

Direct and Maternal Genetic Effects for Ascites-Related Traits in Broilers

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ABSTRACT The objective of the present study was to estimate heritabilities for ascites-related traits in broilers and to assess the importance of maternal genetic effects for these traits. Several traits related to ascites were measured on more than 4,000 broilers kept under cold conditions. Heritabilities were estimated using an animal model with a direct genetic effect and a model with direct and maternal genetic effects. Estimated heritabilities from the direct genetic effects model were 0.46 for hematocrit value, 0.42 for BW, 0.47 for right ventricular weight, 0.46 for total ventricular weight, 0.45 for ratio of right ventricu-

lar weight to the total ventricular weight, 0.32 for total mortality, and 0.18 for fluid accumulation in the heart sac. Maternal effects significantly influenced the traits BW, total ventricular weight, and total mortality. Direct and maternal heritabilities, respectively, for BW were 0.21 and 0.04, for total ventricular weights were 0.29 and 0.03, and for total mortality were 0.16 and 0.05. The heritability estimates for ascites-related traits and the significance of maternal genetic effects for most of these traits indicate that direct and maternal genetic effects play an important role in the development of the ascites syndrome.

(*Key words:* broiler, ascites, heritability, cold stress, maternal genetic effect)

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INTRODUCTION

In all major poultry-producing countries, cases of ascites in broilers have been reported. Ascites syndrome, or water belly, is an increase in the amount of lymph normally found in the peritoneal space (Julian, 1993). This syndrome is a serious economic concern because it results in a loss of broilers (Shapiro, 1993) and has a negative impact on animal welfare. In a survey (Maxwell and Robertson, 1997), information on 18 countries from four continents showed that ascites affects 4.7% of all live broilers worldwide.

Genetically, the modern broiler seems to be more prone to develop ascites, which is probably due to selection for growth rate or feed conversion ratio, which puts high demands on metabolic processes and on the oxygen demand (Decuyper et al., 2000). To alter the metabolic load placed on the birds, the producer can reduce the growth rate, change the diet, or change other aspects of the management system, e.g., by raising the temperature (e.g., Bendheim et al., 1992). Alternatively it might be possible to select for birds that can maintain a high rate of growth without succumbing to the penalties imposed on their health and, consequently, on their welfare. This requires alternative selection strategies, traits that indicate the sus-

ceptibility of birds to ascites, and genetic parameters for these traits. Several studies have suggested traits that can be used as indicator traits for ascites. Lubritz et al. (1995) clearly demonstrated that ascites is strongly related to the ratio of right ventricular weight (RV) to the total ventricular weight (TV). Shlosberg et al. (1996) showed that broilers with high hematocrit values have an increased chance of developing ascites when exposed to cold temperatures and suggested that hematocrit values might be a useful selection tool. Maxwell et al. (1998) indicated that in the presence of ascites, plasma troponin T, an indicator of heart muscle damage, is heritable. Moghadam et al. (2001) showed that heart defects, e.g., pulmonary hypertension, right ventricular failure, and fluid accumulation in the peritoneal cavity, are heritable and have a positive genetic correlation with BW. De Greef et al. (2001) estimated genetic parameters for a number of ascites-related traits. However, they also demonstrated that genetic parameters varied considerably with the severity of the disease.

Koerhuis and Tompson (1997) reported maternal genetic effects on juvenile broiler BW. Other studies have shown positive phenotypic effects of egg weight on juve-

Abbreviation Key: ABDOMEN = fluid in the abdomen; BREAST = color of the breast; HCT = hematocrit value; HEART = fluid in the heart sac; LIVER = liver abnormalities; MORT-TOT = total mortality; RV = right ventricular weight; %RV = right ventricular weight as a percentage of BW; RV:TV = ratio of right ventricular weight to the total ventricular weight; TV = total ventricular weight; %TV = total ventricular weight as a percentage of BW.

