GENETICS AND GENOMICS

Relationships among mortality, performance, and disorder traits in broiler chickens: a genetic and genomic approach

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ABSTRACT Four performance-related traits [growth trait (GROW), feed efficiency trait 1 (FE1) and trait 2 (FE2), and dissection trait (DT)] and 4 categorical traits [mortality (MORT) and 3 disorder traits (DIS1, DIS2, and DIS3)] were analyzed using linear and threshold single- and multi-trait models. Field data included 186,596 records of commercial broilers from Cobb-Vantress, Inc. Average-information restricted maximum likelihood and Gibbs sampling-based methods were used to obtain estimates of the (co)variance components, heritabilities, and genetic correlations in a traditional approach using best linear unbiased prediction (BLUP). The ability to predict future breeding values (measured as realized accuracy) was checked in the last generation when traditional BLUP and single-step genomic BLUP were used. Heritability estimates for GROW, FE1, and FE2 in single- and multi-trait models were similar and moderate (0.22 to 0.26) but high for DT (0.48 to 0.50). For MORT, DIS1, and DIS2, heritabilities were 0.13, 0.24, and 0.34, respectively. Estimates from single- and multi-trait models were also very similar. However, heritability for DIS3 was higher from the single-trait threshold model than for the multi-trait linear-threshold model (0.29 vs. 0.19). Genetic correlations between growth traits and MORT were weak, except for maternal GROW, which had a moderate negative correlation (−0.50) with MORT. The genetic correlation between MORT and DIS1 was strong and positive (0.77). Feed efficiency 1, which was moderately heritable (0.25) and is highly selected for, was not genetically related to MORT of broilers and other disorders. Broiler MORT also had moderate heritability (0.13), which suggests that MORT and FE1 can be improved through selection without negatively impacting other important traits. Selection of heavier maternal GROW also may decrease offspring MORT.

Key words: early mortality, feed efficiency, genetic correlation, growth traits

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INTRODUCTION

In addition to production and feed efficiency traits, a commercially important trait in broiler chicken is mortality (**MORT**). Through genetic improvement, management, and nutrition improvements over the last decades, the mortality of broilers has reduced from 18 to 4.8% since 1925 (National Chicken Council, 2016). Growth rate and feed efficiency also increased (Buzala and Janicki, 2016). Whereas mortality has decreased significantly in the industry, efforts are still focused on reducing this further. Mortality rate first peaks ≤1 week after hatch, then a second peak gradually comes after week 7 (McNaughton et al., 1978; Tabler et al., 2004).

In this study we had access to 3 disorder traits and MORT. All 3 disorders are primary indicators on broiler mortality and are recorded in discrete categories; therefore, classified as categorical traits. To properly account for that, multi-trait threshold models were used as it could improve genetic analyses of categorical traits (Gianola and Foulley, 1983; Gilmour et al., 1985; Janss and Foulley, 1993).

Recently, genotypes from high-density SNP chips have been widely studied as a way to improve accuracy of genetic evaluations for continuous and categorical traits. Lourenco et al. (2015a) showed that using genomic information in genetic evaluation of commercial

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Table 1. Summary of bird-based statistics for growth and efficiency traits.

Trait	Unit	n	n, genotyped	Mean[1]
GROW	g	161,984	17,998	8.583
FE1	g	41,730	16,188	0.002
DT	%	7,087	537	13.421
FE2	g	41,730	16,188	4.950

GROW: growth trait; FE1: feed efficiency trait 1; DT: dissection trait; FE2: feed efficiency 2.
[1]Standardized mean.

broilers increased accuracy compared to traditional evaluations. In addition, predictions of breeding values for growth traits benefited from genomic data for young birds of both sexes (Liu et al., 2014). Accuracy of genetic evaluation for MORT in chickens increased as well (Zhang et al., 2015).

The objectives of this study were to 1) use a multi-trait linear-threshold model to evaluate the genetic associations of underlying MORT with growth, efficiency traits, and disorder traits in broiler chickens, thereby verifying if selection for performance is correlated to MORT, and 2) compare the ability to predict future breeding values for all studied traits when genomic information is included in multi-trait models.

