

ON THE PATHOGENIC ROLE OF LARVAE AND MATURE
STAGES OF *Bunostomum trigonocephalum*
(NEMATODA: ANCYLOSTOMATIDAE)

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Abstract

The authors have followed up experimentally the migration of the larvae of *Bunostomum trigonocephalum* through uninjured skin of an ovine host.

Studies were conducted on the duration of the prepatent and patent period of *B. trigonocephalum* infection.

The larvae are comparatively quick in penetrating the uninjured skin of the host. Five minutes after their infestation they were present under the epidermis and caused significant traumatic changes in the skin. Ten minutes later the larvae were present in the derma layer. After two days the larvae were situated in the host lungs and caused damage of the alveoli. The mature stages of *B. trigonocephalum* are localized primarily in the jejunum, where nematodes attach themselves to the intestinal mucosa and damage it during blood feeding.

Key words: *Bunostomum trigonocephalum*, traumatic pathogenic action, larvae, mature stages

Introduction. *Bunostomum trigonocephalum* nematode belongs to the family of Ancylostomatidae and like the other species of this family has strongly expressed pathogenic action on the host. There are a lot of scientific studies which show the pathogenic role of this nematode [1–5].

This parasite develops by ancylostomidian type of life cycle and completes its metacyclic migration in the organism of the host [2,3,7,8]: beginning from the skin

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and finishing in the small intestines where the nematode reaches mature stage, and its final predilection site. During the long migratory way the parasite renders primary (traumatic) and secondary (toxic) pathogenic action [2].

One of the most important clinical signs of the parasite host is the presence of anemia caused by the pathogenic action of *B. trigonocephalum* [1,2,6]. We considered this pathogenic role and pathomorphological changes a significant reason to undertake more detailed studies on their character by the method of histology. Our purpose was to study only traumatic pathogenic action of larvae and adult stages of *B. trigonocephalum*.

Material and methods. The experiments were carried out with lambs of four months of age, free of infection with other pathogenic agents. The lambs were experimentally infested with infective larvae (L₃ stage) of *B. trigonocephalum* [2]. Samples were obtained from the skin, lungs and small intestine and studied by routine histological techniques [2,3].

Results. Histological study showed that 5 min after percutaneous experimental infection in lamb host the larvae of *B. trigonocephalum* were present under the epidermis. The structure of the epidermis was injured traumatically (Fig. 1a).

Ten minutes later most of the parasite larvae were situated in the derma layer, but still we found some larvae under the epidermis of the skin. Twenty and thirty minutes after the infection we detected larvae in the derma and under the skin tissues. After one hour there were larvae into the subcutaneous tissue only (Fig. 1b). We found also larvae located closely to the blood vessels and high

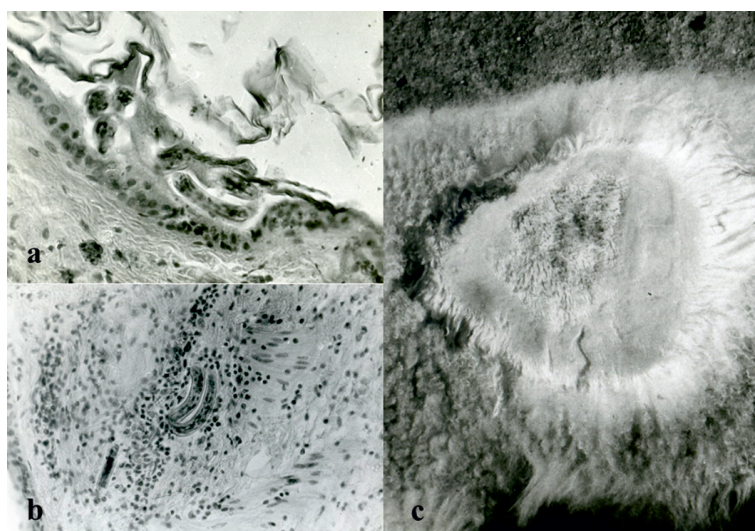


Fig. 1. a. Larvae of *B. trigonocephalum* under the epidermis of the skin 5 min after infestation; b. Larvae of *B. trigonocephalum* into the skin 1 h after infestation; c. Skin area after percutaneous infection. The presence of reddened skin and glued hairs

