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Parasites of Wild and Diseased Juvenile Golden Snapper, *Lutjanus johni* (Bloch), in Floating Cages in Penang, Malaysia

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Abstract

A total of 148 wild healthy juvenile snapper *(Lutjanus johni)* from the Middle Bank, and 197 diseased ones from floating cages in Penang, Malaysia, were examined for parasites. Seventeen species of parasites were recovered. The most abundant parasite was a monogenean, *Haliotrema johni*, found in all samples of healthy and diseased fish. The densities of this monogenean in diseased fish (up to a mean of 314.1) were an order of magnitude higher than that of the healthy ones (28.9). One of the major factors that contributed to the observed disease outbreak was probably the high density of the monogenean. In the diseased fish, areas near the nostril, above and below the eyes, and sides of the operculum were without skin and scales. The exposed areas were reddish. Hemorrhagic caudal fin rot, typical symptoms of vibriosis were also observed in many of the diseased fishes.

Introduction

The culture of marine finfishes in floating cages in Peninsular Malaysia first started in Penang in 1973. Golden snapper, *Lutjanus johni* (Bloch), is one of the finfish cultured, the others being grouper, *Epinephelus salmoides* (Lacépède), and seabass, *Lates calcarifer* (Bloch). Pacific fauna of the seabass have been reported previously (Leong and Wong 1986). All the snapper fry are caught from the wild for culture in the floating cages.

No proper studies have been done on the parasitic fauna of snapper either from wild or cultured populations. Due to frequently observed disease outbreaks in cultured marine finfish, a sampling

program was undertaken to examine both wild and cultured juvenile snappers for parasitic fauna. In the course of this sampling program, a serious disease outbreak occurred in a floating cage. This provided us an opportunity to study the parasitic fauna of the diseased snapper and compare them with that of the healthy population. This paper presents data on the parasitic fauna of wild snapper and diseased snapper which were cultured in floating cages in Penang, Malaysia.

Materials and Methods

The golden snapper are caught along the Middle Bank in the South Channel of Penang Island by fishermen. These wild snapper are sold to fish farmers for stocking in their floating cages. Before stocking these fish, the farmers usually treat the fish with sulfurate drugs. Treatment lasts for several hours depending on the feeding schedule of the the fish farmers. The wild snapper fry investigated here were purchased from fish farmers before such treatment. These fry were examined within 24 hours for parasites.

In July and in August 1986, disease outbreaks occurred in a floating cage containing about 2,500 snapper. During the outbreaks, diseased fish were obtained from the fish farmer for examination before any drugs or chemical treatments were applied to the diseased fish. The diseased fish were then treated with 200 ppm formalin and 10 ppm acriflavin for about 20 minutes for two consecutive days.

The external surfaces of the fish were examined for gross abnormalities such as eroded fins, lesions, etc., and for ectoparasites. The mucus from the body surface and gills was scraped, placed on a glass slide and examined under a compound microscope at 400x magnification.

The length and weight of the fish were then recorded. The gills, stomach and intestine were removed and placed separately in Petri dishes. The lining was scraped and the contents poured into beakers filled with water. The contents were allowed to settle and the supernatant was then decanted frequently until a clear suspension was obtained. The contents were then examined for parasites under a dissecting microscope.

Monogenea, Trematoda, and Cestoda were fixed and preserved in 70% alcohol, stained in Semichon's Acetocarmine and mounted in Canada balsam. Nematoda, Acanthocephala, Isopoda, and Copepoda were preserved in 2% glycerine in 70% alcohol, cleared in glycerine and examined under a dissecting microscope.

A total of 345 golden snappers, of which 197 were diseased fish, were examined for parasites between June and August 1986.

Results

Symptoms of diseased fish

In samples of fish from the first disease outbreak, the skin and scales were absent in reddish areas above and behind the eyes, sides of the operculum and the nostril (Fig. 1). These injuries to the forehead and sides of operculum appeared to be due to the fish rubbing against the side of the cages to rid itself of some irritants. In a small number of fish, ulcers were also observed on the body.