MATERIALS AND METHODS

Data

Cobb-Vantress, Inc. (Siloam Springs, AR) provided data for purebred broiler chickens from 20 overlapping mating groups (**MG**) from multiple breeder flocks. After data edits, 186,596 birds with at least 1 record for any of 8 traits were present in the data set. Pedigree data were available for 188,936 birds. In total, 420 contemporary groups (**CG**) were defined by combining effects of age of hen and rearing pens. Four continuous traits related to growth (**GROW**), feed efficiency (**FE1** and **FE2**), and a dissection trait (**DT**), and 4 categorical traits related to MORT and disorders (**DIS1**, **DIS2**, and **DIS3**) were evaluated.

Summary statistics for the continous traits are in Table 1. The GROW was recorded for 161,984 birds, whereas DT was recorded only for 7,087 male birds. The FE1 and FE2 were measured within a set time period, and each trait had 41,703 birds. The multiple measurements were combined into a unique value for each trait and then analyzed as a single record per trait.

Categorical traits MORT, DIS1, and DIS2 were classified as 1 (alive or normal) or 2 (dead or abnormal). Table 2 shows numbers of birds and incidence rates for the 4 traits. Mortality (n = 180,998; dead = 7.5%) was recorded from hatch through phenotypic grading of GROW and DT. DIS1 and DIS2 were ascertained in a total of 163,971 and 59,124 birds, respectively. Further, random samples of birds were sent each week for dissection where DIS2 and DIS3, as well as DT were recorded. The DIS3 was scored as 1 (normal) to 7 (severe disorder) (R. L. Sapp, Cobb-Vantress, Inc., Siloam Springs, AR, personal communication). Out of 16,870 dissected birds, 65.86% were normal for DIS3, and 13.4 and 8.1% were scored 1 and 2, respectively; scores of 4 through 7 had very low incidence. Because of this low incidence, the DIS3 categories were reduced to 3 (i.e., 1, 2, and 3 to 7).

Genomic data were obtained for 18,047 birds using a moderate-density (60k) Illumina (San Diego, CA) SNP BeadChip for chicken (Groenen et al., 2011). For quality control of genomic data, SNP were retained if call rate was >0.9, minor allele frequency was >0.05, and departure from Hardy-Weinberg equilibrium (difference between expected and observed frequency of heterozygosity) was <0.15. In addition, SNP with an unknown position or located on a sex chromosome were excluded from analysis. After edits, 38,609 autosomal SNP remained for analysis.

Statistical Models

Before evaluating the relationships between performance traits (growth and efficiency) and mortality and disorder traits, all traits were modeled individually. The single-trait models were then combined in an 8-trait multivariate analysis.

Linear models were used for growth and efficiency traits (GROW, FE1, DT, FE2), and threshold models were used for the categorical traits (MORT, DIS1, DIS2, and DIS3). Linear models for all traits included a fixed effect for CG and a random additive direct genetic effect. Additive maternal genetic and maternal permanent environmental effects were added for GROW. Sex

Table 2. Numbers of birds and incidence rates for mortality and disorder traits.

			Category incidence rates (%)					
			1		2		3 to 7	
Trait	n	n, genotyped	All	Geno-typed	All	Geno-typed	All	Geno-typed
MORT[1]	180,998	18,045	92.5	100.0	7.5	0.0	—	—
DIS1[1]	163,971	18,017	98.8	99.8	1.2	0.2	—	—
DIS2[1]	59,124	18,045	96.5	92.4	3.5	7.6	—	—
DIS3[2]	14,840	417	74.9	77.4	15.3	15.0	9.8	7.6

MORT: mortality; DIS1: disorder trait 1; DIS2: disorder trait 2; DIS3: disorder trait 3.
[1]Categories: 1 = alive or normal; 2 = dead or abnormal.
[2]Categories: 1 to 7.

