American Journal of Animal and Veterinary Sciences 2 (3): 62-65, 2007
ISSN 1557-4555


# Study on Mechanism of Ascites Syndrome of Broilers

[1]J.L. Guo, [2]Q.H. Zheng, [2]Q.Q. YIN, [1]W. Cheng and [2]Y.B. Jiang
[1]Zhengzhou College of Animal Husbandry Engineering, Zhengzhou 450011
[2]Department of Animal Science, Henan Agricultural University,
Zhengzhou 450002, People's Republic China

**Abstract:** Two hundred and forty male Cobb broilers were used to study the reasons of causing ascites. The results showed that cold ambient temperature could induce ascites (33.89% vs. 2.50%) and significantly increase triiodothyronine ($T_3$), but reduce thyroxine ($T_4$) concentrations in plasma ($p<0.05$). The ascitic broilers had high concentrations of aldosterone and $K^+$, but low concentrations of $T_3$ and $T_4$ in the plasma ($p<0.05$). The lower body weight and higher relative heart, lung and liver weight of the ascitic broilers demonstrated the metabolic disarrangement. When ascites occurred, hematocrit in blood increased significantly ($p<0.05$). The mash feed could reduce body weight and the onset of ascites, compared with the pellet feed.

**Key words:** Broilers, ascites, plasma, biochemical indexes, body weight and relative organ weight

## INTRODUCTION

The ascites syndrome, a metabolic disorder that accounts for over 25% of overall mortality, has become the most noticeable, non-infectious cause of loss in the broiler industry worldwide[1]. There are many factors that cause ascites, for example, high altitude, rapid growth rate, limiting lung volume, the provision of high energy rations and pelleted diets, cold, poor ventilation, the presence of respiratory disease, high sodium and low dietary phosphorus levels, hepatotoxins, mycotoxins and furazolidone in the feed, vitamin E and Se deficiencies and stress[2,3,4]. Among so many causes, which one is the main trigger is still questionable. It was reported that low temperature was an easy and economical method to trigger ascites[5]. One report has indicated that high nutrient metabolic rate could cause ascites[6], however high levels of the hormones ($T_3$ and $T_4$) in the plasma are related to nutrient metabolism. The aim of this research is to use low temperature to trigger ascites in order to investigate the relationship between some physiological parameters and ascites and to seek the optimum methods of predicting and preventing this disease.

## MAERIALS AND METHODS

**Broilers and treatments:** The experiment was conducted in chicken farm, Henan Agricultural University, from March to June in 2005. Two hundred and forty male Cobb broilers were divided equally into 24 groups and allocated to one of four treatments. The birds in treatment 1 (16 groups) were fed with pelleted feeds in a relatively cold environment to induce ascites from 3 weeks of age; the birds in treatment 2 (4 groups) were fed with pelleted feeds under common condition as control; and the birds allocated to Treatment 3 (4 groups) were fed with mash feeds under common condition as control too. The allocation of birds to the conditions described for treatment 1 was such that there would be sufficient birds showing ascites.

The ambient temperatures for both the control and cold environments were similar for the first 2 wks of the experiment. At wk 3, the cold environment was 2 cooler (23 vs. 25) and from wk 4 was further reduced (13 vs. 25). Fine control of temperature was needed during this time to achieve sensible results in any experiment.

**Diets and managements:** The broilers were fed with commercial corn-soybean diets formulated according to NRC (1994) standards[7]. The crude protein and energy levels for the broilers at the age of 0-3 and 4-7 wks were 23.1% and 13.44 MJ ME $kg^{-1}$, 19.8% and 13.42 MJ ME $kg^{-1}$, respectively. Feed and water were provided ad libitum and the birds were grown under continuous lighting.

**Sample analyses and statistical analyses:** The birds were weighed weekly and blood was taken and analyzed at three wks of age and weekly intervals thereafter from a sample of chickens in each treatment. The hormones, mineral and protein concentrations in plasma were measured using a gamma counter (Diagnostic Products Corporation, Los Angeles, CA., USA), atomic absorption spectrometer (GBC Scientific Equipment Pty Ltd., Dandenong, VIC, Australia) and

**Corresponding Author:** Yin, Q.Q., Department of Animal Science, Henan Agricultural University, Zhengzhou 450002, P.R. China

colorimetric methods (Elisa, 520nm; Helena Laboratories, Beaumont, Texas, USA), respectively.

