Introduction

Buri or the Japanese yellowtail (hereinafter referred to as yellowtail), *Seriola quinqueradiata*, is a species of Carangidae and it exists in the coastal waters of Japan, from Hokkaido in the north to near Taiwan in the south. The spawning grounds are located in a wide area south of Japan, centered in the waters south of Kyushu. Spawning begins in April to May and the fry migrate with the drifting algae when they reach the size of 1 to 1.5 cm. When they reach 15 cm or more they move away from the drifting algae to form a school. These fish migrate northward along the coast of Japan, but return south to the warmer seas between autumn and early winter. Yellowtail reaches maturity 3 years after hatching, or when it has reached about 60 cm in size and 5 kg or more in weight. Full maturation occurs a year later at 70 cm or more in length and 7 kg or more in weight.

The first attempt to culture yellowtail was made by Mr Wasaburo Noami of Hiketa, Kagawa Prefecture, Shikoku, in 1927, who succeeded in rearing yellowtail from captured fry. Commercial yellowtail culture began in diked or net-fenced bays or inlets about 1950. Commercial net-pen culture began around 1960 and the introduction of chemical fiber fishing nets, ropes, and floats led to the development of durable and tractable floating net pens. In addition to this, the development made in the supply and storage of frozen fish for feed led to a rapid expansion of the yellowtail culture industry. It is no exaggeration to say that in almost all bays and inlets in the south of Japan, except those polluted with social and industrial waste, the culture of yellowtail is being carried out. According to government statistics, the production of cultured yellowtail totalled 122 000 tonnes in 1978; this is about 3 times as large as the catch of wild yellowtail in that year.

Yellowtail culture starts with the capture of fry under the drifting algae during the April to June period. The fry are cultured in floating net pens and fed anchovy, sand launce, mackerel, horse mackerel, Pacific saury, etc. of low economic value until they have grown to a profitable marketing size. Formerly they were harvested within the year, but in recent years cultured one- and two-year-old fish have been marketed. They are used widely for the “sashimi”, a raw fish dish. Yellowtail in net pens grow remarkably fast, reaching a length of 30 to 50 cm and a weight of 1 to 2 kg by the end of the first year of culture, and 3 to 5 kg and 6 to 9 kg by the end of the second and third year respectively. The optimum temperature for growth is 24°C to 29°C; growth ceases below 13°C and the fish die at 7°C. In areas where the temperature goes below 15°C in winter, the fish are moved to warmer areas for wintering.

As mentioned before, yellowtail culture has expanded rapidly over the past 20 years. However, the industry is now faced by many problems that require urgent solution, such as seed fish, diseases, and pollution of culture centers, etc. The disease problem is the most serious. For example, in 1978 the output of cultured yellowtail was worth about ¥120 000 million, and the damage caused by diseases was estimated to be ¥10 100 million. Many kinds of diseases are known to exist in cultured yellowtail today, the most important of which are the bacterial diseases, namely vibriosis, pseudotuberculosis, streptococciosis, and nocardiosis. The outbreak of these diseases is now common with great losses suffered every year in most culture areas.

The study of diseases of cultured yellowtail was initiated about 1960 and thereafter much information accumulated rapidly. The purpose of this paper is to review the current knowledge concerning the principal diseases of cultured yellowtail.

Viral disease

Lymphocystis disease

Lymphocystis of cultured yellowtail was reported by Matsusato (1975 b). It is the only viral disease known to date in cultured yellowtail. The disease is called “black spot disease” by farm operators, because lymphocystis cells which form in the skin or fins are surrounded by
many abnormally developed melanophores which look like a black spot macroscopically. The disease occurred occasionally in various areas but did not cause any harmful effect on the fish. The affected fish were observed mainly in the summer months and the disease healed by itself within a few months. The source of infection was believed to be lymphocystis-diseased fish used as feed.

