CHALLENGES IN GENOMIC SELECTION IN POULTRY: FROM CHANGES IN VARIANCE COMPONENTS TO MODELING AND EFFICIENT ANALYSIS OF CATEGORICAL DATA

by

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(Under the Direction of Daniela Lourenco)

ABSTRACT

Proper evaluation of animals in breeding programs is essential to maximize response to selection. While breeding programs continually refine their selection strategies, they face evolving challenges stemming from factors such as the increasing number of genotyped individuals and reduced additive genetic variance due to selection. These challenges are even more pronounced for breeding programs for poultry species due to their rapid generation intervals, which speed up selection responses compared to other livestock species. This dissertation aims to address some of the current challenges affecting the breeding programs of broiler breeders. Accurate genetic parameters are vital for predicting breeding values to maximize the selection response. Changes in genetic parameters and correlations of SNP marker effects were estimated over time to investigate the effect of genomic selection on the population. Under genomic selection, the decline in additive genetic variation is faster, and thus, genetic parameters must be updated frequently with all the information used for selection decisions. In genomic evaluations, it is imperative to make selection decisions using the correct trait definition to ensure continued genetic gains. In the poultry industry, mortality is an economically important trait with financial and societal pressure

for improvement. Alternative trait definitions were investigated for chicken mortality to explore whether any model accuracy improvement existed or whether a maternal genetic effect could be applied to this trait. Splitting mortality into time periods with the inclusion of the maternal genetic effect for early mortality may enhance the genetic evaluation in broiler breeder populations compared to the trait definition currently used for evaluations. Like mortality, many traits evaluated in breeding programs are binary or categorical by nature. Evaluating categorical traits can be costly regarding both time and computing performance, especially with large genomic data. A new method using an EM algorithm was investigated for efficiency without sacrificing breeding value accuracy within categorical trait evaluations. This approach used phenotypes and residuals from a linear model to impute liabilities for the categorical traits.

INDEX WORDS: Poultry, ssGBLUP, Threshold Modeling, Mortality, Genomics, Accuracy, Categorical traits, Genetic Variance

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DEDICATION

To my parents, whose unwavering support and boundless love have been my pillars. I will forever be grateful for your encouragement to embrace my full potential and to reach for the stars.

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CHAPTER 1

INTRODUCTION

The evolution of genetic evaluations in poultry breeding reflects a dynamic journey shaped by historical milestones and continuous technological advancements. Originating in the mid-1900s with the rise of commercial meat chicken production, the "Chicken of Tomorrow" contest marked the onset of systematic breeding programs to enhance meat-type chicken traits. The early breeders, leveraging mass selection and confronting challenges like negative correlations between production and health traits, paved the way for subsequent improvements in breeding strategies.

The emergence of Best Linear Unbiased Prediction (BLUP) in the 1950s played a pivotal role in refining genetic evaluations, enabling the estimation of breeding values with minimized prediction error variance. Over time, BLUP evolved with mixed model equations and efficient methods for inverting the pedigree relationship matrix, laying the foundation for contemporary genetic evaluations.

The genomic era ushered in a paradigm shift with the discovery of single nucleotide polymorphisms (SNPs) and the application of genomic information in breeding programs. Genomic selection (GS), initially explored by Meuwissen in 2001, marked a transformative phase, though initially constrained by high genotyping costs. The advent of large-scale genotyping, starting with cattle, eventually extended to poultry, catalyzing the development of high-density SNP chips in 2011.

Integrating genomic information into genetic evaluations spurred the advancement of methodologies, including multi-step and single-step approaches, which enhanced the precision of genetic evaluations within poultry breeding. Addressing the computational challenges posed by increasing genotyped individuals, the Algorithm of Proven and Young (APY) presented an innovative solution, allowing efficient inversion of the genomic relationship matrix.

Variance component estimation, a crucial aspect of accurate breeding value prediction, has been advanced through statistical methods like Restricted Maximum Likelihood (REML) and Bayesian approaches, such as Gibbs sampling. As poultry breeding faced the challenge of modeling categorical data, linear and threshold models were explored, each with advantages and computational complexities.

In this intricate landscape of poultry genetic evaluations, the validation of genomic predictions became paramount. Cross-validation studies, particularly the innovative approach introduced by Legarra and Reverter in 2018, facilitated the comparison of genetic and genomic evaluation models in terms of accuracy, bias, and dispersion of breeding values.

Navigating the complexities of modern genetic and genomic evaluations demonstrates that continuous research remains imperative to refine methodologies, overcome challenges, and ensure the sustainable progress of poultry breeding programs. Thus, this dissertation aimed to investigate the challenges of genomic selection in poultry through changes in genetic parameters, alternative trait definitions of mortality, and efficient genomic evaluations of categorical data. In Chapter 2, a literature review is presented. Chapter 3 investigates the temporal dynamics of genetic parameters and SNP effects over time for traits in a broiler population undergoing selection. In Chapter 4, alternative trait definitions for broiler mortality are explored and investigated. Chapter 5 presents

an alternative algorithm for evaluations of large categorical genomic datasets. The general conclusions of this dissertation are presented in Chapter 6.

CHAPTER 2

LITERARTURE REVIEW

HISTORY OF POULTRY GENETIC EVALUATIONS

The foundation of modern poultry breeding programs began in the mid-1900s, driven by the profitability of raising commercial meat chickens through mass production methods. In 1950, 616 million birds were raised in the United States (Shrader, 1952). This increase in production inspired "The Chicken of Tomorrow" contest to create an improved meat-type chicken with more meat on the breast, thigh, and drumsticks. A considerable surge in research unfolded, marked by the dawn of long-term selection experiments across diverse poultry types. Concurrently, commercial breeding and development enterprises surfaced, aiming to transform these experimental approaches into practical industrial applications (Hunton, 2006).

Early breeders achieved rapid progress in meat-type chicken breeding, capitalizing on the high heritability of desirable traits. The initial method involved mass selection, where individual birds were chosen based on their phenotype for traits with sufficiently high heritability (Neeteson et al., 2023). This process included testing animals for live body weight in early life, with the best males and females becoming parents for the next generation. However, challenges emerged, including negatively correlated responses between production and reproduction and health traits. These correlations include growth rate leading to issues like excessive fat, leg weakness, ascites, and reproductive health. In an attempt to manage these correlations, male and female lines were created (Hunton, 2006).

Over the years, breeders refined their methods significantly to enhance breeding programs. With advanced computing capabilities, they deployed diverse genetic and economic tools for developing modern chickens. These tools, including statistical approaches, selection index development, evaluation of pureline and crossbreeding, evaluation of siblings, and the deliberate use of stressful environments to identify behavioral differences, reflect the industry's commitment to continually improving and fine-tuning breeding strategies (Wolc et al., 2015).

Best Linear Unbiased Prediction (BLUP)

One of the most widely used statistical approaches for genetic selection is best linear unbiased prediction (BLUP), first proposed by Henderson (1949), allowing for concomitantly estimating of fixed effects and breeding values. BLUP's properties are embedded in its name. *Best* denotes its capability to minimize prediction error variance (PEV). *Linear* signifies that predictors are linear functions of the observations. *Unbiased* indicates that the estimation of realized values for a random variable and estimable functions of fixed effects is unbiased. Lastly, *prediction* involves the prediction of random effects.

Henderson (1950) introduced the mixed model equations (MME) for the simultaneous estimation of solutions for fixed effects and the prediction of solutions for random effects. Considering the following general mixed model:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Zu} + \mathbf{e} \qquad [2.1]$$

where **y** is a vector of observations, **b** is a vector of fixed effects, **u** is a vector of random animal effects, **e** is a vector of random residual effects, **X** and **Z** are incidence matrices for the fixed and random effects, respectively.

Further advancements in BLUP mixed model equations research included the development of methods for recursively constructing the inverse of the pedigree relationship matrix (Henderson,

1976; Quaas, 1976). The devised method for inverting the pedigree relationship matrix enhanced efficiency. It reduced computing costs, which led to the integration of BLUP into the animal model, predicting the additive genetic effect for each individual (Quaas, 1988). The MME applied to an animal model are:

$$\begin{bmatrix} \mathbf{X'X} & \mathbf{X'Z} \\ \mathbf{Z'X} & \mathbf{Z'Z + A^{-1}}\,\lambda \end{bmatrix} \begin{bmatrix} \hat{\mathbf{b}} \\ \hat{\mathbf{u}} \end{bmatrix} = \begin{bmatrix} \mathbf{X'}y \\ \mathbf{Z'}y \end{bmatrix} \quad [2.2]$$

where $\mathbf{A^{-1}}$ is the inverse of the pedigree relationship matrix and λ is a variance ratio, σ_e^2/σ_a^2. These methods laid the foundation for methods used to conduct genetic evaluations which are still used in breeding programs today.

Into the Genomic Era

The discovery of single nucleotide polymorphisms (SNPs) as a significant contributor to genome variation, particularly highlighted by the Human Genome Project in 2001, has significantly impacted livestock genetics (Venter et al., 2001). Single nucleotide polymorphisms (SNP) are responsible for most of the genetic variation and are abundant throughout the genome. The use of SNP has become essential in animal breeding, notably in genomic selection (GS). Not long after the publication of the human genome sequence, the chicken genome was published through the Chicken Genome Project in 2004 (Hillier et al., 2004).

The first study highlighting the potential benefits of incorporating genomic information into genetic evaluations was performed by Meuwissen (2001). Meuwissen's simulation showcased the effectiveness of genomic information in predicting breeding values, marking a transformative era in the application of genomic information in genetics. Although the cost of genotyping individuals was expensive early in the genomic era, costs have decreased drastically and are projected to continue decreasing, allowing more and more individuals to be genotyped for selection purposes (Schaeffer, 2006; Weigel, 2010). Poultry breeding companies began research

on the application of genomics in broiler breeding. These companies tried to make advancements in technology while genotyping large numbers of animals. With large numbers of phenotypic data, near complete pedigrees, and dense genotypes on large numbers of individuals, the efficacy of genomic selection in real populations could be validated and produce promising results (Avendaño et al., 2010, 2012).

LARGE SCALE GENOMIC EVALUATIONS

Large-scale genotyping in livestock populations first started in cattle with the introduction of the SNP 50k bovine chip (Matukumalli et al., 2009). Subsequently, other livestock species followed suit, crafting their own SNP chip panels tailored for genomic evaluations. Although the chicken was the first farm animal whose genome was sequenced entirely, the development of a high-density SNP chip had not been released into the public domain until the release of a 60K SNP chicken chip in 2011 (Groenen et al.).

As the number of genotyped individuals began increasing, the following challenge animal breeders faced was how to incorporate this new information into existing genetic evaluations. Previous genetic evaluations were based on pedigree relationships and phenotypic information. However, for genomic evaluations, methods had to be developed to include genomic information in addition to pedigree relationships and phenotypic information. VanRaden (2008) introduced three methods for creating the genomic relationship matrix (**G**). The first and most common method:

$$\mathbf{G} = \frac{\mathbf{ZZ'}}{\mathbf{2}\sum \boldsymbol{p_i}(\mathbf{1} - \boldsymbol{p_i})} \qquad [2.3]$$

The **G** effectively measures the count of homozygous loci for each individual along the diagonals while also assessing the number of shared alleles among individuals in the off-diagonals. Notably, these measures deviate from simple identity by state (IBS), incorporating allelic frequencies and centering, thus providing a robust approximation of real (IBS). This approximation is better than the accuracy attained through pedigree approximation (Legarra et al., 2021b).

Using actual or realized relationships determined in **G** provides more accurate predictions compared to the expected relationships provided by **A**. Although the differences between **G** and **A** are typically minor, these differences can significantly increase reliabilities, especially if the number of genotyped individuals is large (VanRaden, 2008; VanRaden et al., 2009). Using **G** instead of **A** in BLUP resulted in a new approach for genetic evaluations called genomic BLUP or GBLUP, where only genotyped individuals were used in the relationship matrix.

Multi-Step versus Single-Step Methods

Despite growing numbers of genotyped individuals, most individuals in the pedigree were not genotyped, meaning they could not receive estimated breeding values from GBLUP. One successful attempt to handle this problem is the multi-step procedure, where several steps are conducted to obtain breeding values (VanRaden et al., 2009). First, a traditional evaluation is performed using the pedigree relationship matrix, and then the EBVs are deregressed. Second, GBLUP is used to estimate SNP effects and then a direct genomic value is calculated for genotyped individuals. Lastly, direct genomic values are combined with EBVs to get GEBVs. It has been shown that this procedure may be limited to simple models, and bias and errors might be introduced in the multiple steps (Misztal et al., 2009; VanRaden and Wright, 2013).

Another alternative method proposed is called single-step genomic BLUP (ssGBLUP), where a relationship matrix that combines pedigree and genomic relationships is used instead of

A that could support genomic models. This combined relationship matrix (**H**) was first proposed by Legarra et al (2009). This method extends genomic information to non-genotyped animals based on the joint distribution of breeding values of non-genotyped and genotyped animals. According to Aguilar et al. (2010b) and Christensen and Lund (2010), the inverse of **H** is:

$$\mathbf{H^{-1}} = \mathbf{A^{-1}} + \begin{bmatrix} 0 & 0 \\ 0 & \mathbf{G^{-1}} - \mathbf{A_{22}^{-1}} \end{bmatrix} \quad [2.4]$$

where $\mathbf{A^{-1}}$ is the inverse of a pedigree-based relationship matrix for all animals, $\mathbf{A_{22}^{-1}}$ is the inverse of the pedigree-based relationship matrix for genotyped animals only, and $\mathbf{G^{-1}}$ is the inverse of a genomic relationship matrix. For routine evaluations in livestock, ssGBLUP is currently the favored method. This is especially true for poultry, as broiler datasets are large, and ssGBLUP allows for multi-trait evaluation using the well-known framework of mixed models (Wolc et al., 2016).

Algorithm for proven and young (APY)

Addressing the increased computational cost was one major challenge of increasing numbers of genotyped animals. The computational cost increases cubically with the number of SNPs and quadratically with the number of genotyped animals (Bermann et al., 2021b), and inverting **G** becomes unfeasible once the number of genotyped animals is more than 150,000 (Fragomeni et al., 2015). To address this issue, the Algorithm of Proven and Young (APY) was presented by Misztal et al. (2016). The APY method directly inverts only a small portion of **G** composed of relationships among animals treated as core. Since livestock species have relatively small effective population sizes, **G** has a rank of ~4k, ~5k, to ~15k for chickens, pigs, and cattle, respectively (Pocrnic et al., 2016b). Then, the inverse of **G** can be obtained by recursion on a number of core animals equal to the rank of **G**. When animals are designated as core or non-core, the inverse of **G** can be directly obtained as:

$$\mathbf{G}_{\text{APY}}^{-1} = \begin{bmatrix} \mathbf{G}_{\text{cc}}^{-1} & 0 \\ 0 & 0 \end{bmatrix} + \begin{bmatrix} -\mathbf{G}_{\text{cc}}^{-1}\mathbf{G}_{\text{cn}} \\ \mathbf{I} \end{bmatrix} \mathbf{M}^{-1} [-\mathbf{G}_{\text{nc}}\mathbf{G}_{\text{cc}}^{-1} \quad \mathbf{I}] \qquad [2.5]$$

where **M** is a diagonal matrix with elements:

$$m_i = g_{ii} - \mathbf{g}_{\text{ic}}\mathbf{G}_{\text{cc}}^{-1}\mathbf{g}_{\text{ci}} \qquad [2.6]$$

where i refers to the ith genotyped, noncore animal.

The computational cost under APY is almost linear regarding computations and memory with the number of animals (Fragomeni et al., 2015). Multiple studies have shown that GEBVs are stable when APY is used when the number of core animals is representative of the dimensionality of genomic information, and the correlation between GEBV when using APY or not is greater than 0.99 (Fragomeni et al., 2015; Masuda et al., 2016). The choice of core animals for recursion can be arbitrary for accuracy, but a random choice of core animals is more ideal for convergence (Bradford et al., 2017b). This method has been successfully used to construct the genomic relationship matrix in dairy populations with almost 4 million genotyped animals (Masuda et al., 2019).

Validation of genomic predictions

The adoption of new genetic or genomic evaluation methods in animal breeding prompts considerations about choosing and assessing models. The increased use of genomic selection, encouraging riskier decisions like selecting unproven young candidates in dairy cattle, has highlighted the need for tools to rank, understand, and quantify prediction model behavior. In this animal breeding context, cross-validation studies, uncommon in pedigree-based genetic evaluation, have become the norm for checking genomic predictions (Legarra and Reverter, 2018).

Cross-validation relies on one of two approaches, as outlined by Legarra and Reverter (2017). The first approach compares (G)EBV or predicted phenotypes to pre-adjusted observed phenotypes. The second approach compares (G)EBV to highly accurate EBV obtained from

progeny testing. While these methods are valuable, concerns arise regarding their quality or adequacy. Firstly, there is a dependency on pre-adjusted phenotypes. Secondly, obtaining unbiased estimates of DYD becomes increasingly challenging due to the growing difficulty in acquiring progeny-tested bulls selected based on GEBV. Lastly, these approaches may prove inadequate for complex models, such as those involving maternal effects.

Legarra and Reverter (2018) introduced a new cross-validation approach. This method compares estimated breeding values derived from all available phenotypes with statistics computed in the absence of phenotypic information for a specific group of focal animals. The LR method offers the flexibility to choose between various sets of focal animals, including parents or young animals. The three main statistics calculated under LR validation are accuracy, bias, and dispersion. Bias measures the bias of EBVs for the focal animals and has an expectation of zero if the evaluation is unbiased. The b_1 measures the dispersion of the EBVs for the focal animals and has an expectation of one if there is no over- or under-dispersion.

In this method, the validation statistics are calculated as:

$$\text{acc} = \sqrt{\frac{\text{cov}\left(\hat{\mathbf{u}}_{\text{w}}, \hat{\mathbf{u}}_{\text{p}}\right)}{(1-\bar{\text{F}})\sigma_{\text{u}}^2}} \qquad [2.6]$$

$$\text{bias} = \frac{\bar{\hat{\mathbf{u}}}_{\text{p}} - \bar{\hat{\mathbf{u}}}_{\text{w}}}{\sigma_{\text{u}}} \qquad [2.7]$$

$$b_1 = \frac{\text{cov}\left(\hat{\mathbf{u}}_{\text{w}}, \hat{\mathbf{u}}_{\text{p}}\right)}{\text{var}\left(\hat{\mathbf{u}}_{\text{p}}\right)} \qquad [2.8]$$

where: $\hat{\mathbf{u}}_{\text{w}}$ and $\hat{\mathbf{u}}_{\text{p}}$ are the vectors of GEBV for focal animals from whole and partial datasets, respectively, $\bar{\text{F}}$ is the average inbreeding coefficient of validation animals, σ_{u}^2 (σ_{u}) is the additive genetic variance (standard deviation); and $\bar{\hat{\mathbf{u}}}_{\text{w}}$ and $\bar{\hat{\mathbf{u}}}_{\text{p}}$ are the GEBV averages from whole and partial data, respectively.

VARIANCE COMPONENT ESTIMATION

Accurately predicting breeding values is crucial for the success of animal breeding programs, as the genetic merit of the population can be heavily influenced by a few animals. Achieving high prediction accuracy involves utilizing phenotypic data from a large pool of performance-tested animals to forecast the breeding values of potential candidates for selection. Successful prediction hinges on understanding the (co)variances of random effects, including the additive genetic effect, within the mixed model equations. In practical applications, estimating these variance components becomes necessary for effective implementation (Hofer, 2011).

Restricted Maximum Likelihood (REML)

In order to estimate variance components, there are several methods, two of which are most popular and used in today's breeding programs. The first is restricted maximum likelihood (REML). REML is a modification of the maximum likelihood procedure to account for the loss in degrees of freedom from estimating fixed effects (Patterson and Thompson, 1971). As an alternative to the full likelihood, they proposed a restricted likelihood where the REML estimates are the values in the parameter space that maximize the restricted likelihood. Many different forms for writing the restricted likelihood are presented in literature (Harville, 1977; Graser et al., 1987; Meyer, 1989; Searle et al., 1992). REML is popular due to its resistance to selection bias, and estimates are always within the parameter space. The likelihood of $\boldsymbol{\beta}$ and $\mathbf{V}$:

$$\mathrm{L}(\boldsymbol{\beta}, \boldsymbol{\varphi}; \mathrm{y}) = \frac{1}{\sqrt{2\pi|\mathbf{V}|}} \mathrm{e}^{-\frac{(\mathbf{y}-\mathbf{X}\boldsymbol{\beta})'\mathbf{V}^{-1}(\mathbf{y}-\mathbf{X}\boldsymbol{\beta})}{2}} \quad [2.9]$$

Where $\boldsymbol{\varphi}$ is a vector or a matrix of variance components in V. This can be simplified to:

$$\ln\mathrm{L}(\boldsymbol{\beta}, \boldsymbol{\varphi}; \mathrm{y}) = -\frac{1}{2}\left[\ln(2\pi) + \ln|\mathbf{V}| + (\mathbf{y} - \mathbf{X}\boldsymbol{\beta})'\mathbf{V}^{-1}(\mathbf{y} - \mathbf{X}\boldsymbol{\beta})\right] \quad [2.10]$$

For mixed model equations, the log likelihood is proportional to:

$$\ln L(\boldsymbol{\varphi}; y) \propto \ln|\mathbf{R}| + \ln|\mathbf{G}| + \ln|C^*| + \mathbf{y}'\mathbf{R}^{-1}(\mathbf{y} - \mathbf{X}\widehat{\boldsymbol{\beta}} - \mathbf{Z}\widehat{\mathbf{u}}) \quad [2.11]$$

where C^* is the coefficient matrix of the MME converted to full rank; the determinant of a singular matrix is 0.

The covariance matrices G and R can be written as:

$$\mathbf{R} = \begin{bmatrix} \boldsymbol{R}_1 & 0 & . \\ 0 & \boldsymbol{R}_2 & . \\ . & . & . \end{bmatrix}, \mathbf{G} = \begin{bmatrix} \boldsymbol{G}_1 & 0 & . \\ 0 & \boldsymbol{G}_2 & . \\ . & . & . \end{bmatrix} \quad [2.12]$$

Where Ri is the residual (co)variance matrix for observation i, and **G**j is the (co)variance matrix for random effect j. The likelihood can be further simplified to

$$\ln|\mathbf{R}| = \sum \ln|\mathbf{R}_i|, \ln|\mathbf{G}| = \sum \ln|\mathbf{G}_i| \quad [2.13]$$

REML does have some disadvantages, such as its high computational cost for large models, and has been relatively difficult to program, especially for nonstandard models (Misztal, 2022).

Two algorithms are used for REML: expectation-maximization REML (EM-REML) and average-information REML (AI-REML). The EM-REML algorithm is an iterative procedure for estimating parameters that compute the expected value of the random effects given the observed data and the current estimates of the covariance parameters. Then the algorithm updates the covariance parameters using the REML method to maximize the likelihood of the observed data (Dempster et al., 1977).

The AI-REML algorithm uses the average of the second derivatives, and their expectations (average information matrix) result in the cancellation of terms that are hard to compute based on the ideas of Thompson (1971). The average information is created by averaging of information matrices, combining observed and expected information. It is manipulated to a form that is much easier to calculate than either of the two. This transformation includes creating dummy variables linked to residuals and the calculation of sums of squares and cross-products related to these

variables. Approaches relying on second derivatives may result in estimates falling outside the parameter space (Jensen et al., 1997).

Bayesian Methods via Gibbs Sampling

First implemented by Geman and Geman (1984), Gibbs sampling is a numerical integration technique and is part of the broader category of Markov chain Monte Carlo (MCMC) methods These methods entail drawing samples from defined distributions, earning the "Monte Carlo" designation, and labeling them as Markov Chains because each sample relies on the preceding one (Sorensen and Gianola, 2002). More precisely, Gibbs sampling entails iteratively drawing random samples from marginal posterior distributions by sampling from conditional posterior distributions. Therefore, conditional distributions can be generated when the joint posterior distribution is known up to proportionality. However, creating the joint density requires the application of Bayes' theorem (Mrode, 2014).

Gibbs sampling has been used for estimating variance components in animal and sire models (Wang et al., 1994c), implemented for the study of covariance components in models with maternal effects (Jensen et al., 1994), in threshold models (Sorensen et al., 1995) and in random regression models (Jamrozik and Schaeffer, 1997).

Given a multivariate animal model and proper uniform distributions are defined for the systematic effects, the prior distributions for the residual and additive genetic covariance would be:

$$\mathrm{P}(\mathbf{R}|\mathbf{V}_{\mathrm{e}}, \upsilon_e) \propto |\mathbf{R}|^{-\frac{1}{2}(\upsilon_e+p+1)} exp\,[-\frac{1}{2} tr(\mathbf{R}^{-1}\mathbf{V}_e^{-1})] \qquad [2.14]$$

$$\mathrm{P}(\mathbf{G}|\mathbf{V}_{\mathrm{u}}, \upsilon_{\mathrm{u}}) \propto |\mathbf{G}|^{-\frac{1}{2}(\upsilon_{\mathrm{u}}+\mathrm{p}+1)} \exp\,[-\frac{1}{2} \mathrm{tr}(\mathbf{G}^{-1}\mathbf{V}_{\mathrm{u}}^{-1})] \qquad [2.15]$$

where p is the order of $\mathbf{R}$ or $\mathbf{G}$, $\mathbf{V}_e$ or $\mathbf{V}_u$ is a parameter of the prior distribution and υ_e or $\upsilon_u$is the degrees of freedom. The joint posterior distributions assuming n traits and using equations 2.14 and 2.15, is:

$$\begin{aligned} &p(b_1, \ldots., b_n, u_1, \ldots., u_n, \mathbf{R}, \mathbf{G}) \\ &\propto p(y_1, \ldots., y_n | b_1, \ldots., b_n, u_1, \ldots., u_n, \mathbf{R}) p(u_1, \ldots., u_n | \mathbf{G}) p(\mathbf{G}) p(\mathbf{R}) \end{aligned} \quad [2.16]$$

From equation 2.16, the full conditional distribution of the residual variance and additive genetic variance is:

$$P(\mathbf{R}|\mathbf{b}, \mathbf{u}, \mathbf{y}) \propto P(\mathbf{R}) P(\mathbf{y}|\mathbf{b}, \mathbf{u}, \mathbf{R}) \quad [2.17]$$

$$P(\mathbf{G}|\mathbf{b}, \mathbf{u}, \mathbf{y}) \propto P(\mathbf{G}) P(\mathbf{u}|\mathbf{G}) \quad [2.18]$$

In practice, before inferences about the model's parameters are made, samples of Gibbs sampling may need an initial burn-in period along with a sample of the samples, typically obtained through thinning (Misztal, 2022).

MODELING CATEGORICAL DATA

Many traits of importance for livestock species are not continuous by nature but have a discrete phenotypic distribution. These traits focus on the prevalence and incidence of traits (Gianola, 1982). These traits include degree of calving difficulty, conformation, type scores, mortality, disease resistance, and litter size. Traits of this nature are known as threshold or quasi-continuous. They can be analyzed by postulating an underlying continuous distribution of phenotypes, which maps into the observed distribution via a set of fixed thresholds (Falconer, 1965).

Typically, these traits are either binary or ordered categorical traits meaning their responses are ordered along a gradient. For binary traits, there is only one threshold. However, for more

levels, the observed categorical responses are due to animals exceeding particular threshold levels ($\mathbf{t_i}$) of the underlying trait. Thus, with m categories of responses, there are $m - 1$ thresholds such that $t_1 < t_2 < \cdots t_{m-1}$.

For genetic analysis of categorical traits, both linear and threshold models have been used with the assumption of an underlying customarily distributed liability. Threshold models are typically more complex and require long computing times. The advantage of the linear model may be the ease of implementation; however, there have been some criticisms that some properties of BLUP do not hold with categorical traits. Fernando et al. 1983 presented a study that showed the invariance of BLUP to selection and its inability to maximize the probability of correct pairwise ranking.

Gianola (1982) has also shown that the variance of a categorical trait is a function of its expectation. This application of certain linear models results in heterogeneity of variance. Dempster and Lerner (1950) showed that the relative contribution of non-additive genetic variance to the total genetic variance in the outward scale increases heritability for binary traits. Thus, obtaining estimates of additive genetic parameters free of biases may be difficult if the original scale is used for analysis. Given that assumptions hold, evaluations based on threshold model analysis have a larger rate of response to selection than those based on linear models (Meijering and Gianola, 1985; Höschele, 1986). The advantage of threshold models increases as the incidence of the trait decreases along with lower heritability.

