STUDIES OF THE IMMUNOLOGICAL RESPONSE OF PUPPIES, MONKEYS AND CHILDREN TO TOXOCARA CANIS (WERNER, 1782) INFECTION

A THESIS

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by

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resistant animals.

SUMMARY

The course of the naturally acquired prenatal infection of T. canis in puppies from their birth to 4 months of age was followed. The faecal Toxocara egg count trends, the antibody response and the Toxocara worm burden at post-mortem examination either on death or after being killed at 120 days of age in these puppies have been reported (Experiment 1).

A study of the effects of superinfection on puppies aged 4 to 6 weeks and naturally infected with Toxocara canis was made (Experiment 2 and 3).

The pattern of circulatory anti-Toxocara antibodies has been studied in these puppies superinfected with different doses and in those which received single and multiple doses of T. canis infective ova by means of conglutinating complement absorption test and Ouchterlony agar diffusion tests. Attempts have been made to correlate the serological reactions as measured by the conglutinating complement absorption test (C.C.A.T) with the Toxocara worm egg count trends and pathological effects of superinfection.

Puppies naturally infected with T. canis showed marked resistance to superinfection by doses of 5000 to 20,000 ova. But larger doses of 50,000 to 100,000 ova often resulted in the death of the puppies. It has been shown in this study that the immunity acquired from a natural infection can be reinforced by superinfection with a moderate dose of 10,000 infective ova to enable the animals to withstand a challenge with a large dose of 100,000 infective ova.

In the puppies superinfected with large doses of (50,000 to 100,000) ova most of the existing infection was eliminated by the self-cure phenomenon, however, this was not necessarily accompanied by resistance to the reinfection or to its effects. Acquired immunity to T. canis infection in puppies seems to be manifested at least in two ways: (i) by preventing the development of larvae produced by superinfection beyond the second-stage in the lungs and their migration to the intestine mainly to produce the fourth-stage and (ii) by enabling the host to withstand the pathological effects of superinfection which are fatal in non-immune puppies.

The pathological effect of a superinfection in non-

immune puppies is manifested by extensive necrosis of the hepatic parenchymal cells with concomitant increase of the serum glutamic pyruvate transaminase (SGP-T) activity which are less marked in the immune puppies.

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