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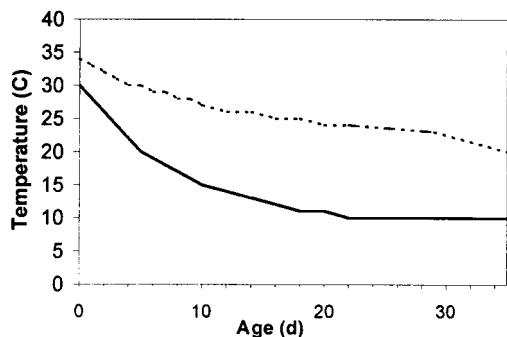


FIGURE 1. The normal temperature schedule (---) and the cold-stressed temperature schedule (—).

nile broiler BW (e.g., Chambers, 1990). Further, Dewil et al. (1996) demonstrated that selection for ascites resistance is linked to several physiological variables at the embryonic stage. These results suggest that maternal genetic effects might play a role in the susceptibility of birds to ascites. To our knowledge no studies have actually established the significance of maternal genetic effects on ascites-related traits. The objective of the current study was to estimate heritabilities for ascites-related traits and to assess the importance of maternal genetic effects for these traits in broilers.

MATERIALS AND METHODS

Birds and Traits

Birds. Ascites-related traits were recorded for 4,202 chickens, 1,736 females and 2,466 males. The experimental population was the result of a cross between two genetically different outcross broiler dam lines (Hybro) originating from the White Plymouth Rock breed. The maternal line had a relatively high reproductive performance and was fast-feathering, and the paternal line had a relatively high growth performance and was slow-feathering. The total pedigree file consisted of 5,096 birds of which 36 were pure-line birds (F_0), 29 were F_1 birds, 829 were F_2 birds, and 4,202 were F_3 birds. Observations were on F_3 birds. A more detailed description of the experimental set up was given by Van Kaam et al. (1998).

The experimental birds were hatched at 6 different wk in 1994 and 1995. Broilers were kept in four different pens; however, most were kept in one pen. Nine batches were allotted by hatching day and pen number. In order to identify individuals that were susceptible to ascites, a cold stress temperature schedule was applied (Figure 1). At the time of hatching, the temperature was 30 C and then was gradually decreased to 10 C by 22 d of age. The temperature remained at 10 C until the end of the experiment. A normal temperature schedule starts at 33 to 34 C and then gradually decreases to 17 to 18 C by 35 d of age. Except for the adjusted temperature schedule, birds were kept under circumstances that closely resemble commercial practice, i.e., a standard commercial feed,

artificially lighted housing for 23 h/d, and group housing with 20 birds/m².

Traits. The hematocrit value (HCT) and BW of birds were measured 1 d before slaughtering at 5 wk of age. After birds were slaughtered, several ascites-related traits were measured. Livers and hearts were removed and visually inspected. Liver abnormalities (LIVER) were scored as follows: 0 represented no abnormalities observed, 1 represented an abnormal liver, and 2 represented serious liver abnormalities. Liver abnormalities were a lighter color, an irregular liver surface, or both. Accumulation of fluid in the heart sac (HEART) was scored with 0 if no fluid had accumulated, 1 if fluid accumulation was observed, and 2 if there was serious accumulation of fluid in the heart sac. Further, the weights of RV and TV were measured. From these measurements, the RV:TV, RV as a percentage of total BW (% RV), and TV as percentage of total BW (%TV) were derived. The accumulation of fluid in the abdomen (ABDOMEN) was scored as 0, 1, or 2. A score of 0 indicated no fluid, 1 indicated the presence of fluid in the abdomen, and 2 indicated a serious accumulation of fluid in this section.

For color of the breast (BREAST), a score of 0 represented normal color, 1 represented a color deviation, and 2 represented serious color deviation. In general, a deviation of breast color meant that the color was more red. Also, the total mortality (MORT-TOT) of the birds was recorded as 0 or 1. A score of 0 represented a bird that was alive at the end of the experiment, and a score of 1 indicated a bird that died before the end of the experiment. For the first three batches of birds, mortality was not recorded. No observations were recorded for other traits of birds died before the end of experiment.

