JOURNAL OF Evolutionary Biology

doi:10.1111/j.1420-9101.2010.02079.x

Combining capture–recapture data and pedigree information to assess heritability of demographic parameters in the wild

J. PAPAÏX*, S. CUBAYNES*†, M. BUORO*, A. CHARMANTIER*, P. PERRET* & O. GIMENEZ*

*Centre d'Ecologie Evolutive et Fonctionnelle UMR 5175, 1919 Route de Mende, 34293 Montpellier Cedex 5, France
†Institut de Mathématiques et Modélisation de Montpellier, UMR 5149, Place E. Bataillon, 34095 Montpellier Cedex 5, France

Keywords:

Binary trait;
Capture–Recapture Animal Model;
Cormack–Jolly–Seber model;
Individual heterogeneity;
State–space model;
Threshold model.

Abstract

Quantitative genetic analyses have been increasingly used to estimate the genetic basis of life-history traits in natural populations. Imperfect detection of individuals is inherent to studies that monitor populations in the wild, yet it is seldom accounted for by quantitative genetic studies, perhaps leading to flawed inference. To facilitate the inclusion of imperfect detection of individuals in such studies, we develop a method to estimate additive genetic variance and assess heritability for binary traits such as survival, using capture–recapture (CR) data. Our approach combines mixed-effects CR models with a threshold model to incorporate discrete data in a standard 'animal model' approach. We employ Markov chain Monte Carlo sampling in a Bayesian framework to estimate model parameters. We illustrate our approach using data from a wild population of blue tits (*Cyanistes caeruleus*) and present the first estimate of heritability of adult survival in the wild. In agreement with the prediction that selection should deplete additive genetic variance in fitness, we found that survival had low heritability. Because the detection process is incorporated, capture–recapture animal models (CRAM) provide unbiased quantitative genetics analyses of longitudinal data collected in the wild.

Introduction

Quantitative genetic models (Falconer & Mackay, 1996; Lynch & Walsh, 1998) allow identification of components of variance observed in a phenotypic trait (either morphological or demographic) by jointly analyzing data on the trait and on genealogical relationships in a pedigree. In particular, the 'animal model' approach allows, through the use of generalized linear mixed models, simultaneous estimation of components of phenotypic variance that can be attributed to genetic factors, environmental factors and other unknown factors (Kruuk, 2004). Heritability of the phenotypic trait can then be estimated from the fraction of the variance that can be attributed to the additive genetic effects.

Correspondence: Olivier Gimenez, Centre d'Ecologie Evolutive et Fonctionnelle UMR 5175, 1919 Route de Mende, 34293 Montpellier Cedex 5, France.
Tel.: +33 467 613 211; fax: +33 467 412 138;
e-mail: olivier.gimenez@cefe.cnrs.fr

Although well developed in animal breeding science, it is only recently that the estimation of heritability using the animal model framework has been advocated for wild animal and plant populations as an alternative to more limited classic regressions between relatives (Kruuk, 2004). The methodological advances of the animal model and the increasing use of quantitative genetics in wild populations has resulted in important applications for identifying management strategies for species of conservation concern (Law & Stokes, 2005), for other conservation biology issues (Coltman *et al.*, 2003; Stockwell *et al.*, 2003) and to address questions of a basic nature in evolutionary biology (Kruuk *et al.*, 2008).

However, there are still problems concerning the estimation of heritability in natural systems (Merilä *et al.*, 2001), which Kruuk (2004) suggests 'can to a certain extent be overcome by resorting to statistical techniques that are more elaborate than the ones adopted in a majority of the investigations in natural settings'. Among

other problems, it is well known that estimating demographic parameters in the wild can be biased and inference can be flawed when the detectability of studied individuals is not accounted for (Gimenez *et al.*, 2008). Typically, estimating individual survival, and hence heritability in survival, can be strongly biased when individuals are missed during population monitoring. Methods using traditional models for inferring heritability of demographic parameters have been developed (Cox or parametric model for survival e.g. Ducrocq & Casella, 1996). However, such methods do not deal with detection probabilities less than 1 (Cam, 2009). In contrast, capture–recapture (CR) models allow estimation of demographic parameters when the detection is imperfect (Lebreton *et al.*, 1992). The basic Cormack–Jolly–Seber (CJS) model (Lebreton *et al.*, 1992) considers survival and recapture probabilities as varying over time but homogeneous among individuals, which is of little use for estimating individual variability in demographic parameters. Recently, the CJS model has been extended to account for individual effects in both survival and recapture probabilities. Royle (2008) proposed a state–space model (SSM) formulation of the CJS model, specifically to incorporate random individual effects (see Gimenez & Choquet (2010) for an alternative approach). The SSM framework distinguishes the underlying demographic process from the observation process (detection), therefore providing much flexibility for decomposing the variability in demographic parameters (Gimenez *et al.*, 2007).

