

# Pathogenicity of *Microtetrameres centuri* Barus, 1966 (Nematoda: Tetrameridae) in Meadowlark

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**Abstract:** Pathogenicity of adult and third-stage *Microtetrameres centuri* Barus, 1966 in meadowlarks (*Sturnella* spp.) is reported for the first time. Adults do not encyst; their effects are mainly mechanical. Some glandular atrophy of its avian host is detected but no marked inflammatory response is exhibited. Juveniles also encyst experimentally in the perivisceral sinus of the grasshopper (alternate host) hemo-coel. **Key words:** *Microtetrameres centuri*, *Sturnella magna*, *Sturnella neglecta*, Parasitism.

*Microtetrameres* spp. are known to parasitize the proventriculi of birds of many species and orders (5). However, numerous studies have failed to report the presence of this parasitic genus in certain wild birds (5, 7, 8, 11) or birds of domestic importance (1, 10, 12, 13, 14, 17, 18). Therefore, it is not surprising that reports of the pathology caused by *Microtetrameres* spp. are unknown.

LaPage (9) ascribed a toxic effect to *Tetrameres americana*, a nematode closely related to *Microtetrameres centuri* Barus, 1966 and also a proventricular parasite of birds. He stated that greatest damage was caused by juveniles of *T. americana* migrating into the proventricular wall where they caused "marked irritation and inflammation." *M. centuri* juveniles also migrate within the tissues of the avian host and under laboratory conditions encyst within grasshoppers as third-stage juveniles (6).

In the present study the pathogenicity of certain stages of *Microtetrameres centuri* is reported for the first time.

## MATERIALS AND METHODS

Adult *Microtetrameres centuri* females were collected from naturally infected meadowlarks (*Sturnella magna* and *S. neglecta*)

and certain other birds which had been infected experimentally. Eggs from these females were fed to nymph grasshoppers as reported earlier (6).

Proventriculi of birds infected with female *M. centuri* were fixed in A.F.A., sectioned and stained with H & E. Sections of females *in situ* were examined by Dr. Frank Ramsey, Head, Department of Veterinary Pathology, Iowa State University.

Infected grasshopper hosts were fixed in alcoholic Bouin's fluid, doubly infiltrated and sectioned at about 20 microns. Sections were stained with Harris' hematoxylin and counterstained either with eosin or triosin.

## RESULTS AND DISCUSSION

Nucleated, ellipsoidal, red blood cells were noted within the digestive tract of adult *M. centuri* females. The lumen of this tract in these females was lined with a black unidentified substance.

Living third-stage juveniles were studied as they migrated within the ventricular tissue of a young laboratory-reared house sparrow (*Passer domesticus*). These nematodes remained within the soft tissue of the ventriculus. However, third-stage juveniles did not penetrate the keratin of a similarly reared house sparrow (6). At this stage the juveniles measured approximately 1500 by 50 microns and were quite vigorous in their movements.

Dr. Ramsey's report on the tissue parasitized by a female *M. centuri* may be sum-

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marized as follows: effects on the host tissue were primarily mechanical, growth of the parasite causing a cystic dilatation of the gland lobule with mild pressure atrophy of glandular epithelium. The involved gland lobules were non-functional and mild hyperemia of the lamina propria was noted. The parasite did not elicit marked inflammatory response. Lymphoid tissue was normal; some evidence of increased mucus secretion leading to "mild" catarrhal proventriculitis was observed. This mild infection of the proventriculus probably caused little apparent effect on general health; however, heavy infection with involvement of many gland lobules would probably affect the health of the bird. No encapsulation in connective tissue was noted.

An *in situ* study of representative sections of an adult female *M. centuri* reveals nucleated red blood cells within the lumen of the gut, indicating this species to be hematophagous. However, the amount of blood lost by an avian host presumably would be negligible except in heavy infections. The greatest infection found in any meadowlark host was 18 females.

Disruption in the production of hydrochloric acid by proventricular gland lobules and consequent disruption of the digestive processes in the bird has been attributed to the presence of *Tetrameres* (4). Perhaps similar conditions occur in birds harboring *Microtetrameres*, although little evidence supports this statement.

Rasheed (15) reported female *M. orioles* to be encysted. However, no evidence of a cyst, either of host or parasite origin, appeared in sections of adult *M. centuri* females *in situ*.

Some authors (16, 19) have reported the crop to be the site of infection for species of *Microtetrameres*. Such reports, however, seem to stem from the incorrect translation of "ventricule succenturie" as "crop" instead

of "proventriculus." Frequently during the course of this study, numerous nymphal grasshoppers were found dead shortly after they had ingested *M. centuri* eggs. While the exact cause of these deaths is not known, examination of the nymphs showed a large mass of eggs and egg shells within the esophageal region of their gut. These masses indicated a blockage of the digestive tract. Such blockage was not noted in nymphs which had ingested eggs and which were alive three or four days post-ingestion.

Numerous grasshopper nymphs were examined for the presence of juveniles after they had eaten nematode eggs. Only third-stage juveniles were found within any insect's hemocoel. However, in a few instances juveniles of an unknown stage were found in the legs of some grasshoppers. Cram (3) reported the same condition in her study of *T. americana*.

While damaged insect tissue was not studied during this investigation, it seems logical to assume that the first-stage juveniles of this species migrated through the intermediate host tissue with the aid of the cephalic projections previously described (6) and as hypothesized by Chabaud (2). These juveniles migrated from the gut of the grasshopper, molted twice and developed to third-stage. The damage caused by these juveniles during this migration is unknown. However, third-stage juveniles eventually encysted within the perivisceral sinus of the insect's hemocoel.

These intact cysts were removed easily from the host tissue. Their walls were about as thick as the diameter of the nematodes they enclosed. Because these cyst walls were transparent (6), the third-stage, encysted juveniles were examined easily. Most of them moved actively within their cysts and thrashed vigorously when experimentally excysted.

Cram (3) mentioned that grasshoppers

infected experimentally with *Tetrameres americana* became "droopy," a condition not noted in this study.

No difference in infectibility or refractoriness was noticeable among the various grasshopper species used (*Melanoplus femurrubrum*, *M. bivittatus*, *M. sanguinipes*, *M. mexicana*, *M. differentialis*). However, all experimental grasshoppers were reared from eggs furnished by the U.S.D.A., Montana State University, Bozeman, Montana; hence, the species was dependent upon its availability from this source. With rare exceptions, grasshoppers that lived 30 days after having ingested embryonated eggs of *M. centuri* were infected with third-stage juveniles.

Because only three known *M. centuri* males have been recovered experimentally (5), their pathological effect is still unknown. However, all studies of presumed and known male *Microtetrameres* indicate that they were recovered from the mucus of the bird's proventriculus.

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