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Pau Navarro, Peter Visscher, Alfons Koerhuis, Dimitrios Chatziplis, Chris Haley

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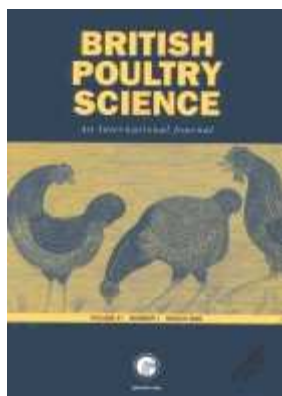
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**Genetic parameters for blood oxygen saturation, body weight and breast conformation in four meat-type chicken lines**

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Keywords:	Growth, Broilers, Genetics, Ascites



1 **Genetic parameters for blood oxygen saturation, body weight and breast conformation in**  
2  
3 **four meat-type chicken lines**

4 P. NAVARRO<sup>\*,1,2</sup>, P. M. VISSCHER<sup>†</sup>, D. CHATZIPLIS<sup>‡</sup>, A. N. M. KOERHUIS<sup>‡</sup> AND C. S.  
5  
6 HALEY<sup>\*</sup>  
7

8 <sup>\*</sup>ROSLIN INSTITUTE, ROSLIN, MIDLOTHIAN EH25 9PS, SCOTLAND (UK)  
9

10 <sup>†</sup> INSTITUTE OF EVOLUTIONARY BIOLOGY, UNIVERSITY OF EDINBURGH, WEST  
11  
12 MAINS ROAD, EDINBURGH EH9 3JT, SCOTLAND (UK)

13 <sup>‡</sup> AVIAGEN LTD., NEWBRIDGE, MIDLOTHIAN, EH28 8SZ, SCOTLAND (UK)  
14  
15

16  
17 Corresponding author: Pau Navarro, Institute of Evolutionary Biology, University of Edinburgh,  
18  
19 West Mains Road, Edinburgh EH9 3JT, Scotland (UK).

20  
21 Tel: +44 (0) 131 650 5442

22  
23 Fax: +44 (0) 131 650 6564

24  
25 E-mail: Pau.Navarro@ed.ac.uk  
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28 SHORT TITLE: GENETIC PARAMETERS BLOOD OXYGEN SATURATION  
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31 ABBREVIATION KEY: - 2LnLR = minus two times the logarithm of the likelihood ratio;  
32  
33 APO<sub>2</sub> = arterial oxygen pressure; Flesh = fleshing score; h<sup>2</sup> = heritability; Heart-API = ratio of  
34  
35 the weight of the right heart ventricle to total heart weight; PCV = packed cell volume; SaO =  
36  
37 blood oxygen saturation; Weight = body weight;  $\sigma_e^2$  = residual variance;  $\sigma_{em}^2$  = maternal  
38  
39 environmental variance;  $\sigma_{gm}^2$  = maternal genetic variance;  $\sigma_p^2$  = phenotypic variance;  $\sigma_u^2$  =  
40  
41 genetic (polygenic) variance.  
42

43  
44 SECTION: GENETICS

45  
46 (*Key words*: ascites, blood oxygen saturation, broiler, genetic parameters, production)  
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49  
50 <sup>1</sup>Present address: Pau Navarro, Institute of Evolutionary Biology, University of Edinburgh,  
51  
52 West Mains Road, Edinburgh EH9 3JT, Scotland (UK).  
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<sup>2</sup>To whom correspondence should be addressed: Pau.Navarro@ed.ac.uk.

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For Peer Review Only

**Abstract**

1                   **Abstract**       1. The objective of the study was to explore the genetic architecture of  
2 blood oxygen saturation (SaO) (an indicator trait, negatively correlated with ascites  
3 susceptibility), body weight (Weight) and fleshing score (Flesh, a measure of breast  
4 susceptibility), body weight (Weight) and fleshing score (Flesh, a measure of breast  
5 conformation) for four meat-type chicken lines reared in commercial conditions.  
6  
7  
8                   2. Genetic components, including heritabilities and genetic correlations, were estimated by  
9 Restricted Maximum likelihood for these traits measured at six weeks of age.  
10  
11                   3. Data were collected over eight generations of selection and pedigrees comprised in excess of  
12 130,000 birds.  
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15                   4. Univariate analyses were performed to allow model definition and to obtain starting values  
16 for trivariate analyses. The basic model included a random animal effect and, in further models  
17 explored, a maternal environmental effect or a genetic maternal effect or both were fitted.  
18 Models were compared using likelihood ratio tests.  
19  
20                   5. Estimated heritabilities for SaO ranged from 0.1 to 0.2, and there was no evidence of genetic  
21 maternal effects for SaO. The environmental maternal component was significant for one of the  
22 populations only. Estimated heritabilities for both Weight and Flesh were between 0.2 and 0.4,  
23 and there was evidence of environmental and genetic maternal effects these traits for all  
24 populations.  
25  
26                   6. Genetic correlations between SaO and Weight and between SaO and Flesh were low and  
27 negative. This suggests that, in principle, genetic selection to simultaneously increase SaO, and  
28 therefore decrease ascites susceptibility, and Weight and Flesh could be performed using  
29 traditional (marker-free) selection methods. We discuss how a putative interaction between  
30 ascites and production traits could jeopardize the success of such methods.  
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## INTRODUCTION

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An increase in the incidence of ascites and ascites-related mortality has occurred in broiler populations reared at low altitudes in recent decades (Julian, 1993). During the last 50 years, improvement in production traits (e.g., growth, conformation, feed efficiency) through genetic selection has been spectacular (Havenstein et al., 2003) and has been paralleled by an increase in ascites incidence. This, and the fact that fast growing broilers are more susceptible to suffer from ascites than slow growing ones (Julian, 1993) has made that the increase in ascites incidence has been linked to the great genetic improvement in production traits. It is now accepted that ascites susceptibility is, at least partially, under genetic control in certain populations. Broiler breeders want to reduce the incidence of the disorder in breeding and commercial flocks and genetic selection against ascites susceptibility provides a more durable means of accomplishing this aim than management solutions, because the benefits of selection accumulate from generation to generation. The study of the genetic basis of ascites, and its relationship with growth traits, in meat-type chicken populations reared in commercial conditions is required in order to implement successful breeding programs that reduce ascites incidence and continue to improve production traits in a sustainable fashion in commercial flocks.

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Because the presence of ascites can only be confirmed by post-mortem examination (except for severe cases), selection against this disorder has been performed using family information. In recent years, a variety of physiological and anatomical measures have been evaluated as indicators of ascites susceptibility. One of these measures is blood oxygen saturation (SaO), which measures oxygen binding to haemoglobin. Julian and Mirsalimi (1992) showed that broilers suffering from ascites had lower SaO than fast or slow growing healthy contemporaries and that fast growing healthy birds had lower SaO than slow growing ones. Wideman et al. (1998) demonstrated that low SaO predicted susceptibility to ascites and Druyan et al. (1999) showed that SaO was heritable and that its genetic correlation with ascites mortality was negative in a cold-challenged broiler line. SaO has the advantage of being easily and non-invasively measured (Wideman et al., 1998) in large numbers of birds that can include selection candidates, which makes it convenient to use in commercial breeding programs.

