微胞子虫 Microsporidium seriolaeによるブリ類のベこ病の感染動態
Infection Dynamics of Microsporidium seriola (Microspora) Causing the Beko Disease of Seriola spp.

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(Received February 15, 2011)

ABSTRACT—Infection dynamics of Microsporidium seriola causing the beko disease of yellowtail Seriola quinqueradiata and amberjack S. dumerili were investigated at fish farms and an experimental facility. A 4-year survey (2006–2009) showed a sharp increase in prevalence of infection with M. seriola in June or July soon after stocking net cages with yellowtail and amberjack wild fingerlings. Most cysts blackened and finally disappeared in November in the following year. However, in 2006 when the levels of infection were relatively high, some visible cysts still remained even in autumn of the following year. Infective periods and effects of fish size on M. seriola infection were investigated via natural exposure of hatchery-reared yellowtail juveniles by transferring to an open sea cage in an endemic area. Invasion of the parasite into fish occurred in June and July but not after the mid-August. A comparison of M. seriola infection among four size classes of yellowtail showed that the smallest fish (1 g in mean weight) had the lowest prevalence of infection.

Key words: Microsporidium seriola, Seriola quinqueradiata, yellowtail, Seriola dumerili, amberjack, infection dynamics, beko disease

Beko disease of yellowtail Seriola quinqueradiata is caused by Microsporidium seriola, which is an obligate intracellular microsporidian parasite (Egusa, 1982). Infection with this parasite in the trunk muscle is visible as elongate white masses which are called "cysts". Disintegration of massive cysts in the trunk muscle results in the characteristic concave fish body surface. This disease has often been reported in yellowtail and goldstriped amberjack S. lalandi cultured in open sea cage from the Kyushu area of Japan (Egusa, 1982; Yokoyama et al., 1996; Sano et al., 1998; Bell et al., 1999, 2001).

Heavy infection with M. seriola causes emaciation and mortality of yellowtail juveniles, whereas lightly or moderately infected fish usually recover from the disease (Egusa, 1982). Beko disease has not been considered as a serious problem by fish farmers. However, in recent years, commercial fish with even a minute scar of M. seriola cysts are often rejected by consumers in terms of hygiene. This public trend has recalled the beko disease of yellowtail.

The recovering process of fish infected with M. seriola has not been well documented. Yokoyama et al. (1996) revealed that disintegration of cysts led to ingestion of liberated spores by host's phagocytes, resulting in formation of melanomacrophage centers in the muscle tissues as well as secondary migration of phagocytized spores into other organs, such as the heart, intestine, kidney, and gills. Also, Sano et al. (1998) showed that encapsulated cysts in the musculature were associated with granulation tissue, causing the scar formation. These findings indicated that recovery of infected fish was caused by host's inflammatory responses. However, it is unknown how long it takes until cysts disappear completely from host fish.

Experimental infections of fish with M. seriola were attempted by immersion, intramuscular injection and oral administration of fresh spores but all were never successful (Yokoyama et al., unpublished data). Failures of direct transmission suggest that an obligate intermediate host is required in the life cycle of M. seriola. Thus, studies on the host-parasite relationships have depended solely on observation of naturally infected fish in the open sea cage or hatchery-reared fish experimentally exposed with the parasite by transferring to the open sea cage in endemic area. Various questions still remain to be answered for the beko dis-
ease, such as the infective period, process of recovery and a size effect of host fish. In the present study, we aimed to monitor the seasonal occurrence of *M. seriolae* as well as to determine several factors responsible for the infection of the parasite in the open sea.