In addition, information from 795 birds kept under a normal temperature schedule (Figure 1) was available. These birds originated from the same F_2 parents and, except for temperature, were kept under similar conditions as the birds kept under cold conditions. These birds were slaughtered between 6 and 7 wk, and the measurements for BW, HCT, RV, TV, and RV:TV were available. These birds were previously used in an experiment by Van Kaam et al. (1998). The data was only used to describe the mean and the distribution of traits under a normal temperature schedule and was not included in genetic analyses.

Genetic Analyses

An animal model including maternal genetic effects was used to calculate direct heritabilities and maternal genetic heritabilities (Model 1):

$$Y_{ijklmn} = \mu + \text{sex}_i + \text{feather}_j + \text{batch}_k + \text{group}_l + d_m + a_n + e_{ijklmn}$$

where Y_{ijklmn} = the dependent variable on chicken n from dam m of sex i, feathering class j from batch k in group l; sex_i = fixed effect of sex i (I = 1,2 female or male); feather_j = fixed effect of feathering j (j = 1,2 fast or slow); batch_k

= fixed effect of batch k of the birds ($k = 1, 2, \dots, 9$), classes were formed based on a combination of hatching day and pen; group_1 = fixed effect of group ($l = 1, 2, \dots, 46$), classes were based on the age of dam and the hatching day of the experimental animals; d_m = random genetic effect of dam m ; a_n = random direct genetic effect of individual n ; e_{ijklmn} = random residual effect.

In matrix notation, $\mathbf{y} = \mathbf{X}\mathbf{b} + \mathbf{Z}_1\mathbf{a} + \mathbf{Z}_2\mathbf{m} + \mathbf{e}$ where \mathbf{y} = vector of observations, \mathbf{b} = vector of fixed effects, \mathbf{a} = vector of direct genetic effects, \mathbf{m} = vector of maternal genetic effects, and \mathbf{e} = vector of residual effects. \mathbf{X} = incidence matrix relating observations to fixed effects. \mathbf{Z}_1 and \mathbf{Z}_2 = incidence matrices relating observations to random effects \mathbf{a} and \mathbf{m} , respectively. The covariance structure for this model was

$$V \begin{bmatrix} \mathbf{a} \\ \mathbf{m} \\ \mathbf{e} \end{bmatrix} = \begin{bmatrix} \mathbf{A}\sigma_a^2 & \mathbf{A}\sigma_{am} & 0 \\ \mathbf{A}\sigma_{am} & \mathbf{A}\sigma_m^2 & 0 \\ 0 & 0 & \mathbf{I}\sigma_e^2 \end{bmatrix}$$

where σ_a^2 = direct genetic variance, σ_m^2 = maternal genetic variance, σ_e^2 = error variance, and σ_{am} = covariance between direct genetic effects and maternal genetic effects. Estimates of variance components were obtained using the ASREML software (Gilmour et al., 2000).

An animal model without a maternal genetic effect (Model 2) was also used:

$$Y_{ijkln} = \mu + \text{sex}_i + \text{feather}_j + \text{batch}_k + \text{group}_l + a_n + e_{ijkln}$$

Fixed effects in this model were identical to fixed effects in Model 1. To test the significance of the maternal genetic effect, a likelihood ratio test with two degrees of freedom was used:

$$\chi_2^2 = 2\log_e L(F) - 2\log_e L(R)$$

where $L(F)$ = likelihood of the full model (Model 1), and $L(R)$ = likelihood of the residual model (Model 2).

In addition to the analyses described, score traits were also analyzed with a binary model and ASREML software (Gilmour et al., 2000). Fixed and random effects in this model were identical to the effects in Model 2. Because some traits were scored using three classes (0, 1, and 2), scores in classes 1 and 2 were combined into one class.