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1 In this study, we explored a range of models to estimate the genetic parameters  
2 (heritabilities and genetic correlations) for SaO, weight (Weight) and fleshing score (Flesh, a  
3 measure of breast conformation) measured at six weeks of age in four large pedigrees of  
4 different meat-type chicken populations. Some analyses were only performed for line 3, and this  
5 line was chosen to illustrate results because further work was to be carried out on it, since it  
6 shows a slightly higher ascites-related mortality than other Aviagen Ltd. lines (A. Koerhuis,  
7 personal communication) and it is the heaviest of the lines studied.

## MATERIALS AND METHODS

### *Data Description*

18 Data on SaO, Weight (kg) and Flesh (1 to 5 scale), measured at six weeks of age, were  
19 available for four lines of meat-type chicken. SaO was directly measured using a pulse  
20 oximeter. A sensor was attached to a Criticare Pulse Oximeter 504US<sup>1</sup> and positioned on the  
21 wing to illuminate the tissue between the radius and ulna for measurement of percentage  
22 saturation of haemoglobin with oxygen (Julian and Mirsalimi, 1992). When the bird was settled,  
23 two readings were taken, and the record for this bird was taken to be an average of both  
24 readings. Fleshing is a conformation score with higher scores relating to superior breast muscle  
25 development relative to the size of the whole bird. Recording is carried out in handheld  
26 terminals grading the birds between 1 and 5, 1 being the birds with the least breast meat and 5  
27 the most. Feel of the breast breadth and depth and length of the keel are also taken into account.  
28 SaO data were only available for male selection candidates whereas Weight and Flesh were  
29 recorded on all birds. Other information included hatch number (with 224 levels for lines 1, 2  
30 and 3 and 113 levels for line 4), sex and age of dam when the egg was laid (in weeks, with 14  
31 levels).

**Deleted:** , where a high score means a good conformation),

32 All four lines have been closed for 30 to 40 generations. Lines 1, 2 and 3 have a history  
33 of selection on wheat diets whereas line 4 has been selected on a maize diet. Lines 1, 3 and 4 are  
34 male lines. Line 1 has been selected heavily on efficiency of growth and yield, with less

35 <sup>1</sup> Criticare Systems Inc., Milwaukee, U.S.A, supplied by R.L. Dolby, Stirling, UK

1 emphasis on growth rate; selection for lines 3 and 4 has been more focused on growth rate and  
2 efficiency and less emphasis on yield. Line 2 is a female that has been selected on growth rate  
3 and efficiency but also on egg production and hatchability. In addition to the abovementioned  
4 production traits all lines have been exposed to considerable family selection against broiler  
5 mortality and leg defects.

**Deleted:** Records on SaO were available only for male selection candidates. Other information included hatch number (with 224 levels for lines 1, 2 and 3 and 113 levels for line 4), sex and age of dam when the egg was laid (in weeks, with 14 levels).

6 Pedigrees for all lines consisted of around eight generations that were overlapping for  
7 all lines except line 4. Table 1 shows an overview of pedigree and data structure. We considered  
8 “base birds” the first generation of birds available in our pedigrees. These birds were not  
9 unrelated or unselected.

10 The size of the sibships was very variable within and between lines. Family sizes  
11 ranged from one to around 100 individuals per full-sib family (with a mean full-sib family size  
12 of 25 birds) and from one to around 580 birds per paternal half-sib family (with a mean half-sib  
13 family size of 160 birds). Not all birds had SaO data (although all had Weight and Flesh  
14 records) and the mean number of males with SaO data per recorded full-sib and paternal half-sib  
15 family was three and 17 respectively. At the time when data were recorded, the overall mortality  
16 for these lines was 5 to 10% and mortality caused by ascites was part of this proportion.

17 [TABLE 1]

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18 Descriptive statistics of the distributions of SaO, Weight and Flesh were obtained with  
19 GENSTAT (GENSTAT 5 COMMITTEE, 1993). In this and the following section, basic results  
20 will be presented for all lines but more detailed descriptions will, in some cases, be presented  
21 only for line 3, which is the line for which further studies were carried out.

### 22 *Genetic Analyses*

23 Genetic parameters were estimated by Restricted Maximum Likelihood in an animal  
24 model using ASREML (Gilmour et al., 2000). Fixed effects fitted were hatch number and age of  
25 dam for all three traits and sex for Weight and Flesh (because SaO was only recorded for  
26 males). The expression of some traits can be influenced by the ability of the individual's dam to  
27 provide a suitable environment. This ability can be partly environmental and partly genetic (see,  
28 for example, Mrode (1996)) and in the case of birds reared in commercial conditions, this  
29 influence is mostly reduced to the quality and composition of the egg (given that, for example,  
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**Deleted:** Age of dam affects egg characteristics and this can affect progeny traits. For example, older parents produce larger eggs, which have a greater oxygen demand, but a low surface-area to volume ratio and this is likely to increase ascites incidence in the pre-hatching period.



brooding behaviour would be irrelevant in this case). Initially, a series of single trait analyses were carried out for all traits. Four different models were considered:

Model 1 included a random animal effect in addition to a residual term.

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Model 2 included a maternal environmental component, modelled as a common dam

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effect for all maternal half-sibs, in addition to the terms in Model 1.

Model 3 included a maternal genetic component in addition to the terms in Model 1.

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Model 4 included a maternal environmental component and a maternal genetic

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component in addition to the terms in Model 1.

Self and Liang (1987) suggested that, when testing the hypothesis that a parameter has an unspecified positive value versus the hypothesis that this parameter is zero, the asymptotic distribution of  $-2 \ln LR$  is a 50 : 50 mixture of a  $\chi^2$  with zero degrees of freedom and a  $\chi^2$  with one degree of freedom. The significance of the maternal environmental and genetic components was tested using this criterion and the best model for each trait was chosen for further (multitrait) analyses. Estimates of variance components from the univariate analyses were used as starting values for variance components for the trivariate analyses. The starting values for the additive, maternal -genetic or environmental- and residual covariances amongst the traits were zero and other covariance components (for example, amongst additive genetic and genetic maternal components) were not estimated.

Deleted: Stram and Lee (1994) suggested (following

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## RESULTS

### *Data Description*

Table 2 presents the means and standard deviations for the raw phenotypic data for the traits analyzed. For all the lines, the distributions of the raw SaO data were non-Normal (negatively skewed, with skewness coefficients ranging from  $-0.7$  to  $-0.8$  and leptokurtic, with kurtosis coefficients of around 0.5). Figure 1 shows the distribution of SaO phenotypes for all individuals in the pedigree that had an SaO record for line 3. Distributions for other lines were similar. Regression lines were fitted to mean SaO phenotypes for each hatch plotted over time (hatch number) as well as intervals covering from the first to the third quartile of each hatch distribution. These fitted lines show that, over time, mean and first and third quartiles hatch SaO

Deleted: Figure 2 shows

Deleted: Regressions

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1 values increased ( $P \leq 0.0001$ ). Fitting different regressions for means and quartiles explained  
 2 the data better than a model for which only one regression coefficient was fitted ( $P \leq 0.005$ ),  
 3 and the slope for the first quartile regression was greater than the slope for the mean regression,  
 4 that was in turn greater than the slope for the third quartile regression. This meant that the  
 5 variance of SaO values decreased over time.  
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9 [TABLE 2], [FIGURE 1]

