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Forecasting Causes of Death using Compositional Data Analysis: the Case of Cancer Deaths

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Abstract

Cause–specific mortality forecasting is often based on predicting cause–specific death rates independently. Only a few methods have been suggested that incorporate dependence among causes. An attractive alternative is to model and forecast cause–specific death distributions, rather than mortality rates, as dependence among the causes can be incorporated directly. We follow this idea and propose two new models which extend the current research on mortality forecasting using death distributions. We find that adding age, time, and cause–specific weights and decomposing both joint and individual variation among different causes of death increased the forecast accuracy of cancer deaths using data for French and Dutch populations.

Keywords: Cause-specific mortality; Cancer forecast; Forecasting methods; Compositional Data Analysis; Population health

JEL Codes: C22, C23, C53, I12

1 Introduction

Forecasting mortality by cause of death can provide valuable information for health care and social services planning in general. In particular, the future distribution of deaths by cause is of interest given the potential to efficiently target public health actions. The future cause-specific death distribution may be related to the relative risk of a disease and be used to predict future incidence rates. In many countries, incidence forecasts are used for planning future hospital capacities, for example, for cancer patients (Rapiti et al., 2014). A major challenge when modelling cause of death data is the competing risks between causes. In a conventional all-causes-combined life table there is a one-to-one relation between the mortality rates and the cumulative incidence of deaths. This relation is lost when cause-specific deaths are analysed because survival may be influenced by all the causes, even if the cause-specific mortality rates are independent, see Andersen et al. (2012). Analysts interested in the future pattern of deaths from a specific cause cannot ignore the effects of the other competing causes.

Among demographers and actuaries, cause specific mortality is conventionally analysed using a multiple decrement life table where age- and cause-specific mortality rates are calculated using age- and cause-specific deaths and the total number of persons at risk in the population at a given age for a specific year. The underlying assumption in the multiple decrement life table is that cause-specific death rates are independent so that a change in the death rate for cause i does not imply a change in the death rate for cause j. However, the survival probabilities in the life table for each cause are not independent because they are calculated on the basis of all the cause-specific death rates so that the proportion of people dying from a specific cause may be affected by the mortality from other causes, see Preston et al. (2001) for further details.

The multiple decrement life table is a standard tool in demography and enables the calculation of cause-specific mortality and thus it is used to calculate the input for all the models analysed. The same approach is followed by all other cause-specific mortality forecasting models. Dependence among cause-specific mortality also relates to the time dependent processes underlying changes in all-cause and cause-specific mortality. For example, behavioural changes in the population may affect multiple causes of death over a longer time period. Modelling of this dependence relates to the choice of model for modelling and forecasting. Generally, this dependence has been ignored as each cause is modelled independently of the others. Sev-

eral studies use the Lee and Carter (1992) model (LC) to forecast cause of death mortality or age-period-cohort models and apply the models independently to each cause: for example, Wilmoth (1995); Peltonen and Asplund (1996); Knorr-Held and Rainer (2001); Cesare and Murphy (2011) . Regression models have also been used to forecast mortality by cause of death, (Mathers and Loncar, 2006), but in their approach causes are treated individually. Girosi and King (2008) relate causes of death in a Bayesian framework by treating the different causes as spatially different groups however the estimation is not carried out jointly for all the causes - data from different countries are included in a similar way.

Independent modelling and forecasting of cause–specific mortality is not only unattractive because it ignores dependence patterns among the causes, but also because forecasts often fail to be coherent in the sense that cause–specific deaths must sum to the total number of deaths, which could lead to implausible forecasts. Recently, some mortality forecasting models have been suggested which include dependence among different causes of death and thus incorporate competing risks among causes of deaths: Oeppen (2008); Arnold-Gaille and Sherris (2013); Foreman et al. (2017) and Hirz et al. (2017). In contrast to the other proposed models, Oeppen (2008) uses life table deaths to forecast cause–specific mortality using compositional data analysis (CoDA). CoDA a is well established set of statistical methods for the analyses of compositional data which is defined as a data vector with positive elements summing to a constant value and thereby only contains relative information (Pawlowsky-Glahn and Buccianti, 2011). Life table deaths sum to the life table's initial birth cohort in each year across all causes and age. Thus, CoDA enables a coherent and correct modelling of dependent causes of death by recognizing the sum constraint.

Oeppen (2008) used a Lee-Carter type of model within the CoDA to forecast both all–cause mortality and cause–specific mortality. Wilmoth (1995) argued that an aggregation of individual causes always leads to more pessimistic forecasts than an all causes combined forecast but Oeppen (2008) showed that this is not the case when modelling life table deaths because of the dynamics in CoDA. CoDA was also used by Bergeron-Boucher et al. (2017) for coherent forecasts of all–cause mortality in different countries. One important difference between modelling cause–specific deaths rates and deaths distributions is that deaths are directly dependent on each other on a aggregated level, as avoided deaths from one cause will result in an increase of deaths from some other cause. The same mechanism does not apply to death rates as death

rates are defined as deaths divided by the number of people at risk. Avoided deaths from one cause thus affect both the numerator and the denominator, and the dependence between rates is therefore not as easy to predict (Preston et al., 2001).

This study examines the limitations of the Oeppen (2008) model by suggesting two new models: the CoDA model with co–integrating vector error correction model (VECM-CoDA) that allows for multiple time trends and the 2-step CoDA model (2S-CoDA) which extends the Oeppen (2008) model by introducing time, age, and cause specific weights and by adding cause specific information to the forecasts. The 2S-CoDA model is found to produce more accurate cancer forecasts for the populations analysed than the Oeppen (2008) model. The VECM-CoDA and 2S-CoDA models make use of compositional data analysis similar to the model presented in Bergeron-Boucher et al. (2017) but differ fundamentally in the way morality is modelled. The VECM-CoDA and 2S-CoDA models model several causes of death from a multiple decrement life table in a single population. In contrast Bergeron-Boucher et al. (2017) consider a single decrement life table but several populations. Further analysis can be found in the supplementary material, which examines the influence of the number of causes of deaths included on the accuracy of the cancer forecasts.

It is well known that the dependence between causes of death is not identifiable for individuals, (see (A, 1975), as it is not possible to distinguish between independent competing risks and a large number of dependent competing risks producing the same cause-specific hazards. CoDA mortality models, which makes use of aggregated data, can respect covariances between ages and causes, but only identify net transitions of shifted deaths between ages and/or causes. They do they suggest that the latent mortality patterns in the absence of competing risks are identifiable.

In this study we focus on forecasting cancer mortality and evaluate the proposed models' ability to forecast cancer deaths. Cancer is selected as it is a major cause of death over the last 50 years, is relatively well diagnosed as the principal cause of death, and has experienced an increasing share of deaths in many industrialised countries. In 2014, 26.4% of all deaths in EU countries (Eurostat, 2017) was caused by cancer and 83.2 billion Euros were spent on health care related to cancer (Jönssona et al., 2016). Despite the importance of cancer mortality, limited research has been carried out on improving current forecasting methods for cancer deaths and, hence,

this article provides valuable information for social planners and society.

2 Notation and Methods

Throughout the paper mortality is measured using life table information in order to account for the age composition of mortality. Standard life table calculations are used following Preston et al. (2001). Cause–specific mortality is often available in the form of actual death counts ($D_{t,x,i}$) by year (t), age (x) and cause of death (i), where $t \in (1, 2, ..., T)$, $x \in (1, 2, ..., N)$ five year age groups, and $i \in (1, 2..., K)$ causes. Knowing the death density for the all–cause mortality in the population i.e. the life table deaths ($d_{t,x}$) allows the calculation of cause–specific life table deaths $d_{t,x,i}$ following equation

$$d_{t,x,i} = d_{t,x} \frac{D_{t,x,i}}{D_{t,x}}, \tag{1}$$

where $D_{t,x}$ denotes the total all–cause deaths count, (Preston et al., 2001). Further, by knowing the number of person-years lived at age x ($L_{t,x}$) it is possible to calculate the associated cause–specific death rates $m_{t,x,i}$ using,

$$m_{t,x,i} = \frac{d_{t,x,i}}{L_{t,x}} \tag{2}$$

These two relations are used to transform the observed data and to relate variables within the life table.

2.1 Data

To evaluate the cause–specific mortality models, data from France and the Netherlands are used to fit the models. Data for France were downloaded from Database (2016) divided into the following causes: infectious diseases, cancer, cardio–vascular diseases, diseases of the respiratory system, diseases of the digestive system, other diseases, injury and poisoning. The death classification is taken as given from the data source. Data were divided into five year age groups, censored at age 100+, and into single years of time from 1925 to 2008. We restricted the analysis to the years 1955 and onwards to avoid fluctuating patterns from the Second World War. As few deaths occur at younger ages we restricted the data to age groups older than 25

which simplified the estimation of the CoDA models. These cannot be estimated for age groups with zero deaths, hence zeros are replaced by half a death - a similar imputation method is used by Bergeron-Boucher et al. (2015). The imputation has no appreciable effect on the results in this study.

Data for the Netherlands were downloaded from Statistics Netherlands (CBS) (2018) and divided into the following causes: infectious diseases, cancer, diseases of the endocrine system, mental diseases, diseases of the nervous system, circulatory diseases, diseases of the respiratory system, diseases of the digestive system, other diseases, and external causes. Some causes were aggregated from the original source to match the French data. A close match between the two data sets is not possible because data are not in general available at a detailed enough ICD level. Hence, the results for the two countries are not directly comparable but are used to illustrate that the models can be applied to different subdivisions of the causes of death. Similar to the French data, analyses where restricted to the ages groups 25 to 95+, where data were censored, for the years 1958 to 2014. 1958 is selected to avoid very frequent shifts in the ICD classification system of causes of death with changes in both 1941, 1950, and 1955 (Koren et al., 2012).

Sex specific all–cause life tables for France and the Netherlands for the relevant years were downloaded from the Human Mortality Database (Human Mortality Database, 2018).

2.2 Compositional Data Analysis

CoDA refers to a broad set of statistical methods used to analyse compositional data. This section briefly describes CoDA essential to the suggested models and further details can be found in (Aitchison, 1982, 1986; Pawlowsky-Glahn and Buccianti, 2011; Bergeron-Boucher et al., 2017). As life table deaths are constructed by multiplying age specific probabilities of dying into an arbitrary initial birth cohort also known as the radix, they only contain relative information and sum to the initial cohort in each year (Bergeron-Boucher et al., 2017). Changes in the number of life table deaths for a specific age group must therefore be offset by changes in the other age groups which is the fundamental feature in compositional data. Traditional decomposition techniques provide inconsistent results when applied to compositional data as they do not recognize the implicit constraints of summing to a constant (Aitchison, 1982, 1986):

mathematically, compositional data lie in the bounded space of the simplex and traditional decomposition techniques are defined for data in the real space. Aitchison (1986) showed that by making log-ratio transformations it is possible to express compositional data in the real space where the data can be analysed with conventional models and then transformed back into the simplex.