was included as a fixed effect for all traits except DT. The full form of the linear models was

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_1\mathbf{a} + \mathbf{Z}_2\mathbf{m} + \mathbf{Z}_3\mathbf{p} + \mathbf{e},$$

where $\mathbf{y}$ is a vector of phenotypes; $\mathbf{b}$, $\mathbf{a}$, $\mathbf{m}$, and $\mathbf{p}$ are vectors of fixed effects, additive direct genetic effect, additive maternal genetic effect**,** and maternal permanent environmental effect, respectively; $\mathbf{X}$, $\mathbf{Z}_1$, $\mathbf{Z}_2$ and $\mathbf{Z}_3$ are incidence matrices for $\mathbf{b}$, $\mathbf{a}$, $\mathbf{m}$, and $\mathbf{p}$, respectively; and $\mathbf{e}$ is a vector of random residuals.

For the categorical traits, the threshold model assumed an underlying distribution $\mathbf{L}$ of the categorical traits (MORT, DIS1, DIS2, and DIS3) with the similar effects as linear model. The model accounted for the fixed effects of sex and MG, whereas random effects were additive direct genetic and CG. The model was

$$L = \mathbf{Xb} + \mathbf{Za} + \mathbf{Wc} + \mathbf{e},$$

where $\mathbf{L}$ is a vector of underlying distribution of phenotype $\mathbf{y}$; $\mathbf{b}$ is a vector of fixed effects; $\mathbf{a}$ is a vector of random additive direct genetic effects; $\mathbf{c}$ is a vector of random CG effects; $\mathbf{X}$, $\mathbf{Z}$, and $\mathbf{W}$ are incidence matrices for $\mathbf{b}$, $\mathbf{a}$, and $\mathbf{c}$, respectively; and $\mathbf{e}$ is a vector of random residuals. The response $\mathbf{y}$ was modeled with the following distribution:

$$f(y|L) = \prod_{i=1}^{n} I(L_i < t_1)I(y_i = 1) + I(t_1 < L_i < t_2) \times I(y_i = 2) + I(t_2 < L_i < t_3)I(y_i = 3),$$

where n is the number of records; $\mathbf{t_1}$, $\mathbf{t_2}$, and $\mathbf{t_3}$ are thresholds that define the three categories of response and $\mathbf{I}$ is an indicator function that takes value 1 if the condition specified is true, otherwise the value is 0. The procedure is a nonlinear transformation of best linear unbiased estimate and best linear unbiased prediction (**BLUP**).

Genetic components were assumed to be correlated, whereas random CG effects were assumed to be uncorrelated. Residual components were also assumed to be uncorrelated, except for the disorder trait that was recorded along with GROW; e.g., if birds were dissected to confirm the disorder status, then only healthy chickens (category 1) which were not dissected had records of traits that were measured afterwards. For categorical traits, heritabilities were reported on the liability scale.

Estimation of Breeding Values and Validation

To evaluate usefulness of genomic information in predicting future breeding values for performance, MORT, and disorder traits, realized accuracies were assessed by twofold cross-validation as described by Ramirez-Valverde et al., 2001. The multi-trait analysis was run using the estimated (co)variance components from traditional BLUP of estimated breeding values (**EBV**) and from single-step genomic BLUP of genomic EBV. The general mixed-model equation for single-step genomic Genomic BLUP (GBLUP) was

$$\begin{bmatrix} \mathbf{X}'\mathbf{X} & \mathbf{X}'\mathbf{Z} \\ \mathbf{Z}'\mathbf{X} & \mathbf{Z}'\mathbf{Z} + \lambda\mathbf{H}^{-1} \end{bmatrix} \begin{bmatrix} \mathbf{b} \\ \mathbf{u} \end{bmatrix} = \begin{bmatrix} \mathbf{X}'\mathbf{y} \\ \mathbf{Z}'\mathbf{y} \end{bmatrix},$$

