title: Glucocorticoid levels are linked to lifetime reproductive success and survival of adult barn owls

short title: Glucocorticoids and Fitness in Barn owls

Paul Béziers[1,2], Fränzi Korner-Nievergelt[1], Lukas Jenni[1], Alexandre Roulin[2,†], Bettina Almasi[1,†]

[1]*Swiss Ornithological Institute, Sempach, Switzerland*

[2]*Department of Ecology and Evolution, University of Lausanne, Lausanne, Switzerland*

[†]*Shared senior authorship*

corresponding author:
Paul Béziers
paul.beziers@unil.ch
tel. +41 79 757 09 58

University of Lausanne
Department of Ecology and Evolution
Biophore building
1015 Lausanne, Switzerland

Abstract

Glucocorticoid hormones, such as corticosterone, are crucial in regulating daily life metabolism and energy expenditure, as well as promoting short-term physiological and behavioural responses to unpredictable environmental challenges. Therefore, glucocorticoids are considered to mediate trade-offs between survival and reproduction. Relatively little is known about how selection has shaped glucocorticoid levels. We used 15 years of capture-recapture and dead recovery data combined with 13 years of corticosterone and breeding success data taken on breeding barn owls (*Tyto alba*) to investigate such trade-offs. We found that survival was positively correlated with stress-induced corticosterone levels in both sexes, while annual and lifetime reproductive success (i.e. the sum of young successfully fledged during the entire reproductive career) was positively correlated with both baseline and stress-induced corticosterone levels in females only. Our results suggest that, in the barn owl, the stress-induced corticosterone response is a good proxy for adult survival and lifetime reproductive success. However, selection pressure appears to act differently on corticosterone levels of males and females.

keywords: glucocorticoids, corticosterone, stress, reproductive success, survival, fitness, bird, barn owl, multistate model

Introduction

Hormones orchestrate many characteristics of an organism's morphology, physiology and behaviour, and are thus critical components in the evolution of many traits (Wingfield et al., 1998, Dallman et al., 2004, Landys et al., 2006, McEwen, 2008). Because of their capacity to regulate and coordinate suites of physiological traits, hormones are thought to be essential in the regulation of fitness components such as reproduction and survival (Ketterson & Nolan, 1992, Romero, 2002, Wingfield & Sapolsky, 2003, Crespi et al., 2013). Although there is prior empirical evidence of relationships between hormones and fitness components (Breuner et al., 2008, Bonier et al., 2009a), a better knowledge of the selection processes and mechanisms that regulate fitness components is essential to understand how different life histories have evolved. In particular, we need more information about how intraspecific variation in the endocrine system mediates different fitness components.

Glucocorticoid hormones (GCs) are important transducers between an organism and its environment, allowing it to adopt appropriate physiological and behavioural responses to cope with environmental perturbations. The environment is a source of unpredictable perturbations and challenges (e.g. social interaction, diseases, predators, competitors), which has led to the evolution of important variations in GC levels among populations and individuals within populations. Baseline GC levels are responsible for maintaining energy homeostasis in relation to energetic demands (Carsia & Harvey, 2000, Sapolsky et al., 2000, Romero, 2002, Landys et al., 2006, Roulin et al., 2010). GCs are also part of the adrenocortical stress response that allows the reallocation of resources to physiological functions and behaviours that are essential to self-maintenance and survival when the environment becomes unpredictably challenging (e.g. predator attack, food restriction, inclement weather).

Therefore, GCs play an essential role in mediating trade-offs between different life history traits and, as a consequence, can be associated with fitness components (Ricklefs & Wikelski, 2002, Zera et al., 2007, Hau et al., 2010, Crespi et al., 2013). Besides, in some species GC levels have been shown to have a heritable component (Brown & Nestor, 1973, Satterlee & Johnson, 1988, Pottinger & Carrick, 1999, Odeh et al., 2003, Evans et al., 2006, Jenkins et al., 2014, Béziers et al., 2019) implying that GC levels can respond to natural selection.

It is commonly assumed that low baseline GC levels favour survival because high and chronically elevated GC levels can impair the health of an individual (de Kloet et al., 1999, Sapolsky et al., 2000, Bremner, 2007, Breuner et al., 2008, Martin, 2009). In contrast, strong stress-induced GC levels favour reallocation of resources towards self-maintenance at the expense of reproduction (Almasi et al., 2008, Ouyang et al., 2012). The empirical evidence supporting these predictions are however mixed, with some studies having found positive, negative or no relation between GCs and fitness components (reviewed in Breuner et al., 2008, Bonier et al., 2009a). Three main hypotheses with different assumptions depending on the measure of GC (baseline or stress-induced levels) and/or the stage at which individuals are sampled (e.g. incubation or parental care stages) have been suggested to explain these equivocal relationships. The *CORT (for corticosterone)-trade-off hypothesis* suggests that GCs mediate the trade-off between survival and reproduction and thus predicts that a short-term increase of (baseline or stress-induced) GC should promote survival at the expense of reproduction (Wingfield & Sapolsky, 2003, Almasi et al., 2013, Patterson et al., 2014). Under this tenet, GC levels are expected to be positively associated with survival and negatively with reproductive success. The *CORT-fitness hypothesis* (Bonier et al., 2009a) proposes that environmental challenges should at the

same time increase (baseline) GC levels and reduce fitness because resources must be reallocated to support these challenges at the expense of current reproduction or self-maintenance. Under this second tenet, GC levels are expected to be negatively associated with both survival and reproductive success. Finally, the *CORT-adaptation hypothesis* (Bonier et al., 2009b) suggests that high (baseline) GC levels increase reproductive success by allocating more resource to behaviours that promote reproductive activities such as increased parental care, and thus GC levels are expected to be positively associated with reproductive success. Despite the evidence of an association between GC and fitness in the literature, so far few studies have investigated the association between GC levels and multiple fitness components (Table 1, Lancaster et al., 2008, Schmid et al., 2013, Patterson et al., 2014, Vitousek et al., 2018). Furthermore, results across these studies are equivocal, potentially because of differences in life history strategies or sex-specific roles across species or other context-dependent factors (e.g. variation in environmental conditions across seasons, years, populations or species, variation in corticosterone levels across life history stages, etc.). Therefore, it is still unclear how selection acts on GC levels, and thus how GCs are implicated in mediating life history trade-offs. Consequently, we need more studies that relate GCs to life history measures. This type of data is difficult to gather because it requires the assessment of hormonal levels in many individuals over a long period of time.

In the present study, we investigated the relation between GC measurements and fitness in a free-living population of barn owls (*Tyto alba*). To do so, we used capture-recapture and death recovery data as well as breeding records collected during 15 years, with corticosterone measurements collected during 13 years in parallel. We evaluated the relationship between the GCs levels and fitness components in view of

current thinking about selection patterns for GCs. The fitness measures used for the analyses were lifetime reproductive success (i.e. the sum of young successfully fledged during the entire reproductive career), average annual reproductive success, and adult survival probability.

Material and Methods

Study site and species

The barn owl is a medium-sized bird of prey that lives in open rural landscapes where it hunts small mammals. In our study area, barn owls commonly breed in artificial nest-boxes fixed to the wall of barns. From mid-February to the beginning of August, females lay one to two clutches, each comprising between 2 and 11 eggs (Béziers & Roulin, 2016). Females start incubation as soon as the first egg is laid, which results in hatching asynchrony of nestlings. This asynchrony can lead to a pronounced age hierarchy within the nest with the oldest nestling being up to 25 days older than its youngest sibling. After hatching, the mother stays in the nest until the offspring can thermoregulate and feed by themselves. The male hunts and supplies the female and offspring with prey during this period. Once the first-hatched nestlings can feed and thermoregulate by themselves, the mother usually assists the male in food provisioning. In our population breeding pairs are rather faithful to their breeding site: 78 % of pairs that stay together from one year to the next remain at the same breeding site (Dreiss & Roulin, 2014). The typical lifespan of barn owls is four years, but individuals up to 15 years have been recorded (Altwegg et al., 2007). Reproduction and survival in our study population are being monitored since 1990.

Our study was conducted between 2004 and 2018 in western Switzerland. All nest-boxes were checked once a month from March until September to determine

where barn owls breed. Additional visits were made to mark nestlings and adults, determine clutch size and number of fledglings. Adult females (n = 338 individuals) and males (n = 191) were captured at the end of the incubation stage or during nestling provisioning (Table 2). If an adult had not been marked as a juvenile, its age was estimated from the moult pattern (i.e. the sequence of replacement of primary feathers) of wing feathers (Taylor, 1993). We distinguished females from males by their incubation behaviour and the presence of a brood patch (only the mother incubates the eggs).