Fungal disease

Ichthyophonosis

Ichthyophonosis was first found in Mie Prefecture (Kubota, 1967). It has been noted in various areas, but the incidence was usually low. It infects fish mainly in the first year of culture, in particular soon after the rearing of seed fish has started. The causative fungus is thought to be *Ichthyophonus hoferi*, which affects almost all internal organs including the heart, liver, spleen, the nervous system, and the gills. The affected organs swell and many white spots up to 4 mm become visible. When the gills are affected hemorrhage occurs resulting in marked anemia. The fish with fungi ultimately dies. The source of infection is believed to be fungi-carrying fish in the feed. Unfortunately, there is no established treatment for this disease. The histopathology of diseased fish, and the morphology, life cycle, and culture of the fungus have been reported by Chien et al. (1979 a–c).

Microsporidian disease

A kind of disease is known that produces an irregular unevenness on the body, stops the growth, and causes the fish to lose weight rapidly; the mortality is rather low, and the majority of the fish which have contracted this disease heal naturally, but their growth is retarded even after recovery. The disease generally breaks out between June and August soon after the rearing of seed fish has begun. It is called microsporidiosis and affects the trunk muscles of the fish, which show the formation of white lumps of trophozoites packed with large numbers of very small microsporidian spores. The parasite has not yet been identified and no scientific paper has been published on this disease. There is so far no established method of eradication or prevention of it. Fortunately the frequency of its outbreak is low and its infectiousness is weak.

Myxosporidian diseases

Pericardial kudoasis

Pericardial kudoasis outbreaks occur often in various areas, mainly among fish in the first year of culture. The disease is characterized by the formation of many white egg-shaped objects 1 to 2 mm in size in the pericardial cavity. These are the trophozoites of the myxosporidian, *Kudoa pericardialis*, encapsulated by host-produced connective tissues (Nakajima and Egusa, 1978). The parasite produces little, if any, harmful effect to the fish; there is no significant difference in the condition factor between healthy and diseased fish. It does not affect the market price because the site of parasitization is limited to the pericardial cavity and is inconspicuous. The source of infection is unknown.

Muscular kudoasis

A similar myxosporidian, *Kudoa amamiensis*, thrives in the muscle, producing white spherical or oval cysts 1 to 5 mm in size consisting of a trophozoite filled with spores and a thick wall of connective tissue (Egusa and Nakajima, 1980). In severe cases several cysts are formed per cm² of the trunk muscle and the market value of affected fish is totally lost. This disease broke out at very high rates among fish cultured in Amami-Oshima and Okinawa areas and was named muscular kudoasis or Amami kudoasis. The parasite was believed to be transmitted to yellowtail from damsel fishes which are the natural hosts (Egusa and Nakajima, 1978). At present yellowtail culture is not taking place in the mentioned areas for fear of this disease, even though the environmental conditions are optimum for the culture.

Myxosporidian green liver

There is an unidentified myxosporidian which is parasitic in the bile duct of the liver. Its trophozoites, when developed, are large enough to block the flow of bile and they cause a condition named green liver, which is frequently observed in some areas (Matsusato, personal communication). Its effect on the host is unknown.

Helminthous diseases

Skin fluke

*Benedenia seriolaе* (Trematoda: Monogenea; Capsaloidae) is a harmful parasite often found on cultured yellowtail in the first year of growth, causing skin flukes. The body is oval and flat dorsally ventrally. It has one big posterior sucker with three pairs of central hooks, and two small anterior suckers. It fastens tightly with the suckers on to the body surface and affects mainly epithelial tissues. Large individual parasites reach a body length of 9 mm, but 5 to 7 mm is the usual size.

The heavily affected fish suffers from a reduced appetite, becomes emaciated, and rubs the affected part of its body against the net owing to the apparent itchiness caused. As a result, the injury of the skin becomes more extreme and provides a portal of entry for bacteria that...
cause vibriosis. Skin fluke which causes much damage occurs from early summer to autumn with the peak in mid-summer, since the development and maturation of the larvae are rapid at high water temperatures. The life cycle of the parasite in relation to temperature has been studied by Harada (1965), Hoshina and Matsusato (1967), Kasahara (1967 a), and Hoshina (1968). During its life the parasite spawns several times at a temperature range of 13° to 29°C, and lays 60 to 200 eggs at a time. The pattern of the life cycle resembles that of B. seriola.

