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Forecasting Cause-Age Specific Mortality Using Two Random Processes

Yousung PARK, Jai Won CHOI, and Hee-Young KIM

Mortality forecasts are critical information for assessing the health of a population and are necessary for making informed decisions about how best to direct health-related resources and activities. Timeliness in making health statistics available is crucial to identify and address current health problems. Being motivated to meet these needs, we propose a method to forecast the number of cause-age specific deaths through a two random processes model. Unlike the previous methods, the new method incorporates both cross-sectional and longitudinal correlations into our model without a high-dimensional problem. A bootstrap confidence interval is presented to measure the validity of our model and to detect an unusual occurrence of deaths. Our data analysis demonstrates that our method gives promising results compared with the true final counts.

KEY WORDS: Bootstrap confidence interval; Cause-specific mortality; Forecasting; Two correlations; Two random processes.

1. INTRODUCTION

Mortality data are used to create fundamental knowledge to guide the assessment, development, and evaluation of health policy. The World Health Organization (WHO) regularly forecasts cause-specific mortality to estimate morbidity, to make ongoing public health recommendations to specific countries and regions, and to use as a source of information to direct the flow of funds in the most effective way. Various government agencies study mortality trends to assess the future security of retirement, public and private insurance plans, and other public policies that depend on specific population and mortality counts. The National Center for Health Statistics (NCHS) stores the U.S. mortality data and publishes yearly cause-specific death counts for public interest. It also wants to publish long-term forecasting of mortality to evaluate the impact of health programs and interventions for major chronic conditions.

Health policy makers and public health, medical, and pharmaceutical researchers are interested primarily in cause-specific mortality. They need cause-specific information to direct appropriate treatments to population subgroups and to estimate the prevalence of some illnesses from the known relationship between cause-specific mortality and these illnesses. On the other hand, economists, sociologists, actuarial scientists, insurance companies, and public and private retirement plans are primarily interested in total number of deaths (Girosi and King 2003). To meet these two interests, the current researchers forecast total and individual cause-specific death counts separately. However, the forecasted cause-specific death counts do not add up to the total forecasted number of deaths, causing internal inconsistency.

The NCHS publishes the yearly preliminary estimates of cause-specific deaths one or more years later than the actual year (e.g., the preliminary estimates for 2002 were published in February 2004). The NCHS also publishes the final cause-specific death counts of that year still at a later time when all of the states finally complete their reports (e.g., the final deaths for 2002 were published in October 2004). Such delay causes public requests for the speedy release of data and hence the U.S. National Committee on Vital and Health Statistics recommends the timely production of health statistics as one of eight recommendations for its strategic twenty-first century vision of health statistics.

The NCHS produces yearly tabulated cause-age-sex-race–specific death counts. These tabulated death counts are cross-sectionally correlated among causes at each fixed time and are also longitudinally correlated to the previous time points. The U.K. Government Actuary's Department (2001) emphasizes that the forecasts of cause-specific deaths are unreliable when mortality forecasts are obtained from a time series model that does not address longitudinal and cross-sectional correlations. One obvious difficulty to incorporate these correlations in a time series model is that the dimension of the time series is too high to control. For example, 50 causes of deaths for each of two age groups would require modeling 100-dimensional time series in our data analysis. To avoid such a high-dimensional problem, researchers have used either each individual time series or a parameterized time series approach, but they have ignored the cross-sectional correlation (Manton, Patrick, and Stallard 1980; McNown and Rogers 1992; King and Signorino 1996; Murray and Lopez 1996).

Lee and Carter (1992) used a principal component analysis to reduce data dimensionality, then forecasted future mortality using only the first principal component, under the assumption that time trends of mortality are the same across classification. However, Lee and Miller (2001) and Girosi and King (2003) pointed out that one principal component is often not sufficient to explain the future behavior of mortality, that the assumption of the same trend across causes of death is often not true, and that there is no way to include exogenous variables (i.e., covariates) in their model for better forecasts.

In our study we overcome the shortcomings of theses methods by using two random processes to jointly model 50 cause-specific death counts. Our model not only addresses the two types of correlations, but also solves the high-dimensional problem and incorporates relevant exogenous variables. The first process is a new integer-valued time series model to forecast the total number of deaths. This process reflects the longitudinal correlation and generalizes the previous stationary integer-valued time series models (McKenzie 1986; Alzaid

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and Al-Osh 1990; McCormick and Park 1997) to a nonstationary model. The second process classifies the total count by cause-age categories using classification probabilities under the multinomial assumption and thus incorporates the cross-sectional correlation. The classification probability in the second process is specified by two hierarchical logistic link functions with relevant covariates. Then the ultimate number of cause-age specific deaths is obtained by multiplying the two results (i.e., total $\times$ classification probability). Because our method forecasts the total counts first, then distributes these total counts to cause-age–specific deaths by classification probabilities, it always preserves the internal consistency. By incorporating relevant covariates in the hierarchical logistic link functions, the main effects of covariates, as well as the interaction effects between covariates and classification variables, are incorporated into the classification probability.

Our model can forecast timely estimates of cause-specific mortality, and yet the estimates are reasonably close to the true values. In addition, our model includes a general trend of population change, as well as seasonality in forecasting future numbers of cause-age–specific deaths. These forecasts estimate the expected number of deaths if past conditions and patterns continue to hold in the future time. Thus it can be used to develop a statistical strategy for public health officials to detect an unusual occurrence of deaths. For example, if the numbers of deaths by pneumonia and chronic obstructive pulmonary disease in the winter of 1997–1998 exceed the upper bound of the 95% forecast confidence interval constructed from the forecast model based on the data before the winter of 1997–1998, then we may conclude that the forecast identifies an unusually large number of deaths for pneumonia and chronic obstructive pulmonary disease in 1997–1998, which is consistent with an influenza outbreak that was widely reported that winter (see Sec. 4). Because it is not easy to obtain the distributional properties of forecasts from the two–random processes model, we use a bootstrap method to calculate forecast confidence intervals according to the approach of Cardinal, Roy, and Lambert (1999).

Section 2 describes the NCHS mortality data used for data analysis. Section 3 discusses the two random processes and the connection of the total number of deaths to the cause-age–specific death counts. We present a new integer-valued AR model and an integer-valued regression model with autocorrelated disturbance. The conditional expectations of these two models are the same as those of continuous counterparts. We use optimal estimation equations to estimate the parameters and investigate their asymptotic behavior and describe how to construct a bootstrap confidence interval. Section 4 applies our method to the data of one decade (1989–1998) of the 49 most frequent causes of death classified by two age groups. We compare the integer-valued time series model with its continuous counterpart [i.e., the integrated autoregressive model (ARI)] and two regression models in terms of goodness of fit or coverage probability of 95% nominal confidence interval. We present 2-year out-of-sample forecasts and their bootstrap confidence intervals for 1998 using the data from 1989 to 1996 to evaluate the validity of our model. We also forecast cause-age–specific death rates per 100,000 population with bootstrap confidence intervals for 2010 and 2020. Section 5 provides some concluding remarks.

2. NATIONAL CENTER FOR HEALTH STATISTICS MORTALITY DATA

The NCHS receives monthly mortality reports from the 50 states and the District of Columbia through the Vital Statistics Cooperative Program. Some states often do not report all deaths in time for various reasons and complete their report months or even years later than the current year. Rather than waiting for the complete counts, the NCHS reports the preliminary mortality estimates through the National Vital Statistics Report (NVSR) with the data available at the time, but even these preliminary estimates are released 1 or more years after the current year.

Since 1997, through the NVSR, the NCHS has provided monthly estimates of the total number of deaths and yearly estimates of cause-age-sex-race–specific deaths using a substantial proportion of all death records for the year. These yearly estimates differ considerably from the corresponding final counts for some causes. These differences arise mainly from the delays in reporting, especially for the causes subject to further investigation (e.g., homicide) and other causes heavily affected by seasonality. The death counts for such causes are systematically overestimated or underestimated by the truncated nature of the preliminary file (Kochanek, Smith, and Anderson 2004). Our forecasts for these causes of death are compared to the NCHS preliminary estimates (see Table 5 in Sec. 4).

We use the 1989–1998 NCHS monthly mortality data, which were classified by the ninth revision of the International Classification of Diseases (ICD). Since 1999, the NCHS has used the tenth revision to code the cause of death. Differences between the two revisions cause problems in directly comparing the data before and after the revision. For example, the NCHS reported 72 causes of death when it used the ninth version, whereas it now reports 113 causes. For this work we use the data from 1989–1998, to avoid the bias introduced from this change.

Our data analysis uses only 49 leading causes of death, which include 98.8% of all deaths in 1998, and two age groups, categorized by young and old age groups based on a normal retirement age (i.e., 65 years). This particular cause-age–specific classification can be easily extended to more extensive cause-age-sex-race death counts by adding two more logistic link functions for the additional sex-race classification, as described in Section 3.3.

3. MODEL AND ESTIMATION

In this section we present two random processes for forecasting the cause-age–specific death counts. To investigate the connection between these two processes and the estimation method, we divide this section into five subsections. Section 3.1 describes the overall picture of our approach to mortality forecasting and the connection between the two processes. Section 3.2 demonstrates how to forecast the total number of deaths through the first process of the integer-valued time series model. It also presents an integer-valued regression model with autocorrelated disturbance to incorporate covariates that may improve long-term forecasts. Section 3.3 describes how to obtain the classification probability specified by two logistic link functions through the second process. Section 3.4 estimates the parameters in the model and reports their asymptotic normality for further inferences, and Section 3.5 describes how to obtain bootstrap confidence intervals.

