -0.0082 & \mathbf{0.1329} \\ -0.0429 & -0.1211 & 0.0337 & -0.0892 & 0.1963 \\ -0.1345 & -0.2944 & -0.0850 & \mathbf{-0.3810} & \mathbf{1.1189} \\ 0.0416 & -0.3232 & 0.1981 & \mathbf{-0.1455} & 0.2267 \end{bmatrix} \Delta \mathbf{y}_{t-1}$$

$$+\begin{bmatrix}-0.0331\\-0.0020\\-0.0030\\-0.0257\\0.0019\end{bmatrix}\begin{bmatrix}1.7716 & -5.4985 & -18.6015 & 13.2167 & 14.1321\end{bmatrix}\mathbf{y}_{t-1}+\boldsymbol{\epsilon}_t$$

The significant coefficients are in bold with the selected significance level of 5%. While many of the $\mathbf{\Gamma}_1$ coefficients are not significant, the cause-specific mortality rates from Cancer, Respiratory, and External causes are influenced by the lagged values of Circulatory and External, Respiratory and External, and Respiratory causes respectively. We see that in this dataset three out of five cause-specific mortality rates experience the short-term adjustments from other causes. Hence, it was justified to use the VAR(2) setup and include the lagged values of $\Delta\mathbf{y}_t$ into the model. Otherwise, an essential piece of information on the development of the cause-specific mortality rates would not have been accounted for. Another interesting observation is that only the Respiratory mortality rate shows the autoregressive feature. In other words, the corresponding cause-specific mortality rate is dependent on the lagged value of itself.

As for the dataset of JP males with the US males population structure, the chosen VECM has two cointegration relations with a constant and a trend, the latter being restricted to the cointegration term:

$$\Delta\mathbf{y}_t=\mathbf{c}+\mathbf{\Gamma_1}\Delta\mathbf{y}_{t-1}+\alpha\beta'(\mathbf{y}_{t-1}+(t-1))+\epsilon_t=$$

$$=\begin{bmatrix}0.3960\\\mathbf{-0.8539}\\-0.7600\\-1.8252\\-0.7695\end{bmatrix}+\begin{bmatrix}-0.1679 & 0.5510 & 0.1982 & 0.0335 & 0.2440\\0.0143 & \mathbf{-0.3818} & -0.0268 & 0.0184 & 0.0406\\0.1343 & 0.7531 & 0.1711 & \mathbf{-0.1692} & 0.0055\\0.1477 & 2.1179 & \mathbf{1.3108} & \mathbf{-0.5537} & -0.1487\\0.0307 & 0.3048 & -0.2898 & 0.1305 & -0.0906\end{bmatrix}\Delta\mathbf{y}_{t-1}$$

$$+\begin{bmatrix}0.0261 & 0.3600\\-0.0186 & -0.0425\\-0.0126 & 0.0399\\0.0054 & 0.8417\\-0.0154 & -0.0149\end{bmatrix}\left[\begin{bmatrix}1.5951 & 7.7055 & 0.9876 & -1.5822 & 1.8454\\-1.0848 & 9.3172 & -10.1473 & -6.6630 & -3.7839\end{bmatrix}\mathbf{y}_{t-1}+\begin{bmatrix}0.1851\\-0.3817\end{bmatrix}(t-1)\right]$$

$$+\boldsymbol{\epsilon}_t$$

Also for this dataset, many of the $\mathbf{\Gamma}_1$ coefficients are not significant. On the other hand, the cause-specific mortality rates corresponding to the causes Cancer, Circulatory, and Respiratory causes are influenced by the lagged values of the Cancer, Respiratory, Circulatory and Respiratory mortality rates respectively. Again, three out of five cause-specific mortality rates experience the short-term adjustments from other causes. Therefore, it would not be justified to use the VAR(1) setup for JP males with the US males population structure. Similar to the US males dataset, the Respiratory cause shows the autoregressive feature as well as the Cancer cause.

After the analysis was repeated for the rest of the datasets using both the US males and the JP females population structures, the results can be summarized as follows:

- In every dataset, there is at least one cause-specific mortality rate that is significantly impacted by other causes in the short run. For this reason, it would not be optimal to use the VAR models with the lag order one instead of two.

- While in the short run the I&P and Cancer causes are rarely impacted by other causes, they also infrequently impact the rest of the causes, i.e., they show a development mostly independent from other causes in the short run.
- On the other hand, the Circulatory, Respiratory, and the External causes are frequently impacted by one or more causes in the short run and also occasionally impact other causes. Hence, these cause-specific mortality rates are more linked in their development to other causes than the I&P and Cancer mortality rates are.
- The Respiratory cause consistently shows the autoregressive feature. In other words, in many datasets the corresponding cause-specific mortality rate is dependent on the lagged value of itself.
- For all datasets, the larger part of the significant coefficients are negative, i.e., more often than not the change in the cause-specific mortality rate goes in the opposite direction of the short-term variation of this and/or other cause-specific mortality rates at the previous point in time. More specifically, this means that if the mortality rate of a particular cause of death increases (decreases), the other causes will tend to decrease (increase) in the short run.

The detailed overview of the significant coefficients in $\mathbf{\Gamma}_1$ matrix for each dataset is presented in the tables A7 to A10 of the Appendix.

3.3 Long-term dynamics

The α matrix allows us to estimate how deviations from the steady-states impact the cause-specific mortality rates. For $r = 1$ (which is the case for the majority of the datasets) we can write the long-term component as follows:

$$\mathbf{\Pi y}_{t-1} = \alpha\beta'\mathbf{y_{t-1}} = \begin{bmatrix}\alpha_1\\ \alpha_2\\ \alpha_3\\ \alpha_4\\ \alpha_5\end{bmatrix} \begin{bmatrix}\beta_1 & \beta_2 & \beta_3 & \beta_4 & \beta_5\end{bmatrix} \mathbf{y}_{t-1} =$$

$$= \begin{bmatrix}\alpha_1\\ \alpha_2\\ \alpha_3\\ \alpha_4\\ \alpha_5\end{bmatrix} (\beta_1 y_{1t-1} + \beta_2 y_{2t-1} + \beta_3 y_{3t-1} + \beta_4 y_{4t-1} + \beta_5 y_{5t-1}) \tag{7}$$

This way, if a particular coefficient α_i is significant, the long-term component on the right-hand side of the equation (2) is important in explaining the past variations of the corresponding cause-specific mortality rate on the left-hand side. Moreover, the value of this coefficient shows the extent to which the long-term component contributes to the variation of the cause-specific mortality rate in question. As in the previous subsection, we calculated the standard deviations and the corresponding t-ratios of the estimates of α as shown in Lütkepohl (2005). The following table shows the p-values for the US males and JP males datasets using the US males population structure.