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 12 The transformation  $\text{Ln}(100 - x)$ , where  $x$  is SaO was found to decrease the skewness of  
 13 the SaO distribution (skewness coefficients from -0.3 to -0.4). Figure 1 also shows the  
 14 distribution of the transformed phenotypes for line 3. Genetic parameters were obtained for both  
 15 untransformed and transformed SaO data.  
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Deleted: [FIGURE 2]¶

### 20 21 22 *Genetic Analyses*

23 Throughout this section results are only presented for analyses of untransformed SaO  
 24 data, because analyses of transformed data yielded similar variance ratios and results obtained  
 25 on the untransformed scale are easier to interpret.  
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29 Table 3 shows the estimates of variance components for untransformed SaO data,  
 30 Weight and Flesh obtained from univariate analyses for Models 1, 2, 3 and 4. Estimates  
 31 obtained for the model chosen for further analyses are shown in bold. For some of the analyses  
 32 of SaO data, the maternal genetic component tended to move past the boundary (zero) and was  
 33 fixed near it. Estimates from these analyses are shown in italics and underlined, and the models  
 34 that produced them (within line and trait) were not used in further analyses.  
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40 [TABLE 3]

41 Table 4 shows  $-2\text{LnLR}$  obtained from comparison of different models and an indication  
 42 of the improvement in fit of the full compared to the reduced model (e.g., Model 2 compared to  
 43 Model 1, etc.). In brief, for Weight and Flesh, the “best model” for all lines was Model 4, that  
 44 included both an environmental and a genetic maternal component. For SaO data, Model 1 was  
 45 the “best model” for lines 1, 3 and 4, whereas Model 2 (including an environmental maternal  
 46 component) was the “best model” for line 2.  
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[TABLE 4]

Although the “best model” for Weight and Flesh for all lines was Model 4, trivariate analyses including a maternal genetic component could not be performed for lines 2 and 4 due to the large size of these pedigrees. Instead results are presented for (trivariate) analyses not including a genetic maternal component but including an environmental maternal component for all traits (line 2) or for Weight and Flesh (line 4).

Table 5 shows heritabilities ( $h^2$ ), genetic and phenotypic correlations and their standard errors, obtained for (untransformed) SaO, Weight and Flesh. Variance ratios and correlations are also shown for the environmental and genetic maternal components when available.  $h^2$  for SaO varied in the range 0.12 to 0.21, the lowest value corresponding to lines 2 and 4 and the highest to line 3. In univariate analyses, only line 2 showed significant environmental maternal effects for SaO, and these explained around 2% of the total variance. The significance of the maternal environmental component for SaO was retested in trivariate analyses. The full model included one variance (environmental maternal component for SaO) and two covariances more than the reduced model and  $-2\text{LnLR}$  was therefore compared to a 50 : 50 mixture of a  $\chi^2$  with two degrees of freedom and a  $\chi^2$  with three degrees of freedom (Self and Liang, 1987). The results obtained from these tests were consistent with univariate results.

[TABLE 5]

$h^2$  for Weight ranged between 0.26 and 0.41, the lowest value corresponding to line 3 and the highest to line 4. The inclusion of both environmental and genetic maternal effects improved significantly the fit of the model in univariate analyses, and these components explained between 2 and 6% and 1% of the total variance respectively (the genetic maternal component was only available for lines 1 and 3). Line 4 was the one for which the environmental maternal component explained the largest proportion of variance compared to other lines.

$h^2$  for Flesh was approximately in the same range as  $h^2$  for Weight and its lowest value (0.22) corresponded to lines 1 and 3, the highest (0.41) corresponding to line 4. Environmental and genetic maternal effects jointly explained less than 3% of the total variance for this trait.

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Genetic correlations of SaO with Weight were low and negative (between -0.17 and -0.02) for all lines and were not significantly different from zero for line 3 ( $P \geq 0.05$ ). Genetic correlations of SaO with Flesh were approximately in the same range and were not significantly different from zero for lines 2 and 3 ( $P \geq 0.05$ ). Genetic correlations between the two production traits were positive and moderate to high, ~~from~~ 0.31 for line 4 ~~to~~ 0.64 for line 2. Correlations of environmental and genetic maternal effects were generally high between these two traits and approached unity in some cases.

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Table 6 shows the estimates of genetic, maternal environmental, maternal genetic, and residual and phenotypic variances ( $\sigma_u^2$ ,  $\sigma_{em}^2$ ,  $\sigma_{gm}^2$ ,  $\sigma_e^2$  and  $\sigma_p^2$ , respectively) obtained from trivariate analyses for SaO, Weight and Flesh for all lines studied. The fact that the estimated  $h^2$  of SaO for line 3 was almost twice the value estimated for lines 2 and 4 reflects differences in estimated  $\sigma_u^2$  rather than differences in  $\sigma_e^2$ .  $\sigma_e^2$  for Weight appeared to be greater for line 3 than for other lines and this was reflected in a lower estimated  $h^2$  for this line. The estimated  $\sigma_u^2$  for Flesh was almost double for line 4 than for the other lines, and  $h^2$  of Flesh for this line was twice the value estimated for the others.

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[TABLE 6]

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The heritabilities obtained when performing the analysis using transformed SaO phenotypes did not differ from the ones obtained from untransformed data and are therefore not presented. Because of the nature of the transformation, genetic and phenotypic correlations of SaO with Weight and Flesh were different in sign but not different in magnitude.

Figure 2 shows the mean additive value (in additive standard deviations) for SaO, Weight and Flesh for each hatch for line 3. Estimated breeding values from all birds in the pedigree (not only the ones with records for SaO) were used to obtain the graph. Figure 2 shows that, although mean hatch values increased over time for all three traits, the rate of increase was a greater for Flesh and Weight than for SaO.

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[FIGURE 2]

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## DISCUSSION

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3 We have demonstrated that there was substantial genetic variation ( $h^2$  of 0.1 to 0.2) for  
4 SaO in the four populations studied and therefore genetic selection on this trait is feasible.  
5  
6 Although a slightly higher heritability and additive genetic variance was obtained for SaO in  
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8 line 3, in general the estimates of variances, heritabilities and genetic correlations with Weight  
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10 and Flesh are consistent across lines. Druyan et al. (1999) obtained estimates of  $h^2$  for SaO of  
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12 around 0.5 to 0.6. Unlike our populations, the population Druyan et al. (1999) used in their  
13 study was cold-challenged, and this is common in studies of ascites-related traits (see Table 7),  
14  
15 with few exceptions (e.g., Moghadam et al. (2001)). Rearing birds in challenging environments  
16  
17 increases the incidence of the disorder and therefore facilitates selection against it. Table 7  
18 shows heritabilities of a selection of ascites-related traits and genetic correlations with body  
19 weight from published studies. All traits presented are heritable, but estimates of heritabilities  
20 varied widely across studies for similar traits. Moghadam et al. (2001) estimated that  $h^2$  for  
21 ascites mortality obtained from male data alone were higher than the ones obtained jointly from  
22 male and female data.  
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[TABLE 7]

