A handbook of diseases of importance to aquaculture in New Zealand

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Cover: Eggs of the monogenean ectoparasite Zeuxapta seriolae *(see p. 102) from the gills of kingfish. Photo by Ben Diggles.*

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Contents

This symbol in the text denotes a disease is exotic to New Zealand

Contents colour key: Black font denotes disease present in New Zealand
Blue font denotes disease exotic to New Zealand
Red font denotes an internationally notifiable disease exotic to New Zealand

Diseases of Fishes

* Virus detected, but never associated with disease

Introduction

Aquaculture in New Zealand has advanced rapidly during the last two decades. This has been mainly due to development of three species; mussels, salmon, and oysters, which have evolved from a few small scale domestic operations to extensive multimillion dollar industries. In 1998, they collectively represented a value of NZ$200M and composed more than 17% of the total value of New Zealand's seafood exports (Holland & Jeffs 2000). Further expansion of these industries is planned in the near future and if current trends continue they will become an increasingly important part of New Zealand's economy, providing seafood for both local and export markets as well as jobs and regional economic development.

Diversification into new species is being increasingly emphasised. Research into the biology of high value marine species which have aquaculture potential, such as rock lobsters, paua, snapper, kingfish, and seahorses, has become a high priority in recent years. However, there is increasing evidence that disease may seriously impede expansion of both the existing and newly developed species on which aquaculture in New Zealand is based.

This handbook is intended as a resource for those working in New Zealand aquaculture. It covers much of what we know about the diseases of fish and shellfish in New Zealand to date, updating information contained in earlier disease guides which dealt mainly with freshwater fish (Hine & Boustead 1974, Boustead 1989), and including for the first time in this country sections on diseases of cultured marine fish, mollusks, and crustaceans. This handbook is intended for the intelligent layperson, though additional material which will help specialists diagnose and control disease outbreaks has also been included for many diseases. (However, please note the disclaimer on page 7).

As well as listing, and in many cases illustrating, the diseases that are known occur in New Zealand aquaculture, we also include a number of diseases exotic to this country to provide an international perspective on diseases of importance to aquaculture and international trade in other parts of the world. An emphasis has been placed on exotic diseases which are listed by the Office International des Epizooties (OIE), the World Animal Health Organization based in Paris, France. The three main aims of the OIE are: to inform governments of the occurrence and course of animal diseases throughout the world, and of ways to control these diseases; to coordinate, at the international level, studies devoted to the surveillance and control of animal diseases; and to harmonise regulations for trade in animals and animal products among member countries. As of June 2001, New Zealand was one of 157 member countries which were signatories to the international agreement (OIE 2001).

This handbook has been produced to raise industry awareness of the pivotal role they play in the front line against incursion of exotic diseases, and also in the control of endemic diseases, which could threaten New Zealand’s reputation as a producer of high quality, "clean and green" seafood.

References

Boustead, N.C. (1989). A guide to diseases of salmon in New Zealand. *New Zealand Freshwater Fisheries Report No. 112.* Freshwater Fisheries Centre, MAF Fisheries, Christchurch.

Hine, P.M.; Boustead, N.C. (1974). A guide to disease in eel farms. *Fisheries Research Division Occasional Publication No. 6.* 28 p..

Holland, R.C.; Jeffs, A.G. (2000). The status of aquaculture in New Zealand. *World Aquaculture 31*: 24–27.

OIE (2001). Office International des Epizooties. http://www.oie.int

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NIWA has used all reasonable care and skill in compiling this information. Every reasonable effort has been made to ensure that all information and advice in this handbook is accurate at the time of publication, however the information is general in nature and is made available on the understanding that NIWA provides no guarantees and accepts no liability for any loss or misfortune which might result from use of information contained in this handbook. Any person using this publication should independently verify the information before relying on it. Treatment and disinfection information contained in this handbook is intended only as a guide to the types of treatments and disinfection procedures commonly used for a given disease. NIWA strongly recommends that a diagnosis is obtained from a qualified aquatic animal health specialist prior to administration of any treatments and that any such treatments are administered under the supervision of suitably qualified persons. Note that in New Zealand antibiotics can be prescribed only by qualified veterinarians.

Disease and stress in the aquatic environment

It is necessary to clarify some of the terms and concepts used in this handbook. Many terms are given in the glossary, but it is necessary to comment on those below in more detail.

Terms such as parasite, parasitism, disease, pathogens, and pathogenicity are used. A parasite is an organism which lives on (ectoparasite) or in (endoparasite) its host and in so doing gains some benefit from its host. In general, when the relationship between the parasite and host is long-standing, the parasite and host have adapted to each other, so that they can co-exist without the parasite causing harm to the host. Such relationships tend to be obligate, that is, the parasite can survive only in or on the host, and is not free-living for any great period of its life. Other potential parasites are normally free living, but can and do become parasitic, from time to time. These opportunistic parasites can slip from one life-style into another. Parasitism is not the core subject of this handbook. Those who are specifically interested in fish parasites are referred to checklists of the known parasites of New Zealand fishes (Hine et al. 2000). If, however, a parasite does cause harm to its host such that the host can die from parasitic infestation, the parasite becomes a pathogen, and pathogens cause disease (Figure 1). Such a situation may arise if the host is being affected by abnormal environmental conditions or another pathogen that has weakened the hosts ability to defend itself because its immune system is impaired. Therefore, when reading this handbook, it is important to distinguish parasites and parasitism from pathogens and disease.

Disease is defined as an abnormal condition that affects the performance of vital functions of the affected animal and which sometimes displays diagnostic signs (called symptoms in human medicine) typical of that disease. The ways in which different infectious agents may cause disease differ between, and sometimes within, the different groups of pathogens. Viruses are very small, very simple infectious agents which can replicate (reproduce) only inside living cells (Figure A). They take over the host cell and use the internal components of the cell to replicate, and in doing so often destroy the host cell. When this happens on a large scale, tissues and organs can be affected. However, in such cases, death may also be partly due to the response of the host immune system as it tries to contain the virus and deal with the toxic debris of dead cells. Water is the ideal medium for viruses, it prevents desiccation, protects from UV light, and actively transports the viral particles to potential hosts.

Bacteria are much larger organisms than viruses (Figure B), and most are capable of living outside host cells. Bacteria are ubiquitous in the aquatic environment and also occur naturally on and inside many parts of healthy fish and shellfish. With a few notable exceptions, bacteria cause disease only in fish and shellfish which are injured, stressed, or otherwise have compromised defence systems. Some types of bacteria, such as intracellular forms such as rickettsias and chlamydias, produce little if any toxin, and tend to be relatively harmless. Other forms may, or may not, produce damaging toxins which cause widespread damage to surrounding host cells and hence disease. Often different strains of bacteria exist which differ in their toxicity and, therefore, their pathogenicity.

Protozoans (single-celled organisms, Figure C) live on or inside their hosts, and some are obligate parasites. Protozoan parasites can infect fish and shellfish in both freshwater and marine areas, and are particularly important pathogens of molluscs. *Bonamia exitiosus* in the Bluff oyster (*Ostrea chilensis*) is an example of an intracellular protozoan as it lives inside the blood cells (haemocytes) of Bluff oysters. Low numbers of protozoans seldom cause disease; however, due to their ability to multiply rapidly, the numbers of protozoans can increase quickly and cause disease under suitable environmental conditions, or when hosts are stressed.

Many larger metazoan (many celled organisms, Figure D) parasites infect fish and shellfish, particularly ecto- and endoparasitic worms, copepods and other types of parasitic crustaceans. Usually fish and shellfish tolerate natural infections of these quite well, because evolution favours co-existence of host and parasite so that the host is not usually killed by the parasite. However, for certain parasites (particularly those with a direct lifecycle without the need for intermediate hosts), aquaculture provides unusually favourable conditions which allow the parasite to flourish to the detriment of the host, due to the high density of hosts in a confined area.

These latter examples highlight how both infectious agents and their hosts are influenced by their environment. Simply because an infectious agent is present, does not mean that disease will develop. However, environmental changes can upset the balance between infectious agents and their hosts, leading to pathogenicity and disease (Figure 1). In such cases the change either favours the infectious agent, or is detrimental to the host. Sometimes, disease may result from a build up in infectious agent numbers, until the host can no longer support such a burden, becomes weakened, and dies.

Environmental changes for aquatic animals are usually related to changes in water quality and stocking density. Water quality parameters such as pH, salinity, temperature, ammonia, nitrite, or oxygen content may all influence the establishment of disease. In fact, it is not uncommon in instances of extremely poor water quality (for example, very high (supersaturated) or very low dissolved oxygen) to have disease without the presence of infectious agents (non-infectious disease, Figure 1). Because of this, naturally occurring events which can change water quality, such as floods, algal blooms, and stratification, can exert substantial influences on the health of cultured fish and shellfish, as can human induced changes in water quality such as pollution. High stocking densities also provide favourable conditions for increased transmission of most infectious agents, often resulting in disease. However, in such cases, if environmental controls can be implemented, or stocking densities reduced, it is possible to reverse the situation and eliminate disease.

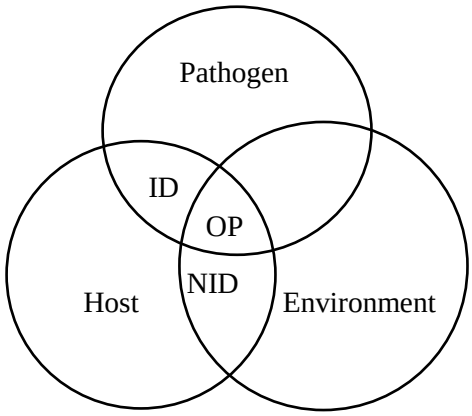
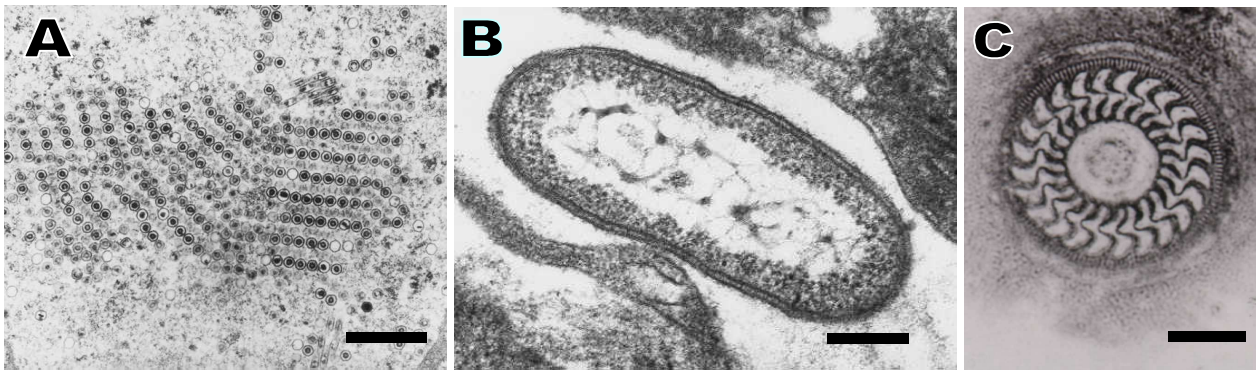
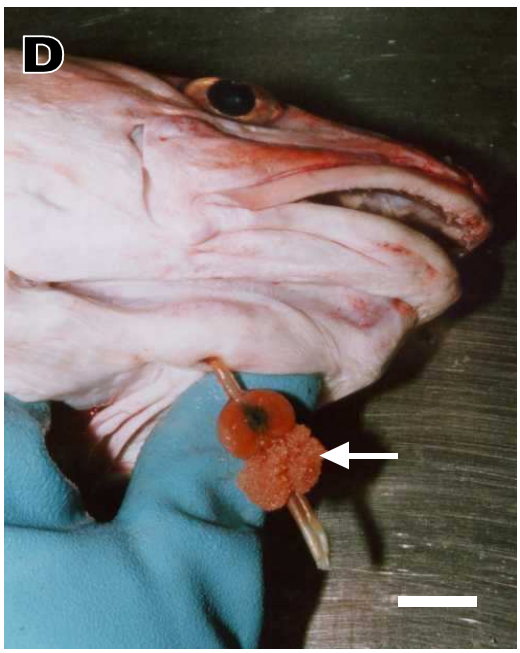


Figure 1: The interaction of pathogens and environmental stressors. Obligate pathogens can cause infectious disease (ID) in the absence of adverse environmental conditions. Opportunistic pathogens (OP) can cause infectious disease under environmental conditions adverse to the host, while adverse environmental conditions can result in non-infectious disease (NID).

Reference

Hine, P.M.; Jones, J.B.; Diggles, B.K. (2000). A checklist of parasites of New Zealand fishes, including previously unpublished records. *NIWA Technical Report 75*. 95 p.





Examples of the main types of disease agents of importance to aquaculture to illustrate their respective sizes.

A: Virus. Transmission electron microscope (TEM) photograph of an array of herpesvirus particles inside the cell of an oyster. Scale bar = 0.0001 mm. Photo by M. Hine.

B: Bacterium. TEM of a *Vibrio* sp. from the kidney of a turbot. Scale bar = 0.0005 mm . Photo by B. Diggles.

C: Protozoan. A ciliate, *Trichodina* sp., from the gills of a turbot. Scale bar = 0.01 mm. Photo by B. Diggles.

D: Metazoan. A copepod crustacean, *Sphryion* sp., on a ling. Scale bar = 10 mm. Photo by B. Diggles.

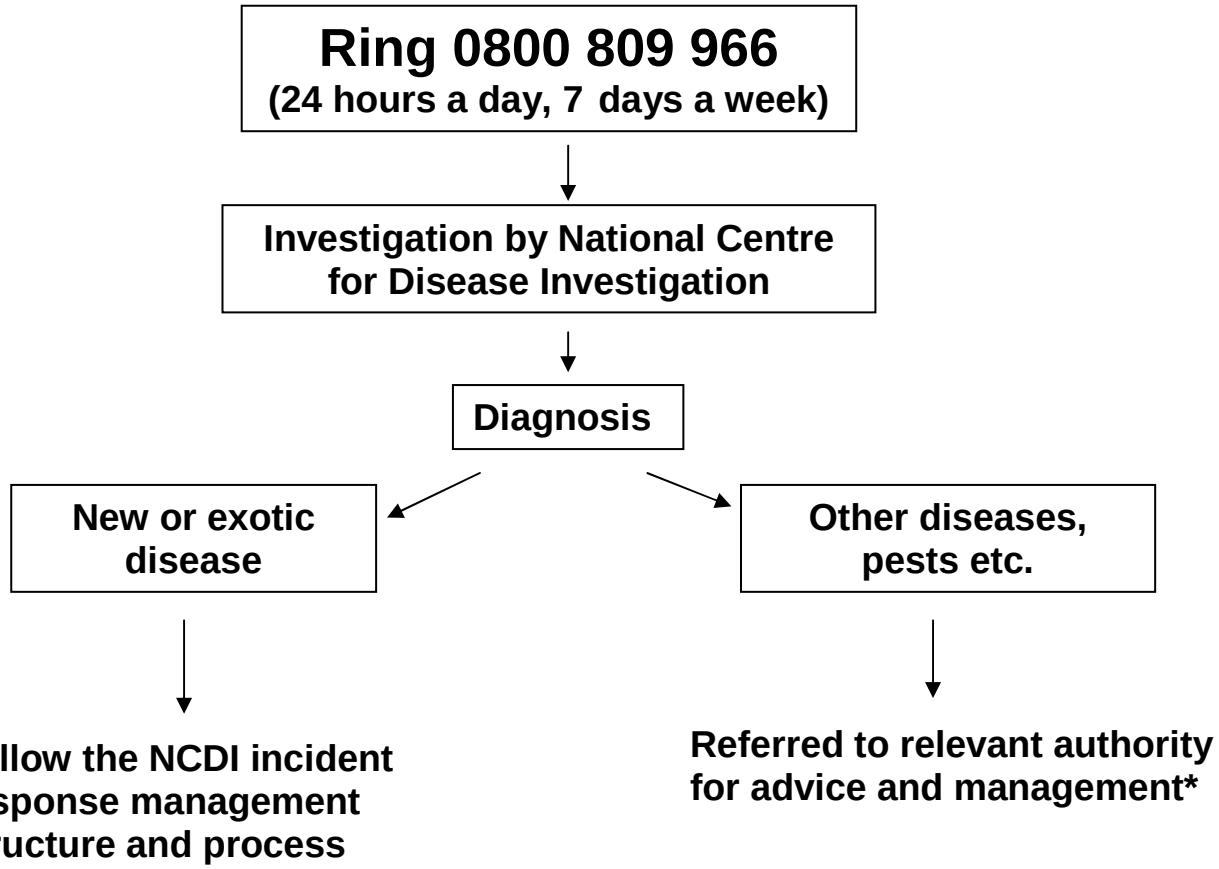
Who to contact when disease is suspected

In New Zealand, the National Centre for Disease Investigation (NCDI) is an operational unit established by the Ministry of Agriculture and Forestry (MAF) to investigate outbreaks of animal disease throughout the country. The unit is specifically concerned with diagnosis of exotic diseases, including those listed by the OIE. MAF define an exotic disease as a disease of animals which:

- is not recognised as previously occurring in New Zealand;
- is capable or potentially capable of causing unwanted harm to any natural and physical resources;
- could potentially have an economically significant impact on the viability of animal production or market access.

MAF consider exotic disease to include any new and emerging diseases which are not known to occur in New Zealand, regardless of their origins. The NCDI has a 24 hour 0800 number which aquaculturists are encouraged to call if an outbreak of a new or exotic disease is suspected in their stock (see below). If the disease outbreak is not suspected to be a listed disease (see appendices 1 and 2), or is already known to exist in New Zealand, or you have reason to suspect the disease outbreak is related to management, water quality, or environmental factors, NCDI advise contacting your normal aquatic animal health advisors, such as NIWA, universities, or veterinarians, rather than using the 0800 number. NIWA staff are integrated with the NCDI notification process, and if during the course of routine investigations an important new or exotic disease is detected, NIWA staff are obliged to notify the NCDI and, where necessary, other Government authorities.

The diagram below outlines the procedure followed if an outbreak of an exotic disease is suspected*



***Note**: If management, water quality, or endemic diseases are suspected, please contact your normal diagnostic advisers such as NIWA, universities, or veterinarians. If in any doubt, ring 0800 809 966.

Submitting a sample for diagnosis

It is not uncommon for aquatic pathologists to receive samples and submissions for diagnosis which have been treated in such a way that obtaining a meaningful diagnosis is impossible. Before sending specimens for diagnosis, first contact your aquatic animal health advisors to discuss the case. This is essential to establish how serious or widespread the problem is and to determine the most appropriate samples to take to help obtain a diagnosis.

Under most circumstances the ideal material for examination is live or moribund fish or shellfish exhibiting representative signs of disease. Receipt of such specimens allows pathologists to note the gross signs of disease, culture causative bacteria when indicated, prepare wet preparations of affected organs, and obtain optimal fixation for histopathology and other diagnostic techniques. Provision of a 500 ml water sample in a glass container is also helpful when algal blooms or toxic contamination are suspected.

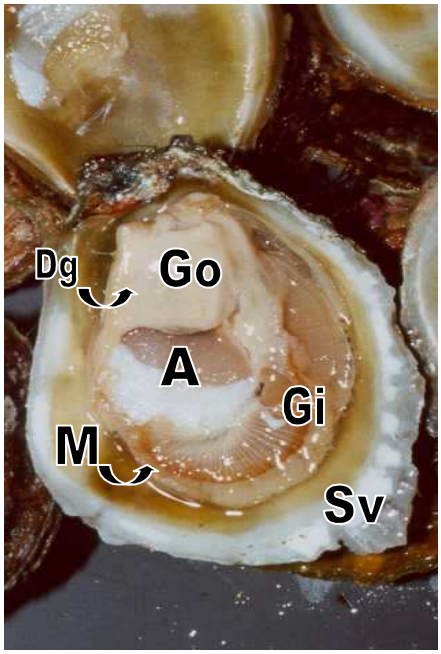
Transport of live and moribund specimens usually necessitates careful packing in sufficient water inside watertight containers lined with plastic bags. Usually an inner and outer container are used to minimise leakage. For fish or delicate shellfish, such as scallops, the water may need to be supplemented with oxygen and ice packs to keep temperatures low. The latter is particularly important during summer, when most disease outbreaks occur. Air freight or fast courier services are recommended for transferring specimens from affected facilities to the diagnostic laboratory in the shortest possible time.

Chilling samples at 4 °C (e.g., on ice or in a refrigerator) is acceptable for short periods of time only (usually less than 12 hours). If transport of live specimens is not feasible, and samples need to be held for longer than 12 hours, fixation is the next preferred method as it allows preservation of specimens for diagnosis. For most marine specimens, including whole small fish, molluscs, and crustaceans, fixation in 10% formalin in filtered seawater (100 ml of formalin (40% formaldehyde) in 900 ml of filtered seawater) provides adequate fixation as long as sufficient fixative is used, e.g., at least 4 or 5 volumes of fixative to each volume of specimen – and provision is made to allow entry of the fixative to areas such as body and mantle cavities by slicing the body cavity or carapace, or shucking. Specimens from freshwater areas are treated similarly except that distilled or tap water is substituted for seawater, though these preparations may need to be buffered with sodium phosphate for long-term storage. Other fixatives such as ethanol, gluteraldehyde, or combinations of these (e.g., Davidson's fixative – 100 ml = 33 ml ethanol, 22 ml formalin, 11.5 ml glacial acetic acid and 33.5 ml distilled water) are sometimes used for particular diagnostic methods. Well fixed material is excellent for histopathology and some other diagnostic techniques, but generally is unsuited for microbiology.

Freezing of samples is usually of limited value and generally should be avoided unless toxic poisoning is suspected or there are no feasible alternatives which would allow access to moribund, chilled, or formalin fixed specimens. This is because many cellular structures are disrupted by the freezing process, making diagnosis by histopathology difficult. However, for some types of testing which require use of genetic probes for identification of disease agents, freezing can be the preferred method of storage. Bacteria can also be cultured from frozen specimens, though diagnosis of causative bacteria is more difficult due to the post mortem proliferation of autolytic bacteria.

Basic anatomy of fish and shellfish

Molluscs



Basic anatomy of a flat oyster (the Bluff oyster, *Ostrea chilensis*). Photo by B. Diggles.

A, adductor muscle, used to close the shell valves

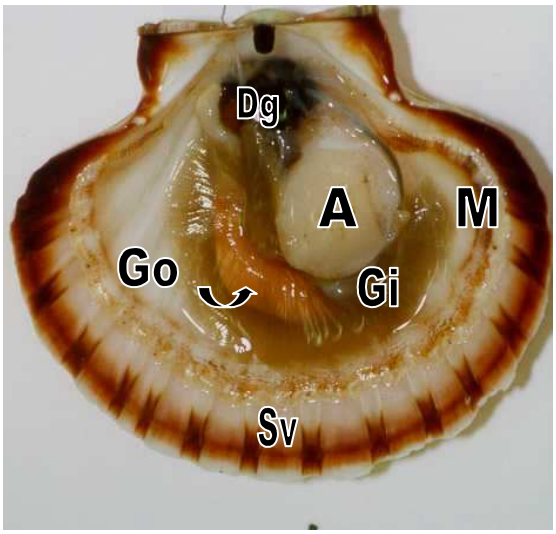
Dg, digestive gland, or gut, inside gonad

Gi, gills, used for feeding and oxygen uptake

Go, gonad. The white coloured gonad surrounds the gut, or digestive gland

M, mantle, a thin layer of tissue located between the gills and the shell valve

Sv, shell valve



Basic anatomy of a scallop (*Pecten novaezelandiae*). Photo by A. Blacklock.

A, adductor muscle, used to close the shell valves

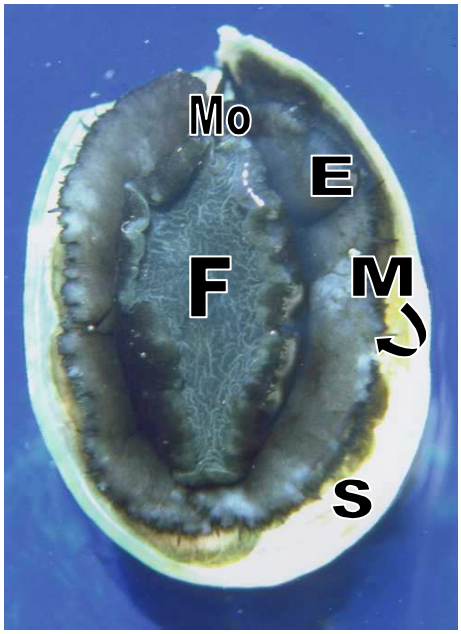
Dg, digestive gland, or gut

Gi, gills, used for feeding and oxygen uptake

Go, gonad. The bright orange (ovary) or white (testis) gonad is located between the gill rows

M, mantle, a thin layer of tissue located along the edge of the shell valve

Sv, shell valve



External anatomy of a paua (*Haliotis iris*). Photo by B. Diggles.

E, epipodium, a skirt-like extension of the foot

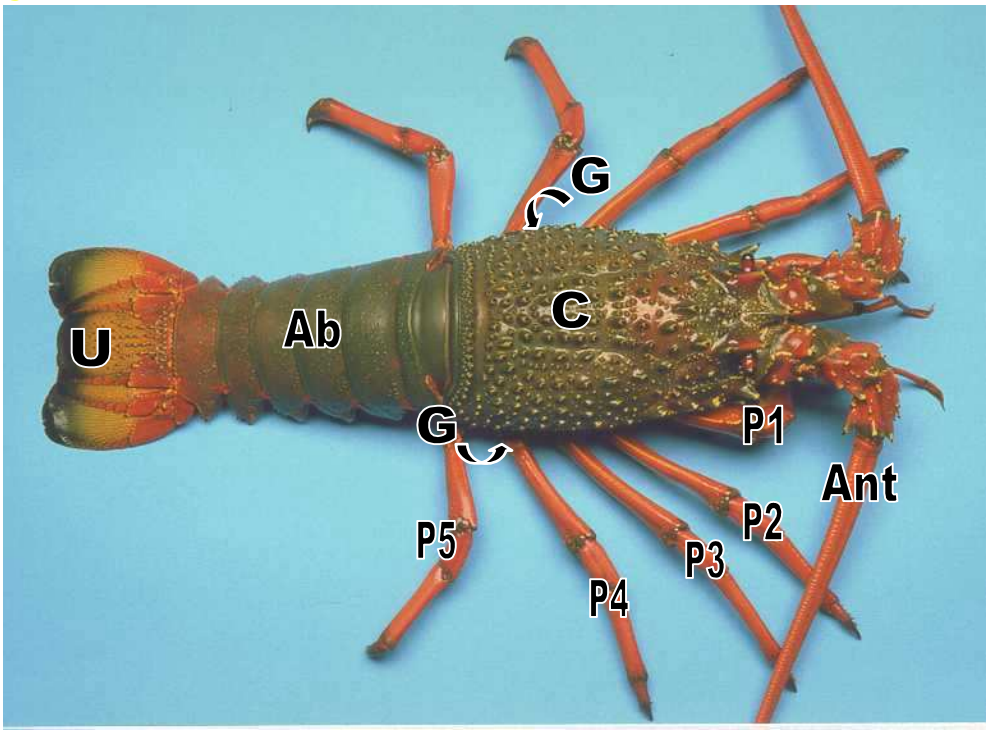
F, foot, a muscular organ used for attachment

M, mantle, a thin layer of tissue located along the edge of the shell, under the epipodium

Mo, mouth

S, shell

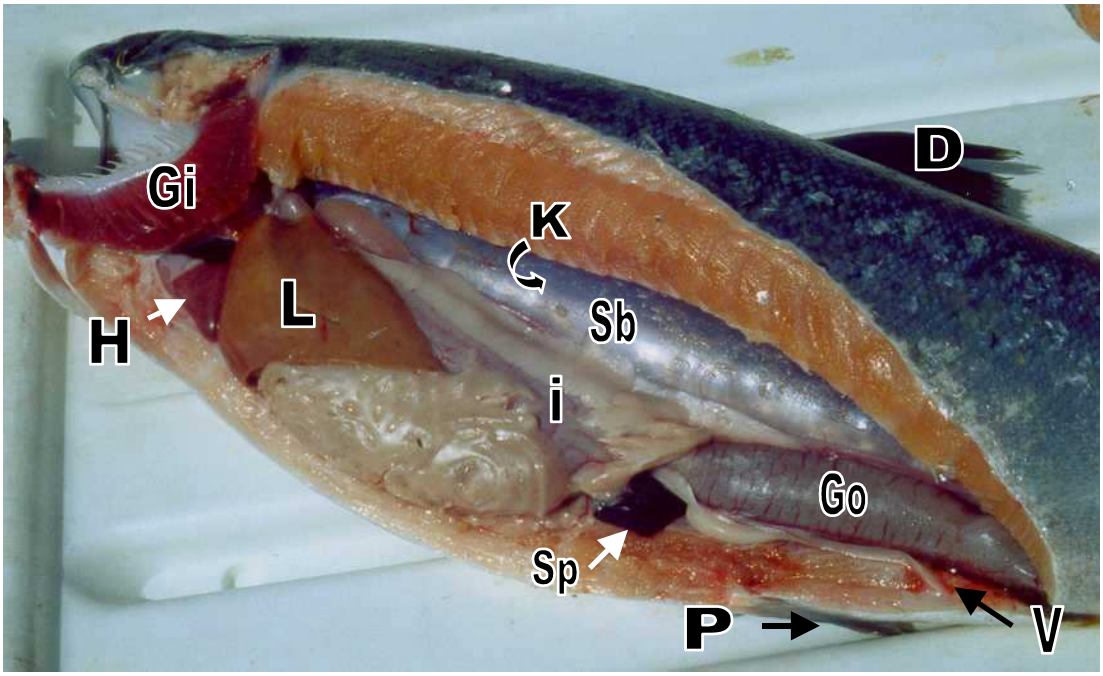
Crustaceans



External anatomy of a rock lobster (packhorse lobster, *Jasus verreauxi*). Photo by J. Booth.

Ab, abdomen, composed of 6 segments; Ant, antennae; C, cephalothorax (or carapace), under which are the internal organs; G, gills (under each side of the carapace); P1, 1st perieopod (walking leg); P2–P5, 2nd to 5th perieopods; U, uropod (tail).

Fish



Basic anatomy of a fish (chinook salmon, *Oncorhynchus tshawytscha*). Photo by N. Boustead.
D, dorsal fin; Gi, gills; Go, gonad; H, heart; I, intestine; K, kidney (behind swim bladder); L, liver; P, pelvic fin; Sb, swim bladder; Sp, spleen; V, vent.

Quick help guide

Freshwater and marine fishes

Gross signs	Possible causes
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Petechiae at base of fins	p. 18, 20, 34, 36, 40, 42, 46, 48, 88
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Quick help guide

Crustaceans

Gross signs	Possible causes
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Blackened lesions in hepatopancreas	p. 130, 132
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Opaque body musculature	p. 120, 130
Reddened haemolymph	p. 130, also sometimes occurs immediately prior to moulting
Swollen, turgid appearance	p. 134
Turbid haemolymph	p. 130
White spots under cuticle	p. 116

Molluscs

Gross signs	Possible causes
Abnormal swimming behaviour in larvae	p. 144, 146
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High mortalities in spat/juveniles	p. 138, 140, 178, also advanced stages of most serious diseases
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Diseases of Freshwater Fishes

FRESHWATER FISHES

Viral diseases of freshwater fishes

Disease: Epizootic haematopoietic necrosis (EHN) (ESV, ECV)

Species and life stage affected: Redfin perch (*Perca fluviatilis*), rainbow trout (*Oncorhynchus mykiss*) under 125 mm long, sheatfish (*Silurus glanis*) (ESV), and catfish (*Ictalurus melas*) (ECV) in Europe. Larger trout may be infected without mortality.

Gross signs: Lethargy, swimming slowly on the surface, or other unusual behaviour. High mortality in susceptible species. Infected perch show reddening around the brain and nostrils, areas of muscular pallor, petechial haemorrhages at the base of fins, particularly the anal fin. Cutaneous ulcers. Pale foci 1–3 mm in diameter in adult fish. An enlarged and bright red gelatinous spleen in juveniles. Sometimes peritoneal fluid present (Langdon & Humphrey 1987, Langdon et al. 1988).

Causative agent: Epizootic haematopoietic necrosis virus (EHNV), a ranavirus (family Iridoviridae).

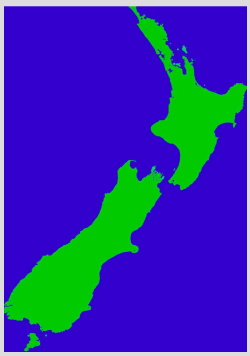
Diagnosis: Virus isolation into fish cell-lines, IFAT, ELISA, and PCR.

Treatment and prevention: No known treatment. Prevention is by controls on movement, destruction of infected stock, and disinfection.

Distribution in New Zealand

Unreported, but both rainbow trout and redfin perch occur in New Zealand.

EHN IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: EHN is confined to Australia, and ESV and ECV to Europe.

