Viral Diseases in Cultured Marine Fish in Japan

Kazuhiro Nakajima¹, Kiyoshi Inouye² and Minoru Sorimachi³

¹National Research Institute of Aquaculture, Nansei, Mie 516-0193, Japan
²National Research Institute of Aquaculture, Inland Station, Tamaki, Mie 519-0414, Japan
³Toyama Prefectural Fisheries Research Institute, Namerikawa Toyama 936-8536, Japan

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In Japan, mass mortalities of cultured fish due to infectious diseases have been often reported. Among the pathogens, viruses are the most devastating infectious agents that afflict fish. This paper reviews the major viral diseases of cultured marine fish in Japan. The major diseases caused by DNA viruses include viral epidermal hyperplasia in flounder and herpesviral disease in coho salmon, both caused by a hepesviruses and lymphocystis disease and another iridoviral disease in red sea bream and other marine fishes. The major diseases caused by RNA viruses are viral ascites (VA), viral deformity (VD) in yellowtail, both caused by a birnavirus (YAV or VDV), erythrocytic inclusion body syndrome (EIIBS) in coho salmon, a rhabdoviral disease in flounder and viral nervous necrosis (VNN) in striped jack and several other fishes by a nodavirus. The causative virus of Kuchijirosho in tiger puffer has not been classified.

Key words: viral disease, cultured marine fish, aquaculture, birnavirus, iridovirus

Marine aquaculture has been developing rapidly in Japan. Annual production of cultured marine fish has constantly increased (Table 1). Total production of cultured marine fish in 1995 was 279,182 t. Yellowtail (Seriola quinqueradiata) was the most important species, with 169,765 t or 60.8% of the total, followed by red sea bream (Pagrus major) and coho salmon (Oncorhynchus kisutch) with 72,185 t and 13,524 t, respectively (Table 1). The damage to finfish aquaculture by diseases was estimated at 28 billion yen in 1994. Thus, 7.7% of the total value of production was lost as a result of diseases.

In the latter half of the 1980s, the diversity of marine species in hatcheries and farms began to increase. However, the rapid development of aquaculture has resulted in the increased occurrence of diseases, including several that have not been previously described. This has been caused by the intensification of marine fish culture exploiting new species of fish and by recent introductions of foreign pathogens. Among pathogens, viruses are generally the most devastating infectious agents that afflict fish. In the 1990s, the list of viral diseases associated with cultured marine fish has continued to grow and mass mortalities have become more frequent. In particular, emerging viral diseases such

Table 1. Changes in annual production of cultured marine fish in Japan

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<tbody>
<tr>
<td>Yellowtail</td>
<td>Seriola quinqueradiata</td>
<td>92,352</td>
<td>149,311</td>
<td>150,961</td>
<td>161,106</td>
<td>169,765</td>
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<td>Red sea bream</td>
<td>Pagrus major</td>
<td>4,303</td>
<td>14,757</td>
<td>28,430</td>
<td>51,616</td>
<td>72,185</td>
</tr>
<tr>
<td>Japanese flounder</td>
<td>Paralichthys olivaceus</td>
<td>-</td>
<td>-</td>
<td>1,572</td>
<td>6,039</td>
<td>6,845</td>
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<tr>
<td>Jack mackerel</td>
<td>Trachurus japonicus</td>
<td>923</td>
<td>2,283</td>
<td>5,008</td>
<td>5,863</td>
<td>4,999</td>
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<tr>
<td>Puffer</td>
<td>Takifugu rubripes</td>
<td>11</td>
<td>69</td>
<td>750</td>
<td>2,895</td>
<td>4,031</td>
</tr>
<tr>
<td>Striped jack</td>
<td>Pseudocaranx dentex</td>
<td>22</td>
<td>228</td>
<td>461</td>
<td>1,368</td>
<td>2,653</td>
</tr>
<tr>
<td>Coho salmon</td>
<td>Oncorhynchus kisutch</td>
<td>-</td>
<td>1,855</td>
<td>6,990</td>
<td>23,608</td>
<td>13,524</td>
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<tr>
<td>Others</td>
<td></td>
<td>385</td>
<td>1,214</td>
<td>1,344</td>
<td>2,991</td>
<td>5,180</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>97,996</td>
<td>169,717</td>
<td>195,516</td>
<td>255,506</td>
<td>279,182</td>
</tr>
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</table>
as viral nervous necrosis (VNN) (Nakai et al., 1995) and iridoviral disease of cultured marine fish (Inouye et al., 1993) are causing serious damages to the industry. This paper reviews the current status of viral diseases of cultured marine fish in Japan. Especially, among them, iridoviral disease, viral ascites and viral deformity are described in detail based on the results of our own studies.

