INTERNATIONAL CAUSE-SPECIFIC MORTALITY RATES: NEW INSIGHTS FROM A COINTEGRATION ANALYSIS

BY

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ABSTRACT

This paper applies cointegration techniques, developed in econometrics to model long-run relationships, to cause-of-death data. We analyze the five main causes of death across five major countries, including USA, Japan, France, England & Wales and Australia. Our analysis provides a better understanding of the long-run equilibrium relationships between the five main causes of death, providing new insights into similarities and differences in trends. The results identify for the first time similarities between countries and genders that are consistent with past studies on the aging processes by biologists and demographers. The insights from biological theory on aging are found to be reflected in the cointegrating relations in all of the countries included in the study.

KEYWORDS

Causes of death, mortality trends, cointegration, dependence, common trends, biological aging.

1. INTRODUCTION

Non-stationary time series have been widely studied by economists for macroeconomic data over many years. An interesting feature of non-stationary variables is that we can distinguish between long-run relations, that are stationary, and short-run adjustments. These long-run relations are known as cointegrating relations and represent long-run equilibria or steady-states. Cointegration analysis has proved to be a powerful methodology for identifying economic relationships between variables such as interest rates and inflation rates, as it allows the testing of relevant economic theories (Johansen and Juselius, 1992, 1994). These techniques have the potential to provide insights into changes in underlying mortality trends by cause of death given that mortality rates have been shown to be non-stationary time series.

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This paper considers long-run equilibrium relations between mortality rates for different causes of death in order to gain insights into the dependence that exists between these competing risks. It extends and complements a recent work by Arnold and Sherris (2015), where cointegrating relations between causes of death were shown to exist, although consistency for countries and genders was not found.

As mentioned by Arnold and Sherris (2015), the nature of the dependence between causes of death is not well understood. The impact of a cause-specific mortality decrease on the remaining cause-of-death mortality rates is not obvious, since these relationships are complex and, strictly speaking, unobservable. Therefore, the assumption usually employed is that the causes of death are independent. Cause-elimination models as well as cause-delay models developed by Manton *et al.* (1980a) and Olshansky (1987) are two well-known examples, still used today; see e.g. Wong-Fupuy and Haberman (2004) and the United States decennial life tables (Bayo, 1968; Greville *et al.*, 1975; Curtin and Armstrong, 1988; Anderson, 1999), amongst others. Cause-specific mortality forecasts are also frequently based on the independence assumption: each cause is independently forecasted and subsequently aggregated to derive total mortality, see e.g. McNown and Rogers (1992), Caselli (1996), Wilmoth (1996), Tabeau *et al.* (1999), and Caselli *et al.* (2006).

In parallel, many studies have been conducted to better understand the relations binding the causes of death to each other and their dependence structure. One may mention models incorporating individual observed risk factors (covariates) or individual unobserved risk factors (frailties) in which cause-specific mortality rates are correlated through their joint dependence on the same risk factors (see e.g. Rosén (2006), Manton (1986)) or the joint distribution of the frailties respectively (see e.g. Vaupel and Yashin (1983), Manton *et al.* (1986) and Hougaard (1984)). More recently, copulas were used to model cause-specific dependence, see e.g. Kaishev *et al.* (2007). When multiple cause-of-death data are available, links between various causes can be investigated, see e.g. Manton *et al.* (1976); Manton and Poss (1979); Manton *et al.* (1980b); Manton and Myers (1987).

In this paper, we complement the above methods by using cointegration techniques in order to extract new insights from cause-of-death data. We extend the methodology of Arnold and Sherris (2015) by using a modified age-standardized death rate and by applying a comprehensive methodology to test the statistical significance of steady-state relationships. Unlike in economic applications, we do not have strong prior hypotheses on the potential long-run relations that may exist between causes of death and that need to be tested. As a result the analysis is exploratory in nature. The aim is to identify meaningful stationary relations between causes of death, based on historical data. This approach is *data-based*. We empirically observe historical trends and use cointegration techniques to determine similarities and differences in long-run trends. We study five developed countries, USA, Japan, France, England & Wales and Australia, to provide robustness to our results.

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The study shows similarities between countries and genders that are consistent with past studies by biologists and demographers. Interestingly, we find that the biological theory on aging is reflected in the cointegrating relations in all the countries included in the study. The application of cointegration techniques to cause-of-death mortality data, provides a first bridge between econometrics and biology, two areas of studies that are essential for life actuaries. The new results lead to specific considerations for the dependence structure between causes of death, that should inform competing risk models for mortality and help practitioners in setting dependence assumptions for cause-specific mortality scenarios. These new results should also be of interest to biologists in further understanding the factors impacting the aging processes of the human body.

The paper begins with a brief description of cointegration in Section 2. Section 3 summarizes the data source and cause-of-death mortality used to estimate the long-run relations. Results from the model fitting are then presented in Section 4, with a discussion on the link existing between the cointegrating relations and theories of aging developed by biologists. Section 5 highlights implications for modeling mortality trends and concludes.

2. Theoretical framework on cointegration

2.1. General concepts

To assess relationships binding economic variables, multiple time series are modeled using Vector AutoRegression (VAR) and Vector Error Correction Models (VECM) developed in the field of econometrics. When variables are stationary, a VAR framework denoted VAR(p) is used, the current level of each variable being explained with p lags of itself and p lags of the other variables in the model. When variables are non-stationary, the non-stationarity can be removed by differencing the variables if the process is integrated of order one. The first difference of each variable is then used in a VAR and explained with $p-1$ lags of its first difference and $p-1$ lags of the first difference of the other variables in the model.

By differencing the variables, potential information present in the levels of the data (original dataset) are lost. Indeed, non-stationary variables may be linked by some relations and thus move together, influenced by common stochastic trends. When a linear combination of non-stationary variables exists such that the resulting relation is stationary, the variables are referred to as cointegrated and the relation as a cointegrating relation. A cointegrating relation represents a long-run equilibrium relationship that is lost when variables are differenced.[1]

The cointegrating relations can then be incorporated in VAR modeling using an alternative VAR(p) representation or a VECM

$$\nabla \mathbf{y}_t = \mathbf{c} + \mathbf{d}t + \xi_1 \nabla \mathbf{y}_{t-1} + \xi_2 \nabla \mathbf{y}_{t-2} + \cdots + \xi_{p-1} \nabla \mathbf{y}_{t-p+1} + \Pi \mathbf{y}_{t-1} + \epsilon_t, \quad (1)$$

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where the n variables at time t are denoted by the $(n \times 1)$ vector $\mathbf{y}_t$, $\mathbf{c}$ and $\mathbf{d}$ are $(n \times 1)$ vectors of constants and ξ_i is a $(n \times n)$ matrix of autoregressive coefficients for $i = 1, 2, \ldots, p-1$ and

$$\begin{aligned}
\nabla \mathbf{y}_t &= \mathbf{y}_t - \mathbf{y}_{t-1};\\
\Pi &= \alpha\beta';\\
&= \text{matrix of rank } r;\\
\alpha &= \text{a } (n \times r) \text{ loading matrix ;}\\
\beta &= \text{a } (n \times r) \text{ matrix containing the } r \text{ cointegrating vectors.}
\end{aligned}$$

The $(n \times 1)$ vector ϵ_t is a vector of white noise terms, with

$$E(\epsilon_t) = \mathbf{0}, \tag{2}$$

$$E(\epsilon_t \epsilon_l) = \begin{cases} \Omega & \text{for } t = l \\ \mathbf{0} & \text{for } t \neq l, \end{cases} \tag{3}$$

where Ω is a symmetric positive definite matrix.

Equation (1) shows that for non-stationary variables, the first difference of each variable is explained with lagged values of the first difference of the variables and the term $\beta' \mathbf{y}_{t-1}$ which contains the cointegrating relations. Each column of the matrix β represents a cointegrating relation. More than one cointegrating relation may exist, each being linearly independent from the others. If r linearly independent cointegrating relations are found and if all other cointegrating relations are a linear combination of these r relations, then there are exactly r cointegrating relations among the elements of $\mathbf{y}_t$ and the matrix β forms a basis of the space of cointegration. Thus, the β matrix represents the long-run steady-states or equilibria and the other parameters (α, $\mathbf{c}$, $\mathbf{d}$ and ξ_i for $i = 1, 2, \ldots, (p-1)$) reflect the short-run dynamic adjustments. Finally, the loading matrix α measures the impacts cointegrating relations have on the variables under study. Hamilton (1994) and Lütkepohl (2005) are comprehensive references on these models.

In order to find the number of cointegrating relations that may exist between a set of variables, two preliminary tests have to be made. First, the number of past values (lag order p in Equation (1)) to be included in the VECM has to be selected. Several criteria exist for that, such as Akaike's Information Criterion (AIC), Hannan-Quinn Criterion (HQ), Schwarz Criterion (SC), Final Prediction Error (FPE). Second, the non-stationarity of the variables has to be checked through unit root tests such as the Kwiatkowski-Phillips-Schmidt-Shin test (KPSS), the Augmented Dickey-Fuller test (ADF), the Phillips-Perron test (PP) or the Elliot-Rothenberg-Stock test (ERS) (Hamilton (1994) and Lütkepohl (2005)).