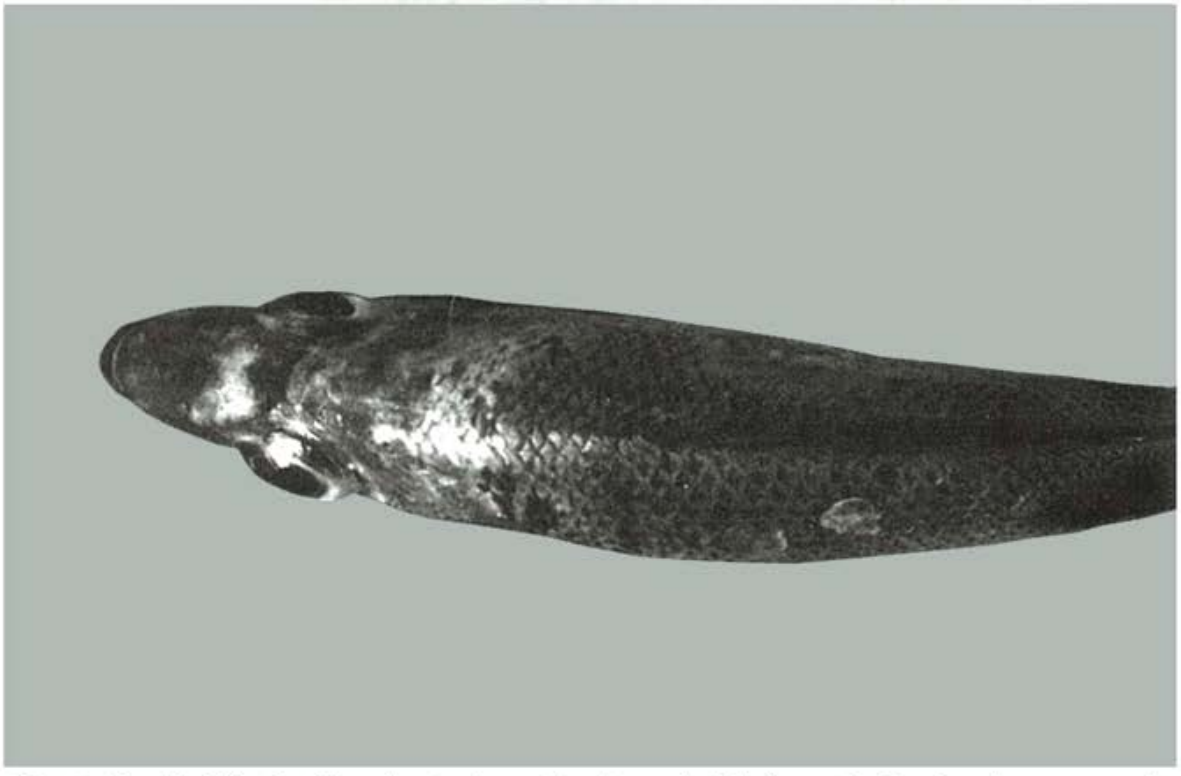

Fig. 1. The "balding" effect due to loss of scales and skin from the head region as a result of heavy infection of *Haliotrema* spp.

The above symptoms were also observed in the sample of fish from the second disease outbreak. However, in this batch of diseased fish, a large number showed symptoms of typical vibriosis with hemorrhagic caudal fin rot (Fig. 2).

