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Inbreeding depression in morphological and growth traits of wild barn owls (*Tyto alba*)

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Despite its detrimental effects, inbreeding depression in wild populations is relatively under-explored. Here, we use whole-genome sequence data from more than 2700 wild barn owls (*Tyto alba*) to investigate the presence, severity and genetic architecture of inbreeding depression in three morphological traits. Using linear models (accounting for age) and nonlinear models (measuring growth effects) we show evidence for inbreeding depression. Moreover, by partitioning developmental stages we improve our ability to detect it, as in some traits we find that it manifests during juvenile growth, and in others during adulthood. These trait-specific patterns may reflect changes in environmental influences, as we also find that juvenile heritability is lower. We additionally use two genomic inbreeding coefficients: F_{ROH} and F_{UniW} , and while both showed the same direction of effect, the strength of evidence for inbreeding depression differs. This discrepancy offers some insight into the frequency distribution of responsible variants, as the coefficients weight variants differently based on their frequencies. Finally, an assessment of local genomic inbreeding effects highlights a handful of genomic regions with significantly deleterious effects when homozygous, alongside many regions with a smaller contribution. Overall, we provide a detailed examination of inbreeding depression affecting morphological and growth traits in this wild population.

1. Introduction

The decreased fitness in the offspring of related individuals—known as inbreeding depression—is crucial in fields from human genetics to conservation biology. In this context, fitness refers to an individual's ability to survive and reproduce, but it can be expressed in a number of related traits. In humans, inbreeding has been linked to numerous diseases [1], whereas in endangered populations, inbreeding depression limits population recovery and increases the risk of extinction [2,3]. However, despite its severity, the prevalence of inbreeding depression and its mechanisms are still poorly understood in wild populations.

Detecting inbreeding depression in wild populations presents unique challenges. Unless the strength of inbreeding depression is exceptionally high, such studies may require a large sample size [4,5]; and/or substantial variation in inbreeding coefficients between individuals to accurately detect inbreeding

depression [6]. Moreover, merely obtaining individual inbreeding coefficients and fitness data required for studies of this nature can be difficult in natural populations. A major advance in the field was the use of genomic inbreeding coefficients, rather than those estimated from a population pedigree. Often, genetic data is easier to acquire than an accurate pedigree in a wild population, and will provide estimates of realized inbreeding rather than predicted inbreeding. Hence, numerous genomic estimators of inbreeding coefficients have been established in recent years (reviewed in Alemu *et al.* [7] and Zhang *et al.* [8]). These estimators are broadly either based on stretches of homozygosity assumed to be descendent from the same common ancestor (identical-by-descent) and range between 0 and 1, e.g. F_{ROH} , or average homozygosity across the genome, treating each variant as unlinked which can range between -1 and 1, e.g. F_{GRM} , F_{Uni} and F_{AS} . The choice of the most appropriate genomic inbreeding coefficient to study inbreeding depression has been widely debated [4,7,9], and is dependent on the demographic history of the focal population or the type of genotyping employed. Despite this, various genomic inbreeding coefficients have since been used to establish a reliable association between inbreeding and a number of fitness-related traits in various wild species, such as breeding success [10] and survival [3,11], highlighting their practical value in real-world conservation research. In addition, the acquisition of high-quality genomic data in recent years has renewed the interest in identifying loci responsible for observed inbreeding depression; for example, the contribution of (slightly) deleterious recessive loci versus over-dominant loci, along with their effect size and distribution across the genome, which still remains largely unknown in wild populations [12,13].

While life-history traits such as survival and fecundity are generally expected to exhibit stronger signs of inbreeding depression [14], they can be difficult to directly measure in a natural population, often requiring individuals to be monitored throughout their lives. Conversely, morphological traits are easily quantifiable and can be a good indication of inbreeding depression in wild populations [10,15]. In particular, since early weight can be a key factor in determining juvenile survival [16], reduced growth rates are likely to be highly disadvantageous, particularly in a natural environment [17]. The effect of inbreeding depression on growth rates has been relatively well explored in farmed species—where the daily growth of an individual represents significant economic gain (e.g. [18,19])—but few studies on growth rates in wild animals exist in the literature. A study on meerkats (*Suricata suricatta*) showed evidence for inbreeding depression in pup mass at emergence from the natal burrow [17], and in Soay sheep (*Ovis aries*) inbreeding depression was detected in four month weight [15], both of which are good indicators of early life growth rates but not direct measures. For farmed species many studies measure individual growth at multiple time points for uniformity, such as eight week weight, meaning the growth rate can be easily calculated. However, in wild populations the exact age is often unknown prior to measurement. Instead, studies on wild populations commonly obtain one morphological phenotypic measure per individual or, when multiple records exist, only one is taken during early juvenile growth stages. This highlights the need for further research to determine how widespread the effects of inbreeding depression on growth rates may be.

Determining the genetic and environmental sources of variation that contribute to a trait may further help our understanding of inbreeding depression in a population. When present, inbreeding depression contributes to the overall phenotypic variance (V_P) of a trait, reflecting a component of non-additive genetic variance, specifically dominance genetic variance (V_D). In addition, high environmental influences could manifest as an inbreeding depression-by-environment interaction, where inbreeding depression is more severe in harsher environmental conditions [20,21]. During the course of an individual's life these phenotypic influences can fluctuate [22], which may then also affect the strength of inbreeding depression.

A population of barn owls (*Tyto alba*) residing in southwest Switzerland has been the subject of an individual based long-term study for over 30 years [23], and presents an ideal opportunity to investigate inbreeding depression in a wild population. Barn owls are wide-spread across Europe, but intense farming and homogenized landscapes has led to a population decline from which they may still be recovering [24]. Here, we calculate genomic inbreeding coefficients and quantify inbreeding depression in three recorded morphological traits for this population. We also take advantage of regular phenotypic measurements covering a range of ages, to investigate inbreeding depression in growth rates. We then disentangle the genetic architecture of inbreeding depression by associating local homozygosity-by-descent with these traits. Finally, we explore other recorded sources of variation in the traits and how these change over the development of an individual.

