Genetic and phenotypic relationships between blood gas parameters and ascites-related traits in broilers

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ABSTRACT Ascites, also called pulmonary hypertension syndrome, is a metabolic disorder in chickens that have an insufficient pulmonary vascular capacity. The tendency of broilers to develop ascites is heritable, and successful selection against this susceptibility would benefit from good and easy-to-measure indicator traits. Blood gas parameters have been suggested as indicator traits for ascites susceptibility. Therefore, the aim of the present study was to estimate the heritability of blood gas parameters and the genetic and phenotypic correlations between blood gas parameters, heart ratio (postmortem indicator for ascites), and BW at 2 different ages. For this purpose, blood gas parameters, including the partial pressure of carbon dioxide in venous blood (pvCO₂), the partial pressure of oxygen in venous blood (pvO₂), and blood oxygen saturation, were measured at an average age of 22 d in nearly 3,000 broilers. To challenge the resistance of the birds to ascites, they were kept under cold conditions. Heritability for heart ratio was 0.43, and the heritability estimates were low: 0.02 for pvCO₂, 0.03 for pvO₂, and 0.07 for blood oxygen saturation. The estimated heritability for pH was 0.15, for bicarbonate was 0.19, and for total carbon dioxide content was 0.19. The genetic correlations between heart ratio and total carbon dioxide content (0.31 ± 0.15) and between heart ratio and bicarbonate (0.31 ± 0.15) were moderate and positive. For pvO₂, the genetic correlation with heart ratio was stronger and negative (−0.62 ± 0.21); however, this correlation could not be estimated accurately because of the low heritability of pvO₂. For pvCO₂, the genetic correlation with the heart ratio was close to zero (−0.04 ± 0.45). Phenotypic correlations between traits were, in general, similar to the genetic correlations. Heritabilities for blood gas parameters and the genetic correlations between blood gas parameters and the heart ratio estimated in the present study do not support the suggestion that blood gas parameters measured during wk 3 or 4 are useful traits to select against the susceptibility for ascites.

Key words: broiler, ascites, blood gas parameter, heritability, correlation

INTRODUCTION

Ascites, also called pulmonary hypertension syndrome, is a metabolic disorder in chickens. The disorder is associated with an insufficient pulmonary vascular capacity and results in right ventricular failure (Julian et al., 1987; Julian, 1998; Balog et al., 2000). In most cases, ascites is caused by a disproportion between the oxygen requirement and the cardiovascular ability to supply oxygen (Julian and Mirsalimi, 1992; Scheele et al., 1992; Decuyper et al., 2000). Oxygen shortage puts pressure on the pulmonary vascular system and can lead to oxygen deficiency in the tissues, which will increase pulmonary arterial pressure. The high blood pressure and high work load of the heart lead to fluid accumulation in the abdominal cavity and eventually death (Shlosberg et al., 1992; Decuyper et al., 2000; Havenstein et al., 2003). Mortality caused by ascites ranges from 5 to 8% in populations worldwide and can be as great as 20 to 30% in heavier broiler flocks (Balog, 2003; Pavlidis et al., 2007).

The increase in the occurrence of ascites has been linked to genetic selection for increased growth rate, greater meat yield, and lower feed conversion ratio (Decuyper et al., 2000; Balog, 2003). It has been shown that fast-growing broilers are more susceptible to ascites than slow-growing broilers (Julian, 1993). Ascites in broiler flocks can be reduced by management measures, such as avoiding low temperatures, maintaining good air quality and high oxygen concentrations, and restricting feeding to restrict growth (Decuyper...
et al., 2000; Julian, 2000; Balog, 2003). A variety of physiological studies have evaluated specific traits as indicators for ascites susceptibility. Two of the most common clinical signs associated with ascites are right ventricular hypertrophy and fluid accumulation in the abdominal cavity (Decuyper et al., 2000; Moghadam et al., 2001; Balog et al., 2003; Pakdel et al., 2005a; Zerehdaran et al., 2006). The ratio of right to total ventricular weight (RATIO), which measures right ventricular hypertrophy, has been suggested as a good indicator for ascites (Julian, 1993; McGovern et al., 1999; Pakdel et al., 2005a).