Table 6: p VALUES FOR THE NULL HYPOTHESIS THAT α_i IS NOT SIGNIFICANTLY DIFFERENT FROM ZERO,
US MALES POPULATION STRUCTURE

Country/Sex	Model		ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US Males	VAR(2), QT, 1 CR	α_i	0.000	0.085	0.243	0.001	0.496
JP Males	VAR(2), TC, 2 CR	α_{1i}	0.026	0.000	0.183	0.837	0.182
		α_{2i}	0.000	0.001	0.500	0.000	0.836

On the one hand, this example shows that for the US males dataset the long-term component enters the equations for the I&P and Respiratory mortality rates with a significant coefficient (at a 5% significance level). On the other hand, the equations for Cancer, Circulatory, and External mortality rates are not significantly impacted by the long-term equilibrium. As for JP males, there are two long-term components that each enter with a significant coefficient the equations for the I&P and Cancer mortality rates; also, the second component enters with a significant coefficient the equation for the Respiratory mortality rate.

We repeated the analysis for the rest of the datasets, and the results can be summarized as follows:

- The I&P and the Respiratory causes seem to be the most impacted by the long-term component: The corresponding α_i coefficients are significant in 15 out of 19 datasets. A similar observation was made using the framework of the impulse-response analysis, as there the I&P and the Respiratory mortality rates showed an important reaction to the shocks from other causes. Hence, these shocks propagate in the system via the cointegration relation(s).

- The External causes seem to be the least impacted: The corresponding α_i coefficients are significant in only 5 out of 19 datasets. Interestingly, the results of the impulse-response analysis for the External causes were equivocal in that there was an important reaction to the shocks from the Cancer and the Circulatory causes, but a low response to the shocks from the I&P and the Respiratory causes. The impact from the Cancer and the Circulatory causes may hence come from the short-term adjustments.

- Results for the Cancer and the Circulatory causes are more difficult to interpret: The corresponding α_i coefficients are significant in respectively 9 and 11 out of 19 datasets. The results of the impulse-response analysis also showed low reactions of the Cancer and the Circulatory mortality rates to the shocks from other causes.

Interestingly, while as was mentioned above, the I&P and the External causes do not participate conjointly in the long-term equilibrium, they show different behaviors when it comes to the impact they experience from this long-term steady-state. Indeed, the cointegration relations often enter the equation for the I&P mortality rate with a significant coefficient, but seldom have an effect on the External causes. Therefore, only the External causes show behavior that is entirely independent from the long-term equilibrium state and, possibly, aging.

The overview of the results for the remaining datasets is shown in the Tables A11 and A12 of the Appendix.

3.4 Long-term vs. short-term dynamics

In the previous sections, we have analyzed the short- and long-term elements separately. Now we want to assess the relative importance of the long- and short-run forces. For this purpose, we break down the expected cause-specific mortality rates at time *t*, based on the information available up to time *t-1*, in two elements: the short-term (ST) and the long-term (LT) components. By comparing the behavior of each of these elements with the realized change in the mortality rates we assess the relative importance of the long- and short-run forces in terms of their contribution to the variation of the cause-specific mortality rates.

For illustrative purposes, we present the results for the Respiratory equation for US males (Figure 3) and the I&P equation for JP males (Figure 4), both datasets using the US males population structure. In the first case, the actual mortality changes fluctuate primarily with the short-term components (the correlation coefficient between $\Delta Resp_t$ and the LT: 0.268, between $\Delta Resp_t$ and the ST: 0.336):

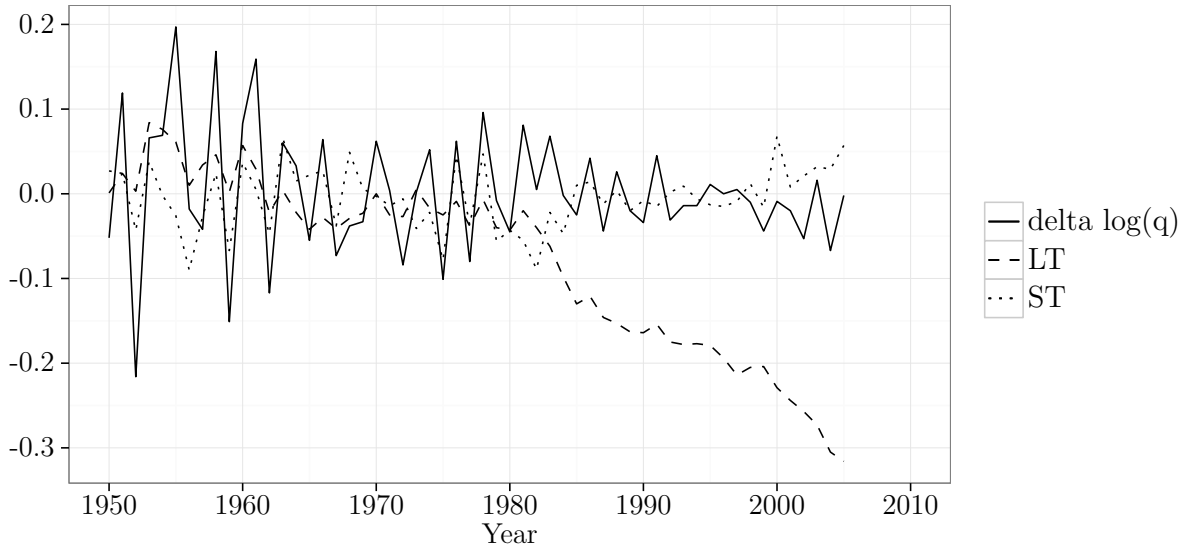

Figure 3: Respiratory cause: actual mortality changes, long- and short-term components (US males, US males population structure)

As for JP males, the actual mortality changes fluctuate primarily with the long-term component (the correlation coefficient between ΔIP_t and the LT: 0.570, between ΔIP_t and the ST: -0.177).

