# Evaluation of Bias, Precision and Accuracy of Mortality Cause Proportion Estimators from Ring Recovery Data

**Michael Schaub**

**Abstract** Knowledge about proportions of specific mortality causes is important for the design of efficient conservation measures or the determination of harvest regulations. Unfortunately, these proportions are difficult to estimate. We (Schaub and Pradel 2004) have recently introduced a multistate capture-recapture model that allows one to estimate proportions of specific mortality causes from recoveries of dead animals with known cause of death. However, parameter estimation was found to be difficult, because the likelihood surface of the model relative to most parameters has a flat ridge, unless the proportions of mortality causes vary with time and the cause-specific recovery rates are constant. These conditions are likely to be violated in most empirical situations. For the application of this model, it is therefore important to study the sensitivity of parameter estimates to violations of these assumptions. I use a Bayesian implementation of the model to evaluate bias, precision and accuracy of parameter estimates under variable means and temporal variation of mortality cause proportions and recovery rates. Survival rate estimates were unbiased in all scenarios. Bias and precision of the proportion of mortality causes and of the cause-specific recovery probabilities decreased with increasing temporal variance of the proportion of mortality causes while their accuracy increased. The bias of these estimates also decreased with decreasing difference between cause-specific recovery probabilities and with decreasing temporal variation of them. Moreover, informative priors affected the posterior distribution of the parameters when temporal variation in the proportion of mortality causes was low. Temporal variance of the proportion of mortality causes could be estimated reliably regardless of bias. This result is important, since it allows one to assess whether accuracy of the estimates of mortality proportions is acceptable for the objectives of a study. The bias of the naïve estimator (quotient of the number of animals reported dying from a particular cause to the total number reported altogether) was usually much larger than the bias of the corresponding estimator from the multistate model. In conclusion, a careful

M. Schaub (✉)
Division of Conservation Biology, Zoological Institute, University of Bern, Baltzerstrasse 6, CH-3012 Bern, Switzerland; Swiss Ornithological Institute, CH-6204 Sempach, Switzerland
e-mail: michael.schaub@nat.unibe.ch

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application of the multistate capture-recapture model can give useful information about the proportion of mortality causes that is otherwise hard to obtain.

**Keywords** Bayesian · Bias · Multistate Model · Proportion of Mortality Causes · Recovery Probability · Simulation

## 1 Introduction

Overall mortality rates are typically due to a variety of mortality causes such as predation, disease, accidents or human harvest, and each of these may influence population dynamics differentially. If the frequency of these causes changes, overall mortality is likely to change also and thereby population growth rate. Thus, studying the proportion of different sources of mortality is important for understanding proximate causes of population dynamics.

Unless animals are radio-tagged (e.g. Bro et al. 2001; Buner and Schaub 2008), the importance of a particular mortality cause is difficult to assess. This is because the probability of finding an animal that has died due to a specific mortality cause depends on the mortality cause. For example, animals that die from human-related mortality causes such as hunting or accidents with cars or windows are usually much more likely to be found and reported than animals that die from predation, disease or starvation. Therefore, estimates of the proportion of mortality causes based on the ratio of the number of animals reported dead from a particular cause and the total number reported (Newton et al. 1999; Hüppop and Hüppop 2002) are likely to be seriously biased.

Recently, we (Schaub and Pradel 2004) introduced a multistate capture-recapture model which allows estimation of overall survival rates (S) and the proportion ($\alpha$) of animals dying from a particular mortality cause under question from recovered dead animal whose cause of death is known. In this model, which I term cause-specific mortality model, the probability of finding and reporting an animal that died from a particular cause of death is estimated, allowing to estimate the parameters of interest (S, $\alpha$) without bias. The parameters of interest are intrinsically identifiable, if $\alpha$ is time-dependent and the cause-specific recovery rates constant across time (Schaub and Lebreton 2004). However, even in intrinsically identifiable models the model likelihood relative to $\alpha$ and the recovery rates has a flat ridge, such that the estimation of these parameters can be difficult (see discussion in Schaub and Pradel 2004). The problem arises because a nested model with fewer parameters, where $\alpha$ is not variable over time, is intrinsically unidentifiable (Schaub and Lebreton 2004). Therefore, the information matrix of the model is near-singular (Catchpole et al. 2001; Catchpole and Morgan 2001) and the model provides only unbiased estimates when $\alpha$ varies across time. However, it is not clear how large the temporal variation of $\alpha$ needs to be to obtain accurate parameter estimates.

A further important issue is that often additional information is available on some of the parameters in the model. For example it is known that the recovery rate of

hunted individuals is larger than the recovery rate of individuals dying from natural causes. In this situation, it may well be that parameter estimation from a Bayesian analysis is less delicate owing to the inclusion of such additional information via the prior distributions or order constraints. I therefore evaluated how much the estimates can be improved through the use of additional information.

Another challenge is that the parameters ($\alpha$, recovery rates) are not separately estimable when the recovery probabilities are time-dependent. One must therefore fit a model with constant recovery probabilities, yet is it not clear how strongly $\alpha$ and the recovery rates will be biased when there is such variation.

The aim of the paper is to enhance the understanding of the cause-specific mortality model to facilitate its application. I developed a Bayesian implementation of the model and used simulation to explore the conditions when the model provides reasonably accurate parameter estimates. The sensitivity of the parameter estimates under the model is explored along several dimensions, (i) magnitude of temporal variance of $\alpha$, (ii) magnitude of temporal variance of the recovery rates, (iii) magnitude of cause-specific recovery rates, (iv) sample size (numbers of data years), and (v) use of additional information. I also studied bias and precision of the naïve estimator (quotient of the number of animals reported dying from a particular cause to the total number reported altogether), to evaluate how much the estimates from the cause-specific mortality model improve over the naïve estimates. Based on the results I propose guidelines for the practical application of the model.