General comments: EHN is a serious disease in redfin perch, but less so in rainbow trout. The viruses causing EHN are also antigenically related to viruses reported from sturgeon (*Acipenser transmontanus*), largemouth bass (*Micropterus salmoides*), and sticklebacks (*Gasterosteus aculeatus*) in the U.S., sturgeon (*Acipenser guldenstadi*) from northern Europe, red sea bream (*Pagrus major*) in Japan, pike-perch (*Stizostedion lucioperca*) in Finland, tilapia (*Oreochromis niloticus*) imported into Canada, and many frog species (Mao et al. 1997, 1999).

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No photographs currently available

Disease: Infectious haematopoietic necrosis (IHN)

Species and life stage affected: Salmonids, particularly species of *Oncorhynchus* (e.g., rainbow trout *Oncorhynchus mykiss*), also Atlantic salmon (*Salmo salar*) and brown trout (*Salmo trutta*).

Gross signs: In acute disease fish may die without clinical signs. More typically fish are lethargic, interspersed with periods of hyperactivity. Fish may darken, have a distended abdomen, exophthalmia, with pale gills and mucoid opaque faecal casts. Petechial haemorrhages may occur at the base of the fins, vent, and sometimes gills, mouth, eye, skin, and muscle. Internally the liver, spleen, and kidney of fry may be pale, with watery fluid in the stomach. Yellowish fluid occurs in the intestines, and petechial haemorrhages occur in the visceral mesenteries, fatty tissues, swim-bladder, peritoneum, meninges, and pericardium. The blood-forming tissues of the kidney and spleen are the most seriously affected.

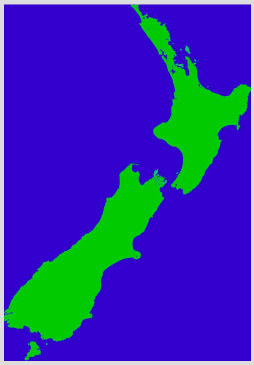
Causative agent: Infectious haematopoietic necrosis virus (IHNV) (family Rhabdoviridae).

Diagnosis: The virus can be isolated by cell culture, with subsequent identification by serum neutralisation, immunofluorescence, or staphylococcal agglutination. There are also several immunoassays available: ELISAs, dot blots, western blots, and immunoperoxidase. The virus may also be identified by electron microscopy, PCR, nucleic acid probes, or serum antibodies.

Treatment and prevention: No known treatment. Control may be exercised by destruction of infected stocks, disinfection of affected facilities, screening of broodstock for the virus and removing carriers, and vaccination.

Distribution in New Zealand

Unreported (Boustead 1993), although farmed salmonids are under active surveillance.



IHN IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: The west coast of the U.S., Japan, and Europe.

General comments: IHN occurs as several types, and the course of disease and clinical signs vary with host species, age and reproductive state. Young fish are more susceptible than older fish. Outbreaks are most likely at water temperatures of 10–12 °C, and are unlikely above 15 °C, but rainbow trout may be diseased at 3–18 °C. Although IHN is predominantly a freshwater disease, it does occur in ocean net pens among Atlantic salmon. Transmission may be vertical or horizontal. The virus may occur as a latent infection in fishes that survive mass mortalities, and may not be reisolated from infected fish until they reach sexual maturity. Because fish may be latently infected, testing for IHN before movement may require holding and testing over an extended period of time.

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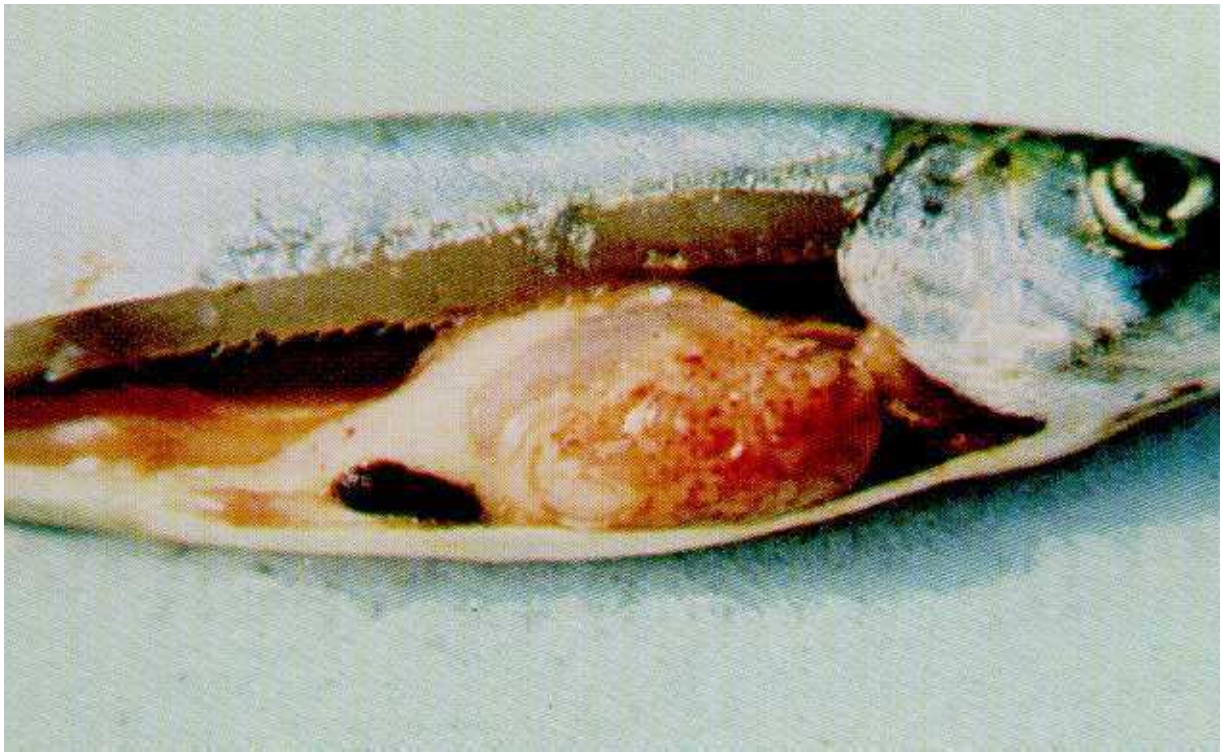


Coho salmon (*Oncorhynchus kisutch*) from Japan infected with infectious haematopoietic necrosis virus. (Photo reprinted from Fish Pathology, 2nd edition, Roberts, R.J. (ed.) 1989, Plate 6.1, by permission of the publisher, Bailliere Tindall).

This fish is exhibiting haemorrhages in the fatty tissues surrounding the pancreatic tissue, a typical sign of IHN in older fish.

Disease: Infectious pancreatic necrosis (IPN) (aquatic birnavirus infection)

Species and life stage affected: Fry and fingerling salmonids (*Salmo* sp., *Oncorhynchus* sp., *Salvelinus* sp.), freshwater eels (*Anguilla* sp.), and many marine fish species, prawns (*Penaeus*) and molluscs (*Meretrix, Tellina*). Aquatic birnaviruses have also been isolated without signs of disease in many families of fishes, molluscs and crustaceans.

Gross signs: There are no specific signs of IPN. In salmonids, behavioural changes include anorexia, corkscrew swimming, and ataxia. Externally the fish may show swollen bellies, hyperpigmentation, exophthalmia, and petechial haemorrhages on the ventral surface. Internally, there may be visceral petechial haemorrhages, and a yellow exudate in an empty gut. These signs may easily be confused with those of infectious haematopoietic necrosis (IHN).

Causative agent: Infectious pancreatic necrosis virus (IPNV), an unenveloped icosahedral birnavirus. Strains exist that vary in virulence in relation to the age and species of host.

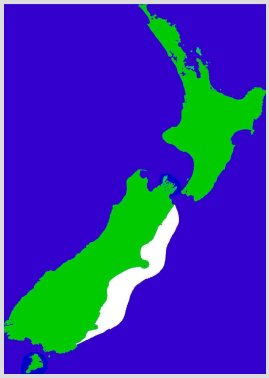
Diagnosis: Primary isolation into one of the many cell-lines that are available. Serological techniques involving serum neutralisation, conjugated antibodies binding to viral antigen, or molecular probes.

Treatment and prevention: No known treatment. IPN is an extremely difficult disease to control because it can be transmitted vertically or horizontally, and the conditions of culture are ideal for transmission. It can also be spread by humans, and because it occurs in wild fish and shellfish, these may act as a reservoir of infection. Effective control may be established using 1.0–1.5 x 10^5 μW s cm^{-2} UV irradiation (Yoshimizu et al. 1986), and treatment of facilities and eggs with iodophors. IPN is inactivated after 14 days in 250 ppm formalin, also by chlorine and iodine (Desautels & MacKelvie 1975), ozone (Liltved et al. 1995), and quaternary ammonium compounds (Dorson & Michel 1987).

Distribution in New Zealand

IPNV has been found in healthy chinook salmon returning from the sea (Tisdall & Phipps 1987, Anderson 1997), and has never been associated with disease in this country.

IPN IS REGARDED AS AN INTERNATIONALLY SIGNIFICANT DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Worldwide. As well as causing IPN in fish, the virus infects and causes disease in clams in Taiwan (Lo et al. 1988).

General comments:. Transmission can occur through the ovarian fluids and milt of salmonids, but these infections are external to the gametes. Horizontal transfer is via infected fish urine and faeces, and invertebrate faeces and tissues. Rainbow trout excrete virus within 2 days of immersion challenge, with the rate of excretion highest at 4–8 days, declining thereafter. IPNV may exist in the carrier state in salmon, trout and char, with replication primarily in head kidney leukocytes (Johansen & Sommer 1995). The presence of IPNV in chinook salmon in New Zealand was recorded from asymptomatic, wild caught, sea run fish after being at sea for 2 or 3 years (Tisdall & Phipps 1987).

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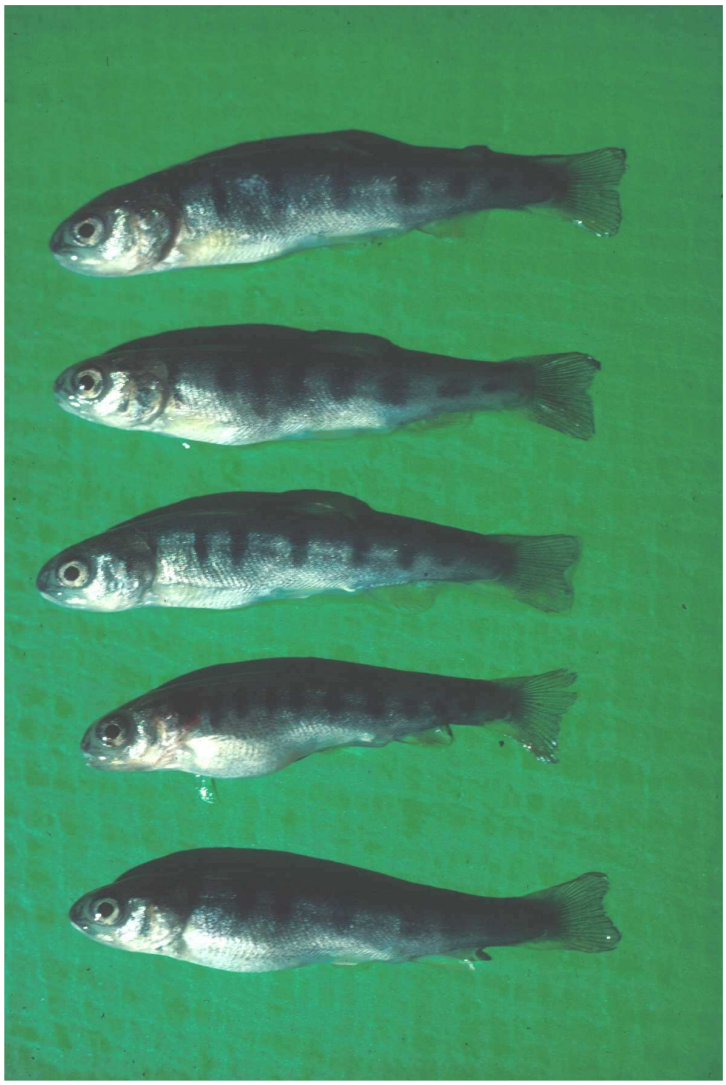
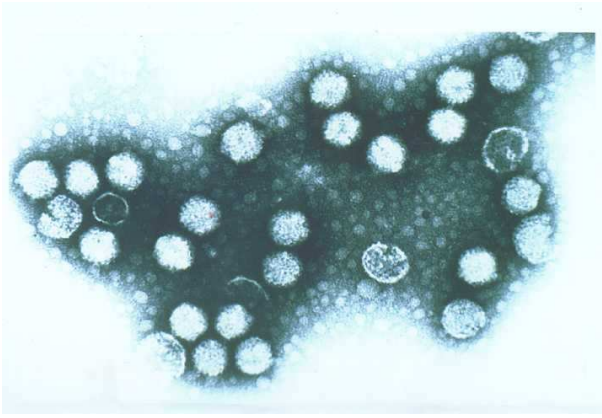


Above: IPN in hatchery reared rainbow trout (*Oncorhynchus mykiss*) from Norway. Fry showing swollen bellies typical of IPN infection. Photo by T. Håstein.

Below: Electron micrograph of negatively stained IPNV, showing typical icosahedral shape. Photo by N. Boustead.



Disease: Infectious salmon anaemia (ISA)

Species and life stage affected: Causes disease in growout sized Atlantic salmon (*Salmo salar*) and coho salmon (*Oncorhynchus kisutch*). Other salmonids, including brown trout (*Salmo trutta*) and rainbow trout (*Oncorhynchus mykiss*), may be covertly infected without showing signs of disease and hence may act as reservoirs of infection.

Gross signs: Infected fish are often lethargic and have difficulty maintaining a horizontal position in the water. The infection is characterised by anaemia (pale gills and heart), ascites (fluid in the body cavity), petechial haemorrhage of the visceral fat, and congestion and enlargement of the liver and spleen. The liver is frequently dark but may range in colour from yellow/pale to black. Exophthalmia (pop eye), haemorrhage in the eye chamber, and inflammation of the foregut may be present.

Causative agent: Infectious salmon anaemia virus (ISAV), an enveloped single-stranded RNA virus belonging to the family Orthomyxoviridae (see Krossoy et al. 1999).

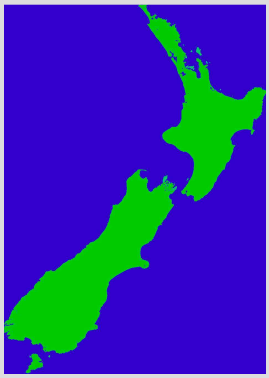
Diagnosis: Isolation of the virus from kidney or spleen in salmon head kidney (SHK-1) or chinook salmon embryo (CHSE-214) cell lines, followed by identification by immunofluorescence and RT-PCR. Other diagnostic methods include examination of kidney imprints by IFAT and histopathology of the liver which demonstrates multifocal haemorrhagic hepatic necrosis that may become confluent, leaving areas around large veins intact – a typical symptom of advanced stages of infection. Broodstock can be screened for ISAV by RT-PCR of ovarian fluids (Melville & Griffiths 1999).

Treatment and prevention:. No known treatment. Prevention is based on slaughter and disinfection of affected facilities, and reducing the risk of ISA transfer by the implementation of good husbandry and sanitary practices (e.g., use of ISA free broodstock, disinfection of eggs with iodophors (100 ppm for 10 minutes), use of footbaths and brushes, detergent and disinfectant sprays, effluent disinfection systems). The virus is inactivated by sodium hypochlorite (bleach) (100–1000 mg/L for a minimum of 10 minutes) or iodophors (100–200 mg/L for 5 minutes) ozone (8 mg/L/min for 3 minutes), and UV radiation (Oye & Rimstad 2001).

Distribution in New Zealand

Unreported.

ISA IS REGARDED AS AN INTERNATIONALLY SIGNIFICANT DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: ISA was first described in Norway in 1984 (Roberts 1998) and spread to Canada in 1997 (Lovely et al. 1999), Scotland in 1998, and Chile in 1999 (Kibenge et al. 2001).

General comments: ISA is a contagious viral disease mainly 0f farmed Atlantic salmon (*Salmo salar*). The virus is transmitted via seawater, fish wastes and mucus, fish blood, and waste waters from processing plants. It is uncertain whether true vertical transmission occurs (Melville & Griffiths 1999). ISA outbreaks have been closely linked with horizontal transmission of infection in seawater, and therefore use of untreated seawater in the production phase in hatcheries is not recommended. Live fish movements have been identified as a major risk factor in the spread of ISA.

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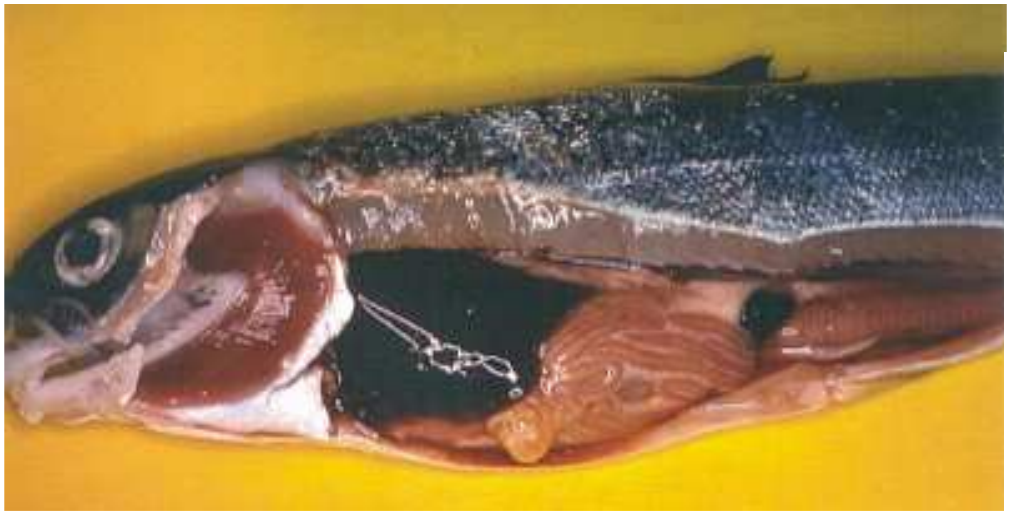
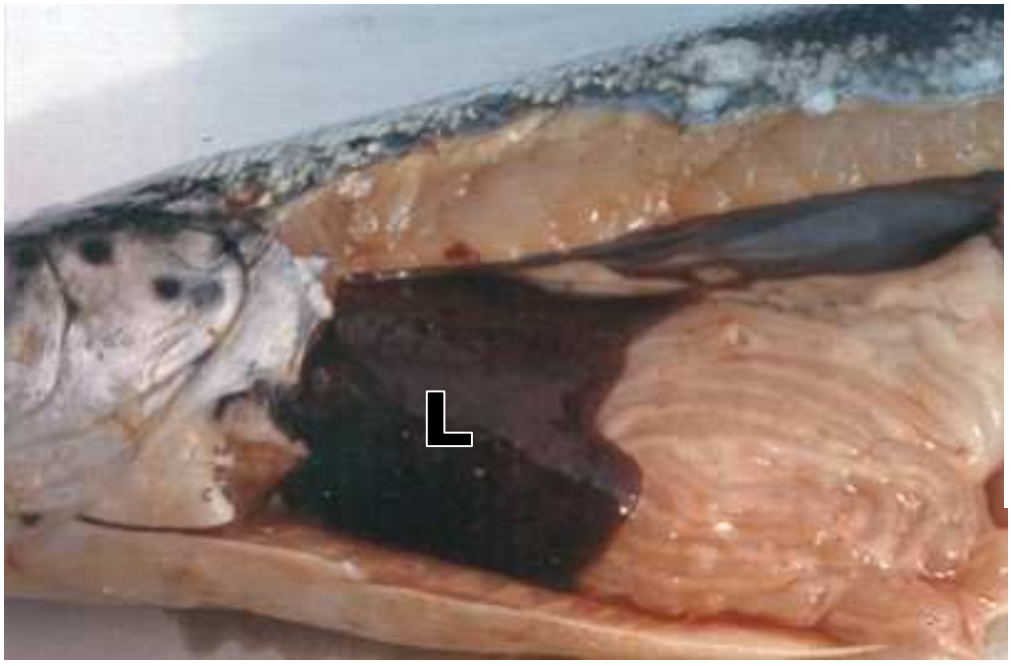


Gross appearance of Atlantic salmon (*Salmo salar*) from Scotland infected with ISA. Photos by D. Bruno.

Above: A salmon with typical ISA signs including petechial haemorrhage of the visceral fat and darkening and enlargement of the liver and spleen.



Below: Closer view of an enlarged and markedly darkened (almost black) liver (L) of a salmon infected with ISA.

Disease: *Oncorhynchus masou* virus disease (OMVD) (yamame tumour virus, nerka virus, coho salmon tumour virus, *Oncorhynchus kisutch* virus, coho salmon herpesvirus, rainbow trout kidney virus).

Species and life stage affected: Masou salmon (*Oncorhynchus masou*), kokanee salmon (*Oncorhynchus nerka*), chum salmon (*Oncorhynchus keta*), coho salmon (*Oncorhynchus kisutch*) and rainbow trout (*Oncorhynchus mykiss*).

Gross signs: Oedema, haemorrhaging, and heavy mortalities in juvenile fish. The virus multiplies in the endothelial cells of the host, causing cell death and the resulting signs. After 4 months some fish exhibit epithelioma (Yoshimizu et al. 1988), mainly around the mouth. The kidney is severely infected, and partial necrosis of the liver, spleen, and pancreas may occur (Tanaka et al. 1984). In coho salmon, 1 year old fish show ulcers in the skin, white spots on the liver, and neoplasia around the mouth or on body surfaces. Rainbow trout may be largely asymptomatic, but may have skin ulcers, intestinal haemorrhages and white spots on the liver.

Causative agent: *Oncorhynchus masou* virus (OMV), a herpesvirus.

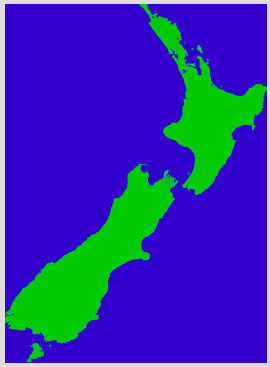
Diagnosis: Isolation of the virus in cell cultures, serum neutralisation, IFAT, and ELISA.

Treatment and prevention: No known treatment. Disinfection of eggs just after fertilisation or at the eyed stage with iodophors gives effective prevention.

Distribution in New Zealand

Unreported.

OMVD IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Japan only.

General comments: Clinical signs and course of disease vary with host species and age.

References

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No photographs currently available

Disease: Spring viraemia of carp (SVC)

Species and life stage affected: Cyprinid fishes. Overt infections occur in common carp and koi carp (*Cyprinus carpio*), grass carp (*Ctenopharyngodon idellus*), silver carp (*Hypophthalmichthys molitrix*), bighead carp (*Aristichthys nobilis*), crucian carp and goldfish (*Carassius auratus*), tench (*Tinca tinca*), roach (*Rutilus rutilus*), sheatfish (*Silurus glanis*), pumpkinseed (*Lepomis gibbosus*), and guppies (*Lebistes reticulatus*). Young more susceptible than adults.

Gross signs: Usually occurs during springtime. Lethargy, lying on the bottom, slow reaction to sensory stimuli. Oedema, haemorrhaging, enteritis and peritonitis, skin darkening, swollen belly, exophthalmia. Petechial and congestive haemorrhaging in the skin, gills, and anterior eye, anaemia, and protrusion and inflammation of the vent. The faecal casts are long, white to yellowish, and mucoid. Abdominal adhesions of the viscera. The virus multiplies in the endothelial cells (which line the blood vessels), haematopoietic tissues of the kidney, and nephron cells.

Causative agent: Spring viraemia of carp virus (SVCV), a rhabdovirus.

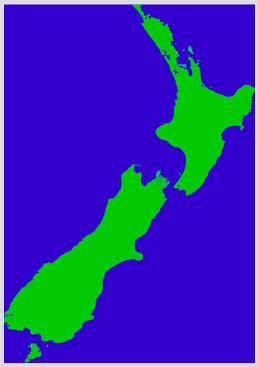
Diagnosis: Isolation and culture of the causative virus from the kidney, spleen, brain or gills of infected fish, serum neutralisation, IFAT, and ELISA.

Treatment and prevention: No known treatment. Prevention is by controls on movement, slaughter of infected stock, and disinfection of affected facilities.

Distribution in New Zealand

Unreported.

SVC IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Continental Europe.

General comments: The reservoirs of infection are clinically infected fish and asymptomatic carriers among cultured or wild fish. The virus is shed via faeces, urine, gill and skin mucus, and exudates of skin blisters. Transmission is horizontal, but possibly also vertical. The virus may also be spread by vectors such as ectoparasitic copepods and leeches. Susceptibility not only varies between host species, but within a host species. Whether this is due to innate immunity or the age of the fish is unclear. Temperature is very important in virulence. For example, under experimental conditions, high mortality occurs within 10–15 days at 16–17 °C, but later at 11–15 °C. The disease does not develop in fish kept constantly at 18 °C. In yearling or older fish, overt infection is not often observed at over 17 °C, whereas fry may be infected at 22–23 °C. The shedding of virions from asymptomatic fish at 13–14 °C during winter may be an important factor in transmission.

Reference

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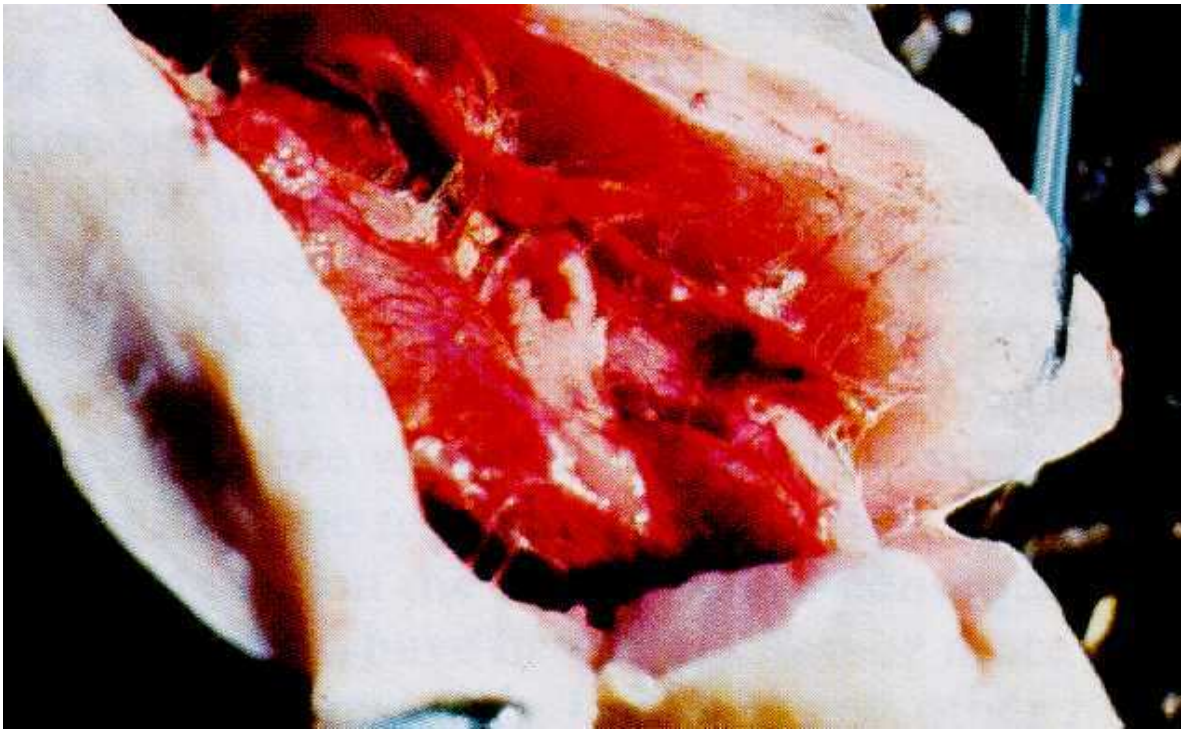


Spring viraemia of carp in a common carp from Europe. (Photo reprinted from Fish Pathology, 2nd edition, Roberts, R.J. (ed.) 1989, Plate 6.3, by permission of the publisher, Bailliere Tindall).

This fish is exhibiting severe haemorrhages, peritonitis and adhesions of the viscera due to infection with SVCV.

Disease: Viral haemorrhagic septicaemia (VHS)

Species and life stage affected: Fry, fingerlings, and adults of most salmonids, and also other species including turbot (*Scophthalmus maximus*), sea bass (*Dicentrarchus labrax*), Atlantic cod (*Gadus morhua*), Pacific cod (*Gadus macrocephalus*), and Pacific herring (*Clupea harengus pallasi*).

Gross signs: Rainbow trout (*Oncorhynchus mykiss*) fry become lethargic, darker in colour, and swim erratically with a corkscrew motion, or on the surface. They may exhibit exophthalmia with haemorrhaging around the eye, and pale gills. Internally, extensive haemorrhaging, with a swollen dark kidney that is necrotic in the anterior and mid-sections. The liver may be pale or yellowish with haemorrhaging giving a mottled appearance. Histopathologically the kidney is the most affected organ, showing extensive necrosis of the haemorrhagic tissues. Focal necrosis occurs in the liver.

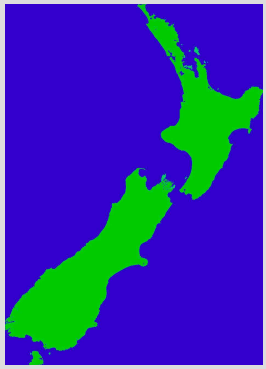
Causative agent: Viral haemorrhagic septicaemia virus (VHSV), a novivirus (family Rhabdoviridae).

Diagnosis: VHSV may be isolated in a variety of cell-lines, and identified by serum neutralisation. The virus may also be identified by IFAT or ELISA tests, with gene probes, by PCR, or by monoclonal antibody capture and PCR.

Treatment and prevention:. No known treatment. Sterilisation of intake and waste water by UV irradiation is useful in controlling VHS in farms (Yoshimizu et al. 1986, Oye & Rimstad 2001). Development of vaccines has provided protective immunity in rainbow trout (Enzmann et al. 1997, Lorenzen et al. 1998), but other vaccines will be needed against other strains of the disease. Transmission is horizontal and fish in waters surrounding farms may become carriers from exposure to farm wastewater. Therefore farms may be re-infected from the wild after the disease has been eradicated from the farms.

Distribution in New Zealand

Unreported, although farmed salmonids are under active surveillance.



VHS IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: Freshwater VHS occurs in continental Europe and the Mediterranean. Marine VHS occurs in the North Sea, off the coast of Alaska (Meyers et al. 1994) and in Japan (Isshiki et al. 2001).

General comments: VHS occurs as many strains and some of the clinical signs may be confused with IHN and IPN. A reverse transcriptase-PCR technique has been developed to rapidly differentiate the VHS and IHN viruses (Miller et al. 1998). Stress, the age of the host (rainbow trout fry are the most susceptible), and temperature (VHS is a cold water disease) predispose stocks to infection. The virus enters the fish through the gills, and spreads quickly via the blood. It enters endothelial cells, and then to the kidney where it causes severe necrosis of the blood forming tissues. It then spreads to the liver and pancreas where it causes further necrosis.

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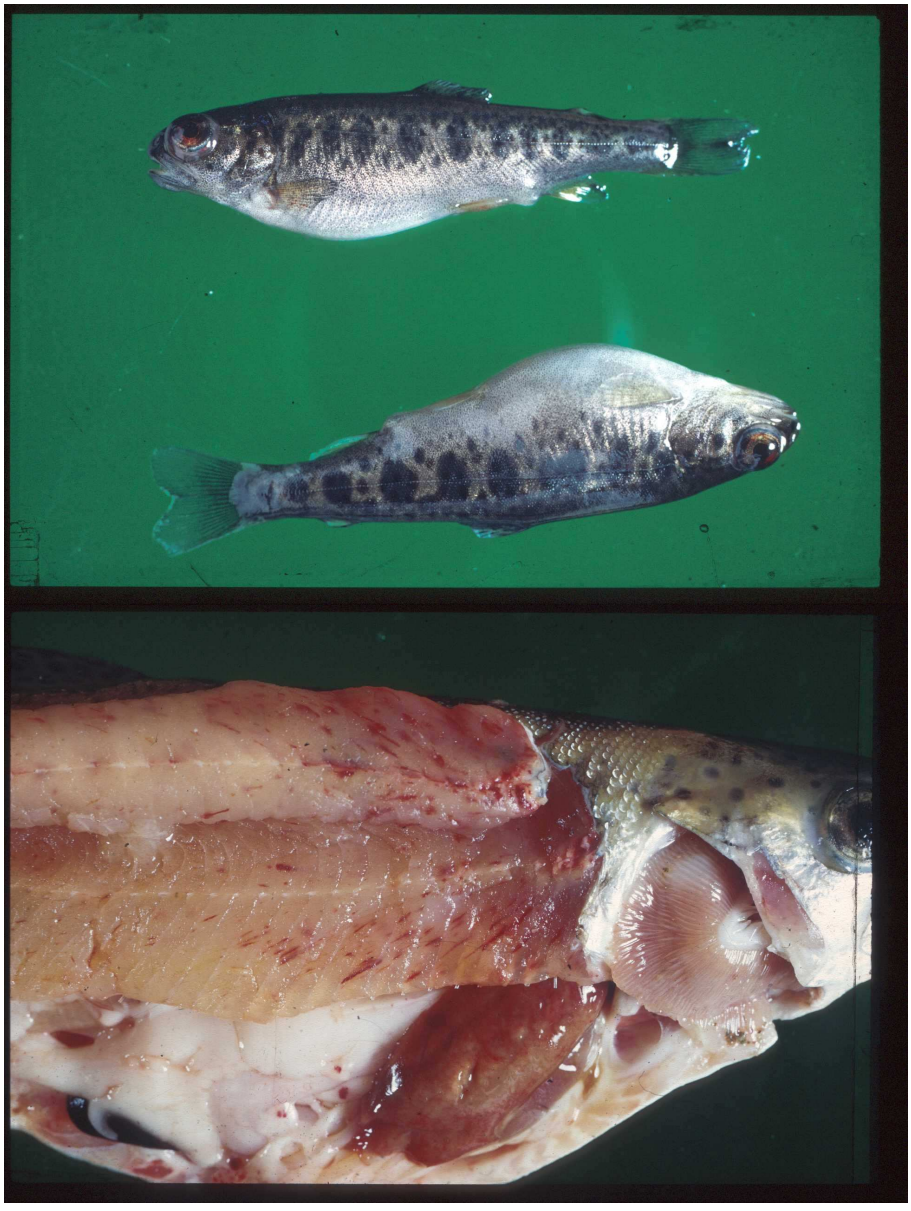


VHS in rainbow trout (*Oncorhynchus mykiss*) from Norway. Photos by T. Håstein.