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31 The heritabilities obtained for body weight are similar to the ones reported in the  
32 literature (0.2 to 0.4) (e.g., Dunnington and Siegel (1996), Koerhuis and Thompson (1997)).  
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34 Despite decades of selection on growth or body weight, genetic variation has not been depleted  
35 for these (Rose, 1997) or related traits such as feed conversion (Emmerson, 1997); heritabilities  
36 are still moderate to high in contemporary broiler strains and have not diminished markedly  
37 compared with earlier estimates (Hill, 1996). The estimates of  $h^2$  obtained for Flesh were of the  
38 same size of those for body weight, and although selection on this trait has not been taking place  
39 for as long as for growth traits, the maintenance of this level of genetic variation is also  
40 remarkable.  
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47 Hill (1996) suggested that mutational variance could be an important source of new  
48 genetic variation. Also, the fact that there is still genetic variation may support the idea that  
49 many genes with very small effects influence those characters. Alternatively, the mode of action  
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1 of individual genes, directly on these production traits or indirectly through actions on fitness  
2 traits, and interactions amongst loci could explain that genetic variation is maintained; Hill  
3 (2002) also pointed out that, under certain circumstances, selection favoured most variable  
4 genotypes. Breeding strategies also influence changes of genetic variance over time: for  
5 example, new variation could be brought in by the incorporation to the population of  
6 immigrants from different lines and fixation of unfavourable alleles could be prevented in this  
7 way; this influence could as well be through the selection strategy implemented, for instance the  
8 use of mass selection during the early years of the poultry breeding industry may have  
9 contributed to maintain high effective population sizes (Hill, 1996).

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17 Pakdel et al. (2002) and Koerhuis and Thompson (1997) demonstrated, respectively, the  
18 significance of genetic maternal effects on ascites related traits and environmental and genetic  
19 maternal effects on body weight. In our study, the inclusion of a maternal environmental  
20 component for SaO only improved significantly the fit of the model for line 2. The structure of  
21 our SaO data may have had an impact on our results: a large proportion (around 30%) of  
22 maternal half-sib families were of size one, and the mean maternal family size for SaO data was  
23 around three. Datasets with larger maternal family sizes could facilitate a better estimation of  
24 environmental maternal effects for SaO and allow exploration of the existence of genetic  
25 maternal effects for this trait in our populations. Significance of the maternal environmental  
26 component was re-tested in trivariate analyses yielding similar results to the ones obtained in  
27 univariate analyses. In the case of Pakdel et al. (2002), although the genetic maternal component  
28 explained under 5% of the total variance for all traits, its inclusion in the analysis model had a  
29 large impact on the estimated heritability, halving it for most traits, and the correlation of the  
30 additive and the maternal genetic components was moderate or high depending on the traits. In  
31 our case, although the inclusion of a maternal environmental component slightly decreased  
32 heritability of SaO for line 2, this decrease was only small, as was the decrease in estimated  
33 additive genetic variance. Maternal family sizes for Weight and Flesh data were considerably  
34 greater than for SaO data, allowing for the estimation of both environmental and genetic  
35 maternal effects.

1 Koerhuis and Thompson (1997) estimated that environmental and genetic maternal  
2 effects explained respectively fewer than 10% and 5% of the total variance for juvenile body  
3 weight. In their case, environmental maternal effects were divided into both common maternal  
4 half-sib effects and common full-sib family effects. Our results show that both heritable and  
5 non-heritable maternal effects are significant for both Weight and Flesh, although the  
6 percentage of the total variance explained jointly by these effects was less than 10% for both  
7 traits across lines. Their inclusion in the analysis model reduced estimated heritabilities  
8 generally more markedly for Weight than for Flesh. We attempted to estimate the correlation  
9 between the direct additive (i.e. animal) component and the genetic maternal component, but in  
10 the cases where the size of the pedigree allowed the analyses to be run, those failed to converge  
11 and therefore no results have been presented. For lines 2 and 4, multitrait analyses including a  
12 genetic maternal component could not be run and results were presented instead for analyses  
13 including only an environmental genetic component. This, most likely implies that  $\sigma_{em}^2$  would  
14 be overestimated and would include part of  $\sigma_{gm}^2$ . Based on results from univariate analyses,  
15 genetic maternal effects would be larger than environmental ones for line 4 but approximately  
16 of the same size for line 2.

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29 Because ascites is a production-related disorder caused by high oxygen requirements of  
30 rapid growth, one would expect a positive correlation between growth-related traits and  
31 incidence of the disorder. The sign of our estimates of genetic correlations between SaO and  
32 Weight and Flesh meets the expectation because SaO is negatively correlated with ascites  
33 susceptibility. Nevertheless, the estimated correlations of SaO with production traits were  
34 relatively low, suggesting that selection to increase SaO (and therefore reduce ascites  
35 susceptibility) and production traits simultaneously would be possible in the populations  
36 studied. Correlations of other ascites indicator traits with body weight are shown in Table 7.  
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1 are affected by the presence of the disorder and that this effect depends on the proportion of  
2 affected individuals. They suggested that this phenomenon can be viewed as a genotype x  
3 environment interaction, in which the internal environment of the bird (ascites presence or  
4 absence) might affect the ability of fast growing animals to fully express their growth potential,  
5 so they would show below-average growth. At the same time, changes in metabolic pressure  
6 caused by increased growth trigger the presence of the disorder in genetically prone birds that  
7 would not be affected if growth were somehow controlled by management practices and growth  
8 acts as an internal environment that affects ascites presence. These ideas were previously  
9 suggested by Julian (1993). To check their hypothesis, they analyzed a subset of data containing  
10 only animals not affected with ascites (the dataset was divided on the basis of heart-API  
11 observed values). The sign of the correlation obtained from this data set changed, meeting their  
12 original expectations. Pakdel et al. (2005) estimated genetic correlations between body weight  
13 and several ascites-related traits from both a cold-challenged and a non-cold-challenged  
14 population. Their estimated correlations between body weight and heart-API and body weight  
15 and packed cell volume (PCV) were different in sign depending on the population they were  
16 estimated from (cold-challenged or not). They argue that the same phenomena described by De  
17 Greef et al. (2001) could be responsible for this observation. Because our populations were not  
18 cold-challenged, presumably the incidence of the disorder was lower than for the population  
19 studied by De Greef et al. (2001) and the effect on estimated genetic correlations would be  
20 smaller. To check the effect that estimating genetic correlations from subsets of potentially  
21 ascitic (i.e., with low SaO records) and non-ascitic birds had in our case we focused on line 3  
22 and divided the dataset into subsets of “ascitic” (with SaO records lower than 60 or 75) and  
23 “non-ascitic” birds (with SaO records higher than 60, 80, 90 or 95). For these analyses, the  
24 complete pedigrees were used and phenotypic records below or above the selected cut-off points  
25 were omitted. Table 8 material, shows estimates of heritabilities and genetic correlations for  
26 these analyses.

