BREEDING AND GENETICS

Research Notes

The Effect of Strain of Broiler on Physiological Parameters Associated with the Ascites Syndrome

F. G. SILVERSIDES,* M. R. LEFRANÇOIS,* and P. VILLENEUVE†

*Département des Sciences Animales, Université Laval, Ste-Foy, Québec, Canada, G1K 7P4 and
†Ministère de l'Agriculture, des Pêcheries et de l'Alimentation du Québec, Service de la zootechnie, 120-A Chemin du Roy, Deschambault, Québec, Canada, G0A 1S0

ABSTRACT Differences among eight commercial broiler strains in factors indicating susceptibility to ascites were investigated. Chicks from eight crosses were obtained from two hatcheries (one was obtained from both hatcheries to give a total of nine) and raised to 6 wk of age on commercially prepared rations containing 3,300 kcal/kg energy with 24% protein to 3 wk of age and 20% protein from 3 to 6 wk. Each cross was represented by four pens of 90 broilers each. At 21 and 42 d, groups of 20 birds per pen were weighed and feed consumption was measured in order to calculate the feed conversion ratio. At 21 and 42 d, a blood sample was taken from two birds per pen to determine the hematocrit value. These birds were killed and dissected to determine heart, liver, lung, and spleen weights. Lung volume was determined by water displacement. Significant differences were observed between crosses for all of the measures with the exception of feed conversion ratio and mortality. These differences were most prominent for hematocrit value, which is an initial adaptation to anoxia, suggesting that broiler lines could be chosen that resist anoxic environmental conditions.

(*Key words*: broiler lines, ascites, hematocrit, lung volume)

1997 Poultry Science 76:663–667

INTRODUCTION

Ascites in broilers is an accumulation of liquid in the abdominal cavity resulting in mortality and condemnations. Originally detected several decades ago at high altitudes (Lamas da Silva *et al.*, 1988), this condition is now also found at low altitudes, where broilers are hypoxic during periods of rapid growth (Peacock *et al.*, 1988). Post-mortem examination of ascetic birds reveals straw-colored fluid in the abdominal cavities, enlarged hearts, pulmonary congestion, and abnormal livers and spleens (Maxwell *et al.*, 1986; Orr *et al.*, 1986; Wilson *et al.*, 1988).

The processes most commonly leading to ascites in broiler chickens are described by Julian (1987) and Hoerr (1988). With an oxygen insufficiency, the heart rate increases as the heart tries to supply more oxygen to the fast-growing tissues, causing pulmonary hypertension. With prolonged hypoxia, the body increases the oxygen carrying capacity of its blood by increasing the number of erythrocytes (Sturkie, 1986), which increases the blood viscosity and aggravates the pulmonary hypertension. Chronic hypertension results in right ventricular hypertrophy and causes malfunction of the right atrioventricular valve, allowing blood to flow backwards into the *vena cava.* This leads to liver congestion and seepage of liquid from the liver surface. When the rate of seepage is greater than the capacity of the abdominal membranes to absorb the liquid, ascites develops. This eventually leads to death by respiratory failure caused by the pressure of the liquid on the air sacs (Julian *et al.*, 1989). The ratio of right ventricle to total ventricle weight (Julian, 1987) and the packed cell volume (Maxwell *et al.*, 1990) are often used to indicate susceptibility to ascites.

The environment is very important in the occurrence of ascites, but it is clear that ascites and measures associated with the condition have a genetic basis. Vidyadaran *et al.* (1990) found that compared to jungle fowl, domestic fowl have low lung capacity relative to body weight. Timmwood *et al.* (1987) found similar differences between a line of turkeys selected for increased muscle mass and an unselected line. Both Smith and Abbott (1961) and Sillau and Montalvo (1982) found that several generations of natural selection at high altitudes reduced either mortality or factors associated with ascites. Lubritz *et al.* (1995) found moderate to high heritabilities for ascites, and moderate heritabilities for the ratio of right ventricle to total heart

Received for publication June 16, 1996.
Accepted for publication December 30, 1996.

weight in three lines of broiler breeders. Selection on the basis of ascites or heart weight requires clinical signs of ascites or slaughter. Lubritz and McPherson (1994) suggested selecting on the basis of packed cell volume, a procedure used by Shlosberg *et al.* (1996), who found that the progeny of selected birds had a much reduced incidence of ascites.