The analysis presented in the present paper makes use of the centred log-ratio (clr) transformation to express the data in the real space. The clr transformation takes the log of each observation divided by the geometric mean. That is,

$$clr(d_{t,x}) = log\left[\frac{d_{t,x}}{g(t,x)}\right], \tag{3}$$

where $g(t,x)$ is the geometric mean by age in the year, that is $g(t,x) = (d_{t,x_1} \cdot d_{t,x_2} \cdots d_{t,x_n})^{\frac{1}{n}}$. The clr transformation maintains the initial constraint in the data as its elements sum to zero by construction but resulting values are real. The inverse clr (clr^{-1}) is defined as,

$$clr^{-1}(d_{t,x}) = C\left[exp(d_{t,x})\right], \tag{4}$$

where C is the closure operator that divides each entry by the sum of all entries and multiplies by the initial number of life table deaths so the result meets the restriction of all life table deaths summing to a certain constant. For a vector $Y = (y_1, ..., y_n)$ the closure of Y is, $C[Y] = \left[\frac{y_1}{\sum y_i}, \frac{y_2}{\sum y_i}, ..., \frac{y_1}{\sum y_n}\right] \cdot K$, where K is a constant (Bergeron-Boucher et al., 2017)

Aitchison (1986) also defined addition and subtraction operations obeying conventional rules of arithmetic, called perturbations, maintaining the result of the operation in the simplex (Pawlowsky-Glahn and Buccianti, 2011). The following operations will be used in the analysis and are essential when modelling death distribution. Assume, $X = (x_1, ..., x_n)$ and $Y = (y_1, ..., y_n)$ then

$$X \ominus Y = C\left[\frac{x_1}{y_1}, ..., \frac{x_n}{y_n}\right]$$

and

$$X \oplus Y = C\left[x_1 y_1, ..., x_n y_n\right].$$

$X \ominus Y$ is called perturbation and measures the distance between X and Y in compositional

data similar to subtraction in some data on the real axis. $X \oplus Y$ is the opposite operation and can be compared with addition on the real axis.

2.3 Forecasting models

The model suggested by Oeppen (2008), denoted CoDA model with common time trend (CT-CoDA), is compared with two new CoDA models which accommodate limitations in the CT-CoDA model. That is: the 2-step CoDA (2S-CoDA) and CoDA with cointegrating vector error correcting model (VECM-CoDA). Finally, all three CoDA models are compared with the Lee-Carter model (LC) as a standard benchmark from the literature.

2.3.1 CoDA model with common time trend

The CT-CoDA model was suggested by Oeppen (2008) for forecasting cause–specific mortality in Japan. The CT-CoDA model uses life table deaths from a multiple decrement life table to forecast mortality and recognises the compositional nature of life table deaths over cause and age where,

$$\sum_i \sum_x d_{x,i} = 1, \tag{5}$$

when the radix of the life table is equal to one. The model makes use of equation (5) by stacking cause–specific death matrices horizontally forming a $T \times NK$ matrix and calculates the clr of each row to map the life table deaths onto the real space. Next, data are centred by subtracting the geometric mean for each year and a singular value decomposition (SVD) is used to estimate the model parameters. The model can be written as,

$$clr(d_{t,x,i} \ominus \alpha_{x,i}) = \beta^1_{x,i} k^1_t + \beta^2_{x,i} k^2_t + ... + \beta^p_{x,i} k^p_t + \epsilon_{t,x,i}, \tag{6}$$

where $\alpha_{x,i}$ is the geometric mean, k^p_t measure changes over time, $\beta^p_{x,i}$ age specific changes, p is the number of extracted components from the SVD, and $\epsilon_{t,x,i}$ is the time, age, and group specific iid. error term. Note that the SVD is constructed so that the first component explains most of the total variation in the data, the second component the second most, etc.

The k_t^p parameter vectors are the only time dependent parameters and constructed to be orthogonal to each other. Thus, forecasts can be calculated by forecasting k_t^p using autoregressive integrated moving average (ARIMA) models (Box and Jenkins, 1970). Forecasts of k_t^p are used in the model and the result is transformed back using the inverse clr procedure and the geometric mean $\alpha_{x,i}$ is added. That is,

$$\hat{d}_{t,x,i} = clr^{-1}\left(\beta_{x,i}^1 \hat{k}_t^1 + \beta_{x,i}^2 \hat{k}_t^2 + ... + \beta_{x,i}^p \hat{k}_t^p\right) \oplus \alpha_{x,i}, \tag{7}$$

where the hat indicates forecast values.

The basic dynamics in the model can most easily be understood by considering the first component alone so that the total variation is decomposed using only the first rank component. Higher order components can be interpreted in a similar way. With one component the CT-CoDA model reduces to,

$$clr(d_{t,x,i} \ominus \alpha_{x,i}) = \beta_{x,i} k_t + \epsilon_{t,x,i}. \tag{8}$$

As the summation constraint, equation (5), is maintained by the clr transformation, deaths are redistributed in the model by $\beta_{x,i}$ when mortality is changing over time. This means that, if some deaths do not occur at a specific age and cause, they will shift to a different age and/or cause group. These deaths are not redistributed randomly but towards the ages and causes where they are most likely to occur according to the parameter estimates in the model. Here, $\beta_{x,i}$ estimates below zero indicate that deaths are reallocated from these ages and causes to ages and causes with a positive $\beta_{x,i}$ when k_t is increasing (Oeppen, 2008; Bergeron-Boucher et al., 2017). Thus, the net effects of competing risks between causes of death are modelled explicitly in the CT-CoDA model. The redistributing effects are one of the main advantages of using the death distribution instead of deaths rates for modelling cause specific mortality. Redistribution is the result of compositional data analysis's ability handle a covariance structure where changes over time in one of the elements in the distribution of deaths must be offset by changes in other elements (Bergeron-Boucher et al., 2017).

2.4 2-step CoDA

The 2S-CoDA model introduces two new elements compared to the CT-CoDA model in order to improve the fit and forecast of the model: 1) Age, cause, and time weights are imposed and 2) cause–specific information is added to a common trend forecast determined by the CT-CoDA model. These two elements also diversifies the 2S-CoDA model from the model presented in Bergeron-Boucher et al. (2017) together with the modelling of multi decrement life tables instead of single decrement life tables as in Bergeron-Boucher et al. (2017). The CT-CoDA model centres the observed life table deaths by perturbating its geometric mean. The importance of each cause of death, i.e. number of deaths, is therefore not relevant when estimating $\beta_{x,i}$ and k_t. A less common cause like infectious diseases is thus weighted equally with a large cause like cancer when determining the common trend k_t^1. The size of the cause of death is potentially important when forecasting due to competing risks and thus the 2S-CoDA model introduces a weighting scheme where age and cause–specific weights are imposed according to the average number of deaths for each age and cause. Further, equal weighting of the causes means that results can depend on how causes are aggregated. For example, if cancer is split into whether or not the deaths are related to smoking, the SVD would effectively give twice the weight to overall cancer compared to the situation where cancer is treated as a single cause. Hence, the 2S-CoDA model neutralises some of the undesired consequences of aggregation.

Further, $\beta_{x,i}$ is determined in the CT-CoDA model by an equal weighting of each year and assumed to be constant over time. As there has been a considerable change in the relative sizes of the different causes in recent years this assumption is likely to fail. Further, improvements in mortality have shifted from the young ages towards improvements at increasingly higher ages (Rau et al., 2008; Bergeron-Boucher et al., 2015). Estimation of $\beta_{x,i}$ where recent years are given more weight may thus produce more accurate forecasts. Hyndman et al. (2013) suggested a declining weighting scheme where the highest weight is placed on the most recent year and thereafter reduced exponentially. A similar approach is introduced in the 2S-CoDA model meaning that both the year and age dimensions are weighted. A comparison between CT-CoDA forecast and 2S-CoDA forecast will determine how useful the described weighting scheme is for forecasting.

The 2S-CoDA model follows the same first steps as the CT-CoDA model but using only a first rank approximation as higher rank approximations are modelled by cause specific terms. That is,

$$clr(d_{t,x,i} \ominus \alpha_{x,i}) = \beta_{x,i} k_t + \epsilon_{t,x,i}. \tag{9}$$

Generalized singular value decomposition (GSVD) is used to estimate the model parameters, imposing weights on the age, cause, and time dimensions. GSVD is a generalization of the SVD which imposes constraints on a rectangular matrix so that a weighting of rows and columns is possible (Loan, 1976; Abdi, 2011). The 2S-CoDA model uses age and cause–specific weights according to the average relative size of each age and cause combination. That is,

$$w_{x,i}^{age} = \frac{\bar{d}_{x,i}}{\sum_{x=1}^{\omega} \sum_{i=1}^{K} \bar{d}_{x,i}}, \tag{10}$$

where, $\bar{d}_{x,i}$ is the temporal mean of the life table deaths.

We adopt the approach described in Hyndman et al. (2013) and impose the following weights on the time dimension when estimating $\beta_{x,i}$ and k_t. That is,

$$w_t^{time} = \rho \cdot (1-\rho)^{(T-t)}, \tag{11}$$

where, $t \in (1, 2, ..., T-1)$ and ρ determines the percentage weight on the recent year, so that $\rho = 0.05$ implies that the last year is weighted with 5%.

The common trend assumption in the CT-CoDA model implies that only the variation shared between the causes is used in the fit and forecast of the model. In the 2S-CoDA this component is approximated by $\beta_{x,i} k_t$ and denoted the joint component. Cause–specific variation is thereby contained in the residuals and is potentially important when forecasting. In order to improve the fit and forecast we suggest decomposing the cause–specific residuals obtained from estimating equation (9) using SVD and the individual information when forecasting. The 2S-CoDA model follows the underlying idea in Lock et al. (2013) by estimating a joint and individual component but differs in the estimation procedure. Lock et al. (2013) use an iterative process for the

estimation of both joint and individual components whereas the 2S-CoDA model uses a more simpler approach without an iterative process. Further, the 2S-CoDA model uses weights based on the number of age-specific deaths whereas Lock et al. (2013) suggest to apply weights based on standardized variation of the data.

The model can be written as,

$$clr(d_{t,x,i} \ominus \alpha_{x,i}) = \beta^J_{x,i} k^J_t + \beta^I_{x,i} k^I_{t,i} + \epsilon_{t,x,i}, \tag{12}$$

where J and I denote the joint and individual cause components of the model, respectively and $\epsilon_{t,x,i}$ an iid. error term.

2.5 CoDA model with co–integrating vector error correction model

The VECM-CoDA model introduces one new element in comparison with the CT-CoDA model by estimating more than one time trend for each rank approximation. The CT-CoDA model implicitly assumes that different causes share one time trend forming stationary relationships. Equivalently, this can be expressed as cointegrating relationships for all the causes. This assumption might be acceptable for some causes but does not necessarily hold for all causes. Furthermore, the assumption may be appropriate for some populations but not for others. The forecast implication of the common trend assumption will be analysed by comparing forecasts from the CT-CoDA model with forecasts from the VECM-CoDA model where multiple stochastic time trends are allowed, in which case the modelling of mortality trends follows the underlying idea in Arnold-Gaille and Sherris (2013).

