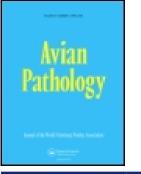


Avian Pathology



ISSN: 0307-9457 (Print) 1465-3338 (Online) Journal homepage: www.tandfonline.com/journals/cavp20

Quantitative comparisons of lung structure of adult domestic fowl and red jungle fowl, with reference to broiler ascites

M.K. Vidyadaran, A.S. King & H. Kassim

To cite this article: M.K. Vidyadaran, A.S. King & H. Kassim (1990) Quantitative comparisons of lung structure of adult domestic fowl and red jungle fowl, with reference to broiler ascites, Avian Pathology, 19:1, 51-58, DOI: 10.1080/03079459008418655

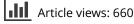
To link to this article: https://doi.org/10.1080/03079459008418655



Published online: 12 Nov 2007.



Submit your article to this journal 🕑



View related articles



Citing articles: 2 View citing articles 🗹

QUANTITATIVE COMPARISONS OF LUNG STRUCTURE OF ADULT DOMESTIC FOWL AND RED JUNGLE FOWL, WITH REFERENCE TO BROILER ASCITES

M.K. VIDYADARAN¹, A.S. KING² and H. KASSIM¹

¹Department of Animal Sciences, Universiti Pertanian, Serdang, Selangor, Malavsia

²Department of Preclinical Veterinary Sciences, University of Liverpool, P.O. Box 147, Liverpool L69 3BX, England

SUMMARY

A stereological comparison has been made of the structure of the lungs of the adult female domestic fowl and its wild progenitor the Red Jungle Fowl. The volume of the lung per unit body weight of the domestic bird is between 20 and 33% smaller than that of the wild bird. The domestic fowl has partly compensated for this by increasing the surface area for gas exchange per unit volume of exchange tissue. However, the blood-gas tissue barrier is about 28% thicker in the domestic fowl than in the Red Jungle Fowl, and this has led to a 25% lower anatomical diffusing capacity for oxygen of the blood-gas tissue barrier per unit body weight in the domestic fowl vulnerable to stress factors such as altitude, cold, heat or air pollution by predisposing to hypoxaemia and perhaps thence to ascites.

INTRODUCTION

Although the qualitative characteristics of the anatomy of the avian lung have been progressively elucidated during the past 100 years (King, 1966; Duncker, 1971), quantitative (stereological) observations have been relatively few (Duncker, 1972, 1973; Abdalla, 1977; Abdalla and Maina, 1981; Dubach, 1981; Maina, 1982, 1984, 1989; Maina and King, 1982a,b; Abdalla *et al.*, 1982; Maina *et al.*, 1982; Powell and Mazzone, 1983; Timmwood and Julian, 1983; Vidyadaran, 1986; Timmwood *et al.*, 1987a,b). These studies have yielded comprehensive pulmonary stereological data for about 26 avian species representing more than 10 orders. When these stereological values are plotted on allometric regression lines relative to body mass for birds in general, all the parameters for gas exchange of the domestic fowl are conspicuously inferior to those of all non-galliform birds (Maina and Settle, 1982; Maina, 1982; Maina, 1982, 1989; Maina *et al.*, 1989). In particular, the anatomical diffusing capacity of the tissue barrier for the

Received 6 March² 1989 Accepted 26 April 1989

uptake of oxygen (which expresses the full anatomical potential of the tissue barrier of a lung for gas exchange, assuming perfect ventilation and blood perfusion) is from 1.5 to 35 times higher in non-galliform birds than in the domestic fowl, when standardised against body weight.