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[TABLE 8]

27 Although estimated heritabilities of production traits and genetic correlations amongst  
28 them were similar for all subsets, this was not the case for SaO and correlations of this trait with  
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1 Flesh and Weight (N.B. these correlations were not different from zero ( $P \geq 0.05$ ) in any case,  
2 possibly due to the small sample size of some subsets). Estimated heritabilities for SaO varied  
3 across subsets, and were close to zero for subsets of birds with SaO records smaller than 75% or  
4 greater than 80% SaO. For subsets with SaO records greater than 80%, the expected sign of the  
5 correlation with production traits would be negative (given that the expected sign of the genetic  
6 correlation between production and ascites incidence would be positive and SaO is negatively  
7 correlated with ascites incidence), and this was not observed. The estimated genetic correlations  
8 of SaO with production traits obtained from the subset of birds with less than 60% SaO were  
9 positive (as expected for affected birds) and moderate (apparently larger than the one estimated  
10 from the whole dataset, but caution must be taken in the interpretation of this result given the  
11 large standard error of the estimate). In conclusion, the proportion of susceptible birds (assigned  
12 on the basis of SaO records) seems to have an effect on the estimated genetic correlations  
13 between SaO and production traits, but no clear trends were identified, and differences in  
14 sample size of the different subsets make results still more difficult to interpret. These analyses  
15 are also difficult to interpret in that the estimation of parameters was carried out for the trait  
16 used to create the subsets of data. Anyhow, if these effects were real, they would have an impact  
17 on the success of programs aiming to improve SaO and production traits simultaneously.

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31 McMillan and Quinton (2002) conducted a simulation study to assess the changes in  
32 ascites incidence when selection was performed on body weight alone or also on an ascites  
33 indicator trait, under a variety of scenarios. They concluded that selection for improved growth  
34 and reduced ascites could be effective, more so if an indicator trait was used together with sib  
35 information prior to selection for growth, but noticed that this reduced gain in the latter trait.  
36 The authors showed that the higher the correlation of the indicator trait with ascites, the greater  
37 was the genetic reduction of the population's ascites susceptibility. We do not have estimates of  
38 correlations between SaO and ascites incidence in our populations, but Druyan et al. (1999)  
39 estimated that the genetic correlation of SaO with ascites mortality was around -0.50. McMillan  
40 and Quinton (2002) also observed that a reduction in the genotypic mean of ascites  
41 susceptibility did not always translate into a decrease in the incidence of the disorder, but rather  
42 the opposite, and suggested that this was an effect of added metabolic pressure of the  
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1 improvement in growth potential and therefore a change in the internal environment of the  
2 birds. However, this result could depend on the model used in their simulations.

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5 Finally, Figure 2 shows that, although heritabilities for SaO, Weight and Flesh were  
6 similar for line 3, and genetic progress was being made for the three traits studied, improvement  
7 was more pronounced for production traits. This could be a consequence of breeding strategies  
8 but could also be caused by a failure of the models used to adequately describe the genetic  
9 architecture of the traits studied and their genetic relationships. The exploration of the data  
10 using more complex models (including, for example, a locus or several loci with large effect, in  
11 addition to a polygenic component) may give a better insight of the genetic architecture of SaO  
12 and its relationship with Weight and Flesh. If such loci existed, their identification could make  
13 the reduction of ascites incidence in broiler flocks an easier task, allowing for direct  
14 manipulation of allele frequencies at this locus or loci, therefore bypassing potential problems  
15 due to effects of allele, gene or genotype x environment interactions that could hinder the  
16 effectiveness of more traditional selection methods.  
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[TABLE S1]¶  
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**Table 1. Pedigree and data structure for blood oxygen saturation (SaO), weight (Weight) and fleshing score (Flesh, a measure of breast conformation) for the four populations**

N	Line 1	Line 2	Line 3	Line 4
Birds in pedigree	179,565	320,550	134,986	206,876
Base generation birds	1,226	1,421	1,018	1,502
Birds with record for SaO	23,134	17,513	11,919	9,055
Birds with record for Weight and Flesh	133,603	318,740	118,782	205,063
Sires/paternal half-sib families	935	1,174	755	1,218
Dams	5,864	8,500	4,809	8,677
Full-sib families	7,788	11,560	6,563	10,640

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Table 2. Means and standard deviations (in brackets) for blood oxygen saturation (SaO), weight (Weight) and fleshing score (Flesh, a measure of breast conformation), for the four lines studied, all birds together (All), males (Male) and females (Female).

Line		SaO (%)	Weight (kg)	Flesh (units)
1	All	79.85 (9.38)	2.55 (0.30)	3.19 (0.91)
	Male	<u>79.85 (9.38)</u>	<u>2.73 (0.26)</u>	<u>3.17 (0.91)</u>
	Female	NA <sup>1</sup>	<u>2.38 (0.22)</u>	<u>3.22 (0.90)</u>
2	All	82.32 (8.52)	2.14 (0.29)	3.04 (0.87)
	Male	<u>82.32 (8.52)</u>	<u>2.31 (0.26)</u>	<u>3.01 (0.88)</u>
	Female	NA	<u>1.97 (0.22)</u>	<u>3.07 (0.86)</u>
3	All	80.02 (9.15)	2.72 (0.34)	3.17 (0.92)
	Male	<u>80.02 (9.15)</u>	<u>2.92 (0.29)</u>	<u>3.12 (0.93)</u>
	Female	NA	<u>2.52 (0.25)</u>	<u>3.23 (0.90)</u>
4	All	75.30 (8.95)	2.26 (0.31)	3.09 (0.96)
	Male	<u>75.30 (8.95)</u>	<u>2.45 (0.27)</u>	<u>3.05 (0.96)</u>
	Female	NA	<u>2.09 (0.23)</u>	<u>3.13 (0.96)</u>

<sup>1</sup> NA: not available.

Table 3. Estimates of genetic, maternal environmental, maternal genetic, residual and phenotypic variances ( $\sigma_u^2$ ,  $\sigma_{em}^2$ ,  $\sigma_{gm}^2$ ,  $\sigma_e^2$  and  $\sigma_p^2$ ) obtained from univariate analyses for blood oxygen saturation (SaO), body weight (Weight) and fleshing score (Flesh, a measure of breast conformation) for all lines. Estimates obtained for the model chosen for further analyses are shown in bold. For some of the analyses of SaO data, the maternal genetic component tended to move past the boundary (zero) and was fixed near it. Estimates from these analyses are shown in italics and underlined.