Our present purpose is to adapt the SSM framework to combine CR with animal models (hereafter CRAM), thus allowing the decomposition of individual variation in demographic parameters into environmental and genetic components (as first suggested by O'Hara *et al.* (2008) in their Fig. 1). We provide the details of the Bayesian inference and its implementation through Markov chain Monte Carlo (MCMC) simulations using the freely available software package OpenBUGS. We refer to O'Hara *et al.* (2008) for a review of the Bayesian approach for quantitative genetic analyses and to Sorensen & Gianola (2002) for exhaustive details on its

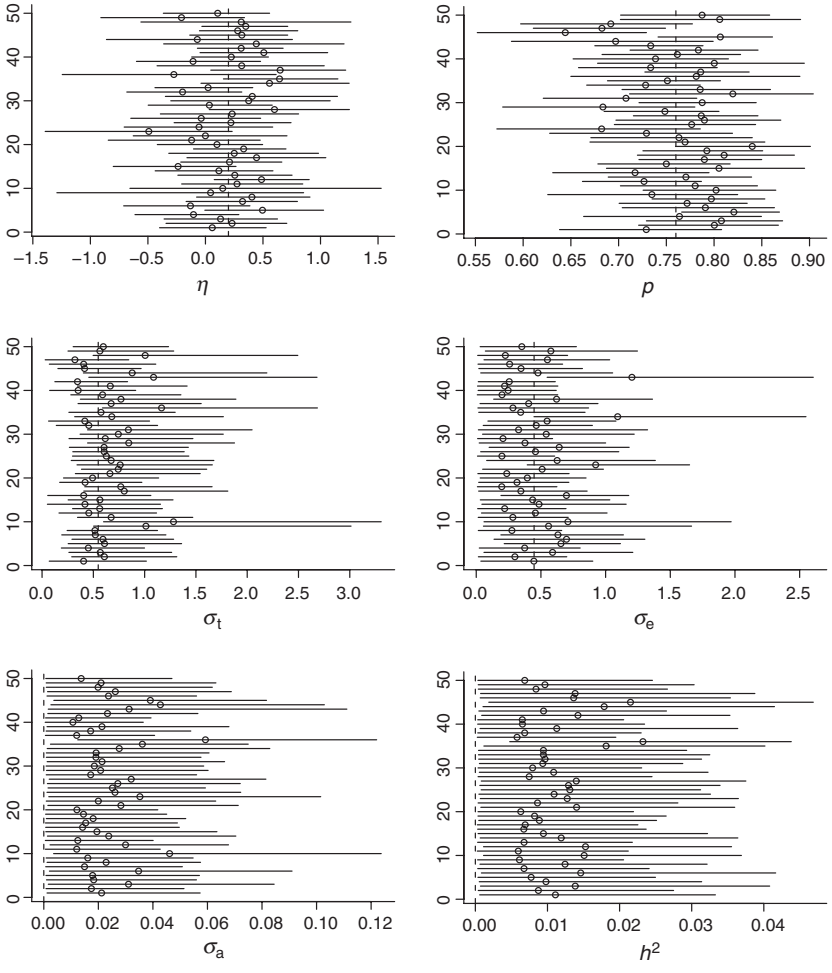

Fig. 1 Performance of the capture–recapture animal model approach – scenario with $\sigma_a^2 = 0$. For each of the 50 simulated data sets, we displayed the median (circle) and the 95% credible interval (horizontal solid line) of the parameter. The actual value of the parameter is given by the vertical dashed line. Notation: η is the mean survival on the probit scale, p is the detection probability, σ_t^2 is the variance of the yearly random effect, σ_e^2 is the variance of the nongenetic individual effect, σ_a^2 is the additive genetic variance and h^2 is the heritability.

implementation. Finally, we illustrate the utility of our method by estimating the heritability of survival using data from a 29-year study of individually marked blue tits (*Cyanistes caeruleus*).

A threshold state–space model for CR–pedigree data

A SSM model can be specified in which there are two layers, one specifying the dynamic process (the state model) and another connecting the demographic process to its observation through the detection of individuals (the observation model). We focused on a binary trait, namely survival, and aimed at calculating its heritability. In standard methods of quantitative genetics, the heritability of a discrete trait is often assessed using so-called threshold models (Gianola, 1982; Falconer & Mackay, 1996; Lynch & Walsh, 1998). This approach assumes that there exists a continuous random variable called a latent variable or liability from which discrete values of the trait are generated.

Under natural conditions, because individual detectability is often less than one, we need to deal with the observation process. To account for this issue in the sampling protocol, we used CR data generally collected under the form of 1's and 0's corresponding to a detection or not of I individuals over T sampling occasions.

The state model

We define X as a binary random variable that represents the demographic process, with $X_{i,t} = 1$ if individual i is alive and available for detection at time t and 0 if it is dead. The state process in the SSM formulation stipulates that if individual i is alive at time $t - 1$, it survives until time t with survival probability $\phi_{i,t-1}$ or dies with a probability $1 - \phi_{i,t-1}$; in other words, $X_{i,t}$ is distributed as a Bernoulli random variable with parameter $\phi_{i,t-1}$ given $X_{i,t-1} = 1$ (Gimenez *et al.*, 2007; Royle, 2008).

The observation model

We define Y as a binary random variable standing for the observation process, with $Y_{i,t} = 1$ if the individual i is detected at time t and 0 otherwise. These observations are generated from the underlying demographic process, which is partially hidden from the observer, because when an individual is not detected, it is not possible to say whether it is alive or not.

If individual i is alive at time t, then it has a probability $p_{i,t}$ of being encountered and a probability $1 - p_{i,t}$ otherwise; in other words, the link between survival and the detection of individuals is made through the observation equation, which states that $Y_{i,t}$ is distributed as a Bernoulli random variable with parameter $p_{i,t}$ given $X_{i,t} = 1$ (Gimenez *et al.*, 2007; Royle, 2008).