The computing difficulty of actual evaluations impairs the usefulness of threshold models. Programs for threshold models required equations involving many normal probability integrals, making programs challenging to write and test solutions in threshold modeling need to be obtained iteratively. A linear system of equations must be solved in each round, which may involve setting

up the coefficient matrix on disk many times and expensive computing procedures (Misztal et al., 1989). Another issue with modeling categorical data is the extreme category problem (ECP), which can cause slow or lack of convergence. The ECP occurs when all responses for a given level of a fixed effect occur in an extreme category. The solutions for these classes are ten to plus or minus infinity. Two solutions have been presented to solve this problem. The first is to treat the fixed effect as random, and the second is to remove any ECP class (Harville and Mee, 1984). Both solutions come with their issues for the final solutions of the model.

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CHAPTER 3

TEMPORAL DYNAMICS OF GENETIC PARAMETERS AND SNP EFFECTS FOR PERFORMANCE AND DISORDER TRAITS IN POULTRY UNDERGOING SELECTION[1]

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ABSTRACT

Accurate genetic parameters are crucial for predicting breeding values and selection responses in breeding programs. Genetic parameters change with selection, reducing additive genetic variance and changing genetic correlations. This study investigates the dynamic changes in genetic parameters for residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) in a broiler population that undergoes selection, both with and without the use of genomic information. Changes in SNP effects were also investigated when including genomic information. The dataset containing 200,093 phenotypes for RFI, 42,895 for BP, 203,060 for GAIN, and 63,349 for FHN was obtained from 55 mating groups (MG). The pedigree included 1,252,619 purebred broilers, of which 154,318 were genotyped with a 60K Illumina Chicken SNP BeadChip. A Bayesian approach within the GIBBSF90+ software was applied to estimate the genetic parameters for single-, two-, and four-trait models with sliding time intervals. For all models, we used genomic-based (GEN) and pedigree-based approaches (PED), meaning with or without genotypes. For GEN (PED), heritability varied from 0.19 to 0.2 (0.31 to 0.21) for RFI, 0.18 to 0.11 (0.25 to 0.14) for GAIN, 0.45 to 0.38 (0.61 to 0.47) for BP, and 0.35 to 0.24 (0.53 to 0.28) for FHN, across the intervals. Changes in genetic correlations estimated by GEN (PED) were 0.32 to 0.33 (0.12 to 0.25) for RFI to GAIN, -0.04 to -0.27 (-0.18 to -0.27) for RFI-BP, -0.04 to -0.07 (-0.02 to -0.08) for RFI-FHN, -0.04 to 0.04 (0.06 to 0.2) for GAIN-BP, -0.17 to -0.06 (-0.02 to -0.01) for GAIN-FHN, and 0.02 to 0.07 (0.06 to 0.07) for BP-FHN. Heritabilities tended to decrease over time while genetic correlations showed both increases and decreases depending on the traits. Similar to heritabilities, correlations between SNP effects

declined from 0.78 to 0.2 for RFI, 0.8 to 0.2 for GAIN, 0.73 to 0.16 for BP, and 0.71 to 0.14 for FHN over the eight intervals with genomic information, suggesting potential epistatic interactions affecting genetic trait architecture. Given rapid genetic architecture changes and differing estimates between genomic and pedigree-based approaches, using more recent data and genomic information to estimate variance components is recommended for populations undergoing genomic selection to avoid potential biases in genetic parameters.

INTRODUCTION

Accurate genetic parameters are crucial for the unbiased prediction of breeding values and selection response in breeding programs. Genetic parameters are expected to change under selection, and the magnitude of the changes depends on the intensity of selection and the initial allele frequencies (Falconer and Mackay, 1996b; Walsh and Lynch, 2018). Genetic variance is one of the critical parameters in a breeding program because it determines the potential for selection (Lush and Shultz, 1938; Falconer and Mackay, 1996b; Lynch and Walsh, 1998; Walsh and Lynch, 2018).

Additive genetic variance, and therefore, heritability, can be reduced under selection due to increased coancestry among animals (Walsh and Lynch, 2018). Another cause of reduction in the genetic variance is the Bulmer effect, which creates negative linkage disequilibrium (i.e., negative covariance between pairs of loci) under directional or stabilizing selection (Bulmer, 1971). Due to strong selection, genetic parameters may change, leading to inaccurate predictions of genetic gain when using outdated values (McMillan et al., 1995). Fluctuations in genetic parameters over time have been presented in populations under selection in chicken (Sosa-Madrid et al., 2023), in swine (Hidalgo et al., 2020), in dairy (Lawlor et al., 2002; Tsuruta et al., 2004b),

and in beef (Meyer, 2004). These changes were theorized to be caused by several potential factors such as the Bulmer effect, changes in trait definition, genetic drift, accumulation of inbreeding, or changes in selection index (Van Grevenhof et al., 2012). Thus, it is essential to update the genetic parameters regularly, especially in the genomic era, because of the faster generation turnover and increased genetic progress (Hidalgo et al., 2020). However, estimating genetic parameters over time is computationally costly with extensive data and under genomic selection because the genomic relationship matrix (**G**) is dense. According to Hidalgo et al. (2020), time intervals are helpful in studying the temporal dynamics of genetic parameters so that only a fraction of the data is utilized in each analysis. Although using time intervals is computationally advantageous, it could be sensitive to the number of records within each interval, any outliers that exist within an interval, or even the genotyping strategy of the population (Cesarani et al., 2019). It is important that intervals must be large enough with sufficient data to avoid biases (Misztal et al., 2021).

Along with estimating genetic parameters over time using intervals, the temporal changes in SNP effects can be helpful in observing the modifications in genetic interactions within a population. In single-step genomic BLUP (ssGBLUP; Aguilar et al., 2010a; Christensen and Lund, 2010), SNP effects can be obtained based on genomic estimated breeding values from a subset of randomly selected genotyped animals that represent the dimensionality of the genomic information (Bermann et al., 2022a). Accurate SNP effects are crucial for calculating reliable indirect predictions for young, genotyped animals without phenotypes or progeny (Garcia et al., 2022). In single-step SNP-BLUP (ssSNPBLUP; Fernando et al., 2014; Liu et al., 2014), SNP effects are directly estimated, then genomic EBV (GEBV) are computed as functions of those effects.

The SNP effects are expected to be stable across generations, even under strong selection, if the genetic architecture underlying the expression of phenotypes is purely additive (Bradford et al., 2017a; Wientjes et al., 2022). However, SNP effects have also been shown to change due to several factors, such as the presence of genotype by environment (GxE) interactions in layer chickens or epistatic interactions existing in the genetic architecture (Romé et al., 2015; Wientjes et al., 2022). Previous studies have advocated the recalculation of SNP effects every generation due to changes in LD or relationship structure under selection (Habier et al., 2007; Sonesson and Meuwissen, 2009; Wolc et al., 2011; Fragomeni et al., 2014).

Earlier simulation studies showed that linkage disequilibrium (LD) identified in one generation decays very slowly over generations (Meuwissen et al., 2001; Solberg et al., 2009). However, under strong selection the decay is much faster (Muir, 2007). Therefore, newer studies advocate continuous genotyping and recalculation of SNP effects (Habier et al., 2007; Sonesson and Meuwissen, 2009; Wolc et al., 2011).

The objectives of this study were to 1) estimate temporal changes in genetic parameters for efficiency, carcass yield, and leg disorder traits in a broiler population under genomic selection using time intervals including or not genomic information and 2) investigate the changes of SNP effects in a population under genomic selection.

MATERIALS AND METHODS

Animal Care and Use Committee approvals were unnecessary because data were obtained from preexisting databases.

Data

Cobb-Vantress, Inc. (Siloam Springs, AR) provided data from 55 overlapping mating groups (MG) of purebred broiler chickens. Three continuous performance traits related to feed efficiency and carcass yield, residual feed intake (RFI), gain (GAIN), and breast percentage (BP), along with one categorical leg disorder trait, femoral head necrosis (FHN), were evaluated. A total of 200,093 animals had phenotypes for RFI, 42,895 for BP, 203,060 for GAIN, and 63,349 were scored for FHN. The latter was scored from 1 (normal) to 7 (severe disorder). Of 63,349 animals, 73% were normal for FHN, 15% scored a 2, and 11% scored a 3. Scores from 4 to 7 had a very low incidence and, therefore, were grouped, resulting in a four-category trait (i.e., 1, 2, 3, 4–7). When animals had all traits missing in the dataset, these records were removed from the analysis. The pedigree included 1,252,619 purebred broilers hatched over seven years, of which 154,318 birds were genotyped with a 60K Illumina Chicken SNP BeadChip. Quality control was performed on genotypes using the PREGSF90 software (Misztal et al., 2014) and excluded duplicated genotyped individuals, and SNP call rate <0.90, SNP with minor allele frequency <0.05 or with departure from Hardy-Weinberg equilibrium >0.15 (difference between the observed and expected heterozygous frequency). SNP were removed if Mendelian conflict rate between parent-progeny pairs was >10% and progenies were eliminated if the conflict rate was >1%. Monomorphic SNP with unknown position or located at sex chromosomes were also removed. After quality control, 44,448 autosomal SNP for 154,318 birds were kept for analysis.

For estimating variance components, we defined 13 intervals: the first interval comprised seven MG, while the remaining 12 intervals were each composed of eight MG. Each subsequent interval had four overlapping MG. For example, the first interval included data from MG one to seven. The second interval included data from MG four to eleven; therefore, MG four to seven

overlapped in intervals one and two (Figure 1). Only the most recent time intervals in the dataset contained genotypes (i.e., MG 24 to 55). Genomic selection within this population started during interval 6, meaning no genomic information was available for intervals 1 through 5. Intervals including genotypes had at least 30,000 genotyped animals per interval, except for the first one, which had ~16,000 genotyped animals. The numbers of animals with genotypes, phenotypes, and pedigree per interval are presented in Table 1.

Model and Analysis

We implemented single-, two-, and four-trait analyses, with and without genomic information. Without genomics, the model of analysis was a regular pedigree-based (PED) BLUP, whereas, with genomics (GEN), single-step genomic BLUP (ssGBLUP) was used. RFI, BP, and GAIN were analyzed under the linear model. A threshold model was applied to FHN, in which a liability with a threshold is assumed, from which phenotypic values change. (Co)variance components were estimated under a Bayesian approach via the Gibbs sampling algorithm implemented in the GIBBSF90+ program (Misztal et al., 2014b; Lourenco et al., 2021). A single Gibbs chain of 100,000 samples was generated. The initial 10,000 samples were discarded as burn-in, and 1 in every ten samples was stored to compute the posterior means and standard deviations. The means were used as estimates of the variance components, and their posterior standard deviations were considered a measurement of their standard errors. Convergence was assessed graphically, by the autocorrelation of samples, and by the Geweke's diagnostic test (Geweke and In, 1995) implemented in the POSTGIBBSF90 software (Tsuruta and Misztal, 2006).

PED analyses were conducted for all intervals (1-13), but GEN analyses were only conducted for intervals that contained genomic information (6-13). Combining RFI, BP, and GAIN

into one vector of performance traits ($\mathbf{y}_p$), and FHN being in the vector of disorder traits ($\mathbf{y}_d$), the four-trait model can be expressed in matrix notation as:

$$\begin{bmatrix}\mathbf{y}_p\\ \mathbf{y}_d\end{bmatrix} = \begin{bmatrix}\mathbf{X}_p & \mathbf{0}\\ \mathbf{0} & \mathbf{X}_d\end{bmatrix}\begin{bmatrix}\mathbf{b}_p\\ \mathbf{b}_d\end{bmatrix} + \begin{bmatrix}\mathbf{Z}_p & \mathbf{0}\\ \mathbf{0} & \mathbf{Z}_d\end{bmatrix}\begin{bmatrix}\mathbf{a}_p\\ \mathbf{a}_d\end{bmatrix} + \begin{bmatrix}\mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{W}_d\end{bmatrix}\begin{bmatrix}\mathbf{0}\\ \mathbf{cg}_d\end{bmatrix} + \begin{bmatrix}\mathbf{e}_p\\ \mathbf{e}_d\end{bmatrix} \quad [3.1]$$

where **y** is the vector of observations; **b** is the vector of systematic effects (sex for all traits and contemporary group for performance traits); **a** is the vector of additive genetic effects; **cg** is the vector of contemporary group random effects only for FHN; **e** is the vector of random residual terms; **X**, **Z**, and **W** are the incidence matrices relating the elements of **y** to the elements of **b**, **a**, and **cg**, respectively. The CG was treated only as random for FHN to avoid issues created by extreme category problem (ECP) in threshold modeling (Harville and Mee, 1984; Misztal et al., 1989). Assuming multivariate normality for **y**, the covariance matrix for the random effects was given by:

$$\mathrm{Var}\begin{bmatrix}\mathbf{a}_p\\ \mathbf{a}_d\\ \mathbf{cg}_d\\ \mathbf{e}_p\\ \mathbf{e}_d\end{bmatrix} = \begin{bmatrix}\mathbf{T}\otimes\mathbf{G}_p & \mathbf{T}\otimes\mathbf{G}_{pd} & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{T}\otimes\mathbf{G}_{dp} & \mathbf{T}\sigma_d^2 & \mathbf{0} & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{I}\sigma_{cg}^2 & \mathbf{0} & \mathbf{0}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{I}\otimes\mathbf{R}_p & \mathbf{I}\otimes\mathbf{R}_{pd}\\ \mathbf{0} & \mathbf{0} & \mathbf{0} & \mathbf{I}\otimes\mathbf{R}_{pd} & \mathbf{I}\sigma_{e_d}^2\end{bmatrix} \quad [3.2]$$

where $\mathbf{G}_p$ is a 3 × 3 covariance matrix for additive genetic effects of performance traits; $\mathbf{G}_{pd}$ is a 3 × 1 vector of covariances between additive genetic effects of the performance traits and the additive genetic effects of the disorder trait; σ_d^2 is the additive genetic variance for the disorder traits; σ_{cg}^2 is the variance for the contemporary group of the disorder trait; $\mathbf{R}_p$ and $\mathbf{R}_{pd}$ contain (co)variances for the residual terms among performance and disorder traits, with dimensions defined as in their counterparts for the additive genetic effects; $\sigma_{e_d}^2$ is the residual variance for the disorder trait. **I** is an identity matrix of proper order. **T** represents the relationship matrix used according to the implemented analysis: either **A** (PED) when only pedigree information is used for

the construction of the covariance structure or **H** (GEN) when genomic and pedigree information are used in conjunction. The inverse of **H**, used in single-step is:

$$\mathbf{H}^{-1} = \mathbf{A}^{-1} + \begin{bmatrix} 0 & 0 \\ 0 & \mathbf{G}^{-1} - \mathbf{A}_{22}^{-1} \end{bmatrix} \quad [3.3]$$

where $\mathbf{A}^{-1}$ is the inverse of a pedigree-based relationship matrix for all animals included in the analysis, $\mathbf{G}^{-1}$ is the inverse of the genomic relationship matrix (Aguilar et al., 2010a), and $\mathbf{A}_{22}^{-1}$ is the inverse of the pedigree-based relationship matrix for the genotyped animals only. For the categorical trait, heritability estimates from the models including **A** or **H** were reported on the liability scale.

Calculation of SNP Effects

Breeding values were predicted using the estimated variance components for each interval, and then SNP effects were estimated by back-solving the predicted breeding values from the four-trait models. SNP effects were derived from the following equation for each interval containing genotyped animals:

$$\hat{\mathbf{a}}_j = \frac{1}{2\sum p_{ij}(1-p_{ij})} \mathbf{Z}_j' \mathbf{G}_j^{-1} \hat{\mathbf{u}}_j \quad [3.4]$$

Where $\hat{\mathbf{a}}_j$ is the vector of SNP effects in interval j, p_{ij} is the minor allele frequency of SNP i in the interval j, $\mathbf{Z}_j$ is a centered and scaled genotyped matrix, $\mathbf{G}_j^{-1}$ is the inverse of the genomic relationship matrix, and $\hat{\mathbf{u}}_j$ is the vector of breeding values for those genotyped animals. Correlations among SNP effects between each interval for each model were calculated to investigate the changes over time.

RESULTS AND DISCUSSION

Changes in variance components and calculated parameters from the four-trait model are presented from the first interval (1) through the last interval (13) for PED and from the sixth (6) interval through the last interval (13) for GEN as genomic selection did not begin in this population until interval 6. The changes in variance components for the four-trait model were similar to those found in the single- and two-trait analyses. Estimates for the last two are in supplementary material, Figures 3.S1, 3.S2, 3.S3, 3.S4, 3.S5, and 3.S6.

Heritability Estimates

The posterior means and standard deviations for heritabilities estimated by GEN and PED for the four-trait model are in Figure 2. Before genomic selection, from intervals 1 to 5, heritability estimates by PED varied from 0.31 to 0.18 for RFI, 0.25 to 0.21 for GAIN, 0.61 to 0.50 for BP, and 0.53 to 0.44 for FHN. After genomic selection began, from intervals 6 to 13, heritability estimates by GEN (PED) varied from 0.19 to 0.2 (0.21 to 0.21) for RFI, 0.18 to 0.11 (0.21 to 0.14) for GAIN, 0.45 to 0.38 (0.52 to 0.47) for BP, and 0.35 to 0.24 (0.37 to 0.28) for FHN, with a gradually declining trend for most traits.

Before genomic selection, heritability estimates showed a declining trend for all traits under PED. Once genomic selection started, estimates of heritability for RFI were stable for several intervals with an increase in heritability in the last two. Estimates for RFI were very similar between GEN and PED, with many intervals having overlapping standard deviations. Estimates between PED and GEN for GAIN had only one interval, with overlapping standard deviations with estimates from GEN being lower than those from PED. Heritabilities for BP showed fluctuations in the first three intervals after genomic selection began, followed by a decreasing trend among many remaining intervals. Under GEN, the heritability estimates after starting genomic selection

for FHN showed a declining trend. In contrast, the estimates from PED stayed stable until the last interval, which showed a decline. For both BP and FHN, estimates of heritability for GEN were mostly lower than those from PED. The declining heritability trends for BP and FHN were steeper than that of RFI and GAIN, possibly due to a greater selection intensity on these traits and the higher estimates of heritability. Walsh and Lynch (2018) stated that the stronger selection and the higher heritabilities, the greater the reduction in additive genetic variance, thus causing a greater reduction in heritability, assuming all the remaining variance components constant. The reduced additive genetic variance comes from the increased correlation between pairs of loci (Bulmer, 1971).

The range of heritabilities for RFI and GAIN was similar to those reported for feed efficiency traits in previous research. Zhang et al. (2018b) reported the heritabilities for two feed efficiency traits for a chicken population under genomic selection to be 0.22 and 0.26. Rekaya et al. (2013a) reported a heritability of 0.26 for residual feed intake in another chicken population. The range of heritabilities for BP was also in agreeance with estimates reported in poultry populations, ranging from 0.41 to 0.5 by Zhang et al. (2018b), Vanderhout et al. (2022), and Bungsrisawat et al. (2018). The heritabilities for FHN were higher before the genomic selection introduction than the heritability reported by Zhang et al. (2018) but became more similar after that.

Hidalgo et al. (2020) reported a reduction of heritability for growth traits in a pig population under genomic selection. They found that the reduction in heritability was primarily caused by the decrease in additive genetic variance and an increase in residual variance over time. Sungkhapreecha et al. (2021) found an increase in heritability in dairy cattle over time; however, this increase was most likely due to the addition of native breeds into the population, which

increased genetic diversity. Before genomic selection began in the US dairy cattle population, Tsuruta et al. (2004a) found constant heritability for productive life; this consistency was associated with additive genetic and residual variances stable over time due to no directional selection detected for this trait in the genetic trends.

Heritability changes in our study were due to the interactions between the additive genetic variances, the cg variance (for FHN), and the residual variances. The posterior means and standard deviations for additive genetic variances, cg variance (for FHN), and residual variances estimated by GEN and PED for the four-trait model are shown in Figure 3, Figure 4, and Figure 5, respectively. Before genomic selection, from intervals 1 to 5, additive genetic variances estimated by PED varied from 1175.1 to 1277.5 for RFI, 3028.80 to 2876.41 for GAIN, 1.40 to 1.54 for BP, and 1.04 to 0.84 for FHN. After genomic selection began, from intervals 6 to 13, changes in additive genetic variances estimated by GEN (PED) were 733.61 to 1246.3 (821.5 to 1279.4) for RFI, 2588.20 to 1482.10 (2957.62 to 1761.00) for GAIN, 1.30 to 1.40 (1.51 to 1.70) for BP, and 0.83 to 0.71 (0.89 to 0.85) for FHN.

Before the start of genomic selection, estimates for the additive genetic variance under PED for RFI and GAIN showed a declining trend and the estimates for BP, and FHN showed a more stable trend. After genomic selection started, estimates for RFI showed a stable trend with an increase in additive variance within the last three intervals. For GAIN, both PED and GEN showed a declining trend after the start of genomics. Additive genetic variances for both BP and FHN showed fluctuations for both GEN and PED, with the estimates at interval 6 being very similar to the final estimates at interval 13. Estimates of additive genetic variance among GEN and PED for RFI were very similar, with overlapping standard deviations just as the estimates of heritability for this trait. Just as was the case for heritability, estimates for RFI were very similar

among GEN and PED but estimates from GEN were typically lower than estimates from PED for GAIN, BP, and FHN. Before genomic selection, estimates for CG variance for FHN varied from 0.15 to 0.17 under PED. Changes in CG variance for FHN estimated by GEN (PED) after genomic selection started, from intervals 6 to 13, were 0.20 to 0.33 (0.20 to 0.32; Figure 4); however, differences between GEN and PED estimates were negligible for almost all intervals. Before genomic selection began, estimates showed a very slow increase but at the onset of genomic selection, CG variance decreased for two intervals before the trend went upward at a faster rate.

Before genomic selection, from intervals 1 to 5, residual variance estimated by PED varied from 2625.30 to 2906.90 for RFI, 8981.20 to 10870.00 for GAIN, 0.89 to 1.54 for BP, and 0.79 to 0.90 for FHN. After genomic selection began, residual variances estimated by GEN (PED) varied from 3223.9 to 5042.00 (3149.5 to 4947.00) for RFI, 11665.00 to 11486.00 (11332.00 to 11170.00) for GAIN, 1.58 to 2.25 (1.38 to 1.92) for BP, and 1.40 to 1.94 (1.36 to 1.8) for FHN (Figure 5). Estimates produced by GEN and PED were very similar with overlapping standard deviations for nearly every interval.

Under both GEN and PED, from intervals 1 to 13, estimates of residual variances showed an overall increasing trend for RFI. The reduction of heritability over time for RFI was associated with genetic and residual variances changes, with the latter contributing more to the reduction. For instance, because of a sharp increase in the residual variance, RFI heritability decreased from intervals 8 to 11, even when the genetic variance was nearly stable. This trait's additive genetic variance seemed reasonably stable over time, possibly because it has reached equilibrium. Under the infinitesimal model assumption, with an infinite population size under selection, an equilibrium is eventually achieved, and any genetic variation lost is restored by recombination (Bulmer, 1973).

A study with two traits completed by Villanueva and Kennedy (1990) showed a reduction of genetic variance and an equilibrium reached in approximately four rounds of selection.

Estimates for residual variances for GAIN prior to genomic selection showed an overall increase; however, once genomic selection started estimates from both PED and GEN showed a declining trend until interval 11 when the residual variances started to increase again. Estimates from GEN were higher than those from PED for residual variances of GAIN. A decrease in additive genetic variance and an increase in residual variance towards the later intervals were associated with the reduction of heritability in GAIN in the later intervals. The additive genetic variance steadily declined, with a variance drop directly after introducing genomic selection at interval six. Macedo et al. (2021) showed roughly a 10% steady reduction of genetic variance for milk yield in dairy sheep during 30 years of selection. Additionally, they associated a 3% reduction of genetic variance due to drift within the population. Within that dairy sheep population, the loss of genetic variance due to selection reached an asymptotic value, potentially due to the nature of the Bulmer effect or stagnancy of selection objectives. Tsuruta et al. (2021) found that heritability declined an average of 3.3% for 18 linear type traits in dairy cattle over 16 years. These authors found the total variance and permanent environmental variance increased while the additive genetic variance declined.

Genetic variances in plant species have also reduced due to selection. Allier et al. (2019) estimated that 23% of the reduction of genetic variance was due to the Bulmer effect in maize. Lara et al. (2022) reported genetic variation gradually reducing through breeding cycles from repeated rounds of selections in a simulated wheat breeding program. These authors compared the additive genetic variance between an existing selected population in linkage disequilibrium to the genetic variance in a hypothetical population under linkage equilibrium. Cesarani et al. (2020)

found that the decrease in heritability for an average daily gain trait in an Italian beef cattle breed was related to a reduction in the genetic variance and an increase in the residual variance. A similar pattern was reported by Haile-Mariam and Pryce (2015) for survival and calving interval in Australian dairy cattle.

Estimates of residual variance for BP before and after the start of genomic selection showed a steady increasing trend for both PED and GEN with estimates from GEN being higher than those from PED. Similar to BP, estimates for FHN before and after the start of genomic selection shows an overall increasing trend but with dynamic changes after the start of genomics. Estimates from GEN are higher than estimates from PED and their standard deviations are similar for several intervals, but they differentiate around interval 10. For BP and FHN, the reduction in heritability was also more associated with the increase in residual variance than changes in the additive genetic variance. For these traits, there was an increase in additive genetic variance after introducing genomic selection.

Walsh and Lynch (2018) showed that contrary to expectations of long-term response, phenotypic and additive genetic variance can increase during the course of selection, often resulting in bursts of response. One source of possible increases in genetic variance is the increase of rare favorable alleles under selection. Additive loci with favorable alleles show an increase of additive genetic variance to a peak, and then the variance decreases, a pattern we observed for BP. Fluctuation trends in additive genetic variances for both traits after starting genomic selection were mirrored by the residual variances, with the residual variance gradually increasing over time. The increase in the residual variances might be due to scaling effects. As the means of each trait changed in their respective direction for improvement, those residual variances increased. Hidalgo

et al. (2020) also reported an increase in residual variance for fitness and growth traits in a pig population under genetic selection.

Genetic Correlations and Covariances

Changes in genetic correlations prior to the start of genomic selection, from intervals 1 to 5, varied from 0.12 to 0.20 for RFI-GAIN, -0.18 to -0.06 for FE-BP, -0.02 to -0.18 for RFI-FHN, 0.06 to -0.04 for GAIN-BP, -0.02 to -0.13 for GAIN-FHN, and 0.06 to 0.06 for BP-FHN. Changes in genetic correlations after the start of genomic selection, from intervals 6 to 13, estimated by GEN (PED) were 0.32 to 0.33 (0.27 to 0.25) for RFI-GAIN, -0.04 to -0.27 (-0.05 to -0.27) for RFI-BP, -0.04 to -0.07 (-0.15 to -0.08) for RFI-FHN, -0.04 to 0.04 (0.02 to 0.20) for GAIN-BP, -0.17 to -0.06 (-0.21 to -0.01) for GAIN-FHN, and 0.02 to 0.07 (0.03 to 0.07) for BP-FHN.

Posterior means and standard deviations for genetic correlations computed by GEN and PED from four-trait models are shown in Figure 6. Before the start of genomics, from intervals 1 to 5, genetic correlations had an increasing trend for RFI-GAIN and RFI-BP, but for RFI-FHN, GAIN-BP, GAIN-FHN, and BP-FHN showed a decreasing trend. Once genomic selection began, genetic correlations for RFI-GAIN, GAIN-BP, GAIN-FHN, and BP-FHN showed slightly increasing trends, with many intervals having overlapping standard deviations from PED and GEN. For RFI-BP and RFI-FHN, the trend after the start of genomic selection increased for a couple of intervals before decreasing. Genetic correlations between PED and GEN for RFI-BP were almost completely overlapping, whereas genetic correlations between PED and GEN for RFI-FHN only had half of the intervals overlapping.

The decreasing trend for genetic correlations in later intervals could be caused by changes in the selection index for these traits. Genetic correlations for GAIN-BP and RFI-BP from intervals 4 to 8 showed some stability, with changes occurring after a few intervals of genomic selection.

Constant genetic correlations between some traits over time might be due to a stabilizing LD pattern between these traits or a balancing change of SNP correlations, resulting in the same overall genetic correlation (Walsh and Lynch, 2018). Hidalgo et al. (2020) found stable genetic correlations between a fitness and a growth trait in a swine population undergoing genomic selection. In contrast, these authors also reported that genetic correlations became more negative over time for other growth and fitness trait combinations, changing from -0.32 (2009-2011) to -0.50 (2015-2018). Cesarani et al. (2020) found genetic correlations between body size and feet and legs to change from a positive to negative or zero correlation when changing from an old dataset to a more current dataset to estimate variance components.

Looking at the components of the genetic correlation formula, changes in genetic covariances are less predictable than in variances because selection modifies the frequency of antagonistic alleles, increasing or decreasing covariances. Linkage disequilibrium can create covariances through the co-inheritance of alleles, and pleiotropy can be complementary or antagonistic when traits change in the same or opposite direction (Walsh and Lynch, 2018). Posterior means and standard deviations estimated for the additive genetic covariances from the four-trait models are shown in Figure 7 and follow a similar pattern as the genetic correlations.