Line	Trait	Model 1			Model 2				Model 3				Model 4				
		$\sigma_u^2$	$\sigma_e^2$	$\sigma_p^2$	$\sigma_u^2$	$\sigma_{em}^2$	$\sigma_e^2$	$\sigma_p^2$	$\sigma_u^2$	$\sigma_{gm}^2$	$\sigma_e^2$	$\sigma_p^2$	$\sigma_u^2$	$\sigma_{em}^2$	$\sigma_{gm}^2$	$\sigma_e^2$	$\sigma_p^2$
1	SaO	<b>11.29</b>	<b>58.00</b>	<b>69.28</b>	10.84	0.23	58.11	69.19	10.72	0.25	58.22	69.19	<i>10.60</i>	<i>0.11</i>	<i>0.21</i>	<i>58.24</i>	<i>69.16</i>
1	Weight	239.50	277.04	516.54	171.76	12.48	308.21	492.45	192.46	13.71	298.45	504.63	<b>168.45</b>	<b>9.87</b>	<b>4.29</b>	<b>309.88</b>	<b>492.48</b>
1	Flesh	0.24	0.62	0.86	0.18	0.01	0.65	0.84	0.21	0.01	0.64	0.85	<b>0.18</b>	<b>0.01</b>	<b>0.00</b>	<b>0.65</b>	<b>0.84</b>
2	SaO	9.69	54.13	63.82	<b>7.66</b>	<b>1.09</b>	<b>54.66</b>	<b>63.42</b>	9.47	0.11	54.22	63.79	<i>7.67</i>	<i>1.09</i>	<i>0.00</i>	<i>54.65</i>	<i>63.41</i>
2	Weight	176.29	173.76	350.05	116.55	9.27	201.95	327.77	130.77	11.59	195.07	337.42	<b>113.59</b>	<b>6.23</b>	<b>4.66</b>	<b>203.41</b>	<b>327.88</b>
2	Flesh	0.26	0.55	0.81	0.18	0.01	0.59	0.78	0.21	0.01	0.58	0.80	<b>0.18</b>	<b>0.01</b>	<b>0.00</b>	<b>0.59</b>	<b>0.78</b>
3	SaO	<b>14.13</b>	<b>54.17</b>	<b>68.29</b>	12.07	1.20	54.60	67.88	13.48	0.37	54.37	68.21	<i>12.07</i>	<i>1.21</i>	<i>0.00</i>	<i>54.61</i>	<i>67.88</i>
3	Weight	271.74	382.80	654.54	164.81	17.87	433.54	616.22	195.25	19.65	419	633.9	<b>161.86</b>	<b>13.49</b>	<b>6.71</b>	<b>434.9</b>	<b>616.96</b>
3	Flesh	0.27	0.62	0.89	0.19	0.02	0.66	0.87	0.22	0.01	0.64	0.88	<b>0.19</b>	<b>0.01</b>	<b>0.00</b>	<b>0.66</b>	<b>0.86</b>
4	SaO	<b>8.02</b>	<b>57.32</b>	<b>65.35</b>	7.98	0.04	57.32	65.34	<i>8.15</i>	<i>0.00</i>	<i>57.23</i>	<i>65.38</i>	<i>8.20</i>	<i>0.01</i>	<i>0.00</i>	<i>57.17</i>	<i>65.38</i>
4	Weight	234.07	214.14	448.21	183.21	28.29	231.01	442.50	185.74	48.48	229.06	463.27	<b>181.22</b>	<b>9.42</b>	<b>32.46</b>	<b>231.21</b>	<b>454.3</b>
4	Flesh	0.47	0.52	0.99	0.41	0.01	0.55	0.97	0.42	0.02	0.54	0.98	<b>0.40</b>	<b>0.01</b>	<b>0.01</b>	<b>0.55</b>	<b>0.97</b>

<sup>1</sup> Model 1 includes a random animal effect and a residual term; Model 2 includes a maternal environmental component, modelled as a common dam effect for all maternal half-sibs, in addition to the terms in Model 1; Model 3 includes a maternal genetic component, in addition to the terms in Model 1; Model 4 includes, in addition to the terms in Model 1, both a maternal environmental component and a maternal genetic component.

**Table 4. Minus two times the logarithm of the likelihood ratio (- 2LnLR) and corresponding P-values (P) obtained for comparison of models used in univariate analyses for blood oxygen saturation (SaO), body weight (Weight) and fleshing score (Flesh, a measure of breast conformation) for all lines**

Line	Trait	Model 1 vs. Model 2		Model 1 vs. Model 3		Model 2 vs. Model 4		Model 3 vs. Model 4	
		- 2LnLR	P	- 2LnLR	P	- 2LnLR	P	- 2LnLR	P
1	SaO	0.4	0.26	0.8	0.19	NA	NA	NA	NA
1	Weight	188.0	*** <sup>2</sup>	142.0	***	16.0	***	62.0	***
1	Flesh	89.8	***	56.4	***	8.8	**	42.2	***
2	SaO	7.8	*** <sup>3</sup>	0.2	0.33	NA	NA	NA	NA
2	Weight	500.0	***	520.0	***	120.0	***	100.0	***
2	Flesh	222.0	***	160.0	***	30.0	***	92.0	***
3	SaO	2.1	0.07	0.8	0.19	NA	NA	NA	NA
3	Weight	162.0	***	132.0	***	18.0	***	48.0	***
3	Flesh	82.2	***	49.0	***	5.2	**	38.4	***
4	SaO	0.0	0.50	NA <sup>4</sup>	NA	NA	NA	NA	NA
4	Weight	1,428.0	***	1,694.0	***	302.0	***	36.0	***
4	Flesh	131.2	***	154.0	***	47.4	***	24.6	***

<sup>1</sup> Model 1 includes a random animal effect and a residual term; Model 2 includes a maternal environmental component, modelled as a common dam effect for all maternal half-sibs, in addition to the terms in Model 1; Model 3 includes a maternal genetic component, in addition to the terms in Model 1; Model 4 includes, in addition to the terms in Model 1, both a maternal environmental component and a maternal genetic component.

<sup>2</sup> \*\*\* P < 0.001.

<sup>3</sup> \*\* P ≤ 0.01.

<sup>4</sup> NA: not available.



**Table 5. Heritabilities (on diagonals and bold at “Direct Genetic and Phenotypic” column), genetic and phenotypic correlations (below and above diagonal respectively) and their standard errors (in brackets), obtained for blood oxygen saturation (SaO), weight (Weight) and fleshing score (Flesh, a measure of breast conformation). Variance ratios and correlations are presented for the environmental and genetic maternal components when relevant**

Line	Trait	Direct	Genetic	and	Maternal	Maternal Genetic		
		Phenotypic	Weight	Flesh	Environmental	SaO	Weight	Flesh
1	SaO	<b>0.17</b> (0.01)	-0.00 (0.01)	-0.04 (0.01)				
1	Weight	-0.17 (0.04)	<b>0.34</b> (0.01)	0.57 (0.00)	0.02 (0.00)		0.01 (0.00)	
1	Flesh	-0.21 (0.04)	0.59 (0.02)	<b>0.22</b> (0.01)	0.76 (0.05)	0.01 (0.00)	0.87 (0.12)	0.00 (0.00)
2	SaO	<b>0.12</b> (0.02)	-0.02 (0.01)	-0.07 (0.01)	0.02 (0.00)			
2	Weight	-0.13 (0.06)	<b>0.35</b> (0.01)	0.58 (0.00)	0.00 (0.12)	0.03 (0.00)		
2	Flesh	-0.11 (0.06)	0.64 (0.01)	<b>0.24</b> (0.01)	-0.07 (0.13)	0.90 (0.02)	0.02 (0.00)	
3	SaO	<b>0.21</b> (0.02)	0.01 (0.01)	-0.04 (0.01)				
3	Weight	-0.02 (0.06)	<b>0.26</b> (0.01)	0.57 (0.00)	0.02 (0.00)		0.01 (0.00)	
3	Flesh	-0.10 (0.06)	0.53 (0.03)	<b>0.22</b> (0.01)	0.68 (0.00)	0.01 (0.00)	0.77 (0.00)	0.00 (0.00)
4	SaO	<b>0.12</b> (0.02)	-0.02 (0.01)	-0.05 (0.01)				
4	Weight	-0.17 (0.07)	<b>0.41</b> (0.01)	0.38 (0.00)	0.06 (0.00)			
4	Flesh	-0.15 (0.06)	0.31 (0.02)	<b>0.41</b> (0.01)	0.62 (0.00)	0.02 (0.00)		