## Materials and Methods

### Field monitoring at a fish farm

Yellowtail and amberjack *S. dumerili* (0-year and 1-year) cultured at a private fish farm in Miyazaki Prefecture were examined for *M. seriolae* between 2006 and 2009. The origin of 2006 and 2007 fingerling cohorts was Kagoshima Prefecture, whereas 2008 and 2009 cohorts were from Miyazaki Prefecture. After the introduction of fingerlings into sea cages in June or July, samples of 10–20 fish were randomly collected from the same lot several times during 6–18 months. Fish were weighed, measured and filleted, and the number of cysts in the trunk muscle was counted by naked eyes. Prevalence and mean intensity of infection was defined as the number of infected fish/total number of fish examined, and the number of cysts/the number of infected fish, respectively. For the 2007 and 2009 cohorts, the presence of spores was also examined by the Uvitex 2B staining with 25 fish (only large fish from which 1 g of the muscle tissue could be collected), because the remaining 63 fish were not large enough to be tested by the Uvitex 2B method. Additionally, single and nested PCR-based detection assays for *M. seriolae* DNA were performed according to Bell et al. (1999). Briefly, ca. 50 mg sample of musculature was collected from each fish, homogenized, and the DNA was extracted using DNA Mini Kit (Qiagen). PCR reactions were carried out in 20 µL total volume. Each PCR mixture contained 0.1 µL of Takara Ex TaqTM HS (5 UI/µL), 2 µL of 10×Ex Taq Buffer, 1.6 µL of dNTP mixture (2.5 mM each), 0.5 µL (25 µg/µL) of each outer primer (MS-F1: 5'-CACCTGTCTGCAATGCGGG-3' and MS-R1: 5'-[CTTCCGGCGTATCTTTAGTC-3') and 3.0 µL of extracted DNA suspension. All PCR reactions were performed in iCycler (Bio-Rad). Denaturation of DNA (95°C for 5 min) was followed by 30 cycles of amplification consisting of 30 s for denaturation at 95°C, 30 s for annealing at 43°C and 1 min for extension at 72°C, and ended by a 10 min extension at 72°C. For nested PCR, 1 µL of the first round PCR product was used as template in the second round PCR using the internal primer sets (MS-F2: 5'-GGTTGTTTGGCCGTACGGGG-3' and MS-R2: 5'-GGTTGTTTGGCCGTACGGGG-3') and 3.0 µL of extracted DNA suspension. The same PCR conditions were used as for the first round PCR. PCR products were analyzed by 1.5% agarose gel electrophoresis with TAE (40 mM Tris-acetate, 1 mM EDTA) running buffer and visualized with SYBR Safe DNA gel stain (Invitrogen). Expected sizes of amplicons for the first and second PCRs were 1,205 bp and 549 bp, respectively.

### Infection of cultured yellowtail juveniles

After wild yellowtail fingerlings were introduced into sea cages of eight farms located in Nagasaki Prefecture in June 2007, fish were sampled (n = 10–20 per each sampling) at 1, 2, 6, 16 and 25 weeks. Details of col-

<table>
<thead>
<tr>
<th>Location of sea cages</th>
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<th>E</th>
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<tbody>
<tr>
<td>South of Goto Islands</td>
<td>X</td>
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<td>X</td>
<td>Y</td>
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* X is located in close proximity to the land, whereas Y and Z are located offshore.

### Table 1. Information on fish farms of cultured yellowtail juveniles in Nagasaki Prefecture

Wild yellowtail fingerlings (mojako) were captured at four different occasions from several locations around Goto and Tsushima Islands, Nagasaki Prefecture from May to June 2008. Immediately after catch, fish samples (n = 88) were iced on board and frozen at −20°C shortly after landing. The samples were thawed at the laboratory and visually examined for cysts. Presence of spores was also investigated by the Uvitex 2B staining with 25 fish (only large fish from which 1 g of the muscle tissue could be collected), because the remaining 63 fish were not large enough to be tested by the Uvitex 2B method. Additionally, single and nested PCR-based detection assays for *M. seriolae* DNA were performed according to Bell et al. (1999). Briefly, ca. 50 mg sample of musculature was collected from each fish, homogenized, and the DNA was extracted using DNA Mini Kit (Qiagen). PCR reactions were carried out in 20 µL total volume. Each PCR mixture contained 0.1 µL of Takara Ex TaqTM HS (5 UI/µL), 2 µL of 10×Ex Taq Buffer, 1.6 µL of dNTP mixture (2.5 mM each), 0.5 µL (25 µg/µL) of each outer primer (MS-F1: 5'-CACCTGTCTGCAATGCGGG-3' and MS-R1: 5'-GGTTGTTTGGCCGTACGGGG-3') and 3.0 µL of extracted DNA suspension. All PCR reactions were performed in iCycler (Bio-Rad). Denaturation of DNA (95°C for 5 min) was followed by 30 cycles of amplification consisting of 30 s for denaturation at 95°C, 30 s for annealing at 43°C and 1 min for extension at 72°C, and ended by a 10 min extension at 72°C. For nested PCR, 1 µL of the first round PCR product was used as template in the second round PCR using the internal primer sets (MS-F2: 5'-GGTTGTTTGGCCGTACGGGG-3' and MS-R2: 5'-GGTTGTTTGGCCGTACGGGG-3'). The same PCR conditions were used as for the first round PCR. PCR products were analyzed by 1.5% agarose gel electrophoresis with TAE (40 mM Tris-acetate, 1 mM EDTA) running buffer and visualized with SYBR Safe DNA gel stain (Invitrogen). Expected sizes of amplicons for the first and second PCRs were 1,205 bp and 549 bp, respectively.

