Strain and sex effects on ascites development in commercial broiler chickens

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Summary

The objective of this study was to assess the importance of strain and sex and their interaction effect on broiler chickens' performance and susceptibility to ascites. Chicks from three strains (Ross 308, Cobb 500 and Arian, labeled as A, B and C respectively) were obtained from the same age breeder and sexed when one-day-old. Each crosses' sex was assigned to four pens of 60 broilers each. Body weight gain (BWG), feed intake, feed conversion ratio (FCR) and ascites mortality were determined. At the age of 28 and 49 d haematocrit values, arterial pressure index (API = right ventricular/total ventricular weight ratio), the values of plasma thyroid hormone concentration (thyroxin, T4 and triiodothyronine, T3) and metabolic lung weight (lung weight/body weight$^{(0.75)}$) were determined. Strain B had the highest BWG but an equal incidence of ascites as strain A, which had low BWG and ascites mortality. Ascites mortality was not correlated with BWG and FCR. A significant strain interaction by sex on feed conversion ($P<0.01$) and final weight ($P<0.05$) was found. The sex effect was significant only in strain C on feed conversion and final weight. Strains B and C had lower thyroid hormones and reduced metabolic lung weights compared with strain A. The sex effect was not significant on the parameters associated with ascites syndrome and its incidence.

Key words: Ascites, Broiler, Performance, Sex, Strain

Introduction

Genetic selection has increased production levels of broiler chickens over the recent decades. Havenstein *et al*. (2003) reported that the genetic selection applied by commercial breeding companies has contributed to about 85 to 90% of the progressive increase in broilers' growth rates over the past 45 years. On the other hand, the mortality rate of modern broilers has also increased considerably (Havenstein *et al*., 2003).

Ascites syndrome (accumulation of non infectious fluid in the peritoneal cavity) is classified as a metabolic disorder resulting in considerable mortality (about 5%) and occurs usually in fast growing chickens (Swire, 1980; Maxwell and Robertson, 1998). However, a high rate of weight gain is not always a good predictor of ascites susceptibility. In addition, this syndrome does not only occur in chickens that show high growth rates in flocks (Wideman and Kirby, 1996; Luger *et al*., 2001).

Ascites is mainly linked with an insufficient supply and high requirement of oxygen in fast-growing broiler chickens (Malan *et al*., 2003). When sufficient oxygen is not supplied, the heart rate increases to supply more oxygen to the tissues, increasing blood flow and pulmonary hypertension (Julian, 1998).

Ascites syndrome has a genetic basis. Heritability for this syndrome has been estimated moderate to high, ranging from 0.11 to 0.57 (Lubritz *et al*., 1995; Moghadam *et al*., 2001; Druyan *et al*., 2007). Variation in the susceptibility of different commercial broiler strains to ascites is related to metabolic rate (Konarzewski *et al*., 2000; Malan *et al*., 2003), lung capacity (Silversides *et al*., 1997; Wideman, 2001; Malan *et al*., 2003), decreased heart weight (Gaya *et al*., 2007), and thyroid activity (Gonzales *et al*., 1999; Luger *et al*., 2001; Luger *et al*., 2002; Guo *et al*., 2007). Malan *et al*. (2003) studied ascites-sensitivity in chicken lines with different growth rate backgrounds. They confirmed that the low relative lung weight of fast-growing lines indicates less lung tissue, and that increased haematocrit value is possibly associated with insufficient lung development. Elevation in haematocrit value is caused by an increased erythropoiesis process rather than diminished plasma volume in ascitic broilers (Luger *et al*., 2003). Triiodothyronine (T3) also plays the main role in red blood cells' differentiation by hemoglobin accumulation (Bauer *et al*., 1998). In ascites syndrome, the activity of the thyroid gland decreases, causing hypothyroidism (Scheele *et al*., 1992; Gonzales *et al*., 1999; Luger *et al*., 2002). Furthermore, in other literature Sceheele *et al*. (2003) it is reported that the tendency for high haematocrit value is linked with low proportional lung weight and low concentrations of T3. This disorder has become an increasingly important cause of mortality and financial loss in commercial broiler units (Maxwall and Robertson, 1998). For this reason, commercial broiler companies have attempted to reduce ascites susceptibility in their chicks.