Major Viral Diseases of Cultured Marine Fish in Japan

Recently, several new viral diseases have been reported in cultured marine fish. In some cases, the causative agents have been observed by electron microscopy and isolated in established fish cell lines. In other cases the virus was observed by electron microscopy but could not be replicated in cultured cells. Viral diseases of cultured marine fish have been grouped in various ways. In this paper, the causative viruses are classified by their nucleic acid as follows: 1) diseases caused by DNA viruses 2) diseases caused by RNA viruses, and 3) diseases caused by unclassified viruses (Table 2).

The diseases caused by DNA viruses

The diseases caused by DNA viruses which belong to the family Iridoviridae and the family Herpesviridae include lymphocystis, iridoviral disease of various marine fishes, viral epidermal hyperplasia of Japanese flounder Paralichthys olivaceus, and herpesviral disease of coho salmon. The causative agent of viral epidermal hyperplasia has not been isolated in cell cultures.

1) Lymphocystis disease

Lymphocystis has been observed in several marine

<table>
<thead>
<tr>
<th>Name of virus</th>
<th>Abbreviation</th>
<th>Status</th>
<th>Name of disease</th>
<th>Major species affected</th>
<th>Reference</th>
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<tr>
<td>DNA virus</td>
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<td>lymphocystis disease virus</td>
<td>LCDV-2</td>
<td>I</td>
<td>Lymphocystis disease</td>
<td>Japanese flounder</td>
<td>Miyazaki &amp; Egusa (1972)</td>
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<tr>
<td>red sea bream iridovirus</td>
<td>RSIV</td>
<td>I</td>
<td>Iridoviral disease</td>
<td>Red sea bream</td>
<td>Inouye et al. (1992)</td>
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<td>Herpesviridae</td>
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<tr>
<td>flounder herpesvirus</td>
<td>FHV</td>
<td>EM</td>
<td>Viral epidermal hyperplasia</td>
<td>Japanese flounder</td>
<td>Iida et al. (1989)</td>
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<td>salmonid herpesvirus 2</td>
<td>SaHV-2</td>
<td>I</td>
<td>Herpesviral disease</td>
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<td>Kumagai et al. (1994)</td>
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<td>RNA virus</td>
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<td>Binnaviridae</td>
<td></td>
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<td>yellowtail ascites virus</td>
<td>YAV</td>
<td>I</td>
<td>Viral ascites</td>
<td>Yellowtail</td>
<td>Sorimachi et al. (1985)</td>
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<tr>
<td>viral deformity virus</td>
<td>VDV</td>
<td>I</td>
<td>Viral deformity</td>
<td>Yellowtail</td>
<td>Nakajima et al. (1993)</td>
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<td>Rhabdoviridae</td>
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<td>hirame rhabdovirus</td>
<td>HIRRV</td>
<td>I</td>
<td>Rhabdoviral disease</td>
<td>Japanese flounder</td>
<td>Kimura et al. (1986)</td>
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<tr>
<td>Nodaviridae</td>
<td></td>
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<tr>
<td>striped jack nervous necrosis virus</td>
<td>SJNNV</td>
<td>EM*</td>
<td>Viral nervous necrosis</td>
<td>Striped jack</td>
<td>Yoshikoshi &amp; Inoue (1990)</td>
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<tr>
<td>Togaviridae (?)</td>
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<tr>
<td>erythrocytic inclusion body syndrome</td>
<td>EIBSV</td>
<td>EM</td>
<td>Erythrocytic inclusion body syndrome</td>
<td>Coho salmon</td>
<td>Takahashi et al. (1992)</td>
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<tr>
<td>Unclassified</td>
<td>?</td>
<td>I</td>
<td>Kuchijirosho (Snout ulcer disease)</td>
<td>Tiger puffer</td>
<td>Inoue et al. (1986)</td>
</tr>
</tbody>
</table>

I = isolated, EM = electron microscope observation
* Piscine neuropathy nodavirus from sea bass was isolated in fish cell line (SNN-1) (Frerichs et al., 1996)
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fish species, such as sea bass *Lateolabrax japonicus*, yellowtail, red sea bream, and Japanese flounder with typical surface lesions (Miyazaki and Egusa, 1972). Lymphocystis cells are observed mainly on the fins or body surface. Although the disease is known in a great variety of marine, estuarine and freshwater fish species in the world, the damages caused by this disease were not serious in Japan. However, the incidence of the disease in Japanese flounder has been increasing in the 1990s (Matsuoka, 1995).