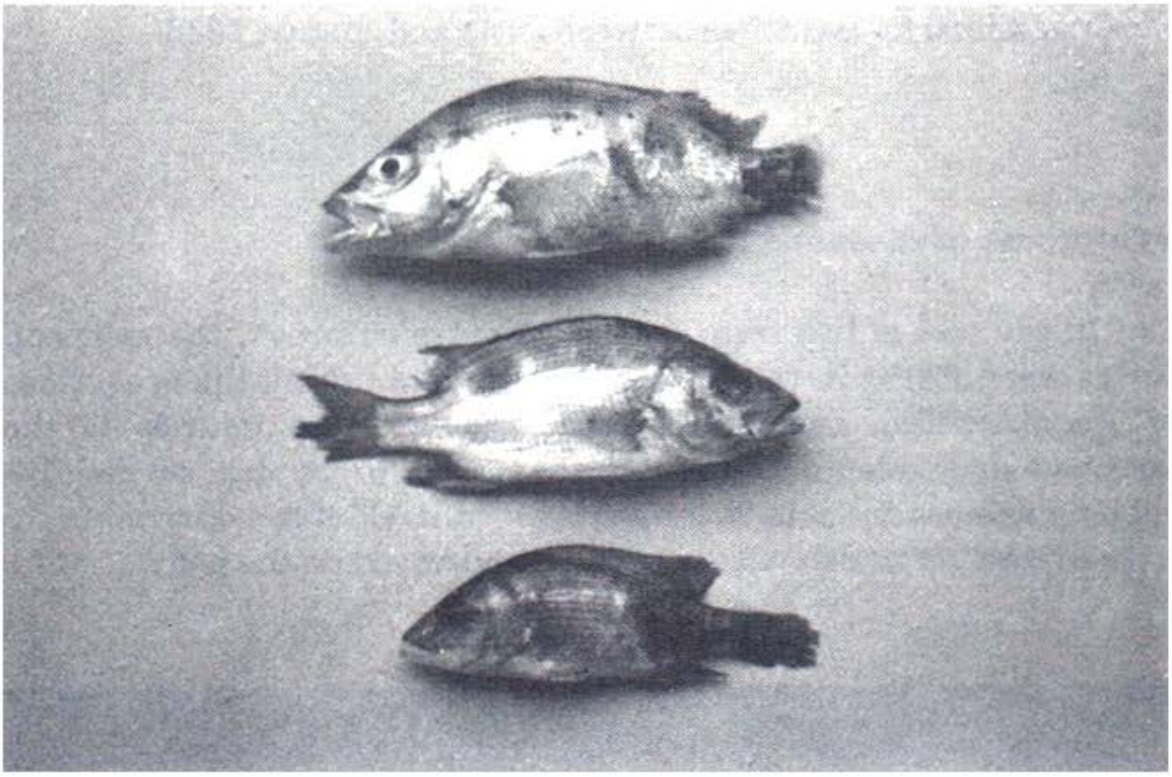

Fig. 2. The "balding" effect as a result of heavy infection of *Haliotrema* spp. followed by ulceration on the body and erosion of the caudal fin symptomatic of vibriosis.

Parasitic Infection

Seventeen species of parasites were recovered (Table 1). The most abundant was a monogenean, *Haliotrema johni*, which was recovered from both diseased and healthy fishes. The incidence of the monogenean ranged from 8.0% in the first batch of wild snapper to nearly 100% in the diseased cultured fish. The mean intensity of *H. johni* (parasites/fish) ranged from 12.8 in the first of batch wild snapper to 314.1 in the first batch of diseased fish.

The first batch of wild snapper sampled had an overall infection rate of only 19.0% compared to 72.9% in the second batch, about a month later than the first batch (Table 1). The first batch of wild snapper was smaller (average fork length 5.9 cm) than in the second batch (9.6 cm). The mean intensity of infection increased from 5.8 to 26.8.

In the diseased snapper, the first batch of diseased fish had a prevalence of 99.2% and a very high mean overall intensity of 325.7. In the second batch of diseased fish, 97.3% of the fishes examined

Table 1. Prevalences and mean intensities of infection of various parasites in wild and diseased cultured golden snapper, *Lutjanus johni* in Penang, Malaysia.

	Wild Stock		Cultured Disease Stock	
	Batch one 20/6/86	Batch two 24/7/86	First disease outbreak 18-23/7/86	Second disease outbreak 22/8/86
Protozoa				
Cryptocaryon irritans	6.0	22.4	-	2.7
Trichodina sp.	-	-	0.8	
Monogenea				
Haliotrema sp.	1.0(1.0)	33.3(4.2)	54.9(6.9)	28(2.8)
Haliotrema johni	8.0(12.8)	60.4(28.9)	99.2(314.1)	97.3(107.1)
Benedenia sp.	-	-	13.0(1.3)	2.7(1.0)
Trematoda				
Cardicola sp.*	-	-	-	2.7(1.0)
Mehracola ovocaudatum	1.0(1.0)	18.8(1.8)	0.8(1.0)	4.0(1.3)
Neoparacryptogonimus ovatus	-	6.3(1.3)	0.8(1.0)	1.3(1.0)
Prosorhynchus pacificus	-	4.2(1.0)	-	1.3(1.0)
Helicometrina nimia	1.0(1.0)	-	-	-
Lecithochirium sp.*	-	-	0.8(1.0)	2.7(1.0)
Didymozoid*	1.0(1.0)	2.1(1.0)	-	2.7(1.0)
Cestoda				
Tetraphyllidea*	-	-	4.1(185.4)	2.7(1.0)
Nematoda				
Raphidascaris sp.*	2.0(1.0)	4.2(1.0)	23.8(2.5)	17.4(1.2)
Acanthocephala				
Acanthocephalus sp.*	2.0(1.0)	-	-	-
Copepoda				
Caligus sp.	-	14.6(1.1)	0.8(1.0)	2.7(1.0)
Isopoda				
Nerocila sp.*		2.1(1.0)	0.8(1.0)	
No. of fish examined	100	48	122	75
Average fork length (cm)	5.9	9.6	8.3	10.6
Average weight (g)	3.6	14.3	9.2	22.1
Overall prevalence (mean intensity)	19.0(5.8)	72.9(26.8)	99.2(325.7)	97.3(108.4)