**Statistical analyses:** Experimental data were expressed as the means and standard errors. The data were analyzed using the ANOVA procedures of Statistical Analysis Systems Institute, 1992. Duncan's multiple range test was used to compare treatment means. Differences were considered statistically significant at $p<0.05$.

## RESULTS AND DISCUSSION

Ambient temperature and $T_3$ and $T_4$ concentrations in plasma: Cold ambient temperature tended to increase $T_3$ concentration but decrease $T_4$ concentration, compared to the other treatments ($p<0.05$). The broilers showing obvious signs of ascites had generally lower $T_3$ concentrations than those without sign (Table 1).

The results showing that low environmental temperature increased plasma $T_3$ concentration agree with Scheele *et al*.[8]. This increase may be due to the need for additional metabolic heat to maintain body temperature in the cooler environment. The subsequent increase in metabolic rate results in an increase in blood pressure as the heart attempts to maintain oxygen supply to the organs and muscles thus leading to pulmonary hypertension and right ventricular failure. Interestingly, even though high $T_3$ concentration could induce ascites[9], the ascetic broilers had lower plasma $T_3$ than the healthy ones in the "cold" control group[5], especially after 5 wks old. The reason may be that metabolic disarrangement could have taken place as soon as ascites occurred and this could influence $T_3$ excretion. $T_4$ had the opposite response, i.e. low temperature reduced $T_4$. It has been assumed that low temperatures improve the excretion of thyroid-stimulating hormone (TSH)[10] and the excretion speed of thyroid[11] to cause the thyroid to produce more $T_4$ and less $T_3$, or make $T_4$ be transferred to $T_3$ because $T_3$ is more effect than $T_4$ to increase metabolic rate for the birds to adapt to the cold stress condition.

**Body weight and relative organ weight:** The ascetic broilers had the lowest body weight ($p<0.05$) and the highest organ ratio (heart, lung, liver, right ventricular weights/body weight) at 7 wks of age ($p<0.05$) (Table 2). These changes are parts of the syndrome of hypoxaemia[5,12]. The provision of a pellet feed increased body weight gain, Ca, Na, P and total protein concentrations in the plasma, compared with the mash feed. The reason was that pellets improved feed conversion. Because the birds had a lower body weight when fed with the mash diet, the feed and oxygen demanded by the body would be less than the pellet fed birds and hence the tendency to develop ascites would

Table1: The relationship between ascites and plasma hormone contents (ng mL$^{-1}$) (means±SE)

| | Week 4 | | Week 5 | | Week 6 | | Week 7 | |
|---|---|---|---|---|---|---|---|---|
| $T_3$ | | n | | n | | n | | n |
| Ascites$^+$ | 3.28±0.11$^a$ | 16 | 2.71±0.17$^b$ | 17 | 2.11±0.24$^b$ | 11 | 1.08±0.26$^{bc}$ | 6 |
| Ascites$^-$ | 2.94±0.16$^a$ | 10 | 3.28±0.23$^a$ | 10 | 2.79±0.54$^a$ | 10 | 1.94±0.23$^a$ | 8 |
| Pellet | 1.64±0.15$^b$ | 10 | 2.18±0.22$^b$ | 10 | 1.42±0.24$^c$ | 10 | 1.48±0.19$^{ac}$ | 10 |
| Mash | 0.38±0.15$^b$ | 9 | 2.31±0.22$^b$ | 10 | 1.35±0.31$^c$ | 10 | 0.49±0.25$^c$ | 6 |
| $T_4$ | | | | | | | | |
| Ascites$^+$ | 3.05±0.39$^d$ | 16 | 3.09±0.49$^b$ | 17 | 1.21±0.35$^b$ | 10 | 1.75±0.84$^c$ | 6 |
| Ascites$^-$ | 4.29±0.49$^c$ | 10 | 3.69±1.49$^b$ | 10 | 2.19±1.10$^b$ | 10 | 4.38±0.73$^b$ | 8 |
| Pellet | 6.76±1.39$^b$ | 10 | 4.66±0.64$^{ab}$ | 10 | 6.39±0.39$^a$ | 9 | 7.23±0.68$^a$ | 10 |
| Mash | 10.38±0.53$^a$ | 9 | 6.04±0.67$^{ba}$ | 10 | 7.38±1.35$^a$ | 10 | 8.46±0.68$^a$ | 9 |