where $\mathbf{y}$ is a vector of phenotypic records in a multi-trait scenario; $\mathbf{X}$ and $\mathbf{Z}$ are incidence matrices that correspond to fixed effects and additive genetic effects, respectively; $\mathbf{b}$ is a vector of fixed effects; $\mathbf{u}$ is the vector of random additive direct genetic effects; λ is the ratio of residual to additive genetic variances; $\mathbf{H}^{-1}$ is the inverse of a matrix that combines pedigree and genomic relationships (Aguilar et al., 2010); and $\mathbf{e}$ is the vector of residual effects, which is assumed to be independent and have a normal distribution [$e \sim$ N(0, $I\sigma_e^2$)]. This general mixed-model was modified to incorporate maternal genetic effect and maternal permanent environmental effect for GROW.

Because a few dead animals were genotyped, the traditional validation techniques did not apply. Therefore, models were compared using a data-splitting technique (Ramirez-Valverde et al., 2001) based on the correlation of EBV from 2 samples that did not overlap, each with half of the phenotypes selected across CG. The correlations were computed only for genotyped animals in the last generation, were averaged across 10 replicates, and are measures of realized accuracy.

This study considered only selected models with a focused discussion from a larger study. For a broader description, see Zhang (2015).

Computation and Software

The AIREMLF90 program (Misztal et al., 2002) was used to estimate the variance components of the single-trait linear models with a convergence criterion of 10^{-12}. The THRGIBBS1F90 program (Tsuruta and Misztal, 2006) was used to estimate variance components of the single-trait and multi-trait threshold models. The POSTGIBBSF90 program (Tsuruta and Misztal, 2006) was used to check convergence and to calculate posterior means. The burn-in size ranged from 5,000 to 150,000 depending on the trait. Traditional and genomic EBV were computed using BLUPF90 (Misztal et al., 2002) with the convergence criterion set to 10^{-14} and THRGIBBS1F90 with an option to store solutions.

RESULTS

Variance components and heritability estimates from the single-trait models are in Table 3 for the growth and efficiency traits and in Table 4 for MORT and disorder traits. Correlations and heritability estimates for

Table 3. Means (± SE) of genetic parameters for growth and efficiency traits using single-trait linear models.

Statistic	GROW	FE1	DT	FE2
σ_a^2	49.25 ± 4.57	10.87 ± 0.56	0.01 ± 0.00	27.79 ± 1.56
σ_m^2	8.53 ± 2.46	—	—	—
σ_{am}	−7.41 ± 2.67	—	—	—
σ_p^2	11.66 ± 1.43	—	—	—
σ_e^2	181.23 ± 2.40	31.50 ± 0.41	0.01 ± 0.00	96.99 ± 1.16
h^2	0.20 ± 0.02	0.26 ± 0.01	0.48 ± 0.03	0.22 ± 0.01
h_m^2	0.04 ± 0.01	—	—	—

GROW: growth trait; FE1: feed efficiency trait 1; DT: dissection trait; FE2: feed efficiency trait 2.

σ_a^2, additive direct genetic variance; σ_m^2, additive maternal genetic variance; σ_{am}, animal-maternal covariance; σ_p^2, maternal permanent environmental variance; σ_e^2, residual variance; h^2, direct heritability; h_m^2, maternal heritability.

Table 4. Posterior means (± SD) of genetic parameters for mortality and disorder traits using single-trait threshold models.

Statistic	MORT	DIS1	DIS2	DIS3
σ_a^2	0.14 ± 0.01	0.31 ± 0.02	0.53 ± 0.05	0.45 ± 0.06
σ_c^2	0.03 ± 0.00	0.09 ± 0.02	0.06 ± 0.01	0.04 ± 0.01
σ_e^2	1.00 ± 0.01	1.00 ± 0.01	1.00 ± 0.01	1.04 ± 0.02
h_L^2	0.12 ± 0.01	0.22 ± 0.02	0.33 ± 0.02	0.29 ± 0.02

MORT: mortality; DIS1: disorder trait 1; DIS2: disorder trait 2; DIS3: disorder trait 3.