Before the start of genomic selection, from intervals 1 to 5, genetic covariances varied from 224.58 to 272.59 for RFI-GAIN, -7.46 to -1.78 for RFI-BP, for -0.59 to -4.14 for RFI-FHN, 4.16 to -2.87 for GAIN-BP, -1.09 to -6.37 for GAIN-FHN, and 0.07 to 0.07 for BP-FHN. Once genomic selection began, changes in genetic covariances estimated by GEN (PED) were 437.55 to 446.05 (412.27to 381.20) for RFI to GAIN, -1.11 to -11.45 (-1.89 to -12.68) for RFI-BP, -0.10 to -2.04 (-1.01 to -2.57) for RFI-FHN, -2.06 to 1.79 (-1.20 to 11.07) for GAIN-BP, -7.82 to -1.95 (-10.75 to

-0.57) for GAIN-FHN, and 0.02 to 0.07 (0.03 to 0.09) for BP-FHN. Genetic covariances followed similar trends to that of the genetic correlations among traits.

Cesarani et al. (2020) found that genetic covariances changes occurred faster than the changes found in genetic variances. McMillan et al. (1995) stated that faster changes in genetic covariances lead to greater changes in genetic correlations. Villanueva and Kennedy (1990) reported that the change in genetic correlation between a trait under direct selection and a trait under indirect selection is maximum when its initial value is close to $\pm$ 0.6 and insignificant when its initial value is close to zero. Their findings agree with our results, as the more stable genetic correlations (GAIN-FHN, RFI-FHN, BP-FHN, and GAIN-BP) had a closer starting value to zero than the genetic correlation showing more changes (RFI-GAIN and RFI-BP).

Absolute genetic correlation values between two traits not under direct selection can either increase or decrease according to the genetic correlation sign and magnitude and heritabilities of the two traits. Strandén et al. (1993) analyzed a simulated dataset under directional selection and found that the change in the genetic correlation was always negative when both traits were under selection. Thus, selecting two traits made the traits less positively correlated if the initial correlation was positive or more negatively correlated if the initial correlation was negative. Changes in genetic covariances and correlations are important for breeding programs to consider in their selection indices to ensure direct selection on traits is not causing undesirable indirect changes on other traits of importance.

SNP effects

In this study, the correlation between SNP effects declined from 0.78 to 0.2 for RFI, 0.8 to 0.2 for GAIN, 0.73 to 0.16 for BP, and 0.71 to 0.14 for FHN over the eight intervals with genomic information. Figure 8 shows the genetic correlations between the SNP effects in intervals six

through 13 for each trait included in the four-trait analysis. The further apart the intervals are, the lower the SNP correlations between those intervals. Correlations between SNP effects from the single trait and bivariate analysis are shown in Figures 3.S7-3.S9 in the supplementary material. Correlations between SNP effects from the four-trait model followed a similar trend to the correlations from the single-trait and bivariate models. Correlations between intervals from RFI and GAIN were slightly higher than those from BP and FHN. Correlations among intervals between single- and two-trait models show negligible differences.

Legarra et al. (2021a) demonstrated that there are three main factors determining the correlation between SNP effects across generations: 1) genetic distance, 2) the magnitude of genetic interactions, and 3) the distribution of allele frequencies. The changes in correlation over intervals may be caused by one of these factors. In a simulation study, Wientjes et al. (2022) proved that the correlation between statistical additive effects decays little when only additive effects and dominance effects govern the expression of the phenotypes. However, with epistatic interactions present, the correlation decayed faster over generations of selection. This suggests epistatic interactions may exist in the genetic architecture of the population used in this study.

Correlations among SNP effects between traits changed from 0.49 to 0.42 for RFI-GAIN, -0.05 to -0.6 for RFI-BP, -011 to -0.26 for RFI-FHN, -0.05 to 0.04 for GAIN-BP, -0.34 to -0.14 for GAIN-FHN, and -0.05 to 0.22 for BP-FHN as shown in Figure 9. These changes in SNP effects were similar to the changes in genetic correlations from the four-trait model. For instance, correlations among SNP effects and genetic correlations for GAIN-BP, GAIN-FHN, and BP-FHN increased from the start of genomic selection to the last interval of information, whereas both values decreased for RFI-BP.

For RFI-GAIN and RFI-FHN, the correlations among SNP effects decreased, whereas the genetic correlations among these traits showed some stability over time. Genetic correlations are influenced by two genetic mechanisms, linkage disequilibrium and pleiotropy. Linkage disequilibrium arises because of selection, and pleiotropy is the result of an allele affecting two or more traits. Given a pair of traits, if the selection pressure is not intense on them, the generation of linkage disequilibrium will not be strong, explaining, at least partially, the stable genetic correlation. On the side of pleiotropy contribution, the lack of change in genetic correlations may be due to the number of underlying complementary and antagonistic pleiotropic alleles being roughly similar, canceling out their effects. Consequently, the net effect of pleiotropy on the genetic covariance is small or even zero, creating a phenomenon called hidden pleiotropy (Turelli, 1985) where there are pleiotropic alleles but their contribution to the genetic correlation is null (Walsh and Lynch, 2018). The latter suggests that SNP effects correlations can reveal hidden pleiotropy; however, more research is needed to test this hypothesis. Another hypothesis is related to selection again. If animals measured for traits T2 and T3 are pre-selected based on trait T1, by chance, the selected animals can be desirable for T2 and T3, then the genetic correlation between them (T2 and T3) will not change, or at least will show some stability over time. Now, if some animals selected for T1 show variation in the breeding values for T2 and T3, the genetic correlation between them will be apparently stable, whereas the allelic substitution effects will change.

Changes among correlations between SNP effects from the start of genomic selection to the last interval show that SNP effects changed over time for this population. A study by Fragomeni et al. (2014) showed three regions with a persistent percentage of variance explained by SNP (i.e., calculated based on SNP effects) for a body weight trait in a broiler breeder population across five consecutive generations. However, these authors also found that the

variances across generations changed for other regions. Wolc et al. (2012) found differing results in a layer chicken population, with consistent SNP effect estimates across six generations. The differences between the results may be due to differing genetic architecture of the traits or potentially due to the generation interval difference between broiler breeders and laying chickens, as the latter may be longer, resulting in a greater percentage of overlap across generations. Changes in SNP effects over time may also occur in the presence of genotype-by-environment interactions, as shown by Romé et al. (2015) in a layer chicken population. If linkage disequilibrium patterns and allele frequencies change over generations, using only data from recent generations may provide more accurate GEBV for current selection candidates (Wolc et al., 2016). In addition, SNP effects can be a good tool to evaluate how the population changes over time and be used as a reflection of the changes in genetic correlations.

Genomic versus pedigree-based analysis

Heritabilities estimates between GEN and PED were very similar for RFI and GAIN, whereas, for BP and FHN, heritabilities were smaller than those calculated by PED (Figure 1). A possible explanation is the varying sources of information being included in the analysis. For example, when including genomic information in the analysis when the population is under genomic selection, the analysis is better able to capture the variance in the population. Without the inclusion of all information (i.e. genomic information) that was used at the time of selection, there may be bias in the estimates of variances. Our results agree with those found in Hidalgo et al. (2020) and Sungkhapreecha et al. (2021), in which estimates from analysis with genomic information were typically smaller than those found using only pedigree-based analysis. Hidalgo et al. (2020) found less difference between genomics and pedigree-based analysis for fitness traits, but they saw more considerable differences for growth traits. Several studies looked at single-point

estimates of variance components before genomic selection was implemented, which showed varying differences between genomic and pedigree-based analysis. Agreeing with this study's results of RFI and GAIN, Forni et al. (2011) and Veerkamp et al. (2011) found similar estimates of variance components between genomic and pedigree-based analysis for several traits in a dairy population. These authors found that variance components estimated using pedigree BLUP were higher than when using genomic information whether with the genomic relation matrix (**G**) or the combined relationship matrix (**H**). Raiden et al. (2018) reported greater heritabilities in genomic analysis for adaptive growth in beef cattle, and Momen et al. (2017) found similar results in a chicken population for body weight.

Differences between GEN and PED were mostly negligible for genetic correlations between RFI-BP, GAIN-BP, GAIN-FHN, and BP-FHN. For genetic correlations between RFI-GAIN and RFI-FHN, the estimates from GEN started higher than those from PED but became similar after about four intervals of genomic selection. Sungkhapreecha et al. (2021) found slightly larger estimates in a genomic-based analysis than in a pedigree-based analysis for the genetic correlation between permanent environment and permanent environment heat tolerance effects in dairy cattle. Momen et al. (2017) found more negative correlations from genomic-based estimation between body weight and hen-housed egg production and between the latter and ultrasound area breast meat. Hidalgo et al. (2020) saw lower genetic correlations from genomic analysis for several pairwise correlations between fitness and growth traits.

One consideration when using genomic-based estimation of variance components versus pedigree-based is the genotyping strategy and genotyping proportion, primarily when ssGBLUP is used. In a broiler population, Wang et al. (2020) reported that the additive genetic variance for a body weight trait was severely overestimated when using ssGBLUP compared to pedigree-based

analysis and GBLUP. The authors concluded that this overestimation was due to the genotyping proportion and the genotyping strategy within that population. We found significant differences between estimates from GEN and PED in a preliminary study using the same body weight trait as Wang et al. (2020). Before the start of genomic selection, heritability estimates under a single-trait model varied from 0.24 to 0.20; however, once genomic selection began, changes in heritability estimates by GEN (PED) were 0.71 to 0.65 (0.27 to 0.18). Here, heritabilities from GEN were severely overestimated in the analyses with genomic information. After investigating phenotypic means and standard deviations among genotyped and non-genotyped males and females, these differences seem most likely due to the genotyping strategy adopted for females, and the genotyping proportion used in this population. Pre-selecting animals only at one tail of the phenotypic distribution for genotyping may lead to biased estimates. Thus, genotyping strategy and genotyping proportion need to be important considerations when estimating variance components with genomics under ssGBLUP to ensure no pre-selection bias.

Studies from that literature on variance components estimation have consistently shown that the most unbiased estimators are produced when all pedigree and all data on which selection decisions were based are included in the analysis. We make selection decisions based on pedigree, phenotypic, and genomic information when conducting genomic selection. Thus, all information should be available when estimating variance components to avoid bias (Jensen and Mao, 1991; Hofer, 1998; Schaeffer et al., 1998).

CONCLUSIONS

This study reinforces the importance of accounting for the temporal dynamics of genetic parameters, especially for populations under genomic selection. Genetic variances decrease over time, and residual variances tend to increase. Additionally, genetic correlations between traits and

correlations between SNP effects also present temporal variation, the latter suggesting epistatic interactions. Therefore, under a changing genetic architecture, periodically updating variance components is crucial, and the estimation process should account for all the information sources available at the selection time. As the estimates from genomic and non-genomic models differ across generations, using genomic information is recommended when estimating variance components once the population is under genomic selection. This will result in an unbiased prediction of breeding values and selection response.

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TABLES

Table 3.1. Number of genotyped, phenotyped and pedigreed animals for residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) among each interval

Interval	Pedigree[1]	Genotypes	RFI[2]	GAIN[2]	BP[2]	FHN[2]
1	118540 (40020)	0	24060 (0)	24754 (0)	5831 (0)	8586 (0)
2	138261 (46084)	0	27637 (0)	28318 (0)	6847 (0)	10103 (0)
3	135704 (45,749)	0	28532 (0)	29109 (0)	6784 (0)	9291 (0)
4	130979 (46813)	0	29157 (0)	29659 (0)	6521 (0)	10517 (0)
5	133212 (48,881)	0	29625 (0)	30080 (0)	5602 (0)	12469 (0)
6	142779 (47,859)	16514	29390 (12379)	29693 (12488)	5114 (2694)	12055 (2733)
7	138826 (46,499)	33465	29308 (24849)	29548 (25059)	5702 (5645)	10890 (5695)
8	142322 (46061)	36285	30101 (26480)	30393 (26751)	6355 (6308)	8934 (6349)
9	153943 (45760)	39395	30001 (28695)	30337 (29022)	6467 (6442)	7723 (6487)
10	149191 (45751)	40611	29918 (29844)	30274 (30199)	6377 (6355)	7503 (6396)
11	137063 (45074)	40107	29712 (29633)	30083 (30003)	6369 (6341)	7355 (6390)
12	124729 (44591)	39628	27879 (27791)	28212 (28124)	5994 (5808)	6821 (5846)
13	128498 (46,493)	41351	28862 (28769)	29156 (29063)	6147 (5948)	7042 (5990)

[1]Total number of animals in the interval (number of animals used in analysis after tracing back all animals with phenotypes or genotypes up to 3 generations of their ancestors).
[2]Number of animals with phenotypes (number of genotyped animals with phenotypes)

FIGURES

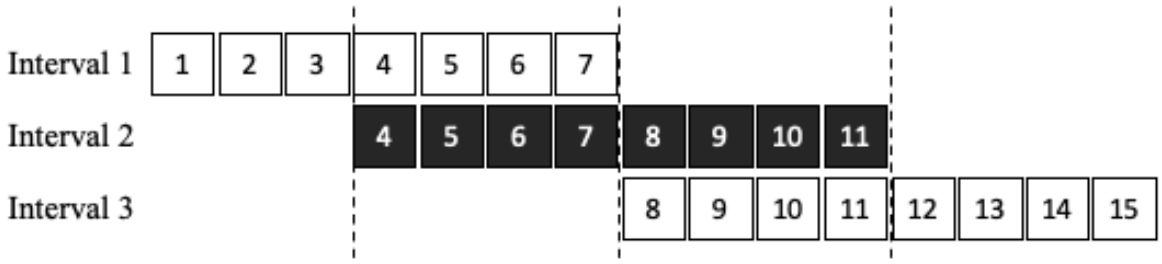

Figure 3.1. Illustration showing how the first three intervals were composed of mating groups.

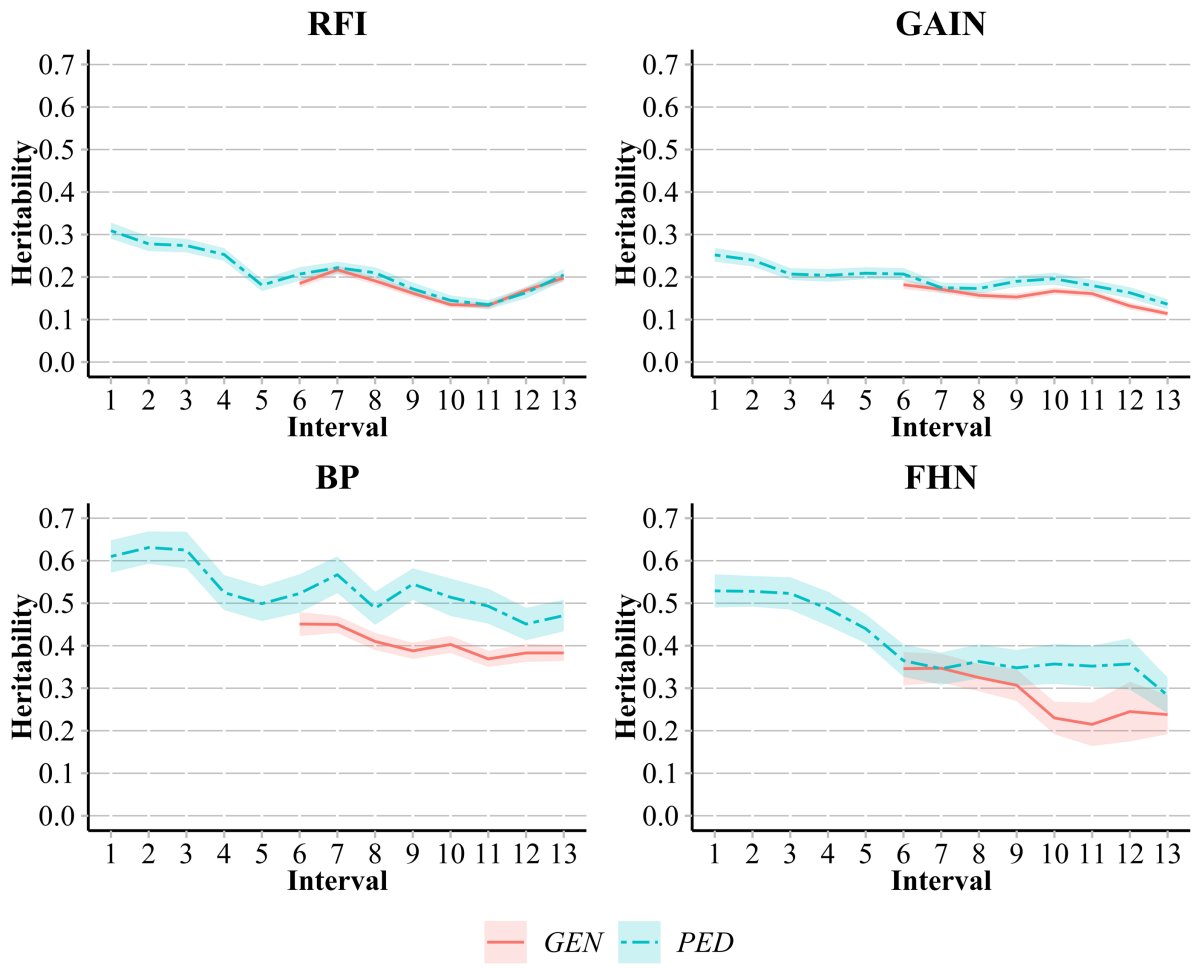

Figure 3.2. Posterior means and standard deviations for heritabilities from residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) in four-trait models.

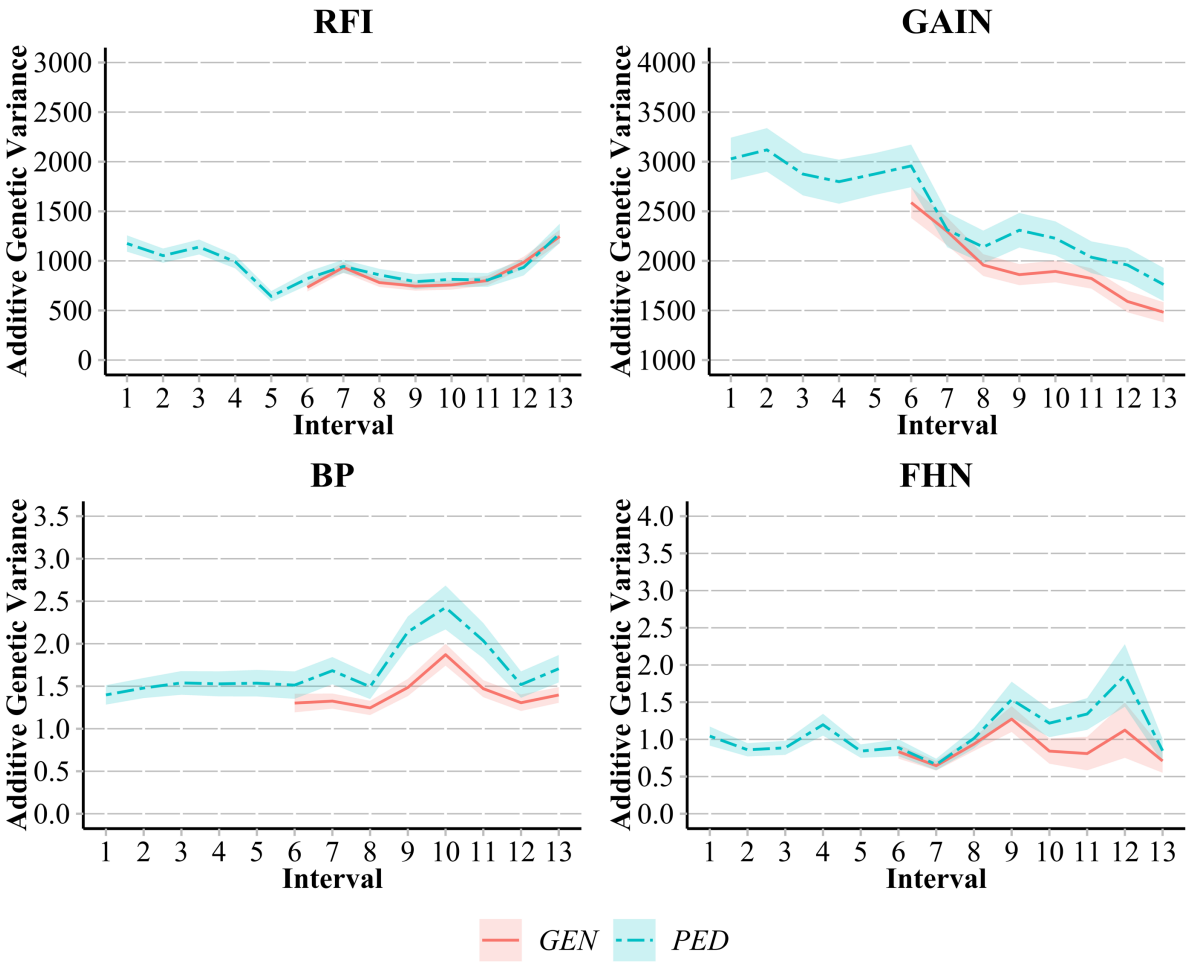

Figure 3.3. Posterior means and standard deviations for additive genetic from residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) in four-trait models.

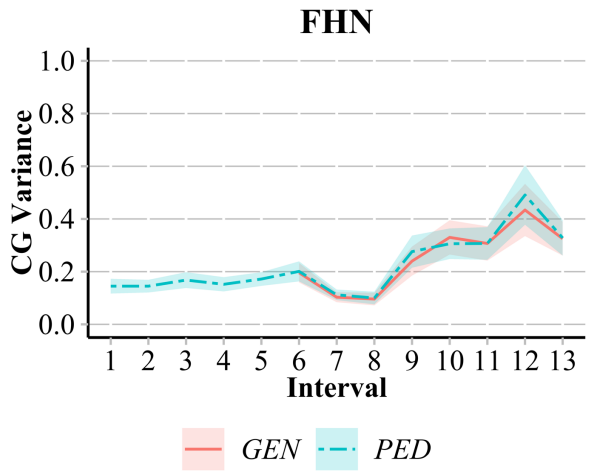

Figure 3.4. Posterior means and standard deviations for contemporary group (CG) of femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) in four-trait models.

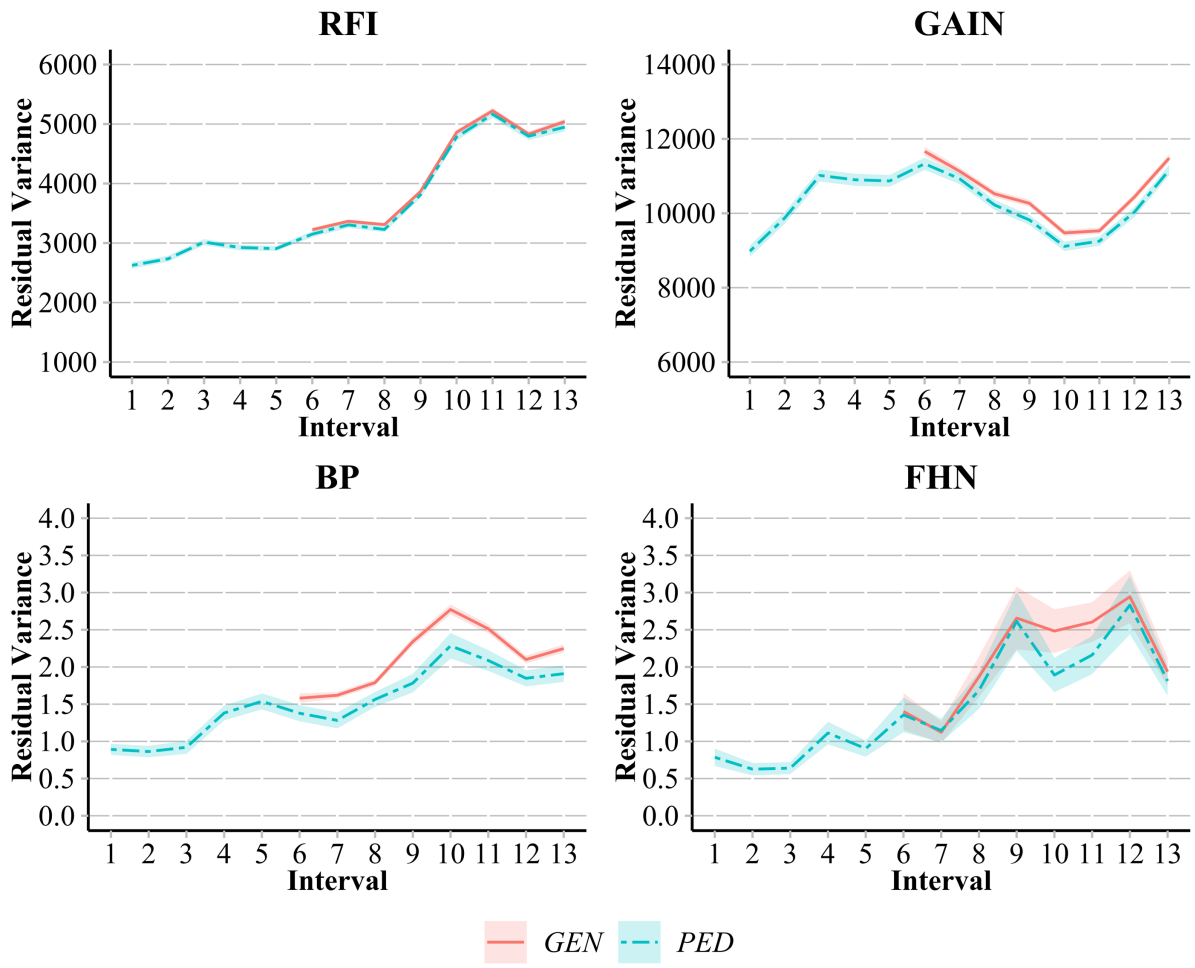

Figure 3.5. Posterior means and standard deviations for residual variances from residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) in four-trait models.

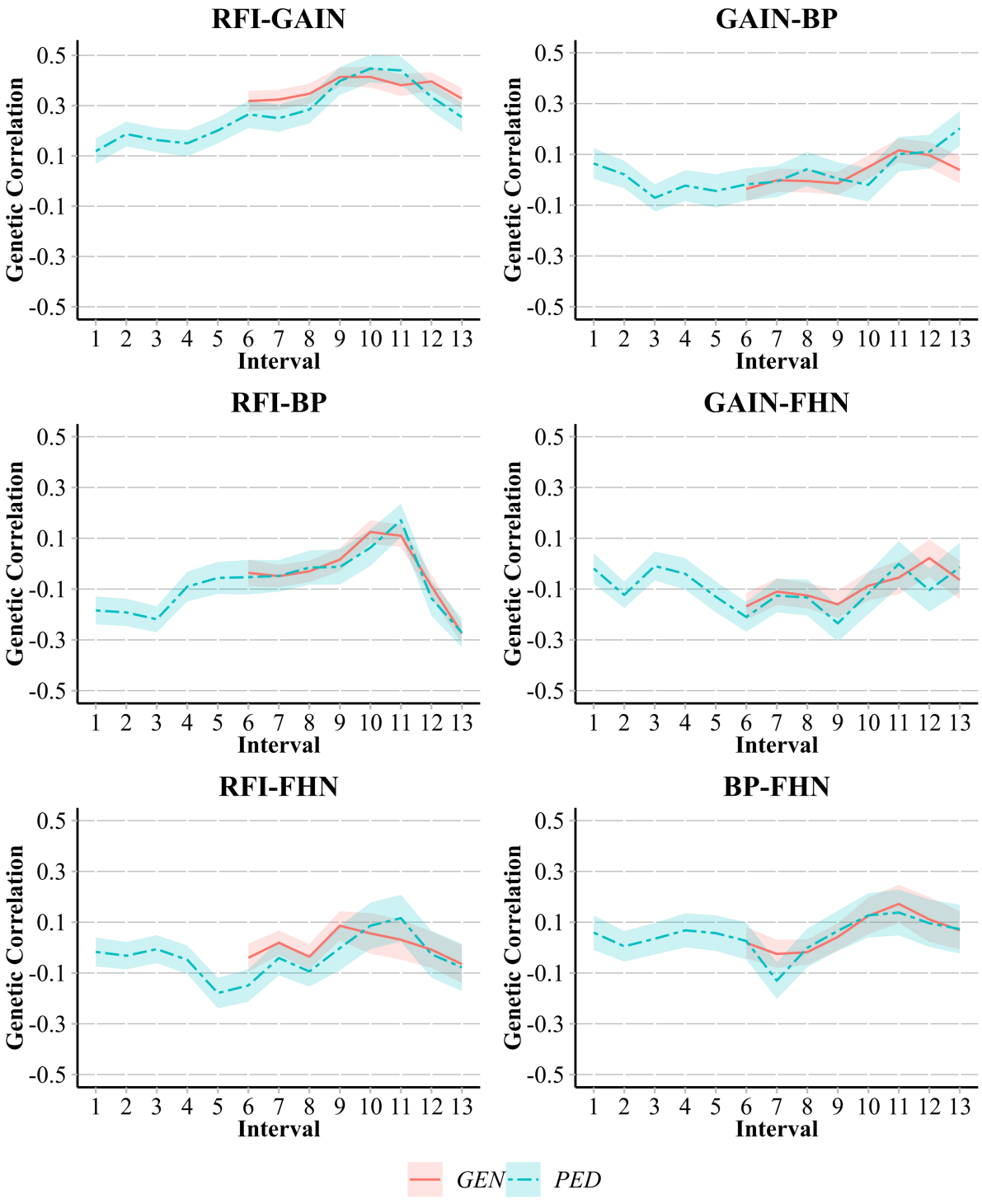

Figure 3.6. Posterior means and standard deviations for genetic correlations among residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) in four-trait models.