Above: Fry with VHS showing markedly swollen bellies and haemorrhages in the eye.

Below: A juvenile rainbow trout with extensive haemorrhaging of the musculature, pale gills and pale, mottled liver, all classical signs of VHS.

Microbial diseases of freshwater fishes

Disease: Atypical *Aeromonas salmonicida* (carp erythrodermatitis, ulcer disease of goldfish, head ulcer disease of Japanese eels).

Species and life stage affected: Most salmonids and other freshwater fish. Also affects marine fish, particularly flatfish.

Gross signs: Skin ulceration.

Causative agents: Bacteria of the genus *Aeromonas*, including *Aeromonas salmonicida masoucida* (salmonids, Japan), *A. salmonicida achromogenes* (salmonids and non-salmonids, in many countries), *A. salmonicida nova* (salmonids and non-salmonids, many countries), *A. salmonicida smithia* (non-salmonids, England), and other un-named *A. salmonicida.*

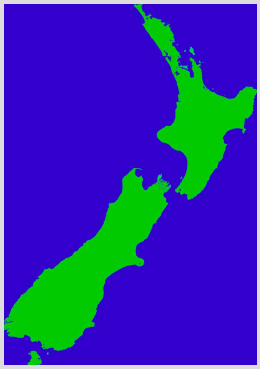
Diagnosis: Isolation onto microbiological media, biochemical profiling, serological identification.

Treatment and prevention: Treatment with antibiotics and, in a few cases such as in carp erythrodermatitis, vaccination.

Distribution in New Zealand

Unreported despite investigation.

ATYPICAL *A. SALMONICIDA* ARE IMPOSSIBLE TO DISTINGUISH FROM FURUNCULOSIS, EXCEPT IN THE LABORATORY. IF YOU SUSPECT A DISEASE MAY BELONG TO THIS GROUP, RING MAF ON 0800-809-966



Worldwide distribution: Atypical strains occur worldwide, except in New Zealand. In Australia they infect goldfish (*Carassius auratus*), silver perch (*Bidyanus bidyanus*), and greenback flounder (*Rhombosolea taparina*).

General comments: Whereas the strains of *A. s. salmonicida* are all very similar, the atypical strains are much more heterogeneous.

References

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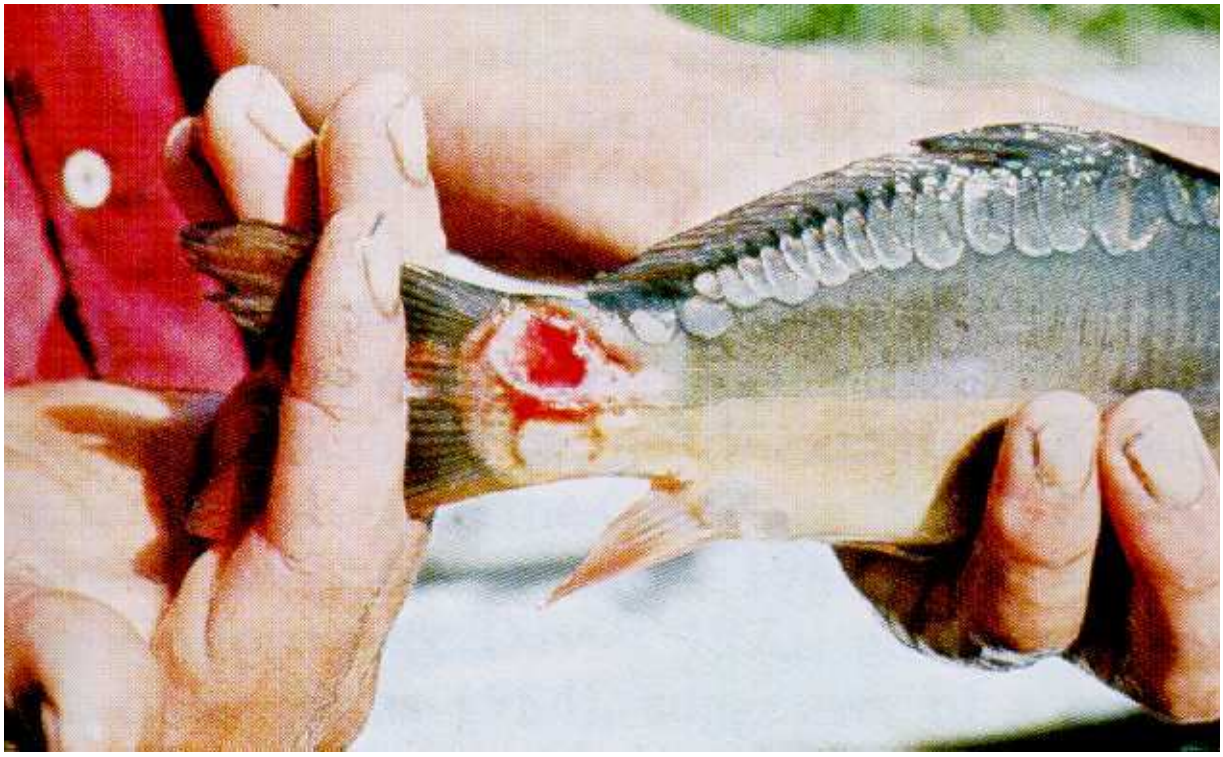


Carp erythrodermatitis in a mirror carp caused by infection with an atypical strain of *Aeromonas salmonicida*. (Photo reprinted from Fish Pathology, 2nd edition, Roberts,R.J. (ed.) 1989, Plate 6.1, by permission of the publisher, Bailliere Tindall).

Note the shallow haemorrhagic ulceration of the skin of the caudal peduncle.

Disease: Bacterial kidney disease (BKD)

Species and life stage affected: Salmonids (*Oncorhynchus* sp., *Salmo* sp., *Salvelinus* sp.) (Fryer & Sanders 1981). Natural infections in Danube salmon (*Hucho hucho*) and grayling (*Thymallus thymallus*).

Gross signs: From complete lack of signs to darkening of the body, distended abdomen, exophthalmia, petechial haemorrhaging, and haemorrhaging near the fins. The overt disease occurs only at advanced stages of infection, when fish have completed their first year of life. Systemic granulomatous lesions can be found in all organs, particularly the kidney. Greyish necrotic abscesses in the kidney multiply until they merge, resulting in diffuse granulomatous lesions throughout the swollen kidney.

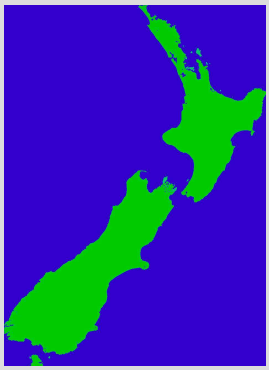
Causative agent: *Renibacterium salmoninarum,* a gram-positive bacterium most closely related to the eubacterial division of the actinomycetes.

Diagnosis: Isolation and bacteriological identification, antigen detection and identification by serological methods, including agglutination testing and immunofluorescence, ELISA, or by PCR. Seropositive results and molecular techniques do not indicate the disease is present in the absence of bacterial isolation.

Treatment and prevention: Erythromycin sulphate given by injection (11 mg/kg), or feeding (200 mg/kg) for 21 days. Controls on movement, segregation of infected and uninfected fish.

Distribution in New Zealand

Unreported even though there have been concerted efforts to find the pathogen in New Zealand salmonids.



BKD IS REGARDED AS AN INTERNATIONALLY SIGNIFICANT DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: North America, western Europe, Japan, Chile, Scandinavia.

General comments: BKD is a chronic disease that may take up to 9 months to present clinical signs after infection. Therefore the disease may become well established before it is detected. Even when clinical signs are apparent, the causative bacterium can be difficult to grow. The bacterium can be transmitted both horizontally and vertically. The condition must be distinguished from proliferative kidney disease (PKD), in which kidney hypertrophy is not associated with any discoloration.

Reference

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BKD in chinook salmon (*Oncorhynchus tshawytscha*) from British Columbia. Photo by N. Boustead.

Note granulomatous lesions evident in the kidney (arrows).

Disease: Enteric redmouth disease (ERM)

Species and life stage affected: Salmonids, and non-salmonids, including sturgeon (*Acipenser baeri*), European eels *(Anguilla anguilla*), goldfish (*Carassius auratus*), channel catfish (*Ictalurus punctatus*), turbot (*Scophthalmus maximus*), and Dover sole (*Solea solea*).

Gross signs: Bacterial septicaemia with anorexia, darkening of the skin, lethargy, subcutaneous haemorrhaging and reddening of throat and mouth. Erosion of the jaw and palate may occur. Haemorrhaging occurs on the body surface, at the gill tips, at the base of the fins, in the eye, along the lateral line, and at the vent. Internally there is congestion of the blood vessels of the peritoneum, and petechial haemorrhaging in the liver, pancreas, swim bladder, lateral muscles, and adipose tissues. The kidney and spleen may be swollen, with mucoid yellowish matter in the gut.

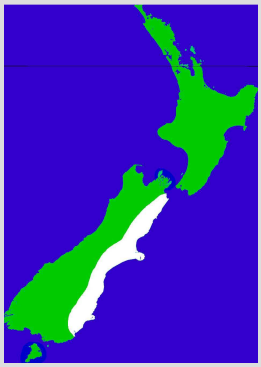
Causative agent: *Yersinia ruckeri*, a gram-negative rod of the Enterobacteriaceae, 1.0 μm in diameter and 2–3 μm in length.

Diagnosis: Isolation on to media and biochemical identification, monoclonal antibodies, ELISA, antibody detection by latex-agglutination, PCR. There have been problems with validation of PCR (Hiney & Smith 1998).

Treatment and prevention: Avoidance of contaminated stocks, disinfection of eggs, vaccination, antibiotic treatment (potentiated sulfonamides). ERM is often an indicator that fish are stressed, possibly due to poor water quality, poor nutrition, overcrowding, high temperatures (peak mortality in summer, no infection below 10 °C), or high levels of suspended organic matter. Handling may trigger the disease in fish that are already stressed but appear healthy. Avoidance of these conditions will prevent disease.

Distribution in New Zealand

ERM has been isolated in salmon hatcheries on the east coast of the South Island. Improvements in fish husbandry have reduced the incidence of ERM in recent years.



Worldwide distribution: North America, Europe, Iran, South Africa, Australasia, Chile.

General comments: In chinook salmon in New Zealand the most obvious sign of ERM is the presence of blood spots in the eye: however, these signs can be confused with other septicaemias.

Reference

Horne, M.T.; Barnes, A.C. (1999). Enteric redmouth disease (*Yersinia ruckeri*). *In*: Woo, P.T.K.; Bruno, D.W. (eds), Fish diseases and disorders, Vol. 3. pp. 455–477. CAB International, Wallingford.

ERM in rainbow trout. Photos by H. Schlotfeldt.

Above: Classical reddening of the mouth associated with subcutaneous haemorrhaging.
Below: Reddening of the mouth and haemorrhage in the eye.

Disease: Flavobacterial diseases (columnaris, cold-water disease, bacterial gill disease, fin rot)

Species and life stage affected: Probably all species of freshwater fish can suffer some form of flavobacterial disease (Shotts & Starliper 1999).

Gross signs: Columnaris: infection begins at the mouth, fins and gills, as an increase in mucus on the head and upper body, appearing as greyish patches with a yellowish tint. Gills initially have whitish tips, followed by overgrowth of the filament. Spots may appear on the surface, followed by necrosis, septicaemia, and death. Cold-water disease: tissue necrosis (fin rot), which starts as rough skin and loss of fin tips, whitish growth along the fin margin, followed by fin erosion that may finally erode the musculature of the body, exposing the spine. Bacterial gill disease: anorexia, with the fish lining up at the freshwater inlet. Small yellowish-white to grey spots appear on the gills, bleeding may occur, and eventually the gills are smothered in bacteria, leading to death.

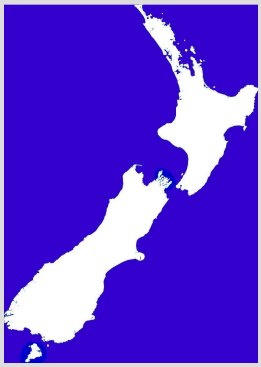
Causative agents: Columnaris (*Flavobacterium columnare*), cold-water disease (*Flavobacterium psychrophilum*), bacterial gill disease (*Flavobacterium branchiophilum*).

Diagnosis: Clinical signs, taken together with prevailing water temperatures, and microscopic examination of scrapings from an eroded area may allow presumptive diagnosis. Definitive diagnosis requires isolation on to cytophaga medium and biochemical identification, though this can sometimes be difficult due to overgrowth by opportunistic bacteria. PCR can be used to distinguish the three pathogens (Bader & Shotts 1998, Wiklund et al. 2000). An ELISA also exists for *F. branchiophilum.*

Treatment and prevention: Improvement in water quality and husbandry are the best preventatives for columnaris and cold-water disease. Similarly, bacterial gill disease thrives when fish are stressed by overcrowding and build up of faeces and uneaten food in the environment. Experimental vaccines have been developed, but chemical treatments are favoured. *F. branchiophilum* can be treated with choramine-T (Bowker & Erdahl 1998), benzalkonium chlorides at 1–2 mg/L (Piper et al. 1983), hydrogen peroxide (Lumsden et al. 1998), or a 5% salt bath (Heo et al. 1990).

Distribution in New Zealand

These bacteria are ubiquitous in the environment and probably occur throughout New Zealand's freshwaters. They usually cause disease only under poor environmental conditions or when fish are stressed.



Worldwide distribution: Ubiquitous worldwide.

General comments: Flavobacterial diseases are often the first to appear when fish are stressed by poor environmental conditions. In New Zealand, bacterial gill disease occurs in chinook salmon fry and yearling sockeye salmon when ammonia levels and turbidity are high, and columnaris disease has been recorded in overcrowded cultured eels and as the cause of bleeding of gills of chinook salmon at water temperatures of 18 °C (Boustead 1989). Cold-water disease has been recorded in New Zealand salmonids and eels at water temperatures of 8–10°C (Jones et al. 1983, Boustead 1989).

References

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Heo, G.J.; Kasai, K.; Wakabayashi, H. (1990). Occurrence of *Flavibacterium branchiophila* associated with bacterial gill disease at a trout hatchery. *Fish Pathology 25*: 99–105.

Jones, J.B.; Astill, M.; Kerei, E. (1983). The pond culture of *Anguilla australis* in New Zealand – with special reference to the experimental farm at TeKaha. *Rivista Italiana di Piscicoltura e Ittiopatologia 18*: 85–117, 138–166.

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Wiklund, T.; Madsen, L.; Bruun, M.S.; Dalsgaard, I. (2000). Detection of *Flavobacterium psychrophilum* from fish tissue and water samples by PCR amplification. *Journal of Applied Microbiology 88*: 299–307.

Flavobacterial disease in chinook salmon (*Oncorhynchus tshawytscha*). Photo by N. Boustead.

Tissue necrosis associated with flavobacterial infection has caused extensive erosion of the caudal fin and musculature of the caudal peduncle, exposing the spine.

Disease: Furunculosis

Species and life stage affected: Salmonids, eels, carp, and many other species of freshwater fish.

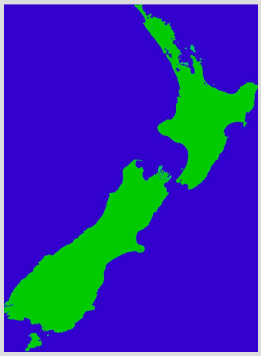
Gross signs: Boil-like lesions on the skin and in the muscle, although furuncles do not always develop on the dorsal body and are more common on older fish with chronic disease. In acute infections of younger fish, death can be so rapid that only exophthalmia will be present. Microcolonies occur in many organs without an inflammatory response or much necrosis. Cardiac damage may be the cause of death. Covert infections display none of these signs. Acute infections in growing fish are characterised by darkening of the skin, poor appetite, lethargy, and haemorrhaging at the base of the fins.

Causative agent: *Aeromonas salmonicida* subspecies *salmonicida*, a non-motile aeromonad bacterium.

Diagnosis: Internally the fish may have haemorrhages in the abdominal wall, viscera, and heart, a soft liquefied kidney, enlarged cherry-red spleen, pale liver with haemorrhages, an empty digestive tract except for mucus and blood, and muscular furuncles. The causative organism can be readily cultured from affected organs on nutrient media and identified as *A. salmonicida.* Several techniques exist to separate typical *A. salmonicida* from atypical strains (antibiogram typing, biotyping, multilocus enzyme electrophoresis, phage typing, *Pseudomonas* inhibitory assay, LPS antibody binding, serotyping, plasmid profiling, DNA techniques). In covert infections the *A. salmonicida* may be amplified by stressing the host.

Treatment and prevention: As diseased fish lose their appetites, oral antibiotic treatment only prevents new infections. Standard treatments in freshwater are oxytetracycline at 80 mg/kg, oxolinic acid at 10 mg/kg, Romet at 50 mg/kg, florfenicol at 20 mg/kg, and amoxycillin at 40–80 mg/kg, and in seawater are oxytetracycline at 120 mg/kg, oxolinic acid at 30 mg/kg, methasul at 40 mg/kg, and sulfatrim at 60 mg/kg. Vaccination using oil-adjuvant injection vaccines. Site rotation and farm fallowing, by removing the host biomass for a period, reduces infection pressure.

Distribution in New Zealand



Absent. Furunculosis has never been found, despite investigation (Anderson et al. 1994).

FURUNCULOSIS PROBABLY POSES THE GREATEST SINGLE THREAT TO FISH AQUACULTURE IN THIS COUNTRY. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: Worldwide except New Zealand and South America.

General comments: Should this disease be introduced into New Zealand, it would affect not only salmonid aquaculture, but many other species of freshwater fish. This disease is particularly likely to affect native galaxiid fishes, which are salmoniform fishes.

References

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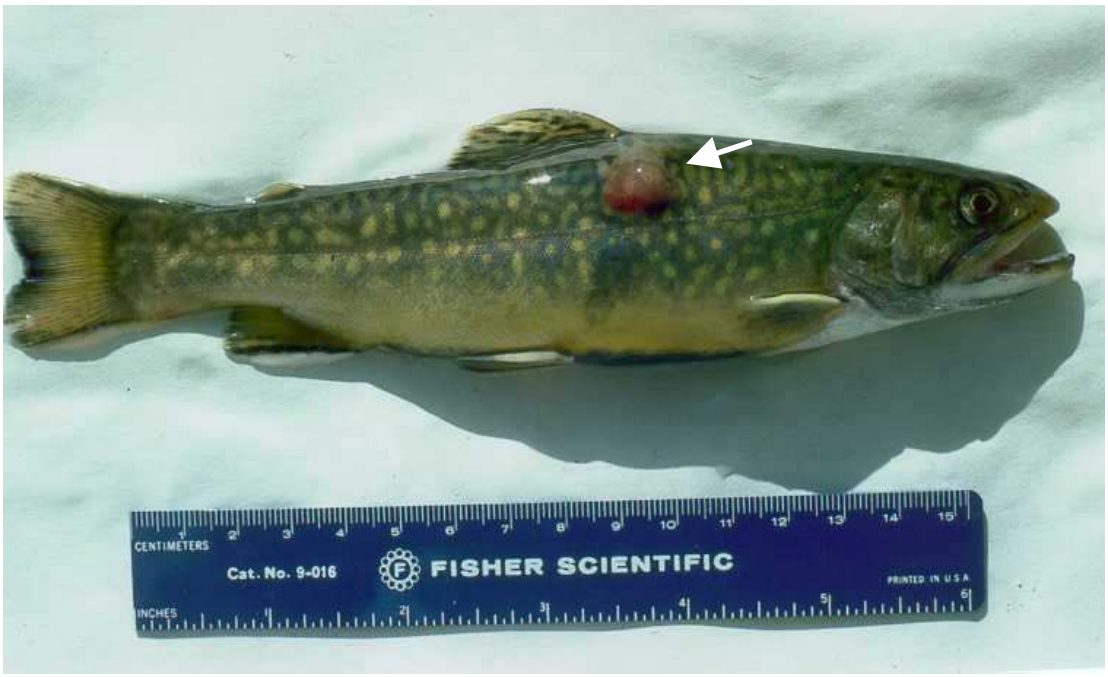
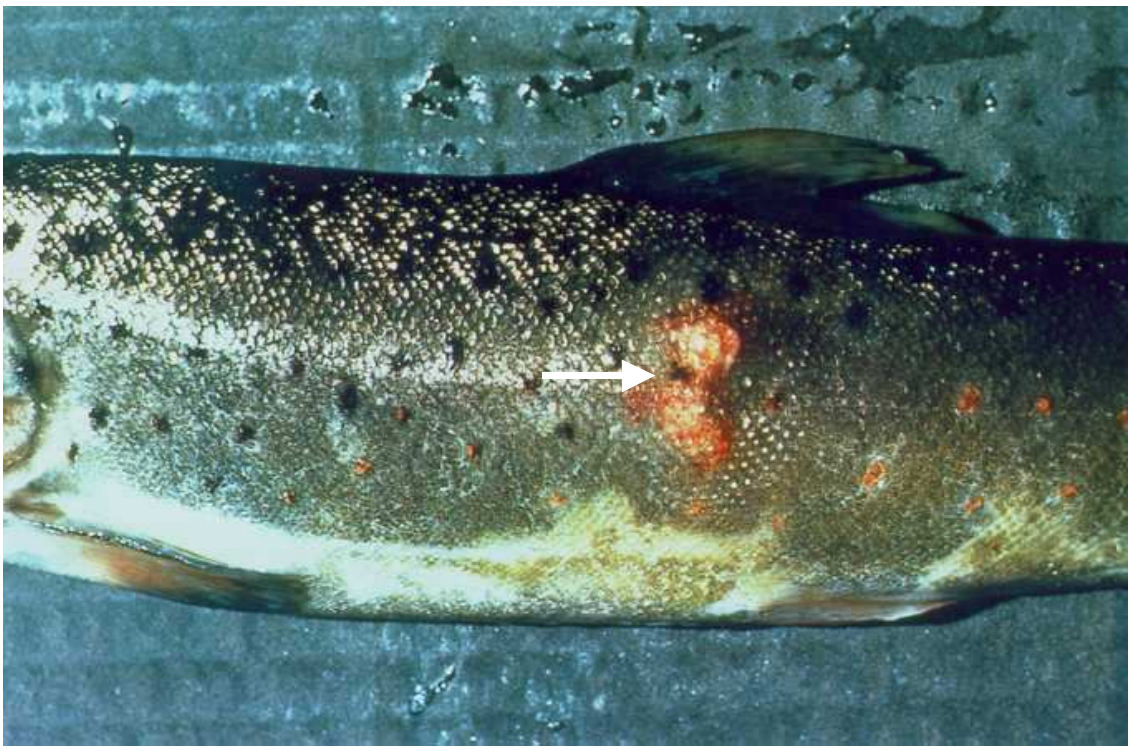


Furunculosis caused by *Aeromonas salmonicida*. Photos by N. Boustead.

Above: A brook trout from the USA with furunculosis exhibiting a furuncle (arrow) in the dorsal muscle.
Below: A classic furuncle (arrow) on the side of a brown trout with furunculosis.



Disease: Haemorrhagic septicaemia (*Aeromonas hydrophila* complex)

Species and life stage affected: Most cultured and wild freshwater fish are susceptible to infection, particularly coldwater species (salmonids, cyprinids, anguillids).

Gross signs: Haemorrhagic septicaemia. Cutaneous haemorrhage and ulceration of the fins, trunk, and stomach. In carp, tail and fin rot may be present.

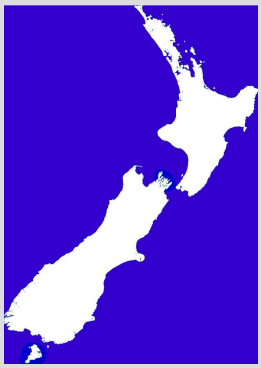
Causative agent: *Aeromonas hydrophila* , a motile aeromonad bacterium.

Diagnosis: Isolation on to media and biochemical identification. Techniques such as gel-diffusion, direct and indirect fluorescent antibody techniques, immunoblotting, and ELISA are available, but are of limited use because many strains of *A. hydrophila* exist in fish farms. Ribotyping, ISH, and PCR give more precise results.

Treatment and prevention: *A. hydrophila* is common and free-living in water, but it becomes an opportunist pathogen when fish are stressed by overcrowding, high temperatures, sudden temperature change, handling, transport, and low dissolved oxygen. The rectification of such husbandry problems is the best and quickest solution. Antibiotic (florfenicol, tetracycline, sulphonamides, nitrofuran derivatives, and pyridonecarboxylic acids) treatment may also be effective. However, if the poor husbandry is not rectified, the disease will return.

Distribution in New Zealand

Aeromonas hydrophila is common in freshwater all around New Zealand.

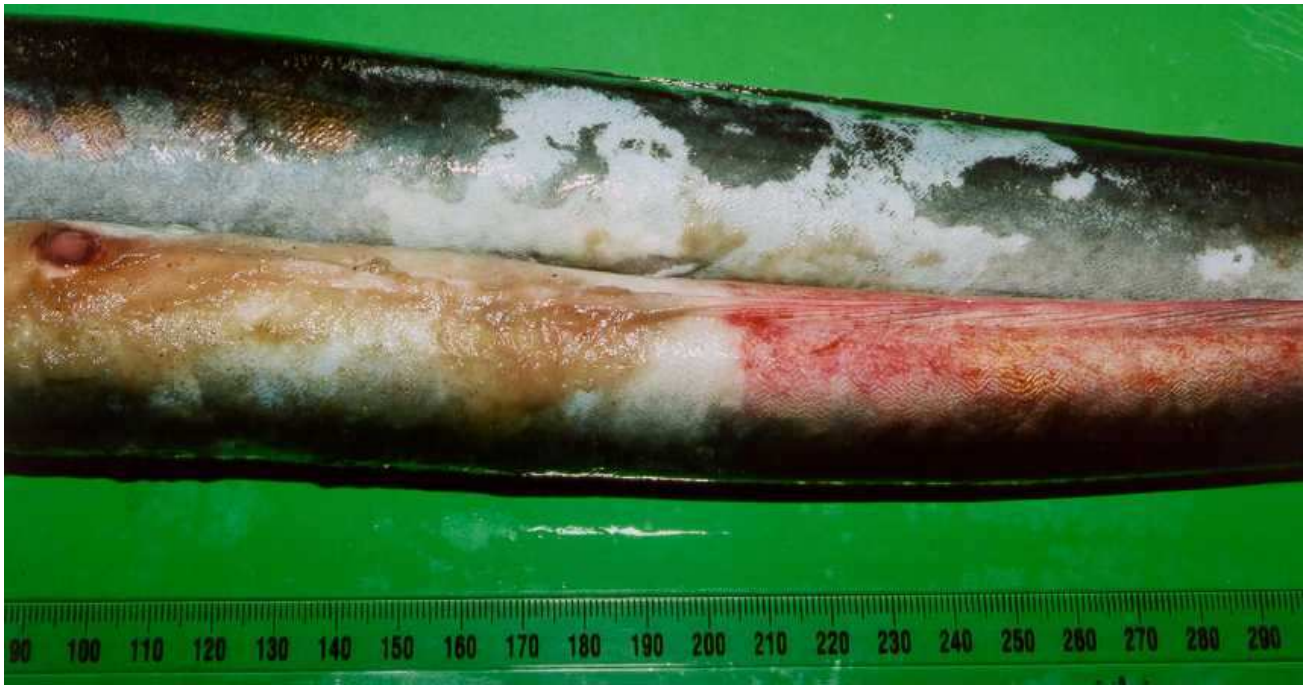


Worldwide distribution: Occurs worldwide.

General comments: In some circumstances, signs of *A. hydrophila* septicaemia may be confused with furunculosis, but as *A. hydrophila* is a motile aeromonad, and *A. salmonicida* a non-motile aeromonad, they can be presumptively distinguished in smears of infected organs.

Reference

Aoki, T. (1999). Motile aeromonads (*Aeromonas hydrophila*). *In*: Woo, P.T.K.; Bruno, D.W. (eds), Fish diseases and disorders, Vol. 3. pp. 427–453. CAB International, Wallingford.



Haemorrhagic septicaemia in eels (*Anguilla australis*). Photos by B. Diggles.

Above: Note large necrotic ulcers in the skin on the flanks of both eels. The lower eel also exhibits haemorrhage of the anal fin, and rectal prolapse.
Below: Typical appearance of ulcerative lesions in the skin of an eel with haemorrhagic septicaemia.

Disease: Mycobacteriosis (tuberculosis)

Species and life stage affected: Juveniles and adults of many species of freshwater fish, particularly ornamental fish.

Gross signs: Cessation of feeding, emaciation, exophthalmia, external ulceration, discoloration, multiple nodules in the skin and internal organs. This disease is chronic and may take several years to progress from the asymptomatic state to clinical illness.

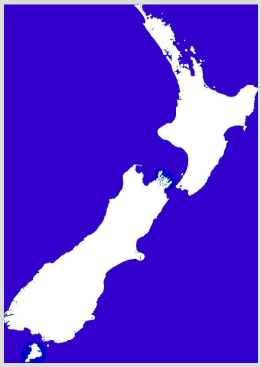
Causative agents: Acid-fast, gram-positive bacteria of the genus *Mycobacterium*, usually *M. fortuitum*, *M. marinum*, or *M. chelonei* but also other species.

Diagnosis: Isolation of the bacteria in culture is difficult. Wet smears or routine histopathology, using Zeihl Neelsen stain for acid fast organisms, is used to demonstrate the presence of strongly acid-fast bacilli 1–3 µm long from inside granulomatous lesions, particularly in the liver, kidney, and spleen. Sometimes confused with infections by *Nocardia* sp. Specific diagnosis requires isolation of the bacteria and identification (Chinabut 1998).

Treatment and prevention: Outbreaks of mycobacteriosis in cultured fish are usually related to management factors such as poor water quality and/or high stocking density. However, the disease can also be spread and transmitted vertically to fish larvae via eggs (Chinabut et al. 1994). Chemical treatments are often unsuccessful, especially if administered during the later stages of infection. Antibiotic treatments (rifampicin) may be practicable when used to treat expensive ornamental fish (Dulin 1979).

Distribution in New Zealand

Probably present in ornamental fish throughout New Zealand.



Worldwide distribution: *Mycobacterium* is ubiquitous in the water and sediment (Chinabut 1998), and mycobacteriosis has been recorded in over 150 species of marine and freshwater fish worldwide (Nigrelli & Vogel 1963, Austin 1988).

General comments: These bacteria can also infect humans, causing allergic skin reactions or infections (Barrow & Hewitt 1971), hence caution should be exercised when immersing arms or hands with open wounds into affected aquariums.

