# Testing the additive versus the compensatory hypothesis of mortality from ring recovery data using a random effects model

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**Abstract**

*Testing the additive versus the compensatory hypothesis of mortality from ring recovery data using a random effects model.*— The interaction of an additional source of mortality with the underlying "natural" one strongly affects population dynamics. We propose an alternative way to test between two forms of interaction, total additivity and compensation. In contrast to existing approaches, only ring–recovery data where the cause of death of each recovered individual is known are needed. Cause–specific mortality proportions are estimated based on a multistate capture–recapture model. The hypotheses are tested by inspecting the correlation between the cause–specific mortality proportions. A variance decomposition is performed to obtain a proper estimate of the true process correlation. The estimation of the cause–specific mortality proportions is the most critical part of the approach. It works well if at least one of the two mortality rates varies across time and the two recovery rates are constant across time. We illustrate this methodology by a case study of White Storks *Ciconia ciconia* where we tested whether mortality induced by power line collision is additive to other forms of mortality.

Key words: Additive mortality, Compensatory mortality, Ring recoveries, White stork, Variance components, Power line collision.

**Resumen**

*Estudio comparativo entre la hipótesis de la mortalidad aditiva y la hipótesis de la mortalidad compensatoria mediante el empleo de un modelo de efectos aleatorios basado en datos de recuperación de anillas.*— La interacción de una fuente adicional de mortalidad con la fuente subyacente "natural" incide de forma considerable en la dinámica poblacional. Proponemos un método alternativo para comprobar los dos tipos de interacción: la aditividad total y la compensación. A diferencia de lo que sucede con los modelos empleados actualmente, en este caso sólo se precisan datos de recuperación de anillas de cada uno de los individuos recuperados cuando se conoce la causa que ha provocado su muerte. Los porcentajes de mortalidad inducida por una causa específica se estiman a partir de un modelo de captura–recaptura multiestado. Las hipótesis se comprueban examinando la correlación existente entre los porcentajes de mortalidad inducida por una causa específica. Posteriormente, se efectúa una descomposición de varianza a fin de obtener una estimación apropiada de la verdadera correlación del proceso. La estimación de los porcentajes de mortalidad provocada por una causa específica representa el punto más crítico de este planteamiento. Funciona adecuadamente si por lo menos una de las dos tasas de mortalidad varía con el tiempo y las dos tasas de recuperación se mantienen constantes en el tiempo. Para ilustrar esta metodología, presentamos un estudio de la cigüeña blanca *Ciconia ciconia*, en el que verificamos si la mortalidad inducida por colisiones con los tendidos eléctricos se suma a otras formas de mortalidad.

Palabras clave: Mortalidad aditiva, Mortalidad compensatoria, Recuperación de anillos, Cigüeña blanca, Componentes de varianza, Colisión con tendidos eléctricos.

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## Introduction

Many animal populations are subjected to man–induced sources of mortality. These include, e.g., harvesting and hunting, collisions with vehicles or objects such as power lines, or contamination with pesticides. All of this man–induced mortality can be viewed as a form of population exploitation. In the context of harvesting the exploitation is direct and intentional, in other contexts it may be indirect and unintentional. In any case, exploitation is an additional source of mortality to which the population is submitted. Determining the impact of exploitation in the broad sense on the dynamics of the population is a key question. Examples of this question are the determination of a harvesting rate which does not result in a population crash (e.g. Nichols et al., 2001), assessment of the long–term population persistence when an additional mortality cause emerges (e.g. Tavecchia et al., 2001), or the evaluation of pest control strategies (e.g. Brooks & Lebreton, 2001). Central for the evaluation of all these examples and in general is the knowledge of how the additional mortality interacts with the natural mortality.

Two extreme hypotheses about the interaction between natural and an additional mortality rate can be formulated: the totally additive and the completely compensatory hypothesis (Anderson & Burnham, 1976; Burnham & Anderson, 1984). The totally additive hypothesis of mortality assumes that deaths due to a specific mortality cause represent an additional component of mortality in the population. Hence individuals that die due to this mortality cause would, if this mortality cause wouldn't have existed, not have died during the time interval considered. If this hypothesis is true, the overall natural survival rate drops by the amount of the additional mortality rate (fig 1). Under the completely compensatory hypothesis of mortality, deaths due to the additional mortality cause would be compensated for by lowering the natural mortality rate. Hence, individuals that die due to the additional mortality cause would, if this mortality cause wouldn't have existed, have died because of another reason within the time interval considered. If this hypothesis is true, an increase of the additional mortality rate does not reduce the overall survival rate (fig. 1). Complete compensation is only possible when the additional mortality rate is lower or equal to the overall mortality rate in the absence of the additional mortality cause (Anderson & Burnham, 1976; fig. 1). Between these two extreme hypotheses any degree of partial compensation is possible (fig. 1). Under partial compensation the overall survival rate decreases when animals are subjected to an additional mortality rate, but the decrease is lower than the value of the additional mortality rate.

Complete or partial compensation of mortality can occur as a result of density–dependent mortality or of heterogeneity in survival among individuals (Burnham & Anderson, 1976). However, Lebreton (in press) showed by means of simple calculations that the resulting compensation must generally be weak even under strong density–dependence or heterogeneity.