The VECM-CoDA model transforms horizontally stacked life table death matrices using the clr operator and centres the data by perturbation with the geometrical mean, similar to the CT-CoDA model. But instead of applying SVD on the stacked $T \times NK$, the VECM-CoDA model decomposes each centred cause–specific matrix using SVD. Thus, K $k_{t,i}$ time trends and $\beta_{x,i}$ age specific responses are estimated. That is,

$$clr(d_{t,x,i} \ominus \alpha_{x,i}) = \beta^1_{x,i} k^1_{t,i} + ... + \beta^p_{x,i} k^p_{t,i} + \epsilon_{t,x,i}, \tag{13}$$

where $k^p_{i,t}$ is cause specific and $\epsilon_{t,x,i}$ an iid. error term. The different $k_{t,i}$ time trends might be dependent i.e. some causes might be sharing trends. The Johansen trace test, described in the supplementary material section A, provides a statistical test of stationary and non–stationary relationships among time series i.e. whether some $k^1_{i,t}$'s follow the same time trends. The number of stationary relations among $k^1_{i,t}$ is found (denoted r) with the Johansen test and thereby also the number of trends (denoted $n = (K - r)$) that drive the $k^1_{i,t}$ system. Having determined stationary relations in $k^1_{i,t}$, a cointegrated VECM model is used when forecasting $k^1_{i,t}$. Dependence among the causes is thereby taken into account when forecasting cause of death. The cointegrated VECM model can be written as,

$$\boldsymbol{\Delta k_t = \Pi k_{t-1} + \sum_{j=1} \Gamma_j \Delta x_{t-j} + B + \epsilon_t,} \tag{14}$$

where $\mathbf{k_t} = (k^1_{1,t}, \ldots, k^1_{K,t})'$, $\mathbf{\Pi}$ is a matrix with rank r determining the stationary relations among $k^1_{i,t}$, $\mathbf{\Gamma_j}$ a matrix measuring the autoregressive part of the system, and $\mathbf{B}$ a $(k \times 1)$ vector specifying deterministics in the model, further details are in appendix A. We specify a deterministic trend in the non–stationary part of the model meaning that the variables are trending: this is similar to a drift term in the ARIMA specification used for the other models. Equation (14) is estimated and used to calculate forecasts of $k_{i,t}$ which are multiplied by $\beta_{x,i}$ and used to calculate forecasts of causes of death.

2.6 Lee–Carter model

Forecast results from the CoDA models will be compared with the LC model, which is one of the most–used mortality forecasting models. The LC model was suggested by Lee and Carter (1992) for forecasting all–cause mortality, but also used to forecast cause–specific mortality (Peltonen and Asplund, 1996). The LC model centres age–specific deaths rates by subtracting the age-specific arithmetic time mean and decomposes the result using SVD. That is,

$$m_{t,x,i} = \alpha_{x,i} + k_{t,i}\beta_{x,i} + \epsilon_{t,x,i}, \tag{15}$$

where $\alpha_{x,i}$ is the arithmetic mean, $k_{t,i}$ is a cause–specific index describing the general pattern of mortality, $\beta_{x,i}$ is age- and cause–specific response to changes in $k_{t,i}$ and $\epsilon_{t,x,i}$ is an iid. error term. The LC model forecasts mortality rates and life table deaths are calculated from these when comparing with the CoDA models. Standard life table calculations are used following Preston et al. (2001). We do not include a jump-off correction in the LC model as argued by Lee and Miller (2001) as it was shown to introduce a forecast bias (Booth et al., 2006). A jump-off correction might be useful for short term mortality forecasts but as we focus on long forecasts of 20 years we want to avoid introducing a forecast bias.

2.7 Comparing model forecasting performance

The models' ability to forecast future cancer deaths are evaluated by fitting the models to a subset of the data and forecasting the remaining years. Observed and forecast cancer life table deaths are compared by averaging the root-mean-square error (RMSE) over age. That is,

$$aRMSE^{d_{x,t}} = \frac{1}{N}\sum_{x=1}^{X}\sqrt{\frac{\sum_{h=1}^{M}(\hat{d}_{h,x,cancer} - d_{h,x,cancer})^2}{M}} \tag{16}$$

where a denotes an average over age, h the forecast year, and M the total number of forecast years.

The aRMSE [1] is calculated for different forecast periods, rolling the origin of the forecasts from 10 to 20 years. For example when using the French data, a 20 years forecast is calculated by fitting the models to the years 1955 to 1988. Next, a 19 years forecast is calculated by rolling the origin of the forecast one year so data from 1955 to 1989 are included in the fitting period. This procedure is continued until a 10 years forecast is calculated.

The average forecast error over the different forecasts is calculated and used for comparing the models. Thus, the impact of particular years used for forecasting is reduced as different forecast periods are considered. By using the RMSE, ages with a large number of life table deaths are implicitly weighted more than ages with few life table deaths. We do this as the main objective is to predict the total number of cancer deaths most accurately.

[1] This statistics is used to give an idea about forecast comparisons of different models. Asymptotically, the test statistic follows a normal distribution for large $d_{h,x,cancer}$ following the arguments in Czado et al. (2009)

As the LC model is formulated in death rates we also evaluate the models with the RMSE measured on death rates. That is,

$$aRMSE^{m_{x,t}} = \frac{1}{N}\sum_{x=1}^{X}\sqrt{\frac{\sum_{h=1}^{M}(\hat{m}_{h,x,cancer} - m_{h,x,cancer})^2}{M}} \tag{17}$$

2.8 Other specifications in the models

The time–weighting parameter, in the 2S-CoDA model, is set to $\rho = 0.1$ throughout the paper as determined by cross validation of a 15 years out–of–sample window across the four populations. A rank three approximation is used for the CT-CoDA model and a rank two approximation for the VECM-CoDA model as adding higher ranks only changes the fit and forecast negligibly. The specific ARIMA models are selected using the Augmented Dickey-Fuller test (Dickey and Fuller, 1979) to determine the order of integration and the number of AR and MA terms selected based on the Akaike Information Criterion (Akaike, 1974). For all rank 1 k_t terms, a deterministic drift term is included to account for upward and downward slopes.

3 Results

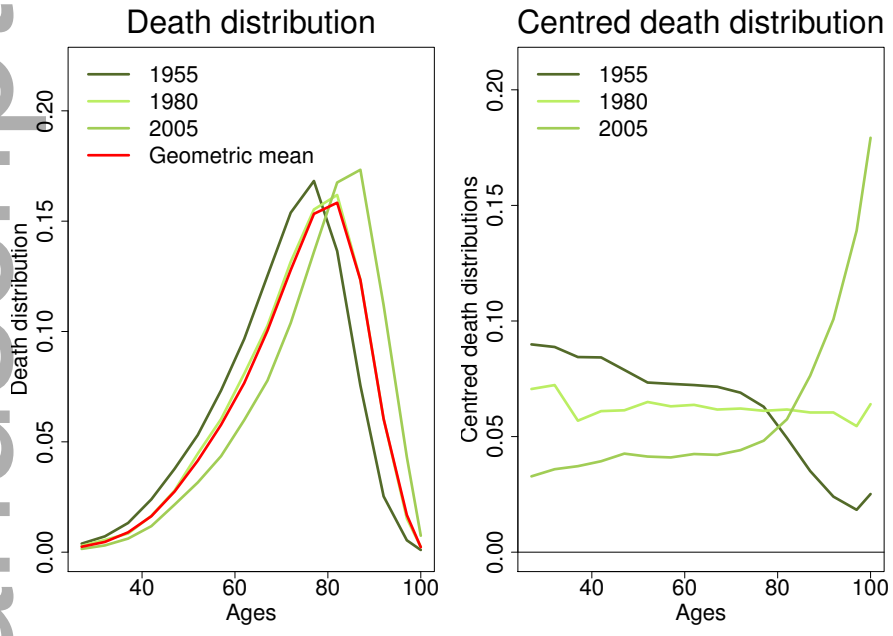
Fits of the four models are illustrated for French females and Dutch males and results of the forecast performance of the models are shown for all populations. Remaining results for French males and Dutch females are shown in supplementary material Figures D3 and D11.

3.1 Distributions of cancer deaths

The cancer deaths distribution has both shifted and been compressed over the data period meaning that cancer deaths are occurring at higher and higher ages (Figure 1) for French females (results for Dutch males in supplementary material Figure D4). The geometric mean is the geometric average over time and close to the distribution in 1980. The log–transformed and centred life table life table deaths are positive in all years and ages which means that deaths are relatively transferred towards cancer throughout the data period (Figure 1). The centred distribution in 2005 is increasing at older ages meaning that deaths for these ages especially are

increasing. The CoDA models fit the changes in the centred deaths distributions for all causes when modelling cause–specific mortality.

Figure 1: Distribution and centred distribution of cancer life table deaths for French females in selected years

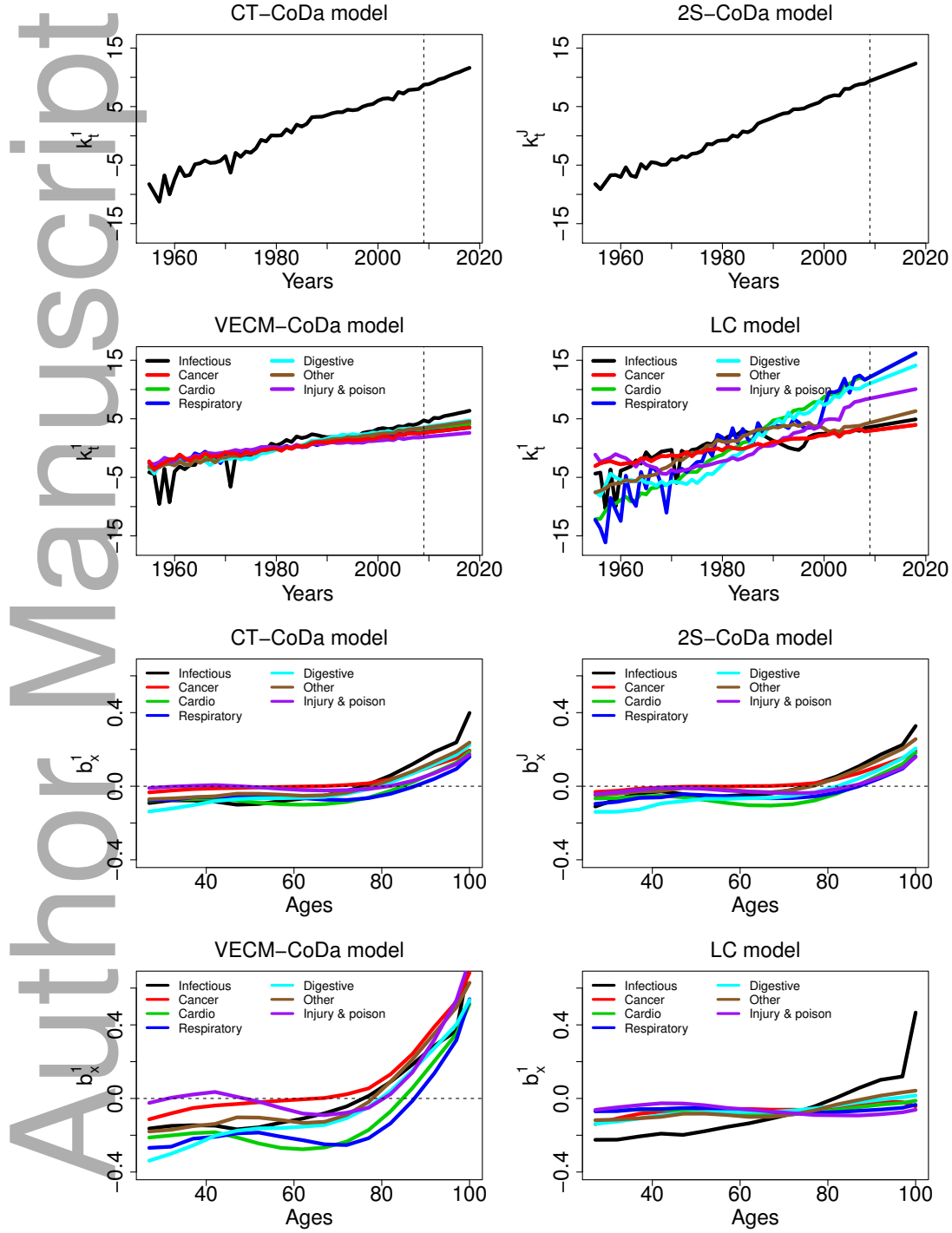


3.2 Parameter estimates

To illustrate the four different models we show the parameters estimated using data for French females. For k_t^1 a 20 years forecast is shown together with the model estimates. Parameter estimates for the first rank are shown in this section and higher orders are presented in the supplementary material Figures D5 and D6. The negative estimates of the LC model are plotted to be comparable with the CoDA models.