References

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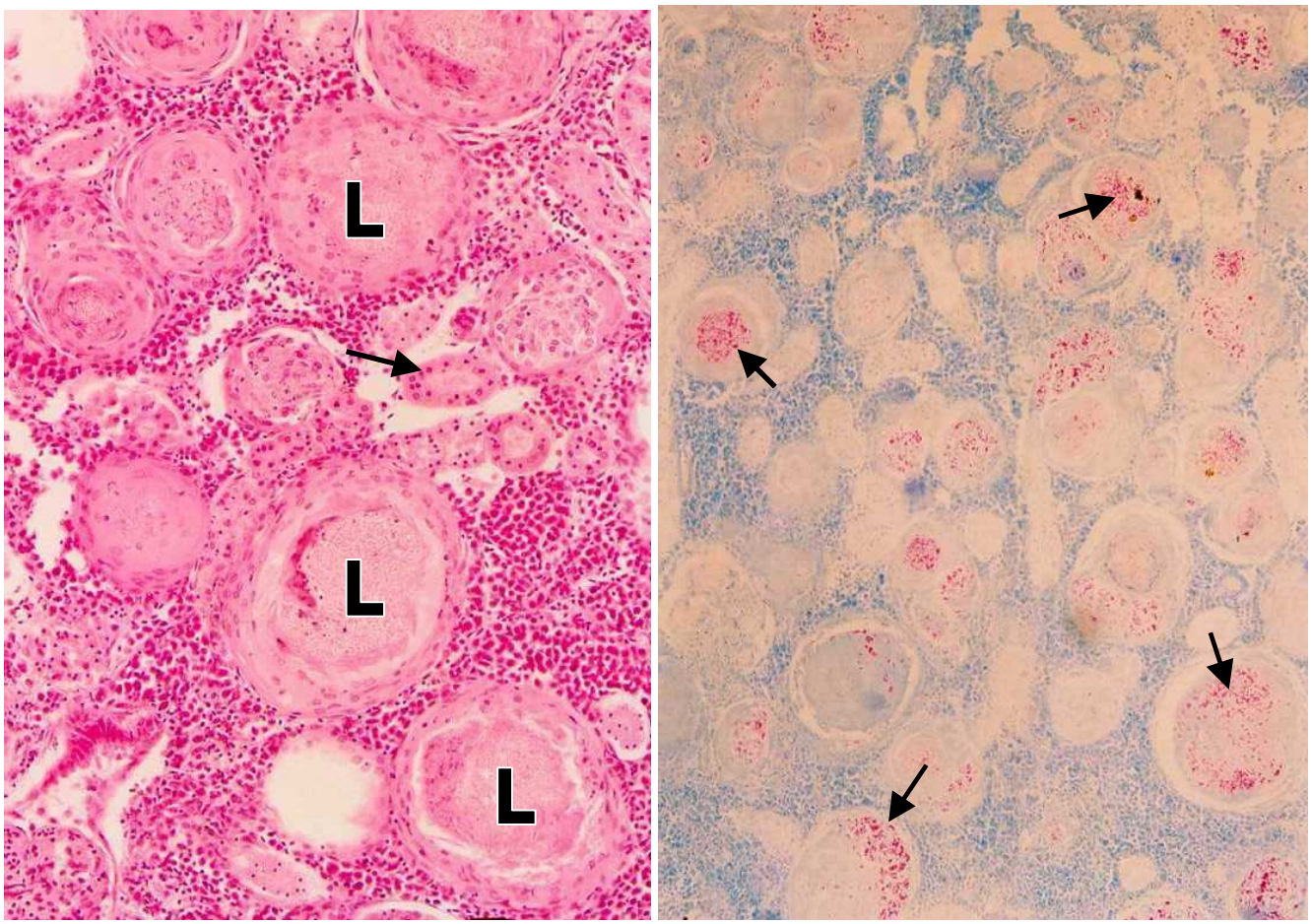


Mycobacteriosis in an ornamental fish (Australian rainbowfish, *Melanotaenia* sp.). Photos by B. Diggles.

Left: Histological section of the kidney showing numerous necrotic granulomatous lesions (L) associated with massive destruction of the kidney tubules (arrow).

Right: Same kidney stained with Zeihl Neelsen stain showing numerous red staining acid fast *Mycobacterium* sp. (arrows) inside the lesions.

Disease: Vibriosis (*Vibrio anguillarum*)

Species and life stage affected: Affects most species of freshwater fish, including salmonids and eels, particularly when they occur in brackish waters or are reared in seacages.

Gross signs: Red spots on the ventral and lateral areas of the fish, and swollen regions of skin which develop to ulcers and release a bloody exudate. Corneal lesions. In severe infection, mortality is so rapid that no signs may be present.

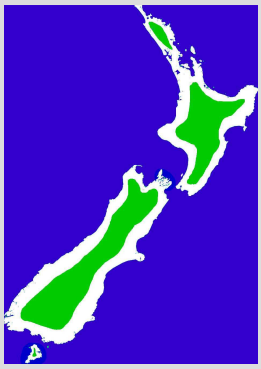
Causative agent: *Vibrio anguillarum*, a gram-negative, curved bacterium with a polar flagellum.

Diagnosis: Isolation on to media and biochemical identification. Serotyping, fluorescent antibody tests, monoclonal antibodies (Hanna et al. 1991), ELISA, and specific oligonucleotide probes are available (Ito et al. 1995). Bacteraemia develops in the early stages of infection, with changes in the blood, loose connective tissue, kidney, spleen, gills, and posterior intestinal tract. This contrasts with *Vibrio ordalii* infection, in which bacteraemia occurs late in infection, with changes mainly in the skeletal and cardiac muscle, digestive tract, and gills (Ransom et al. 1984).

Treatment and prevention: Antibiotics (ampicillin, oxytetracycline, Romet, oxolinic acid, sarafloxacin, erythromycin, streptomycin, nalidixic acid derivatives, nitrofuran derivatives, sulphonamides, and trimethoprim) may be used (Giles et al. 1991), but are not favoured because of the development of antibiotic resistance. Effective vaccines are available, and also probiotics may be used to suppress proliferation (Austin et al. 1995). Disinfectants may be used to clean contaminated sites (Sako et al. 1988), and UV to sterilise water (Sako & Sorimachi 1985).

Distribution in New Zealand

Vibrio anguillarum occurs in New Zealand estuarine and marine waters.



Worldwide distribution: Occurs worldwide.

General comments: Again the occurrence of disease is a combination of many factors including, virulence of the strain, environmental factors, stressors among the fish stocks, fish genetics, and age.

References

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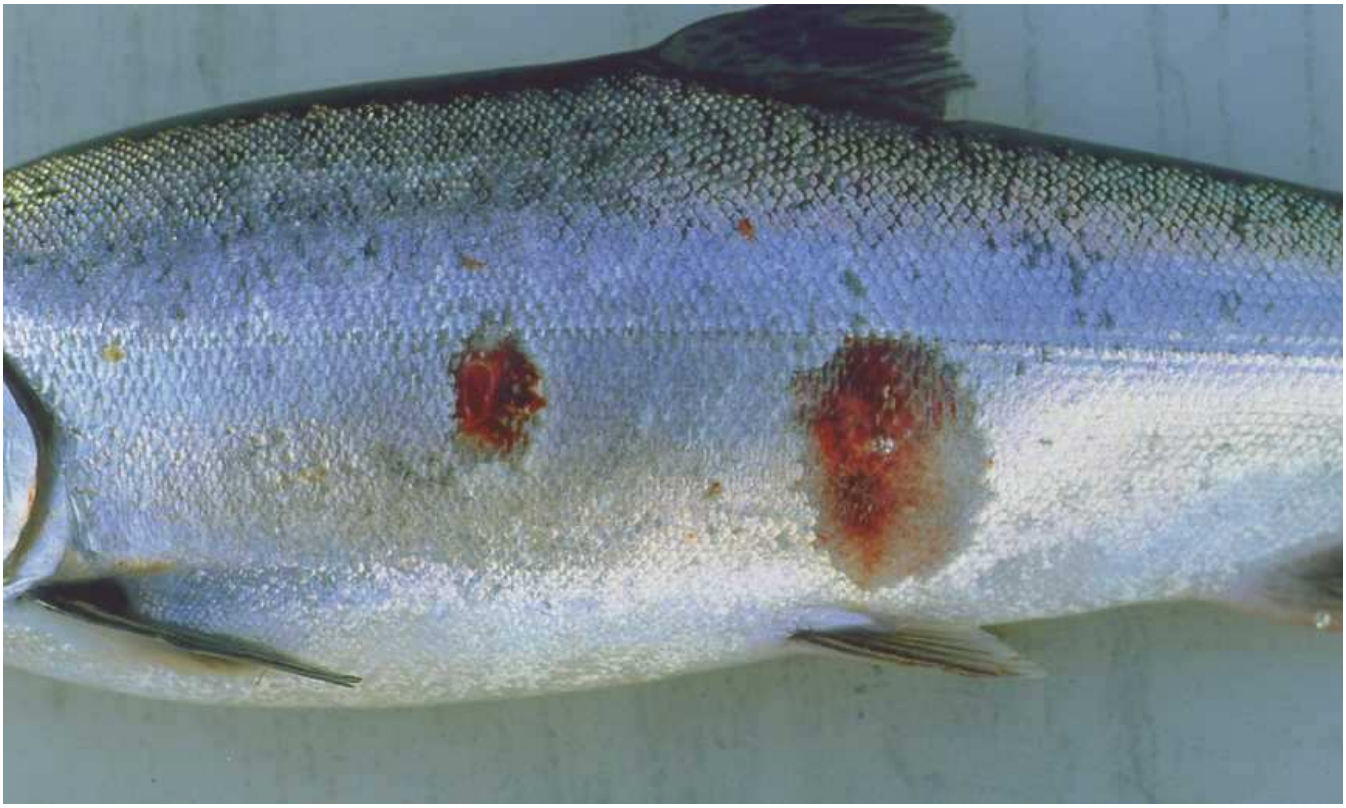


Vibriosis in chinook salmon (*Oncorhynchus tshawytscha*). Photo by N. Boustead.

These two reddened, focal ulcerative lesions in a seacage reared salmon are suggestive of infection by *Vibrio anguillarum*.

Disease: Vibriosis (*Vibrio ordalii*)

Species and life stage affected: Chinook salmon (*Oncorhynchus tshawytscha*), sockeye salmon (*Oncorhynchus nerka*), and other salmonids**.**

Gross signs: Selectively infects the muscle and skin and may be able to actively penetrate the skin. It is also found in the gills, and digestive tract which may act as other portals of entry. The spleen, liver, and blood are less often infected.

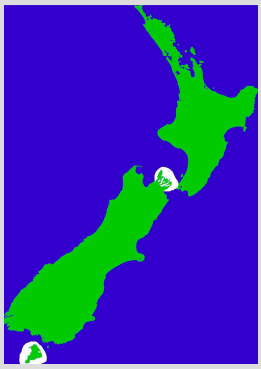
Causative agent: *Vibrio ordalii* , a gram-negative, curved bacterium with a polar flagellum.

Diagnosis: Isolation on to media and biochemical identification. Serotyping, fluorescent antibody tests, monoclonal antibodies (Hanna et al. 1991), ELISA, and specific oligonucleotide probes are available (Ito et al. 1995). In *Vibrio ordalii* infection, bacteraemia occurs late in infection, with changes mainly in the skeletal and cardiac muscle, digestive tract, and gills. This contrasts with *V. anguillarum* infection in which bacteraemia develops in the early stages of infection, with changes in the blood, loose connective tissue, kidney, spleen, gills, and posterior intestinal tract (Ransom et al. 1984).

Treatment and prevention: Antibiotics (oxytetracycline, Romet-30, oxolinic acid, sarafloxacin, erythromycin, streptomycin) may be used (Giles et al. 1991), but are not favoured because of the development of antibiotic resistance. Effective vaccines are available, and probiotics may be used to suppress proliferation (Austin et al. 1995). Disinfectants may be used to clean contaminated sites (Sako et al. 1988), and UV to sterilise water (Sako & Sorimachi 1985).

Distribution in New Zealand

Reported from chinook salmon (*Oncorhynchus tshawytscha*) from Owaka, Stewart Island, and the Marlborough Sounds, and sockeye salmon (*Oncorhynchus nerka*) from the Marlborough Sounds (Wards et al. 1983).



Worldwide distribution: Pacific northwest of the U.S.and Japan.

General comments: It is thought that *V. ordalii* infections are acquired while fish are kept in sea cages. All infections recorded in New Zealand were from fish in estuaries or marine areas.

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Vibriosis in chinook salmon (*Oncorhynchus tshawytscha*). Photo by N. Boustead.

The vibriosis lesion near the vent of this salmon was probably associated with handling damage.

Protozoan diseases of freshwater fishes

Disease: Amoebic granulomatosis

Species and life stage affected: Juvenile and adult goldfish, *Carassius auratus*.

Gross signs: None in early stages. In advanced infections cessation of feeding, multiple internal lesions, rapid increase in mortalities.

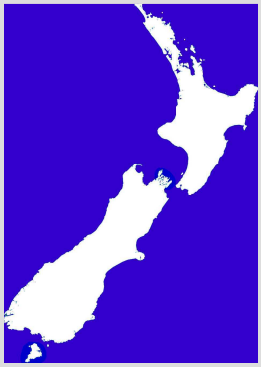
Causative agent: Unclassified amoeba-like organisms within internal organs.

Diagnosis: Definitive diagnosis requires histology to detect numerous small (2–3 µm) amoebae-like protozoans at the periphery of granulomatous lesions in internal organs, particularly the kidney and liver. The protozoans can also be found inside macrophages, where they apparently can survive and divide. Staining with the Zeihl Neelsen method is required to eliminate mycobacteriosis, which causes grossly similar lesions.

Treatment and prevention: No known treatment. Transmission is horizontal, hence quarantine of new introductions before stocking tanks is recommended. This disease appears to increase in prevalence in the warmer months and is associated with high stocking densities at water temperatures above 20 °C. Lowering of the water temperature and reducing stocking density would therefore appear likely methods for limiting mortalities associated with this disease.

Distribution in New Zealand

Probably present in goldfish throughout New Zealand.



Worldwide distribution: Amoeba-like organisms occur throughout the range of cyprinid fish worldwide, including Asia, Europe, the Middle East and the United States (Voelker et al. 1977, Lom & Dykova 1992).

General comments: Amoebae recorded to be parasitic in the internal organs of freshwater fishes include members of the genera *Acanthamoeba*, *Hartmanella*, *Naegleria*, *Vahlkampfia* and *Vexillifera* (see Lom & Dykova 1992, Dykova et al. 1996). Most of these are non-pathogenic in the digestive tract, however, hence the identity of the amoeba-like organisms which cause granulomatous lesions in internal organs in goldfish remains unconfirmed (Dykova et al. 1996). Other potential causative agents include poorly known *Dermocystidium*-like organisms (Landsberg & Paperna 1992).

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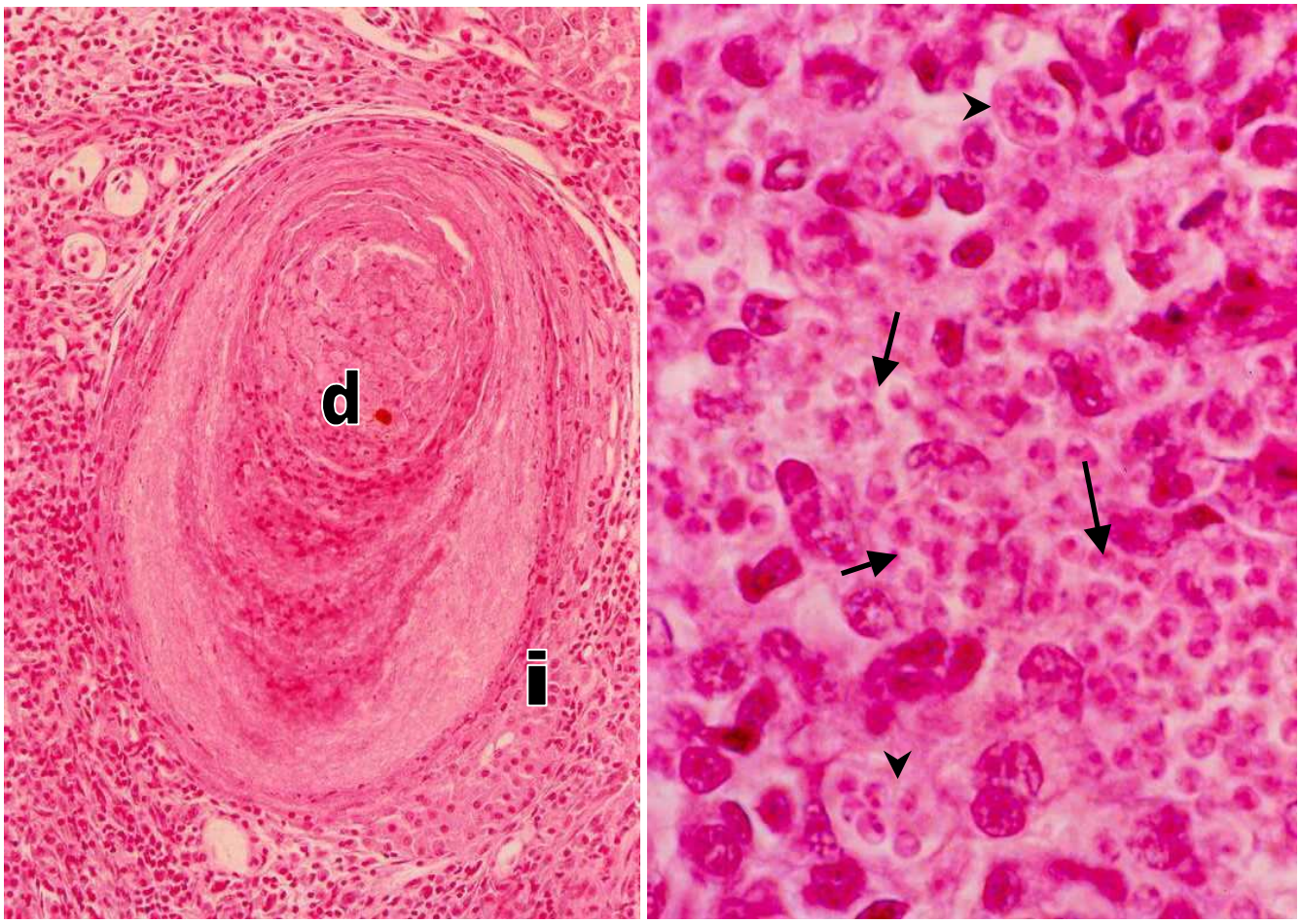


Amoebic granulomatosis in goldfish. Photos by B. Diggles.

Left: Low power view of a histological section of a granulomatous nodule in the kidney. The nodules consist of an inner area of cellular debris (d), surrounded by a layer of amoeba-like protozoans and host inflammatory cells (i) which may be surrounded by a fibrous layer.

Right: High power view of the edge of a nodule showing numerous amoeba-like protozoans both between host inflammatory cells (arrows) and intracellularly within host cells (arrowhead).

Disease: White spot disease (ich)

Species and life stage affected: Juveniles and adults of all species of freshwater fish.

Gross signs: Pinpoint white spots in the epidermis of the skin and fins, flashing and rubbing behaviour, decreased appetite. Lethargy, excessive mucus production, osmotic imbalance, and secondary infections occur in heavy infections.

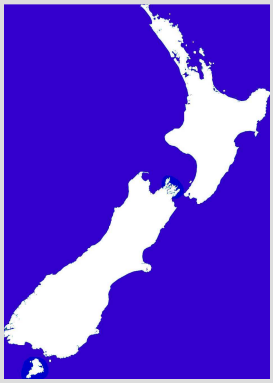
Causative agent: *Ichthyophthirius multifiliis*, a ciliate.

Diagnosis: A presumptive diagnosis can be obtained by observation of pinpoint white spots in the epidermis of the skin and fins. Confirmatory diagnosis by microscopic examination of mucous scrapings containing ciliates under a microscope to demonstrate the distinctive horseshoe shaped macronucleus of *I. multifiliis*.

Treatment and prevention: Heavy infections can quickly lead to epizootics in aquaria or aquaculture when fish are held at high densities, unless effective treatment is administered. UV radiation of 9×10^5 µW s ml^{-1} kills the infective theront stage, as do formalin baths of 25 ppm for 24 hours or adding common salt to the water at a rate of 5 g/L (Selosse & Rowland 1990). Ornamental fish can be treated with 0.1 mg/L malachite green or 2 mg/L methylene blue at 3–4 day intervals until the infections are resolved. Interruption of the lifecycle by daily transfer of fish to different aquaria for 5–7 days is also effective, but places additional stress on affected fish.

Distribution in New Zealand

Ichthyophthirius multifiliis infects wild and captive freshwater fish throughout New Zealand.



Worldwide distribution: *Ichthyophthirius multifiliis* occurs worldwide in wild and ornamental freshwater fish (Dickerson Dawe 1995).

General comments: White spot disease has been the only parasite to cause significant losses in eel culture in New Zealand (Boustead 1982), despite being easily controlled. The life cycle of *I. multifiliis* includes an obligate parasitic stage on the fish called a trophont, and a free living cyst stage called a tomont. Mature trophonts exit the fish and settle on a convenient substrate and encyst, forming a tomont. The tomont divides internally to produce large numbers of infective stages, called theronts. Upon excystment (which occurs less than 24 hours after encystment at 23 °C) the theronts emerge and swim rapidly towards light and fish mucus, which helps them find a fish host (Dickerson & Dawe 1995).

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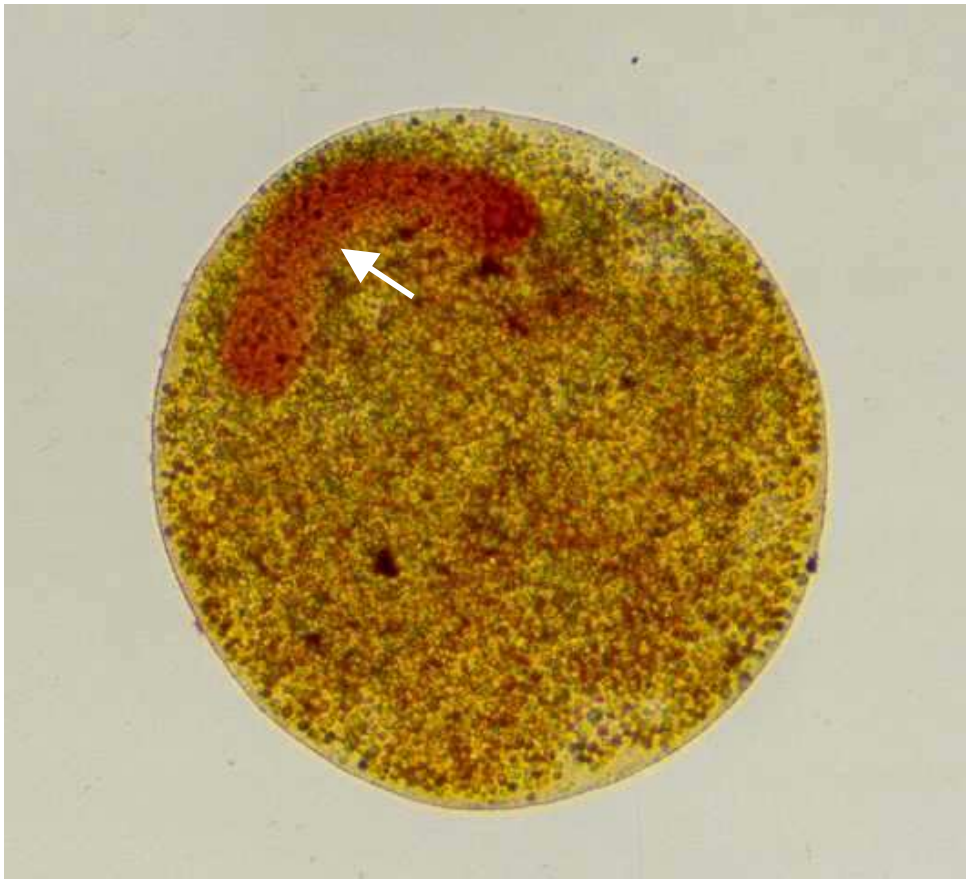
White spot disease caused by *Ichthyophthirius multifiliis*. Photos by B. Diggles.

Above: A juvenile guppy with a white spot infection. Note relatively large white spots which are the trophont stage (arrow), and prominent epithelial hyperplasia (arrowheads).



Below: A trophont of *I. multifiliis* as seen in a wet smear after silver staining. Note the prominent horseshoe-shaped macronucleus (arrow).

Metazoan diseases of freshwater fishes

Disease: Gyrodactylosis (*Gyrodactylus salaris* infection)

Species and life stage affected: Salmonids, mainly Atlantic salmon (*Salmo salar*), but also rainbow trout (*Oncorhynchus mykiss*), brook trout (*Salvelinus fontinalis*), and brown trout (*Salmo trutta*). Other closely related parasites cause disease in a wide variety of non salmonid freshwater fishes.

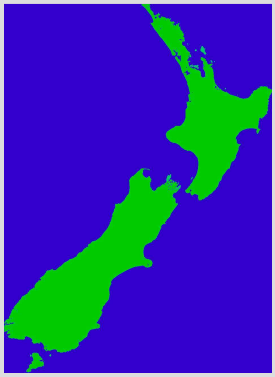
Gross signs: Affected fish produce excessive mucus in areas where parasites attach to the fins and skin and may perform flashing and rubbing behaviour. As disease progresses, secondary infection by bacteria or fungi may occur and under suitable conditions mortalities result as parasite numbers build up rapidly.

Causative agent: Monogenean ectoparasites of the genus *Gyrodactylus*. *Gyrodactylus salaris* causes disease in wild Atlantic salmon and cultured rainbow trout. Other species of *Gyrodactylus* cause disease in various species of freshwater and marine fishes.

Diagnosis: Diagnosis is by examination of the whole surface of affected fish, including gills and mouth, under a dissecting microscope. Members of the genus *Gyrodactylus* are easily identified by the presence of embryos inside the uterus. Identification of *Gyrodactylus salaris* is based on morphology and morphometry of hooks and bars on the opisthaptor, or by DNA analysis (OIE 2000).

Treatment and prevention: *Gyrodactylus salaris* is normally eradicated from aquaculture facilities by destocking and disinfection. Most chemical treatments are not 100% effective, hence methods of control of *G. salaris* in wild salmonid populations in Norway have included radical measures such as rotenone treatment of entire rivers to remove all fish. Novel alternative treatments include addition of aluminium (200 µg/L) to rivers, which removed 100% of *G. salaris* in 4 days (Soleng et al. 1999).

Distribution in New Zealand



Gyrodactylus salaris is unreported, though monogeneans of the genus *Gyrodactylus* have been reported from imported grass carp and wild estuarine fish such as flounder.

GYRODACTYLOSIS IS REGARDED AS AN INTERNATIONALLY SIGNIFICANT DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: Members of the genus *Gyrodactylus* parasitise a variety of fish species worldwide (Cone 1995), though *Gyrodactylus salaris* is confined to northern Europe.

General comments: The only record of monogeneans of the genus *Gyrodactylus* in freshwater fishes in New Zealand is by Edwards & Hine (1974) who reported *G. ctenopharyngodontis* on the external surfaces of imported grass carp (*Ctenopharyngodon idella*) during quarantine. An undescribed species of *Gyrodactylus* has also been reported from yellowbelly flounder (Hine et al. 2000).

References

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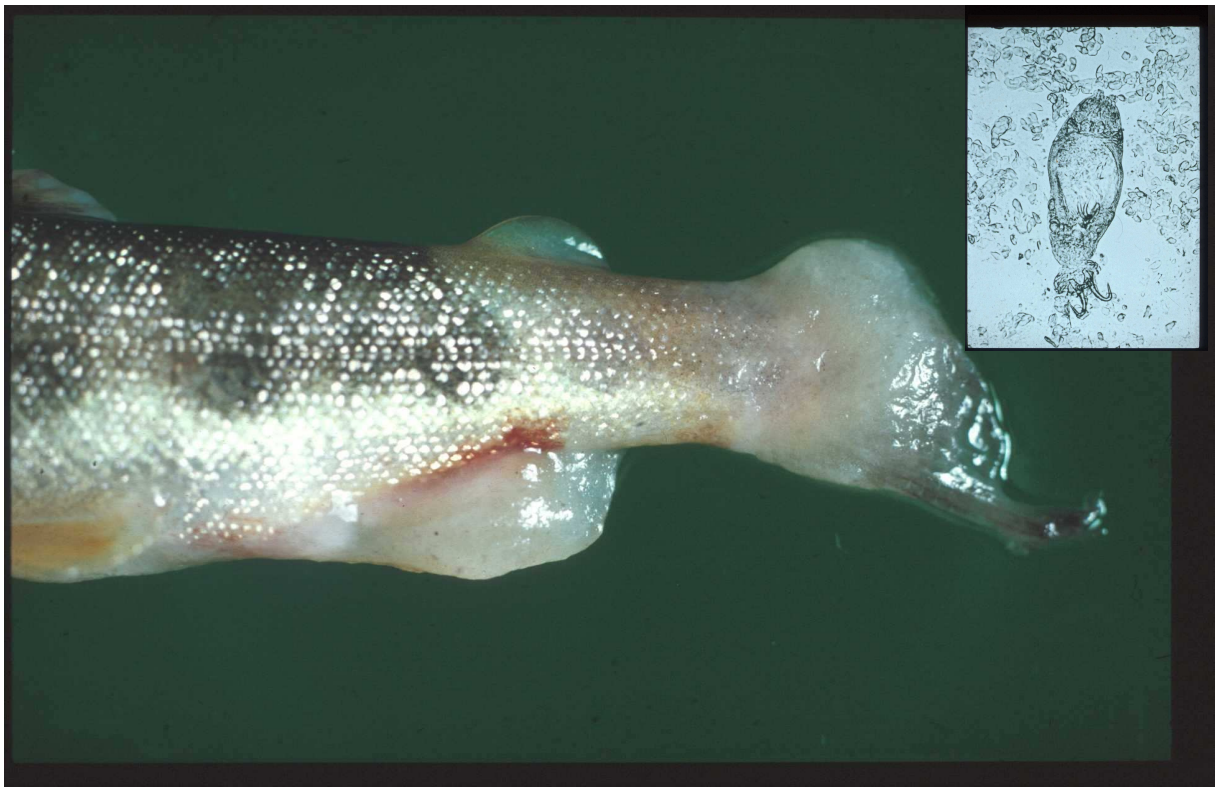
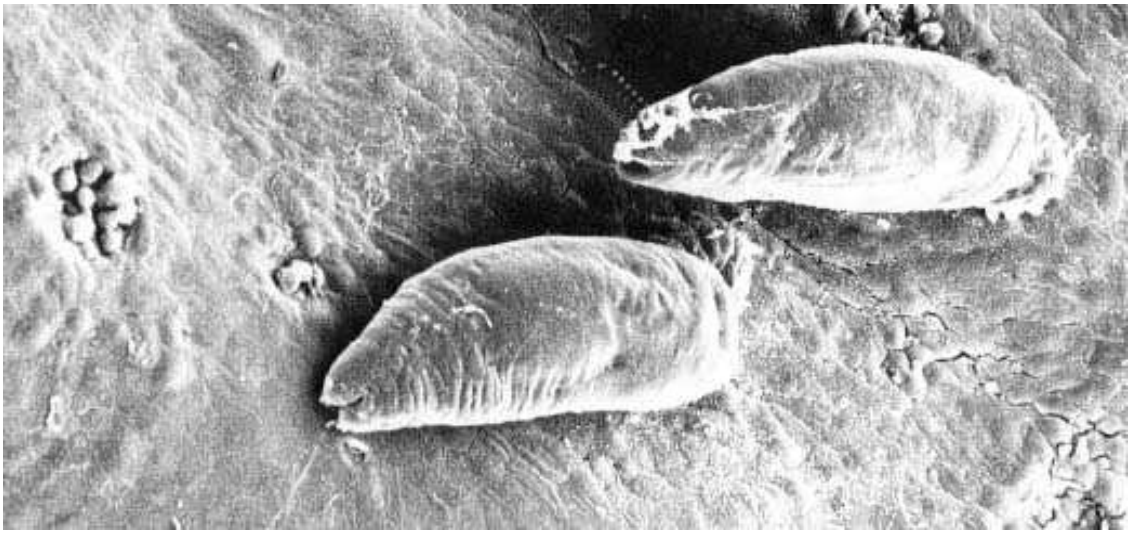


Gyrodactylosis in Atlantic salmon.

Above: Gross signs of disease include necrosis and excessive mucus production on the fins and on other parasite attachment sites, and evidence of secondary bacterial infection. **Inset:** Wet preparation of *Gyrodactylus salaris*. Photos by T. Håstein. **Below:** SEM micrograph showing two *G. salaris* attached to the skin of an Atlantic salmon. Photo by T. Bakke.



Disease: Myxozoan diseases of eels

Species and life stage affected: Juvenile and adult longfinned and shortfinned eels, *Anguilla dieffenbachii* and *A. australis*.

Gross signs: Numerous moderately large white cysts up to 6 mm diameter on the gills, gill arches, and skin. Heavily infected eels may show signs of respiratory distress, swimming slowly and gulping at the water surface.

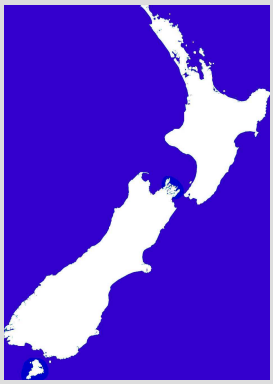
Causative agents: Myxozoan parasites of the genera *Myxidium* (including *M. giardi* (synonym *M. zelandicum*), *M. acinum* and *Myxidium* sp.) and *Myxobolus* sp.

