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Evaluating modelling strategies and genotype by environment for mortality in crossbred layers

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Introduction

Layer breeding has delivered substantial gains in egg number and persistency, enabling extended laying cycles (90–100 weeks) while maintaining commercially acceptable egg quality. However, production in intensive regimes increase exposure to cumulative health and welfare risks that can ultimately manifest as on farm mortality (Bain *et al*., 2016). In contrast to birds in breeding nuclei, which are monitored under highly controlled conditions with individual traceability, commercial crossbred hens are typically managed in large groups within cages or cage-free systems, where social interactions are perversive (Peeters *et al*., 2012). Under group housing, injurious feather pecking can lead to feather cover deterioration, increase mortality and create strong shared-cage dependencies (Rodenburg *et al*., 2013). Additionally, across commercial farms, mortality varies because environments differ in climate, management intensity, health pressure, and group dynamics, even when the same genetic material is used (Weeks *et al*., 2016). This creates a clear genotype by environment (G×E) interaction, where sire or line rankings can shift when hens are evaluated in contrasting environments, as demonstrated in laying hens across temperate and tropical conditions (Mathur and Horst, 1994).

Mortality can be modelled either as an end-of-cycle binary trait (dead/alive) or as a time-to-event outcome that leverages when death occurs and appropriately accommodates right-censoring (Yazdi *et al*., 2002). Linear models applied to 0/1 records are simple but can attenuate variance components for low-incidence traits because distributional assumptions are misaligned. Threshold models address this by mapping observed categories to an underlying continuous liability (Gianola and Foulley, 1983). Survival models, model the time to event directly and handle censoring explicitly (Yazdi *et al*., 2002). The choice of model affects parameter estimates and selection decisions, underscoring the need for such comparisons (Ødegård *et al*., 2006).

This objective of this study is two-fold, we first compare linear, threshold, and Weibull models using single-farm data and, then evaluate multiple farms data using random regression to capture genotype-by-environment interaction.

Materials & Methods

The dataset included 18,510 crossbred hens from reciprocal crosses A×B (n=10,297) and B×A (n=8,213), with 26,843 pedigree records tracing ancestry up to six generations. Crossbreds represented two generations; each housed in a separate facility (generation and house were confounded). Birds were allocated to 10 houses across six farms, with 721–3,233 hens per house. All farms operated group cage systems, varying from 106–886 cages per farm and 3–13 hens per cage. Across sires, recorded offspring ranged from 6 to 271 per sire (90 ± 61). Sire connectedness across environments was high, with progeny recorded in 4 farms on average, 84% of sires had offspring in ≥3 farms. Outcomes were end-of-lay mortality (0=alive, 1=dead)

and survival time (weeks to death); overall mortality was 12%, with 16,294 hens surviving to the end of the cycle.

Models comparison. For the initial comparison of the three modelling approaches, analyses were restricted to one farm (6,095 hens) with 1,303 cages (4–6 hens per cage). All three modelling approaches shared the same linear predictor with fixed effects of cross and generation/house and random effects of cage and animal additive genetic:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_1\mathbf{c} + \mathbf{Z}_2\mathbf{a} + \mathbf{e} \quad (1)$$

In Eq.1, **y** is the response vector (binary mortality for the linear and threshold models; time-to-event outcome for the survival model), **X** is the incidence matrix for fixed effects (cross, generation/house), **b** is the fixed-effects vector, $\mathbf{Z}_1$ and $\mathbf{Z}_2$ are incidence matrices for the random cage (**c**) and additive genetic animal (**a**) random effects, assumed to be independent and normally distributed , $\mathbf{c} \sim \mathcal{N}(0, \sigma_c^2\mathbf{I})$ and $\mathbf{a} \sim \mathcal{N}(0, \sigma_a^2\mathbf{A})$, where **I** is an identity matrix and **A** is the pedigree-based relationship matrix. For the linear model, mortality was coded $y_i \in \{0,1\}$ and analysed directly under Eq.1 assuming $\mathbf{e} \sim \mathcal{N}(0, \sigma_e^2\mathbf{I})$. In threshold, the observed binary outcome $y_i \in \{0,1\}$ was linked to an unobserved continuous liability λ_i via a probit threshold ($y_i = 1$ if $\lambda_i > 0$; $y_i = 0$ if $\lambda_i \leq 0$), with λ modelled using Eq.1. Survival model. Time-to-death was analysed using a Weibull proportional hazards frailty model with right censoring at $T = 100$ weeks; t_i denotes the observed time and δ_i the event indicator ($\delta_i = 1$ for death; $\delta_i = 0$ for censoring). The hazard followed a Weibull baseline and incorporated the same linear predictor as in Eq.1.