Archaeological and palaeoclimatic evidence indicates that the domestic fowl originated in SE Asia and from the Red Jungle Fowl (Gallus gallus) about 8000 years ago (West and Zhou, 1988). The gas exchange structures of the wild progenitor were presumably adapted by natural selection to meet the animal's energetic requirements. On the other hand the domestic descendant has been intensively subjected to artificial selection for meat and eggs. For instance, it took 70 days for a table bird to achieve a live weight of 1815 g in 1960 and only 40 days in 1985 (Smith, 1985). Coinciding with genetic and nutritional changes there has been an increasing and world-wide incidence of ascites in young broilers (Julian and Wilson, 1986; Julian, 1987) in which Maxwell et al. (1986a,b) have found widespread pathological changes closely resembling those found in hypoxic birds. Julian and Wilson (1986) and Huchzermeyer and De Ruyck (1986) have reported an association between the ascites and right ventricular hypertrophy. In a review of the possible pathogenesis of the ascites, Huchzermeyer (1986) suggested that hypoxia causes pulmonary vasoconstriction, with consequent pulmonary hypertension and right ventricular hypertrophy. All broiler flocks contain both genetically susceptible and resistant birds, the susceptible birds undergoing exaggerated pulmonary vasoconstriction. The initial cause of the hypoxia remains unknown.

These events in the poultry industry, and particularly the evidence of serious problems of hypoxia, caused us to return to Maina's (1982) discovery that the morphometric parameters of the lung of the domestic fowl are remarkably out-of-line with those of birds in general. To examine this further, we have investigated in the posthatching chick (Vidyadaran *et al.*, 1987) and in the adult female (this paper) the quantitative anatomy of the lung of the Red Jungle Fowl, and compared the pulmonary stereological characteristics of this wild form of *Gallus gallus* with those of its domestic relative.

MATERIALS AND METHODS

Five female domestic fowls of a commercial layer strain (Euribred Hisex Brown) were obtained from a poultry farm in England, and five female Euribred Hisex Brown were also bought from a Malaysian poultry farm. All the birds were in their first year of lay. The English birds were subjected to a comprehensive stereological pulmonary examination, and the Malaysian birds were investigated for lung volume only.

Five adult female wild Red Jungle Fowl were trapped at night in the jungle, transported to the laboratory on the following morning, and kept on free range during that day with access to water and standard commercial chicken feed. They were starved overnight, though water was provided, and killed on the next morning. They were then exposed to the same stereological pulmonary survey as the English domestic fowl. All of the Red Jungle Fowl had functionally active ovaries with large follicles.

All birds were killed by an intraperitoneal injection of sodium pentobarbitone. The techniques of fixation of the lung, processing for light and electron microscopy under conditions of controlled osmolarity, multistage sampling with uniform randomness, and stereological analysis were the same as those described by Abdalla *et al.* (1982) and Maina and King (1982b). For the reasons given by Abdalla *et al.* (1982) and Maina and King (1982a,b), it is believed that these procedures for fixation and subsequent processing and measurement of the lung and its component parts should yield estimates of volumes, areas and thicknesses which are likely to represent approximately the values in life.

The anatomical diffusing capacity of the blood-gas tissue pathways was estimated from the model of Weibel (1970/71). Thus

$$\frac{DtO_2 = St \times KtO_2}{\gamma ht}$$

where St is the surface area of the tissue barrier, γht is the harmonic mean thickness of the tissue barrier, and KtO_2 is a physical coefficient for the permeation of oxygen through tissue.

Symbols are defined in Table 1.

Table 1. Definition of symbols.

 $Dt\theta_2$ Anatomical diffusing capacity of the blood-gas (tissue) barrier for oxygen. St Surface area of the blood-gas (tissue) barrier. Harmonic mean thickness of the blood-gas (tissue) barrier. yht Volume of the lumen of the air capillaries. Va Volume of the lumen of the blood capillaries. Vc . VL Volume of the fixed lungs (left lung x 2). VxVolume of the exchange tissue of the lung. WBody weight.

Specific values are those standardised against body weight. For example, VL/W is the specific lung volume.