3.1 General Description of Two Random Processes

This section relates the total death count from the first process to the cause-age–specific death count from the second process. Let D_t be the total death count, and let D_{tij} be the cause-age–specific death count belonging to the ith cause and the jth age at month t. Then D_{tij} can be written as, for $i = 1, \ldots, c$ and $j = 1, \ldots, a$,

$$D_{tij} = \sum_{m=1}^{D_t} d^c_{tm}(i) \cdot d^a_{tm}(j), \tag{1}$$

where $d^c_{tm}(i) = 1$ if the mth death at month t belongs to the ith cause, $d^a_{tm}(j) = 1$ if the mth death at month t belongs to the jth age, and $d^c_{tm}(i) = d^a_{tm}(j) = 0$ otherwise.

Denote the classification probability by $P(d^c_{tm}(i)d^a_{tm}(j) = 1) = \pi_{tij}$ and assume that

$$P\big(d^c_{tm}(i)d^a_{tm}(j) = 1, d^c_{tm'}(i')d^a_{tm'}(j') = 1\big) = \begin{cases} 0 & \text{for } m = m' \text{ and } (i,j) \neq (i',j') \\ \pi_{tij}\pi_{ti'j'} & \text{for } m \neq m'. \end{cases}$$

Then (1) implies that when the number of total deaths, D_t, is given, the cause-age–specific death counts, D_{tij}, are the multinomial random variates with classification probability π_{tij}. This incorporates the cross-correlation in (1) among cause-age–specific death counts at each time point. Because D_t is a time-correlated random process rather than a fixed number, in Section 3.2 we propose a new time series model that reflects the longitudinal correlation among the death counts at different time points.

It can be easily shown from (1) that for $k \geq 1$, the conditional expectation of the number of cause-age–specific deaths at month $t + k$ given all information up to month t is

$$E(D_{t+kij}|\mathcal{F}_t) = E(D_{t+k}|\mathcal{F}_t)\pi_{t+kij}, \tag{2}$$

where $\mathcal{F}_t$ is a sigma field generated by $\{D_{t'ij}, t' \leq t\}$.

It is known that $E(D_{t+kij}|\mathcal{F}_t)$ and $E(D_{t+k}|\mathcal{F}_t)$ are respective minimum variance k-month-ahead predictors of D_{t+kij} and D_{t+k} given $\mathcal{F}_t$. Equation (2) is expressed as

$$\hat{D}_{t+kij} = \hat{D}_{t+k} \times \pi_{t+kij}, \tag{3}$$

where $\hat{D}_{t+kij} = E(D_{t+kij}|\mathcal{F}_t)$ and $\hat{D}_{t+k} = E(D_{t+k}|\mathcal{F}_t)$. By (3), the forecast of the cause-age–specific death count is calculated by total death counts $\times$ the corresponding classification probability.

Therefore, our focus is now on how to derive $\hat{D}_{t+k}$ and π_{t+kij}. We encounter two difficulties in deriving these $\hat{D}_{t+k}$ and π_{t+kij}. One difficulty is that D_t is an integer-valued time series satisfying the random equation (1). We handle this problem in Section 3.2. The other difficulty is that there are as many π_{tij}'s as the observed counts. In Section 3.3 we show how to reduce the number of parameters to be estimated.

3.2 First Process for Total Number of Deaths

In this section we introduce a new integer-valued time series model for monthly total death D_t. This model describes the distributional characteristic better than the continuous ARMA model for count data, retaining the same conditional expectation as that of the usual ARMA model. Although the monthly total death count D_t is sufficiently large to be approximated by the ARMA model, previous studies have shown that the ARMA approximation cannot describe the true variance structure of an integer-valued time series (McKenzie 1985, 1986; Alzaid and Al-Osh 1990; Park and Oh 1997). Because the variance of D_{tij} depends on the variance of D_t by (1), the incorrect variance estimate with the usual ARMA model leads to the incorrect inferences related not only to $E(D_{t+k}|\mathcal{F}_t)$, but also to π_{t+kij}.

Before we present a new integer-valued time series model dealing with the nonstationary nature of monthly total deaths, we briefly discuss the stationary model of integer-valued AR process with order p [INAR(p)] (Alzaid and Al-Osh 1990; Jin-Guan and Yuan 1991). Let $\{\omega_{\alpha r}, r = 1, 2, \ldots\}$ be an iid sequence of Bernoulli random variables with $P[\omega_{\alpha r} = 1] = \alpha$. INAR($p$) is then defined as

$$D_t = \alpha_1 \circ D_{t-1} + \alpha_2 \circ D_{t-2} + \cdots + \alpha_p \circ D_{t-p} + \epsilon_t, \tag{4}$$

where $\alpha_q \circ D_{t-q} = \sum_{r=1}^{D_{t-q}} \omega_{\alpha_q r}$ with $P[\omega_{\alpha_q r} = 1] = \alpha_q$ for $q = 1, \ldots, p$ and for all r, the $\circ$-operation is referred to as the binomial thinning operator, and random disturbances $\{\epsilon_t\}$ are iid nonnegative integer-valued random variables. It has been shown that the correlation structure of INAR(p) behaves in the same way as the usual continuous AR(p), but the variances of the two models are different (McKenzie 1986; Jin-Guan and Yuan 1991).

Examining U.S. monthly total deaths from the NCHS final reports, we found the apparent seasonality that the monthly deaths peaked in either every January or December and bottomed out in every June, August, or September, implying that the counts are not stationary. To eliminate the seasonality from the monthly total death count D_t, we use differencing as used in the usual continuous ARMA model; that is, define $\nabla^d_s D_t \equiv (1 - B^s)^d D_t$ to be the differenced series of D_t, where d and s are nonnegative integers and B is the back-shift operator defined as $B^k D_t = D_{t-k}$. For example, $\nabla_{12} D_t = D_t - D_{t-12}$ when $d = 1$ and $s = 12$. The binomial thinning operator used in the INAR(p) given in (4) can be applied only to a nonnegative integer-valued process. Thus the differenced series (i.e., $\nabla^d_s D_t$) cannot be used in the INAR(p) model, because the differenced series can be negatively valued.

To overcome this problem, we introduce a new operator with the symbol $\odot$, which we call the "signed binomial thinning" operator. This new operator generalizes the previous binomial thinning operator to handle negatively valued difference series. Let $\{w_{\alpha tr}\}$ be the iid Bernoulli random variables at time t with the probability $P(w_{\alpha tr} = 1) = |\alpha|$, where $-1 \leq \alpha \leq 1$. Define

$$\alpha \odot \nabla^d_s D_t \equiv \operatorname{sgn}(\alpha) \operatorname{sgn}(\nabla^d_s D_t) \sum_{r=1}^{|\nabla^d_s D_t|} w_{\alpha tr}, \tag{5}$$

where $\operatorname{sgn}(x) = 1$ if $x \geq 0$ and $\operatorname{sgn}(x) = -1$ if $x < 0$. When $d = 0$, $\alpha \geq 0$, and $D_t > 0$, the signed binomial thinning is reduced to the binomial thinning because $\nabla^d_s D_t = D_t$ for $d = 0$ and $\alpha \odot D_t = \sum_{r=1}^{D_t} w_{\alpha tr} = \alpha \circ D_t$ for $D_t \geq 0$ and $\alpha \geq 0$. Thus all properties of the binomial thinning operator shown by Park and Oh (1997) are special cases of the signed binomial thinning operator.

We now define the integer-valued INARI process with order p [INARI(p)] for $\nabla_s^d D_t$ when the series of $\nabla_s^d D_t$ follows

$$\nabla_s^d D_t = \sum_{q=1}^{p} \alpha_q \odot \nabla_s^d D_{t-q} + \epsilon_t, \qquad t = 0 \pm 1, \pm 2, \ldots, \quad (6)$$

where $\{\epsilon_t\}$ is a sequence of iid integer-valued random variables with mean μ_ϵ and variance σ_ϵ^2, $0 \le |\alpha_q| \le 1$ for $q = 1, \ldots, p$, and all counting series $\{w_{\alpha_q,t,r}\}$ are mutually independent. The $\{\epsilon_t\}$'s are uncorrelated with $\nabla_s^d D_{t-q}$ for $q \ge 1$.

The INARI(p) process is developed to remove the seasonality and to handle a negative series of $\nabla_s^d D_t$. When $\nabla_s^d D_t \ge 0$ and $\alpha_q \ge 0$ for all t and $q = 1, 2, \ldots, p$, INARI(p) is reduced to the previous INAR(p) process. Thus INARI(p) extends the previous INAR(p) process.

By the same argument used by Jin-Guan and Yuan (1991), we can show that if all of the roots of the polynomial

$$\lambda^p - \alpha_1 \lambda^{p-1} - \cdots - \alpha_{p-1}\lambda - \alpha_p = 0 \quad (7)$$

are inside the unit circle, then the INARI(p) model given in (6) uniquely exists in L_2 space and is stationary. Hereafter we assume that INARI(p) satisfies the stationary condition of (7), and then the difference series $\nabla_s^d D_t$ becomes stationary even though the original series D_t is nonstationary.