Assessment of baseline and stress-induced corticosterone levels

To assess corticosterone levels (i.e. the main GC in birds), adult barn owls were captured and submitted to the same standardised capture-restrain protocol (Wingfield et al., 1994). A first blood sample was taken within 3 minutes (mean ± SD: 2 min 22 s ± 31 s) after first disturbance (e.g. when entering the barn or triggering the trap) to assess baseline corticosterone levels (n = 741 samples; Table 2). Although the increase in corticosterone level during the first 3 minutes after an acute stress is marginal (Romero & Reed, 2005, Roulin et al., 2010), we considered sampling time in our statistical analyses (Table 3). The blood sample was taken by puncturing the brachial vein and then collecting with heparinised capillaries. Blood samples were directly centrifuged, the plasma was separated and flash-frozen in liquid nitrogen. Once back from the field (within <24 hours), the samples were stored at –20 °C until analysis within the next six months. After having collected this first blood sample (i.e. baseline sample) the birds were weighed, the length of their wing measured to the nearest mm, and then placed in an opaque cloth bag until a second blood sample was taken 25 minutes (mean ± SD: 23 min 59 s ± 1 min 42 s) after first disturbance to

146 measure the stress-induced corticosterone response (n = 753; Table 2). This time span
147 represents the peak of stress-induced corticosterone levels in the barn owl (Almasi et
148 al., 2015).
149
150 *Total corticosterone assay*
151 Plasma corticosterone was extracted with dichloromethane and determined with an
152 enzyme immunoassay (Munro & Stabenfeldt, 1984, Munro & Lasley, 1988)
153 following Müller et al. (2006). Ten microliters of plasma were added to 190 µl of
154 water and extracted with 4 ml of dichloromethane. The solution was mixed for 30
155 minutes on a vortex machine and then incubated for two hours. After separating the
156 water phase, the dichloromethane was evaporated at 48 °C and corticosterone was re-
157 suspended in a phosphate buffer. The dilution of the corticosterone antibody
158 (Chemicon; cross-reactivity: 11-dehydrocorticosterone 0.35 %, progesterone 0.004 %,
159 18-OH-DOC 0.01 % cortisol 0.12 %, 28-OH-B 0.02 % and aldosterone 0.06 %) was
160 1:8,000. We used Horseradish Peroxidase (HRP) (1:400,000) linked to corticosterone
161 as enzyme label and 2,2'-azino-bis (3-ethylbenzothiazoline-6-sulphonic acid) (ABTS)
162 as substrate. We determined the concentration of corticosterone in triplicate by using
163 a standard curve run in duplicate on each plate. If the corticosterone concentration
164 was below the detection threshold of 1 ng ml^{-1}, the analysis was repeated with 15 µl
165 or 20 µl plasma. Eighteen of these repeated determination samples were still below
166 the detection limit and were therefore assigned 1 ng ml^{-1} (the detection limit of the
167 assay). This corresponds to 1.2 % of the total corticosterone samples used for our
168 study (18 of total 1,494 samples). Plasma pools from chicken with a low and high
169 corticosterone concentration were included as internal controls on each plate. Intra-
170 assay variation ranged from 3 % to 14 % (mean ± SD: 8.8 % ± 3.8) and inter-assay

171 variation from 7 % to 22 % (mean ± SD: 16.5 % ± 5.8), depending on the
172 concentration of the internal control and the year of analysis.
173
174 *Survival Analyses*
175 We used a multistate capture-recapture and death recovery model to estimate survival
176 of barn owl adults as related to plasma baseline and stress-induced corticosterone
177 levels (Lebreton et al., 2009). The model allowed us to estimate the probability of
178 recapturing an individual given it is alive and did not permanently emigrate from the
179 study area at time t (p), and its apparent survival from time t to time t +1, (ϕ). Our
180 multistate model had three states: "alive", "freshly dead" and "dead", and the possible
181 observations were "captured alive", "recovered dead" and "not seen, not captured, or
182 not recovered". We modelled apparent survival probability (ϕ) in relation to different
183 covariates, including age (linear and quadratic) and sex in interaction with
184 corticosterone levels, using the logit-link function.
185 For examining correlations between corticosterone and apparent annual
186 survival, corticosterone values should be measured comparably across years and
187 individuals, because the sampling unit of the mark-recapture model is an individual /
188 year combination (capture history matrix). The raw corticosterone measurements,
189 however, were collected at every capture of an individual, i.e. from zero to several
190 times within the same year (range: 0 – 4). Furthermore, raw measurements were
191 influenced by sex, age, body mass, sampling date (i.e. day of the year), time (i.e.
192 hour), sampling latency (i.e. time span between capture and blood sampling), brood
193 size (a proxy of reproductive investment), and stage at which individuals were
194 sampled, i.e., during the incubation vs. offspring provisioning period (Table 3). All
195 these influencing factors hamper a comparison of raw corticosterone measurements

196 across individuals and years. Therefore we used corticosterone values per individual
197 and year estimated from linear mixed models using year and individual-specific
198 random effects (Robinson, 1991), one for baseline and one for stress induced-
199 corticosterone as the outcome variable (see Fig. 1 for a schematic representation of
200 the survival modelling process). In the mixed models the influencing factors (listed
201 above; Table 3) were fixed effects, with identity of the individuals, brood identity and
202 year included as random factors (intercepts). We thus obtained one mean
203 corticosterone estimate typical for an individual and year corrected for age, body
204 mass, sampling date and time, sampling latency, brood size and stage. Importantly, by
205 estimating random intercepts for year, brood and individual, corticosterone values
206 could be estimated for individual and year combinations even when an individual was
207 not captured (5.47 % of individual-year combinations), thereby assuming no
208 interaction between individual and year (% explained variance 0.02). Before fitting
209 the model, all numeric variables were centered and scaled ((x-mean[x]) / (2 × standard
210 deviation[x])). Baseline corticosterone was ln-transformed (ln[y] + 1) to improve the
211 normality assumption, whereas for stress-induced corticosterone the model fit was
212 better using untransformed measurements. As a consequence of the different
213 transformations of the two corticosterone variables, the estimated effect sizes of the
214 baseline and stress-induced corticosterone and survival models cannot directly be
215 compared. However, the purpose of the corticosterone models was to obtain
216 comparable corticosterone estimates for each individual and year, although a good
217 model fit is more important than obtaining comparable effect sizes. In order to make
218 the effects of the two variables on apparent survival comparable, we nevertheless
219 present the effects of each corticosterone variable on apparent survival graphically
220 using the original (untransformed) scale.

The two normal linear mixed models (one for baseline and one for stress-induced corticosterone) were fitted to the data using the function *lmer* from the package *lme4* (Bates et al., 2015) in R (version 3.3.2). The uncertainties of the model (including the variance) parameters were measured by Monte Carlo simulation. Five thousand random values from the joint posterior distribution of the model parameters were simulated using the function sim from the R-package arm (Gelman & Hill, 2007). The 2.5 % and 97.5 % quantiles of these simulated values were used as the lower and upper bands of the 95 % credible interval (i.e. 95 % CrI of the marginal posterior distribution for each parameter).

The individual and year-specific corticosterone values were then, together with sex and age (linear and quadratic), included as predictors for apparent survival in the mark-recapture/recovery model (Fig. 1). Recapture probability was modelled in relation to the number of available nest-boxes in our study area (the number of nest-boxes varied from 123 to 291 between 2004 and 2018). We modelled the probability of recovering a dead individual as constant throughout the study period. The capture-recapture/recovery matrix started with the individual at age 1 (i.e. hatching year + 1) and included only individuals from which we had at least one corticosterone measurement as an adult. We used uninformative priors for the different parameters (intercepts: normal [mean = 0, SD = 1.5], coefficients: normal [0, 5]). All live recapture and death recovery data were derived from the European Union for Bird Ringing (EURING) data bank for birds ringed inside or outside our area, so were thus not restricted to our study area.

The capture-recapture/recovery models were fitted in JAGS (Plummer, 2003) version 4.2, using the package *RJAGS* version 4.6 (Plummer, 2016) in R version 3.3.2 (R Core Team, 2018). JAGS uses Markov Chain Monte Carlo (MCMC) simulations

to estimate parameters. We simulated three chains with 510,000 iterations, a burn-in phase of 10,000 iterations, and a sampling interval of 100 iterations. The mean effective sample size for the model parameters was 12,961 and the lowest 4,723. We visually inspected the chains and used the R-hat statistics to assess the convergence of all chains (Gelman & Hill, 2007). R-hat values for each parameter were close to 1. We also performed posterior predictive model checking to estimate the goodness of fit of our models. To do so, we simulated 1,000 data sets from our models and compared the observed data with the replicated data (Nichols et al., 1984, Table S1).

For the corticosterone effect on survival, we calculated the selection coefficient for each sex separately averaged over all age classes from 1 to 12 (the maximum age observed) following Janzen and Stern (1998).

Annual and lifetime reproductive success

We measured correlations between corticosterone levels and reproductive success. We used lifetime reproductive success as the outcome variable in two different normal linear mixed models, one including the individual's lifespan (here, lifespan refers to the maximum age that a bird was found reproducing) and one excluding its lifespan. In the first model, correlations between corticosterone and average annual reproduction were estimated, whereas the second model estimated correlations between corticosterone and lifetime reproductive success. Both models were used once with baseline and once with stress-induced corticosterone as predictor. In each model, we included the individual-specific corticosterone values (averaged across the lifetime of the individual), the sex of the individual, as well as the interaction sex $\times$ corticosterone as fixed effects, plus the cohort (year of birth) as a random factor. As reproductive success we counted all nestlings that survived until fledging for all the

271 breeding individuals used in the survival model, excluding individuals younger than
272 four years old in 2018 (four years corresponds to the mean lifespan of barn owls).
273 Such a procedure is possible in the barn owl because adults are sedentary and very
274 few emigrate from the study area (Altwegg et al., 2003, van den Brink et al., 2012,
275 Dreiss & Roulin, 2014). However, given that we cannot be sure to have access to the
276 entire reproductive history of each individual because some individuals may have
277 reproduced out of our study area or somewhere we did not notice, our lifetime
278 reproductive success estimate corresponds to a "minimum lifetime reproductive
279 success". For the normal linear mixed models we used the function *lmer* from the
280 package *lme4* (Bates et al., 2015) and the function sim (Gelman and Hill, 2007) to
281 obtain 95 % CrI of the parameter estimates as described above for the corticosterone
282 linear mixed models.

283 Since corticosterone in eggs increases with laying order (Love et al., 2008), we
284 analysed whether baseline and stress-induced corticosterone levels during incubation
285 were associated with clutch size, to determine whether the correlation of
286 corticosterone with our lifetime reproductive success measure could be an indirect
287 effect of the number of eggs laid by a female. We fitted a normal linear mixed model
288 to the clutch size data with laying date (i.e. day of the year), sex and baseline
289 corticosterone levels of individuals during incubation stage plus their interaction (sex
290 × corticosterone levels) as fixed factors, and year and individual identity as random
291 factors (random intercept). The 95 % CrI were obtained as described above for the
292 corticosterone models. The same model was fitted separately with stress-induced
293 corticosterone instead of baseline as predictor variable.