2) Iridoviral disease

Introduction: The first outbreak of an iridoviral disease was recorded in cultured red sea bream in Shikoku Island, Japan, in 1990. Since 1991, the disease has produced mass mortalities of cultured fish populations in the western part of Japan, mainly among juvenile red sea bream. However, mortalities of market-sized fish have also been reported.

Clinical signs and gross pathology: The affected fish were lethargic and exhibited severe anemia, petechiae of the gills, and enlargement of the spleen (Inouye et al., 1993). The disease was histopathologically characterized by the appearance of enlarged cells that stained deeply with Giemsa solution in the spleen, heart, kidney, liver, and gills.

Causative virus: It was reported that the causative agent was a large, icosahedral, cytoplasmic DNA virus classified as a member of the family *Iridoviridae*. The virus was tentatively designated as red sea bream iridovirus (RSIV). Each virion consists of a central electron-dense core (120 nm) and an electron translucent zone, measuring 200–240 nm in diameter. Biological and physico-chemical properties of the virus have been reported (Nakajima and Sorimachi, 1994b). RSIV is grown on GF, BF-2, CHSE-214, FHM, JSKG, KRE-3, RTG-2 and YTF cell lines. The titers of the virus on GF, BF-2 and KRE-3 are higher than those on the other cell lines. RSIV is replicated at 15°C, 20°C, 25°C and 30°C but not at 37°C. The suitable temperature for viral growth is 20°C or 25°C. The virus is sensitive to acid (pH3), chloroform and ether and unstable to heat but stable to ultrasonic treatment and repeated freezing and thawing. Treatment with 5-iodo-2-deoxyuridine (IUdR) reduced the titer of the virus.

Diagnosis: Diagnostic methods such as the observation of stamped or sectioned specimens stained with Giemsa, an immunofluorescence test with a MAb and PCR have been reported (Nakajima and Sorimachi, 1995, Nakajima et al., 1995, Kurita et al., 1998). The indirect immunofluorescence test with a MAb is commonly used for the rapid diagnosis of RSIV infected fish in the field.

Host range: The same type of disease has also seriously damaged stocks of several kinds of cultured marine fish such as yellowtail, sea bass, and Japanese parrotfish (*Oplegnathus fasciatus*). It is known that the iridoviral disease has afflicted 20 species of cultured marine fish in 17 prefectures located in the south-western part of Japan from 1991 to 1995. The infected fish include 18 Perciformes, 1 Pleuronectiformes and 1 Tetradontiformes (Matsuoka et al., 1996).

Transmission: Iridoviral disease has not occurred in hatcheries. So the possibility of vertical transmission seems to be little. The horizontal infection of RSIV was confirmed by experimental infection. The disease was transmittable through co-habitation or the water from the tanks rearing RSIV infected fish (unpublished data).

Comparison with other fish iridoviruses: The antigenic relationship between RSIV and two iridovirus-like agents associated with systemic infection in fish, epizootic haematopoietic necrosis virus (EHNV) and the iridovirus isolated from sheatfish (SFIV) were examined. Although cross reactivities were observed between RSIV and other fish iridoviruses by IF test or immunoprecipitation test using anti-RSIV serum, none of the MAbs against RSIV reacted with EHNV or SFIV-infected cells by the IF test (Nakajima et al., 1998a). Pathogenicities of EHNV or SFIV to red sea bream were not shown by experimental challenge (Nakajima et al., 1998b, in press).

Antibody response of RSIV-infected fish: The sera obtained from experimentally and naturally RSIV-infected fish reacted with 46 kDa polypeptide by radioimmunoprecipitation test (unpublished data). The sera obtained from vaccinated fish also reacted with 46 kDa polypeptide. This show the possibility that the antibody to this polypeptide is important on the humoral immunity against iridoviral disease.