Diagnosis: The white cysts are easily confused with whitespot infections caused by *Ichthyophthirius multifiliis*, unless the cysts are removed and examined under a microscope for the presence of large numbers of spores inside the cysts. Members of the genus *Myxidium* have fusiform spores 8–11.5 µm long with pointed ends containing polar capsules at the opposite ends of the spore (Hine 1978). *Myxobolus* has rounded to oval spores with two polar capsules adjacent to each other at one end of the spore (Lom & Dykova 1992).

Treatment and prevention: None reported. However, treatment with the antibiotic Fumagillin (0.1% in medicated pellets fed 1% body weight per day) is successful in preventing clinical outbreaks of the closely related *Myxobolus cerebralis*, the causative agent of whirling disease (El-Matbouli & Hoffmann 1991), and thus similar treatments may be effective against myxozoan diseases of eels.

Distribution in New Zealand

Myxidium sp. and *Myxobolus* sp. are common on wild eels throughout New Zealand



Worldwide distribution: Members of the genera *Myxidium* and *Myxobolus* commonly parasitise a variety of fish species worldwide (Lom & Dykova 1992).

General comments: The life-cycle of these parasites probably requires (an as yet unknown) intermediate host. If these parasites caused disease in cultured eels, it might be possible to identify the intermediate host(s) and investigate methods of eliminating these in order to interrupt the life cycle of the parasite.

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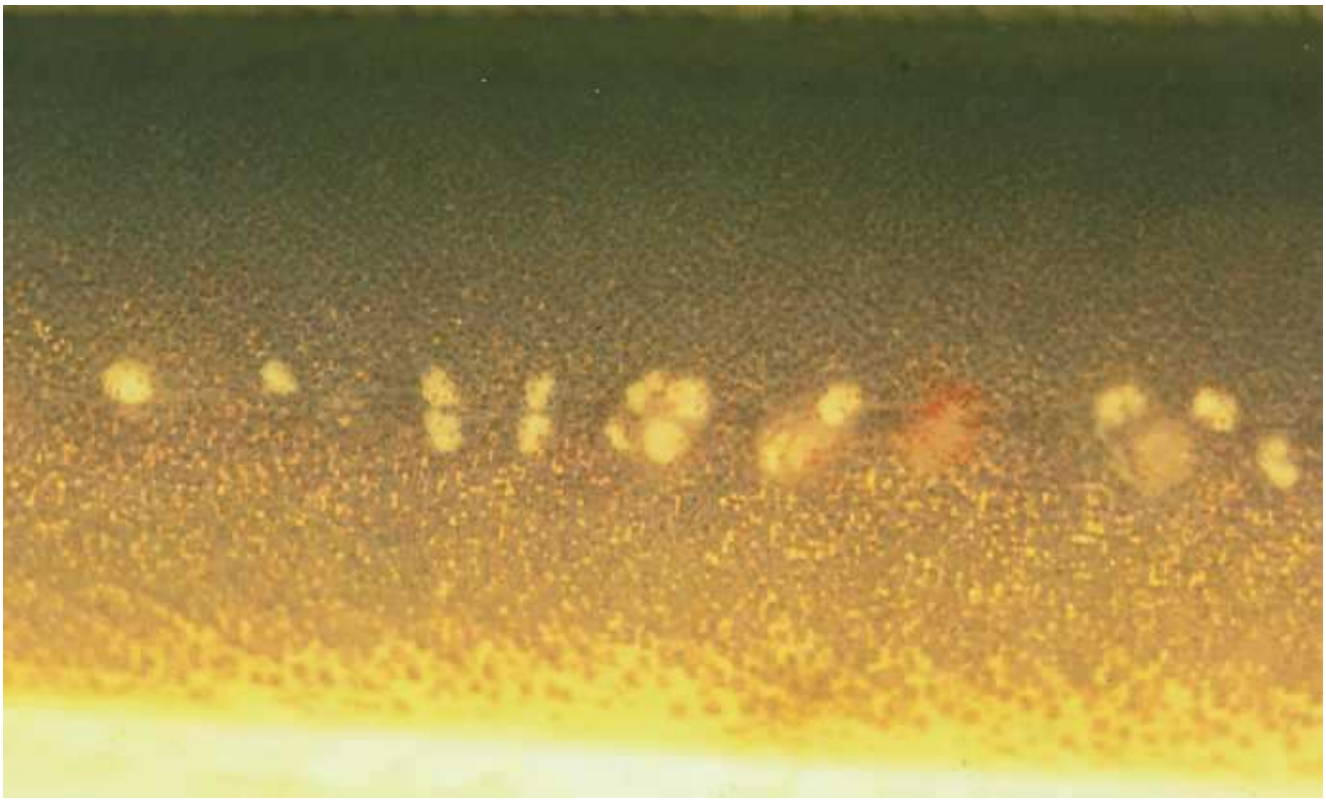
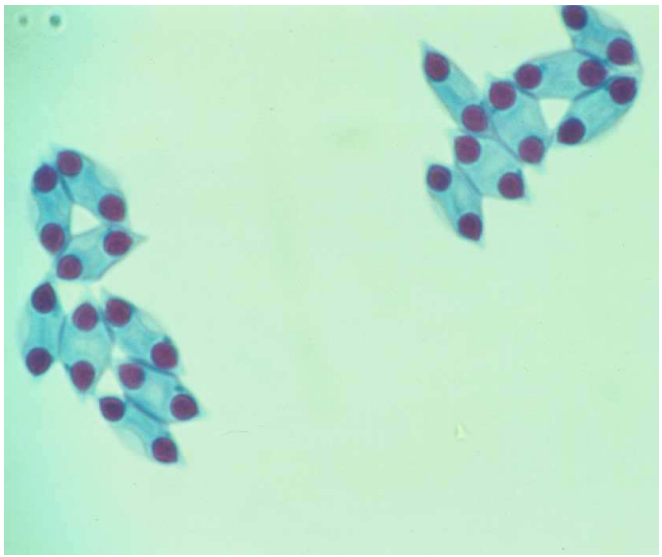
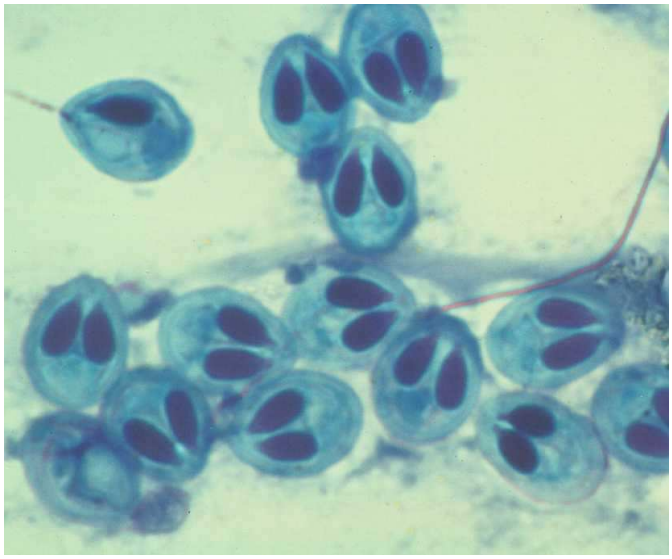


Myxozoan disease of eels. Photos by N. Boustead.

Above: Cysts of *Myxidium* sp. embedded in the lateral line of an eel.
Below left: Stained spores of *Myxidium* sp. from the lateral line of an eel.
Below right: Stained spores of *Myxobolus* sp.





Disease: Whirling disease (WD)

Species and life stage affected: Most salmonids, particularly rainbow trout (*Oncorhynchus mykiss*). Some species may act as asymptomatic carriers.

Gross signs: Acute infections of juvenile fish may cause blackening of the tail and the characteristic whirling ("tail chasing") swimming behaviour. Other signs of disease include cranial or skeletal deformities and erratic darting movements, but most infected fish do not display signs of disease.

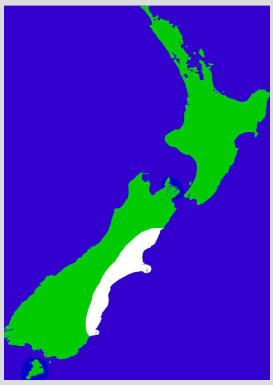
Causative agent: *Myxobolus cerebralis*, a myxozoan parasite with a mud worm (*Tubifex* sp.) intermediate host.

Diagnosis: Presumptive diagnosis of acute infections may follow observation of the characteristic darkened tail and abnormal whirling behaviour in clinically affected fish, but these signs can be caused by other diseases (Margolis et al. 1996). Histology of sections of cartilage from the skull, enzymatic digestion of head cartilage, or plankton centrifuge methods are required to confirm infection with *M. cerebralis*. A DNA based diagnostic test is also available (Hedrick et al. 1999).

Treatment and prevention: Treatment with the antibiotic Fumagillin (0.1% in medicated pellets fed 1% body weight per day) is successful in preventing clinical outbreaks of whirling disease in fish infected under laboratory conditions (El-Matbouli & Hoffmann 1991a). Infective stages survive at least 5 months in mud, freezing at –20 °C for 3 months, and passage through the gut of fish and birds (El-Matbouli & Hoffman 1991b), but are inactivated by exposure to 1300 mW s ml^{-1} UV irradiation (Hedrick et al. 2000).

Distribution in New Zealand

Whirling disease has been recorded in 16 locations on the east coast of the South Island, including seven hatcheries (some of which are no longer operating).



Worldwide distribution: Salmonid whirling disease was discovered in Europe in 1893 and has since been spread with shipments of cultured and wild fish to North America, South Africa, and New Zealand (Hoffman 1990), but has not been detected in Australia.

General comments: In New Zealand, whirling disease has been found in chinook and sockeye salmon as well as brown, rainbow, and brook trout (Boustead 1993). Whirling disease was detected only through disease testing of wild or hatchery fish except for two occurrences of clinical disease in rainbow trout reared in hatcheries for sports fisheries. *Myxobolus cerebralis* has caused clinical disease and substantial mortalities in wild rainbow trout juveniles in the United States (Nehring & Walker 1996).

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Hoffman, G.L. (1990). *Myxobolus cerebralis* , a worldwide cause of salmonid whirling disease. *Journal of Aquatic Animal Health 2*: 30–37.

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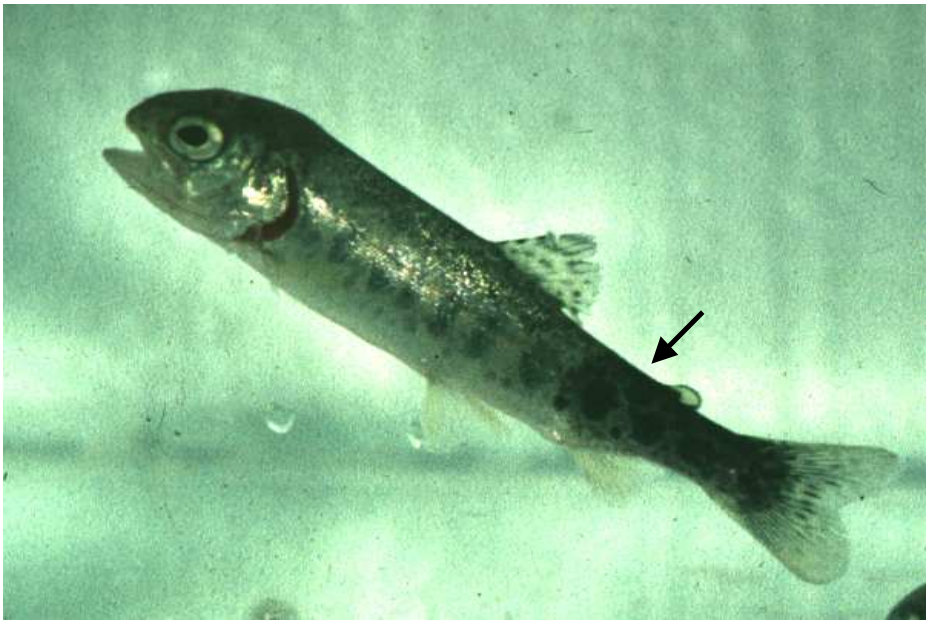
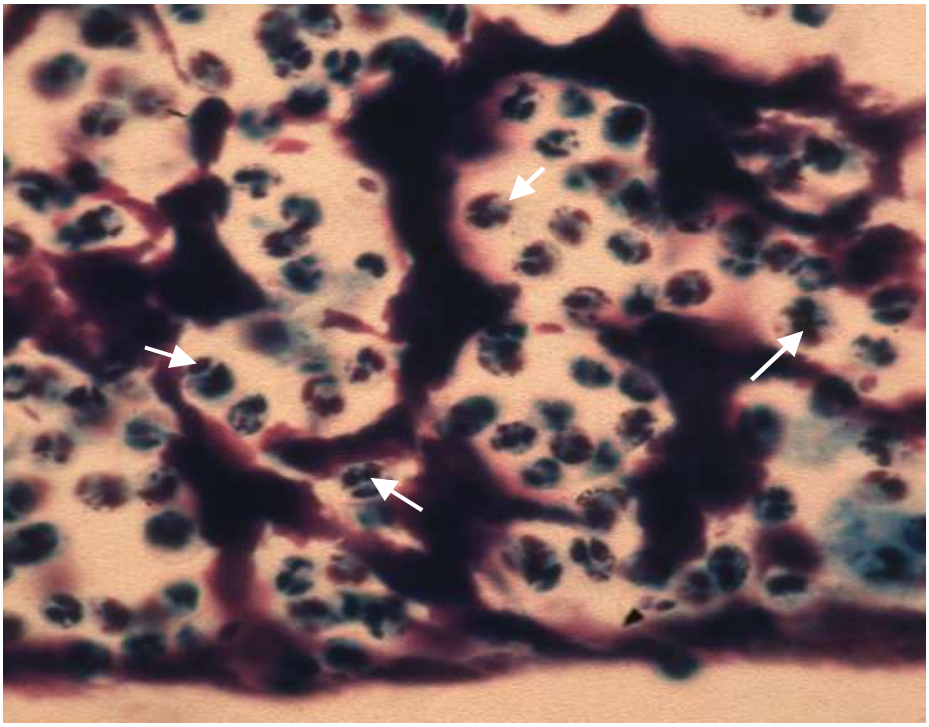
Nehring, B.R.; Walker, P.G. (1996). Whirling disease in the wild: a new reality in the intermountain west. *Fisheries 21*: 28–30.



Whirling disease in rainbow trout.
Photos by M. El-Matbouli.

Above: A classical sign of acute whirling disease in juvenile rainbow trout is darkening of the tail (arrow).



Below: Histological section of cranial cartilage of a trout with acute whirling disease showing numerous *Myxobolus cerebralis* spores (arrows) embedded in digested cartilage.

Husbandry related diseases of freshwater fishes

Disease: Gas bubble disease

Species and life stage affected: Juveniles and adults of all species of freshwater fish, including chinook salmon (*Oncorhynchus tshawytscha*) and longfinned and shortfinned eels, *Anguilla dieffenbachii* and *A. australis*.

Gross signs: Unusual behaviour, gas bubbles evident in fins, eyes, gills, lateral line and other tissues, exophthalmia.

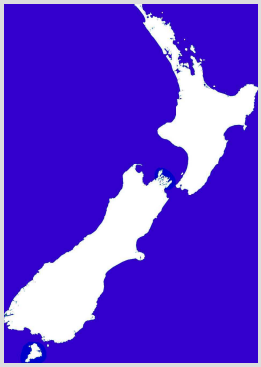
Causative agent: Oxygen supersaturation of the water.

Diagnosis: Diagnosis is through visualisation of gas bubbles throughout various tissues.

Treatment and prevention: Remove source of oxygen supersaturation (e.g., broken or cavitating pumps). Also, aerate cold water which has been heated before use to minimise the chances of supersaturation (cold water carries more oxygen than does warm water). Bubbles may be reabsorbed if fish are pressurised to simulate deeper water (Elston et al. 1997).

Distribution in New Zealand

Gas bubble disease is a husbandry related disease which can occur wherever freshwater fish are cultured throughout the country.



Worldwide distribution: Gas bubble disease has been recorded in fish and shellfish worldwide.

General comments: Fish can usually tolerate quite high levels of supersaturation for short periods of time (minutes, hours), but disease and mortalities can occur if chronic exposure to even very low levels of supersaturation is allowed. There may be an extreme amount of variation in susceptibility to gas bubble disease, both within a given population of fish (Mesa et al. 2000) and between fish species. Fish in the wild may also be exposed to human-induced supersaturated water discharged from dams and reservoirs, and photosynthetic activity of aquatic plants can be a natural source of supersaturation (Schisler 2000). See also p. 108 for marine fishes.

References

Elston, R.; Colt, J.; Abernethy, S.; Maslen, W. (1997). Gas bubble reabsorption in chinook salmon: pressurization effects. *Journal of Aquatic Animal Health 9*: 317–321.

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Please refer to the marine fish section (p. 109) for photos of some gross signs of gas bubble disease.

Disease: Nephrocalcinosis

Species and life stage affected: Juveniles and adults of a variety of freshwater and marine fish, including salmonids, particularly rainbow trout (*Oncorhynchus mykiss*), and white bream (*Diplodus sargus*).

Gross signs: Upon dissection white calcareous deposits are evident in the kidney, particularly in the posterior part of the kidney and the ureters, which may appear as thick, white convoluted threads.

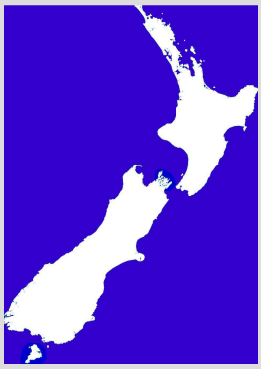
Causative agents: Usually associated with high carbon dioxide (CO_2) content in hard water supplies, but is also associated with feeding diets with high levels of calcium, cadmium, and other metals (Hicks et al. 1984, Romestand et al. 1986).

Diagnosis: Presumptive diagnosis requires observation of white calcareous deposits in the kidney. This can be supported by histological assessment of kidney damage (calcification, granuloma formation) and detection of the presence of high levels of carbon dioxide in the rearing water or high levels of calcium, cadmium, and other metals in the diet.

Treatment and prevention: Can be treated and prevented by feeding diets low in metals and calcium and rearing fish in water with safe levels of free CO_2, i.e., less than 6 mg/L and ideally 1–2 mg/L (Boustead 1989). Levels of CO_2 can be reduced by vigorous aeration.

Distribution in New Zealand

Nephrocalcinosis is a husbandry related disease which can occur wherever freshwater or marine fish are cultured throughout the country.



Worldwide distribution: Has been recorded worldwide in sea-farmed and freshwater farmed rainbow trout (Schlotfeldt & Ahne 1979), and also in white bream in Spain (Gomez 2000).

General comments: Nephrocalcinosis in sockeye and chinook salmon in New Zealand has been associated with minimal mortalities (Boustead 1989). The reported incidence of nephrocalcinosis in rainbow trout at different CO_2 concentrations was as follows: free CO_2 concentrations of 12 mg/L, 24 mg/L, and 55 mg/L, prevalence of nephrocalcinosis was 4.8%, 9.7%, and 47.2%, respectively (Boustead 1989). Ninety percent of rainbow trout fed a diet containing high levels of selenium developed nephrocalcinosis (Hicks et al. 1984).

References

Boustead, N.C. (1989). A guide to diseases of salmon in New Zealand. New Zealand Freshwater Fisheries Report No. 112. Freshwater Fisheries Centre, MAF Fisheries, Christchurch.

Gomez, S. (2000). Nephrocalcinosis in Mediterranean cultured white bream (*Diplodus sargus* L.). *Bulletin of the European Association of Fish Pathologists 20*: 40–42.

Hicks, B.D.; Hilton, J.W.; Ferguson, H.W. (1984). Influence of dietary selenium on the occurrence of nephrocalcinosis in rainbow trout, *Salmo gairdneri* Richardson. *Journal of Fish Diseases 7*: 379–389.

Romestand, B.; Halsband, E.; Halsband, I.; Pump, H.; Dzuvic, A.; Schmidt, G. (1986). Investigations on nephrocalcinosis pathogeny of the trout (*Salmo gairdneri*). Action of cadmium, calcium, and vitamin D3 enriched food on blood constants and calcification of certain organs. *Ichtyophysiologia Acta 10*: 5–25.

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Nephrocalcinosis in salmonids. Photos by N. Boustead.

Above: Nephrocalcinosis in the kidney of a sockeye salmon (*Oncorhynchus nerka*). Note the white calcareous deposits in the ureters (arrow). **Below:** Nephrocalcinosis in chinook salmon (*Oncorhynchus tshawytscha*). Note the massively enlarged kidney with white calcareous deposits (arrow).

Disease: Pinhead syndrome

Species and life stage affected: Fry and juveniles of most species of cultured freshwater and marine fish.

Gross signs: Extreme emaciation of fry or juvenile fish so that they appear to have a disproportionately large head.

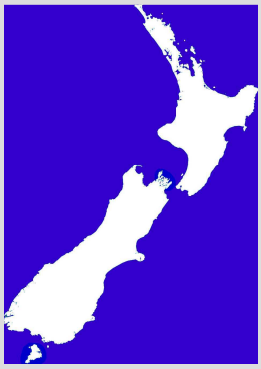
Causative agent: Starvation, failure to feed.

Diagnosis: Observations of the gross signs of emaciation in the absence of other known disease agents is required to diagnose this syndrome. Histopathology may demonstrate a lack of food in the gut together with a reduction in height of the epithelial cells of the gut mucosa and reduced fat storage in the liver (Margulies 1993). Note that infection by a wide variety of disease agents can cause cessation of feeding in fry and juvenile fish.

Treatment and prevention: This syndrome is thought to be due to feeding problems related to poor palatability of diet or unsuitable conditions for first feeding, such as the incorrect tank colour, lighting regime, etc. To reduce the incidence of pinhead syndrome a change in diet and/or modification of husbandry conditions is recommended.

Distribution in New Zealand

Pinhead syndrome is a husbandry related syndrome which can occur wherever freshwater or marine fish are cultured throughout the country.



Worldwide distribution: This syndrome occurs worldwide wherever freshwater or marine fish are cultured.

General comments: It is important that the diagnosis of pinhead syndrome is made only after undertaking a diagnostic process which includes elimination of other disease agents which might cause similar gross signs or cessation of feeding.

References

Boustead, N.C. (1989). A guide to diseases of salmon in New Zealand. *New Zealand Freshwater Fisheries Report No. 112.* Freshwater Fisheries Centre, MAF Fisheries, Christchurch.

Margulies, D. (1993). Assessment of the nutritional condition of larval and early juvenile tuna and spanish mackerel (Pisces: Scombridae) in the Panama Bight. *Marine Biology 115*: 317–330.

Pinhead syndrome in chinook salmon (*Oncorhynchus tshawytscha*) fry. Photo by N. Boustead.

One normally developed fry (arrow) is surrounded by numerous emaciated fry which are smaller and have disproportionately large heads.

Disease: Sunburn

Species and life stage affected: Juveniles and adults of many species of marine and freshwater fish, but particularly salmonids.

Gross signs: Sunburn usually begins as a slight darkening of the dorsal surfaces of fish, followed by erosion of the skin which eventually exposes the white epidermis and underlying tissue. Secondary infection by opportunistic bacteria and fungi may follow.

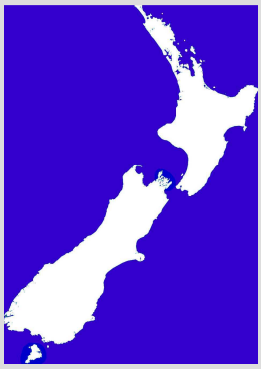
Causative agent: Excessive ultraviolet radiation, mostly due to UV-B.

Diagnosis: Presumptive diagnosis can be based on the characteristic lesions on the dorsal surface of the fish. A definitive diagnosis requires histopathology to rule out involvement of other possible disease agents and demonstrate the presence of characteristic sunburn cells with fragmented nuclear material and a reduction in mucous cells in affected areas of epidermis.

Treatment and prevention: Provision of shade or deeper water (over 1 metre) to allow fish to escape from excess solar radiation. Upon providing shade the restoration of normal epidermis occurs rapidly by processes of cell proliferation and tissue closure resembling those of wound healing.

Distribution in New Zealand

Sunburn is a husbandry related disease which can occur wherever susceptible freshwater or marine fish are cultured throughout the country.



Worldwide distribution: Sunburn can occur worldwide wherever susceptible fish are held in shallow, clear water.

General comments: Mortalities caused by sunburn may be insignificant, but the lesions reduce the marketability of affected fish (Boustead 1989). Some species, such as channel catfish (*Ictalurus punctatus*) and salmonids, are quite sensitive to UV-B radiation (Blazer et al. 1997, Ewing et al. 1999). These species appear to lack a methanol-extractable skin substance that is associated with resistance to sunburn in other fish species (Ewing et al. 1999). Ultraviolet B radiation can penetrate water up to 1 metre deep (Roberts 1989). Under natural conditions fish avoid exposure to potentially damaging UV radiation by moving to deeper water or shade during the middle of the day.

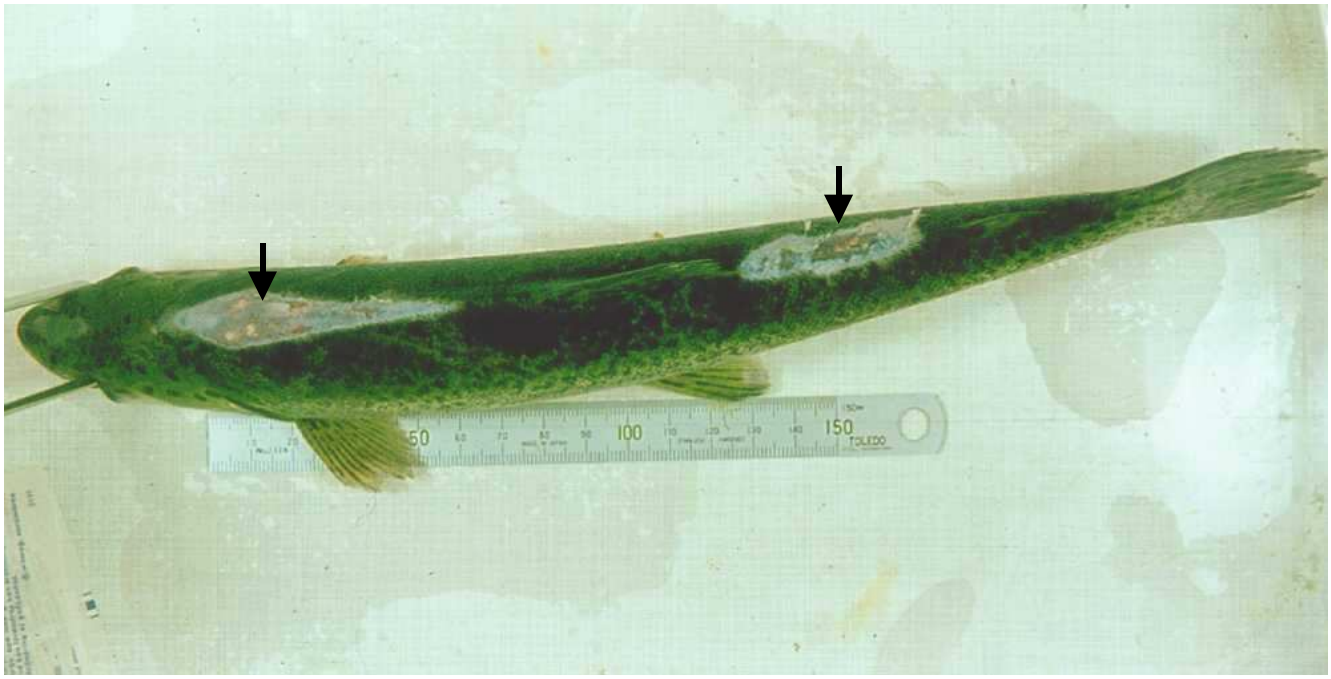
References

Blazer, V.S.; Fabacher, D.L.; Little, E.E.; Ewing, M.S.; Kocan, K.M. (1997). Effects of ultraviolet-B radiation on fish: Histologic comparison of a UVB-sensitive and a UVB-tolerant species. *Journal of Aquatic Animal Health 9*: 132–143.

Boustead, N.C. (1989). A guide to diseases of salmon in New Zealand. *New Zealand Freshwater Fisheries Report No. 112*. Freshwater Fisheries Centre, MAF Fisheries, Christchurch.

Ewing, M.S.; Blazer, V.S.; Fabacher, D.L.; Little, E.E.; Kocan, K.M. (1999). Channel Catfish Response to Ultraviolet-B Radiation. *Journal of Aquatic Animal Health 11:* 192–197.

Roberts, R.J. (1989). Fish pathology , 2nd edition. Bailliere Tindall, London. 467 p.



Sunburn in cultured chinook salmon (*Oncorhynchus tshawytscha*). Photo by N. Boustead.

Note the erosion of the skin in two areas along the dorsal surfaces of this fish (arrows) exposing the lighter coloured epidermis and underlying tissue.

Diseases of unknown aetiology of freshwater fishes

Disease: GDAS (gastric dilation air saculitus) of salmon (bloat, water belly)

Species and life stage affected: Salmonids such as rainbow trout (*Oncorhynchus mykiss*), Atlantic salmon (*Salmo salar*), and chinook salmon (*Oncorhynchus tshawytscha*), predominantly growers in seawater, up to harvest size.

Gross signs: Distended and enlarged abdomen. Internally, the swim bladder and/or stomach are stretched, enlarged and filled with fluid. Sometimes no external abnormality is visible, but internally the stomach and/or swim bladder are affected.

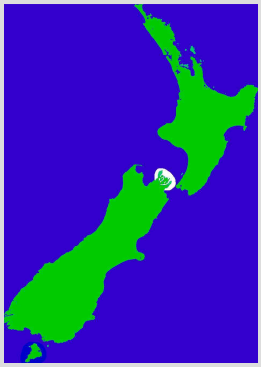
Causative agents: GDAS in chinook salmon appears not to be caused by infectious organisms but is suspected to be caused by dietary or feeding factors. Similar conditions in other species can be multifactorial, however, (Rorvik et al. 2000) and the exact cause is unknown.

Diagnosis: Diagnosis of the condition is based on the distinctive massive accumulation of fluid in the stomach and swim bladder and the absence of known pathogens.

Treatment and prevention: A change to the source of food used has been associated with a significant reduction in the incidence of this condition.

Distribution in New Zealand

The condition has been found predominantly in sea cage culture of chinook salmon in the Marlborough Sounds, but has also been reported at a low incidence in freshwater and some other marine farming locations elsewhere in the South Island.



Worldwide distribution: Similar conditions have been reported from pen-reared salmonids on the Pacific Northwest of America (Kent 1992) and from marine farmed rainbow trout in Europe (Staurnes et al. 1990, Rorvik et al. 2000).

General comments: Gastric dilation syndromes have been recorded in several species of farmed salmonids. Although apparently multifactorial events (Rorvick et al. 2000), many cases have been temporally linked to changes in feeding programmes (Staurnes et al. 1990, Speare 1998).

References

Kent, M.L. (1992). Diseases of seawater netpen-reared salmonid fishes in the Pacific Northwest. *Canadian Special Publication of Fisheries and Aquatic Sciences 116*. 64 p.

Rorvik, K.A.; Skervold, P.O.; Fjaera, S.O.; Steien, S.H. (2000). Distended, water filled stomach in seawater farmed rainbow trout, *Oncorhynchus mykiss* (Walbaum), provoked experimentally by osmoregulatory stress. *Journal of Fish Diseases 23:* 15–18.

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Staurnes, M.; Andorsdottir, G.; Sundby, A. (1990). Distended water-filled stomach in sea-farmed rainbow trout. *Aquaculture 90*: 333–343.

GDAS in chinook salmon. Photo by N. Boustead.

Note the abdominal distention caused by a massive accumulation of fluid in the stomach and swim bladder.

Diseases of Importance to Aquaculture in New Zealand

Diseases of Marine Fishes

MARINE FISHES

Viral diseases of marine fishes

Disease: Red sea bream iridoviral disease (RSIVD)

Species and life stage affected: Has been reported in 20 species of cultured marine fish in Japan, including red sea bream (*Pagrus auratus*) (snapper), yellowtail (*Seriola quinqueradiata*), seabass (*Lateolabrax* sp.), and parrotfish (*Oplegnathus fasciatus*). Mainly juvenile fish are affected.

Gross signs: Affected fish are lethargic, exhibit severe anaemia, petechiae of the gills, and enlargement of the spleen.

Causative agent: Red sea bream iridovirus (RSIV).

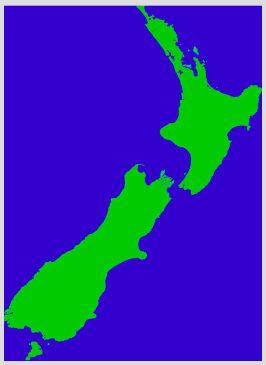
Diagnosis: Isolation of RSIV in cell culture (BF-2 cells are preferred (Nakajima & Sorimachi 1994)) from kidney or spleen tissues followed by identification with monoclonal antibodies, IFAT, or PCR based diagnostic tests (Oshima et al. 1998).