Studies have shown genetic variation within lines (Wideman and French, 1999; Wideman et al., 1999; Deeb et al., 2002; Pakdel et al., 2002) and between lines (Lubritz et al., 1995; Buys et al., 1999a,b; Wideman and French, 2000; De Greef et al., 2001; Druyan et al., 2007, 2008) for susceptibility to ascites. However, current indicator traits, such as RATIO and fluid accumulation in the abdominal cavity, can only be measured postmortem. Therefore, selection against ascites susceptibility by using these indicators is complicated, and information for selection relies heavily on information from relatives (McMillan and Quinton, 2002; Pakdel et al., 2005a). Thus, there is a need for alternative indicator traits that can be measured on living birds; blood gas parameters might be a good alternative. It has been shown that broilers with right ventricular failure have significantly lower blood oxygen saturation (sO2) compared with broilers with a normal heart (Julian and Mirsalimi, 1992). Wideman et al. (2003) found that chickens with an elevated RATIO had a greater partial pressure of oxygen in arterial blood, and greater bicarbonate (HCO3) concentrations in arterial blood compared with chickens with a normal RATIO. Furthermore, by comparing 2 different broiler lines, Schaele et al. (2003) observed a relationship between ascites susceptibility and high pvCO2 at d 11 in juvenile chickens and suggested that ascites could be eliminated by selecting for low pvCO2. Navarro et al. (2006) demonstrated that sO2 is heritable and suggested that ascites susceptibility could be decreased by selecting for increased sO2 values. Druyan et al. (2007) reported a moderate heritability for sO2 and indicated that sO2 might serve as an indicator in selection against ascites susceptibility, although with limited efficacy.

In addition to the studies by Navarro et al. (2006) and Druyan et al. (2007), to our knowledge, no other studies have reported heritability estimates for blood gas parameters. Furthermore, to our knowledge, only Druyan et al. (2007) reported genetic correlations between blood gas parameters and other ascites indicator traits such as RATIO.

The objective of the present study was to estimate heritability, heart ratio, and genetic and phenotypic correlations between blood gas parameters measured during wk 3 and 4 and BW at 2 different ages in broilers.

### MATERIALS AND METHODS

#### Experimental Population and Phenotyping

**Animal Material.** The experiment was carried out by licensed and authorized personnel under approval of Hendrix Genetics. The experimental population consisted of 5,987 broilers. The chickens were from generations 7 and 8 of an advanced intercross line, which was a cross between 2 genetically different dam lines originating from the White Plymouth Rock breed. The data consisted of 2,413 males, 2,452 females, and 1,122 chickens of unknown gender. Birds from generations 3 of this population have been used in previous studies on ascites and meat quality traits (van Kaam et al., 1998; Pakdel et al., 2002). The chickens in the experiment were kept under a cold temperature regimen to induce ascites. The temperature was 30°C at the time of hatching and was gradually reduced to 10°C at 22 d of age. The temperature remained at 10°C until the end of the experiment when the chickens were 5 wk of age. The chickens were group housed with 20 birds/m², they had ad libitum access to a commercial broiler feed containing 12,970 KJ/kg, and they were exposed to 23 h of light per day during the entire experiment. Except for the temperature schedule applied, the chickens were kept under conditions that closely resemble commercial practice.

Venous blood samples were taken when the chickens were, on average, 22 d old (ranging from 19 to 27 d old). The blood gas parameters measured (GEM Premier 3000, Instrumentation Laboratories, Lexington, MA) were blood pH, pvCO2, and partial pressure of oxygen in venous blood (pvO2). Bicarbonate and total carbon dioxide content (TCO2) were calculated from the pH and pvCO2 by the following equations:

\[
\log \text{HCO}_3 = \text{pH} + \log \text{pvCO}_2 - 7.608, \quad \text{and}
\]

\[
\text{TCO}_2 = \text{HCO}_3 + 0.03 \text{pvCO}_2.
\]

Blood sO2 is an indicator of the percentage of hemoglobin saturated with oxygen at the time of the measurement; pvO2, pH, and HCO3 were used to calculate sO2 with the following equation:

\[
s\text{O}_2 = 100 \frac{X^3 + 150X}{X^3 + 150X + 23400},
\]

where \(X = \text{pvO}_2 \times 10^{\left[(H\text{pH}-7.4)-0.0013(\text{HCO}_3-25)\right]}

The weight of the heart ventricles was determined at 5 wk of age. The RATIO was the weight of the right ventricle as a percentage of the total ventricle weight (TV). The chickens were weighed at 2 wk (BW2) and 5 wk of age (BW5). No postmortem dissection was performed on the animals that died before the end of the experiment; therefore, the cause of death was unknown.
Animals that died before the end of the experiment were assigned a total mortality (MORT-TOT) score of 1 and birds that survived got a score of 0.