2. Methods

(a) Study population, trait measurements and genetic sampling

Individual barn owls (*T. alba*) born within an area of southwest Switzerland have been monitored as part of a long-term study since approximately 1986 (figure 1; [23,24]). Currently there are 400 nest boxes at 316 sites, which are visited regularly during the breeding season between March and October. When a clutch is found, the developmental stage of each egg is estimated. As hatching is asynchronous over multiple days, the first brood visit occurs when the oldest chick is predicted to be 30 days old, based on a 32 day egg incubation time, reducing the impact of visits on young growing chicks. Nestlings are then ringed with an individual identifier, have a blood sample taken, and are measured, including, but not limited to: bill length (mm), tarsus length (mm) and body mass (g)—the three focal traits of this study. The age of each nestling is estimated based on their wing length at this first visit [25]. Between 2015 and 2024 instead of a visit at 30 days, two chick measurements were scheduled at 25 and 35 days old. Final measurements are taken before fledging (approx. 55–60 days) when the oldest chick is 55 days. Measurements are also taken of the parents, and when a parent has not been ringed previously (i.e. born outside of the study area) they are ringed, measured and have a blood sample taken.

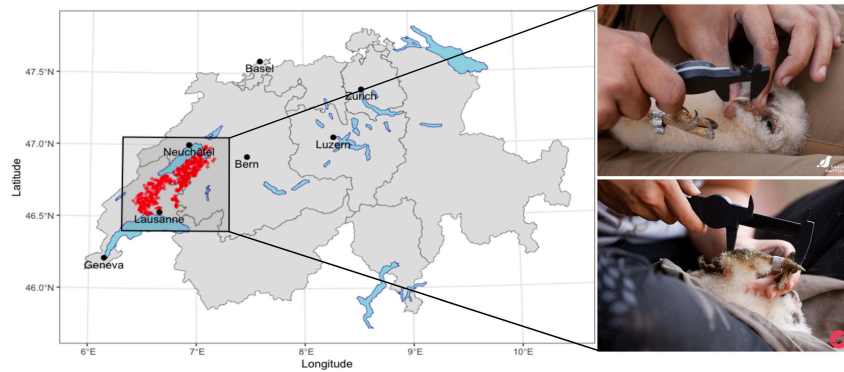


Figure 1. Barn owl (*Tyto alba*) research study area in Switzerland. Red dots show monitored nest-boxes as of the 2025 field season, with examples of individual measurements being taken for bill and tarsus length. Photo credit to Sandra Mattsson. All animal handling was performed in accordance with international animal welfare standards and the Swiss Animal Welfare Act, and under permission from the Veterinary Office of the Canton of VD, Fr, BE and the Federal Office for the Environment.

As part of an experiment, in some years partial and full cross fostering of individuals between nests occurred [26]. In order to account for this in subsequent analysis we created a new variable which combined the ID of the born clutch and the raised clutch. A total of 3102 individuals in the study population have been whole genome sequenced at a mix of high and low coverage. Five hundred and two individuals from the *T. alba* species range across the western Palearctic (including 346 individuals from the focal population) were sequenced at high-coverage (ave. 14x), phased using WHATSHAP v1.4 [27,28] and SHAPEIT v4.1.2 [29]; and used to build a reference panel. The remaining individuals were sequenced at low coverage with a mean genome coverage of 1.95x, a minimum of 0.2x and maximum of 4.15x. Their full genotypes were imputed with GLIMPSE v1.1.1 [30] using the previously established reference panel. See Topaloudis *et al.* [31] and Cumer *et al.* [32] for full details on imputation methods and accuracy. For estimation of genomic inbreeding coefficients, we used genomic markers distributed across 37 high-quality autosomal super scaffolds which showed strong synteny with chicken (*Gallus gallus*) chromosomes, as previously inferred in Topaloudis *et al.* [33]. Hereafter ‘chromosome’ will refer to the chromosome number used for chicken rather than the super scaffold number. After phasing and quality control, 10 074 749 high-quality called variants remained, covering approximately 85.5% of the 1.2 Gb genome. We used individuals sequenced at low coverage (<5x) for our analysis, to avoid any possible bias from sequencing depth (electronic supplementary material, figure S1). Following quality control with expected pedigree kinships, we calculated inbreeding coefficients for 2744 individuals.

(b) Estimating inbreeding coefficients

Because barn owls from this population are in an open landscape and can breed outside the study area, the pedigree for this populations is relatively shallow, and inbreeding coefficients estimated from pedigree relatedness will underestimate the true level of inbreeding. To more accurately capture individual inbreeding levels we used two widely used inbreeding coefficients from each category outlined in the introduction: F_{UniW} and F_{ROH} . The former uses the correlation between uniting parental gametes relative to the reference population, giving greater weight to loci with larger expected heterozygosity [8]. Used in combination with a genomic relatedness matrix (GRM), F_{UniW} has been shown to give the most accurate estimations of inbreeding depression in structured populations [5]. F_{ROH} on the other hand considers Single-Nucleotide Polymorphisms (SNPs) as non-independent by focusing on long consecutive identical-by-descent genomic regions, and has been suggested to be a better measure of homozygosity-by-descent (HBD) [34].

F_{UniW} , described in [8] was calculated as:

$$F_{\text{UniW}_j} = \frac{\sum_{l=1}^L (x_{jl}^2 - (1 + 2p_l)x_{jl} + 2p_l^2)}{\sum_{l=1}^L 2p_l(1 - p_l)},$$

where, $x_{jl} \in \{0, 1, 2\}$ is the minor allele count for individual j at locus l , p_l is the allele frequency of the minor allele at the same locus and L is the total number of loci.