Let $\hat{\alpha}_q$ $(q = 1, 2, \ldots, p)$ be the estimators of the coefficients of $\nabla_s^d D_t$ in the model (6) satisfying

$$\hat{\gamma}_k = \hat{\alpha}_1 \hat{\gamma}_{k-1} + \cdots + \hat{\alpha}_q \hat{\gamma}_{k-q} + \cdots + \hat{\alpha}_p \hat{\gamma}_{k-p}, \quad (8)$$

where $\hat{\gamma}_k = \frac{1}{n-k}\sum_{t=1}^{n-k}(\nabla_s^d D_t - \bar{\bar{D}})(\nabla_s^d D_{t-k} - \bar{\bar{D}})$ with $\hat{\gamma}_k = \hat{\gamma}_{-k}$ and $\bar{\bar{D}} = \frac{1}{n}\sum_{t=1}^{n} \nabla_s^d D_t$. Using $\hat{\alpha}_q$, define $\hat{\sigma}_\epsilon^2 = \frac{1}{n}\sum_{t=1}^{n}(\hat{\epsilon}_t - \bar{\epsilon}_n)^2 + \frac{1}{n}\sum_{t=1}^{n} |\nabla_s^d D_t| \sum_{q=1}^{p} |\hat{\alpha}_q|(1 - |\hat{\alpha}_q|)$ and $\bar{\epsilon}_n = \frac{1}{n}\sum_{t=1}^{n} \hat{\epsilon}_t$, where $\hat{\epsilon}_t = \nabla_s^d D_t - \hat{\alpha}_1 \nabla_s^d D_{t-1} - \cdots - \hat{\alpha}_p \nabla_s^d D_{t-p}$. Then we have the following lemma.

Lemma 1. Let $\sigma_\epsilon^2 = \mathrm{var}(\epsilon_t)$ and $\mu_\epsilon = E(\epsilon_t)$. Then $\hat{\alpha}_q$, $\hat{\sigma}_\epsilon^2$, and $\bar{\epsilon}_n$ are strong consistent estimators of parameters α_q, σ_ϵ^2, and μ_ϵ.

The proof of Lemma 1 and other proofs are given in the Appendix. We use the $\hat{\sigma}_\epsilon^2$ to derive other estimators, because σ_ϵ^2 is a nuisance parameter in the subsequent sections.

Lemma 1 together with (8) imply that the estimated correlations for the continuous ARI(p) and the discrete INARI(p) are the same. However, the consistent estimator $\hat{\sigma}_\epsilon^2$ indicates that the variance estimator under ARI(p) [i.e., $\frac{1}{n}\sum_{t=1}^{n}(\hat{\epsilon}_t - \bar{\epsilon}_n)^2$] is biased for σ_ϵ^2 by $n^{-1}\sum_{t=1}^{n} |\nabla_s^d D_t| \sum_{i=1}^{p} |\hat{\alpha}_i|(1 - |\hat{\alpha}_i|)$. This implies that a continuous time series approximation to INARI(p) causes incorrect inference for the α_i's.

Under the stationary INARI(p) model, the immediate results by conditional expectation technique and the signed binomial thinning operator $\odot$ are

$$\begin{aligned} E(D_{t+k}|\mathcal{F}_t) &= E(D_{t+k} - \nabla_s^d D_{t+k}|\mathcal{F}_t) \\ &\quad + \sum_{i=1}^{p} \alpha_i E(\nabla_s^d D_{t+k-i}|\mathcal{F}_t) + \mu_\epsilon, \\ &\quad k = 1, 2, \ldots, \qquad \text{and} \\ \mathrm{var}(D_t|\mathcal{F}_{t-1}) &= \sum_{i=1}^{p} |\alpha_i|(1 - |\alpha_i|)|\nabla_s^d D_{t-i}| + \sigma_\epsilon^2, \end{aligned} \quad (9)$$

where $D_{t+k} - \nabla_s^d D_{t+k}$ is $\mathcal{F}_{t+k-1}$ measurable, $\mu_\epsilon = E(\epsilon_t)$, and $\sigma_\epsilon^2 = \mathrm{var}(\epsilon_t)$.

The conditional expectation of (9) indicates that INARI(p) has the same minimum variance k-months-ahead predictor of D_{t+k} as the usual ARI(p) does. Therefore, the forecasts and fitted values are basically same for these two models. However, the conditional variance indicates that INARI(p) always has larger conditional variance than ARI(p) by $\sum_{i=1}^{p} |\alpha_i|(1 - |\alpha_i|)|\nabla_s^d D_{t-i}|$. This additional conditional variance term makes INARI(p) conditionally heteroscedastic, demonstrating another difference from ARI(p).

The relation $D_{tij} = \sum_{m=1}^{D_t} d_{tm}^c(i) \cdot d_{tm}^a(j)$ of (1) also leads to

$$\mathrm{var}(D_{tij}|\mathcal{F}_{t-1}) = \pi_{tij}(1 - \pi_{tij})E(D_t|\mathcal{F}_{t-1}) + \pi_{tij}^2 \mathrm{var}(D_t|\mathcal{F}_{t-1}). \quad (10)$$

By (10), the continuous ARI(p) approximation to INARI(p) underestimates $\mathrm{var}(D_{tij}|\mathcal{F}_{t-1})$, because the approximation underestimates $\mathrm{var}(D_t|\mathcal{F}_{t-1})$. This underestimated conditional variance by ARI(p) causes incorrect inferences for estimating parameters and for confidence interval of forecasts. We discuss this in detail in Section 4 with real data.

To include an useful covariate X_t in the model, we can easily extend the INARI(p) model to the integer-valued analog of the regression model with autocorrelated disturbance by defining

$$\nabla_s^d D_t = \alpha_0 \odot X_t + \nu_t, \qquad \nu_t = \sum_{q=1}^{p} \alpha_q \odot \nu_{t-q} + \epsilon_t, \quad (11)$$

where ν_t is assumed to satisfy the stationary condition given in (7) and ϵ_t is the same as before. Observe that (11) becomes a typical form of the continuous regression model with autocorrelated disturbance when the thinning operator $\odot$ in (11) is replaced with multiplication.

We can rewrite (11) as

$$\nabla_s^d D_t - \alpha_0 \odot X_t = \sum_{q=1}^{p} \alpha_q \odot (\nabla_s^d D_{t-q} - \alpha_0 \odot X_{t-q}) + \epsilon_t.$$

This is a special form of the INARI(p) model given in (6) with $\nabla_s^d D_t$ replaced by $\nabla_s^d D_t - \alpha_0 \odot X_t$. Thus an argument similar to that used in INARI(p) yields

$$E(\nabla_s^d D_t|\mathcal{F}_{t-1}) = \alpha_0 X_t + \sum_{q=1}^{p} \alpha_q (\nabla_s^d D_{t-q} - \alpha_0 X_{t-1}) + \mu$$

and

$$\begin{aligned} &\mathrm{var}(\nabla_s^d D_t|\mathcal{F}_{t-1}) \\ &\quad = |\alpha_0|(1 - |\alpha_0|)|X_t| + \sum_{q=1}^{p} \alpha_0^2 |\alpha_q|(1 - |\alpha_q|)|X_{t-q}| \\ &\qquad + \sum_{q=1}^{p} |\alpha_q|(1 - |\alpha_q|)E_B\big(|\nabla_s^d D_{t-q} - B(X_{t-q}, \alpha_0)|\big) + \sigma_\epsilon^2, \end{aligned}$$

where E_B denotes the expectation over random variable B, where $B(X, \alpha)$ is a binomial random variable with sample size X and success probability α.

The foregoing conditional expectation again implies that basically the integer-valued regression model (11) provides the

same fitted and predicted values as the continuous regression model. But, the conditional variance of model (11) is larger than the conditional variance, σ_ϵ^2, of the continuous regression model with autocorrelated disturbance. An integer-valued regression model with autocorrelated disturbance can be constructed similarly for more than two covariates. In the data analysis in Section 4, we consider two regression models with population as a covariate and compare them with the INARI(p) model.

3.3 Second Process of Classification Probability

We now extend the second process introduced in Section 3.1 to show how the classification probability connects two numbers (i.e., total and cause-age–specific death numbers). We discuss how the classification probability is specified by two logistic functions.

Let the classification probability be $\pi_{tij} = P(d_{tm}^c(i) \cdot d_{tm}^a(j) = 1|\mathbf{x}_t) = \pi_{ti|j}\pi_{tj}$, where $\pi_{ti|j} = P(d_{tm}^c(i) = 1|d_{tm}^a(j) = 1, \mathbf{x}_t)$ and $\pi_{tj} = P(d_{tm}^a(j) = 1|\mathbf{x}_t)$. Then we link the relevant covariates $\mathbf{x}_t$ to $\pi_{ti|j} = \pi_{ti|j}(\boldsymbol{\beta}_i'\mathbf{x}_t)$ and $\pi_{tj} = \pi_{tj}(\boldsymbol{\eta}_j'\mathbf{x}_t)$, so that π_{tij} is completely determined by much smaller number of parameters, $\boldsymbol{\beta}_i$ and $\boldsymbol{\eta}_j$, for $i = 1, \ldots, c$ and $j = 1, \ldots, a$.