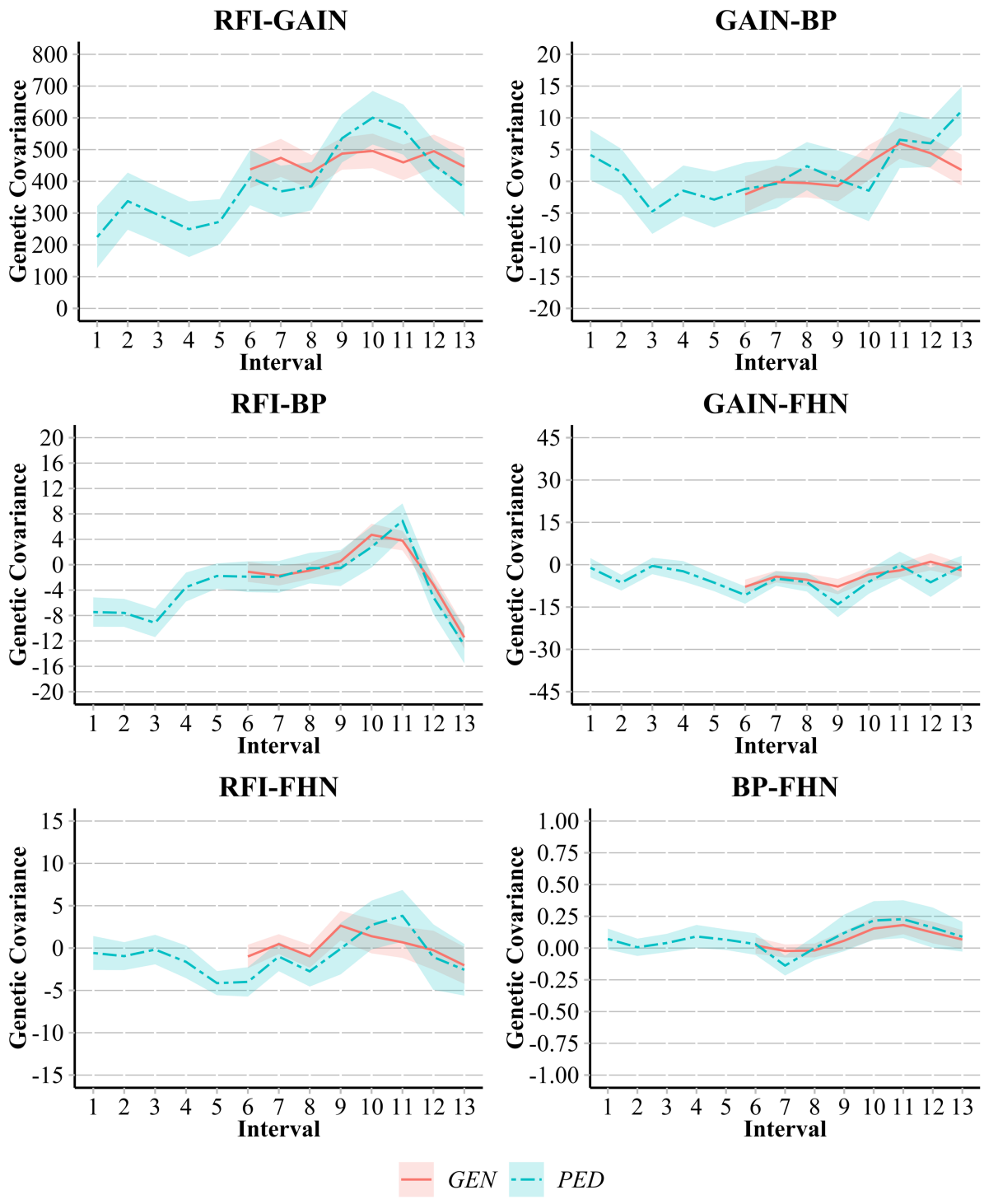

Figure 3.7. Posterior means and standard deviations for genetic covariances among for residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) in four-trait models.

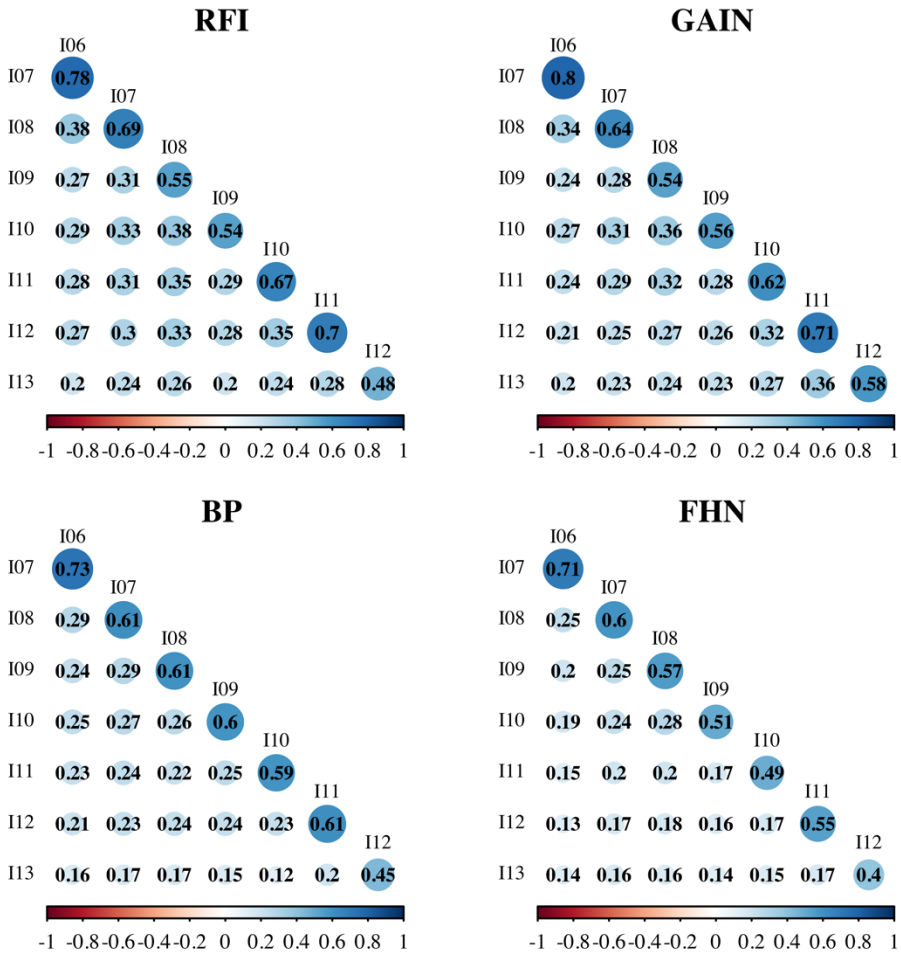

Figure 3.8. Correlation plots between SNP marker effects from interval 6-13 (intervals with animals that have genotypes) from each of the traits in the four-trait model.

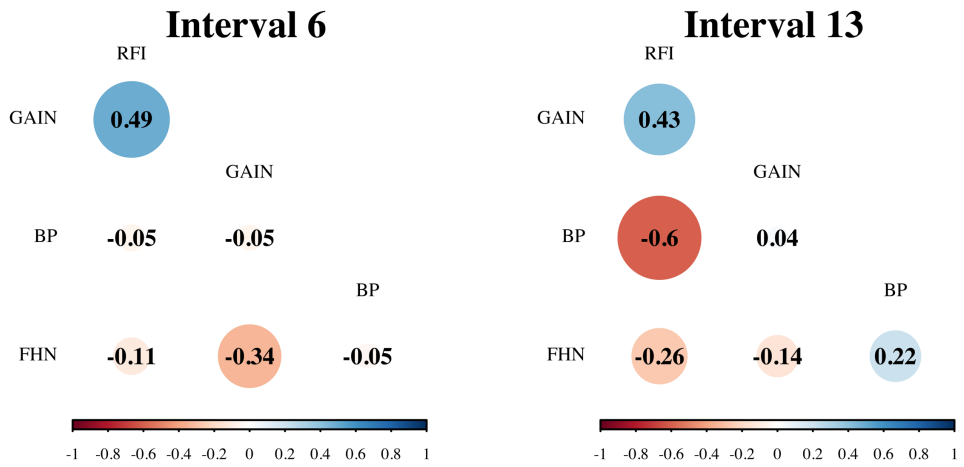

Figure 3.9. Correlation plots between SNP marker effects between traits for interval 6 (first interval of genomic selection) and interval 13 (last interval of genomic selection) from the four-trait model.

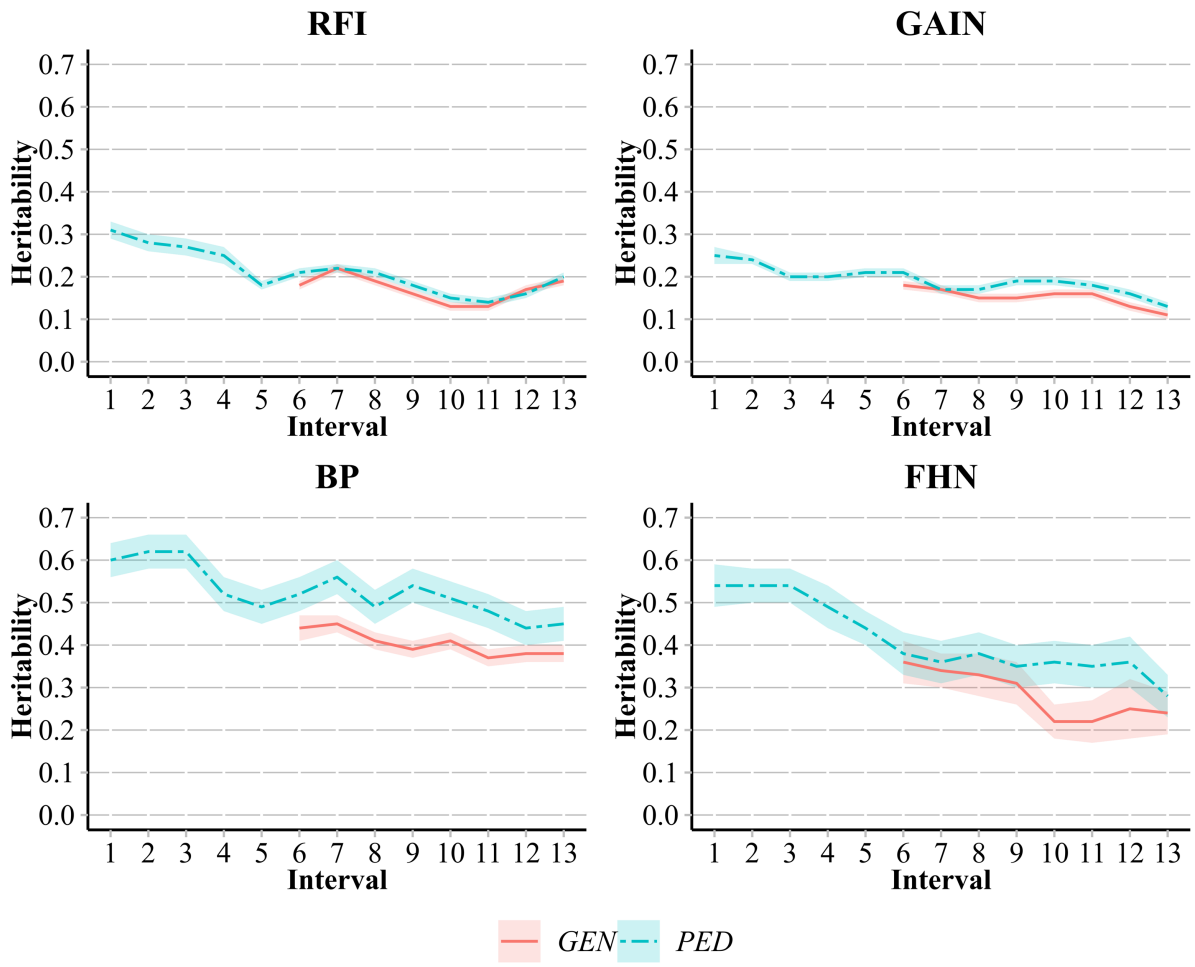

Figure 3.S1. Posterior means and standard deviations for heritability from residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) using single-trait models.

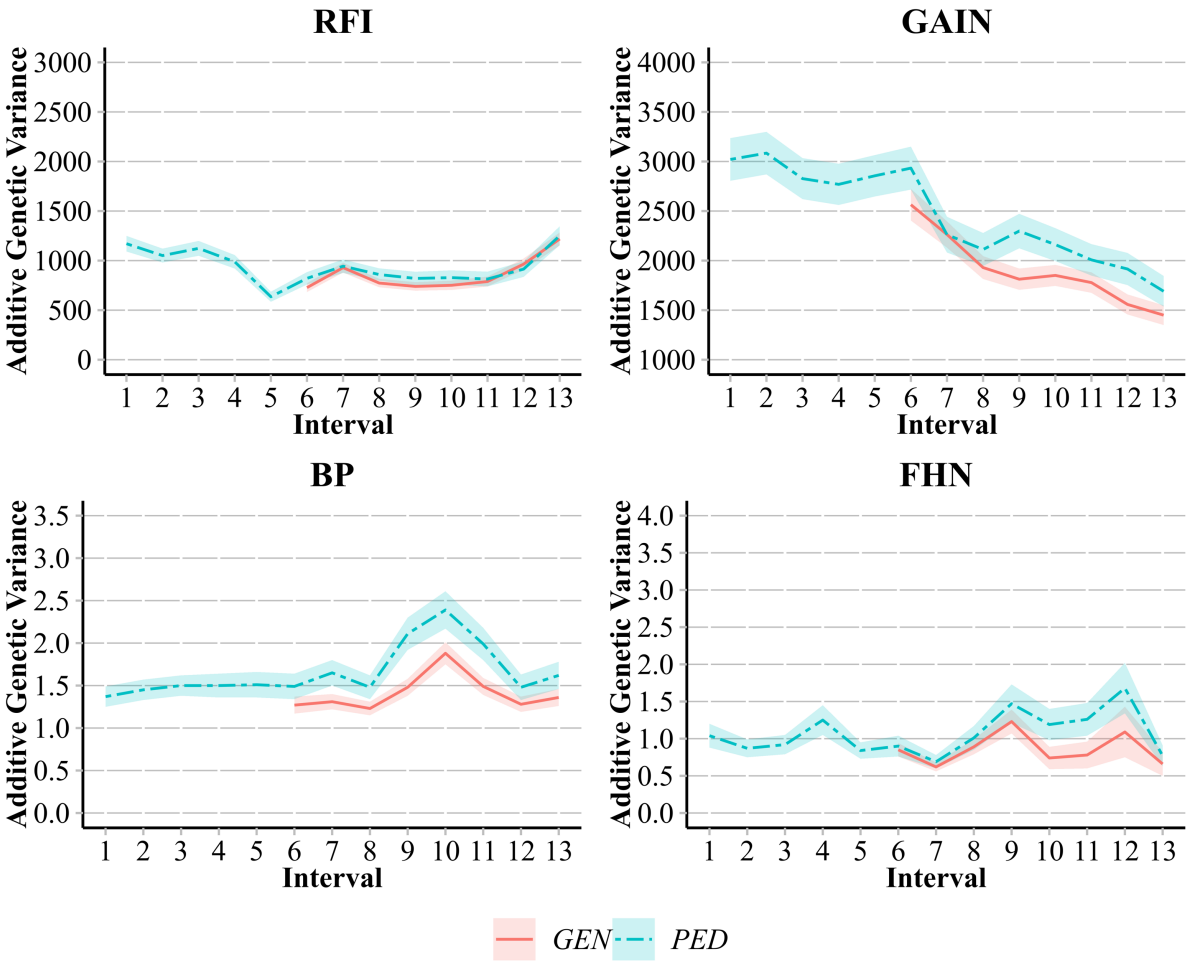

Figure 3.S2. Posterior means and standard deviations for additive genetic variances from residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) using single-trait models.

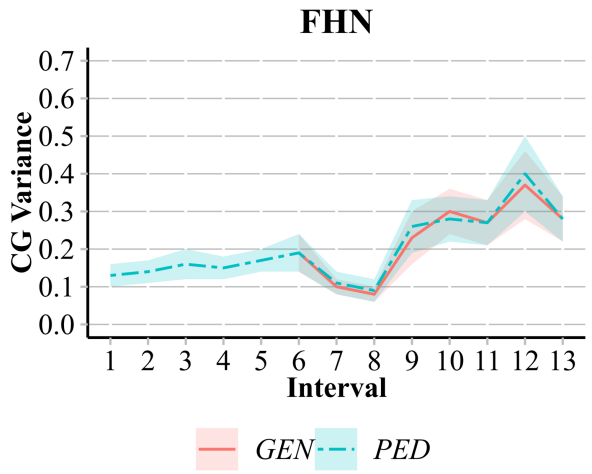

Figure 3.S3. Posterior means and standard deviations for contemporary group (CG) from femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) using single-trait models.

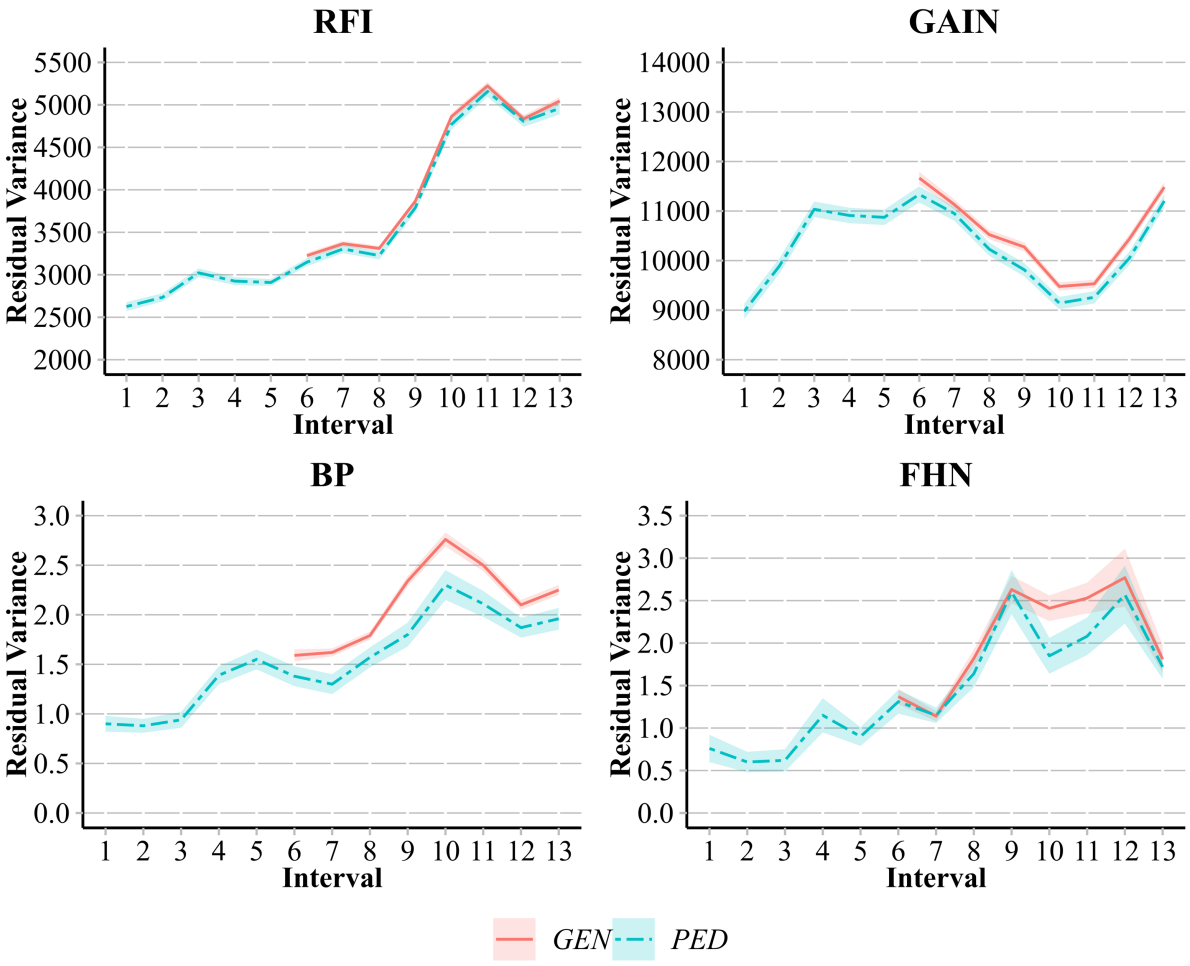

Figure 3.S4. Posterior means and standard deviations for residual variances from residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) using single-trait models.

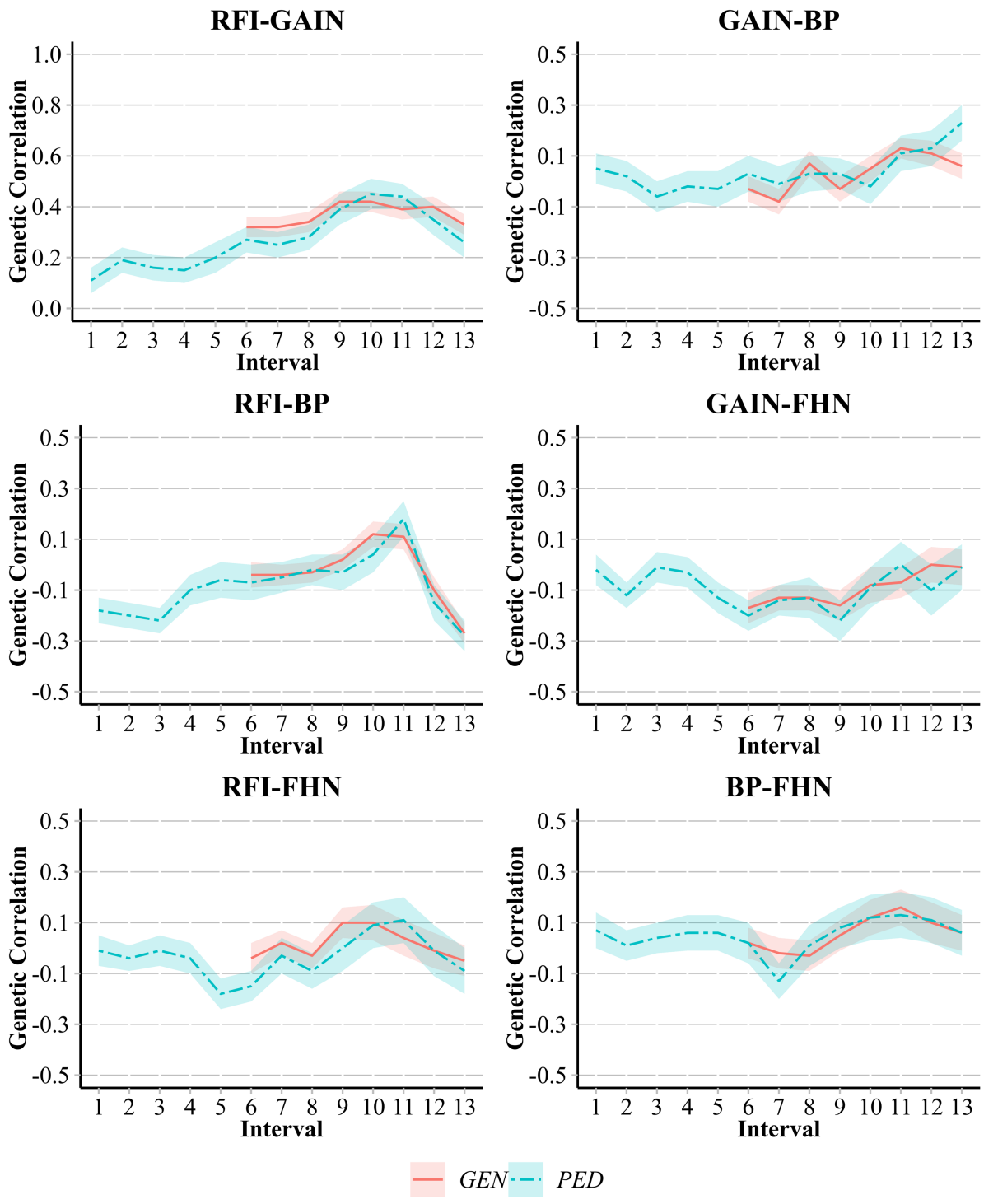

Figure 3.S5. Posterior means and standard deviations for genetic correlations among residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) using the two-trait models.

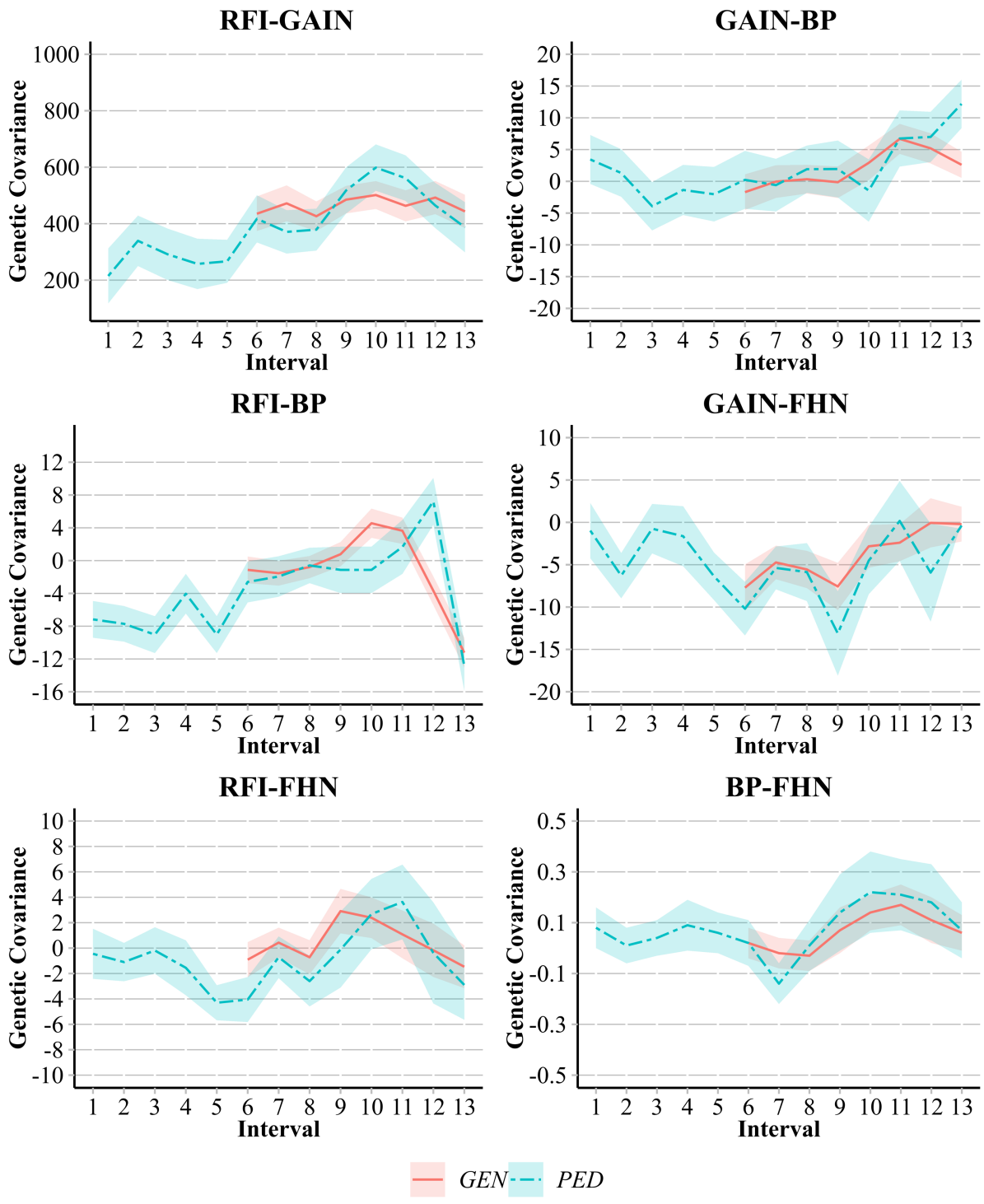

Figure 3.S6. Posterior means and standard deviations for genetic covariances among residual feed intake (RFI), gain (GAIN), breast percentage (BP), and femoral head necrosis (FHN) estimated with or without genotypes (GEN or PED) using the two-trait models.