Treatment and prevention: No known treatment. Control methods currently rely on implementation of hygiene practices, though a vaccine may soon be commercially available (Nakajima et al. 1999).

Distribution in New Zealand

Unreported, though significant as the major host species in Japan (*P. auratus*) also occurs in New Zealand waters.

RSIVD IS REGARDED AS AN INTERNATIONALLY SIGNIFICANT DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Known only from Japan.

General comments:. An acute and highly contagious disease of snapper, particularly troublesome during the summer months. The principal means of transmission of the virus is horizontal via the water. The disease affects mainly juvenile snapper, but mortality of market sized fish has also been reported.

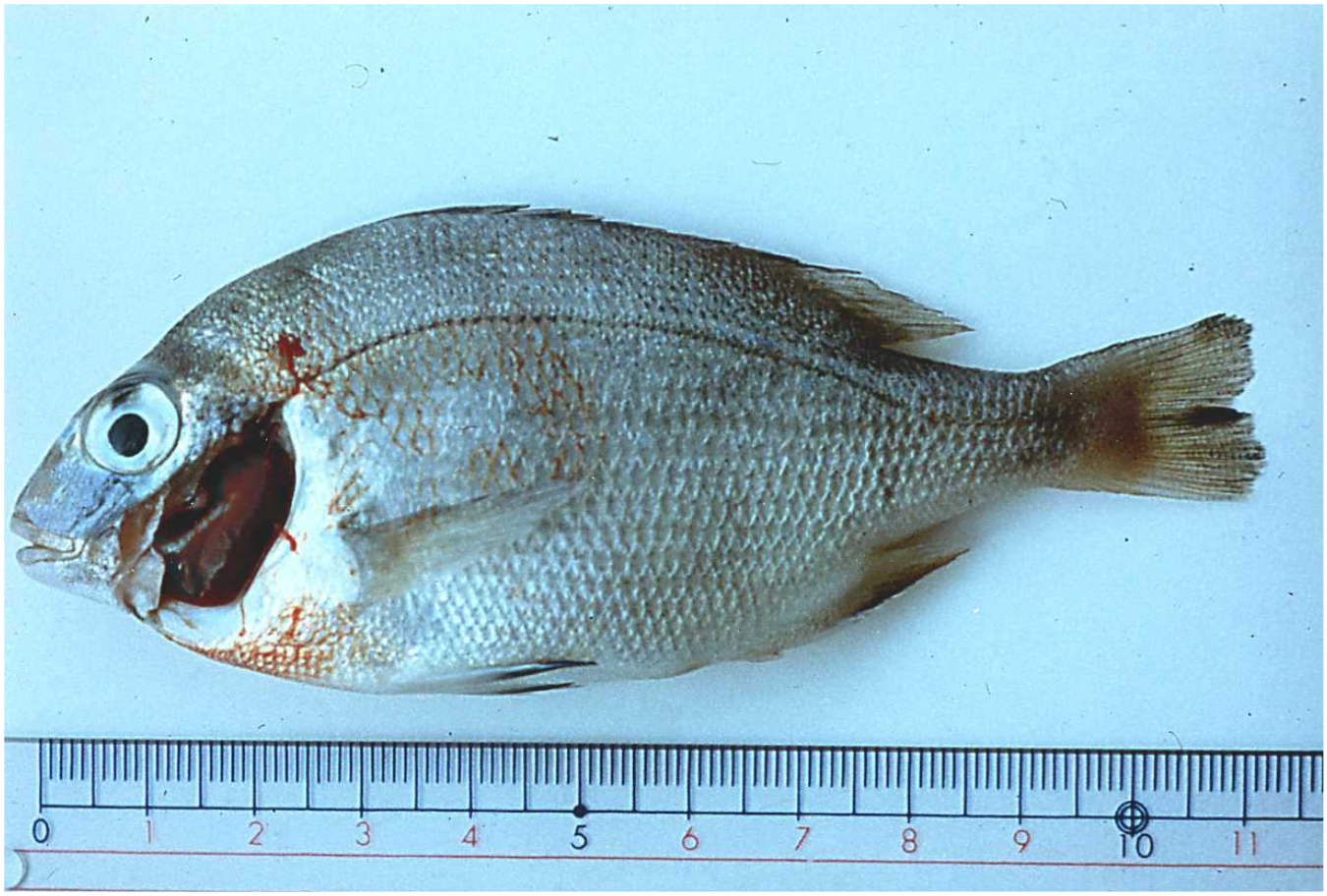
References

Matsuoka, S.; Inouye, K.; Nakajima, K. (1996). Cultured fish species affected by red sea bream iridoviral disease from 1991 to 1995. *Fish Pathology 31*: 233–234.

Nakajima, K.; Sorimachi, M. (1994). Biological and physio-chemical properties of the iridovirus isolated from cultured red sea bream, *Pagrus major*. *Fish Pathology 29*: 29–33.

Nakajima, K.; Maeno, Y.; Honda, A.; Yokoyama, K.; Tooriyama, T.; Manabe, S. (1999). Effectiveness of a vaccine against red sea bream iridoviral disease in a field trial test. *Diseases of Aquatic Organisms 36*: 73–75.

Oshima, S.; Hata, J.I.; Hirasawa, N.; Ohtaka, T.; Hirono, I.; Aoki, T.; Yamashita, S. (1998). Rapid diagnosis of red sea bream iridovirus infection using the polymerase chain reaction. *Diseases of Aquatic Organisms 32*: 87–90.



A Japanese red sea bream (*Pagrus auratus*) with RSIVD. Photo by K. Nakajima.

This fish exhibited severe anaemia, petechiae of the gills, and enlargement of the spleen, typical signs of infection with red sea bream iridovirus disease. The operculum was removed to show bleeding from the gills.

Disease: Viral encephalopathy and retinopathy (VER), (viral nervous necrosis (VNN))

Species and life stage affected: Known to infect 22 species of marine fish from 11 families, including trevally (*Pseudocaranx dentex*) and flatfish such as halibut (*Hippoglossus hippoglossus*) and turbot (*Scophthalmus maximus*). Some species, such as sparids (e.g., *Sparus aurata* in Europe) may be asymptomatic carriers of the virus (Castric et al. 2001).

Gross signs: Neurological abnormalities, erratic swimming behaviour and abnormal coloration, particularly evident in very young fish (less than 40 days old). Microscopically evident lesions include vacuolation and necrosis of the central nervous system tissue and in the retina. Endocarditis can occur in halibut (*Hippoglossus hippoglossus*) (see Grotmol et al. 1997).

Causative agents: Betanodoviruses, family Nodaviridae. Four genotypes are known to exist (Nishizawa et al. 1997).

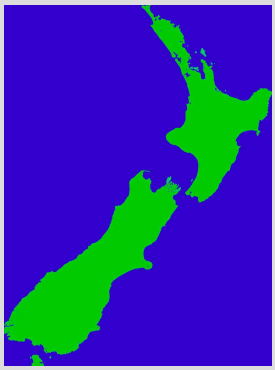
Diagnosis: Presumptive diagnosis in fish exhibiting erratic swimming behaviour and other gross signs of VER may be obtained by demonstrating vacuolation of nervous tissues and the retina using histopathology. Electron microscopy and examination by negative and positive staining. Isolation into cell-lines, IFAT, ELISA, PCR (Mushiake et al. 1994), and immunohistochemistry.

Treatment and prevention: No known treatment. Prevention is by screening of broodstock for the virus and removing carriers from the spawning group. Surface disinfection of eggs may increase survival (Grotmol & Totland 2000), though this does not guarantee prevention as the virus is vertically transmitted in some species (OIE 2000), including *P. dentex*. Avoidance by controls on movement, slaughter and disinfection of affected stocks, not feeding trash fish to farm fish, and culturing susceptible species in separate areas from possible carrier species (Castric et al. 2001).

Distribution in New Zealand

Unreported.

VNN IS REGARDED AS AN INTERNATIONALLY SIGNIFICANT DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Regarded as worldwide (Munday & Nakai 1997). More specifically it has been reported from Europe, Japan, Australia, Asia, and most recently in the U.S.

General comments: This disease is included here because it infects a New Zealand marine species, trevally (*P. dentex*) (see Nguyen et al. 1996), and this species may be processed for feeding as trash fish to cultured species. Such feeding may transmit the disease. The course of the disease varies greatly between host species, but in all cases it is likely that the virus is horizontally transmitted. However, it may also be vertically transmitted in *P. dentex.* Hatchery reared juvenile turbot (*Colistium nudipinnis*) with minor vacuolation of the spinal chord sampled during mortality events in New Zealand were negative for VER using the IFAT technique (Diggles et al. 2000).

References

Diggles, B.K.; Carson, J.; Hine, P.M.; Hickman, R.W.; Tait, M.J. (2000). *Vibrio* species associated with mortalities in hatchery-reared turbot (*Colistium nudipinnis*) and brill (*C. guntheri*) in New Zealand. *Aquaculture 183*: 1–12.

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Grotmol, S.; Totland, G.K. (2000). Surface disinfection of Atlantic halibut *Hippoglossus hippoglossus* eggs with seawater inactivates nodavirus and increases survival of larvae. *Diseases of Aquatic Organisms 39*: 89–96.

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Munday, B.; Nakai, T. (1997). Special topic review: Nodaviruses as pathogens in larval and juvenile marine finfish. *World Journal for Microbiology and Biotechnology 13*: 375–381.

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Nishizawa, T.; Furuhashi, M.; Nagai, T.; Nakai, T.; Muroga, K. (1997). Genomic classification of fish nodaviruses by molecular phylogenetic analysis of the coat protein gene. *Applied Environmental Microbiology 63*: 1633–1636.

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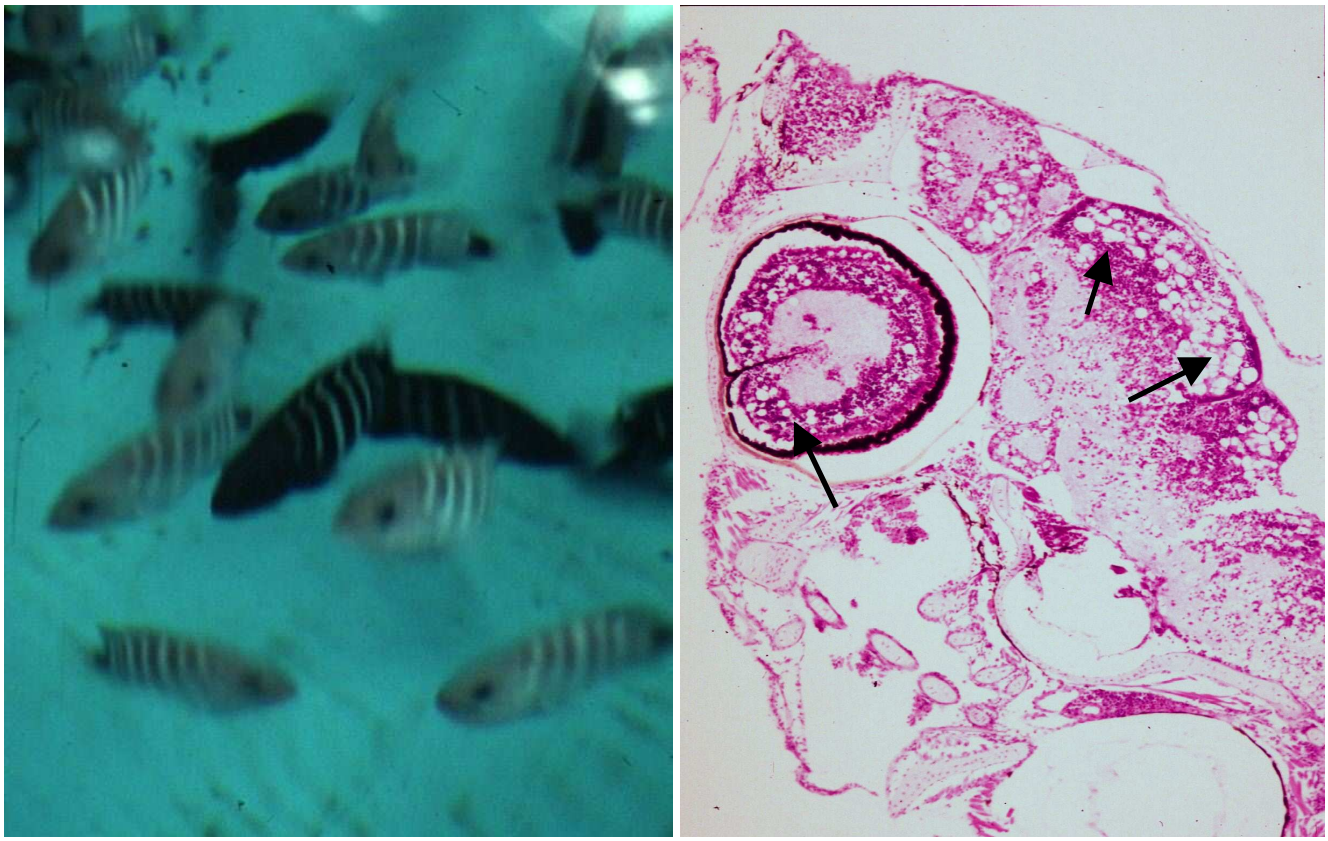


VER in barramundi and groupers. Photos by B. Munday.

Left: VER in convict groupers. Dark coloured fish have clinical VER (retinal lesions).
Right: Histology of a VER infected barramundi showing massive vacuolation in brain and retina (arrows).

Microbial diseases of marine fishes

Disease: Enterococcal infection

Species and life stage affected: Primarily a disease of yellowtail (*Seriola quinqueradiata*) in Japan, but can affect numerous other species, including red sea bream (*Pagrus auratus*) and eels (*Anguilla japonica*). Affects both juveniles and adult fish.

Gross signs: Bilateral exophthalmia, petechiae on the inside walls of the opercula, and congestion and haemorrhage of the intestine, liver, spleen, and kidney. An infectious systemic disease characterised by haemorrhagic septicemia.

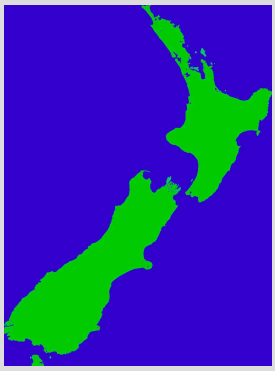
Causative agent: *Lactococcus garvieae* (previously known as *Enterococcus seriolicida*, see Eldar et al. (1996)), a bacterium.

Diagnosis: Presumptive diagnosis can be obtained by detection of typical gross signs of the disease (e.g. bilateral exophthalmia, petechiae on the inside walls of the opercula). Definitive diagnosis requires isolation of 0.7 x 1.4 µm gram-positive ovoid cells forming short chains from the kidney, spleen, and intestine of infected fish. Culture on BHI agar with or without 1.5–2 % NaCl. Rapid and reliable direct or indirect fluorescent antibody tests are available (Kusuda & Kawahara 1987).

Treatment and prevention: Maintenance of good water quality and adoption of husbandry techniques which minimise handling or other damage are strongly recommended. Antibiotics such as erythromycin and spiramycin were widely used in Japan, but resistant strains of bacteria evolved. Vaccines have now been developed and show promise for control of the disease (Kawai & Hatamoto 1999), and bacteriophages may also be useful for controlling *L. garvieae* infections (Nakai et al. 1999).

Distribution in New Zealand

Unreported. This disease is included as it occurs in Japan in species which are being farmed in New Zealand.



ENTEROCOCCAL INFECTIONS MAY BE EXOTIC. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: Besides Japan, *Lactococcus* and *Enterococcus*-like bacteria have been reported in marine and freshwater fish in Europe and Australia (Eldar & Ghittino 1999).

General comments: Enterococcal infection is a chronic septicaemic disease. This disease is the major bacterial disease of yellowtail in Japan, being responsible for 69% of total losses due to bacterial infection in 1989. It was also responsible for about 5% of total losses of cultured *Pagrus auratus* in Japan in 1989 (Kusuda & Salati 1993).

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No photographs currently available

Disease: Epitheliocystis

Species and life stage affected: Juveniles and adults of a wide variety of marine fish, including snapper (*Pagrus auratus*) and pilchards (*Sardinops sagax neopilchardus*).

Gross signs: No obvious external signs, usually benign, but can be associated with epizootics in confined, hyperinfected fish, especially those which mount a proliferative response. Small whitish nodules on gills are visible in heavy infections. Juvenile fish appear more susceptible, but also found in adults.

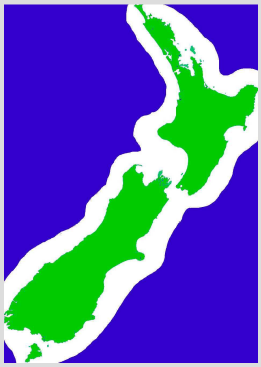
Causative agents: Infection of the gill epithelium by rickettsia and/or chlamydia-like organisms.

Diagnosis: Presumptive diagnosis can be obtained by taking a wet squash of a gill biopsy. Epitheliocystis affected cells appear whitish and are enlarged and roughly spherical. Histology will demonstrate the presence of characteristic basophilic inclusion bodies in the lamellar epidermis of gills. The inclusions contain numerous chlamydia and or rickettsia-like cells 0.5–1 µm long. Definitive diagnosis requires TEM and/or molecular techniques.

Treatment and prevention: Occurs in wild fish, including pilchards (*Sardinops sagax neopilchardus*) around New Zealand. Direct fish to fish transmission occurs (Hoffman et al. 1969), hence exclusion of possible carrier species from culture facilities may be warranted. Exclusion of the bacteria from intake water using UV irradiation is also highly recommended. Isolation of infected batches of fish is also recommended to prevent spread of infection. Antibiotic treatments are usually ineffective.

Distribution in New Zealand

Has been recorded from wild pilchards and cultured snapper in the North Island. Probably occurs throughout New Zealand waters.



Worldwide distribution: Affects numerous species of wild and cultured fish throughout the world (Bradley et al. 1988), including *Sparus aurata* in Europe (Paperna 1977) and *P. auratus* in Japan and Hong Kong (Miyazaki et al. 1986).

General comments: Epitheliocystis hyperinfection has been associated with mortality of a variety of cultured fish, including lake trout in the U.S. (Bradley et al. 1988), and *Sparus aurata* (see Paperna 1977), flounder (*Paralichthys* sp.) and Pacific yellowtail (*Seriola* sp.) in Europe (Benetti 1997). It is a potentially serious problem in the culture of snapper in New Zealand.

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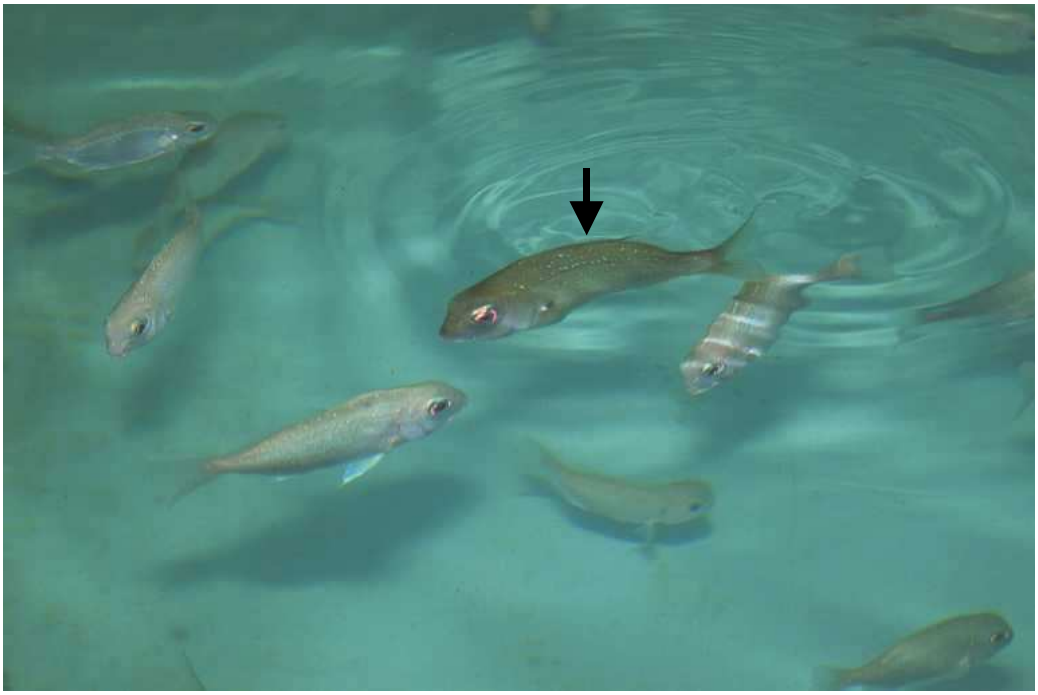
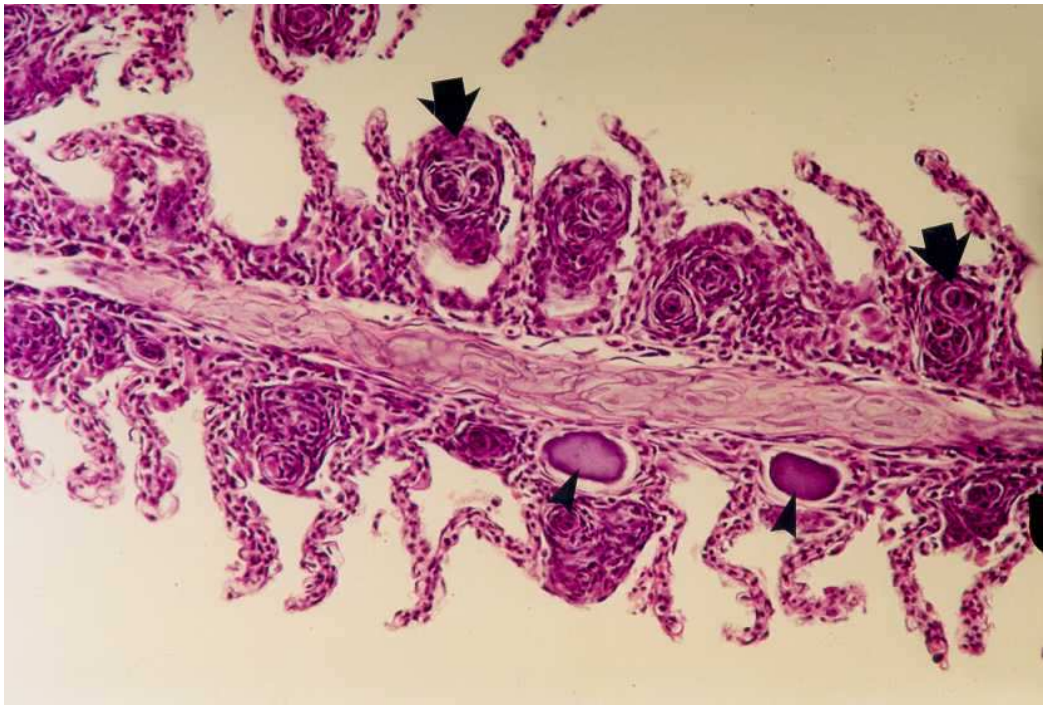


Epitheliocystis in cultured snapper (*Pagrus auratus*). Photos by B. Diggles.

Above: A juvenile snapper swimming near the surface of the tank (arrow) heavily infected with epitheliocystis disease. Note the darkened colour. **Below:** Histology of affected gills showing hypertrophied cells in the lamellar epithelium (arrowheads) with basophilic inclusions. A granulomatous proliferative response is also evident between the secondary gill lamellae (arrows).



Disease: Epizootic ulcerative syndrome (EUS) (red spot disease, mycotic granulomatosis)

Species and life stage affected: As many as 100 species of estuarine and marine fish may be susceptible, including mullet (*Mugil* spp.) and rainbow trout (*Oncorhynchus mykiss*).

Gross signs: Loss of appetite, darkening, erratic movements, red spots on the body surface, head, operculum, or caudal peduncle, with large red or grey shallow ulcers, often with brown necrosis, at later stages. Most species will die at this stage, but more susceptible species, such as snakeheads and mullets, may show complete erosion of the posterior body, or erosion of the cranium, exposing the brain.

Causative agents: A fungus, *Aphanomyces invadans.* However, the disease is a syndrome and other pathogens may be involved, such as the bacterium *Aeromonas* (see Iqbal et al. 1999), or viruses (Frerichs et al. 1993, Kanchanakhan et al. 1999), once the primary pathogen *A. invadans* infects the fish. Historically, *A. invadans* has been called *Aphanomyces piscicida*, *Aphanomyces invaderis*, and ERA (EUS-related *Aphanomyces*).

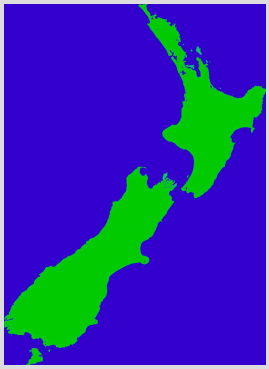
Diagnosis: Histopathology (large branching hyphaeabout 10 µm diameter deeply penetrating into tissues with an intense granulocytic response), isolation of the fungus and sporulation of the fungus for positive identification. Gross signs are similar to those of other ulcerative diseases, and hence are not reliable for diagnosis.

Treatment and prevention: Hydrogen peroxide may be used to treat the disease, and Proxitane 0510 to disinfect contaminated facities (Lilley & Inglis 1997). Controls on movements, slaughter, and disinfection and surveillance are also recommended.

Distribution in New Zealand

Unreported, but red cod (*Physiculus bachus*) do sometimes show signs of a similar disease.

EUS IS REGARDED AS AN INTERNATIONALLY SIGNIFICANT DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: EUS was first reported from Japan in 1971, and a single clonal genotype (Lilley et al. 1997) has since spread throughout Southeast Asia, northern Australia, India, and Pakistan. EUS-like lesions associated with fungal infections also occur along the east coast of the U.S. (Noga & Dykstra 1986). One probable mode of spread of this disease is via imports of aquarium fishes.

General comments: EUS is generally regarded as a warm water pathogen, but was first observed in a temperate country, Japan. It probably could not survive in New Zealand at present, but may spread southwards with global warming. It is included here as it infects many families of fishes, and those in New Zealand would be immunologically naïve. Also, grey mullet (*Mugil cephalus*), which is particularly susceptible and may have aquaculture potential, occurs around New Zealand.

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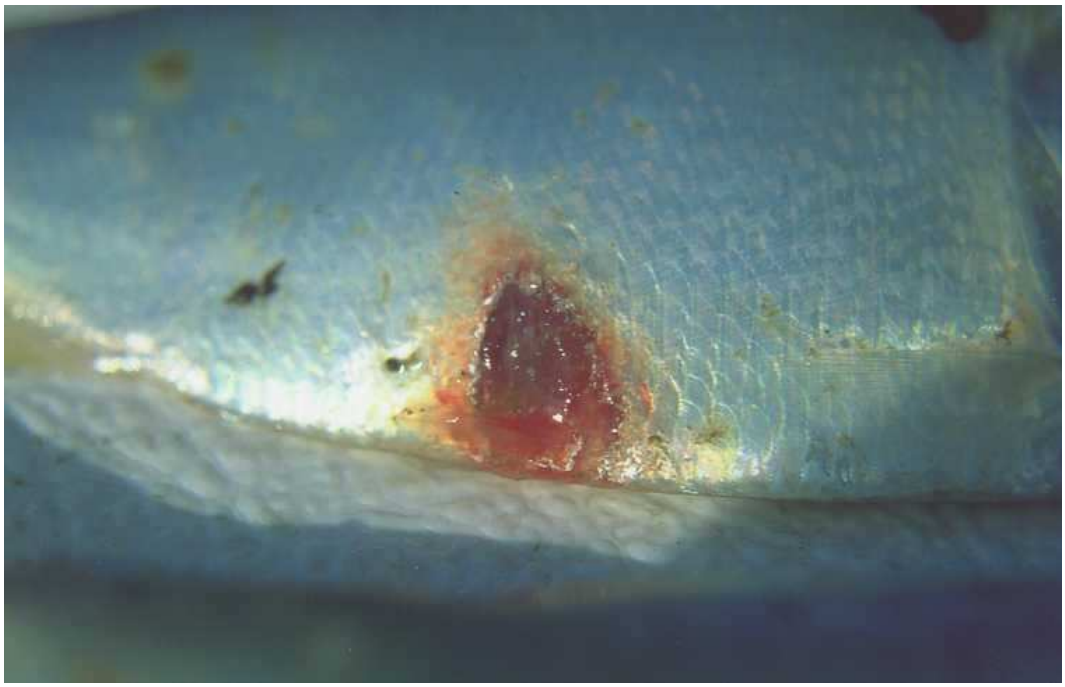
EUS-like lesions in Atlantic menhaden (*Brevoortia tyrannus*) from Virginia,U.S. Photos by B. Diggles.



Above and below: EUS-like lesions on the ventral surface associated with mixed fungal- bacterial infection.

Disease: Flavobacterial diseases (fin rot)

Species and life stage affected: Juveniles and adults of virtually all species of marine fish, including snapper (*Pagrus auratus*).

Gross signs: Grossly visible, yellowish tinged erosive lesions of the pectoral, caudal, and anal fins, reddened ulcerative lesions on the mouth, operculum, flanks, and caudal peduncle, congested gills.

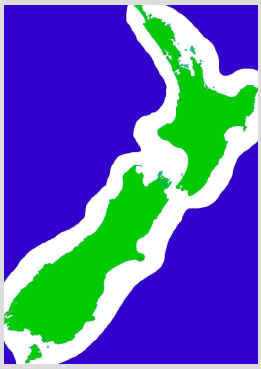
Causative agents: Long, thin, gliding bacteria of the *Flexibacter*/*Flavobacterium*/*Cytophaga* group, including *Flexibacter maritimus* and *Flavobacterium* sp.

Diagnosis: Presumptive diagnosis requires detection of long (up to 30 µm), thin (1–2 µm) rod shaped bacteria which exhibit gliding motility in wet squashes of affected fin or skin. Definitive diagnosis requires culture of yellow/orange pigmented, gram-negative bacteria using cytophaga agar made with 70% seawater (Pazos et al. 1996), or marine agar 2216, followed by identification using biochemical or molecular techniques. Often it is difficult to culture the causative agents due to their fastidiousness and the large numbers of opportunistic bacteria (particularly *Vibrio* sp.) that invade external lesions (Kimura & Kusuda 1983).

Treatment and prevention: The disease can be minimised by good husbandry, i.e. provision of good water quality, sensible stocking densities, and minimising handling and other damage to fish. Use of immunostimulants can improve the non-specific immune response (Mulero et al. 1998) and may be useful in preventing this disease. Treatment with antibiotics may be possible in some circumstances once the identity and antimicrobial sensitivities of the bacteria involved are known, but routine use of antibiotics will promote development of resistant strains of bacteria and is not a viable long term alternative to good husbandry practices.

Distribution in New Zealand

These bacteria are ubiquitous in the marine environment and usually cause disease under poor environmental conditions and in stressed or damaged fish.



Worldwide distribution: Flavobacterial diseases occur worldwide (Austin & Austin 1993).

General comments: *Flexibacter maritimus* was originally described from an outbreak of disease in cultured red sea bream (*P. auratus*) in Japan (Masumura & Wakabayashi 1977, Wakabayashi et al. 1986). *Flexibacter* outbreaks in *P. auratus* are particularly problematic in Japan during winter (Wakabayashi et al. 1984). This may correspond to the apparent predisposition of snapper in New Zealand to flavobacterial disease during the winter months.