RESULTS

The mean body weights were 1.87 ± 0.35 kg for the English and 1.88 ± 0.25 kg for the Malaysian domestic fowl, and 0.48 ± 0.07 kg for the Red Jungle Fowl. The mean volumes of the left and right fixed lungs combined were 26.59 ± 2.10 cm³ in the English and 22.53 ± 3.72 cm³ in the Malaysian domestic fowl (the difference being not statistically significant) and 8.7 ± 1.91 cm³ in the Red Jungle Fowl. The volume proportion of exchange tissue in the lung (VX%) was similar in the English domestic fowl and Red Jungle Fowl, giving means of $49.66 \pm 0.88\%$ and $53.15 \pm 1.49\%$ respectively; because the lung was larger in the domestic fowl (13.22 ± 1.23 cm³ than in the Red Jungle Fowl (4.61 ± 0.88 cm³).

Further details of the stereological analysis of the five English domestic fowl and the five Red Jungle Fowl are summarised in Table 2. The weight-specific volume of the

M.K. Vidyadaran et al.

Table 2. Summary of stereological observations on the lungs of the domestic fowl and Red Jungle Fowl, values being expressed as means ± SD. All birds were adult females. All values except for those for thickness pertain to the combined left and right fixed lungs.

Red Jungle Fowl		Domestic fowl	
L L	Measure		
	14.65 ± 3.17	18.10 ± 1.94	*
St/W (cm ² /g)	12.46 ± 1.96	12.97 ± 1.59	NS
$St/Vx (mm^2/mm^3)$	172.84 ± 12.45	135.17 ±13.54	**
Va/Vx (mm ³ /cm ³)	555.88 ±66.54	648.22 ± 48.25	**
Vc/Vx (mm ³ /cm ³)	279.08 ± 27.85	212.96 ±22.45	**
τht (µm)	0.322 ± 0.01	0.252 ± 0.02	**
DtO_2/W (ml02min ⁻¹ mmHg ⁻¹ kg ⁻¹)	12.79 ± 2.20	17.01 ± 1.95	**

* P<0.05; ** P<0.01; NS not significant.

lung (*i.e.* the volume of the lung standardised against body weight, VL/W) of the Red Jungle Fowl was about 24% greater than that of the domestic fowl from England and about 52% greater than that of the Malaysian domestic fowl. Thus the domestic fowl suffers the immediate disadvantage of having a substantially smaller lung volume per unit body weight than its wild ancestor.

However, the weight-specific surface area of the blood-gas tissue barrier (*St/W*) was almost the same in the domestic fowl and the Red Jungle Fowl. This is consistent with the ratio of the surface area of the tissue barrier to the volume of exchange tissue (*St/Vx*), which was 28% higher in the domestic fowl than in the Red Jungle Fowl (Table 2). Therefore the domestic fowl has partly compensated for its relatively small lung by packing in almost the same surface area of tissue for gas exchange as there is in the lung of the Red Jungle Fowl, but in a much smaller volume of exchange tissue. However, this has entailed changes in the relative proportions of air and blood in the exchange tissue. Thus each unit volume of exchange tissue contained a significantly smaller proportion of air (*Va/Vx*) and a larger proportion of blood (*Vc/Vx*) in the domestic fowl than in the Red Jungle Fowl (Table 2).

The harmonic mean thickness of the blood-gas (tissue) (γht) (Table 2) was about 28% thicker in the domestic fowl than in the Red Jungle Fowl. The weight-specific anatomical diffusing capacity of the tissue barrier for oxygen (DtO_2/W) was 25% lower in the domestic fowl than in the adult Red Jungle Fowl (Table 2). Since DtO_2 is derived from the surface area of the tissue barrier (St) and the harmonic mean thickness of the tissue barrier in the domestic fowl accounts for the relatively low DtO_2 .