The objective of this study was to evaluate the importance of strain and sex effects on the performance of broilers and their susceptibility to ascites syndrome.

Materials and Methods

Fourteen hundred and forty broiler chicks (720 of each sex) of three strains (480 of each strain; Ross 308, Cobb 500 and Arian, represented here as A, B and C, respectively) were obtained from different commercial hatcheries. The Arian and Cobb 500 were vent sexed and Ross 308 was feather sexed on the day of hatch. Body weight for all chicks was 40 ± 2 g at one day age. Chicks were randomly assigned to pens by strain cross and sex. Each sex of the crosses was assigned to four pens of 60 broilers each. All birds were raised in floor pens (160 × 300 cm) in a controlled house with a temperature of 35°C for the first three days, decreasing daily by 1°C to 20°C on day 15. Continuous light was applied during the whole experiment. The chickens were fed with starter diets from 1 to 14 d (3010 Kcal ME/kg, 22% crude protein), grower diets from 15 to 28 d (3175 Kcal ME/kg, 20% crude protein) and finisher diets from 29 to 49 d of age (3227 Kcal ME/kg, 18% crude protein). Water and feed were supplied *ad libitum*. The diet was formulated according to the recommendations of the NRC (1994) using WUFFDA formulation software program.

Measurements

Performance data including body weight gain and feed intake was collected weekly. Average feed intake was calculated and corrected for mortality per pen. Feed conversion rate was calculated on a pen basis. Dead birds were weighed, necropsied and recorded daily throughout the experiment. Special attention was given to the identification of ascetic mortality according to abdominal fluid accumulation and the ratio of the right ventricle to total ventricle weight (Gonzales *et al.*, 1999).

Blood samples were collected from wing veins at 28 and 49 days of age from 3 birds per pen. Plasma was stored at -20°C until assayed. At the age of 28 and 49 days, two birds of each pen were weighed and exterminated to calculate their metabolic lung weight (lung weight/body weight$^{(0.75)}$). Arterial pressure index (API= RV/TV, right ventricle to total ventricle weight ratio) was determined for the removed hearts of the birds (Julian, 2005).

Blood for haematocrit measurement was collected in heparinized microcapillary tubes and recorded as volume percentage in all samples after the centrifugation of blood at 15000 rpm for 10 min. Radioimmunoassay was performed on plasma samples to measure thyroxin (T4) and T3, using available kits (Izotop Ltd., Budapest, Hungary). T4 and T3 hormones were characterized by intrassay variation (CV) of 4.3 and 5.1, respectively.

Statistical analysis

The study was conducted with a completely randomized design with factorial arrangement using strain and sex classification as fixed treatment effects. Results were analyzed by running an ANOVA of SAS. Mean separation was carried out using Duncan's multiple range test. Within treatment correlations between group records (such as feed intake and FCR) and individual records (such as blood parameter and API) were calculated using the average of individual records per pen.

Results

Production performance, incidence and total mortality

Production performances including body weight gain (BWG), feed intake and feed conversion ratio (FCR) for the entire period (from 1 to 49 d of age) are shown in Table 1. Body weight gain was significantly different among strains (P<0.01). There was no difference in the feed intake of different strains, but strain effect was evident with regard to FCR (P<0.01).

The strain interaction by sex on feed conversion was found to be significant (P<0.01). Only in strain C females had higher feed conversion than males (Table 2). In general, strain B had the highest daily body weight gain. Also, males had higher daily BWG than females (P<0.01), whereas for the mean daily feed intake, strain, sex and their interaction effects were not significant.