**Statistical Analysis.** Genetic parameter estimates were obtained by using ASREML software (Gilmour et al., 2006). To determine the importance of maternal effects, a model without a maternal effect and a model with a maternal environmental effect were used. The following model without a maternal environmental effect was used:

\[ y_{ijkl} = \mu + \text{sex}_i + \text{IHD}_j + \text{date}_k + a_1 + e_{ijkl}, \quad [1] \]

where \( y_{ijkl} \) is the dependent variable of chicken \( ijkl \) of sex \( i \), which is the fixed effect of sex (i = female, male, or unknown); IHD \( j \) is the fixed effect of individual hatching day (\( j = 1, 2, \ldots, 34 \) d at hatching); date \( k \) is the fixed effect for date of blood gas measurement (\( k = 1, 2, \ldots, 37 \)); \( a_1 \) is the random direct genetic effect of individual \( l \) with \( a \sim N(0, \Lambda^{-1}) \); and \( e_{ijkl} \) is the random residual effect with \( e \sim N(0, I) \). The effect date \( k \) was used only in the model for the blood gas parameters.

The second model with a maternal environmental effect was:

\[ y_{ijklm} = \mu + \text{sex}_i + \text{IHD}_j + \text{date}_k + a_1 + d_m + e_{ijklm}, \quad [2] \]

This model is identical to the first model, except for the random maternal environmental effect of dam \( m \) (\( d_m \)) with \( d \sim N(0, I^{-1}) \). The fraction of the variation attributable to maternal environmental effects (\( m^2 \)) was calculated as

\[ m^2 = \frac{\sigma_d^2}{\sigma_a^2 + \sigma_d^2 + \sigma_e^2}. \]

To test the significance of the maternal environmental effect, a likelihood ratio test with 1 df was used:

\[ X^2_1 = 2 \log_e L(F) - 2 \log_e L(R), \]

where \( L(F) \) is the likelihood of the full model (model [2]), and \( L(R) \) is the likelihood of the reduced model (model [1]). Univariate analysis was used to estimate heritabilities and maternal environmental effects. Bivariate analysis was used to estimate genetic and phenotypic correlations between the traits.

Some of the animals died before the end of the experiment and had an observation for only BW2. The animals that died might have been the ones that were most susceptible to ascites, and this selection might have had an impact on the estimated genetic parameters. Selection related to BW2 can be accounted for by performing a multivariate analysis including BW2 (Ouweltjes et al., 1988). Therefore, we also estimated heritabilities by using a bivariate analysis with BW2 as a permanent trait. The effect of selection on genetic correlations was studied by performing a trivariate analysis with BW2 as a permanent trait.

### RESULTS

#### Data Description

Means, SD, and CV of the traits measured under cold stress conditions are presented in Table 1. Of the 5,987 chickens retained for measurement of BW2, 5,222 also had measurements for BW5, 5,155 had measurements for RATIO, and 2,956 chickens were used for measuring blood gas parameters. Mortality recordings were missing for 210 chickens because of the loss of wing bands or because the trait was not recorded.

The average venous blood pH was 7.38, the average \( sO_2 \) was 84%, the average \( pCO_2 \) was 45.4 mmHg, and the average \( HCO_3 \) concentration was 26.88 mmol/L (Table 1). The average BW of broilers under cold stress conditions was 360 g at 2 wk and 1,146 g at 5 wk, and the average RATIO was 25%. The MORT-TOT was 10%. Coefficients of variation were moderate to high for most of the traits (e.g., 14.6% for \( pCO_2 \), 20% for \( pO_2 \), 29.4% for BW2, 18.9% for BW5, and 21.2% for RATIO). However, the CV for pH was very low (0.7%).
**Table 2. Phenotypic variance, heritability (h²), and maternal effect (m²) for model [1] and model [2]**