294

295 **Results**

296 The baseline corticosterone survival model was based on 78 males and 145 females
297 marked as juveniles plus 59 males and 144 females marked as adults. From these 426
298 individuals, 29 (including two recovered outside our study area) were recovered dead
299 during the study period (mean ± SD age at recovery: 4.2 ± 2.1 years), and 426
300 individuals were recaptured a total of 794 times (Fig. 2; mean ± SD age at recapture:
301 2.6 ± 1.9; including 98 live recaptures outside of our study area). The stress-induced
302 corticosterone survival model was based on a total of 82 males and 134 females
303 ringed as juveniles plus 54 males and 137 females ringed as adults. From these 407
304 individuals, 19 (including one outside our study area) were recovered dead during the
305 study period (mean ± SD age at recovery: 3.6 ± 1.6), and 407 different individuals
306 were recaptured a total of 764 times (Fig. 2; mean ± SD age at recapture: 2.4 ± 1.8
307 years; including 81 recaptured outside our study area). The age of the oldest
308 individual recovered dead was 11 years, and that of the oldest individual recaptured
309 alive was 12 years (Fig. 2).
310
311 *Individual and annual corticosterone values*
312 The number of baseline and stress-induced corticosterone samples per individual
313 ranged from 1 to 12 (mean ± SD: 1.7 ± 1.3) and from 1 to 11 (mean ± SD: 1.8 ± 1.5),
314 respectively. Most of the baseline and stress-induced corticosterone samples were
315 from individuals between one and three years old (> 81 %; Fig. 2C). Average baseline
316 and stress-induced corticosterone levels decreased over the season (Table 3), whereas
317 corticosterone levels increased from the incubation stage to the rearing period (Table
318 3). Older birds had on average higher baseline corticosterone levels than younger
319 individuals, whereas the age relationship of stress-induced corticosterone tended to be
320 the opposite. Adults with large broods had higher baseline corticosterone levels than

individuals with small broods. Body mass was more strongly negatively associated with stress-induced than with baseline corticosterone levels. Sampling latency and brood size were not clearly associated with stress-induced corticosterone levels. The time of the day and sex of individuals (Fig. 3) were neither clearly associated with baseline corticosterone levels nor with stress-induced corticosterone levels (all statistics in Table 3).

Survival estimates

We found no strong association between baseline corticosterone levels and survival in male barn owls (β = -0.12; 95 % CrI [-1.25 – 1.01]) or female (0.29 [-0.54 – 1.11]; Fig. 4A). However, females with higher baseline corticosterone levels tended to show higher survival than females with lower baseline levels (posterior probability of the hypothesis that baseline corticosterone levels is positively associated with female survival was 0.7). There was a clear positive association between stress-induced corticosterone levels and survival probability in females (1.19 [0.44 – 1.93], Table 4; Fig. 4B, posterior probability of the hypothesis that stress-induced corticosterone levels is positively associated with female survival was 0.99). Stress-induced corticosterone levels were also positively associated with male survival (0.76 [-0.24 – 1.78]); however, the relationship was less pronounced than for females (Table 4; Fig. 4B, posterior probability of the hypothesis that stress-induced corticosterone levels is positively associated with male survival was 0.91). Survival probability was further associated with age in a curvilinear way (Table 4). Survival increased slightly during the first six years of life before decreasing again at older ages (Fig. S1). The probabilities of recapturing an individual were constant through time and hence not clearly associated with the number of nest-boxes available in the study area (Table 4).

Finally, the probability of dead recovery and recapture was similar for the baseline model (probability of dead recovery: 0.08 [0.05 – 0.11], recapture probability: 0.88 [0.84 – 0.91]) and the stress-induced model (probability of dead recovery: 0.05 [0.03 – 0.08], recapture probability: 0.89 [0.86 – 0.92]). Correspondingly, we found no clear directional selection gradient for female or male baseline corticosterone levels (Table 4). On the other hand, female but not male stress-induced corticosterone levels are under directional selection (Table 4). Posterior model checking showed that the observed frequency of individual recapture as well as the number of recaptures and dead recoveries was within the range of the simulated data set for both the baseline and the stress-induced models (Table S1). Overall, our models fitted well to our dataset.

Annual and lifetime reproductive success

There was a clear relationship between female baseline and stress-induced corticosterone levels and her annual and lifetime reproductive success. Females with high baseline or stress-induced corticosterone levels produced more fledglings per year (Table 5A), and also a larger total number of fledglings during their lifetimes (Table 5B; Fig. 5). On the other hand, there was no clear relationship between male baseline and stress-induced corticosterone levels and his annual and lifetime reproductive success (Table 5). Correspondingly, we found a clear directional selection gradient for female baseline and stress-induced corticosterone levels (Table 5) but not for males (Table 5). Unsurprisingly, lifespan explained part of the variation in lifetime reproductive success, for both sexes (Table 5A).

Finally, clutch size was positively associated with laying date. Baseline and stress-induced corticosterone levels were not clearly associated with clutch size

(Table 6). Consequently, we found no evidence of directional selection for female or male baseline or stress-induced corticosterone levels with clutch size (Table 6).

Discussion

We here used a long-term data set from barn owls to investigate the relationships between corticosterone levels and major fitness components. Overall our results show that our two different corticosterone measures (baseline and stress-induced) correlate positively with survival, annual and lifetime reproductive success. However, we observed that these relationships differed between the sexes, suggesting that selection may differ between the sexes and different components of the stress axis. Given that our study is correlative, we are aware that we cannot exclude the possibility that selection is operating on other traits associated with fitness and corticosterone.

Survival

Our results merely suggest with high uncertainty that females with high baseline corticosterone levels during the breeding season survive better, while no relationship whatsoever was found for males. Overall, therefore, we have no clear evidence of positive directional selection on baseline corticosterone levels (Table 4; Fig. 4A). As for other fitness traits, the association between corticosterone and survival might be complex and context-dependent, as various studies investigating this link in other species have yielded mixed results (Bonier et al., 2009a). The lack of strong association between baseline corticosterone and survival in our study may also result from the incapacity of controlling all parameters potentially affecting baseline corticosterone levels (e.g. environmental factors, disease, climate, etc.).

In contrast, for stress-induced corticosterone levels we found a positive link with survival, suggesting positive directional selection on stress-induced corticosterone levels (Table 4; Fig. 4B). However, the effect for males was less marked than for females. Brief increases in corticosterone levels are thought to promote functions that are vital to self-maintenance at the expense of less-essential functions like reproduction (i.e. *CORT trade-off hypothesis*). For instance, individuals with higher stress-induced corticosterone levels show better fight- or flight- capacities (Overli et al., 2002a). A large increase in corticosterone levels is also known to stimulate activity, energy acquisition and storage through the glucocorticoid type II receptor (Dallman et al., 1995, Dallman et al., 2004, see also Vera et al., 2017 for discussion). Note that the role of the stress response is to reduce allostatic load, i.e. when energy demand exceeds energy income and hence maintaining homeostasis becomes costly for an organism, and to restore homeostasis as rapidly as possible to prevent damage (Sapolsky et al., 2000, McEwen & Wingfield, 2003). Therefore, an organism with high stress-induced corticosterone levels might regain homeostasis faster when foraging (Overli et al., 2002b, Lynn et al., 2003) or feeding more actively (Cote et al., 2006). For example, female tree swallows (*Tachycineta bicolor*) with higher corticosterone stress response levels and a stronger negative-feedback were shown to be more resilient to stressors (Zimmer et al., 2019). Evidence of selection on stress-induced corticosterone levels in other species is mixed, however, with some studies having found positive (Cabezas et al., 2007, Angelier et al., 2009, Patterson et al., 2014) and others negative associations with fitness components (Romero & Wikelski, 2001, Blas et al., 2007, MacDougall-Shackleton et al., 2009), or relationships only in one sex (Jimeno et al., 2018). These equivocal results suggest that selection may operate differently between sexes, species, and life history stages.

420

421 *Annual and lifetime reproductive success*

422 We found similar positive relationships with reproductive success for baseline and
423 stress-induced corticosterone levels but contrasting results for males and females (Fig.
424 5). Females with high baseline and stress-induced corticosterone levels have higher
425 annual and lifetime reproductive success than females with low corticosterone levels.
426 These results are not mediated by corticosterone affecting clutch size, as we found no
427 relationship between baseline or stress-induced corticosterone levels and clutch size.
428 By contrast, we found no relationships between corticosterone levels and annual and
429 lifetime reproductive success in males.

430 In the barn owl, females incubate the eggs alone while males provide most of
431 the food. Although offspring provisioning is mainly done by the male, there is
432 considerable variation between females in their participation in food provisioning,
433 with some females actively participating and others not at all (personal observation).
434 The positive relationship between female baseline corticosterone levels and both
435 annual and lifetime reproductive success could, therefore, result from corticosterone
436 promoting maternal food provisioning. Accordingly, several studies have shown that
437 moderate increase of baseline corticosterone during the breeding season can promote
438 parental care (Bonier et al., 2009a, Crossin et al., 2012, Ouyang et al., 2013a). In our
439 study, baseline corticosterone levels could reflect different levels of investment in
440 maternal care. Such results are in line with the *CORT-adaptation hypothesis* (Bonier
441 et al., 2009b).

