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Myxobolus sandrae Reuss, 1906, the agent of vertebral column deformities of perch *Perca fluviatilis* in northeast Scotland

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ABSTRACT: *Myxobolus sandrae* Reuss, 1906 has been identified as a pathogen causing severe lesions of the spinal cord, vertebral collapse and marked curvature of the vertebral column of perch *Perca fluviatilis* in northeast Scotland. The taxonomic determination is based on spore morphology and on the taxonomic relatedness of perch to the type host of *M. sandrae*, *Stizostedion lucioperca*.

INTRODUCTION

The pathogenicity of Myxosporea in their fish hosts may be manifested by a wide range of changes, including skeletal deformities. The best known is that caused by *Myxobolus cerebralis* in rainbow trout (Halliday 1976), which causes cranial lesions and also spinal deformities. Recently Egusa (1985) reported scoliosis of the vertebral column of Japanese cultured yellowfins *Seriola quinqueradiata* infected with *M. buri*. In Australia, Langdon (1987) described spinal curvature in imported *Perca fluviatilis* due to infection with *Triangula percae*. We report here on an isolated population of *P. fluviatilis* in Sandloch near Aberdeen, northeast Scotland, which showed visible evidence of skeletal abnormalities in 0-group individuals. Parasitological examination revealed a *Myxobolus* species to be associated with these deformities, which was determined to be *M. sandrae*.

MATERIALS AND METHODS

Two samples of ca 30 0-group perch *Perca fluviatilis* were caught by dip net in the narrow exit stream to Sandloch (57° 21′ N, 1° 56′ W), ca 25 km north of Aberdeen, during August and October 1988. They were maintained live in aquaria until needed. Fish were killed with an overdose of benzocaine and either fixed in 10 % neutral buffered formalin or radiographed fresh with a Hewlett-Packard Faxitron 43805N machine. Exposures were made on Kodak Kodalith type 3 sheet film using 5 min exposures at 30 kV, 2.8 mA at a source-to-film distance of 45 cm. Spores were examined from formalin-fixed material. Histology was done on this material and sections stained in haematoxylin and eosin or Giemsa.

RESULTS

The fish exhibited clear signs of stunting (Fig. 1) but with normal skin colouration when first caught; some did not swim effectively. In other respects the fish were healthy and were not emaciated. When in laboratory aquaria it was obvious that some individuals did not swim in the normal orientation, and allowed the tail to sink. Within a few days some individuals displayed black pigmentation caudally and many of these fish subsequently died. Non-pigmented fish also died during this time. Mortalities were high and most fish from the first sample died within 2 wk. Survival was slightly longer for the second sample. It is not possible to attribute cause of death in any of these cases. One 1+ perch was caught in the second sample which was normal in appearance except for obvious shortening of the tail region. This was estimated to be about 25 % of the body length based on measurement of normal vertebrae and calculating the expected length from this measurement.

Perca fluviatilis possesses 40 vertebrae. The radiographs (Fig. 2) of each of 13 individuals show severe