The time pattern of mortality captured by k_t^1 in Figure 2, shows an increasing pattern for the three CoDA models measuring that across all causes of deaths there has been a decline in mortality. Comparing k_t^1 from the CT-CoDA model and the 2S-CoDA model shows that weighting the causes by size reduces the fluctuations in k_t^1 making it more linear and potentially easier to forecast. From the VECM-CoDA model it is clear that infectious disease contributes significantly to this fluctuating pattern in k_t^1 for the CT-CoDA model, as it is the only cause

Figure 2: k_t^1 (measuring the general development in mortality) and β_x^1 (measuring the redistribution of deaths) estimates for French females from the CT-CoDa, 2S-CoDa, VECM-CoDa and LC models for the years 1955 - 2009



Note: The negative estimates are plotted for the LC model to be comparable with the CoDa models. For the 2S-CoDA model the joint components are plotted i.e. k_t^J and β_t^J

with three major from 1955 to approximately 1972. The $k^1_{t,i}$ estimates in the VECM-CoDA model also indicate that causes do not follow the same time trend through the fitting period. In particular infectious diseases and injury & poisoning follow separate patterns: this is tested with the Johansen trace test (Johansen, 1991) and results are shown in the supplementary material. The LC model's parameters are not directly comparable with the CoDA models as it uses death rates as input. An increase in the negative of k_t in the LC model implies that mortality is declining and thus the LC also estimates a decline in mortality for all the causes. The LC model and the VECM-CoDA model show diverging time trends but with different patterns.

The estimated $\beta^1_{x,i}$ parameters show similar patterns across the three CoDA models. Negative $\beta^1_{x,i}$ indicate redistribution of deaths away from these ages and causes when k_t is positive. Thus, Figure 2 shows that deaths are transferred from younger ages towards older ages, and away from cardio-vascular and respiratory diseases when time progresses, as the lowest estimates are found for these causes. The LC parameter estimates are again not directly comparable to the CoDA models as it uses death rates and causes which are not related in the model. A positive $\beta_{x,i}$, implies that mortality declines for this age group, but an automatic redistribution between causes cannot be claimed because the deaths rates are not closed by a closure operator as in the CoDA models. The negative $\beta_{x,i}$ estimates for the LC model for infectious diseases are increasing by age with large positive values for high ages meaning that the LC finds an increasing number of deaths for these ages groups when considering infectious diseases only.

The time, age, and cause weights used for the 2S-CoDA model are shown in Figure D1 in the supplementary material. As most French females die from cardio-vascular disease and cancer at an age of around 80 years, these causes and ages are given the highest weights.

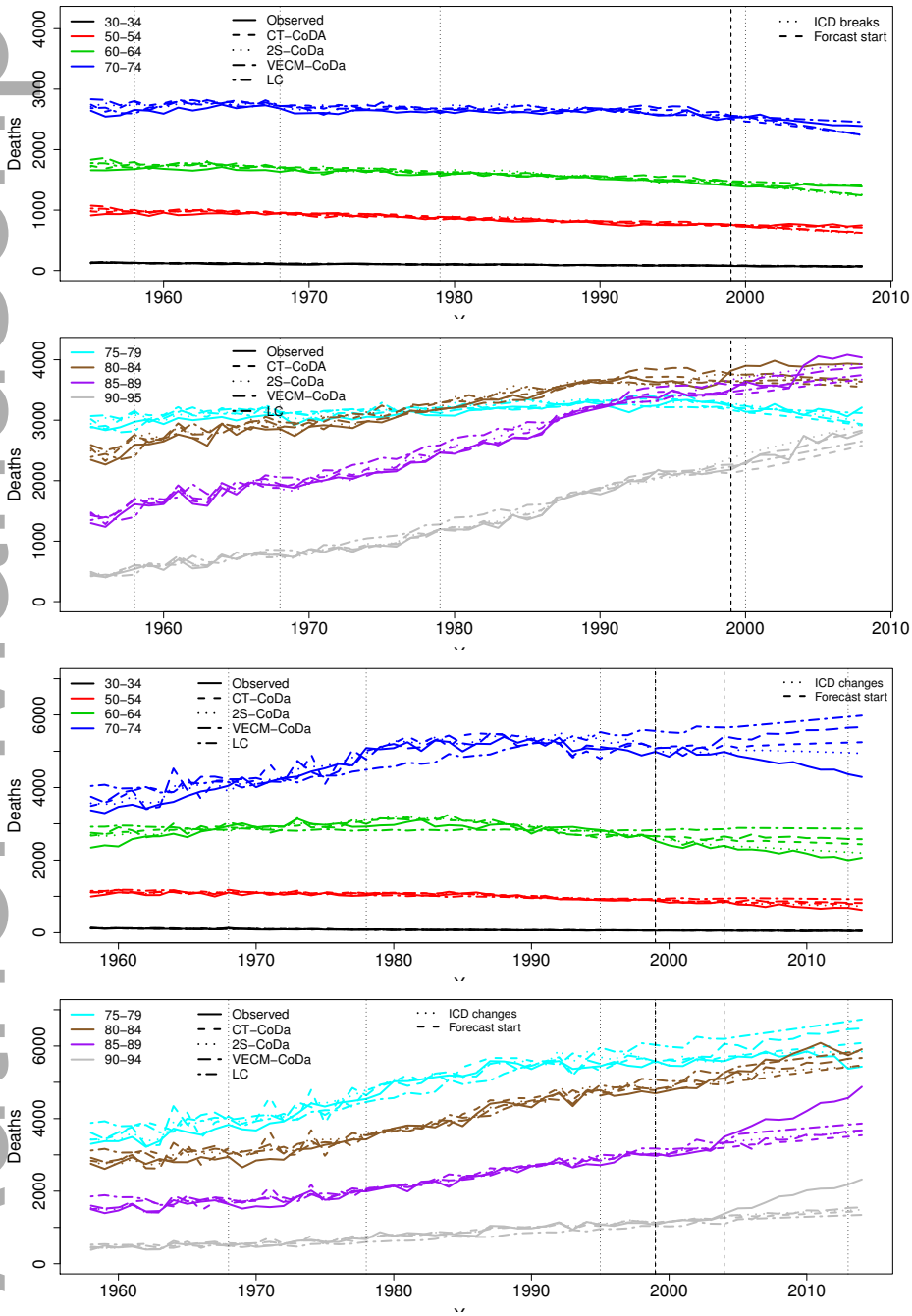
3.3 Model fit in- and out-of-sample

3.3.1 In-sample fit and forecast of withheld years

The main objective of this paper is to forecast the number of cancer deaths, hence observed, fitted, and 10 out-of-sample forecasts for cancer are shown for different age groups illustrated for French females and Dutch males. Similar plots are shown for French males and Dutch

females in supplementary material Figure D3.

Figure 3: 10 year out-of-sample forecasts of cancer life table deaths across age groups for French females and Dutch males using the CT-CoDA, 2S-CoDA, VECM-CoDA, and LC models



Note: Vertical dotted lines indicate changes in the classification of causes of deaths for each country.

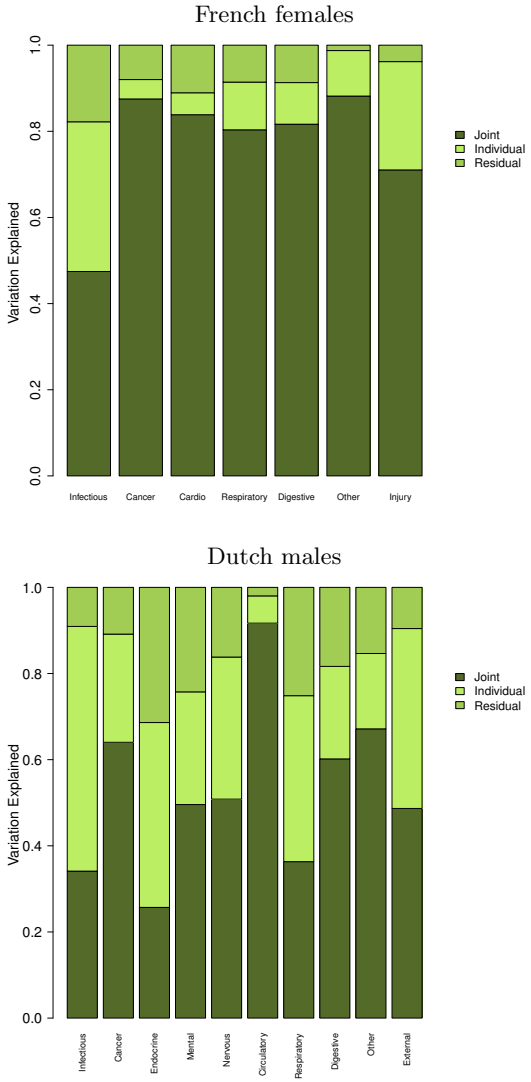
Figure 3 shows that all models produce a reasonable fit and forecast for most age groups. For French females, the older age groups 85-89 and 95-99 years old are harder to fit and forecast

compared to the younger age groups. As the 2S-CoDA model is weighted on the time dimension it produces a worse fit compared to the CT-CoDA model in the beginning of the fitting period but a better fit at the end. The VECM-CoDA model has the worst forecast of the four models whereas the LC model produces a good fit and forecast at the younger ages but largely deviates at the older age groups 80-84 and 85-89 years old. This could be a consequence of the restricted age dynamics allowed in the LC model due the constant $\beta_{x,i}$ assumption. For completeness, Figure D10 in the supplementary material shows the average RMSE over age for each year across the whole fitting period for French females and Dutch males. It is important to note that all the applied models are trend models, which cannot predict breaks and deviations from a trend. For example, with French females at age 85-89 we see that the observed number of life table deaths is increasing very rapidly around 2003 and none of the models is able to predict this break. This is a general limitation with extrapolative trend models but a successful mortality forecasting model which can predict breaks does not exist in the literature.

The fit of the 2S-CoDA model is illustrated in Figure 4 where the variation explained by the joint and individual components of the models is shown. The explained variation is calculated using the Frobenius norm (Golub and Loan, 1996).
The joint component is the first rank approximation from the GSVD, which for French females captures a high proportion of the variation for the diseases cancer, cardio-vascular, respiratory, digestive, other diseases and injury & poisoning. These diseases are therefore also well described by the CT-CoDA model. On the other hand much less variation is captured by the joint component for infectious diseases and the 2S-CoDA model improves the fit by adding an individual component. For Dutch males the joint component only exceeds 60% of the variation for cancer, circulatory diseases and other diseases, whereas a low proportion of the variation is explained for the rest of the causes. Thus the 2S-CoDA has the potential to perform much better than the CT-CoDA model as the individual component explains a large part of the variation for Dutch males. Note that the CT-CoDA model captures more variation than the joint component as higher order rank approximations are added but, in contrast to the 2S-CoDA model, these higher order terms are still joint for all causes.