**Note:** Birds from treatments denoted with ascites$^+$ and ascites$^-$ were sourced from treatment 1, Pellet from treatment 2, and Mash from treatment 3. Means lacking a common superscript letter in each line differ ($p<0.05$). n---- Number of broilers

Table 2: Body weights and relative organ weights (at 7 wks old) (means±SE)

| | Ascites$^+$ | Ascites$^-$ | Pellet | Mash |
|---|---|---|---|---|
| Number of broilers | 19 | 15 | 10 | 10 |
| Body weight (kg) | 2.13±0.09$^a$ | 2.89±0.10$^b$ | 3.32±0.05$^c$ | 2.89±0.06$^b$ |
| Organ weight/body weight (%) | | | | |
| Heart | 0.72±0.04$^a$ | 0.59±0.03$^b$ | 0.45±0.02$^c$ | 0.44±0.01$^c$ |
| Lung | 0.59±0.03$^a$ | 0.42±0.02$^b$ | 0.38±0.01$^b$ | 0.42±0.02$^b$ |
| Liver | 2.66±0.13$^a$ | 1.98±0.05$^{bc}$ | 1.81±0.12$^c$ | 2.18±0.11$^b$ |
| Right ventricular | 0.18±0.02$^a$ | 0.13±0.02$^b$ | 0.11±0.01$^b$ | 0.09±0.01$^b$ |
| Right ventricular to total ventricular (RV/TV, %) | 25.85±1.24$^a$ | 21.27±0.69$^b$ | 22.16±0.54$^b$ | 22.31±1.27$^b$ |
| Rate of ascites (%) | --- | 33.89 | 2.50 | 0.00 |

**Note:** Means lacking a common superscript letter in each column differ ($p<0.05$)

be less.

**Mineral, protein and albumin concentrations in plasma:** Table 3 showed that the ascetic broilers had higher concentrations of $K^+$ in plasma at the age of 6 wks, compared with the healthy broilers. This result corresponds with the higher aldosterone concentration in plasma because the function of aldosterone is to discharge high concentration of $K^+$ from animal body. The higher $K^+$ concentration in plasma may have been due to leakage of plasma from the circulatory system caused by right ventricular failure.

Table 4 indicated the changes of total protein and albumin concentrations in plasma. At the age of 5 wks, the lower temperature increased total protein and albumin concentrations ($p<0.05$) and the ascetic birds had lower total protein content. At the age of 7 wks, the pellet diet made the total protein content higher, compared with the mash diet. The concentration of total and albumin in plasma was not significantly different between ascitic broilers and non-ascitic broilers, which supports the theory that the ascitic fluid is plasma from the circulatory system, except for the birds at the age of 5 weeks.

**Aldosterone and renin contents in plasma and hematocrit changes in blood:** Table 5 indicated that the ascetic birds could make the concentration of aldosterone in plasma higher (37.43 vs. 12.10 pg $mL^{-1}$). This result may be induced by higher concentration of $K^+$ in plasma. Table 5 also showed that there was a

Table 3: Mineral Concentrations in plasma (mg $L^{-1}$) (means±SE)