σ_a^2, additive direct genetic variance; σ_c^2, contemporary group variance; σ_e^2, residual variance; h_L^2, heritability on the liability scale.

the multi-trait model are in Table 5. For the continuous traits (Table 3), heritability was highest (0.48) for DT and moderate for FE2 (0.22), FE1 (0.26), and GROW (0.20). For maternal GROW, heritability was low (0.04). For the categorical traits (Table 4), heritability estimates were 0.12 for MORT, 0.22 for DIS1, 0.33 for DIS2, and 0.29 for DIS3. Residual variances were very close to 1 for all categorical traits, which suggests reliable estimates.

Multi-trait heritability estimates (Table 5) for the continuous traits were similar to single-trait estimates except for direct and maternal GROW. The multi-trait heritability for GROW (0.26) was higher than the single-trait heritability (0.20); multi-trait heritability for maternal GROW (0.08) was twice as high as single-trait heritability (0.04). Multi-trait heritability estimates for MORT, DIS1, and DIS2 also were similar to those from single-trait threshold analyses. For DIS3, however, multi-trait heritability (0.19) was lower than single-trait heritability (0.29).

Genetic correlations between continuous traits were generally weak (≤0.28) except between direct and maternal GROW, which was moderate and negative (−0.53). Genetic correlations between GROW and FE2 (0.28) and FE1 and FE2 (0.22) were slightly stronger than correlations between other growth/efficiency traits. Genetic correlations among disorder and mortality traits also were weak except between MORT and DIS1 (0.77). This result is most likely due to the fact that all DIS1 birds that are affected are also considered dead. Genetic correlations between continuous and categorical traits varied greatly. Moderate positive correlations were found between GROW and DIS1 (0.27), FE2 and DIS1 (0.25), and GROW and DIS3 (0.23). Maternal GROW had a moderate negative correlation with MORT (−0.50) and DIS1 (−0.37).

Realized accuracies in the last generation for genotyped animals are shown in Figure 1 and Supplementary Table S1. The realized accuracy was lowest (0.40) for DIS1 evaluations without genomic information and highest (0.81) for GROW evaluations with genomic information. The gain in realized accuracy as a result of genotyping broilers averaged 0.17 for performance traits and 0.07 for mortality and disorder traits. The largest gain in realized accuracy obtained when including genomic information compared to traditional evaluation was for FE1 (0.25), whereas the smallest gain was for DIS2 (0.03). Although DIS2 had the highest heritability among categorical traits (0.34, Table 5), the incidence rate for genotyped animals was low (7.6%) as was the total number of birds (59,124). Among the categorical traits, DIS1 had the greatest gain in realized accuracy (0.10), although this gain is still small compared with gains for performance traits.

DISCUSSION

In threshold model using maximum likelihood method for binary or categorical response variable,

Table 5. Genetic correlations (above diagonal) and heritabilities (on diagonal) from the multi-trait threshold-linear model for continuous growth and efficiency traits and categorical mortality and disorder traits.

Trait	GROW	$GROW_m$	FE1	DT	FE2	MORT	DIS1	DIS2	DIS3
GROW	**0.26**	−0.53[a]	0.00	−0.12[a]	0.28[a]	0.13	0.27[a]	0.17[a]	0.23[a]
$GROW_m$		**0.08**	0.09	0.11	0.03	−0.50[a]	−0.37[a]	0.00	−0.13[a]
FE1			**0.25**	0.14[a]	0.22[a]	0.01	0.08[a]	0.01	0.18[a]
DT				**0.50**	0.00	0.04	−0.06	−0.01	−0.15[a]
FE2					**0.21**	0.14[a]	0.25[a]	−0.03	−0.10
MORT						**0.13**	0.77[a]	−0.02	0.10
DIS1							**0.24**	0.02	0.08
DIS2								**0.34**	0.11
DIS3									**0.19**

GROW: growth trait; $GROW_m$: growth maternal trait; FE1: feed efficiency trait 1; DT: dissection trait; FE2: feed efficiency 2; MORT: mortality; DIS1: disorder trait 1; DIS2: disorder trait 2; DIS3: disorder trait 3.