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<table>
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<tr>
<th>Fish farms</th>
<th>A</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Site of collection of wild fingerlings</td>
<td>South of Goto Islands</td>
<td>Danjo Islands</td>
<td>South of Goto Islands</td>
<td>Danjo Islands</td>
<td>Danjo Islands</td>
<td>West of Goto Islands</td>
<td>South of Goto Islands</td>
<td>South of Goto Islands</td>
</tr>
<tr>
<td>Location of sea cages</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>Y</td>
<td>Z</td>
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</table>

* X is located in close proximity to the land, whereas Y and Z are located offshore.
lection sites of wild fingerlings and the location of sea
cages for the eight farms are summarized in Table 1.
Location of fish farms designated as A–E was located in
close proximity to the land (ca. 20–80 m), whereas sea
cages of farms F, G and H was set up offshore (ca.
100–350 m from the land). The samples were visually
examined for cysts and for spores in Uvitex 2B-stained
smears by fluorescent microscopy as described above.

Exposure of hatchery-reared yellowtail juveniles to
infective water

(1) Determination of days for cyst formation: To
decide the appropriate rearing period of exposed fish in
the next experiment, days required for cyst formation
were investigated. Hatchery-produced yellowtail juve-
niles were obtained from the Goto Station, National
Center for Stock Enhancement (NCSE) for the following
experiments. Experimental fish (n = 150, body length =
ca. 5 cm, body weight = ca. 2 g) were stocked in a sea
cage of Nagasaki Prefectural Institute of Fisheries
Sciences (NPIFS), where persistent infection with M.
seriolae has been detected every year. After being
transferred to sea cage on 25 June 2009, five fish were
randomly collected every day for 4 weeks. The sam-
ple were examined for cysts macroscopically and for
spores microscopically as described above.

(2) Determination of infective period: Hatchery-
reared fish were kept in a land-based tank (200 L) sup-
plied with sand-filtered sea water which was further
passed through a cartridge filter with 1-µm openings.
The inflow was adjusted to ca. 10 L/min. The fish were
divided into eight groups (30–40 fish per one group),
which were held in a sea cage of NPIFS for various 1-
week periods between 18 June and 25 August 2009 to
confirm the infective stage of M. seriolae in the marine
environment. After each exposure period, the yellow-
tail were transferred back to the land-based tanks
receiving flow-through of filtered sea water to prevent
their re-exposure to the parasite's infective stage.
After 3 weeks of subsequent rearing (the period deter-
dined in the above experiment), the fish were examined
for the presence of cysts as described above. The
negative control group was kept in the land-based tank
during the study period. To avoid size effects on the
parasite infection, fish were constrained from growing
by feeding only twice a week.