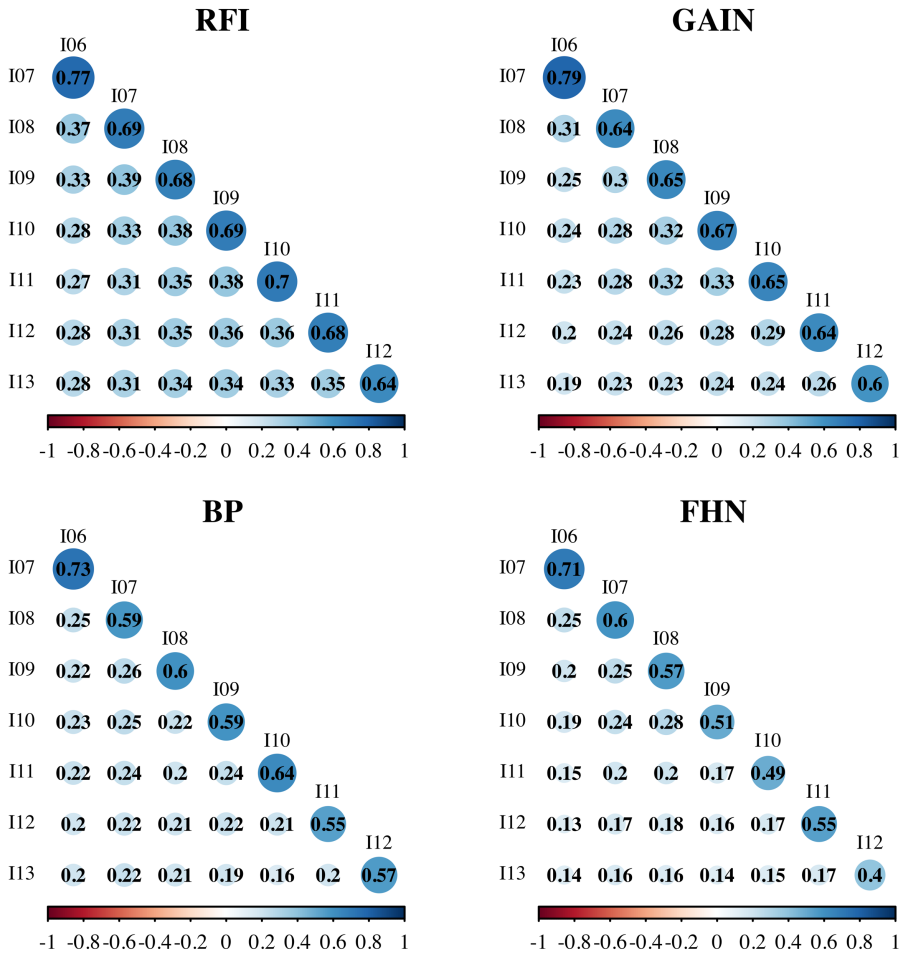

Figure 3.S7. Correlation plots between SNP marker effects from interval 6-13 (intervals with animals that have genotypes) from the single-trait models.

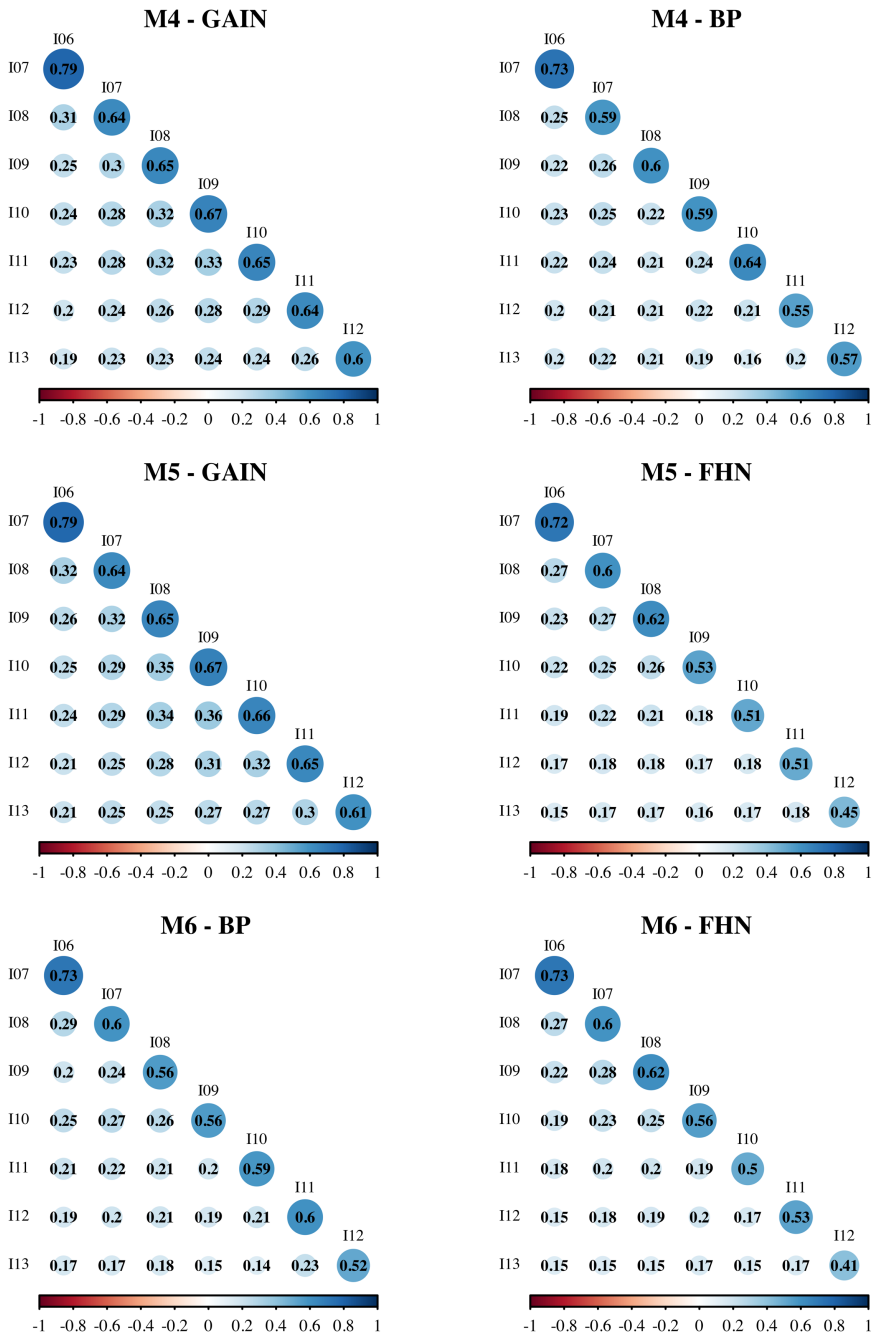

Figure 3.S8. Correlation plots between SNP marker effects from interval 6-13 (intervals with animals that have genotypes) from the bivariate models: Model 1 (M1) – RFI and GAIN, Model 2 (M2) – RFI and BP, and Model 3 (M3) – RFI and FHN.

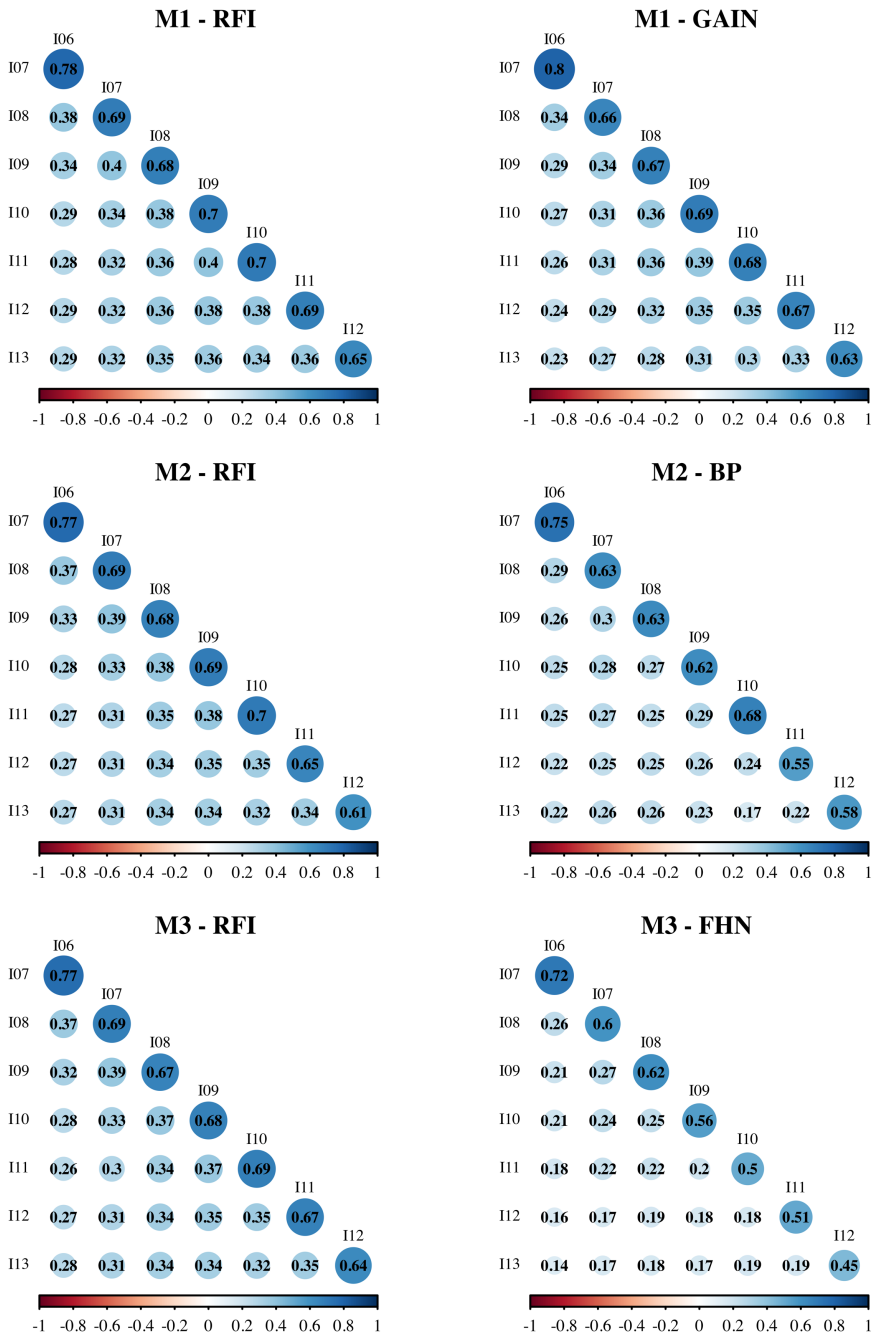

Figure 3.S9. Correlation plots between SNP marker effects from interval 6-13 (intervals with animals that have genotypes) from the bivariate models: Model 4 (M4) – GAIN and BP, Model 5 (M5) – GAIN and FHN, and Model 6 (M6) – BP and FHN.

CHAPTER 4

REVIEWING THE TRAIT DEFINITION IN BROILER CHICKENS AND ITS IMPLICATIONS IN GENOMIC EVALUATIONS[2]

[2] Jennifer Richter, Fernando Bussiman, Jorge Hidalgo, Vivian Breen, Ignacy Misztal, Daniela Lourenco. Submitted to Journal of Animal Science.

ABSTRACT

Mortality is an economically important trait usually handled as a binary outcome from hatch time until selection in most broiler breeder programs. However, in other species, it has been shown that not only does the genetic component change over time, but there are maternal genetic effects to be considered when mortality is recorded early in life. This study aimed to investigate alternative trait definitions of mortality with varying models and effects. Approximately three years' worth of data were provided by Cobb-Vantress, Inc. and included two mortality traits. The first trait was binary, whether the bird died or not (OM), and the second mortality trait was a categorical weekly mortality trait (WM), with the phenotype being the week the bird died. After data cleaning, six weeks of data for the two given mortality traits were used to develop five additional trait definitions. The definitions were broiler mortality (BM), early and late mortality (EM & LM), and two traits with repeated records as cumulative or binary (CM and RM, respectively). Variance components were estimated using linear and threshold models to investigate whether either model had a benefit. The variance components from the threshold models were then transformed into the linear scale. Genomic breeding values were predicted using the BLUPf90 software suite, and linear regression validation (LR) was used to compare trait definitions and models. Heritability estimates ranged from 0.01 to 0.15 under linear and 0.04 to 0.21 under threshold models, indicating genetic variability within the population across these trait definitions. The genetic correlation between EM and LM ranged from 0.48 to 0.81, indicating they have divergent genetic backgrounds and should be considered different traits. The LR accuracies showed that EM and LM used together in a two-trait model have comparable accuracies to that of

OM while giving a more precise picture of mortality. When including the maternal effect, the direct heritability considerably decreased for EM, indicating that the maternal effect plays an important role in early mortality. Therefore, a suitable approach would be a model with EM and LM while considering the maternal effect for EM. Single nucleotide polymorphism effects were estimated under each trait definition and model, and no individual SNP explained more than 1% of the additive genetic variance. Additionally, the SNP with the largest effect size and variance were inconsistent across trait definitions. Chicken mortality can be defined in different ways, and reviewing these definitions and models may benefit poultry breeding programs.

INTRODUCTION

Genetic selection in broiler chickens has revolutionized growth, enabling them to attain market weight swiftly (Havenstein et al., 2003; Zuidhof et al., 2014). However, this rapid growth is coupled with significant mortality concerns (Meseret, 2016). Modern commercial strains of broiler chickens often struggle to perform natural behaviors and may experience lameness, worsening with their increasingly body weight (Bassler et al., 2013). The accelerated growth rate is directly linked to cardiovascular diseases, leg disorders, bone deformities, and other issues resulting in mortality and, thus, economic losses in the industry (Kalmar et al., 2013; Zhang et al., 2018a; Hartcher and Lum, 2020).

Mortality is influenced by genetic factors but also heavily influenced by environmental factors. Long et al. (2007) reported that most of the variability in an early mortality study (0 – 14 days) between chicks was due to non-identifiable environmental factors (e.g., residual effect). Other studies have shown that first-week mortality is higher when personnel walk through the house more quickly (Cransberg et al., 2000). In contrast, others have shown that leg health is one

of the most prevalent causes of culling and late mortality during the grow out of heavy broilers (Cransberg et al., 2000; Oviedo-Rondón, 2008). The literature has shown that mortality has a low heritability, making it a trait that takes several generations and lots of data to show a significant response to selection (González-Recio et al., 2008; Zhang et al., 2018c; Bermann et al., 2021a). Additionally, when working with mortality, extra complexity comes from the fact that there are many reasons for death.

Maternal genetics may also be an important factor to consider when assessing early mortality. In pigs, for example, the maternal effect has been shown to be equally, if not more influential, than the direct effect (Leite et al., 2021). Working with chickens, Zhang et al. (2018c) showed that the additive maternal variance was not negligible for growth traits. At the commercial level, growth is measured early in life; hence, maternal effects play a role early in a chick's life. Although the impact of maternal effect has been investigated for broiler growth, its influence on mortality remains to be observed.

In broilers, survival is typically measured as a binary response, whether the bird is alive (0) or dead (1). The evaluation of mortality is commonly performed with linear models with the assumption of a continuous distribution for phenotypes. However, it has been presented that in evaluations of categorical traits, it is more suitable to use non-linear models, such as threshold models, to better accommodate the natural distribution of the binary responses (Gianola, 1982). Using genomic information, Leite et al. (2021) successfully applied threshold models to evaluate pig survival in different production phases. Nonetheless, with the growing amount of genomic information used in evaluations, threshold models have been reported to have convergence and computing-time issues, primarily due to the non-linearity of the set of equations involved.

Using genomic information allows not only the identification of variants/genes associated with the trait but also helps understand the underlying variability of the traits. The changes in single-nucleotide polymorphisms (SNP) effects can help infer the genetic dynamics of different trait definitions within a population. This study aimed to assess alternative trait definitions of mortality as well as the inclusion of maternal genetic effects for genomic evaluations of mortality in a broiler population. Furthermore, we investigated linear and threshold models for variance component estimation and breeding value prediction. Lastly, we explored the genetic architecture of these trait definitions by examining the behavior of SNP effects and how the SNPs influenced the additive genetic effect.

MATERIALS AND METHODS

Animal Care and Use Committee approvals were unnecessary because data were obtained from preexisting databases.

Data

Cobb-Vantress, Inc. (Siloam Springs, AR) provided data for three lines of broiler breeder containing approximately three years' worth of mortality records. Among the datasets, two distinct mortality traits were available: a binary mortality trait (OM) and a categorical weekly mortality trait (WM). The OM was assigned a value of 1 for birds that were alive and 2 for those that were deceased, recorded from hatching until the first selection point. The WM was recorded during the initial ten weeks after hatching, with the bird's phenotype indicating the week of death. If a bird survived beyond these ten weeks, no phenotype was assigned.

To explore alternative trait definitions and models, a new mortality phenotype was created using OM and WM. Using information from OM, phenotypes for animals that survived past ten

weeks were generated. Inconsistent records between OM and WM were resolved by assuming OM to be the gold standard since it had been accepted in previous studies (Zhang et al., 2018c; Bermann et al., 2021a). Any remaining inconsistent records were removed from the dataset. Birds that received a WM record of 0 were considered missing because the week of death was unknown. Due to the limited number of records in weeks seven, eight, nine, and ten, they were combined with week six, such that the phenotype of week six indicated birds that survived up to week 6. After data editing, the remaining records covered weeks one to six. The pedigrees contained at least 245,000 individuals, with at least 77,000 genotyped individuals. Those individuals were genotyped with a 60K Illumina Chicken SNP BeadChip. Furthermore, genotyping was limited to animals selected after the initial selection date, resulting in all genotyped animals having a corresponding phenotypic record. After quality control, 54,305 SNP were retained for line 1 (L1), 42,156 SNP remained for line 2 (L2), and 54,518 SNP were kept for line 3 (L3). A summary of the original data is in Table 1.

Trait definitions

The first trait definition, termed "broiler mortality" (BM), was analogous to OM. In this approach, a binary phenotype was created whether the bird survived (1) or died (2) within the first six weeks of life. The subsequent trait definitions involved dividing the six weeks into two distinct periods: "early mortality" (EM), encompassing the first three weeks, and "late mortality" (LM), covering the last three weeks. Once again, both EM and LM were represented as binary phenotypes, receiving 1 if the birds survived or 2 otherwise, within three (EM) or six (LM) weeks.

Based on WM two other approaches were formulated. The first followed a cumulative strategy (CM), resulting in six repeated records for each animal. Starting at 1 (first week), every week the animal survived, the phenotype increased by one; if the animal was dead, the phenotypic

record remained the same until the sixth week of life. For example, if death occurred in week one, the animal would receive the phenotypic record 1 for all six weeks, but if it survived week one and died at week two its records would be all 2. Using the same reasoning, if the animal survived week two, its phenotype would change to 3 and either remain constant (death at week 3) or be increased by one every week, meaning it survived up to six weeks. The second approach treated weekly mortality as a binary, repeated (RM) measurement. Once again, using 1 (survived) or 2 (died) for every week, but contrary to CM, no additional record was attributed when the animal died. For example, if a bird was dead at week three, it would have three repeated records, being 1 for the first two weeks and 2 for the third. A summary of the new trait definitions is in Table 2.

Variance components estimation

Variance components were estimated for all different trait definitions using either a linear or a threshold model without including genomic information. Three different models were implemented:

$$\mathbf{y} = \mathbf{X\beta} + \mathbf{Zu} + \mathbf{e} \quad [4.1]$$

$$\mathbf{y} = \mathbf{X\beta} + \mathbf{Z}_1\mathbf{u} + \mathbf{Z}_2\mathbf{p} + \mathbf{e} \quad [4.2]$$

$$\mathbf{y} = \mathbf{X\beta} + \mathbf{Z}_1\mathbf{u}_\mathrm{d} + \mathbf{Z}_3\mathbf{u}_\mathrm{m} + \mathbf{e} \quad [4.3]$$

Where: $\mathbf{y}$ is the vector of phenotypic records (OM, WM, and BM in eq. [4.1]; CM and RM in eq. [4.2]; EM and LM in eq. [4.3]); $\boldsymbol{\beta}$ is the vector of fixed/systematic effects (contemporary group and/or age), assumed as $\boldsymbol{\beta}\sim\mathrm{N}\{\mathbf{0}, \mathbf{I}\sigma^2_\beta\}$ (only for threshold models); $\mathbf{u}$ is the vector of animal additive genetic random effects ($\mathbf{u}\sim\mathrm{N}\{\mathbf{0}, \mathbf{A}\sigma^2_\mathrm{u}\}$); $\mathbf{p}$ is the vector of permanent environmental random effects ($\mathbf{p}\sim\mathrm{N}\{\mathbf{0}, \mathbf{I}\sigma^2_\mathrm{p}\}$ and $\mathrm{Cov}(\mathbf{u}, \mathbf{p}') = \mathbf{0}$); $\mathbf{u}_\mathrm{d}$ and $\mathbf{u}_\mathrm{m}$ are the vectors of additive direct and additive maternal random effects, respectively assumed as $\mathbf{u}_\mathrm{d}\sim\mathrm{N}\{\mathbf{0}, \mathbf{A}\sigma^2_{\mathrm{u_d}}\}$ and $\mathbf{u}_\mathrm{m}\sim\mathrm{N}\{\mathbf{0}, \mathbf{A}\sigma^2_{\mathrm{u_m}}\}$ ($\mathrm{Cov}(\mathbf{u}_\mathrm{d}, \mathbf{u}_\mathrm{m}') = \mathbf{A}\sigma_{\mathrm{u_d u_m}}$); $\mathbf{e}$ is the vector of random residuals ($\mathbf{e}\sim\mathrm{N}\{\mathbf{0}, \mathbf{I}\sigma^2_\mathrm{e}\}$); $\mathbf{X}$

and $\mathbf{Z}_i$ are incidence matrices for fixed/systematic and random effects, respectively. Single-trait models were implemented for OM, WM, BM, CM, and RM, whereas a two-trait model (with maternal genetic effects) was implemented for EM and LM.

Variance components were estimated using BLUPF90+ (linear model) and GIBBSF90+ (threshold model) software (Misztal et al., 2014a). Since Bayesian inference was used for the threshold models, a single-chain of 300,000 samples was generated, assuming a burn-in of 100,000 and thinning interval of 100. Thus, inferences about the variance components were made over 2,000 samples from the posterior distribution. Convergence evidence was assessed graphically and by applying Geweke's diagnostic test (Geweke and In, 1995), implemented in the POSTGIBBSF90 software (Tsuruta and Misztal, 2006). Variance components from threshold models were transformed from the liability to the observed scale following Dempster and Lerner (1950) and Gianola (1979).

Prediction of breeding values

Breeding values were predicted through ssGBLUP employing iteration on data with preconditioned conjugate gradient using the BLUPIOD2OMP1 software from the BLUPF90 suite of programs (Misztal et al., 2014a). Due to the number of genotyped animals, APY (Misztal et al. (2014a)) was used to calculate the inverse of genomic relationship matrix (**G**). The number of core animals was defined as the number of eigenvalues explaining at least 98% of the variation in **G** (Pocrnic et al., 2016a). For L1, L2, and L3, the number of core animals was 5800, 6100, and 9700, respectively. Core animals were kept consistent throughout each model's prediction of breeding values. The implemented statistical models were as before, except for replacing $\mathbf{A}^{-1}$ by $\mathbf{H}^{-1}$ (Aguilar et al., 2010a), the inverse of the realized relationship matrix in ssGBLUP.

Estimation of SNP effects

Using ssGBLUP with APY, GEBV were predicted for each trait definition with each model. Then, SNP effects were estimated by backsolving GEBV for each model according to Bermann et al., (2022) taking advantage of the equivalence between APY ssSNP-BLUP and APY ssGBLUP:

$$\hat{\mathbf{a}} = \frac{1}{2\sum_{i=1}^{m} p_i(1-p_i)} \mathbf{Z_c}'\mathbf{G_{cc}^{-1}}\widehat{\mathbf{u_c}} \qquad [4.4]$$

Where: $\hat{\mathbf{a}}$ is the vector of SNP effects, p_i is the allelic frequency of the i^{th} SNP, $\mathbf{Z_c}$ is a centered SNP content matrix for core animals, $\mathbf{G_{cc}^{-1}}$ is the inverse of the genomic relationship matrix among core animals, and $\widehat{\mathbf{u}}_\mathbf{c}$ is the vector of GEBV for core animals. Additionally, the percentage of additive genetic variance explained by each SNP was also calculated, as well as the p-value associated with each SNP following Bermann et al. (2022b) and Leite et al. (2023).

Validation of genomic predictions

The linear regression (LR) method (Legarra and Reverter, 2018) was employed to assess the performance of each model and trait definition with respect to OM. This approach compares genetic evaluations, including whole and partial datasets, based on differences in means and covariances using a set of focal individuals. The number of focal animals changed from line to line since they were selected as young genotyped animals in the partial data. Generally, 1,914 (L1), 709 (L2), and 4,376 (L3) animals were used for validation. The partial dataset was created by removing phenotypes from the focal animals, their siblings, and contemporaries. Let the partial dataset be denoted by subscript p, while the whole dataset is denoted by subscript w, then validation statistics are:

$$\text{acc} = \sqrt{\frac{\text{cov}(\hat{\mathbf{u}}_w, \hat{\mathbf{u}}_p)}{(1-\bar{F})\sigma_u^2}} \quad [4.5]$$

$$\text{bias} = \frac{\bar{\hat{\mathbf{u}}}_p - \bar{\hat{\mathbf{u}}}_w}{\sigma_u} \quad [4.6]$$

$$b_1 = \frac{\text{cov}(\hat{\mathbf{u}}_w, \hat{\mathbf{u}}_p)}{\text{var}(\hat{\mathbf{u}}_p)} \quad [4.7]$$

Where: $\hat{\mathbf{u}}_w$ and $\hat{\mathbf{u}}_p$ are the vectors of GEBV for focal animals from whole and partial datasets, respectively, $\bar{F}$ is the average inbreeding coefficient of validation animals, σ_u^2 is the additive genetic variance; and $\bar{\hat{\mathbf{u}}}_w$ and $\bar{\hat{\mathbf{u}}}_p$ are the GEBV averages from whole and partial data, respectively.

RESULTS AND DISCUSSION

Variance components and genetic parameters

Posterior means and standard deviations of heritability estimates from threshold and linear models are presented in Tables 3 and 4, respectively. Direct heritabilities ranged from 0.01 to 0.16 for OM, WM, BM, CM, and RM in linear models and from 0.11 to 0.21 for OM, WM, and BM in threshold models, indicating a low but existing genetic variability across trait definitions. Threshold models produced higher estimates of heritability in the liability scale, but when converted to the observed scale, variances were reduced, and heritability estimates were also reduced almost to the same value as the linear model. The heritability estimates for WM under the linear models are much larger than those for the other trait definitions. This larger heritability could be due to the structure of WM, where there are several more levels of responses, thus approaching normality. The heritability estimates for OM and BM were very similar across lines, most likely due to OM and BM being very similar traits. Heritability estimates for the CM and RM were consistently slightly lower than OM, WM, and BM, with RM having lower estimates than CM.

When comparing overall estimates across lines, L2 showed higher heritability estimates among these trait definitions than L1 and L3, with L3 consistently having lower estimates. This may suggest the environmental factors have a more considerable impact on mortality for L3.

Zhang et al. (2018c) reported heritability for a general chicken mortality trait to be 0.14 on the liability scale using a threshold model with sex as a systematic effect and contemporary group as random effect. Bermann et al. (2021a) also reported heritability for mortality to be 0.14 on the liability scale using threshold models. In a small experimental group of chickens, Pakdel et al. (2005b) reported heritability of 0.32 for a total mortality trait that spanned the first seven weeks of the bird's life. These results suggest changes in heritability are expected from population to population due to size, genetic makeup, number of records, different statistical models, and, in the case of categorical traits, the incidence levels.

