A New Disease in Cultured Yellowtail *Seriola quinqueradiata* Characterized by Encephalomyelitis

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**ABSTRACT**—In many yellowtail *Seriola quinqueradiata* farms in the vicinity of Uwajima city, Ehime Prefecture, Japan, a new disease has caused heavy mortalities of yearlings, mainly in summer and autumn. Symptoms included anorexia, lethargy with occasional bursts of swimming and redness in the central nervous system. In a histological survey, the most noticeable sign was severe encephalomyelitis. It is suspected that densely packed parasite-like cells (about 4 μm in diameter) in the spinal cords are the causative agent of this disease.

**Key words**: mass mortality, encephalomyelitis, *Seriola quinqueradiata*, yellowtail

In recent years, yellowtail *Seriola quinqueradiata* farms near Uwajima city, Ehime Prefecture in western Japan have been hit by a disease that has caused heavy mortalities. The disease appears in summer and autumn when water temperatures are 23–28°C, and affects yearlings (100–1,000 g in body weight). Symptoms include anorexia, lethargy with occasional sudden bursts of swimming, emaciation and skin ulceration from the jaw to the anterior abdomen. Internally, redness in the brain and spinal cord was the most noticeable sign (Fig. 1A). These gross signs thus suggested disorders of the central nervous system. However, preliminary surveys using ordinary bacteriological isolation using tryptic soy, brain heart infusion, cytophaga and Ogawa media agar plates failed to identify any pathogen. PCR techniques targeting viral nervous necrosis (VNN) and iridoviral disease were not able to amplify the fragment of these viruses. In experimental infection trials, intraperitoneal injection of healthy fish with brain homogenates from affected fish failed to cause infection. In the present study, we used histological techniques to elucidate the pathogenesis and to identify a possible causative pathogenic agent.

**Materials and Methods**

Sixty-five yearlings (100–1,160 g in body weight) affected in various extents by the disease were taken from three fish farms near Uwajima city every month from July to November of 2006. For the healthy control, five yearlings (450 g in average body weight) were obtained from a farm in Miyazaki Prefecture where the disease had not occurred yet. Their brains, spinal cords, lateral organs, noses, eyes, visceral organs and gills were fixed in 20% formalin and subjected to usual histological procedures. The spinal cords of three diseased yearlings (450 g in average body weight) sampled from one fish farm in September were serially sectioned. The sections were stained with H-E and examined by light microscopy.

**Results and Discussion**

In all the affected fish, the most serious histological lesions were found in the anterior half of the spinal cord. The damage was characterized by necrotic nerve cells with cytoplasmic vacuoles (Fig. 1B), neurophagia, swollen eosinophilic axons, multifocal glial nodules and hemorrhages. The spinal cord sections and other central nervous tissues showed various degrees of nerve cell necrosis with neurophagia, glialosis, multi-focal glial nodules and hemorrhages. The spinal cord sections and other central nervous tissues showed various degrees of nerve cell necrosis with neurophagia, glialosis, multi-focal glial nodules (Fig. 1C), degenerative axons, blood congestion and encephalomeningitis. These characteristics indicate that the affected fish had severe neurotic dysfunction. Although granulomatous peritonitis was seen in all of the affected fish, the healthy control fish also had similar lesions. Other remarkable lesion was observed neither in affected nor in healthy fish. Therefore, it appears that the central nervous dysfunction associated with anorexia leads to death of the fish.

Exotic, parasite-like bodies were found in seriously damaged spinal cords of 18% (12 out of 65 fish) affected fish (Figs. 1B and 1D). The serially sectioned spinal cords of the three fish had four to six exotic bodies, which suggested that actual rate of infection of these bodies could be much higher than 18% in the spinal cord. Thus, the presence of these bodies seems to be linked to the encephalomyelitis. They were elongated in shape (10–80 μm long), surrounded by a thin membrane, and packed with many parasite-like cells (Figs. 1B and 1D), which were round in shape and about 4 μm in diameter, having one or two small basophilic nuclei in the center.
The myxozoan parasite, *Myxobolus acanthogobii*, is known to infect the brain of the yellowtail [1] and to cause compressive damages (i.e., mechanical damage) to the nerve tissues, causing anomalies of the vertebrae. In the case of *M. acanthogobii*, however, the encephalomyelitis with necrotic disfunction has not been observed, suggesting that the pathogenic effects, if any of the exotic bodies found in the present study are non-mechanical.

Over 20 species of microsporidians and myxozoans have been reported to infect the central nervous system of teleosts [2–4]. The present observations cannot be attributed to any of these known parasites. In some cases, pathological changes were found in close contact with parasite-like bodies (Fig. 1D), suggesting that these bodies are the causative agents. The mass mortality of yellowtail accompanied by encephalomyelitis might be caused by this unidentified parasite. However, there were cases, where no clear associations of the parasite with histological changes was noticed (Figs. 1B and 1C). Further studies are needed to identify the parasite-like cells and to clarify the link between these cells and the disease.

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**References**