## 2 Material and Methods

### *2.1 The Cause Specific Mortality Model*

The data required for the cause-specific mortality model (Schaub and Pradel 2004) are capture-mark-recovery data where the cause of death of each recovered animal is known without error. Such data is frequently available, for example in the database of the European ringing schemes. The various causes of death observed among recovered individuals are then allocated to the two groups A and B, where group A refers to the mortality cause one is particularly interested in (e.g. hunting) and B refers to all other mortality causes (not A). A multistate capture-recapture history is then constructed for each individual. For example, capture history 010A0 denotes an individual that was marked in the second year and died from cause A and was recovered in year four.

To obtain estimates of $\alpha$, survival and the recovery rates, I used a multistate capture-recapture model with the three states "alive", "died due to cause A", and "died due to cause B". The model is presented here with a matrix of transition probabilities and a state-specific vector of "sighting" probabilities. Note that states are from top to bottom (states of arrival) and from left to right (states of departure) in the order as indicated above:

$$\begin{bmatrix} S_t & (1-S_t)\alpha_t & (1-S_t)(1-\alpha_t) \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} 0 \\ \lambda_{A,t} \\ \lambda_{B,t} \end{bmatrix}.$$

Here, $S_t$ is the probability that an individual survives from time $t$ to time $t+1$; $\alpha_t$ is the probability that the mortality cause of an individual is A if it dies between $t$ and $t+1$; and $\lambda_{A,t}$ and $\lambda_{B,t}$ are the probabilities that an individual dying from cause A or B, respectively, between $t$ and $t+1$ is found and reported. A more detailed description of the model is presented in Schaub and Pradel (2004) and in Schaub and Lebreton (2004).

While we used a frequentist approach in Schaub and Pradel (2004) and Schaub and Lebreton (2004), here I apply a Bayesian analysis. The likelihood is formed from the products of multinomial distributions whose cell probabilities are functions of S, $\alpha$, $\lambda_A$ and $\lambda_B$ (see also Brooks et al. (2000) for an equivalent formulation of a classic ring-recovery model). I used several different priors (see below more details) and Markov chain Monte Carlo (MCMC) methods to sample from the posterior distribution.

## 2.2 Intrinsic Identifiability of the Model

Evaluation of the model using formal methods (Catchpole and Morgan 1997; Catchpole et al. 2002; Gimenez et al. 2003) has shown that only models in which $\alpha$ is time-dependent and the recovery probabilities are constant across time, i.e. models [S, $\alpha_t$, $\lambda_A$, $\lambda_B$] and [$S_t$, $\alpha_t$, $\lambda_A$, $\lambda_B$], are intrinsically identifiable. Other models, where either all parameters are constant across time or where one or both recovery probabilities are time-dependent, are not intrinsically identifiable (Schaub and Lebreton 2004). The fact that model [S, $\alpha$, $\lambda_A$, $\lambda_B$] is not fully identifiable (i.e. only the quantities S, $\alpha^*\lambda_A$, and $(1-\alpha)^*\lambda_B$ are separately estimable) can pose problems for the parameter estimation also under the two identifiable models: it is the time variation of $\alpha$ which renders $\alpha$, $\lambda_A$ and $\lambda_B$ separately estimable. One aim of my study was therefore to understand how large the temporal variation of $\alpha$ needs to be in order to get useful estimates of S, $\alpha$, $\lambda_A$, and $\lambda_B$.

## 2.3 Simulation Methods

To evaluate estimator performance I conducted a simulation study with different scenarios with 500 generated data sets each. Generation of a data set first required selection of those parameters values ($\theta$) that were variable across time. I chose the beta distribution to model temporal variation in a parameter. For time interval $t$, a $\theta_t$ was taken from a beta distribution with mean $\bar{\theta}$ and variance $\sigma_\theta^2$. The two parameters a and b of the beta distribution were calculated as $a = \bar{\theta}(\bar{\theta}(1-\bar{\theta})/\sigma_\theta^2 - 1)$, and $b = (1-\bar{\theta})(\bar{\theta}(1-\bar{\theta})/\sigma_\theta^2 - 1)$, respectively. Considering a specific number of study years,

then I assumed that 1000 individuals were newly marked in each year and created a multistate m-array (Burnham et al. 1987) using multinomial distributions. Parameter estimates were then obtained from analysis of the m-array under the cause-specific mortality model described above.

In a first set of simulations, I studied the effect of different means of $\alpha$ and the two recovery probabilities on the parameter estimates. I generated data from a model with constant survival and recovery probabilities and with time-dependent mortality cause probabilities [S, $\alpha_t$, $\lambda_A$, $\lambda_B$]. I considered five different mean values of $\alpha$ ($\bar{\alpha} = \{0.1, 0.35, 0.5, 0.65, 0.9\}$), seven different temporal variances of $\alpha$ ($\sigma_\alpha^2 = \{0.0001, 0.0005, 0.001, 0.005, 0.01, 0.05, 0.1\}$) and two different options of recovery probabilities. In the first option the values of the two recovery probabilities were closer ($\lambda_A = 0.2$, $\lambda_B = 0.1$) than in the second option ($\lambda_A = 0.25$, $\lambda_B = 0.05$). I considered 10 and 30 study years, whereas the mean value for survival ($S = 0.4$) remained the same in all scenarios. These values appeared to cover realistic levels of variation to be expected in practical applications of the model. Non-informative (uniform) $\beta(1,1)$ priors were considered for all parameters and all scenarios. In total I considered 140 different scenarios in this set.