Residuals for the four models are plotted in supplementary material Figure D8 and D9 for French females and Dutch males. No evidence of cohort effects are found for residuals for French females whereas some cohort effects are seen for Dutch males. Including cohort effects

Figure 4: Explained variation by the joint and individual component in the 2S-CoDA model for French females and Dutch males



could therefore potentially improve the forecasts for Dutch males, but it is beyond the scope of this paper to explore the inclusion of cohort effects.

3.3.2 Out-of-sample comparison

Cancer forecasts based on the three CoDA models and the LC model are compared using the scheme described in Section 2.7. Table 1 shows the average RMSE comparing observed and forecast life table deaths and for death rates using data from France and the Netherlands.

Table 1: 20-year out-of-sample rolling-window RMSE's for observed vs. forecast cancer life table deaths, for French and Dutch populations

Model	FRA females	FRA males	NLD females	NLD males
RMSE measured in life table deaths (equation 16)				
CT-CoDa	105.4	292.3	140.03	316.9
2S-CoDa	**90.9**	**217.2**	168.55	**259.1**
VECM-CoDa	114.6	369.4	**108.13**	391.6
LC	99.6	263.9	153.56	484.3
RMSE measured in death rates (equation 17)				
CT-CoDa	0.00076	0.00320	0.00631	0.01366
2S-CoDa	0.00070	**0.00239**	**0.00586**	**0.01349**
VECM-CoDa	0.00085	0.00570	0.00634	0.01490
LC	**0.00056**	0.00324	0.00654	0.01571

Note: Lowest RMSE forecast error is indicated with bold font.

Considering life table deaths, the 2S-CoDA model produces the lowest RMSE for both French populations and for Dutch males and the VECM-model produces the lowest RMSE for Dutch females. The results are similar when measured in death rates for the male populations but the LC model produces the lowest RMSE for French females and the 2S-CoDA model for Dutch females when death rates are used to measure mortality.

As the 2S-CoDA model is performing better than the CT-CoDA model for three out of the four populations it indicates that the 2S-CoDA can improve the forecast over the CT-CoDA model when forecasting cancer. Accommodating the limitations about equal weighting and modelling of cause–specific behaviour in the CT-CoDA model thus leads to a better fit and a better forecast performance for most of the populations we consider. The VECM-CoDA on the other hand did not improve the forecast of the CT-CoDA model indicating that allowing different $k^1_{t,i}$ trends did not improve the forecast of cancer, or that the VECM-CoDA model could not forecast these well for the selected populations. A significance test of the out-of-sample errors was carried out and results shown in the supplementary material.

3.3.3 20 years forecast

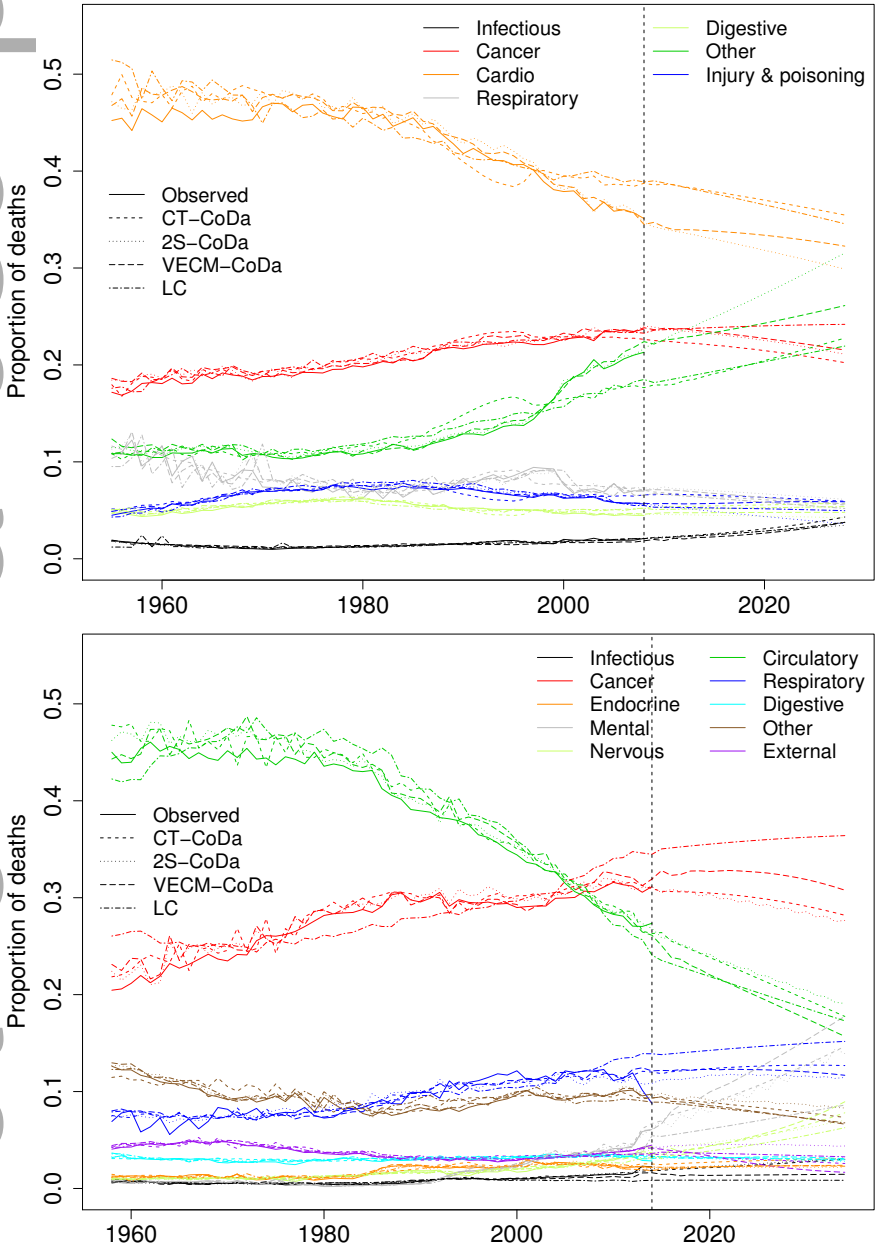
Even though the main objective of this paper is to forecast the number of cancer deaths, it is important to check how the models forecast the other causes because of dependence among the causes. Hence, the proportion of life table deaths are calculated across age for the causes and 20 year forecasts calculated for each model. The compositional analysis in the three CoDA models ensures that the proportions sum to 1 in each year, while this is not ensured in the LC model which fits each cause independently.

Figure 5 shows fitted and a 20 years forecast of proportions of life table deaths across age for French females and Dutch males, and similar plots are shown for French males and Dutch females in the supplementary material Figure D11. The improved fit for the 2S-CoDA model, compared to the CT-CoDA model, not only applies to cancer but also to the other causes: e.g. for French females the 2S-CoDA model provides a better fit for other diseases. All the models provide similar fit and forecast for the causes respiratory diseases, digestive diseases, infectious diseases and injury & poisoning, but differ for cancer, other and cardio-vascular which are the largest causes of deaths. (Table E1 and E2 in the supplementary material present the RMSE of the proportion of life table deaths for cause for French females and Dutch males, respectively). Hence, the better forecast performance of the 2S-CoDA model in terms of cancer is not only due to cancer alone but also the other causes to which it is linked through the sum constraint (see equation 5). Similar results are found for Dutch males where fit and forecast differ among the models for circulatory diseases, cancer, respiratory diseases, and mental diseases, whereas fit and forecasts for the rest of the causes are similar for all models. Note that mental diseases in the French data are aggregated in other diseases which is likely to be the cause of the predicted increase in deaths from other diseases.

4 Discussion

The analysis presented in this paper extends and improves cause–specific death forecasts using CoDA by developing a CoDA based forecasting model with the benefit of explicit modelling of dependence among cause-specific mortality, introduction of age-, period, and cause-specific weights, and modelling of both joint and cause specific variation. We find that the new 2S-

Figure 5: Proportion of life table deaths: fitted and 20 year forecasts for French females and Dutch males



Note: The vertical dashed line indicates start of forecast.

CoDA model provides more accurate forecasts of the number of cancer deaths. Compositional data analysis enables coherent modelling of cause–specific mortality where dependencies between causes are explicitly modelled, so a relative improvement in survival for one cause leads to decline in the relative survival for the remaining causes. Thus, CoDA models provide a more satisfactory modelling of cause–specific mortality than applying the LC model to each cause separately as dependence among causes is ignored in the LC model. This article focuses on forecasting cancer by improving the CT-CoDA model, suggested by Oeppen (2008), and finds that through adding age, cause, and time specific weights and by introducing a cause–specific decomposition the forecast accuracy of the CT-CoDa model can be improved. The best performing model (denoted the 2S-CoDA model) has a better forecast performance in three out of the four populations used in the analysis compared with the CT-CoDA and LC models. The 2S-CoDA model is also less sensitive to aggregation among causes of deaths as each cause is weighted by its size. The VECM-CoDA model which allows for multiple trends does not provide better forecasts compared to the CT-CoDA model despite a rejection of the CT-CoDA model's assumption about all causes being described by one common trend. Assuming one time trend for all causes thus constitutes a reasonable assumption when forecasting as a simplification of a more complicated set of different time trends. Hence, a simpler model such as the CT-CoDA model or the 2S-CoDA model performs better when compared to the more complex VECM-CoDA model. That simpler models perform better than complex models is not a new phenomenon in forecasting. For example Green and Armstrong (2015) find, in a meta study, that complex models fail to improve the forecast of simple models in 81 out of 97 comparisons.

The suggested CoDA models provide valuable information that can be used for the planning of health care and targeting of public health actions. In health care planning, the CoDA-models provide information about the future number of cancer deaths relative to the total population. Thus potentially, implied incidence rates could be calculated for cancer forecasts with information about the relative risk of cancer. The proportion of cancer deaths is expected to decline slightly for French females and Dutch females and males in 2030 when considering forecasts from the 2S-CoDA model, which was the most accurate of the models considered. Only for French males is the proportion of cancer deaths expected to increase in 2030. Hence, cancer mortality is expected to decline faster relative to the other causes for three of the four population. As the CoDA models are forecasting cause–specific mortality coherently for all

causes they also predict which causes are going to be the main causes of death in the future. For example, for Dutch males the relative number of circulatory diseases and cancer deaths are predicted to decrease whereas an increase for mental diseases is forecast. In order to improve the general survival for Dutch males it is thus necessary to consider treatments or life extending procedures for mental diseases. Our results demonstrate that causes which, today, are considered as the natural public health target because of their size might not be large in the future. As medical research takes years before better treatments are ready, research resources should be allocated so that they contribute to future survival. The suggested CoDA models have the potential to inform such public health strategies.

The CoDA models are, like the LC model, trend models which extrapolate time trends identified in the data. Thus, the models are sensitive to breaks in the data and are not necessarily able to predict new trends after a break. The 2S-CoDA model is less sensitive to new trends as recent observations are weighted more than past observations and hence new trends will be fitted better in the 2S-CoDA model. Users of the models should be careful when using them on data with many trend breaks.

