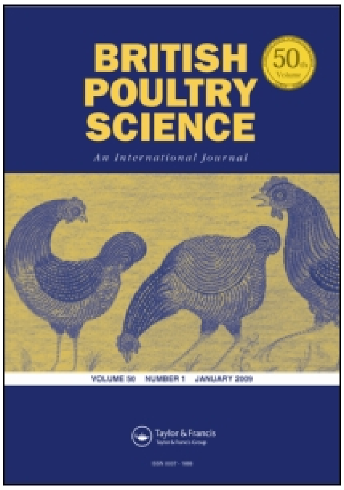

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Genetic parameters of ascites-related traits in broilers: correlations with feed efficiency and carcase traits

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Abstract 1. Pulmonary hypertension syndrome followed by ascites is a metabolic disorder in broilers that occurs more often in fast-growing birds and at cool temperatures.

2. Knowledge of the genetic relationships among ascites-related traits and performance traits like carcase traits or feed efficiency traits is required to design breeding programmes that aim to improve the degree of resistance to ascites syndrome as well as production traits. The objective of this study was to estimate these genetic correlations.

3. Three different experiments were set up to measure ascites-related traits (4202 birds), feed efficiency traits (2166 birds) and carcase traits (2036 birds). The birds in different experiments originated from the same group of parents, which enabled the estimation of genetic correlations among different traits.

4. The genetic correlation of body weight (BW) measured under normal conditions and in the carcase experiment with the ascites indicator trait of right ventricle to total ventricle ratio (RV:TV) measured under cold conditions was 0·30. The estimated genetic correlation indicated that single-trait selecting for BW leads to an increase in occurrence of the ascites syndrome but that there are realistic opportunities of multi-trait selection of birds for improved BW and resistance to ascites.

5. Weak but positive genetic relationships were found between feed efficiency and ascites-related traits suggesting that more efficient birds tend to be slightly more susceptible to ascites.

6. The relatively low genetic correlation between BW measured in the carcase or in the feed efficiency experiments and BW measured in the ascites experiment (0·49) showed considerable genotype by environment interaction. These results indicate that birds with high genetic potential for growth rate under normal temperature conditions have lower growth rate under cold-stress conditions due to ascites.

INTRODUCTION

Consistent selection by poultry breeding companies has dramatically increased the growth rate of broiler stocks. As a result, today's chickens as compared to typical broilers from the 1950s consume more feed per unit time, have better feed efficiency, grow faster and have greater carcase yield (Havenstein *et al.*, 1994). However, apart from a favourable increase in production traits, birds that have been selected for high production efficiency seem to be more sensitive to suboptimal conditions. Furthermore, there seems to be an increase in the incidence of metabolic and physiological disorders of which ascites syndrome is an example (Julian, 1998; Rauw *et al.*, 1998). Ascites syndrome is an accumulation of fluid in the abdominal cavity and the lay term 'waterbelly' is an appropriate description of its appearance in affected chickens. It occurs when broilers fail to take up sufficient oxygen to support their metabolic demands. This syndrome is a challenge to the poultry industry because it is an end-stage lesion with multiple causes and heavy, rapidly growing broilers appear to be more susceptible as a result of an increased metabolic rate (Hoerr, 1988).

Previous studies have shown that traits related to ascites syndrome have a relatively high heritability (Lubritz *et al.*, 1995; De Greef *et al.*, 2001; Pakdel *et al.*, 2002). This indicates that selection against this syndrome should be feasible. Furthermore, Pakdel *et al.* (2005) demonstrated a strong genetic correlation between the same ascites-related trait measured under cold and normal conditions, which indicates that the same

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genes affect these traits in both environments. Genetic correlations among ascites-related traits and productivity traits like body weight (BW) have been reported (De Greef *et al.*, 2001; Moghadam *et al.*, 2001; Pakdel *et al.*, 2005), but seem to depend on environmental conditions. Little information is available on genetic correlations with feed efficiency or carcase traits. Under normal climatic conditions a positive genetic correlation was reported between ascites-related traits and BW (Moghadam *et al.*, 2001; Pakdel *et al.*, 2005). However, under cold conditions a negative genetic correlation was reported between ascites and BW (De Greef *et al.*, 2001; Pakdel *et al.*, 2005). Deeb *et al.* (2002) found that birds with a higher potential for growth rate under normal temperature conditions are more likely to suffer from ascites under cold-stress conditions as compared to birds with a lower potential for growth rate. Pakdel *et al.* (2005) reported that the genetic correlation between ascites-related traits and BW depends upon the conditions under which birds are kept. They suggest that high production of birds under cold conditions is due to a favourable combination of resistance to ascites and production potential. Under normal conditions however, the production potential of animals is important while genetic differences in resistance to ascites syndrome are less relevant.

Knowledge of the genetic relationships among ascites-related traits and performance traits like carcase traits or feed efficiency traits is required to design breeding programmes that aim to improve the degree of resistance to ascites syndrome as well as production traits. The objective of this study was to estimate these genetic correlations.

