# Effects of Cold Stress on Broiler Performance and Ascites Susceptibility

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**ABSTRACT:** The present study was conducted to determine the effect of cold stress on broiler performance and ascites susceptibility. Male chicks were obtained from a commercial strain of broiler breeders. The trial was divided into two treatments (control and cold stress groups). Ascites was induced in broiler chickens in the trial by exposing the chickens to low temperature (Ta) and by supplying a pelleted diet. The two experimental treatments consisted of: 1) Control group, 33.3°C the 1<sup>st</sup> wk, 30.2°C the 2<sup>nd</sup> wk, and 27.5°C the 3<sup>rd</sup> wk. 2) Cold stress group, 29.0°C the 1<sup>st</sup> wk, 26.4°C the 2<sup>nd</sup> wk, and 23.1°C the 3<sup>rd</sup> wk. From the end of the 3<sup>rd</sup> wk all broilers were reared to 6 wk of age at a constant temperature of 21°C. There was significant difference in live BW during wk 1 to 5. The control group was consistently the heaviest; however, at 6 wk of age, both groups weighed the same. Body weight gain up to 3 wk was significantly decreased by cold stress. During wk 3 and 6 the chicks in the cold stress group had greater BW gain compared with the chicks in the control group. There were significant differences in mortality due to ascites between the groups. During wk 3 and 6 the cold stress group exhibited the most ascites mortality (9.52%) when compared with the control group (1.90%). At 5 wk of age cold stress condition caused significant changes in packed cell volume (PCV), hemoglobin (Hb) and red blood cell counts (RBC). Right ventricle weight was significantly heavier in the cold stress group than the control. There were also significant differences in right ventricle/total ventricle (RV/TV) ratios at 5 wk. the right ventricle/total ventricle ratios in the cold stress group was higher (0.25) than the control group (0.20). It was concluded that, fast growth and cold temperatures are the primary triggers for ascites during commercial broiler production. (*Asian-Aust. J. Anim. Sci. 2006. Vol 19, No. 5 : 734-738*)

Key Words: Broiler, Ascites, Performance Parameters, Cold Stress, Physiological Traits

#### INTRODUCTION

Extreme selection pressure towards either growth rate and feed conversion ratio (FCR) puts high demands on the metabolic processes in broilers (Decuypere et al., 2000). Rapidly growing broiler chickens have a high metabolic requirement for oxygen that requires a high volume of blood flow through their lungs (Julian et al., 1989).

Cool temperatures are the primary triggers for ascites during commercial broiler production (Wideman, 2001). The ascites has been a worldwide source of concern to the poultry industry for several decades. It has been estimated that ascites accounts for losses of about US \$1 billion annually worldwide (Maxwell and Robertson, 1997). The incidence of ascites is higher in the colder environmental temperatures (Wideman, 1988; Shlosberg et al., 1992; Yahav et al., 1997), because cold ambient temperatures increase the oxygen requirement, cardiac output, and blood flow and may result in increased pulmonary arterial pressure overload on the right ventricle (Julian et al., 1989). A reduction in environmental temperature from 20 to 2°C for example, almost doubles the oxygen requirement in White Leghorn hens (Gleeson, 1986). If chicks are chilled before day 6, it may affect their metabolic rate for several weeks and increase the ascites. However, Malan et al. (2003) found that ascites can relate to a low metabolic rate. Cold temperature is closely associated with the ability of the broiler to produce heat. During the development of ascites, birds exhibit classic hematological changes. Hematocrit, hemoglobin and red blood cell counts (RBC) all increase dramatically (Cueva et al., 1974; Maxwell et al., 1986; 1987; Yersin et al., 1992).

Right ventricle to total ventricle (RV/TV) ratio, hemoglobin, hematocrit, blood gases and specific clinical chemistries can be used to determine the ascites status of a bird before gross lesions are apparent, and can be considered an accurate measure of the onset of ascites (Huchzermeyer and DeRuyck, 1986).

The aim of the study was to determine the effects of cold stress on broiler performance and ascites susceptibility.

#### **MATERIALS AND METHODS**

Day-old chicks were obtained from a commercial strain of broiler breeder (Ross 308) at 47 wk of age. At 1 d of age, "210 male broilers" were selected from a population of 300. Those with extreme weights were discarded (more than 2 SD of the mean). The trial was divided into two treatments (control and cold stress group). Ascites was induced in broiler chickens in the trial, by exposing the chickens to low Ta and by supplying a pelleted diet.