In a second set of simulations, I studied the impact of the inclusion of additional information on the parameter estimates. I generated data under a model with constant survival and recovery probabilities and with time-dependent mortality cause probabilities [S, $\alpha_t$, $\lambda_A$, $\lambda_B$]. The mean values of survival ($S = 0.4$), the proportion dying ($\bar{\alpha} = 0.35$) and the number of study years (10) remained the same in all scenarios. I evaluated different magnitudes of temporal variances of $\alpha$ ($\sigma_\alpha^2 = \{0.0001, 0.0005, 0.001, 0.005, 0.01, 0.05, 0.1\}$), two different options of recovery probabilities ($\lambda_A = 0.2$, $\lambda_B = 0.1$, and $\lambda_A = 0.25$, $\lambda_B = 0.05$, respectively) and the inclusion of additional information. For the latter I used four possibilities. First, I assumed that no additional information is available and used non-informative $\beta(1,1)$ priors for all parameters. Second, I used a $\beta(1,1.857)$ prior for $\alpha$ and non-informative $\beta(1,1)$ priors for the other parameters. The $\beta(1,1.857)$ distribution is an almost linearly decreasing distribution with mean 0.35. This is exactly the same as the mean value of $\bar{\alpha}$ used to simulate the data. This prior distribution was chosen in order to explore the potential gain in parameter accuracy if the mean of $\alpha$ were known. Third, I used a uniform prior for $\alpha$ within the interval $\{0, 0.5\}$, and non-informative $\beta(1,1)$ priors for the other parameters. This choice was motivated because in practice it may be possible to have an idea about likely magnitude of $\alpha$. Fourth, additional structural information was incorporated into the model in form of an order constraint on the recovery probability. Typically, it will be known which mortality cause is associated with a higher recovery probability, which can be translated into an order constraint such as $\lambda_A > \lambda_B$ (specifically I used $\beta(1,1)$ priors for $\lambda_B$ and $\Delta$, and calculated $\lambda_A = \lambda_B + \Delta$). For example, in hunted species it may be sensible to assume that the recovery probability associated with a mortality cause related to human activity is larger than the recovery probabilities associated to other mortality causes (e.g. "natural" mortality). Non-informative $\beta(1,1)$ priors were used for $\alpha$ and S. In total I considered 56 different scenarios in this set of simulations.

In a third set of simulations, I assessed the impact of the temporal variance of the recovery probabilities on the parameter estimates. Consequently, I constructed the data under model [S, $\alpha_t$, $\lambda_{A,t}$, $\lambda_{B,t}$]. As before, the mean parameter values (S = 0.4, $\bar{\alpha}$ = 0.35, $\bar{\lambda}_A$ = 0.2, $\bar{\lambda}_B$ = 0.1) and the number of study years (10) were constant in all simulations. I considered the effects of the temporal variance of $\alpha$ ($\sigma^2_\alpha$ = {0.0001, 0.0005, 0.001, 0.005, 0.01, 0.05, 0.1}), two common levels of temporal variance for both recovery probabilities ($\sigma^2_\lambda$ = 0.001, 0.01), and two different prior distributions for $\alpha$ (non-informative: $\beta$(1,1), informative: $\beta$(1,1.857)). In total 28 scenarios were considered for this set.

## 2.4 Data Analyses

All data sets were analyzed with an intrinsically identifiable model [S, $\alpha_t$, $\lambda_A$, $\lambda_B$]. In addition the naïve estimate of the proportion dying due to cause A ($\eta_t$: quotient of the number of recoveries associated with cause A in year $t$ and the total number of recoveries in year $t$) was calculated for each data set. For each parameter $\theta$ I calculated the mean bias as,

$$B(\hat{\theta}) = \frac{1}{500}\sum_{sim=1}^{500}\frac{1}{T}\sum_{t=1}^{T}\left(\theta_{sim,t} - \hat{\theta}_{sim,t}\right),$$

i.e., as the mean over the 500 simulated data sets and T years of the difference between the generating parameter and its recovered estimate, where $\theta_{sim,t}$ is the parameter value at time $t$ to construct the simulated data and $\hat{\theta}_{sim,t}$ is the estimated parameter at time $t$. To evaluate the precision of the estimators, I calculated the coefficient of variation as,

$$CV(\hat{\theta}) = 100\sqrt{\text{var}\left(\frac{1}{T}\sum_{t=1}^{T}\hat{\theta}_{sim,t}\right)}\Bigg/\frac{1}{500}\sum_{sim=1}^{500}\frac{1}{T}\sum_{t=1}^{T}\hat{\theta}_{sim,t}.$$

Finally, to evaluate the accuracy of the estimators I calculated the mean squared error as,

$$MSE(\hat{\theta}) = \text{var}\left(\frac{1}{T}\sum_{t=1}^{T}\hat{\theta}_{sim,t}\right) + B(\hat{\theta})^2.$$

Low MSE indicate high accuracy of the estimator, high MSE indicate low accuracy. I also calculated the mean variance of the estimated $\alpha$ across time in order to evaluate how well this estimated the true underlying variance $\sigma^2_\alpha$.

I used R (R Development Core Team 2004) to simulate the data and analyzed them in WinBUGS (Spiegelhalter et al. 2004) from R using the package R2WinBUGS (Sturtz et al. 2005). Initial trials showed that convergence of the

MCMC chains occurred very quickly (after about 50 iterations) as evidenced by the Brooks–Gelman-Rubin diagnostics (Brooks and Gelman 1998). I used 2000 MCMC samples and conservatively discarded the first 1000 to avoid transient (preconvergence) effects in all simulations.

## 3 Results

In the first set, I evaluated bias, precision and accuracy of the estimators under different mean values of $\alpha$, $\lambda_A$, $\lambda_B$ and different temporal variation of $\alpha$. Generally, bias and precision of $\hat{\alpha}$ decreased and accuracy increased with increasing $\sigma_\alpha^2$ (Fig. 1). The bias of $\hat{\alpha}$ was mostly positive, but it could become negative when $\alpha$ was high. When the difference between the two cause-specific mortality rates became larger, bias of $\hat{\alpha}$ increased, but the precision did not change. The pattern of bias of the two recovery probabilities was similar to that of $\hat{\alpha}$: when $\hat{\alpha}$ was strongly biased, at least one the recovery probabilities was biased as well (Table 1). Absolute bias of survival rate estimates was low ($< 0.002$) in all scenarios. The number of study years only had a marginal effect with slightly lower bias of $\hat{\alpha}$ and the two recovery probabilities with more study years (Table 1). The bias of the naïve estimate $\hat{\eta}$ depended on $\alpha$, but only slightly on $\sigma_\alpha^2$, and it increased with increasing difference of the two recovery rates. Moreover, the bias of $\hat{\eta}$ was smaller when $\hat{\alpha}$ was either high or low compared to when $\hat{\alpha}$ was medium. Bias of $\hat{\eta}$ was much larger than bias of $\hat{\alpha}$, while precision of both were similar in almost all conditions (Fig. 1). Consequently, accuracy of $\hat{\eta}$ was lower than accuracy of $\hat{\alpha}$. Exceptions occurred when $\sigma_\alpha^2$ was low and the two recovery rates were close.