RESULTS

Description of Traits

Means and standard deviations for the traits measured under cold conditions are presented in Table 1. The average weight of broilers at 5 wk was 1,604 g, and total mortality in the current experiment was 16%. Figure 2 shows the distribution of HCT, RV, and RV:TV under cold and normal conditions. Under cold conditions the mean and SD HCT of the birds was $35.4 \pm 4.2\%$ and 28.3

$\pm 2.3\%$ under normal conditions. This difference in means, as well as in variance, between traits measured in normal or cold conditions is clearly illustrated in Figure 2. Mean values and SD for other ascites-related traits under normal conditions were 1.15 g (± 0.30) for RV, 5.60 g (± 0.94) for TV, and 20.64% (± 4.66) for RV:TV.

Sex had significant effects on all traits analyzed, except for BREAST and LIVER. The most pronounced effects of sex were found for BW, RV:TV, MORT-TOT, and ABDOMEN. The average weight of males was 1,676 g, whereas this value was 1,503 g for females. For RV:TV the average for males was 29.3%, and for females it was 26.0%. The MORT-TOT in males was 17.7% and in females 14.2%, and the mean value of ABDOMEN in males was 0.10 and in females was 0.04.

Feathering had a significant effect on all continuous traits analyzed; however, no significant effects of feathering on score traits were found. The most pronounced effects of feathering were found for HCT and RV:TV. The average HCT for slow-feathering birds was 35.7% and for fast-feathering birds it was 34%. Also RV and TV were higher for slow-feathering birds, but the increase in RV was higher than the increase in TV, which resulted in a higher RV:TV ratio for slow-feathering birds. The average of the RV:TV ratio in slow-feathering birds was 28.2% and in fast-feathering birds it was 25.9%.

Genetic Parameters

The genetic parameters for ascites-related traits from Models 1 and 2 are presented in Table 2. For continuous traits, heritabilities obtained using Model 2 were around 0.45, but for score traits, heritabilities were lower, especially for ABDOMEN, BREAST, and LIVER. For most of the continuous traits, the maternal genetic effects model (Model 1) gave a significantly better fit. Heritabilities for maternal genetic effects were low (0.02 to 0.05). Heritabilities for direct genetic effects of the continuous traits under Model 1 ranged from 0.2 to 0.3. Genetic correlations between direct genetic effects and maternal effects showed moderately positive values, but the high SD of the estimates indicated that this parameter could not be accurately estimated. For the traits HCT, RV, BREAST, and HEART, convergence problems were encountered when using the maternal genetic effects model. For these traits, no estimates are reported for Model 1.

Additional analyses for binary traits were performed using a binary model. Estimated heritabilities using this model were higher than heritabilities reported in Table 2. Using a binary model without a maternal genetic effect resulted in heritability estimates for the direct genetic effect of 0.46 (± 0.07) for MORT-TOT, 0.49 (± 0.09) for ABDOMEN, 0.47 (± 0.12) for BREAST, 0.36 (± 0.10) for LIVER, and 0.23 (± 0.06) for HEART.

DISCUSSION

Susceptibility to Ascites

In the present experiment, traits related to ascites were analyzed. Birds that were most susceptible to ascites prob-

TABLE 1. Statistical description of the traits in cold conditions

Trait	Abbreviation	Number	Mean	SD
Hematocrit value (%)	HCT	3,547	35.40	4.21
BW at 5 wk (g)	BW	3,693	1,604	263
Right ventricular weight (g)	RV	3,660	1.95	0.68
Total ventricular weight (g)	TV	3,658	6.97	1.17
Ratio of right ventricular weight to total ventricular weight (%)	RV:TV	3,658	27.94	8.07
Right ventricular weight as percentage of BW (%)	%RV	3,646	0.125	0.050
Total ventricular weight as percentage of BW (%)	%TV	3,644	0.439	0.070
Total mortality ¹	MORT-TOT	2,494	0.16	0.37
Fluid in the abdomen ²	ABDOMEN	3,697	0.08	0.38
Color of the breast ²	BREAST	3,697	0.03	0.18
Liver abnormalities ²	LIVER	3,697	0.07	0.29
Fluid in the heart sac ²	HEART	3,696	0.59	0.62

¹Trait scored as 0 or 1. The number of birds for each class: $N_0 = 2,095$ and $N_1 = 399$.