We also estimated the runs of homozygosity (ROH) based inbreeding coefficient, F_{ROH} . ROHs of a given length were inferred through homozygous-by-descent (HBD) segments called using the RZooRoH package v.0.3.1 using the genetic positions of SNPs with 13 HBD classes and 1 non-HBD class with rates (R) of 2, 4, 8, 16, 32, 64, 128, 256, 502, 1024, 2048, 4096 and 8192 for the non-HBD class [35]. We then averaged the probability of belonging to a HBD segment in order to estimate inbreeding coefficients using a T -value of 1024, therefore only considering HBD segments coalescing less than 512 generations ago as identical-by-descent. R (v4.3.2 [36]); functions and R scripts to calculate F_{UniW} and F_{ROH} are available at: https://github.com/annamayh/Inbreeding_morph_growth_BarnOwl.

(c) Statistical models

We ran all following statistical models using the *brms* package v2.21.0 [37] in R STATISTICAL SOFTWARE v4.3.2 [36]. Convergence was verified with: trace plots, potential scale reduction factors on split chains (Rhat) and effective sample size measures. See the electronic supplementary material, tables S1–S3 for details on warm-up iterations, total number of iterations and thinning intervals. All model code, including priors, is currently available at: https://github.com/annamayh/Inbreeding_morph_growth_BarnOwl.

(i) Inbreeding depression in traits

To estimate the effects of inbreeding depression on three morphological traits we ran a standard repeated measures linear mixed model (LMM) with either bill length, body mass or tarsus length as the response variable. To account for age with a single predictor, we fitted age of the individual at the time of measurement as a fixed effect following a Gompertz growth curve: $\text{asym} \times e^{(-b \times (c)^{\text{age in days}})}$, where ‘asym’ is the asymptote, b is the initiation of the growth curve, c is the growth slope and ‘age in days’ refers to the number of days since the estimated hatch date when measurements were taken. Population-average estimates of these parameters were used to derive an age-dependent growth variable for each individual at a certain age, which was then included as a fixed effect to model nonlinear growth of the trait. The variable was also mean-centred so the intercept represents the expected trait value at the mean age, for interpretation and visualization purposes (see the electronic supplementary material, figures S2–S4 for parameter estimates and curve). Sex and rank (numeric order of individuals born within a clutch, from 1 to 8) of the individual were also fitted as fixed effects. Random effects were fitted for: observer ($n = 46$), birth year (as factor, $n = 24$), birth month of the individual (as numeric, $n = 7$), individual nestbox identity code ($n = 303$), individual clutch identity ($n = 699$) and individual to account for repeated measurements. Of the 2744 individuals with calculated inbreeding coefficients, 1996 (1995 for tarsus length) had all appropriate phenotypic records for the fixed and random effects included in all our models. For bill length, body mass and tarsus length respectively we had 5596, 9001 and 5731 records in total. We also included an allele-sharing GRM to account for population and family structure when estimating inbreeding depression [38], calculated using the R package *Hierfstat* [39]. Finally, we fitted a genomic inbreeding coefficient, either F_{UniW} or F_{ROH} as defined above, as a fixed effect to estimate the effect of inbreeding coefficient on the trait value (i.e. inbreeding depression).

(ii) Inbreeding depression in growth rates

Given the availability of multiple records for each individual over its lifespan, particularly during the juvenile growth period, we then further explored at what point in an individual's life inbreeding depression may be most severe. To determine the effect of inbreeding on growth we estimated the effect of inbreeding coefficients on a three parameter version of the Gompertz growth curve (commonly used to model biological and economic growth [40]), using a hierarchical nonlinear mixed model (NLMM) with the following structure:

$$\begin{aligned} y &\sim \text{asym} \times e^{(-b \times (c)^{\text{age in days}})}, \\ \text{asym} &\sim 1 + X + m + e, \\ b &\sim 1, \\ c &\sim 1 + X + m + e, \end{aligned}$$

where, in this case ‘age in days’ is the age of the individual, ‘asym’ represents the asymptote of the trait, b the initiation of the growth curve and c the growth slope. Here, X , m and e represent the fixed, random and residual effects on each parameter respectively. During refinement of the model, we also tested the double exponential Gompertz growth curve and a logistic growth curve. The former failed to converge and the latter was not a good representation of the data. For all three traits we fixed the intercept of b , assuming no fixed effect variable influenced the initiation of the growth. This was to aid model mixing and, as the age of an individual is an estimate (see §2a), effects on this specific parameter may be more confounded. In each trait, certain modifications were made to optimize convergence times: for bill length we exponentiated the c parameter to prevent estimates from being below zero (impossible for a growth parameter), and for tarsus length, two asymptotes were used to allow the growth curve to start further from the origin i.e. $\text{asym}1 + (\text{asym}2 \times e^{(-b \times (c)^{\text{age in days}})})$. For the ‘asym’ parameter, X (fixed effects) and m (random effects) were the same as in the LMM above, excluding the GRM to aid convergence as this had only a minor improvement on estimates of inbreeding depression in the LMM (electronic supplementary material, Models SM1–SM6). During model refinement, co-variables affecting the growth rate parameter (c) were simplified to allow for model convergence, whereby, variables were removed if they did not explain any variation in the trait. This led to the final model of c including inbreeding coefficient, rank and sex of the individual as fixed effects and individual ring identity as a random effect to account for repeated measurements. We additionally ran a supplementary model including clutch identity to model c in mass, as despite it having a minimal effect (electronic supplementary material, Models SM7–SM10), it accounts for parental feeding during this time.

(iii) Trait heritability at different developmental stages

To further disentangle other sources of genetic and environmental variation over different life stages, we firstly split records into juvenile stages (≤ 180 days old) and adult stages (> 180 days old) as most barn owls have fledged by approximately 90 days and can breed the following year more than six months later [41]. Then, we ran a repeated measures animal model with the following model structure:

$$y = X\beta + Z_{\text{juvenile}}u + Z_{i\text{juvenile}}m_i + Z_{\text{adult}}u + Z_{i\text{adult}}m_i + e_{\text{juvenile}} + e_{\text{adult}}$$

where for the i th individual, y is a vector of the trait measurements and X is an incidence matrix relating individual measures to the vector of fixed effects, β . Z_{juvenile} and $Z_{i\text{juvenile}}$ are incidence matrices relating individual juvenile measures to additive genetic and the other remaining random effects respectively. By contrast, Z_{adult} and $Z_{i\text{adult}}$ are incidence matrices relating individual adult measures to additive genetic and other remaining random effects. u is the relatedness matrix estimated from genomic data (GRM) as above, and m_i is a vector of additional random effects. Finally, e_{juvenile} and e_{adult} are vectors of residual effects in the juvenile and adult stages, respectively. We used the same fixed and random effects described in the LMM, with the exception that rank was treated as a random effect. R code was modified from Martin & Videler [42].