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**Table 6. Estimates of genetic, maternal environmental, maternal genetic, residual and phenotypic variances ( $\sigma_u^2$ ,  $\sigma_{em}^2$ ,  $\sigma_{gm}^2$ ,  $\sigma_e^2$  and  $\sigma_p^2$ ) obtained from trivariate analyses for blood oxygen saturation (SaO), body weight (Weight) and fleshing score (Flesh, a measure of breast conformation) for all lines**

Line	Trait	$\sigma_u^2$	$\sigma_{em}^2$	$\sigma_{gm}^2$	$\sigma_e^2$	$\sigma_p^2$
1	SaO	11.53	NA <sup>1</sup>	NA	57.88	69.41
1	Weight	1.66x10 <sup>-02</sup>	1.03x10 <sup>-03</sup>	4.08x10 <sup>-04</sup>	3.11x10 <sup>-02</sup>	4.91x10 <sup>-02</sup>
1	Flesh	0.19	0.01	0.00	0.64	0.85
2	SaO	7.97	1.09	NA	54.61	63.67
2	Weight	1.15x10 <sup>-02</sup>	9.47x10 <sup>-04</sup>	NA	2.03x10 <sup>-02</sup>	3.27x10 <sup>-02</sup>
2	Flesh	0.19	0.01	NA	0.58	0.79
3	SaO	14.37	NA	NA	54.06	68.43
3	Weight	1.59x10 <sup>-02</sup>	1.38x10 <sup>-03</sup>	6.80x10 <sup>-04</sup>	4.36x10 <sup>-02</sup>	6.16x10 <sup>-02</sup>
3	Flesh	0.19	0.01	0.00	0.66	0.87
4	SaO	8.09	NA	NA	57.34	65.43
4	Weight	1.81x10 <sup>-02</sup>	2.88x10 <sup>-03</sup>	NA	2.32x10 <sup>-02</sup>	4.42x10 <sup>-02</sup>
4	Flesh	0.40	0.01	NA	0.55	0.96

<sup>1</sup> NA: not available.

**Table 7. Selection of ascites-related traits and estimates of their heritabilities and genetic correlations with body weight ( $r_g W$ )**

Trait <sup>1</sup>	Heritability	$r_g W$	Cold? <sup>2</sup>	Comments	Ref <sup>3</sup>
Ascites mortality	0.11 to 0.44	NA <sup>4</sup>	Yes		1
	0.06	NA	Yes		2
	0.12 to 0.21	NA	No	Data on males and females	3
Total mortality	0.22 to 0.41	0.22 to 0.35	No	Data on males	3
	0.22	-0.46	Yes		2
	0.16	NA	Yes	Maternal genetic effects significant	4
Heart-API	0.32	-0.06	Yes	Maternal genetic effects not considered	5
	0.21 to 0.27	NA	Yes		1
	0.54	-0.26	Yes		2
PCV	0.28	NA	Yes	Maternal genetic effects significant	4
	0.45	-0.27	Yes	Maternal genetic effects not considered	5
	0.12	0.50	No	Maternal genetic effects not considered	5
	0.42 to 0.72	NA	Yes		6
	0.5	-0.54	Yes		2
SaO	0.5	NA	Yes		4
	0.46	-0.23	Yes	Maternal genetic effects not considered	5
	0.17	0.55	No	Maternal genetic effects not considered	5
	0.74 to 0.88	NA	Yes		6
APO <sub>2</sub>	0.53 to 0.63	NA	Yes		6
Troponin T	0.13	0.12	Yes		2
Heart rate	0.38	NA	NA		7
	0.52 to 0.69	NA	Yes		6

<sup>1</sup> Heart-API = ratio of the weight of the right heart ventricle to total heart weight; PCV = packed

cell volume; SaO = blood oxygen saturation; APO<sub>2</sub> = arterial oxygen pressure (related with SaO).

<sup>2</sup> Cold? = Was the population cold-challenged?

<sup>3</sup> Reference key: 1 = Lubritz et al. (1995); 2 = De Greef et al. (2001); 3 = Moghadam et al. (2001); 4 = Pakdel et al. (2002); 5 = Pakdel et al. (2005); 6 = Druyan et al. (1999); 7 = Maxwell et al. (1998).

<sup>4</sup> NA: not available.

Table 8. Estimates of heritabilities for blood oxygen saturation (SaO), weight (Weight) and fleshing score (Flesh, a measure of breast conformation) ( $h^2S$ ,  $h^2W$  and  $h^2F$ ) and genetic correlations amongst these traits ( $r_{gSW}$  is the correlation between SaO and Weight,  $r_{gSF}$  is the correlation between SaO and Flesh and  $r_{gWF}$  is the correlation between Weight and Flesh) obtained from different subsets of data from line 3. The original data set was divided on the basis of SaO measures to separate ascites-susceptible from ascites-resistant birds. Standard errors of estimates are shown in brackets. n is the number of birds with records per subset of data

Data	Cut-off SaO	n	$r_{gSW}$	$r_{gSF}$	$r_{gWF}$	$h^2S$	$h^2W$	$h^2F$
Ascites susceptible	< 60 %	315	0.30 (0.36)	0.37 (0.38)	0.48 (0.03)	0.19 (0.29)	0.23 (0.01)	0.18 (0.01)
	< 75 %	3062	-0.23 (0.22)	-0.09 (0.21)	0.48 (0.03)	0.03 (0.02)	0.23 (0.01)	0.19 (0.01)
Full dataset	None	11,919	-0.02 (0.06)	-0.10 (0.06)	0.53 (0.03)	0.21 (0.02)	0.26 (0.01)	0.22 (0.01)
	> 60 %	11,506	-0.04 (0.06)	-0.12 (0.06)	0.54 (0.03)	0.16 (0.02)	0.26 (0.01)	0.22 (0.01)
Ascites resistant	> 80 %	6,499	0.16 (0.13)	-0.02 (0.12)	0.50 (0.03)	0.04 (0.01)	0.24 (0.01)	0.20 (0.01)
	> 85 %	3,871	0.08 (0.17)	0.10 (0.16)	0.48 (0.03)	0.04 (0.02)	0.24 (0.01)	0.19 (0.01)
	> 90 %	1,299	0.29 (0.32)	0.07 (0.28)	0.47 (0.03)	0.04 (0.05)	0.23 (0.01)	0.18 (0.01)