The eradication of this parasite is best achieved by immersing the affected fish for 2 to 2.5 minutes in sea water saturated with salt (Akazaki et al., 1965). Baths in seawater solutions of pyrophosphate peroxyhydrate (Kasahara, 1967 b) or disodium phosphate peroxyhydrate (Kasahara, 1971) are also effective eradication methods.

The outbreak of gill fluke, like that of skin fluke, has decreased in recent years, ever since the aforementioned paint that contains TBTO has come into use.

Philometrosis

Philometrosis is caused by Philometoides seriola (Nematoda: Philometridae; Philometridae) which bores a hole in the trunk muscles including the red muscle of 1-year-old fish after the summer and in those of 2-year-olds before the autumn. Fish affected before September in its first year of life carry the larvae under the serous membrane of the liver and not in the muscle. Usually 3 to 5 worms, sometimes 10, 2 to 40 cm in length are found in the muscle (Nakajima and Egusa, 1969 e). The parasite has little, if any, detrimental effect on the fish, but the market value of infected fish is totally lost. When the parasite is present in the muscle, a pouch-like nest is often formed which occasionally has abscess-like materials in it. The life cycle of the worm has been studied by Nakajima and Egusa (1970) and Nakajima et al. (1970). The worm that matures in 2-year-old fish moves to the subcutaneous tissue from late April to late August with the peak in May and June; it bores a hole through the skin, extends the rear half of the body outside, releases the larvae and dies. The intermediate host of the parasite is assumed to be copepod plankton and the fry that feed on zooplankton presumably take in the larvae and the cycle is repeated. Therefore, when the parasite is found in a cultured fish, it means that the fry was infected before capture. However, the growth of the fish will not be impaired and usually the disease is not noted before the fish is shipped to the market. If the parasite is noted during culture, the marketing of the fish should be made when the 2-year-olds have passed the summer, i.e. when the parasite has died upon releasing the larvae and the wound has healed. There is no established method for eradicating the parasite.

Measles (bladder worm disease)

Measles was once a common disease in the farms along the coast of the Bungo Channel between Shikoku and Kyushu. It was caused by the parasitism of plerocercus larvae of Callo tetrarhynchus nipponica (Cestoda: Trypanorhyncha; Dasyrhynchidae) in the abdominal cavity. These parasites are usually 2 to 3 cm long and covered
with a light-brown sac of host-produced connective tissue. The number of parasites varies between several to 20 per fish. The mass parasitism of the worm causes the fish physiological disturbance and retards the growth.

The parasite does not occur in the edible part of the fish, but the market value of affected fish is greatly reduced. The taxonomy and life cycle of the parasite and its effect on the host and controlling measures have been studied by Nakajima and Egusa (1968 a, b; 1969 a–d; 1971 a, b; 1972 a–g; 1973). The stage before the procercus, which was tentatively named procercus by Nakajima and Egusa (1972 a), is found in the anchoy (Engraulis japonica). It is a leech-like worm, 3 to 4 cm long, which parasitizes the abdominal cavity. The procercus taken in by yellowtail penetrates the stomach wall and shifts arbitrarily in the body cavity. The final hosts of the worm are the sharks. The first intermediate host is assumed to be copepods but this has not been confirmed.

The high infection rate of this disease among cultured yellowtail in the above-mentioned area was at one time related to the feeding of fresh anchovy caught in the area. The worm can be killed by freezing or mincing the anchovy. There is no problem of measles in the area today.

Copepod disease

Caligiosis

Caligiosis is a gill disease caused mainly on first-year fish by Caligus spinousus (Crustacea; Caligidae; Caligidae). Its morphology and life history have been reported in detail by Izawa (1969). The carapace is circular, flattened, and ventrally concave. It acts as an adhesive disc. The lunules and the second antennae are also adhering organs. The mean body length of the adults is 2.73 mm for females and 2.19 mm for males. However, a specimen of C. spinousus found on wild yellowtail is much larger than that found on cultured yellowtail; the mean body length of females and that of males were reported to be 5.5 mm and 3.76 mm, respectively (Shiino, 1960).