The standard approach used to identify the potential cointegrating relations among non-stationary variables is the Johansen procedure, especially when the

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number of cointegrating relations has to be found and there is no knowledge on the form of the cointegration. Johansen developed two tests to determine the cointegration order, that are the trace test and the maximum–eigenvalue test. Depending on the model specifications, namely the inclusion/exclusion of a vector of constants and/or a vector of trends in Equation (1) (vectors **c** and **d**), the cointegrating relations may be stationary around a constant level or a trend. Therefore, the Johansen approach also allows us to test the significance of the vector of constants/vector of trends. We will focus on three different model specifications (for details, see Johansen (1994)):

- Case 1: The process has a linear trend, which is eliminated by the cointegrating relations. Thus, the cointegrating relations do not contain a trend, but only a constant. The long-run equilibria are then stationary and the process contains no trend stationary component. This model refers to Equation (1) with an unrestricted vector of constants **c** and no trend, $\mathbf{d} = 0$.
- Case 2: The process does not have any quadratic trend, but a linear trend is allowed in all the components of the process, a trend which cannot be eliminated by the cointegrating relations. A linear trend is thus allowed in the cointegrating relations and the long-run equilibria are allowed to be trend stationary. This model refers to Equation (1) with an unrestricted vector of constants **c** and a vector of trends **d** restricted such that $\mathbf{d} = \alpha \cdot \beta_1$, with β_1 a $(r \times 1)$ vector. Equation (1) becomes

$$\nabla \mathbf{y}_t = \mathbf{c} + \xi_1 \nabla \mathbf{y}_{t-1} + \cdots + \xi_{p-1} \nabla \mathbf{y}_{t-p+1} + \alpha(\beta' \mathbf{y}_{t-1} + \beta_1 \cdot t) + \epsilon_t.$$

- Case 3: The process has a quadratic trend, which is eliminated by the cointegrating relations. Thus, the cointegrating relations do not contain a quadratic trend, but allow for a linear trend. This model refers to Equation (1) with an unrestricted vector of constants **c** and an unrestricted vector of trends **d**. Indeed, since the linear trend **d** appears in the first difference of the variables (Equation (1)), a quadratic trend is present in the level of the process.

Johansen developed two tests to compare the three different model specifications. The first statistic (we will refer to it as H1) compares the model with a quadratic trend (Case 3) against the model without a quadratic trend (Case 2). He showed that this statistic has an asymptotic χ^2 distribution with $(n - r)$ degrees of freedom. The second statistic (we will refer to it as H2) tests the significance of the linear trend in the cointegrating relations. This is a comparison of Case 2 with Case 1. He showed that this statistic has an asymptotic χ^2 distribution with r degrees of freedom. Naturally, if no cointegrating relation is found, that is r equals zero in Equation (1), the VECM reduces to a VAR$(p-1)$, that is a VAR applied to the first difference.

Finally, model validation tests should be performed. The Portmanteau test is applied to check for any remaining autocorrelation among the residuals up to lag l, while the normality of the residuals is tested with statistics based on the third and forth central moments (skewness and kurtosis) of a normal

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distribution. Details can be found in Gaille and Sherris (2011), Arnold and Sherris (2015), Hamilton (1994) and Lütkepohl (2005).

2.2. Testing the cointegrating relations

Johansen approach allows us to test if some of the coefficients of the cointegrating relations are not significantly different from zero, which means that some coefficients of the β matrix can be equaled to zero. That allows us to assess if only q of the n variables are required in the r cointegrating relations. This test can be done through a likelihood ratio test based on standard asymptotic distribution theory. Johansen (1988, 1991) showed that the likelihood ratio statistic found with his procedure has an asymptotic χ^2 distribution with $r \cdot (n - q)$ degrees of freedom.

3. DATA

We use the following dataset: age-sex-cause-specific death numbers and age-sex-specific mid-year populations for each calendar year in several countries. The first divided by the second produces central death rates, more specifically we have

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

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, for 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;

Data were obtained from the Mortality Database administered by the World Health Organization (World Health Organization, 2012). This database contains the underlying cause of death and is generally divided into five-year age-groups for the last 50 or 60 years.

Five countries were chosen for the analysis:[2] the USA (1950–2007); Japan (1950–2009); France (1952–2008); England and Wales (1950–2009), thereafter E&W; Australia (1950–2004). The first four countries are the developed countries with the highest population and represent three different parts of the world, namely America, Asia and Europe. Australia was added to the analysis as a country representing Oceania.

Causes of death are defined by the International Classification of Diseases (ICD), which ensures consistencies between countries (Table 1). Under the ICD, the underlying cause of death is specified as *the disease or injury which initiated*

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TABLE 1

INTERNATIONAL CLASSIFICATION OF DISEASES — CODING SYSTEM.

Causes of death	ICD 7	ICD 8	ICD 9	ICD 10
Circulatory system	A079-A086	A080-A088	B25-B30	I00-199
Cancer	A044-A060	A045-A061	B08-B17	C00-D48
Respiratory system	A087-A097	A089-A096	B31-B32	J00-J99
External causes	A138-A150	A138-A150	B47-B56	V00-Y89
Infectious and parasitic diseases	A001-A043	A001-A044	B01-B07	A00-B99

the train of morbid events leading directly to death, or the circumstances of the accident or violence which produced the fatal injury. We consider the five main ICD causes, which are: diseases of the circulatory system, cancer, diseases of the respiratory system, external causes, and infectious and parasitic diseases (I&P). The major causes account for more than 80% of deaths in recent years, and made up approximately 60%–70% of deaths 50 years ago, as mentioned by Arnold and Sherris (2013, 2015).

The same database as in Arnold and Sherris (2013, 2015) is used.[3] In these two papers, the time evolution of mortality rates for different causes of death is analyzed with an age-standardized country-specific central death rate. The standard population used to compute this age-standardized death rate is equal to the population of the last year under observation. They denote by $m^*_{t,d,s,c}$ the age-standardized central death rate in year t for cause d, gender s and country c, assuming that the age structure of the population is constant over the complete period under observation and fixed at the level of the last observed year,

$$m^*_{t,d,s,c} = d^*_{t,d,s,c} / l_{LY_c,s,c},$$
$$d^*_{t,d,s,c} = \sum_x (m_{x,t,d,s,c} \times l_{x,LY_c,s,c}),$$

with

$$l_{LY_c,s,c} = \sum_x (l_{x,LY_c,s,c});$$
$$= \text{mi-year population in year } LY_c, \text{ for gender } s \text{ and country } c;$$
$$LY_c = \text{last year under observation for country } c.$$

In applying this methodology, the age structure of the populations differs between countries. Differences observed between countries may then reflect population age structure differences. In order to compare cause-specific mortality evolution between countries, we will standardize the population across countries.

For that purpose, two different populations are used as reference to construct the age-standardized death rates: (1) the US male population in 2007; (2) the

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Japanese female population in 2009. In this way, the age structure of the population is kept constant across countries. By using two different standard populations, one relatively young (USA) and the other one relatively old (Japan), we analyze if cause-specific death rates for young populations behave differently to cause-specific death rates in older populations. We denote by $m^{US}_{t,d,s,c}$ the age-standardized central death rate in year t for cause d, gender s and country c, assuming that the age structure of the population is constant over the complete period under observation and equal to the age structure of the 2007 US male population,

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

and by $m^{\text{Jap}}_{t,d,s,c}$ the age-standardized central death rate in year t for cause d, gender s and country c, assuming that the age structure of the population is constant over the complete period under observation and equal to the age structure of the 2009 Japanese female population,

$$m^{\text{Jap}}_{t,d,s,c} = d^{\text{Jap}}_{t,d,s,c} / l_{2009,\text{females,Japan}},$$
$$d^{\text{Jap}}_{t,d,s,c} = \sum_x (m_{x,t,d,s,c} \times l_{x,2009,\text{females,Japan}}).$$

4. Long-run equilibrium for causes of death

Arnold and Sherris (2015) showed that there exist long-run equilibrium relationships between cause-specific mortality rates. By applying additional analysis, we extend these results and identify similarities between the five selected countries. To do this, the model described in Section 2 is applied independently to each country for males and females, with the age structure of US males and the age structure of Japanese females, so twenty times altogether. To be concise, the vector $\mathbf{y}_t$ in Equation (1) contains here the logarithm of the age-standardized central death rates of the five studied causes of death, and thus five variables. Since we have a different vector $\mathbf{y}_t$ for each gender s and country c, we can write

$$\mathbf{y}^{US}_{t,s,c} = \begin{pmatrix} \log\left(m^{\text{US}}_{t,\text{circulatory},s,c}\right) \\ \log\left(m^{\text{US}}_{t,\text{cancer},s,c}\right) \\ \log\left(m^{\text{US}}_{t,\text{respiratory},s,c}\right) \\ \log\left(m^{\text{US}}_{t,\text{external},s,c}\right) \\ \log\left(m^{\text{US}}_{t,I\&P,s,c}\right) \end{pmatrix}, \tag{4}$$

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and

$$\mathbf{y}_{t,s,c}^{Jap} = \begin{pmatrix} \log\left(m_{t,\text{circulatory},s,c}^{\text{Jap}}\right) \\ \log\left(m_{t,\text{cancer},s,c}^{\text{Jap}}\right) \\ \log\left(m_{t,\text{respiratory},s,c}^{\text{Jap}}\right) \\ \log\left(m_{t,\text{external},s,c}^{\text{Jap}}\right) \\ \log\left(m_{t,I\&P,s,c}^{\text{Jap}}\right) \end{pmatrix}. \tag{5}$$

We are then looking for long-run steady-states between the logarithm of the following variables: First, $m_{t,\text{circulatory},s,c}^{\text{US}}$, $m_{t,\text{cancer},s,c}^{\text{US}}$, $m_{t,\text{respiratory},s,c}^{\text{US}}$, $m_{t,\text{external},s,c}^{\text{US}}$ and $m_{t,I\&P,s,c}^{\text{US}}$ for each country and gender (Equation (4)); Second, $m_{t,\text{circulatory},s,c}^{\text{Jap}}$, $m_{t,\text{cancer},s,c}^{\text{Jap}}$, $m_{t,\text{respiratory},s,c}^{\text{Jap}}$, $m_{t,\text{external},s,c}^{\text{Jap}}$ and $m_{t,I\&P,s,c}^{\text{Jap}}$ for each country and gender (Equation (5)).