(3) Effects of fish size on infection: Hatchery-reared
yellowtail juveniles were provided from the Goto Station,
NCSE in February (the first batch of breeding) and June
(the second batch of breeding), 2010. Fish were kept
in the land-based tank described above until use. Fish
from the first batch were divided into three groups
based on their size; mean body weight and fork length
were 123.8 g and 20.0 cm for 'large-size', 45.8 g and
15.2 cm for 'medium-size', and 27.0 g and 12.7 cm for
'small-size'. Fish from the second batch (mean body
weight and fork length were 1.0 g and 4.4 cm, respec-
tively) were used as an 'extremely small-size' group.
The four different size classes were hold separately in
columnar net cages which were placed in a 2-ton cir-
cular tank with flow-through unfiltered sea water. After 1
week of exposure, all fish were transferred to land-based
tanks (500 L tank for the large, medium and small-size
groups, and 200 L tank for the extremely small-size
group) supplied with seawater filtered with a sand filter
and with 1.0 µm-cartridge filters. After 1 month of rear-

Fig. 1. Prevalence (lines) and mean intensity (bars) of Microsporidium seriolae in the 2006–2009 cohorts of amberjack (closed
circle, black bar) and yellowtail (open circle, gray bar) cultured at a fish farm in Miyazaki Prefecture. Vertical lines for preva-
ience and mean intensity of infection show the confidence intervals at 95% in reliability and standard deviations, respectively.
Data of mean intensity of infection in 2007 cohort amberjack is not shown.
2006 cohort reached up to 16 cysts/fish in August 2006, and visually detectable cysts still remained at the end of the survey. In contrast, 2007 cohort yellowtail and amberjack had lower prevalence and mean intensity than those of the 2006 cohort even at the early period of infection (10% in July for yellowtail). Mean intensity was as low as 2 cysts/fish over the study period both for yellowtail and amberjack. Most cysts became brown in color, progressed to black, and then shrunk to negligible size (< 1 mm long) by the end of the survey (Fig. 2). However, spores were detected by Uvitex 2B staining from more than 90% fish of the 2007 cohort even in the early period of infection (Fig. 3). For the 2008 cohort, no infection was observed in yellowtail fingerlings just after catch (May 2008), but cysts appeared in more than 80% fish in June 2008 (Fig. 1). Prevalence declined substantially over time, and only a few cysts were found in November 2009. Although monitoring for the 2009 cohort was ended in November 2009, overall levels of infection in yellowtail were relatively moderate. In all cohorts of fish groups, neither relapse of prevalence nor young cysts (visible as small and white cysts) were observed in the following year.

Histological observations showed that cysts degenerated by host responses over time (Fig. 4). Masses of spores were encapsulated by host’s connective tis-

**Results**

**Seasonal infection**

Annual and seasonal patterns in prevalence and mean intensity of infection were observed in the 4-year cohorts (Fig. 1). Prevalence in the 2006 cohort of amberjack was 100% in July and August 2006, decreased in autumn and winter, but was still 80% in September 2007. Mean infection of infection in the

![Image](https://example.com/image.png)

Fig. 2. Recovery process of the beko disease in yellowtail. Arrows show a developing cyst (A: 3 months post-introduction to sea cage, 2009-cohort), a blackened cyst (B: 6 months post-introduction, 2008-cohort) and degenerative scars (C: 17 months post-introduction, 2007-cohort).

![Image](https://example.com/image.png)

Fig. 3. Prevalence of *Microsporidium seriolae* in the 2007 and 2009 cohorts of yellowtail. Closed and open circles represent the prevalence determined by visual observation of cysts and microscopic examination for Uvitex 2B-stained spores, respectively.
Infection dynamics of bekofus disease

**Infection of wild yellowtail fingerlings**

Neither cysts nor spores were observed in any of wild yellowtails caught around Goto or Tsushima islands (88 fish for visual observation and 25 fish for UVitex 2B examination). Using the nested-PCR, none of the 88 fish were positive for *M. seriolae* DNA in the first round PCR but six fish were positive (6.8%) after the second amplification (Table 2). There was no remarkable difference in the prevalence of infection among the four fish lots investigated.

**Infection of cultured yellowtail juveniles**

Cysts appeared at 2 weeks post-introduction to sea cages in seven out of eight farms, and prevalence of infection increased to more than 90% at 6-25 weeks in all farms studied (Fig. 5). Distribution of *M. seriolae* seemed homogenous, regardless of different origin of wild fingerlings and different locations of sea cages.