Deciding between additivity or compensation based on empirical data has been proven to be difficult (Williams et al., 2002; Lebreton, in press). Anderson & Burnham (1976) and Burnham & Anderson (1984) were the pioneers in formulating these hypotheses and in establishing methods to test them. Since then, there has been little effort to refine the existing or to develop further methods. The basic principle approach proposed by Anderson & Burnham (1976) is to estimate the overall survival rate and the mortality rate induced by the additional mortality cause (called kill rate), and then to estimate the slope of survival against kill rate while taking account of the sampling variation. The complete compensation hypothesis is supported if this slope does not differ from 0. The critical step in this approach is the estimation of the kill rate. Because only recoveries of animals that died from the particular mortality cause are considered, an independent estimate of the recovery rate (and crippling loss rate) is required to work out the kill rate. Reward band experiments can help to obtain these independent estimates (Henny & Burnham, 1976; Nichols et al., 1991). Another approach is to test whether overall survival rate is a function of the mortality intensity due to the cause in question (e.g. harvest rate) using an ultrastructural model (e.g. Smith & Reynolds, 1992; Sedinger & Rexstad, 1994; Gauthier et al., 2001). The complete compensation hypothesis is supported if overall survival is not a function of the varying mortality intensity. Both approaches need information independently from the capture–recovery data. Because the independent variable is estimated with some sampling variance (and even bias), the slope of the regression line which serves to test the hypotheses will be biased to some degree (Lebreton, in press). Lebreton (in press) pointed out that the potential bias or uncertainty in this information tends to bias the additivity test towards the alternative hypothesis, i.e., compensation, which is quite an undesirable property of a statistical test. The case of seasonal compensation is addressed by Boyce et al. (1999).

Here we attempt to develop an alternative approach for testing the total additivity hypothesis which does not need additional independent information. Rather, this approach uses knowledge about the cause of death of each recovered, marked animal. Schaub & Pradel (2004) showed recently that it is possible to estimate separately the overall survival and the proportions of different mortality causes from capture–recovery data when the cause of death of each recovered individual is known. We use a different parameterisation of their model to estimate directly two mortality rates ("natural" and "kill" rate). We develop a random

effects model, in order to estimate in a similar way as Burnham & Anderson (1976) the correlation between the two mortality rates which serves as a test for the two opposing hypotheses. The additive hypothesis is supported if the correlation does not differ from zero. We illustrate our approach with a case study of White Storks (*Ciconia ciconia*), where we tested whether the mortality induced by power line collisions is completely additive or compensated for by other forms of mortality. Finally we discuss advantages, drawbacks and perspectives of this approach.

## Methods

### The proposed approach

The data needed for our approach are capture–mark–recovery data where the cause of death of each recovered individual is known. We then allocate all individuals that died because of the mortality cause under consideration to cause A, all other dead individuals to cause B. A multistate capture–history is then constructed for each individual, in which resightings, recoveries due to mortality cause A and recoveries due to mortality cause B are coded differently.

Over a defined time interval (usually one year) an individual has three possible fates: it may survive with probability $S$, it may die because of cause A with probability $M_A$, or it may die because of cause B with probability $M_B$. Conditional on the three fates the individual may be observed with resighting probability $p$ (probability to resight a marked individual that is alive), with recovery probability $r_A$ (probability that an animal that has died because of cause A is recovered and reported) and with recovery probability $r_B$ (probability that an animal that has died because of cause B is recovered and reported), respectively. A three–states capture–recapture model serves to estimate the unknown parameters. Written with a transition matrix (departure states are written in rows, arriving states in columns, states from top to down and from left to right are "alive", "dead due to cause A" and "dead due to cause B") and a vector of recapture probabilities, the model is,

$$\begin{bmatrix} S & M_A & M_B \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}_t \begin{bmatrix} p \\ r_A \\ r_B \end{bmatrix}_t \qquad (1),$$

where, subscript $t$ of the matrix and the vector denote time–dependence. In fact this model would contain a fourth state "dead for at least one year", but as it is absorbing and non–observable it is not necessary to consider it explicitly (Lebreton et al., 1999). An alternative notation of this model is $\{M_A(t),\ M_B(t),\ p(t),\ r_A(t),\ r_B(t)\}$.

Originally, Schaub & Pradel (2004) used another parameterisation of this model. Instead of directly estimating $M_A$ and $M_B$, they estimated the proportion ($\alpha$) of animals that have died due to cause A among all animals that have died in the specified time interval, and the overall survival rate ($S$). These parametrisaitons are equivalent, since linked by: $M_A = (1 - S)\ \alpha$ and $M_B = (1 - S)(1 - \alpha)$.

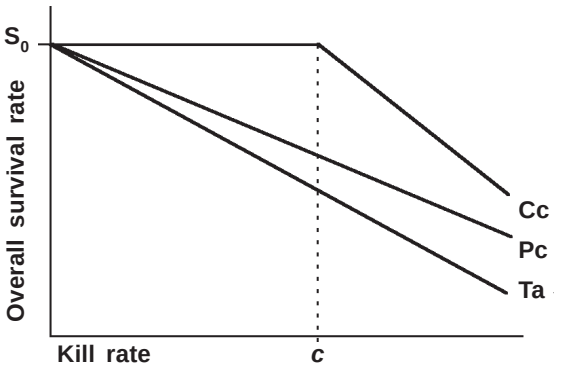


Fig. 1. Simple illustration of the complete compensatory, partial compensatory and totally additive hypotheses of mortality. $S_0$ is the survival that would be observed in the absence of the additional mortality cause (kill rate = 0). Complete compensation can occur maximally up to the threshold given by $c = 1 - S_0$: Cc. Complete compensation; Pc. Partial compensation; Ta. Total additivity.

*Fig. 1. Ilustración simple de las hipótesis de mortalidad compensatoria total, compensatoria parcial y aditiva total.* $S_0$ *es la supervivencia que se observaría ante la ausencia de la causa de mortalidad adicional (tasa de mortalidad = 0). La compensación completa sólo puede darse como máximo hasta el umbral indicado por* $c = 1 - S_0$*: Cc. Compensación total; Pc. Compensación parcial; Ta. Aditividad total.*

Schaub & Pradel (2004) pointed out that the indentifiablity of their model depends on the model structure. Using formal calculus software (Catchpole & Morgan, 1997; Catchpole et al., 2002; Gimenez et al., 2003), we tested the intrinsic identifiablity of several models with different complexity regarding time–dependence of the parameters. The models were intrinsically identifiable (i.e., not parameter redundant) when at least one of the two mortality rates is time–dependent and the two recovery rates are constant across time, or when only one mortality rate (e.g. $M_A$) and the recovery rate associated with the other cause of death ($r_B$) are time–dependent (table 1). As the model with time–constant mortality and recovery rates $\{M_A(.),\ M_B(.),\ p(t),\ r_A(.),\ r_B(.)\}$, is not identifiable, parameter estimation using identifiable models can nevertheless be negatively affected.