Table 1: Average daily body weight gain (g/day), final body weight (g), average daily feed intake (g/day) and feed conversion ratio (g/g) for the three commercial broilers and both sexes, from 1 to 49 d of age (mean±SE)

		[1]BWG (g/day)	Final BW (g)	Daily feed intake (g/day)	[2]FCR (g/g)
Strain	A	46.1 ± 0.6^{b}	2336 ± 26^{b}	84.9 ± 1.3	1.82 ± 0.01^{b}
	B	50.7 ± 1.0^{a}	2641 ± 50^{a}	87.0 ± 1.4	1.71 ± 0.04^{b}
	C	46.5 ± 1.9^{b}	2548 ± 114^{a}	90.4 ± 2.2	1.96 ± 0.10^{a}
Sex	Male	50.2 ± 1.0^{a}	2622 ± 66^{a}	87.5 ± 1.0	1.74 ± 0.03^{b}
	Female	45.8 ± 1.0^{b}	2394 ± 56^{b}	87.6 ± 2.0	1.91 ± 0.06^{a}
[3]SE		0.9	50	1.1	0.04
Source of variation		P-value			
Strain		0.0038	0.0008	0.1045	0.0021
Sex		0.0010	0.0006	0.9261	0.0019
Strain × sex		0.0504	0.0193	0.1896	0.0034

Means in the same column in each comparison group of strains and both sexes with no common superscript differ significantly. [1] Body weight gain, [2] Feed conversion ratio, and [3] Pooled standard error

Table 2: The significant interaction effect of strain with sex on final body weight at 49 d of age (P<0.05) and feed conversion ratio (P<0.01) from 1 to 49 d of age (mean±SE)

	Strain × sex interaction effect						P-value
Strain	A		B		C		
Sex	Male	Female	Male	Female	Male	Female	
Final BW (g)	2372 ± 34^{c}	2299 ± 29^{c}	2719 ± 54^{ab}	2562 ± 30^{b}	2775 ± 30^{a}	2320 ± 111^{c}	0.0003
[1]FCR (g/g)	1.85 ± 0.02^{b}	1.81 ± 0.02^{b}	1.67 ± 0.01^{b}	1.76 ± 0.07^{b}	1.75 ± 0.07^{b}	2.17 ± 0.05^{a}	0.0004

Means with in the same row in each comparison group with no common superscript differ significantly. [1] Feed conversion ratio

Table 3: Percentage of mortality caused by ascites syndrome of the three commercial broilers and both sexes from 1 to 49 d of age (% of bird at the onset of each period)

		Ascites mortality (%)			
		1 to 14 d	15 to 28 d	29 to 49 d	1 to 49 d
Strain	A	0.20	0.20^{b}	1.04^{b}	1.25^{b}
	B	0.41	0.63^{b}	3.85^{b}	4.58^{b}
	C	0.00	1.89^{a}	11.92^{a}	13.33^{a}
Sex	Male	0.13	0.70	6.39	6.94
	Female	0.27	1.12	4.82	5.83
[1]SE		0.11	0.24	1.16	1.22
Source of variation		P-value			
Strain		0.3874	0.0087	0.0001	0.0001
Sex		0.5709	0.3125	0.2605	0.4055
Strain × sex		0.7209	0.2630	0.2771	0.2823

Means in the same column in each comparison group of strains and sex with no common superscript differ significantly. [1] Pooled standard error

Table 4: Metabolic lung weight[1], arterial pressure index ([2]API value = RV/TV) and haematocrit value (PCV) of the three commercial broilers and both sexes at 28 and 49 d of age (mean±SE)