In our data analysis we use population and monthly dummy variables as common covariates and link them through the logistic link functions. More precisely, we let

$$\begin{aligned}
\pi_{ti|j} &= \exp\left(\beta_{0i} + \beta_{1i}POP_t + \sum_{k=2}^{12}\beta_{ki}M_{t,k-1} + \sum_{k=13}^{a+11}\beta_{ki}A_{k-12}\right) \\
&\quad \times \left(1 + \sum_{i=1}^{c-1}\exp\left(\beta_{0i} + \beta_{1i}POP_t + \sum_{k=2}^{12}\beta_{ki}M_{t,k-1} \right.\right. \\
&\quad \left.\left. + \sum_{k=13}^{a+11}\beta_{ki}A_{k-12}\right)\right)^{-1} \quad \text{for } i = 1, \ldots, c; \\
\pi_{tj} &= \exp\left(\eta_{0j} + \eta_{1j}POP_t + \eta_{2j}\frac{POP_{jt}}{POP_{at}} + \sum_{k=3}^{13}\eta_{kj}M_{t,k-2}\right) \\
&\quad \times \left(1 + \sum_{j=1}^{a-1}\exp\left(\eta_{0j} + \eta_{1j}POP_t + \eta_{2j}\frac{POP_{jt}}{POP_{at}} \right.\right. \\
&\quad \left.\left. + \sum_{k=3}^{13}\eta_{kj}M_{t,k-2}\right)\right)^{-1} \quad \text{for } j = 1, \ldots, a-1,
\end{aligned} \tag{12}$$

where POP_t and POP_{jt} are U.S. total population and the population of the jth age (both divided by 100,000) at month t, $M_{t,k} = 1$ when t is k for $k = Jan., \ldots, Nov.$, and $A_{k-12} = 1$ for $k - 12 = j$ and 0 otherwise, by which $\pi_{ti|j}$ depends on the jth age. Therefore, we have

$$\begin{aligned}
\pi_{tij} &= \exp\left(\beta_{0i} + \eta_{0j} + (\beta_{1i} + \eta_{1j})POP_t \right. \\
&\quad \left. + \sum_{k=2}^{12}(\beta_{ki} + \eta_{k+1,j})M_{t,k-1} + \sum_{k=13}^{a+11}\beta_{ki}A_{k-12}\right) \\
&\quad \times \left(1 + \sum_{i=1}^{c-1}\exp\left(\beta_{0i} + \beta_{1i}t + \sum_{k=2}^{12}\beta_{ki}M_{t,k-1} \right.\right. \\
&\quad \left.\left. + \sum_{k=13}^{a+11}\beta_{ki}A_{k-12}\right)\right)^{-1}\eta_{2j}\frac{POP_{jt}}{POP_{at}} \\
&\quad \times \left(1 + \sum_{j=1}^{a-1}\exp\left(\eta_{0j} + \eta_{1j}POP_t \right.\right. \\
&\quad \left.\left. + \eta_{2j}\frac{POP_{jt}}{POP_{at}} + \sum_{k=3}^{13}\eta_{kj}M_{t,k-2}\right)\right)^{-1}.
\end{aligned} \tag{13}$$

We observe from (13) that the π_{tij} has different population and monthly effects on different age group j for each cause i. This implies that the hierarchical expressions of (12) also reflect the interaction effects between age group j and the common covariates (i.e., POP_t and M_{tk}) on π_{tij}.

This hierarchical expression can be easily extended to more complex classification probabilities. For example, we may consider the cause-specific deaths further classified by age-sex-race. Then the classification probability can be written as π_{tijkl} for the ith cause in the jth age, the kth sex, and the lth race at time t. As long as $\pi_{ti|jkl}$, $\pi_{tj|kl}$, $\pi_{tk|l}$, and π_{tl} include some common covariates in their link functions, all possible interaction effects between the common covariates and age-sex-race categorical variables can be reflected on π_{tijkl}.

3.4 Estimation

In this section we discuss a method of estimating parameters that reflects conditional variance and covariance of D_{tij} given $\mathcal{F}_{t-1}$. The parameters are denoted by $\boldsymbol{\alpha} = (\alpha_0, \alpha_1, \alpha_2, \ldots, \alpha_p)'$ and μ_ϵ for the INARI(p) model of (6) or the integer-valued regression model of (11), and by $\boldsymbol{\beta}_i$ and $\boldsymbol{\eta}_j$ for the two logistic functions of (12). All of these parameters are expressed as $\boldsymbol{\theta} = (\boldsymbol{\beta}_1', \ldots, \boldsymbol{\beta}_{c-1}', \boldsymbol{\eta}_1', \ldots, \boldsymbol{\eta}_{a-1}', \boldsymbol{\alpha}', \mu_\epsilon)'$. Denote $\mathcal{Y}_t = (D_{t11}, D_{t12}, \ldots, D_{tca})'$ and $\mathcal{K}_t(\boldsymbol{\theta}) = \mathcal{Y}_t - E(\mathcal{Y}_t|\mathcal{F}_{t-1})$. Then optimal estimating functions in the sense of Godambe (1985) are defined by

$$S_n(\boldsymbol{\theta}, \sigma_\epsilon^2) = \sum_{t=1}^{n}\frac{\partial\mathcal{K}_t(\boldsymbol{\theta})}{\partial\boldsymbol{\theta}}V_{t-1}^{-1}\mathcal{K}_t(\boldsymbol{\theta}), \tag{14}$$

where the variance–covariance matrix $V_{t-1} = \text{var}(\mathcal{Y}_t|\mathcal{F}_{t-1})$ whose diagonal elements are given in (9), (10), and (12). The off-diagonal elements of V_{t-1} are given by $\text{cov}(D_{tij}, D_{tk\ell}) = \pi_{tij}\pi_{tk\ell}(\text{var}(D_t|\mathcal{F}_{t-1}) - E(D_t|\mathcal{F}_{t-1}))$ for $(i, j) \neq (k, \ell)$. This variance–covariance matrix V_{t-1} reflects both longitudinal and cross-sectional correlations into estimation. Let

$$G_n(\boldsymbol{\theta}) = \sum_{t=1}^{n}\frac{\partial\mathcal{K}_t(\boldsymbol{\theta})}{\partial\boldsymbol{\theta}}V_{t-1}^{-1}\frac{\partial\mathcal{K}_t(\boldsymbol{\theta})}{\partial\boldsymbol{\theta}}'.$$

Following Fokianos and Kedem (1998), with appropriate regularity conditions for $G_n(\boldsymbol{\theta})$, it can be shown that

$$G_n^{-1/2}(\boldsymbol{\theta}, \sigma_\epsilon^2)S_n(\boldsymbol{\theta}, \sigma_\epsilon^2) \xrightarrow{d} Z, \tag{15}$$

where Z is a standard normal random vector. We replace σ_ϵ^2 with its consistent estimator $\hat{\sigma}_\epsilon^2$ provided by Lemma 1 for further development as follows.

Let $\hat{\boldsymbol{\theta}}_n$ be the solution of

$$S_n(\boldsymbol{\theta}, \hat{\sigma}_\epsilon^2) = \mathbf{0}. \tag{16}$$

We call $\hat{\boldsymbol{\theta}}_n$ the generalized estimating equation estimator. The first-order Taylor expansion of the optimal estimating equations $S_n(\hat{\boldsymbol{\theta}}_n, \hat{\sigma}_\epsilon^2)$ is

$$S_n(\hat{\boldsymbol{\theta}}_n, \hat{\sigma}_\epsilon^2) = 0 = S_n(\boldsymbol{\theta}, \hat{\sigma}_\epsilon^2) + \frac{\partial S_n(\boldsymbol{\theta}^*, \hat{\sigma}_\epsilon^2)}{\partial \boldsymbol{\theta}}(\hat{\boldsymbol{\theta}}_n - \boldsymbol{\theta}), \tag{17}$$

where $\boldsymbol{\theta}^*$ is an intermediate point between $\hat{\boldsymbol{\theta}}_n$ and $\boldsymbol{\theta}$. We assume that the following results hold:

(R1) $\frac{\partial S_n(\boldsymbol{\theta}, \tilde{\sigma}_\epsilon^2)}{\partial \sigma_\epsilon^2} = O_p(\sqrt{n})$ for any consistent estimator $\tilde{\sigma}_\epsilon^2$.

(R2) $\frac{1}{n}(\frac{\partial S_n(\boldsymbol{\theta}^*, \hat{\sigma}_\epsilon^2)}{\partial \boldsymbol{\theta}} - G_n(\boldsymbol{\theta}, \sigma_\epsilon^2)) = o_p(1)$, where $\frac{1}{n}G_n \xrightarrow{p} G$ for positive definite matrix G.

Using (15), the asymptotic normality of $\hat{\boldsymbol{\theta}}_n$ is established by

Theorem 1. Under (R1) and (R2),

$$\sqrt{n}(\hat{\boldsymbol{\theta}}_n - \boldsymbol{\theta}) \longrightarrow \mathrm{N}(\mathbf{0}, G^{-1}).$$

A conventional time series approach to the series of D_t leads to the incorrect variances of $V_{t-1} = \mathrm{var}(\mathcal{Y}_t|\mathcal{F}_{t-1})$ and $\hat{\sigma}_\epsilon^2$ in defining $S_n(\boldsymbol{\theta}, \hat{\sigma}_\epsilon^2)$ of (16). Thus the usual ARI(p) approximation to INARI(p) gives a different solution $\hat{\boldsymbol{\theta}}_n$ and an incorrect asymptotic distribution of the estimators.