We calculated narrow sense heritability per stage as:

$$h^2 = V_A/V_P$$

and evolvability as:

$$I_A = V_A/\bar{z}^2,$$

where, V_A , V_P and $\bar{z}^2$ are the additive genetic variance, phenotypic variance, and trait mean squared respectively, for each growth stage (juvenile or adult). We removed the variance due to the observer in our estimates of V_P as these are biases created through sampling with little biological interpretation and may lead to an underestimation of narrow sense heritability [43], although they are still necessary to account for within the model.

(iv) Genetic architecture of inbreeding depression

In order to uncover the genomic regions underlying inbreeding depression, we firstly calculated local HBD probabilities for non-overlapping windows of 2500 SNPs (approx. 100 kbp on average naively assuming 1 SNP every 40 bp equally spaced). RZooRoH provides a convenient framework to calculate these metrics, quantifying the probability of belonging to an HBD segment for any given window size. Then, for each window we ran separate LMMs including both the local HBD probability and the genome-wide HBD probability (F_{HBD}) as fixed effects. We repeated this process using local F_{UniW} and genome-wide F_{UniW} as fixed effects. All other fixed and random effects were kept the same as the previous models with the exception of the GRM which was omitted to aid model convergence time as in the LMM it was shown to have a very minimal effect on estimates (see the electronic supplementary material, models SM1–SM6). Bayesian equivalent p -values for windows were calculated as the proportion of post-warm up draws that were of the opposite value to the mean estimated effect of inbreeding for the focal window [44]. To account for the 4010 independent tests genome-wide, we used a Bonferroni corrected threshold of 1.25×10^{-5} , where any window with a Bayesian p -value below this threshold was then treated as a significant window. We then searched for genes within significant windows of F_{ROH} only (see §3c for details) using the *T. alba* genome assembly (*T. alba* DEE v4.0) on NCBI [45]. For each trait, G-PROFILER [46] was used to analyse gene ontology (GO) enrichment terms of biological processes, molecular function and cellular components, with chicken selected as the organism and using a false discovery rate correction.

3. Results

(a) Evidence for inbreeding depression in morphological traits

Across the 1996 individuals used in our analysis we found moderate levels of inbreeding but minimal close inbreeding. The two inbreeding coefficients (F_{UniW} and F_{ROH}) were highly positively correlated ($r = 0.81$; electronic supplementary material, figure S6), and both showed a range of individual inbreeding levels (figure 2A).

For bill length, we showed consistent evidence for inbreeding depression in the LMM, regardless of whether F_{ROH} or F_{UniW} was used as the inbreeding coefficient (figure 2B; electronic supplementary material, tables S4 and S5). An individual with an inbreeding coefficient of 0.25 (i.e. the product of a father–daughter or full-sibling mating) is predicted to have a bill length approximately 3.1% and 5.2% shorter using F_{UniW} and F_{ROH} respectively in comparison with an outbred individual ($F = 0$; electronic supplementary material, tables S4 and S5). The NLMM with F_{UniW} further suggests that these differences are most pronounced after growth (electronic supplementary material, table S16). That is, inbred and outbred individuals do not differ during the hatchling/nestling period (0 to approx. 30 days) but their ‘final’ bill length is different. Moreover, the estimate is within the same magnitude as the estimate from the LMM—approximately 2.4% shorter (electronic supplementary material, table S16). However, this was not the case using F_{ROH} in the NLMM, where no parameters showed a significant effect of F_{ROH} , despite finding evidence for inbreeding depression with the LMM (figure 2F; electronic supplementary material, table S17).

