

Effective Factors on Genetic Parameters of Day-Old Chicken Body Weight in a Commercial Broiler Line

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Abstract: Genetic and non-genetic factors on day-old chicken body weights (BWT1) were investigated in a commercial broiler line with restricted maximum likelihood (REML) procedures. Ten different animal models were used to estimate the genetic parameters. All models consisted of the direct additive genetic effect but differed in combinations of maternal additive genetic, maternal environmental, generation-hatch-sire interaction (GHS) and covariance between direct and maternal genetic effects (cov_{am}). When cov_{am} was considered in the models, higher estimations of direct and maternal genetic variances were obtained that could be because of negative covariance between these effects. Also adding GHS factor in analysis reduced the cov_{am} . Although GHS variance represented only 5% of the phenotypic variance, but ignoring it in the models made up 70% of negative covariance between direct and maternal genetic effects. Based on the full model, direct heritability (h^2_a), maternal heritability (h^2_m), maternal environmental variance as a proportion of the phonotypic variance (c^2), GHS variance as a proportion of the phonotypic variance (s^2) and direct-maternal additive genetic correlation (r_{am}) were estimated 0.02, 0.411, 0.107, 0.046 and -0.168, respectively.

Key words: Genetic Parameters % Day-old Chicken % Body Weight % Commercial Broiler Line¹

INTRODUCTION

Considering the maternal effects in biometrical models causes to obtain better estimations of (co)variance components and genetic parameters for production and reproduction traits in poultry and ignoring this effect in statistical models tend to overestimate direct additive genetic variance and heritability [1, 2]. In addition, genetic correlation between direct and maternal additive genetic effects (r_{am}) was reported to be negative. This negative correlation has been observed for different traits in other domestic species [3-5]. Those researchers showed that when the data, containing sire by year interaction effect (SY) were analysed by ignoring the SY, the direct and maternal variances were inflated and a negative covariance was observed between these effects. But ignoring the SY for the data without significant interaction effect has not changed the genetic parameters. In a study on body weight at 6 weeks in a commercial broiler line, a reduction in direct and maternal heritability has been reported when fitting SY effect in the analysis model [2]. In the present study, the data from a commercial broiler

line were used to explore the consequences of including random effects, such as maternal and generation-hatch-sire interaction (GHS) effects on the estimates of genetic parameters for day-old chicken's body weight.

MATERIAL AND METHODS

The data included day-old chicken's body weights information from a commercial maternal broiler line. The main breeding goal of this line is to evaluate the reproductive traits such as egg number, age at sexual maturity and egg weight. The day-old chick weights were recorded randomly on the 50% of the selected birds. The structure of the data is summarized in Table 1. The structure of the data is summarized in Table 1.

To investigate the maternal effects on day-old chickens' body weight (BWT1) and the effect of generation-hatch-sire interaction (GHS) on maternal models, 10 different animal models were fitted. The animal models in matrix notations are presented in Table 2. In these models, y is a vector of observations,

Table 1: The structure of data

Information	Record
Number of generations	15
Number of pedigree records	67365
Number of animals with records	34430
Trait mean \pm standard deviation (gr)	46.28 \pm 4.37
Coefficient variation (%)	9.44
Minimum value (gr)	30
Maximum value (gr)	62

Table 2: Matrix forms of the studied models

Models Num.	Models Matrix Notations	
1	$y = Xb + Z_1a + e$	
2	$y = Xb + Z_1a + Wc + e$	
3	$y = Xb + Z_1a + Z_2m + e$	$cov_{am} = 0$
4	$y = Xb + Z_1a + Z_2m + e$	$cov_{am} \dots 0$
5	$y = Xb + Z_1a + Z_2m + Wc + e$	$cov_{am} \dots 0$
6	$y = Xb + Z_1a + Z_2m + Wc + e$	$cov_{am} \dots 0$
7	$y = Xb + Z_1a + Z_2m + Ss + e$	$cov_{am} = 0$
8	$y = Xb + Z_1a + Z_2m + Ss + e$	$cov_{am} \dots 0$
9	$y = Xb + Z_1a + Z_2m + Wc + Ss + e$	$cov_{am} = 0$
10	$y = Xb + Z_1a + Z_2m + Wc + Ss + e$	$cov_{am} \dots 0$