MATERIAL AND METHODS

Birds and traits

Birds

The experimental population was the result of a F_2 cross between two genetically different broiler dam lines (Hybro) originating from the White Plymouth Rock breed, as described by Pakdel *et al.* (2005). The same F_2 parents were used to produce three groups of F_3 birds, which were used in the three different experiments.

Ascites experiment. This experiment was to measure ascites-related traits under cold conditions. This group consisted of 4202 birds, hatched in 6 different weeks as described by Pakdel *et al.* (2005). At the time of hatching, the temperature was 30°C for this group of birds and was gradually decreased to 10°C by 22 d of age, where it remained until the end of the experiment. Except for the cold-stress temperature schedule, birds were kept under circumstances that closely resemble commercial practice.

Carcase experiment. This experiment was to measure carcase traits. This group consisted of 2036 birds and hatched in 6 different weeks. They were reared in 6 hatches and housed in a litter system until the age of 48 d. These birds were kept under normal temperature schedule, starting at 33 to 34°C and gradually decreasing to 17 to 18°C at 35 d of age. The animals were in the same pen from d 0, where they received feed and water *ad libitum*. The birds in this experiment were used previously to detect quantitative traits loci (QTL) affecting carcase traits (Van Kaam *et al.*, 1999*a*). In addition to the carcase traits, information on ascites-related traits was collected on 795 birds in this experiment (Pakdel *et al.*, 2005).

Feed efficiency experiment. This experiment was to measure traits related to feed efficiency. This group consisted of 2166 birds, hatched in 5 different weeks. They were reared consecutively in the same floor pens up to 22 d of age and housed individually in another building between the age of 22 and 48 d. Individual cages were used for individual measurements of feed intake. Throughout, feed and water were supplied *ad libitum*. The temperature schedule of this group of birds was normal and similar to the carcase experiment. The birds in this experiment have been used previously to detect QTL affecting feed efficiency traits (Van Kaam *et al.*, 1999*b*).

All three groups of birds originated from the same parents, making it possible to estimate genetic correlations between traits measured in different experiments. There were a few important differences in the housing conditions of the three experimental groups. In the ascites and carcase experiment, birds were kept in groups (20 birds/m^2), whereas in the feed efficiency experiment birds were housed individually. Furthermore, in the ascites experiment birds were kept under cold conditions whereas in the two other experiments the temperatures were normal. Otherwise, all three groups of birds were kept under similar conditions: a standard commercial broiler feed, consisting of crumbled concentrates (12·97 MJ/kg and 210 g protein/kg) and artificial lighting for 23 h/d. All three experiments were conducted strictly in line with the regulations of the Dutch law on the protection of animals.

Traits

Ascites experiment. Birds were slaughtered at 5 weeks of age. The body weight ($BW_{AS\text{-}35}$) and haematocrit value (HCT) of birds were measured one day before slaughtering. After slaughtering a number of ascites-related traits were measured (Pakdel *et al*., 2005). These were liver abnormalities (LIVER), accumulation of fluid in the heart sac (HEART), weight of right ventricle (RV), total ventricles (TV) and ratio of RV:TV, accumulation of fluid in the abdomen (Abdomen), colour of the breast (BREAST) and, finally, total mortality (MORT-TOT).

Carcase experiment. Around d 47, the legs of the birds were scored on a scale from 1 to 9, by looking at the lateral deviation of the legs (the hock joints). Straight legs received a score of 9. Birds with legs further away from this optimum received a lower score. Leg problems were considered as an effect of weak hock ligaments or tendons, which could result in both *varus* (proximal hocks) as well as *valgus* (distal hocks). Therefore, both *varus* and *valgus* birds had a score below maximum. In practice, most birds with leg abnormalities showed *varus*. At 48 d, body weight ($BW_{C\text{-}48}$) was measured, animals were slaughtered and carcase weight (CW) was measured. CW was measured on the chilled carcase after removal of feathers, head, lungs, liver, kidneys, gastrointestinal tract, abdominal body fat, subcutaneous leg fat, lower legs and blood loss due to bleeding. For one hatch, CW was measured on d 2. On the same day that CW was measured, meat colour (MC) was measured at three spots on the chilled breast fillet, using a fibre optic meat probe (TBL Fibre Optics Ltd, Leeds, UK). These three measurements were averaged to obtain a single value for MC. The last hatch of birds was measured on a different scale due to problems with the fibre optic meat probe. To remove this effect, data were standardised within every hatching week (by dividing the deviation of each observation from the mean with standard deviation). For about 700 birds in this experiment measurements for the ascites-related traits HCT, RV, TV and RV:TV were available. BW and HCT of this group of birds were also measured one day before slaughtering.