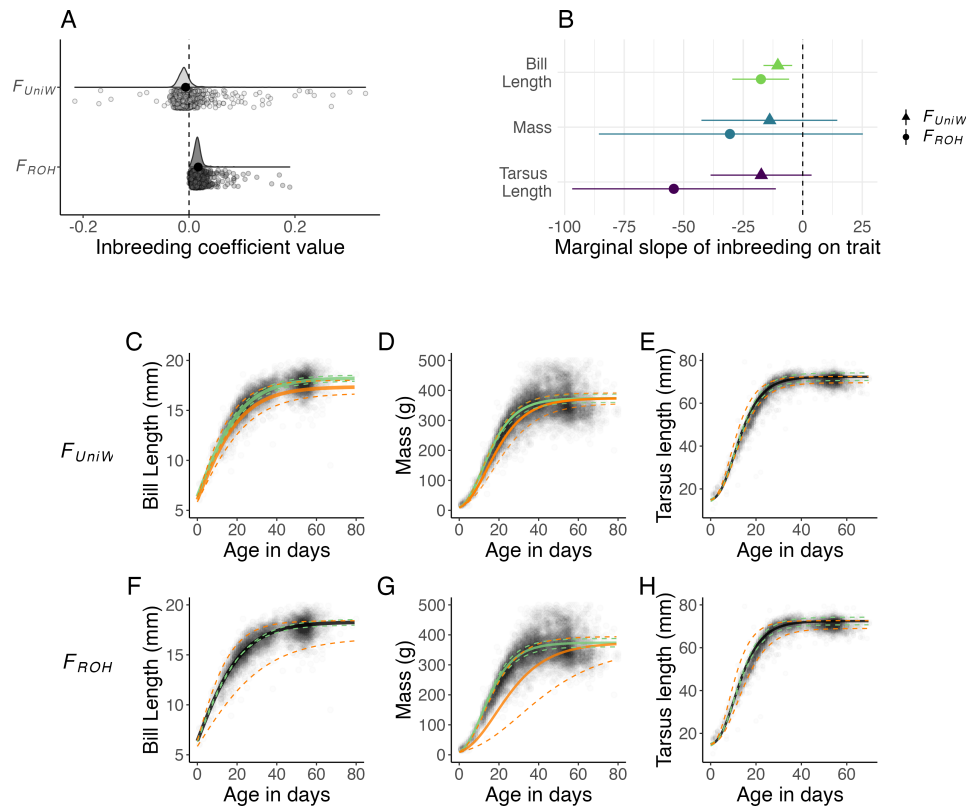


Figure 2. (A) Distribution of two inbreeding coefficients: F_{UnIW} and F_{ROH} in 1996 individuals used for analysis. (B) Inbreeding depression estimated from linear mixed models (LMM) in three morphological traits using F_{ROH} or F_{UnIW} . The mean posterior estimate is shown with 95% credible intervals. Estimates are relative to the scale of the trait and show the difference between an inbred ($F = 0.5$) and outbred individual ($F = 0$). All traits are plotted using the same x-axis but scales differ between traits e.g. mean bill length is 16.5 mm (scaled to three digits), mean mass is 323 g and mean tarsus length is 65.4 mm (scaled to three digits). (C–H) Inbreeding depression in growth rates estimated from nonlinear mixed models (NLMM). The three models where inbreeding coefficient was significant are shown with coloured solid lines (C,D and G), the predicted growth curve for inbred individuals ($F = 0.5$) shown in orange and an outbred individual ($F = 0$) shown in green. The coloured dashed lines in all plots are 95% credible intervals for predictions. Predicted slopes are overlaid on top of raw data points. The x-axis is truncated at an age when the growth asymptotes but estimates and data extend beyond the axes.

For mass, using the LMM we found no statistical evidence for inbreeding depression (figure 2B; electronic supplementary material, tables S6 and S7). While the point estimates for inbreeding coefficients showed an overall negative trend with the mass of an individual, suggesting inbred individuals may be lighter, the 95% credible intervals overlapped zero for both F_{UnIW} and F_{ROH} (electronic supplementary material, tables S6 and S7). By contrast, the NLMM showed a significant effect of individual inbreeding coefficients (both F_{UnIW} and F_{ROH}) in the early juvenile weight gain period (growth parameter c ; figure 2D and G; electronic supplementary material, tables S20 and S21). For example, an inbred individual with an F_{UnIW} of 0.25 was predicted to grow approximately 6% slower in the first 25 days of life, gaining on average 273 g compared with 290 g in its outbred counterpart. Using F_{ROH} , we predicted a more pronounced difference, with inbred individuals growing approximately 17% slower, gaining 250 g compared with 293 g over the same period. However, we also show that including a random term of clutch identity to account for parental feeding led to larger credible intervals which then touch 0 (electronic supplementary material, models SM7–SM10)—probably because most individuals within a clutch have similar inbreeding coefficients. Nevertheless, we still show a consistent indication of inbreeding depression in juvenile weight gain, as estimated growth rates for inbred individuals remain lower than outbred individuals (electronic supplementary material, models SM7–SM10).

Finally, for tarsus length, we found inconsistent results between the inbreeding coefficients used in the LMM. Both F_{UnIW} and F_{ROH} in general indicated that higher inbreeding coefficients lead to a shorter tarsus length, but only F_{ROH} showed a significant association (figure 2; electronic supplementary material, tables S8 and S9). An individual with F_{ROH} equal to 0.25 was estimated to have a tarsus length approximately 4.5% shorter. However, using the NLMM we found no significant association with F_{ROH} or F_{UnIW} for growth rate (c) or the asymptote (figure 2E and H; electronic supplementary material, tables S24 and S25).

Regarding the other fixed effects in the model, using the LMM we found that females were heavier, had longer bills but a shorter tarsus (electronic supplementary material, tables S4–S9). We also found that lower ranked individuals (i.e. those born later than their siblings) were lighter and had shorter bills and tarsi (electronic supplementary material, tables S4–S9). We found no association of rank or sex with any growth rates (c) and our results for the ‘final’ asymptotic body mass reflect those in the LMM (electronic supplementary material, tables S16–S27), with the exception that lower rank individuals had a heavier final body mass—probably because only the heavier individuals at a lower rank survived, i.e. selective disappearance.

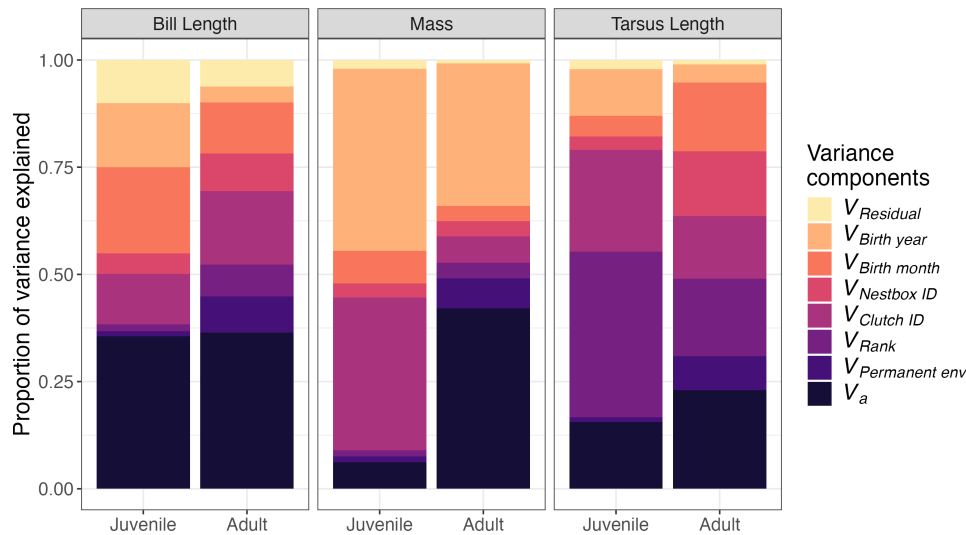


Figure 3. Proportion of variance explained by different components across the juvenile (≤ 180 days old) and adult stages (> 180 days old) for three morphological traits. Thus, additive genetic variance (V_a) in dark purple (bottom) gives an indication of the narrow sense heritability for each trait at different life stages.