**Days required for cyst formation**

Cysts were first observed in yellowtails 20 days post-exposure (Fig. 6). The water temperature increased gradually and over 26°C when cysts formed in the muscles. At that time, mean intensity of infection was 8.3 cysts/infected fish, and the mean length of cyst was 3.8 mm.

**Infective period**

Almost 100% of the fish in the initial four groups exposed between 18 June and 16 July were found to be infected with *M. seriolae* (Fig. 7). Starting with the 5th group exposed between 16 and 23 July, the prevalence of infection gradually decreased and was down to zero in the last group exposed between 18 and 25 August. Mean intensity of infection peaked at 25.8 cysts/infected fish for the 2nd group, and declined over time. Mean length of cysts was almost constant at approximately 6
Fig. 6. Daily monitoring of Microsporidium seriola in infection in hatchery-reared yellowtail juveniles stocked in a net cage of Nagasaki Prefectural Institute of Fisheries Sciences. Solid and broken lines represent the prevalence of infection (n = 5 for each sampling) and water temperature, respectively.

Fig. 7. Prevalence (lines) and mean intensity (bars) of Microsporidium seriola in juvenile yellowtail stocked in a net cage of Nagasaki Prefectural Institute of Fisheries Sciences. Each fish group was reared for eight 1-week intervals from 18 June to 25 August, 2010, followed by examination for the presence of cysts after 3 weeks.

Relationship between fish size and infection

Water temperature varied between 22.5°C and 25.1°C during the experimental period (from 23 June to 26 July, 2010). Prevalence of infection was not significantly different (approximately 70–80%) among the large, medium and small-size groups, while it was significantly lower (2.3%) in the extremely small-size group compared to the others (Table 3). Mean intensity of infection was significantly lower (7.9 cysts/infected fish)

Table 3. Comparison of prevalence and intensity of infection with Microsporidium seriola among four different size classes of yellowtail juveniles (n = 44 for each class). Different letters (a and b) in the columns on the prevalence and intensity of infection mean significant differences among different size groups (P < 0.05; Ryan’s test and Steel-Dwass test for prevalence and intensity, respectively). The data of mean intensity of infection for extremely small-size group is not included in the statistical analysis due to the small sample size (n=1).

<table>
<thead>
<tr>
<th>Fish size group (initial mean weight and fork length)</th>
<th>Mean body weight at the end of the experiment (g)</th>
<th>Mean fork length at the end of the experiment (cm)</th>
<th>Prevalence of infection (%)</th>
<th>Mean intensity of infection (mean number of cysts/infected fish)</th>
</tr>
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<tbody>
<tr>
<td>Large (123.8 g, 20.0 cm)</td>
<td>151.2</td>
<td>22.2</td>
<td>81.8a</td>
<td>7.9b</td>
</tr>
<tr>
<td>Medium (45.8 g, 15.2 cm)</td>
<td>65.6</td>
<td>17.3</td>
<td>66.2b</td>
<td>12.5b</td>
</tr>
<tr>
<td>Small (27.0 g, 12.7 cm)</td>
<td>54.4</td>
<td>15.9</td>
<td>70.5b</td>
<td>13.6b</td>
</tr>
<tr>
<td>Extremely small (1.0 g, 4.4 cm)</td>
<td>5.4</td>
<td>7.8</td>
<td>2.3c</td>
<td>6.0</td>
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in the large group compared to the medium (12.5) and small-size (13.6) groups (Table 3).

Discussion

This is the first study that describes the whole infection dynamics of the beko disease from the infection period to the recovery phase in cultured yellowtail. The reason for annual variation in the prevalence and mean intensity of *M. seriolae* among 2006–2009 cohorts is unknown. Because it is unlikely that the variation is caused by the origin of fingerlings and the culture site, it may be partly explained by different water temperature, variable biomass of the unknown intermediate host or prevalence of infective stage in the intermediate host. Disease occurrence must be subject to complex interactions between the host and the parasite under specific environmental influences (Sano et al., 1998). It is notable that most of fish were found to be subclinically infected in the early period of infection in the 2007 cohorts. Most of parasites might have cleared without developing into visible cysts. The recovery phase depended on severity of infection in fingerlings during the initial 1–2 months. Fish could recover from the infection in the second year when the mean intensity of infection was as low as 5 cysts/fish in June or July in the first year. No recurrence of the beko disease was found in the following year, suggesting that a resistance to the disease was established in surviving fish.

