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Comparisons of lung structure of fully developed domestic fowl with duck, through orientation to broiler ascites

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ABSTRACT

This comparative study of the structure in lungs of the fully developed female domestic fowl and the duck. In the avian lung gas exchanging region, while proportionally smaller than mammalian lung, competently manages respiration to meet the high active necessities of flapping flight. The domestic fowl has partially remunerated for this by raising the surface area for gas exchange per unit volume of exchange tissue. . In domestic bird the size of the lung per unit body weight is between 20 and 33% lesser than that of the wild bird.

The blood-gas tissue barrier is about 28% thicker in the domestic fowl than in the duck, and this has led to a 25% lower anatomical diffusing ability for oxygen of the blood-gas tissue barrier per unit body weight in the domestic fowl. At hatching (day 21), with a thin blood-gas barrier and a large respiratory surface area, the lung is well ready for gas exchange. Gas exchange in the bird lung is better, in part, by an extremely thin blood-gas barrier. These structural characteristics may make the modern domestic fowl weak to stress factors such as elevation, cold, heat or air pollution by predisposing to hypoxaemia.

Key words: Lung, Domestic Fowl, Duck, Broiler.

INTRODUCTION

Although the qualitative characteristics of the anatomy of the avian lung have been progressively elucidated during the past 100 years[1], quantitative (stereological) observations have been relatively few[2-9]. 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[10-12]. In particular, the anatomical diffusing capacity of the tissue barrier for the 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 standardized against body weight. All horses bred and trained for competitive racing show evidence of exercise-induced pulmonary hemorrhage (EIPH), which manifests as bleeding from the nose after exertion or the presence of red blood cells or hemosiderophages in the airways[13-15].

Archaeological and palaeoclimatic evidence indicates that the domestic fowl originated in SE Asia and from the Duck (*Anas platyrhynchos*) about 8000 years ago[22]. 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[16]. Coinciding with genetic and nutritional changes there has been an increasing and world-wide incidence of ascites in young broilers[17-18] in which have found widespread pathological changes closely resembling those found in hypoxic birds[19, 20]. They have reported an association between the ascites and right ventricular hypertrophy[21-24]. In a review of the possible pathogenesis of the ascites[25] 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[26] 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[27] and in the adult female the quantitative anatomy of the lung of the Duck, and compared the pulmonary stereological characteristics of this wild form *of Gallus gallus* with those of its domestic relative. The diameter of the air capillaries in an avian lung is a fraction of the size of the mammalian alveoli, the resultant surface tension forces in the air capillaries may be very strong and patency is ensured by the presence of a unique surfactant-like material[28, 29, 30].

MATERIAL AND METHODS

Ten female domestic fowls of a commercial layer strain were obtained from a poultry farm in Uttar Pradesh and five female were also bought from a near Madhya Pradesh. All the birds were in their first year of lay. The Uttar Pradesh birds were subjected to a comprehensive stereological pulmonary examination, and the Madhya Pradesh birds were investigated for lung volume only.

Five adult Duck were kept in cage, 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 Uttar Pradesh domestic fowl. All of the Duck 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[31, 32]. For the reasons given by and 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 [33]. Thus

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

where *St* is the surface area of the tissue barrier, *yht* 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.

RESULTS

The mean body weights were 1.87 ± 0.35 kg for the Uttar Pradesh and 1.88 ± 0.25 kg for the Madhya Pradesh domestic fowl, and 0.48 ± 0.07 kg for the Duck. The mean volumes of the left and right fixed lungs combined were 26.59 ± 2.10 cm^3 in the Uttar Pradesh and 22.53 ± 3.72 cm^3 in the Madhya Pradesh domestic fowl (the difference being not statistically significant) and 8.7 ± 1.91 cm^3 in the Duck. The volume proportion of exchange tissue in the lung (VX%) was similar in the Uttar Pradesh domestic fowl and Duck, giving means of 49.66 ± 0.88% and 53.15 ± 1.49% respectively; because the lung was larger in the domestic fowl, the absolute volume of the exchange tissue (VX) was larger in the domestic fowl (13.22 ± 1.23 cm^3 than in the Duck (4.61 ± 0.88 cm^3).