Table 1. Proportion of variance explained by additive genetic variance, i.e. trait heritability, h^2 , across two developmental stages: juvenile (≤ 180 days old) and adult (> 180 days old) for three traits, with 95% credible intervals. (Sample size indicates the number of unique individuals in each category with the number of records—since many individuals are recorded multiple times. Note: both developmental stages are used to inform variance estimates (see S2).)

trait	developmental stage	h^2	95% credible Intervals	sample size (no. records)
bill length	juvenile	0.357	0.156–0.511	1970 (5164)
	adult	0.364	0.046–0.717	265 (432)
mass	juvenile	0.063	0.012–0.119	1994 (8156)
	adult	0.419	0.146–0.687	277 (845)
tarsus length	juvenile	0.157	0.067–0.256	1969 (5298)
	adult	0.216	0.007–0.524	266 (433)

(b) Heritability at different developmental stages

Estimates of heritability, h^2 , increased between juvenile and adult stages for all traits, although this increase was only significant in mass (table 1; figure 3; electronic supplementary material, tables S28–S30 and figure S5). Evolvability also significantly increased between juvenile and adult mass, but decreased for the other two traits (electronic supplementary material, figure S5).

In juveniles, environmental factors such as birth year and clutch identity consistently had a greater contribution to the overall phenotypic variance compared with their contribution to adult phenotypic variance (figure 3; electronic supplementary material, tables S28–S30). For mass in particular, the birth month and year as well as the clutch identity, explained the majority of the phenotypic variance in juveniles. Conversely, for adults, in all traits additive genetic variance was the largest contributor to overall phenotypic variance, exceeding the contribution of any other variance components (figure 3; electronic supplementary material, tables S28–S30).

(c) Genetic architecture of inbreeding depression

We ran this analysis with both F_{ROH} and F_{UniW} , yielding non-concordant results (electronic supplementary material, figures S7 and S8, table S31). In the main text, we continue with F_{ROH} because using a local model based estimate of HBD segments probably incorporates any fine-scale uncertainty or bias existing from low coverage genotyping errors, imputation or ascertainment bias [47]. F_{UniW} is more sensitive to local fluctuations in homozygosity, as windows are treated as fully independent, whereas F_{ROH} calls long stretches of homozygosity spanning over multiple windows. After accounting for multiple testing, we found nine windows with significantly deleterious effects (negative estimates) and three windows with significantly beneficial effects (positive estimates) across all traits (figure 4; electronic supplementary material, tables S32–S34 and S37). In bill length—which had the strongest and most consistent estimate of inbreeding depression in our previous analyses—three windows were significantly deleterious on chromosomes 8, 10 and 17; while one window was significantly beneficial on chromosome 2E (figure 4A; electronic supplementary material, tables S32 and S37). We also found a marginally negative skew of effects direction with the average effect of local inbreeding being slightly negative (mean and median window estimates -0.12 and -0.08 , respectively, and see figure 4A right). We identified several genes within the significant genomic windows (electronic supplementary material, table S37). GO enrichment analysis revealed an over-representation of broad developmental and cellular processes, driven by *NOTCH1*, a key regulator of developmental patterning and cell fate determination [48]. No

GO terms specifically related to ossification or keratinization were enriched, suggesting that variation in bill length may be influenced primarily by early developmental regulatory mechanisms rather than by genes directly involved in bone or keratin growth (electronic supplementary material, table S38).

For body mass, where we did not detect inbreeding depression using the LMM in the previous analysis, three significant windows were detected—two with beneficial effects on chromosomes 9 and 15, and one deleterious on chromosome 4A (figure 4B; electronic supplementary material, tables S33 and S37). Here, the mean effect size was slightly positive (0.01) and the median was negative (−0.01) but the distribution mainly centres around zero with limited skew (figure 4B right). GO analysis revealed significant over-representation of molecular functions related to proteolysis and its regulation, driven primarily by *CAPN6* and *LXN* (electronic supplementary material, table S37; [49,50]). This suggests that variation in body mass may be associated with differences in protein turnover, tissue remodelling and growth signalling rather than skeletal elongation directly (electronic supplementary material, tables S37 and S39).

Finally, for tarsus length, for which inbreeding depression was detected only with F_{ROH} in the previous analysis, we identified five windows with deleterious effects, occurring on chromosomes 2A, 2C, 4B, 7 and 23 (figure 4C; electronic supplementary material, table S34). Interestingly, the average effect size of windows were positive (mean and median window estimates 0.13 and 0.11, respectively), but the distribution was negatively skewed and leptokurtic (figure 4C right). This indicates a number of outliers with deleterious effects contributing to inbreeding depression in this trait. In context, given the average tarsus length of an adult is 70 mm, an individual fully identical-by-descent ($F = 1$) for the most highly associated window in tarsus length on chromosome 7 would have a tarsus approximately 2 mm smaller. Furthermore, GO analysis of genes within these outlier windows identified significant enrichment of numerous cellular and developmental categories; specifically, a gene responsible for collagen synthesis (*COL1A2* [51]), two involved in cytoskeletal organization (*MACF1* [52], *CAP1* [53]), and in growth-related signalling pathway (*BMP8A* [54]; electronic supplementary material, tables S27 and S40). As these processes are consistent with underlying skeletal growth and bone elongation, it further suggests that the deleterious effect of inbreeding may be caused by a handful of influential genes.

