Journal of Modern Applied Statistical Methods

Volume 7 | Issue 2 Article 22

11-1-2008

Application of Dynamic Poisson Models to Japanese Cancer Mortality Data

Shuichi Midorikawa
Tokyo University of Science, shuu@m2.dion.ne.jp

Etsuo Miyaoka
Tokyo University of Science, miyaoka@rs.kagu.tus.ac.jp

Bruce Smith
Dalhousie University, Bruce.Smith@Dal.Ca

Journal of Modern Applied Statistical Methods
November, 2008, Vol. 7, No. 2, 577-587

1538 – 9472/08/$95.00

Application of Dynamic Poisson Models to Japanese Cancer Mortality Data

Shuichi Midorikawa
Tokyo University of Science

Etsuo Miyaoka
Tokyo University of Science

Bruce Smith
Dalhousie University

A dynamic Poisson model is used with a Bayesian approach to modeling to predict cancer mortality. The complexity of the posterior distribution prohibits direct evaluation of the posterior, and so parameters are estimated by using a Markov Chain Monte Carlo method. The model is applied to analyze lung and stomach cancer data which have been collected in Japan.

Key words: Dynamic Poisson model, Markov Chain Monte Carlo, cancer mortality data, age-period-cohort model prediction.

Introduction

The number of cases of stomach cancer in the Japanese male population is tabulated in Table 1, by five year period, and by 5 year age group. Periods are identified in the Table by their central year. For example, the period labeled 1970 includes all data for the years 1968 through 1972, inclusive. These data were obtained from the Japanese Ministry of Health and Welfare. (http://wwwdbtk.mhlw.go.jp/toukei/index.html)

The goal of this article is the development trend models for these data, and in particular, the development of methods for short to medium term prediction, which will be important from the perspective of public health planning. The entries in Table 1 for the 2005 and 2010 periods are, in fact, predictions, calculated as described in section 5 below. In assessing trends in such data, care must be taken to accommodate for trends in the underlying population structure.

Shuichi Midorikawa is in the Department of Mathematics. Email: shuu@m2.dion.ne.jp Etsuo Miyaoka is Professor of Statistics and Probability in the Department of Mathematics. Email:miyaoka@rs.kagu.tus.ac.jp. Bruce Smith is Professor in the Department of Mathematics and Statistics. Email: Bruce.Smith@Dal.Ca.

In particular, the reduction in numbers of cancers at increased age is due primarily to the reduction in the associated number of individuals at risk. To accommodate for the number of individuals at risk, we focus on the incidence rate, equal to the number of events divided by the number at risk. Table 2 shows the incidence rate as numbers of stomach cancers per million males, calculated by dividing the raw incidence numbers from Table 1 by the population size (the total number of males in the associated age group), and multiplying by one million. The population cohort numbers were obtained from the Japanese Ministry of Internal Affairs and Communications.

There are some notable trends in the incidence rate data of Table 2. In particular, except for the oldest few age groups, the incidence rate is increasing with age, in each period. On the other hand, at least up until age 65 or 70, the incidence rate within age group appears to be more or less decreasing over time. Incidence rates for female stomach cancer, and for male and female lung cancer, were similarly calculated, and are illustrated in the appendix, together with predictions for the periods centered at 2005 and 2010. Patterns similar to the males for the rate of female stomach cancer are noted (Table 5), and with respect to rates of lung cancer in both males and females, the data appear to show increasing rates over age group, and over time (Tables 6 and 7).

Table 1: Numbers of cases of stomach cancer - males

Age Group	5 year period centered at												
	1950	1955	1960	1965	1970	1975	1980	1985	1990	1995	2000	2005	2010
15-19	9	9	15	17	19	9	5	5	11	5	4	5	5
20-24	27	30	46	72	80	59	28	21	18	20	18	18	18
25-29	65	106	127	158	162	162	104	87	49	49	30	45	45
30-34	166	196	300	353	346	309	308	196	102	77	70	87	95
35-39	359	395	470	615	628	562	526	453	315	207	142	171	165
40-44	788	854	790	781	1004	1003	799	719	646	494	303	322	300
45-49	1406	1568	1517	1309	1387	1638	1583	1192	1101	1027	724	766	581
50-54	2206	2470	2488	2402	1991	1922	2465	2203	1772	1626	1608	1491	1231
55-59	3024	3398	3717	3666	3214	2871	2632	3253	2992	2592	2458	2209	2068
60-64	3602	4125	4569	4993	4638	4201	3603	3467	4263	4034	3408	3310	2929
65-69	3465	4195	4799	5483	5699	5334	5013	4082	4081	5210	5237	4213	3998
70-74	2505	3244	4147	4483	5228	5594	5472	4952	4258	4869	6009	4757	4257
75-79	1063	1743	2289	3010	3459	4132	4743	4702	4613	4571	4859	4234	4005
80-84	261	479	829	1034	1457	2000	2636	3273	3512	4073	3977	2976	2971
85-89	61	78	170	241	313	536	804	1283	1727	2361	2767	1747	1580
90-	3	15	16	28	35	77	134	269	460	806	1184	628	598