Ascites and rapid growth are not inherently linked (Dale and Villacres, 1988; Julian *et al.*, 1989). Since the 1940s, broilers have been selected primarily for growth rate (Hunton, 1990). In the absence of a specific need, it is unlikely that organ capacity has been selected for directly. We might expect differences in susceptibility to ascites among commercial lines because each breeding company follows a slightly different selection program. Madrigal *et al.* (1993) and Buys *et al.* (1993) found no differences between commercial strains in susceptibility to ascites, but Huchzermeyer *et al.* (1988) found differences in both the incidence of ascites and in the ratio of right ventricle to heart weight.

We tested eight commercial broiler crosses for measures related to genetic predisposition to ascites (lung weight and volume), to the normal physiological response to hypoxia (hematocrit value), and to the pathological response to hypoxia (heart and liver weights, and lung density).

MATERIALS AND METHODS

Males from eight commercial broiler crosses were obtained from two commercial hatcheries. One cross was obtained from both hatcheries to give a total of nine groups, arbitrarily assigned numbers from 1 to 9. The primary breeding companies represented by these crosses were, in alphabetical order, Arbor Acres, Avian Farms, Hubbard, Peterson, Ross, and Shaver. Crosses 5 and 8 were high-yield crosses. An attempt was made to obtain chicks from older flocks because the egg weight change with increasing age is less important for old hens. Eight of the nine crosses came from hens between 42 and 63 wk of age. Cross 9 (from one hatchery) was from a 33-wk-old flock and Cross 4 (the same cross from the other hatchery) was from a 44-wk-old flock.

The birds were housed at a density of 0.075 m^2 (0.83 ft^2) per broiler (90 birds per pen) in four complete blocks (four repetitions). Feed was commercially prepared and pelleted.[1] Diets contained 3,300 kcal/kg energy and 24% protein to 3 wk of age, and 20% protein from 3 to 6 wk. Mortality was recorded and dead birds were weighed. Because of a problem in the final few days of the experiment, only 63.9% of the mortality was necropsied. Ascites was diagnosed if the ratio of right ventricle to total heart weight was greater than 0.25, or if liquid was visible in the abdomen. Body weights were measured on 40 birds per cross before housing and on 20 birds per pen at 21 and 42 d of age. Feed consumption was recorded at 21 and 42 d of age. Feed conversion was calculated from BW, mortality, and feed consumption data.

[1]Nutribec Ltée Québec, QC, Canada, G1K 6W3.

At 21 and 42 d of age, two birds per pen were randomly chosen and blood samples were taken into heparinized tubes by brachial venipuncture for hematocrit (packed cell volume) measurement. These birds were killed by cervical dislocation and weighed, and the heart, liver, spleen, and lungs were removed and weighed. The volume of the lungs was measured immediately by water displacement in a graduated cylinder. Heart, liver, spleen, and lung weights were calculated as percentages of BW, lung volume was expressed as milliliters per gram BW, and apparent lung density was calculated as the weight in grams divided by the volume in milliliters.

The values for the pen or the means of individuals within a pen were used in the statistical analyses. Differences between lines were tested using the general linear models procedure of SAS® (Littell *et al.*, 1991). Least squares means were calculated by the SAS® program. The effect of the hatchery was not significant for any variable when tested using the cross nested within the hatchery as an error term. Hatchery was therefore not included in the final model and the effect of the cross was tested in a model including the cross and the block as fixed effects. The incidence of ascites among necropsied mortality was compared between the lines using a contingency chi-square test (Steel and Torrie, 1980). Regression analyses were used to test the effect of the hens' age on the means of the crosses and the effect of the 42-d weight of the crosses on feed conversion, mortality, organ weights, lung density, and hematocrit value.

RESULTS

Chick weight was significantly affected by hen age ($P < 0.05$, $R^2 = 0.48$). Body weights at 21 and 42 d, feed conversion, mortality, and variables obtained by dissection were statistically independent of hen age (R^2 values between zero and 0.22). The only measure statistically associated with 42-d BW was lung volume at 21 d ($b = -0.25\%$/kg BW, $P = 0.046$, $R^2 = 0.46$).

Body weights, feed conversion ratios, and mortality for the nine crosses are shown in Table 1. In this table and those that follow, the crosses are presented in the order of 6-wk BW. Differences between crosses were significant for BW at hatch and at 21 and 42 d of age. Weights at hatch and at 42 d were each separated into six statistically different groups. The two similar crosses (4 and 9) were statistically different at hatch and at 42 d. At 42 d of age, the lightest cross was 5% lighter than the overall average and the heaviest cross was 3% heavier. The feed conversion ratio did not differ significantly between the crosses. The difference between the lowest and the highest values was only 3% of the overall average. Mortality was between 11.6 and 20.8% over the 42 d. This large apparent difference between crosses was not significant. Ascites was diagnosed in between 34 and 53% of the birds that were necropsied in the nine lines; these differences were not significant.