This is because the non–identifiable model {$M_A$(.), $M_B$(.), $p(t)$, $r_A$(.), $r_B$(.)} is a nested submodel of identifiable models. An inadequate performance can be made apparent by unrealistic estimates of some parameters and very large or zero standard errors. Catchpole et al. (2001) provide a thorough examination of the same problem found in a different model.

For testing the additivity hypothesis we estimated the correlation between the two mortality rates. If the mortality rate due to cause A were totally additive to the mortality rate due to cause B, the two mortality rates would vary independently from each other over time, and hence their correlation would be zero. However, as mortality events of the two types compete over a non negligible time period, the numbers at risk of mortality over time are affected by both sources of mortality. As a consequence, even under the assumption of additivity, the proportions dying from the two causes of mortality will be slightly negatively correlated (see appendix). However this correlation will be small in absolute value (Burnham & Anderson, 1984; Lebreton, in press) and the null hypothesis of a correlation equal to 0 remains a good approximation. In contrast, if the mortality rate due to cause A would be compensated by decreasing mortality rate due to cause B, their correlation would be negative (–1, if compensation is complete). The correlation of the two estimated mortality rates cannot be used directly for this purpose, because it is affected by sampling correlation to an unknown degree. Instead we have to estimate and decompose the different variance components, i.e. the true process variance and the sampling variance.

The covariation over time (indexed by $i$) between $M_A$ and $M_B$ is examined using a random effect model according to:

$$M_A(i) = \mu_A + U_A(i) + V(i) \qquad (2)$$

$$M_B(i) = \mu_B + U_B(i) + V(i) \qquad (3)$$

where $U_A(i)$, $U_B(i)$ and $V(i)$ are independent and normally distributed random variables with respective variances, independent of $i$, $\sigma_A^2$, $\sigma_B^2$, and $\sigma^2$.

The true process correlation between $M_A$ and $M_B$ can then be calculated as

$$\mathrm{corr}(M_A, M_B) = \frac{-\sigma^2}{\sqrt{\left(\sigma_A^2 + \sigma^2\right)\left(\sigma_B^2 + \sigma^2\right)}} \qquad (4)$$

The null (additive) hypothesis $H_0$ corr ($M_A$, $M_B$) = 0 translates then into $H_0$ var ($V$) = 0. It can thus be tested simply by a Wald test once estimates of the variance component $\sigma^2$ and of its standard error have been obtained.

In practice, the components of variance have to be estimated based on estimates $\hat{M}_A(i)$ and $\hat{M}_B(i)$ obtained from the multistate capture–recapture model. We used the following general procedure:

Let $\theta$ be a vector of parameters in a probabilistic model for which maximum likelihood estimates $\hat{\theta}$ are available together with an estimate $\hat{\Sigma}$ of their covariance matrix $\Sigma$. The maximum likelihood estimates are normally distributed and asymptotically it follows that $\hat{\theta} \sim N(\theta, \Sigma) \approx N(\theta, \hat{\Sigma})$. When the number of parameters has been reduced by some model selection, this approximation will be quite valid (Besbeas et al., 2002), even considering $\hat{\Sigma}$ as known without uncertainty. Then let us assume that $\theta$ is modeled as mixed models with fixed effects described by a design matrix $X$ and components of variance being part of a covariance matrix $W$, as:

$$\theta \sim N(X\beta, W) \qquad (5).$$

It follows that:

$$\hat{\theta} \approx N(X\beta, \hat{\Sigma} + W) \qquad (6).$$

The likelihood of this overall mixed model can then be easily maximized to find MLEs of $\beta$ and of the variance components in $W$. Maximum likelihood is among the standard methods for fitting mixed models and appears as a good competitor to more sophisticated methods such as REML (Searle et al., 1992, ch. 6). Otis & White (2004) showed that variance components are estimated accurately from band recovery data. Obviously, a Bayesian model could also be used.

This simple two–step maximum likelihood approach was used for estimating the components of variance in the model with the two sources of mortality and to test for var($V$) = 0, i.e., for additivity.

## Application to data: the White Stork and power line collisions

To illustrate this approach, we consider capture–recovery data of White Storks from Switzerland. A significant source of mortality in White Storks is collision with overhead powerlines (Riegel & Winkel, 1971; Schaub & Pradel, 2004). Evaluation of how strongly the population dynamics of Swiss White Storks are affected by power line accidents is of conservation relevance. Reconstruction of power lines is an efficient conservation option if mortality due to power lines would be additive, but less so, if power line mortality would be compensated for by other forms of mortality.

From 1984 to 1999 2912 nestlings have been ringed, of which 61 were later resighted at the breeding sites, 195 were recovered as due to power line collision and 221 as due to other sources of mortality (table 2). According to *a priori* knowledge we constructed our candidate models in the following way. The resighting effort was low and highly variable between the study years, therefore we always kept the resighting probability ($p$) time–dependent. White Storks start to breed at age 3 to 4 years before this age they may return to the breeding colonies without breeding or they may stay elsewhere. To reduce heterogeneity, we only considered resightings of

Table 1. Test results of intrinsic identifiability of constant and time–dependent mortality causes models obtained by computer algebra methods (Gimenez et al., 2003). The parameters in the model are $M_A$ (mortality rate due to cause A), $M_B$ (mortality rate due not cause B), $p$ (resighting rate), $r_A$ (recovery rate due to cause A), and $r_B$ (recovery rate due to cause B). $t$ denotes time–dependence, and $k$ is the number of capture occasions.

