DEFICIENCY OF TRYPsin-INHIBITING CAPACITY IN
SERUM OF TURKIES WITH
ROUND HEART DISEASE

By

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INTRODUCTION

Round heart disease (RHD) in turkey (*Meleagris gallapavo*) poults was first described by Magwood and Bray (1962) in Canada. Although the clinical signs of RHD are not pathognomonic, the pathological changes are quite characteristic. Affected birds show dilatation of the right or both ventricles of the heart, enlarged liver with hardened discoloured parenchyma and ascites. Histologically, the liver contains intracytoplasmic globules (Neumann, Klopfer, Nobel, Dison and Bendheim, 1973) that react positively with antibodies against serum alpha globulins (Meirom, Trainin, Neumann, Klopfer, Dison and Rattner, 1975). These globules also react positively with the periodic-acid Schiff reagent technique, indicating the presence of glyco-proteins (Neumann et al., 1973). The serum of affected turkeys displays a marked reduction in total protein and a characteristic deficiency in alpha globulins (Meirom, Trainin, Barnea, Neumann, Klopfer, Nobel, Dison and Plesser, 1974).

As the presence of intracytoplasmic globules in the liver suggests a similarity to the condition of alpha 1-antitrypsin deficiency, a hereditary condition in man (Liebermann, Mittman and Gordon, 1972; DeLellis, Balogh, Merk and Cherife, 1972), we decided to investigate the trypsin inhibition capacity (TIC) in the serum of turkeys with RHD.

MATERIALS AND METHODS

The TIC assay was performed as described by Eriksson (1965). The decrease in activity of bovine pancreatic trypsin on the synthetic substrate benzoyl-d l-arginine-p-nitroanilide HCl (BAPNA) in the presence of a measured amount of test serum was determined, the results being expressed in mg. trypsin inhibited by 1 ml. of serum.

Total protein estimation of serum was carried out by the biuret method as described by Fey, Nicolet, Fellenberg and Margadant (1964).

RESULTS

In the first trial, sera from 19 Holland miniature turkey poults that had died with RHD during the first 10 weeks of life were tested. Control sera were obtained from 22 normal poults, aged 7 to 8 weeks, from the same flock. The mean TIC for the RHD group was 0.655 ± 0.071, and that for the control group 1.229 ± 0.049. The difference is highly significant (*P* < 0.001). A second trial was performed to confirm these results on living material. Twelve poults of
the Nicholas strain, aged 4 weeks, originated in a flock in which RHD occurred, were killed and blood samples were collected from the heart. Seven poults showed typical RHD lesions, while the other 5 birds did not show lesions and served as controls. The mean TIC of the affected birds was \(0.698 \pm 0.081\) and of the controls \(1.086 \pm 0.126\). The difference between the 2 groups is significant \((0.01 < P < 0.025)\). The range of TIC values in both diseased groups was wider than in the controls (Fig. 1).

\[\text{Fig. 1. Values of trypsin inhibiting capacity in 26 turkey poults with RHD and 27 control birds expressed as mg. trypsin inhibited by 1 ml. serum. (○) Holland miniature strain; (+) Nicholas strain.}\]

In the second trial the trypsin inhibition was related to the serum protein fraction. The results were \(0.0196\) mg. trypsin/mg. protein for the RHD poults compared with \(0.0246\) mg. trypsin/mg. protein for the controls.

**DISCUSSION**

These results suggest a relationship between TIC deficiency in turkeys and RHD. Such a deficiency may be hereditary and predispose the birds to the disease, while the disease itself is a result of interaction between the genetical determinant and environmental factors. The findings of Hunsaker (1971) are
in favour of the existence of genetic determinants in the etiology of RHD. If this is true, the working hypothesis of Neumann et al. (1973) concerning the similarity between RHD in turkeys and alpha 1-antitrypsin deficiency in man would be strengthened, and RHD in turkeys might act as a suitable model for the study of the disease in man. In addition, the existence of a spontaneous proteinase inhibitor deficiency in connection with pathological changes may enable a better understanding of the physiological role of these naturally occurring substances.

**SUMMARY**

The trypsin inhibition capacity in the sera of turkeys with round heart disease is significantly lower than in controls. This finding gives further support for the similarity between round heart disease of turkeys and alpha 1-antitrypsin deficiency in man.

**REFERENCES**


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