TABLE 1. Body weights, feed efficiency, and mortality of nine broiler crosses at 21 and 42 d of age. The order is given in increasing BW at 42 d of age[1]

Cross	Hatch weight	21-d BW	42-d BW	Feed conversion	Mortality
	(g)			(g:g)	(%)
7	43.3^{bc}	701.9^{ab}	2,108^{f}	2.06	17.9
9	38.7^{f}	688.1^{b}	2,143^{ef}	2.04	11.6
2	42.3^{cd}	670.6^{b}	2,174^{de}	2.02	11.9
3	40.8^{e}	707.5^{ab}	2,216^{cd}	2.04	14.4
4	43.3^{bc}	675.6^{b}	2,237^{bc}	1.98	12.5
8	43.0^{bc}	695.6^{ab}	2,287^{ab}	2.03	20.8
1	44.3^{b}	746.3^{a}	2,292^{ab}	2.01	19.4
6	41.3^{de}	705.6^{ab}	2,294^{ab}	2.01	12.5
5	46.7^{a}	711.3^{ab}	2,305^{a}	2.03	13.6
SE	0.5	18.0	21	0.04	4.0

$^{a–f}$Means in the same column with no common superscript differ significantly ($P < 0.05$).

[1]Each number represents the mean of four pens.

At 21 d, the highest and lowest values for heart weight differed by 11.6% (Table 2); other differences were not significant. Heart weights were divided into three statistically different groups at 42 d, when the highest and lowest crosses differed by 15%. At 21 d, only the lowest and the highest liver weights were different, and at 42 d there were no significant differences. Spleen weights were not different at 21 d, but were divided into three statistically different groups at 42 d.

Lung weights, volumes, and densities are shown in Table 3. At 21 d, only the highest and lowest values for lung weight and volume were significantly different, but lung density measurements divided the crosses into three groups. At 42 d, lung densities separated the crosses into two groups but weights and volumes each divided the crosses into three groups. Hematocrit values for the nine crosses were divided statistically into four groups at 21 d and into three groups at 42 d. At 21 d, the lowest value was 7% lower than the overall average (30.9%) and the highest was 6% higher. At 42 d, the lowest value was 9% lower than the average (29.9%) and the highest was 13% higher.

DISCUSSION

The significant effect of the age of the breeder flock on hatch weight was expected because egg weight increases with age of the hen, and egg and chick weights are highly correlated (North and Bell, 1990). Hatch weights for the two similar lines from different hatcheries were likely different because of the 11-wk difference in hen age. Hen age was of minimal importance on other variables measured.

These data do not provide information on other characters of economic value, but do indicate that considerable variation exists in the growth rate of commercial broilers. The difference found between the same cross obtained from two hatcheries in 42-d weight could suggest that the primary breeders had overlapping generations of breeders in Quebec. Data on feed conversion and mortality are often variable and significant differences are difficult to find with only four repetitions. The mortality rate appears to be high, but comparable rates of mortality have been obtained in other research situations (Owen *et al.*, 1994; Shlosberg *et al.*, 1996), probably due to periodic handling and the fragility of modern broilers.

TABLE 2. Heart, liver, and spleen weights of nine broiler crosses at 21 and 42 d of age. The order is given in increasing BW at 42 d of age[1]

	21 d			42 d		
Cross	Heart	Liver	Spleen	Heart	Liver	Spleen
	(% of BW)					
7	0.632^{ab}	4.07^{ab}	0.102	0.487^{abc}	2.53	0.110^{c}
9	0.586^{ab}	3.91^{ab}	0.120	0.520^{ab}	2.38	0.168^{a}
2	0.576^{b}	4.64^{a}	0.115	0.457^{c}	2.51	0.118^{bc}
3	0.646^{a}	3.56^{b}	0.121	0.481^{abc}	2.56	0.120^{abc}
4	0.614^{ab}	4.45^{a}	0.134	0.532^{a}	2.39	0.139^{abc}
8	0.575^{b}	4.56^{a}	0.126	0.520^{ab}	2.54	0.114^{bc}
1	0.615^{ab}	4.39^{a}	0.136	0.489^{abc}	2.68	0.113^{bc}
6	0.635^{ab}	3.96^{ab}	0.121	0.471^{bc}	2.49	0.160^{ab}
5	0.613^{ab}	4.45^{a}	0.111	0.526^{ab}	2.37	0.127^{abc}
SE	0.023	0.27	0.014	0.020	0.13	0.016

$^{a–c}$Means in the same column with no common superscript differ significantly ($P < 0.05$).