As not every equation contains the long-term component, the results for the rest of the datasets are formulated for those cases where the long-term component is present with a significant coefficient α_i.

- Out of 15 datasets for which the I&P mortality rate equation contains the long-term component, in 13 cases the data fluctuates primarily with the long-term component

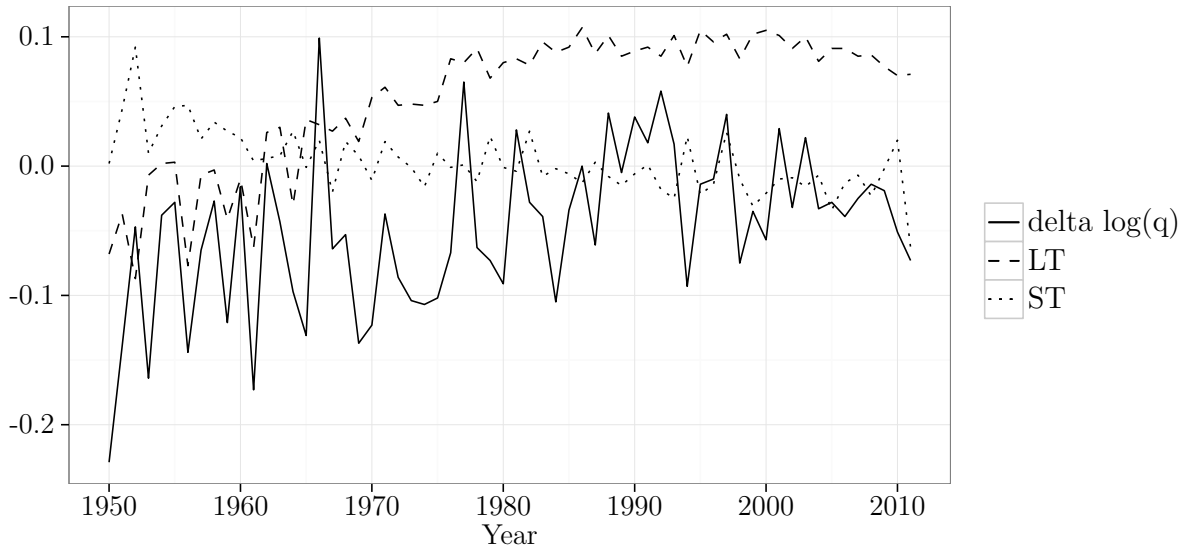

Figure 4: I&P cause: actual mortality changes, long-term and short term components (JP males, US males population structure)

(i.e., the correlation coefficient between the data points and the long-term component is higher than that between the data points and the short-term component).

- Out of 9 datasets for which the Cancer mortality rate equation contains the long-term component, in 8 cases the data fluctuates primarily with the long-term component.
- Out of 11 datasets for which the Circulatory mortality rate equation contains the long-term component, in 4 cases the data fluctuates primarily with the long-term component.
- Out of 15 datasets for which the Respiratory mortality rate equation contains the long-term component, in 7 cases the data fluctuates primarily with the long-term component.
- Out of 5 datasets for which the External mortality rate equation contains the long-term component, only in 1 case does the data fluctuate primarily with the long-term component.

Summarizing the results stated above, we can say that every time the equation contains the long-term component, the cause-specific mortality rate resembles in its behavior the long-term component rather than the short-term one for the causes I&P and Cancer. The opposite is true for the Circulatory and External causes. The Respiratory mortality rate resembles in its behavior the long-term component as often as it resembles the short-term component.

This observation is not surprising for the I&P and the Respiratory causes. As we have seen in previous sections, only these cause-specific mortality rates are often impacted by the long-term equilibrium state. In the same time, the I&P mortality rate is rarely, and

the Respiratory is frequently impacted by the short-term component. It could have been expected that the I&P mortality rate data will fluctuate with the long-term component in the majority of cases where the I&P mortality rate contains the cointegration relation(s). In its turn, the Respiratory mortality rate fluctuates either with the short-term component or with the long-term component in roughly similar proportions. Therefore, the correlation analysis reinforces the conclusions of the previous sections for these two rates.

A similar conclusion holds for the Circulatory mortality rate: as it is frequently impacted in the short run and only occasionally in the long, the short-term components play unsurprisingly a more important role in the correlation analysis.

Regarding the Cancer mortality rate, we have seen that it was infrequently impacted by both short- and long-term components. As none of the effects can be called dominant, the correlation analysis helps to identify the component that plays a more important role in the development of this cause-specific mortality rate, in this case, the long-term.

As for the External causes, the corresponding cause-specific mortality rate is often impacted by the short-term component and virtually never by the long-term one. Even in those rare cases in which the cointegration relation enters the equation with a significant coefficient, the data fluctuate more with a short-term component.

The detailed results for each dataset are shown in Tables A13-A16 of the Appendix.

4 Conclusions

The analysis of dynamics of the cause-specific mortality rates shows that they are dependent from each other in both short- and long-run. Although the observed experience will never exactly repeat itself in the future, the following observations can help practitioners set more informed assumptions on the future development of mortality rates:

- The common long-run trend shared by the cause-specific mortality rates is contingent on the evolution of the Cancer, the Circulatory, and the Respiratory mortality rates, as these are the causes that significantly contribute to the cointegration relation between the mortality rates.

- Once the common long-run trend is defined, it more heavily impacts the development of the I&P and the Respiratory mortality rates and to a lesser extent the development of the Cancer and the Circulatory mortality rates. The External causes are exempt from the influence of the common long-term relationship between the causes.

- In the short run, the Respiratory mortality rate consistently shows the autoregressive feature.

- Although the short-run dependencies are more challenging to model, they are significantly pronounced in the development pattern of the Circulatory, Respiratory, and the External mortality rates. In other words, these rates are dependent on each other in the short run.