<table>
<thead>
<tr>
<th>Trait</th>
<th>Phenotypic variance</th>
<th>h² (SE)</th>
<th>h² (SE)</th>
<th>m² (SE)</th>
<th>Significance of log-likelihood test²</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW₁</td>
<td>4,606</td>
<td>0.51 (0.06)</td>
<td>0.15 (0.09)</td>
<td>0.12 (0.04)</td>
<td>0.000</td>
</tr>
<tr>
<td>BW₂</td>
<td>34,280</td>
<td>0.37 (0.05)</td>
<td>0.17 (0.08)</td>
<td>0.07 (0.03)</td>
<td>0.029</td>
</tr>
<tr>
<td>RATIO</td>
<td>25.5</td>
<td>0.43 (0.06)</td>
<td>0.17 (0.07)</td>
<td>0.06 (0.03)</td>
<td>NS</td>
</tr>
<tr>
<td>RV</td>
<td>0.106</td>
<td>0.42 (0.06)</td>
<td>0.02 (0.02)</td>
<td>0.05 (0.01)</td>
<td>0.006</td>
</tr>
<tr>
<td>TV</td>
<td>0.613</td>
<td>0.37 (0.05)</td>
<td>0.02 (0.02)</td>
<td>0.05 (0.01)</td>
<td>0.006</td>
</tr>
<tr>
<td>pH</td>
<td>0.002</td>
<td>0.15 (0.04)</td>
<td>0.00 (0.00)</td>
<td>0.00 (0.00)</td>
<td>NS</td>
</tr>
<tr>
<td>pCO₂</td>
<td>26.19</td>
<td>0.15 (0.04)</td>
<td>0.02 (0.02)</td>
<td>0.05 (0.01)</td>
<td>0.006</td>
</tr>
<tr>
<td>pO₂</td>
<td>93.25</td>
<td>0.03 (0.01)</td>
<td>0.01 (0.00)</td>
<td>0.00 (0.00)</td>
<td>NS</td>
</tr>
<tr>
<td>HCO₃</td>
<td>5.36</td>
<td>0.19 (0.05)</td>
<td>0.02 (0.02)</td>
<td>0.03 (0.01)</td>
<td>NS</td>
</tr>
<tr>
<td>TCO₂</td>
<td>5.80</td>
<td>0.19 (0.05)</td>
<td>0.00 (0.00)</td>
<td>0.01 (0.00)</td>
<td>NS</td>
</tr>
<tr>
<td>sO₂</td>
<td>36.3</td>
<td>0.07 (0.02)</td>
<td>0.01 (0.00)</td>
<td>0.00 (0.00)</td>
<td>NS</td>
</tr>
<tr>
<td>MORT-TOT</td>
<td>0.087</td>
<td>0.05 (0.02)</td>
<td>0.02 (0.01)</td>
<td>0.00 (0.00)</td>
<td>0.006</td>
</tr>
</tbody>
</table>

¹BW₂ = BW at 2 wk; BW₅ = BW at 5 wk; RATIO = ratio of right to total ventricular weight; RV = right ventricular weight; TV = total ventricular weight; pCO₂ = partial pressure of carbon dioxide in venous blood; pO₂ = partial pressure of oxygen in venous blood; HCO₃ = bicarbonate; TCO₂ = total carbon dioxide in venous blood; sO₂ = oxygen saturation in venous blood; MORT-TOT = total mortality.

²Log-likelihood results indicate the significant difference between model [1] and model [2]. A univariate model was used for the estimations. NS = not significant.

**Genetic Analyses**

Phenotypic variance, heritability, and maternal environmental effects for the ascites-related traits obtained from the univariate models are given in Table 2. The heritability for RATIO was 0.43. For some of the blood gas parameters, the heritabilities were close to zero: 0.02 for pCO₂, 0.03 for pO₂, and 0.07 for sO₂. However, for pH, HCO₃, and TCO₂ moderate heritabilities were found: 0.15, 0.19, and 0.19, respectively. The estimated heritabilities for the 2 BW measurements were 0.15 for BW₂ and 0.17 for BW₅.

The traits BW₂, BW₅, TV, pCO₂, and MORT-TOT were significantly affected by maternal environmental effects. The fraction of the total variation explained by maternal environmental effects was 0.05 for pCO₂, 0.12 for BW₂, and 0.07 for pO₂. No significant evidence for the presence of maternal environmental effects was found for the traits RATIO, pH, pO₂, HCO₃, TCO₂, and sO₂. Using a model without a maternal effect gave a heritability estimate of 0.51 for BW₂, of 0.37 for BW₅, and of 0.15 for pCO₂ (Table 2). Bivariate analysis with BW₂ as a permanent trait resulted in slightly greater heritability estimates; they were at maximum 0.03 greater than the heritabilities estimated by using a univariate model (results not shown). The heritability estimates for MORT-TOT were also analyzed by using a binary model (results not shown), and the results increased compared with heritabilities estimated from the linear model. These results were in agreement with heritabilities estimated by transforming the heritabilities from the linear model to the underlying scale (Lynch and Walsh, 1998).