[1]Each number represents the mean of four pens.

TABLE 3. Lung weights, volumes, and densities and hematocrit values of nine broiler crosses at 21 and 42 d of age. The order is given in increasing BW at 42 d of age[1]

	21 d				42 d			
Cross	Weight	Volume	Density	Hematocrit	Weight	Volume	Density	Hematocrit
	(%)	(mL / g)	(g / mL)	(%)	(%)	(mL / g)	(g / mL)	(%)
7	0.499^{a}	0.584^{a}	0.853^{ab}	31.7^{abc}	0.446^{abc}	0.528^{abc}	0.847^{b}	29.8^{bc}
9	0.456^{ab}	0.588^{a}	0.779^{c}	32.4^{ab}	0.446^{abc}	0.536^{ab}	0.834^{b}	30.4^{abc}
2	0.440^{ab}	0.559^{ab}	0.798^{bc}	28.8^{d}	0.427^{bc}	0.513^{abc}	0.834^{b}	28.0^{bc}
3	0.452^{ab}	0.530^{ab}	0.853^{ab}	30.1^{bcd}	0.471^{ab}	0.507^{abc}	0.930^{a}	29.4^{bc}
4	0.448^{ab}	0.542^{ab}	0.827^{abc}	31.9^{abc}	0.459^{abc}	0.531^{abc}	0.868^{ab}	30.9^{ab}
8	0.428^{b}	0.500^{b}	0.857^{a}	30.0^{bcd}	0.397^{c}	0.472^{c}	0.842^{b}	29.8^{bc}
1	0.465^{ab}	0.557^{ab}	0.834^{abc}	30.4^{bcd}	0.502^{a}	0.566^{a}	0.887^{ab}	27.1^{c}
6	0.469^{ab}	0.543^{ab}	0.864^{a}	29.9^{cd}	0.427^{bc}	0.515^{abc}	0.832^{b}	29.8^{bc}
5	0.462^{ab}	0.551^{ab}	0.840^{ab}	32.9^{a}	0.411^{bc}	0.475^{bc}	0.868^{ab}	33.8^{a}
SE	0.020	0.022	0.019	0.8	0.022	0.022	0.022	1.2

$^{a–d}$Means in the same column with no common superscript differ significantly ($P < 0.05$).

[1]Each number represents the mean of four pens.

Genetic variation in lung capacity of chickens (Vidyadaren *et al.*, 1990) and turkeys (Timmwood *et al.*, 1987) has been demonstrated, and lung weight or volume may indicate a genetic susceptibility to ascites. Differences between the broiler lines included in this experiment in lung weight and volume were more pronounced at 42 than at 21 d.

The first reaction of the body to oxygen deficiency is an increase in the heart rate. With prolonged exposure to anoxia, the number of erythrocytes increases, increasing the hematocrit value. These are normal physiological responses of the bird. Lubritz and McPherson (1994) and Sholsberg *et al.* (1996) suggested using hematocrit values after exposure to cold to measure susceptibility of broilers to ascites. Hematocrit values were found to differ between broiler lines in this trial.

With continued exposure to anoxic conditions, a series of events eventually leads to the clinical symptoms of ascites. An increased heart rate and more viscous blood lead to increased pressure in the lungs and lung congestion. The right ventricle of the heart undergoes hypertrophy and the liver becomes congested. Spleen weight is a general indicator of stress. Lung density and heart, liver, and spleen weights can all be used to measure this pathological response to oxygen deficiency. Differences were observed among the nine commercial crosses for these measures, but they were smaller than the differences observed in hematocrit value.

These data suggest that there are differences among commercial broiler lines in susceptibility to ascites. Hematocrit values differed between the commercial broiler lines tested. This character is associated with ascites and can be measured in live birds without imposing conditions that produce ascites. It could therefore be useful in choosing commercial lines to minimize the incidence of ascites.

REFERENCES

Buys, N., J. Buyse, and E. Decuypere, 1993. Ascites syndrome mortality and growth in two commercial broiler strains subjected to different early temperature programs and T_3-diet. Poultry Sci. 72 (Suppl. 1):135. (Abstr.)

Dale, N., and A. Villacres, 1988. Relationship of two-week body weight to the incidence of ascites in broilers. Avian Dis. 32:556–560.

Hoerr, F. J., 1988. Pathogenesis of ascites. Zootec. Int. (Sept., 1988):60–61.

Huchzermeyer, F. W., A.M.C. De Ruijck, and H. Van Ark, 1988. Broiler pulmonary hypertension syndrome III. Commercial broiler strains differ in their susceptibility. Onderstepoort J. Vet. Res. 55:5–9.