None of the considered models include cohort effects to account for specific survival in some cohorts. It is very likely that cohort components could improve the fit and forecast in some populations, as has been found by Renshaw and Haberman (2006) for all–cause mortality. However, it is not straightforward to implement cohort effects due to the unique relationship between age, time, and cohort which makes it problematic to identify each component (Holford, 1983). A natural extension of cause–specific mortality forecasting using CoDA models is to include cohort components. Further, models forecasting all–cause mortality which relate a forecast for a single country to international trends have been suggested in order to provide more stable and accurate forecasts (Li and Lee, 2005; Cairns et al., 2009; Hyndman et al., 2013). It is possible that changes in the relative importance of causes are shared by multiple countries since medical interventions and health trends can be shared: for example a decline in circulatory diseases is found in both France and the Netherlands. Another possible extension of the cause–specific forecasting models is thus to include shared trends among countries.

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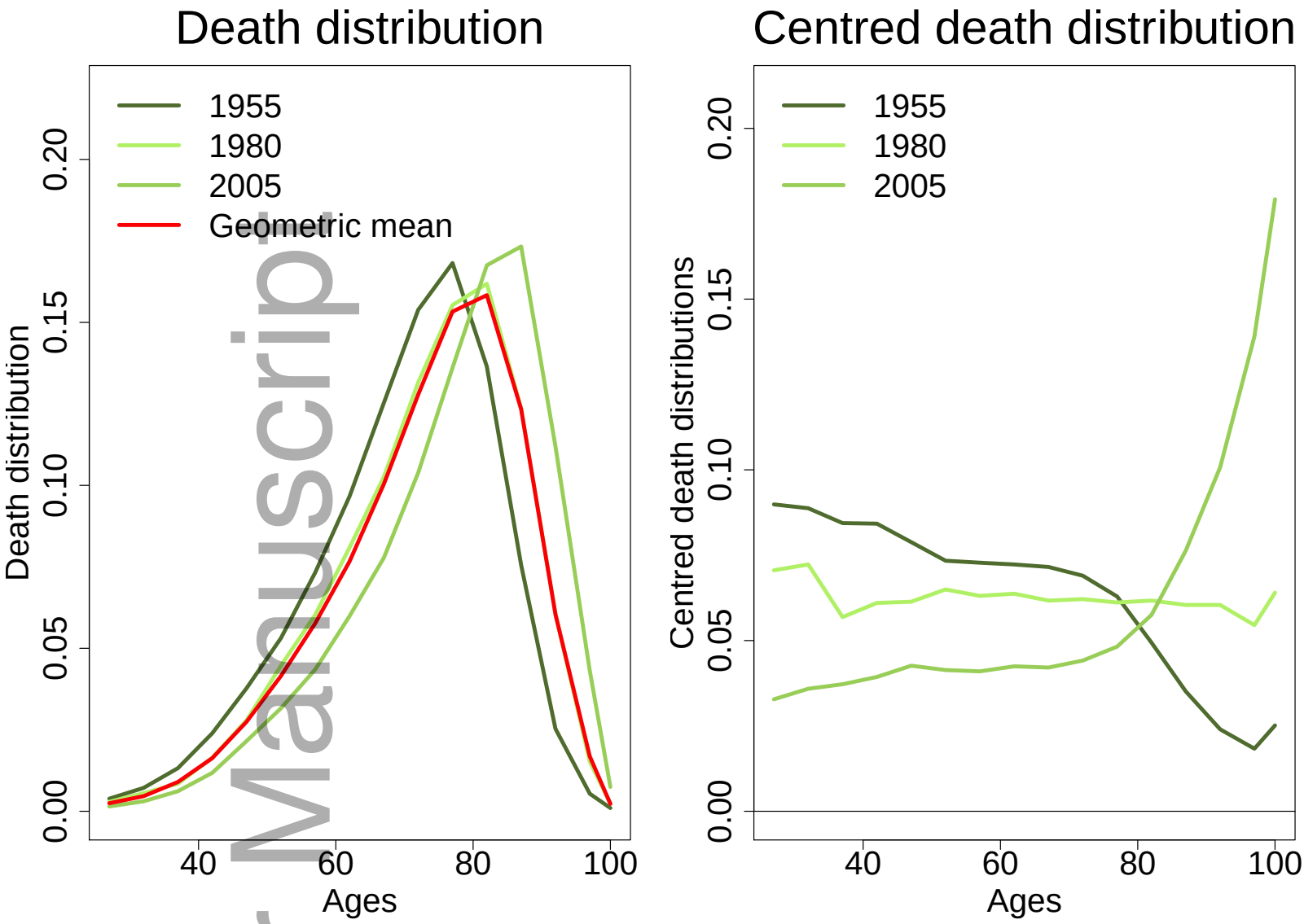

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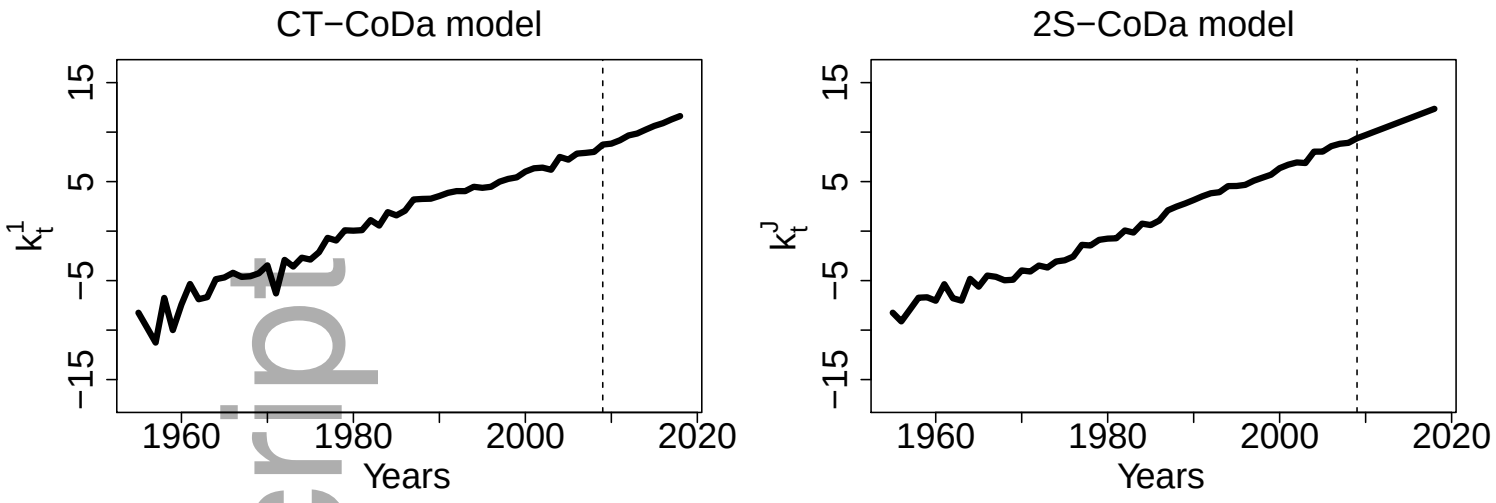