The parasite occurs mainly on the branchial arch and occasionally in the oral cavity; serious infestations cause anemia. The infected fish is emaciated owing to appetite depression, rubs its head against the pen net, and develops ulcerations around the mouth which provide a portal of entry for bacteria which cause vibriosis. Caligiosis is a year-round disease but the peak occurs in summer. It has two nauplius stages which lead a pelagic life for 12 to 18 hours at 20°C. The copepodite larvae swim for some time and then adhere to the host.

Eradication is achieved by immersing the fish in 100 ppm solutions of trichlorfon in sea water for 50 seconds (Fujita et al., 1968). Bathing fish in 50 to 100 ppm solutions of the same chemical in fresh water for 5 min is said to be effective.

Bacterial diseases

Among the various kinds of diseases experienced, the bacterial ones are the most important because they cause most of the serious economic losses to the industry. The damage has been estimated to ¥9750 million, i.e., 96.6% of the total disease damage in 1978.

Vibriosis

Vibriosis is a common disease in cultured yellowtail, which has been known since the inception of the industry. It attacks mainly fish in the first year, in the peak of their growth in the summer and it also affects fish aged one year or more.

Vibriosis in cultured yellowtail was first reported by Kubota and Takakuwa (1963) and subsequently by Kusuda and Akazawa (1963). Thereafter pathological features were reported by Kusuda (1965), Kubota (1967), and Kimura (1968). Vibriosis occurs either in the form of dermatitis, a skin abrasion accompanied by hemorrhage appearing on the head, body, or fins, or as enteritis. The former is more common than the latter. Vibriosis occurs mainly in summer at temperatures of 25°C or higher. When it occurs in wintering fish at relatively low temperatures the ulceration may be superficial but wider.

The cause of the disease was studied by Kusuda and Akazawa (1963), Kusuda (1965), Akazawa et al. (1966), and Hatai et al. (1975). Until now at least two kinds of vibrios responsible for vibriosis in cultured yellowtail have been reported, namely Vibrio anguillarum and a group of vibrios closely related to V. parahaemolyticus (Kusuda et al., 1979; Ezura et al., 1980). According to Ezura et al. (1980) the majority of the V. anguillarum strains isolated from yellowtail are serologically identical with Type I reported by Harrel et al. (1976) or group 2 reported by Johnsen (1977). However, V. anguillarum strains which could not be classified according to any established serological type have also been reported (Jo et al., 1979).

These bacteria are ubiquitous, facultative pathogens and are often found in the sea water and subsoil of the culture area and also on the body and gills, and in the intestines of healthy fish. The fish, however, will not be affected as long as it is healthy. Fish attacked by skin flukes are vulnerable to vibriosis of the dermatic type. Any injury on the skin caused by handling for fresh-water or salt-saturated seawater bathing, or moving fish into the net pen for some reason, can also lead to an infection of vibriosis. When the fish are repeatedly fed poor-quality feed resulting from improper storage and preparation, the noxious substances such as oxidized fat, will cause an abnormal consumption of vitamins by the body, thus resulting in vitamin deficiency and convulsions. The continuous feeding of poor-quality feed will result in enteritis and often vibriosis of the dermatic form, too.
Providing good-quality feed and eradicating skin flukes should prove to be effective measures in reducing vibriosis. In fact, proper culture management has drastically reduced its outbreak in recent years. As mentioned above, the application of a preservative paint containing TBTO on the pen net has beneficial effects in preventing the occurrence of skin flukes and, as a result, in the reduction of the outbreaks of vibriosis.

For the treatment of vibriosis various sulfonamids, antibiotics, and synthetic antibacterial agents are widely used. Although drug-resistant strains of vibrios were used. Although drug-resistant strains of vibrios were detected in yellowtail (Aoki et al., 1972), this problem is negligible from the practical point of view. Vibriosis is not a menace to the yellowtail culture industry today. The damage resulting from vibriosis was estimated to be about ¥ 500 million, approximately 5% of the total loss in 1978.