4. Discussion

(a) Inbreeding depression in morphology and growth

Using a long-term dataset of Swiss barn owls, we reveal that despite only moderate levels of inbreeding, there is appreciable evidence for inbreeding depression in this population across three morphological traits: bill length, tarsus length and body mass. Crucially for survival, we also show that inbred individuals were slower at gaining body mass during juvenile development stages. As we were unable to directly measure inbreeding depression in fitness or survival in this study because of insufficient sample numbers, we used morphological measures as a proxy for fitness. Our findings align with previous work from just a handful of studies in wild birds showing that inbred individuals are lighter [55,56], have smaller skeletal sizes [57] and shorter bills [56]. Such inbreeding depression then probably reflects a broader reduction in fitness, as smaller individuals are less likely to survive in a natural setting. This is particularly the case for early life growth, where fledging mass is known to be positively correlated with juvenile survival in other bird species [16,58]. Therefore, the inbreeding depression we find in these morphological traits probably also impacts the overall fitness of individuals.

We further uncovered that the severity of inbreeding depression varies across developmental stages (figure 2). To our knowledge, this is the first study to directly measure inbreeding depression in early growth rates in a wild population, and only the third to indicate its occurrence [15,17]. Similar early life effects have been shown in domesticated birds [59], but it was previously unknown whether wild birds would also be affected at such an early life stage, particularly as the inbreeding level is often lower than in artificially selected populations. As it manifests predominately at very early life stages this may also explain why we were unable to detect inbreeding depression in mass using the LMM. Interestingly, we also find that inbred individuals appeared to ultimately reach a lower asymptotic bill length despite showing no significant difference in growth rate during the juvenile stage. Rather than any biological explanation, a more likely rationale is that measuring the length of a juvenile bill can be challenging because of its small size. Therefore, in this trait adult measurements are probably more precise, allowing for more accurate estimates of inbreeding depression. For bill length and body mass using the NLMM showed which life stage the severity of inbreeding depression (or our ability to detect it) is the strongest. For tarsus length, we show inbreeding depression using the LMM, but this is not consistent in the NLMM. In this case, breaking down the trait into two parameters (asymptote and growth slope) did not improve detection. In future studies, where such records are available, we recommend assessing early developmental stages as well as late, to avoid missing key evidence for inbreeding depression.

We should also consider the role of selective disappearance in our results. For consistency, adult records only include individuals ringed as juveniles (see table 1), but many juveniles do not return to the study area after fledging and their survival is unknown. As a result, the data used to estimate the asymptote consists of adults that have survived the first selection process. Furthermore, our study uses data from another project where individuals with extensive phenotype records were prioritized for genotyping. Given that we expect highly inbred individuals to be less likely to survive, individuals with more extreme inbreeding coefficients may also be missing from our dataset. Additionally, the predicted age of very young chicks may have affected our results. Age is estimated from wing length at the first visit (see §2a). However, since we show inbred individuals tend to be smaller, this could bias estimates of inbreeding depression in growth. For example, if an inbred individual is small for its true age, its age will be underestimated, as will the level of inbreeding depression. Nevertheless, estimates of the asymptote should be unaffected since age has little effect on trait variation after adulthood. Overall, while we found convincing evidence

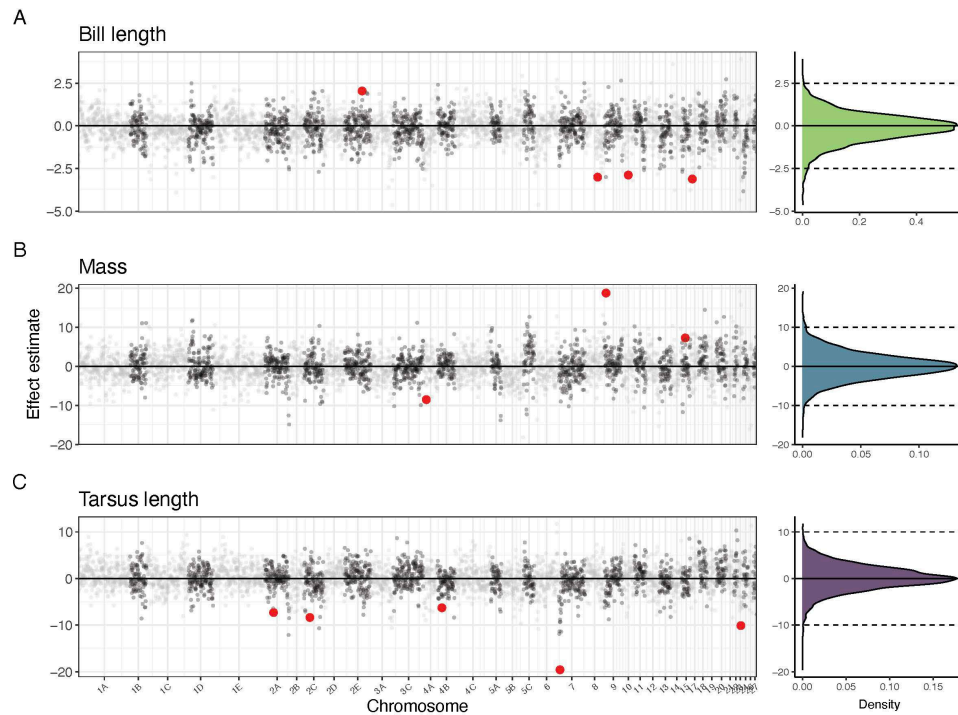


Figure 4. (A–C) Genetic architecture of inbreeding depression in three traits. Left: each dot represents the estimated effect size at the trait scale of local inbreeding within a 2500 SNP window when local and genome-wide inbreeding F_{ROH} are fitted together in the same model. Chromosome numbers are the chicken chromosomes with strong synteny to the recombination super scaffolds identified in Topaloudis *et al.* [33]. Red dots show significant windows crossing the Bonferroni corrected significance threshold where the p -value is calculated as the number of post-warm up iterations with an opposite values to the mean estimate (see §2). Right: density plots of estimated local inbreeding effects. Solid line shows zero effect in both plots.

of inbreeding depression in this wild population, we may be underestimating its true extent because of our data collection methods, highlighting the value of such continuous long-term studies.