The estimates for genetic correlations (above the diagonal) and the phenotypic correlations (below the diagonal) of the blood gas parameters RATIO, BW, and MORT-TOT are presented in Table 3. The greatest genetic correlation between RATIO as a postmortem indicator for ascites and blood gas parameters was found for pO₂ (−0.62 ± 0.21). However, genetic correlations between pO₂ and other traits have high SE, mainly because of the low heritability for pO₂. The genetic correlations between RATIO and the blood gas parameters TCO₂ (0.31 ± 0.15) and HCO₃ (0.31 ± 0.15) were positive and moderate. For pCO₂ and RATIO, the genetic correlation was close to zero (−0.04 ± 0.45). The genetic correlation between BW₂ and RATIO was 0.19, whereas the genetic correlation between BW₅ and RATIO was −0.18. The genetic correlation between BW₂ and BW₅ was high (0.88). Phenotypic correlations between traits were, in general, similar to the genetic correlations. A trivariate model with BW₂ as a permanent trait had hardly any effect on the estimated genetic correlation between the traits: genetic correlations between RATIO and the blood gas parameters increased from 0.01 to 0.02 when using a trivariate model instead of a bivariate model (results not shown).

**DISCUSSION**

The objective of the present study was to estimate the heritability and genetic and phenotypic correlations between blood gas parameters measured at an average age of 22 d, BW at 2 different ages, and heart ratio in broilers. Body weight and RATIO were measured on 5,987 birds, and the blood gas parameters were measured on a subset of 2,956 birds. The study was performed under cold stress conditions to stimulate the metabolic rate resulting in an increased requirement for oxygen, which is known to increase the incidence of ascites in chickens (Decuyper et al., 2000). To evaluate whether specific blood gas parameters could be used in selecting against ascites susceptibility, we studied the heritability and genetic correlations with RATIO.

**Severity of the Challenge**

Previous studies have indicated that correlations between BW and ascites traits are dependent on the frequency of ascitic birds in the population, and there-
Table 3. The genetic correlations (above the diagonal) and the phenotypic correlations (below the diagonal)\(^1\)