		Lungs (g/kg$^{0.75}$)		API (RV/TV)		PCV (%)	
		28 d	49 d	28 d	49 d	28 d	49 d
Strain	A	6.10 ± 0.34^{a}	6.51 ± 0.55^{a}	0.207 ± 0.017	0.196 ± 0.006^{b}	35.1 ± 0.9^{b}	37.3 ± 0.6^{b}
	B	5.36 ± 0.30^{ab}	4.93 ± 0.33^{b}	0.211 ± 0.015	0.211 ± 0.008^{b}	34.4 ± 1.0^{b}	38.5 ± 0.7^{b}
	C	4.79 ± 0.19^{b}	5.14 ± 0.14^{b}	0.249 ± 0.009	0.256 ± 0.020^{a}	38.5 ± 0.6^{a}	41.6 ± 0.9^{a}
Sex	Male	5.39 ± 0.29	5.44 ± 0.30	0.227 ± 0.012	0.233 ± 0.013	36.4 ± 0.8	39.7 ± 0.9
	Female	5.44 ± 0.26	5.62 ± 0.43	0.217 ± 0.013	0.209 ± 0.011	35.6 ± 0.9	38.6 ± 0.7
[3]SE		0.19	0.26	0.008	0.008	0.6	0.6
Source of variation		P-value					
Strain		0.0250	0.0292	0.1491	0.0058	0.0101	0.0035
Sex		0.8826	0.7216	0.5872	0.0944	0.4348	0.2463
Strain × sex		0.9923	0.9977	0.9565	0.1402	0.9559	0.8683

Means in the same column in each comparison group of strains and sexes with no common superscript differ significantly. [1] Metabolic lung weight (lung weight/body weight$^{(0.75)}$), [2] API: Right ventricle to total ventricular weight ratio, and [3] Pooled standard error

Strain and sex had significant effects on final body weight at 49 d (P<0.01), and their interaction was significant (P<0.05). The effect of sex on final body weight was only significant in strain C (Table 2).

Mortality rates from ascites syndrome for three periods (1-14, 15-28 and 29-49 d) and the entire period (1-49 d) are presented in Table 3. Strain, sex and their interaction did not significantly effect ascites mortality from 1-14 d. Strain C showed the highest incidence of ascites mortality (P<0.01) at 15-28, 28-49 d and the entire experimental period. Sex effect and strain interaction by sex were not significantly related to ascites mortality (P>0.05) at any stage.

Metabolic lung weight, arterial pressure index, haematocrit values and plasma thyroid hormones

Strain was found to have an effect (P<0.05) on the metabolic weight of the lungs at ages 28 and 49 days (Table 4), whereas sex did not significantly affect the metabolic weight of the lungs at any age. Strain A was characterized by the highest metabolic lung weight (P<0.05), while strain C had low metabolic lung weights. Strain C manifested the highest API (P<0.01) at the age of 49 d and the highest haematocrit values at 28 d (P<0.05) and 49 d (P<0.01) compared with strains A and

B. There was no significant difference among the strains on API at 28 d of age. Also, no significant difference was observed between the sexes on either API or haematocrit values at 28 and 49 d of age (P>0.05).

Data on plasma thyroid hormones at ages 28 d and 49 d are shown in Table 5. At the age of 28 and 49 d, strain A had the highest T3 and T4 concentration (P<0.01 and P<0.05, respectively). Strains B and C were characterized by the lowest T3 plasma concentrations at both ages (P<0.01).

The partial correlation coefficients for performance and physiological parameters (at ages 28 and 49 d) associated with the ascites are given in Table 6. Total ascites mortality was positively correlated with haematocrit at the age of 28 and 49 d (P<0.05 and P<0.001, respectively), arterial pressure index at 49 d (P<0.01) and feed intake (P<0.05). Ascites mortality was also negatively correlated with metabolic lung weight, T3 (P<0.01) at 28 d, T4 at 28 d (P<0.01) and 49 d (P<0.05), but not with DWG and FCR. Only DWG was negatively correlated with T3 as a physiological parameter associated with the ascites condition. FCR showed no correlation with any of the ascites parameters measured at 28 and 49 d in this experiment.