*Tabla 1. Resultados de la identificabilidad intrínseca de los modelos de causas de mortalidad constantes y dependientes del tiempo obtenidos mediante el empleo de métodos algebraicos asistidos por ordenador (Giménez et al., 2003). Los parámetros utilizados en el modelo son* $M_A$ *(tasa de mortalidad inducida por la causa A),* $M_B$ *(tasa de mortalidad no inducida por la causa B),* p *(tasa de reavistaje),* $r_A$ *(tasa de recuperación debida a la causa A), y* $r_B$ *(tasa de recuperación debida a la causa B).* t *indica la dependencia del tiempo y* k *es el número de casos de captura.*

| Model | Separately identifiable parameters | Number of estimated quantities |
|---|---|---|
| $M_A(t),\ M_B(t),\ p(t),\ r_A(t),\ r_B(t)$ | $p_2,\ldots,p_{k-1}$ | $5k-10$ |
| $M_A(t),\ M_B(t),\ p(t),\ r_A(t),\ r_B(.)$ | $p_2,\ldots,p_{k-1}$ | $4k-5$ |
| $M_A(t),\ M_B(t),\ p(t),\ r_A(.),\ r_B(.)$ | All | $3k-1$ |
| $M_A(t),\ M_B(.),\ p(t),\ r_A(.),\ r_B(.)$ | All | $2k+1$ |
| $M_A(.),\ M_B(.),\ p(t),\ r_A(.),\ r_B(.)$ | $p_2,\ldots,p_k$ | $k+2$ |
| $M_A(.),\ M_B(.),\ p(t),\ r_A(.),\ r_B(t)$ | $p_2,\ldots,p_k$ | $2k$ |
| $M_A(.),\ M_B(.),\ p(t),\ r_A(t),\ r_B(t)$ | $p_2,\ldots,p_k$ | $3k-2$ |
| $M_A(t),\ M_B(.),\ p(t),\ r_A(t),\ r_B(t)$ | $p_2,\ldots,p_{k-1}$ | $4k-5$ |
| $M_A(t),\ M_B(.),\ p(t),\ r_A(.),\ r_B(t)$ | All | $3k-1$ |

storks older than 4 years and fixed the resighting probabilities of the younger storks to zero. The two mortality rates ($M_E$. Electrocution mortality; $M_N$. Natural mortality) were always considered to be age– (two age classes, the first refer to the first year of life, the second to all later years) and time–dependent. Time–dependence was required to test the hypotheses. An age structure was enforced because we know that overall mortality strongly differs between young and adult storks (Lebreton, 1978; Barbraud et al., 1999; Doligez et al., 2004). The recovery rate ($r_E$) associated with electrocuted White Storks is unlikely to be age–dependent, but may be constant or time–dependent. In contrast, the recovery rate ($r_N$) associated with all other mortality causes may be age–dependent, as it compromises different sources of mortality to which young and adult storks may be differently sensitive. In addition this recovery rate may vary over time or may be constant. In summary, we used eight candidate models, that differ only in the complexity of the two recovery rates. We tested the intrinsic identifiability of all candidate models using formal calculus (Gimenez at al., 2003).

Compared to Schaub & Pradel (2004), who made a similar analysis of the data, we only considered storks ringed as nestlings and did not include natal dispersal in the model. This made the model simpler. Since Schaub & Pradel (2004) did not find significant temporal variation in natal dispersal, its omission is unlikely to have altered the estimated temporal pattern of the two mortality rates.

A goodness–of–fit test for multistate models including nonobservable states doesn't currently exist (Pradel et al., 2003). In order to have some indication of the goodness–of–fit we used the following ad hoc approach. We only considered the recovery data but did not distinguish between different causes of death. According to Brownie et al. (1985) we compared the observed number of dead storks for each cohort and year to the expected value under model $\{S(t), r(t)\}$. This model fitted the data well ($\chi^2_{32} = 37.11$, $P = 0.25$). Compared to the model we would like to test, it makes very strong assumptions, e.g., it does not allow for different recovery rates due to the mortality causes or for age–dependence of the recovery rates. We argue that because the simple model fitted the data, the more complicated model which accounts for more heterogeneity would also fit. This goodness–of–fit test does not consider the resighted storks. However the bulk of the data are the recoveries and the few resightings are therefore unlikely to significantly induce lack of fit. We used program M–SURGE (Choquet et al., 2003) to fit the different models and to estimate the parameters and their associated variance–covariance matrix.

Table 2. Capture–recovery data for White Storks from Switzerland summarized in m–array format. White Storks can be encountered dead due to power line collision (*E*), encountered dead due to a natural cause (*N*), or can be resighted alive (*R*). All White Storks are initially released as juveniles ($R_j$), but when resighted, they are "released" again as adult ($R_a$): Y. Year; NR. Number of releases.

*Tabla 2. Datos de captura–recuperación correspondientes a la cigüeña blanca de Suiza, resumidos en formato de matriz m. Las cigüeñas blancas se pueden encontrar muertas por haber colisionado con tendidos eléctricos (*E*), por causas naturales (*N*), o se pueden reavistar vivas (*R*). En un principio, todas las cigüeñas blancas se liberan siendo jóvenes ($R_j$), pero cuando son reavistadas, se "liberan" de nuevo como adultas ($R_a$): Y. Año; NR. Número de liberaciones.*