| Ascites+ | Ascites- | Pellet | Mash | |
|---|---|---|---|---|
| Wk 5 | | | | |
| Number of broilers | 17 | 9 | 10 | 10 |
| Ca | 98.42±3.15[a] | 110.18±2.72[b] | 99.44±1.72[a] | 99.03±1.01[a] |
| Mg | 19.68±0.97[a] | 19.34±0.67[a] | 18.97±0.38[a] | 19.81±0.51[a] |
| Na | 3522.83±66.34[a] | 3398.84±85.51[a] | 3318.89±26.44[a] | 3353.77±27.05[a] |
| K | 237.60±5.51[a] | 237.69±9.02[a] | 232.58±3.76[a] | 252.11±6.75[a] |
| P | 163.86±5.97[a] | 168.83±3.67[a] | 179.91±4.37[a] | 174.26±3.71[a] |
| Wk 7 | | | | |
| Number of broilers | 8 | 15 | 10 | 11 |
| Ca | 134.78±2.88[a] | 132.68±4.23[ab] | 121.75±4.42[b] | 104.73±1.44[c] |
| Mg | 27.09±0.64[a] | 24.03±0.73[ab] | 25.64±2.46[ab] | 21.41±0.31[b] |
| Na | 3582.39±69.59[a] | 3491.35±40.63[ab] | 3372.30±60.46[b] | 2898.55±61.09[c] |
| K | 292.40±22.17[a] | 219.02±5.75[b] | 231.70±17.00[b] | 206.88±5.29[b] |
| P | 186.52±31.87[ab] | 180.00±3.74[ab] | 190.58±14.48[a] | 145.26±3.04[b] |

Note: Means lacking a common superscript letter in each column differ ($p<0.05$).

Table 4: Total protein and albumin content in plasma (g $L^{-1}$) (means±SE)

| | Ascites+ | | Ascites- | | Pellet | | Mash | |
|---|---|---|---|---|---|---|---|---|
| Total Protein | | n | | n | | n | | n |
| Wk 3 | 31.3±0.5[a] | 16 | 30.5±0.7[a] | 10 | 31.6±0.8[a] | 10 | 31.1±0.9[a] | 10 |
| Wk 5 | 34.8±0.9[a] | 16 | 38.1±1.1[b] | 10 | 33.7±0.7[ac] | 10 | 31.2±0.6[c] | 10 |
| Wk 7 | 31.3±2.1[ab] | 8 | 34.1±0.9[a] | 15 | 34.0±1.4[a] | 10 | 27.0±0.8[b] | 11 |
| Albumin | | | | | | | | |
| Wk 3 | 12.1±0.1[a] | 16 | 11.9±0.3[a] | 10 | 12.2±0.2[a] | 10 | 12.1±0.3[a] | 10 |
| Wk 5 | 14.1±0.4[a] | 16 | 14.4±0.3[a] | 10 | 13.1±0.2[b] | 10 | 12.8±0.2[b] | 10 |
| Wk 7 | 12.3±0.8[a] | 8 | 12.2±0.2[a] | 15 | 11.6±0.3[ab] | 10 | 10.5±0.2[b] | 11 |

Note: Means lacking a common superscript letter in each column differ ($p<0.05$).

Table 5: Aldosterone and renin contents in plasma and hematocrit in blood (means±SE)

| | Ascites+ | Ascites- | | Pellet | | Mash | | |
|---|---|---|---|---|---|---|---|---|
| Wk 6 | | n | | n | | n | | n |
| Aldosterone (pg ml) | 37.43±6.18[a] | 8 | 12.10±6.18[b] | 8 | 5.82±7.81[b] | 5 | 15.42±8.74[ab] | 4 |
| Renin (ng $mL^{-1}$) | 0.62±0.07[a] | 17 | 0.67±0.09[a] | 10 | 0.68±0.09[a] | 10 | 0.43±0.09[a] | 9 |
| Hematocrit (%) | | | | | | | | |
| Wk 3 | 26.29±0.53[a] | 19 | 26.27±0.44[a] | 15 | 26.17±0.43[a] | 10 | 25.16±0.33[a] | 10 |
| Wk 4 | 31.44±0.55[a] | 18 | 30.51±0.48[ab] | 15 | 28.88±0.32[bc] | 10 | 28.67±0.58[c] | 10 |
| Wk 5 | 35.58±1.25[a] | 18 | 33.48±0.69[a] | 15 | 29.19±0.69[b] | 10 | 28.36±0.88[b] | 10 |
| Wk 6 | 39.79±2.59[a] | 13 | 33.17±0.67[b] | 15 | 28.88±0.54[bc] | 7 | 27.60±0.73[c] | 10 |
| Wk 7 | 39.85±3.89[a] | 8 | 34.50±0.78[b] | 15 | 28.60±0.73[bc] | 10 | 29.10±0.78[c] | 10 |

Note: Means lacking a common superscript letter in each column differ ($p<0.05$)

tendency towards a greater haematocrit value of the birds in the ascites+ treatment from wk 5 and which thereafter was significantly greater than the other three treatments ($p<0.05$). The highest hematocrits values of the ascetic broilers indicated that a (subjective) rapid compensatory erythropoiesis had been triggered in response to the hypoxaemia[5], but Luger *et al.*[13]' report showed that hematocrits was low before the ascetic broiler died, which was different from this result.