442 Females with stronger stress-induced response produced more offspring over
443 their lifetime. Because high GC levels are thought to promote physiology and
444 behaviours that enhance survival at the expense of other functions like reproduction,

individuals having stronger stress-induced GC response are generally expected to have lower reproductive success according to the *CORT-trade-off hypothesis* (Wingfield & Sapolsky, 2003, Lendvai et al., 2007, Breuner et al., 2008, Almasi et al., 2013). Although, such relationships have been found in short-lived bird species like passerines (Schmid et al., 2013, Patterson et al., 2014, Vitousek et al., 2014), this may not hold for long-lived bird species. Indeed, to maximise fitness, life history theory predicts that an individual should balance costs between current reproduction and future survival (Stearns, 1989). Whether an individual should invest more resources into reproduction or survival depends on how each of these traits helps to maximise its fitness. Long-lived birds are expected to favour survival over reproduction because their lifetime reproductive success depends more on their lifespan (and perhaps experience) than on their annual reproduction, an assumption that holds for barn owls given that longer-lived individuals produce more fledglings over their lifetime (Table 5) than short-lived individuals. Although barn owls live on average four years (Altwegg et al., 2007; maximum in our population: 15 years), surviving one more year may significantly increase their fitness. These results support the idea that GCs are involved in the mediation of trade-offs between current and future reproduction (Bókony et al., 2009, Schoenle et al., 2018).

In contrast to the significant relationships between female corticosterone levels and annual and lifetime reproductive success, male corticosterone levels were surprisingly unrelated to reproductive success. Although males do most of the food provisioning, our results indicate that corticosterone levels during the breeding season are not a good predictor of male reproductive success. An alternative could be that male reproductive success may depend to a large extent on female behaviour and how much females invest or not in parental care. Further studies investigating the

interaction of parental care and corticosterone levels will be necessary to understand the role of corticosterone for male reproductive success.

Conclusion

Our study showed that (baseline and stress-induced) corticosterone levels during the breeding season were positively related to female but not male barn owl annual and lifetime reproductive success. Further, stress-induced but not baseline levels were positively correlated with female and male barn owl survival. The lack of viability selection on baseline corticosterone levels may be due to reduced statistical power or to the high sensitivity of baseline corticosterone levels to several environmental factors or life history stages (Angelier et al., 2009, Cockrem et al., 2009, Rensel & Schoech, 2011, Hennessy et al., 2015, Schoenemann & Bonier, 2018). Overall, our results suggest that corticosterone levels in the barn owl are under positive directional selection (if at all). However, the relative strength of selection varies between sexes and corticosterone levels.

So far, most studies having investigated the link between GCs and fitness parameters explored either reproductive success or survival and rarely multiple fitness components. Moreover, studies exploring reproductive success mostly used seasonal reproductive success rather than lifetime reproductive success parameters as a reproductive proxy. Nevertheless, GCs are thought to mediate the trade-offs between current and future reproduction, implying that selection could have differently shaped GCs levels depending on species and life history traits. Thus, species may differ in their circulating hormone levels during the reproductive period due to their different sex-specific fitness optima (e.g. short- versus long-lived organism). Our study provides evidence that the selection pressures on GC levels can differ between males

and females, and thus contributes to a better understanding of the evolution of GC levels and their associated fitness traits.

Acknowledgements

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Table 1. Studies reporting relationships between reproduction and survival and (baseline and stress-induced) corticosterone (CORT) levels of adult vertebrates during the breeding season.

			Reproductive success*		Survival		
Species	Method	Breeding stage	Baseline CORT†	Stress-induced CORT†	Baseline CORT†	Stress-induced CORT†	Reference
Side-blotched lizard (*Uta stansburiana*)	E_c	prior to reproduction		+/–♀^{p}		+/–♀^{p}	Lancaster et al. (2008)
Tree swallows (*Tachycineta bicolor*)	C	incubation	–♀		–♀		Bonier et al. (2009b)
		brooding	+♀				
Great tits (*Parus major*)	C	prior to clutch initiation	+♂	n.s. ♂	n.s. ♂	n.s. ♂	Ouyang et al. (2013b)
		brooding	–♂	n.s. ♀, –♂	n.s. ♂	n.s. ♂	
Eurasian hoopoe (*Upupa epops*)	C	brooding		–♀, n.s. ♂		n.s. ♂	Schmid et al. (2013)
Mountain white-crowned sparrow (*Zonotrichia leucophrys oriantha*)	C	prior to clutch initiation	+♀^{a}		+ ♂	+ ♂	Patterson et al. (2014)
Tree swallow (*Tachycineta bicolor*)	C	incubation	–♀^{c}, n.s. ♂	+♀^{i}, n.s. ♂	n.s. ♂	n.s. ♂	Vitousek et al. (2018)
		brooding	+/–♀^{i,c}, n.s. ♂	–/+♀^{i}, n.s. ♂	n.s. ♂	n.s. ♂	
Great tits (*Parus major*)	E_W	brooding	n.s. ♂		n.s. ♂		Casagrande and Hau (2018)

Method employed: C, correlative study; E_c, experimental manipulation of CORT levels; E_W, experimental manipulation of workload by feather clipping.

*depending on the study, reproductive success refers to the total number of fledglings produced in a breeding season (annual fledgling success) and/or rearing success (proportion of eggs that produced fledglings) and condition of nestlings.

† depending on the study, baseline CORT corresponds to free and/or total CORT while stress-induced CORT corresponds to free CORT, max free CORT, max total CORT response, or fold increase in free and total CORT.

Results: n.s., non-significant; –, negative relationship; +, positive relationship; +/–, suggest that there is an interaction and therefore the relation may be positive or negative depending on the context. Some of the results reported in these studies depend on sex (♀, ♂, ⚥), ^{p} genotype (i.e. yellow- and orange-throated), ^{c} environmental condition (i.e. good vs bad year), ^{a} age class (adult vs yearling), or ^{i} interaction between baseline CORT × stress-induced CORT levels.

Table 2. Number of corticosterone samples assessed from 2004 to 2018 in male and female breeding barn owls ringed as juveniles or adults.

Number of baseline samples	2004	2005	2006	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	**Total**
Males (78 ringed as juveniles, 59 ringed as adults)	22	45	9	3	5	4	26	3	2	14	16	20	7	**176**
Females (145 ringed as juveniles, 144 ringed as adults)	34	32	37	13	25	26	94	14	34	68	116	57	15	**565**
Total	56	77	46	16	31	29	120	17	36	82	132	77	22	**741**
Number of stress-induced samples														
Males (82 ringed as juveniles, 54 ringed as adults)			10	9	5	7	40	8	15	24	27	21	8	**174**
Females (134 ringed as juveniles, 137 ringed as adults)			30	14	25	29	129	13	42	82	135	61	19	**579**
Total			40	23	30	36	169	21	57	106	162	82	27	**753**

Table 3. Parameter estimates (with 95 % Bayesian CrI) of the models for baseline and stress-induced corticosterone levels assessed between 2004 and 2018 in breeding barn owls. The data used for the baseline model included 741 samples (mean CORT ± SD: 11.37 ± 9.46) taken from 426 individuals measured in 13 different years, whereas the data used for the stress-induced corticosterone model included 753 samples (mean CORT ± SD: 63.27 ± 25.20) taken from 407 individuals measured in 11 different years (cf. Table 2). The random effects are given as among year, among brood, or among individual standard deviation. We also provide the % of variance explained by the random effects. Baseline corticosterone levels were log-transformed to approach normality, while the stress-induced corticosterone values remained untransformed.

Model coefficients (SD base; SD stress)	**Baseline model** estimate (95 % CrI)	**Stress-induced model** estimate (95 % CrI)
Intercept male incubating	-0.20 (-0.35 – -0.05)	-0.13 (-0.35 – 0.10)
Intercept female incubating	-0.12 (-0.26 – 0.02)	-0.12 (-0.29 – 0.05)
Intercept male nursing	0.01 (-0.16 – 0.17)	0.14 (-0.11 – 0.39)
Intercept female nursing	0.08 (-0.05 – 0.22)	0.14 (-0.03 – 0.33)
Date of sampling† (39.5 d; 38.3 d)	-0.35 (-0.43 – -0.27)	-0.27 (-0.34 – -0.20)
Hour of sampling† (4.1 h; 3.9 h)	0.03 (-0.04 – 0.10)	0.05 (-0.02 – 0.15)
Sampling latency†* (31.5 s; 104.6 s)	0.10 (0.03 – 0.18)	0.03 (-0.05 – 0.12)
Age† (1.7 yr; 1.6 yr)	0.20 (0.13 – 0.27)	-0.06 (-0.13 – 0.01)
Body mass† (51.35 g; 51.0 g)	-0.12 (-0.23 – 0.02)	-0.20 (-0.30 – -0.10)
Brood size† (1.9; 2)	0.09 (0.02 – 0.15)	0.04 (-0.02 – 0.10)
Random effects (variance components)	estimate (95 % CrI); explained variance	estimate (95 % CrI); explained variance
Year (among year SD)	0.24 (0.17 – 0.32); 28%	0.27 (0.19 – 0.36); 39%
Brood ID (among brood SD)	0.17 (0.16 – 0.18); 4%	0.11 (0.10 – 0.12); 1%
Individual ID (among individual SD)	0.12 (0.11 – 0.13); 1%	0.20 (0.18 – 0.21); 7%
Residual (residual SD)	0.36 (0.34 – 0.38); 67%	0.34 (0.32 – 0.35); 53%

† *Estimates based on standardised data (centred and scaled to 2 SD)*

* *Latency between time (s) of capture and blood sampling for baseline and stress-induced models*

Table 4. Parameter estimates of the multistate survival model for the log-odds of survival, recapture, and recovery probability. Presented are the posterior means with Bayesian credible interval (95 % CrI) for all model parameters, as well as the selection coefficients for corticosterone following Janzen and Stern (1998). Sample size is 426 individuals for the model with baseline corticosterone as predictor, and 407 individuals for the model with stress-induced corticosterone as predictor. Both data sets span 15 years of capture-recapture data of adults (between 2004 and 2018; cf. Fig. 1). Baseline corticosterone levels were log-transformed to approach normality, while for stress-induced model corticosterone values remained untransformed.