Discussion

According to previous studies, the susceptibility of broiler chickens to ascites syndrome is not associated only to individuals which have high growth rates as long as their high rate of BWG pressures their cardiopulmonary system (Luger *et al*., 2001; Malan *et al*., 2003). In this study, strain B had a higher weight

Table 5: Plasma thyroid (T4, ng/ml), triiodothyronine (T3, ng/ml) of three strains (A to C) and both sex at 28 and 49 d of age

		T3 (ng/ml)		T4 (ng/ml)	
		28 d	49 d	28 d	49 d
Strain	A	2.08 ±0.10[a]	1.68 ± 0.08[a]	7.41 ±0.17[a]	7.74 ± 0.17[a]
	B	1.27 ± 0.09[b]	1.27 ± 0.11[b]	6.47 ± 0.18[ab]	6.65 ± 0.32[b]
	C	1.22 ± 0.25[b]	1.27 ± 0.06[b]	6.17 ± 0.47[b]	6.68 ± 0.26[b]
Sex	Male	1.42 ± 0.15	1.35 ± 0.07	6.50 ± 0.25	6.79 ± 0.29
	Female	1.63 ± 0.19	1.47 ± 0.10	6.85 ± 0.31	7.26 ± 0.18
[1]SE		0.12	0.20	0.06	0.18
Source of variation		P-value			
Strain		0.0027	0.0071	0.0355	0.0135
Sex		0.3321	0.2881	0.3450	0.1453
Strain × sex		0.9811	0.9390	0.7552	0.8386

Means in the same column in each comparison group of strains and sex with no common superscript differ significantly. [1] Pooled standard error

Table 6: Partial correlation coefficients among various parameters measured where table/prob<[r]

		Total [1]AS mortality	T4	T3	[2]API	[3]PCV	[4]Lungs	Feed intake (1-28 d)	[5]FCR (1-28 d)
Physiological parameters at age of 28 d	[6]BWG (1-28 d)	NS	NS	-0.517*	NS	NS	NS	NS	-0.590*
	FCR (1-28 d)	NS	NS	NS	NS	NS	NS	0.591*	
	Feed intake (1-28 d)	0.579*	-0.590**	-0.512**	NS	NS	NS		
	Lungs	-0.591**	NS	NS	NS	NS			
	PCV	0.509*	NS	NS	NS				
	API	NS	NS	NS					
	T3	-0.554**	0.850***						
	T4	-0.603**							
Physiological parameters at age of 49 d	BWG (1-49 d)		NS	NS	NS	NS	NS		
	FCR (1-49 d)		NS	NS	NS	NS	NS		
	Feed intake (1-49 d)		NS	NS	NS	0.519*	NS		
	Lungs	NS	0.494*	0.747***	NS	-0.430*			
	PCV	0.714***	-0.601**	-0.458*	0.456*				
	API	0.575**	NS	-0.456*					
	T3	NS	0.477*						
	T4	-0.438*							

NS: Not significant. * P<0.05, **P<0.01, and ***P<0.001. [r] is indicated when it was significant. [1] Ascite syndrome, [2] API: Right ventricle to total ventricular weight ratio, [3] PCV: Haematocrit value, [4] Metabolic lung weight (lung weight/body weight$^{(0.75)}$), [5] FCR: Feed conversion ratio, and [6]BWG: Daily body weight grain

gain rate than the ascites-sensitive strain C. At the same time, males had higher body weight gain than females, but both sexes had similar ascites development. Also, strain C females gained lower final body weights and had higher feed conversion than males of the same strain, but no difference was found in their ascites susceptibility. In general, daily weight gain and FCR were not correlated with ascites mortality. Among the performance data of this study, only feed intake was positively correlated to ascites incidence. Previous findings, such as the reduced surface area of the intestines of ascites-susceptible chickens (Solis de los Santos *et al*., 2005) and high oxygen consumption of the gastrointestinal tract (Yen *et al*., 1989) may provide clues as to why positive correlations were found between feed intake and ascites incidence in the present study.

As Table 6 shows, API at age 49 d was positively correlated with the incidence of ascites ($P<0.01$). Lubritz *et al*. (1995) reported positive genetic correlations between ascites and API. The high API (at 49 d) and haematocrit values (at 28 and 49 d) of strain C compared with strains A and B, were related to the hypertrophy of the right ventricular as a sign of ascites syndrome (Maxwell *et al*., 1986; Mirsalimi and Julian, 1991; McGovern *et al*., 1999).