A dermatitis caused by a filamentous bacterium

A dermatitis having external symptoms similar to those of vibriosis has recently been reported (Miyazaki et al., 1975). The initial sign is an ulceration on the body or fins with hemorrhage and a partial collapse occurring in the affected part. The disease has hitherto occurred in winter and is not highly virulent. Many filamentous bacteria were observed in the affected part of the skin, but the causative agent was not identified. Incidentally, it can be noted that a similar disease characterized by eroded mouths and rotten tails occasionally breaks out among cultured young sea breams (Pagrus major, Acanthopagrus schlegeli) (Masumura and Wakabayashi, 1977). The cause of the disease is a marine gliding bacterium for which the name Flexibacter marinus has been proposed (Hikida et al., 1978).

Pseudotuberculosis

Among virulent diseases reported for the first time in the past 10 years is pseudotuberculosis caused by Pasteurella piscicida. The first nationwide outbreak occurred in 1969. It appears that the disease had existed before localized to the areas along the Bungo Channel coast. It suddenly broke out in almost all culture areas in 1969, and has thereafter caused serious damage to the industry every year. The loss in 1978 was estimated to be about ¥2800 million, i.e., about 28% of the total loss caused by diseases.

The symptoms, histopathological characteristics, and pathogenesis of the disease were described by Kubota et al. (1970 a, b, 1972). The initial sign of the disease is the loss of the ability of the fish to descend to the bottom of the net pen. A small eruption developing into an ulcer is observed occasionally on the body. The most characteristic signs are numerous tubercles, white in color and up to 3 mm in size, which develop in the internal organs, especially the spleen and kidneys. The causative bacterium is resistant to phagocytosis, being able to multiply in the phagocyte. As a result, the phagocyte swells into a large globule sometimes large enough to block the capillary blood flow. The direct cause of death is believed to be this blockage of the capillaries in vital organs. The disease is transmitted orally (Wakabayashi et al., 1977). The infection may also occur through the skin or gills, although this has not been demonstrated.

The ones infected are mainly fish in the first year, which suffer high mortalities. One-year-olds or older are seldom affected. The disease usually breaks out in early summer when the water temperatures rise to about 20°C and abates when the temperature reaches 25°C.

The morphology, cultural, and biochemical characteristics of the causative agent have been studied by Kimura and Kitao (1971); Shimizu and Egusa (1972); Kusuda and Yamaoka (1972); Kitao and Kimura (1974); and Koike et al. (1975). The bacterium can grow at temperatures ranging from 17°C to 32°C and at 0.5 to 4.0% NaCl, the optimum temperatures and NaCl concentrations for growth being 23°C to 28°C and 1 to 3% respectively. Pasteurella piscicida is serologically homogeneous (Kusuda et al., 1978), and for a rapid and exact diagnosis of the disease a direct fluorescent antibody technique was developed (Kitao and Kimura 1974; Mori et al., 1976). Unpublished data show that many kinds of marine fishes such as Seriola purpurascens, Acanthopagrus schlegeli (= Mylio macrocephalus), Pagrus major (= Chrysopteryx major), Oplegnathus fasciatus, Laterorhabax japonicus (Perciformes), and Sephaholepsis cirrifer (Tetraodontiformes) are susceptible to this bacterium. Therefore, it is concluded that there must have been natural host species other than yellowtail on the Bungo Channel coast.

For the control of the disease ampicillin is reported to be effective (Kusuda and Inoue 1976, 1977 a, b; Hatai et al., 1978). Sulfonamide and chloramphenicol are also said to be effective (Matsusato, 1975 a). Oxolinic acid, sodium nifurtynenate, dihydroxymethyl florotin, and thiamphenicol are currently on sale for the treatment of this disease, but no scientific report has been published on the efficacy of these drugs.

Streptococciosis

In the summer of 1974 streptococciosis broke out explosively in many farms in Kochi, Kagoshima, and some other areas in the south of Japan, causing heavy losses. The disease spread to almost all of the farming areas in the following year and thereafter it broke out every year with serious losses. The damage this disease has caused the industry is greatest of all the known diseases. In 1978 it was estimated to be about ¥6400 million, 63% of the total disease damage. Although
many observations and experiments have been and still are being done on this disease, the published literature is sparse. The disease infects not only fish in the first year but also those 1 year old or older. It is observed from early summer to winter, with the peak occurring in mid-summer, and abates when the water temperatures drop below 20°C and cease at about 15°C.