Feed efficiency experiment. Traits measured were BW at 23 d of age ($BW_{FE\text{-}23}$), BW at 48 d of age ($BW_{FE\text{-}48}$) and feed intake from 23 d to 48 d (FI). Inferred traits were growth between 23 and 48 d (GAIN), feed efficiency (in percentage) between 23 and 48 d (FE) and residual feed intake (RFI). FE was defined as the ratio between GAIN and feed intake and can be seen as gross efficiency. RFI is a measure of net feed efficiency, which accounts for both maintenance and growth requirements. RFI was calculated as:

$$\mathrm{RFI} = \mathrm{FI} - [a + (b_1 * \mathrm{BW}_{\mathrm{FE\text{-}23}}) + (b_2 * \mathrm{GAIN})]$$

where a is the intercept and b_1 and b_2 are the partial regression coefficients of feed intake on $BW_{FE\text{-}23}$ and GAIN, respectively (Van Bebber and Mercer, 1994).

Genetic analysis

An animal model was used to calculate heritabilities and genetic correlations of the traits:

$$Y_{ijklm} = \mu + \mathrm{Sex}_i + \mathrm{Feather}_j + \mathrm{Batch}_k + \mathrm{Group}_l + a_m + e_{ijklm}$$

where Y_{ijklm} = the dependent variable on chicken 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); $\mathrm{Feather}_j$ = fixed effect of feathering j ($j = 1$, 2 fast or slow); Batch_k = fixed effect of batch k ($k = 1, 2 \ldots 9$ only for birds in the ascites experiment), classes were formed based on a combination of hatching day and pen; Group_l = fixed effect of group ($l = 1, 2 \ldots 46$ for birds in the ascites experiment, $l = 1, 2 \ldots 47$ for birds in the carcase experiment and $l = 1, 2 \ldots 40$ for birds in the feed efficiency experiment), classes were formed based on the age of the dam and the hatching day of the animals; a_m = random direct genetic effect of individual m; e_{ijklm} = random residual effect. The model was based on previous analysis reported by Pakdel *et al*. (2005). Except for the batch effect, which was included only in the model for birds in the ascites experiment, fixed and random effects in the model were identical for all experiments. Bivariate analyses were performed to compute correlations between all combinations of traits. Estimates of variance components were obtained using the ASREML software (Gilmour *et al*., 2000).

RESULTS

Descriptive statistics

The traits measured in the three experiments are presented in Table 1. In the ascites experiment, the broilers weighed 1604 g on average at 35 d of age and total mortality was 16%. Under normal conditions (carcase and feed efficiency experiment) the broilers weighed 2199 g on average at 48 d of age and total mortality was 4% in the carcase experiment (data not shown). For a more detailed description of ascites-related traits under cold and normal conditions, see Pakdel *et al*. (2005). In the feed efficiency experiment, average feed intake of birds from 23 to 48 d of age was 3222 g and average weight gain was 1582 g. Average feed efficiency was 49%, average residual

Table 1. *Descriptive statistic of the traits*

Trait	Abbreviation	Number	Mean	SD
Ascites experiment[1]				
BW at 35 d of age (g)	$BW_{AS\text{-}35}$	3693	1604	263
Haematocrit value (%)	HCT	3547	35·40	4·21
Right ventricular weight (g)	RV	3660	1·95	0·68
Total ventricular weight (g)	TV	3658	6·97	1·17
Ratio of right ventricular to total ventricular weight (%)	RV:TV	3658	27·94	8·07
Total mortality[2]	MORT-TOT	2494	0·16	0·37
Fluid in the abdomen[3]	ABDOMEN	3697	0·08	0·38
Colour of the breast[3]	BREAST	3697	0·03	0·18
Liver abnormalities[3]	LIVER	3697	0·07	0·29
Fluid in the heart sac[3]	HEART	3696	0·59	0·62
Feed efficiency experiment[4]				
BW at 23 d of age (g)	$BW_{FE\text{-}23}$	2166	603	100
BW at 48 d of age (g)	$BW_{FE\text{-}48}$	2085	2187	332
Feed intake between 23 and 48 d (g)	FI	2085	3222	490
Growth between 23 and 48 d (g)	GAIN	2085	1582	278
Feed efficiency	FE	2085	0·49	0·05
Residual feed intake from 23 to 48 d (g)	RFI	2085	0	253
Carcase experiment[4]				
BW at 48 d (g)	$BW_{C\text{-}48}$	1999	2212	337
Carcase weight (g)	CW	1987	1495	237
Carcase percentage	CP	1966	67·42	1·86
Meat colour[5]	MC	1943	31·73	4·12
Leg score[6]	LS	1959	5·76	1·98
Haematocrit value (%)	HCT_C[7]	780	28·28	2·30
Right ventricular weight (g)	RV_C	659	1·15	0·31
Total ventricular weight (g)	TV_C	746	5·60	0·94
Ratio of right ventricular to total ventricular weight (%)	$RV{:}TV_C$	659	20·65	4·66

[1] Traits measured at 5 weeks of age under cold conditions.
[2] Trait scored as 0 or 1.
[3] Trait scored as 0, 1 or 2.
[4] Traits measured at 7 weeks of age under normal conditions.
[5] Higher values represent darker or reddish meat.
[6] Legs scored from 1 to 9. Higher score means the legs are straighter and they were considered as the optimum.
[7] C means trait measured in carcase experiment.

feed intake was, by definition, zero and standard deviation was 253 g. The mean weight of 48-d-old birds in the carcase experiment was 2212 g and CW was 1495 g, which corresponds to a carcase percentage (CP) of 67%.