Metabolic lung weight differed significantly across the strains. Strain A had the highest metabolic lung weight, especially at 49 d of age. The high metabolic lung weight of this strain supports its ability to supply sufficient oxygen for body metabolic requirements. The low lung weights of ascites-sensitive broilers coincided with decreased arterial oxygen tension along with high carbon dioxin pressure (Julian, 1998; Malan *et al*., 2003). Hypoxemia, as a causal factor for increasing haematocrit (Bond *et al*., 1999), may arise from underdeveloped lungs due to their insufficient growth (Scheele *et al*., 2003). On other hand, sex made no difference to API, haematocrit value, or lung metabolic weight. In addition, both sexes had similar, ascites mortality in this study, which could be related to their same API, haematocrit value, or lung metabolic weight.

A slightly hypothyroid state was found to be linked with sensitivity to metabolic disorders such as ascites (Gonzales *et al*., 1999). Thyroid hormones were lower in strains B and C compared with strain A (Table 5). In this study, strain B had the same thyroid hormone concentration as strain C, but a lower incidence of ascites mortality. Luger *et al*. (2001) reported that a decrease in thyroid hormone concentrations can provide a good indication of ascites development only during the last week of life and not in all cases. Males and females were not different in their thyroid hormone concentrations at 28 and 49 d of age (Table 5). In this study, T3 ($P<0.01$) at age 28 d and T4 at age 28 and 49 d ($P<0.01$ and $P<0.05$, respectively) were negatively correlated with total ascites syndrome mortality (Table 6).

It can be concluded that high BWG is not directly related to ascites susceptibility in commercial broiler chickens, and males and females in each strain are equally susceptible to ascites syndrome. Therefore, utilizing strategies to increase ascites syndrome resistance in commercial broiler chickens can affect both sexes.

References

Bauer, A; Mikulits, W; Lagger, G; Stengl, G; Brosch, G and Beug, H (1998). The thyroid hormone receptor functions as a ligand-operated developmental switch between proliferation and differentiation of erythroid progenitors. The EMBO J., 17: 4291-4303.

Bond, JM; Julian, RJ and Squires, EJ (1999). Effect of dietary flax oil and hypobaric hypoxia on right ventricular and ascites in broiler chickens. Bri. Poult. Sci., 37: 731-741.

Druyan, S; Ben-David, A and Cahaner, A (2007). Development of ascites-resistant and ascites-susceptible broiler lines. Poult. Sci., 86: 811-822.

Gaya, LG; Costa, AMMA; Ferraz, JBS; Rezende, FM; Mattos, EC; Eler, JP; Michelan Filho, T; Mourão, GB and Figueiredo, LGG (2007). Genetic trends of absolute and relative heart weight in a male broiler line. Gen. Mol. Res., 6: 1091-1096.

Gonzales, E; Buyse, J; Sartori, JR; Loddi, MM and Decuypere, E (1999). Metabolic disturbances in male broilers of different strains. 2-relationship between the thyroid and somatotropic axes with growth rate and mortality. Poult. Sci., 78: 516-521.

Guo, JL; Zheng, QH; Yin, QQ; Cheng, W and Jiang, YB (2007). Study on mechanism of ascites syndrome of broilers. Am. J. Anim. Vet. Sci., 2: 62-65.

Havenstein, GB; Ferket, PR and Qureshi, MA (2003). Growth, livability and feed conversion of 1957 versus 2001 broilers when fed representative 1957 and 2001 broiler diets. Poult. Sci., 82: 1500-1508.

Julian, RJ (1998). Rapid growth problems: ascites and skeletal deformities in broilers. Poult. Sci., 77: 1773-1780.

Julian, RJ (2005). Production and growth related disorders and other metabolic disease of poultry – a review. Vet. J., 169: 350-369.

Konarzewski, M; Gavin, A; McDevitt, R and Wallis, IR (2000). Metabolic and organ mass responses to selection for high growth rates in the domestic chicken (*Gallus domesticus*). Phys. Bio. Zoo., 73: 237-248.