* larval

were infected, but the mean intensity of infection was only 108.4. In both batches, the density of the monogenean, *Haliotrema johni* (314 and 107, respectively) was found to be very much greater than that of other parasites. These cultured snappers had grown from an average fork length of 8.3 cm in the first batch to 10.6 cm in the second batch, which was about a month older.

A small proportion of the wild as well as diseased snapper was found to be infected with the pathogenic protozoans *Cryptocaryon irritans* Brown 1951 and *Trichodina* sp.

Discussion

The present data have contributed to the understanding of the parasitic fauna of the juvenile golden snapper of comparable size in their natural habitat and in floating cages. A large variety of parasites was found in the first batch of wild snapper, which was at the most 2-3 weeks old. A fair proportion of the fish was already infected with a high intensity of the monogenean, *Haliotrema johni*. The ubiquitous ciliate protozoan *Cryptocaryon irritans*, which is very pathogenic to fish in intensive culture, was also present in the wild fish population.

The second batch of wild snapper (6-7 weeks old) exhibited a similar variety of parasites. However, the proportion of fish infected with the parasites increased tremendously, particularly that of the monogenean *Haliotrema johni* which had doubled.

Both batches of diseased fish had a similar variety of parasites to the wild stock. One noticeable difference between the wild and cultured snappers was the incidence of a monogenean, *Haliotrema* sp. In each batch of the diseased fish, the mean intensity of *Haliotrema* sp. was one order of magnitude higher than in the wild stock. The second batch of wild snappers and the two batches of diseased snappers were of about the same age. This indicates that under intensive culture, infections of fish by monoxenous parasites is easily enhanced. Leong and Wong (unpublished data) found that the monogenean *Pseudorhabdosynochus* (= *(Cycloplectanum) epinepheli* Yamaguti 1938 in cultured grouper increased threefold compared to that of the wild population. Such epizootic infection by monogeneans and other parasites on farmed fish has been reported for cultured yellowtail (Eugusa 1983), for sole, plaice and turbot (McVicar and MacKenzie 1977), and for grey mullets, seabass and gilthead seabream (Paperna 1983, 1984). Epizootic infections by monogeneans are often pathogenic to farmed fish.

The tremendous increase in intensity of *Haliotrema johni* infections (up to 1,204) may have caused the fish to rub themselves against the side of the net to rid themselves of the parasites. In the process, the scales were rubbed off and the skin injured. Two adverse effects on the fish may result: physiological imbalance as a result of

the large exposed area and the introduction of ubiquitous *Vibrio* into the fish through wounds on the epithelium as well as injury by the anchors of the many monogeneans.

Similar disease symptoms in golden snapper have been reported by Chong and Chao (1986) in Singapore. However, they attributed the causative organism of the disease to the ciliated protozoan *Cryptocaryon irritans*. In our samples, very few of the diseased fish were infected by this protozoan. It is very unlikely that the protozoan is the causative organism in this case.

One of the major contributing factors to the observed disease outbreak of golden snapper was the constant introduction of wild juvenile snapper into the floating cages. Although the wild juvenile snappers are given prophylactic treatment of Diameton and Furanace-related drugs for about 6 hours, the drugs may not be effective against monogeneans and the snappers may not be freed of the parasites. Additions of new infected fish, together with the adverse growth conditions, including overcrowding, may have resulted in the clinical and pathological manifestations observed. This epizootic infection of ectoparasites was followed by an outbreak of vibriosis.

The treatment of dipping diseased fish in 200 ppm formalin and 10 ppm acriflavin for 20 minutes for two consecutive days appeared to reduce the density of the monogeneans. However, the treatment did not save the diseased fish from dying because of other complications, especially of vibriosis.

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