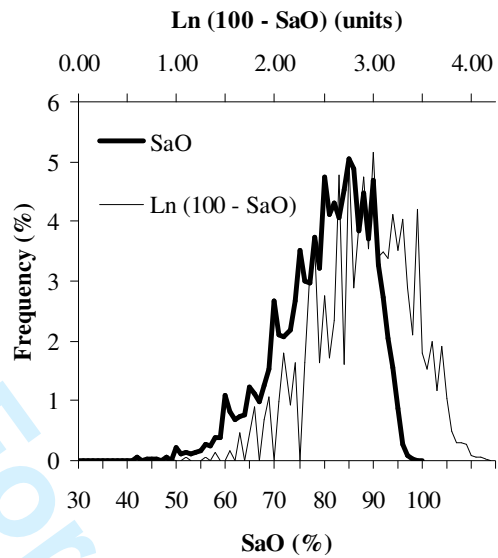
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1 **Figure 1. Distribution of blood oxygen saturation (SaO) and Ln (100-SaO) for line 3.**  
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3 **Phenotypes of all individuals in the pedigree with SaO record were used to obtain the**  
4 **distribution**  
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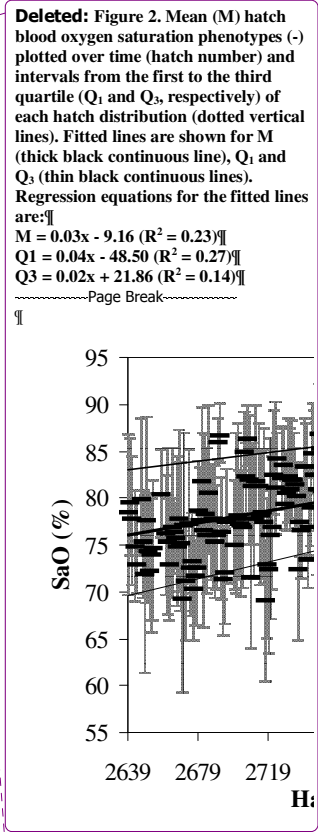
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GENETIC PARAMETERS BLOOD OXYGEN SATURATION, FIGURE 1

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Figure 2. Mean additive value for each hatch (shown in additive standard deviation (SD) units) for blood oxygen saturation, weight and fleshing score, obtained for line 3.

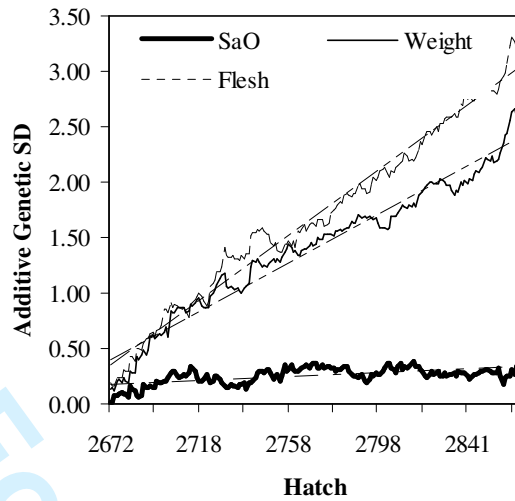
Regression equations of fitted lines (— · — · —) are:

$$\text{SaO} = 0.001x + 0.174 \quad (R^2 = 0.357)$$

$$\text{Weight} = 0.011x + 0.385 \quad (R^2 = 0.961)$$

$$\text{Flesh} = 0.015x + 0.335 \quad (R^2 = 0.982)$$





GENETIC PARAMETERS BLOOD OXYGEN SATURATION, FIGURE 2

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 Non-base generation birds 178,339 319,129 133,968 205,374

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Table S1. Minus two times the logarithm of the likelihood ratio (- 2LnLR) and corresponding P-values (P) obtained for comparison of models used in univariate analyses for blood oxygen saturation (SaO), body weight (Weight) and fleshing score (Flesh, a measure of breast conformation) for all lines

Line	Trait	Model 1 vs. Model 2		Model 1 vs. Model 3		Model 2 vs. Model 4		Model 3 vs. Model 4	
		- 2LnLR	P	- 2LnLR	P	- 2LnLR	P	- 2LnLR	P
1	SaO	0.4	0.26	0.8	0.19	NA	NA	NA	NA
1	Weight	188.0	*** <sup>2</sup>	142.0	***	16.0	***	62.0	***
1	Flesh	89.8	***	56.4	***	8.8	**	42.2	***
2	SaO	7.8	** <sup>3</sup>	0.2	0.33	NA	NA	NA	NA
2	Weight	500.0	***	520.0	***	120.0	***	100.0	***
2	Flesh	222.0	***	160.0	***	30.0	***	92.0	***
3	SaO	2.1	0.07	0.8	0.19	NA	NA	NA	NA
3	Weight	162.0	***	132.0	***	18.0	***	48.0	***
3	Flesh	82.2	***	49.0	***	5.2	**	38.4	***
4	SaO	0.0	0.50	NA <sup>4</sup>	NA	NA	NA	NA	NA
4	Weight	1,428.0	***	1,694.0	***	302.0	***	36.0	***
4	Flesh	131.2	***	154.0	***	47.4	***	24.6	***

<sup>1</sup> Model 1 includes a random animal effect and a residual term; Model 2 includes a maternal environmental component, modelled as a common dam effect for all maternal half-sibs, in addition to the terms in Model 1; Model 3 includes a maternal genetic component, in addition to the terms in Model 1; Model 4 includes, in addition to the terms in Model 1, both a maternal environmental component and a maternal genetic component.

<sup>2</sup> \*\*\* P ≤ 0.001.

<sup>3</sup> \*\* P ≤ 0.01.

<sup>4</sup> NA: not available.

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Table S2. Heritabilities (on diagonals and bold at “Direct Genetic and Phenotypic” column), genetic and phenotypic correlations (below and above diagonal respectively) and their standard errors (in brackets), obtained for blood oxygen saturation (SaO), weight (Weight)

and fleshing score (Flesh, a measure of breast conformation). Variance ratios and correlations are presented for the environmental and genetic maternal components when relevant

Line	Trait	Direct Phenotypic			Genetic and Maternal Environmental			Maternal Genetic		
		SaO	Weight	Flesh	SaO	Weight	Flesh	SaO	Weight	Flesh
1	SaO	<b>0.17</b> (0.01)	-0.00 (0.01)	-0.04 (0.01)						
1	Weight	-0.17 (0.04)	<b>0.34</b> (0.01)	0.57 (0.00)		0.02 (0.00)			0.01 (0.00)	
1	Flesh	-0.21 (0.04)	0.59 (0.02)	<b>0.22</b> (0.01)		0.76 (0.05)	0.01 (0.00)		0.87 (0.12)	0.00 (0.00)
2	SaO	<b>0.12</b> (0.02)	-0.02 (0.01)	-0.07 (0.01)	0.02 (0.00)					
2	Weight	-0.13 (0.06)	<b>0.35</b> (0.01)	0.58 (0.00)	0.00 (0.12)	0.03 (0.00)				
2	Flesh	-0.11 (0.06)	0.64 (0.01)	<b>0.24</b> (0.01)	-0.07 (0.13)	0.90 (0.02)	0.02 (0.00)			
3	SaO	<b>0.21</b> (0.02)	0.01 (0.01)	-0.04 (0.01)						
3	Weight	-0.02 (0.06)	<b>0.26</b> (0.01)	0.57 (0.00)		0.02 (0.00)			0.01 (0.00)	
3	Flesh	-0.10 (0.06)	0.53 (0.03)	<b>0.22</b> (0.01)		0.68 (0.00)	0.01 (0.00)		0.77 (0.00)	0.00 (0.00)
4	SaO	<b>0.12</b> (0.02)	-0.02 (0.01)	-0.05 (0.01)						
4	Weight	-0.17 (0.07)	<b>0.41</b> (0.01)	0.38 (0.00)		0.06 (0.00)				
4	Flesh	-0.15 (0.06)	0.31 (0.02)	<b>0.41</b> (0.01)		0.62 (0.00)	0.02 (0.00)			

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