Genetic parameters for feed efficiency and carcase traits

The estimated heritabilities and genetic correlations among feed efficiency traits and carcase traits are presented in Table 2. Heritability estimates for most of the traits in the carcase experiment were 0·3 to 0·4. The heritability for leg score was lower and had a value of 0·12. In the feed efficiency experiment heritabilities ranged from 0·16 for GAIN to 0·52 for $BW_{FE\text{-}23}$.

The estimated genetic correlation between $BW_{C\text{-}48}$ and CW was close to unity (0·97). The genetic correlation between body weight measured when birds were kept in groups ($BW_{C\text{-}48}$) and individually housed birds ($BW_{FE\text{-}48}$) was 0·87. Body weight ($BW_{C\text{-}48}$ or $BW_{FE\text{-}48}$) and CP were genetically unrelated (0 or −0·03). Leg score had a positive genetic correlation with CP (0·41). No correlations significantly different from zero were found between leg score and other traits. Furthermore, no significant correlations were found between MC and other traits.

Genetically, birds with a higher initial body weight ($BW_{FE\text{-}23}$) also tended to be heavier at 48 d of age, they had a higher GAIN, a higher FI and a lower FE (Table 2). Genetic correlations of $BW_{FE\text{-}48}$ with FI and GAIN were positive and high (0·78 and 0·93, respectively). The correlations between FE and $BW_{FE\text{-}48}$ or $BW_{C\text{-}48}$ had opposite signs but both correlations did not differ significantly from zero. Chickens with higher GAIN during the 23- to 48-d period consumed more feed, which was reflected in the genetic correlation between GAIN and FI of 0·71. The negative relationship between FI and FE (−0·72) indicates that more efficient birds, with

Table 2. *Estimates of genetic parameters for feed efficiency traits and carcase traits*[1]

Trait[2]	Carcase experiment					Feed efficiency experiment					
	$BW_{C\text{-}48}$	CW	CP	MC	LS	$BW_{FE\text{-}23}$	$BW_{FE\text{-}48}$	FI	GAIN	FE	RFI
$BW_{C\text{-}48}$	**0·31 (0·07)**[3]										
CW	0·97 (0·01)	**0·28 (0·06)**									
CP	0·00 (0·16)	0·27 (0·14)	**0·41 (0·07)**								
MC	0·29 (0·16)	0·22 (0·16)	−0·15 (0·14)	**0·36 (0·07)**							
LS	0·10 (0·23)	0·16 (0·23)	0·41 (0·18)	−0·13 (0·22)	**0·12 (0·05)**						
$BW_{FE\text{-}23}$	0·70 (0·12)	0·64 (0·13)	−0·13 (0·13)	0·08 (0·13)	0·21 (0·19)	**0·52 (0·07)**					
$BW_{FE\text{-}48}$	0·87 (0·11)	0·83 (0·12)	−0·03 (0·16)	−0·08 (0·16)	−0·08 (0·24)	0·86 (0·05)	**0·27 (0·06)**				
FI	0·55 (0·15)	0·50 (0·15)	−0·17 (0·14)	−0·05 (0·15)	−0·11 (0·22)	0·73 (0·07)	0·78 (0·06)	**0·35 (0·07)**			
GAIN	0·71 (0·15)	0·70 (0·15)	0·02 (0·19)	−0·14 (0·19)	−0·20 (0·27)	0·64 (0·11)	0·93 (0·02)	0·71 (0·09)	**0·16 (0·05)**		
FE	0·10 (0·17)	0·18 (0·17)	0·26 (0·15)	−0·16 (0·15)	0·06 (0·23)	−0·32 (0·13)	−0·19 (0·18)	−0·72 (0·11)	0·00 (0·20)	**0·29 (0·06)**	
RFI	−0·03 (0·15)	−0·09 (0·15)	−0·22 (0·13)	0·04 (0·14)	−0·10 (0·20)	0·12 (0·12)	0·13 (0·15)	0·72 (0·07)	0·07 (0·18)	−0·93 (0·03)	**0·49 (0·07)**

[1] Genetic correlation below and heritabilities (bold) on the diagonal.
[2] $BW_{C\text{-}48}$ = BW at 48 d in carcase experiment; CW = carcase weight; CP = carcase percentage; MC = meat colour; LS = leg score; $BW_{FE\text{-}23}$ = BW at 23 d in feed efficiency experiment; $BW_{FE\text{-}48}$ = BW at 48 d in feed efficiency experiment; FI = feed intake between 23 and 48 d; GAIN = growth between 23 and 48 d; FE = feed efficiency; RFI = residual feed intake.
[3] Standard errors in parentheses. The standard errors were high for genetic correlations involving leg score.

a higher FE, consumed less feed. RFI showed a strong negative genetic correlation with FE (−0·93) and a positive genetic correlation with FI (0·72).