(b) Heritability increases over developmental stages

For all traits, we show that sources of trait variation change throughout the development of an individual and heritability tends to be larger in adults than in juveniles. This change in heritability with age is an established pattern [22], usually attributed to the decrease in environmental sources of variation [60]. In our analysis, both clutch ID (reflecting parental investment) and birth year (reflecting food availability and weather conditions) contributed greatly to the phenotypic variance of a trait, particularly for body mass (figure 3). In this species, juvenile barn owls rely entirely on parental care for food and protection until fledging, but the level of parental care can fluctuate, making chick mortality and competition more likely if parental care is poor or brood size is large [23,61]. Moreover, food availability can fluctuate between years and a low food year will also result in stress. During these stressful periods in early life, inbreeding depression is probably heightened [20,21]. For example, in meerkats, Nielsen *et al.* [17] showed that inbreeding depression was only present in juvenile survival but not at the earlier stage of pup survival. They attributed this trend to more stressful environmental conditions following independence as helper care provisioned the pups before independence. Therefore, the stressful environmental conditions experienced during early development of barn owl chicks such as food competition, poor parental care and low food availability may heighten inbreeding depression during this time; specifically in the case of body mass, which is the only trait showing inbreeding depression in early life growth (figure 2) and has the highest proportion of environmental trait variance (figure 3).

(c) Genetic architecture of inbreeding depression

Our last aim was to determine whether particular regions of the genome contribute more (or less) to the observed inbreeding depression in our study population. In recent years, other studies have had the same goal also using fitness data from their respective populations—Soay sheep [12], hihi [13] and red deer [62]. Here, across our three traits we identified nine windows that showed a significant negative association with trait values and three with a positive association, the latter may be regions where being homozygous for an allele is beneficial [12]. On the other hand, windows with a negative association probably contain loci harbouring deleterious (recessive) alleles, particularly in tarsus length which showed a skewed distribution of effect sizes with a handful of more negatively associated windows, and genes within these windows which were associated with bone elongation. Although, strongly deleterious alleles are expected to be kept at a lower frequency because of purging. In Stoffel *et al.* [12], the existence of strongly deleterious genomic regions was attributed to long-term small population size (N_e) and genetic drift allowing these alleles to persist. In our case—where N_e is large [5] and extreme inbreeding is relatively uncommon (figure 2A)—the relatively modest effect sizes of deleterious windows (figure 4) may have prevented them from being fully purged. Overall, the combination of several mildly deleterious loci genome-wide, and a few loci with moderate/severe effects probably

causes the observed inbreeding depression, highlighting the importance of considering both widespread and localized effects of inbreeding.

(d) Comparison of inbreeding coefficients

Finally, using two widely used inbreeding coefficients, we have highlighted that the inference of inbreeding depression can be sensitive to the metric used to quantify individual inbreeding. While both inbreeding coefficients consistently showed a negative effect of inbreeding on the trait values, the statistical significance depended on which was used. These differences may arise from the architecture of traits or the specific variants driving inbreeding depression. In traits for which F_{ROH} shows a significant association but F_{UniW} does not (e.g. tarsus length in this study), inbreeding depression may be caused by recessive deleterious mutations, which have recently come together in ROHs [7]. Conversely, traits that only show an association with F_{UniW} and not with F_{ROH} (e.g. bill length in the NLMM), could demonstrate inbreeding depression caused by more ancient relatedness in the population—i.e. not in long ROHs. On the other hand, while F_{UniW} is less heavily influenced by rare alleles than its unweighted counterpart F_{Uni} [9], it may still be more so than F_{ROH} . In this case, F_{UniW} can capture inbreeding depression associated with slightly rarer alleles within the population. Moreover, our equivalent genetic architecture analysis with local F_{UniW} contained no overlapping significant windows, further suggesting that different causal variants are being captured between metrics. Alternatively, these differences could be because of localized fluctuations in allele frequency (either legitimate or owing to fine-scale imputation errors) which F_{UniW} will be more sensitive to. Overall, further research is needed to fully understand how specific deleterious variants contributing to inbreeding depression can affect each coefficient, and in turn the association level.

5. Conclusions

In conclusion, we now have a more complete picture of how inbreeding depression affects this population. We find compelling evidence for inbreeding depression in three morphological traits using multiple complementary methods and show that the severity of inbreeding depression—or our ability to detect it—changes over the course of an individual's growth period. Interestingly, the choice of inbreeding coefficient also affects the ability to detect inbreeding depression. Notably, we reveal that inbreeding depression appears to affect weight gain at an early age. We also show that environmental impacts on traits change over the same time period which may play a role in determining the severity of inbreeding depression. Finally, we show that the genetic architecture of inbreeding depression probably consists of a number of mildly deleterious (recessive) alleles genome-wide, alongside a handful of loci with a more severe impact.

Ethics. All animal handling was performed in accordance with international animal welfare standards and the Swiss Animal Welfare Act, and under permission from the Veterinary Office of the Canton of VD, Fr, BE 3213 and 3571 and the Federal Office for the Environment.

Data accessibility. The genomic data in this study have been previously used in Cumer et al. [32] and Topaloudis et al. [33]. Raw reads are available on NCBI under BioProject: PRJNA1391678. Dataframes used for analysis are available on Figshare [63–65], with full code used to create and analyse said datasets [66].

Supplementary material is available online [67].

Declaration of AI use. AI-assisted technologies were used in some cases to improve readability and language of the work.

Authors' contributions. A.H.: conceptualization, formal analysis, investigation, methodology, visualization, writing—original draft, writing—review and editing; E.L.: conceptualization, data curation, formal analysis, investigation, methodology, writing—original draft, writing—review and editing; A.T.: data curation, writing—review and editing; T.C.: data curation, writing—review and editing; A.-L.D.: data curation, writing—review and editing; C.S.: data curation; B.A.: data curation, resources, writing—review and editing; A.R.: data curation, resources, writing—review and editing; J.G.: conceptualization, data curation, resources, supervision, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

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