Genotype-by-environment analysis. G×E interaction for end-of-cycle mortality was evaluated using a threshold random-regression animal model implemented in the BLUPF90 suite. Mortality was analysed as a two-category trait (0 = alive, 1 = dead) under a probit threshold model. The environmental covariate was cage occupancy (number of hens per cage), scaled to $s \in [-1, 1]$, and modelled using first-order Legendre polynomials P0(s) and P1(s).

$$\boldsymbol{\lambda} = \mathbf{Xb} + \mathbf{Z}_1\mathbf{c} + \mathbf{Z}_2\mathbf{a} + \mathbf{e} \quad (2)$$

Where, **λ** is the vector of latent liabilities; **X** is the incidence matrix for fixed effects (**b**) including Farm×House, cross, and fixed regression on $P0(s)$ and $P1(s)$; $\mathbf{Z}_1$ and $\mathbf{Z}_2$ relate records to random cage effects (**c**) and additive genetic animal effects (**a**), respectively; and **e** is the residual. Cage effects were assumed independent with $\mathbf{c} \sim \mathcal{N}(0, \sigma_c^2\mathbf{I})$. The additive genetic effect for an individual at environment s was specified as a random regression, $\mathbf{a}(s) = \mathbf{a}0P0(s) + \mathbf{a}1P1(s)$, where **a**0 and **a**1 are vectors of animal-specific intercept and slope coefficients. The joint distribution of (**a**0, **a**1) was assumed multivariate normal with covariance structured by the pedigree relationship matrix **A** and a 2×2 (co)variance matrix **G**, where $\mathbf{G} = \begin{bmatrix} \sigma_{a0}^2 & \sigma_{a0a1} \\ \sigma_{a0a1} & \sigma_{a1}^2 \end{bmatrix}$. Under the probit parameterisation, the binary outcome was linked to **λ** with standard-normal liability residuals $\mathbf{e} \sim \mathcal{N}(0, \mathbf{I})$ for identifiability.

Using Bayesian inference, posterior marginal densities of the variance parameters and breeding values for linear and threshold models were estimated with the *gibbsf90+* program from the *BLUPF90* package, using Gibbs sampling under *BLUPF90*'s default weakly informative priors. The survival model was fitted in R-INLA (Laplace-approximation Bayesian inference), assigning weakly informative penalised complexity (PC) priors to cage and additive genetic precision parameters. Posterior means and standard deviations are reported.

Results & Discussion

Comparing the three modelling approaches in the first dataset (n = 6,095) showed that mortality variance partitioning and breeding values ranking was sensitive to model choice. The linear

mixed model, fitted directly to the observed 0/1 outcome, yielded low heritability of 0.10 ± 0.03, and a modest cage contribution of 0.14 ± 0.01. This is common when binary traits are analysed as Gaussian on the observed scale (Gianola, 1982). Moving to threshold model increased both the heritability to 0.25 ± 0.07 and cage shares (0.29 ± 0.04). The probit threshold model operates on an unobserved liability (probit) scale with fixed residual variance and accordingly shifted more signal into both additive and cage components, but it still treats mortality as binary and does not exploit death timing (Gianola 1982). The Weibull proportional-hazards frailty model exploits time-to-event data and explicitly handles right censoring (Cox, 1972; Yazdi *et al*., 2002), yielding a heritability comparable to the threshold model (0.24 ± 0.02) but a slightly larger cage component (0.35 ± 0.02) with tighter posterior uncertainty. Because the survival model is formulated on a latent log-hazard (linear predictor) scale, heritability is not uniquely defined and depends on the assumed residual variation on that scale (Yazdi *et al*., 2002); here we adopted the standard extreme-value assumption, fixing $\mathrm{Var}(\varepsilon) = \pi^2/6$ (Yazdi *et al*., 2002). Across models, EBV rankings were highly concordant between linear and threshold analyses (Spearman ρ = 0.98), whereas concordance with the survival model was moderate (ρ = 0.70–0.71), indicating meaningful reranking once timing and censoring are incorporated, which aligns with reports from longevity evaluations where proportional-hazards approaches can differ from linear evaluations in prediction (Caraviello *et al*., 2004). Overall, the linear model is convenient but overly simplistic for binary mortality, whereas the Weibull model is information-rich but introduces additional complexity due to scale and parameterization dependent variance components (Yazdi *et al*., 2002). Therefore, we prioritized the threshold model for the subsequent random-regression G×E analysis.