Early and late mortality

For lines L1 and L3, heritabilities for EM were slightly lower than for LM whereas for L2, LM was lower than EM. This was true for both linear and threshold models. González-Recio et al. (2008) reported a heritability of 0.02 from linear models for a "late mortality" trait (14-42 days of age) in chickens. The authors stated that it seems unlikely that the true value of this parameter is higher than 0.05 due to the amplitude of their posterior intervals. Our results from the linear models agree with those from González-Recio et al. (2008), most likely because when modeling a binary trait as a linear output, a small variance is captured by the model. Gianola (1982) also stated that discrete traits, when analyzed as continuous, may have underestimated heritabilities, especially when incidences are low. However, other studies found ascites-related mortality, heart-failure mortality, and heart-lung failure mortality to have higher heritabilities ranging from 0.06 to 0.15 (de Greef et al., 2001). These conditions contributing to mortality are typically found to cause

issues later during the chicken grow out period. Therefore, there may be more prominent underlying genetic factors for conditions leading to mortality during the later periods of grow-out, making heritabilities larger. Another reason may be caused by how their definition of mortality was developed in that the heart-lung failure mortality encompassed that of the other two mortality traits.

Genetic correlations between EM and LM in the two-trait models are in Table 5. The values ranged from 0.48 to 0.81 in the linear model (among the three lines) and from 0.52 to 0.64 in the threshold model. These genetic correlations suggest that EM and LM may be considered different traits. In other species, such as pigs, it has been shown that pre-weaning and post-weaning mortality are two genetically different traits with low correlations and sometimes even divergent. Leite et al. (2021) reported a strong, positive correlation of 0.83 between nursery and finishing survival traits, while Dufrasne et al. (2014) reported a moderately positive correlation of 0.59. Due to the lower correlations in the latter, mortality at different phases was considered different traits, and both were included in the evaluation. It may also be necessary to include both traits in the selection index for poultry to make better-informed decisions.

When including the maternal genetic effect into the linear model with EM and LM, heritability estimates for the direct effects of EM decreased by 50% for L1, 88% for L2, and 60% for L3, whereas the decrease for LM was 33% for L1 and L2, and no change was observed for L3. Under the threshold model, including the maternal genetic effect caused a decrease of 60% for L1, 81% for L2, and 50% for L3 for EM. Meanwhile, for LM, the direct heritability decreased only by 27% for L1 and 33% for L2 and increased by 7% for L3. Maternal heritability estimates for EM (EM_m) were 0.01 (L1), 0.02 (L2), and 0.01 (L3) for the linear approach, and 0.04 (L1), 0.06 (L2), 0.03 (L3), for the threshold approach. As for LM maternal effects (LM_m), those accounted for 0.01

(L1) and 0.00 (L2 and L3) of the total variance for the linear models and 0.02 (L1) and 0.01 (L2 and L3) for the threshold models.

Grandinson et al. (2005) reported a low maternal heritability for pre-weaning mortality (0.02), whereas Lund et al. (2002) reported a maternal heritability of 0.08 under linear models. Leite et al. (2021) reported maternal heritability from threshold models for piglet farrowing survival and piglet lactation survival to be 0.09 and 0.08, respectively, thus indicating that both piglet and sow genetics impact piglet survival. These authors also found that the maternal heritability remained constant over time. However, the piglet additive genetic variance increased as the piglet aged, suggesting that the importance of the piglet's own genes increases as they age. Our results agree with studies mentioned above on including the maternal genetic effects. Adding the maternal genetic effect reduced the direct heritability by an average of 0.04 for EM and 0.01 for LM under linear models (across all lines), with a larger average reduction of 0.10 for EM and 0.03 for LM under threshold models. Including the maternal genetic effect considerably decreased direct heritability for EM but not for LM under linear and threshold models, indicating the maternal effect plays a more important role earlier in the chick's life and should be considered in the models.

Validation of genomic predictions

Validation statistics from the LR method are presented in Figure 1 for all trait definitions except for RM and CM. Both CM and RM had comparable accuracy to other trait definitions, but the bias average standard deviation for the repeatability models was over eight-fold larger than the average of the other trait definitions. With this bias, it was determined that the repeatability models were not viable, and thus, their results were not shown. Compared to the baseline scenario from the linear model for L1 (OM – 0.43), the accuracy increased by 12% for WM (0.48), 2% for BM

(0.44), and for LM_d (0.46), except for EM, LM, and EM_d. The accuracy remained constant between OM and LM, decreasing by 12% for EM and EM_d (0.38 and 0.38).

For L2, compared to the baseline linear scenario (OM – 0.53), the accuracy decreased by 66% for WM (0.18), 34% for BM (0.35), 28% for EM (0.38), 19% for LM (0.43), 32% for EM_d (0.36), and 13% for LM_d (0.46). Following a similar trend as L2, in L3, the comparison between the baseline linear scenario (OM – 0.39), the accuracy only increased by 5% for LM (0.41). The accuracy decreased by 28% for WM (0.28), 8% for BM (0.36), 13% for EM (0.34), 15% for EM_d (0.33), and 3% for LM_d (0.39). When using linear models, OM generally produced an overall better accuracy than any of the other trait definitions explored across the three lines of data.

When comparing the baseline linear scenario from the linear model to that of the threshold model, the accuracy increased by 2% for L1 (0.45 vs. 0.46) but then decreased by 6% for L2 (0.53 vs. 0.50) and by 10% for L3 (0.39 vs. 0.35). When comparing the linear baseline to the remaining threshold model trait definitions for L1, the accuracy increased by 9% for WM (0.47), by 5% for BM (0.45), by 18% for LM (0.87), and by 26% for LM_d (0.54). The accuracy decreased by 12% for EM (0.38) and by 7% for EM_d (0.40) for L1. For L2, the comparison between the linear baseline and the threshold trait definitions showed a decrease in accuracy for all trait definitions except for LM_d, which showed an increase of 6% (0.56). The remaining trait definitions showed a decrease by 9% for WM (0.48), by 28% for BM (0.38), by 36% for EM (0.34), by 9% for LM (0.48), and by 23% for EM_d (0.41) for L2. Furthermore, in L3, the comparison between baseline linear OM with all remaining threshold trait definitions, revealed an increase in accuracy by 51% for WM (0.59), by 3% for BM (0.40), by 28% for LM (0.50), and by 21% for LM_d (0.47). The accuracy decreased by 13% for both EM (0.34) and EM_d (0.34).

Consistently, the accuracy for EM and EM_d was lower than the other trait definitions and OM. This lower accuracy may be due to EM containing less information than the other trait definitions since it only contained records from the first three weeks of the bird's life. The changes in accuracy were line-specific, probably because of the amount of information available, genetic architecture of the trait, selection intensity, and/or the genotyping strategy within each line. The accuracies from the threshold models were generally better than those from linear models, indicating that threshold models might be more appropriate when modeling binary chicken mortality. By decomposing mortality in EM and LM, part of the accuracy is lost for EM but regained and improved for LM. Thus, considering EM and LM in a two-trait model will be comparable, if not better, than OM. This is also true when including the maternal effect into the model with EM and LM as the accuracies further increased.

The estimated bias for all trait definitions was ranged from -0.10 to 0.10. Among trait definitions, the bias ranged from -0.05 to 0.01 for L1, -0.06 to 0.07 for L2, and 0.00 to 0.05 for L3 among the linear models. Among the threshold models, the bias ranged from -0.03 to 0.01 for L1, -0.06 to 0.09 for L2, and -0.01 to 0.06 for L3. On average, L3 had the largest bias compared to L2, which had the lowest bias. Bias estimates were comparable from linear and threshold models with similar trends across trait definitions; however, specific trends of bias were not consistent across lines. Predictions were mostly over dispersed for all trait definitions, with a b_1 ranging from 0.81 to 0.94 for L1, 0.76 to 0.95 for L2, and 0.77 to 0.86 for L3 for linear models. For the threshold models, dispersion estimates ranged from 0.86 to 1.02 for L1, 0.81 to 1.01 for L2, and 0.79 to 0.88 for L3. Bias and dispersion estimates were not consistent across lines, and thus, once again, these results are line specific and cannot be generalized across these lines.

Rank Correlations

Spearman's rank correlations were calculated between OM and the remaining trait definitions from linear models and threshold models (Figure 2). Line 2 had an overall smaller reranking compared to L1 and L3. Rank correlations from the linear models indicated a smaller reranking for BM, EM, LM, and EM_m across all three lines, with values above 0.70. A larger reranking existed between OM and WM and LM_d. For all three lines, the correlation between WM and the rest of the trait definitions ranged from small negative to small positive values, centering around zero, indicating that the ranking for WM is not the same as the original mortality as well as the remaining trait definitions.

Rank correlations from threshold models followed a similar trend to linear models where values indicated a smaller reranking for BM, EM, LM, and EM_m and a larger reranking for WM and LM_d across all three lines. For L2 and L3 from threshold models, rank correlations between WM and other trait definitions were mostly smaller except for EM and LM_d. Across all lines and models, rank correlation values from LM_d with all other traits, evidencing that when the maternal effect is modeled with LM, there is a much larger reranking of individuals. For a breeding program, it is important to be aware of how changes in modeling may affect the ranking of selection candidates. These correlations show that for most trait definitions, reranking should be expected.

Detection of SNPs associated with mortality

Each trait definition and model were used to perform a genome-wide association study (GWAS) to investigate the underlying complex genetic architecture of mortality. Our GWAS resulted in no SNP having significant associations (p-value > 0.05/number of SNP in each line) with any trait definitions from all available datasets. These results indicate that for these data, there are no major regions or genes greatly influencing mortality under these trait definitions. For all

traits, no SNP explained more than 1% of the additive genetic variance, with the largest peak explaining not more than 0.20%. One peak with a linkage disequilibrium trail was consistent among OM, EM_d, and LM_d. However, this peak still did not explain more than 1% of the additive genetic variance. Most peaks for OM, EM_d, and LM_d are not consistent across models. As dead animals were not genotyped in these populations, the results are one-sided in that we only have genomic information of the animals that survived. When considering the SNP effects, the magnitude was different when comparing OM to EM, as shown in Figure 3. This difference is most likely caused by changes in the GEBV distributions, as shown in Figure 4. When comparing OM to LM, the magnitude of SNP effects is more similar, just as the distribution of GEBV is more similar for these trait definitions.

Tsuruta et al. (2017) reported SNP variance peaks that reached 2.5% for mortality around the location of the DGAT1 gene on BTA 14, which is also associated with milk production in dairy cattle. These results indicate that for this population, there is a relationship between cow mortality and milk production. However, Berry et al. (2010) reported no effect of DGAT1 on dairy cow longevity, which is related to with mortality. In pigs, Guo et al. (2016) reported that QTL regions identified for early mortality overlapped with the regions associated with the total number born alive. Fragomeni et al. (2014) found that the variation explained by the top SNP windows in a chicken population changed over generations, indicating that SNP with large variance detected in one dataset may not be detected in the following generations. Thus, the usefulness of those SNP over time is limited. As our peaks differ depending on the trait definition, working with mortality as a single trait may not be optimal. Instead, splitting up mortality into time periods may help get a clearer understanding of the underlying genetic architecture of the different phases of mortality.

Practical Implications

Overall, mortality is a complex trait to select against because of many differing factors, both genetic and environmental. As this trait has genetic variability, sustained improvement is possible if it is included in the index as a long-term breeding goal. By using alternative trait definitions for mortality, small increases in the accuracy may be achieved, allowing for improvements at the phenotypic level. Reverter et al. (2022). showed that even small changes in accuracy of breeding values can have a significant impact on phenotypic changes. As shown in this study, using threshold models allows for higher estimates of accuracy, indicating that by using this modeling strategy, there could be better improvements if the costs of using threshold models are feasible. Other methods of modeling mortality, such as survival models, have been shown to add no real benefits compared to animal models (Poulsen et al., 2022). Thus, further investigation of mortality models is needed to continue to make progress on this trait.

CONCLUSIONS

Diversifying trait definitions offers a promising approach to enhance the accuracy of predicting mortality GEBV and refine selection strategies for future generations of broiler breeders. Our exploration and testing of various trait definitions advocate for segmenting mortality into distinct time phases to consider the varying genetic influences on survival. Notably, incorporating maternal effects, especially during the early stages of a chick's life when the maternal genetic impact is significant, holds the potential to improve the understanding of mortality mechanisms. This study highlights the suboptimal nature of treating mortality as a simple binary trait, clearly demonstrated by the fluctuations in variance explained by SNP based on different trait definitions as well as the fluctuations of additive genetic variances reflected by the different

heritability estimates. Seeking better ways to model mortality will undoubtedly advance our understanding of its underlying genetic architecture and enable more effective breeding practices to improve overall broiler breeder survival, performance, and welfare.

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TABLES

Table 4.1: Number of records, animals in the pedigree, and genotyped animals for the three chicken lines provided for this study

Line	Pedigree	Genotype	OM[1]	WM[1]
1	353,293	100,881	322,039	20,136
2	291,836	80,283	264,607	33,214
3	245,506	77,099	223,882	35,019

[1] Number of records for mortality (OM) and weekly mortality (WM)

Table 4.2: Number of records for each new trait definition used in the analysis

Line	BM	EM	LM	CM[1]	RM[1]
1	321,458	321,458	311,279	1,928,749	1,874,786
2	264,457	264,457	247,493	1,586,743	1,502,638
3	223,882	223,882	207,729	1,343,017	1,267,782

[1] Number of records for broiler mortality (BM), early mortality (EM), late mortality (LM), cumulative repeated records (CM), and binary repeated records (RM).

Table 4.3: Estimates of heritabilities and standard deviations from threshold models

Line	Model	h^2_d	h^2_m
1	OM[a]	0.11 ± 0.01	-
	WM[a]	0.13 ± 0.02	-
	BM[a]	0.11 ± 0.01	-
	EM[b]	0.10 ± 0.01	-
	LM[b]	0.15 ± 0.01	-
	EM[c]	0.04 ± 0.01	0.04 ± 0.01
	LM[c]	0.11 ± 0.01	0.02 ± 0.00
2	OM[a]	0.19 ± 0.01	-
	WM[a]	0.18 ± 0.02	-
	BM[a]	0.19 ± 0.01	-
	EM[b]	0.21 ± 0.01	-
	LM[b]	0.18 ± 0.01	-
	EM[c]	0.04 ± 0.01	0.06 ± 0.01
	LM[c]	0.12 ± 0.01	0.01 ± 0.00
3	OM[a]	0.13 ± 0.01	-
	WM[a]	0.12 ± 0.02	-
	BM[a]	0.13 ± 0.01	-
	EM[b]	0.12 ± 0.01	-
	LM[b]	0.15 ± 0.01	-
	EM[c]	0.06 ± 0.01	0.03 ± 0.01
	LM[c]	0.16 ± 0.02	0.01 ± 0.00

[1]h^2_d direct heritability; h^2_m maternal heritability

[a]Single-trait models for mortality (OM), weekly mortality (WM), broiler mortality (BM)

[b]Two-trait models for early (EM) and late mortality (LM)

[c]Two-trait model with maternal genetic effect for early (EM) and late mortality (LM) with EM_d and LM_d being the additive direct effect and EM_m and LM_m being the maternal genetic effect

Table 4.4: Estimates of heritabilities and standard deviations from linear models

Line	Model	h^2_d	h^2_m
1	OM[a]	0.03 ± 0.00	-
	WM[a]	0.15 ± 0.01	-
	BM[a]	0.03 ± 0.00	-
	CM[a]	0.02 ± 0.00	-
	RM[a]	0.01 ± 0.00	-
	EM[b]	0.02 ± 0.01	-
	LM[b]	0.03 ± 0.01	-
	EM[c]	0.01 ± 0.00	0.01 ± 0.00
	LM[c]	0.02 ± 0.00	0.01 ± 0.00
2	OM[a]	0.08 ± 0.00	-
	WM[a]	0.16 ± 0.01	-
	BM[a]	0.09 ± 0.00	-
	CM[a]	0.08 ± 0.01	-
	RM[a]	0.07 ± 0.01	-
	EM[b]	0.08 ± 0.01	-
	LM[b]	0.06 ± 0.01	-
	EM[c]	0.01 ± 0.00	0.02 ± 0.00
	LM[c]	0.04 ± 0.00	0.00 ± 0.00
3	OM[a]	0.06 ± 0.00	-
	WM[a]	0.12 ± 0.01	-
	BM[a]	0.05 ± 0.00	-
	CM[a]	0.04 ± 0.01	-
	RM[a]	0.01 ± 0.01	-
	EM[b]	0.05 ± 0.01	-
	LM[b]	0.06 ± 0.01	-
	EM[c]	0.02 ± 0.00	0.01 ± 0.00
	LM[c]	0.06 ± 0.01	0.00 ± 0.00

[1]h^2_d direct heritability; h^2_m maternal heritability

[a]Single-trait models for mortality (OM), weekly mortality (WM), broiler mortality (BM), cumulative repeated approach (CM), binary repeated approach (RM)

[b]Two-trait models for early (EM) and late mortality (LM)

[c]Two-trait model with maternal genetic effect for early (EM) and late mortality (LM) with EM_d and LM_d being the additive direct effect and EM_m and LM_m being the maternal genetic effect

Table 4.5: Genetic correlations between early and late mortality from two-trait models for all three chicken lines

	Correlation[a]	L1	L2	L3
Linear	$r_{EM,LM}$	0.81 ± 0.00	0.52 ± 0.00	0.48 ± 0.00
	r_{EM_d,LM_d}	0.25 ± 0.02	0.17 ± 0.03	0.24 ± 0.02
	r_{EM_d,EM_m}	-0.01 ± 0.02	0.01 ± 0.04	-0.02 ± 0.02
	r_{EM_d,LM_m}	0.01 ± 0.03	0.02 ± 0.02	-0.04 ± 0.02
	r_{EM_m,LM_d}	0.00 ± 0.04	0.04 ± 0.04	0.01 ± 0.03
	r_{LM_m,LM_d}	-0.01 ± 0.05	0.07 ± 0.03	-0.06 ± 0.03
	r_{EM_m,LM_m}	0.40 ± 0.02	0.22 ± 0.05	0.08 ± 0.10
Threshold	$r_{EM,LM}$	0.52 ± 0.05	0.64 ± 0.04	0.58 ± 0.04
	r_{EM_d,LM_d}	0.47 ± 0.09	0.51 ± 0.09	0.69 ± 0.05
	r_{EM_d,EM_m}	-0.13 ± 0.10	0.05 ± 0.11	-0.15 ± 0.09
	r_{EM_d,LM_m}	0.11 ± 0.20	0.43 ± 0.14	-0.44 ± 0.13
	r_{EM_m,LM_d}	-0.03 ± 0.12	0.07 ± 0.09	-0.00 ± 0.12
	r_{LM_m,LM_d}	0.14 ± 0.09	0.29 ± 0.20	-0.38 ± 0.13
	r_{EM_m,LM_m}	0.41 ± 0.18	0.83 ± 0.04	0.39 ± 0.21

[a]Genetic correlation between early (EM) and late mortality (LM) and late mortality with the maternal genetic effect (EM_d, LM_d, EM_m, LM_m)

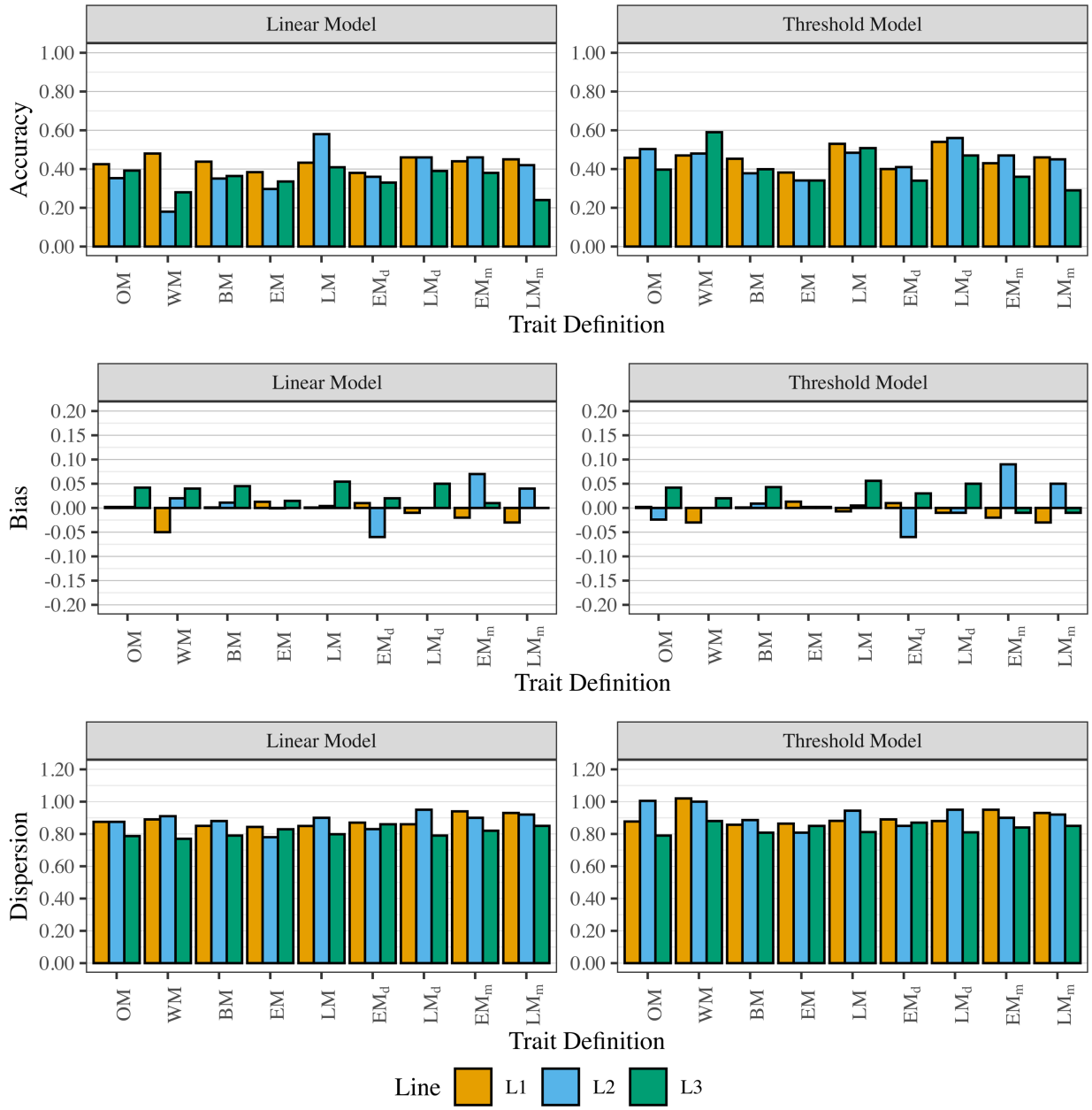

Figure 4.1. Prediction accuracy, bias, dispersion breeding values from ssGBLUP evaluations between whole and partial datasets for both linear and threshold models for each trait definition: mortality (OM), weekly mortality (WM), broiler mortality (BM), early and late mortality (EM and LM) and early and late mortality with maternal genetic effect (EM_d, LM_d, EM_m, LM_m) among all three lines.

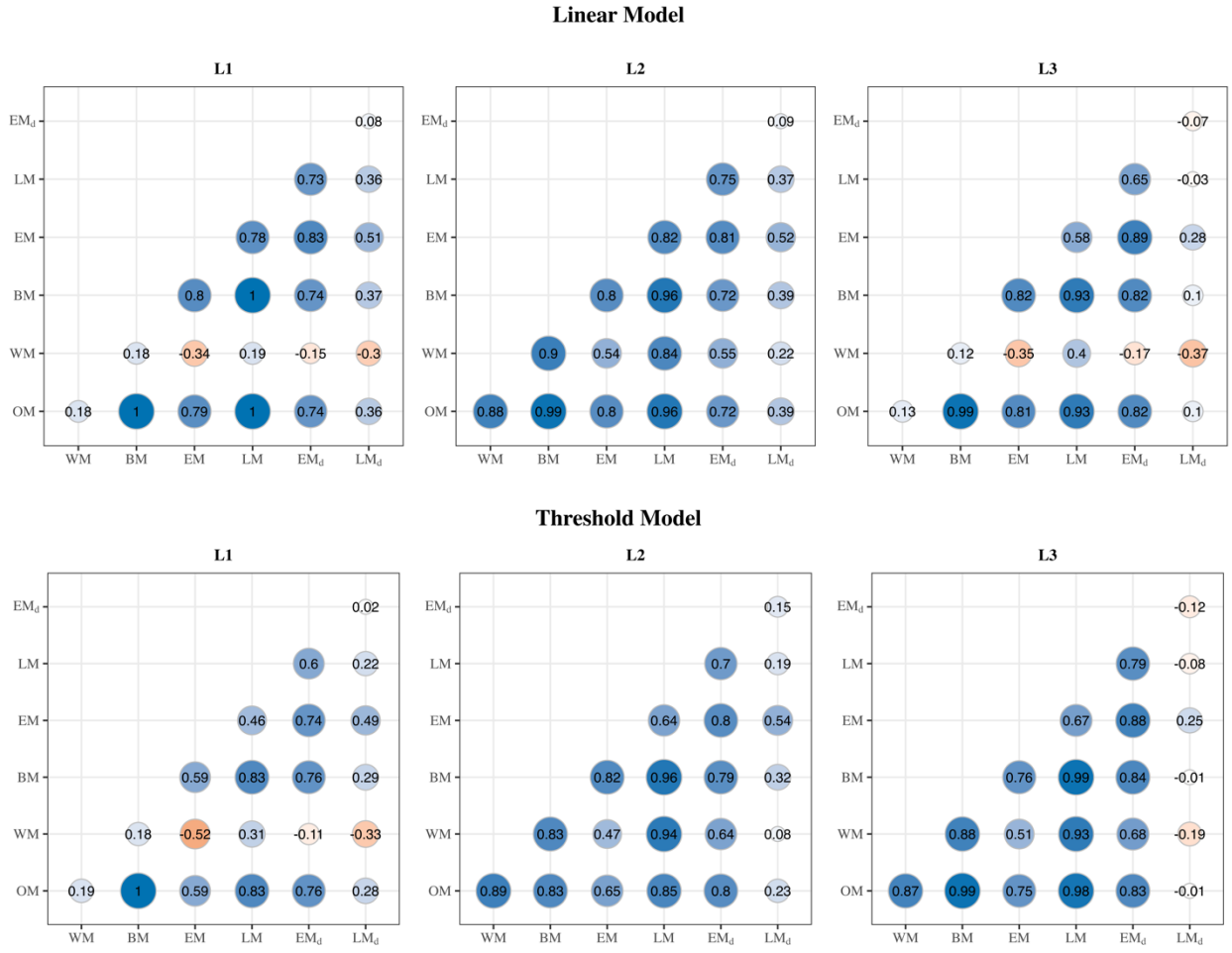

Figure 4.2. Rank correlations of breeding values for L1, L2, and L3 from use of linear models for each trait definition: mortality (OM), weekly mortality (WM), broiler mortality (BM), early and late mortality (EM and LM) and early and late mortality with maternal genetic effect (EM_d, LM_d) among all three lines.