| | | Encounter period | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 1985 | | | 1986 | | | 1987 | | | 1988 | | | 1989 | | | 1990 | | | 1991 | | | |
| Y | NR | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | |
| 1984 | $R_j$ = 101 | 9 | 6 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | |
| | $R_a$ = 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 1985 | $R_j$ = 100 | | | | 7 | 5 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 1 | 0 | |
| | $R_a$ = 0 | | | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 1986 | $R_j$ = 80 | | | | | | | 6 | 6 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 1 | 0 | |
| | $R_a$ = 0 | | | | | | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 1987 | $R_j$ = 123 | | | | | | | | | | 8 | 8 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 3 | |
| | $R_a$ = 0 | | | | | | | | | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 1988 | $R_j$ = 162 | | | | | | | | | | | | | 9 | 12 | 0 | 1 | 0 | 0 | 0 | 2 | 0 | |
| | $R_a$ = 1 | | | | | | | | | | | | | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |
| 1989 | $R_j$ = 140 | | | | | | | | | | | | | | | | 19 | 1 | 0 | 0 | 0 | 0 | |
| | $R_a$ = 1 | | | | | | | | | | | | | | | | 0 | 0 | 1 | 0 | 0 | 0 | |
| 1990 | $R_j$ = 229 | | | | | | | | | | | | | | | | | | | 16 | 7 | 0 | |
| | $R_a$ = 2 | | | | | | | | | | | | | | | | | | | 0 | 0 | 0 | |
| 1991 | $R_j$ = 151 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 3 | | | | | | | | | | | | | | | | | | | | | | |
| 1992 | $R_j$ = 265 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 9 | | | | | | | | | | | | | | | | | | | | | | |
| 1993 | $R_j$ = 211 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 16 | | | | | | | | | | | | | | | | | | | | | | |
| 1994 | $R_j$ = 131 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 5 | | | | | | | | | | | | | | | | | | | | | | |
| 1995 | $R_j$ = 117 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 16 | | | | | | | | | | | | | | | | | | | | | | |
| 1996 | $R_j$ = 300 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 14 | | | | | | | | | | | | | | | | | | | | | | |
| 1997 | $R_j$ = 334 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 6 | | | | | | | | | | | | | | | | | | | | | | |
| 1998 | $R_j$ = 337 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 3 | | | | | | | | | | | | | | | | | | | | | | |
| 1999 | $R_j$ = 131 | | | | | | | | | | | | | | | | | | | | | | |
| | $R_a$ = 9 | | | | | | | | | | | | | | | | | | | | | | |

Encounter period

| 1992 | | | 1993 | | | 1994 | | | 1995 | | | 1996 | | | 1997 | | | 1998 | | | 1999 | | | 2000 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* | *E* | *N* | *R* |
| 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 1 | 0 | 0 | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 1 | 1 | 4 | 0 | 0 | 3 | 0 | 0 | 1 | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 1 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 1 | 1 | 0 | 0 | 1 | 5 | 0 | 0 | 1 | 0 | 2 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 2 | 2 | 0 | 0 | 2 | 0 | 0 | 2 | 2 | 0 | 1 | 5 | 0 | 2 | 2 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| 4 | 2 | 0 | 2 | 1 | 0 | 1 | 1 | 0 | 1 | 0 | 2 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | | 24 | 11 | 0 | 1 | 6 | 0 | 2 | 2 | 0 | 1 | 2 | 5 | 1 | 0 | 2 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 0 | 0 |
| | | | 0 | 0 | 5 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | | | | | 9 | 18 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 4 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 1 |
| | | | | | | 0 | 0 | 1 | 0 | 0 | 4 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 |
| | | | | | | | | | 1 | 7 | 0 | 0 | 2 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 |
| | | | | | | | | | 0 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | | | | | | | | | | | 4 | 4 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 |
| | | | | | | | | | | | | 1 | 0 | 3 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | | | | | | | | | | | | | | 6 | 20 | 0 | 1 | 1 | 0 | 2 | 0 | 0 | 0 | 1 | 0 |
| | | | | | | | | | | | | | | | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 1 | 0 |
| | | | | | | | | | | | | | | | | | | 23 | 17 | 0 | 1 | 1 | 0 | 1 | 2 | 0 |
| | | | | | | | | | | | | | | | | | | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 | 0 |
| | | | | | | | | | | | | | | | | | | | | | 14 | 21 | 0 | 1 | 2 | 0 |
| | | | | | | | | | | | | | | | | | | | | | 0 | 0 | 1 | 0 | 0 | 0 |
| | | | | | | | | | | | | | | | | | | | | | | | | 2 | 11 | 0 |
| | | | | | | | | | | | | | | | | | | | | | | | | 0 | 0 | 1 |

Table 3. Selection among different recovery models of Swiss White Storks. $r_E$ represents the recovery rate of storks killed by power lines and $r_N$ denote the recovery rate of storks that died because of other causes. The expression in parentheses denote whether the parameter is constant (.), time–dependent (*t*), age–dependent (*a*2), or age– and time–dependent (*a*2**t*). The other parameters in the models, the mortality rate due to power line collision ($M_E$), the mortality rate due to other causes ($M_N$) and recapture rate (*p*) were always kept age and time–dependent and time–dependent, respectively ($M_E$(*a*2**t*), $M_N$(*a*2**t*), *p*(*t*)).

*Tabla 3. Selección entre los diferentes modelos de recuperación de cigüeña blanca de Suiza.* $r_E$ *representa la tasa de recuperación de cigüeñas muertas tras haber colisionado con tendidos eléctricos, mientras que* $r_N$ *revela la tasa de recuperación de cigüeñas que murieron por otras causas. La expresión entre paréntesis indica si el parámetro es constante (.), dependiente del tiempo (*t*), dependiente de la edad (*a2*), o dependiente de la edad y del tiempo (*a2**t*). El resto de parámetros empleados en los modelos, tasa de mortalidad debida a la colisión con tendidos eléctricos (*$M_E$*), tasa de mortalidad debida a otras causas (*$M_N$*) y tasa de recaptura (*p*), siempre se mantuvieron dependientes de la edad y del tiempo y dependientes del tiempo, respectivamente (*$M_E$(*a2**t*), $M_N$(*a2**t*), p(*t*)).*

| Model | Deviance | Parameters | ΔAIC | AIC–weight |
|---|---|---|---|---|
| $r_E$(.), $r_N$(*a*2) | 4481.38 | 79 | 0.00 | 0.60 |
| $r_E$(.), $r_N$(.) | 4486.50 | 77 | 1.13 | 0.34 |
| $r_E$(*t*), $r_N$(*a*2) | 4462.65 | 91 | 5.29 | 0.04 |
| $r_E$(.), $r_N$(*t*) | 4467.88 | 90 | 8.52 | 0.01 |
| $r_E$(*t*), $r_N$(.) | 4470.26 | 89 | 8.89 | 0.01 |
| $r_E$(.), $r_N$(*a*2**t*) | 4460.25 | 100 | 20.88 | 0.00 |
| $r_E$(*t*), $r_N$(*t*) | 4464.13 | 105 | 34.77 | 0.00 |
| $r_E$(*t*), $r_N$(*a*2**t*) | 4459.40 | 118 | 56.03 | 0.00 |

Table 4. Test results of intrinsic identifiability of the mortality causes models used in the case study (table 2) as obtained by computer algebra methods (Gimenez et al., 2003). *t* denotes time–dependence, and *k* is the number of capture occasions. See table 2 for a description of the model notations.