²Trait scored as 0, 1, or 2. The number of birds for each class: fluid in the abdomen: $N_0 = 3,542$, $N_1 = 23$, and $N_2 = 132$; color of the breast: $N_0 = 3,585$, $N_1 = 110$, and $N_2 = 2$; liver abnormalities: $N_0 = 3,488$, $N_1 = 170$, and $N_2 = 39$; and fluid in the heart sac: $N_0 = 1,781$, $N_1 = 1,653$, and $N_2 = 262$.

ably died before the end of the experiment. However, no information was available with respect to the cause of death, and therefore MORT-TOT did not fully identify

the birds that were most susceptible to ascites. Also no measurements for HCT, RV:TV, and other traits were available on the birds that died before 5 wk of age.

Ascites is defined as accumulation of fluid in the abdominal cavity and therefore is best described by the trait ABDOMEN. Birds with a score of 1 or 2 for ABDOMEN showed ascites symptoms and, therefore, also could be qualified as susceptible toward ascites. However, these birds were less susceptible than birds that actually died due to ascites. The third category of birds that could be distinguished is birds that have not yet accumulated fluid in the abdomen but have high values for RV:TV, HCT, or both.

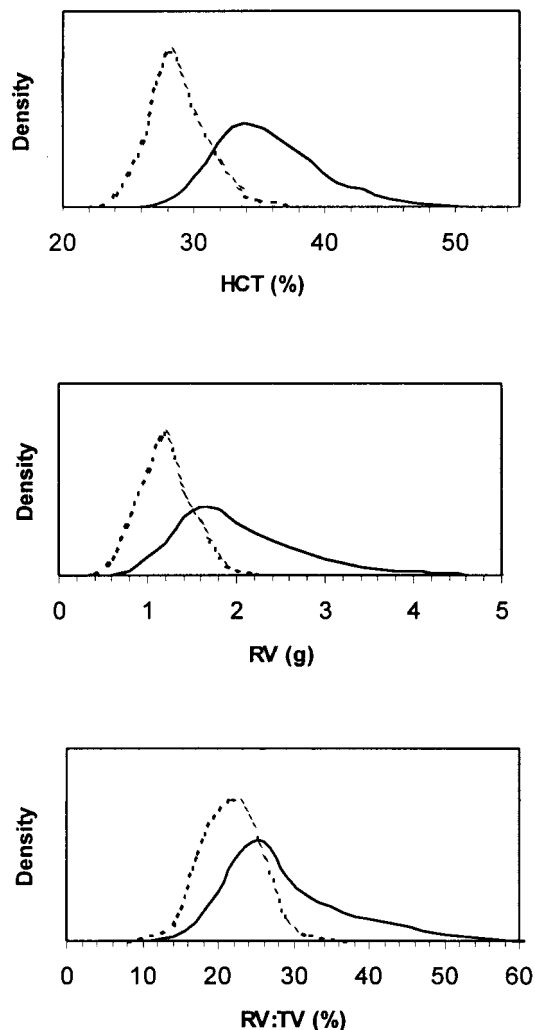


FIGURE 2. The distribution of hematocrit value (HCT), right ventricular weight (RV) and ratio of right ventricular weight to total ventricular weight (RV:TV) in normal (-----) and cold (—) conditions.

Effect of Cold Stress, Sex, and Feathering

Cold stress increases metabolic demands for oxygen and elevates HCT values and RV:TV (Shlosberg et al., 1992; Lubritz and Mcpherson, 1994). In the present study, cold stress had a marked effect on the mean values for HCT, RV, and RV:TV as well as on the variance of these traits. These results indicate that birds react to the cold stress by increased values for HCT and RV. Apparently, the adaptive capacity of some of the birds is insufficient to meet the increased oxygen demands and results in increased incidence of ascites.