Table 2: Stomach cancer - males, rate per million

Age Group	5 year period centered at										
	1950	1955	1960	1965	1970	1975	1980	1985	1990	1995	2000
15-19	2	2	3	3	4	2	1	1	2	1	1
20-24	7	7	11	16	15	13	7	5	4	4	4
25-29	23	28	31	38	36	30	23	22	12	11	6
30-34	70	70	80	85	83	67	57	43	26	19	16
35-39	151	170	170	164	153	134	115	84	70	53	35
40-44	358	367	347	286	275	244	193	160	121	110	78
45-49	696	734	672	588	522	450	394	294	246	194	163
50-54	1283	1280	1219	1105	930	740	698	565	444	370	310
55-59	2193	2114	2062	1899	1584	1395	1055	959	791	667	575
60-64	3246	3362	3178	3072	2656	2183	1864	1476	1318	1121	911
65-69	4353	4564	4673	4498	4090	3411	2890	2305	1864	1744	1562
70-74	4636	5463	5979	5682	5455	4892	4170	3332	2735	2521	2253
75-79	3971	5096	6076	6661	6517	6021	5607	4718	3855	3644	2997
80-84	2730	3596	4901	5531	6048	6511	6326	5978	5176	4957	4355
85-89	2489	2304	3527	4008	4381	5321	5805	6696	6259	6540	5806
90-	706	2573	1937	2040	2003	3554	4041	4873	5647	6894	6715

The numbers of deaths from cancer represent count data, and as such, statistical models for counts, rates or proportions are appropriate. Cancer mortality rates have often been modeled using a classical age-period-cohort model, which is a type of Poisson regression model, and was used to make predictions for lung cancer mortality rates in England and Wales (Osmond, 1985), for example. In particular, for the data in Tables 1 and 2, there are 16 age groups, 11 periods (the 5 year time intervals), and 26 cohorts. Individual cohorts are represented as diagonal slices in the Table. For example, in Table 2, two cohorts are identified by boldface type. The oldest cohort includes those individuals who where were 90 years or older in the period labeled 1950, and this is the only period in which data was recorded for this cohort. The youngest cohort includes those individuals who were 15-19 in the period labeled 2000, and there is again only one year of incidence data for this cohort. There are 6 cohorts which include a maximum of 11 periods of incidence data.

Let $i \in (1,2,\ldots,16)$ index age group, where age group 1 includes 15-19 year olds, age group 2 includes 20-24 year olds, and so on; $j \in (1,2,\ldots,11)$ index 5 year period, with period 1 centered at 1950, period 2 centered at 1955, and so on; and $k \in (1,2,\ldots,26)$ index cohort, where, for example, cohort 26 includes individuals who were 15-19 in 2000, cohort 2 includes individuals 85-89 in 1950, and so on.

Let Y_{ijk} denote the number of cases in age group i, period j and cohort k. The classical age-period-cohort model assumes that Y_{ijk} is a Poisson random variable with mean λ_{ijk}, where

$$\log(\lambda_{ijk}) = \log(n_{ijk}) + \alpha_i + \beta_j + \gamma_k. \quad (1)$$

Here α_i, β_j and γ_k are the effects of age group i, period j and cohort k respectively. The size of the population at risk, assumed to be known without error from census data, is denoted as n_{ijk}, and was used to transform the raw incidence data in Table 1 to the rates in Table 2. Inclusion of the offsets n_{ijk} in the model for the Poisson mean implies that we are effectively modeling incidence rates λ_{ijk} / n_{ijk}, thereby correcting for the number at risk.

It is clear that the parameterization is not identifiable, as we are using three co-ordinates to index into a two dimensional Table of counts. In particular, $k = 16 - i + j$.

Detailed discussions of this model, including identifiability issues, are included, for example, in Osmond and Gardner (1982), Clayton and Schifflers (1987a, b), and Holford (1991), and various methods have been suggested to overcome the non-identifiability problem, for example, imposing constraints on the parameters (Osmond & Gardner, 1982; Holford, 1991), or restricting consideration to certain estimable functions of the parameters (Clayton & Schifflers, 1987a; Holford, 1991). Clayton & Schifflers (1987a, b) advised the use of a reduced age-period or age-cohort model whenever possible and the use of the full age-period-cohort model only when no other model provides a satisfactory fit. Tango (1985) showed that nonlinear effect parameters can be uniquely determined by imposing restrictions on each block of parameters, for example, $\sum \alpha_i = \sum \beta_j = \sum \gamma_l = 0$, with the nonlinear age effects being specified as:

$$\tilde{\alpha}_j = \alpha_j - \frac{\sum_{j=1}^{A} L(j,A)\alpha_j}{\sum_{j=1}^{A} L(j,A)^2}, \quad (2)$$

$$\text{where } L(j,A) = j - \left(\frac{A+1}{2}\right).$$

It is important to note that while individual age, period and cohort parameters are not identifiable, forward prediction is possible (Holford, 1985).