[a]Different ($P < 0.05$) from 0 by 2 SD.

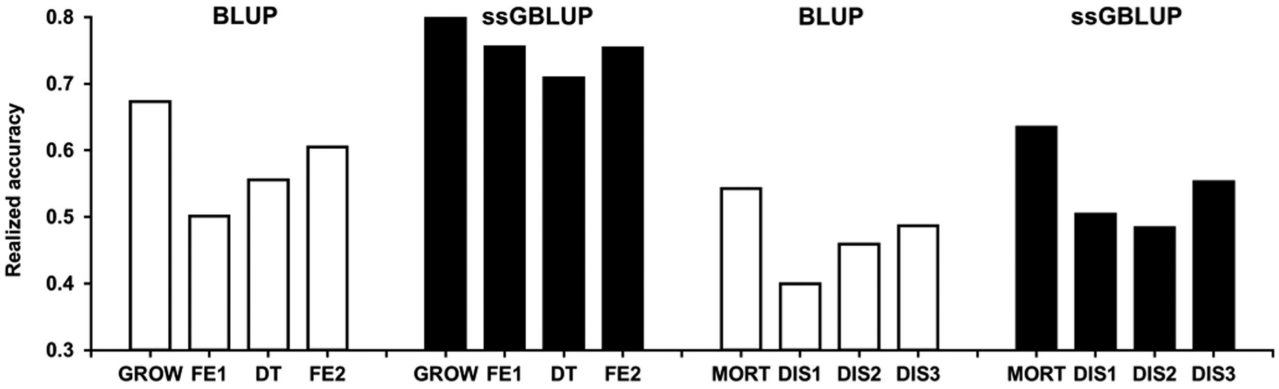

Figure 1. Realized accuracy of estimated breeding values from best linear unbiased prediction (BLUP) and of genomic estimated breeding values from single-step genomic BLUP (ssGBLUP) using multi-trait linear-threshold models. GROW: growth trait; FE1: feed efficiency trait 1; DT: dissection trait; FE2: feed efficiency 2; MORT: mortality; DIS1: disorder trait 1; DIS2: disorder trait 2; DIS3: disorder trait 3.

heritability tends to be biased upward when the amount of information per fixed effect is small (Hoeschele and Tier, 1995; Moreno et al., 1997; Tempelman, 1998). This is also denoted as extreme category problem (**ECP**) (Misztal et al., 1989), were only "0" or "1" observational value emerges at a certain level of a fixed effect. In the full dataset of the current study, MG has at least 2,651 samples at one level, sex has at least 26,788 samples at one level, and CG has at least 39 samples at one level for a single binary or categorical trait. When splitting the data randomly in half, CG would have more serious ECP. The small sample size for levels of CG was not a problem in the current study since 1) the data was split by CG, guaranteeing that in each subset each level contains all samples, 2) it was treated as random effect with a Gaussian distribution so the bias in Monte Carlo error, auto-correlations, and variance estimates would be decreased (Hoeschele and Tier, 1995; Moreno et al., 1997; Luo et al., 2001).

Multi-trait models are expected to have higher heritability estimates than single-trait models because of additional genetic information from links with other traits. This was the case for direct and maternal GROW, DT, MORT, DIS1, and DIS2 but not for DIS3. Heritability estimates with the multi-trait model for FE1, FE2, and DSI1 were almost identical to those from single-trait analyses. Heritability differed most for DIS3, for which the single-trait estimate was remarkably higher than the multi-trait estimate. However, DIS3 was the only categorical trait with rather small numbers of observations per CG. In addition, a slightly higher SE was observed for the single-trait DIS3 variance components, which suggests that the estimated heritability may have been overestimated.