Our observations of wild fingerlings and cultured juveniles suggest that *M. seriolae* infection occurred after introduction of wild fingerlings to sea cages. Although some wild fingerlings were found to be infected, the infection was extremely light. Considering the lack of direct transmission of *M. seriolae* among fish hosts, it is unlikely that the infected wild fingerlings have caused a horizontal transmission of the parasite from fish to fish within a sea cage. These findings imply that selection of wild fingerling lots is not effective in preventing the beko disease. The parasite infection occurred both in coastal and offshore cages, suggesting that the site selection is not a promising prevention method at least in the farming area examined in this study.

The results on the infective period indicate that infectivity of *M. seriolae* decreased after the end of July. This seems to correspond with the findings by Sano et al. (1998), who observed a low infection level in yellowtail juveniles stocked in a sea cage in the end of July. However, we could not determine a starting point of the infective period, because prevalence of infection in the initial group was already almost 100%. Delayed introduction of yellowtail juveniles from non-endemic areas after end of the infective period (in August) is likely effective in avoidance of *M. seriolae* infection.

The prevalence and mean intensity of infection in the extremely small-size group (mean weight; 1 g) was the lowest among the fish groups studied. It may be explained by alteration of feeding habit, although the route of infection with *M. seriolae* has not yet been elucidated. On the other hand, Sano et al. (1998) reported that two different fish size classes (mean weight; 0.3 g and 22.0 g) were infected with *M. seriolae* to a similar extent (64.5% and 95%, respectively). The two experimental designs are different in exposure periods; fish in the present study were exposed only for one week, whereas fish in Sano et al. (1998) were exposed continuously for 43 days. It is possible that fish in Sano et al. (1998) became susceptible to *M. seriolae* with the growth of fish. Low susceptibility of extremely small-size fish seems to last only for a short time, because fish grow rapidly. Thus, it may be difficult to put the result into practice. As far as the three groups larger than 27 g in initial weight, larger fish had a lower intensity than smaller fish. This suggests that it is effective to promote growth of fingerlings as large as possible before the peak period of infection. Generally, no practical chemical treatment has been available for microsporidian infections. Effective control may be achieved with integrated management of a combination of strategies (Ogawa & Yokoyama, 1998). Sano et al. (1998) indicated that sand filtration of rearing water is effective in removing possible infective agents of *M. seriolae*, although this method is not applicable for cage culture. Considering the findings of the infective period obtained by Sano et al. (1998) and the present study, it is recommended to use a land-based tank supplied with filtered seawater until after the peak period of infection (from mid-June to mid-July). In a practical point of view, it seems to be most reasonable to depend on natural recovery process. However, heavily infected fish (like the 2006 cohort) hardly recover, suggesting that juveniles with signs of the disease should be disposed as early as possible, e.g. at the time of a vaccination for viral/bacterial infections usually applied around July in the first year.

Acknowledgements

We greatly acknowledge the Goto Station, National Center for Stock Enhancement, Fisheries Research Agency, for providing yellowtail fingerlings as the experimental fish, and the fish farmers in Miyazaki and Nagasaki Prefectures for kind offer of fish samples. We also thank Dr. Daniel Grabner of the University of Tokyo for his reviewing this manuscript.

References


微胞子虫 Microsporidium seriola によるブリ類のぺこ病の感染動態

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高見生雄・横山文彦・小川和夫

ブリ類のぺこ病について、感染成立から治癒に至る過程を調べた。養殖魚の4年間にわたり調査の結果、感染は稚苗導入直後の6〜7月に起こり、寄生率は約1ヶ月で最大100%に達した。シストは翌年春までに徐々に黒化して消滅したが、初期の寄生強度が高い年は一部が残存した。発生海域への人工稚苗の移入実験により、8月中旬以後には魚への侵入が終息すること、魚のサイズが寄生率や寄生強度に影響することが明らかになった。

魚病研究. 46(2), 51-58 (2011)