Different cohorts are typically unequally represented in age-period-cohort data. In the present case, there are single observations on cohorts 1 and 26, two observations on cohorts 2 and 25, eleven observations on each of cohorts 11 through 16, and so on. Therefore, the precision of estimated cohort effects will differ markedly, which has important consequences for prediction. For example, simple predictive

models that carry forward estimated cohort effects may lead to predictions with a high degree of variability. Recently, Bayesian models have been used to smooth predictions by incorporating a priori beliefs about the smoothness of the model parameters. Berzuini and Clayton (1994) predicted lung cancer mortality rates using a Bayesian age-period-cohort model. Besag, et al. (1995) fit a Bayesian logistic regression to prostate cancer mortality rates in the USA, with age, period and cohort as explanatory variables, and Bray (2000, 2002) used Gaussian autoregressive priors for incidence rates of Hodgkin's disease.

This paper is organized as follows: A dynamic Poisson model and a dynamic age-period-cohort model are specified, Markov Chain Monte Carlo is reviewed and the estimation method is discussed in detail, a prediction method is described, and the result of the analysis of Japanese cancer data is provided. Finally, concluding remarks are given.

Model Specification

Throughout in this section, y_t denotes the *t*-th in a sequence of observations, $t = 1, \ldots, T$, θ_t is a *p*-dimensional parameter vector, F_t is a known *p*-dimensional vector of regressors, G_t is a known $p \times p$ matrix, w_t is a *p*-dimensional vector of errors with covariance matrix *W*, and $g(\cdot)$ is a link function.

Dynamic Poisson Model

A dynamic Poisson model is a state space time series model consisting of observation and system equations, as follows:

Observation equation:

$$P(y_t \mid \lambda_t) = \frac{\exp(-\lambda_t)\lambda_t^{y_t}}{y_t!}, g(\lambda_t) = F_t'\theta_t. \quad (3)$$

System (state) equation:

$$\theta_t = G_t\theta_{t-1} + \eta_t, \quad \eta_t \sim N_p[0, W]. \quad (4)$$

When there is no system equation, the dynamic Poisson model becomes the usual Poisson regression model. The dynamic Poisson model is a particular case of the general state space model, discussed, for example, in Kitagawa and Gersch (1996). There is currently much activity in the development of algorithms for general state space models, focusing primarily on so-called particle filters. For example, see Kitagawa (1998) or Doucet, et al. (2001).

Dynamic Age-Period-Cohort Model

To incorporate the age-period-cohort model within the dynamic Poisson model, let $i(t)$, $j(t)$ and $k(t)$ denote the age, period and cohort indices associated with observation Y_t, and denote the associated age, period and cohort effects as $\alpha_{i(t)}$, $\beta_{j(t)}$ and $\gamma_{i(t)}$. Assume $F_t' = (\log(n_t), 1, 1, 1)$, where n_t is the number at risk for observation *t*, and let $\theta_t' = (1, \alpha_{i(t)}, \beta_{j(t)}, \gamma_{k(t)})$.

Let $\eta_t' = (0, \eta^{\alpha}_{i(t)}, \eta^{\beta}_{j(t)}, \eta^{\gamma}_{k(t)})$, and $G_i = I$ be the 4×4 identity matrix. In this case the dynamic state space model is specified by the following observation and system equations.

Observation equation:

$$P(y_t \mid \lambda_t) = \frac{\exp(-\lambda_t)\lambda_t^{y_t}}{y_t!}, \quad \log(\lambda_t) = \log(n_t) + \alpha_{i(t)} + \beta_{j(t)} + \gamma_{k(t)}. \quad (5)$$

System (state) equation:

$$\alpha_{i(t)} = \alpha_{i(t)-1} + \eta^{\alpha}_{i(t)}, \quad \eta^{\alpha}_{i(t)} \sim N[0, W_\alpha],$$
$$\beta_{j(t)} = \beta_{j(t)-1} + \eta^{\beta}_{j(t)}, \quad \eta^{\beta}_{j(t)} \sim N[0, W_\beta], \quad (6)$$
$$\gamma_{k(t)} = \gamma_{k(t)-1} + \eta^{\gamma}_{k(t)}, \quad \eta^{\gamma}_{k(t)} \sim N[0, W_\gamma].$$

This assumes that the system equation corresponds to three independently evolving random walks for age, period and cohort effect - the same model as considered by Knorr-Held and Rainer (2001). The state variables $\{\alpha_{i(t)}, \beta_{j(t)}, \gamma_{k(t)}, t = 1, \ldots, T\}$ take the form of time varying parameters, while the variances W_α, W_β, and W_γ are assumed not to depend on time.