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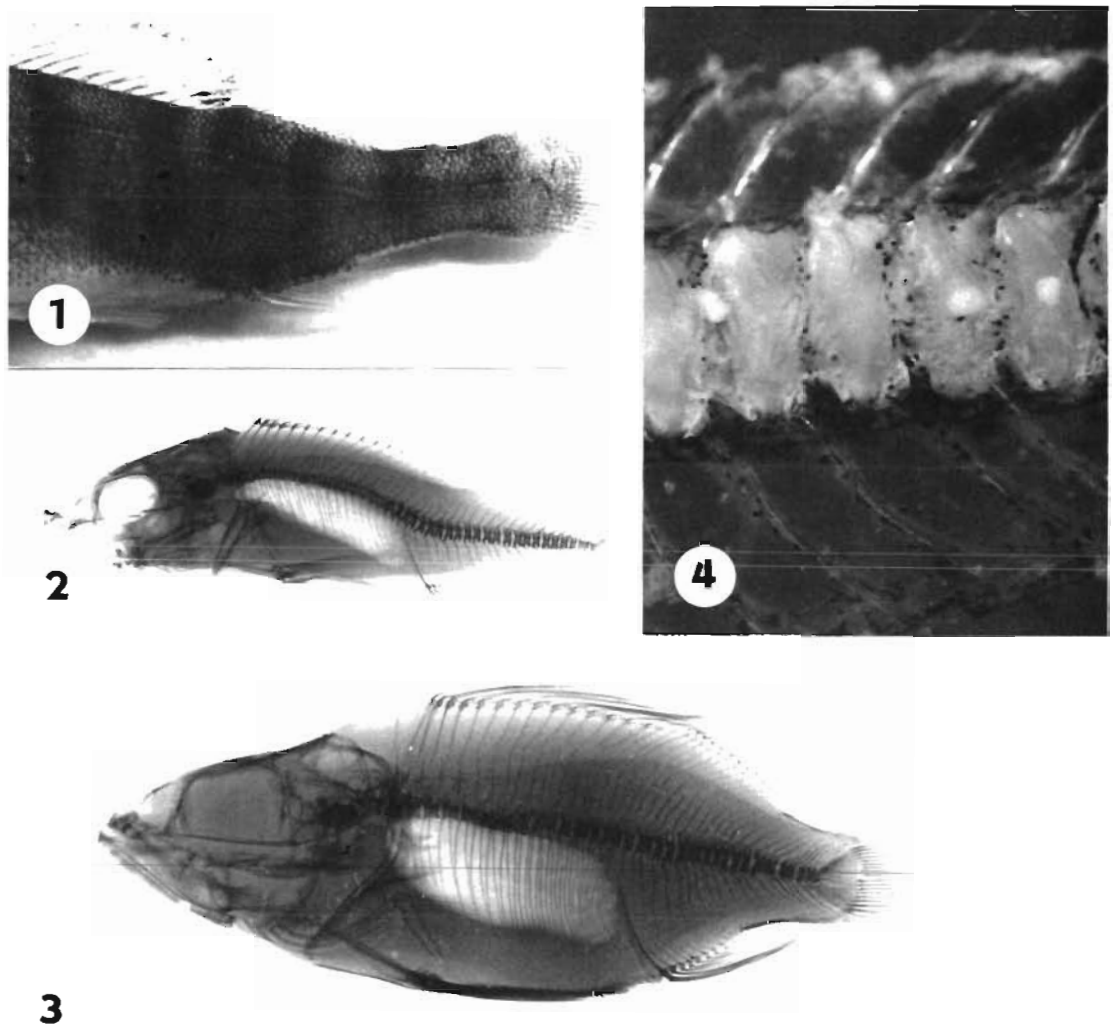
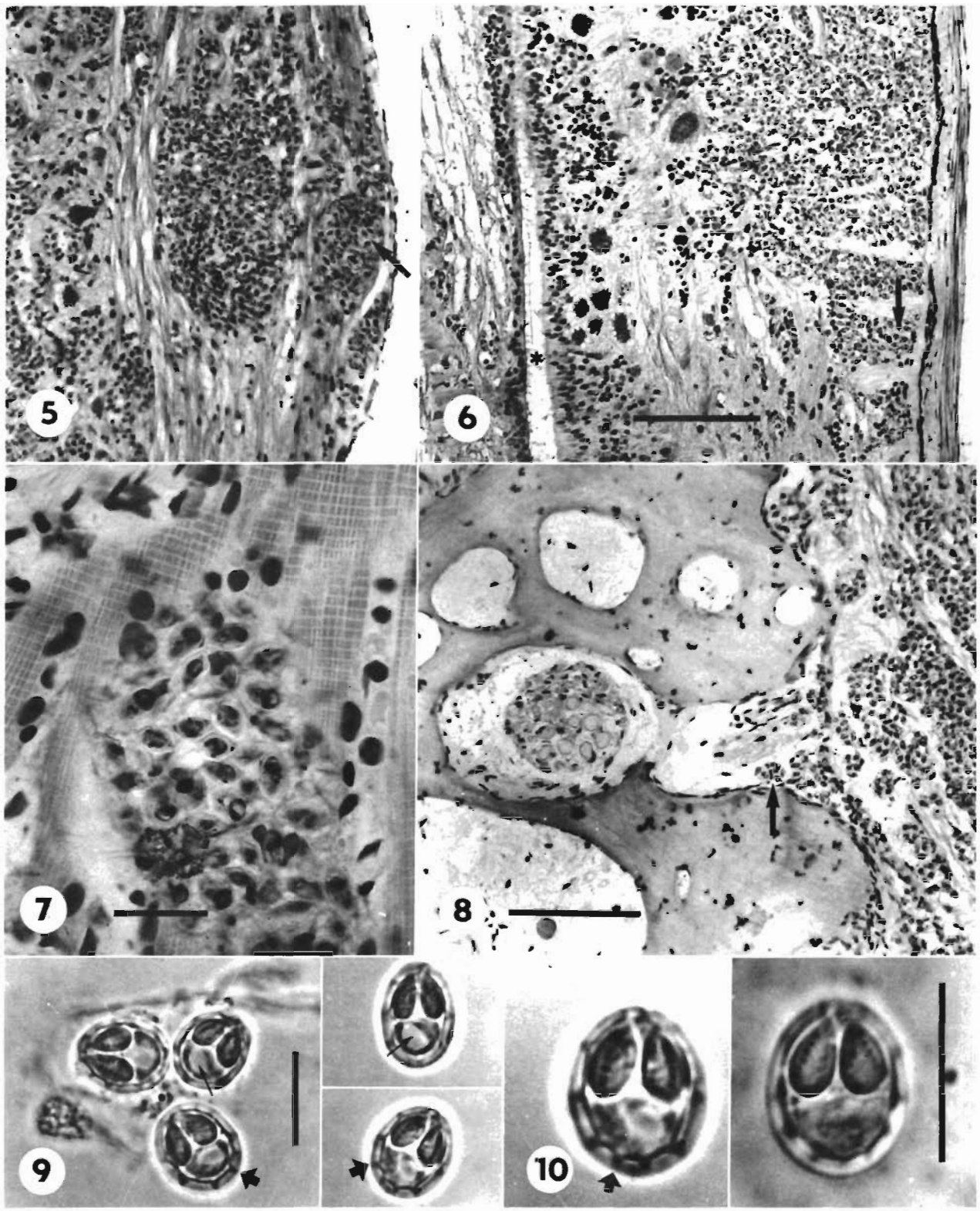