This disease was first reported in 1990 and spread various kinds of cultured marine fishes in Japan. But the infection source of this disease is not clear. There is a possibility that the disease was imported from foreign countries with seedlings, but the confirmation has not been made. This virus is detected from diseased fish mainly in summer but not in winter time. The existence of the virus is not sure in winter, therefore, the epidemiological studies are needed to clarify the ecology of the pathogen.
RSIVs isolated from various fish species in Japan are shown to be closely related to each other by reaction patterns against antibodies and virion polypeptide profiles (Nakajima et al., 1998a). In relation to the infection source, the comparison between RSIV and other iridoviruses isolated in foreign countries especially in south east Asia from where Japan has imported a lot of seedlings is needed.

At present, there is no control measures for this disease in the farms. For the control of this disease, we have developed a vaccine which shows a significant effectiveness in red sea bream under both experimental and field conditions (Nakajima et al., 1997). However, further studies are needed on the route of administration of this vaccine and the effectiveness of the vaccine for other cultured marine fishes such as yellowtails.

3) Viral epidermal hyperplasia

Viral epidermal hyperplasia was first observed in the late 1980s in larval and juvenile Japanese flounder (Iida et al., 1989) and is still causing mass mortalities in hatchery-reared flounder larvae. The disease most commonly occurred in 10–30 day old fish that were reared at about 18–20°C, and sometimes the mortality reached 80 to 90% in 1 week. Hyperplasia was observed in the epidermal layer of the fins and skin by histopathological methods. In addition, herpesvirus particles were observed in the nucleus and cytoplasm of infected epidermal cells by electron microscopy. Electron microscopy revealed hexagonal virus particles in the nucleus (100–140 nm in diameter without an envelope) and cytoplasm (190–230 nm with an envelope) of the affected epidermal cells. Replication of the virus in commonly available fish cell lines has not been demonstrated. Fertilized eggs are disinfected with iodine, however proper prophylactic measures have not been suggested.

4) Herpesviral disease of maricultured coho salmon

Herpesviral disease of maricultured coho salmon was first reported in Miyagi Prefecture in 1988 (Kumagai et al., 1994). Cumulative mortalities ranged from 10 to 30%. Diseased fish were 100–1000 g in body weight and exhibited excoriations of the fin, erosion and ulcers on the body surface and pale spots in the liver. Necrosis of hepatocytes was the most apparent histopathological change in the diseased fish. Numerous virions having an icosahedral shape were observed in the necrotic hepatocytes. Negatively stained virus particles revealed nonenveloped virions (112 nm diameter) and envelope particles (242 nm diameter). The virus was identified as salmonid herpesvirus type 2 by morphological characteristics and serological tests. Recently, Kumagai et al. (1997) suggested that the source of infection might be asymptotically infected other salmonid species reared with coho salmon in freshwater at hatcheries and the transportation of infected fish has spread the virus. Based on these, they established practical control methods that have been effective for reducing the prevalence.

The diseases caused by RNA viruses

The diseases caused by RNA viruses which belong to the families Birnaviridae, Rhabdoviridae, Nodaviridae, and Togaviridae (?) include viral ascites, viral deformity of yellowtail, and rhabdoviral disease and viral nervous necrosis (VNN) of various cultured marine fishes. The causative agents of these diseases have been isolated using established fish cell lines except for the causative agent of VNN.

1) Viral ascites

Introduction: In the summer of 1983, an acute disease characterized by ascites occurred among cultured juvenile yellowtail in the Inland Sea of Japan. From 1983 to 1985, the disease caused serious damages to the industry with 80–90% mortalities but in recent years, the disease has not seriously affected fish stocks. Epizootics generally occur during May through June at water temperatures of 18–22°C. Fish less than 10 g in body weight are most susceptible to this virus.

Clinical signs and gross pathology: The moribund juveniles typically show anemic gills, hemorrhaging in the liver, and severe ascites. The most characteristic pathological changes observed in naturally infected fish are extensive necrosis in the pancreatic acinar cells, and hepatic parenchymal cell necrosis and hepatic hemorrhages (Egusa and Sorimachi, 1986).

Causative virus: A birnavirus designated as yellowtail ascites virus (YAV) was identified as the causative agent of the disease (Sorimachi and Hara, 1985). Electron microscopy revealed pentagonal or hexagonal particles of 62–69 nm in diameter. The virus is replicated in CHSE-214, RTG-2 and EK-1 cells at temperatures between 5 and 30°C. The virus is resistant to pH3, pH11, ether and chloroform and stable at 56°C for 30 min. Replication of the virus is not inhibited by 5-Iodo-2'-deoxyuridine.