In addition to the observation and system equations, a Bayesian dynamic age-period-cohort model requires the specification of prior distributions for model parameters. However, because of the recursive nature of the state equation, the Bayesian model requires prior distributions only for W_α, W_β, W_γ, α_0, β_0, γ_0. Where $N[\mu,\sigma^2]$ denotes the normal distribution with mean μ and variance σ^2 and $IG[\nu,S]$ denotes the inverse gamma distribution with scale parameter S and shape parameter ν, assume the following prior distributions: $\alpha_0 \sim N[\mu_\alpha, R_\alpha]$, $\beta_0 \sim N[\mu_\beta, R_\beta]$, $\gamma_0 \sim N[\mu_\gamma, R_\gamma]$, $W_\alpha \sim IG[\nu_\alpha, S_\alpha]$, $W_\beta \sim IG[\nu_\beta, S_\beta]$ and $W_\gamma \sim IG[\nu_\gamma, S_\gamma]$. Non-informative priors are achieved by letting R_α^{-1}, R_β^{-1}, R_γ^{-1}, ν_α, ν_β, ν_γ, S_α, S_β, and $S_\gamma \to 0$. Other prior was applied to the dynamic age-period-cohort model, but the result was similar to non-informative priors.

Where there are A age groups, P periods and C cohorts, it follows that the joint posterior for $\alpha_0,\alpha_1,\ldots,\alpha_A$, $\beta_0,\beta_1,\ldots,\beta_P$, $\gamma_0,\gamma_1,\ldots,\gamma_C$, W_α, W_β and W_γ is given by:

$$\pi(\alpha_0,\ldots,\alpha_A,\beta_0,\ldots,\beta_P,\gamma_0,\ldots,\gamma_C,W_\alpha,W_\beta,W_\gamma \mid y) \propto$$
$$\prod_{t=1}^{T}\frac{\exp(-\lambda_t)\times\lambda_t^{y_t}}{y_t!}\times\prod_{j=1}^{A}(W_\alpha)^{\frac{1}{2}}\exp(-\frac{1}{2}W_\alpha^{-1}(\alpha_j-\alpha_{j-1}))$$
$$\times\prod_{k=1}^{P}(W_\beta)^{\frac{1}{2}}\exp(-\frac{1}{2}W_\beta^{-1}(\beta_k-\beta_{k-1}))$$
$$\times\prod_{l=1}^{C}(W_\gamma)^{\frac{1}{2}}\exp(-\frac{1}{2}W_\gamma^{-1}(\gamma_l-\gamma_{l-1}))$$
$$\times\exp\left[-\frac{1}{2}R_\alpha^{-1}(\alpha_0-\mu_\alpha)^2\right]\times\exp\left[-\frac{1}{2}R_\beta^{-1}(\beta_0-\mu_\beta)^2\right]$$
$$\times\exp\left[-\frac{1}{2}R_\gamma^{-1}(\gamma_0-\mu_\gamma)^2\right]$$
$$\times f_{IG}(W_\alpha;\nu_\alpha,S_\alpha)\times f_{IG}(W_\beta;\nu_\beta,S_\beta)\times f_{IG}(W_\gamma;\nu_\gamma,S_\gamma). \quad (7)$$

where $\lambda_t = n_t\exp(\alpha_{i(t)}+\beta_{j(t)}+\gamma_{k(t)})$, and $f_{IG}(\cdot;\nu,S)$ is the inverse gamma density function with parameters ν and S.

More generally, the independence structure of the priors could be removed by assuming that $W=(W_\alpha,W_\beta,W_\gamma)'$ follows a trivariate inverse Wishart distribution with kernel $|W|^{-\frac{1}{2}\nu W}\exp\left(-\frac{1}{2}tr\left(W^{-1}S_W\right)\right)$, and that $(\alpha_0,\beta_0,\gamma_0)'$ has a trivariate Gaussian distribution with mean vector μ and covariance matrix R.

Estimation Method

A Bayesian approach is taken to estimate parameters using posterior means. As analytical calculation of integrals with respect to the posterior distribution is typically intractable, a Markov chain Monte Carlo method has been used to approximate the posterior means. The Gibbs sampler was used to generate samples from the joint posterior distribution. General discussions of the Gibbs sampler are provided, for example, by Geman and Geman (1984) and Gammerman (1997). The WinBugs implementation was used to carry out computations (Spiegelhalter, et al., 2003), with non-informative hyper-priors referred to previously.