In the second set of scenarios I assessed the effect of the inclusion of additional information on the estimator bias, precision and accuracy. As before, bias and precision of $\hat{\alpha}$ decreased strongly with increasing $\sigma_\alpha^2$, regardless of whether additional information was considered (Fig. 2). The use of informative priors for $\alpha$ or the order constraint for the recovery probabilities had strong impacts, but their impact decreased with increasing $\sigma_\alpha^2$. The bias of $\hat{\alpha}$ was reduced and the accuracy increased when an appropriate prior for $\alpha$ was chosen. For example, the use of a $\beta(1,1.857)$ prior distribution for $\alpha$, which has the same mean as the values used to simulate $\alpha$, resulted in considerably reduced bias of $\hat{\alpha}$. The uniform prior for $\alpha$ within the interval $\{0, 0.5\}$ also considerably reduced the bias and increased accuracy of $\hat{\alpha}$, when $\sigma_\alpha^2$ was low. However, as $\sigma_\alpha^2$ increases, bias of $\hat{\alpha}$ increases as well and accuracy declined. The prior distribution which constrained $\hat{\lambda}_A$ to be higher than $\hat{\lambda}_B$ also resulted in slightly reduced bias of $\hat{\alpha}$ and higher accuracy. However, the impact of this choice of prior on parameter accuracy decreased with increasing difference between means of the two recovery probabilities. Absolute bias of survival rate was again negligible ($< 0.002$) in all scenarios (results not shown). Bias of the recovery probabilities followed the same pattern as that of $\hat{\alpha}$: when bias of $\hat{\alpha}$ was low, bias of both recovery probabilities was low as well (results not shown).

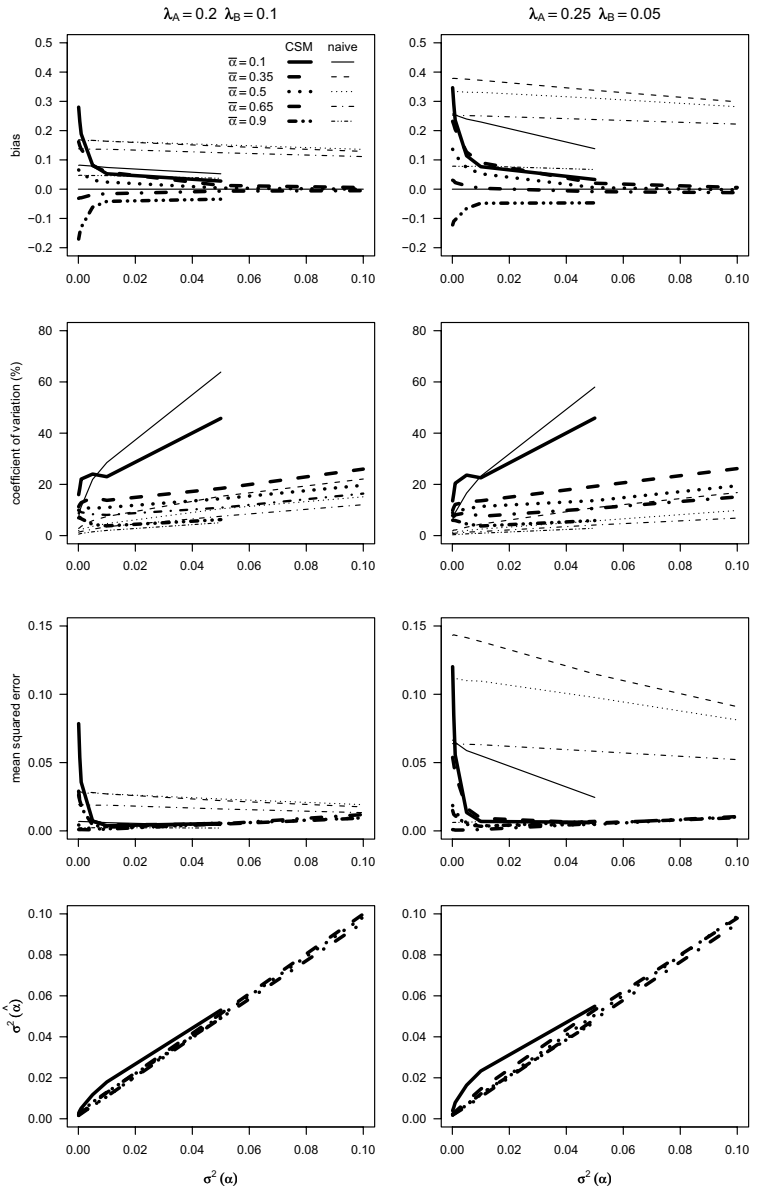


**Fig. 1** Bias, coefficient of variation, mean squared error and temporal variance of the proportion of mortality causes estimated from the cause-specific mortality rate model (CSM) and the naïve estimator (naive), respectively, in relation to the simulated temporal variance of $\alpha$ ($\sigma^2_\alpha$), the cause-specific recovery probabilities and different levels of $\bar{\alpha}$. Shown are mean values originating from 500 simulations. These simulations were performed assuming a survival rate of 0.4, 10 study years and 1000 newly released individuals in each year. Non-informative $\beta$ (1,1) priors were used for all parameters and simulations. Some combinations of $\bar{\alpha}$ and $\sigma^2_\alpha = 0.1$ are not possible, because the beta distribution is not defined