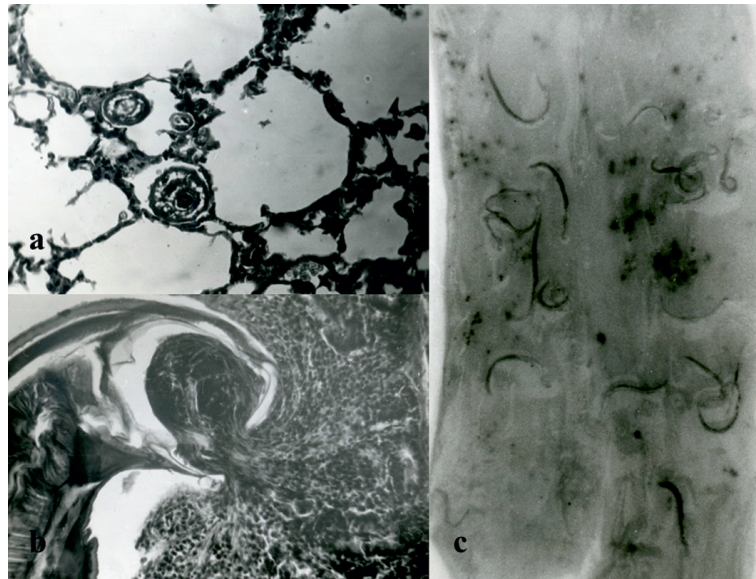


Fig. 2. a. Two days after infection. Lungs: broken alveoli; b. *B. trigonocephalum* attached to the mucosa of the host's jejunum; c. Multiple ulcers into the mucosa of the jejunum caused by *B. trigonocephalum* feeding

concentration of lymphocytes and eosinophils around these larvae. At the place of experimental infection we detected reddened skin and glued hairs (Fig. 1c). The experimental lambs showed symptoms of itching.

Two days later we found *B. trigonocephalum* larvae in the lungs of the experimental hosts. In this internal organ the alveoli were broken (Fig. 2a). In that stage of larvae migration the lambs showed signs of rapid breathing.

The infestation with *B. trigonocephalum* becomes patent on the 48th day post infection.

During the necropsy of the experimental lambs we found that 65% of the adult worms were located in the jejunum, 31% in the ileum, and only 4% of them into the duodenum. One part of the parasites were attached to the intestinal mucosa (Fig. 2b) and the other part were available into the intestinal content. In many places on the mucosal surface we found ulcers with a diameter around 2 mm caused by the feeding of parasites (Fig. 2c). In the point of fixation the adult parasites damaged deeply the host's intestinal mucosa.

Discussion. The results of our study show that all stages in the development of *B. trigonocephalum* cause traumatic pathogenic action on the host organism [1-5,8].

The traumatic pathogenic action in the skin layers is caused by the penetrating larvae, which mechanically damage the skin, searching for the blood vessels [3]. The next severe pathogenic action of the larvae leads to trauma in the

lungs parenchyma following their ancylostomidian type of life cycle. These larvae necessarily damage the lung's capillaries and the alveoli to continue their migration to the small intestine [2,7]. Like other ancylostomids the adult nematodes attach themselves to the intestinal mucosa, damage it after blood sucking. That is the end of migration and the final predilection site of the parasite [2-4,6].

Conclusion. Using the presented results we conclude that the larvae of *B. trigonocephalum* very quickly penetrate the intact skin of the typical ovine host and cause traumatic damages into the skin layers. During their migration the parasites in their larval stage cause traumatic changes in the lungs and of the small intestines as well. The injuries in the small intestinal mucosa are severe, because of the nematode final location and feeding. *B. trigonocephalum* lives as an adult parasite for a very long period in this predilection site. Our studies present and discuss the traumatic (mechanical) pathogenic action of *B. trigonocephalum* only.

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