As described, Tango (1985) was followed in defining nonlinear age and period effects after applying zero sum constraints. Such mean constraints were also used by Berzuini and Clayton (1994) and Bray (2000, 2002).

In order to assess convergence of the sampler, two chains of 10,000 iterations were run from different initial values and time series plots of the MCMC samples were examined. As an example, Figure 1 shows a plot of the sampled values of γ_1, for the male stomach cancer data. And Figure 2 shows the autocorrelation function of γ_1. The plot suggests that convergence was achieved, and it was confirmed that all other parameters were convergent in the same manner.

Prediction

Osmond (1985) used a standard age-period-cohort model (1) to project lung cancer mortality rates for England and Wales. In this method, unknown period and cohort effects for

future periods are estimated using linear regression, while estimated age effects need not be extrapolated.

Figure 1: Time series plots of MCMC iterations for γ_1.

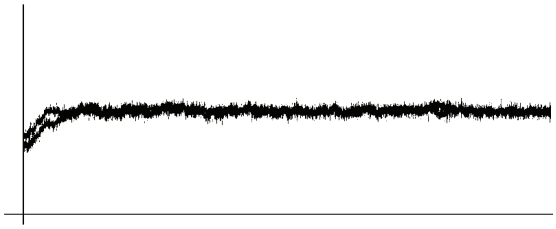
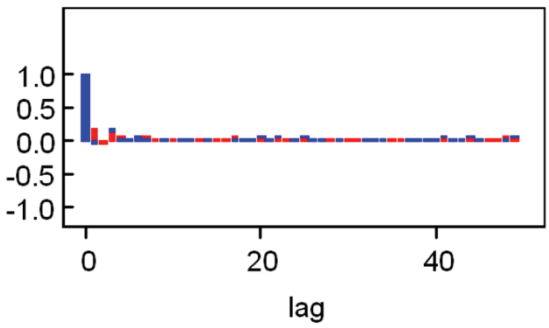


Figure 2: The autocorrelation function of γ_1.



A criticism of the regression, while estimated age effects need not be extrapolated. A criticism of the method is the arbitrariness introduced by the choice of past values to use in the regression, and the type of regression model (e.g., weighted or unweighted).

More recently, parametric bootstrap methods have been used to make projections, for example, by Berzuini and Clayton (1994) and Bray (2000, 2002). In particular, to obtain a prediction for $\tilde{\lambda}_{T+1}$ given data $Y_1,\ldots,Y_T$, sample

$$\tilde{\theta}_{T+1} \sim N\left[G_{T+1}\hat{\theta}_T, \hat{W}\right], \quad (8)$$

where $\hat{W}$ and $\hat{\theta}_T$ are estimates based on $Y_1,\ldots,Y_T$. Then set

$$\tilde{\lambda}_{T+1} = F'_{T+1}\tilde{\theta}_{T+1}. \quad (9)$$

This process is repeated J times leading to $\left\{\tilde{\lambda}^{(j)}_{T+1}, j=1,\ldots,J\right\}$, which are then averaged to provide the overall prediction $\tilde{\lambda}_{T+1}$ of Y_{T+1}. The prediction at time $T+2$ is then based on the combined data $Y_1,\ldots,Y_T$ and prediction $\tilde{\lambda}_{T+1}$. In carrying out the calculations, $J=100$ was used. The Table 3 shows predicted values and simulated 95% prediction intervals for male stomach cancer in 2005.

Predictions were also made using the traditional age-period-cohort model (1). To estimate age and period effects, a simple linear regression was used on one previous period or age group.

Table 3: Predicted value and simulated 95% prediction intervals for male stomach cancer in 2005

Age Group	Lower	Predicted Value	Upper
15-19	5.580305162	5.745869588	5.91469553
20-24	17.96144371	18.07019342	18.17972061
25-29	45.17424256	45.46220645	45.7517975
30-34	86.88730352	87.43865363	87.9901386
35-39	170.0798945	171.1925396	172.2686358
40-44	320.5989165	322.6740741	324.7104773
45-49	762.0522524	766.9412402	771.9716268
50-54	1481.543376	1491.174459	1500.812711
55-59	2195.522161	2209.505762	2223.767449
60-64	3288.881735	3310300914	3331.643898
65-69	4186.238369	4213.082323	4240.180761
70-74	4727.281932	4757.428491	4788.496251
75-79	4207.928407	4234.477476	4262.194609
80-84	2957.042947	2976.174262	2995.429859
85-89	1736.78588	1747.876571	1759.049125
90-	624.2118163	628.1242619	632.1725958

To assess the adequacy of models for fitting and prediction, the first nine periods for model fitting were used and projections for the tenth and eleventh periods were constructed. Estimates and predictions were compared with observed counts using the following estimates of residual and prediction error.