**Table 1** Mean bias of survival probability ($\hat{S}$), proportion of mortality cause A ($\hat{\alpha}$), recovery probability associated with cause A ($\hat{\lambda}_A$), recovery probability associated with other causes than A ($\hat{\lambda}_B$) and of the naïve estimate of proportion of mortality cause A ($\hat{\eta}$) from the analysis of 500 generated data sets in relation to different levels of temporal variation of $\alpha$ ($\sigma_\alpha^2$), different mean $\bar{\alpha}$, different recovery probabilities ($\lambda_A$, $\lambda_B$) and number of study years. Non-informative $\beta$ (1,1) priors were used for all parameters and simulations. Data were generated under model [S, $\alpha_t$, $\lambda_A$, $\lambda_B$] and analyzed with the same model

| | 10 years | | | | | 30 years | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| $\sigma_\alpha^2$ | $\hat{S}$ | $\hat{\alpha}$ | $\hat{\lambda}_A$ | $\hat{\lambda}_B$ | $\hat{\eta}$ | $\hat{S}$ | $\hat{\alpha}$ | $\hat{\lambda}_A$ | $\hat{\lambda}_B$ | $\hat{\eta}$ |
| $\bar{\alpha} = 0.35,\ \lambda_A = 0.2,\ \lambda_B = 0.1$ | | | | | | | | | | |
| 0.0001 | 0.0007 | 0.1622 | −0.0561 | 0.0408 | 0.1689 | 0.0002 | 0.1460 | −0.0567 | 0.0308 | 0.1685 |
| 0.005 | 0.0005 | 0.0806 | −0.0291 | 0.0175 | 0.1668 | −0.0001 | 0.0702 | −0.0312 | 0.0128 | 0.1670 |
| 0.1 | 0.0008 | 0.0055 | −0.0025 | 0.0014 | 0.1291 | −0.0001 | 0.0043 | −0.0021 | 0.0007 | 0.1314 |
| $\bar{\alpha} = 0.35,\ \lambda_A = 0.25,\ \lambda_B = 0.05$ | | | | | | | | | | |
| 0.0001 | 0.0011 | 0.2320 | −0.0936 | 0.0342 | 0.3781 | 0.0002 | 0.2253 | −0.0960 | 0.0283 | 0.3789 |
| 0.005 | −0.0001 | 0.1267 | −0.0583 | 0.0146 | 0.3758 | −0.0001 | 0.1172 | −0.0608 | 0.0115 | 0.3761 |
| 0.1 | 0.0007 | 0.0058 | −0.0035 | 0.0008 | 0.2984 | 0.0002 | 0.0038 | −0.0028 | 0.0003 | 0.3067 |
| $\bar{\alpha} = 0.65,\ \lambda_A = 0.2,\ \lambda_B = 0.1$ | | | | | | | | | | |
| 0.0001 | 0.0001 | 0.0306 | −0.0055 | 0.0117 | 0.2527 | −0.0001 | −0.0432 | 0.0160 | −0.0092 | 0.1381 |
| 0.005 | −0.0006 | −0.0115 | 0.0004 | 0.0060 | 0.2514 | 0.0002 | −0.0221 | 0.0081 | −0.0043 | 0.1368 |
| 0.1 | 0.0001 | −0.0117 | 0.0047 | −0.0014 | 0.2223 | −0.0003 | −0.0055 | 0.0016 | −0.0015 | 0.1162 |
| $\bar{\alpha} = 0.65,\ \lambda_A = 0.25,\ \lambda_B = 0.05$ | | | | | | | | | | |
| 0.0001 | 0.0001 | 0.0306 | −0.0055 | 0.0111 | 0.2527 | 0.0001 | 0.0286 | −0.0091 | 0.0063 | 0.2527 |
| 0.005 | −0.0006 | 0.0115 | −0.0004 | 0.0060 | 0.2514 | 0.0001 | 0.0077 | −0.0017 | 0.0023 | 0.2516 |
| 0.1 | −0.0001 | −0.0118 | 0.0047 | −0.0014 | 0.2222 | 0.0004 | −0.0096 | 0.0037 | −0.0011 | 0.2282 |