Continuous Traits (Growth and Efficiency)

The GROW heritability estimates from both single-trait and multi-trait were smaller than the estimate of 0.33 reported by Rekaya et al. (2013) but comparable to the estimates of 0.17 to 0.25 reported by Chen et al. (2011) measured at 6 weeks of age in Cobb-Vantress commercial lines. However, maternal effect was considered in this study, and it also accounted for part of the genetic variation. The heritability for FE1 was close to the estimate of 0.26 reported by Rekaya et al. (2013), and the DT heritability was higher than the estimate of 0.39 reported by Liu et al. (2014) on an intercross commercial line. In previous studies of feed efficiency recorded during 5 to 6 weeks of age on unselected chicken (Aggrey et al., 2010; González-Cerón et al., 2015), heritabilities ranged from 0.19 to 0.51, and estimates from this study were close to those estimates.

The genetic correlation between direct and maternal GROW was moderately negative but lower than the correlation estimated by Maniatis et al. (2013) with a similar model. Furthermore, GROW had no genetic correlation with FE1, which was expected because FE1 was adjusted for GROW. Similarly, DT was a measurement related to GROW, and GROW was slightly negatively correlated with DT, which differed from the correlation of 0.20 reported by de Greef et al. (2001), with both traits recorded at 35 days of age. A weak genetic correlation was estimated between GROW and FE2. A small positive relationship (NS) was found between FE1 and DT. A weak positive genetic correlation was found between FE1 and FE2, which was smaller than the correlation of 0.27 reported by González-Cerón et al. (2015). Selection for higher FE1 could potentially result in greater FE2. No genetic correlation was found between DT and FE2.

Categorical Traits (Mortality and Disorders)

Heritability estimates for all binomial traits (MORT, DIS1, and DIS2) were almost identical for both models. However, single-trait heritability estimates for DIS3 were remarkably higher than from the multi-trait model. The heritability for MORT was lower than what was reported in other studies at up to 7 weeks of age (Pakdel et al., 2002; González-Recio et al., 2008). Heritability differences among studies could be a

consequence of differences in definitions used by various researchers, animal age at measurement, sample size, and statistical and computational strategies used for estimation (Rekaya et al., 2013).

The estimates for DIS1 heritabilities agreed with other findings. Pakdel et al. (2002) reported that the heritability of continuous traits related to disorders varied from 0.18 to 0.47. Although the incidence of DIS2 was low, heritabilities on the liability scale were higher than estimates reported by other studies. The most similar estimate was a DIS2 heritability of 0.27 for commercial-broiler breeder lines recorded on males only at 5 weeks of age with a prevalence of 7.8% using a linear animal model (Kapell et al., 2012).

Genetic correlations between disorder traits were generally negligible, except between MORT and DIS1. Selection against DIS1 might reduce MORT in broiler chickens. de Greef et al. (2001) reported a genetic correlation of 0.9 between MORT and disorder traits related to MORT, which is similar to the value of 0.77 found in this study.

Genetic Correlations between Continuous and Categorical Traits

With the multivariate model, 4 continuous and 4 categorical traits were simultaneously evaluated to provide the combined distribution information and thus less biased compared to separated distributions. The genetic correlation between GROW and MORT did not differ significantly ($P > 0.05$) from 0, which was probably caused by bias introduced by data truncation. de Greef et al. (2001) reported a moderate negative genetic correlation of -0.46 ± 0.11 between MORT and GROW at 35 days of age. Although the genetic correlation in this study was negligible, the genetic correlation between maternal GROW and MORT was moderate and negative (-0.50; $P < 0.05$) and revealed the role of maternal genetics in hen mortality.

Genetic associations between GROW and other disorder traits (DIS1, DIS2, and DIS3) were low and positive; selection for GROW may slightly impair health. Pavlidis et al. (2007) reported genetic correlations of 0.28 and 0.24 between GROW at 21 days of age and disorder traits linked to MORT in susceptible and resistant lines selected against disorders, respectively, which implies that selection on disorder traits reduces GROW. Other studies (Pakdel et al., 2005; Zerehdaran et al., 2006) have reported negative genetic correlations (-0.23 to -0.37) between GROW and disorder indicator traits, which suggests a positive relationship between GROW and susceptibility to disorders. Closter et al. (2012) found that the genetic correlation between GROW and a disorder indicator trait changed from slightly positive to moderately negative from 2 to 7 weeks of age and that the change was more pronounced in males than in females, an indication that males and females should be studied separately.