Genetic relationships among ascites-related and performance traits

Cold conditions

The estimated genetic correlations among traits measured under cold conditions in the ascites experiment and traits measured in the carcase and feed efficiency experiments are presented in Table 3. Body weight in the ascites experiment ($BW_{AS\text{-}35}$) showed a relatively low genetic correlation (0·49) with body weight in the carcase experiment ($BW_{C\text{-}48}$). The genetic correlation between $BW_{C\text{-}48}$ and the ascites indicator trait RV:TV was 0·30 and with MORT-TOT the genetic correlation was 0·35. CW had very similar genetic correlations with these traits, which was expected given the high genetic correlation between $BW_{C\text{-}48}$ and CW. These results suggest that birds with higher genetic potential for body weight measured under normal commercial circumstances ($BW_{C\text{-}48}$) are more susceptible to ascites. MC showed a positive genetic correlation with RV:TV (0·32) that differed significantly from zero. A higher value for MC represents darker or more reddish meat.

The genetic correlations between body weight in the ascites experiment ($BW_{AS\text{-}35}$) and body weights measured in the feed efficiency experiment were 0·36 and 0·53 for $BW_{FE\text{-}23}$ and $BW_{FE\text{-}48}$, respectively. These results confirm that BW measured in a high-ascites incidence environment (cold temperatures) is a genetically different trait from BW measured in a low ascites incidence environment (normal temperatures). The trait $BW_{FE\text{-}48}$ showed a positive genetic correlation with RV (0·47), TV (0·54) and RV:TV (0·27). The genetic correlations between $BW_{FE\text{-}48}$ and other ascites-related traits did not differ significantly from zero. The genetic correlations between GAIN and $BW_{AS\text{-}35}$ and ascites-related traits were similar to those found for $BW_{FE\text{-}48}$. This is expected given the high genetic correlation between $BW_{FE\text{-}48}$ and GAIN. Feed intake also showed a positive genetic correlation with $BW_{AS\text{-}35}$, RV and TV. Correlations between feed intake and ascites indicator traits like RV:TV or total mortality were close to zero. Feed efficiency was positively correlated to RV:TV (0·20) and ABDOMEN (0·17) suggesting an increased susceptibility towards ascites for more efficient birds. However, these correlations are not very strong and have relatively high standard errors. RFI showed a negative genetic correlation with HEART (−0·31). Correlations between RFI and other ascites-related traits are similar to those found for FE but have opposite signs.

Normal conditions

The genetic correlations among ascites-related traits under normal temperature conditions in the carcase experiment and performance traits are presented in Table 4. Ascites-related traits under normal conditions were measured on approximately 700 of the animals in the carcase experiment. Because of this smaller number of animals, the accuracy of the estimated genetic correlations is lower than estimates reported in Table 3.

The trait $BW_{C\text{-}48}$ showed a relatively high genetic correlation with heart measurements like RV and TV (0·83 and 0·69, respectively). Positive genetic correlations were found between $BW_{C\text{-}48}$ and ascites indicator traits like HCT and RV:TV. MC showed a high positive genetic correlation with RV (0·72) and RV:TV (0·73) which means that birds with high values for MC have a higher right ventricular weight and also a higher RV:TV. No significant correlations were found between leg score and ascites-related traits.

Body weight measured at 48 d in the feed efficiency experiment ($BW_{FE\text{-}48}$) had high genetic correlations with heart measurements like RV and TV (0·63 and 0·83, respectively), but the correlation with RV:TV was close to zero. The relationships between GAIN and ascites-related traits were very similar to those for $BW_{FE\text{-}48}$ which was to be expected given their high genetic correlation (Table 1). FI showed a positive genetic correlation of 0·46 with TV. The genetic correlations between HCT and FE or RFI were of the same magnitude but with opposite signs (0·58 and −0·57, respectively).