The main external symptoms of the diseased fish are cloudy lenses, bulging eyeballs with severe hemorrhage, hemorrhage in the inner wall of the operculum, lacerations with hemorrhage on the fin, and ulcerations on the body or at the base of the caudal fin. The main internal symptoms are operculum surface of the heart, intensive hemorrhage in the liver, petechiae in the heart, pyloric caeca, spleen, and kidney, and adhesion of the internal organs and the peritonium. Generally there is a severe enteritis. It was shown by Kusuda and Kimura (1975) that the causative agent grows well and remains for a long time in the intestine. Granuloma develop in the affected tissues in which bacterial colonies can be observed.

The disease is of a subacute nature. The affected fish suffers from a reduced appetite, swims listlessly, and finally sinks to the bottom of the net pen and dies. Records show that the disease frequently recurs; a conjugate is that upon recovery from the disease the fish is not immune to it.

The causative agent is a α-hemolytic Streptococcus sp. closely related to S. faecalis or S. faecium (Kusuda et al., 1976; Kusuda and Komatsu, 1978). A β-hemolytic strain of Streptococcus which was identical with or very closely related to S. equisimilis was reported as the cause of a very similar disease of yellowtail by Minami et al., (1979), but the Streptococcus sp. reported by Kusuda et al. seems to be the agent responsible for most of the outbreaks of streptococcosis.

The Streptococcus sp. can grow well in media with 0 to 4 % NaCl and temperatures between 15° and 40°C, the optimum NaCl concentration and temperature being 0 % and 27° to 30°C respectively. It can also survive for a long time in the sea and subsoil. Kitao et al. (1979) reported that the bacteria were detected in the sea water and bottom mud of one culture area throughout the year. On the other hand, Minami (1979) reported that sardine, anchovy, and round herring caught near the culture areas carried Streptococcus sp. This suggests that the bacterium exists widely in and around the culture areas today.

The source of infection is obscure. As mentioned above, the disease broke out explosively in the summer of 1974 in Kochi, Kagoshima, and other areas in the south of Japan. There may be a possibility that the disease existed formerly as a local disease of minor importance in these areas and suddenly increased in virulence because of unknown factors that year. On the other hand, the following reasoning may also be possible. The feed which had been accidently contaminated with the bacterium might be the source of infection. It is common for feed fish landed in great numbers at a certain place to be sent to various culture areas.

No chemotherapeutic agent has been effective against this disease which is the main reason why the damage has been so great. Recently a nitrofurans compound, sodium nifurstyrenate, (Kashiwagi et al., 1977 b; Kashiwagi et al., 1977 a), erythromycin (Shiomitsu et al., 1980; Katae et al., 1980), and spiramycin (unpublished data) were proved experimentally to be effective for the control of streptococcosis. It is expected that the use of these drugs will reduce the damage of the disease.

**Nocardiosis**

Nocardiosis of yellowtail, caused by a Gram-positive, acidfast bacterium Nocardia kampachi, is an incurable disease, but it occurs rather sporadically. Therefore, the damage it causes cultured yellowtail is not very great, being estimated to ¥ 34 million, only 0.4 % of the total disease damage in 1978. This disease has been reviewed by Kusuda (1975) and Kusuda and Nakagawa (1978).

The disease broke out for the first time in Mie Prefecture in 1967 and was studied by Kariya et al. (1968) and Kubota et al. (1968). It infects not only fish in the first year but also those 1 year old or older. It occurs between July and February, with the peak in September/October. The disease affects the dermis, subcutaneous tissues, muscles, liver, spleen, kidneys, gills, and almost all other organs. In the visceral ones many tubercles are formed, which appear as small white spots; on the gill a tubercle is formed and around the lips ulcers occur. Transmission is believed to occur mainly orally or through the gills (Kusuda, 1975). Kusuda et al. (1974) reported that there is also a type of nocardiosis which forms tubercles only on the gills. Tubercles formed in the dermis and subcutaneous tissues are seen as small wart-like protuberances on the body surface. A large abscess appears under the skin causing a soft swelling on the skin above. Haematological features of the diseased fish have been described by Kusuda (1975) and Ikeda et al. (1976).