Further details of the stereological analysis of the five Uttar Pradesh domestic fowl and the five Duck are summarized in Table 2. The weight-specific volume of the lung *(i.e.* the volume of the lung standardized against body weight, VL/W) of the Duck was about 24% greater than that of the domestic fowl from Uttar Pradesh and about 52% greater than that of the Madhya Pradesh domestic

Table 1: Definition of symbols

S.No.	Symbols	Description
1	DtO_2	Anatomical diffusing capacity of the blood-gas (tissue) barrier for oxygen
2	St	Surface area of the blood-gas (tissue) barrier
3	γht	Harmonic mean thickness of the blood-gas (tissue) barrier
4	Va	Volume of the lumen of the air capillaries
5	Vc	Volume of the lumen of the blood capillaries
6	VL	Volume of the fixed lungs (left lung x 2)
7	Vx	Volume of the exchange tissue of the lung
8	W	Body weight

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

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 Duck. This is consistent with the ratio of the surface area of the tissue barrier to the volume of exchange tissue *(St/Vx).* This was 28% higher in the domestic fowl than in the Duck (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 Duck, 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 Duck (Table 2).

Table 2 Summary of stereological observations on the lungs of the domestic fowl and Duck, 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

S.No.	Duck	Measure	Domestic fowl	
1	VL/W (mm^3/g)	14.65 ± 3.17	18.10 ± 1.94	*
2	St/W (cm^2/g)	12.46 ± 1.96	12.97 ± 1.59	NS
3	St/Vx ($mm^2/mm3$)	172.84 ±12.45	135.17 ±13.54	**
4	Va/Vx ($mm^3/cm3$)	555.88 ±66.54	648.22 ±48.25	**
5	Vc/Vx ($mm^3/cm3$)	279.08 ±27.85	212.96 ±22.45	**
6	τht (μm)	0.322± 0.01	0.252± 0.02	**
7	$DtO_2/W(mlO_2min^{-1}mmHg^{-1}kg^{-1})$	12.79 ± 2.20	17.01 ± 1.95	**

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

The harmonic mean thickness of the blood-gas (tissue) *(yht)* (Table 2) was about 28% thicker in the domestic fowl than in the Duck. 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 Duck (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 *(yht).* The greater thickness of the 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 Duck. A relatively small weight-specific lung volume has been found by[34] 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 duck, although this has entailed changes in the relative proportions of air and blood in the exchange tissue *(Va/Vx* and *Vc/Vx).* In contrast[35] 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 *(yht)* of the domestic fowl to be 0.322 µm, and this is about 28% thicker than that of the duck. Thickness of the pulmonary blood-gas barrier (BGB) is affected by opposing selective pressures. The barrier thickness is minimized to allow efficient exchange of oxygen and carbon dioxide, yet the capillary walls must also be strong enough to withstand high pulmonary capillary pressures that are incurred during extreme physical exertion.

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, have shown that almost the same thickness of the tissue barrier of the domestic fowl was reported by 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[36, 37]. 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 Duck. 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[38] 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[39]. Suggested that the high oxygen demand of this rapid growth may be the most important factor in broiler ascites[40].

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 which normally limit the expression of genetic potential". 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. 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. 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 48 After further similar experiments on six female domestic fowl in breeding condition raised on free range[41], 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[42], but these were "highly trained" birds; moreover this would leave nothing in reserve for take-off and climbing[43].The impression that emerges is that the modern domestic fowl. In duck are capable of strenuous

exercise; in Madhya Pradesh 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. A similar process seems likely in birds, although avian lymphatics are poorly[44, 45]. It is interesting that "village fowls" are reported to be completely resistant to ascites. 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.*

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