Chicks were weighed on an electronic balance to 0.01 g before being wing-banded and placed in environmentally controlled pens. Chicks were randomly distributed into 6 pens (three replicates of 35 chicks per pen, for each of the

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Diet composition	Starter diet 1-14 <sup>th</sup> day	Grower diet 15-28 <sup>th</sup> day	Finisher diet 29-42 <sup>nd</sup> day	
Organic matters (g/kg)	934.7	912.5	931.5	
Crude protein (g/kg)	220.0	220.0	210.0	
Ether exract (g/kg)	59.5	61.3	73.0	
Crude fibre (g/kg)	24.0	25.0	23.0	
Crude ash (g/kg)	65.1	77.5	68.5	
Nitrogen free exract (g/kg)	631.4	616.2	625.5	
ME (MJ/kg)	12.8	13.3	13.5	
Vitamin premix	0.27	0.27	0.27	
Trace mineral premix	0.16	0.16	0.16	

**Table 1.** Composition and feeding value of the diets (g/kg DM dry matter)

two groups) with a surface area of  $1.25 \times 2.0 \text{ m}^2$ . Chicks were reared on fresh wood shavings at a depth of 8-10 cm. The trial was conducted at an experimental farm located 150 m above sea level, during the cold winter months.

The two experimental treatments consisted of: 1) control group, 33.3°C the 1<sup>st</sup> wk, 30.2°C the 2<sup>nd</sup> wk, and 27.5°C the 3<sup>rd</sup> wk 2) cold stress group, 29.0°C the 1<sup>st</sup> wk, 26.4°C the 2<sup>nd</sup> wk, and 23.1°C the 3<sup>rd</sup> wk. From the end of the 3<sup>rd</sup> wk all broilers were reared to 6 wk of age at a constant temperature of 21°C.

The chicks received pelleted broiler starter diet (22.0% CP and ME 12.8 MJ/kg of diet) between the 1st and 14th day. A grower diet (22.0% CP and 13.3 MJ/kg of diet) was fed between the 15th and 28th d. A finisher diet (21.0% CP and ME 13.5 MJ/kg of diet) was administered between the 29th and 42nd d. The composition of the diets was ascertained by using the Weende analysis according to the findings of Akyıldız (1984) (Table 1). Feed and water were provided for *ad libitum*. The lighting schedule was 24 h of light from days 1 to 5 and 23 h light /1 h dark thereafter.

Live weight gain values were monitored through weekly individual weighing until the end of the 6<sup>th</sup> wk, FCR was calculated using the live weight gains and feed consumption value. The mortality and mortality due to ascites values in each pen were recorded on the pen charts daily and the mortality rates in the groups were determined depending on these data. All dead chicks were examined for the presence of typical ascites lesions, which have been previously published (Maxwell et al., 1986; Julian et al., 1989; Julian, 1993).

After 5 wk of this experiment, 10 chicks were randomly chosen from each group and were killed by decapitation and the hearts were removed and dissected to obtain heart weights for calculating the RV/TV ratio as an index of ascites (Huchzermeyer et al., 1988). Approximately 1 ml of blood was collected for glucose concentration and haematological tests including RBC, packed cell volume (PCV) and haemoglobin (Hb). Blood glucose was determined by the glucose oxidase method (Sigma Chemical Co).

Data were analyzed as one-way ANOVA using General

Linear Model procedure of the SAS (SAS Institute, 1989). A software programme using Duncan's multiple range test to compare treatment means was applied. Mortality data were analyzed using Chi-Square.

#### **RESULTS AND DISCUSSION**

In the present study, initial body weights on day 1 of the control and cold exposed group were similar. There was a significant difference in live BW during wk 1 to 5. The control group was consistently heavier, however, at 6 wk of age the groups did not differ in weight (Table 2).

Body weight gain up to 3 wk was significantly affected by cold stress. The chicks in the control environment had greater BW gain compared with the chicks in the cold stress treatment. If the chicks are in a cool rather than in a warm environment, a greater portion of their nutrient intake must be used to generate heat thus adversely affecting BW gain (Bruzual et al., 2000). However, during wk 3 to 6 compensatory growth was observed in the cold-stress group so that this group had greater BW gain than the control group. The FCR was affected by cold stress. During the 6 wk life span, the cold stress group had a significantly poorer feed efficiency when compared with the control group (Table 2).