b is a vector of fixed effects (Generation-Hatch, Sex and Dam age), a is an unknown random vector of direct additive genetic effect, m is an unknown random vector of maternal additive genetic effect, c is an unknown random vector of maternal environmental effect, s is an unknown random vector of GHS effect and e is an unknown random vector of residuals. X , Z_1 , Z_2 , W and S are known incidence matrices relating observations to b , a , m , c and s , respectively. Cov_{am} is the covariance between direct and maternal additive genetic effects. Estimates of genetic parameters and (co)variance components were obtained by restricted maximum likelihood (REML) method, using the DFREML software [6]. Determination of superiority of one model over another was made by likelihood ratio test. This test is based on the chi-square (X^2) distribution with k degrees of freedom, where k is equal to the number of additional parameters in the more complex model [7]. This way, X^2 can be described as

$$c^2 = -2(LogL_{M_i} - LogL_{M_j})$$

Where, $LogL_{M_i}$ and $LogL_{M_j}$, are the likelihoods of the full model and the residual model, respectively.

RESULTS AND DISCUSSION

Estimates of (co)variance components and genetic parameters for day-old chickens body weight (BWT1) with logarithm of likelihood ratio from each animal model are shown in Table 3. Based on model 1, direct additive genetic variance and direct heritability for BWT1 were estimated 11.21 and 0.770, respectively. Adding maternal environmental (model 2) and maternal additive genetic (model 3) effects in model 1 caused to significant reduction in additive genetic variance and its heritability ($p<0.01$). Direct heritability (h^2_a) by using models 2 and 3 were 0.217 and 0.069, respectively. Based on these models, maternal environmental variance as a proportion of the phenotypic variance (c^2) and maternal heritability (h^2_m) were estimated 0.398 and 0.485, respectively. Comparison the three models indicated that ignoring maternal effects in the analysis tended to overestimate direct additive genetic variance and heritability.

According to Table 3, introducing the covariance between direct and maternal additive genetic effects (model 4), increased the direct and maternal additive genetic variances by approximately 30% and 11% in comparison to model 3, respectively ($p<0.01$). This increase is because of negative covariance (-0.96) between direct and maternal additive genetic effects. Estimations of 0.091 and 0.542 were obtained for h^2_a and h^2_m , respectively, by using model 4. Comparing model 5 with model 3 showed that omitting maternal environmental effect in the analysis of BWT1 tended to overestimate the maternal additive genetic variance (c^2_m) and heritability (h^2_m). Maternal heritability (h^2_m) was estimated 0.371 with model 5, while it was 0.485 by model 3. When we put covariance between direct and maternal additive genetic effects in model 5 (model 6), a significant increase ($p<0.01$) was observed in logarithm of likelihood ratio. Using model 6, h^2_a , h^2_m and c^2 were estimated 0.092, 0.433 and 0.085, respectively. Comparing models 1 and 6 clearly showed that maternal additive genetic variance has the most affective effect on day-old chickens' body weight.

As shown in Table 3, generation-hatch-sire interaction (GHS) effect was calculated for 5% of the phenotypic variance. Introducing the GHS effect reduced direct heritability in all corresponding models by approximately 64 to 78%. Fitting GHS effect in full model ($a + m + c + cov_{am}$) resulted in the lowest negative estimates of direct-maternal correlation in all corresponding models (Table 3). Whereas set of the GHS

Table 3: Estimates of (co)variance components and genetic parameters for day-old chicken body weight (BWT1)

