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CLINICAL AND EXPERIMENTAL STUDIES IN SPONTANEOUS AND EXPERIMENTAL ALOPECIA IN HENS. STUDY ON THE ACID-BASE STATUS

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Summary

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The aim of the study was to follow out the acid-base status in spontanoues and experimental alopecia in hens. For this purpose, 40 Hissex layer hens at the age of 1 year were used. The studies lasted for 2 monts after the appearance of clinical signs.
The results showed that the spontaneous alopecia in hens was accompanied by compensated acidosis whereas the experimental alopecia – by decompensated metabolic acidosis with hypercapnia (mixed acidosis).

Key words: acid-base status, alopecia, hens

INTRODUCTION

The intensive poultry breeding is accompanied by infectious, parasitic and some non-infectious diseases, such as the spontaneous alopecia (SA). It is clinically manifested with feather loss (Nachev *et al.*, 1964) and decreased productivity (Tsokova, 2002). The inducing of experimental models of alopecia could be achieved by various factors as food and water deprivation, darkness (Stevenson and Jackson, 1984; Berry and Brake, 1985), thyroxin administrations (Szelenyi and Peczely, 1988) or via increased intake of sodium chloride or zinc oxide (Sekimoto *et al.*, 1990).

The studies of acid-base status in various avian pathological states are relatively few (Scott, 1982; Ait–Bonlashen *et al.*, 1989; Angelov and Koinarski, 1997) and acid-base changes occurring during SA in hens are not studied.

The aim of the present study was to follow out the acid-base changes after the onset of spontaneous or experimental alopecia in hens in order to elucidate some aspects of the pathogenesis of the disease.

MATERIALS AND METHODS

Animals

The experiments were performed on 40 Hissex laying hens at the age of about 1 year, weighing 1.8–2 kg, obtained from the Chirpan poultry farm.

The birds were housed at the Clinic of Internal Non-infectious Diseases in one

premises equipped with separate sections for each group on a litter, with a density of 10 hens/2 m^2. Each section was provided with places for perch and two nests for egg laying. The light regimen was of 17 h light daily with intensity of 3.5 W/m^2. All hens received daily 100 g of a standard diet and had free access to drinking water.

Experimental design

Two experiments were performed.

The first experiment was carried out in February, when for several years the poultry farm had recorded a feather loss among birds aged over 1 year. The plumage was reconstituted for about 2–3 months. Ten hens with spontaneous alopecia with feather loss amounting for about half of body surface (the whole back, the tail and part of the neck) were chosen. The anamnesis revealed that in the poultry farm, the hens were housed in three-floor cell batteries with 3 hens in a cell (40 cm height and 350 cm^2 per bird; feeding front of 8–10 cm and drinking front of 3 cm per bird). The zoohygienic conditions were not optimal – the air temperature was about 5–10 ^oC and air humidity – over 70%. The light regimen was optimal. Another ten healthy hens from the same flock were used as controls.

In the second experiment, two groups were also used: experimental group (n=10) where experimental alopecia (EA) was induced by the method of Zavadovski (cited after Pahmurin, 1919) via a single oral treatment with 15 g cut and dried tissue of thyroid glands obtained from calves and control group (n=10) of healthy birds. Both experiments were paralelly accomplished.

The studies of experiment one were done by months 1 and 2 after the SA appearance. In experiment two, the analyses were done by months 1 and 2 after the thyroid tissue administration. The severity of alopecia was similar to that in experiment 1.

Acid-base status

Blood samples of 150 μL were anaerobically obtained from v. cutanea ulnaris in heparinized capillary tubes and immediately analysed on an automated acid-base analyser (ABL–3, Radiometer, Denmark).

Blood pH and partial pressures of blood carbon dioxide and oxygen (pCO_2 and pO_2) were measured with electrodes and haemoglobin content – by photometry. The actual bicarbonates (HCO_3^-), total carbon dioxide content (TCO_2), the actual base excess (ABE), the standard base excess (SBE). the oxygen saturation (SAT) and the oxygen content (O_2CT) were calculated on the basis of measured parameters.