A severe induction of ascites was demonstrated. If chicks are chilled before day 6, it may affect their metabolic rate for several weeks and increase ascites (Vogelaere et al., 1992). The propensity for broilers to develop ascites increases as ambient temperatures decrease, because the broilers need more oxygen in order to maintain their body temperature (Wideman and Tackett, 2000). Growth rate, oxygen requirement, cardiac output, heat production and metabolic rate are closely linked (Julian, 2000). Rapid growth has a major influence on oxygen requirement. Increased susceptibility of broilers to ascites has previously been linked with high growth rate (Maxwell et al., 1990; Vereijken and Albers, 1990; Arce et al., 1992; Robinson et al., 1992; Acar et al., 1995; Balog et al., 2000). Some authors indicated that the growth rate per se is not related to ascites (Barbato, 1997; Balog et al., 2001). In the present

**Table 2.** Means of body weight, growth rate, feed consumption, cumulative feed consumption and feed conversion ratio for control and cold stress group of broilers ( $\overline{X} \pm SEM$ )

Variables T	Tuaatmant	Initial weight (g)	Wks			
	Treatment		1	3	5	6
		NS	**	**	*	NS
Body weight	Control	41.6±0.35	138.7±9.30	778.6±27.42	1,851.9±54.62	2,300.18±64.21
(g/bird)	Cold	41.2±0.29	111.1±5.81	562.5±20.13	1,760.6±49.54	2,260.73±65.20
			**	**	**	**
Growth rate	Control	-	97.1±4.77	639.9±15.42	1,073.3±19.76	448.1±11.21
(g/bird)	Cold	-	69.9±4.42	451.4±11.69	1,198.1±22.35	499.4±14.24
			*	**	**	*
Feed consumption	Control	-	101.8±6.95	764.3±28.44	1,967.2±71.23	1,071.5±38.86
(g/bird)	Cold	-	115.7±7.62	812.4±30.12	2,082.6±74.51	1,120.8±42.47
				**	**	**
Cumulative feed	Control	-	-	866.1±41.47	2,833.3±84.75	3,904.8±125.26
consumption (g)	Cold	-	-	928.1±49.13	3,010.7±94.26	4,131.5±133.15
			**	**	*	**
Feed	Control	-	1.05±0.08	1.19±0.14	$1.83\pm0.47$	$2.39\pm0.17$
conversion ratio	Cold	-	1.65±0.14	1.80±0.33	$1.74\pm0.44$	2.24±0.13

Column means without either \* or \*\* do not differ (\*\* p<0.01, \* p<0.05).

Table 3. Mortality due to other reasons and mortality due to ascites, of the control and cold stress groups of broilers

Treatments	Other than ascites (%)			Due to ascites (%)	
			n = 10	n = 9	
	1-20 d	21-42 d	1-42 d	21-42 d	
	NS	NS	NS	*	
Control	2.85	2.85	5.71	1.90	
	(3/105)	(3/105)	(6/105)	(2/105)	
Cold stress	4.76	3.80	8.57	9.52	
	(5/105)	(4/105)	(9/105)	(10/105)	

Column means without either \* do not differ (\* p<0.05).

Numbers in the parentheses are no. of dead bird/total no. of birds.

study, this result was apparent in the cold stress group during wk 3 and 6. There was significant difference in mortality due to ascites between the groups (Table 3). During wk 3 and 6 the cold stress group exhibited the higher ascites mortality (9.52%) when compared with the control group (1.90%). Deeb et al. (2002) demonstrated a higher susceptibility of the fast growing vs. slow growing broilers to cold stress. The incidence of ascites will increase by about 6% for each 100 g increase in body weight at 37 d. These results were not in agreement with those of Wideman et al. (2000) who emphasized that broilers susceptible to ascites do not have to be fastest growing members of the flock.

The intensive selection of broilers for maximal body mass has resulted in anatomical and physiological limitations of blood flow through the lungs, with consequent insufficient oxygenation of the tissues (Julian, 1993). Cold ambient temperatures increase the oxygen requirement (Julian et al., 1989). The lack of oxygen stimulates RBC proliferation to the vascular system causing an increase in hematocrit (Shlosberg et al., 1998). Increases in blood volume, hematocrit and Hb concentration have

been observed in broilers acclimatized to low temperature (Vogelaere et al., 1992; Shlosberg et al., 1996; Yahav et al., 1997; Wideman et al., 1998). The PCV value is a reflection of physiological oxygen transport capacity. A high value for hematocrit in broilers with a high metabolic rate and under cold stress is an adaptive advantage because the blood's oxygen carrying capacity is enhanced. However, the downside of high PCV values is that the blood becomes more viscous and resistant to flow and as a result pulmonary vascular pressure increases (Maxwell et al., 1992).

