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AVIAN PULMONARY HYPERTENSION SYNDROME. IV. INCREASED RIGHT VENTRICULAR MASS IN TURKEYS EXPERIMENTALLY INFECTED WITH *PLASMODIUM DURAE*

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ABSTRACT

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Infection of young turkeys with *Plasmodium durae* produced a significant increase in the mean relative right ventricular mass from 0,172 in the controls to 0,208 in the infected group.

Although turkeys react less strongly to hypoxia than broiler chickens, the similarity of this finding to that previously reported in broilers infected with *Aegyptianella pullorum* suggests that the hypoxic effect of the anaemia produced by these blood parasites causes the pulmonary hypertension and subsequent right ventricular hypertrophy.

INTRODUCTION

Right ventricular (RV) hypertrophy, an essential feature in broiler ascites, has been described in broilers experimentally infected with *Aegyptianella pullorum* (Huchzermeyer, Cilliers, Diaz Lavigne & Bartkowiak, 1987). In order to elucidate the question of whether pulmonary hypertension and the consequent RV hypertrophy was caused by the hypoxic effect of anaemia or, alternatively, by a specific metabolic or biochemical action of the parasite, it was decided to investigate whether an infection with another unrelated avian blood parasite, *Plasmodium durae*, would have a similar effect. This parasite, several strains of which are maintained at present in the laboratory, was first described by Herman (1941) from an outbreak in turkeys in Kenya.

MATERIALS AND METHODS

Experimental animals and infection

Juvenile turkeys from the Institute's breeding flock were reared in isolation and infected intravenously or intramuscularly during routine passaging as well as during certain experiments unrelated to this work with different passages of 2 local isolates of *P. durae*, designated O and N, over a period of 12 months. The O strain is highly virulent and often causes death at the peak of parasitaemia, while the N strain rarely produces mortality but often a sustained high parasitaemia lasting several weeks. The uninfected controls originated from the same stock and were maintained under identical conditions.

Heart dissection

Either when the bird died or when it was sacrificed after the completion of the trial, the heart was removed and dissected for mass-measuring and for the calculation of the pulmonary arterial pressure index (API). This was done by dividing the right ventricular mass (RV) by the total ventricular mass (TV), i.e, API = RV:TV, as previously described (Huchzermeyer & De Ruyck, 1986).

Statistical analysis

Students' t test for the analysis of variates with unequal variance (Clarke, 1969) was used in the statistical analysis of the API values obtained.

RESULTS

Natural death of 46 turkeys occurred between 11 and 39 days after infection with *P. durae* with a mean of 17,1 days. A further 5 birds were sacrificed after the end of the trials, 20 to 41 days after infection and 2 to 25 days after the peak of parasitaemia.

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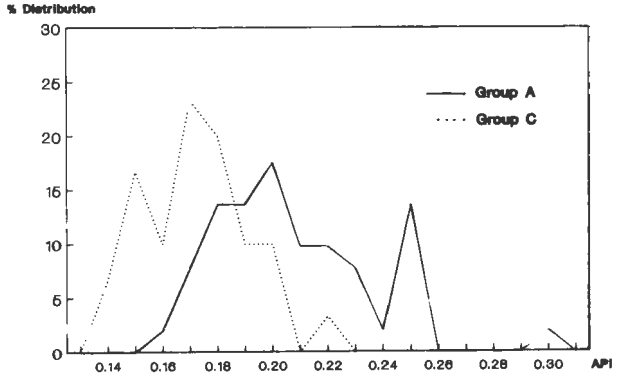

FIG. 1 Percentage distribution of API values of turkeys infected with *Plasmodium durae* (Group A) and of normal controls (Group C)

TABLE 1 API values of turkeys infected with *Plasmodium durae* (Group A) and of normal controls (Group C)

	Group A infected	Group C controls
$\bar{x}$	0,208	0,172
Range	0,16 – 0,30	0,14 – 0,22
s	0,0286	0,0209
n	51	30

The API values obtained from these birds as well as those from 30 controls are presented in Table 1. The distribution of the API values of the 2 groups is shown in Fig. 1. The mean API values of the groups are significantly different at the 5 % level.

DISCUSSION

The values obtained in both groups are considerably lower than those produced by aegyptianellosis in broilers (Huchzermeyer *et al.*, 1987). This is most probably due to the fact that the turkey is more resistant to hypoxia, as no cases of altitude disease in this species have ever been reported. The lower values in the infected birds can probably be attributed to the facts that *P. durae* does not cause as severe an anaemia as *Aegyptianella pullorum* (own unpublished data) and that, as already stated, in many birds death occurred at or shortly after the peak of

parasitaemia, not allowing time for the anaemia to develop and for the pulmonary vasculature and heart to react to it.

The similarity in reaction, however, points to a common mechanism involved in the pathogenesis of pulmonary hypertension and RV hypertrophy, which is most likely the hypoxic effect of the anaemia. This effect has not been reported previously by other sources.

It would be interesting to observe the effect of plasmodia pathogenic to the domestic fowl *(e.g. Plasmodium gallinaceum* or *Plasmodium juxtanucleare)* on broilers.

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