	Baseline model		**Stress-induced model**	
Survival probability	estimate (95% CrI)	selection coefficient (95% CrI)	estimate (95% CrI)	selection coefficient (95% CrI)
Intercept male	0.42 (0.16 – 0.68)		0.46 (0.16 – 0.77)	
Intercept female	0.06 (-0.14 – 0.26)		0.19 (-0.03 – 0.41)	
Corticosterone levels† – male	-0.12 (-1.25 – 1.01)	-0.03 (-0.29 – 0.24)	0.76 (-0.24 – 1.78)	0.18 (-0.06 – 0.41)
Corticosterone levels† – female	0.29 (-0.54 – 1.11)	0.07 (-0.13 – 0.26)	1.19 (0.44 – 1.93)	**0.23 (0.11 – 0.45)**
Age†	0.98 (-0.14 – 2.11)		1.49 (0.55 – 2.47)	
Age^{2}†	-2.35 (-4.72 – -0.03)		-1.3 (-2.75 – 0.15)	
Recapture probability				
Intercept	1.97 (1.66 – 2.29)		2.12 (1.79 – 2.48)	
Number of nest boxes	-0.06 (-0.74 – 0.59)		-0.09 (-1.04 – 0.81)	
Recovery probability				
Recovery estimate	-2.47 (-2.85 – -2.11)		-2.85 (-3.31 – -2.42)	

† *Estimates based on standardised data (centred and scaled to 2 SD)*

Table 5. Parameter estimates of the models for annual (A) and lifetime (B) reproductive success. Parameters are given as means of the posterior distribution with 95 % Bayesian credible intervals (95 % CrI). The linear mixed model with baseline corticosterone as predictor was fitted to data from 349 individuals (mean number of offspring during lifetime ± SD: 10.35 ± 9.16). The model with stress-induced corticosterone as predictor was fitted to data from 340 individuals (mean number of offspring during their lifespan ± SD: 9.08 ± 8.92). The random effect "cohort" is given as among-cohort standard deviation and the % of variance explained. The selection coefficients were calculated following Lande and Arnold (1983). Baseline corticosterone levels were log-transformed to approach normality, while the stress-induced corticosterone values remained untransformed.

A. Annual reproductive success	**Baseline model**		**Stress-induced model**	
Model coefficients (SD base; SD stress)	estimate (95% CrI)	selection coefficient (95% CrI)	estimate (95% CrI)	selection coefficient (95% CrI)
Intercept male	3.21 (2.89 – 3.53)		3.02 (2.69 – 3.36)	
Intercept female	3.00 (2.74 –3.27)		2.72 (2.43 – 3.01)	
Lifespan† (1.63 yr; 1.52 yr)	1.00 (0.72 – 1.30)		0.97 (0.68 – 1.27)	
Mean corticosterone male† (9.15 ng/ml; 22.68 ng/ml)	0.03 (-0.37 – 0.41)	0.01 (-0.18 – 0.20)	-0.02 (-0.45 – 0.41)	-0.01 (-0.22 – 0.20)
Mean corticosterone female† (6.91 ng/ml; 19.54 ng/ml)	1.02 (0.58 – 1.48)	**0.51 (0.29 – 0.74)**	0.95 (0.39 – 1.50)	**0.47 (0.20 – 0.75)**
Random effects (variance components)	estimate (95 % CrI)	explained variance	estimate (95 % CrI)	explained variance
Cohort (among cohort SD)	0.46 (0.34 – 0.58)	9%	0.38 (0.27 – 0.51)	5%
Residual (residual SD)	1.15 (1.08 – 1.24)	91%	1.23 (1.14 – 1.33)	95%

B. Lifetime reproductive success	**Baseline model**		**Stress-induced model**	
Model coefficients	estimate (95% CrI)	selection coefficient (95% CrI)	estimate (95% CrI)	selection coefficient (95% CrI)
Intercept male	3.51 (3.08 – 3.92)		3.31 (2.91 – 3.70)	
Intercept female	3.22 (2.83 – 3.60)		2.95 (2.59 – 3.30)	
Mean corticosterone male† (9.15 ng/ml; 22.68 ng/ml)	0.09 (-0.32 – 0.49)	0.05 (-0.15 – 0.24)	-0.23 (-0.68 – 0.23)	-0.12 (-0.34 – 0.11)
Mean corticosterone female† (6.91 ng/ml; 19.54 ng/ml)	0.89 (0.52 – 1.23)	**0.44 (0.26 – 0.62)**	0.49 (0.10 – 0.88)	**0.25 (0.05 – 0.44)**
Random effects (variance components)	estimate (95% CrI)	explained variance	estimate (95% CrI)	explained variance
Cohort (among cohort SD)	0.71 (0.55 – 0.89)	21%	0.53 (0.39 – 0.70)	10%
Residual (residual SD)	1.21 (1.12 – 1.30)	79%	1.28 (1.19 – 1.39)	90%

† *Estimates based on standardised data (centred and scaled to 2 SD)*

Table 6. Parameter estimates for the models of female clutch size. Parameters were estimated with 95 % Bayesian credible intervals (95 % CrI) from separate linear mixed models fitted to data for 409 baseline (mean clutch size ± SD: 6.54 ± 1.5) and 460 stress-induced samples (mean clutch size ± SD: 6.62 ± 1.54) measured in 309 and 327 individuals, respectively. The random effects are given as among year and among individual standard deviation, respectively, plus the % of variance explained. The selection coefficients were calculated following Lande and Arnold (1983).

	Baseline model		**Stress-induced model**	
Model coefficients (SD base; SD stress)	estimate (95% CrI)	selection coefficient (95% CrI)	estimate (95% CrI)	selection coefficient (95% CrI)
Intercept male	6.52 (5.75 – 7.30)		7.29 (6.57 – 8.02)	
Intercept female	6.37 (5.82 – 6.93)		6.78 (6.27 – 7.03)	
Laying date† (30.78 d; 32.46 d)	0.65 (0.35 – 0.95)		0.62 (0.33 – 0.91)	
Corticosterone male† (10.04 ng/ml; 21.94 ng/ml)	0.54 (-0.50 – 1.59)	0.27 (-0.25 – 0.80)	-0.85 (-2.00 – 0.33)	-0.43 (-1.01 – 0.15)
Corticosterone female† (7.8 ng/ml; 22.67 ng/ml)	0.36 (-0.14 – 0.86)	0.18 (-0.07 – 0.43)	-0.53 (-1.11 – 0.05)	-0.27 (-0.55 – 0.03)
Random effects (variance components)	estimate (95% CrI)	explained variance	estimate (95% CrI)	explained variance
Year (among year SD)	0.91 (0.64 – 1.23)	27%	0.80 (0.54 – 1.13)	19%
Individual ID (among individual SD)	0.41 (0.38 – 0.46)	1%	0.25 (0.23 – 0.28)	0%
Residual (residual SD)	1.30 (1.21 – 1.39)	62%	1.38 (1.30 – 1.49)	81%

† *Estimates based on standardised data (centred and scaled to 2 SD)*

Figures

Fig. 1. Schematic representation of the survival modelling process. The 741 baseline and 753 stress-induced corticosterone (CORT) samples were separately analysed with a linear mixed model to estimate the parameters that best predict baseline and stress-induced CORT levels of individuals. The individualised estimates of these two models were then used to model survival via mark-recapture models.

Data

Capture history matrix*

ID	2004	2005	2006	2007	...
1	–	1	1	0	1
2	1	0	1	0	1
3	–	1	2	0	0
4	–	1	1	0	0
...					

* 0 = not seen, 1 = recaptured alive, 2 = recovered dead

CORT history matrix†

ID	2004	2005	2006	2007	...
1	–	5.3	6.5	x	6.0
2	10.5	x	11.1	x	7.4
3	–	2.3/5.0	–	–	–
4	–	15.7	13.5/12.0	x	–
...					

† x = missing CORT measurement, note that some individuals were sampled twice or more within the same year.

Linear mixed model

dependent variables: baseline or stress-induced CORT levels
fixed effects: sex, age, body mass, sampling date, sampling time, sampling latency (i.e. time between capture and blood sampling), brood size, and breeding stage
random effects: individual identity, brood identity, year

n for baseline CORT model: 741 samples of 426 individuals
n for stress-induced CORT model: 753 samples of 407 individuals

Extracted from linear model
Estimated CORT values for each individual given its sex, brood identity, and year. An average value for age, body mass, brood size, breeding stage, sampling date, sampling hour, and sampling latency was used to estimate CORT.