*Tabla 4. Resultados de ensayo de la identificabilidad intrínseca de los modelos de causas de mortalidad empleados en nuestro estudio (tabla 2), obtenidos mediante el empleo de métodos algebraicos asistidos por ordenador (Giménez* et al.*, 2003).* t *indica la dependencia del tiempo, mientras que* k *es el número de casos de captura. Ver tabla 2 para una descripción de las anotaciones sobre los modelos.*

| Model | Separately identifiable parameters | Number of estimated quantities |
|---|---|---|
| $M_E$(*a*2**t*), $M_N$(*a*2**t*), *p*(*t*), $r_E$(*t*), $r_N$(*t*) | All, but the last in all parameters | 7*k*–8 |
| $M_E$(*a*2**t*), $M_N$(*a*2**t*), *p*(*t*), $r_E$(*t*), $r_N$(*a*2) | All | 6*k*–4 |
| $M_E$(*a*2**t*), $M_N$(*a*2**t*), *p*(*t*), $r_E$(*t*), $r_N$(.) | All | 6*k*–5 |
| $M_E$(*a*2**t*), $M_N$(*a*2**t*), *p*(*t*), $r_E$(.), $r_N$(*a*2**t*) | $p_2$,...,$p_{k-2}$ | 7*k*–8 |
| $M_E$(*a*2**t*), $M_N$(*a*2**t*), *p*(*t*), $r_E$(.), $r_N$(*t*) | All | 6*k*–5 |
| $M_E$(*a*2**t*), $M_N$(*a*2**t*), *p*(*t*), $r_E$(.), $r_N$(*a*2) | All | 5*k*–2 |
| $M_E$(*a*2**t*), $M_N$(*a*2**t*), *p*(*t*), $r_E$(.), $r_N$(.) | All | 5*k*–3 |

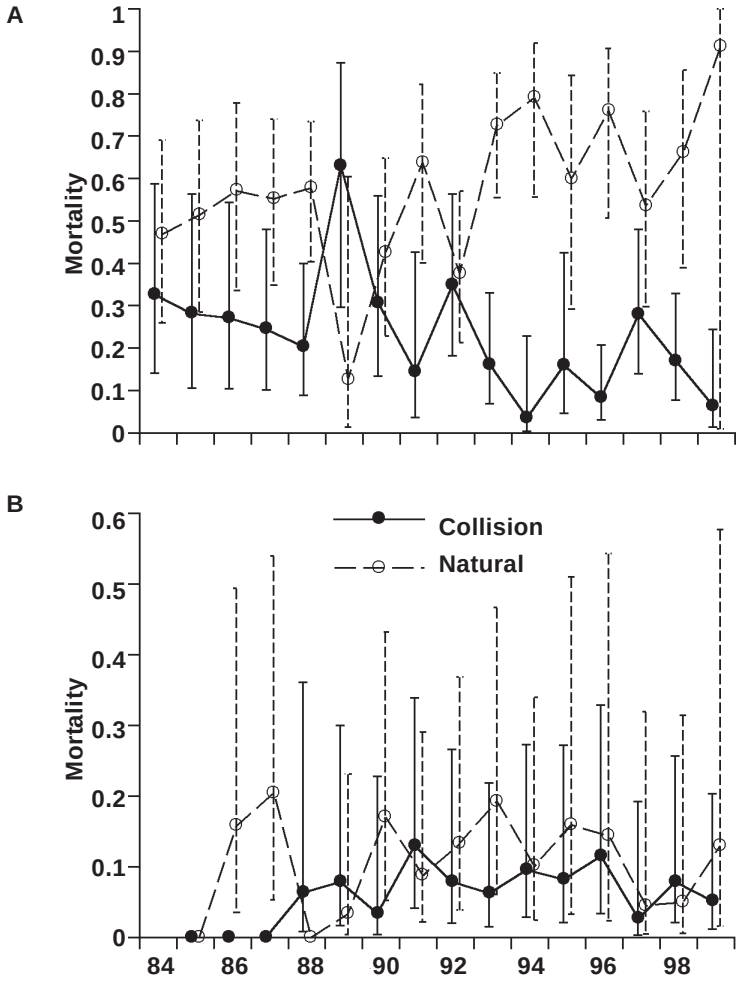


Fig. 2. Mortality rates due to collisions with overhead power lines (filled dots) and due to natural causes (open dots) in juvenile (A) and adult (B) Swiss White Storks estimated with the most parsimonious model {$M_E$(a2*t), $M_N$(a2*t), $p$(t), $r_E$(.), $r_N$(a2)}. The vertical lines show the range of the 95% confidence interval.

*Fig. 2. Tasas de mortalidad por colisiones con tendidos eléctricos aéreos (círculos negros) y por causas naturales (círculos blancos) en cigüeñas blancas de Suiza jóvenes (A) y adultas (B), estimadas mediante el empleo del modelo más moderado {*$M_E$(a2*t), $M_N$(a2*t), p(t), $r_E$(.), $r_N$(a2)*}. Las líneas verticales indican el rango del intervalo de confianza del 95%.*

## Results

Model selection revealed no evidence that the recovery rates varied over time (table 3). There was some uncertainty about whether the recovery rate due to other causes than collision with power lines was age–dependent. The best model with age–dependent recovery rate had 1.76 times more support than the model with constant recovery rates (table 3). Still, for the presentation of the results and the calculations that follow we considered only the most parsimonious model.

Six of the eight candidate models are intrinsically identifiable (table 4), including the most parsimonious one. A presumption that the estimates from that model are suitable is therefore fulfilled.