$$\text{Scaled residual error} = \sum_{i=1}^{9} \frac{(y_i - \hat{y}_i)^2}{\hat{y}_i}, \quad (10)$$

$$\text{Scaled prediction error} = \sum_{j=1}^{11} \frac{(y_j - \tilde{y}_j)^2}{\tilde{y}_j}, \quad (11)$$

where $\hat{y}_i$ is the fitted value for period i and $\tilde{y}_j$ is the predicted value for period j. Table 4 shows these estimates of residual and prediction errors for the age-period-cohort model and a dynamic Poisson models. The estimates of residual error are consistently a bit smaller for the age-period-cohort model, as compared to the dynamic Poisson model.

For the male stomach cancer data, the estimated prediction error is a bit smaller using the age-period-cohort model. However, in the other three cases, the prediction error is smaller using the dynamic Poisson model, and dramatically so in the case of male lung cancer. This suggests that the dynamic Poisson model is the preferred method for making future predictions.

The latter two columns of Tables 1, 5, 6 and 7 contain predictions of lung and stomach cancer rates to the periods centered at 2005 and 2010 using the dynamic Poisson model.

Modeling Variance Heterogeneity

Thus far, we have assumed constant variances for each of the system variables $\alpha_{i(t)}$, $\beta_{i(t)}$, and $\gamma_{i(t)}$ of the dynamic Poisson model. Under this assumption, we have observed that some estimated variances were very large, leading to imprecision of predictions. For example, children born during the years when war occurred, might be faced high risk, then the cohort effect become extremely large than children born at another time. In an attempt to reduce the variability in predictions, the model has been generalized to include non-constant variance, as follows.

$$\begin{aligned}
\alpha_{i(t)} &= \alpha_{i(t)-1} + \eta^{\alpha}_{i(t)}, \quad \eta^{\alpha}_{i(t)} \sim N\left[0, W_{\alpha_{i(t)}}\right], \\
\beta_{j(t)} &= \beta_{j(t)-1} + \eta^{\beta}_{j(t)}, \quad \eta^{\beta}_{j(t)} \sim N\left[0, W_{\beta_{j(t)}}\right], \quad (12) \\
\gamma_{k(t)} &= \gamma_{k(t)-1} + \eta^{\gamma}_{k(t)}, \quad \eta^{\gamma}_{k(t)} \sim N\left[0, W_{\gamma_{k(t)}}\right],
\end{aligned}$$

Again using non-informative priors, this led, for example, to estimates $\hat{W}_{\beta_1}, \hat{W}_{\beta_2}, \ldots, \hat{W}_{\beta_P}$ for the P period effect variances, which were averaged to produce an overall estimate $\tilde{W}_{\beta} = {}^{1}\!/\!_{P} \sum_{j=1}^{P} \hat{W}_{\beta_j}$. This latter quantity was then used to predict the \$N+1\$'st period effect, as

$$\tilde{\beta}_{j(N+1)} \sim N\left[\tilde{\beta}_{j(N)}, \tilde{W}_{\beta}\right], \quad (13)$$

For moderately large P, $\tilde{W}_{\beta}$ should typically be less than $\hat{W}_{\beta}$, thereby increasing the stability of

Table 4: Scaled residual and scaled prediction error

		Scaled Residual	Scaled Prediction Error
Stomach Man	Dynamic Poisson model	6.62314	79.83967
	Age-Period-Cohort model	6.575341	73.35086
Stomach Woman	Dynamic Poisson model	8.740782	80.4833
	Age-Period-Cohort model	8.169002	100.3244
Lung Man	Dynamic Poisson model	2.066267	48.64581
	Age-Period-Cohort model	2.26717	234.6294
Lung Woman	Dynamic Poisson model	1.150069	49.20646
	Age-Period-Cohort model	1.12215	61.80142

the forecast. Indeed, a small $\widetilde{W}_\beta$ could be obtained when the number of death at a specific period group was increased. Table 5 shows the estimated variances for the fixed and heterogeneous variance models.

Conclusion

In the data sets considered, it was observed that the classical age-period-cohort model provided a better fit to past data than did the dynamic age-period-cohort model. On the other hand, when the focus is on making projections, it was found that the classical age-period-cohort model, which makes strong parametric and regression assumptions, was out performed by the dynamic model. Under the assumption of homogeneous error variances in the system equations of the dynamic age-period-cohort model, large standard errors were observed in several cases. It is possible that at least some of this imprecision is the result of natural variation in the Monte Carlo algorithm. Further research will focus on incorporating heterogeneous variances into the model.

The focus has been on the dynamic Poisson model, but the dynamic model can be extended in a straightforward manner to incorporate generalized linear models.