3.5 Bootstrap Confidence Interval

The confidence interval of the forecast is often more informative than the forecast itself. The confidence interval can be used by policy makers to detect when a significantly high incidence of deaths has been occurred. This interval can also be used to validate our model by checking whether the confidence interval contains the NCHS true final death count. But, it is not easy to obtain distributional properties of forecasts from the INARI(p) model, because the integer-valued model introduces the complexity accrued in using the signed binomial thinning operator $\odot$.

An alternative approach is the bootstrap methods used in time series models (Thombs and Schucany 1990; Cardinal et al. 1999; Alonso, Peña, and Romo 2002). Following the approach Cardinal et al., we proceed as follows:

1. Compute the residuals,
$$\hat{\epsilon}_t = \nabla_s^d D_t - \sum_{i=1}^{p} \hat{\alpha}_i \nabla_s^d D_{t-i} \quad \text{for } t = p+1, \ldots, T,$$
where T is the last observation.
2. Because each error $\hat{\epsilon}_t$ may include a fractional part and our ϵ_t defined in (6) is integer-valued, modify it to the modified error $\hat{\epsilon}_t^*$ defined by $\hat{\epsilon}_t^* = [\hat{\epsilon}_t]$, where $[\cdot]$ denotes the usual integer operator. Then construct the empirical distribution F_n of the modified errors.
3. Draw a resample, $\hat{\epsilon}_t^*$, of iid observations from F_n.
4. Compute future bootstrap observations by the recursion
$$D_{T+h}^* = (D_{T+h}^* - \nabla_s^d D_{T+h}^*) - \sum_{i=1}^{p} \hat{\alpha}_i \odot \nabla_s^d D_{T+h-i}^* + \hat{\epsilon}_t^*,$$
where $D_t^* = D_t$ for $t \le T$ and $D_t^* = 0$ for $D_t^* < 0$.
5. Compute D_{tij}^* for $t > T$ by multiplication: $D_{tij}^* = D_t^* \times \hat{\pi}_{tij}$ for $t > T$.

Finally, repeat steps 3–5 B times to approximate the unknown distributions of D_{T+h} and $D_{T+h,i,j}$ given the observed sample. As usual, the bootstrap approximation is obtained by $\hat{F}_{D_{T+h}^*}(x) = \#\{D_{T+h}^* \le x\}/B$ and $\hat{F}_{D_{T+h,i,j}^*}(x) = \#\{D_{T+h,i,j}^* \le x\}/B$. The $(1-\alpha)\%$ forecast intervals for D_{T+h} and $D_{T+h,i,j}$ are obtained by taking $\alpha/2$th and $(1-\alpha)/2$th percentiles from the respective bootstrap distributions. A bootstrap procedure can be defined similarly, for an integer-valued regression model.

4. ANALYSIS OF NATIONAL CENTER FOR HEALTH STATISTICS MORTALITY DATA

We analyze 120 months of NCHS mortality data from January 1989 to December 1998 using the methods developed in the previous sections. During this period, NCHS classified deaths by 72 causes, we use the 49 main causes in our analysis. These 49 causes include 98.8% of all deaths in 1998. The remaining 23 causes are grouped into one class that we use as our reference group for the classification probability. The death records of these 50 causes are further classified by two age groups: young (under 65 years) and old (65 and over). In Section 4.1 we estimate the parameters of the INARI model for forecasting total number of deaths and those of the two logistic functions for estimating the classification probability. In Section 4.2 we show that the INARI model is better than the competitive two regression models. Bootstrap forecasts demonstrate that forecasts from INARI(p) and its continuous analog ARI(p) are negligibly different, as noted in Section 3.2. But the bootstrap confidence interval shows that the continuous ARI(p) badly distorts the nominal 95% confidence interval because it underestimates the conditional variance. In Section 4.3 we present out-of-sample forecast and use the 95% bootstrap confidence interval to check whether the interval contains the NCHS final death count. We also forecast long-term death rates and their 95% bootstrap confidence intervals.

4.1 Estimation

Using the sample autocorrelation function of $\nabla_{12} D_t$ that eliminates seasonality from the monthly total number of death D_t, we establish the INARI(13) model for our data as

$$\begin{aligned}\nabla_{12} D_t = {} & \alpha_1 \odot \nabla_{12} D_{t-1} + \alpha_2 \odot \nabla_{12} D_{t-2} \\ & + \alpha_3 \odot \nabla_{12} D_{t-12} + \alpha_4 \odot \nabla_{12} D_{t-13} + \epsilon_t. \end{aligned} \tag{18}$$

As competitors to the INARI model, we also consider two integer-valued regression models,

$$\begin{aligned}\nabla_{12} D_t &= \alpha_0 \odot \nabla_{12} POP_{t-1}^* + \nu_t, \\ \nu_t &= \alpha_1 \odot \nu_{t-1} + \alpha_2 \odot \nu_{t-2} \\ &\quad + \alpha_3 \odot \nu_{t-12} + \alpha_4 \odot \nu_{t-13} + \epsilon_t, \end{aligned} \tag{19}$$

and

$$\begin{aligned} D_t &= \alpha_0 \odot POP_{t-1}^* + \nu_t, \\ \nu_t &= \alpha_1 \odot \nu_{t-1} + \alpha_2 \odot \nu_{t-6} \\ &\quad + \alpha_3 \odot \nu_{t-12} + \alpha_4 \odot \nu_{t-13} + \epsilon_t, \end{aligned} \tag{20}$$

where $POP_t^* = 100{,}000 \times POP_t$, so that POP_t^* is the unscaled U.S. population at time t.

We used POP_{t-1}^* in both models because it is more reasonable that the current number of deaths depends on the population of the previous month $t-1$ rather than that of the current month t. The model (19) is reduced to model (18) when $\alpha_0 = 0$, whereas the model (20) is equivalent to a simple regression model with population as an independent variable, but its disturbance is autocorrelated.

Because we analyze 50 causes of death with the reference cause $i = 50$ (i.e., the 23 minor causes combined) and two age groups with the reference age group $j = 2$ (i.e., 65 years or over), the two logistic functions given in (12) can be written as

$$\log \frac{\pi_{ti|j}}{\pi_{t,50|j}} = \beta_{0i} + \beta_{1i} POP_t + \sum_{k=2}^{12} \beta_{ki} M_{t,k-1} + \beta_{13i} A_1 \quad \text{for } i = 1, \ldots, 49 \text{ and } j = 1, 2; \tag{21}$$

$$\log \frac{\pi_{t1}}{\pi_{t2}} = \eta_0 + \eta_1 POP_t + \eta_2 \frac{POP_{1t}}{POP_{2t}} + \sum_{k=3}^{13} \eta_j M_{t,k-2},$$

where all covariates are the same as those of Section 3.3 with $A_1 = 1$ for $j = 1$ and $A_1 = 0$ for $j = 2$ for dependence of $\pi_{ti|j}$ on age j.

Although (21) shows the general relationship between log-odd ratios and the covariates, its main purpose is to reduce the number of parameters to be estimated (i.e., from 12,000 π_{tij}'s to 700 β's and η's).

The generalized estimating equation $S_n(\boldsymbol{\theta}, \hat{\sigma}_\epsilon^2) = \mathbf{0}$ defined in (16) is solved by Fisher scoring iterations to estimate all parameters in (18) and (21) for the INARI model and in (19), (20), and (21) for the regression models,

$$\hat{\boldsymbol{\theta}}^{(r+1)} = \hat{\boldsymbol{\theta}}^{(r)} + \left[\sum_{t=1}^{n} \frac{\partial \mathcal{K}_t(\hat{\boldsymbol{\theta}}^{(r)})}{\partial \boldsymbol{\theta}} V_{t-1}^{-1}\left(\hat{\boldsymbol{\theta}}^{(r)}, \hat{\sigma}_\epsilon^2\right) \times \left(\frac{\partial \mathcal{K}_t(\hat{\boldsymbol{\theta}}^{(r)})}{\partial \boldsymbol{\theta}} \right)' \right]^{-1} S_n\left(\hat{\boldsymbol{\theta}}^{(r)}, \hat{\sigma}_\epsilon^2\right). \tag{22}$$

Here we present the estimation results only for the INARI model, because INARI model (18) is better than the competitive two regression models (19) and (20), as shown in Section 4.2. Using the convergence criterion

$$\max_i \left| \frac{\hat{\theta}_i^{(r+1)} - \hat{\theta}_i^{(r)}}{\hat{\theta}_i^{(r)}} \right| \le 10^{-5},$$

after the sixth iteration, we have 705 estimates [the 14 parameters for each of 49 causes in the first logistic model, i.e., 686 estimates in total, 14 estimates for the second logistic model, and five estimates for the INARI(13) model]. Table 1 gives the estimates for the five leading causes of death for the first logistic model, the second logistic model, and INARI(13) model.

In the first logistic model, all log-odd ratios increase as population increases. Log-odd ratios are the highest in November for the two myocardinal infarctions (OM and AM). The odd ratios for the two malignant neoplasms (RI and DP) are higher in the warm season from April to November than in the cold season from December to March; this is completely reversed in pneumonia with the exception of March; showing the different seasonality for the different causes. All negative estimates of β_{13i} on the last column of Table 1 imply that all of the odds ratios are higher in the old age group than the young age group. The second logistic model adjusts the impacts of population and monthly effects in the first logistic function by further incorporating the interaction effects [see (13)]. The current differenced monthly total death in the bottom part of Table 1 (i.e., $\nabla_{12} D_t$) is positively correlated with the differenced monthly total of the previous month $(t-1)$ and that of 13 months earlier $(t-13)$, but negatively correlated with the differenced total of the same month of the previous year $(t-12)$.

