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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 process 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 shortrun 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.

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.

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 longrun 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 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

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

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

 $\Pi = \alpha \beta';$ = matrix of rank r; $\alpha = a (n \times r) \text{ loading matrix };$ $\beta = a (n \times r) \text{ matrix containing the } r \text{ cointegrating vectors.}$

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

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

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

¹In 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).

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 tests exist for that, such as Akaike's Information Criteria (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 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, 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 cointegrat-

ing 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 \mathbf{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 no quadratic trend appears in the process.
- 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**.

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 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 significantly different from 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-sexspecific mid-year populations for each calendar year in several countries. The first divided by the second produces central death rates. 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 un-

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

derlying cause of death is specified as the disease or injury which initiated 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) and Arnold and Sherris (2015).

Causes of death	ICD 7	ICD 8	ICD 9	ICD 10	
Circulatory system	A079-A086	A080-A088	B25-B30	100-199	
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	

Table 1: International Classification of Diseases - Coding system

The same database as in Arnold and Sherris (2013) and Arnold and Sherris (2015) is used. 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. 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 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 $m_{t,d,s,c}^{US}$ 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 and by $m_{t,d,s,c}^{Jap}$ 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 population is constant over the complete period under observation and equal to the age structure of the 2009 Japanese female population.

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 the age structure of US males and the age structure of Japanese females, so twenty times altogether. To be concise, we are looking for long-run steady-states between the logarithm of the following variables: First, $m_{t,circulatory,s,c}^{US}$, $m_{t,cancer,s,c}^{US}$, $m_{t,respiratory,s,c}^{US}$, $m_{t,cancer,s,c}^{Jap}$, for each country and gender; Second, $m_{t,circulatory,s,c}^{Jap}$, $m_{t,cancer,s,c}^{Jap}$, $m_{t,external,s,c}^{Jap}$, for each country $m_{t,I\&P}^{Jap}$, for each country and gender.

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 fitted models 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 tests 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 nonstationary 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.

4.1.2 Contegration

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 maximumeigenvalue statistic. 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.

r	Trace sta	atistics	Critical values					
	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		

Table 2: Tests for the number of cointegrating relations, males with US male population age structure

	Maximum-e	igenvalue	Critical values					
r	statistics							
	USA	SA 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 5%, 2.5% and 1% significance level in Japan.

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.

Table 3: Tests on residuals of the fitted VECM, males with US male population age structure

		<i>p</i> -value				
Type of test	Name of test	USA	Japan			
		Quadratic	No	Quadratic		
		trend	trend	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.

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 coefficients using a likelihood ratio statistic with an asymptotic χ^2 distribution. We have no strong prior hypothesis on the causes of death that will be significant in the cointegrating relations, so we systematically test the significance of each coefficient. 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 would be in the cointegrating relation at a 1% significance level in the USA. In Japan, circulatory diseases, cancer and diseases of the respiratory system would be included with the external causes of death and the I&P not statistically significant at a 1% significance level.

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.

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.

Table 6: Tests for the simultaneous significance of the coefficients in the cointegrating relation, US males

	I&P and	I&P and	I&P and	Cancer and	Cancer and	Circulatory
	Cancer	Circulatory	External	Circulatory	External	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.

In order to test if a variable is trend stationary, we can test the null hypothesis that all the variables 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, 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. Long-run steady-states are studied in 20 different settings, for two genders in five countries using two reference populations. This analysis allows us to more reliably identify similarities in longrun 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 $m_{t,d,females,Japan}^{Jap}$ 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 $m_{t,d,females,Japan}^{US}$ 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 the two cointegrating relations (Table 8). As with the $m_{t,d,females,Japan}^{Jap}$ variables, only I&P with the external causes of death are not significantly appearing in the cointegrating relations in both models.

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

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

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

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

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	-

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

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

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 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 (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 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.

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 can not 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 (Carnes et al. (2013); Holliday (2004)), 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 countries under study, for both males and females. Results are presented in Tables 10 and 11.

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

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Table 10:

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

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. These results show, for example, that males in the United States have an estimated long-run equilibrium relationship given by

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

where z_t is a stationary variable.

Females
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espiratory -9.555 573
ulatory Res 11.974
incer Circu
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Model

Table 11: Restricted cointegrating relations, Japanese female population age structure

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.These results show, for example, that males in the United States have an estimated long-run equilibrium relationship given by

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

where z_t is a stationary variable.

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 (nonsignificant 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 causeof-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.

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 impacts age-groups in a similar way.

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 longrun equilibria for both genders in E&W. Some of the variability in results may be impacted by the fact that we only use mortality data for around 60 years, 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)). It is difficult to be too conclusive 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.

As recognized internationally, there is a need for a better understanding of the fundamental mechanisms of health, the causes of death and the underlying aging processes (Robertson et al. (2013); Olshansky et al. (2002)). The cointegration analysis presented has provided insights that bridge concepts developed in biology and in econometrics. It provides a foundation for further research on cause-of-death mortality trends. By applying contegration techniques to a wider range of countries and to a more refined cause-of-death classification, similarities between countries could be confirmed and theories developed by biologists assessed. 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.

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 causespecific mortality rates.

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