The following sections initially present a detailed analysis for US and Japanese males, using the US male age structure of the population. The procedure and tests used are illustrated in detail using these two countries. In the later part of the section, only the main results and most important test statistics for the twenty settings are presented along with a discussion. Details of the test statistics for each country and additional figures are available from the authors upon request.

4.1. Detailed case study: US and Japanese males, with US male population age structure

4.1.1. *Preliminary tests: Lag order and unit root.* As previously mentioned, in order to look for potential cointegrations between a set of variables, the lag order of the VAR or VECM is first required. Out of the four criteria performed, a lag order of one is indicated as optimal for US males. In Japan, HQ, SC, FPE reveal a lag of one, while the AIC statistic indicates a lag of five as optimal. Since we only have 60 years of observation and since the residuals of the resulting one-lag model are normally distributed and non-autocorrelated, a lag of one is used for both countries.

Second, the non-stationarity of the variables needs to be checked. KPSS, ADF, PP and ERS tests are performed on the data. A cause of death is said non-stationary when at least three out of the four tests accept it at a five percent significance level. Following this procedure, all the causes of death except I&P are shown to be non-stationary in both countries, while contradictory results are found for I&P. Since the stationarity of the variables may also be checked through the Johansen procedure for cointegration, it is shown in the next section that I&P are also non-stationary. Therefore, the five main causes of death are considered as non-stationary in both countries, and thus to have stochastic trends.

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TABLE 2

TESTS FOR THE NUMBER OF COINTEGRATING RELATIONS, MALES WITH US MALE POPULATION AGE STRUCTURE.

	Trace statistics		Critical values			
r	USA	Japan	10%	5%	2.5%	1%
4	0.35	3.21	2.70	3.84	5.25	6.98
3	12.23	15.22	15.74	18.08	20.26	22.40
2	24.74	31.15	31.67	34.27	36.98	40.10
1	40.77	54.43	50.62	54.02	57.01	61.03
0	81.49	109.78	73.73	77.61	81.29	85.56
	Maximum-eigenvalue statistics		Critical values			
r	USA	Japan	10%	5%	2.5%	1%
4	0.35	3.21	2.70	3.84	5.25	6.98
3	11.88	12.01	14.64	16.69	18.84	20.88
2	12.51	15.93	21.44	23.75	25.68	28.31
1	16.03	23.28	27.39	29.93	32.22	35.57
0	40.71	55.35	33.45	36.46	39.00	41.87

A null hypothesis is accepted at a α% significance level when the statistic is lower than the corresponding critical value. Thus, these tables indicate that one cointegrating relation is accepted at 10%, 5% and 2.5% significance levels in the USA and at 2.5% and 1% significance levels in Japan.

4.1.2. *Cointegration.* According to the trace and maximum-eigenvalue tests of the Johansen procedure, one cointegrating relation exists in both countries (see Table 2). The null hypothesis of r cointegrating relations against the alternative of n cointegrating relations is tested using the trace statistic, while the null hypothesis of r cointegrating relations against the hypothesis of $r+1$ cointegrating relations is tested with the maximum-eigenvalue statistic.[4] In the USA, the trace statistics for $r = 1, 2, 3, 4$ are smaller than the critical values, and thus we accept the null hypotheses that there are one, two, three or four cointegrating relations against the alternative of five cointegrating relations. The US maximum-eigenvalue statistic for $r = 1$ is 16.03, which is smaller than the indicated critical values. Therefore, we accept the null assumption that there is one cointegrating relation against the alternative of two. However, we do not accept that there is zero cointegrating relation against the alternative of one at 10%, 5% and 2.5% significance levels. Thus, one long-run equilibrium relationship exists among the causes of death, showing that these rates have changed with common stochastic trends. This long-run equilibrium relationship determines how changes in causes of death move relative to each other and represents historical evolutions. Mortality was evolving stochastically, but death rates were also driven by this long-run equilibrium relationship between the causes, which was maintained stationary over the past 50 years. Similar conclusions are drawn for Japan.

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TABLE 3

TESTS ON RESIDUALS OF THE FITTED VECM, MALES WITH US MALE POPULATION AGE STRUCTURE.

		p value		
			Japan	
Type of test	Name of test	USA Quadratic trend	No trend	Quadratic trend
Autocorrelation	Portmanteau (15 lags)	0.189	0.055	0.144
	Portmanteau (25 lags)	0.336	0.209	0.300
	Portmanteau (35 lags)	0.376	0.179	0.159
Normality	Skewness	0.640	0.050	0.085
	Kurtosis	0.011	0.038	0.106
	Both	0.051	0.011	0.043

The null hypothesis of no-autocorrelation among the residuals is tested through the Portmanteau statistic, with a lag of 15, 25 and 35. The skewness statistic, the kurtosis statistic and a combination of these are used to test the normality of the residuals.

In the USA, tests performed using the Johansen procedure indicate that a quadratic trend should be included in the process, and thus a trend is included in the cointegrating relation. The null hypothesis of no linear trend is rejected at a five percent significance level, the p value of the second test statistic H2 being 0.019, while the null hypothesis of no quadratic trend is also rejected, the p value of the first statistic H1 being 2.80e-06.

In Japan, the results of the two tests are less definitive. The p value of the second test statistic H2 is 0.060, indicating that the null hypothesis of no linear trend in the cointegrating relation is accepted at a five percent significance level. However, the p value of the first test statistic H1 is 1.60e-08, which indicates that a quadratic trend should be included in the process with a linear trend in the cointegrating relation. We applied the two model specifications, that are (1) Case 1 — no linear trend in the cointegrating relation and (2) Case 3 — quadratic trend in the process and analyzed the residuals. The residuals of both models are normally distributed and non-autocorrelated (the null hypotheses of normality and non-autocorrelation are accepted at 1% significance level). Nevertheless, results for the model specification of Case 1 are less straightforward than the results for the model specification of Case 3 (Table 3). Therefore, for both countries, a quadratic trend should be included. The resulting fitted models are introduced in Appendix A.

Arnold and Sherris (2015) found limited similarities in the cointegrating relations between countries and genders based on the rates they used where the age structure of the population differed between countries (and, to a lesser extent, between genders as well). They also did not test the statistical significance of the coefficients in the cointegrating relations. Johansen approach allows us to test the significance of the β matrix coefficients using a likelihood ratio statistic with an asymptotic χ^2 distribution. We have no strong prior hypothesis on the

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TABLE 4

TESTS FOR THE SIGNIFICANCE OF THE COEFFICIENTS IN THE COINTEGRATING RELATION, US MALES.

	Circulatory	Cancer	Respiratory	External	I&P
Statistic	2.10194	1.38777	8.53566	3.84887	0.99454
p values	0.14711	0.23878	0.00348	0.04978	0.31864

A null hypothesis is accepted at a α% significance level when the p value is higher than α%. Thus, this table indicates that the null hypothesis that diseases of the circulatory system, cancer, external causes of death or I&P do not appear individually in the cointegrating relation is accepted at a 1% significance level.

TABLE 5

TESTS FOR THE SIGNIFICANCE OF THE COEFFICIENTS IN THE COINTEGRATING RELATION, JAPANESE MALES WITH US MALE POPULATION AGE STRUCTURE.

	Circulatory	Cancer	Respiratory	External	I&P
Statistic	18.92912	15.18568	27.72206	0.00383	5.67024
p values	1.35666e-05	9.74394e-05	1.40057e-07	0.95062	0.01726

A null hypothesis is accepted at a α% significance level when the p value is higher than α%. Thus, this table indicates that the null hypothesis that the external causes of death or I&P do not appear individually in the cointegrating relation is accepted at a 1% significance level.

causes of death that will be significant in the cointegrating relations, so we systematically test the significance of each coefficient of the β matrix. Tables 4 and 5 present the test statistics for the USA and Japan respectively.

According to the tests in Table 4, testing individual causes separately, only diseases of the respiratory system appear significantly different from zero at a 1% significance level in the USA. In Japan, circulatory diseases, cancer and diseases of the respiratory system are significant with the external causes of death and the I&P not statistically significant at a 1% significance level.

The next step is to test if several causes of death, together, may not be part of the cointegrating relations. We tested all the possible combinations of the causes of death that do not appear significant in Tables 4 and 5. Results are presented in Table 6 for US males and in Table 7 for Japanese males. In both countries, the null hypothesis that I&P and the external causes of death do not appear in the cointegrating relations is accepted at a 1% significance level, although in the USA the null hypotheses of several other combinations are also accepted (I&P and cancer; cancer and circulatory; circulatory and external). Finally, we tested the null hypothesis that three causes of death, together, do not appear in the cointegrating relation for US males. All of theses cases were rejected.