<table>
<thead>
<tr>
<th>Trait</th>
<th>BW(_2) (SE)</th>
<th>BW(_5) (SE)</th>
<th>RATIO (SE)</th>
<th>RV (SE)</th>
<th>TV (SE)</th>
<th>pH (SE)</th>
<th>pCO(_2) (SE)</th>
<th>pO(_2) (SE)</th>
<th>HCO(_3) (SE)</th>
<th>TCO(_2) (SE)</th>
<th>sO(_2) (SE)</th>
<th>MORT-TOT (SE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW(_2)</td>
<td>0.88 (0.10)</td>
<td>0.19 (0.23)</td>
<td>0.52 (0.16)</td>
<td>0.49 (0.26)</td>
<td>0.79 (0.35)</td>
<td>NC</td>
<td>−0.24 (0.43)</td>
<td>0.59 (0.20)</td>
<td>0.58 (0.18)</td>
<td>0.40 (0.29)</td>
<td>−0.63 (0.63)</td>
<td></td>
</tr>
<tr>
<td>BW(_5)</td>
<td>0.75 (0.01)</td>
<td>−0.18 (0.16)</td>
<td>0.23 (0.27)</td>
<td>0.30 (0.23)</td>
<td>0.67 (0.23)</td>
<td>−0.90 (0.89)</td>
<td>0.13 (0.31)</td>
<td>0.45 (0.19)</td>
<td>0.53 (0.18)</td>
<td>0.60 (0.21)</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>RATIO</td>
<td>−0.02 (0.03)</td>
<td>−0.15 (0.03)</td>
<td>0.81 (0.16)</td>
<td>−0.02 (0.10)</td>
<td>0.06 (0.17)</td>
<td>−0.04 (0.45)</td>
<td>−0.62 (0.21)</td>
<td>0.31 (0.15)</td>
<td>0.31 (0.15)</td>
<td>−0.12 (0.20)</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>RV</td>
<td>0.36 (0.03)</td>
<td>0.35 (0.01)</td>
<td>0.76 (0.10)</td>
<td>0.62 (0.17)</td>
<td>0.13 (0.42)</td>
<td>0.00 (0.24)</td>
<td>−0.41 (0.14)</td>
<td>0.41 (0.14)</td>
<td>0.42 (0.20)</td>
<td>0.06 (0.20)</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>TV</td>
<td>0.56 (0.03)</td>
<td>0.71 (0.02)</td>
<td>−0.05 (0.02)</td>
<td>0.60 (0.22)</td>
<td>0.27 (0.90)</td>
<td>−0.60 (0.29)</td>
<td>0.10 (0.20)</td>
<td>0.33 (0.20)</td>
<td>0.32 (0.20)</td>
<td>0.25 (0.23)</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>0.12 (0.03)</td>
<td>0.10 (0.03)</td>
<td>−0.04 (0.03)</td>
<td>−0.02 (0.03)</td>
<td>0.04 (0.22)</td>
<td>NC</td>
<td>−0.08 (0.26)</td>
<td>0.40 (0.30)</td>
<td>0.12 (0.19)</td>
<td>−0.47 (0.17)</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>pCO(_2)</td>
<td>NC</td>
<td>−0.01 (0.03)</td>
<td>0.12 (0.03)</td>
<td>0.13 (0.03)</td>
<td>NC</td>
<td>−0.48 (0.45)</td>
<td>0.69 (0.16)</td>
<td>0.71 (0.15)</td>
<td>−0.82 (0.31)</td>
<td>−0.14 (0.67)</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>pO(_2)</td>
<td>0.01 (0.02)</td>
<td>0.00 (0.02)</td>
<td>−0.06 (0.02)</td>
<td>−0.04 (0.02)</td>
<td>−0.01 (0.02)</td>
<td>0.05 (0.02)</td>
<td>−0.14 (0.21)</td>
<td>−0.59 (0.21)</td>
<td>0.77 (0.13)</td>
<td>−0.58 (0.22)</td>
<td>NC</td>
<td></td>
</tr>
<tr>
<td>HCO(_3)</td>
<td>0.10 (0.03)</td>
<td>0.04 (0.03)</td>
<td>0.15 (0.03)</td>
<td>0.15 (0.03)</td>
<td>0.09 (0.03)</td>
<td>0.21 (0.02)</td>
<td>0.58 (0.02)</td>
<td>−0.14 (0.02)</td>
<td>NC</td>
<td>−0.11 (0.22)</td>
<td>0.30 (0.32)</td>
<td></td>
</tr>
<tr>
<td>TCO(_2)</td>
<td>0.14 (0.03)</td>
<td>0.10 (0.03)</td>
<td>0.15 (0.03)</td>
<td>0.15 (0.03)</td>
<td>0.10 (0.03)</td>
<td>0.15 (0.03)</td>
<td>0.62 (0.02)</td>
<td>−0.14 (0.02)</td>
<td>NC</td>
<td>−0.15 (0.22)</td>
<td>0.49 (0.36)</td>
<td></td>
</tr>
<tr>
<td>sO(_2)</td>
<td>0.08 (0.02)</td>
<td>0.07 (0.02)</td>
<td>−0.08 (0.02)</td>
<td>−0.03 (0.02)</td>
<td>0.0004 (0.02)</td>
<td>0.32 (0.02)</td>
<td>−0.34 (0.02)</td>
<td>0.77 (0.01)</td>
<td>−0.07 (0.02)</td>
<td>−0.09 (0.02)</td>
<td>−0.70 (0.46)</td>
<td></td>
</tr>
<tr>
<td>MORT-TOT</td>
<td>−0.19 (0.02)</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
<td>−0.07 (0.02)</td>
<td>0.04 (0.02)</td>
<td>NC</td>
<td>0.002 (0.02)</td>
<td>0.01 (0.02)</td>
<td>−0.06 (0.02)</td>
<td></td>
</tr>
</tbody>
</table>

\(^1\)BW\(_2\) = BW at 2 wk; BW\(_5\) = BW at 5 wk; RATIO = ratio of right to total ventricular weight; RV = right ventricular weight; TV = total ventricular weight; pCO\(_2\) = partial pressure of carbon dioxide in venous blood; pO\(_2\) = partial pressure of oxygen in venous blood; HCO\(_3\) = bicarbonate; TCO\(_2\) = total carbon dioxide in venous blood; sO\(_2\) = oxygen saturation in venous blood; MORT-TOT = total mortality; NC = nonconverged.
fore on the severity of the challenge (De Greef et al., 2001; Zerehdaran et al., 2006). In the current study, the average MORT-TOT was 10%. In comparison with the MORT-TOT of 16% found by Pakdel et al. (2002) under cold conditions, the mortality in the present study was not very high. However, mortality was much greater than the 4 to 5% mortality found in chickens reared under normal commercial conditions (Pakdel et al., 2002). In the study by Pakdel et al. (2002), an average RATIO of 28% was found, which is greater than the average value of 25% that was obtained in the current study. Julian et al. (1987) suggested that a RATIO of greater than 25% indicates susceptibility to ascites. This threshold would imply that, in our experiment, 45% of the birds showed signs of ascites. The BW5 in the present study was also lower than under commercial conditions. This suppressed growth rate was likely due to the cold stress conditions under which the birds were kept.