In the present study, at 5 wk of ages cold stress caused significant changes in PCV, Hb, RBC (Table 4). These results are in agreement with many other workers (Maxwell et al., 1986 and 1987; Yersin et al., 1992; Owen et al., 1995; Luger et al., 2001).

Cold exposure requires corresponding increase in cardiac output and a greater blood volume is delivered to the gas exchange areas of the lungs and also to the systemic somatic tissues (Odom et al., 1995). It is also known that various organ changes and an increase in the RV/TV ratio indicate the onset of ascides (Cueva et al., 1974; Peacock et

**Table 4.** Mean hematological values and blood glucose concentrations of the control and cold stress groups at  $5^{th}$  wk of age ( $X \pm SEM$ )

Blood parameters	Control $(n = 10)$	Cold stress $(n = 10)$	
Glucose (mg/dL)	275.00±2.79	278.9±3.15	NS
PCV (%)	33.2±1.32	38.7.40±1.26	**
Hb $(g/dL)$	9.05±0.33	9.80±0.35	*
RBC/mm <sup>3</sup>	1,886,071±11,825	2,286,214±12,136	*

Row means without either \* or \*\* do not differ (\*\* p<0.01; \* p<0.05).

**Table 5.** Mean values of heart parameters of the control and cold stress groups at  $5^{th}$  wk of age. ( $\overline{X} \pm SEM$ )

Heart parameters	Control $(n = 10)$	Cold stress $(n = 10)$	
Chick weight (g/bird)	1,848.2±51.41	1,775.8±55.65	**
Right ventricle (RV) weight (g/bird)	1.80±0.03	$2.40\pm0.03$	**
Left ventricle+septum weight (g/bird)	7.10±0.09	7.25±0.10	NS
Total ventricle (TV) weight (g/bird)	8.90±0.13	9.65±0.12	*
RV:TV ratio	$0.20\pm0.02$	$0.25 \pm 0.02$	**

Row means without either \* or \*\* do not differ (\*\* p<0.01; \* p<0.05).

al., 1990; Owen et al., 1995).

The heart parameters of the control and cold stress groups at 5<sup>th</sup> wk of age are given in Table 5. Right ventricle weight was significantly higher in the cold stress group than the control. There were also significant differences in RV/TV ratios at 5 wk. RV/TV ratios in the cold stress group was higher (0.25) than the control group (0.20). However, it is generally accepted that an RV/TV index greater than 0.28 is indicative of right ventricular hypertrophy, ascites (Julian 1988, 1993; Lubritz et al., 1995; Owen et al., 1995; Wideman, 2001). In this study, neither of the two groups exceeded this 0.28 limit. It is unclear whether or not these birds would have eventually developed full ascites, but early heart changes seem to indicate that they would.

### **CONCLUSIONS**

It was concluded that cold temperature is a primary triggers for ascites during commercial broiler production. The cold stress treatment had no effect on the final body weight. However, during wk 3 and 6 the broilers in the cold stress group had greater BW gain than the control group and this group exhibited more ascites mortality. Significantly, different PCV and RV/TV ratio were found between the cold stress group and the control. Fast BW gain, PCV, Hb and RV/TV ratio are good predictors of ascites development.

#### **REFERENCES**

Acar, N., F.G. Sizemore, G. R. Leach, R. F. Jr. Wideman, R. L. Owen and G. F. Barbato. 1995. Growth of broiler chickens in response to feed restriction regimes to reduce ascites. Poult. Sci. 74:833-843.

Akyıldız, R. 1984. Yemler Bilgisi Laboratuar Kılavuzu (Turkish of: Laboratory guide of Feeds and Feed Technology). Ank. Univ. Zir. Fak. Yay. 895, Uygulama Kılavuzu Ankara. pp. 213-236.

Arce, J., M. Berger and C. L. Coello. 1992. Control of the ascites syndrome by feed restriction techniques. J. Appl. Poult. Res. 1:1-5.