Plugging the animal model in CR models: CRAM

We assume that the random survival process is related to a continuous underlying latent variable $l_{i,t}$, which, given $X_{i,t-1} = 1$, is satisfied as:

$$X_{i,t} = \begin{cases} 1 & \text{if} \quad l_{i,t} > \kappa, \\ 0 & \text{if} \quad l_{i,t} \leq \kappa. \end{cases}$$

for $t = f_i + 1,\ldots,T$, where f_i is the first time individual i is detected and κ was a threshold value. We assumed that the so-called liability $l_{i,t}$ was normally distributed with mean $\mu_{i,t}$ and variance σ_e. For identifiability issues, and without loss of generality, we fixed σ_e to 1 and κ to 0 (Harville & Mee, 1984; Sorensen *et al.*, 1995).

From this construction, usually referred to as a threshold model (Gianola, 1982), we have $\phi_{i,t-1} = \Pr(X_{i,t} = 1 | X_{i,t-1} = 1) = F(\mu_{i,t})$ where F is the cumulative function of a normal distribution with mean 0 and variance 1. Noting that F^{-1} is the probit function often used to analyse binary data, we specified an animal model on the mean of the liability:

$$\mu_{i,t} = \text{probit}(\phi_{i,t-1}) = \eta + b_t + e_i + a_i$$

where η is a constant term for the mean survival on the probit scale, b_t is a random yearly effect (i.e. year specific), e_i is an individual random effect that has no genetic basis and a_i is the genetic value for individual i. Note that covariates can be incorporated as fixed effects possibly affecting survival, e.g. climate effects (Grosbois *et al.*, 2008) or anthropogenic pressures (Véran *et al.*, 2007). We assumed that the temporal effect b_t is normally distributed with mean zero and variance σ_t^2, e_i normally distributed with mean 0 and variance σ_e^2 whereas the distribution of $\mathbf{a}$, the vector of the a_i's, was multivariate normal with mean 0 and variance–covariance matrix $\sigma_a^2\mathbf{A}$, where σ_a^2 is the additive genetic variance and $\mathbf{A}$ the additive genetic relationship matrix (Sorensen & Gianola, 2002). The additive genetic relationship matrix $\mathbf{A}$ is built up from the pedigree. For example, for a given individual, $A_{i,i} = 1$, whereas between parents and their offspring, $A_{i,j} = 0.5$. To handle with the complexity of the animal model, we adopted a procedure proposed by Damgaard (2007) (see also Waldmann (2009) for an alternative), which combines a reparametrization (Henderson, 1976) and a recursive algorithm (Quaas, 1989). Heritability was calculated as the ratio of the additive genetic variance to the total variance:

$$h^2 = \frac{\sigma_a^2}{\sigma_t^2 + \sigma_e^2 + \sigma_a^2 + 1}.$$

Implementation

Estimating variance components and heritability

The frequentist approach for fitting our model would require maximizing the likelihood of the data. This is a

complex issue because of the high-dimensional integral of the SSM likelihood and the presence of random effects and latent variables. Therefore, we opted for a Bayesian approach through MCMC methods, which provide powerful computer-intensive methods for handling complex models. Bayesian statistical methods are becoming increasingly popular in evolutionary ecology, in particular to analyse CR data (Gimenez *et al.*, 2006), as well as in quantitative genetics, in particular to fit animal models (Damgaard, 2007) and threshold models (Sorensen *et al.*, 1995).

In order to completely specify the Bayesian model, we provided prior distributions for all parameters. All priors were selected as sufficiently vague in order to induce little prior knowledge. Specifically, we chose $p \sim U[0,1]$ and $\eta \sim N(0,100)$. We assigned uniform distributions to the standard deviation of the random effects, $\sigma_t \sim U[0,10]$, $\sigma_e \sim U[0,10]$ and $\sigma_a \sim U[0,10]$ (Gelman, 2006; Royle, 2008).

The simulations were performed using OpenBUGS (Thomas *et al.*, 2006) (which performs block-updating), using the program R (Ihaka & Gentleman, 1996) and package R2WinBUGS (Sturtz *et al.*, 2005); R was particularly useful for manipulating the pedigree and post-processing the MCMC results (see Supporting Information for the R and OpenBUGS codes).

Simulation study

The ability of our model to estimate the genetic basis of individual variation in survival was verified using simulations. We considered two scenarios, without ($\sigma_a^2 = 0$) and with additive genetic variance ($\sigma_a^2 = 0.4$). All other parameters were chosen to mimic the case study on blue tits (see next section). Specifically, we used $p = 0.76$, $\eta = 0.2$ (mean survival ≈ 0.6), $\sigma_t^2 = 0.3$, $\sigma_e^2 = 0.2$ (heritability ≈ 0.2). We simulated 50 pedigrees with 50 individuals (25 dams and 25 sires) over 5 generations (250 individuals in total). In association with the pedigrees, we simulated 50 capture–recapture datasets with 10 sampling occasions. Parent group was assumed to be unobserved. We divided the progeny group into 5 cohorts (every 2 years) of 40 individuals. For five randomly chosen data sets, we first ran two overdispersed parallel MCMC chains to check whether convergence was reached. As a result, we decided to use 60 000 iterations with 20 000 burned iterations for posterior summarization. We then applied our capture–recapture animal model approach on each data set.