Lubritz, DL; Smith, JL and McPherson, BN (1995). Heritability of ascites and the ratio of right total ventricle weight in broiler breeder male lines. Poult. Sci., 74: 1237-1241.

Luger, D; Shinder, D; Rzepakovsky, V; Rusal, MN and Yahav, S (2001). Association between weight grain, blood parameters, and thyroid hormones and the development of ascites syndrome in broiler chickens. Poult. Sci., 80: 965-971.

Luger, D; Shinder, D; Wolfenson, D and Yahav, S (2003). Erythropoiesis regulation during the development of ascites syndrome in broiler chickens: a possible role of corticosterone. J. Anim. Sci., 81: 784-790.

Luger, D; Shinder, D and Yahav, S (2002). Hyper- or hypothyroidism: its association with the development of ascites syndrome in fast - growing chickens. Gen. Comp. End., 127: 293-299.

Malan, DD; Scheele, CW; Buyse, J; Kwakernaak, C; Siebrits, FK; Van Der klis, JD and Decuypere, E (2003). Metabolic rate and its relationship with ascites in chicken genotypes. Bri. Poult. Sci., 44: 309-315.

Maxwell, MH and Roberston, GW (1998). UK survey of

broiler ascites and sudden death syndromes in 1993. Bri. Poult. Sci., 39: 203-215.

Maxwell, MH; Roberston, GW and Spence, S (1986). Studies on an ascitic syndrome in young broilers. 2. Ultrastructure. Avi. Path., 15: 525-538.

McGovern, RH; Feddes, JJR; Robinson, FE and Hanson, JA (1999). Analysis of right ventricular areas to the severity of ascites syndrome in broiler chickens. Poult. Sci., 78: 62-65.

Mirsalimi, SM and Julian, RJ (1991). Reduced erythrocyte deformability as a possible contributing factor to pulmonary hypertension and ascites in broiler chickens. Avi. Dis., 35: 374-379.

Moghadam, HK; McMillan, I; Chambers, JR and Julian, RJ (2001). Estimation of genetic parameters for ascites syndrome in broiler chickens. Poult. Sci., 80: 844-848.

NRC (1994). Nutrient Requirements for Poultry. 9th Rev. Edn., NY, NRC Publications.

Scheele, CW; Decuypere, E; Vereijken, PFG and Schreurs, FJG (1992). Ascites in broilers. 2. Disturbance in the hormonal regulation of metabolism rate and fat metabolism. Poult. Sci., 71: 1971-1984.

Scheele, CW; Vanderklis, JD; Kwakernaak, C; Buys, N and Decuypere, E (2003). Haematological characteristics predicting susceptibility for ascites. 2. High haematocrit values in juvenile chickens. Bri. Poult. Sci., 44: 484-489.

Silversides, FG; Lefrançois, MR and Villenevue, P (1997). The effect of strain of broiler on physiological parameters associated with ascites syndrome. Poult. Sci., 76: 663-667.

Soils De Los Santos, F; Tellez, G; Farnell, MB; Balog, JM; Anthony, NB; Pavlidis, HO and Donoghue, AM (2005). Hypobaric hypoxia in ascites resistant and susceptible broiler genetic lines influences gut morphology. Poult. Sci., 84: 1495-1498.

Swire, PW (1980). Ascites in broilers. Vet. Rec., 107: 541.

Wideman, RF (2001). Pathophysiology of heart/lung disorders: pulmonary hypertension syndrome in broiler chicks. World's Poult. Sci. J., 57: 289-307.

Wideman, RF and Kirby, YK (1996). Electrocardiographic evaluation of broilers during the onest of pulmonary artery occlusion. Poult. Sci., 75: 407-416.

Yen, JT; Nienaber, JA; Hill, DA and Pond, WG (1989). Oxygen consumption by portal vein-drained organs and by whole animal in conscious growing swine. Pro. Soc. Exp. Bio. Med., 190: 393-398.