Balog, J. M., N. B. Anthony, M. A. Cooper, B. D. Kidd, G. R. Huff, W. E. Huff and N. C. Rath. 2000. Ascites syndrome and related pathologies in feed restricted broilers raised in a hypobaric chamber. Poult. Sci. 79:318-323.

Balog, J. M., N. B. Anthony, B. D. Kidd, X. Liu, M. A. Cooper, G.
R. Huff, W. E. Huff, R. F. Widemann and N. C. Rath. 2001.
Genetic selection of broiler lines that differ in their ascites susceptibility 2. response of the ascites lines to cold stress and bronchus occlusion. In Proceedings 13<sup>th</sup> European Symposium On Poultry Nutrition, Blankenberghe, Belgium, pp. 329-330.

Barbato, G. F. 1997. Selection for growth and poultry welfare: a genetic perspective. In: Proceedings the fifth european symposium on poultry welfare, Wageningen Agricultural University, The Netherlands. pp. 63-71.

Bruzual, J. J., S. D. Peak, J. Brake and E. D. Peeblest. 2000. Effect of relative humidity during the last five days of incubation and brooding temperature on performance of broiler chicks from young broiler breeders. Poult. Sci. 79:1385-1391.

Cueva, S., H. Sillau, A. Valenzuela and H. Ploog. 1974. High altitude induced pulmonary hypertension and right heart failure in broiler chickens. Res. Vet. Sci. 16:370-374.

Decuypere, E., J. Buyse and N. Buys. 2000. Ascites in broiler chickens: exogenous and endogenous structural and functional causal factors. World's Poult. Sci. 56(4):367-377.

Deeb, N., A. Shlosberg and A. Cahaner. 2002. Genotype by environment interaction with broiler genotypes differing in growth rate. 4. association between responses to heat stress and to cold induced ascites. Poult. Sci. 81:1454-1462.

Gleeson, M. 1986. Respiratory adjustments of the unanaesthetized chicken, gallus domesticus, to elevated metabolism elicited by 2,4 dinitrophenol or cold exposure. Comp. Biochem. Phys. 83a: 283-289.

Huchzermeyer, F. W. and A. M. C. De Ruyck. 1986. Pulmonary hypertension syndrome associated with ascites in broilers. Vet. Rec. 119:94.

Huchzermeyer, F. W., A. M. C. De Ruyck and H. Van Ark. 1988.Broiler pulmonary hypertension syndrome. iii. commercial broiler strains differ in their susceptibility. Onderstepoort J.

- Vet. Res. 55:5-9.
- Julian, R. J. 1988. Pulmonary hypertension as a cause of right ventricular failure and ascites in broilers. Zootecnica Intern. November, 58-62.
- Julian, R. J., I. Mcmillan and M. Aquinton. 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.
- Julian, R. J. 1993. Ascites in poultry. Avian Pathol. 22:419-454.
- Julian, R. J. 2000. Physiological, management and environmental triggers of the ascites syndrome: a review. Avian Pathol. 6:519-527.
- Lubritz, D. L., J. L. Smith and B. N. Mcpherson. 1995. Heritability of ascites and the rato of right to total ventricle weight in broiler breeder male lines. Poult. Sci. 74:1237-1241.
- Luger, D., D. Shinder, V. Rzepakovsky, M. Rusal and S. Yahav. 2001. Association between weight gain, blood parameters, and thyroid hormones and the development of ascites syndrome in broiler chickens. Poult. Sci. 80:965-971.
- Malan, D. D., C. W. Scheele, J. Buyse, C. Kwakernaak, F. K. Siebrits, J. D. Van Der Klis and E. Decuypere. 2003. Metabolic rate and its relationship with ascites in chicken genotypes. Br. Poult. Sci. 44:309-315.
- Maxwell, M. H., G. H. Robertson and S. Spence. 1986. Studies on an ascitic syndrome in young broilers. 1: hematology and pathology. Avian Pathol. 15:511-524.
- Maxwell, M. H., S. G. Tullett and F. G. Burton. 1987. Haemotology and morphological changes in young broiler chicks with experimentally induced hypoxia. Res. Vet. Sci. 43:331-338.
- Maxwell, M. H., S. Spence, G. W. Robertson and M. A. Mitchell. 1990. Heamatological and morphological responses of broiler chicks to hypoxia. Avian Pathol. 19:23-40.
- Maxwell, M. H., G. W. Robertson and C. C. Mccorquodale. 1992. Whole blood and plasma viscosity values in normal and ascitic broilers. Br. Poult. Sci. 33:871-877.
- Maxwell, M. H. and G. W. Robertson. 1997. World broiler ascites survey 1996. Poult. Int. 36:16-19.
- Odom, T. W., L. M. Rosenbaum and J. S. Jeffery. 1995. Experimental reduction of eggshell conductance during incubation i. effect on the susceptibility to ascites syndrome. Avian Disease. 39:821-829.
- Owen, R. L., R. F. Jr. Wideman, R. M. Leach, B. S. Cowen, P. A. Dunn and B. C. Ford. 1995. Effect of age of exposure and dietary acidification or alkalinization on mortality due to pulmonary hypertension syndrome. J. Appl. Poult. Res. 3:244-252.
- Peacock, A. J., C. Pickett, K. Morris and J. T. Reeves. 1990. Spontaneous hypoxaemia and right ventricular hypertrophy in fast growing broiler chickens reared at sea level. Comp. Biochem. Physiol. 97a:537-541.