DISCUSSION

In poultry breeding, traits like growth rate and FE are of great interest as these traits have a major economic effect. Therefore, breeding programmes aim to select birds with a higher growth rate and/or a higher FE. However, it has been suggested that the improvement in growth rate or FE is not without undesirable correlated responses such as increased incidence of defects in heart and lung function and reduced adaptability to environmental conditions (Olkowski *et al.*, 1999; McKay *et al.*, 2000). This is in line with the resource allocation theory (Beilharz *et al.*, 1993) which states that when a population is genetically driven towards higher production, and thus allocates a higher proportion of resources to these traits, fewer resources remain to respond adequately to other demands, like

Table 3. *Estimates of genetic correlation among ascites-related traits measured under cold conditions and performance traits measured under normal conditions*

Trait[1]	Carcase experiment					Feed efficiency experiment					
	$BW_{C\text{-}48}$	CW	CP	MC	LS	$BW_{FE\text{-}23}$	$BW_{FE\text{-}48}$	FI	GAIN	FE	RFI
$BW_{AS\text{-}35}$	0·49 (0·12)[2]	0·52 (0·12)	0·16 (0·12)	−0·09 (0·13)	−0·36 (0·20)	0·36 (0·10)	0·53 (0·13)	0·43 (0·12)	0·52 (0·15)	−0·10 (0·13)	0·11 (0·11)
HCT	0·21 (0·13)	0·21 (0·13)	0·03 (0·12)	−0·03 (0·13)	0·48 (0·17)	−0·03 (0·11)	−0·10 (0·14)	−0·19 (0·12)	−0·11 (0·16)	0·19 (0·13)	−0·19 (0·11)
RV	0·44 (0·13)	0·40 (0·13)	−0·09 (0·12)	0·22 (0·12)	0·17 (0·18)	0·34 (0·10)	0·47 (0·12)	0·26 (0·12)	0·49 (0·15)	0·16 (0·13)	−0·12 (0·11)
TV	0·42 (0·12)	0·41 (0·12)	−0·01 (0·12)	−0·05 (0·12)	−0·11 (0·19)	0·34 (0·10)	0·54 (0·12)	0·41 (0·11)	0·59 (0·14)	0·03 (0·13)	0·05 (0·11)
RV:TV	0·30 (0·13)	0·27 (0·13)	−0·06 (0·12)	0·32 (0·12)	0·28 (0·18)	0·22 (0·11)	0·27 (0·13)	0·09 (0·13)	0·26 (0·16)	0·19 (0·13)	−0·16 (0·11)
MORT-TOT	0·35 (0·16)	0·33 (0·16)	0·02 (0·15)	−0·11 (0·16)	0·09 (0·23)	−0·04 (0·14)	−0·19 (0·18)	−0·04 (0·16)	−0·27 (0·20)	−0·20 (0·17)	0·15 (0·14)
ABDOMEN	0·08 (0·21)	0·06 (0·21)	0·00 (0·20)	0·11 (0·20)	0·25 (0·25)	0·23 (0·17)	0·31 (0·20)	0·20 (0·20)	0·29 (0·23)	0·17 (0·21)	−0·06 (0·19)
BREAST	0·10 (0·29)	0·07 (0·30)	0·10 (0·28)	0·26 (0·25)	0·07 (0·37)	0·34 (0·23)	0·03 (0·31)	−0·01 (0·29)	−0·27 (0·35)	−0·06 (0·30)	−0·09 (0·26)
LIVER	−0·02 (0·22)	−0·03 (0·22)	0·07 (0·20)	0·09 (0·21)	0·37 (0·25)	0·20 (0·18)	0·23 (0·21)	0·05 (0·21)	0·17 (0·25)	0·21 (0·22)	−0·18 (0·19)
HEART	0·28 (0·17)	0·26 (0·17)	−0·09 (0·15)	0·08 (0·16)	0·42 (0·21)	0·00 (0·14)	0·05 (0·18)	−0·18 (0·17)	0·11 (0·21)	0·35 (0·18)	−0·31 (0·15)

[1] $BW_{AS\text{-}35}$ = BW at 35 d of age; HCT = haematocrit value; RV = right ventricular weight; TV = total ventricular weight; RV:TV = ratio of right ventricular weight to total ventricular weight; MORT-TOT = total mortality; ABDOMEN = fluid in the abdomen; BREAST = colour of the breast; LIVER = liver abnormalities; HEART = fluid in the heart sac; $BW_{C\text{-}48}$ = BW at 48 d in carcase experiment; CW = carcase weight; CP = carcase percentage; MC = meat colour; LS = leg score; $BW_{FE\text{-}23}$ = BW at 23 d in feed efficiency experiment; $BW_{FE\text{-}48}$ = BW at 48 d in feed efficiency experiment; FI = feed intake between 23 and 48 d; GAIN = growth between 23 and 48 d; FE = feed efficiency; RFI = residual feed intake.
[2] Standard errors in parentheses.