The random regression model attributed a significant share of variation to the shared cage environment (σ_c^2 = 0.43 ± 0.05). The large cage component is biologically plausible in layers, given the role of shared micro-environment and social interactions. Previous work on laying-hen emphasises the importance of grouping structure and social effects for mortality-related outcomes (Ellen *et al*., 2008).

The model suggested additive genetic heterogeneity in both baseline mortality liability (**a**0) and sensitivity to cage occupancy (**a**1), but with considerable uncertainty. The posterior mean of the genetic intercept variance was σ_{a0}^2= 0.31 ± 0.15, while the variance of the genetic slope was σ_{a1}^2= 0.36 ± 0.23. These estimates indicate that survival in laying hens is partly heritable and therefore can respond to selection, rather than being driven only by management (Ellen *et al*., 2008).

Additionally, the slope variance (σ_{a1}^2) shows that genotypes differ in how their mortality liability changes with cage occupancy, providing direct evidence of genotype-by-environment interaction (Schaeffer, 2004). This indicates that the selection program should account not only for average genetic merit, but also for sensitivity to the environment, because birds can differ genetically in how their mortality risk responds to changes in cage occupancy (Benyi *et al*., 2006). The intercept–slope covariance was positive but imprecise (σ_{a0a1} = 0.14 ± 0.17), leading to a moderate yet highly uncertain genetic correlation (r_{a0a1} = 0.32 ± 0.43). This implies weak evidence that higher baseline liability is systematically coupled with greater (or lower) sensitivity to cage occupancy. Consequently, selection for improved average survival and for robustness to cage occupancy may be pursued as partially independent objectives, but the relationship is uncertain.

Environment-specific predictions indicate that stocking density modulates both the magnitude of genetic variation for mortality and, at the extremes, may affect genetic rankings. At low to moderate densities (3–6 hens/cage), additive genetic variance was stable ($\sigma_a^2 \approx$ 0.25–0.29) and heritability was moderate ($h^2 \approx$ 0.15–0.17). At higher cage occupancy (10–13 hens/cage), σ_a^2 increased (≈ 0.42–0.57) and h^2 rose (≈ 0.21–0.26), although estimates were less precise, supporting a scaling component of G×E, where genetic differences are larger at higher densities

(Fodor *et al*., 2023). In parallel, together with the non-zero slope variance, results suggest potential reranking under divergent densities: the genetic correlation between low and high density (3 vs 13 hens/cage) was moderate on average ($r_a = 0.37$) but highly uncertain (−0.20 to 0.99), and EBV agreement was only moderate (Spearman $\rho = 0.54$) with limited overlap among 5% top-ranked animals (193/938). Collectively, these findings imply that selection decisions could be density-sensitive at the extremes, supporting consideration of environment-specific evaluations when breeding for survival across heterogeneous commercial conditions (Rauw and Gomez-Raya, 2015).

Conclusions

Model choice affected both variance estimates and EBV rankings for mortality. The linear model yielded lower heritability and cage effects estimates, whereas the threshold model recovered higher additive and cage variance by modelling mortality on a liability scale. The Weibull survival model produces similar heritability to the threshold model but a slightly larger, more precisely estimated cage component. EBV rankings were highly consistent between the linear and threshold models, but only moderately aligned with the Weibull survival model. Given the objective and interpretability, the threshold model was better aligned with the binary nature of mortality and provides a more appropriate inferential framework for modelling G×E.

The random regression analysis provided evidence of genotype-by-environment interaction for mortality across the stocking-density gradient. The non-zero genetic slope variance confirmed that genotypes differ in sensitivity to stocking density. Consequently, genetic variance increased at higher densities and rankings shifted at the extremes, implying that selection decisions could be density-sensitive and may benefit from environment-specific evaluations in heterogeneous commercial conditions.

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