The results are shown in Fig. 1 (without additive genetic variance) and Fig. 2 (with additive genetic variance). For each of the two scenarios, our approach was successful in estimating the various parameters. In particular, the value of σ_a was well recovered by our model (see Fig. 2, bottom-left panel), with only one 95% credible interval (out of 50) that did not contain the actual value.

Application to the blue tit data

To illustrate our approach, we used a long-term dataset of individually marked blue tits (*Cyanistes caeruleus*) in a natural population in Pirio, on the island of Corsica (France). The study site is made of evergreen forest, composed essentially of Holm Oaks (*Quercus ilex*). Blue tits are hole-nesting birds that readily breed in artificial nest boxes, which facilitates the individual manipulation required for the marking process (Blondel *et al.*, 2006). We used a total of 614 breeding individuals that were banded, released and recaptured in spring during breeding seasons between 1979 and 2007. We recorded 1366 detection events, from which 41% individuals were captured only once (initial marking) and 25% twice (initial marking and a subsequent recapture). A pedigree was constructed based on nest observations; chicks that were marked in a nest box were considered as the progeny of the male and female captured in the same nest box. Within the 614 observed individuals, 287 individuals have no parents identified, 218 fathers and 215 mothers were recorded. The pedigree counts 327 offspring–parent links, 112 full-sib and 126 half-sib links. The maximal pedigree depth is 11 generations. In addition to the observed individuals, 40 dummy individuals were added to retain sib links when constructing the relationship matrix **A**. We used the R package PEDANTICS to manipulate the pedigree (Morrissey & Wilson, 2010).

We assessed the fit of the CJS model to the data using standard goodness of fit techniques (Lebreton *et al.*, 1992) implemented in program U-CARE (Choquet *et al.*, 2009). Overall, the model with both time-dependent survival and recapture probabilities provided a satisfactory fit to the data ($\chi^2_{82} = 65.32$, $P = 0.91$). A preliminary analysis using program M-SURGE (Choquet *et al.*, 2005) suggested that the recapture probability could be simplified by considering it constant over time. Because this test was only valid for the CJS model, we also used a posterior predictive assessment to specifically judge the fit of our CRAM to the observed data (Gelman *et al.*, 1996). The results showed that our model fitted the data adequately well (see Supporting Information).

Two MCMC–chains of 15 000 iterations took around 50 min on a PC (1.8 GHz) with 2 GB of RAM. Convergence was assessed using the Gelman and Rubin statistic which compares the within to the between variability of chains started at different and dispersed initial values (Gelman, 1996). Burn-in was set to 5000, and thinning of each 10th iteration resulted in acceptable mixing and convergence (Fig. 3).

The posterior distributions are displayed in Fig. 4, and the resulting summary estimates are presented in Table 1. Detection probability p was high. Survival probability was in agreement with what we were expecting for a small passerine. The additive genetic