Table 4. *Estimates of genetic correlation among ascites-related traits and performance traits measured under normal conditions*

Trait[1]	Carcase experiment					Feed efficiency experiment					
	$BW_{C\text{-}48}$	CW	CP	MC	LS	$BW_{FE\text{-}23}$	$BW_{FE\text{-}48}$	FI	GAIN	FE	RFI
HCT_C[2]	0·47 (0·24)[3]	0·40 (0·25)	0·35 (0·22)	0·21 (0·28)	0·10 (0·34)	−0·25 (0·23)	0·12 (0·29)	−0·33 (0·26)	0·14 (0·31)	0·58 (0·20)	−0·57 (0·19)
RV_C	0·83 (0·14)	0·73 (0·18)	−0·07 (0·32)	0·72 (0·20)	0·18 (0·42)	0·25 (0·30)	0·63 (0·25)	0·32 (0·30)	0·61 (0·26)	0·50 (0·25)	−0·21 (0·29)
TV_C	0·69 (0·13)	0·67 (0·13)	0·04 (0·18)	0·27 (0·17)	0·24 (0·25)	0·46 (0·16)	0·83 (0·14)	0·46 (0·19)	0·78 (0·16)	0·29 (0·18)	−0·11 (0·17)
$RV{:}TV_C$	0·46 (0·29)	0·35 (0·30)	−0·14 (0·34)	0·73 (0·23)	0·08 (0·41)	−0·13 (0·30)	−0·01 (0·34)	−0·17 (0·33)	−0·04 (0·38)	0·38 (0·30)	−0·19 (0·30)

[1] HCT = haematocrit value; RV = right ventricular weight; TV = total ventricular weight; RV:TV = ratio of right ventricular weight to total ventricular weight; $BW_{C\text{-}48}$ = BW at 48 d in carcase experiment; CW = carcase weight; CP = carcase percentage; MC = meat colour; LS = leg score; $BW_{FE\text{-}23}$ = BW at 23 d in feed efficiency experiment; $BW_{FE\text{-}48}$ = BW at 48 d in feed efficiency experiment; FI = feed intake between 23 and 48 d; GAIN = growth between 23 and 48 d; FE = feed efficiency; RFI = residual feed intake. [2] C means trait measured in carcase experiment. [3] Standard errors in parentheses.

coping with unexpected stressors. The results of the current study show that birds that grow fast under normal commercial circumstances (normal temperatures) are more susceptible to ascites syndrome. Further, weak but positive genetic relationships were found between feed efficiency and ascites-related traits suggesting that more efficient birds tend to be slightly more susceptible to ascites.

Growth rate and susceptibility to ascites

Birds that grow fast under normal commercial circumstances, that is, birds with a high genetic potential for $BW_{C\text{-}48}$, also tend to have larger hearts as is indicated by the positive genetic correlations with RV and TV. This holds true under both cold and normal temperature conditions. Based on the ascites experiment it can be concluded that these birds are also genetically more susceptible to develop the ascites syndrome as is indicated by the correlation with RV:TV. The ratio of RV:TV is an ascites heart index that quantifies the degree of hypertrophy of the heart in broilers (Huchzermeyer and De-Ruyck, 1986). The literature generally reports that RV:TV is higher for ascitic than non-ascitic broilers (Wideman and French, 2000). Further, fast growth in the carcase experiment, where the housing system (group housing) was similar to that in the ascites experiment, is positively correlated to total mortality. Based on these results we can conclude that broilers with higher genetic potential for growth rate are more susceptible to ascites. However, the estimated genetic correlations among body or carcase weights and ascites-related traits in the present study were moderate, which indicates that there is scope for simultaneous improvement of growth rate and susceptibility to ascites.

The favourable genetic correlations between TV measured under cold or normal conditions and productivity traits like BW, CARCASE and GAIN are in agreement with the results previously obtained by Rance *et al.* (2002), who reported that the genetic correlations between production traits and support organs in broilers were generally low, however, heart mass was positively correlated with all carcase components; the genetic correlations ranged between 0·55 with breast mass to 0·64 with eviscerated body mass.

Feed efficiency and susceptibility to ascites

The estimated genetic correlations between FE and ascites-related traits measured under cold-stress conditions were in general low. Correlations between FE and the important ascites-related traits RV:TV and ABDOMEN were positive but not very strong. Relationships between RFI and the ascites-related traits were negative but also not very strong. Based on the strong negative genetic correlation between FE and RFI these results were expected. Relations between FE and RV:TV measured under normal temperatures also indicate that more efficient birds are more susceptible towards ascites. Previous studies have reported that birds with a high feed efficiency have lower heat production and therefore have less flexibility in metabolic adaptation to a changing environment, which can account for the development of ascites (Scheele *et al.*, 1991; Scheele, 1996). We found some evidence that supports this hypothesis, however, it should be pointed out that the evidence is not very strong given the high standard errors of the estimated correlations.

It should be realised that the genetic correlations among feed efficiency and ascites-related traits in the present study are not only the result of differences in environmental conditions (including system of housing and temperature effect) but also differences in age between the groups of birds should be considered. Leenstra and Cahaner (1991) reported that age and temperature have significant effects on feed efficiency. Younger birds (0 to 4 weeks) showed higher FE than older birds (4 to 6 weeks). On the other hand the FE of birds was higher under normal temperatures than under low temperatures (Leenstra and Cahaner, 1991). In the current study FE was measured under conditions where birds were using feed mainly for growth. However, under cold-stress conditions birds were younger (5 *vs* 7 weeks of age) and they might have higher FE than birds under the FE experiment.