In the present study, birds under cold conditions (5 wk) were younger than those under normal conditions (6 to 7 wk). Therefore, results might be influenced by an effect of age. From the results of Shlosberg et al. (1992), it can be concluded that there is no significant difference between HCT values at 5, 6, and 7 wk of age. However, there is a significant increase of RV:TV from Weeks 5 to 7. In the current study, the younger birds under cold condition (5 wk) showed higher RV:TV than the older birds under normal conditions (6 to 7 wk). These results emphasize that cold conditions markedly increased RV:TV.

In the present study, total mortality was recorded, but no information was available about mortality due to ascites. In comparison to the rate of mortality under normal

TABLE 2. Genetic parameters of ascites-related traits using a maternal genetic effects model (Model 1) or a direct genetic effects model (Model 2)

Trait ¹	Model 1 ²			Model 2	Likelihood ratio test ³
	h_a^2	r_{am}	h_m^2	h_a^2	
HCT	NC ⁴	NC	NC	0.46 (0.05) ⁵	NC
BW	0.21 (0.06)	0.57 (0.40)	0.04 (0.02)	0.42 (0.05)	13.76**
RV	NC	NC	NC	0.47 (0.05)	NC
TV	0.29 (0.07)	0.30 (0.40)	0.03 (0.03)	0.46 (0.05)	9.14*
RV:TV	0.28 (0.06)	0.90 (0.43)	0.02 (0.02)	0.45 (0.05)	6.72*
%RV	0.22 (0.06)	0.86 (0.31)	0.04 (0.02)	0.45 (0.05)	12.44**
%TV	0.31 (0.07)	0.44 (0.43)	0.03 (0.02)	0.46 (0.05)	7.53*
MORT-TOT	0.16 (0.07)	0.21 (0.45)	0.05 (0.04)	0.32 (0.06)	8.50*
ABDOMEN	0.07 (0.03)	0.00 (0.00)	0.00 (0.01)	0.08 (0.03)	0.24
BREAST	NC	NC	NC	0.03 (0.02)	NC
LIVER	0.06 (0.03)	0.05 (0.75)	0.00 (0.00)	0.08 (0.03)	0.52
HEART	NC	NC	NC	0.18 (0.04)	NC

¹HCT = hematocrit value; RV = right ventricular weight; TV = total ventricular weight; RV:TV = ratio of right ventricular weight to total ventricular weight; %RV = right ventricular weight as percentage of BW; %TV = total ventricular weight as percentage of BW; MORT-TOT = Total mortality; ABDOMEN = Fluid in the abdomen; BREAST = Color of the breast; LIVER = Liver abnormalities; and HEART = Fluid in the heart sac.

² h_a^2 = heritability of direct genetic effect; r_{am} = genetic correlation between direct and maternal effects; h_m^2 = heritability of maternal genetic effect.

³Test if Model 1 fit better than Model 2.

⁴NC = convergence problems.

⁵Empirical standard errors are in parentheses.

* $P \leq 0.05$.

** $P \leq 0.01$.

conditions (4 to 5%), mortality under cold conditions was relatively high (16%). Therefore, it could be concluded that cold stress resulted in higher mortality, which was likely due to ascites.

Higher mean values for ascites-related traits in male broilers suggest that males are more prone to develop ascites. These results confirm previous reports that ascites is more prevalent in male broilers (e.g., Fredric, 1988). Also higher mean values for ascites-related traits in slow-feathering birds suggest that under cold conditions, slow-feathering birds are more susceptible to ascites.

Maternal Genetic Effects

The heritabilities for continuous traits related to ascites under the cold condition indicated that direct genetic effects play an important role. The significance of maternal genetic effects for most of the continuous traits suggested that maternal effects play a role in the development of the ascites syndrome. However, maternal genetic effects might be partly confounded with dominance effects, especially if maternal genetic effects were estimated based on full-sib family relationships. In the presence of additive genetic, maternal genetic, and dominance effects, resemblance of full sibs is $\frac{1}{2}\sigma_a^2 + r_a\sigma_a\sigma_m + \sigma_m^2 + \frac{1}{4}\sigma_d^2$ where σ_d^2 = variance due to dominance effects. In the current study, however, information regarding maternal genetic effects is also based on maternal half-sib family relationships. Resemblance between maternal half-sibs is $\frac{1}{2}\sigma_a^2 + r_a\sigma_a\sigma_m + \sigma_m^2$ and not affected by dominance effects.