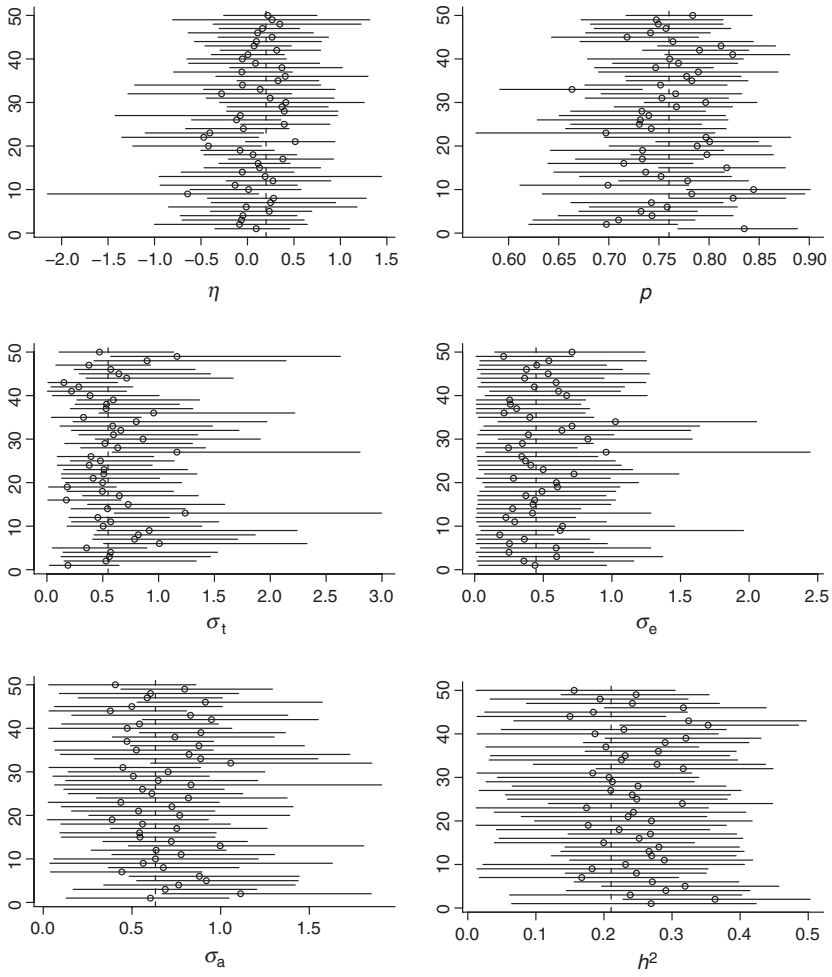

Fig. 2 Performance of the capture–recapture animal model approach – scenario with $\sigma_a^2 = 0.4$. For each of the 50 simulated data sets, we displayed the median (circle) and the 95% credible interval (horizontal solid line) of the parameter. The actual value of the parameter is given by the vertical dashed line. Notation: η is the mean survival on the probit scale, p is the detection probability, σ_t^2 is the variance of the yearly random effect, σ_e^2 is the variance of the nongenetic individual effect, σ_a^2 is the additive genetic variance and h^2 is the heritability.

variance σ_a^2 was low, resulting in a low heritability h^2. The environmental variance σ_t^2 was moderate, suggesting temporal variation in survival should not be neglected.

Finally, we compared the results of this CR study with a naive analysis in which we considered all individuals as being detected with certainty. In practice, we assumed that time to death was obtained as the occasion following that when an individual was last captured. As expected, the naive analysis led to a downward–biased survival estimate (posterior mean $\text{probit}^{-1}(\eta) = 0.530$, SD = 0.034), because tits that were observed for the last time before the end of the study were wrongly assumed as dead by the naive approach, whereas they might actually have been alive but undetected. While the additive genetic variance was greater in the naive analysis (posterior mean $\sigma_a^2 = 0.187$, SD = 0.107) and the estimate of heritability twice as large as in the CRAM analysis (posterior mean $h^2 = 0.040$, SD = 0.037), the inference remained unchanged as heritability was negligible.

Discussion

We developed a model to estimate and make statistical inference about the genetic basis of survival, an important component of fitness. We combined CR data and pedigree information using up-to-date CR and animal models within a Bayesian framework using MCMC techniques. In particular, because survival is a binary trait, we introduced a threshold model that is frequently used in assessing the heritability of qualitative traits. Our approach relies on the SSM methodology, which has the appealing advantage of disentangling the demographic process under investigation from its observation through the detection process.

The analysis of the blue tit data showed that heritability of survival was low. Following the classical interpretation of Fisher's fundamental theorem of natural selection, this is an expected result because traits strongly associated with fitness should be weakly heritable (Fisher, 1958). Yet, estimates of the heritability of longevity in the wild are scarce and, with regard to the

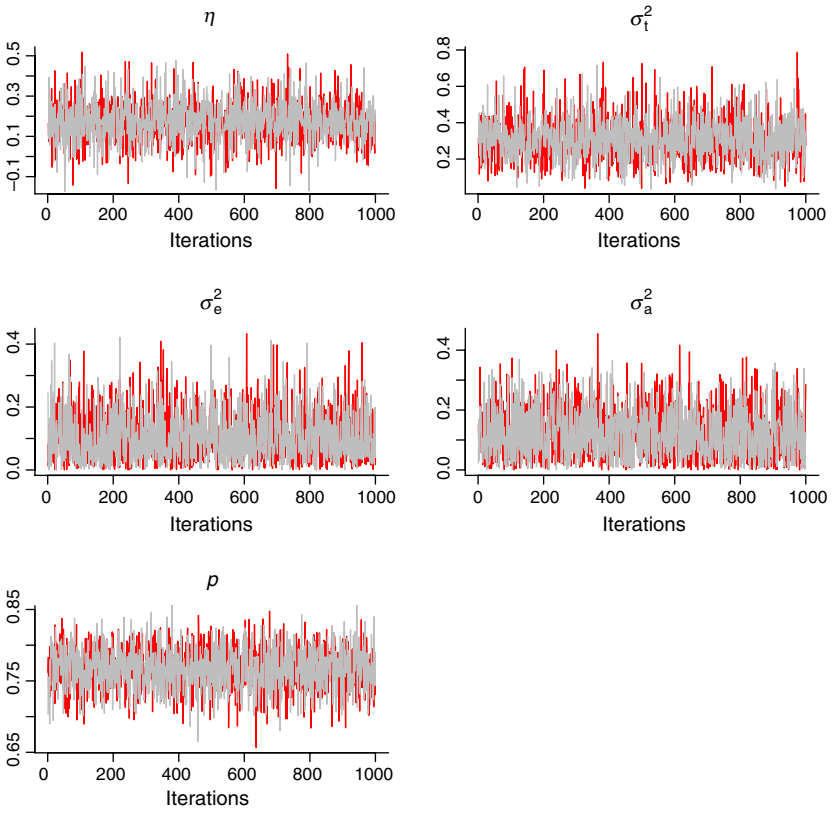

Fig. 3 Mixing of the MCMC algorithm used to fit the capture–recapture animal model (CRAM) to the blue tit data. Two chains of 15 000 iterations were used, with a thinning of each 10th iteration, and 5000 iterations as a burn–in, resulting in 1000 iterations for each chain (one in red/dark grey, the other in light grey) used to summarize the posterior results. Notation: η is the mean survival on the probit scale, σ_t^2 is the variance of the yearly random effect, σ_e^2 is the variance of the nongenetic individual effect, σ_a^2 is the additive genetic variance and p is the detection probability.