Survival model

State matrix††

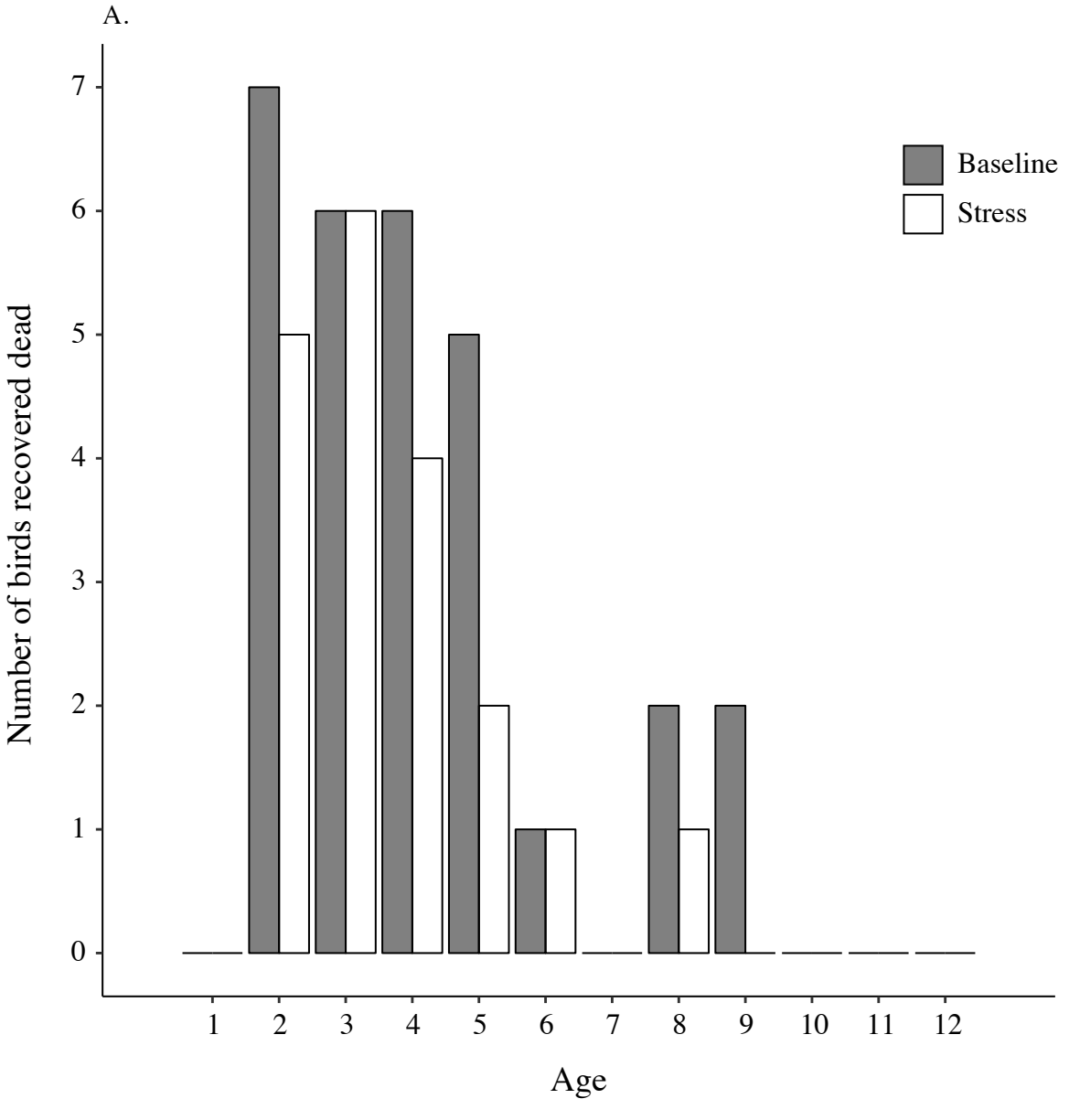
ID	2004	2005	2006	2007	...
1	0	1	1	1	1
2	1	1	1	1	1
3	0	1	2	3	3
4	0	1	1	1	2
...					

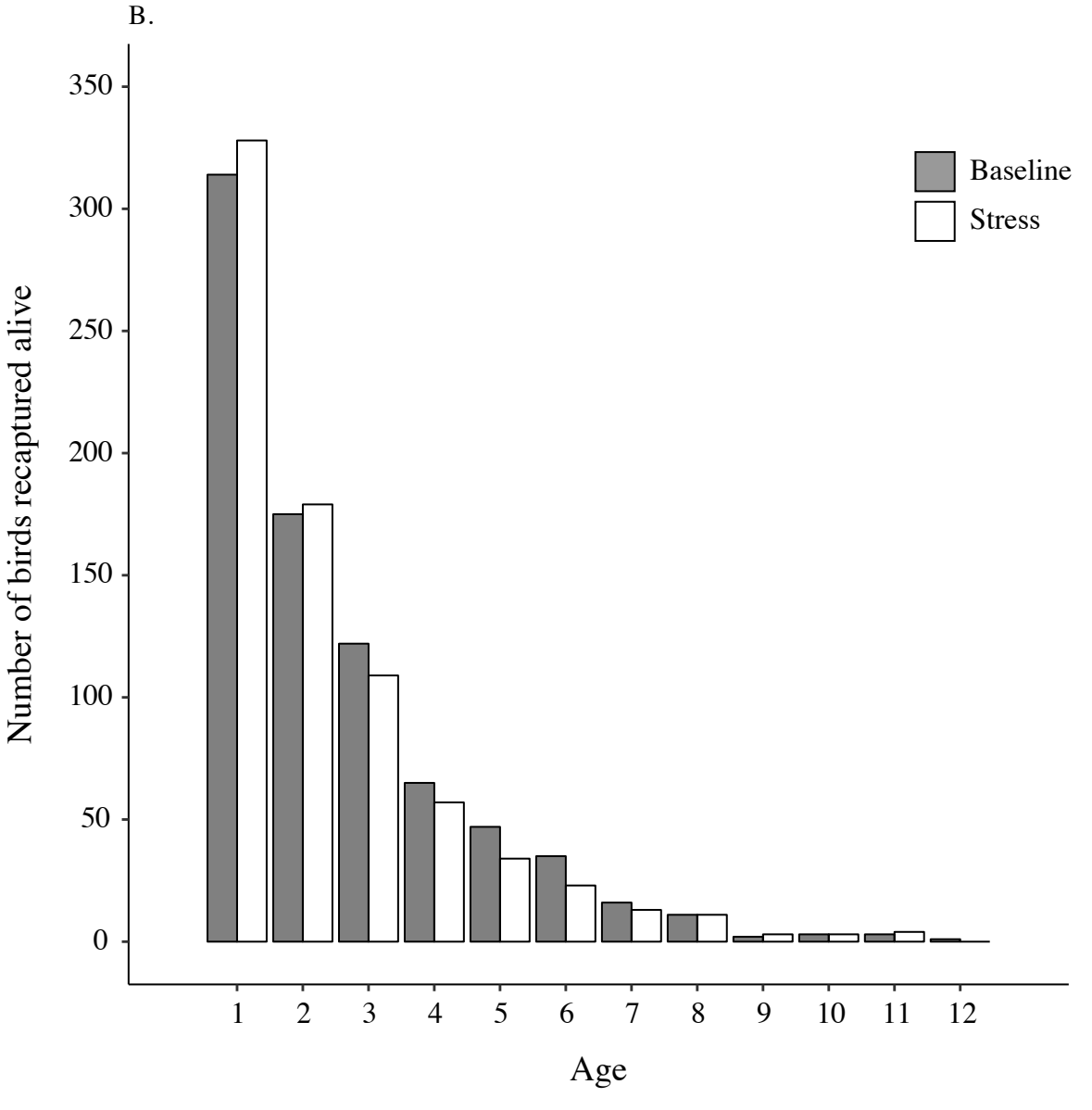
†† 1 = alive, 2 = freshly dead, 3 = dead

Comparable CORT values

ID	2004	2005	2006	2007	...
1	–	5.4	6.7	6.1	6.5
2	10.3	10.8	12.0	9.4	7.9
3	–	1.8	5.0	–	–
4	–	15.5	12.7	13.5	–
...					

Fig. 2. Number of (A) birds recovered dead, (B) birds recaptured alive, and (C) corticosterone samples in relation to age (in years). The dark and light grey bars represent the baseline and stress-induced CORT survival models, respectively.