Susceptible species: The susceptibility of juveniles of 5 different marine fishes to YAV was studied by experimental infection (Isshiki and Kusuda, 1987). Among the fish examined, Japanese parrotfish, hybrid
of goldstriped amberjack (Seriola aureovittata) and amberjack (Seriola dumerili) were sensitive to the virus, whereas spotted parrotfish (Oplegnathus punctatus) and red sea bream were not sensitive to the virus. However, spotted parrotfish was sensitive to the virus in our experiment (unpublished data).

**Transmission:** Presence of YAV was examined in the brood stocks of yellowtail before and after maturation-inducing treatment and YAV was frequently detected from the eggs and ovarian fluid and occasionally from the seminal fluid of gonadotropic hormone (GTH)-treated fish, indicating that YAV is transmitted vertically to offsprings (Isshiki et al., 1993). Horizontal transmission was also suspected because the YAV-infected wild fish such as file fish (Stephanolepis cirrhifer) was found. But the spread of the disease among net pens is rare. So the possibility of the horizontal transmission is low. Until now, the horizontal transmission has not been shown by experimental infection.

2) **Viral deformity**

**Introduction:** A new viral disease characterized by abnormal swimming behavior, deformity of the body, and high mortalities was detected in juvenile yellowtail at a hatchery in Kyushu Island, Japan (Nakajima et al., 1993). Fish less than 10 g in body weight are most susceptible to the virus.

**Clinical signs and gross pathology:** Severe congestion of the liver, edema, anemia in the kidney and spleen, and congestion in various parts of the brain were observed in diseased fish (Maeno et al., 1995; Maeno and Nakajima, 1997). In the brain of fish exhibiting an abnormal swimming behavior and deformity, severe congestion and hemorrhages were routinely observed.

**Causative virus:** A birnavirus designated as viral deformity virus (VDV) belonging to the family Birnaviridae was identified as the causative agent of the disease. Electron microscopy revealed that the virions are non-enveloped and hexagonal in shape with a diameter of 65–69 nm. The virus is replicated and produces cytopathic effects on CHSE-214, EPC, BF-2, FHM, EK-1 and RTG-2 cells. Replication of the virus is inhibited by IUdR. The virus is resistant to pH3, pH11, ether and chloroform and stable at 56°C for 30 min.

**Comparison between YAV and VDV:** Two birnaviruses, viral deformity virus (VDV) and yellowtail ascites virus (YAV) isolated from cultured yellowtail were examined for their serological and biochemical properties (Nakajima and Sorimachi, 1994a). Cross-neutralization studies showed that VDV was closely related to YAV but clearly distinct from the 3 type strains of infectious pancreatic necrosis virus (IPNV). VDV and YAV contained two genome segments and mobility of the smaller segment slightly differed between the two. Polypeptide electropherotypes showed a distinct difference between VDV and YAV. The differences in biochemical properties, clinical signs and histopathological changes between YAV- and VDV-infected fish indicate that YAV is distinct from VDV. However, MAbs against YAV and VDV showed similar reaction patterns to YAV and VDV but distinct from those to IPNV (Nakajima and Sorimachi, 1996a, b). These results showed that these two viruses are serologically similar and antigenic epitopes are conserved, but the clinical signs caused by these viruses are completely different. So, further studies are in progress to identify the regions of the genome which are important for the pathogeneicities of these viruses.

3) **Rhabdoviral disease**

Rhabdovirus infection of Japanese flounder first occurred in 1984. The causative virus was designated hirame rhabdovirus (HIRRV = HRV, Kimura et al., 1986). The signs of the disease are congestion of the gonads, focal hemorrhages in the skeletal muscle and fins, and the accumulation of ascitic fluid (Oseko et al., 1988a). HIRRV has a wide host range including ayu Plecoglossus altivelis, black sea bream Acanthopagrus schlegeli, and darkbanded rockfish Sebastes inermis (Yoshimizu et al., 1987). Virus particles are bullet-shaped and measure 80 nm × 189 to 200 nm. HIRRV has been placed into the lyssavirus genus because it is composed of a negative-sense, single-stranded RNA genome (Nishizawa et al., 1991) and 5 structural proteins. Keeping the water temperature at 15°C or higher seems to be an effective measure for minimizing the occurrence of the disease (Oseko et al., 1988b).