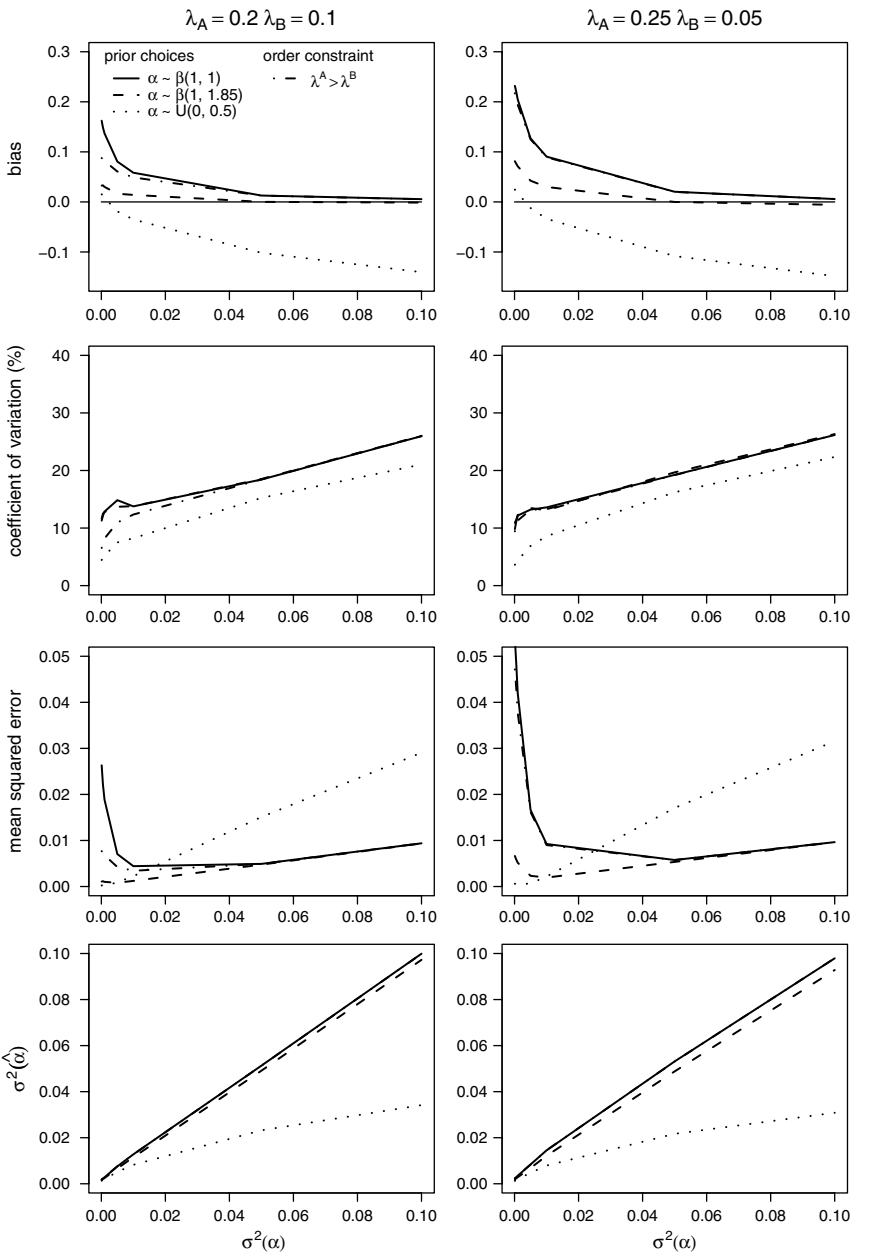


**Fig. 2** Bias, coefficient of variation, mean squared error and temporal variance of the proportion of mortality causes estimated from the cause-specific mortality rate model ($\hat{\alpha}$) in relation to the simulated temporal variance of $\alpha$ ($\sigma^2_\alpha$), the cause-specific recovery probabilities and different prior distributions or order constraints. Shown are mean values originating from 500 simulations. These simulations were performed assuming a survival rate of 0.4, $\bar{\alpha} = 0.35$, 10 study years and 1000 newly released individuals in each year

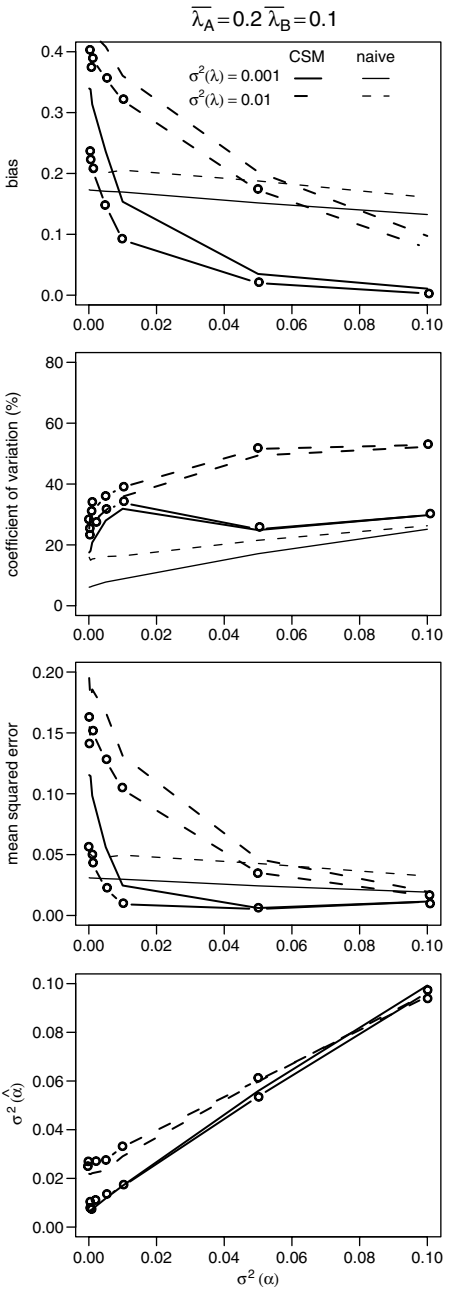


**Fig. 3** Bias, coefficient of variation, mean squared error and temporal variance of the proportion of mortality causes estimated from the cause-specific mortality rate model (CSM) and the naïve estimator (naive), respectively, in relation to the simulated temporal variance of $\alpha$ ($\sigma^2_\alpha$), temporal variance of cause-specific recovery probabilities and different prior distributions. The lines without dots refer to analyses using a $\beta$ (1,1)-prior for $\alpha$, the lines with open dots refer to analyses using a $\beta$ (1,1.857)-prior for $\alpha$. Shown are mean values originating from 500 simulations. These simulations were performed assuming a survival rate of 0.4, 10 study years and 1000 newly released individuals in each year

In the third set, I studied the estimator performance when the recovery probabilities were variable across time. The pattern of bias, precision and accuracy of $\hat{\alpha}$ remained the same: bias and precision decreased and accuracy increased with increasing $\sigma^2_\alpha$ (Fig. 3). Bias of $\hat{\alpha}$ increased and precision of $\hat{\alpha}$ decreased with increasing $\sigma^2_\lambda$. Use of an informative prior distribution for $\alpha$ reduced the bias and slightly increased accuracy. As in the other sets, bias in survival estimates was always minimal. Bias of recovery probabilities followed again the same pattern as bias of $\hat{\alpha}$ (results not shown). Bias of $\hat{\eta}$ was important in all conditions, and it increased with increasing $\sigma^2_\lambda$ (Fig. 3). Precision of $\hat{\eta}$ decreased with increasing $\sigma^2_\lambda$ and $\sigma^2_\alpha$. Bias of $\hat{\eta}$ was larger than bias of $\hat{\alpha}$ when $\sigma^2_\lambda$ was low, otherwise bias of both estimators was important.