A.
Number of birds recovered dead
0
1
2
3
4
5
6
7
Baseline
Stress
1
2
3
4
5
6
7
8
9
10
11
12
Age


B.
Number of birds recaptured alive
0
50
100
150
200
250
300
350
1
2
3
4
5
6
7
8
9
10
11
12
Age
Baseline
Stress

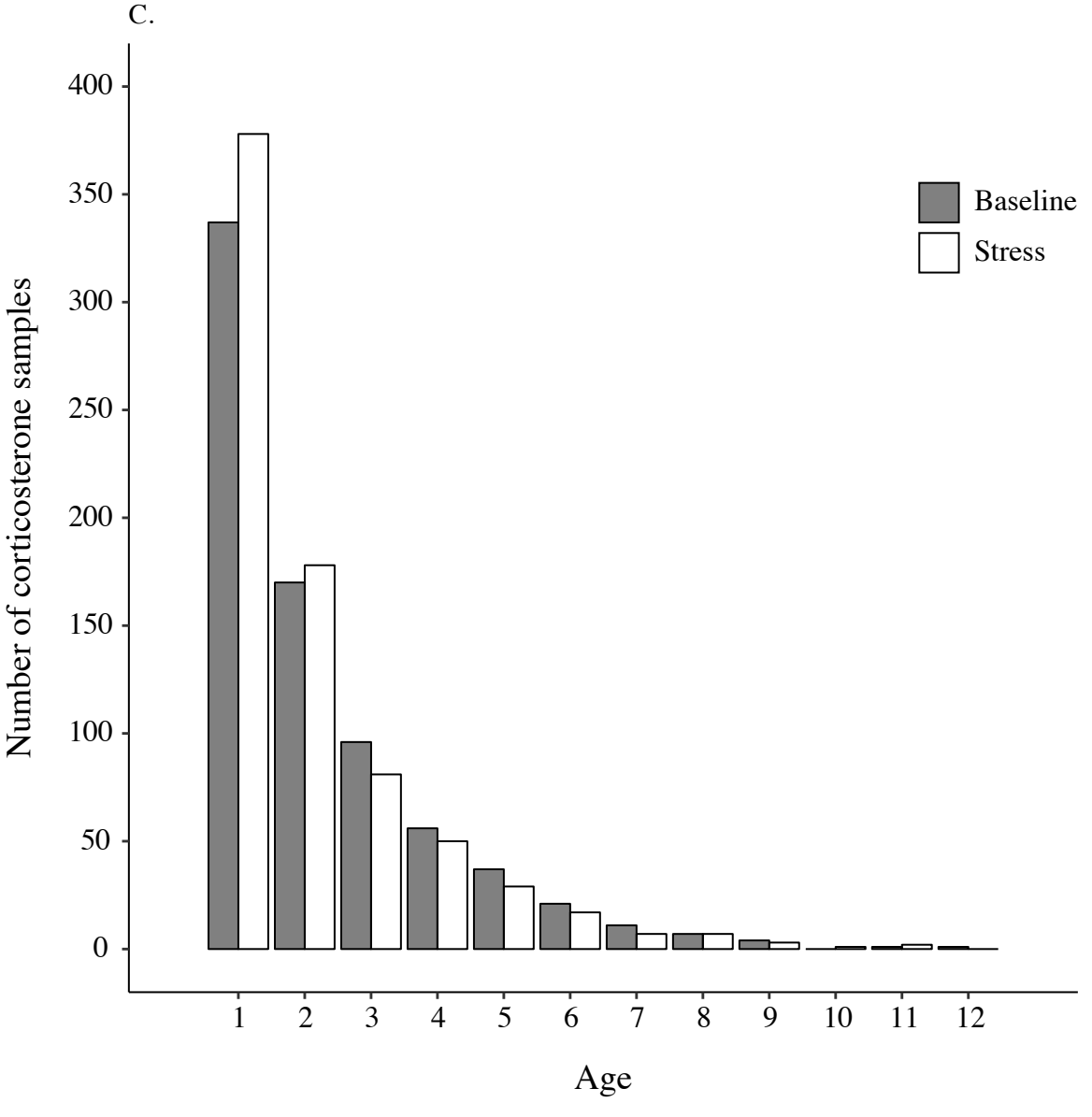

C.
Number of corticosterone samples
400
350
300
250
200
150
100
50
0
Baseline
Stress
1
2
3
4
5
6
7
8
9
10
11
12
Age

Fig. 3. Mean baseline and stress-induced corticosterone levels with 95 % Bayesian credible intervals for females (filled symbols) and males (open symbols) during incubation (circle) and nursing (square) (posterior distribution estimates).

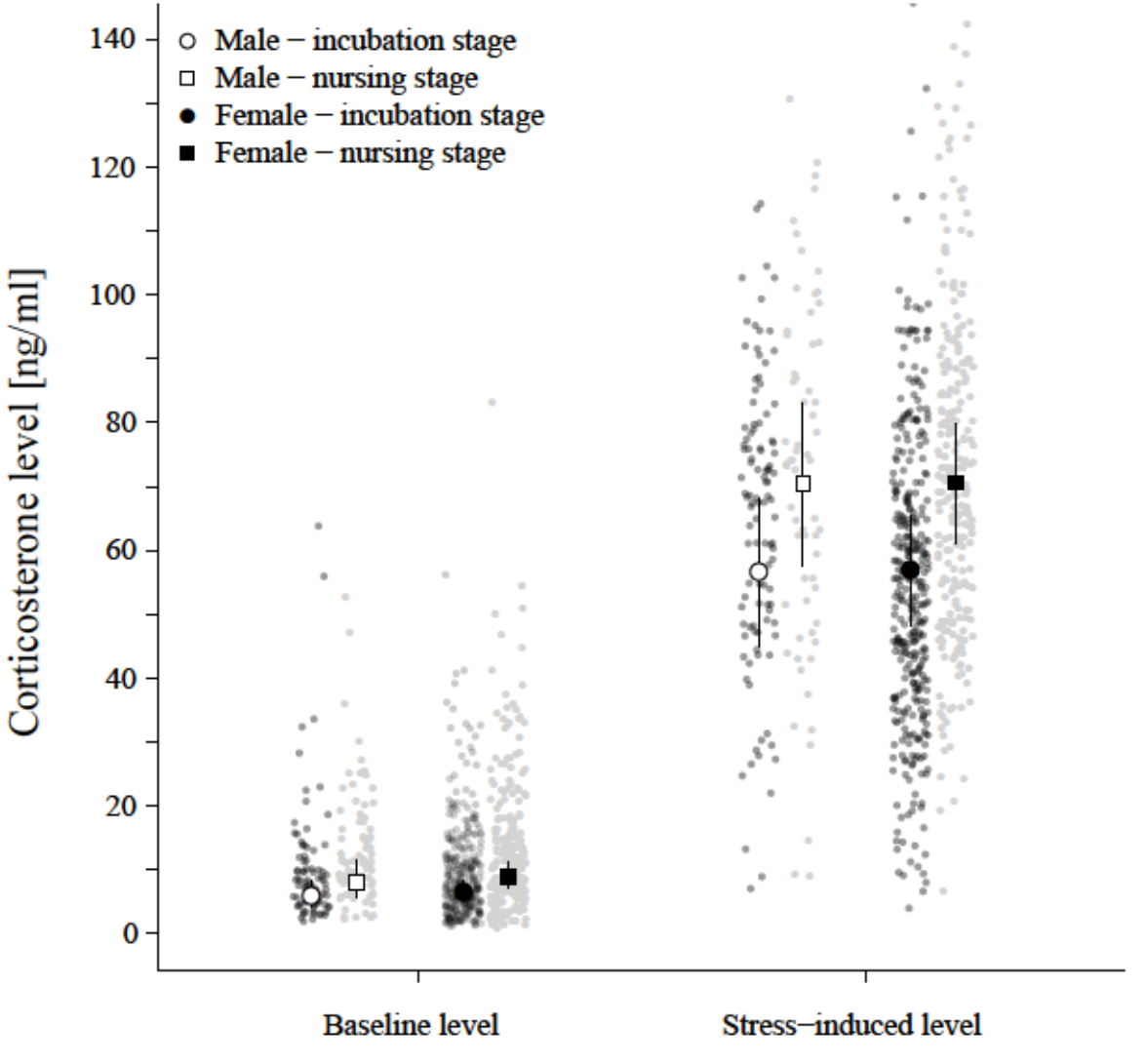

Male – incubation stage
Male – nursing stage
Female – incubation stage
Female – nursing stage
Corticosterone level [ng/ml]
0
20
40
60
80
100
120
140
Baseline level
Stress–induced level

Fig. 4. Estimated survival probability of breeding barn owls in relation to their (left) baseline and (right) stress-induced corticosterone levels. The solid (male) and dashed lines (female) represent the mean of the posterior distribution, with shaded 95 % CrI regions. The tick marks displayed along the x-axes represent the individual data.

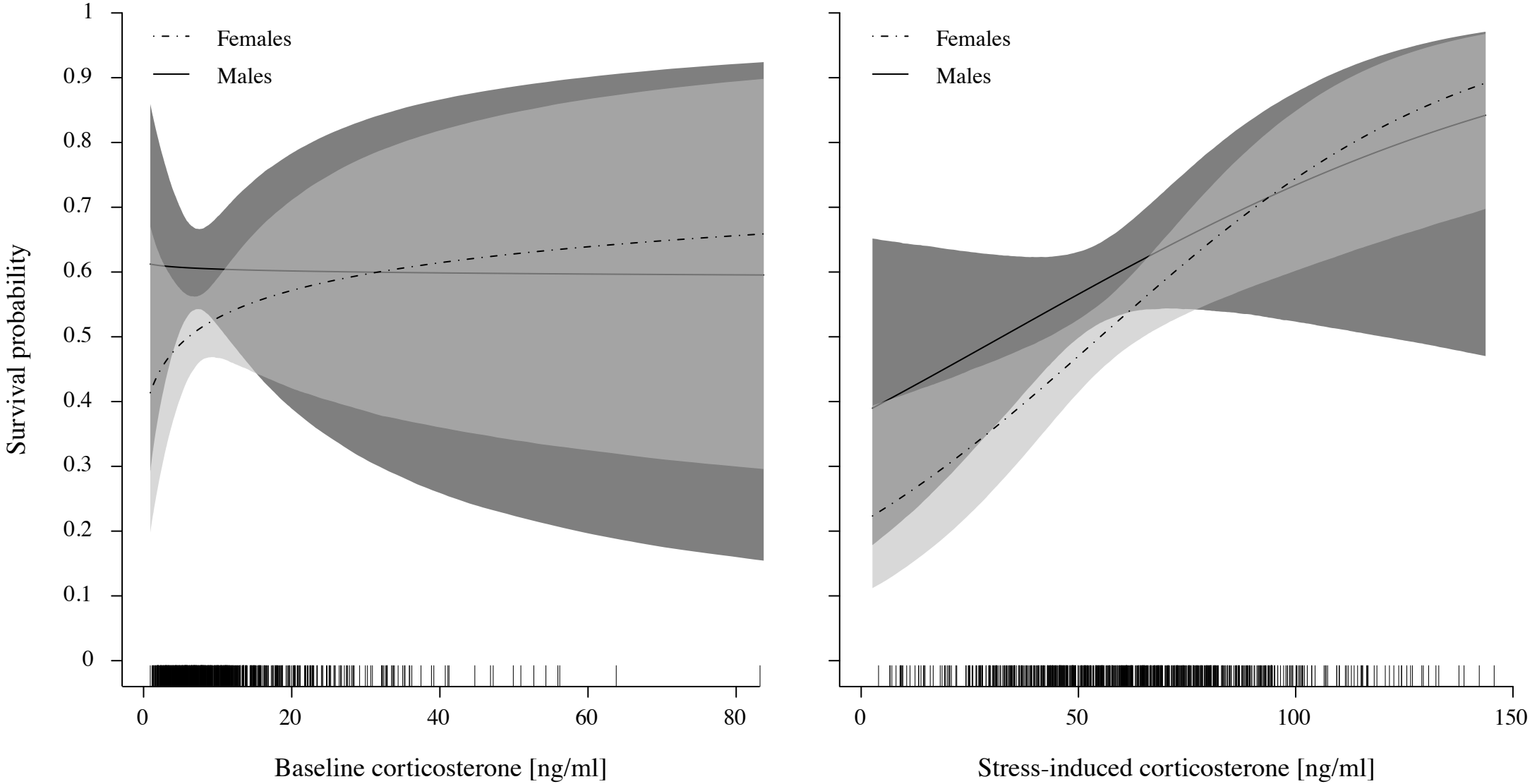
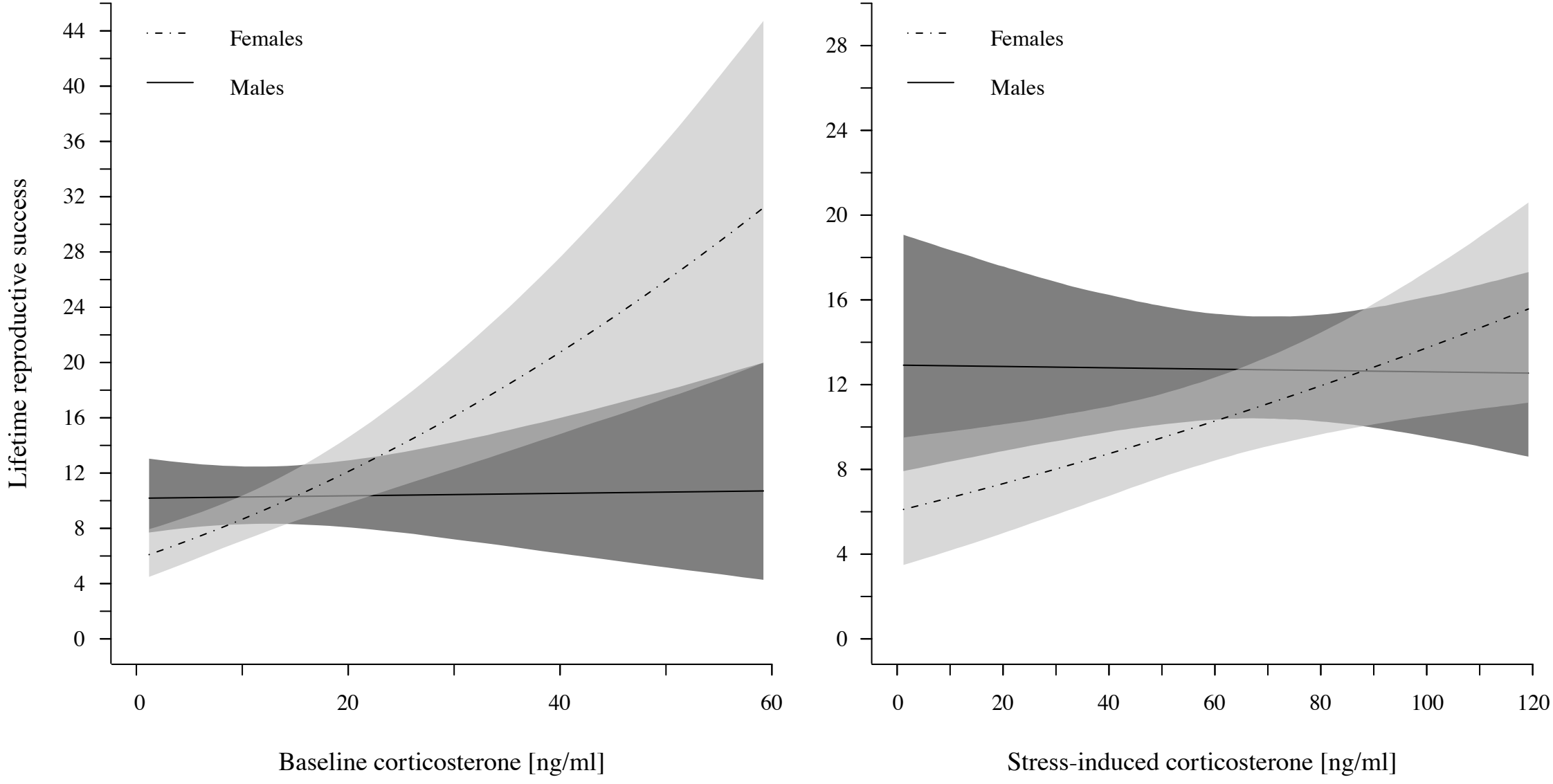