Coming back to the conclusion made in Arnold and Sherris (2016) that the I&P and the External causes do not participate conjointly in the long-term steady-state, we

see that these causes differ in the way they are impacted by the long-term equilibrium. Though the I&P mortality rate is often impacted by the cointegration relation(s) and when it is, fluctuates more with the long-term component, the External causes show the opposite behavior: the corresponding rate is almost never impacted by the cointegration relation(s), and when it is, it fluctuates more with the short-term component.

We see that the development of the External causes mortality rate is completely independent from the long-term equilibrium both in terms of the contribution to, and influence experienced from, the steady-state. This is a behavior of what could be called a genuinely exogenous cause of mortality as we observe no long-term impact to or from this cause. It develops in a way that is entirely independent of the observed equilibrium between the rest of the cause-specific mortality rates and is subject to only short-term shocks from other causes. Basically, this observation is not surprising, as under the External causes are grouped such causes as transport and other accidents (falls, poisoning, accidental fire, drowning), suicides, homicides, and war injuries. So it is rather difficult to imagine a link connecting these mortality rates to the rest of the mortality causes that could be observed over a long time. On the contrary, these causes of mortality can rather be characterized by randomness and "bad luck" rather than by a steady long-term development.

In turn, the I&P mortality rate does not influence the long-term equilibrium observed between the cause-specific mortality rates but is rather sensitive to the impacts received from this equilibrium. Occasionally, it is also subject to short-term shocks from other causes. We can conclude that while the evolution of the I&P mortality rate does not influence the development of other cause-specific mortality rates, i.e., a sudden increase or drop in the I&P mortality rate will not affect the rest of the cause-specific mortality rates, its own development depends to a great extent from other causes of death, especially in the long run. Such behavior cannot be described as fully independent, and so the I&P cause cannot be classified as a truly exogenous cause in the same way the External causes can.

These observations are consistent with the intuition that the biological processes of aging are reflected in the common stochastic trend shared by the cause-specific mortality rates. Indeed, while it can seem that the infectious or parasitic diseases are similar to the external causes in that the origin of the force affecting the human body lies outside, the underlying biological processes are more complicated, as human beings are not equal when they face an infection. Even during severe epidemics, the probability of getting sick and dying depends to a large extent on the internal immune forces of the person, which in turn, depend, among other factors, on age. A well-known example is influenza that is the most dangerous for the elderly. When advancing in age, we are more and more confronted with competing risks such that a decrease in mortality from the circulatory diseases, for example, would leave more vulnerable persons alive who could then die from an infectious disease during an epidemic. It is then understandable that the I&P mortality rate, while not being a part of the long-term equilibrium, is substantially affected by it. Our results then confirm and reinforce the link between the cointegration relations observed within a set of cause-specific mortality rates and the biological processes of aging.

One further possible application of the present study is the calibration of copula-based models for the cause-specific mortality rates that remains an open question. Indeed, due to the indentifiability issue raised by Tsiatis (1975), one usually has to assume that the

dependence is represented by a known copula with known parameters. In the same time, copula-based models are highly sensitive with respect to these choices (Dimitrova et al., 2013). As pointed out in Kaishev et al. (2007), "the free parameters could be set according to a priori available (medical) information, about the degree of pairwise dependence between the two competing risks, expressed through Kendall's τ and/or Sperman's ρ". In the absence of additional information and to demonstrate the sensitivity of the results, the authors use four different copulas and five different values of Kendall's τ, ranging from -0.91 (extreme negative dependence) to 0.91 (extreme positive dependence). In Li and Lu (2019), the authors go further and by introducing hierarchical Archimedean copulas succeed in building a model that allows for different levels of association between the causes of death. For this, they group the causes in different clusters based on the (assumed) level of dependence between the causes, but also admit that the introduced hierarchical structure is not unique. Although our study cannot provide an exact value of parameters to be used in copula-based models, a certain degree of pairwise association (correlation) between the causes of death can be inferred from the results of the impulse-response analysis (Section 3.1). This could help researchers working with copula-based models further reduce the possible range of free parameters that otherwise have to be chosen arbitrarily. Also, the revealed differences in the long- and short-term development of the cause-specific mortality rates can serve as the basis for building clusters of the causes.

In the current study, we limited our analysis to the total cause-specific mortality rates and did not differentiate by age. Yet, it is intuitively clear that when analyzed by age, the mortality rates will present different development patterns. As the cause-specific death numbers are available in the WHO database by five-year age groups, it seems to be a promising path to integrate the age specifics of the mortality rates into the modeling process. However, this remains a challenging task, as, on the one hand, the cointegration tests have been developed for systems with maximum 12 variables (Osterwald-Lenum, 1992), and, on the other, the observation horizon, which goes back as far as 1950, is also rather brief. In our opinion, analysis trying to overcome these difficulties while preserving the information on the age profile has the potential to deliver additional insights on the interaction of the cause-specific mortality rates. Moreover, the biological processes of aging may probably be easier to measure once the data on young ages are excluded from the analysis, as by definition, the aging risk factor becomes more important the longer we live. For this reason, the analysis of the cause-specific mortality rates excluding young ages may provide a better measure of the aging process.

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APPENDIX

Table A1: TESTS ON RESIDUALS OF THE FITTED VECM, MALES, US MALES POPULATION BASE, VAR(2) MODELS

		p value					
		Autocorrelation			Normality		
Country	Model	15 lags	25 lags	35 lags	Skewness	Kurtosis	Both
US	VAR(2), QT, 1 CR	0.212	0.146	0.181	0.546	0.020	0.066
JP	VAR(2), TC, 2 CR	0.526	0.764	0.810	0.749	0.002	0.015
FR	VAR(2), NT, 1 CR	0.354	0.644	0.824	0.467	0.154	0.244
E&W	VAR(2), QT, 1 CR	0.209	0.146	0.207	0.510	0.558	0.607
AU	VAR(2), QT, 1 CR	0.594	0.297	0.405	0.876	0.002	0.027