Clinical and biochemical analyses

The respiratory rate was determined using routine methods. Blood uric acid and blood creatinine were determined on a automated biochemical analyser (Reflotron, Boerhinger, Germany) with diagnostic kits (Roche Diagnostics, Germany).

Statistical analysis

The data (mean± SEM) were statistically processed by the Student's t-test at a level of significance $p \leq 0.05$.

RESULTS

The changes in the acid–base status are presented in Tables 1 and 2. It was seen that during the first month, the experimental birds with SA did not exhibit any considerable changes in pH, pCO_2, pO_2, SBC, SAT, O_2CT and haemoglobin. Actual bicarbonates and TCO_2 were however ele-

Table 1. Changes in acid-base parameters in laying hens – healthy (controls) and with spontaneous alopecia by months 1 and 2 after the appearance of clinical symptoms (mean ±SEM; n=10)

Parameters	Month 1		Month 2	
	Control group	Experimental group	Control group	Experimental group
pH	7.26±0.02	7.22±0.02	7.34±0.02	7.26±0.01
pCO_2, mmHg	52.3±1.82	57.31±3.20	56.57±3.57	55.22±2.45
pO_2, mmHg	52.02±3.01	52.64±3.92	58.42±7.54	63.72±3.89
HCO^-_3,mmol/L	19.74±0.55	23.72±0.91**	22.43±0.91	22.70±0.63
TCO_2, mmol/L	21±0.53	25.14±0.94**	23.88±0.94	24.08±0.70
ABE, mmol/L	6.12±0.80	2.61±0.76*	3.28±1.00	2.87±0.47
SBE, mmol/L	5.96±0.76	2.59±0.67*	3.15±0.99	2.72±0.51
SBC, mmol/L	18.86±0.61	22.26±2.24	21.25±0.81	21.65±0.37
SAT, %	65.60±3.15	61.48±4.14	69.63±8.43	78.35±3.47
O_2CT, %	8.14±0.49	8.56±0.54	7.47±0.95	9.48±0.45
Hb, g/L	87.80±1.70	89.90±1.80	86.00±2.3	85.70±1.90

* $p < 0.05$, ** $p<0.01$ vs controls.

Table 2. Changes in acid-base parameters in laying hens – healthy (controls) and with experimental alopecia, induced by treatment with thyroid gland tissue by months 1 and 2 after the appearance of clinical symptoms (mean ±SEM; n=10)

Parameters	Month 1		Month 2	
	Control group	Experimental group	Control group	Experimental group
pH	7.33±0.03	7.23±0.03*	7.35±0.10	7.25±0.09
pCO_2, mmHg	58.52±1.59	69.38±2.03**	57.55±1.28	60.8±2.65
pO_2, mmHg	56.28±1.38	52.22±2.68	55.88±1.71	54.07±3.10
HCO^-_3,mmol/L	18.95±0.35	25.75±0.67*	20.28±1.39	22.07±1.50
TCO_2, mmol/L	21.0±0.93	26.83±0.65***	20.02±0.55	22.17±1.37
ABE, mmol/L	6.02±0.48	2.42±0.58***	4.9±0.57	3.97±0.66
SBE, mmol/L	6.25±0.47	2.02±0.32***	5.52±0.42	4.07±0.53
SBC, mmol/L	18.58±0.40	23.15±0.73***	20.9±0.98	21.62±1.05
SAT, %	65.57±3.17	66.18±5.17	62.8±2.71	64.87±4.00
O_2CT, %	8.22±0.40	8.68±0.67	8.28±0.51	7.7±0.65
Hb, g/L	81.0±1.66	97.55±2.37***	84.95±2.96	89.95±5.87

* $p < 0.05$, ** $p<0.01$, ***$p<0.001$ vs controls.