Figs. 1 to 4. *Perca fluviatilis* infected with *Myxobolus sandrae*. Fig. 1. Posterior body part of a (0 group) perch showing signs of stunting, × 3. Fig. 2. Radiograph of a 0-group perch showing compression of vertebrae with some distortion of the dorsal projection, × 2. Fig. 3. Radiograph of a 1+ class perch showing severe compression of the vertebrae, × 2. Fig. 4. Spore agglomerations in ectopic localisation in the muscles outside the vertebral column, × 25

compression of the vertebrae, particularly in numbers 15 to 40, but in 3 fish there was also compression of vertebrae 1 to 6. The neural and haemal spines and the neural arch appeared normal but there was some distortion of the dorsal projections. Lordosis was also evident in the posterior region of the vertebral column. The 1+ perch examined (Fig. 3) did not exhibit lordosis but there was severe compression of the vertebrae.

Only mature spores were found in the material available. These were found in small groups and in large, confluent agglomerations in the white matter of the spinal cord (Figs. 5 & 6). Some of these spore aggregates were quite obviously the original plas-

Figs. 5 to 10. *Perca fluviatilis* infected with *Myxobolus sandrae*. Figs. 5, 6. Spores in small and large confluent agglomerations (some of which can be considered to have arisen from the original plasmodia – arrow) in the white matter of the spinal cord as seen in the parasagittal section of the cord. Asterisk marks the central canal of the spinal cord. H & E. For both figures bar = 100 µm. Fig. 7. Spores in ectopic location in the muscle tissue. H & E Bar = 20 µm. Fig. 8. Spores (arrow) are found to migrate passively along the roots of the spinal nerves into the perineural spaces of the latter. H & E. Bar = 100 µm. Fig. 9. Formalin-fixed spores of *M. sandrae* in front view; thin arrow points at the iodinophilous vacuole, arrowhead points to the sutural markings appearing as folds on the spore surface. Bar = 10 µm. Fig. 10. Spores at a higher magnification. Bar = 10 µm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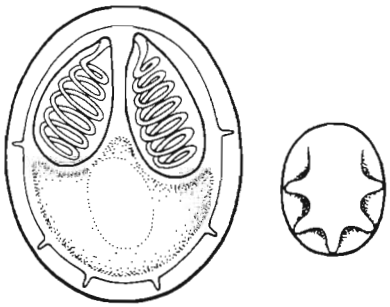

Fig. 11 *Myxobolus sandrae*. Line drawing of a typical spore from perch (frontal view); at right: folds on the surface of the shell valve (at a smaller scale than the spore drawing)

modia turned into pouches of mature spores. The grey matter concentrated around the central canal was compressed by giant aggregates of spores as were the horns of grey matter extending towards the periphery. The pathological changes can be characterised as atrophy and demyelinisation.

There were many small groups of spores in the vicinity of the central canal, exceptionally even in the ependymal cell layer Small aggregates of spores were found in the perineural spaces, i.e. in connective tissue surrounding spinal nerves and in the vicinity of the haemal arch (Fig. 8), around the dorsal aorta and close to the haemal spine which projects into the myosepta. The spores were also found in ectopic locations in the intermuscular connective tissue outside the vertebral column (Fig. 7), their masses appearing as whitish nodules or foci on the dissected vertebral column (Fig. 4) especially between vertebrae 16 and 34, but not in parenchymatous organs. The brain was not examined for presence of spores.

Spores are ellipsoidal in front view (Figs. 9 to 11), 8 (7.4 to 8.5) × 10.1 (9.2 to 11.3) µm in size (n = 20), with a 1 µm wide sutural edge revealing about 6 sutural markings mostly around the posterior half of the spore. The markings correspond to the folds visible on the spore surface when focused on this surface. Ellipsoidal polar capsules have anteriorly tapering and converging ends. Polar filaments make 6 or 7 slightly slanted turns. There is no intercapsular appendix. The sporoplasm contains a large iodinophilous vacuole.

Further investigation of the perch population in Sandloch has been inhibited by its nature reserve status. It was hoped that older fish could be obtained to establish whether infection persists and what skeletal effects might be observable. Despite repeated attempts to obtain perch in subsequent breeding seasons in 1989 and 1990 no young perch appeared in the exit stream. It would be interesting to establish the pathogenicity of this species in natural conditions. The large populations of three-spined sticklebacks *Gasterosteus aculeatus*, also in the stream and infected with *Myxobilatus* and *Sphaerospora*, have not been affected.

DISCUSSION

The mechanism of changes of the vertebral column is probably similar to that in infections with *Myxobolus cerebralis* in salmonids (Schäperclaus 1954, Hoffman et al. 1962, Halliday 1976) or in infections of *Perca fluviatilis* with *Triangula percae* Langdon 1987). The flexure of the vertebral column may be the result of myopathy of axial muscles and/or of asymmetrical changes in muscle tone, both resulting from lesions or damage inflicted upon caudal nerves that control contraction of the respective muscles. It is interesting to note, however, that *T. percae*, as well as other species causing spinal curvatures in fish – *Myxobolus buri* in *Seriola quinqueradiata* (Egusa 1985) and *Myxobolus* sp. in sandflathead *Platycephalus bassensis* (Rothwell & Langdon 1990) –, were not recorded from the spinal cord but only from the brain. Other brain-infecting species, such as *M. encephalicus* Mulsow, 1911 (see Dyková et al. 1986), *M. lairdi* Moser & Noble, 1977, *M. bilineatus* Bond, 1938, *M. kisutchi* Yasutake & Wood, 1957 and *M. neurobius* Schuberg & Schröder, 1905 although they may induce clinical disease are not known to elicit spinal deformities.