Females
Males
Survival probability
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
20
40
60
80
Baseline corticosterone [ng/ml]
Females
Males
0
50
100
150
Stress-induced corticosterone [ng/ml]

Fig. 5. Estimated lifetime reproductive success of adult barn owls in relation to their (left) baseline and (right) stress-induced corticosterone levels. The solid (male) and dashed lines (female) represent the mean of the posterior distribution, with shaded 95 % CrI regions.

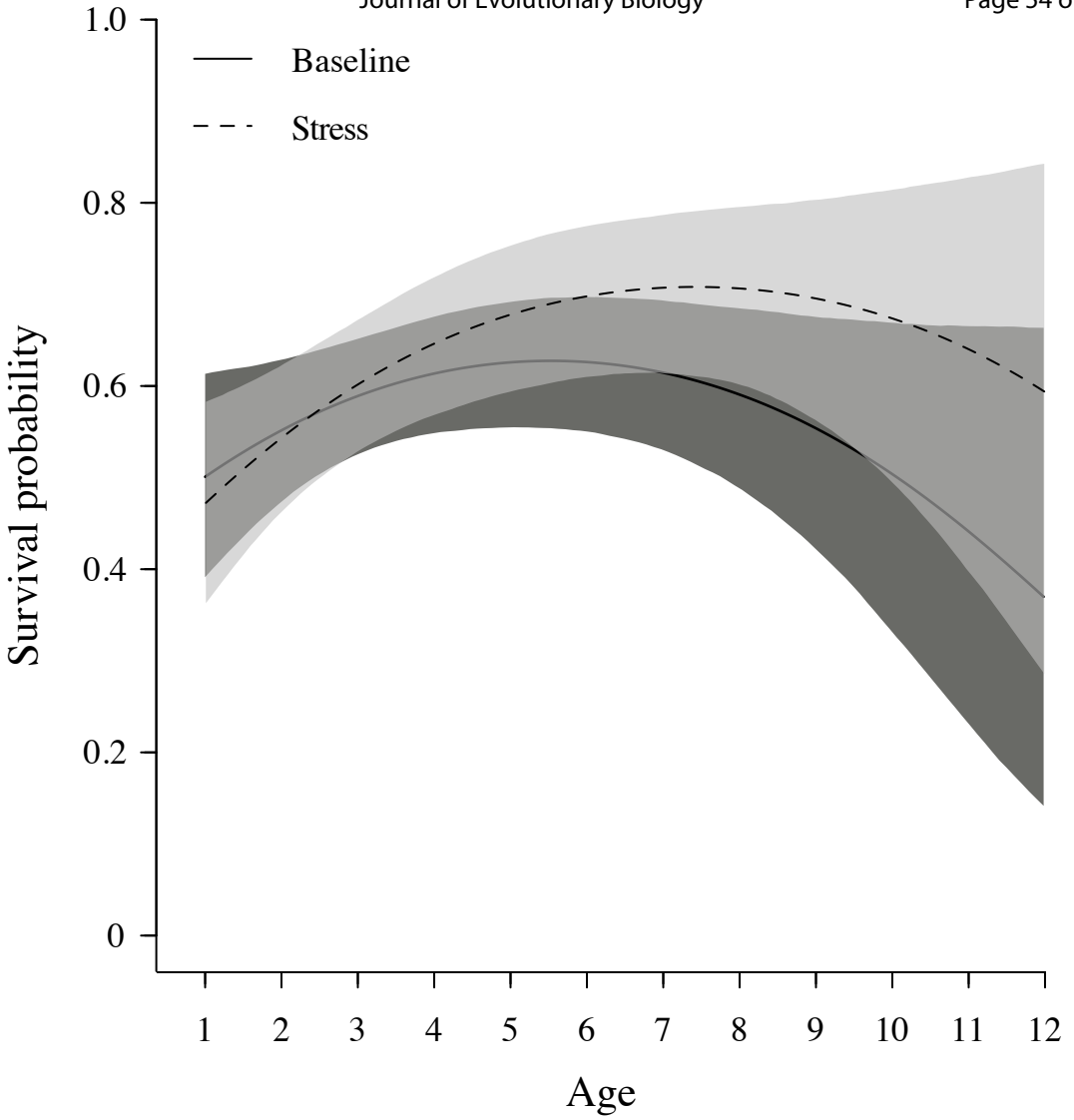

Females
Males
Lifetime reproductive success
0
4
8
12
16
20
24
28
32
36
40
44
0
20
40
60
Baseline corticosterone [ng/ml]
Females
Males
0
4
8
12
16
20
24
28
0
20
40
60
80
100
120
Stress-induced corticosterone [ng/ml]

Supplementary material

Table S1. Results of posterior predictive model checking: 1,000 data sets using the parameters from our models (Tables 3 & 4) were simulated and compared to our observed data.

Models	Test statistics	Observed data	Replicated data (1–99 % quantiles)
Baseline CORT model	live recapture	794	717 – 872
	dead recovery	29	15 – 46
	number of times an individual gets captured		
	1	219	194 – 256
	2	112	87 – 133
	3	59	34 – 67
	4	22	11 – 32
	5	4	3 – 17
	6	6	1 – 11
	7	3	0 – 8
	8	0	0 – 4
	9	1	0 – 3
	10	0	0 – 2
	11	0	0 – 1
	12	0	0 – 1
	13	0	0 – 1
	14	0	0 – 0
	15	0	0 – 0
Stress-induced CORT model	live recapture	763	672 – 822
	dead recovery	19	7 – 34
	number of times an individual gets captured		
	1	212	191 – 250
	2	105	82 – 129
	3	56	31 – 64
	4	18	9 – 28
	5	4	2 – 15
	6	6	0 – 10
	7	4	0 – 7
	8	1	0 – 4
	9	1	0 – 3
	10	0	0 – 2
	11	0	0 – 1
	12	0	0 – 1
	13	0	0 – 1
	14	0	0 – 0
	15	0	0 – 0

Fig. S1. Estimated survival of adult barn owls in relation to their age (in years) for the baseline (solid line, dark grey) and stress-induced (dashed line, light grey) CORT survival models with shaded 95 % CrI regions.


Baseline
Stress
Survival probability
1.0
0.8
0.6
0.4
0.2
0
1
2
3
4
5
6
7
8
9
10
11
12
Age

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