In order to test if a variable is trend stationary, we can test the null hypothesis that all the variables simultaneously except the one of interest do not appear in the cointegrating relation. If this hypothesis is accepted, the cointegrating relation is then the variable of interest, plus a linear trend and so the variable is trend stationary. We can verify if I&P is trend stationary. By applying the tests,

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TABLE 6

TESTS FOR THE SIMULTANEOUS SIGNIFICANCE OF THE COEFFICIENTS IN THE COINTEGRATING RELATION, US MALES.

	I&P and Cancer	I&P and Circulatory	I&P and External	Cancer and Circulatory	Cancer and External	Circulatory and External
Statistic	2.02264	10.65785	4.58128	8.46790	12.31029	4.15345
p values	0.36374	0.00485	0.10120	0.01450	0.00212	0.12534

A null hypothesis is accepted at a α% significance level when the p value is higher than α%. As an example, this table indicates that the null hypothesis that I&P together with cancer do not appear in the cointegrating relation is accepted at a 5% significance level.

TABLE 7

TESTS FOR THE SIMULTANEOUS SIGNIFICANCE OF THE COEFFICIENTS IN THE COINTEGRATING RELATION, JAPANESE MALES WITH US MALE POPULATION AGE STRUCTURE.

	I&P and external
Statistic	8.50870
p values	0.01420

A null hypothesis is accepted at a α% significance level when the p value is higher than α%. This table indicates that the null hypothesis that I&P together with the external causes do not appear in the cointegrating relation is accepted at a 1% significance level.

we find a p value of 0.2% and 0.00001% in USA and Japan respectively. The two null hypotheses are rejected. I&P is thus confirmed to be non-stationary.

We conclude that the cointegrating relation for males in Japan only includes diseases of the circulatory system, cancer and diseases of the respiratory system. It is more difficult to draw any conclusion for males in the USA, since several combinations of two causes may not appear in the cointegrating relation. In order to gain additional insights and to detect potential recurrent patterns, the same procedure as the one described for males in Japan and in the USA is applied for males in E&W, France and Australia, as well as for females in the five countries. The main results are covered in the following section.

4.2. International comparison

The procedure described in section 4.1 is used to analyze cause-specific mortality for males and females in USA, Japan, France, E&W and Australia. Two different mortality rates are studied: (1) age-standardized central death rate with the 2007 US male population age structure; (2) age-standardized central death rate with the 2009 Japanese female population age structure. The model generally fits well the data with normally distributed and non-autocorrelated residuals in these 20 different settings, that are for two genders in five

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TABLE 8

p VALUES FOR THE NULL HYPOTHESIS THAT I&P AND THE EXTERNAL CAUSES OF DEATH ARE NOT SIGNIFICANTLY DIFFERENT FROM ZERO, US MALE POPULATION AGE STRUCTURE.

Country	Model	Males	Females
USA	VAR(1), QT, one CR	0.10120	0.00538
Japan	VAR(1), QT, one CR	0.01420	0.34110
	VAR(2), TC, two CR	–	0.05292
France	VAR(2), no trend, one CR	0.13400	–
	VAR(1), QT, one CR	0.00053	0.00000
	VAR(1), no trend, one CR	–	0.00204
E&W	VAR(1), QT, one CR	0.00011	0.55270
Australia	VAR(1), QT, one CR	0.09199	–
	VAR(2), QT, one CR	0.25699	–
	VAR(2), no trend, one CR	–	0.04383

QT = Quadratic trend in the VAR (Case 3 of Section 2.1); TC = Linear trend in the cointegrating relation and in the VAR (Case 2); no trend = No trend in the cointegrating relation (Case 1); CR = cointegrating relation. A null hypothesis is accepted at a α% significance level when the p value is higher than α%.

countries using two reference populations (see Appendix B for the results of the tests applied on the residuals). Long-run steady-states are thus studied in detail in these 20 settings. This analysis allows us to more reliably identify similarities in long-run equilibrium relationships between countries and genders. In what follows, we describe the similarities and recurrent patterns from this analysis.

The main finding is that I&P and the external causes of death usually do not appear significantly in the cointegrating relations. The p values for the 20 models are introduced in Tables 8, 9. The second column of the two tables presents the best models describing the dataset, according to the methodology described in Section 4.1. When several models describe equivalently well the dataset and no test reveals the most appropriate one, the results for the different models are included. An interesting example is for females in Japan. By applying the methodology described in Section 4.1, it is not clear whether the best model describing the process for $\log m^{\text{Jap}}_{t,d,\text{females,Japan}}$ is a VAR(1) or a VAR(2) with a quadratic trend and one cointegrating relation or a VAR(2) with a linear trend in the process and in the two cointegrating relations (Table 9). The three models have normally distributed and non-autocorrelated residuals and thus capture the features of the dataset. In the three models, I&P as well as the external causes of death are not significantly different from zero, while the remaining three other causes of death are significantly different from zero. The process for $\log m^{\text{US}}_{t,d,\text{females,Japan}}$ is also well described by a VAR(1) with a quadratic trend and one cointegrating relation or a VAR(2) with a linear trend in the process and in

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TABLE 9

p VALUES FOR THE NULL HYPOTHESIS THAT I&P AND THE EXTERNAL CAUSES OF DEATH ARE NOT SIGNIFICANTLY DIFFERENT FROM ZERO, JAPANESE FEMALE POPULATION AGE STRUCTURE.

Country	Model	Males	Females
USA	VAR(1), QT, one CR	0.05509	0.00008
Japan	VAR(1), QT, one CR	0.00443	0.05775
	VAR(2), QT, one CR	–	0.05683
	VAR(2), TC, two CR	–	0.03781
France	VAR(2), no trend, one CR	0.03180	0.03787
E&W	VAR(1), QT, one CR	0.00014	0.62422
Australia	VAR(1), QT, one CR	0.01881	–
	VAR(2), QT, one CR	0.05698	0.06906
	VAR(2), no trend, one CR	0.19169	–

QT = Quadratic trend in the VAR (Case 3 of Section 2.1); TC = Linear trend in the cointegrating relation and in the VAR (Case 2); no trend = No trend in the cointegrating relation (Case 1); CR = cointegrating relation. A null hypothesis is accepted at a α% significance level when the p value is higher than α%.

the two cointegrating relations (Table 8). As with the $\log m^{\text{Jap}}_{t,d,\text{females,Japan}}$ variables, only I&P with the external causes of death are not significantly appearing in the cointegrating relations in both models.

In the other countries, the I&P and the external causes of death are usually not significantly different from zero, even if in some countries some other combinations of the causes may also be non significantly different from zero. For example, for French females using the age structure of the Japanese female population, the I&P and the diseases of the circulatory system are also not significantly different from zero with a p value of 11%, while for E&W females (with Japanese female age structure), the I&P and cancer (p value of 4.7%), or the diseases of circulatory system and cancer (p value of 2.9%), or the external causes of death and the diseases of the circulatory system (p value of 4.7%) are also not significant. For males in France (with US male or Japanese female population age structure), we cannot reject the null hypothesis that the diseases of the respiratory system are stationary in the VAR(2) framework (p value of 21% and 13% respectively), and thus that the steady-state may represent the stationary variable, namely the diseases of the respiratory system. There is then some difficulties to detect which cointegrating relation reflects a potential underlying pattern existing in the data of some countries. However, by applying the analysis to different countries, only one pattern regularly appears, namely the non-significance of the coefficient for the I&P and the external causes of death in the cointegrating relation.

There are a few exceptions. Males in E&W represent the only situation where both the I&P and the external causes of death do appear significantly in the

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cointegrating relation (with Japanese female population age structure). Indeed, only cancer and the diseases of the respiratory system are not significantly different from zero (p value of 39.7%). For females in the USA (with US male or Japanese female population age structure), the I&P are not significantly different from zero (p value of 84% and 2.1% respectively), while the external causes of death appear in the cointegrating relation. For males in Japan (with Japanese female population age structure), for females in France (with US male population age structure, VAR(1) with or without a quadratic trend) and for males in E&W (with US male population age structure), the external causes of death are not significantly different from zero (p value of 29%, 51%, 41% and 1.5% respectively), while the I&P appear in the cointegrating relation. To summarize, for 14 times out of the 20 cases considered, the I&P combined with the external causes of death do not significantly appear in the steady-states.

4.3. What drives the results?

The significance of this result becomes clearer if we consider theories that have been developed and studied by biologists and demographers, namely the distinction that is made between what we will call *exogenous* causes of death and *endogenous* causes of death. Historically, the idea to separate mortality into two groups comes from Gompertz (1825); a first mortality group related to *chance, without previous disposition to death or deterioration*; a second mortality group referring to *an unspecified force that destroyed the material of organization necessary for life*. Forty years later, Makeham suggested that each disease could be classified in one of the two categories, but he did not think that the medical knowledge at that time was sufficient to define a clear classification (Makeham, 1867). Many researchers attempted to refine Gompertz's description of *an unspecified force that destroyed the material of organization necessary for life* and a nice review is provided in Carnes and Olshansky (1997).