In identifying the parasite responsible for the pathology associated with the perch, account has been taken of *Myxobolus* spp. already known in *Perca fluviatilis* and related species, their location in the host tissues and the spore morphology.

Perca fluviatilis is known to harbour *Myxobolus karelicus* Petrushevski, 1940 in its gill. However, the morphology of this species, which has also been recorded from *Aspro zingell* and *Stizostedion lucioperca* (Shulman 1984), differs from the present form. *M. dujardini* Thélohan, 1899, *M. minutus* Nemeczek, 1911, *M. permagnus* Wegener, 1910 and *M. pfeifferi* Auerbach, 1908 have also been reported from *Perca fluviatilis* (Shulman 1984), but all differ from this species in spore morphology. *M. guénoti* Naville, 1928, reported by Naville (1928) from perch gills, is probably a junior synonym of *M. permagnus* and thus also differs in spore morphology

Three *Myxobolus* species were reported from the closely related North American *Perca flavescens*. *M. percae* described by Fantham et al. (1939) from the base of the pectoral fins has spores of different structure. Guildford (1963) described *M. neurophila* in the optic tectum and brain and *M. scleroperca* in the eyes of *P. flavescens* but both parasites differ in spore morphology from the present form.

Myxoboli that infect nervous tissue and/or the brain, such as *Myxobolus encephalicus, M. neurobius, M. hendricksoni* Mitchell, Seymour & Gamble, 1985, *M. bilineatus, M. buri* Egusa, 1985, *M. arcticus* Pugachev

& Khokhlov, 1979 and *M. spinacurvatura* Maeno et al., 1990 have different spores and, in addition, infect hosts of taxonomically different position.

Looking for morphological confirmation of our finding, although there is a slight similarity with *Myxobolus saidovi* Gasimagomedov, 1970 and *M. nemachili* Weiser, 1949 sensu Chen (1973), these are not sufficient for safe identification. However, *M. sandrae* Reuss, 1906 has spores which appear completely identical with our findings. The original drawing of Reuss (1906) is rather poor, but our observations on spores from the type host, *Stizostedion lucioperca*, as well as data of Soltynska (1967) and Grabda & Grabda (1971) correspond to the present finding as well as with each other. There is the same wide sutural edge, shape and size of spore, absence of triangular appendix and polar capsule configuration. The host is different but it seems that *M. sandrae* may not exhibit strict host specificity. Wegener (1910) recorded a *Myxobolus* sp. appearing identical with our finding, from the gills and opercula of *Perca fluviatilis*, in the former eastern Prussia. *M. magnus* Averinzew, 1913, found by Averinzew (1913) in the iris and corpus vitreum of *Gymnocephalus cernuus* from Peterburg, appears morphologically identical, and hence a junior synonym of *M. sandrae*. Shulman (1984) quotes *G. cernuus* as the host of *M. sandrae*, too, in addition to *Stizostedion lucioperca* and *S. volgensis*, and lists as sites of infection the gill arches and filaments, opercula, skin, fins, iris of the eye, subcutaneous tissue, muscle and intestinal wall. Even if some of these organs may have harboured only mature spores, conveyed there passively by the blood circulation, as is common in myxosporean infection, it appears that *M. sandrae* is neither strictly host nor site specific. It may infect several hosts of the related genera *Gymnocephalus, Perca* and *Stizostedion* with site preference differing in each of the hosts.

According to Maitland (1981) the area of distribution of *Stizostedion lucioperca* in Britain is limited to southeast England, *Gymnocephalus cernuus* is absent from Scotland whereas *Perca fluviatilis* is widely distributed.

Myxobolus sandrae is thus believed to belong to the group of myxosporeans with low host and site specificity, unlike for example *M. encephalicus* which is only known, thus far, from the brain of *Cyprinus carpio*. Further investigation of this species is warranted on its observed pathogenicity to young perch and in view of its potentially wide host range.

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