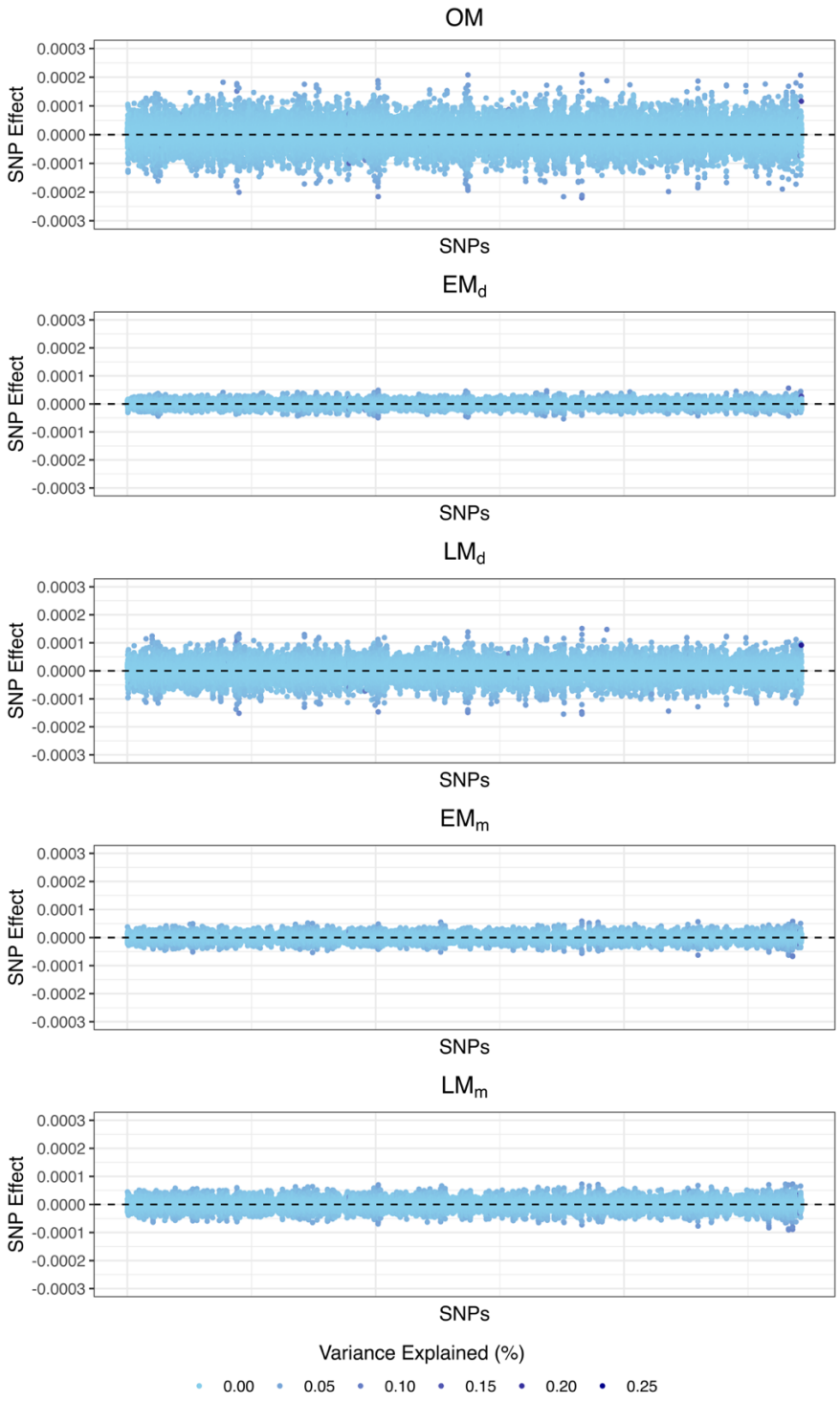

Figure 4.3. SNP effects for baseline mortality (OM) and early and late mortality with the inclusion of the maternal genetic effect (EM_d, LM_d, EM_m, LM_m) for L1. The colors show the percentage of additive genetic variance explained by each SNP.

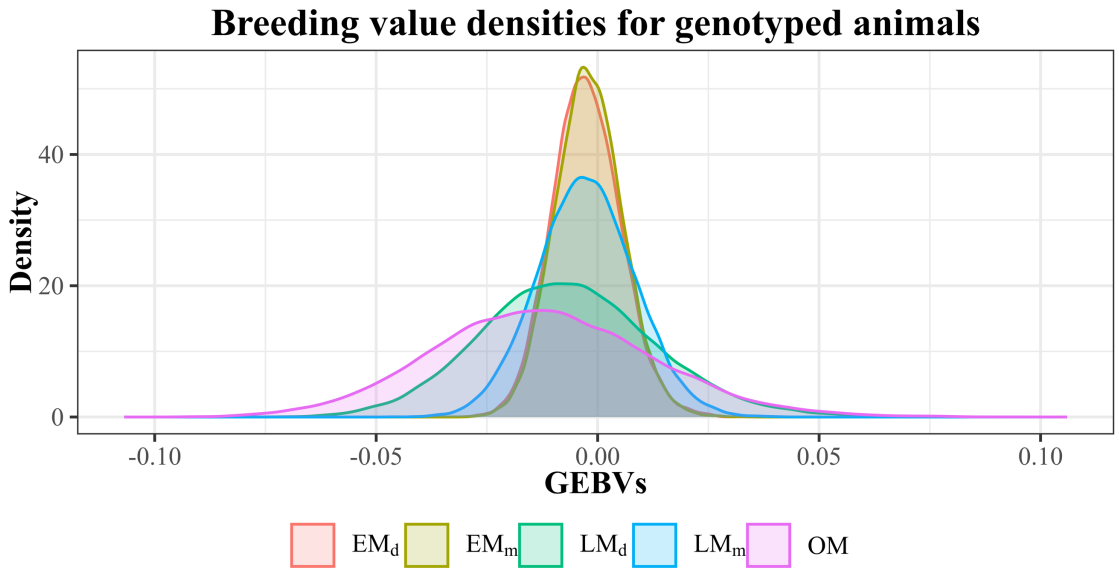

Figure 4.4. Densities of genomic breeding values for baseline mortality (OM) and early and late mortality with the inclusion of the maternal genetic effect (EM_d, LM_d, EM_m, LM_m) for L1 for all genotyped individuals.

CHAPTER 5

ANALYSIS OF CATEGORICAL DATA IN BROILER CHICKEN: AN ALTERNATIVE ALGORITHM FOR EFFICIENCY[3]

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ABSTRACT

Categorical traits can be assessed through threshold modeling, a statistical approach used for dealing with such characteristics. In this framework, the prediction of breeding values for these traits relies on the assumption that the observed "realized" phenotype results from truncating an underlying liability, normally distributed, into distinct categories with thresholds. This study aimed to investigate an alternative approach to threshold models that utilizes the Expectation Maximization algorithm and imputes liabilities using phenotypes and residuals from a linear model. This approach, implemented in the CATEGF90 software, was compared to solutions from the regular threshold models regarding predictive ability and computing performance. Approximately three years' worth of data was provided by Cobb-Vantress, Inc. for four categorical traits. Three traits were binary: mortality (MT), tibial dyschondroplasia (TD), and ascites (AC), each with differing levels of incidence. The last trait was a multiple-ordered categorical trait, femoral head necrosis (FN), with three levels after data cleaning. Five different methods were used for the analysis of the four categorical traits. Variance components were estimated, and linear regression validation (LR) was performed. The approaches included a threshold model based on Gibbs sampling (GIB), two threshold models based on the frequentist approach, one with the default option of estimating threshold (CET) and another with the option to fixate thresholds (CFT), and the alternative approach with two different convergence criterion, 1E-4 (CA4) and 1E-8 (CA8). The LR accuracies showed that GIB had consistently the highest accuracies across traits. CET and CFT had comparable accuracies with a reduced computational cost. CA4 performed well for MT and FHN but had significantly lower accuracies for the traits with low incidence, TD, and AC. The computing performance of CA4 was similar to that of CET and CFT. CA8 had a higher computing cost than CA4 but had more comparable accuracies to

GIB for AC and TD. The alternative approach may not be more efficient than some of the available algorithms to evaluate categorical data using threshold models, but its implementation is straightforward as it utilizes solutions from linear models.

INTRODUCTION

Many evaluations performed in animal breeding include both continuous and categorical traits. Categorical traits do not have a normal distribution of phenotypes and possess unique features that distinguish them from continuous traits. These categorical traits are often binary, with two possible outcomes, or ordered categorical, with several possible outcomes (Gruneberg, 1952; Gianola, 1981; Falconer and Mackay, 1996a). Such traits of interest might include calving ease, gait scores, mortality, disorder traits, and disease resistance.

The categorical traits can be evaluated using threshold modeling, which is well-suited for handling traits of this nature. In this statistical framework, breeding values for such traits are predicted through the assumption that the observed "realized" phenotype is a response from the truncation of an underlying, normally distributed liability into discrete categories guided by a series of thresholds (Wright, 1934; Gianola and Foulley, 1983). The determination of thresholds is contingent upon the number of categories assigned to the trait, with binary traits requiring a single threshold and traits with m categories necessitating $m-1$ thresholds. One notable advantage of employing threshold models in animal breeding evaluations is their ability to assimilate diverse information in a valid manner (Wang et al., 1997). Also, it has been presented that in evaluations of categorical traits, it is more suitable to use non-linear models, such as threshold models, to accommodate better the natural distribution of the binary responses (Gianola, 1982).

A complication in modeling categorical data arises from the extreme category problem (ECP), which can hinder or delay convergence (Misztal et al., 1989). The ECP manifests when all responses for a specific level of a fixed effect fall into an extreme category, resulting in solutions tending toward plus or minus infinity. Two approaches have been proposed to address the ECP. The first involves treating the fixed effect as random, while the second suggests removing any class affected by ECP (Harville and Mee, 1984). However, both solutions introduce challenges in arriving at final model solutions.

The practical implementation of threshold models faces challenges that diminish their utility. The complexity of computations required for these evaluations poses a hindrance, demanding equations involving numerous normal probability integrals. This complexity not only makes programming arduous but also complicates the testing process. Solutions in threshold modeling necessitate iterative approaches, where each round involves solving a system of equations. This, in turn, requires frequent setup of the coefficient matrix on disk, leading to computationally expensive procedures (Misztal et al., 1989). With the growing amount of genomic information used in evaluations, threshold models have been reported to have convergence and computing-time issues, primarily due to the non-linearity of the set of equations involved.

Various approaches have been proposed for integrating categorical traits into genetic evaluations. One such method is Bayesian analysis employing Gibbs sampling, initially introduced by Geman and Geman (1984). This approach considers all model parameters, including fixed effects, breeding values, genetic and residual covariance matrices, and thresholds based on marginal posterior densities while considering all uncertainties associated with other parameters. Initial applications of Gibbs sampling in analyzing categorical data were presented by Zeger and

Karim (1991) in a simulation study analyzing infectious disease data and by Albert and Chib (1993) on a binary response of election data.

Gibbs sampling was originally used in an animal breeding context by Wang et al. (1993) to describe Gibbs sampler for a univariate mixed linear model. Gibbs sampler provided a method to construct marginal densities of variance components, variance ratios, and marginal distributions of fixed and random effects. Further applications were made by Wang et al. (1994b), where Bayesian marginal inferences were made about fixed and random effects, variance components, and functions of variance components in a univariate Gaussian mixed linear model. Additionally, Wang et al. (1994a) reported promising results of response to selection using Bayesian analysis via Gibbs Sampler in comparison to classical analysis techniques. Although inferences from this method are robust, the computational expense of implementing Gibbs sampling makes it challenging to routinely conduct large-scale analyses (Wang et al., 1997).

An alternative approach, initially developed by Foulley et al. (1983), was designed to evaluate a binary trait and two continuous traits without considering missing data. Subsequently, Janss and Foulley (1993) developed the method further to accommodate missing data. Expanding upon this, Hoeschele et al. (1995) further enhanced the method to handle an ordered categorical trait with any number of levels, along with several continuous traits exhibiting various patterns of missing data.

This approach obtained iterative solutions for location parameters within each Fisher scoring step. During each scoring iteration, new solutions for the residual covariances among categorical and continuous traits were computed through maximum likelihood estimation. These solutions were then used to reevaluate all partial regression coefficients of liability on continuous traits for each missing data pattern. Breeding values were estimated as part of the mode of the joint

posterior density of fixed and random effects, and dispersion parameters were determined using approximate marginal maximum likelihood or maximum likelihood (Gianola and Foulley, 1983; Hoeschele et al., 1995). The use of iteration on data was suggested to handle solving large systems of equations for this type of approach (Schaeffer and Kennedy, 1986).

Using this methodology allowed practical prediction of breeding values for field data that included threshold characters in a more applicable way. This methodology became the basis for the program CBLUPF90IOD2 in the BLUPF90 software suite (Misztal et al., 2014a). This program includes a conversion to the threshold model using this approach and a conversion to iteration on data by the preconditioned conjugate gradient algorithm (PCG), making it easier to implement and faster to converge (Wiggans et al., 1998; Tsuruta et al., 2001). This method is much quicker than the previous method via Gibbs Sampler; however, one of the limitations of this method is the inability to include more than one categorical trait within the evaluations. The objective of this study was to investigate the performance of an alternative algorithm to analyze binary and ordered categorical traits using a broiler population under selection. This approach utilizes the Expectation Maximization (EM) algorithm and imputes liabilities using phenotypes and residuals from a linear model.

MATERIALS AND METHODS

Animal Care and Use Committee approvals were unnecessary because data were obtained from preexisting databases.

Alternative algorithm

The alternative algorithm uses an expectation maximization (EM) algorithm to deal with threshold traits. First proposed by Quaas (1994), an outer loop successively updates the phenotypes (y) based on current solutions of the inner BLUP loop. The EM algorithm forms an "imputed" trait in the underlying scale (U). To get the conditional expectations of residuals of one trait given the others, considering one trait at a time, let:

$$\mathrm{Var}(\mathrm{e}) = \mathbf{R} \qquad [5.1]$$

Using multivariate normal theory:

$$\mathrm{e_i}|\mathbf{e}_{-\mathrm{i}} = N(\mathbf{R}_{-\mathrm{I,i}}\left(\mathbf{R}_{-\mathrm{I},-\mathrm{i}}\right)^{-1}\mathbf{e}_{-\mathrm{I}}, \mathbf{R}_{\mathrm{ii}} - \mathbf{R}_{-\mathrm{I,i}}\left(\mathbf{R}_{-\mathrm{I},-\mathrm{i}}\right)^{-1}\mathbf{R}_{\mathrm{I},-\mathrm{i}}) \qquad [5.2]$$

Then, two partitioned matrix inverses are used. Let:

$$\mathbf{A} = \begin{pmatrix}\mathbf{A}_{11} & \mathbf{A}_{12}\\ \mathbf{A}_{21} & \mathbf{A}_{22}\end{pmatrix} \text{ and } \mathbf{A^{-1}} = \begin{pmatrix}\mathbf{A}^{11} & \mathbf{A}^{12}\\ \mathbf{A}^{21} & \mathbf{A}^{22}\end{pmatrix} \qquad [5.3]$$

Then, $\mathbf{A}^{12} = -\mathbf{A}^{11}\mathbf{A}_{12}\mathbf{A}_{22}^{-1}$, which leads to:

$$-(\mathbf{A}^{11})^{-1}\mathbf{A}^{12} = \mathbf{A}_{12}\mathbf{A}_{22}^{-1} \qquad [5.3.1]$$

and

$$\mathbf{A}^{11} = (\mathbf{A}_{11} - \mathbf{A}_{12}\mathbf{A}_{22}^{-1}\mathbf{A}_{21})^{-1} \text{ or } (\mathbf{A}^{11})^{-1} = \mathbf{A}_{11} - \mathbf{A}_{12}\mathbf{A}_{22}^{-1}\mathbf{A}_{21} \qquad [5.3.2]$$

Then from [5.3.2]:

$$\mathbf{R}_{\mathrm{ii}} - \mathbf{R}_{-\mathrm{i,i}}\left(\mathbf{R}_{-\mathrm{i},-\mathrm{i}}\right)^{-1}\mathbf{R}_{\mathrm{i},-\mathrm{i}} = \left(\mathbf{R}^{\mathrm{ii}}\right)^{-1} = \frac{1}{\mathbf{R}^{\mathrm{ii}}} \qquad [5.4]$$

where the conditional variance of $\mathrm{e_i}|\mathbf{e}_{-\mathrm{i}}$ is $\frac{1}{\mathbf{R}^{\mathrm{ii}}}$,

And from [5.3.1]:

$$\mathbf{R}_{\mathrm{ii}} - \mathbf{R}_{-\mathrm{i,i}}\left(\mathbf{R}_{-\mathrm{i},-\mathrm{i}}\right)^{-1}\mathbf{R}_{\mathrm{i},-\mathrm{i}} = \left(\mathbf{R}^{\mathrm{ii}}\right)^{-1} = \frac{1}{\mathbf{R}^{\mathrm{ii}}} \qquad [5.4.1]$$

where the conditional expectation of $\mathrm{e_i}|\mathbf{e}_{-\mathrm{I}}$ is $\mathrm{e_i}|\mathbf{e}_{-\mathrm{i}} = -\frac{1}{\mathbf{R}^{\mathrm{ii}}}\mathbf{R}^{(\mathrm{I},-\mathrm{i})}\mathbf{e}_{-\mathrm{i}}$.

Thus,

$$\mathrm{e_i}|\mathbf{e}_{-\mathrm{i}} = N\left(-\frac{1}{\mathbf{R}^{\mathrm{ii}}}\mathbf{R}^{\mathrm{i},-\mathrm{i}}\mathbf{e}_{-\mathrm{i}}, \frac{1}{\mathbf{R}^{\mathrm{ii}}}\right) \quad [5.5]$$

Then, $\widehat{\mathrm{e_i}} = E(\mathrm{e_i}|\mathbf{e}_{-\mathrm{i}}) = \frac{1}{\mathbf{R}^{\mathrm{ii}}}\mathbf{R}^{(\mathrm{i},-\mathrm{i})}\hat{\mathrm{e}}_{-i}$

which is the actual implementation of data augmentation in the Thurstonian Model, by Varona and Legarra (2020), which uses the single inversion of **R** instead of inverting the submatrices of **R**. This corresponds to "solving" the following equations for $\mathrm{e_i}$:

$$(R_{ii} \quad \boldsymbol{R}^{i,-i})\begin{pmatrix} e_i \\ e_{-1} \end{pmatrix} = (0) \quad [5.5.1]$$

The EM strategy uses the conditional expectations to compute the pseudo-trait by using the residuals, provided that to "impute" trait i, the observed y for linear traits or the "imputed" U for categorical traits is used. Assume one categorical trait and several continuous traits. Then the conditional distributions of $\mathrm{e_i}$ (Equation 5.5), then b, the regression coefficient of the residual is:

$$b = \mathbf{R}_{-\mathrm{i},\mathrm{i}}\left(\mathbf{R}_{-\mathrm{i},-\mathrm{i}}\right)^{-1} = -\frac{1}{\mathbf{R}^{\mathrm{ii}}}\mathbf{R}^{(\mathrm{i},-\mathrm{i})} \quad [5.6]$$

and, σ^2, its conditional variance is:

$$\sigma^2 = \mathbf{R}_{\mathrm{ii}} - \mathbf{R}_{-\mathrm{i},\mathrm{i}}\left(\mathbf{R}_{-\mathrm{i},-\mathrm{i}}\right)^{-1}\mathbf{R}_{\mathrm{i},-\mathrm{i}} = \frac{1}{\mathbf{R}^{\mathrm{ii}}} \quad [5.7]$$

The expressions hold for any number of continuous traits, provided that the matrix R that is used contains the categorical and the observed traits only. If no continuous traits are observed, then $(\mathbf{R}_{-\mathrm{i},-\mathrm{i}})$ is a null matrix and σ^2 is obtained with $\sigma^2 = \mathbf{R}_{\mathrm{ii}}$ and b is undefined and not used. Using these equations, a pseudo-trait (U) can be computed by:

$$U = \mu + \sigma\lambda \quad [5.8]$$

where

$$\mu = x\hat{\beta} + z\hat{u} + \hat{e}_i = \hat{y} + \hat{e}_i \quad [5.8.1]$$

where $\hat{\beta}$and $\hat{u}$ are the current estimates for the categorical trait, $\hat{e}_i = \mathbf{b}'\hat{e}_{-i}$ computed as $\hat{e}_i = -\frac{1}{R_{ii}}R^{(i,-i)}\hat{e}_{-i}$, and $\sigma^2 = \frac{1}{\mathbf{R}^{ii}}$. Then λ is:

$$\lambda = -\frac{(\phi(Z_k) - \phi(Z_{k-1}))}{(\Phi(Z_k) - \Phi(Z_{k-1}))} \quad [5.8.2]$$

where $Z = \frac{(t-\mu)}{\sigma}$ is the standardized distance from the mean to the threshold, and k is the observed category, and ϕ is the normal density function and Φ is the cumulative density function.

Finally,

$$U = x\hat{\beta} + z\hat{u} + \hat{e}_i + \lambda\sigma = \mu + \lambda\sigma \quad [5.9]$$

Those formulas are implemented in CATEGF90 (from the BLUPF90 software suite), which is an iterative program that calls a BLUP MME solver like BLUP90IOD3 (from the BLUPF90 software suite) for the linear model and then uses the residuals and phenotypes to create "imputed liabilities" (U) using the above methodology. Thresholds and variance components in the underlying scale need to be known in advance. Theoretically, this program should handle any pattern of missing data. Convergence is measured in the external loop of the program. The external loop measures how the "imputed" liability U changes for each trait i as

$$\text{eps} = \frac{\sum(u^t - u^{t-1})^2}{\sum(u^{t-1})^2} \quad [5.10]$$

where the program stops once eps is less than the convergence criteria (default convergence criteria is 1E-4).

Data and variance components

Cobb-Vantress, Inc. (Siloam Springs, AR) provided a dataset comprising 15 overlapping mating groups. The dataset included four discrete traits, being three binary traits and one ordered

categorical trait. The three binary traits were mortality (MT), tibial dyschondroplasia (TD), and ascites (AC), each of which had differing levels of incidence. These traits were recorded as 1 (alive or normal) or 2 (dead or abnormal). Mortality was recorded from the time of chick placement after hatch until the first selection point. The multiple-ordered categorical trait was femoral head necrosis (FN). The FN was scored as 1 (normal) to 7 (severe disorder). A random sample of birds was sent for dissection, with TD and FN records collected at the first selection point (Zhang et al., 2018c). However, they were combined due to low incidences for the last four levels in both datasets, leaving only possible phenotypes of 1, 2, or 3 to 7. The number of records and incidences within each level for each categorical trait are presented in Table 1. In the dataset, there were 186,596 animals in the pedigree, of which 18,047 animals had genomic information.

All individuals were genotyped with a 60K Illumina Chicken SNP BeadChip. Quality control was performed on genotypes using the PREGSF90 software (Misztal et al., 2014) and excluded duplicated genotyped individuals, and SNP call rate <0.90, SNP with minor allele frequency <0.05. Also, SNP were removed if Mendelian conflict rate between parent-progeny pairs was >10% and progenies were eliminated if the conflict rate was >1%. Monomorphic SNP with unknown position or located at sex chromosomes were also removed. After quality control, 41,156 autosomal SNP for 18,047 birds were kept for analysis.

Variance components were estimated for all traits using single-trait threshold models without including genomic information. The models implemented were:

$$\mathbf{y} = \mathbf{X\beta} + \mathbf{Z_1 u_1} + \mathbf{Z_2 u_2} + \mathbf{e} \qquad [5.11]$$

Where: $\mathbf{y}$ is the vector of observations; $\boldsymbol{\beta}$ is the vector of systematic effects (sex for TD,AC, and FN and mating group (MG) for MT), assumed as $\boldsymbol{\beta} \sim N\{\mathbf{0}, \mathbf{I}\sigma^2_{\beta}\}$; $\mathbf{u_1}$ is the vector of additive genetic effects, assumed as $\mathbf{u_1} \sim N\{\mathbf{0}, \mathbf{A}\sigma^2_{u}\}$; $\mathbf{u_2}$ is the vector of contemporary group random effects

$\mathbf{u_2} \sim N\{\mathbf{0}, \mathbf{I}\sigma^2_{cg}\}$; **e** is the vector of random residual effects ($\mathbf{e} \sim N\{\mathbf{0}, \mathbf{I}\sigma^2_e\}$); **X**, $\mathbf{Z_1}$, and $\mathbf{Z_2}$ are the incidence matrices relating the elements of **y** to the elements of **b**, $\mathbf{u_1}$, and $\mathbf{u_2}$, respectively.

Variance components were estimated using GIBBSF90+ software (Misztal et al., 2014a). A single-chain of 300,000 samples was generated, assuming a burn-in of 100,000 and thinning interval of 100. Inferences about variance components were made over 2,000 samples from the posterior distributions. Convergence was assessed graphically, by the autocorrelation of samples, as well as the Geweke's Diagnostic test (Geweke and In, 1995) as implemented in the POSTGIBBSF90 software (Tsuruta and Misztal, 2006).

Statistical Analysis

Breeding values were predicted using five different methods. The implemented statistical models were as before, except for replacing $\mathbf{A}^{-1}$ with $\mathbf{H}^{-1}$ (Aguilar et al., 2010), the inverse of the realized relationship matrix in ssGBLUP. For a benchmark, we first predicted breeding values under single-trait threshold models using GIBBSF90+ (GIB) (Tsuruta and Misztal, 2006). A single-chain was generated with 30,000 samples, assuming a burn-in of 10,000 and thinning interval of 10; thus, inference about the breeding values was made over 2,000 samples from the posterior distribution. Secondly, CBLUP90IOD2 (Misztal et al., 2014) was used to predict breeding values using the default method of estimating thresholds in outer rounds and solving the system of equations in inner PCG rounds (CET) and testing the fixation of the thresholds (CFT). A single threshold was fixed at 0 for binary traits (MT, TD, and AC), and thresholds of 0 and 1 were fixed for FN. Lastly, the alternative algorithm, implemented in CATEGF90 (still an in-house software) was performed using the default convergence criteria of 1e-4 (CA4) and testing stricter convergence criteria of 1e-8 (CA8) for the pseudo phenotypes.

Comparison of Methods

The linear regression (LR) method (Legarra and Reverter, 2018) was employed to assess each method with respect to GIB. This approach compares genetic evaluations, including whole and partial datasets, based on differences in means and covariances using a set of focal individuals. There were 2,382 animals selected as focal that had genotypes and phenotypes in the whole dataset. The partial dataset was created by removing phenotypes from the focal animals, their siblings, and contemporaries. Let the partial dataset be denoted by subscript p, while the whole dataset is denoted by subscript w, then validation statistics are:

$$\text{acc} = \sqrt{\frac{\text{cov}\left(\hat{\mathbf{u}}_{\text{w}}, \hat{\mathbf{u}}_{\text{p}}\right)}{(1-\bar{\text{F}})\sigma_{\text{u}}^2}} \quad [5.12]$$

$$\text{bias} = \frac{\bar{\hat{\mathbf{u}}}_{\text{p}} - \bar{\hat{\mathbf{u}}}_{\text{w}}}{\sigma_{\text{u}}} \quad [5.13]$$

$$\text{b}_1 = \frac{\text{cov}\left(\hat{\mathbf{u}}_{\text{w}}, \hat{\mathbf{u}}_{\text{p}}\right)}{\text{var}\left(\hat{\mathbf{u}}_{\text{p}}\right)} \quad [5.14]$$

where: $\hat{\mathbf{u}}_{\text{w}}$ and $\hat{\mathbf{u}}_{\text{p}}$ are the vectors of GEBV (for focal animals) from whole and partial datasets, respectively, $\bar{\text{F}}$ is the average inbreeding coefficient of validation animals, σ_{u}^2 is the additive genetic variance; and $\bar{\hat{\mathbf{u}}}_{\text{w}}$ and $\bar{\hat{\mathbf{u}}}_{\text{p}}$ are the GEBV averages from whole and partial data, respectively. Bias evaluates the bias present in the predicted breeding values (GEBVs) for the focal animals, with an expected value of zero indicating an unbiased prediction. Dispersion measures the inflation of GEBVs for the focal animals and is expected to be one in the absence of inflation.

RESULTS AND DISCUSSION

Variance components are presented for each trait in Table 2. The heritability estimates indicate that enough genetic variation exists within these traits. As expected, the residual variances were 1.00 for all binary traits but not for the ordered categorical trait. These variance components are still comparable across presented literature for mortality and these health traits (de Greef et al., 2001; Pakdel et al., 2005a; Kapell et al., 2012; Zhang et al., 2018c; Richter et al., 2022). Additionally, any differences in genetic parameters across the literature might be due to different trait definitions, animal age at measurement, sample size, and statistical and computational strategies used for estimation (Rekaya et al., 2013).

Validation of methods

Validation statistics from the LR method are presented in Figure 1. Accuracies ranged from 0.18 to 0.45 for AC, 0.16 to 0.39 for TD, 0.33 to 0.40 for MT, and 0.35 to 0.47 for FN. When comparing GIB with the other methods for the lowest incidence trait, AC, the accuracy decreased by 13.3% for both CET and CFT, 60.0% for CA4, and 15.6% for CA8. For the next highest incidence trait, TD, accuracy decreased by 15.4% for both CET and CFT, 59.0% for CA4, and 18.0% for CA8. For the highest incidence binary trait, MT, the accuracy decreased by 7.5% for CET and CFT, 17.5% for CA4, and 5% for CA8. For the multiple ordered categorical trait, FN, the accuracy decreased 10.6% for CET, 4.3% for CFT, 25.5% for CA4, and 21.3% for CA8.

GIB consistently produced better accuracies than any other method across traits, with CET and CFT producing higher accuracies than CA4 and CA8 Using GIB in practical breeding programs would allow for better improvements over time, but there are limitations due to fluctuations across subsequent evaluations and extensive computing time (Lee et al., 2002). For all binary traits, there was no difference between CET and CFT. However, for FHN, accuracy was

higher for CFT than CET, even though this difference was slight. Both CET and CFT produced comparable accuracies with GIB, especially as the incidence of binary traits increased and when multiple categories were present. Previous studies have shown that a similar approach produced promising results in Australian Angus Cattle foot and leg score and docility evaluations (Jeyaruban, 2012; Walkom et al., 2016).