The exogenous causes of death represent external or environmental factors that produce death, while the endogenous causes of death represent biological forces that lead to death, namely aging or senescent (Makeham, 1867; Shryock *et al.*, 1975; Carnes and Olshansky, 1997). The endogenous causes refer then to Gompertz's *unspecified force that destroyed the material of organization necessary for life*. As mentioned by Shryock *et al.* (1975), the classification of the causes of death in the exogenous or the endogenous group is still not well defined today and stays somewhat arbitrary. The causes of death classified as exogenous or endogenous differ then slightly between studies (see e.g. the classifications in Carnes *et al.* (2006) and a review in Carnes and Olshansky (1997)). However, the exogenous mortality usually includes *mortality mainly from infections and accidents* (Shryock *et al.*, 1975), represented by the I&P and the external causes of death in our death classification. The remaining three causes of death (diseases of the circulatory system, cancer and diseases of the respiratory system) can be grouped under the endogenous category.

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An interesting aspect in separating mortality into exogenous and endogenous components relies on the idea that endogenous mortality reflects fundamental and underlying processes of the human body referred to as the aging processes or the biological processes of aging. As noted by Strehler (1959), *there exist gradual changes in the structure of organisms which are not due to preventable diseases or other gross accidents and which eventually lead to the increased probability of death of the individual with advancing age*. In a natural, unprotected environment, aging is rare. Most wild animals die from predators, infections, accidents or starvations. However, in a sheltered environment, where the hazards in the natural environment are minimized, animals live longer and experience the loss of some functions associated with aging (Adams and White, 2004). Biological aging is then usually defined as *the incremental, universal, and intrinsic degeneration of physical and cognitive functioning and the ability of the body to meet the physiologic demands that occur with increasing chronologic age* (Robertson *et al.*, 2013). It is due to *the imperfect operation of maintenance mechanisms and the resultant accumulation of cellular damage* (Adams and White, 2004). The process of aging is not well understood (Jin, 2010) and thus cannot be reliably measured today (Olshansky *et al.*, 2002, 2004; Hayflick, 2004). As mentioned by Carnes *et al.* (2006), *knowledge about underlying mechanisms of senescence and disease has been and remains incomplete*. Even for the simpler question of whether processes of aging exist, no common agreement is reached (Butler *et al.*, 2004).

To summarize, the aging processes are underlying forces that affect endogenous causes, which would explain why we find the dependencies we observe between the causes across our countries. These forces can be seen as *intrinsic and currently immutable forces* (Olshansky *et al.*, 2002). Since a process with cointegrating relations has, by definition, common stochastic trends, it is reasonable that the cointegrating relations in the data will usually only include the diseases of the circulatory system, cancer and diseases of the respiratory system, namely endogenous causes of death. The common stochastic trends of the cause-specific mortality process represent the aging processes. Indeed, the aging process is known to be stochastic (Hayflick, 2004; Carnes *et al.*, 2013) and to be a potential mixture of several stochastic processes (Holliday, 2004; Carnes *et al.*, 2013), which is exactly the definition of the common trends affecting a cointegrating system. The long-run equilibrium relationships we found are then representing somehow the aging processes and the theories developed by biologists and demographers that endogenous causes are manifestations of the aging processes and not its cause (Carnes *et al.*, 2006) are reenforced. The biological aging of the body is the underlying risk factor — even the greatest risk factor according to Hayflick (2004) — influencing the causes of death (Olshansky *et al.*, 2002) and is captured by the common stochastic trends of the cointegrating system.

Since long-run equilibrium relationships including only diseases of the circulatory system, cancer and diseases of the respiratory system are found in most cases analyzed in our study and this is supported by theories of aging used by demographers and biologists, we consider and report these relations for the five

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countries under study, for both males and females. Estimates of the components of the β matrix in Equation (1) are presented in Tables 10 and 11.[5]

It is worthy to note that no matter what population age structure is used the results remain unchanged. By comparing Table 10 with Table 11, we see that the most appropriate models are similar and so are the cointegrating relations. For example, the long-run equilibrium relationship for males in the USA is similar in both tables, since in both tables cancer and diseases of the circulatory system have the same sign, while diseases of the respiratory system are of opposite sign. A decrease in mortality due to the diseases of the respiratory system was associated, in the past, with a decrease in log-death rates of either or a combination of the two remaining causes. The causes of death appearing in the long-run steady-states and the relations existing between them are robust to the population age structure used. The aging processes impact all age groups in a similar way.

Also worthy of noting is that male and female steady-states are similar within each country, as opposed to what was first found by Arnold and Sherris (2015) (with the only exception referring to Australia). For example, in E&W, diseases of the respiratory system and cancer have the same sign, while diseases of the circulatory system are of opposite sign. Thus, E&W males and females show similar relative past changes. An important remark should be made with respect to the six models for which I&P combined with the external causes of death do appear significantly in the cointegrating relations. Similarities between males and females remain when I&P and/or the external causes of death are kept, but the other (non-significant cointegration parameter) causes of death are removed. For example, only the external causes of death do not appear significant in the cointegrating relation for Japanese males (with the age structure of Japanese female population). By removing only the external causes of death from the cointegrating relation, similar relations are still found between males and females in Japan: I&P and cancer coefficients have similar sign, while diseases of the circulatory and respiratory system are of opposite sign. Thus, males and females within a country have similar long-run equilibria.

5. Conclusion

The aim of this paper has been to provide a better understanding of the dependence between causes of death. Potential links between causes of death are analyzed through cointegration. It is indeed possible to estimate long-run equilibrium relationships existing between causes of death by using age-standardized cause-of-death mortality rates and considering models with long-run common stochastic trends. The paper derives long-run relations from the data since today no prior knowledge or theory on these relations has been internationally recognized. Cointegrations are analyzed for 20 different cases including both males and females in five developed countries with two different population age structures used to derive the age-standardized death rates. From this analysis, a number of significant conclusions are drawn.

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TABLE 10

RESTRICTED COINTEGRATING RELATIONS, US MALE POPULATION AGE STRUCTURE.

		Males			Females		
Country	Model	Cancer	Circulatory	Respiratory	Cancer	Circulatory	Respiratory
USA	VAR(1), QT, one CR	11.231	9.257	−8.577	−7.411	−9.528	5.892
Japan	VAR(1), QT, one CR	−9.915	9.423	5.813	−33.268	12.186	5.504
	VAR(2), TC, two CR	–	–	–	−18.686	1.800	0.972
	VAR(2), TC, two CR	–	–	–	−52.867	19.853	8.953
France	VAR(2), no trend, one CR	−2.364	2.166	2.298	–	–	–
	VAR(1), QT, one CR	5.604	5.660	7.943	−45.379	14.930	5.236
	VAR(1), no trend, one CR	–	–	–	25.144	−4.593	−3.101
E&W	VAR(1), QT, one CR	−35.738	19.046	−6.464	−43.785	24.806	−8.016
Australia	VAR(1), QT, one CR	−22.845	18.352	−13.692	–	–	–
	VAR(2), QT, one CR	32.901	−28.535	21.382	–	–	–
	VAR(2), no trend, one CR	–	–	–	−8.232	−2.425	−5.129

QT = Quadratic trend in the VAR (Case 3 of Section 2.1); TC = Linear trend in the cointegrating relation and in the VAR (Case 2); no trend = No trend in the cointegrating relation (Case 1); CR = cointegrating relation.

This table presents estimates of the components of the β matrix introduced in Equation (1). For example, males in the United States have an estimated long-run equilibrium relationship given by

$$11.231 \times \log(m^{\text{US}}_{t,\text{cancer,males,USA}}) + 9.257 \times \log(m^{\text{US}}_{t,\text{circulatory,males,USA}}) - 8.577 \times \log(m^{\text{US}}_{t,\text{respiratory,males,USA}}) = z_t,$$

where z_t is a stationary variable.

TABLE 11

RESTRICTED COINTEGRATING RELATIONS, JAPANESE FEMALE POPULATION AGE STRUCTURE.

		Males			Females		
Country	Model	Cancer	Circulatory	Respiratory	Cancer	Circulatory	Respiratory
USA	VAR(1), QT, one CR	10.866	11.974	−9.555	5.741	8.310	−6.254
Japan	VAR(1), QT, one CR	12.045	−8.055	−5.773	−25.917	9.391	5.601
	VAR(2), QT, one CR	–	–	–	−40.856	15.106	9.366
	VAR(2), TC, two CR	–	–	–	4.729	1.794	1.545
	VAR(2), TC, two CR	–	–	–	42.901	−14.992	−9.177
France	VAR(2), no trend, one CR	−1.951	0.818	3.629	−42.434	6.866	5.949
E&W	VAR(1), QT, one CR	28.790	−18.847	5.328	−32.441	18.261	−7.432
Australia	VAR(1), QT, one CR	−23.892	18.853	−13.363	–	–	–
	VAR(2), QT, one CR	−36.463	29.397	−20.931	−34.964	−12.098	−9.287
	VAR(2), no trend, one CR	−13.433	1.267	1.349	–	–	–

QT = Quadratic trend in the VAR (Case 3 of Section 2.1); TC = Linear trend in the cointegrating relation and in the VAR (Case 2); no trend = No trend in the cointegrating relation (Case 1); CR = cointegrating relation.
This table presents estimates of the components of the β matrix introduced in Equation (1). For example, males in the United States have an estimated long-run equilibrium relationship given by

$$10.866 \times \log(m^{\text{Jap}}_{t,\text{cancer,males,USA}}) + 11.974 \times \log(m^{\text{Jap}}_{t,\text{circulatory,males,USA}}) - 9.555 \times \log(m^{\text{Jap}}_{t,\text{respiratory},males,USA}) = z_t,$$

where z_t is a stationary variable.