Table 5: The value of $\widetilde{W}_\beta$ and $\hat{W}_\beta$ to each data in Japan

		Variance For Period	Variance For Cohort
Stomach Man	Homogeneous	0.001324854 $\hat{W}_\beta$	0.034891835 $\hat{W}_\gamma$
	Heterogeneous	0.003901684 $\widetilde{W}_\beta$	0.02093981 $\widetilde{W}_\gamma$
Stomach Woman	Homogeneous	0.001282216 $\hat{W}_\beta$	0.034843206 $\hat{W}_\gamma$
	Heterogeneous	0.003719764 $\widetilde{W}_\beta$	0.029620221 $\widetilde{W}_\gamma$
Lung Man	Homogeneous	0.035637919 $\hat{W}_\beta$	0.049188392 $\hat{W}_\gamma$
	Heterogeneous	0.031894849 $\widetilde{W}_\beta$	0.040851123 $\widetilde{W}_\gamma$
Lung Woman	Homogeneous	0.049800797 $\hat{W}_\beta$	0.039339103 $\hat{W}_\gamma$
	Heterogeneous	0.043756821 $\widetilde{W}_\beta$	0.028132685 $\widetilde{W}_\gamma$

References

Berger, J. (1985) *Statistical decision theory and Bayesian analysis* (2nd ed). NY:Springer Verlag.

Berzuini, C., & Clayton, D. (1994). Bayesian analysis of survival on multiple time scales. *Statistics in Medicine*, *13*, 823-838.

Besag, J., Green, P., Higdon, D., & Mengersen, K. (1995) Bayesian computation and stochastic systems. *Statistical Science*, *10*, 3-66.

Bray, I., Brennan, P., & Boffetta, P. (2000). Projections of alcohol- and tobacco-related cancer mortality in central Europe. *International Journal of Cancer*, *87*, 122-128.

Bray, I. (2002). Application of Markov chain Monte Carlo methods to projecting cancer incidence and mortality. *Journal of the Royal Statistical Society*, *51*, Part 2, 151-164.

Clayton, D., & Schifflers, E. (1987a). Models for temporal variation in cancer rates II: age-period-cohort models. *Statistics in Medicine*, *6*, 469-481.

Clayton, D., & Schifflers, E. (1987b). Models for temporal variation in cancer rates I: age-period-cohort models. *Statistics in Medicine*, *6*, 449-467.

Doucet, A., Freitas, N., & Gordon, N., eds. (2001). *Sequential Monte Carlo methods in practice*. NY: Springer-Verlag.

Frome, E. L. (1983). The analysis of rates using Poisson regression models. *Biometries*, *39*, 665-674.

Gamerman, D. (1998).Markov chain Monte Carlo for dynamic generalized linear models. *Biometrika*, *85*, 215-227.

Geman, S., & Geman, D. (1984). Stochastic relaxation, Gibbs distributions and the Bayesian restoration of images. *IEEE Transcation on Pattern Analysis and Machine Intelligence*, *6*, 721-741.

Gamerman, D. (1997). *Markov chain Monte Carlo*. Boca Raton, FL: Chapman & Hall/CRC.

Hastings, W. K. (1970). Monte Carlo sampling methods using Markov chains and their applications. *Biometrika*, *57*, 97-109.

Holford, T. R. (1985). An alternative approach to statistical age-period-cohort analysis. *Journal of Chronic Diseases*, *38*, 831-836.

Holford, T. R. (1991). Understanding the effects of age, period and cohort on incidence and mortality rates. *Annual Review of Public Health*, *12*, 425-457.

Kitagawa, G. (1998). Self-organising state space model. *Journal of the American Statistical Association*, *93*, 1203-1215.

Kitagawa, G., & Gersch, W. (1996). *Smoothness Priors Analysis of Time Series*, NY: Springer-Verlag.

Knorr-Held, L., & Rainer, E. (2001) Projection of lung cancer mortality in West Germany: a case study in Bayesian prediction. *Biostatistics*, *2*, 109-129.

Nelder, & Wedderburn, (1972). Generalized linear models. *Journal of the Royal Statistical Society, Series A*, *135*, 370-384.

Osmond, C., & Gardner, M. J. (1982). Age, period and cohort models applied to cancer mortality rates. *Statistics in Medicine*, *1*, 245-259.

Spiegelhalter, D., Thomas, A., & Best, N. (2003). *WinBUGS Version 1.3 User Manual* Medical Research Council Biostatistics Unit, Cambridge, UK.

Tango, T. (1985) Estimation of age, period and cohort effects (in Japanese). *Applied statistics in Japanese*, *14*, 45-49.

West, M., Harrison, P. J., & Migon, H. S. (1985). Dynamic generalized linear models and Bayesian forecasting (with discussion). *Journal of the American Statistical Association*, *80*, 73-83.