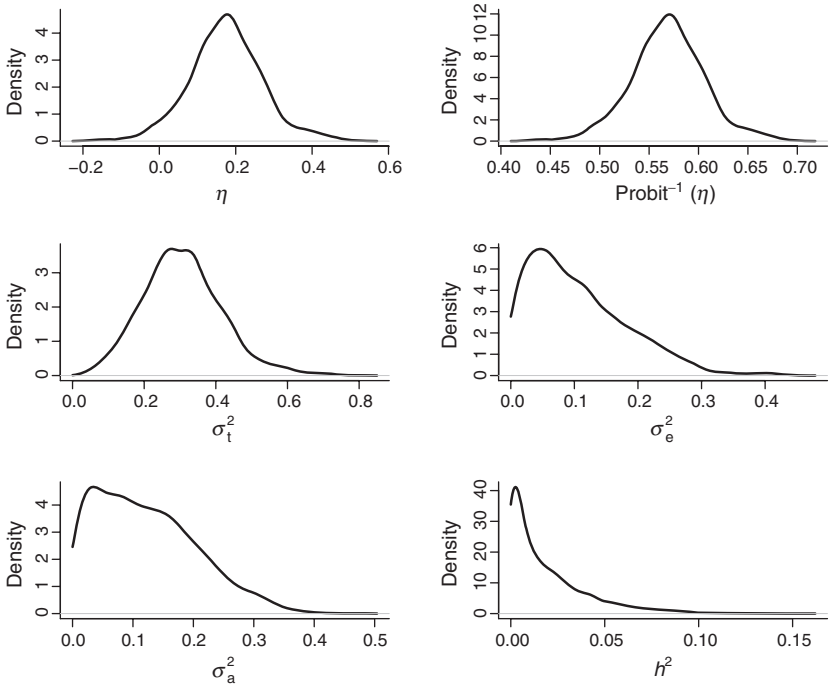

Fig. 4 Posterior density distributions for parameters of the capture–recapture animal model (CRAM) used for the blue tit data. Notation: η is the mean survival on the probit scale, $probit^{-1}(\eta)$ is the mean survival after back-transformation, σ_t^2 is the variance of the yearly random effect, σ_e^2 is the variance of the nongenetic individual effect, σ_a^2 is the additive genetic variance and h^2 is the heritability.

depletion of genetic variation for fitness traits, are inconsistent (e.g. Kruuk *et al.*, 2000; Coltman *et al.*, 2005). Besides, heritability of adult survival in the wild has simply not been estimated until now, to our knowledge. Hence, we present here the first estimate of heritability of survival between breeding seasons in a

Table 1 Parameter estimates for the capture–recapture animal model (CRAM) applied to the blue tits. Posterior means, medians, standard deviations (SD) and 95% credible intervals (CI) are provided: η is the mean survival on the probit scale, $probit^{-1}(\eta)$ is the mean survival after back-transformation, σ_t^2 is the variance of the yearly random effect, σ_e^2 is the variance of the nongenetic individual effect, σ_a^2 is the additive genetic variance, h^2 is the heritability and p is the detection probability.

Parameter	Mean	Median	SD	CI
η	0.175	0.175	0.096	[–0.018, 0.385]
$\text{probit}^{-1}(\eta)$	0.569	0.569	0.035	[0.493, 0.650]
σ_t^2	0.307	0.301	0.112	[0.105, 0.558]
σ_e^2	0.105	0.089	0.078	[0.005, 0.284]
σ_a^2	0.122	0.110	0.083	[0.006, 0.308]
h^2	0.018	0.011	0.021	[0.000, 0.077]
p	0.767	0.769	0.030	[0.707, 0.822]

wild vertebrate using a model accounting for the detection process. Although using a naive analysis assuming perfect detection did not change the inference, this was probably because of a relatively high detection probability, constant through time. This will not be always the case, and, because it is difficult to give guidelines about when the issue of detectability less than one could be ignored and a naive analysis could be conducted, we recommend that joint analysis of CR and pedigree data be undertaken using our new CRAM methodology.

To formally assess the relevance of including an additive genetic variance term, an individual nongenetic effect or both in the model, a model selection procedure could be undertaken. Adapting a method developed by Kuo & Mallick (1998), Royle (2008) recently implemented a way to compute the posterior model probability of a model. In our context, this requires introducing two indicator variables, say w_e and w_a, both having Bernoulli (0.5) prior distributions, and premultiplying the random effects e_i and a_i, respectively, in the expression $\text{probit}(\phi_{i,t-1})$ (see Plugging the animal model in CR models: CRAM). For example, if $w_a = 1$, then the genetic additive effect is present in the model, whereas if $w_a = 0$, it is not. Therefore, a model with $w_e = 1$ and $w_a = 1$ corresponds to $\text{probit}(\phi_{i,t-1}) = \eta + b_t + e_i + a_i$ (both effects). The posterior model probability is calculated from the MCMC histories, using the ratio between the number of iterations giving a particular model over the total number of iterations. In the blue tit data analysis, the simplest model was by far the most visited by the MCMC chains (posterior probability = 96%), indicating that neither individual random effect was needed as suggested by the estimates of variance components. The Bayesian framework offers several alternative approaches that are reviewed in O'Hara & Sillanpää (2009).