DISCUSSION

Several of the quantitative anatomical parameters of the lungs of the domestic fowl are substantially different from those of its wild ancestor. Firstly, the weight-specific volume of the lung (VL/W) is between 20 and 33% smaller in the domestic fowl than in the Red Jungle Fowl. A relatively small weight-specific lung volume has been found by Timmwood and Julian (1983) in certain fast-growing strains of turkey which are also

subject to ascites. They suggested that in these strains of turkey the oedema, an insufficient capacity to respond to stress, and susceptibility to aspergillosis, could perhaps be attributed to the inadequate matching of the volume of the lung to the excessive body weight. The lung of the domestic fowl appears to have partly compensated for its relatively low weight-specific volume (VL/W) by packing in a weight-specific surface area for gas exchange (St/W) which is similar to that of the Red Jungle Fowl, although this has entailed changes in the relative proportions of air and blood in the exchange tissue (Va/Vx and Vc/Vx). In contrast Timmwood *et al.* (1987b) found that the lung of a genetically highly selected line of turkey had not so compensated for a low weight-specific volume, but had a smaller surface area for gas exchange per unit body weight (St/W) than the lung of the unselected line.

Secondly, we found the harmonic mean thickness of the blood-gas tissue barrier (γht) of the domestic fowl to be 0.322 µm, and this is about 28% thicker than that of the Red Jungle Fowl. The thickness of the tissue barrier is the single most important structural determinant of oxygen uptake, and this extra thickness therefore has a pronounced adverse effect on the anatomical diffusing capacity for oxygen of the tissue barrier. Theoretically such a thickening could be of pathological origin. However, Vidyadaran *et al.* (1988) have shown that almost the same thickness of the tissue barrier of the domestic fowl was reported by Duncker (1973), Dubach (1981), Abdalla *et al.* (1982) and Maina (1982), who gave values of 0.346, 0.346, 0.314 and 0.318 µm respectively, and it appears unlikely that four other groups of workers would all have encountered blood-gas tissue barriers with such closely similar pathological thickening. Therefore it seems that this thickness of the barrier in the domestic fowl must be genetically determined.

Thirdly, the weight-specific anatomical diffusing capacity of the blood-gas tissue barrier (DtO_2/W) is 25% lower in the adult domestic fowl than in the Red Jungle Fowl. This is due to two factors, *i.e.* the relatively thicker barrier and the greater body weight of the domestic fowl.

The hazards of the increased growth rate of the modern domestic fowl are shown by the particular vulnerability to ascites of the fastest growing broilers (Julian and Wilson, 1986) and by the observation that reducing the growth rate by feeding mash instead of pellets or crumbs can reduce the incidence of ascites in a flock (Huchzermeyer, 1986). Julian and Wilson (1986) suggested that the high oxygen demand of this rapid growth may be the most important factor in broiler ascites.

The artificial selection of poultry for earlier maturation and greater body weight has taken place in the absence of natural selection for respiratory adequacy. Statements in the literature on animal production sometimes reveal an alarming dedication to achieving the maximum genetic "improvement", including "the removal of *all environmental constraints* which normally limit the expression of genetic potential" (Wilson, 1973). In the absence of environmental challenge, adverse pulmonary mutations might not be noticed at first. However, could there come a point when the margin of reserve for gas exchange could be overreached by stress factors such as cold, altitude, air pollution, social pressures, handling, or local airway obstruction, thus predisposing to hypoxaemia and possibly thence to ascites? Timmwood *et al.* (1987b) raised a similar question about genetic lines of turkeys selected for early rapid growth and enlargement of the pectoral muscles; are the sudden deaths of such turkeys at 30 weeks of age during handling attributable to insufficient capacity for gas exchange?

M.K. Vidyadaran et al.

Attempts have been made to measure the metabolic rate of the domestic fowl whilst running on a treadmill. After "preliminary training" followed by further training for 10 weeks, cockerels were able to achieve a maximum of about 9 km/h in 10 to 12 min runs; of eleven females subjected to preliminary training only one went on to further training for 10 weeks, and was then able to move in excess of about 5 km/h for only 2 to 3 min (Brackenbury and Avery, 1980). After further similar experiments on six female domestic fowl in breeding condition raised on free range (Brackenbury *et al.*, 1981), concluded that "hens are only just capable of running, or are extremely unwilling to run at faster speeds". The aerobic capacity of the males would be just sufficient to sustain level flight in a bird of the same body weight (Brackenbury, 1984), but these were "highly trained" birds; moreover this would leave nothing in reserve for take-off and climbing (Brackenbury and Avery, 1980). The impression that emerges is that the modern domestic fowl is not an athletic animal. In contrast, wild female Red Jungle Fowl are capable of strenuous exercise; in Malaysia they can be observed to fly for about 30 m at a height of about 6 m and then scuttle rapidly into the bush.