In the current study, the average pvCO2 was 45.4 mmHg. Scheele et al. (2003) found an average pvCO2 at 3 wk of age of 53.8 mmHg in a high-risk broiler line and an average pvCO2 of 43.9 mmHg in a low-risk line. Interestingly, however, the pvO2 was lower (44.6 mmHg for the high-risk line and 46.9 mmHg for the low-risk line) than in the current study (52.46 mmHg). However, it should be noted that Scheele et al. (2003) used only male broilers, whereas in the current study, the average measurements were based on results from both males and females. In addition, the previous study compared ascites susceptibility between 2 genetically different stocks (high- and low-risk lines), whereas the current study investigated ascites susceptibility within one crossed line.

It can be concluded that birds in the current study were kept under circumstances that caused a mild increase in ascites. Because estimates of genetic parameters depend on the severity of the challenge (De Greef et al., 2001; Zerehdaran et al., 2006), the estimates presented in this study should be interpreted in this context.

**Correlations Between BW and RATIO**

Pakdel et al. (2005c) found a negative genetic correlation between BW5 and RATIO (−0.27). This is consistent with the negative genetic correlation (−0.18) found between RATIO and BW5 in the present study. A positive genetic correlation (0.19) was observed between RATIO and BW2. These results suggest that susceptible chickens tend to have a greater BW early in life (BW2) and a lower BW later in life (BW5). These results are in agreement with the general finding that correlations between traits are dependent on the frequency of ascitic birds in the population.

**Maternal Effects**

A maternal environmental effect may influence the phenotype of the individual, which, in case these effects play a role, should be accounted for in the statistical analysis (Clément et al., 2001). In the current study, noticeable changes were found in the heritabilities for TV, pvCO2, and MORT-TOT when the maternal environmental effect was included in the model. De Smit et al. (2008) showed that ascites resistance is related to several physiological variables at the embryonic stage, which suggests that maternal effects might play a role in susceptibility. Several studies have reported a maternal effect for BW (Koerhuis and Thompson, 1997; van Kaam et al., 1998; Pakdel et al., 2002). Pakdel et al. (2002) found a significant maternal effect for RATIO, which could not be confirmed in the present study. Navarro et al. (2006) found little evidence for maternal (environmental) effects on sO2; of the 4 broiler lines investigated, one of them exhibited significant evidence for the presence of maternal effects. In that line, maternal environmental effects explained approximately 2% of the total variance. In the present study, we did not find significant maternal effects on sO2; however, a significant maternal effect was found for pvCO2, which explains 5% of the phenotypic variation.

**Blood Gas Parameters as Indicator Traits for Ascites Susceptibility**

In the present study, we evaluated the suitability of blood gas parameters as indicator traits for ascites based on heritabilities and correlations with RATIO. For some of the blood gas parameters, heritabilities were close to zero (pvCO2, pvO2, and sO2), whereas for others, they were moderate (pH, HCO3, and TCO2). The heritability estimate for sO2 was in agreement with results by Navarro et al. (2006); however, in that study, sO2 was measured on 6-wk-old chickens that were not cold stressed. This might have affected the heritability estimates. Druyan et al. (2007) reported a considerably greater heritability estimate (0.49 ± 0.23) for sO2 in chickens that were 7 d old. However, this estimate is not significantly different from the heritability estimate reported in the present study. The low heritabilities indicate that accurate estimates of breeding values for these traits cannot be obtained based on single observations. Accuracies might be improved by using repeated observations, but this will depend on the repeatability of the traits. Repeatability could not be estimated based on the present data; therefore, this is still an option that can be explored. In addition to high heritability, a suitable indicator trait should also have a high genetic correlation with ascites susceptibility. This was evaluated by studying correlations with RATIO. Several authors have suggested that RATIO is a good indicator trait for ascites susceptibility (Lubritz et al., 1995; Pakdel et al., 2002, 2005b). Julian et al. (1987) recommended the use of RATIO as an objective method for assessing right ventricular failure, and therefore of diagnosing ascites. However, others have questioned whether RATIO is a good indicator trait for birds kept under normal conditions (i.e., conditions that do not...
stimulate ascites (Pavlidis et al., 2007). In the present study, birds were kept under cold stress conditions.