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A2: TESTS ON RESIDUALS OF THE FITTED VECM, FEMALES, US MALES POPULATION BASE, VAR(2) MODELS

		p value					
		Autocorrelation			Normality		
Country	Model	15 lags	25 lags	35 lags	Skewness	Kurtosis	Both
US	VAR(2), QT, 1 CR	0.133	0.007	0.021	0.768	0.141	0.369
JP	VAR(2), TC, 2 CR	0.539	0.601	0.769	0.007	0.000	0.000
FR	VAR(2), QT, 1 CR	0.066	0.286	0.491	0.420	0.038	0.080
E&W	VAR(2), QT, 1 CR	0.389	0.307	0.353	0.025	0.000	0.000
AU	VAR(2), NT, 1 CR	0.238	0.284	0.262	0.652	0.059	0.175

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A3: TESTS ON RESIDUALS OF THE FITTED VECM, MALES, JP FEMALES POPULATION BASE, VAR(2) MODELS

		p value					
		Autocorrelation			Normality		
Country	Model	15 lags	25 lags	35 lags	Skewness	Kurtosis	Both
US	VAR(2), QT, 1 CR	0.496	0.202	0.202	0.879	0.189	0.511
JPn	VAR(2), QT, 1 CR	0.168	0.103	0.081	0.225	0.047	0.052
FR	VAR(2), NT, 1 CR	0.398	0.615	0.534	0.139	0.009	0.009
E&W	VAR(2), QT, 1 CR	0.225	0.094	0.099	0.722	0.556	0.743
AU	VAR(2), NT, 1 CR	0.521	0.397	0.437	0.407	0.013	0.034

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A4: TESTS ON RESIDUALS OF THE FITTED VECM, FEMALES, JP FEMALES POPULATION BASE, VAR(2) MODELS

		p value					
		Autocorrelation			Normality		
Country	Model	15 lags	25 lags	35 lags	Skewness	Kurtosis	Both
US	VAR(2), QT, 1 CR	0.168	0.005	0.021	0.123	0.010	0.009
JP	VAR(2), QT, 1 CR	0.469	0.625	0.664	0.000	0.000	0.000
FR	VAR(2), NT, 1 CR	0.096	0.426	0.381	0.324	0.097	0.127
E&W	No suitable VAR(2) model						
AU	VAR(2), QT, 1 CR	0.548	0.688	0.353	0.488	0.046	0.108

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A5: IMPULSE-RESPONSE ANALYSIS: RESPONSE OF THE MORTALITY RATE Y FROM THE SHOCK GIVEN TO THE CAUSE X, US MALES POPULATION BASE

Cause		Males					Females				
X \ Y	Country	I&P	Canc	Circ	Resp	Ext	I&P	Canc	Circ	Resp	Ext
I&P	US	0.53 (Med)	-0.01 (Low)	-0.02 (Low)	-0.2 (Low)	0.1 (Low)	-0.05 (Low)	-0.12 (Low)	-0.11 (Low)	-0.47 (Med)	0.24 (Low)
I&P	JP	0.97 (High)	-0.13 (Low)	-0.09 (Low)	-0.12 (Low)	-0.14 (Low)	0.58 (Med)	0.03 (Low)	0.13 (Low)	0.01 (Low)	0.01 (Low)
I&P	FR	1.15 (High)	-0.17 (Low)	-0.07 (Low)	0.38 (Med)	-0.21 (Low)	1.2 (High)	-0.03 (Low)	0.07 (Low)	0.22 (Low)	-0.18 (Low)
I&P	E&W	1.59 (High)	-0.14 (Low)	-0.09 (Low)	-0.33 (Low)	-0.38 (Med)	0.37 (Low)	-0.03 (Low)	-0.08 (Low)	-0.33 (Low)	0.17 (Low)
I&P	AU	0.73 (Med)	0.01 (Low)	-0.02 (Low)	-0.12 (Low)	-0.03 (Low)	-0.1 (Low)	0.01 (Low)	0.16 (Low)	-0.1 (Low)	0.2 (Low)
Canc	US	2.05 (High)	1.13 (High)	-0.15 (Low)	0.75 (Med)	-0.78 (Med)	3.39 (High)	1.64 (High)	-0.61 (Med)	0.8 (Med)	-1.78 (High)
Canc	JP	1.06 (High)	0.24 (Low)	-0.27 (Low)	0.75 (Med)	-0.26 (Low)	4.37 (High)	0.5 (Med)	0.03 (Low)	3.38 (High)	0.98 (High)
Canc	FR	1.46 (High)	0.16 (Low)	-0.92 (High)	0.64 (Med)	-0.86 (Med)	2.27 (High)	0.36 (Low)	0.15 (Low)	4.56 (High)	-0.45 (Med)
Canc	E&W	1.61 (High)	0.58 (Med)	-0.1 (Low)	-2.57 (High)	-1.42 (High)	-4.09 (High)	0.7 (Med)	-0.69 (Med)	-2.96 (High)	0.59 (Med)
Canc	AU	-0.18 (Low)	0.68 (Med)	-0.22 (Low)	-1.33 (High)	-0.37 (Low)	0.97 (High)	0.77 (Med)	-0.01 (Low)	0.57 (Med)	0.03 (Low)
Circ	US	2.26 (High)	-0.04 (Low)	1.07 (High)	1.14 (High)	0.01 (Low)	1.34 (High)	0.05 (Low)	1.42 (High)	1.19 (High)	0.22 (Low)
Circ	JP	-0.79 (Med)	-0.09 (Low)	0.91 (High)	-1.09 (High)	-0.52 (Med)	0.1 (Low)	-0.09 (Low)	0.22 (Low)	-1.03 (High)	-0.61 (Med)
Circ	FR	-1.82 (High)	1.16 (High)	1.96 (High)	-1.23 (High)	1.29 (High)	-0.23 (Low)	0.13 (Low)	0.86 (Med)	-0.51 (Med)	0.17 (Low)
Circ	E&W	0.67 (Med)	-0.11 (Low)	0.69 (Med)	-0.03 (Low)	-0.33 (Low)	0.92 (High)	0.06 (Low)	1.01 (High)	1.04 (High)	-0.2 (Low)
Circ	AU	0.4 (Med)	0.05 (Low)	0.83 (Med)	0.67 (Med)	0.16 (Low)	-0.2 (Low)	0.01 (Low)	0.71 (Med)	-0.7 (Med)	0.01 (Low)
Resp	US	-1.37 (High)	-0.08 (Low)	-0.07 (Low)	0.01 (Low)	0.06 (Low)	-0.77 (Med)	-0.11 (Low)	-0.12 (Low)	0.19 (Low)	-0.01 (Low)
Resp	JP	-0.27 (Low)	0.05 (Low)	-0.05 (Low)	0.15 (Low)	0.07 (Low)	-0.02 (Low)	0.01 (Low)	-0.14 (Low)	0.35 (Low)	0.13 (Low)
Resp	FR	-0.41 (Med)	0.2 (Low)	0.05 (Low)	0.05 (Low)	0.13 (Low)	-0.19 (Low)	0.01 (Low)	-0.08 (Low)	0.19 (Low)	-0.02 (Low)
Resp	E&W	-0.05 (Low)	-0.02 (Low)	-0.05 (Low)	0.53 (Med)	-0.04 (Low)	-0.56 (Med)	-0.03 (Low)	-0.13 (Low)	0.31 (Low)	0.07 (Low)
Resp	AU	-0.34 (Low)	-0.05 (Low)	-0.11 (Low)	-0.02 (Low)	-0.07 (Low)	0.65 (Med)	-0.02 (Low)	-0.16 (Low)	0.84 (Med)	-0.17 (Low)
Ext	US	-4.06 (High)	-0.08 (Low)	0.16 (Low)	-1.01 (High)	1.71 (High)	-3.39 (High)	-0.38 (Med)	-0.16 (Low)	-1.47 (High)	1.38 (High)
Ext	JP	0.42 (Med)	-0.19 (Low)	-0.26 (Low)	-0.32 (Low)	0.68 (Med)	-1.35 (High)	0.16 (Low)	0.58 (Med)	-0.13 (Low)	0.79 (Med)
Ext	FR	0.44 (Med)	-0.28 (Low)	-0.08 (Low)	1.08 (High)	0.63 (Med)	-0.6 (Med)	0.07 (Low)	-0.01 (Low)	0.16 (Low)	1.07 (High)
Ext	E&W	-0.38 (Med)	-0.06 (Low)	-0.05 (Low)	-0.56 (Med)	0.53 (Med)	0.01 (Low)	-0.04 (Low)	-0.05 (Low)	-0.35 (Low)	0.98 (High)
Ext	AU	0.07 (Low)	-0.04 (Low)	0.04 (Low)	0.42 (Med)	0.84 (Med)	-1.1 (High)	0.02 (Low)	0.32 (Low)	-0.16 (Low)	1.05 (High)