Since $\sigma^2_\alpha$ plays a major role for bias and accuracy of $\hat{\alpha}$ and the two recovery probabilities, there is clearly an interest to know whether the estimated temporal variance of $\hat{\alpha}$ is a good estimator of the true underlying temporal variation of $\hat{\alpha}$ ($\sigma^2_\alpha$); owing to the bias of $\hat{\alpha}$, this was not clear a priori. As shown in Figs. 1, 2 and 3, the temporal variance of $\hat{\alpha}$ was mostly a good indicator of $\sigma^2_\alpha$. However, when the uniform prior (U(0, 0.5)) for $\alpha$ was chosen and $\sigma^2_\alpha$ was high, the temporal variance of $\hat{\alpha}$ underestimated $\sigma^2_\alpha$ (Fig. 2). Moreover, when recovery probabilities were variable across time, $\sigma^2_\alpha$ was slightly overestimated (Fig. 3).

## 4 Discussion

I evaluated bias, precision and accuracy of the estimators of the cause-specific mortality model of Schaub and Pradel (2004). While survival rate estimates were never strongly biased and always very accurate, bias, precision and accuracy of the proportion of the mortality causes ($\hat{\alpha}$) and of the cause-specific recovery probabilities ($\hat{\lambda}_A$, $\hat{\lambda}_B$) strongly depended on the specific situation: generally biases and precision decreased and accuracy increased with increasing temporal variance of $\hat{\alpha}$, with decreasing difference between the two recovery probabilities and with decreasing temporal variation of the two recovery probabilities. Increasing the number of study years only had limited impact on parameter accuracy and bias. The commonly used naïve estimator $\hat{\eta}$ was in almost all cases much more strongly biased and less accurate than $\hat{\alpha}$. These results followed expectations and are in line with our previous assessment of the intrinsic identifiablity of the parameters in the cause-specific mortality model (Schaub and Pradel 2004; Schaub and Lebreton 2004).

The inclusion of additional information sometimes had a strong effect on the parameter estimates, in particular when the temporal variation of $\hat{\alpha}$ was low. According to the evaluation of the intrinsic identifiability this result was expected. When the temporal variation of $\hat{\alpha}$ decreases, the likelihood of the model becomes a more flat ridge, and thus the information in the data to separately estimate all parameters becomes smaller. Consequently, the prior distributions obtain more weight compared to the likelihood until they dominate the posterior distribution (Brooks 1998).

I also found that the estimated temporal variation of the proportion of mortality causes is a fairly good estimator of the true variability of this proportion. Since bias, precision and accuracy of the parameters of interest strongly depend on the temporal variation of the proportion of mortality causes, this is an important result that may help in practical applications of the model. Based on the estimated parameters it can be judged whether accuracy is acceptable.

The naïve estimate $\hat{\eta}$ is only unbiased if the cause-specific recovery rates are identical. As shown here, bias of $\hat{\eta}$ increases the more this condition is violated. In addition, bias of $\hat{\eta}$ was usually much stronger than bias of $\hat{\alpha}$. In contrast to $\hat{\alpha}$, where the magnitude of bias can be assessed by the inspection of the temporal variance of $\hat{\alpha}$, magnitude of possible bias of $\hat{\eta}$ cannot be assessed, as the cause-specific recovery rates remain unknown. This clearly shows that $\hat{\alpha}$ is a superior and more rigor estimator of the proportion of mortality causes than $\hat{\eta}$, even if $\hat{\alpha}$ is biased in some situations.

For the practical application of the model, it would be important to evaluate how often or to what degree the assumptions of high level of variation of mortality proportions across time and constant recovery probabilities are met in practice. Classical analyses of dead recovery data have shown that sometimes recovery probabilities may be fairly constant across years (Thomson et al. 1997); yet, perhaps more often, they appear to vary across time (Piper 1995; Frederiksen and Bregnballe 2000; Schaub et al. 2005; Altwegg et al. 2006). Because these analyses did not distinguish between recovery probabilities and proportions of mortality causes, the estimated recovery probability is a combination of the two. Therefore, it is impossible to know which of these parameters was variable across time or whether both were. In the only empirical analysis so far that separated these parameter types, we (Schaub and Pradel 2004) found little evidence for temporal variation in the recovery probabilities and strong evidence for fairly large temporal variation of the proportion of white storks killed by power lines.

Based on the present study, I here propose some guidelines about how the model will be most fruitfully applied in practice. Such guidelines are important, because parameter estimates may not be adequate in terms of unbiasedness or accuracy in every situation. The first step in the application of the cause-specific mortality model should be the selection of a proper model using AIC or similar methods, although the parameter estimation will later be performed with a model in which the parameters of interest are intrinsically identifiable, i.e. model $[S, \alpha_t, \lambda_A, \lambda_B]$ or $[S_t, \alpha_t, \lambda_A, \lambda_B]$. The candidate set should include all combinations of time-specific and constant proportion of the mortality causes and time-specific and constant recovery probabilities. Despite the fact that some models in the candidate set may have parameters that are not separately identifiable, such models can validly be used for model selection. This model selection exercise will help to evaluate the expected accuracy of the parameter estimates. There are three possible outcomes. (1) If it turns out that the recovery probabilities are variable across time, it will be very difficult to obtain useful estimates of $\hat{\alpha}$, even if $\hat{\alpha}$ is highly variable over time. In this situation it may be best to conduct a simulation study that mimics the current situation in order to explore how large the bias in the parameters of interest

may possibly be. The estimates under the model could then be reported along with an acknowledgement of the likely magnitude and direction of bias. (2) If recovery probabilities are constant and $\hat{\alpha}$ is variable across time, the parameter estimates will be fairly accurate. For a further assessment of the potential bias, temporal variation in $\hat{\alpha}$ may be considered. This is best combined with a simulation study adapted to the specific situation, which will indicate the likely magnitude and direction of the bias. (3) If model selection will favor a model with constant parameters, $\alpha$ is probably not strongly variable over time and $\hat{\alpha}$ will be biased. To minimize bias a wise use of additional information, if available, may be helpful. Because the true proportion of mortality causes is not likely to be known in practice and because of their strong impact, I would not recommend to use informative priors for $\alpha$. Constraining the range of the prior for $\alpha$ or forcing one recovery probability to be larger than the other one seems reasonable, because this kind of information is more likely to be available and is quite confident. Constraining the prior range of $\alpha$ (i.e. U(0,0.5)) worked well in cases where the temporal variation of $\hat{\alpha}$ was low. $\hat{\eta}$ may be useful to define the upper limit of this uniform prior. Forcing one recovery probability to be larger than the other, resulted in some bias reduction, in particular when the values of the two recovery probabilities were not far apart.

