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IMMUNOPATHOLOGICAL STUDIES OF TOXOCARA VITULORUM

IN BUFFALO CALVES AND RODENTS

by

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## SUMMARY

Studies on the migratory behaviour of Toxocara vitulorum in mice reveal that the larvae of this species undergo extensive somatic migration through the body (liver, lungs, kidneys etc) without undergoing a substantial degree of development beyond the second (infective) stage. On oral infection, larvae migrate to the liver by 8 hours. They migrate to the lungs, kidneys and uteri by 48 hours but the brain is reached by 96 hours. By the third and fourth weeks most of the larvae migrate to the skeletal muscles (carcase). The period for which the larvae persist in the tissue of mice is not known.

In general, immunization of mice with two small doses of either Toxocara vitulorum or Toxocara canis induced a strong resistance to a challenge with a large infection of T. vitulorum. But the manifestation of immunity showed a difference in the mice which received the two types of immunization namely the homologous and heterologous. Homologous immunization was effective against the establishment of a challenge in the liver and migration presumably from the liver to the lungs whereas heterologous immunization did not affect significantly the establishment of the larvae in the liver but was mostly effective against the migration of larvae presumably from the liver to the lungs.

Studies on the natural infection of the buffalo calves with T. vitulorum from birth reveal that T. vitulorum eggs appear in the faeces from about days 19-21 of birth. The patency

of this (T. vitulorum) infection as indicated by the presence of homologous species is preceded by that of Strongyloides species from days 9-16 of birth. The patent period of T. vitulorum lasts till the calves were  $2\frac{1}{2}$ -3 months, presumably in the absence of a reinfection. The peak egg counts show a very marked variation. Colostral antibodies passively transmitted to calves from the dam do not have any effect on the patency of T. vitulorum infection. Thus, it was noted that in spite of the presence of precipitins passively transmitted to the calf with the colostrum, T. vitulorum infection became patent indicating that these antibodies are not protective in the calf against the development to maturity of an infection acquired by the calf pre- or peri- or post-natally. In general, reinfection of the buffalo calves with large doses of infective eggs after they had cleansed themselves of the natural infection acquired from birth, did not result in a patent infection but it induced strong or stronger precipitin reaction in the calves. The drawbacks in the experimental set ups in the studies since buffalo calves reared free of T. vitulorum infection were not available for comparison after appropriate treatment are discussed along with the results. It was noted also in the course of these studies that in three buffalo calves which had apparently cleansed themselves of a natural infection the reinfection developed to the third and fourth stages indicating a progressive development.

Serum samples from buffalo calves at different stages of natural and experimental infection were fractionated by 'Sephadex' gel-filtration and DEAE-A 25 ion exchange

chromatography. On 'Sephadex' gel-filtration, the buffalo calf sera in general resolved into three main peaks. The first peak consisted largely of IgM and  $\alpha_2$ -macroglobulin. The second peak consisted mainly of IgG's. The third peak contained mostly the non-antibody proteins. The non-antibody proteins were not identified. On DEAE-A 25 chromatography the IgG's resolved into two subclasses IgG<sub>1</sub> and IgG<sub>2</sub>. The antibodies detectable by the Enzyme Linked Immunosorbent Assay (ELISA) were present in the IgM and IgG's. But the gel-diffusion and in vitro larval precipitins were mainly confined to the IgG<sub>1</sub> and not to the IgG<sub>2</sub>. Even in the ELISA IgG<sub>1</sub> showed markedly higher activity than IgG<sub>2</sub>.