The causative agent was first studied by Kariya et al. (1968), who identified it as a new species of the genus Nocardia and gave it the name N. kampachi. This was verified later by Kusuda and Taki (1973) and Kusuda et al. (1974). The bacterium can grow in media with 0 to 4·5 % NaCl, the optimum NaCl concentration being 0 to 0·5 %, which suggests that the bacteria may be naturally terrestrial or limnetic.

This disease was first noted in 1967, but the source of the infection was not demonstrated. The possibility was suggested that fish used as feed had been contaminated with the bacterium. Another possibility was that naturally terrestrial or limnetic bacteria were brought into the culture area through some route. There is no established effective treatment for this disease.
Concluding remarks on the prevention of infectious diseases

The three kinds of virulent bacterial diseases, namely pseudotuberculosis, nocardiosis, and streptococcosis, have appeared one after another over the past 12 years. Pseudotuberculosis, which formerly existed only as a local disease in the Bungo Channel area, spread nationwide in 1969 and thereafter it broke out annually with heavy loss. Nocardiosis was first noted in Mie Prefecture in 1967, and spread thereafter to Shikoku, Kyushu, and other areas. Streptococcosis first occurred explosively in the summer of 1974 in Kochi, Kagoshima, and other areas in the south of Japan, and in the following year it spread to almost all culture areas, causing heavy losses every year. The nationwide spread of these diseases is believed to be due to the transportation of seed fish or the movement of fish of various ages for different purposes including wintering. It may be concluded that all of the yellowtail culture areas are now contaminated with these three diseases plus, of course, vibriosis.

Once a bacterial disease breaks out among fish in a net pen in a given area, the bacterium is discharged from the diseased fish and spreads to the surrounding net pens and to the whole area. Since it is not always easy to remove diseased fish from a net pen, dead fish sink to the bottom and release the bacterium in large numbers, giving rise to a most dangerous source of infection. It is very difficult to get rid of the disease after an outbreak.

Streptococcus sp. can survive for long periods of time in the sea water and bottom mud, contrary to Nocardia kampachi which cannot last long in open sea water, dying out within a few days. However, this species can survive fairly long in polluted or enriched sea water (Kasuda and Nakagawa, 1978).

Janssen and Surgalla (1968) reported that Pasteurella piscicida died within 3 days in sterile brackish water. It is believed that this bacterium, like Nocardia kampachi, can survive well in polluted or enriched sea water, though this has not been scientifically demonstrated.

The rapid expansion of the yellowtail culture industry over the past 20 years has led to an indiscriminate establishment of farms so that almost all the suitable bays and inlets are currently overcrowded by cultured yellowtail causing serious environmental pollution. The water quality at culture centers has deteriorated seriously as a result of the fluids and particles dispersed from minced fish used as feed, the leftover feed which settles on the sea bottom, and the excrement of the fish itself. This is called “self pollution”. Thus the disease agents are supported continuously in the sea water of culture areas throughout the culture seasons and constitute a constant menace to the yellowtail cultured there.

The deterioration of environmental conditions imposes stress directly or indirectly on cultured yellowtail, thereby lessening resistance to infection. Overcrowding of yellowtail in the net pen is also an important stress-causing factor. This is common knowledge among farm operators, now that the incidence and mortalities from any infectious disease are much higher in overcrowded net pens than in uncrowded ones in any given bay or inlet.

It has been pointed out that each farm operator should adhere to the following principles to reduce to a minimum the damage caused by infectious diseases: (1), not to move diseased fish to other areas; (2), to keep a proper stocking density in each net pen; (3), to use good-quality feed; (4), to periodically eradicate external parasites; (5), to remove dying and dead fish and to incinerate them on land; (6), to apply proper medication as soon as a disease occurs (it is important to prevent its spread); and (7), to cooperate with all of the farm operators in the area to stamp out the disease.

Of utmost importance, however, is the maintenance of good environmental conditions in each culture area; in other words, prevent self pollution. This is not possible unless all of the operators unite in their efforts. Fisheries biologists concerned with this problem should make every effort to accumulate the scientific knowledge necessary to give rational guidance to the farm operators and their leaders.

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