Model performance might be improved if additional auxiliary data could be added, that must, however, contain information about mortality causes. For example, the inclusion of live recapture data would not help to increase accuracy of $\hat{\alpha}$ and the recovery probabilities, since this kind of data only adds more information about survival rate. Rather, an independent covariate that is correlated with the proportion of mortality causes might lead to progress. Such a covariate could be included using ultrastructural modeling (Link 1999). In the context of hunting, the annual proportion of hunted individuals among all individuals in a population might be useful. Another kind of information would be the mortality causes evaluated with radio tagged individuals. The frequencies obtained from such smaller scale studies could be used as a priori knowledge in the model. In practice we are often interested in long term trends. For example, we may want to know whether a disease over time becomes more or less important as a mortality cause, or whether the impact of harvesting on the overall mortality of a population changes over time. Future research ought therefore to investigate whether such trends can accurately be estimated in the presence of bias in the parameter estimates themselves.

This evaluation of the cause-specific mortality rate model suggests that in only few situations may the required assumptions be sufficiently well met that the model provides completely unbiased parameter estimates. Encouragingly, in many cases resulting biases are not very strong though and it is possible to assess the likely magnitude of bias. Furthermore, it is important to keep in mind that even if the estimated proportion of mortality causes under the model may be biased somewhat, these estimates will be an improvement over the usual naïve estimate based on the actual numbers of animals reported dead from some cause. Therefore, I conclude that a combination of careful application of the cause-specific mortality model along with custom-designed simulations can provide useful inference about the proportion of mortality causes.

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## Appendix

WinBUGS code used to estimate the parameters under model $[S,\ \alpha_t,\ \lambda_A,\ \lambda_B]$. I also provide a test data set in the format of a multistate m-array that was generated using the following parameter values $S = 0.4,\ \alpha = \{0.4835, 0.0150, 0.5636, 0.0003, 0.9408, 0.0476, 0.3089, 0.8420, 0.6130, 0.0276\}$ (corresponding to $\sigma_\alpha^2 = 0.1$), $\lambda_A = 0.2,\ \lambda_B = 0.1$, and a set of initial values.

The input data is a matrix consisting of the m-array matrix and an additional column (the last one) with the total number of animals from each cohort that were never recovered. The odd columns (except the last) contain the number of recovered animals dying from cause A, the even columns the number of recovered animals dying from other causes than A. The different lines refer to release cohorts.

### *Code*

```
{
# priors
S ~ dbeta(1,1)
for (i in 1:T) {alpha[i] ~ dbeta(1,1)}
lambdaA ~ dbeta(1,1)
lambdaB ~ dbeta(1,1)

# likelihood
for (i in 1:ni) {m[i,1:(2*nj+1)] ~ dmulti(p[i, ],
r[i])}

# calculate the number of birds released each year
for(i in 1:ni) {r[i]<- sum(m[i, ]) }

# cell probabilities of the multistate m-array
# above main diagonal
for (i in 1:(ni-1)) {
   p[i, 2*i+1]<- S*(1-S)*alpha[i]*lambdaA
   p[i, 2*i+2] <- S*(1-S)*(1-alpha[i])*lambdaB}
```

```
# main diagonal
for (i in 1:ni) {
   p[i, 2*i-1] <- (1-S)*alpha[i]*lambdaA
   p[i, 2*i] <- (1-S)*(1-alpha[i])*lambdaB

# further above
for (j in (i+2):nj) {
   p[i, 2*j-1] <- pow(S,(j-i-1))*S*(1-
S)*alpha[i]*lambdaA
   p[i, 2*j] <- pow(S,(j-i-1))*S*(1-S)*(1-
alpha[i])*lambdaB}

# below main diagonal
for (j in 1:(2*i-2)) {p[i,j] <- 0}

# last column: probability of non-recovery
p[i, 2*nj+1] <- 1-sum(p[i, 1:2*nj])
}
}
```

### *Test Data Set*

```
list(ni=10, nj=10, m= structure(.Data= c(58, 32, 17,
11, 7, 5, 3, 1, 1, 1, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0,
864, 0, 0, 0, 59, 0, 18, 0, 4, 0, 3, 0, 2, 0, 2, 0,
0, 0, 0, 0, 0, 912, 0, 0, 0, 0, 71, 22, 28, 9, 12,
4, 7, 1, 1, 0, 1, 1, 0, 0, 1, 0, 842, 0, 0, 0, 0, 0,
0, 0, 61, 0, 19, 0, 9, 0, 5, 0, 1, 0, 0, 0, 0, 905,
0, 0, 0, 0, 0, 0, 0, 0, 100, 5, 46, 1, 17, 1, 9, 0,
3, 0, 1, 0, 817, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 4,
63, 2, 29, 0, 5, 0, 3, 0, 0, 894, 0, 0, 0, 0, 0, 0,
0, 0, 0, 0, 0, 0, 49, 48, 19, 17, 3, 7, 2, 3, 852,
0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 88, 10,
37, 9, 11, 3, 842, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0,
0, 0, 0, 0, 0, 73, 16, 34, 9, 868, 0, 0, 0, 0, 0, 0,
0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 4, 54, 942),
.Dim=c(10, 21)), T=10)
```

### *Initial Values*

```
list(alpha=c(0.5, 0.5, 0.5, 0.5, 0.5, 0.5, 0.5, 0.5,
0.5, 0.5), S=0.5, lambdaA=0.5, lambdaB=0.5)
```

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