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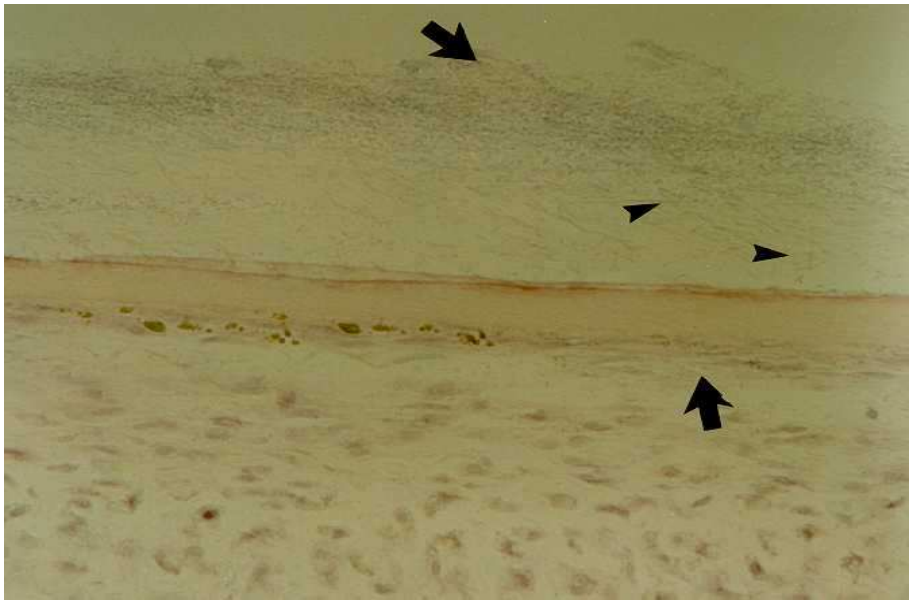
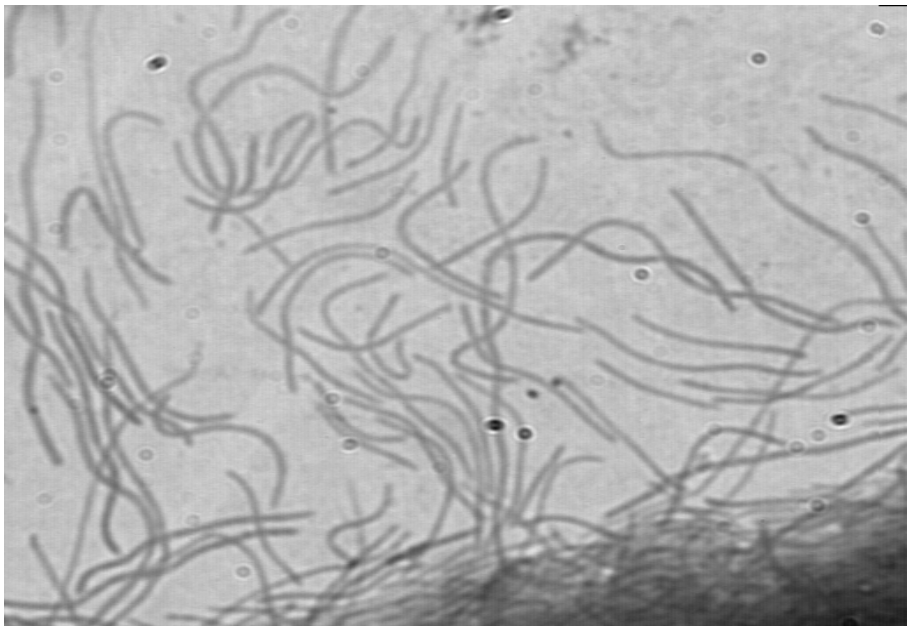




Flavobacterial disease of snapper. Photos by B. Diggles.

Above: Histopathology of an eroded fin showing large numbers of long, thin, rod shaped *Flavobacterium* sp. bacteria outside (arrowheads) and inside (arrow) the surface of a fin ray. Many *Vibrio*-like bacteria are also present.

Below: Gram-stained wet preparation from an eroded fin ray showing numerous long thin *Flavobacterium*-like bacteria which exhibit gliding motility.

Disease: Pasteurellosis (pseudotuberculosis)

Species and life stage affected: Japanese yellowtail (*Seriola quinqueradiata*), gilthead sea bream (*Sparus aurata*), sea mullet (*Mugil cephalus*), turbot (*Scophthalmus maximus*), and many more freshwater, brackish, and marine fishes, both wild and cultured.

Gross signs: Few reliable external signs of disease. Internally, numerous white bacterial colonies are visible throughout internal organs, especially the spleen and kidney. The spleen may be enlarged in some species. Granuloma formation occurs in chronic infections, causing the appearance of numerous white tubercles in internal organs – hence the name pseudotuberculosis.

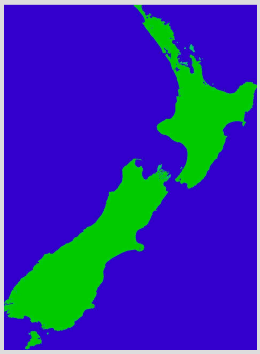
Causative agent: *Photobacterium damsela* subsp. *piscicida* (formerly known as *Pasteurella piscicida*, see Gauthier et al. 1995), a bacterium.

Diagnosis: Presumptive diagnosis from gross or internal signs of disease is unreliable. Histology may show extensive multifocal necrosis in the spleen and kidney. Definitive diagnosis requires isolation of non motile, gram-negative rod-shaped bacteria with bipolar staining characteristics from internal organs, especially spleen and kidneys, followed by identification using biochemical, antibody, or molecular techniques. Will grow on most bacteriological media provided 0.5% NaCl is present.

Treatment and prevention: Maintenance of water temperature below 20 °C, good water quality, and adoption of husbandry techniques which minimise handling or other damage are strongly recommended. Antibiotics may provide effective treatment, but resistant strains readily evolve and vaccination is the preferred method for controlling the disease (Kusuda et al. 1988).

Distribution in New Zealand

Unreported. This disease is included here as it infects a range of fish, including species closely related to those being cultured in New Zealand.



PASTEURELLA INFECTIONS MAY BE EXOTIC. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: Pasteurellosis occurs in Japan, Taiwan,U.S., and Europe (Daly 1998).

General comments: In Japan and Spain, pasteurellosis tends to occur in summer when water temperatures are in the range of 20– 25 °C. The disease is a serious problem in culture of yellowtail (*Seriola quinqueradiata*) in Japan and can result in stock losses of up to 50% in individual farms (Daly 1998). Yellowtail appear to be particularly susceptible to infection compared with other species of *Seriola* (see Kawakami et al. 2000). *Photobacterium damsela* subsp. *piscicida* is one of the few fish pathogens that can cause massive mortalities in wild fish (Snieszko et al. 1964).

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No photographs currently available

Disease: Streptococcosis

Species and life stage affected: Juvenile and adult Japanese yellowtail (*Seriola quinqueradiata*), sea mullet (*Mugil cephalus*), flounder (*Paralichthys olivaceus*), and many more freshwater, brackish water, and marine fishes, both wild and cultured.

Gross signs: Exophthalmia, petechial haemorrhaging on the inside of the opercula, and congestion at the pectoral and caudal fins and mouth. Skin lesions may occur in some species. Some species can be asymptomatic carriers.

Causative agent: *Streptococcus iniae*, a bacterium.

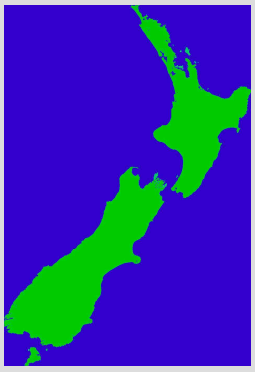
Diagnosis: Presumptive diagnosis from gross signs is unreliable due to similarities with other diseases. Definitive diagnosis requires isolation and biochemical identification of the causative bacterium from the internal organs of affected fish.

Treatment and prevention: Maintenance of good water quality and adoption of husbandry techniques which minimise handling or other damage are strongly recommended. Antibiotics (penicillins, cephalosporins, rifampicin, bacitracin, sodium nifurstyrenate, diaminopyrimidines) may be effective treatments, but resistant strains are likely to evolve if antibiotics are used routinely as an alternative to good husbandry practices.

Distribution in New Zealand

Unreported. This disease is included here as it occurs in Australia, infects a range of fish, and also can infect humans.

STREPTOCOCCAL INFECTIONS MAY BE EXOTIC. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Japan,U.S. (Eldar et al. 1999), South Africa, Australia (Bromage et al. 1999), Spain, Italy, Israel, Bahrain, Venezuela.

General comments: Streptococcosis is a chronic septicaemic disease with systemic involvement. Cellular infiltration and bacteria occur in most organ systems (Perera et al. 1998). Gross signs of this disease may be confused with those of *Lactococcus garvieae* (see p. 76), but histology can reveal notable differences between the two diseases (Eldar & Ghittino 1999).

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No photographs currently available

Disease: Vibriosis

Species and life stage affected: All life stages of virtually all species of marine fish, including turbot (*Colistium nudipinnis*), brill (*Colistium guntheri*), snapper (*Pagrus auratus*), kingfish (*Seriola lalandi*), and seahorses (*Hippocampus abdominalis*).

Gross signs: Larvae: weak swimming, cessation of feeding, blackened gut, rapid increase in mortalities. Juveniles and adults: darkened colour, lethargy, loss of appetite, haemorrhages at the bases of fins, exophthalmia, red external lesions on various parts of the body and fins.

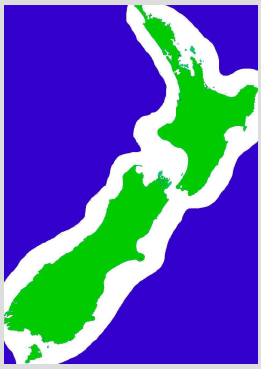
Causative agents: Infection and/or septicaemia from various species of opportunistic marine bacteria of the genus *Vibrio*, including *V. anguillarum*, *V. harveyi*, and *V. splendidus* I.

Diagnosis: Presumptive diagnosis can be obtained by observation of the clinical signs of infection, i.e., cessation of feeding, blackened gut in larvae, reddened lesions on the body and fins in juvenile and adult fish. Definitive diagnosis is by culturing and identifying bacteria from the blood or internal tissues of affected fish on TCBS agar using standard microbiological methods.

Treatment and prevention: The disease can be minimised by good husbandry, i.e. provision of good water quality, good quality live food, and minimising handling and other damage to fish. Manipulating the microbial flora of the rearing water or live food cultures with probiotic bacteria may reduce vibriosis and other bacterial diseases in larval and juvenile fish (Gatesoupe 1999). Immunostimulants may also improve resistance of fish to vibriosis (Vadstein 1997). Treatment with antibiotics may be possible in some circumstances once the identity and antimicrobial sensitivities of the bacteria involved are known.

Distribution in New Zealand

Opportunistic bacteria of the genus *Vibrio* are ubiquitous in the marine environment and can infect damaged or compromised fish anywhere throughout New Zealand.



Worldwide distribution: Vibriosis occurs worldwide (Egidius 1987).

General comments: May occur whenever sub-optimal culture conditions exist (Diggles et al. 2000). Routine use of antibiotics to control vibriosis will promote development of resistant strains of bacteria and is not a viable long term alternative to good husbandry practices.

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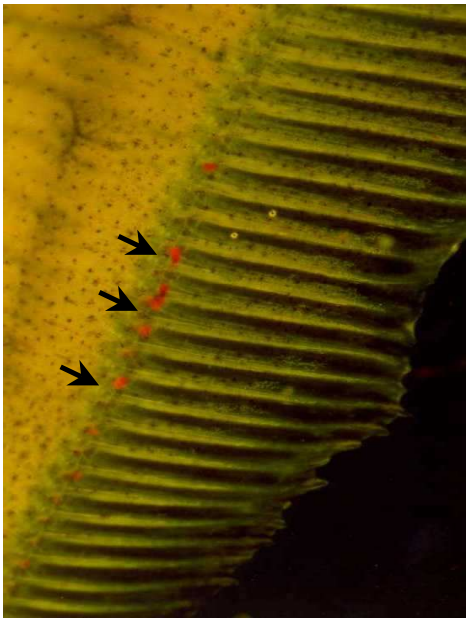
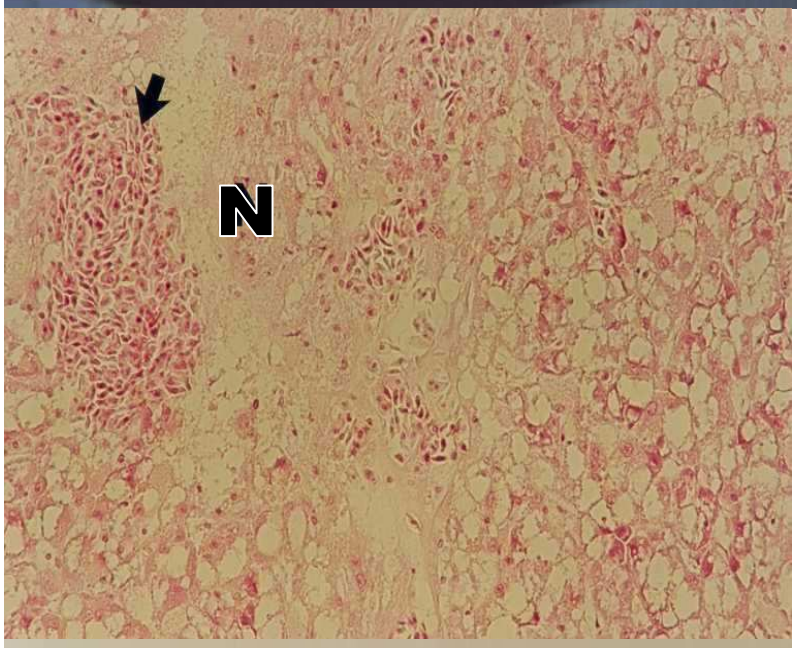
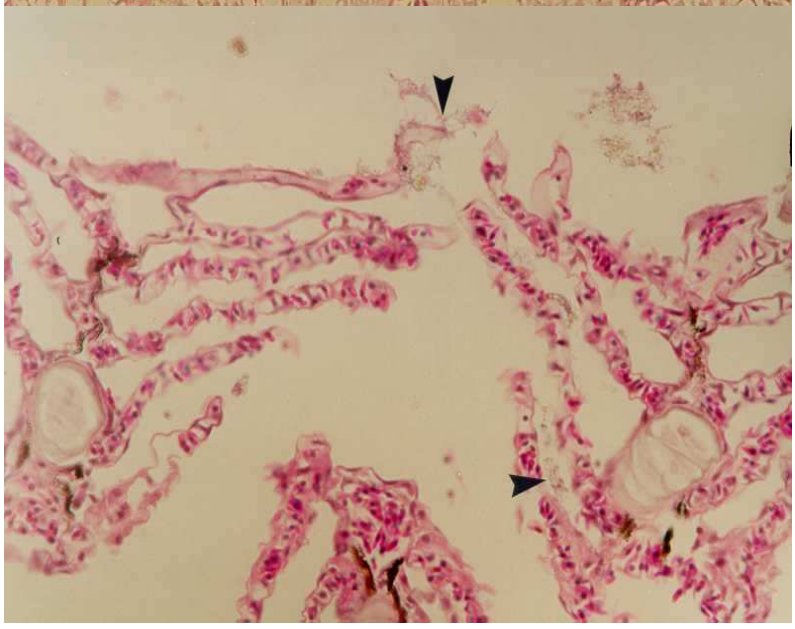






Vibriosis in marine fish. Photos by B. Diggles.

Above left: A large red lesion on the side of a snapper (*Pagrus auratus*) associated with handling damage.

Above right: Haemorrhages at the base of fins of a turbot (*Colistium nudipinnis*) associated with systemic vibriosis.

Left: Histology of the liver of a juvenile turbot (*C. nudipinnis*) with vibriosis, showing necrosis (N) and haemorrhage (arrows).

Below left: Histology of the gills of a seahorse (*H. abdominalis*) suffering from vibriosis, showing clumps of bacteria (arrowhead) adhering to gill filaments.

Protozoan diseases of marine fishes

Disease: Amoebic gill disease (AGD)

Species and life stage affected: Juvenile and adult salmonids reared in marine areas are most commonly affected, particularly Atlantic salmon (*Salmo salar*) and rainbow trout (*Oncorhynchus mykiss*), but occasionally chinook (*O. tshawytscha*) and coho salmon (*O. kisutch*). Also recorded in European turbot (*Scophthalmus maximus*), sea bass (*Dicentrarchus labrax*), and sharpsnout seabream (*Diplodus puntazzo*).

Gross signs: Reduced appetite, respiratory distress, and lethargic swimming near the top of the cage. Pale areas and congestion in the gills. AGD is usually associated with high water temperatures (12–20 °C) and high salinities, but recently has been reported at water temperatures of 9–10 °C (Douglas-Helders et al. 2001).

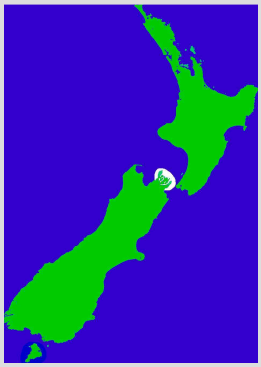
Causative agent: Amoebae of the genus *Neoparamoeba* (formerly *Paramoeba*), usually *N. pemaquidensis*.

Diagnosis: Experienced personnel can sometimes obtain a presumptive diagnosis by viewing the gross morphology of the gills. The presence of the amoebae results in prominent epithelial hyperplasia, complete fusion of the secondary lamellae, and gill congestion. Confirmatory diagnosis via IFAT or stained wet preparations of affected areas of the gill (Zilberg et al. 1999) is required to demonstrate amoebae.

Treatment and prevention: Freshwater baths are used to treat AGD in Atlantic salmon in Tasmania (Parsons et al. 2001). Some fish can acquire resistance to AGD, probably due to non-specific immune factors (Munday et al. 2001).

Distribution in New Zealand

Amoebae have been reported in the gills of caged chinook salmon in the Marlborough Sounds, but they have not been associated with any significant disease outbreaks in New Zealand.



Worldwide distribution: AGD has been recorded in salmonids in Australia (Roubal et al. 1989), the United States (Kent et al. 1988), and Europe (Palmer et al. 1997) and in turbot (*Scophthalmus maximus*) in Spain (Dykova et al. 1998, 1999).

General comments: *Neoparamoeba pemaquidensis* is an opportunistic pathogen that is normally free-living in seawater. Similar amoebae occur on seacaged chinook salmon in New Zealand but appear to have no significant impact on production. This is in contrast to Tasmania where heavy losses of Atlantic salmon are caused by AGD. This difference could be due to chinook salmon having a degree of innate resistance to *Neoparamoeba* infection.

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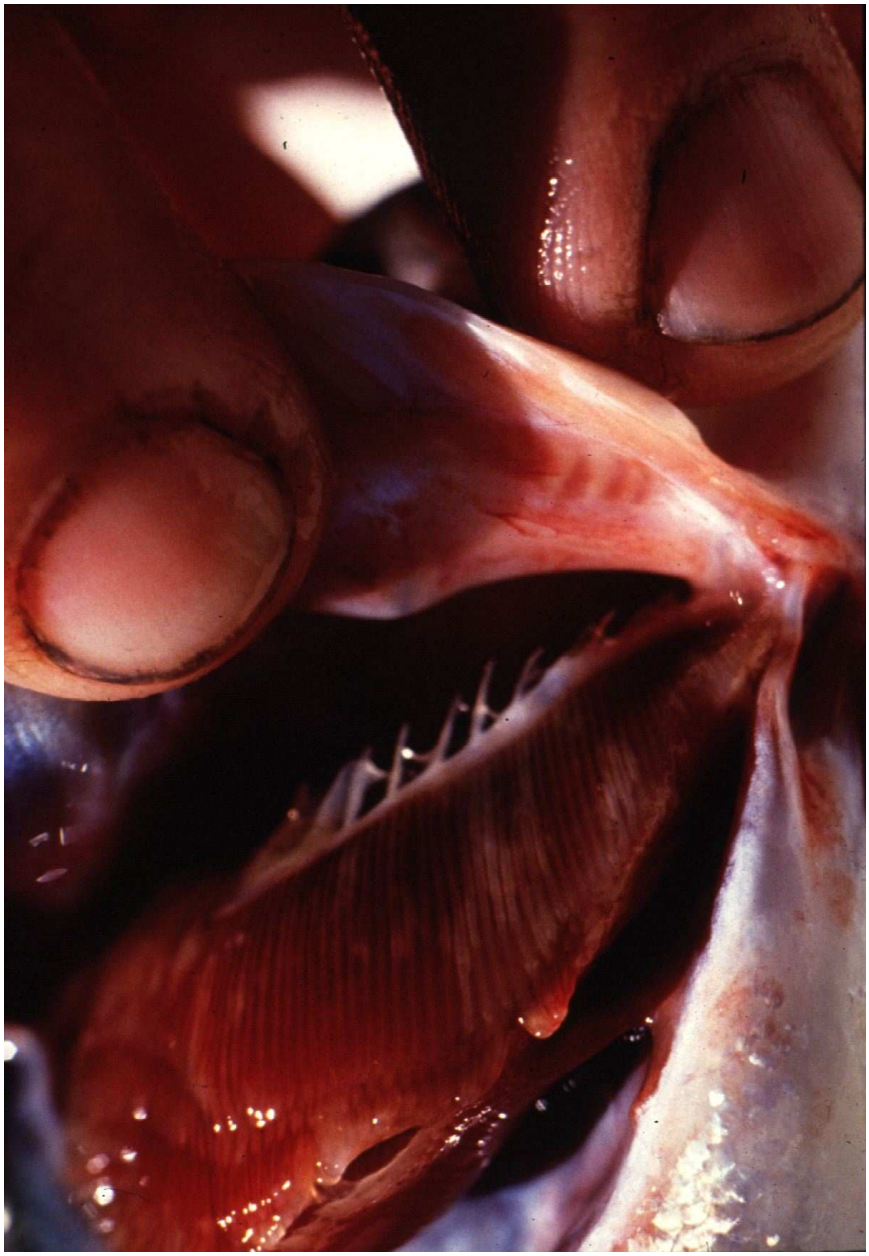


Gill of a Tasmanian Atlantic salmon with AGD. Photo by B. Munday and reproduced with permission from *Journal of Fish Diseases.*

Note multiple pale areas and congested appearance of the gills.

Disease: Trichodiniasis

Species and life stage affected: Larvae, juveniles, and adults of most species of wild and cultured marine fish, including snapper (*Pagrus auratus*) and turbot (*Colistium nudipinnis*).

Gross signs: Heavily infected fish can lose their appetite, become emaciated, and exhibit flashing behaviour and respiratory distress.

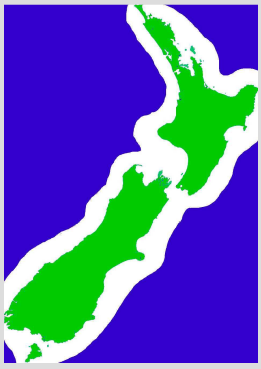
Causative agents: Ciliates of the genus *Trichodina*.

Diagnosis: Trichodinids with their distinctive round denticulate rings and "flying saucer" shape, can be observed in gill smears or squash preparations of gill biopsies.

Treatment and prevention: Formalin baths of 200 ppm for 30 minutes are the recommended treatment for commercial situations (Diggles 2000). Heavy ciliate ectoparasite infections usually occur as a result of poor water quality and or stressors which predispose fish to infection. Healthy fish in a clean environment are seldom heavily infected. Many species of wild fish carry trichodinid infections in New Zealand (Laird 1953), so exclusion of these from the culture facility and quarantine of new introductions is recommended.

Distribution in New Zealand

Trichodinids occur throughout New Zealand waters on wild and cultured fish.



Worldwide distribution: Trichodinid ciliates infect fish worldwide (Lom 1984).

General comments: Though wild fish may harbour heavy infections of trichodinids, these usually do not cause disease. However, ciliate numbers can build up quickly on confined fish, especially at higher temperatures. If no action is taken in instances of heavy *Trichodina* infection, skin and gill lesions can develop, probably from damage to the epithelium by the sharp borders of the adhesive disc (Lom 1984). These lesions can become colonised by secondary bacterial and fungal invaders and can contribute to mortalities (Liewes 1984, Lom 1984).

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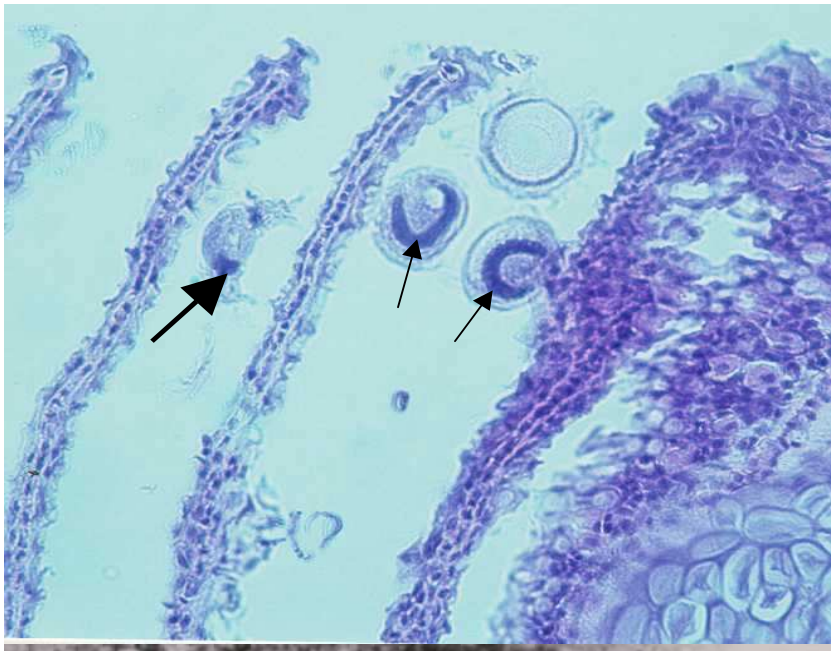
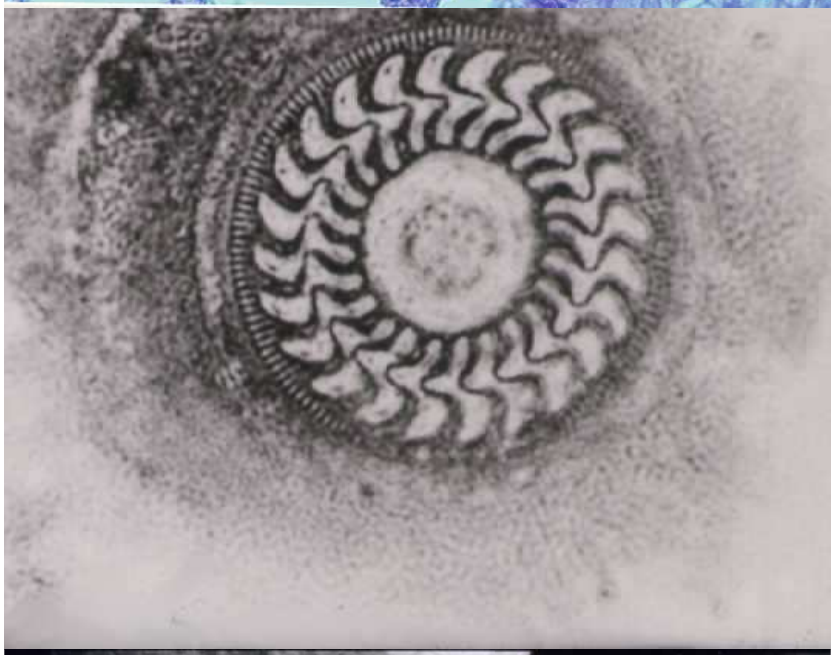
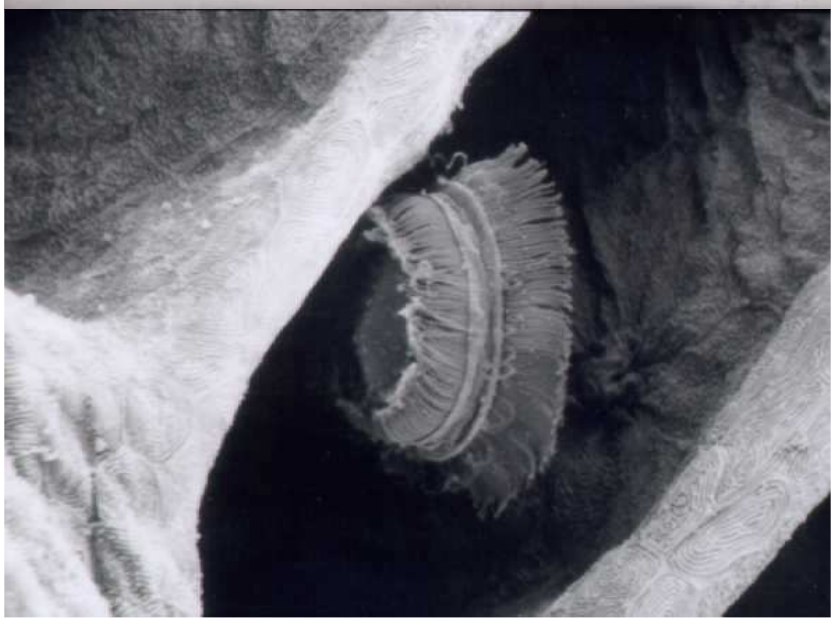






Trichodiniasis of marine fish. Photos by B. Diggles

Above: *Trichodina* sp. in a histology section of the gill of a turbot (*Colistium nudipinnis*). Note the prominent dark staining, and semicircular macronucleus (small arrows) which is less evident in side view (large arrow).

Middle: A silver stained *Trichodina multidentis* from a wild caught flounder (*Rhombosolea leporina*), showing the intricate pattern of denticles of the adhesive disc.

Below: SEM of a *Trichodina* in the gills of a flounder, showing saucer shape and rows of adoral (left side) and locomotory (right side) cilia.

Disease: White spot disease (cryptocaryonosis)

Species and life stage affected: Juveniles and adults of most species of wild and cultured marine fish.

Gross signs: Multiple, pinpoint white spots on the skin, fins, eyes, and gills, usually associated with flashing and rubbing behaviour. Heavily infected fish may have clouded eyes, produce excessive amounts of mucus, lose their appetite, become lethargic and emaciated, and exhibit respiratory distress.

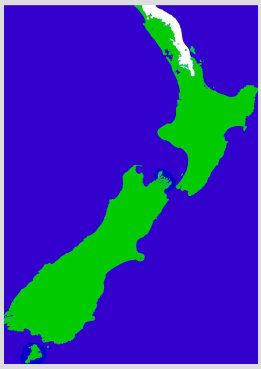
Causative agent: A ciliate, *Cryptocaryon irritans*. A number of strains exist, including at least two strains adapted to waters temperatures of 15 °C or below (Diggles & Adlard 1997, Jee et al. 2000).

Diagnosis: Presumptive diagnosis can be made from the appearance of white spots on the fins, skin and gills. Definitive diagnosis requires visualisation of large (50–750 µm) round to pear-shaped ciliates in wet preparations of scrapes of affected organs. Infective stages have a quadripartite nucleus.

Treatment and prevention: The direct lifecycle can be disrupted by transferring fish to new holding tanks every 3 days for at least 10 days (Colorni 1987), but this can be stressful to fish. Chemical treatments which kill the theront infective stage include 3–10 day baths of copper sulphate (0.15–0.25 ppm) and formalin (50 ppm), while 8 x 10^5 µW s^{-1} UV irradiation and 8 mg/h ozonation are also effective (Colorni & Burgess 1997). Oral administration of lactoferrin (40 mg/kg body weight per day for 28 days) prevented *C. irritans* infections of red sea bream in Japan (Kakuta & Kurokura 1995).

Distribution in New Zealand

Cryptocaryon irritans has been observed in captive snapper held in glass tanks in Auckland (Hine 1982). It probably occurs in wild fish along the north east coast of the North Island. It may also be present in imported marine ornamental fish.



Worldwide distribution: *C. irritans* has caused disease in many species of cultured and ornamental marine fish worldwide (Colorni & Burgess 1997), mostly at water temperatures above 19 °C.

General comments: Global warming may predispose New Zealand fish to this disease. Wild fish can be infected by *C. irritans* at very low intensities (Diggles & Lester 1996), which do not cause disease. However, on confined fish ciliate numbers can build up quickly. If no action is taken in these instances, fish can die from respiratory failure, osmotic imbalance, and secondary bacterial infection. If fish survive a heavy infection, they may become refractory to further infection, suggesting that fish can develop acquired immunity (Burgess & Matthews 1995, Bryant et al. 1999). Any attempts at treatment and control must consider the direct lifecycle, which includes an encysted tomont stage, and an infective theront stage as well as the adult ciliate on the fish (Colorni 1987).