To investigate the presence of dominance effects, additional analyses were performed. In these analyses, a ran-

dom full-sib family effect was included in the model in addition to additive genetic and maternal genetic effects. This effect should account for additional resemblance between full-sib family members due to dominance effects. Analyses showed that the variance component of full-sib families was very small and not significant for all traits. This finding suggests that the significantly better fit of Model 1 was due to true maternal genetic effects.

Egg characteristics such as shell conductance or shell porosity, volume of air cell, and weight or the composition of the egg might influence the early embryonic development and, thereby, susceptibility toward ascites. Chineme et al. (1995) indicated that interference with eggshell conductance during incubation can increase postnatal HCT. Other findings suggest possible effects of hatching conditions, length of incubation, and interval between internal pipping (start of breathing) and hatching on the incidence of ascites (Visschedijk, 1968a,b; Coleman and Coleman, 1991; Dewil et al., 1996). These effects might be due to maternal effects or due to direct effects of the embryo itself.

Clément et al. (2001) demonstrated that if maternal genetic effects exist but are neglected in the model, the direct heritability is overestimated. Sometimes this results in a direct heritability that is more than twice the true direct heritability (Clément et al., 2001). In the current study, large increases in the direct heritability were found for traits with a significant maternal genetic effect when this factor was neglected from the model (Model 2). For BW, the heritability of the direct genetic effect increased from 0.21 under Model 1 to 0.42 under Model 2. This finding indicated that heritabilities that have been re-

ported for some of the ascites-related traits are likely to be overestimates of the true heritability.

Literature

De Greef et al. (2001) estimated heritabilities for some ascites-related traits on 2,788 chicks in cold-stressed conditions. Heritability estimates were 0.50 for HCT, 0.15 for ABDOMEN, 0.41 for RV, 0.54 for RV:TV, 0.17 for liver cirrhosis, 0.57 for BW, and 0.22 for MORT-TOT. These estimates are in agreement with the results reported using Model 2. Also Lubritz et al. (1995) estimated heritabilities for two ascites-related traits using a paternal half-sib model in all-male populations ($n = 3,436$) originating from three different lines. Heritability estimates for ABDOMEN ranged from 0.11 to 0.44, and for RV:TV estimates ranged from 0.21 to 0.27. The estimate for ABDOMEN using Model 1 was lower (0.07), but for RV:TV the estimates are similar.

In the present study, information on F_3 individuals was used to estimate genetic parameters for base population. If the two pure lines differed with respect to allele frequencies of genes involved in the traits under study, it was expected that the genetic variation in the F_2 and F_3 would be increased. Therefore, heritability estimates in the present study might be somewhat higher when compared to previously reported estimates.

Score Traits

For discrete or score traits, heritabilities might be underestimated using a linear model, especially when the incidences are low (Gianola, 1982). In the current experiment, estimated heritabilities for score traits were especially low for traits for which a majority of individuals had a score of 0 and therefore a low mean value. When the data were analyzed using a binary model, the estimated heritabilities for these traits increased. The heritability estimates from a binary model were in agreement with estimates that were obtained by transforming the heritabilities from the linear model to the underlying scale (Lynch and Walsh, 1998).

Prospects

The present study showed that some of the ascites-related traits were significantly influenced by maternal genetic effects. Heritability estimates for ascites-related traits suggested that selection for these traits is possible. However, selection should be done, taking into account maternal genetic effects. Before a selection strategy to reduce the incidence of ascites can be implemented, correlations among traits measured in the present study and genetic correlations between production traits measured under commercial circumstances and under cold-stressed circumstances are required.

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