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STUDIES ON THE PATHOPHYSIOLOGY OF PARASITIC DISEASE

Published Papers Submitted to the University of Adelaide
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by

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- A2. Biochemical studies on *Nippostrongylus muris* infestation.
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L.E.A. Symons (1960), *Australian Journal of Biological Sciences*, 13, 163-170.
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L.E.A. Symons (1966), *Nature*, 210, 422-423.

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PART B

- B1. Pathology, absorption, transport and activity of digestive enzymes in rat jejunum parasitized by the nematode *Nippostrongylus brasiliensis*.
L.E.A. Symons and Donald Fairbairn (1962), *Federation Proceedings*, 21, 913-918.
- B2. Pathology of gastrointestinal helminthiases.
L.E.A. Symons (1969), *International Review of Tropical Medicine*, 3, 49-100.
- B3. Digestion, absorption and protein synthesis in intestinal nematode infections.
L.E.A. Symons (1971). Pathology of parasitic Diseases, pp. 187-195 (Ed. S.M. Gaafar). Proceedings of the Fourth International Conference of the World Association for the Advancement of Veterinary Parasitology, University of Glasgow, 1969. Purdue University Studies, Lafayette, Indiana.
- B4. Anaemia of fascioliasis.
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PART C

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C2. The use of Clioxanide as an anthelmintic in sheep infected with *Haemonchus contortus*.

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SUMMARY

The principal aim of the work described in these published papers was an explanation of the depressed growth rate or loss of weight in young mammals infected with parasitic intestinal nematodes. For this purpose, the pathophysiology of the rat infected with *Nippostrongylus brasiliensis* (Syn. *N. muris*), the mouse with *Nematospiroides dubius* and the sheep and guinea pig with *Trichostrongylus colubriformis* was studied. The cause of anaemia in the sheep infected with the trematode *Fasciola hepatica* was also investigated. A more detailed outline of the work is given in the Introduction, in which the significance of the experimental results is discussed.

In general, these investigations, which are presented in the first group of papers, may be divided into two: (1) measurements of function of the host, (2) the relationship between the morphological and biochemical changes and any physiological abnormality.

The general conclusion was that derangements of digestion and absorption could not explain depression of growth or weight loss in young infected animals, but that they were explained by changes in protein metabolism. An important, but not necessarily sole cause of these changes in protein metabolism was inappetance of the infected host.

When digestion was allowed to continue for some hours there was no evidence of depression, particularly of protein digestion, in nippostrongylosis, nematospiroidosis or trichostrongylosis, except possibly in the most severely affected animals close to death. There

was evidence of a slow rate of digestion in the early stage of the process, which was associated with the upper parts of the small intestine, the site of infection.

There was no malabsorption when this was measured in the small intestine as a whole, but there was jejunal malabsorption in the infected rat, in which there was some compensatory absorption in the distal ileum. In spite of jejunal mucosal hyperplasia, and hence a greater number of epithelial cells per cm length of intestine, jejunal malabsorption was due to immature cells on the villi. These crypt-like cells had migrated from the crypts of Lieberkühn following faster rates of epithelial cell turnover and proliferation. The immaturity of these crypt cells was believed to explain the low activity of certain mucosal enzymes.

Synthesis of skeletal muscle protein was depressed, while that of liver protein was elevated by infection in the growing mouse and guinea pig. In the very young guinea pig poor muscle growth was due to a combination of poor protein synthesis and hypoplasia due to failure to include new nuclei in fibres. Changes in the ratios of RNA to DNA and of polyribosomes to lighter ribosomes were consistent with the protein synthetic changes.

Ferrokinetic studies revealed that ingestion of blood by fluke was the cause of anaemia in chronic ovine fascioliasis, but that haemolysis of erythrocytes in the liver was important in the early stages of more acute infections.

A second group of papers was presented at a symposium or at international meetings, and includes a review article prepared by invitation.

The third group of two papers describes work outside the principal subject of this collection.