## CONCLUSION

(1) the increase of $T_3$ concentration in plasma and hematocrits in blood and decrease of $T_4$ concentration in plasma are the signals to predict that the ascites will take place; (2) the decrease of $T_3$ and $T_4$ concentrations in plasma and increase of hematocrits in blood are the signals to predict that the ascites has taken place; (3) the practical suggestions for preventing ascites in broilers are to maintain heat stable throughout the growing period[14], decrease heat slowly and feed balanced mash diets as the final rations.

## REFERENCES


1. De Smit, L., K. Tona, V. Bruggeman, O. Onagbesan, M. Hassanzadeh, L. Arckens and E. Decuypere, 2005. Comparison of three lines of broilers differing in ascites susceptibility or growth rate. 2. Egg weight loss, gas pressures, embryonic heat production and physiological hormone levels. Poult. Sci., 84: 1446-1452.
2. Vanhooser, S. L., A. Beker and R. G. Teeter, 1995. Bronchodilator, oxygen level and temperature effects on ascites incidence in broiler chickens. Poult. Sci., 74:1586-1590.
3. Diaz-Cruz, A., C. Nava, R. Villanueva, M. Serret, R. Guinzberg and E. Pina, 1996. Hepatic and cardiac oxidative stress and other metabolic changes in broilers with the ascites syndrome. Poult. Sci., 75: 900-903.
4. Gordon, S., 1997. Effect of light programmes on broiler mortality with reference to ascites. World's Poul. Sci. J., 53: 68-70.
5. Scheele, C.W., J.D. van Der Klis, C. Kwakernaak, N. Buys and E. Decuypere, 2003. Haematological characteristics predicting susceptibility for ascites. 2. High haematocrit values in juvenile chickens. Br. Poult. Sci., 44: 484-489.
6. Olkowski, A.A. and H.L. Classen, 1998. Progressive bradycardia, a possible factor in the pathogenesis of ascites in fast growing broiler chickens raised at low altitude. Br. Poult. Sci., 39: 139-146.
7. National Research Council, 1994. Nutrient Requirements of Poultry. 9th Edn. National Academy Press, Washington, DC.
8. Scheele, C.W., E. Decuypere, P.F. Vereijken and F.J. Schreurs, 1992. Ascites in broilers. 2. Disturbances in the hormonal regulation of metabolic rate and fat metabolism. Poult. Sci., 71: 1971-1984.
9. Chineme, C.N., J. Buyse, N. Buys, H.M. Ladmakhi, G.A.A. Albers and E. Decuypere, 1995. Ascites syndrome induced by low temperature. Archiv fur Geflugelkunde. 59: 129-134.
10. Hendrich, C.E. and C.W. Turner, 1964. Estimation of thyroid stimulating hormone (TSH) Secretion rates of New Hampshire fowls. Proc. Soc. Exp. Biol. Med., 117: 218.
11. Mueller, W.J. and A.A. Amezcua, 1959. The relationship between certain thyroid characteristics of pullets and their egg production, body weight and environment. Poult. Sci., 38: 620.
12. Julian, R.J., G.W. Friars, H. French and M. Quinton, 1987. The relationship of right ventricular hypertrophy, right ventricular failure and ascites to weight gain in broiler and roaster chickens. Avian Dis., 31: 130-135.
13. 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.
14. Sato, T., K. Tezuka, H. Shibuya, T. Watanabe, H. Kamata and W. Shirai, 2002. Cold-induced ascites in broiler chickens and its improvement by temperature-controlled rearing. Avian Dis., 46: 989-996.