Both mortality rates were higher in juveniles than in adults (fig. 2). Power line kill rate in both age classes was usually lower than the mortality rate due to other causes. The confidence intervals of the estimates were rather wide, resulting either from the possible over–parameterisation of the model (no model selection was performed for the mortality rates) and/or from the near non–identifiability of the

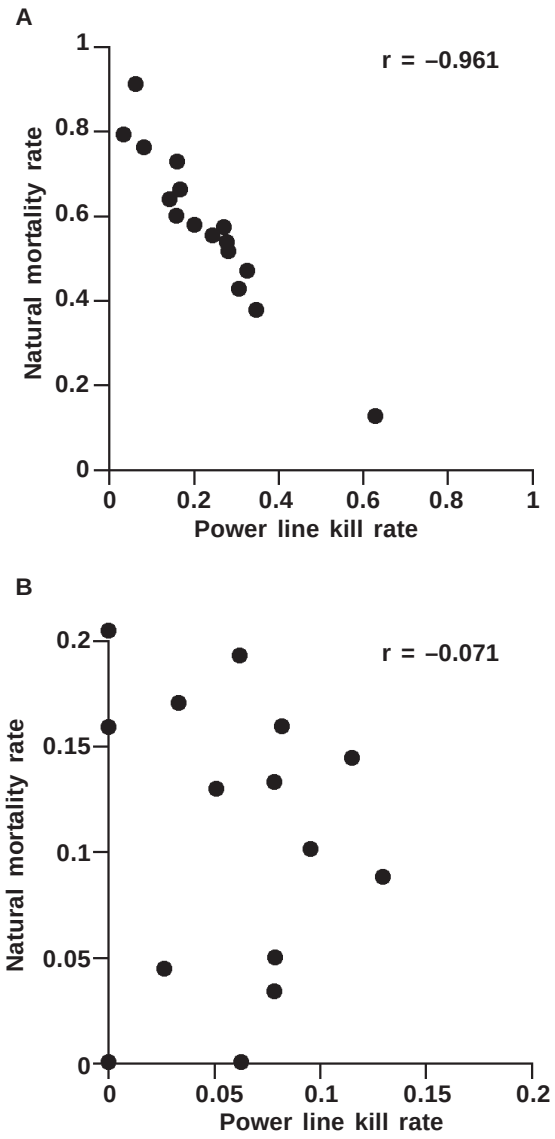


Fig. 3. Correlation of the mortality rates due to power line collision and the mortality rate due to natural causes in Swiss White Storks in juvenile (A) and adult (B) (estimated using model {$M_E$(a2*t), $M_N$(a2*t), p(t), $r_E$(.), $r_N$(a2)}). The correlations are subject to sampling and true process correlation, and thus not suited to reject or support the additive hypothesis of mortality causes.

*Fig. 3. Correlación de las tasas de mortalidad por colisiones con tendidos eléctricos y por causas naturales en la cigüeña blanca de Suiza en juveniles (A) y adultos (B) (estimadas mediante el empleo del modelo {$M_E$(a2*t), $M_N$(a2*t), p(t), $r_E$(.), $r_N$(a2)}). Las correlaciones están sujetas a muestreo y a la verdadera correlación del proceso, por lo que no resultan apropiadas para desestimar o defender la hipótesis aditiva de las causas de mortalidad.*

Table 5. Estimated variance components and their standard errors. $\sigma_E^2$ and $\sigma_N^2$ are the temporal variances of the independent components of powerline and natural mortality rates, respectively. $\sigma^2$ is the variance of their common component. A non–null value for $\sigma^2$ results in a negative correlation over time between mortality rates (see text for further explanations). All variance components are not statistically different from zero ($P > 0.05$): P. Parameter; E. Estimate; SE. Standard Error.

*Tabla 5. Componentes de varianza estimados y sus errores estándar. $\sigma_E^2$ y $\sigma_N^2$ son las varianzas temporales de los componentes independientes de las tasas de mortalidad por colisión con tendidos eléctricos y las tasas de mortalidad por causas naturales, respectivamente. $\sigma^2$ es la varianza de su componente común. Un valor de no nulidad para $\sigma^2$ se traduce en una correlación negativa a lo largo del tiempo entre las tasas de mortalidad (para más detalles al respecto, ver el texto). Ningún componentes de varianza difiere estadísticamente de cero (P > 0.05): P. Parámetro; E. Estimado; SE. Error estándar.*

| P | E | SE |
|---|---|---|
| Juveniles | | |
| $\sigma_E^2$ | 0.028915 | 0.03297 |
| $\sigma_N^2$ | 0.040161 | 0.02911 |
| $\sigma^2$ | 0.021737 | 0.01368 |
| Adults | | |
| $\sigma_E^2$ | 0.000000 | Not available |
| $\sigma_N^2$ | 0.000000 | Not available |
| $\sigma^2$ | 0.000000 | Not available |

model. There was a strong negative correlation between the two mortality rates $M_E$ and $M_N$ in juveniles, but not in adults (fig. 3). This suggest that there is some form of compensation in the juveniles. However, the observed correlation results from correlation between real (i.e. parameter) values and sampling correlation.

The variance components of the two mortality rates were not different from zero in juveniles (table 5; $P > 0.05$), thus variation over time was small. Yet the correlation between the two mortality rates was negative [corr($M_E$, $M_N$) = –0.3882]. This suggest that power line mortality is slightly compensated for by other forms of mortality in juveniles. In the adults the variation of the two mortality rates over time was very low, rendering the estimation of the variance components and the correlation between the two mortality causes impossible. Consequently the hypothesis could not be tested in the adults.

## Discussion

The population growth rate of the long–lived White Stork is much more sensitive to changes in adults survival than to changes in juvenile survival (Schaub et al., 2004). The weak compensation of the power line induced mortality in juveniles may therefore not have a very strong significance for the population dynamics. Based on the data at hand we could not test whether power line mortality of adults is compensated for by other forms of mortality, which is a pity, because this evaluation would be more important for the population dynamics. However, if compensation in the adults would occur, it would presumably be weak because the overall natural mortality rate is low (Lebreton, in press). Hence our conservative and preliminary conclusion is that power line collision is likely to have a negative impact on White Stork survival rates. We encourage other studies using data from other areas or time–periods to get more conclusive results.