In this study, FE1 and FE2 had a nonsignificant ($P > 0.05$) genetic correlation with MORT and a weak genetic correlation with some of the disorder traits; those traits likely are not affected by selection for performance traits. The correlation between MORT and DT was low in this study, but DT was only recorded in one gender and the sample size was very small (7,087 birds). Although high DT is widely considered to affect phenotypic health traits negatively, few studies have examined the genetic correlations between those traits. de Greef et al. (2001) reported a genetic correlation of 0.02 ± 0.01 between MORT and dissection traits, which was close to the correlation of 0.04 ($P > 0.05$) in this study.

Zavala et al. (2011) suggested examining losses on growth traits and some disorders to determine the cause of MORT because disorders that change physiological condition at the time of death are highly related to MORT. For this study, the major cause of MORT was considered to be DIS1; other MORT factors may include maternal effect from hens, management, and sex. According to Zavala et al. (2011), female and male broilers have different causes and rates of MORT. Higher MORT rates in broiler chicks up to 8 weeks of age has been found to be correlated with younger age of hen at laying and lighter egg weight (McNaughton et al., 1978).

Realized Accuracy

The use of genomic information increased the accuracy of predicting future breeding values for all 8 traits, especially performance traits. An average gain of 18 percentage points (0.76 vs. 0.58) over traditional BLUP EBV was observed for all performance, whereas the gain for MORT and disorders was 7 percentage points (0.54 vs. 0.47). Increasing accuracy is important for both sets of traits, but the ability to predict future breeding values for MORT or disorder traits is of greatest interest. Of the disorder traits, DIS1 had the lowest EBV accuracy when genomic information was not included, probably because this trait has more importance than other disorder traits included in the evaluation. Lourenco et al. (2015a) reported lower realized accuracy for highly selected traits. The small increase in accuracy for MORT, DIS2, and DIS3 can be explained by the lower incidence rate, especially among genotyped animals, as only live animals were genotyped. A low incidence rate has also been related to small gains in accuracy for American Angus calving ease (Lourenco et al., 2015b). González-Recio et al. (2008) showed a doubling of the realized accuracy for MORT (0.2 and 0.1 with and without genomic information, respectively) when evaluating 200 genotyped male broilers based on progeny information in a cross-validation study where MORT had an incidence of 5%. Proportionally bigger gains have been observed in small genotyped populations (VanRaden et al., 2009).

Performance traits that are heavily selected for in broiler chickens were weakly correlated with MORT and disorder traits; selecting for heavier and more efficient animals will likely not increase the incidence of MORT and disorders. Furthermore, offspring MORT can be reduced by selecting for heavier maternal GROW. Genetic correlations between MORT and disorder traits showed that disorders affect mortality with different intensities. The ability to predict future breeding values for performance, MORT, and disorder traits increased when genomic information was available. Although the increase was more evident for performance traits, genomic information was also extremely valuable for MORT and disorder traits.

STATEMENT

Research on live animals in this article met the guidelines approved by the institutional animal care and use committee (IACUC).

SUPPLEMENTARY DATA

Supplementary data are available at *Poultry Science* online.

Table S1. Realized accuracy of estimated breeding values from best linear unbiased prediction (BLUP) and of genomic estimated breeding values from single-step genomic BLUP (ssGBLUP) using multi-trait linear-threshold models. GROW: growth trait; FE1: feed efficiency trait 1; DT: dissection trait; FE2: feed efficiency 2; MORT: mortality; DIS1: disorder trait 1; DIS2: disorder trait 2; DIS3: disorder trait 3.

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