Model	\mathbf{s}_a^2	\mathbf{s}_c^2	\mathbf{s}_s^2	\mathbf{s}_m^2	\mathbf{s}_{am}^2	\mathbf{s}_e^2	\mathbf{s}_p^2	h^2_a	c^2	s^2	h^2_m	r_{am}	LogL
1	11.21	-	-	-	-	3.35	14.55	0.770	-	-	-	-	-56442/02
2	2.94	5.39	-	-	-	5.22	13.55	0.217	0.398	-	-	-	-54988/71
3	0.97	-	-	6.79	-	6.25	14.01	0.069	-	-	0.485	-	-54756/69
4	1.27	-	-	7.58	-0.96	6.09	13.98	0.091	-	-	0.542	-0.31	-54749/39
5	0.963	1.42	-	5.06	-	6.20	13.64	0.071	0.104	-	0.371	-	-54732/18
6	1.27	1.16	-	5.93	-0.70	6.04	13.71	0.092	0.085	-	0.433	-0.26	-54728/70
7	0.32	-	0.68	6.82	-	6.06	13.89	0.023	-	0.049	0.492	-	-54400/30
8	0.45	-	0.68	7.32	-0.57	5.99	13.88	0.033	-	0.049	0.528	-0.31	-54397/33
9	0.25	1.46	0.67	5.57	-	6.08	14.02	0.018	0.104	0.048	0.397	-	-54376/56
10	0.27	1.50	0.65	5.76	-0.21	6.06	14.03	0.020	0.107	0.046	0.411	-0.17	-54375/46

\mathbf{s}_a^2 Direct Additive Genetic Variance, \mathbf{s}_c^2 Maternal Environmental Variance, \mathbf{s}_s^2 Generation-Hatch-Sire Interaction Effect, \mathbf{s}_m^2 Maternal Additive Genetic Variance, \mathbf{s}_{am}^2 Covariance between Direct and Maternal Additive Genetic Effects, \mathbf{s}_e^2 Residual Variance, \mathbf{s}_p^2 Phenotypic Variance, h^2_a Direct Heritability, c^2 Maternal Environmental Variance as a Proportion of the Phenotypic Variance, s^2 Generation-Hatch-Sire Interaction as a Proportion of the Phenotypic Variance, h^2_m Maternal Heritability, r_{am} Correlation between Direct and Maternal Additive Genetic Effects, LogL Logarithm of Likelihood Ratio.

effect without fitting maternal environmental effect (model 8 = a + m + s + cov_{am}) could not decrease in negative direct-maternal genetic correlation in comparison to model 4 (a + m + cov_{am}). A comparison between models 6 and 10 showed that although GHS had a minor proportion of phenotypic variance, but ignoring it from the model made up 70% of negative covariance between direct and maternal genetic effects.

Many studies demonstrated the importance of maternal effect on production and reproduction traits in poultry. Hartmann *et al.* [8] reported an intermediate (0.5) maternal heritability (h^2_m) for chick weight, whereas the direct heritability was close to zero. This result is in line with the finding of Koerhuis *et al.* [9] who found substantial variation in chick weight due to the dam. Prado-Gonzalez *et al.* [10] showed that direct heritability (h^2_a) was low for body weight of Mexican Creole chickens during rearing. They also showed that maternal additive and environmental were not important source of variation after 4 weeks of age in Creole chickens. But in a study on Iranian native fowls, Seraj *et al.* [1] reported that maternal additive genetic and environmental effects with the covariance between direct and maternal additive genetic effects were important for body weight at 8 weeks. In these studies, the estimation of direct-maternal genetic correlation (r_{am}) was negative. Different reasons such as adaptation of a species to a trait optimum emerging as a result of natural selection [11], decreased of egg shell quality, inattention to the maternal effect during previous generations [12], linkage disequilibrium and pleiotropic effects of the same gene [13] were suggested for this negative correlation. Negative correlation between direct and maternal additive genetic effects has been observed

in other domestic species. Lee and Pollak [4] reported that if sire by year (SY) interactions are detected, then they are true effects, not spurious results due to incorrect direct and maternal covariance. According to these authors, the SY effect is either a true interaction, perhaps caused by different environmental factors associated with different years, or indication of confounding of sire effects with other unidentified sources of covariance between progeny records in the same year.

CONCLUSION

The results of present study indicated that ignoring maternal effects in the analysis of BWT1 tended to overestimate direct additive genetic variance and heritability. With considering the cov_{am} in the models, higher estimations of direct and maternal genetic variances were obtained that could be because of negative covariance between these effects. The investigations showed that negative estimates of cov_{am} are not only because of genetic antagonism, but also because of the influence of GHS interaction effects. Comparison between different models of this study indicated that although GHS had a small part of phenotypic variance (5%), but 70% of negative covariance between direct and maternal genetic effect resulted in ignoring this effect from the analysis model.

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