The main advantage of our approach to test the additive versus the compensatory hypothesis of mortality is that there is no need to have data other than ring recoveries. This is in contrast to traditional approaches. Most of them need additional, independent information (kill rate intensity, reporting rate, crippling loss rate) resulting in biased test results (see Otis & White, 2003 for an exception). This seems particularly relevant for studies on other mortality causes than hunting, where usually information about "intensity" is completely lacking. The test results based on the new approach are more rigorous. Finally, this methodology allows the use of data which are widely available. For example in the EURING data base the mortality cause of all recovered birds has been stored routinely since years (Speek et al., 2001). Hence it is possible to test whether particular mortality risks have changed over time and whether they have significantly affected population dynamics.

Testing the additive versus the compensatory hypothesis of mortality is hampered by the need for temporal variation in at least one of the two mortality rates. The reasons are twofold. First, it is impossible to study the interaction of two mortality rates or the survival and the kill rate if there is no temporal variation. This is true for all approaches attempting to test these hypotheses. Second, and specific to our approach, the estimation of the two mortality rates only works properly when there is temporal variation in at least one of the mortality rates. As pointed out, the reason for this difficulty is the non–identifiability of the simplest submodel (see Catchpole el al., 2001). Poor estimates result when the underlying true parameter values follow the non–identifiable model, e.g. when the two mortality rates are not or only slightly variable over time or when the two recovery rates are strongly variable over time. Hence, the estimation will work accurately with some data, but not with others. We recommend the approach be used with care — parameter estimates and their variances must be checked in order to decide whether the results are sound.

Another problem for testing the additive hypothesis is the fairly strong negative correlation between the two mortality rates that arose from competing risks (see appendix). Hence, the proper null hypothesis $H_0$ would not be corr($M_A$, $M_B$) = 0, but rather corr($M_A$, $M_B$) = $–x$, where $x$ has an unknown value. The difficulty of formulating the proper $H_0$ exists also in other approaches and appears as a general difficulty in testing the additive and compensatory hypotheses of mortality.

What could be done to render the described approach more generally applicable? First, for a wider use of this method it would be very valuable to conduct a simulation study. Such a study could evaluate how accurately the parameters can be estimated depending on different degrees of temporal variation in the two mortality or recovery rates, and how strongly the outcome of the hypothesis test is compromised by inaccurate estimates of mortality rates. Second, it is worthwhile to explore how additional information could be used to stabilise the estimation of the parameters. The use of Bayesian priors for the recovery rates is certainly a promising possibility to explore.

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Appendix. The correlation between two competing sources of mortality.

*Apéndice. Correlación entre dos causas de mortalidad competitivas.*

The starting point is the approximate equation for survival: $S = 1 - M_N - M_E \approx S_0 - S_0 M_E$

form which one deduces: $M_N \approx (1 - S_0)\ (1 - M_E)$

The three terms in this equation are random variables changing from year to year. The property X and Y independent implies $E(XY) = E(X)E(Y)$ (e.g., Mood et al., 1974, p.181) and leads then to:

$$E(M_N) \approx (1 - \mathrm{E}(S_0))(1 - E(M_E))$$

$$E(M_N M_E) \approx (1 - \mathrm{E}(S_0))\ \mathrm{E}(M_E) - (1 - E(S_0))E(M_E^2)$$

Hence:

$$E(M_N M_E) - E(M_N) E(M_E) \approx (1 - E(S_0))\ E(M_E) - (1 - E(S_0))E(M_E^2) - (1 - E(S_0))E(M_E) + (1 - E(S_0)E(M_E)^2$$

i.e., $$\mathrm{cov}(M_N, M_E) \approx -(1 - E(S_0))\ \mathrm{var}(M_E)$$

This first result implies a negative correlation between $M_N$ and $M_E$ even with additivity of instantaneous sources of mortality. The next step is to calculate the correlation. First, using the formula for the variance of a product of independent random variables (Mood et al. 1974, p. 181):

$$\mathrm{var}(M_N) \approx \mathrm{var}((1 - S_0)\ (1 - M_E)) = (1 - E(S_0))^2\ \mathrm{var}(M_E) + (1 - E(M_E))^2\ \mathrm{var}(S_0) + \mathrm{var}(M_E)\ \mathrm{var}(S_0)$$

Then, using the various results above, with $\sqrt{\mathrm{var}(M_E)} = \sigma(M_E)$

$$\mathrm{corr}(M_N, M_E) \approx \frac{-(1 - E(S_0))\mathrm{var}(M_E)}{\sqrt{(1 - E(S_0))^2\,\mathrm{var}(M_E) + (1 - E(M_E))^2\,\mathrm{var}(S_0) + \mathrm{var}(M_E)\mathrm{var}(S_0)}\sqrt{\mathrm{var}(M_E)}}$$

which simplifies to:

$$\mathrm{corr}(M_N, M_E) \approx \frac{-(1 - E(S_0))}{\sqrt{(1 - E(S_0))^2 + (1 + E(M_E))^2\ \mathrm{var}(S_0)/\mathrm{var}(M_E) + \mathrm{var}(S_0)}}$$

or still:

$$\mathrm{corr}(M_N, M_E) \approx \frac{-1}{\sqrt{1 + (1 - E(M_E))^2\ \mathrm{var}(S_0)/(1 - E(S_0))^2 / \mathrm{var}(M_E) + \mathrm{var}(S_0)/(1 - E(S_0))^2}}$$

or still:

$$\mathrm{corr}(M_N, M_E) \approx \frac{-1}{\sqrt{(1 + \mathrm{cv}^2\ (1 - S_0)\ / \mathrm{cv}^2\ (1 - M_E) + \mathrm{cv}^2 (1 - S_0)}}$$

For the first year White Stork with $E(S_0) \approx 0.65$, $E(M_E) \approx 0.25$, $\mathrm{var}(S_0) \approx 0.04$, and $\mathrm{var}(M_E) \approx 0.03$, we expect the correlation between $M_N$ and $M_E$ to be $\mathrm{corr}(M_N, M_E) = -0.36$ also if the two mortality causes are completely additiv.