First, I&P and the external causes of death do not significantly appear in cointegrating relations in 14 out of the 20 cases we consider. This pattern is the only one regularly observed across countries. This finding is consistent with previous studies made by biologists and demographers where exogenous factors impacting mortality are considered separately to endogenous factors. I&P and the external causes of death are considered as exogenous causes of death and as such not directly affected by any underlying biological aging processes, in contrast to endogenous causes. As mentioned by Hayflick (2004), accidents, infectious diseases and genetic anomalies are not driven by the aging processes. Cointegrating relations capture common stochastic trends among endogenous causes of death, and have the potential to capture the statistical characteristics of the biological processes of aging.

Second, no matter which age structure we use to compute age-standardized death rates, steady-states are similar. Biological aging seems then to impact age-groups in a similar way. However, further analyses are required to confirm this statement, as for example by applying cointegration techniques to a restricted number of age-groups.

Third, the long-run steady-states are consistently similar between males and females. The aging processes do not depend significantly on gender.

Finally, comparing these trends across countries allows us to identify countries with similar trends. Both genders in France and Japan have similar steady-states, while the long-run equilibrium for males in Australia behaves similarly to the long-run equilibria for both genders in E&W. Some of the variability in results may be explained by the 60-year time horizon of our database, a rather short period for a cointegration analysis. Non-similarities between countries may also be due to differences in death classification, in interpretation of international rules, in coding practices and in training of physicians (Booth and Tickle, 2008) or in different specific diseases that dominate each of the five broad categories studied. It is difficult to be too conclusive at this stage whether or not a country-specific environment has an important impact on the relationships between the causes of death, nor if applying results from one country to another may be misleading.

As mentioned by Olshansky *et al.* (2002), *because aging is the greatest risk factor for the leading causes of death and other age-related pathologies, more attention must be paid to the study of these universally underlying processes.* Such study may *hold the key to an understanding of all of the causes of death presently written on the death certificates of elderly people.* Unfortunately, as underlined by Hayflick (2004), resources available for research *to increase our understanding of the underlying aging process* are extremely low and *very little research is conducted on efforts to understand the biology of aging*.

There is then an internationally recognized need for a better understanding of the fundamental mechanisms of health, the causes of death and the underlying aging processes (Olshansky *et al.*, 2002; Robertson *et al.*, 2013). The cointegration analysis presented has provided insights in that direction that also bridge concepts developed in biology and in econometrics. Even if very broad

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categories of causes of death were analyzed in order to minimize the impact of ICD changes, it provides a foundation for further researches on cause-of-death mortality trends. By applying cointegration techniques to a wider range of countries and to a more refined cause-of-death classification, similarities and non-similarities between countries could be confirmed as well as better explained and theories developed by biologists assessed. An application of cointegration techniques to a more refined cause-of-death classification would avoid the loss of information due to the presence of: (1) a dominant cause in a broad category (e.g. ischaemic heart diseases under diseases of the circulatory system); (2) opposite trends being compensated in a broad category. These two aspects may differ across countries and explain the non-similarities that have been observed between some countries. Besides, a more detailed cause-specific mortality analysis would allow a more precise distinction between exogenous and endogenous causes: for example, according to some researchers, smoking-related cancers should be classified as exogenous causes of death (Carnes and Olshansky, 1997). Cointegration techniques are useful tools to test such assumptions.

Besides applying cointegration analysis to a wider range of countries and to a more refined cause description, two additional research questions may be worth investigating. First, cointegration techniques could be helpful in generating hypotheses regarding chronic disease mortality, such as for coronary heart diseases, ischaemic stroke, lung cancer, etc. Valuable epidemiological interpretation could be gain and complement the already existing extensive literature on the etiology, prevention and prognosis of leading chronic causes of death.

Second, it would be worth exploring alternative explanations for the significant cointegration found, which may complement the shared biological basis given in this study. Indeed, since our analysis is based on the observed mortality rates over the last 60 years or so, the common stochastic trends between the endogenous causes may also be partly due to the observed changes in for example smoking habits or diets (e.g. fat and salt consumption), which simultaneously affect coronary heart diseases, some cancers and chronic obstructive pulmonary diseases. Past medical advances, development of health-care facilities (number of hospitals, specialists, etc) and of national health care systems as well as other societal improvements (such as better living and working conditions, technological developments or GDP changes) would also be worth investigating to see potential connections with cointegrating processes. It would be interesting to determine if these major medical and societal improvements have an impact on the long-run steady-states (the β matrix in Equation (1)) or only have an impact on the remaining parameters of Equation (1), that are reflecting short-run adjustments towards a more fundamental equilibrium related to biology.

However, cointegration and related models need to be used with caution. These techniques were originally developed to assess and to test several economic theories, through the distinction made between short-run and long-run dynamics. As mentioned by Johansen and Juselius (1994), *the possibility to ask interesting questions within a well-defined statistical model such that they can be validly tested makes this approach potentially useful for economic investigation*.

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Even so, these models are not meant for forecasting purposes. How to use cointegrating relations, such as the ones developed in this paper, in order to improve current forecasting techniques remains a very interesting and open question.

To conclude, similarities exist between patterns of endogenous mortality across countries. These similarities are reflected in the cointegrating relations for causes of death and explained by the biological aging processes that impact all human bodies. New perspectives for modeling the dependence between causes of death have been provided. Taking these new relations into account in the modeling and forecasting processes should provide a basis for improving the analysis of cause-specific mortality rates and this needs to be carefully considered.

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Notes

1. In this paper, we consider variables that are integrated of order one. In this case, cointegrating relations are necessarily stationary. For a more general framework, see Hamilton (1994) and Lütkepohl (2005).

2. Developing countries are not included, their data being less reliable.

3. This database was adjusted in order to analyze data consistently over time and across countries: the number of deaths of unknown age is proportionally distributed across the age range; ages 85 and above as well as ages one to four are grouped together; comparability ratios are used to remove discontinuities in the death rates due to changes of classification over time (the ICD changed three times between 1950 and 2010, from ICD-7 to ICD-10). Details are provided in Arnold and Sherris (2013, 2015).

4. Assuming that the error term ϵ_t in Equation (1) is Gaussian, these tests are based on the comparison of the log likelihood function of $\mathbf{y}_t$ with r and n cointegrating relations for the trace test, while the log likelihoods including r and $r+1$ cointegrating relations are compared for the maximum-eigenvalue test.

5. It is important to mention that the goodness of fit of the VECM using these restricted cointegating relations (we will call it the *restricted VECM*) may not be optimal for the genders and countries using only one lag (mentioned as *VAR(1)* in Tables 10 and 11), even if these models appear to be the best ones with unrestricted cointegrating relations (as shown in Appendix B). However, by increasing the number of lags to two (e.g. instead of using a VAR(1) with a quadratic trend and one cointegrating relation for males in USA, we use a VAR(2) with a quadratic trend and one cointegrating relation), the residuals of the restricted VECM do not present any remaining autocorrelation and also become normally distributed and thus, the model fits perfectly well the data. By increasing the number of lags, the cointegrating relations remain however similar to the ones introduced in Tables 10 and 11 and the conclusions that are derived in our analysis remain

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valid. Increasing the number of lags results in an increase in the number of parameters ξ_i in Equation (1) that reflect short-run dynamics. The short-run adjustments do not have an impact on the form of the long-run equilibrium relationships and thus, do not have a significant impact on the cointegrating relations. We will discuss the cointegrating relations presented in Tables 10 and 11, since they result from standard practice in cointegration analysis and are not altered by increasing the number of lags included in the model in order to improve the goodness of fit of the restricted VECM.

References

ADAMS, J.M. and WHITE, M. (2004) Biological aging: A fundamental, biological link between socio-economic status and health? *European Journal of Public Health,* **14**(3), 331–334.

ANDERSON, R.N. (1999) US decennial life tables: 1989–91. United States life tables eliminating certain causes of death. *DHHS Publication No (PHS) 99-1150-4*, **1**(4).

ARNOLD, S. and SHERRIS, M. (2013) Forecasting mortality trends allowing for cause-of-death mortality dependence. *North American Actuarial Journal,* **17**(4), 273–282.

ARNOLD, S. and SHERRIS, M. (2015) Causes-of-death mortality: What do we know on their dependence? *North American Actuarial Journal,* **19**(2), 116–128.

BAYO, F. (1968) Life tables: 1959–61. United States life tables by causes of death: 1959–61. *Public Health Service Publication No 1252*, **1**(6).

BOOTH, H. and TICKLE, L. (2008) Mortality modelling and forecasting: A review of methods. *Annals of Actuarial Science*, **3**(1–2), 3–43.

BUTLER, R.N., SPROTT, R., WARNER, H., BLAND, J., FEUERS, R., FORSTER, M., FILLIT, H., HARMAN, S.M., HEWITT, M., HYMAN, M., JOHNSON, K., KLIGMAN, E., MCCLEARN, G., NELSON, J., RICHARDSON, A., SONNTAG, W., WEINDRUCH, R. and WOLF, N. (2004) Biomarkers of aging: From primitive organisms to humans. *Journal of Gerontology: Biological Sciences*, **59A**(6), 560–567.