4.2 Model Comparison

We compare INARI(13) of model (18) with the two competitive regression models, (19) and (20), in terms of the difference between model-fitted values (or forecasts) and the NCHS true final death counts. This difference is measured by the mean squared error (MSE) and the mean absolute percentage error (MAPE) for the total number of death $\hat{D}_t$, deaths by the

Table 1. Coefficient Estimates for Two Logistic Models and INARI(13)

First logistic equations: $\log \frac{\pi_{ti/j}}{\pi_{t,50/j}} = \beta_{0i} + \beta_{1i} POP + \sum_{k=2}^{12} \beta_{ki} M_{t,k-1} + \beta_{13i} A_1$

Cause	β_{0i}	β_{1i} (POP)	β_{2i} (Jan.)	β_{3i} (Feb.)	β_{4i} (Mar.)	β_{5i} (Apr.)	β_{6i} (May)	β_{7i} (Jun.)	β_{8i} (Jul.)	β_{9i} (Aug.)	β_{10i} (Sep.)	β_{11i} (Oct.)	β_{12i} (Nov.)	β_{13i} (age)
OM	1.07	.00098	−.123	−.047	−.021	.009*	−.007*	−.022	−.020	−.044	−.034	−.018*	.024	−3.003
AM	3.35	.00003*	−.149	−.077	−.059	−.013*	−.018*	−.048	−.043	−.064	−.042	−.018*	.031	−2.508
RI	−.521	.00129	−.176	−.058	−.014*	.051	.064	.079	.104	.112	.109	.097	.098	−1.87
DP	−.403	.00118	−.189	−.062	−.009*	.054	.084	.104	.131	.140	.135	.106	.104	−2.109
PM	−1.515	.00159	.066	.006*	−.036	−.100	−.210	−.294	−.325	−.351	−.317	−.220	−.141	−3.181

Second logistic equation: $\log \frac{\pi_{t1}}{\pi_{t2}} = \eta_0 + \eta_1 POP + \eta_2 \frac{POP_1}{POP_2} + \sum_{j=3}^{13} \eta_j M_{t,j-2}$

η_0	η_1	η_2	η_3 (Jan.)	η_4 (Feb.)	η_5 (Mar.)	η_6 (Apr.)	η_7 (May)	η_8 (Jun.)	η_9 (Jul.)	η_{10} (Aug.)	η_{11} (Sep.)	η_{12} (Oct.)	η_{13} (Nov.)
.0928	.0872	1.934	−11.28	.003	−.117	.052*	.110	.174	.197	.248	.215	.203	.136

Monthly total deaths, INARI(13) model

$$\nabla_{12} D_t = 2089.4 + .451 \odot \nabla_{12} D_{t-1} - .119^* \odot \nabla_{12} D_{t-2} - .654 \odot \nabla_{12} D_{t-12} + .321 \odot \nabla_{12} D_{t-13}$$

NOTE: OM, old myocardinal infarction; AM, acute myocardinal infarction; RI, malignant neoplasms of respiratory and intrathoracic organs; DP, malignant neoplasms of digestive organs and peritoneum; PM, pneumonia.

*Nonsignificant under $\alpha = .05$.

Table 2. Two Statistics for Model Adequacy Using the Data Used for Model Fitting

Model	$\hat{D}_t$		$\hat{D}_{ti}$		$\hat{D}_{tj}$		$\hat{D}_{tij}$	
	$\sqrt{MSE}$	MAPE	$\sqrt{MSE}$	MAPE	$\sqrt{MSE}$	MAPE	$\sqrt{MSE}$	MAPE
INARI(13) [model (18)]	1,504.7	.016	212.8	.036	1,064.0	.016	150.5	.062
Regression model 1 [model (19)]	2,566.9	.036	363.0	.050	1,815.1	.036	256.7	.073
Regression model 2 [model (20)]	4,834.4	.093	683.7	.102	3,418.5	.093	483.4	.120

ith cause and the jth age group $\hat{D}_{tij}$, the cause-specific deaths $\hat{D}_{ti}$ ($= \sum_j \hat{D}_{tij}$), and the age-specific deaths $\hat{D}_{tj}$ ($= \sum_i \hat{D}_{tij}$).

Table 2 gives the two statistics for the four fitted values of the three models. The two statistics are calculated using predictions for time periods within the scope of the data used for model fitting. The INARI(13) model has apparently smaller MSE and MAPE than the two regression models for all four fitted values. The MAPE of the INARI(13) indicates that the $\hat{D}_t$ deviates from the true value D_t by 1.6% on average, from $\hat{D}_{ti}$ by 3.6%, from $\hat{D}_{tj}$ by 1.6%, and from $\hat{D}_{tij}$ by 6.2%.

Although we did not include all of the results in this article, it is noteworthy that the continuous counterparts of the two regression models [i.e., models (19) and (20)] have almost the same MSE and MAPE as presented in Table 2. The reason for this is that both discrete models and continuous counterparts have the same conditional expectation. However, they have different inference for the parameter estimates as illustrated in Table 3 for ARI(13) and INARI(13).

Because bootstrapping represents distributional behavior better than its underlying model, the MSE and MAPE for bootstrap $\hat{D}_t$ by INARI(13) in the first column of Table 3 are smaller than those calculated from the underlying INARI(13) in Table 2. This indicates the validity of the bootstrap procedure provided in Section 3.5.

Table 3 shows the bootstrap approach to comparing the INARI(13) model with the ARI(13) model. The first column provides the MSE and MAPE for these two models using predictions for time periods within the scope of data used for model fitting. The MSE and MAPE of INARI(13) are negligibly different from those of ARI(13), as expected. However, 13.7% of the observations are outside of the 95% confidence interval of ARI(13), whereas 7.35% are outside of that of INARI(13), showing a serious underestimation of conditional variance in the ARI(13) model. The second column of Table 3 also compares the two models with 2-year out-of-sample forecasts. The 2-year (24-month) forecasts are obtained for 1997 and 1998 with new estimates based on data from 1989 to 1996. Although the MSE and MAPE of ARI(13) are slightly smaller than those of INARI(13), the 95% confidence interval of ARI(13) excludes 6 of 24 true death counts (i.e., 25%), whereas that of INARI(13) excludes 2 true death counts (i.e., 8.3%). This shows that the ARI(13) approximation seriously distorts the nominal 95% confidence interval.

4.3 Forecasting

The NCHS releases the final death data 1 or more years later than the actual year. To validate our model in practical situation, we use the 96-month data from January 1989 to December 1996 to obtain new estimates and to forecast the cause-specific death counts for 1998 as if they were unknown. We then compare our forecasts with the true 1998 final data. Because our forecasts (Fore) for 1998 use neither the 1997 data nor the 1998 data, our model can eliminate the time delay of the 1998 NCHS preliminary estimates, which were released in October 1999. Our model forecasts slightly larger numbers of total deaths than the preliminary estimate, which used 99% or more of the total final counts; our model overestimates by 1.6% in 1997 and .9% in 1998, whereas the preliminary report overestimates by .2% for both years. However, our forecasts give better numbers than the preliminary estimates for the causes of death which are subject to medicolegal investigation (see Table 5).

Table 4 shows the forecasts and their bootstrap 95% confidence intervals for the 33 leading causes of death among the 49 causes of death used in our analysis. The first four columns are for February 1998, whereas the second five columns are for the total counts of 1998. For February 1998, our monthly forecasts (Fore) are close to the final cause-specific final counts (Final), and most confidence intervals contain their true final cause-specific counts. Exceptions are pneumonia and its allied conditions (e.g., chronic obstructive pulmonary diseases), whose final death counts exceed their 95% upper bounds. This implies that these forecasts identify unusually large numbers of deaths for pneumonia and its allied conditions in February 1998, which is consistent with a severe influenza outbreak reported that winter. Our forecasts show that this outbreak substantially increased the number of deaths ranging from 13.1% to 22.1% for these causes and can detect unusual incidence of deaths that deviated from the previous pattern.

The second five columns in Table 4 show the 1998 NCHS final counts, our forecast with the 95% bootstrap confidence bounds, and the NCHS preliminary estimates. Our forecasts are very close to the NCHS final true numbers for most of the cause-specific deaths. The 95% bootstrap confidence bounds contain all of the true final death counts with the two exceptions (i.e., 5.2, cerebral thrombosis and occlusion of cerebral arteries and 5.3, all other and late effects of cerebrovascular disease), which are barely below their lower bound. This indicates

Table 3. Bootstrapping Comparison of INARI(13) and Its Continuous Analog ARI(13) for Total Number of Deaths D_t

Model	Model fit			2-year out-of-sample forecast for 1997 and 1998		
	$\sqrt{MSE}$	MAPE	% of observations outside the 95% CI	$\sqrt{MSE}$	MAPE	% of observations outside the 95% CI
INARI(13)	1,458.2	.0150	7.36%	5,453	.031	8.33%
ARI(13)	1,480.5	.0153	13.7%	5,282	.028	25.0%

Table 4. The Forecasts and Their 95% Bootstrap Confidence Bounds for February 1998 and for the Total of 1998 Using the Data From 1989 to 1996, and Final Death Counts and NCHS Preliminary Estimates