Our stereological comparisons of the lung of the highly bred domestic fowl with that of its wild progenitor do not prove that anatomical deficiencies in the lung are the cause of ascites, but perhaps they suggest that the modern domestic form has been 'overengineered' to a point closely approaching pulmonary inadequacy, thus predisposing to ascites. It is interesting that "village fowls" are reported to be completely resistant to ascites (Pizarro *et al.*, 1970). Huchzermeyer *et al.* (1988) concluded that the varying degrees of susceptibility to ascites in different commercial broiler strains "can only be explained as genetic", and that "the practical solution of the broiler ascites problem therefore rests with the selection for resistance to ascites in broiler stock". Such selection may include selection for the pulmonary characteristics of the more primitive forms of *Gallus gallus*.

Acknowledgements

We thank the British Council and Universiti Pertanian Malaysia for making our collaboration possible. We are grateful to Mr G. Settle of the University of Liverpool and Dr Mak Tian Kuan of the Universiti Pertanian Malaysia for statistical advice, to Mr P. Ganesamurthi and Mr Rosley bur Sidek for technical assistance and Mrs Shamala for typing the manuscript. M.K. Vidyadaran is especially grateful to Dr J.N. Maina, University of Nairobi, for advice on stereological techniques.

REFERENCES

Abdalla, M.A. (1977). Morphometry of the avian lung. Journal of Anatomy, 123: 262.

Abdalla, M.A. and Maina, J.N. (1981). Quantitative analysis of the exchange tissue of the avian lung. Journal of Anatomy, 133: 677-680.

Abdalla, M.A., Maina, J.N., King, A.S., King, D.Z. and Henry, J. (1982). Morphometrics of the avian lung. I. The domestic fowl (Gallus gallus variant domesticus). Respiration Physiology, 47: 267-278.

Brackenbury, J. (1984). Physiological responses of birds to flight and running. Biological Reviews, 59: 559-575.

Brackenbury, J. and Avery, P. (1980). Energy consumption and ventilatory mechanisms in the domestic fowl. Comparative Biochemistry and Physiology, 66A: 439-445.

Brackenbury, J., Avery, P. and Gleeson, M. (1981). Respiration in exercising fowl. I. Oxygen consumption, respiratory rate and respired gases. Journal of Experimental Biology, 93: 317-325.

Dubach, M. (1981). Quantitative analysis of the respiratory system of the House Sparrow, Budgerigar and Violet-eared Hummingbird. Respiration Physiology, 46: 43-60.

Duncker, H.-R. (1971). The lung air sac system of birds. A contribution to the functional anatomy of the respiratory apparatus. Ergebnisse der Anatomie, 45: 1-171.

Duncker, H.-R. (1972). Structure of avian lungs. Respiration Physiology. 14: 44-63.