Table A6: IMPULSE-RESPONSE ANALYSIS: RESPONSE OF THE MORTALITY RATE Y FROM THE SHOCK GIVEN TO THE CAUSE X, JP FEMALES POPULATION BASE

Cause		Males					Females				
X \ Y	Country	I&P	Canc	Circ	Resp	Ext	I&P	Canc	Circ	Resp	Ext
I&P	US	0.41 (Med)	-0.02 (Low)	-0.04 (Low)	-0.21 (Low)	0.09 (Low)	1.25 (High)	0.04 (Low)	-0.07 (Low)	0.16 (Low)	0.03 (Low)
I&P	JP	0.92 (High)	-0.02 (Low)	0.18 (Low)	0.64 (Med)	0.11 (Low)	0.81 (Med)	0 (Low)	0.09 (Low)	0.1 (Low)	0.1 (Low)
I&P	FR	1.07 (High)	-0.17 (Low)	0 (Low)	0.37 (Low)	-0.14 (Low)	1.26 (High)	-0.04 (Low)	-0.14 (Low)	0.37 (Low)	-0.48 (Med)
I&P	E&W	0.78 (Med)	-0.03 (Low)	-0.08 (Low)	0.05 (Low)	0.23 (Low)	No suitable VAR(2) model				
I&P	AU	0.3 (Low)	0.03 (Low)	0.08 (Low)	-0.08 (Low)	0.04 (Low)	0.71 (Med)	0.03 (Low)	0 (Low)	0.15 (Low)	-0.02 (Low)
Canc	US	1.22 (High)	1.26 (High)	-0.02 (Low)	0.78 (Med)	-0.48 (Med)	-2.25 (High)	0.94 (High)	-0.8 (Med)	-2.37 (High)	-1.13 (High)
Canc	JP	0.9 (High)	0.95 (High)	0.73 (Med)	2 (High)	0.79 (Med)	0.46 (Med)	0.99 (High)	1.3 (High)	2.44 (High)	0.76 (Med)
Canc	FR	0.69 (Med)	0.5 (Med)	-0.88 (High)	-0.39 (Med)	-0.75 (Med)	2.95 (High)	0.35 (Low)	-0.8 (Med)	5.79 (High)	-2.01 (High)
Canc	E&W	-0.57 (Med)	1.15 (High)	0.4 (Med)	-0.87 (Med)	0.08 (Low)	No suitable VAR(2) model				
Canc	AU	1.15 (High)	0.65 (Med)	-0.24 (Low)	-0.19 (Low)	-0.27 (Low)	0.1 (Low)	0.84 (Med)	0 (Low)	-0.92 (High)	0.29 (Low)
Circ	US	2.65 (High)	-0.06 (Low)	1.16 (High)	1.52 (High)	0.02 (Low)	3.3 (High)	0.44 (Med)	1.53 (High)	2.48 (High)	0.02 (Low)
Circ	JP	-0.41 (Med)	-0.06 (Low)	0.91 (High)	-0.67 (Med)	-0.48 (Med)	-0.46 (Med)	-0.03 (Low)	0.6 (Med)	-1.05 (High)	-0.52 (Med)
Circ	FR	-1.39 (High)	1.28 (High)	2.15 (High)	-1.13 (High)	1.5 (High)	0.03 (Low)	0.09 (Low)	0.97 (High)	0.08 (Low)	0.09 (Low)
Circ	E&W	1.01 (High)	0.03 (Low)	1.04 (High)	0.51 (Med)	-0.55 (Med)	No suitable VAR(2) model				
Circ	AU	-0.33 (Low)	0.13 (Low)	0.91 (High)	-0.11 (Low)	0.07 (Low)	0.27 (Low)	0.03 (Low)	0.59 (Med)	-0.63 (Med)	-0.25 (Low)
Resp	US	-1.12 (High)	-0.05 (Low)	-0.12 (Low)	-0.03 (Low)	-0.06 (Low)	-0.75 (Med)	-0.14 (Low)	-0.11 (Low)	0.16 (Low)	-0.03 (Low)
Resp	JP	-0.07 (Low)	0.02 (Low)	-0.21 (Low)	0.13 (Low)	-0.01 (Low)	-0.03 (Low)	0.02 (Low)	-0.15 (Low)	0.25 (Low)	0.06 (Low)
Resp	FR	-0.3 (Low)	0.21 (Low)	0.06 (Low)	0.14 (Low)	0.16 (Low)	-0.23 (Low)	0.02 (Low)	-0.03 (Low)	0.13 (Low)	0.06 (Low)
Resp	E&W	-0.36 (Low)	-0.02 (Low)	-0.11 (Low)	0.61 (Med)	0.19 (Low)	No suitable VAR(2) model				
Resp	AU	-1.36 (High)	0.13 (Low)	0.27 (Low)	0.67 (Med)	0.14 (Low)	-0.15 (Low)	-0.03 (Low)	-0.08 (Low)	0.14 (Low)	0.09 (Low)
Ext	US	-4.06 (High)	-0.11 (Low)	0.11 (Low)	-1.4 (High)	1.47 (High)	-5.92 (High)	-0.93 (High)	-0.27 (Low)	-3.36 (High)	1.56 (High)
Ext	JP	0.14 (Low)	0.01 (Low)	-0.04 (Low)	-0.53 (Med)	0.75 (Med)	0.08 (Low)	0.01 (Low)	0.07 (Low)	0.13 (Low)	0.68 (Med)
Ext	FR	1.11 (High)	-1.05 (High)	-0.73 (Med)	2.26 (High)	-0.09 (Low)	-1.49 (High)	0.18 (Low)	0.52 (Med)	-1.41 (High)	2.39 (High)
Ext	E&W	1.17 (High)	0.09 (Low)	0.39 (Med)	-0.18 (Low)	-0.06 (Low)	No suitable VAR(2) model				
Ext	AU	0.72 (Med)	-0.15 (Low)	-0.18 (Low)	0.08 (Low)	0.78 (Med)	0.16 (Low)	0.03 (Low)	0.17 (Low)	0.69 (Med)	0.94 (High)