Cause of death (based on the ninth revision of ICD)	February 1998				Total of 1998				
	Final	Fore	L95	U95	Final	Fore	L95	U95	NCHS
1. Septicemia(Septi)	2,083	2,007	1,944	2,111	23,753	23,237	22,381	24,376	23,645
2. Malignant neoplasms(Cancer)									
2.1 Malignant neoplasms of lip, oral cavity, and pharynx	640	632	612	664	7,974	7,949	7,659	8,345	7,902
2.2 Malignant neoplasms of digestive organs and peritoneum	10,025	10,126	9,809	10,646	128,783	130,069	125,318	136,554	127,685
2.3 Malignant neoplasms of respiratory and intrathoracic organs	12,684	12,776	12,376	13,432	159,819	161,956	156,035	170,014	159,207
2.4 Malignant neoplasms of breast	3,406	3,433	3,325	3,609	42,125	43,122	41,550	45,270	41,862
2.5 Malignant neoplasms of genital organs	4,543	4,698	4,541	4,939	58,419	59,910	57,841	63,022	58,079
2.6 Malignant neoplasms of urinary organs	1,868	1,874	1,815	1,970	23,782	24,041	23,162	25,237	23,722
2.7 Malignant neoplasms of all other and unspecified sites	4,977	5,225	5,061	5,493	65,467	67,406	64,944	70,765	65,305
2.8 Leukemia	1,574	1,632	1,573	1,716	20,382	20,823	20,062	21,859	20,160
2.9 Other neoplasms of lymphatic and hematopoietic tissues	2,817	2,887	2,796	3,035	35,266	36,393	35,063	38,203	35,007
3. Diabetes mellitus	5,558	5,579	5,404	5,865	64,808	66,563	64,113	69,838	64,574
4. Heart diseases(Heart)									
4.1 Rheumatic fever and rheumatic heart disease	424	413	340	434	4,799	4,842	4,665	5,081	4,833
4.2 Hypertensive heart disease	2,469	2,392	2,316	2,514	27,808	28,203	27,163	29,588	27,304
4.3 Hypertensive heart and renal disease	223	215	208	226	2,412	2,471	2,380	2,592	2,395
4.4 Acute myocardinal infarction	17,917	17,513	16,966	18,413	203,888	206,403	198,810	216,549	203,835
4.5 Other acute and subacute forms of ischemic heart disease	226	232	224	244	2,912	2,858	2,752	2,998	2,824
4.6 Old myocardinal infarction and other forms of ischemic heart	22,371	22,330	21,632	23,478	253,045	261,213	251,607	274,056	253,074
4.7 Other diseases of endocardium	1,731	1,625	1,574	1,709	18,547	18,687	18,000	19,607	18,562
4.8 All other forms of heart disease	19,102	18,380	17,805	19,324	211,953	215,366	207,431	225,939	210,820
5. Cerebrovascular diseases(Cereb)									
5.1 Intracerebral and other intracranial hemorrhage	2,174	2,209	2,140	2,322	26,297	26,258	25,290	27,549	26,010
5.2 Cerebral thrombosis and occlusion of cerebral arteries	975	1,012	973	1,064	10,967	11,584	11,161	12,156	11,024
5.3 All other and late effects of cerebrovascular disease	10,600	10,751	10,415	11,304	120,795	126,168	121,523	132,366	120,461
6. Pneumonia(Pneum)	11,148	8,889	8,611	9,346	90,193	88,866	85,564	93,121	92,718
7. Chronic obstructive pulmonary diseases and allied conditions(Chronic)									
7.1 Bronchitis, chronic and unspecified	392	318	307	334	3,034	3,030	2,919	3,176	3,120
7.2 Emphysema	1,844	1,670	1,618	1,756	17,562	17,754	17,101	18,620	17,918
7.3 Asthma	554	489	474	514	5,460	5,658	5,475	5,963	5,344
7.4 Other obstructive pulmonary diseases and allied conditions	9,137	8,122	7,819	8,540	86,576	87,334	84,112	91,584	87,999
8. Nephritis and nephrotic syndrome(Nephri)									
8.1 Chronic glomerulonephritis, nephritis and nephropathy	163	138	134	145	1,717	1,688	1,630	1,776	1,706
8.2 Renal failure, disorders resulting from impaired renal function	2,138	2,060	1,996	2,166	24,157	24,173	23,283	25,360	24,274
9. Symptoms, signs, and ill-defined conditions	2,056	2,181	2,113	2,293	25,945	26,874	25,871	28,184	35,491
10. Accidents and adverse effects(Accident)									
10.1 Motor vehicle accidents	2,974	2,986	2,893	3,140	43,765	44,311	42,692	46,540	41,826
10.2 All other accidents and adverse effects	4,193	4,230	4,078	4,447	54,603	54,669	52,675	57,400	51,382
11. Suicide	2,413	2,428	2,352	2,552	30,617	31,124	30,003	32,692	29,264

that our model is a valid method for forecasting cause-specific death counts and can be used as an alternative to the NCHS preliminary estimates.

Because of the nature of the preliminary data (Kochanek et al. 2004), the NCHS preliminary estimates systematically underestimate the number of deaths for the causes subjected to medicolegal investigation, such as accidents and adverse effects and suicide, whereas it overestimates for symptoms, signs, and ill-defined conditions. As shown in the second five columns of Table 4, our 95% confidence intervals confirm these systematic biases by showing that the NCHS preliminary estimates in the last column for accidents and adverse effects and suicide are below our lower 95% bounds, whereas those for the symptoms, signs, and ill-defined conditions clearly exceed the upper 95% bound. Generally, our model forecasts quite accurately and even better than the NCHS preliminary estimates for these causes.

To see whether this pattern is consistent for other years, we perform 2-year out-of-sample forecasts: monthly forecasts for 1996 using data from January 1989 to December 1994, those for 1997 using data from January 1989 to December 1995, and those for 1998 using data from January 1989 to December 1996. Table 5 shows that our forecasts are generally better than the preliminary estimates and are consistently better for symptoms, signs, and ill-defined conditions, and other external causes for all three years, 1996, 1997, and 1998.

Table 5. Comparison of Our Forecast (Fore) With the NCHS Preliminary Estimate and Final Number of Deaths for Causes Subject to Medicolegal Investigation

	1996			1997			1998		
Cause of death	*Fore*	*NCHS*	*Final*	*Fore*	*NCHS*	*Final*	*Fore*	*NCHS*	*Final*
Symptoms, signs, and ill-defined conditions	26,289 (1.005)	30,371 (1.161)	26,164	26,700 (1.038)	33,569 (1.305)	25,716	26,817 (1.033)	35,491 (1.368)	25,945
Motor vehicle accidents	44,015 (1.002)	43,449 (.989)	43,939	44,484 (1.016)	42,420 (.969)	43,767	44,237 (1.011)	41,826 (.956)	43,765
All other accidents and adverse effects	52,202 (1.012)	50,425 (.976)	51,582	53,763 (1.025)	49,722 (.949)	52,420	54,554 (.999)	51,382 (.941)	54,603
Suicide	31,265 (1.010)	30,862 (.997)	30,945	31,382 (1.026)	29,725 (.973)	30,563	31,061 (1.014)	29,264 (.956)	30,617
Homicide and legal intervention	21,520 (1.025)	20,738 (.988)	20,994	20,505 (1.032)	18,774 (.945)	19,865	19,274 (1.055)	17,350 (.950)	18,268
All other external causes	3,560 (1.028)	3,185 (.920)	3,462	3,747 (1.024)	3,255 (.890)	3,659	3,876 (1.035)	3,315 (.885)	3,745

NOTE: The numbers in parentheses indicate the ratios to final death count.

The death rate (the number of deaths for each age group divided by the corresponding population) is often used to study mortality trends. We use the U.S. Bureau of the Census' population projection and our forecasted number of deaths to obtain the rates for 2010 and 2020 using all of the data from 1989 to 1998. Table 6 shows the forecasted death rates (per 100,000 U.S. population) and their bootstrap 95% confidence bounds of the 10 leading causes and two age groups for 2010 and 2020. The death rates for 1998 are the true observation. The death rates behave quite differently for different causes and age groups. As expected, the death rates are much higher for the old age group than for the young age group in all causes.

All death rates for the old age group are decreased, especially for cancer and heart disease, whereas the death rates for the young age group are generally increased as time progresses. The 2020 lower bounds for cancer (631.8) and heart diseases (890.5) indicate that their death rates can be about 50% of the 1998 rates (1,134.9 and 1,790.8) for the old age group. It is interesting to see that the death rates of diabetes mellitus and the remaining seven causes are generally increased for the young age group.

The U.S. Social Security Administration (SSA) (2002) forecasted death rates for six causes of death. It is impossible for a direct comparison between our and the SSA's long-term death rates, because the two calculation methods of death rates are different. But Table 7 provides two sets of death rates to demonstrate general trends for heart disease, cancer, and diabetes over all ages for 2010 and 2020. Both death rates for heart disease have similar decreasing patterns, whereas our death rate for cancer decreases more rapidly than the SSA's rate. Our death rate for diabetes shows a monotone increasing pattern, but that of SSA increases until 2010 and decreases thereafter. The NCHS report (Kochanek et al. 2004) indicated that the death rate for cancer increased by 1.4% between 1979 and 1993 and showed a gradual and consistent downward trend since 1993. On the other hand, this report indicated that the death rate for diabetes decreased steadily from 1968 to 1986 and increased rapidly since 1986. Based on this report, our rates for cancer and diabetes reflect these recent trends more reasonably than the SSA's rates do.