- Duncker, H.-R. (1973). Der quantitative Aufbau des Lungen-Luftsacksystem der Vögel. Verhandlung anatomischen Gesellschaft, 67: 194-204.
- Huchzermeyer, F.W. (1986). Causes and prevention of broiler ascites. S.A.P.A. Poultry Bulletin, 346.
- Huchzermeyer, F.W., and De Ruyck, A.M.C. (1986). Pulmonary hypertension syndrome associated with ascites in broilers. The Veterinary Record, 119: 94.
- Huchzermeyer, F.W., De Ruyck, A.M.C. and Van Ark, H. (1988). Broiler pulmonary hypertension syndrome. III. Commercial broiler strains differ in their susceptibility. Onderstepoort Journal of Veterinary Research, 55, 5-9.
- 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 Pathology, 16: 61-71.
- Julian, R.J. and Wilson, J.B. (1986). Right ventricular failure as a cause of ascites in broiler and roaster chickens. Proceedings of the 1Vth International Symposium of Veterinary Laboratory Diagnosticians, pp. 608-611.
- King, A.S. (1966). Structural and functional aspects of the avian lung and air sacs. International Review of General and Experimental Zoology, 2: 171-267.
- Maina, J.N. (1982). Qualitative and quantitative observations on the lungs of Aves with comments on the lung of a species of Chiroptera: A morphological study. Ph.D. Thesis, University of Liverpool, England.
- Maina, J.N. (1984). Morphometrics of the avian lung. 3. The structural design of the passerine lung. Respiration Physiology, 55: 291-307.
- Maina, J.N. (1989). The morphometry of the avian lung. In: Form and Function in Birds, 4 pp. 369-391. Edited by King, A.S. and McLelland, J. London: Academic Press.
- Maina, J.N. and King, A.S. (1982a). The thickness of the avian blood-gas barrier: qualitative and quantitative observations. Journal of Anatomy, 134: 553-562.
- Maina, J.N. and King, A.S. (1982b). Morphometrics of the avian lung. 2. The wild mallard (Anas platyrhynchos) and graylag goose (Anser anser). Respiration Physiology. 50: 299-310.
- Maina, J.N. and Settle, J.G. (1982). Allometric comparisons of avian and mammalian lungs. Journal of Physiology, 330: 28P.
- Maina, J.N., Abdalla, M.A. and King, A.S. (1982). Light microscopic morphometry of the lung of 19 avian species. Acta Anatomica, 112: 264-270.
- Maina, J.N., King, A.S. and Settle, J.G. (1989). An allometric study of pulmonary morphometric parameters in birds, with mammalian comparisons. *Philosophical Transactions of the Royal Society. Series B, in press.*
- Maxwell, M.H., Robertson, G.W. and Spence, S. (1986a). Studies on an ascitic syndrome in young broilers. 1. Haematology and pathology. Avian Pathology, 15: 511-524.
- Maxwell, M.H., Robertson, G.W. and Spence, S. (1986b). Studies on an ascitic syndrome in young broilers. 2. Ultrastructure. Avian Pathology, 15: 525-538.
- Pizarro, B., Salas, A. and Paredes, J. (1970). Mal de altura en aves. Instituto Veterinario de Investigaciones Tropicales y de Altura, Cuarto Boletin Extraordinario. 147-151. Cited by Huchzermeyer et al. (1988).
- Powell, F.L. and Mazzone, R.W. (1983). Morphometrics of rapidly frozen goose lungs. Respiration Physiology, 51: 319-332.
- Smith, J.H. (1985). Breeders must respond to market trends. Poultry: Misset International, January, 34.
- Timmwood, K.I. and Julian, L.M. (1983). Early lung growth in the turkey. Proceedings of the 32nd Western Poultry Disease Conference, Davies, California, 21-23.
- *Timmwood, K.I., Hyde, D.M. and Plopper, C.G. (1987a).* Lung growth of the turkey, *Meleagris gallopavo:* 1. Morphologic and morphometric description. *American Journal of Anatomy, 178: 144-157.*
- Timmwood, K.I., Hyde, D.M. and Plopper, C.G. (1987b). Lung growth of the turkey, Meleagris gallopavo: II. Comparison of two genetic lines. American Journal of Anatomy, 178: 158-169.
- Vidyadaran, M.K. (1986). Quantitative observations on the pulmonary anatomy of the domestic fowl and other ground dwelling birds. Ph.D. Thesis, Universiti Pertanian, Malaysia.
- Vidyadaran, M.K., King, A.S. and Kassim, H. (1987). Deficient anatomical capacity for oxygen uptake of the developing lung of the female domestic fowl when compared with the Red Jungle Fowl. Schweizer Archiv für Tierheilkunde, 129: 225-237.
- Vidyadaran, M.K., King, A.S. and Kassim, H. (1988). Quantitative studies of the lung of the domestic fowl (Gallus gallus var. domesticus). Pertanika, 11: 229-238.
- Weibel, E.R. (1970/71). Morphometric estimation of pulmonary diffusion capacity. I. Model and method. Respiration Physiology, 11: 54-75.
- West, B. and Zhou, B.-X. (1988). Did chickens go north? New evidence for domestication. Journal of Archaeological Science, 15: 515-533.
- Wilson, P.N. (1973). Livestock physiology and nutrition. Philosophical Transactions of the Royal Society, 267: 101-112.