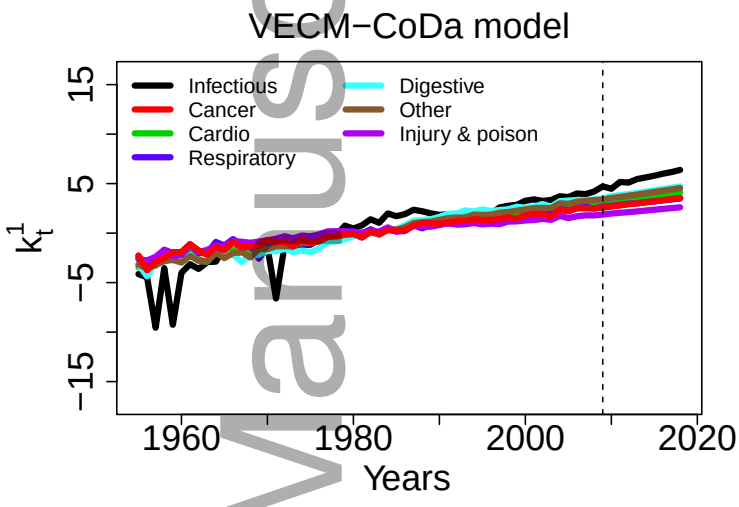

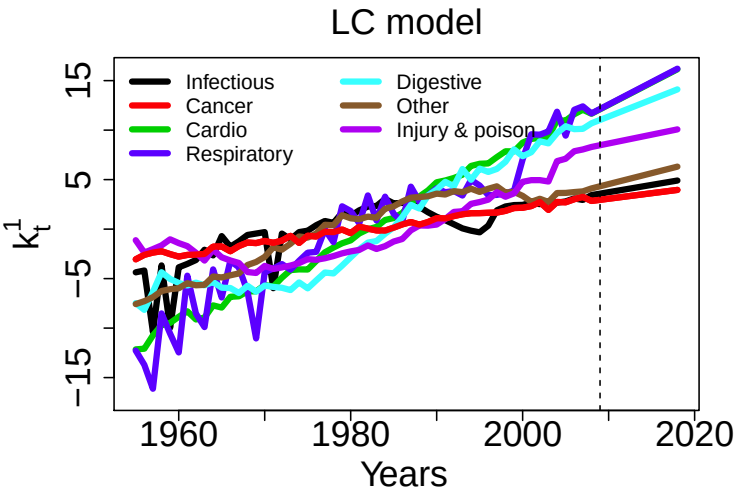

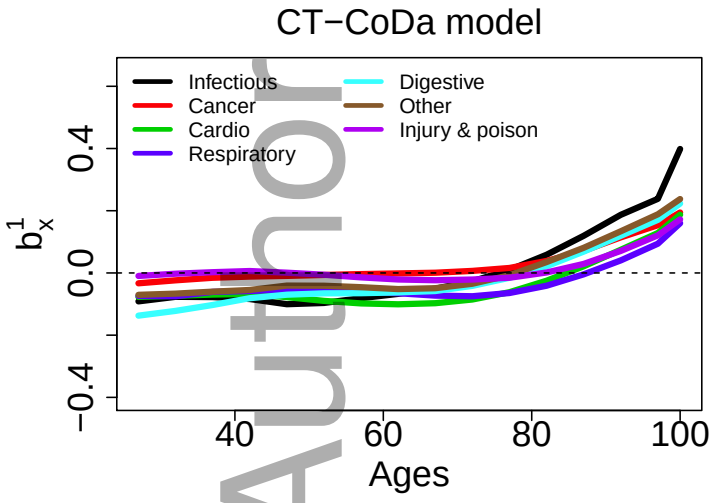

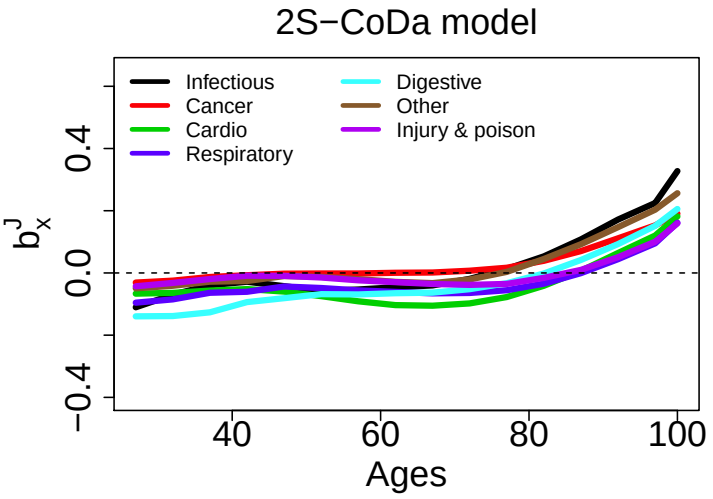

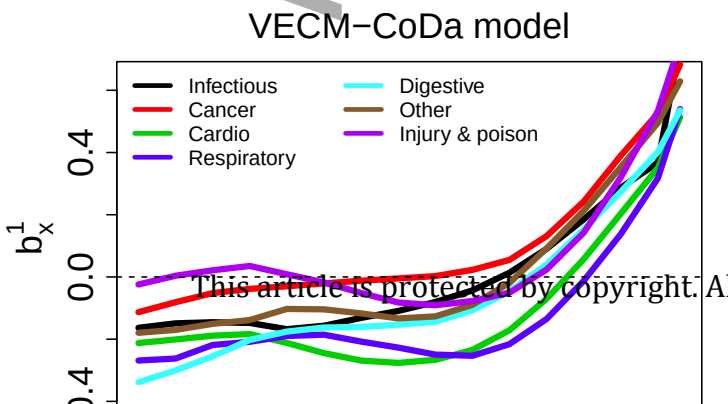

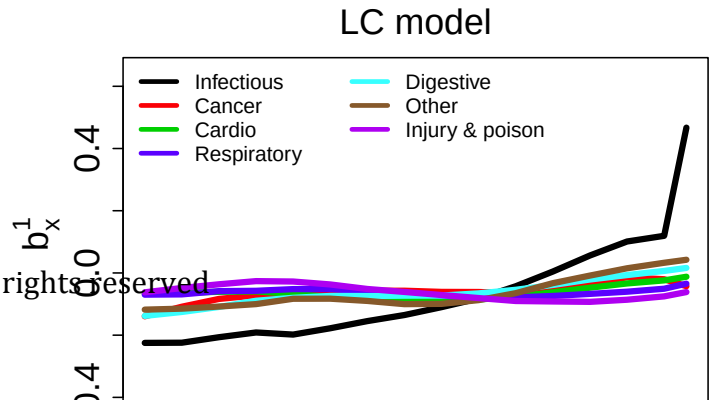

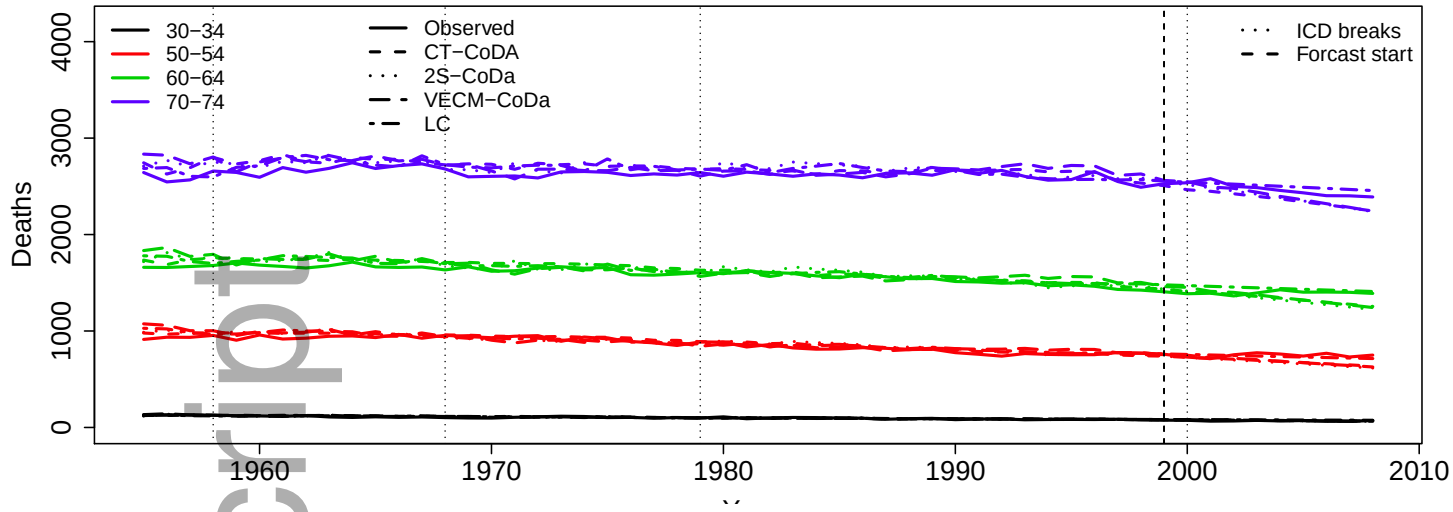

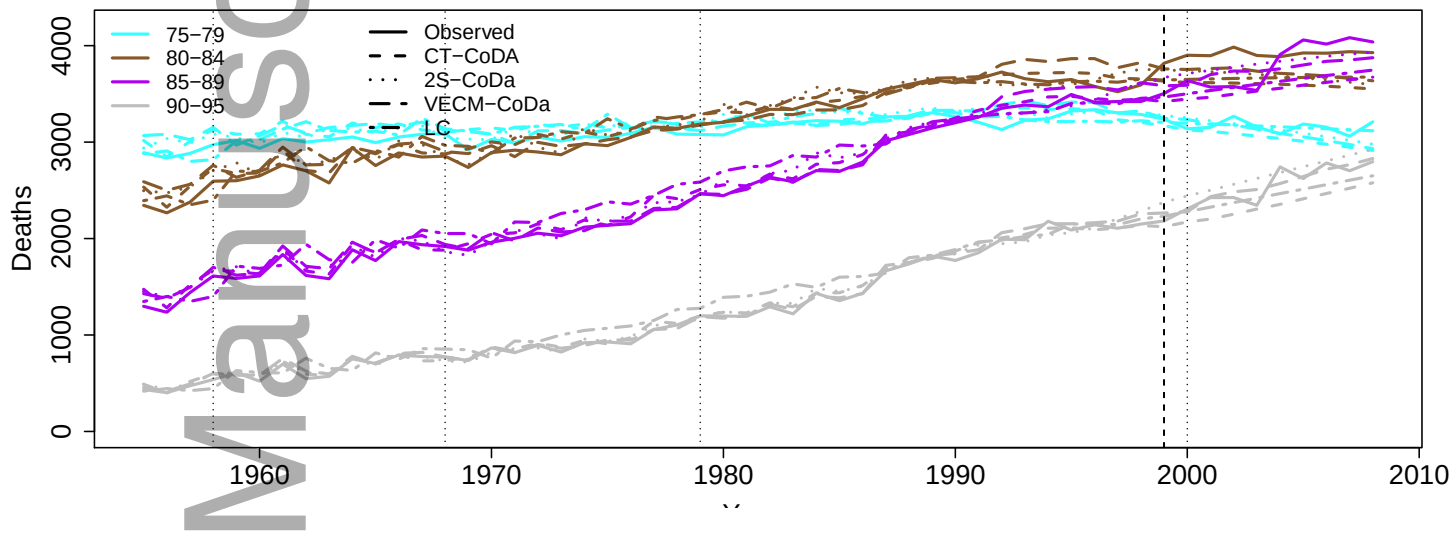