The CA4 performed adequately in terms of accuracy compared to GIB for MT and FN; however, it performed poorly for AC and TD. This is most likely due to low incidence levels, which causes a lack of proper convergence. Once a stricter convergence criterion was used (CA8), the AC and TD accuracies became more comparable to GIB. Theoretically, CA4 should be strict enough to encompass proper convergence as this measure is not the convergence criteria of GEBVs but instead the convergence of pseudo-phenotypes created in CATEGF90. When traits have this low incidence levels, GEBVs have low accuracies unless there is a large enough dataset, even when the software has no flaws. Simulation studies using threshold models with the EM algorithm have shown good theoretical results (Pettitt, 1986; Carriquiry et al., 1987; Smith and Helms, 1995).

When making selection decisions in genetic evaluations, it is essential to account for the potential impact of bias and dispersion in breeding values to avoid suboptimal “biased” decisions (Legarra and Reverter, 2018). The estimated bias range from each method were from -0.06 to 0.02 for AC, -0.26 to -0.10 for TD, -0.05 to 0.06 for MT, and 0.02 to 0.04 for FN (Figure 1). For TD, there was a higher amount of bias compared to other traits, potentially due to the reduced number of records compared to the other binary traits. Another possible explanation is that there could be a lack of a proper model used for this particular trait. The bias for FN was the lowest among all methods compared to other traits. This may be due to this trait having more levels within the phenotype. The trend of bias among methods was unclear, with some methods having over- and

underestimated GEBVs depending on the trait. Small deviations from zero within these results could be due to small noise (Legarra and Reverter, 2018).

Most GEBVs were over-dispersed among all methods but were still reasonably close to one. The estimated dispersion ranged from 0.92 to 1.09 AC, 1.01 to 1.49 for TD, 0.84 to 0.86 for MT, and 0.87 to 0.94 for FN. The dispersion of GBEVs was more stable across MT and FN than in the lower-incidence traits AC and TD. The method used to predict GEBVs had a greater impact on these traits. Bias and dispersion of GEBVs for MT may also be due to selective genotyping, where only survivors are genotyped, or the lack of parent phenotype variation due to survivors being the only ones to get genotyped. This can reduce the predictability of breeding values (Grandinson et al., 2005; Garcia et al., 2018).

Scatterplots showing GEBVs among methods are shown in Figures 2, 3, 4, and 5 for AC, TD, MT, and FHN, respectively. The predicted GEBVs among each method showed high correlations for MT and FHN. For TD and AC, GEBVs had high correlations among methods except for CA4. Spearman rank correlations were also calculated among the methods tested. All correlations were greater than 0.99, meaning there was no major reranking of animals within the pedigree. Thus, if any of these methods were selected for evaluation, rankings among the animals for selection decisions should remain the same.

Computational Cost

The wall-clock time for each method in minutes is presented in Table 3, and the number of iterations to reach convergence is presented in Table 4. Even though the analysis was performed on a shared server, the wall-clock time and the number of iterations were consistent among the methods. The GIB consistently took more minutes to reach the necessary samples to ensure convergence. On average, across traits, GIB was 196-fold slower than CET, 82-fold slower than

CFT, 89-fold slower than CA4, and 35-fold slower than CA8. The wall-clock time difference between CET and CFT was less than 10 minutes among all traits. The time difference between CA4 and CA8 was larger but within 2 hours. This time difference was much larger for AC and TD than for MT and FHN. This is most likely due to the increased number of iterations to reach convergence under the stricter convergence criterion for CA8.

A chain of 30,000 samples was created for GIB to get adequate samples to make inferences. It took less than 4000 iterations for CET, CFT, and CA4 to reach convergence. CET had 47% and 26% fewer iterations compared to CFT and CA4 among all traits, respectively. On average, CA8 had 147% necessary iterations to reach convergence compared to CA4. Along with the number of iterations to reach convergence, the number of times the external round (BLUP90IOD3) is called within the CATEGF90 program can be used to access computing costs. For CA4, less than 20 rounds of the external loop were necessary to reach convergence. This number increased significantly for CA8, especially for the low incidence traits, AC and TD.

Methods like GIB that uses a Gibbs sampler are known to require a large number of samples for adequate convergence, and the computational costs can be prohibitive with large data sets (Lee et al., 2002). The implementation of Gibbs sampling has been shown to require around six times more computing time relative to other methods (Arakawa et al., 2009). The delay in sampling can be caused by extremely slow mixing due to single-component updating intensified in generalized linear models (Tempelman, 1998).

Practical Implications

Gibbs allows for the analysis of several continuous and categorical variables with any number of levels and unequal models per trait. However, due to the large computation cost, this may not be feasible for large-scale genomic evaluations. The approach developed by Hoeschele et

al. (1995) can significantly improve the computing cost for running evaluations with categorical data without losing out on the accuracy of predicted breeding values. However, this method does come without limitations as it only considers one categorical trait in the analysis. Also, this methodology is better performed using fixed thresholds, especially for traits with multiple ordered categories. Using the EM algorithm to analyze categorical traits has been well-developed over the years. It shows promising results for computational time and adequate prediction of breeding values. The advantage of the EM algorithm approach, in this case as implemented in CATEGF90, is that multiple categorical traits can be used in the evaluation while still getting adequate prediction of breeding values with reasonable computing costs. One consideration is that with growing genomic datasets being used in breeding programs, the computing cost of each of these methods will increase, so for the application of these methods, additional tests with a large number of genotyped animals may be needed.

CONCLUSIONS

The expectation maximization algorithm to impute liabilities using phenotypes and residuals from a linear model provides a robust tool for evaluating categorical data. This method can produce breeding values with comparable accuracies to a Bayesian approach via Gibbs sampling without the long computing time. More research needs to be completed to understand how this approach compares with larger genomic datasets and multiple-trait models.

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TABLES

Table 5.1: Number of records for each level of each trait (percentage of incidence)

Trait	# of Records	1	2	3 to 7
MT	180,998	167,389 (92.4%)	13,609 (7.5%)	-
TD	59,124	57,995 (98.1%)	1,129 (1.9%)	-
AC	163,971	161,950 (98.8%)	2,021 (1.2%)	-
FN	16,870	13,112 (77.7%)	2,295 (13.6%)	1,463 (8.7%)

MT: Mortality; TD: Tibial Dyschondroplasia; AC: Ascites; FN: Femoral Head Necrosis
[1]Levels: 1 (normal or alive); 2 (abnormal or dead)
[2]Levels: 1 (normal) to 7 (severe disorder)

Table 5.2: Posterior means (±SD) of genetic parameters for mortality and disorder traits using single-trait threshold models

Trait	h^2	σ_u^2	σ_{cg}^2	σ_e^2
MT	0.13 (0.08)	0.15 (0.01)	0.03 (0.00)	1.00 (0.00)
TD	0.33 (0.03)	0.53 (0.07)	0.09 (0.01)	1.00 (0.01)
AC	0.20 (0.03)	0.29 (0.01)	0.13 (0.02)	1.00 (0.05)
FN	0.29 (0.03)	0.82 (0.08)	0.11 (0.02)	1.91 (0.09)

MT: Mortality; TD: Tibial Dyschondroplasia; AC: Ascites; FN: Femoral Head Necrosis

Table 5.3: Wall-clock time for each method (in minutes)

Method	Real Time (m)			
	AC	**TD**	**MT**	**FN**
GIB	852	388	658	305
CET	12	1	21	1
CFT	21	4	18	2
CA4	9	5	6	4
CA8	96	42	17	8

MT: Mortality; TD: Tibial Dyschondroplasia; AC: Ascites; FN: Femoral Head Necrosis

Table 5.4: Number of iterations to convergence for each method performed

Method	Total number of iterations			
	AC	**TD**	**MT**	**FN**
GIB	30000	30000	30000	30000
CET	2270	575	2912	1188
CFT	3194	1836	3712	1293
CA4	2472	2148	2602	2472
CA8	27217	24364	7651	5486
	Number of external rounds[1]			
CA4	19	17	11	9
CA8	100	84	32	20

MT: Mortality; TD: Tibial Dyschondroplasia; AC: Ascites; FN: Femoral Head Necrosis
[1]Number of times the external loop, BLUP90IOD3 is called within CATEGF90

FIGURES

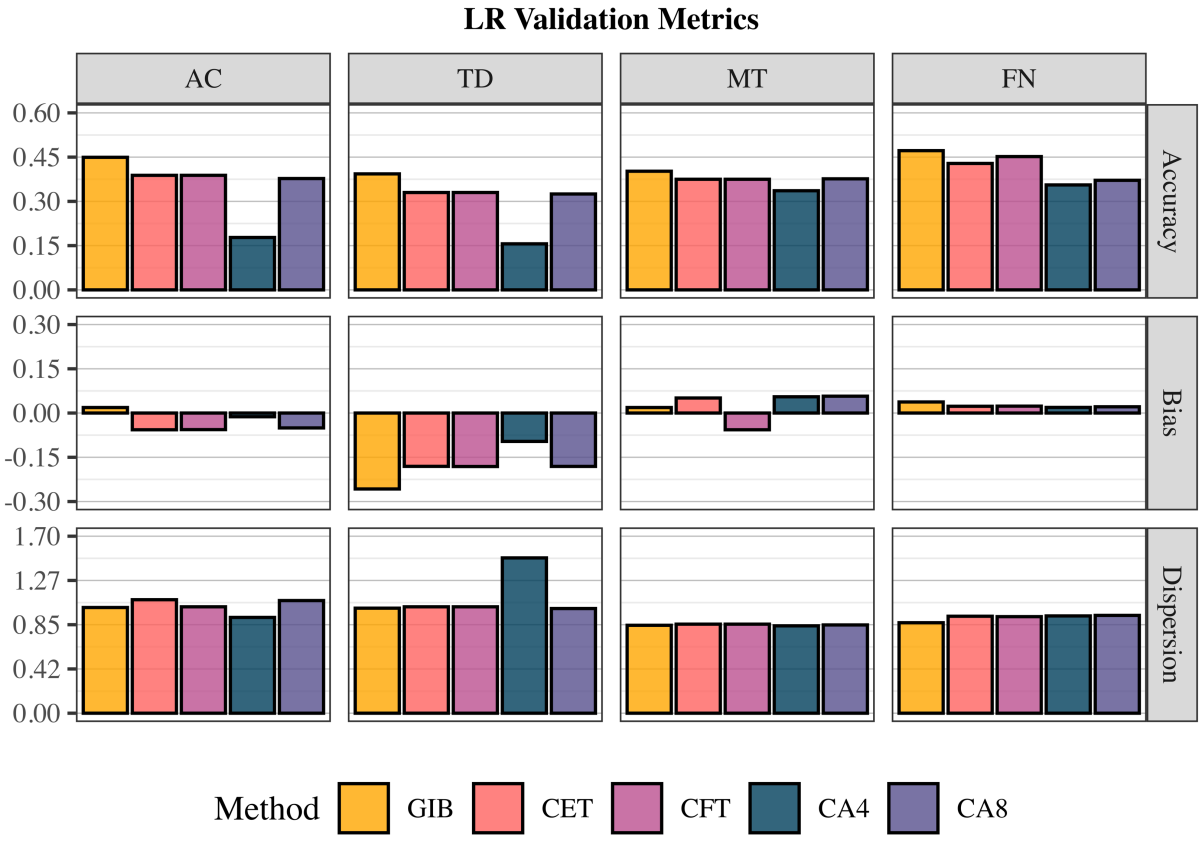

Figure 5.1: Prediction accuracy, bias, and dispersion of breeding values from evaluations between whole and partial datasets among each of the 5 methods: GIBBSF90+ (GIB), CBLUP90IOD3 with default of estimating thresholds (CET) and option to fix the thresholds (CFT), and CATEGF90 with default convergence criteria of 1e-4 (CA4) and stricter convergence criteria of 1e-8 (CA8).

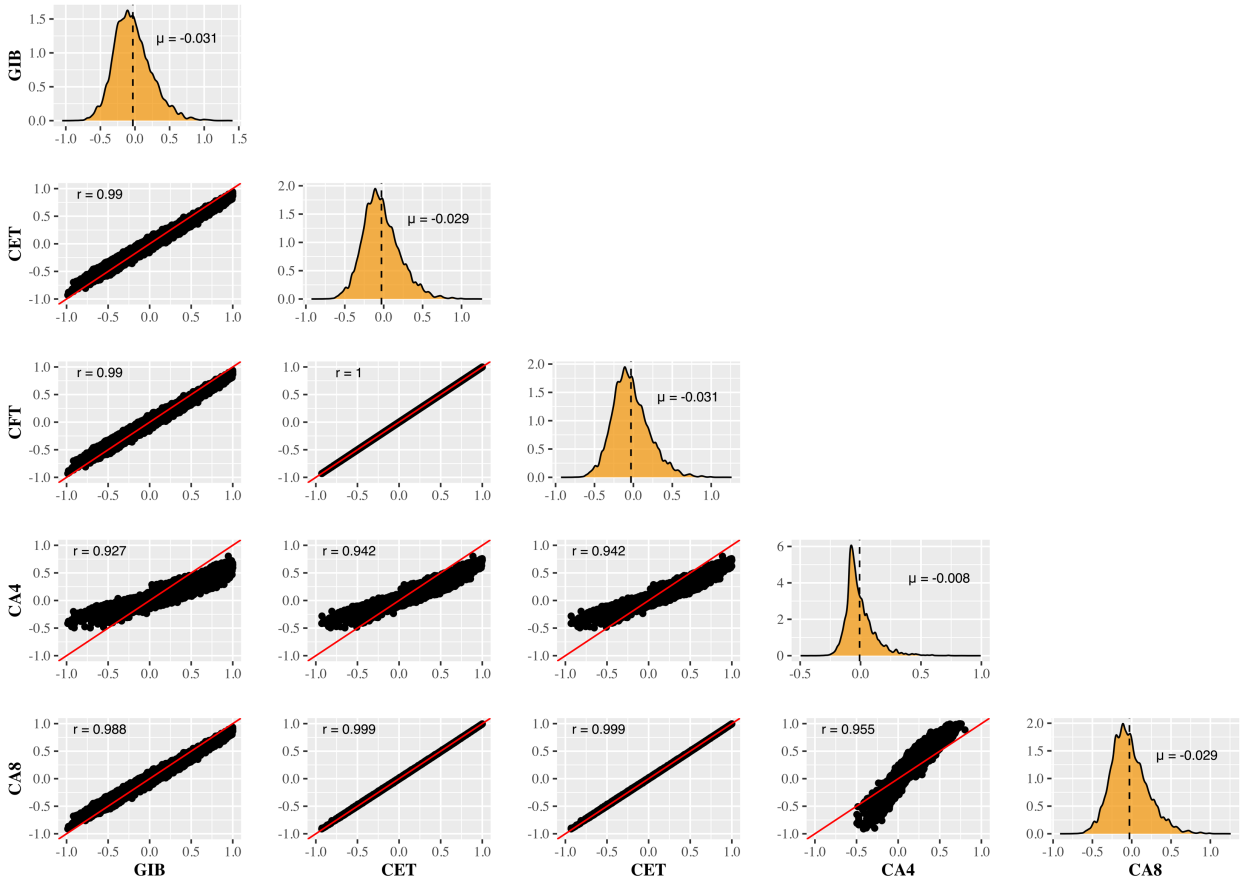

Figure 5.2: Scatter plots showing comparison of GEBVs and densities of GEBVs among methods for AC among each of the 5 methods: GIBBSF90+ (GIB), CBLUP90IOD3 with default of estimating thresholds (CET) and option to fix the thresholds (CFT), and CATEGF90 with default convergence criteria of 1e-4 (CA4) and stricter convergence criteria of 1e-8 (CA8).

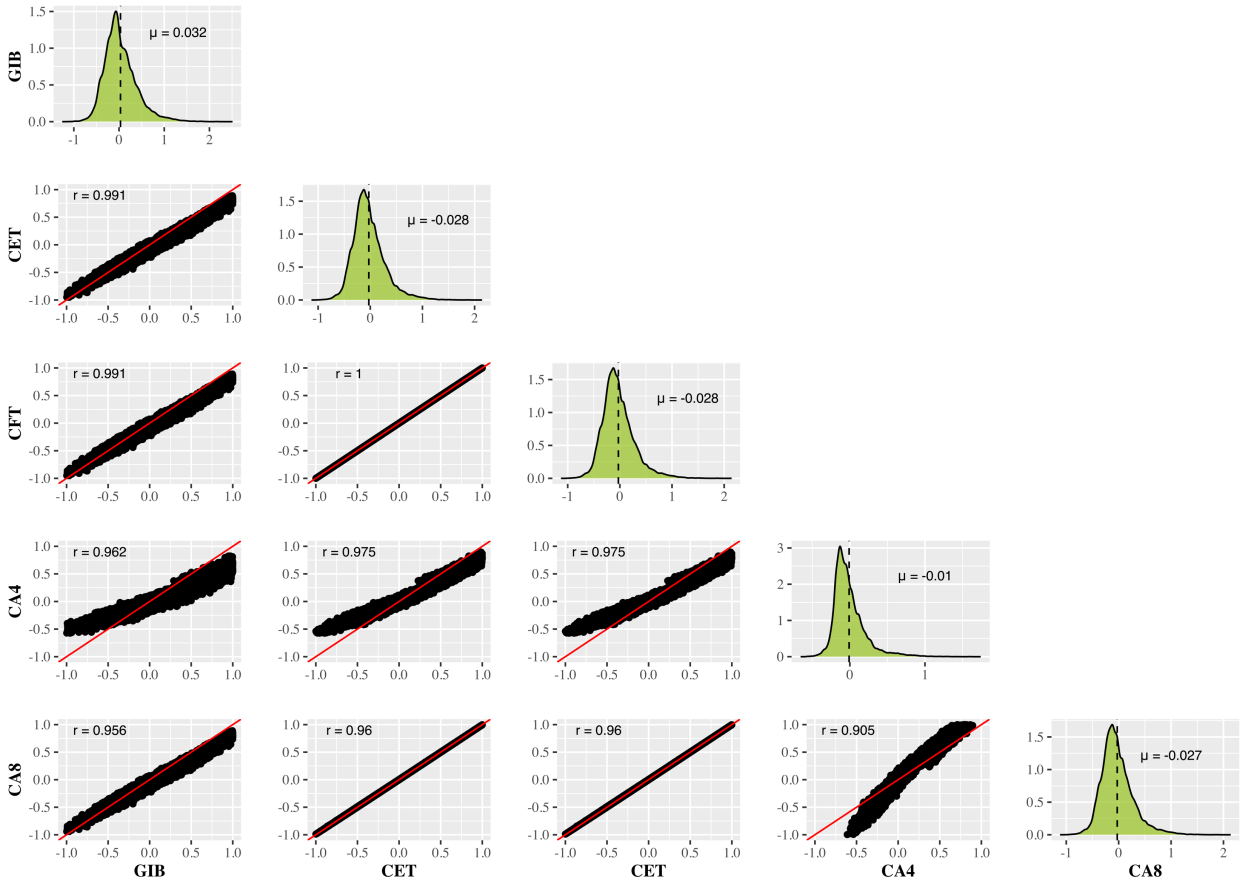

Figure 5.3: Scatter plots showing comparison of GEBVs and densities of GEBVs among methods for TD among each of the 5 methods: GIBBSF90+ (GIB), CBLUP90IOD3 with default of estimating thresholds (CET) and option to fix the thresholds (CFT), and CATEGF90 with default convergence criteria of 1e-4 (CA4) and stricter convergence criteria of 1e-8 (CA8).

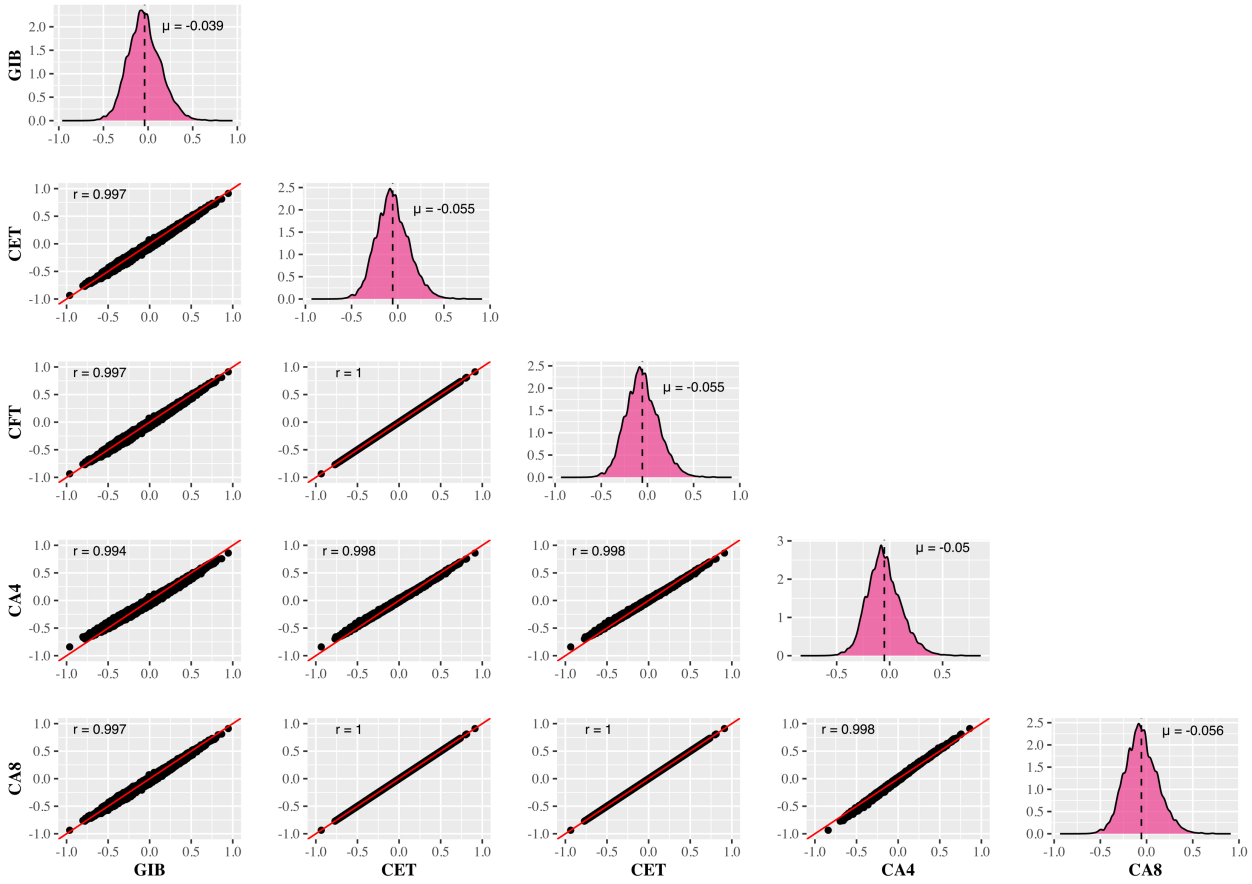

Figure 5.4: Scatter plots showing comparison of GEBVs and densities of GEBVs among methods for MT among each of the 5 methods: GIBBSF90+ (GIB), CBLUP90IOD3 with default of estimating thresholds (CET) and option to fix the thresholds (CFT), and CATEGF90 with default convergence criteria of 1e-4 (CA4) and stricter convergence criteria of 1e-8 (CA8).

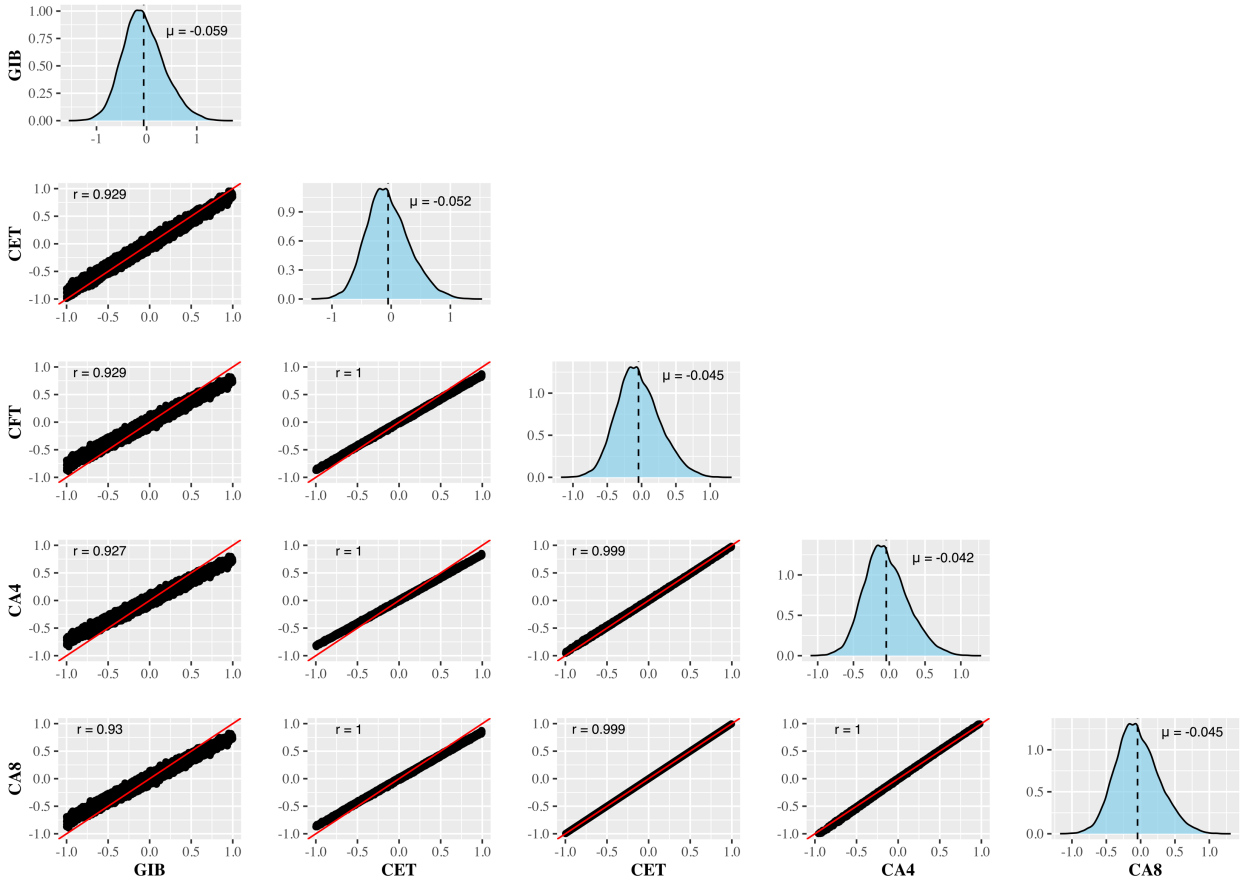

Figure 5.5: Scatter plots showing comparison of GEBVs and densities of GEBVs among methods for FN among each of the 5 methods: GIBBSF90+ (GIB), CBLUP90IOD3 with default of estimating thresholds (CET) and option to fix the thresholds (CFT), and CATEGF90 with default convergence criteria of 1e-4 (CA4) and stricter convergence criteria of 1e-8 (CA8).

CHAPTER 6

CONCLUSIONS

To sum up, the evolution of genetic evaluations in poultry breeding represents a dynamic journey, shaped by historical landmarks and technological strides. From the inception with the "Chicken of Tomorrow" contest in the mid-1900s, through the pivotal era of Best Linear Unbiased Prediction (BLUP) in the 1950s, to the transformative genomic phase marked by the discovery of single nucleotide polymorphisms (SNPs), each stage has contributed to the refinement of breeding strategies. The integration of genomic information, innovative methodologies, and critical validation techniques have significantly improved the precision of predicting breeding values. As the field encounters ongoing challenges, the need for continuous research becomes evident, through development of new methodologies and the incorporation of new technologies to aid in the progress of genetic evaluations in poultry breeding programs.

This dissertation recognized the temporal variation in genetic parameters, including decreasing genetic variances and increasing residual variances over time, highlighting the necessity of periodic updates and consideration of all available information sources in the estimation process. Utilizing genomic information in estimating variance components under genomic selection provides unbiased predictions of breeding values which in turn can advance breeding practices for improved broiler breeder performance and welfare.

Refining how mortality is defined in broiler breeders can improve predictions for survival and expand selection strategies. This dissertation recommends breaking down mortality into different time phases to better understand genetic influences. Including maternal effects, especially in the early stages of a chick's life, could enhance our understanding of mortality mechanisms.

Using these updated mortality definitions will improve the understanding of its genetic basis, ultimately leading to more effective breeding practices for better survival in broiler breeders.

Many traits evaluated in breeding programs are categorical in nature and are challenging to include in evaluations due to slow convergence and limitations on the number of categorical traits that can be included. This dissertation explored using an alternative method based on the EM algorithm for categorical data analysis. This method shows feasible computing performance without the limitations of previous methods. By utilizing this approach, improved breeding strategies for categorical traits in broiler breeder populations can be achieved.