Table A7: $\mathbf{\Gamma}_1$ COEFFICIENTS THAT ARE SIGNIFICANTLY DIFFERENT FROM ZERO, SIGNIFICANCE LEVEL OF 0.05, MALES, US MALES POPULATION BASE

Country	Model	ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US	VAR(2), QT, 1 CR	-	Circ,Ext	-	Resp,Ext	Resp
JP	VAR(2), TC, 2 CR	-	Canc	Resp	Circ,Resp	-
FR	VAR(2), NT, 1 CR	-	-	Resp	Circ,Resp	Circ,Resp
E&W	VAR(2), QT, 1 CR	Ext	-	-	IP,Circ Resp	Ext
AU	VAR(2), QT, 1 CR	IP	Canc, Ext	Circ	-	Ext

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A8: $\mathbf{\Gamma}_1$ COEFFICIENTS THAT ARE SIGNIFICANTLY DIFFERENT FROM ZERO, SIGNIFICANCE LEVEL OF 0.05, FEMALES, US MALES POPULATION BASE

Country	Model	ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US	VAR(2), QT, 1 CR	Canc,Ext	Ext	Canc	Resp	Canc,Resp
JP	VAR(2), TC, 2 CR	IP,Resp	-	Resp	-	Circ,Resp Ext
FR	VAR(2), QT, 1 CR	Canc,Circ	-	-	Canc,Circ Resp	IP
E&W	VAR(2), QT, 1 CR	-	-	Resp	Resp	Resp
AU	VAR(2), NT, 1 CR	IP,Resp	Canc	Circ	Circ,Resp	-

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A9: $\mathbf{\Gamma}_1$ COEFFICIENTS THAT ARE SIGNIFICANTLY DIFFERENT FROM ZERO, SIGNIFICANCE LEVEL OF 0.05, MALES, JP FEMALES POPULATION BASE

Country	Model	ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US	VAR(2), QT, 1 CR	Ext	-	-	Resp,Ext	Resp
JP	VAR(2), QT, 1 CR	Ext	-	-	-	Resp
FR	VAR(2), NT, 1 CR	-	-	Canc,Resp	Circ,Resp	Resp
E&W	VAR(2), QT, 1 CR	-	-	Circ	Resp	Ext
AU	VAR(2), NT, 1 CR	Circ	Canc	Circ	Resp	Ext

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A10: $\mathbf{\Gamma}_1$ COEFFICIENTS THAT ARE SIGNIFICANTLY DIFFERENT FROM ZERO, SIGNIFICANCE LEVEL OF 0.05, FEMALES, JP FEMALES POPULATION BASE

Country	Model	ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US	VAR(2), QT, 1 CR	Canc,Ext	Ext	-	Resp,Ext	Canc,Resp
JP	VAR(2), QT, 1 CR	-	-	-	-	Circ,Ext
FR	VAR(2), NT, 1 CR	-	-	Resp	Canc,Circ Resp	IP
E&W	No suitable VAR(2) model					
AU	VAR(2), QT, 1 CR	-	Canc	Circ	-	Circ

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A11: EQUATIONS TO WHICH THE LONG-TERM COMPONENT ENTERS WITH A STATISTICALLY SIGNIFICANT COEFFICIENT α_i, SIGNIFICANCE LEVEL OF 0.05, US MALES POPULATION BASE