5. CONCLUSION

We present the forecasts and their bootstrap confidence intervals for the monthly and yearly number of deaths using the U.S. mortality data from 1989 to 1998. We use the two–random processes model to achieve our forecasting and to avoid the overflowing dimension problem. The first process, the INARI(p) model for the total monthly deaths, handles nonstationary integer-valued time series and incorporates serial dependency of the monthly death counts. The second process is a

Table 6. Yearly Mean Death Rates and Their Bootstrap 95% Confidence Bounds per 100,000 Population for Two Age Groups

Age	*Year*		*Cancer*	*Diabetes*	*Heart*	*Cereb*	*Pneum*	*Chronic*	*Nephri*	*Septi*	*Accident*	*Suicide*
≤64	1998		66.1	6.68	49.5	8.27	3.74	6.39	1.51	1.79	27.8	10.4
	2010	L95	49.3	7.33	34.2	7.36	3.12	5.86	1.37	1.61	24.1	7.73
		Mean	54.8	8.14	37.9	8.17	3.46	6.51	1.52	1.78	26.8	8.58
		U95	58.6	8.70	40.6	8.74	3.69	6.95	1.62	1.91	28.7	9.18
	2020	L95	49.6	10.2	33.2	8.77	3.42	7.13	1.62	1.87	27.6	7.71
		Mean	56.7	11.6	37.9	10.0	3.91	8.14	1.84	2.14	31.5	8.80
		U95	61.4	12.6	41.1	10.9	4.22	8.80	2.00	2.32	34.2	9.54
>64	1998		1,134.9	145.8	1,790.8	415.4	230.3	283.8	64.1	54.6	93.8	18.2
	2010	L95	947.0	178.7	1,386.7	388.5	214.4	301.6	65.1	54.4	95.6	15.1
		Mean	1,051.2	198.3	1,539.1	431.2	238.1	334.7	72.3	60.5	106.2	16.8
		U95	1,124.0	211.9	1,664.9	460.8	254.0	357.5	77.2	64.6	113.6	17.9
	2020	L95	631.8	164.3	890.5	291.8	156.3	249.9	51.0	42.1	75.6	10.0
		Mean	721.4	187.6	1,016.6	333.1	178.5	285.3	58.3	48.1	86.3	11.4
		U95	781.0	203.0	1,100.4	360.6	192.8	308.6	63.1	52.1	93.5	12.4

Table 7. Comparison of Our Death Rates to Those of the Social Security Administration for Heart Disease, Cancer, and Diabetes Over All Ages

	Ours			Social security		
Year	Heart	Cancer	Diabetes	Heart	Cancer	Diabetes
1998	270.6	201.8	24.3	255.5	213.2	23.9
2010	237.5	187.3	33.4	205.1	204.9	26.8
2020	200.4	167.1	40.8	173.9	196.2	25.8

multinomial process with the classification probabilities specified by logistic link functions. This process incorporates the categorical correlation among cause-age–specific death counts. We obtain the cause-age–specific number of deaths by multiplying the results of the two processes (i.e., total death × classification probability).

We have compared our INARI model with several continuous models (e.g., continuous regression model with autocorrelated disturbance and ARI model) and showed that our model provides nominal confidence interval more reasonably than the ARI model does. We have demonstrated that our yearly forecasts are generally as good as the NCHS preliminary estimates. In particular, for the causes subject to medicolegal investigation, our forecasts are better than the preliminary estimates. However, when the preliminary estimates are based on 99% or more of the total deaths, these estimates are often slightly more reliable than our forecasts. But the important benefits of our model are that our forecast does not have the time-delay problem, that our model can predict future mortality, and that we can get better estimates for the death of medicolegal causes. Our bootstrap confidence band can be used to detect an unusual situation, and this can be a useful tool for statistical strategists.

APPENDIX: PROOFS

Proof of Lemma 1

By propositions 6.31 and 6.32 and theorem 3.12 of Breiman (1968), the INARI(p) process satisfying the stationary condition described in Section 3.2 is also ergodic. Thus, by the approach of Durrett (1991),

$$\bar{\tilde{D}} = \frac{1}{n}\sum_{t=1}^{n} \nabla_s^d D_t \xrightarrow{\text{a.s.}} E(\nabla_s^d D_t),$$

$$\frac{1}{n}\sum_{t=1}^{n} |\nabla_s^d D_t| \xrightarrow{\text{a.s.}} E(|\nabla_s^d D_t|), \tag{A.1}$$

$$\frac{1}{n}\sum_{t=1}^{n} \nabla_s^d D_t \cdot \nabla_s^d D_{t-k} \xrightarrow{\text{a.s.}} E(\nabla_s^d D_t \cdot \nabla_s^d D_{t-k}) \quad \text{for } k = 0, 1, 2, \ldots.$$

Thus $\hat{\alpha}_i$ for $i = 1, 2, \ldots, p$ is strongly consistent, because $\hat{\gamma}_k \xrightarrow{\text{a.s.}} \gamma_k$ by (A.1).

Observe that $\epsilon_t = \nabla_s^d D_t - \sum_{i=1}^{p} \alpha_i \odot \nabla_s^d D_{t-i}$ to have $E(\epsilon_t) = (1 - \sum_{i=1}^{p} \alpha_i)\mu$, where $E(\nabla_s^d D_t) = \mu$ by stationarity. A little calculation shows that

$$E(\epsilon_t^2) = (\gamma_0 + \mu^2)\left(1 + \sum_{i=1}^{p} \alpha_i^2\right) + \sum_{i=1}^{p} |\alpha_i|(1 - |\alpha_i|)E|\nabla_s^d D_{t-i}|$$

$$- 2\sum_{i=1}^{p} \alpha_i(\gamma_i + \mu^2) + 2 \sum_{1 \le i < j \le p} \alpha_i \alpha_j \big(\gamma_{|i-j|} + \mu^2\big). \tag{A.2}$$

Because

$$\frac{1}{n}\sum_{t=1}^{n} \hat{\epsilon}_t = \frac{1}{n}(\nabla_s^d D_t - \hat{\alpha}_1 \nabla_s^d D_{t-1} - \cdots - \hat{\alpha}_p \nabla_s^d D_{t-p}),$$

(A.1) and strong convergence of the $\hat{\alpha}$'s imply that $\bar{\epsilon}_n$ converges $(1 - \sum_{i=1}^{p} \alpha_i)\mu$ almost surely. Similarly, one also can show that, by (A.1) and consistency estimators $\hat{\alpha}$,

$$\frac{1}{n}\sum_{t=1}^{n} \hat{\epsilon}_t^2 \xrightarrow{\text{a.s.}} (\gamma_0 + \mu^2)\left(1 + \sum_{i=1}^{p} \alpha_i^2\right) - 2\sum_{i=1}^{p} \alpha_i(\gamma_i + \mu^2)$$

$$+ 2 \sum_{1 \le i < j \le p} \alpha_i \alpha_j \big(\gamma_{|i-j|} + \mu^2\big). \tag{A.3}$$

Consequently, (A.2) and (A.3) show that $\hat{\sigma}_\epsilon^2$ converges to σ_ϵ^2 almost surely.

Proof of Theorem 1

By Taylor expansion,

$$S_n(\boldsymbol{\theta}, \hat{\sigma}_\epsilon^2) = S_n(\boldsymbol{\theta}, \sigma_\epsilon^2) + \frac{\partial S_n(\boldsymbol{\theta}, \tilde{\sigma}_\epsilon^2)}{\partial \sigma_\epsilon^2}(\hat{\sigma}_\epsilon^2 - \sigma_\epsilon^2), \tag{A.4}$$

where $\tilde{\sigma}_\epsilon^2$ is between σ_ϵ^2 and $\hat{\sigma}_\epsilon^2$. Thus, condition (R1) and (15) yield that

$$G_n^{-1/2} S_n(\boldsymbol{\theta}, \hat{\sigma}_\epsilon^2) \xrightarrow{d} Z. \tag{A.5}$$

From (17), we have that

$$\sqrt{n}(\hat{\boldsymbol{\theta}}_n - \boldsymbol{\theta}) = -\left[\frac{1}{n}\frac{\partial S_n(\boldsymbol{\theta}^*, \hat{\sigma}_\epsilon^2)}{\partial \boldsymbol{\theta}}\right]^{-1} \frac{1}{\sqrt{n}} S_n(\boldsymbol{\theta}, \hat{\sigma}_\epsilon^2)$$

$$= -\left[\frac{1}{n}\frac{\partial S_n(\boldsymbol{\theta}^*, \hat{\sigma}_\epsilon^2)}{\partial \boldsymbol{\theta}}\right]^{-1} \frac{1}{\sqrt{n}}$$

$$\times G_n^{1/2}(\boldsymbol{\theta}, \sigma_\epsilon^2) G_n^{-1/2}(\boldsymbol{\theta}, \sigma_\epsilon^2) S_n(\boldsymbol{\theta}, \hat{\sigma}_\epsilon^2). \tag{A.6}$$

Because (R2) implies that $(1/\sqrt{n})G_n^{1/2}(\boldsymbol{\theta}, \sigma_\epsilon^2) \xrightarrow{p} G^{1/2}$ and $[\frac{1}{n}\frac{\partial S_n(\boldsymbol{\theta}^*, \hat{\sigma}_\epsilon^2)}{\partial \boldsymbol{\theta}}]^{-1} = [\frac{1}{n}G_n + o_p(1)]^{-1}$, (A.5) and (A.6) yield the desired result.

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