CARNES, B.A., HOLDEN, L.R., OLSHANSKY, S.J., WITTEN, T.M. and SIEGEL, J.S. (2006) Mortality partitions and their relevance to research on senescence. *Biogerontology*, **7**(4), 183–198.

CARNES, B.A. and OLSHANSKY, S.J. (1997) A biologically motivated partitioning of mortality. *Experimental Gerontology,* **32**(6), 615–631.

CARNES, B.A., OLSHANSKY, S.J. and HAYFLICK, L. (2013) Can human biology allow most of us to become centenarians? *Journal of Gerontology: Biological Sciences,* **68**(2), 136–142.

CASELLI, G. (1996) Future longevity among the elderly. In *Health and Mortality among Elderly Populations* (ed. G. Caselli and A.D. Lopez), pp. 235–265. Oxford: Clarendon Press.

CASELLI, G., VALLIN, J. and MARSILI, M. (2006) How useful are the causes of death when extrapolating mortality trends. An update. *Social Insurance Studies from the Swedish Social Insurance*, **4**, 9–36.

CURTIN, L.R. and ARMSTRONG, R.J. (1988) US decennial life tables: 1979-81. United States life tables eliminating certain causes of death. *DHHS Publication No (PHS) 88-1150-2*, **1**(2).

GAILLE, S. and SHERRIS, M. (2011) Modeling mortality with common stochastic long-run trends. *The Geneva Papers on Risk and Insurance - Issues and Practice,* **36**(4), 595–621.

GOMPERTZ, B. (1825) On the nature of the function expressive of the law of human mortality and on a new mode of determining life contingencies. *Philosophical Transactions of the Royal Society of London*, **115**, 513–585.

GREVILLE, T.N.E., BAYO, F. and FOSTER, R.S. (1975) Life tables: 1969–71. United States life tables by causes of death: 1969–71. *DHEW Publication No (HRA) 75-1150*, **1**(5).

HAMILTON, J.D. (1994) *Time Series Analysis*. Princeton: Princeton University Press.

HAYFLICK, L. (2004) "Anti-Aging" is an oxymoron. *Journal of Gerontology: Biological Sciences*, **59A**(6), 573–578.

HOLLIDAY, R. (2004) The multiple and irreversible causes of aging. *Journal of Gerontology: Biological Sciences*, **59A**(6), 568–572.

HOUGAARD, P. (1984) Life table methods for heterogeneous populations: Distributions describing the heterogeneity. *Biometrika,* **71**(1), 75–83.

https://doi.org/10.1017/asb.2015.24 Published online by Cambridge University Press

JIN, K. (2010) Modern biological theories of aging. *Aging and Disease,* **1**(2), 72–74.
JOHANSEN, S. (1988) Statistical analysis of cointegration vectors. *Journal of Economic Dynamics and Control*, **12**(2–3), 231–254.
JOHANSEN, S. (1991) Estimation and hypothesis testing of cointegration vectors in Gaussian vector autoregressive models. *Econometrica,* **59**(6), 1551–1580.
JOHANSEN, S. (1994) The role of the constant and linear terms in cointegration analysis of nonstationary variables. *Econometric Reviews,* **13**(2), 205–229.
JOHANSEN, S. and JUSELIUS, K. (1992) Testing structural hypotheses in a multivariate cointegration analysis of the PPP and the UIP for UK. *Journal of Econometrics*, **53**(1–3), 211–244.
JOHANSEN, S. and JUSELIUS, K. (1994) Identification of the long-run and the short-run structure. An application to the ISLM model. *Journal of Econometrics*, **63**(1), 7–36.
KAISHEV, V.K., DIMITROVA, D.S. and HABERMAN, S. (2007) Modelling the joint distribution of competing risks survival times using copula functions. *Insurance: Mathematics and Economics,* **41**(3), 339–361.
LÜTKEPOHL, H. (2005) *New Introduction to Multiple Time Series Analysis*. Berlin: Springer.
MAKEHAM, W. (1867) On the law of mortality. *Journal of the Institute of Actuaries,* **13**(6), 325–358.
MANTON, K.G. (1986) Past and future life expectancy increases at later ages: Their implications for the linkage of morbidity, disability, and mortality. *Journal of Gerontology,* **41**(5), 672–681.
MANTON, K.G. and MYERS, G.C. (1987) Recent trends in multiple-caused mortality 1968 to 1982: Age and cohort components. *Population Research and Policy Review*, **6**, 161–176.
MANTON, K.G., PATRICK, C.H. and STALLARD, E. (1980a) Mortality model based on delays in progression of chronic diseases: Alternative to cause elimination model. *Public Health Reports*, **95**(6), 580–588.
MANTON, K.G. and POSS, S.S. (1979) Effects of dependency among causes of death for cause elimination life table strategies. *Demography,* **16**(2), 313–327.
MANTON, K.G., STALLARD, E. and POSS, S.S. (1980b) Estimates of U.S. multiple cause life tables. *Demography,* **17**(1), 85–102.
MANTON, K.G., STALLARD, E. and VAUPEL, J.W. (1986) Alternative models for the heterogeneity of mortality risks among the aged. *Journal of the American Statistical Association,* **81**(395), 635–644.
MANTON, K.G., TOLLEY, H.D. and POSS, S.S. (1976) Life table techniques for multiple-cause mortality. *Demography,* **13**(4), 541–564.
MCNOWN, R. and ROGERS, A. (1992) Forecasting cause-specific mortality using time series methods. *International Journal of Forecasting,* **8**(3), 413–432.
OLSHANSKY, S.J. (1987) Simultaneous/multiple cause-delay (SIMCAD): An epidemiological approach to projecting mortality. *Journal of Gerontology,* **42**(4), 358–365.
OLSHANSKY, S.J., HAYFLICK, L. and CARNES, B.A. (2002) Position statement on human aging. *Journal of Gerontology: Biological Sciences*, **57A**(8), B292–B297.
OLSHANSKY, S.J., HAYFLICK, L. and PERLS, T. (2004) Anti-aging medicine: The hype and reality - I. *Journal of Gerontology: Biological Sciences*, **59A**(6), 000–000.
ROBERTSON, T., BATTY, G.D., DER, G., FENTON, C., SHIELS, P.G. and BENZEVAL, M. (2013) Is socioeconomic status associated with biological aging as measured by telomere length? *Epidemiologic Review*, **35**(1), 98–111.
ROSÉN, M. (2006) Forecasting life expectancy and mortality in Sweden–some comments on methodological problems and potential approaches. Technical Report 4, Social Insurance Studies from the Swedish Social Insurance.
SHRYOCK, H.S., SIEGEL, J.S. and ASSOCIATES (1975) *The Methods and Materials of Demography*, volume 2, Washington, DC: U.S. Dept of Commerce, Bureau of the Census, U.S. Govt. Printing Office.
STREHLER, B. (1959) Origin and comparison of the effects of time and high-energy radiations on living systems. *The Quarterly Review of Biology*, **34**, 117–142.
TABEAU, E., EKAMPER, P., HUISMAN, C. and BOSCH, A. (1999) Improving overall mortality forecasts by analysing cause-of-death, period and cohort effects in trends. *European Journal of Population*, **15**(2), 153–183.
VAUPEL, J.W. and YASHIN, A.I. (1983) The deviant dynamics of death in heterogeneous populations. Technical Report RR-83-001, International Institute for Applied Systems Analysis (IIASA).

WILMOTH, J.R. (1996) Mortality projections for Japan: A comparison of four methods. In *Health and Mortality among Elderly Populations* (eds. G. Caselli and A.D. Lopez), pp. 266–287. Oxford: Clarendon Press.
WONG-FUPUY, C. and HABERMAN, S. (2004) Projecting mortality trends: Recent developments in the United Kingdom and the United States. *North American Actuarial Journal,* **8**(2), 56–83.
WORLD HEALTH ORGANIZATION (2012) WHO mortality database. `http://www.who.int/whosis/mort/download/en/index.html`.

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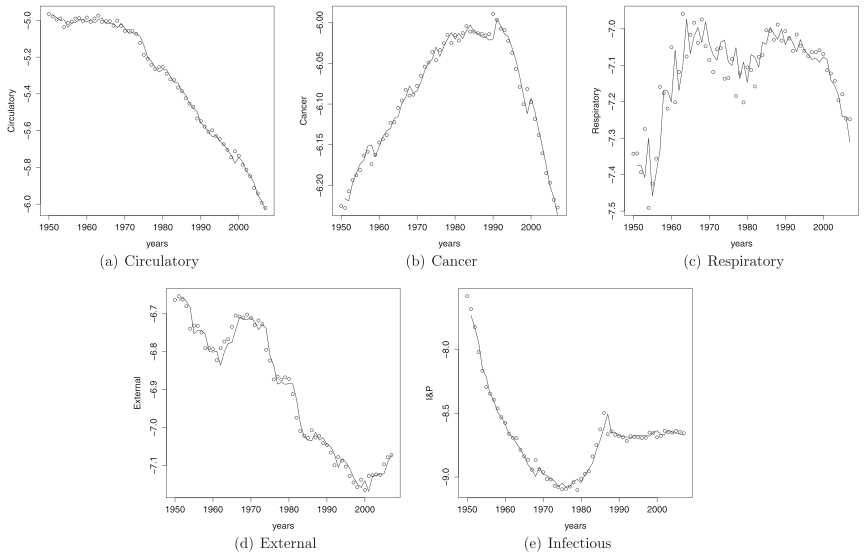
APPENDIX

A. FITTED MODEL FOR US AND JAPANSES MALES, WITH US MALE POPULATION AGE STRUCTURE



(a) Circulatory (b) Cancer (c) Respiratory

(d) External (e) Infectious

FIGURE A.1: Observed and fitted cause-specific log-death rates, US males.
The observed age-standardized log-death rates are depicted by the dots. The curve represents the fitted VECM described in Section 4.1, that is including one lag, one cointegrating relation and a quadratic trend (Case 3 of Section 2.1).