Genetic correlations between \( \text{RATIO} \) and both \( \text{HCO}_3^- \) and \( \text{TCO}_2^- \) were moderate, and correlations between \( \text{RATIO} \) and \( \text{pO}_2 \) or \( \text{pCO}_2^- \) were close to zero. The correlation between \( \text{RATIO} \) and \( \text{pO}_2^- \) was \(-0.62\); however, the estimated heritability for \( \text{pO}_2^- \) was very low, resulting in very high SE for the genetic correlations with this trait. Therefore, results from the present study suggest that blood gas parameters are not useful as indicators for ascites susceptibility when measured at an average age of 22 d.

Experimental results from juvenile chickens (Korte et al., 1999; Scheele et al., 2003) showed that at the age of 11 d, a high \( \text{pCO}_2^- \) is associated with a greater incidence of ascites at the age of 5 to 7 wk. Scheele et al. (2005) stated that genetic selection for low \( \text{pCO}_2^- \) values at 11 d of age will be an effective method of reducing the occurrence of the ascites syndrome. However, results from the present study do not confirm this. The first explanation for the discrepancy between results from the present study and results by Scheele et al. (2003, 2005) may be the different ages of the chickens at which the blood gas parameters were measured. The pulmonary pressure index values are known to change rapidly over the first 2 wk of the life of a chicken. Particularly during the period of juvenile growth, the metabolic rate is high and these conditions impose greater metabolic demands. The increased metabolism requires high \( \text{O}_2 \) intake and, at the same time, high maintenance requirements. These factors lead to the maximal potential delivery capacity of oxygen in the respiratory and cardiovascular systems, which is then exceeded and triggers the events that lead to ascites (Decuypere et al., 2000). In the current study, the blood gas parameters were measured when the chickens were, on average, 22 d old. However, in the study by Scheele et al. (2005), the differences in \( \text{pCO}_2^- \) values between the lines remained consistent until the end of the experiment, but did increase as the chickens became older. The \( \text{pO}_2^- \) values decreased as the chickens aged, and the differences between the mean \( \text{pO}_2^- \) values in the 2 lines became greater (Scheele et al., 2003). It should be mentioned that the severity of the challenge differed between the study by Scheele et al. (2003, 2005) and the current study. In the current study, the temperature was gradually reduced to 10°C at 22 d of age, whereas Scheele et al. (2003, 2005) gradually reduced the temperature to 15°C at 16 d of age. Although this did not result in a greater mortality, it is possible that even ascites-resistant broilers experienced problems with breathing because of the low temperature. This might have had an effect on the blood gas parameter values. The second explanation could be the lines that were used. Scheele et al. (2003) compared ascites susceptibility between 2 genetically different stocks (high- and low-risk lines), and several different lines were compared in the other study (Scheele et al., 2005), whereas in the current study, ascites susceptibility was investigated within one crossed line. A third explanation could be the fact that only males were used by Scheele et al. (2003, 2005), whereas both males and females were used in the current study. The female growth rate is slower than the male growth rate; thus, ascites-susceptible females will have a lower \( \text{pCO}_2^- \), on average. Therefore, differences in \( \text{pCO}_2^- \) between healthy and affected chickens will be smaller. Although previous studies (Scheele et al., 2003, 2005) reported the use of \( \text{pCO}_2^- \) as an indicator trait for ascites susceptibility in male broilers, the results from the current study do not support this. It might be concluded that the severity of the challenge, the genetic lines used, the sex of the chickens, and the time of measurement are critical factors.

**Conclusion**

The estimated heritabilities for the blood gas parameters \( \text{pCO}_2^- \) and \( \text{pO}_2^- \) were almost zero. This indicates that selection based on single measurements of these blood gas parameters is not feasible. The heritabilities of \( \text{HCO}_3^- \) and \( \text{TCO}_2^- \) showed enough variation in the population to be used for selection. However, the low genetic correlation between \( \text{RATIO} \) and these 2 blood gas parameters suggests that they are not useful as indicators for ascites susceptibility. Therefore, the current data suggest that blood gas parameters measured at an average age of 22 d will not be very effective when used for selecting against susceptibility.

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