4) **Viral nervous necrosis**

VNN has been reported to cause high mortalities in hatchery-reared larvae and juveniles of marine fishes in Japan such as Japanese parrotfish, red spotted grouper (Epinephelus akaara), striped jack (Pseudocaranx dentex), Japanese flounder, tiger puffer (Takifugu rubripes), kelp grouper (Epinephelus moara), and barfin flounder (Verasper moseri) (Yoshikoshi and Inouye, 1990; Mori et al., 1991; Arimoto et al., 1993; Nakai et al., 1994; Nguyen et al., 1994). The disease is characterized histopathologically by vacuolation of the retinal and brain tissues (Yoshikoshi and Inouye, 1990). The
causative virus is nonenveloped, round-shaped virions, approximately 25–30 nm in diameter that has been identified as a member of the family Nodaviridae (Mori et al., 1992). Recently the causative nodavirus of sea bass (Dicentrarchus labrax) was isolated in SSN-1 cell derived from snakehead (Frerichs et al., 1996). The striped jack nervous necrosis virus (SJNNV) genome consists of two (RNA1 and RNA2) single-stranded positive sense RNAs lacking poly(A). On the basis of the nucleotide sequence of the genome, primers were designed to amplify a portion of the SJNNV genome by PCR. This technology has been applied to the detection of SJNNV from affected larval striped jack (Nishizawa et al., 1994) and has been used to prevent transmission of the disease in larval production facilities (Mushiake et al., 1994).

5) Erythrocytic inclusion body syndrome
Erythrocytic inclusion body syndrome (EIBS) is a serious viral disease of salmonid fish. Epizootics attributed to EIBS has occurred among populations of coho salmon cultured in seawater in Japan (Takahashi et al., 1992). The principal signs in moribund fish include severe anemia, erythrocytes with characteristic inclusion bodies, and a yellowish colored liver. Characteristic inclusion bodies in erythrocytes contain enveloped viral particles with a diameter of approximately 77 nm. Although the causative viral agent has not been isolated, it most likely is a member of the family Togaviridae (Arakawa et al., 1989).

Diseases caused by unclassified virus
1) Kuchijirosho (Snout ulcer disease)
Tiger puffer affected with Kuchijirosho (snout ulcer disease) showed necrosis around the mouth and aggressive biting behavior. Viral particles were observed in the brain of diseased fish by electron microscopy. The causative virus was isolated in primary cells (PFG cells) but has not been classified (Inouye et al., 1992). Based on the results of the virus isolation, experimental infection and filtration tests, it has been hypothesized that Kuchijirosho is an infectious disease caused by a virus with a size less than 50 nm in diameter.

Discussion
The list of viral diseases for marine fish continues to grow with increasing economic losses resulting in Japan in recent years. This could be attributed to the rapid development of aquaculture, particularity intensive culture system, and species diversification and introduction of seed fish from foreign countries without any quarantine system. These diseases are predisposed by reduced resistance of the farmed fish, owing to environmental deterioration and over stocking, or caused by new foreign pathogens. Therefore, the prevention and control of viral diseases requires fundamental improvements in culture system including legislations. A quarantine system requiring health certification for specific diseases for the importation of fish and shellfish was introduced in 1996.

At present, no antibiotics or chemotherapeutic compounds are effective as virustats or virucides. Destruction of infected stocks and disinfection of the facilities continue to be the main methods used to control viral diseases. To avoid the spread of the pathogen from infected stocks, early and sensitive disease diagnosis is required. SJNNV appears to be transmitted vertically from female and male spawners to their offsprings through the fertilized eggs (Mushiake et al., 1994). By introducing a PCR-based method of detecting the virus from asymptomatic spawners, VNN outbreaks among offsprings in seed production facilities have been significantly reduced. However, detection methods for identifying carriers remain to be established for other viral diseases in mariculture. Strict quarantine and epidemiological studies are also necessary to prevent horizontal transmission. The development of preventive measures such as vaccines is very important. For the iridoviral disease of cultured marine fish, the effectiveness of vaccination with formalin-inactivated vaccine against iridoviral disease has been demonstrated as mentioned before.

References