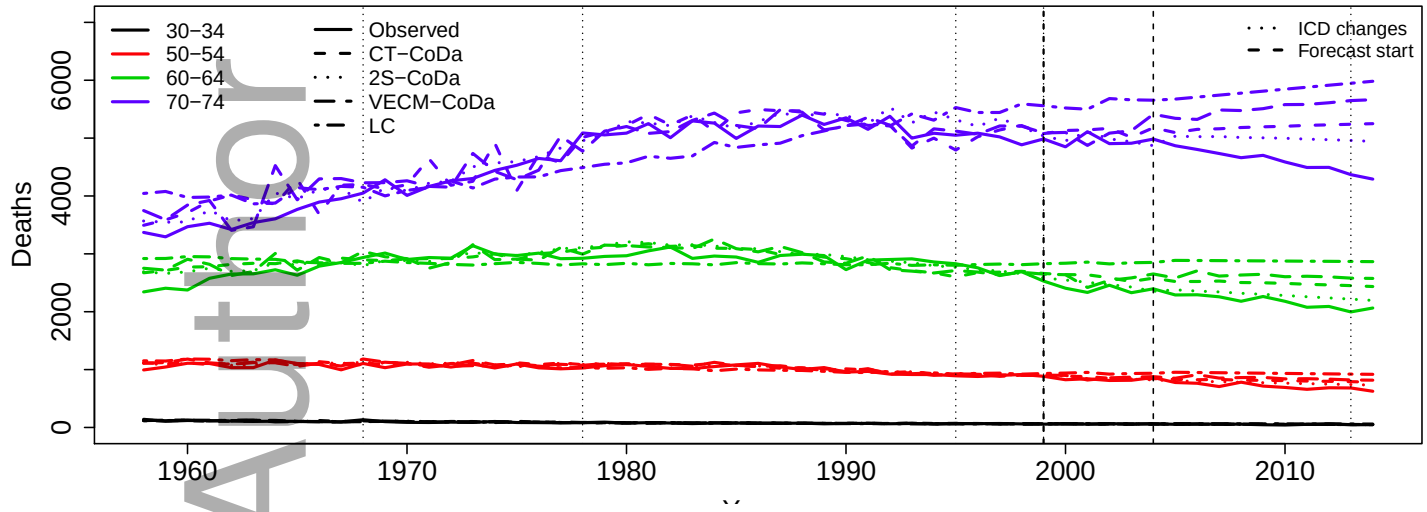

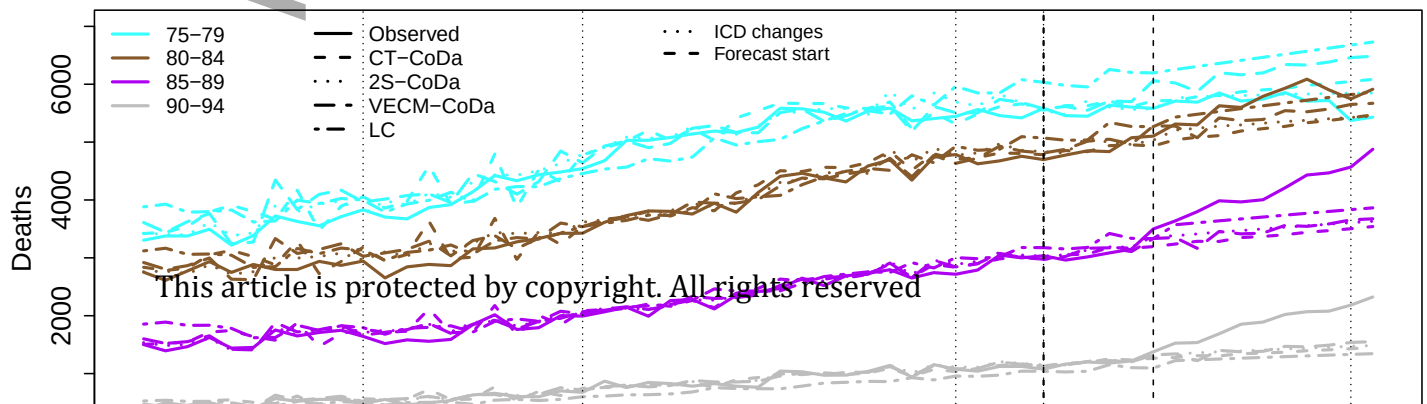

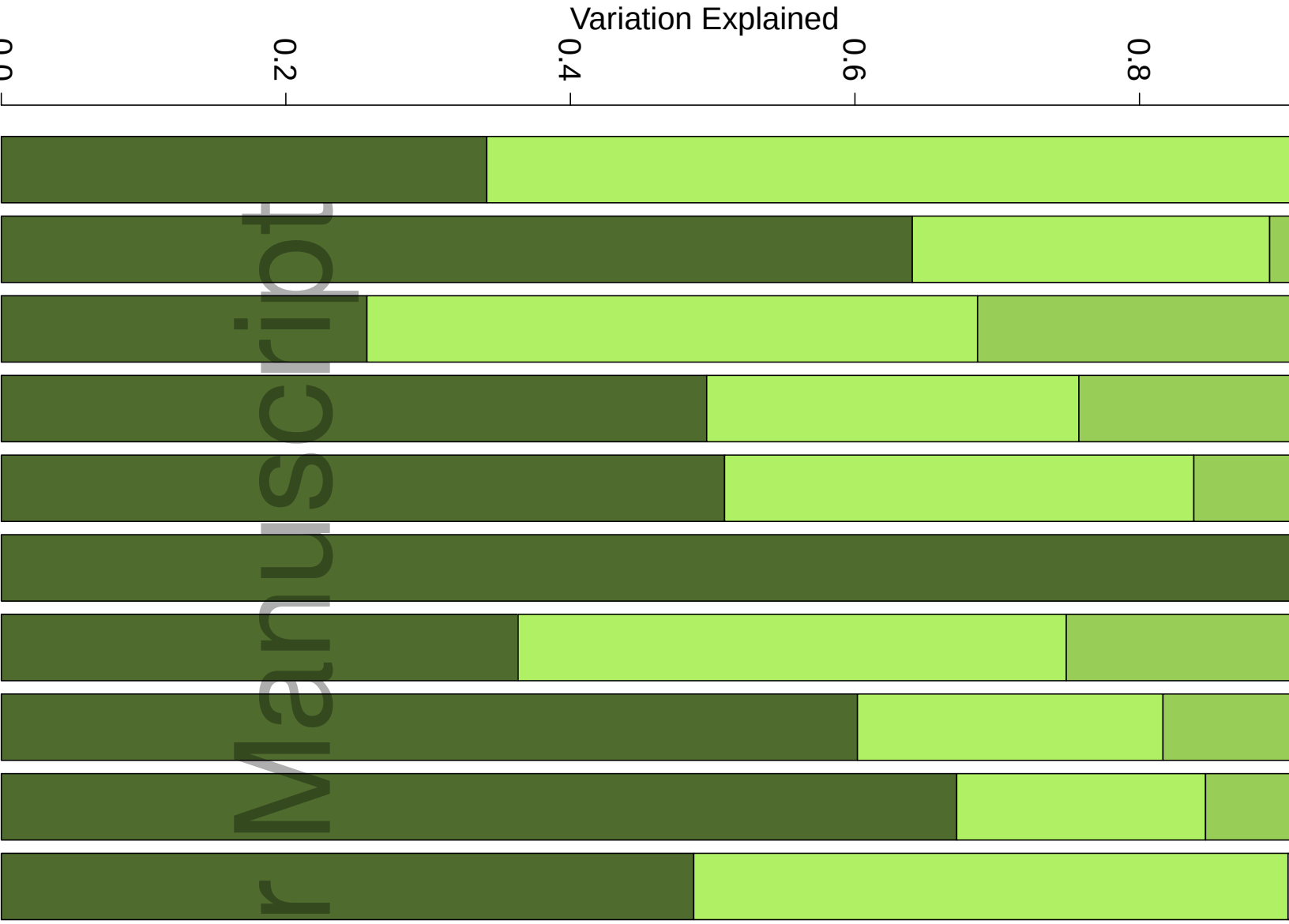

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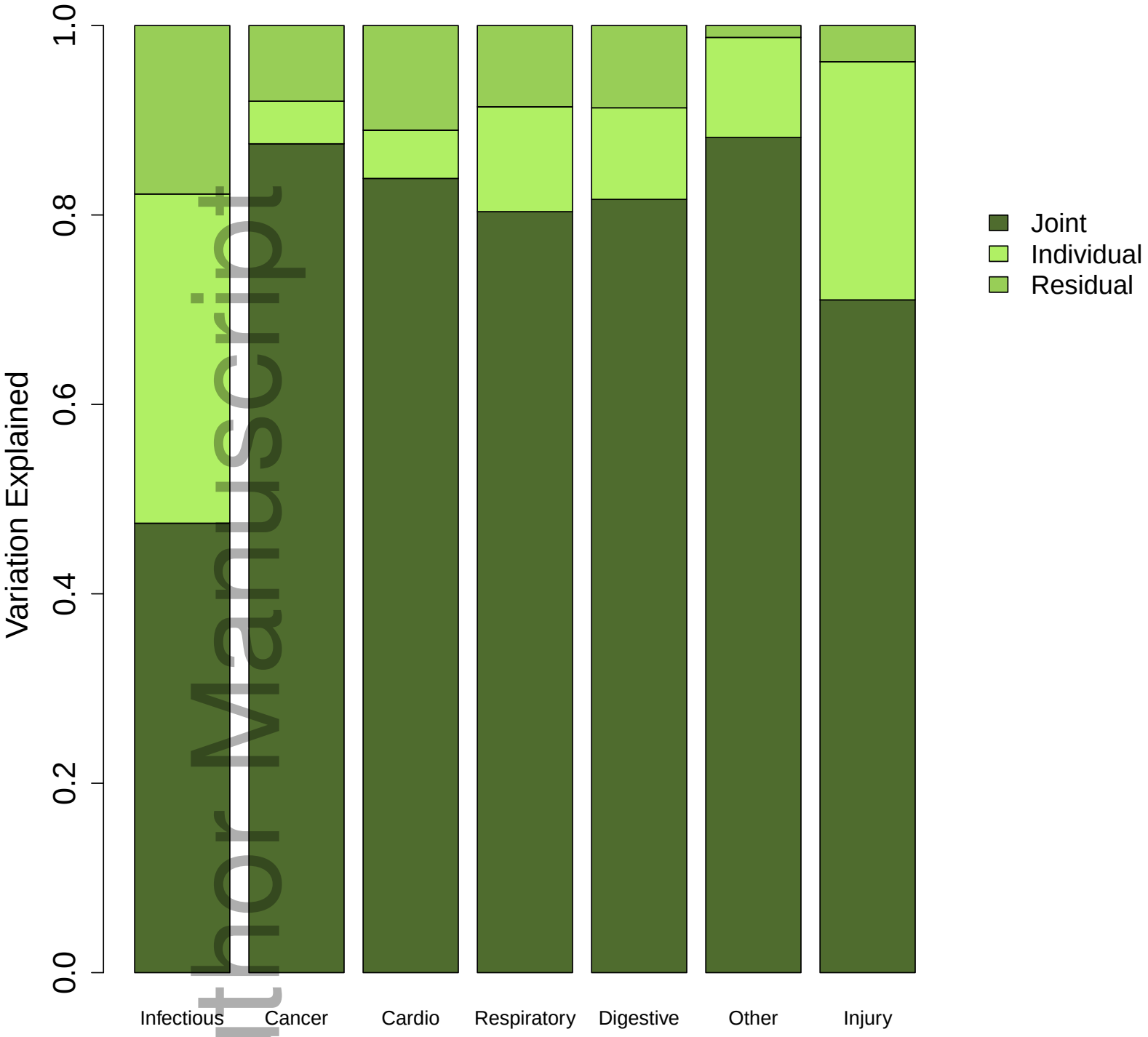

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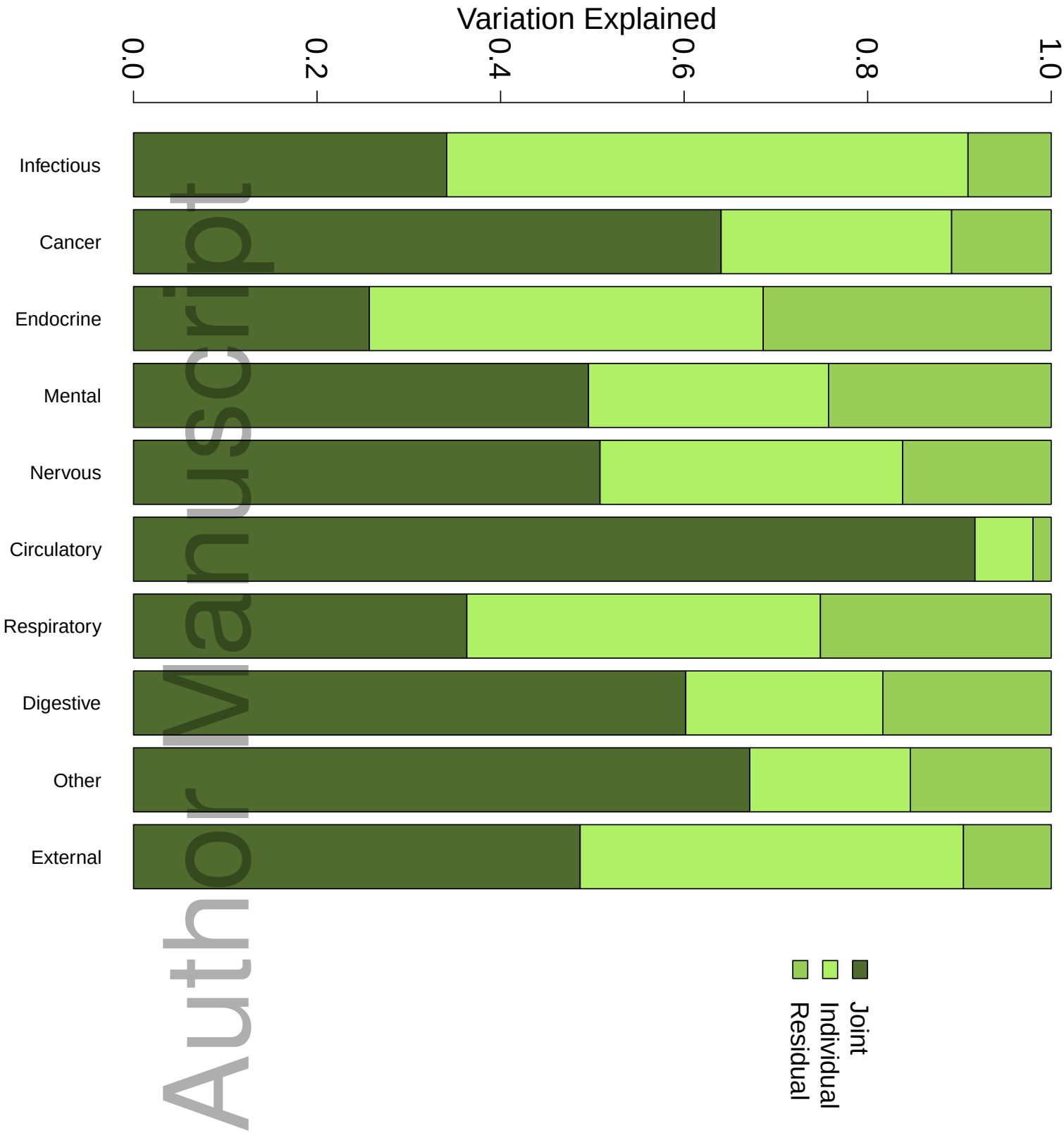

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