Genotype by environment interaction

If the estimated genetic correlation between the same traits measured under different conditions is significantly less than unity, there is genotype by environmental interaction (Falconer, 1989). In the present study there was a relatively low genetic correlation between body weight measured under cold and normal conditions (around 0·50) which indicates that BW is very sensitive to the applied temperature schedule. Also the difference in age between the groups of birds could have contributed to the lower correlation. However, genetic correlations among body weights of broilers at various ages are in general high (Chambers *et al.*, 1984; Wang *et al.*, 1991): in the present study the genetic correlation between $BW_{FE\text{-}23}$ and $BW_{FE\text{-}48}$ was 0·87. Therefore, it can be concluded that the relatively low genetic correlation between BW measured in the carcase or feed efficiency experiments and BW measured in the ascites experiment is mainly due to the temperature effect. The low estimated genetic correlation between BW measured under cold and normal conditions is in agreement with the results obtained in a previous study (Pakdel *et al.*, 2005). In a previous study we reported a genetic correlation between $BW_{AS\text{-}35}$ and $BW_{C\text{-}48}$ using 770 birds of 0·29, whereas in the current study this correlation was 0·49 and was estimated based on 1999 observations (Pakdel *et al.*, 2005).

Effect of housing

Different correlations were observed between some of the ascites-related traits measured in the ascites experiment and body weight in the carcase experiment ($BW_{C\text{-}48}$) or body weight in the feed efficiency experiment ($BW_{FE\text{-}48}$). Birds with a higher genetic potential for $BW_{C\text{-}48}$ showed a higher total mortality and higher HCT values under cold-stress conditions. However, the genetic correlations among $BW_{FE\text{-}48}$ and total mortality or HCT were negative. Furthermore, under normal conditions, the genetic correlations between $BW_{C\text{-}48}$ and HCT or RV:TV were higher than those for $BW_{FE\text{-}48}$. The other point that suggests correlations are affected by the system of housing is the relationship among BW, ABODOMEN and HEART. The genetic correlation between $BW_{C\text{-}48}$ and ABDOMEN was 0·08, and with HEART 0·28. However, the genetic correlation between $BW_{FE\text{-}48}$ and ABDOMEN was 0·31, and with HEART 0·05.

Although standard errors of these estimates are relatively high, the difference between the genetic correlations among $BW_{C\text{-}48}$ or $BW_{FE\text{-}48}$ and ascites-related traits indicate that correlations are affected by a higher incidence of ascites syndrome, which might be due to the difference in the housing system: individual *vs* group housing. It is probable that birds under group housing are more susceptible to ascites than birds under individual housing: under individual housing birds have less movement and no competition for food as might be the case in the carcase or ascites experiment which influence the metabolic rate of the chickens and eventually the incidence of ascites. Moreover, under group housing (ascites or carcase experiment) stocking density is very high towards the end of the rearing period. The concentration of air pollution and respiratory irritants (ammonia, carbon dioxide and carbon monoxide) is higher under group housing (Madelin and Wathes, 1989). The contamination in the air of broiler houses may increase the incidence of ascites syndrome in susceptible broilers by posing a greater respiratory challenge to the lungs (Wang *et al.*, 2002).

CONCLUSIONS

Selecting for BW increases susceptibility to ascites. However, the weak genetic correlation among BW and ascites-related traits indicates that there is considerable scope for simultaneous selection for birds with high BW and low susceptibility to ascites. Further, the estimated genetic correlations in the present study do not support the view that selecting for FE will result in an increase of the ascites problem. However, the results showed a substantial genotype by temperature interaction for BW. Our results indicate that birds with high genetic potential for growth rate under normal temperature conditions have a lower growth rate under cold-stress conditions due to ascites.

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ABBREVIATIONS

$BW_{AS\text{-}35}$ = BW of the birds in the ascites experiment at 35 d; HCT = haematocrit value; RV = right ventricular weight; TV = total ventricular weight; RV:TV = ratio of right ventricular weight to the total ventricular weight; MORTTOT = total mortality; ABDOMEN = fluid in the abdomen; BREAST = colour of the breast; LIVER = liver abnormalities; HEART = fluid in the heart sac; $BW_{C\text{-}48}$ = BW of the birds in carcase experiment at 48 d; CW = carcase weight; CP = carcase percentage; MC = meat colour; LS = leg score; $BW_{FE\text{-}23}$ = BW of the birds in feed efficiency experiment at 23 d; $BW_{FE\text{-}48}$ = BW of the birds in feed efficiency experiment at 48 d; FI = feed intake; GAIN = growth between 23 and 48 d; FE = feed efficiency from 23 to 48 d; RFI = residual feed intake from 23 to 48 d.

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