Table 3. Changes in blood uric acid and blood creatinine in laying hens – healthy (controls), hens with spontaneous and experimental alopecia, by months 1 and 2 after the appearance of clinical symptoms (mean ±SEM; n=10)

Groups	Spontaneous alopecia		Experimental alopecia[1]	
	Uric acid (mmol/L)	Creatinine (mmol/L)	Uric acid (mmol/L)	Creatinine (mmol/L)
Month 1				
Control	500.60±19.20	46.02±1.50	440.13±95.97	51.16±5.55
Experimental	631.60±33.82**	44.20±0.25	793.68±52.31**	53.18±3.57
Month 2				
Control	512.50±26.15	45.60±1.24	419.53±87.88	48.34±4.37
Experimental	560.30±23.46	45.40±1.13	589.86±51.54	50.26±3.93

[1] following treatment with thyroid gland tissue; ** $p<0.01$vs controls.

vated ($p<0.01$ vs controls) whereas ABE and SBE were lowered ($p<0.05$ vs controls). During the second month, weak variations of studied parameters within the reference ranges were observed.

In birds with experimental alopecia (Table 2), blood pH, ABE and SBE were statistically significantly lower than controls during the first month. A significant increase in pCO_2, HCO_3^-, TCO_2 and SBC also was present. A tendency towards insignificant decrease in pO_2 and considerable elevation of haemoglobin content was noticed.

During the second month, no substantial changes in studied acid–base parameters occurred.

Blood uric acid levels in birds with both SA and EA were increased during the first month ($p<0.01$ vs controls) (Table 3).

The respiratory rate in SA hens was slightly enhanced vs controls (35±2.57 min^{-1} and 24.4 ± 2.38 min^{-1} respectively). Similar changes were observed with EA birds – 42.3 ± 3.78 min^{-1} ($p<0.05$) vs controls (26.2 ± 2.52 min^{-1}). By the second month, respiratory rates in SA and EA birds were similar to those in controls.

DISCUSSION

The results showed that in SA birds, a compensated acidosis occurred by the end of the 1st month (normal pH and decreased ABE and SBE). It could hardly be defined as metabolic or respiratory by origin, because there are compensatory mechanisms as enhanced respiratory rates, that would probably resulted in normalizng of pCO_2 and pH at the moment of sampling by the end of the second month. Probably, the increase in bicarbonates was also a compensatory manifestation (Iluchev and Kostyanev, 1998).

The data obtained from birds with EA showed that by the 1st month, a decompensated metabolic acidosis with hypercapnia was present (mixed acidosis),

manifested by statistically significant decrease in pH, ABE and SBE and increased pCO_2, HCO_3^-, TCO_2 and SBC.

The elevated pO_2 values in EA hens could be attributed to the increased bicabonate levels. A similar correlation between those acid-base parameters is reported by Stocker *et al.* (1999). The higher pCO_2 could be however due to pulmonary hypoventilation, resulting in hypercapnia and a trend towards decrease in pO_2 that probably reflected in a compensatory increase in haemoglobin, observed by us at the end of the 1st month.

The decrease in ABE and SBE could be explained by the prevailing of anaerobic pathway of glucose degradation and the formation of acidic metabolites (Bardos and Mezes, 1980).

According to some authors (Hartmann *et al.*, 1997), the impaired protein and amino-acid metabolism has also an impact on blood actual base excess. ABE and SBE changes could be further influenced by the amount of both standard and actual bicarbonates acting as buffer mechanism of hydrogen ions neutralization (Goundasheva, 2000).

The lack of changes in blood creatinine concentrations in both diseased groups indicated that the regulatory function of kidneys on acid-base status was normal. The increased uric acid concentrations were probably due to increased thyroid gland function (Nobukuni *et al.*, 1991; Machal and Jerabek, 2000).

CONCLUSIONS

The spontaneous alopecia in hens was accompanied by compensated acidosis that is manifected one month after its appearance.

The induction of experimental alopecia in hens via treatment with dried thyroid gland tissue from calves resulted in decompensated mixed acidosis by the 1st post treatment month.

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