West, M., & Harrison, P. J. (1997). *Bayesian Forecasting and Dynamic Models.* (2nd ed.). NY: Springer Verlag.

Appendix

Age Group	Table 6: Stomach cancer - females, count 5 year period centered at												
	1950	1955	1960	1965	1970	1975	1980	1985	1990	1995	2000	2005	2010
15-19	9	9	10	11	23	8	5	5	1	5	4	3	3
20-24	39	43	55	65	86	63	43	21	26	10	9	15	15
25-29	115	119	153	198	232	246	179	123	75	64	48	43	54
30-34	242	289	389	432	380	465	431	293	180	126	98	90	93
35-39	415	495	597	672	651	629	619	561	387	237	162	204	152
40-44	679	784	857	857	937	874	745	693	677	448	306	330	318
45-49	902	982	1152	1139	1194	1152	1003	870	714	831	596	641	489
50-54	1166	1331	1398	1588	1430	1421	1296	1116	938	824	890	997	881
55-59	1490	1675	1779	1836	1956	1658	1634	1423	1180	1072	987	1251	1272
60-64	1891	1951	2124	2217	2339	2274	2031	1903	1583	1432	1291	1533	1657
65-69	2139	2337	2366	2592	2766	2726	2579	2117	1991	1910	1638	1895	1972
70-74	1743	2111	2462	2628	2976	3009	2957	2706	2211	2340	2131	2091	2221
75-79	959	1517	1858	2036	2362	2653	2974	2864	2727	2545	2610	2247	2199
80-84	306	622	964	1076	1354	1596	2076	2388	2606	2976	2820	2123	2033
85-89	81	132	268	343	420	569	845	1301	1693	2194	2685	1557	1502
90-	9	22	45	63	74	123	202	373	585	1054	1592	1503	775

Age Group	Table 7: Lung cancer - males, count 5 year period centered at												
	1950	1955	1960	1965	1970	1975	1980	1985	1990	1995	2000	2005	2010
15-19	9	5	10	6	5	1	5	1	1	5	4	5	5
20-24	4	9	9	18	11	10	4	5	5	5	5	7	7
25-29	6	12	17	21	18	22	23	12	17	14	10	13	14
30-34	3	14	27	34	34	42	49	41	39	49	57	28	29
35-39	10	19	31	68	66	80	115	157	149	102	97	101	67
40-44	20	47	62	99	168	202	207	275	363	287	288	282	247
45-49	43	105	174	190	226	415	450	450	577	757	635	710	627
50-54	85	195	323	407	431	451	933	983	918	1138	1463	1444	1449
55-59	107	250	550	618	826	904	1218	1818	2020	1831	2210	2453	2578
60-64	153	362	669	1078	1249	1534	1856	2321	3655	3760	3352	3787	4310
65-69	175	398	734	1135	1742	2180	2727	3172	4165	6044	5804	4898	5772
70-74	111	306	603	935	1501	2408	3316	4228	4675	6105	8193	6549	6157
75-79	58	125	309	570	854	1636	2784	4018	5022	5703	7326	6881	6576
80-84	9	36	106	198	306	589	1340	2355	3495	4730	5445	4958	5375
85-89	0	6	17	27	63	142	342	849	1466	2206	3146	2580	2720
90-	1	0	5	3	8	15	74	153	313	660	1017	790	874

Age Group	Table 8: Lung cancer - females, count 5 year period centered at												
	1950	1955	1960	1965	1970	1975	1980	1985	1990	1995	2000	2005	2010
15-19	5	5	5	6	5	0	0	1	1	0	0	4	4
20-24	1	5	9	10	6	0	4	5	0	5	0	6	6
25-29	4	8	5	13	10	17	14	8	8	5	10	9	9
30-34	6	20	31	37	34	33	43	18	27	36	30	25	26
35-39	11	31	43	68	49	72	78	96	89	65	75	61	54
40-44	19	50	50	88	107	98	121	151	164	169	146	147	111
45-49	22	67	126	106	166	200	207	220	258	379	261	299	276
50-54	46	85	139	232	211	297	350	374	425	551	667	557	542
55-59	50	106	194	293	337	412	492	587	618	733	806	908	822
60-64	49	120	250	356	482	545	688	843	886	1020	1069	1284	1368
65-69	49	139	251	374	506	671	932	1044	1239	1416	1601	1751	1892
70-74	44	113	203	344	483	728	1083	1353	1562	1767	2018	2239	2384
75-79	19	51	147	270	344	557	1001	1392	1811	2205	2459	2903	2894
80-84	6	16	63	109	194	277	610	1061	1382	2104	2453	3672	3113
85-89	0	5	17	27	43	117	190	480	864	1320	1989	3538	3327
90-	2	0	6	3	10	21	54	125	285	580	1096	2253	2493