For the sake of illustration, we focused on a relatively simple model, although our approach can be fruitfully adapted to address questions involving more complex analyses. We see at least two promising extensions that are the object of our ongoing research. First, our present focus was on survival, but the CRAM framework could easily handle other parameters such as dispersal or age at first reproduction. It would require extending the SSM to multinomial data (Gimenez *et al.*, 2007) and the liability approach to several thresholds (Sorensen *et al.*, 1995). Second, additive genetic variance and heritability are known to vary in natural populations. In particular, changes with age make quantitative genetics tools particularly relevant for investigating senescence in natural populations. CRAM can be extended to incorporate a relationship between the additive genetic contribution and age using a 'random regression' model (Meyer, 1998; Averill *et al.*, 2006).

With the accumulation of longitudinal data on natural populations of most taxa and the constant improvement of methods for assignment of genetic relationships among individuals, an important goal of evolutionary ecology is to predict evolutionary change in the face of natural or anthropogenic influences in wild populations. Our new approach, combining up-to-date quantitative genetic tools and recent methods for the analysis of longitudinal data with imperfect detection, provides reliable quantitative genetic estimates for both applied and basic research.

Acknowledgments

The authors thank C. Trottier and C. Lavergne for helpful discussions and F. Stephen Dobson for his huge help in improving the English of our MS. This research was supported in part by a grant from the 'Jeunes Chercheuses et Jeunes Chercheurs' program of the French ANR (ANR–08–JCJC–0028–01).

References

Averill, T., Rekaya, R. & Weigel K. 2006. Random regression models for male and female fertility evaluation using longitudinal binary data. *J. Dairy Sci.* **89**: 3681–3689.

Blondel, J., Thomas, D.W., Charmantier, A., Perret, P., Bourgault, P. & Lambrechts, M.M. 2006. A thirty-year study of phenotypic and genetic variation of blue tits in Mediterranean habitat mosaics. *Bioscience* **56**: 661–674.

Cam, E. 2009. Contribution of capture-mark-recapture modeling to studies of evolution by natural selection. In: *Modeling Demographic Processes in Marked Populations* (D.L. Thomson, E.G. Cooch, & M.J. Conroy, eds), pp. 83–129. Series: Environmental and Ecological Statistics, Vol. 3, Springer Science, New York.

Choquet, R., Reboulet, A.M., Pradel, R., Gimenez, O. & Lebreton, J.-D. 2005. M-SURGE: new software specially designed for multistate capture–recapture models. *Anim. Biodivers. Conserv.* **27**: 207–215.

Choquet, R., Lebreton, J.-D., Gimenez, O., Reboulet, A. & Pradel, R. 2009. U-CARE: Utilities for performing goodness of fit tests and manipulating CApture–REcapture data. *Ecography* **32**: 1–4.

Coltman, D., O'Donoghue, P., Jorgenson, J., Hogg, J., Strobeck, C. & Festa-Bianchet, M. 2003. Undesirable evolutionary consequences of trophy hunting. *Nature* **426**: 655–658.

Coltman, D., O'Donoghue, P., Hogg, J. & Festa-Bianchet, M. 2005. Selection and genetic (co)variance in bighorn sheep. *Evolution* **59**: 1372–1382.

Damgaard, L.H. 2007. Technical note: How to use WinBUGS to draw inferences in animal models. *J. Anim. Sci.* **85**: 1363–1368.

Ducrocq, V. & Casella, G. 1996. A Bayesian analysis of mixed survival models. *Genet. Sel. Evol.* **28**: 505–529.

Falconer, D. & Mackay, T. 1996. *Introduction to Quantitative Genetics*, 4th edn. Prentice Hall, Harlow, UK.

Fisher, R. 1958. *The Genetical Theory of Natural Selection*, 2nd edn. Dover Pubications, New York.

Gelman, A. 1996. Inference and monitoring convergence. In: *Markov chain Monte Carlo in Practice*. (W.R. Gilks, S. Richardson, & D.J. Spiegelhalter, eds), pp. 131–143. Chapman and Hall, London.

Gelman, A. 2006. Prior distributions for variance parameters in hierarchical models. *Bayesian Anal.* **1**: 515–533.

Gelman, A., Meng, X. & Stern, H.S. 1996. Posterior predictive assessment of model fitness via realized discrepancies (with discussion). *Stat. Sin.* **6**: 733–807.

Gianola, D. 1982. Theory and analysis of threshold characters. *J. Anim. Sci.* **54**: 1079–1096.

Gimenez, O. & Choquet, R. 2010. Incorporating individual heterogeneity in studies on marked animals using numerical integration: capture–recapture mixed models. *Ecology* **91**: 951–957.

Gimenez, O., Covas, R., Brown, C.R., Anderson, M.D., Bomberger Brown, M. & Lenormand, T. 2006. Nonparametric estimation of natural selection on a quantitative trait using mark-recapture data. *Evolution* **60**: 460–466.

Gimenez, O., Rossi, V., Choquet, R., Dehais, C., Doris, B., Varella, H., Vila, J.-P. & Pradel, R. 2007. State–space modelling of data on marked individuals. *Ecol. Modell.* **206**: 431–438.