Hunton, P., 1990. Industrial breeding and selection. Pages 985–1028 *in*: Poultry Breeding and Genetics. R. D. Crawford, ed. Elsevier, New York, NY.

Julian, R. J., 1987. The effect of increased sodium in the drinking water on right ventricular hypertrophy, right ventricular failure and ascites in broiler chickens. Avian Pathol. 16:61–71.

Julian, R. J., I. McMillan, and M. Quinton, 1989. The effect of cold and dietary energy on right ventricular hypertrophy, right ventricular failure and ascites in meat-type chickens. Avian Pathol. 18:675–684.

Lamas da Silva, J. M., N. Dale, and J. B. Luchesi, 1988. Effect of pelleted feed on the incidence of ascites in broilers reared at low altitudes. Avian Dis. 32:376–378.

Littell, R. C., R. J. Freund, and P. C. Spector, 1991. SAS® System for Linear Models. 3rd ed. SAS® Series in Statistical Applications. SAS Institute Inc., Cary, NC.

Lubritz, D. L., and B. N. McPherson, 1994. Effect of genotype and cold stress on incidence of ascites in cockerels. J. Appl. Poult. Res. 3:171–178.

Lubritz, D. L., J. L. Smith, and B. N. McPherson, 1995. Heritability of ascites and the ratio of right to total ventricle weight in broiler breeder male lines. Poultry Sci. 74:1237–1241.

Madrigal, S. A., S. E. Watkins, A. L. Waldroup, and P. W. Waldroup, 1993. Comparison of feeding programs designed to reduce incidence of ascites, sudden death syndrome, and leg disorders in male broilers grown for further processing. Poultry Sci. 72 (Suppl. 1):179. (Abstr.)

Maxwell, M. H., G. W. Robertson, and S. Spence, 1986. Studies on an ascetic syndrome in young broilers. 1. Haematology and pathology. Avian Pathol. 15:511–524.

Maxwell, M. H., S. Spence, G. W. Robertson, and M. A. Mitchell, 1990. Haematological and morphological responses of broiler chicks to hypoxia. Avian Pathol. 19: 23–40.

North, M. O., and D. D. Bell, 1990. Commercial Chicken Production Manual. 4th ed. Van Nostrand Reinhold, New York, NY.

Orr, J. P., K. S. Little, M. Schoonderwoerd, and A. J. Rehmtulla, 1986. Ascites in broiler chickens. Can. Vet. J. 27:99–100.

Owen, R. L., R. F. Wideman, Jr., R. M. Leach, B. S. Cowen, P. A. Dunn, and B. C. Ford, 1994. Effect of age of exposure and dietary acidification or alkalization on broiler pulmonary hypertension syndrome. J. Appl. Poult. Res. 3: 244–252.

Peacock, A. J., C. K. Pickett, K. M. Morris, and J. T. Reeves, 1988. Spontaneous pulmonary hypertension in rapidly growing broiler chickens reared at sea level. Am. Rev. Resp. Dis. 137:106. (Abstr.)

Shlosberg, A., M. Bellaiche, G. Zeitlin, M. Ya'acobi, and A. Cahaner, 1996. Hematocrit values and mortality from ascites in cold-stressed broilers from parents selected by hematocrit. Poultry Sci. 75:1–5.

Sillau, A. H., and C. Montalvo, 1982. Pulmonary hypertension and the smooth muscle of the pulmonary arterioles in chickens at high altitude. Comp. Biochem. Physiol. 71A: 125–130.

Smith, A. H., and U. K. Abbott, 1961. Adaptation of the domestic fowl to high altitude. Poultry Sci. 40:1459. (Abstr.)

Steel, R.G.D., and J. H. Torrie, 1980. Principles and Procedures of Statistics. A Biometrical Approach. 2nd ed. McGraw Hill Book Co., Inc., New York, NY.

Sturkie, P., 1986. Body fluids: Blood. Pages 103–121 *in*: Avian Physiology. P. D. Sturkie, ed. Springer-Verlag, New York, NY.

Timmwood, K. I., D. M. Hyde, and C. G. Plopper, 1987. Lung growth in the turkey, *Meleagris gallopavo*: II. Comparison of two genetic lines. Am. J. Anat. 178:158–169.

Vidyadaran, M. K., A. S. King, and H. Kassim, 1990. Quantitative comparisons of lung structure of adult domestic fowl and red jungle fowl, with reference to broiler ascites. Avian Pathol. 19:51–58.

Wilson, J. B., R. J. Julian, and I. K. Barker, 1988. Lesions of right heart failure and ascites in broiler chickens. Avian Dis. 32: 246–261.