- Robinson, F. E., H. L. Classen, J. A. Hanson and D. K. Onderka. 1992. Growth performance, feed efficiency and the incidence of skeletal and metabolic disease in full fed and feed restricted broiler and roaster chickens. J. Appl. Poult. Res. 1:33-41.
- SAS Institute, Inc. 1989. A user's guide to SAS. SAS Institute, Inc. Cary, NC.
- Shlosberg, A., G. Pano, J. Handji and E. Berman. 1992. Prophylactic and therapeutic treatment of ascites in broiler chickens. Br. Poult. Sci. 33:141-148.
- Shlosberg, A., M. Bellaiche, V. Hanji, A. Nyska, M. Lubln, M. Shemesh, L. Shore, S. Perk and E. Berman. 1996. The effect of acetylsalicylic acid and cold stress on the susceptibility of broilers to the ascites syndrome. Avian Pathol. 25:581-590.
- Shlosberg, A., M. Bellaiche, E. Berman, S. Perk, N. Deeb, E. Neumark and A. Cahaner. 1998. Relationship between broiler chicken hematocrit-selected parents and their progeny, with regard to hematocrit, mortality from ascites and bodyweight. Res. Vet. Sci. 64:105-109.
- Vereijken, A. L. J. and G. A. A. Albers. 1990. The genetics of ascites susceptibility in broilers. In: Proceedings of the Vin European Poultry Conference Barcelona, Spain. pp. 525-528.
- Vogelaere, P., G. Savourey, G. Daklunder, J. Lecroart, M. Brasseur, S. Bekaert and J. Bittel. 1992. Reversal of cold induced haemoconcentration. European J. Appl. Physiol. 64:244-249.
- Wideman, R. F., Jr., 1988. Ascites in poultry. Monsanto Nutr. Update 6:1-7.
- Wideman, R. F., Jr., T. Wing, Y. K. Kirby, M. F. Forman, N. Marson, C. D. Tackett and C. A. Ruiz-Feria. 1998. Evaluation of minimally invasive indices for predicting ascites susceptibility in three successive hatches of broilers exposed to cool temperatures. Poult. Sci. 77:1565-1573.
- Wideman, R. F., Jr., M. R. Fedde, C. D. Tackett and G. E. Weigle. 2000. Cardio-pulmonary function in preascitic (hypoxemic) or normal broilers inhaling ambient air or 100% oxygen. Poult. Sci. 79:415-425.
- Wideman, R. F. and C. Tackett. 2000. Cardio-pulmonary function in preascitic (hypoxemic) or normal broilers reared at warm or cold temperatures: effect of acute inhalation of 100% oxygen. Poult. Sci. 79:257-264.
- Wideman, R. F. 2001. Pathophysiology of heart/lung disorders: pulmonary hypertension syndrome in broiler chickens. World's Poult. Sci. 57(3):289-307.
- Yahav, S., A. Straschnow, I. Plavnik and S. Hurwitz. 1997. Blood system response of chickens to changes in environmental temperature. Poult. Sci. 76:627-633.
- Yersin, A. G., W. E. Huff, L. F. Kubena, M. H. Elissalde, R. B. Harvey, D. A. Witzel and L. E. Giror. 1992. Changes in haematological, blood gas, and serum biochemical variables in broilers during exposure to simulated high altitude. Avian Diseases 36:189-196.