Gimenez, O., Viallefont, A., Charmantier, A., Pradel, R., Cam, E., Brown, C., Anderson, M., Bomberger Brown, M., Covas, R. & Gaillard, J.-M. 2008. The risk of flawed inference in evolutionary studies when detectability is less than one. *Am. Nat.* **172**: 441–448.

Grosbois, V., Gimenez, O., Gaillard, J.-M., Pradel, R., Barbraud, C., Clobert, J., Mller, A., & Weimerskirch, H. 2008. Assessing the impact of climate variation on survival in vertebrate populations. *Biol. Rev. Camb. Philos. Soc.* **83**: 357–399.

Harville, D. & Mee, R. 1984. A mixed-model procedure for analyzing ordered 1 categorical data. *Biometrics* **40**: 393–408.

Henderson, c.R. 1976. Simple method for computing inverse of a numerator relationship matrix used in prediction of breeding values. *Biometrics* **32**: 69–83.

Ihaka, R. & Gentleman, R. 1996. R: A language for data analysis and graphics. *J. Comput. Graph. Stat.* **5**: 299–314.

Kruuk, L. 2004. Estimating genetic parameters in natural populations using the 'animal model'. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **359**: 873–890.

Kruuk, L., Clutton-Brock, T., Pemberton, J., Brotherstone, S. & Guinness, F. 2000. Heritability of fitness in a wild mammal population. *Proc. Natl Acad. Sci. USA* **97**: 698–703.

Kruuk, L., Slate, J. & Wilson, A. 2008. New answers for old questions: the quantitative genetics of wild animal populations. *Annu. Rev. Ecol. Evol. Syst.* **35**: 525–548.

Kuo, L. & Mallick, B. 1998. Variable selection for regression models. *Sankhya Ser. B* **60**: 6581.

Law, R. & Stokes, T. 2005. Evolutionary impacts of fishing on target populations. In: *Marine Conservation Biology: The Science of Maintaining the Sea's Biodiversity* (E. Norse & L. Crowder, eds), pp. 232–246. Island Press, Washington.

Lebreton, J.-D., Burnham, K.P., Clobert, J. & Anderson, D.R. 1992. Modeling survival and testing biological hypotheses using marked animals: A unified approach with case studies. *Ecol. Monogr.* **62**: 67–118.

Lynch, M. & Walsh, B. 1998. *Genetics and Analysis of Quantitative Traits*. Sinauer Associates, Sunderland, MA.

Merilä, J., Sheldon, B. & Kruuk, L. 2001. Explaining stasis: microevolutionary studies in natural populations. *Genetica.* **112–113**: 199–222.

Meyer, K. 1998. Estimating covariance functions for longitudinal data using a random regression model. *Genet. Sel. Evol.* **30**: 221–240.

Morrissey, M. & Wilson, A. 2010. PEDANTICS: an R package for pedigree-1 based genetic simulation and pedigree manipulation, characterization and viewing. *Mol. Ecol. Resour.* 10.1111/j.1755-0998.2009.02817.x.

O'Hara, R. & Sillanpää, M. 2009. A review of bayesian variable selection methods: what, how, and which. *Bayesian Anal.* **4**: 85–118.

O'Hara, R., Cano, J., Ovaskainen, O., Ahlo, J. & Teplitsky, C. 2008. Bayesian approaches in evolutionary quantitative genetics. *J. Evol. Biol.* **21**: 949–957.

Quaas, R.L. 1989. Transformed mixed model-equations – A recursive algorithm to eliminate A^{-1}. *J. Dairy Sci.* **72**: 1937–1941.

Royle, J.A. 2008. Modeling individual effects in the Cormack–Jolly–Seber model: a state–space formulation. *Biometrics* **64**: 364–370.

Sorensen, D. & Gianola, D. 2002. *Likelihood, Bayesian and MCMC Methods in Quantitative Genetics*. Springer, New York.

Sorensen, S., Erson, D., Gianola, D. & Korsgaard, I. 1995. Bayesian inference in threshold models using gibbs sampling. *Genet. Sel. Evol.* **27**: 229–249.

Stockwell, C., Hendry, A. & Kinnison, M. 2003. Contemporary evolution meets conservation biology. *Trends Ecol. Evol.* **18**: 94–100.

Sturtz, S., Ligges, U. & Gelman, A. 2005. R2WinBUGS: A package for running winbugs from r. *J. Stat. Softw.* **12**: 1–16.

Thomas, A., O'Hara, R., Ligges, U. & Sturtz, S. 2006. Making BUGS open. *R News* **6**: 12–17.

Véran, S., Gimenez, O., Flint, E., Kendall, B., Doherty P. & Lebreton, J.-D. 2007. Quantifying the impact of longline fisheries on adult survival in the black-footed albatross. *J. Appl. Ecol.* **44**: 942–952.

Waldmann, P. 2009. Easy and flexible bayesian inference of quantitative genetic parameters. *Evolution* **63**: 1640–1643.

Supporting information

Additional supporting information may be found in the online version of this article:

Data S1 R script and BUGS code to implement the 5 capture–recapture animal model.

Data S2 Posterior predictive checking.

As a service to our authors and readers, this journal provides supporting information supplied by the authors. Such materials are peer-reviewed and may be re-organized for online delivery, but are not copy-edited or typeset. Technical support issues arising from supporting information (other than missing files) should be addressed to the authors.

Received 27 April 2010; revised 25 June 2010; accepted 8 July 2010