RESUME

Comparaisons quantitatives de la structure du poumon de la volaille domestique adulte et de la volaille rouge sauvage en référence avec les ascites du poulet de chair

Une comparaison stéréologique a été faite sur la structure du poumon de la volaille femelle domestique adulte et de son ancêtre la volaille rouge sauvage. Le volume du poumon par unité de poids du corps de la volaille domestique est entre 20 et 33% plus petit que celui de l'oiseau sauvage. La volaille domestique a partiellement compensé cette diminution en augmentant la surface d'échange des gaz par unité de volume de tissu d'échange. Cependant la barrière de tissu sang-gaz est d'environ 28% plus épaisse chez la volaille domestique que chez la volaille rouge sauvage et ceci a conduit à une capacité anatomique de diffusion 25% moindre pour l'oxygène de la barrière de tissu sang-gaz par unité de poids du corps chez la volaille domestique. Ces caractéristiques structurales peuvent rendre la volaille domestique moderne vulnérable aux facteurs de stress tels que: l'altitude, le froid, le chaud ou la pollution de l'air en prédisposant à l'hypoxie et peut-être aux ascites.

ZUSAMMENFASSUNG

Quantitative Vergleiche der Lungenstruktur von erwachsenen Haushühnern und dem Roten Dschungelhuhn unter Bezug auf die Broilerascites

Ein serologischer Vergleich wurde zwischen der Lungenstruktur des erwachsenen weiblichen Haushuhnes und seinem wilden Vorfahren, dem Roten Dschungelhuhn angestellt. Das Lungenvolumen pro Körpergewichtseinheit des Haushuhnes ist zwischen 20 bis 33% kleiner als das des Wildvogels. Das Haushuhn hat dies teilweise durch Vermehrung der Gasaustauschoberfläche pro Volumeneinheit des Austauschgewebes kompensiert. Doch ist die Blut-Gasgewebeschranke beim Haushuhn zu einer 25% dicker als beim Roten Dschungelhuhn und dies hat beim Haushuhn zu einer 25% niederen anatomischen Diffusionskapazität für Sauerstoff bei der Blut-Gasgewebeschranke pro Einheit Körpergewicht geführt. Diese Struktureigenschaften können wahrscheinlich das moderne Haushuhn verwundbar gegen Stressfaktoren wie Höhe, Kälte. Hitze oder Luftverunreinigung und anfällig für Hypoxaemie und daher vielleicht auch für Ascites machen.

RESUMEN

Estudio comparativo cuantitativo de la estructura del pulmon de la gallina doméstica adulta y de la gallina red jungle, en relación a la ascitis de los broilers

Se realizó una comparición estereológica de la estructura de los pulmones de la gallina doméstica adulta y de su progenitor salvaje. la gallina Red Jungle. El volumen pulmonar por unidad de peso corporal del ave doméstica fue de un 20 a un 33% menor que el del ave salvaje. La gallina doméstica compensa parcialmente esta situación incrementando la superficie para el intercambio gaseoso por unidad de volumen de tejido intercambiado. No obstante, la barrera tisular aire-sangre es aproximadamente un 28% más gruesa en las aves domésticas que en las salvajes, llevando este hecho a una dismunición de la capacidad de difusión del oxígeno en la barrera aire-sangre de un 25% por unidad de peso en las aves domésticas. Estas características estructurales pueden hacer a la moderna gallina doméstica vulnerable a los factores del strés tales como la altitud, el frio, el calor o la polución del aire, predisponiéndola a una hipoxemia y quizás a una ascitis.