Country	Model		Males	Females
US	VAR(2), QT, 1 CR	α_i	ΔIP_t, $\Delta Resp_t$	ΔIP_t, $\Delta Canc_t$ $\Delta Circ_t$, $\Delta Resp_t$
JP	VAR(2), TC, 2 CR	α_{1i}	ΔIP_t, $\Delta Canc_t$	$\Delta Circ_t$, $\Delta Resp_t$
		α_{2i}	ΔIP_t, $\Delta Canc_t$, $\Delta Resp_t$	ΔIP_t, $\Delta Canc_t$ $\Delta Circ_t$, $\Delta Resp_t$
FR	VAR(2), NT, 1 CR	α_i	ΔIP_t, $\Delta Canc_t$, $\Delta Resp_t$	-
	VAR(2), QT, 1 CR	α_i	-	ΔIP_t, $\Delta Canc_t$, $\Delta Resp_t$
E&W	VAR(2), QT, 1 CR	α_i	$\Delta Canc_t$, $\Delta Resp_t$ ΔExt_t	ΔIP_t, $\Delta Circ_t$ ΔExt_t
AU	VAR(2), NT, 1 CR	α_i	-	ΔIP_t, $\Delta Circ_t$, ΔExt_t
	VAR(2), QT, 1 CR	α_i	ΔIP_t, $\Delta Circ_t$, $\Delta Resp_t$	-

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A12: EQUATIONS TO WHICH THE LONG-TERM COMPONENT ENTERS WITH A STATISTICALLY SIGNIFICANT COEFFICIENT α_i, SIGNIFICANCE LEVEL OF 0.05, JP FEMALES POPULATION BASE

Country	Model		Males	Females
US	VAR(2), QT, 1 CR	α_i	ΔIP_t, $\Delta Resp_t$	ΔIP_t, $\Delta Canc_t$ $\Delta Circ_t$, $\Delta Resp_t$
JP	VAR(2), QT, 1 CR	α_i	$\Delta Circ_t$, $\Delta Resp_t$	ΔIP_t, $\Delta Circ_t$, $\Delta Resp_t$
FR	VAR(2), NT, 1 CR	α_i	ΔIP_t, $\Delta Canc_t$, $\Delta Circ_t$ $\Delta Resp_t$, ΔExt_t	ΔIP_t, $\Delta Resp_t$
E&W	VAR(2), QT, 1 CR	α_i	ΔExt_t	No suitable VAR(2) model
AU	VAR(2), NT, 1 CR	α_i	ΔIP_t, $\Delta Circ_t$, $\Delta Canc_t$	-
	VAR(2), QT, 1 CR	α_i	-	$\Delta Circ_t$, $\Delta Resp_t$

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A13: CORRELATION COEFFICIENTS BETWEEN THE ACTUAL CHANGES IN MORTALITY RATES AND THE LONG- AND SHORT-TERM COMPONENTS, MALES, US MALES POPULATION BASE

Country	Model		ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US	VAR(2), QT, 1 CR	LT	-0.271			0.268	
		ST	-0.164			0.336	
JP	VAR(2), TC, 2 CR	LT	0.570	0.797		0.530	
		ST	-0.177	-0.434		0.325	
FR	VAR(2), NT, 1 CR	LT	0.478	0.597		0.368	
		ST	0.023	-0.311		0.613	
E&W	VAR(2), QT, 1 CR	LT		-0.589		0.069	0.018
		ST		0.023		0.575	0.155
AU	VAR(2), QT, 1 CR	LT	-0.389		0.450	0.239	
		ST	-0.058		0.203	-0.059	

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A14: CORRELATION COEFFICIENTS BETWEEN THE ACTUAL CHANGES IN MORTALITY RATES AND THE LONG- AND SHORT-TERM COMPONENTS, FEMALES, US MALES POPULATION BASE

Country	Model		ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US	VAR(2), QT, 1 CR	LT	0.076	0.477	0.290	0.192	
		ST	-0.172	0.012	0.343	0.451	
JP	VAR(2), TC, 2 CR	LT	0.667	0.511	0.763	0.744	
		ST	-0.181	-0.146	-0.315	-0.386	
FR	VAR(2), QT, 1 CR	LT	0.415	0.272		0.582	
		ST	0.083	0.226		0.259	
E&W	VAR(2), QT, 1 CR	LT	0.131		0.410		0.069
		ST	0.286		0.425		0.240
AU	VAR(2), NT, 1 CR	LT	0.529		0.291		0.192
		ST	0.183		0.457		0.311

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A15: CORRELATION COEFFICIENTS BETWEEN THE ACTUAL CHANGES IN MORTALITY RATES AND THE LONG- AND SHORT-TERM COMPONENTS, MALES, JP FEMALES POPULATION BASE

Country	Model		ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US	VAR(2), QT, 1 CR	LT	-0.266			0.275	
		ST	-0.200			0.372	
JP	VAR(2), QT, 1 CR	LT			0.538	0.729	
		ST			0.522	0.069	
FR	VAR(2), NT, 1 CR	LT	0.355	0.553	0.033	0.366	0.271
		ST	0.259	-0.276	0.405	0.621	0.189
E&W	VAR(2), QT, 1 CR	LT					0.130
		ST					0.272
AU	VAR(2), NT, 1 CR	LT	0.600	0.191	0.081		
		ST	-0.017	0.432	0.487		

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

Table A16: CORRELATION COEFFICIENTS BETWEEN THE ACTUAL CHANGES IN MORTALITY RATES AND THE LONG- AND SHORT-TERM COMPONENTS, FEMALES, JP FEMALES POPULATION BASE

Country	Model		ΔIP_t	$\Delta Canc_t$	$\Delta Circ_t$	$\Delta Resp_t$	ΔExt_t
US	VAR(2), QT, 1 CR	LT	0.617	0.589	0.088	0.301	
		ST	-0.160	-0.006	0.365	0.438	
JP	VAR(2), QT, 1 CR	LT	0.533		-0.054	0.378	
		ST	0.142		-0.236	-0.411	
FR	VAR(2), NT, 1 CR	LT	0.406			0.623	
		ST	0.084			0.339	
E&W	No suitable VAR(2) model						
AU	VAR(2), QT, 1 CR	LT			0.514	0.576	
		ST			0.450	0.383	

Note: QT = quadratic trend in the VAR; TC = linear trend in the cointegration relation; NT = no trend; CR = cointegration relation.

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