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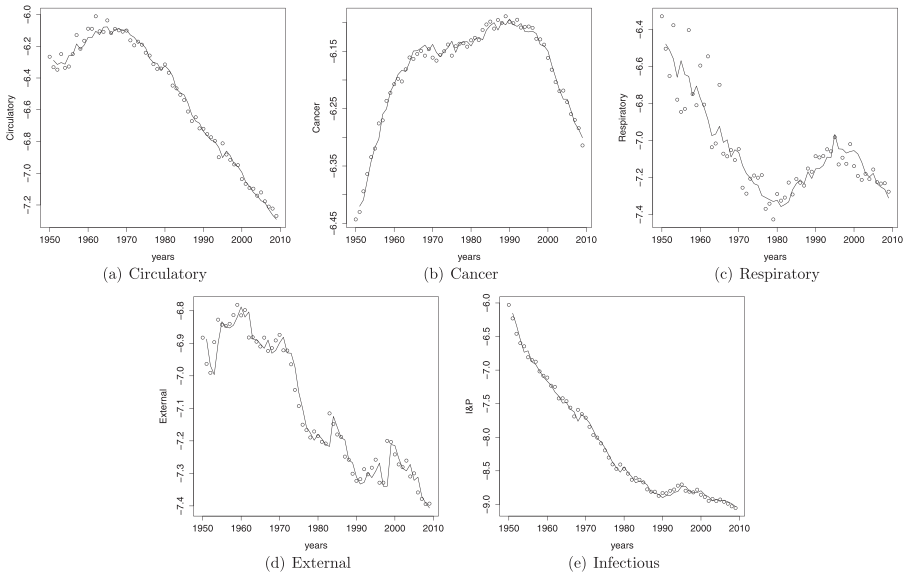

(a) Circulatory (b) Cancer (c) Respiratory

(d) External (e) Infectious

FIGURE A.2: Observed and fitted cause-specific log-death rates, Japanese males.
The observed age-standardized log-death rates are depicted by the dots. The curve represents the fitted VECM described in Section 4.1, that is including one lag, one cointegrating relation and a quadratic trend (Case 3 of Section 2.1).

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B. TESTS ON RESIDUALS OF THE FITTED VECM

TABLE B.1

TESTS ON RESIDUALS OF THE FITTED VECM, MALES WITH US MALE POPULATION AGE STRUCTURE.

		p value					
		Autocorrelation Portmanteau			Normality		
Country	Model	15 lags	25 lags	35 lags	Skewness	Kurtosis	Both
USA	VAR(1), QT, 1 CR	0.189	0.336	0.376	0.640	0.011	0.051
Japan	VAR(1), QT, 1 CR	0.144	0.300	0.159	0.085	0.106	0.043
France	VAR(2), NT, 1 CR	0.336	0.715	0.839	0.467	0.086	0.163
	VAR(1), QT, 1 CR	0.680	0.881	0.739	0.324	0.667	0.529
E&W	VAR(1), QT, 1 CR	0.652	0.537	0.100	0.917	0.443	0.794
Australia	VAR(1), QT, 1 CR	0.108	0.002	0.007	0.331	0.047	0.074
	VAR(2), QT, 1 CR	0.594	0.297	0.405	0.876	0.002	0.027

QT = Quadratic trend in the VAR (Case 3 of Section 2.1); TC = Linear trend in the cointegrating relation and in the VAR (Case 2); NT = No trend in the cointegrating relation (Case 1); CR = cointegrating relation. The null hypothesis of no-autocorrelation among the residuals is tested through the Portmanteau statistic, with a lag of 15, 25 and 35. The skewness statistic, the kurtosis statistic and a combination of these are used to test the normality of the residuals. A null hypothesis is accepted at a α% significance level when the p value is higher than α%. Normality is only rejected in Australia at a 1% significance level, while some autocorrelations only remain in Australia too.

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TABLE B.2

TESTS ON RESIDUALS OF THE FITTED VECM, FEMALES WITH US MALE POPULATION AGE STRUCTURE.

		p value					
		Autocorrelation Portmanteau			Normality		
Country	Model	15 lags	25 lags	35 lags	Skewness	Kurtosis	Both
USA	VAR(1), QT, 1 CR	0.420	0.066	0.064	0.514	0.569	0.617
Japan	VAR(1), QT, 1 CR	0.085	0.652	0.367	0.150	0.039	0.031
	VAR(2), TC, 2 CR	0.028	0.325	0.320	0.598	0.113	0.248
France	VAR(1), QT, 1 CR	0.270	0.384	0.384	0.070	0.206	0.066
	VAR(1), NT, 1 CR	0.048	0.388	0.575	0.699	0.285	0.511
E&W	VAR(1), QT, 1 CR	0.718	0.258	0.250	1.08e-2	1.53e-6	2.82e-7
Australia	VAR(2), NT, 1 CR	0.238	0.284	0.262	0.652	0.059	0.175

QT = Quadratic trend in the VAR (Case 3 of Section 2.1); TC = Linear trend in the cointegrating relation and in the VAR (Case 2); NT = No trend in the cointegrating relation (Case 1); CR = cointegrating relation. The null hypothesis of no-autocorrelation among the residuals is tested through the Portmanteau statistic, with a lag of 15, 25 and 35. The skewness statistic, the kurtosis statistic and a combination of these are used to test the normality of the residuals. A null hypothesis is accepted at a α% significance level when the p value is higher than α%. Normality is only rejected in E&W, while no autocorrelation remains in any country.

TABLE B.3

TESTS ON RESIDUALS OF THE FITTED VECM, MALES WITH JAPANESE FEMALE POPULATION AGE STRUCTURE.

		p value					
		Autocorrelation Portmanteau			Normality		
Country	Model	15 lags	25 lags	35 lags	Skewness	Kurtosis	Both
USA	VAR(1), QT, 1 CR	0.288	0.229	0.284	0.911	0.802	0.954
Japan	VAR(1), QT, 1 CR	0.076	0.168	0.143	0.404	0.356	0.388
France	VAR(2), NT, 1 CR	0.449	0.711	0.642	0.207	0.185	0.143
E&W	VAR(1), QT, 1 CR	0.795	0.560	0.363	0.887	0.225	0.566
Australia	VAR(1), QT, 1 CR	0.011	0.001	2.82e-4	0.516	0.155	0.268
	VAR(2), QT, 1 CR	0.571	0.513	0.444	0.829	0.002	0.018
	VAR(2), NT, 1 CR	0.521	0.397	0.437	0.407	0.013	0.034

QT = Quadratic trend in the VAR (Case 3 of Section 2.1); TC = Linear trend in the cointegrating relation and in the VAR (Case 2); NT = No trend in the cointegrating relation (Case 1); CR = cointegrating relation. The null hypothesis of no-autocorrelation among the residuals is tested through the Portmanteau statistic, with a lag of 15, 25 and 35. The skewness statistic, the kurtosis statistic and a combination of these are used to test the normality of the residuals. A null hypothesis is accepted at a α% significance level when the p value is higher than α%. Normality is only rejected in Australia at a 1% significance level, while some autocorrelations only remain in Australia too.

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TABLE B.4

TESTS ON RESIDUALS OF THE FITTED VECM, FEMALES WITH JAPANESE FEMALE POPULATION AGE STRUCTURE.

		p value					
		Autocorrelation Portmanteau			Normality		
Country	Model	15 lags	25 lags	35 lags	Skewness	Kurtosis	Both
USA	VAR(1), QT, 1 CR	0.262	0.019	0.023	0.032	0.057	0.011
Japan	VAR(1), QT, 1 CR	0.003	0.065	0.022	0.301	0.295	0.274
	VAR(2), QT, 1 CR	0.004	0.042	0.040	0.255	0.112	0.115
	VAR(2), TC, 2 CR	0.008	0.097	0.170	0.542	0.073	0.167
France	VAR(2), NT, 1 CR	0.049	0.354	0.200	0.322	0.043	0.068
E&W	VAR(1), QT, 1 CR	0.914	0.578	0.413	2.26e-3	7.31e-12	5.78e-13
Australia	VAR(2), QT, 1 CR	0.548	0.668	0.353	0.488	0.046	0.108

QT = Quadratic trend in the VAR (Case 3 of Section 2.1); TC = Linear trend in the cointegrating relation and in the VAR (Case 2); NT = no trend in the cointegrating relation (Case 1); CR = cointegrating relation. The null hypothesis of no-autocorrelation among the residuals is tested through the Portmanteau statistic, with a lag of 15, 25 and 35. The skewness statistic, the kurtosis statistic and a combination of these are used to test the normality of the residuals. A null hypothesis is accepted at a α% significance level when the p value is higher than α%. Normality is only rejected in E&W at a 1% significance level, while some autocorrelations seem to remain in Japan.

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