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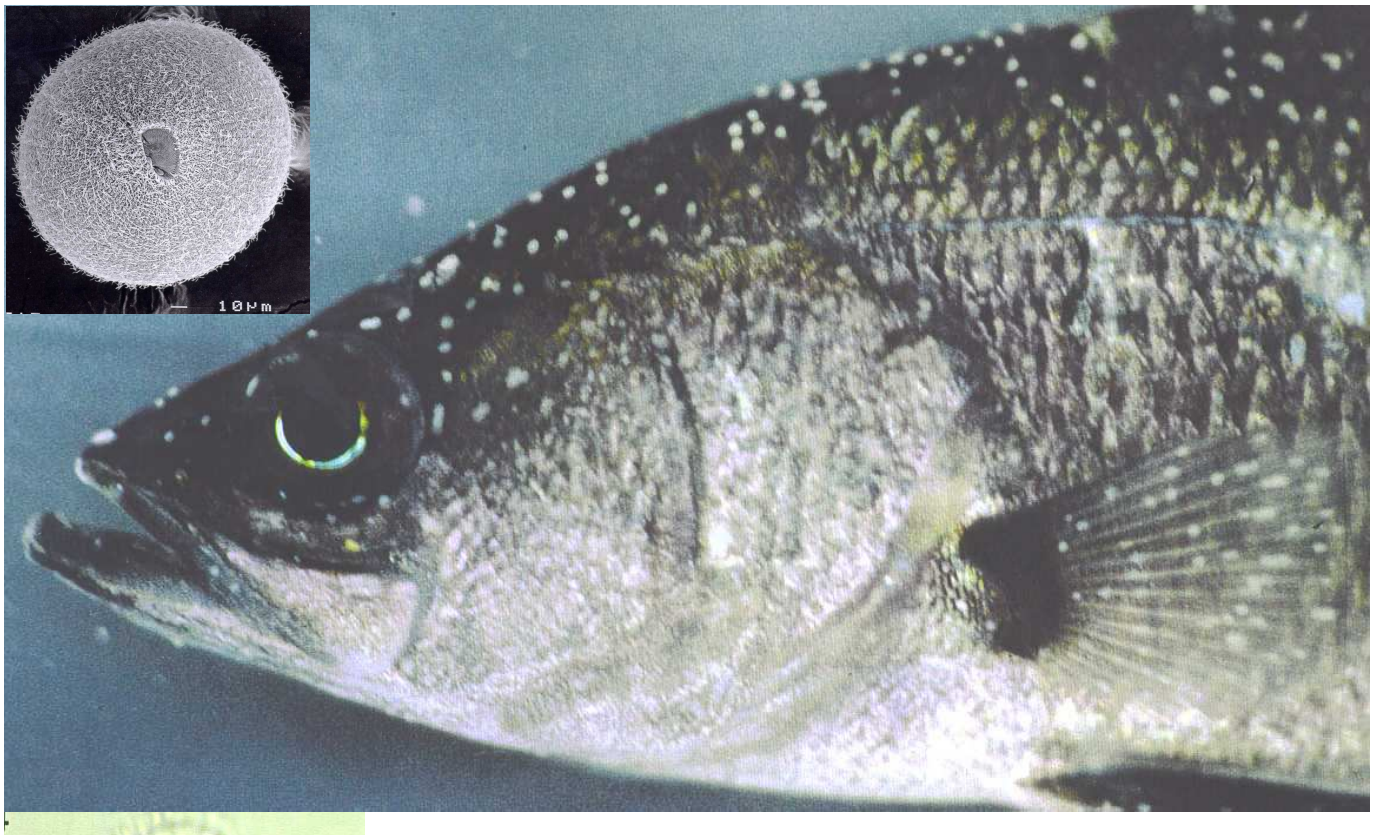
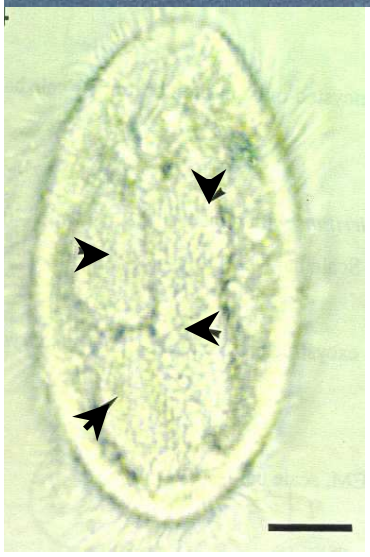




White spot infections of marine fish caused by *Cryptocaryon irritans*.

Above: A barramundi (*Lates calcarifer*) heavily infected with *C. irritans*. Individual trophonts appear as distinct white spots in the skin and fins. Photo by M. Bryant.
Inset: SEM of a trophont, showing the oral apparatus. Scale bar = 10 µm. Photo by B. Diggles.

Left: Unstained wet preparation of the theront infective stage of *C. irritans*. Note the prominent quadripartite macronucleus (arrowheads). Scale bar = 10 µm. Photo by B. Diggles.

Metazoan diseases of marine fishes

Disease: Copepod infestation ("sea lice")

Species and life stage affected: Ectoparasitic copepods occur on many marine fishes. *Abergasilus amplexus* exhibits low host specificity and may infect kahawai (*Arripis trutta*), longfinned eel (*Anguilla dieffenbachi*), shortfinned eel (*A. australis*), yellowbelly flounder (*Rhombosolea leporina*), and others. *Caligus longicaudatus* occurs on sea-caged sockeye salmon (*Oncorhynchus nerka*) and unknown species of wild fish. *Caligus lalandei* and *C. aesopus* occur on wild and seacaged kingfish (*Seriola lalandi*).

Gross signs: None recorded for *A. amplexus*. For *Caligus* sp., heavily infected fish may exhibit appetite suppression, flashing behaviour and may rub against nets and other objects while trying to remove the ectoparasitic copepods, which can be seen with the naked eye attached to the surface of the skin. External lesions and secondary bacterial infections are common in heavy infections.

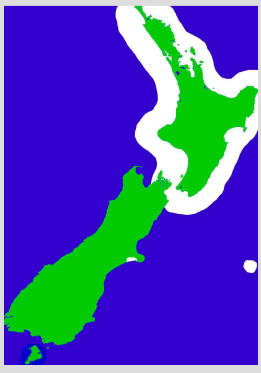
Causative agents: Ectoparasitic copepods. *Abergasilus amplexus* occurs on the gills; members of the family Caligidae (*Caligus longicaudatus*, *C. aesopus,* and *C. lalandei*) occur mainly on the skin and fins.

Diagnosis: Detection on the gills of ergasilid copepods with only three pairs of legs, or detection of copepods conforming to the family Caligidae (see Jones 1988) on the skin or gills.

Treatment and prevention: *Arbergasilus amplexus*: Movement of fish either to freshwater or full strength seawater may remove them from the range of the parasite, which occurs only in euryhaline areas. Caligidae: A variety of chemotherapeutic agents have been used against sea lice (*Lepeophtheirus salmonis*) in the Northern Hemisphere, including organophosphate insecticides (trichlorfon, dichlorvos), ivermectin, and hydrogen peroxide (Roth et al. 1993). Because of problems with development of resistance (e.g., Treasurer et al. 2000) and/or environmental concerns (e.g. Davies et al. 1998), long term use of most chemotherapeutic agents has not been feasible. This has increased research into biological control using cleaner wrasse (e.g., Deady et al. 1995), and development of vaccines (Raynard et al. 1994) and lice-resistant fish stocks (Mustafa & MacKinnon 1999).

Distribution in New Zealand

A. amplexus has been recorded from euryhaline environments, including Lake Ellesmere, the Awakino River, and the Chatham Islands. *Caligus longicaudatus* was recorded from sockeye salmon in the Marlborough Sounds, and *C. lalandei* and *C. aesopus* occur on kingfish in the North Island.



Worldwide distribution: *Abergasilus* is a genus so far recorded only from New Zealand (Hewitt 1979). Members of the family Caligidae occur on marine fish worldwide and some are important pathogens of seacaged salmonids in the Northern Hemisphere (Roth et al. 1993).

General comments: *Abergasilus amplexus* is a potentially dangerous pathogen, but its impact on aquaculture is likely to be minimal, unless fish culture is undertaken in estuarine areas (Hewitt 1979). Members of the Caligidae are widespread on wild marine fish but to date apparently have not been problematic in sea-cage culture of salmonids or other species in New Zealand.

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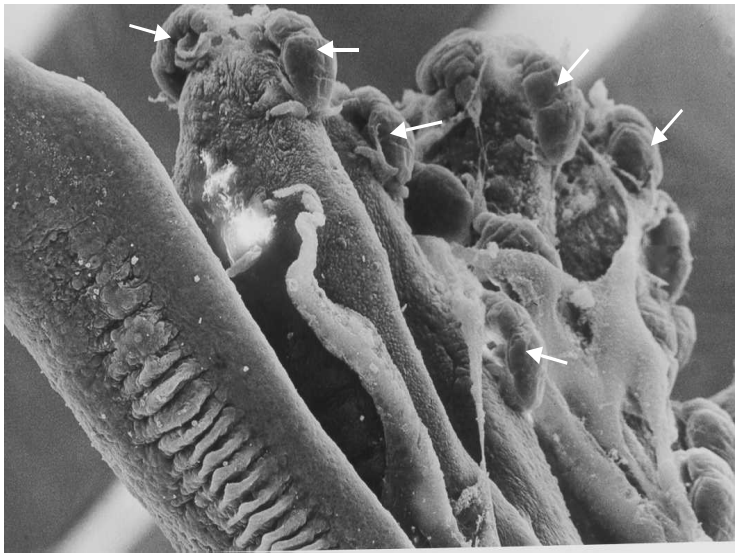
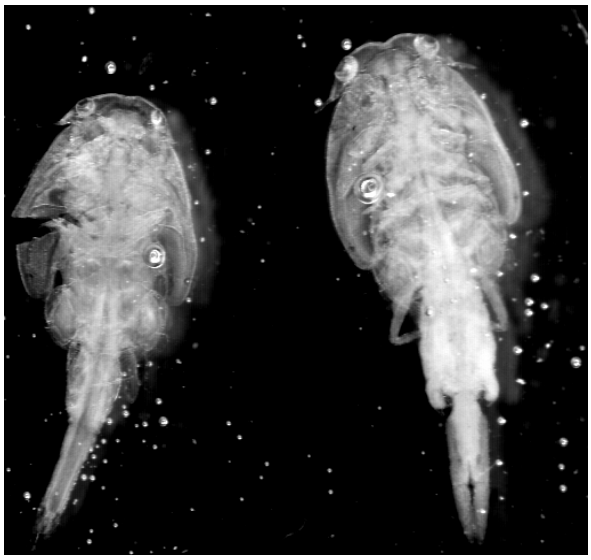




Left: SEM of gills of an eel (*Anguilla dieffenbachii*) infected with *Abergasilus amplexus*. Note numerous copepods (arrows) attached to the gill filaments, many of which exhibit clubbing, hyperplasia, and occlusion of the secondary lamellae. Photo by M. Hine.

Right: Wet preparations of *Caligus lalandei* collected from the skin of wild caught kingfish (*Seriola lalandei*). The copepod on the left is the male; the female is on the right. Photo by B. Diggles.

Disease: Ectoparasitic worms on the skin (benedenisis)

Species and life stage affected: Juvenile and adult kingfish (*Seriola lalandi*) and snapper (*Pagrus auratus*).

Gross signs: Flashing and rubbing behaviour associated with the presence of medium to large flattened worms adhering to the flanks of affected fish, reduced appetite, skin lesions.

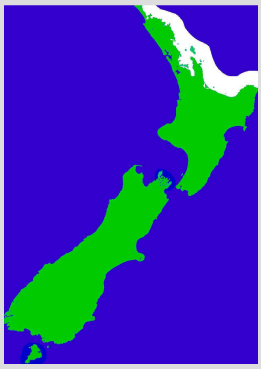
Causative agents: Monogenean worms (Phylum Platyhelminthes). Kingfish are infected by *Benedenia seriolae*; snapper are infected by *Benedenia sekii*. These worms exhibit high host specificity.

Diagnosis: Diagnosis involves detection of relatively large (2–10 mm), flattened, clear, or pigmented capsalid monogeneans conforming to *B. seriolae* on the flanks of kingfish, or *B. sekii* under the fins of snapper. A simple method of detecting infections without handling fish is by placing plankton netting (50–100 µm pore size) over the water outlet from holding tanks for a few hours and then examining the netting at 40x magnification with a dissecting microscope for tetrahedral eggs.

Treatment and prevention: Freshwater baths of 5 minutes are 100% effective in killing *B. seriolae* (see Sharp 2001) and *B. sekii* (Diggles unpublished). Anthelmintics (e.g., praziquantel, levamisole) may also be effective (Thoney & Hargis 1991), but larvae inside eggs can survive most treatments and will quickly reinfect treated fish unless treatments are repeated two or three times at 2 week intervals.

Distribution in New Zealand

Benedenia seriolae and *B. sekii* are known to occur on kingfish and snapper along the east coast of the North Island, and probably occur throughout the New Zealand range of their respective host species.



Worldwide distribution: Monogenea of the genus *Benedenia* cause disease in captive fish worldwide.

General comments: Although these parasites occur naturally on wild fish without causing disease, numbers of monogenean ectoparasites such as *Benedenia* tend to increase on confined fish (Roubal et al. 1996) because confinement increases the chances of completion of their direct lifecycle.

References

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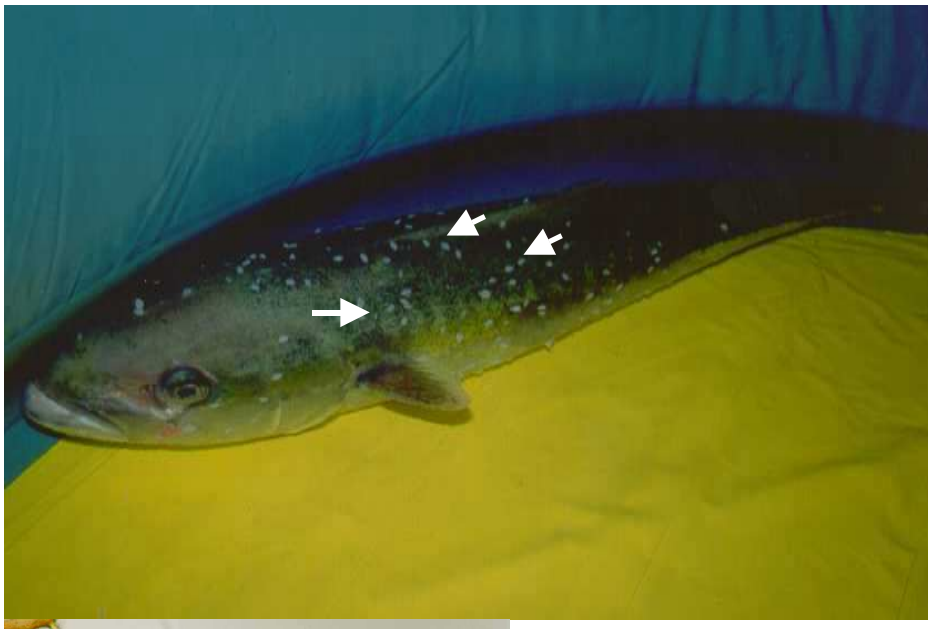
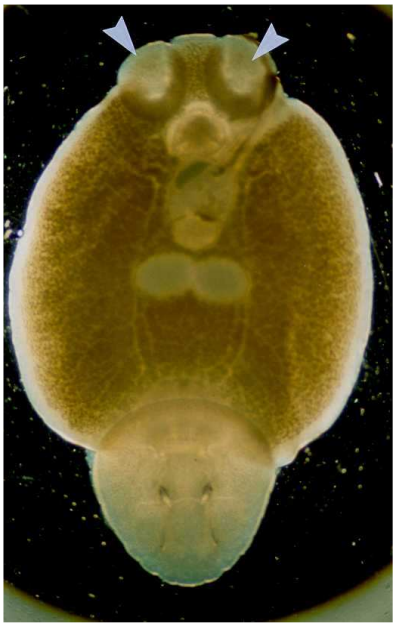




Benedenisis. Photos by B. Diggles.

Above left: A kingfish with numerous *Benedenia seriolae* worms (arrows) on its flanks. The worms are normally clear but have turned white after a freshwater bath treatment.
Above right: An adult *B. seriolae*, with distinctive oral suckers (arrowheads).
Left: Tetrahedral eggs of *B. seriolae*. Some eggs have larvae with eyespots (arrow), others have hatched (arrowheads).
Below left: Characteristic reddened lesions under the fins of snapper caused by site-specific attachment of *Benedenia sekii* in these areas.
Below right: An adult *B. sekii*, with pigment stripes which aid camouflage.

Disease: Ectoparasitic worms on the fins (anoplodiscisis)

Species and life stage affected: Juvenile and adult snapper (*Pagrus auratus*).

Gross signs: Fish exhibit flashing and rubbing behaviour in attempts to remove ectoparasites. Numerous oval shaped monogeneans 1–2 mm long attached to the pectoral and caudal fins and occasionally on the flanks and inside the nasal cavity. Emaciation and partial loss of fins on heavily infected fish.

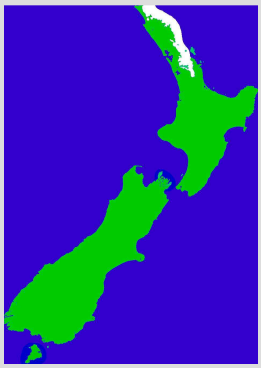
Causative agent: The monogenean worm, *Anoplodiscus cirrusspiralis* (Phylum Platyhelminthes).

Diagnosis: Scrapes of fins detect the presence of *Anoplodiscus*-type monogeneans with oval bodies and a small opisthaptor.

Treatment and prevention: Freshwater baths (15 min, after determining the susceptibility of fish to freshwater) or low salinity (5‰ for 1 hour) kill 100% of *A. cirrusspiralis* (see Roubal et al. 1992). Anthelmintics such as praziquantel and levamisole may also be effective (Thoney & Hargis 1991), but as larvae inside eggs can survive most treatments, they will quickly reinfect treated fish unless treatments are repeated two or three times at 2 week intervals. These parasites occur on wild snapper in New Zealand, so exclusion of wild fish from aquaculture facilities, adequate water filtration, and quarantine of all new broodstock introductions is recommended.

Distribution in New Zealand

Anoplodiscus cirrusspiralis has been recorded from snapper along the east coast of the North Island north from Auckland. However, they probably occur throughout the range of snapper in New Zealand.



Worldwide distribution: *Anoplodiscus cirrusspiralis* has been recorded from *P. auratus* in Australia and New Zealand (Roubal et al. 1983, 1992, West & Roubal 1998, Sharples & Evans 1995a, 1995b, 1995c) and the closely related *Anoplodiscus tai* is found on cultured *P. auratus* in Japan (Ogawa, 1994).

General comments: A monogenean which feeds on host mucus and epidermal cells on the fins, causing increased chance of secondary bacterial infection. Mortalities do occur, but the major problem is reduced marketability due to numerous visible ectoparasites (Ogawa 1994). Because of their direct life cycle these parasites build up quickly on caged snapper, but some fish are resistant to infection (West & Roubal 1998).

References

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Left: The monogenean worm *Anoplodiscus cirrusspiralis* from the pectoral fin of snapper *Pagrus auratus*.
Photo by B. Diggles.

Disease: Ectoparasitic worms on the gills (bivaginisis, zeuxaptisis)

Species and life stage affected: Juvenile and adult kingfish (*Seriola lalandi*), snapper (*Pagrus auratus*).

Gross signs: Darkening and lethargy, pale gills, severe gill irritation, coughing, appetite suppression, rapid gill movements suggestive of hypoxia, emaciation, anaemia.

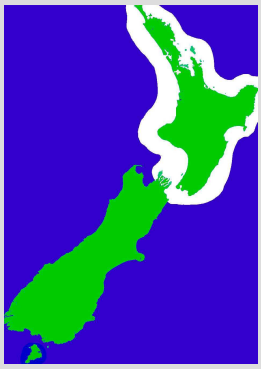
Causative agents: Blood feeding monogenean worms (Phylum Platyhelminthes) on the gills. *Zeuxapta seriolae* on kingfish and *Bivagina pagrosomi* on snapper. These worms exhibit high host specificity.

Diagnosis: Detection of long (up to 10 mm) thin worms strongly attached to the gills by an attachment organ with numerous clamps. For *Z. seriolae* the clamps are larger and more numerous on one side of the attachment organ than the other. For *Bivagina pagrosomi* equal numbers of clamps occur on each side of the attachment organ. A simple method of detecting infections without handling fish is to place plankton netting (50–100 µm pore size) over the water outlet from holding tanks for a few hours and then examine the netting with a dissecting microscope for fusiform eggs.

Treatment and prevention: Baths of 250 ppm formalin for 1 hour are 100% effective for removing *B. pagrosomi* from snapper (Diggles, unpublished), and 400 ppm formalin for 1 hour is 100% effective in removing *Z. seriolae* from kingfish (Sharp 2001). Anthelmintics such as praziquantel and levamisole may also be effective, but as larvae inside eggs can survive most treatments, they will quickly reinfect treated fish unless treatments are repeated two or three times at 2 week intervals. These parasites occur on wild snapper and kingfish in New Zealand, so exclusion of wild fish from aquaculture facilities, adequate water filtration, and quarantine of all new broodstock introductions is recommended.

Distribution in New Zealand

Zeuxapta seriolae has been recorded from kingfish along the east coast of the North Island. *Bivagina pagrosomi* is known from snapper throughout the North Island and also from the Marlborough sounds. Both parasites probably occur throughout the New Zealand range of their respective host species.



Worldwide distribution: Gill dwelling monogeneans occur on many species of wild and cultured fish worldwide. *Zeuxapta seriolae* and *Bivagina pagrosomi* both occur in Australia (Rohde, 1978, Roubal et al. 1996), and *Bivagina tai* occurs on *Pagrus auratus* in Japan (Ogawa 1988).

General comments: These parasites occur naturally on wild fish without causing disease, but their numbers increase on confined fish due to increased chances of completion of their direct lifecycle. Heavy infections of blood feeding monogeneans on the gills can cause anaemia, emaciation, and a low packed cell volume (Eto et al. 1976, Roubal et al. 1996).

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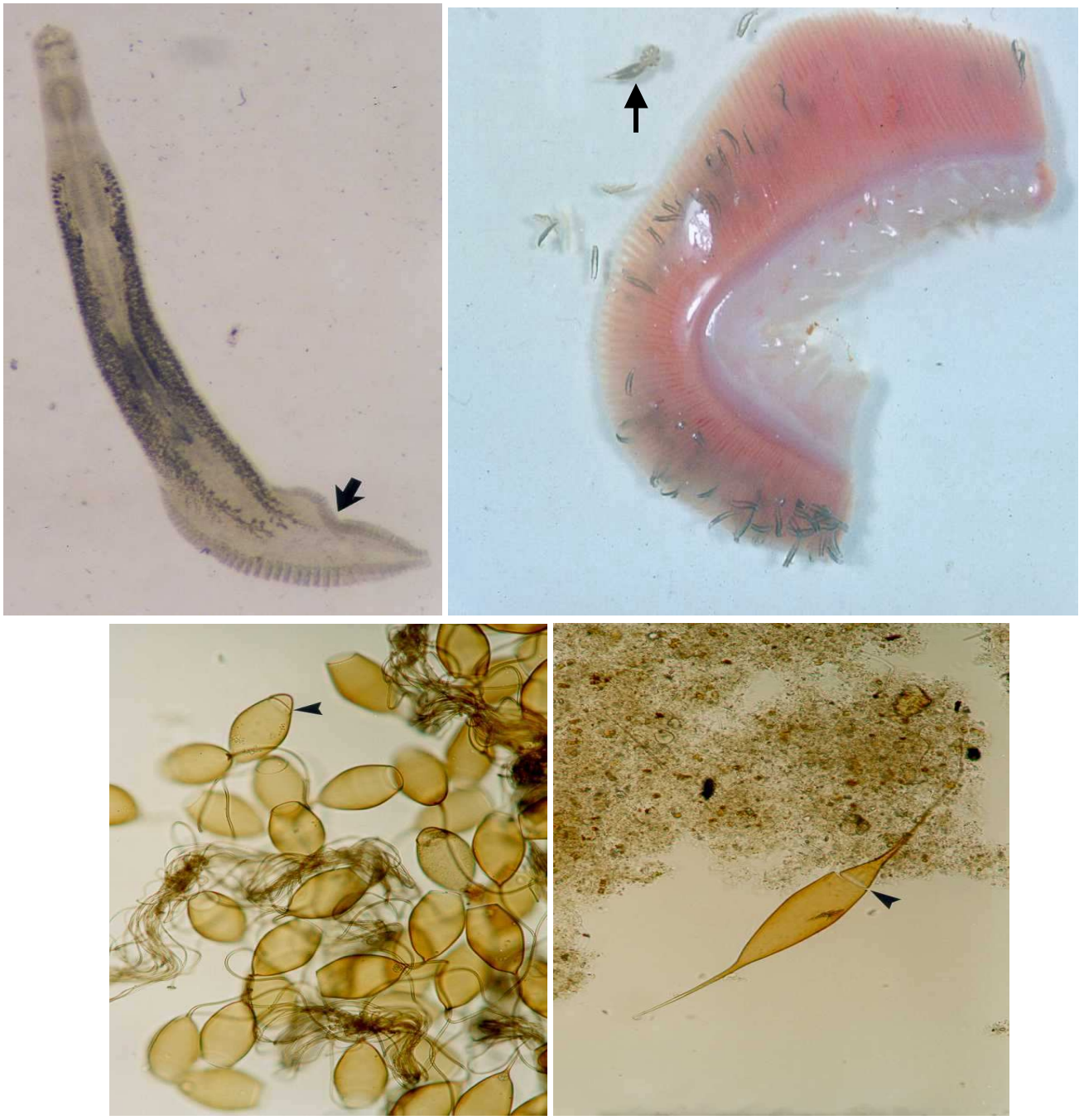


Ectoparasitic monogeneans on the gills. Photos by B. Diggles and N. Boustead.

Above left: *Zeuxapta seriolae* from the gills of a kingfish. Note numerous clamps on the asymmetrical attachment organ (arrow). **Above right:** A heavy infection of *Bivagina pagrosomi* on the gills of a snapper. Note also one specimen of *Choricotyle australiensis* (arrow). **Below left**: A clump of fusiform eggs of *Z. seriolae*. Note operculum of egg (arrowhead). **Below right:** A fusiform egg of *B. pagrosomi*. Note operculum (arrowhead).

Disease: *Myxidium* disease of snapper

Species and life stage affected: Juveniles and adults of snapper (*Pagrus auratus*).

Gross signs: None known in New Zealand snapper, but gilt head sea bream (*Sparus aurata*) in Europe affected by *Myxidium leei* are emaciated and display a bloated abdomen.

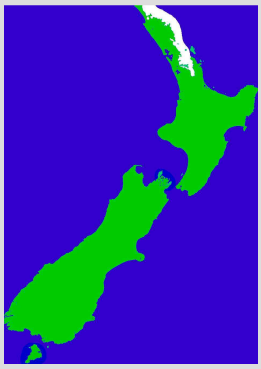
Causative agents: Myxosporeans of the genus *Myxidium*. *Myxidium* sp. occurs in New Zealand snapper, and *M. leei* is a pathogen of gilt head sea bream in Europe.

Diagnosis: Diagnosis by histopathology is required to demonstrate large *Myxidium* sp. plasmodia in bile ducts. In *Myxidium leei* infections, smears from the intestinal lining (can be done non lethally through the vent) show numerous arc-shaped myxosporean spores, 15 x 5 µm with two polar capsules (with seven turns of the polar filament) at each end.

Treatment and prevention: Oral administration of the antibiotic Fumagillin and baths of toltrazuril have been shown to be effective against some myxosporean stages (Schmahl et al. 1991). Prevention is by exclusion of wild fish which can be carrier species from aquaculture facilities and quarantine of all new introductions to avoid introducing myxosporeans. Filtration of incurrent water to 5 µm or less to remove infective stages is recommended.

Distribution in New Zealand

To date known only from snapper on the east coast north of Auckland.



Worldwide distribution: The closely related *Myxidium leei* has caused mortalities in gilt-head sea bream (*Sparus aurata*) in Europe (Diamant 1992, Diamant et al. 1994), and can also infect cultured red drum (*Sciaenops ocellatus*) (see Diamant 1998).

General comments: Although freshwater myxosporeans may require an intermediate oligochaete host to complete their life-cycle, there is evidence that some marine myxosporeans, including *M. leei*, may have a direct lifecycle (Diamant 1997, Diamant & Wajsbrot 1997), and thus may become problematic in culture. *Myxidium leei* causes a chronic infection which can be pathogenic and result in significant mortalities (Diamant 1992).

References

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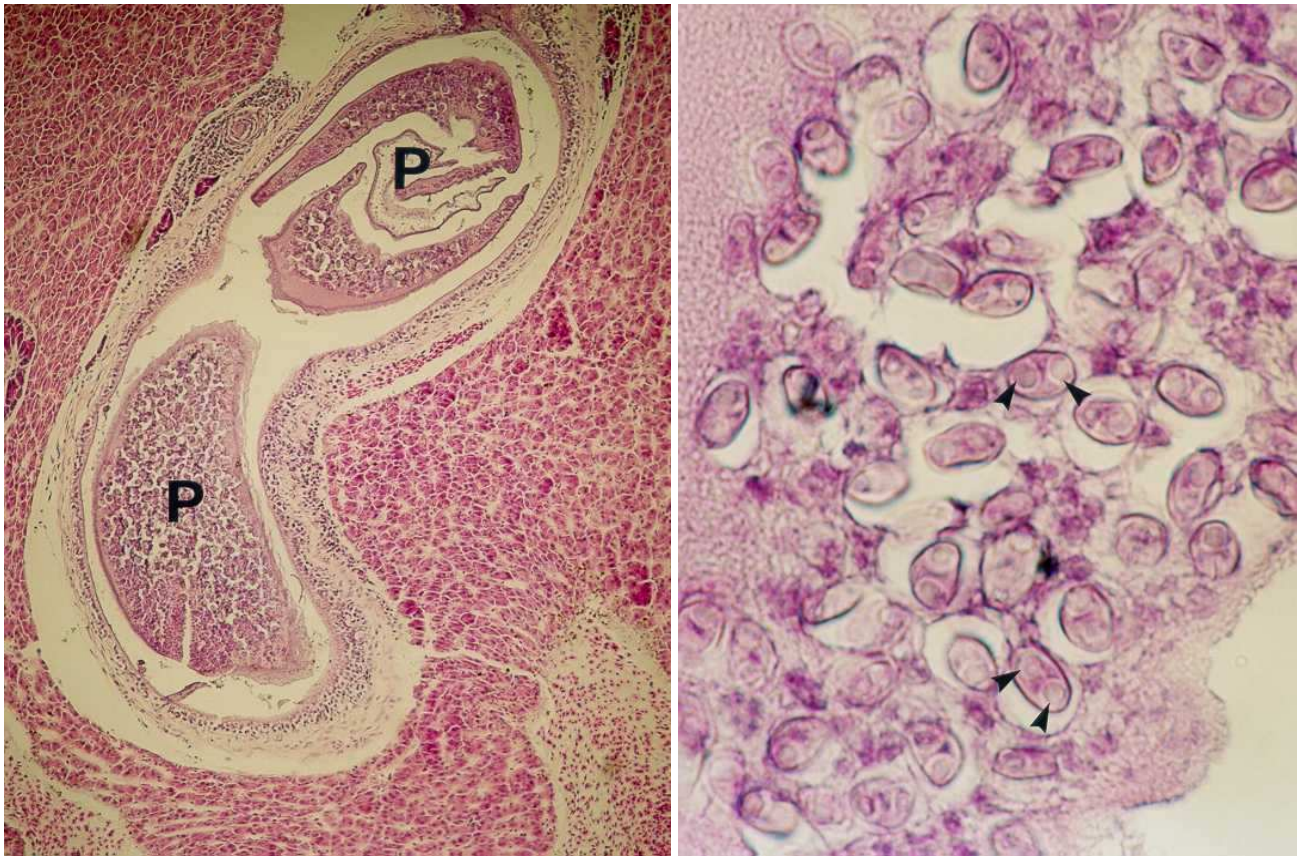


Myxidium sp. infection in cultured snapper (*Pagrus auratus*). Photos by B. Diggles.

Left: Histology of a bile duct in the liver of small snapper infected with *Myxidium* sp. Note enlargement and fibrosis of the duct due to a large plasmodium (P) containing numerous *Myxidium* sp. spores.

Right: Higher magnification of the plasmodium showing individual *Myxidium* sp. spores with two polar capsules (arrowheads).

Husbandry related diseases of marine fishes

Disease: Algal blooms

Species and life stage affected: All life stages of all species of marine fish and shellfish.

Gross signs: Varies between species and types of algal bloom. Fish may exhibit signs of respiratory distress, including slow swimming and gulping at the water surface. Shellfish may show minimal signs of disease but may harbour high levels of toxins which are harmful to humans when consumed.

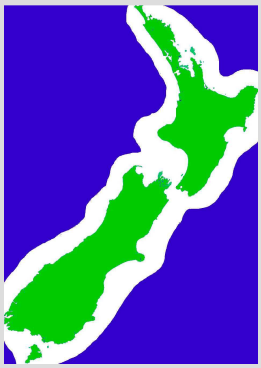
Causative agents: Blooms of harmful marine algae, including *Alexandrium* sp., *Chattonella* sp., *Chaetoceros* sp., *Dinophysis* sp., *Gambierdiscus* sp., *Gymnodinium* sp., *Heterosigma* sp., and *Pfiesteria* sp. Disease may result from abrasion or clogging of gills caused by high numbers of algal cells in the water, anoxia due to high biological oxygen demand at night or chemical oxygen demand when algal cells break down, or when algae produce extracellular chemical compounds which are toxic to fish and shellfish.

Diagnosis: Diagnosis is based on detection of high numbers of harmful species of algae in water around aquaculture facilities in the absence of other disease causing agents. Usually algal blooms will result in disease or death of a wide range of species of fish and shellfish, rather than just one particular species. Some toxic algae produce distinctive lesions in the gills and internal organs of fish and shellfish which are detectable using histology (Chang et al. 1990, Brusle 1995).

Treatment and prevention: Algal blooms require suitable environmental conditions of temperature, rainfall, and a source of nutrients. Aquaculturists have little control over water temperature and rainfall, but may be able to limit nutrient loading in semi-enclosed systems such as bays and estuaries by minimising wastage of food and using moderate stocking densities. Avoidance through maintaining an ability to move stock away from blooms (e.g., moving racks of shellfish, towing sea cages) is one way of reducing the impact of blooms once they develop.

Distribution in New Zealand

Disease caused by algal blooms can occur wherever marine fish and shellfish are cultured.



Worldwide distribution: Algal blooms are becoming increasingly problematic in marine aquaculture worldwide (Hallegraeff 1993, Brusle 1995, Patterson 2000).

General comments: Disease involves interactions between organism, pathogen, and environment. Algal blooms are an example of environmental events which can cause disease and death of cultured fish and shellfish in the absence of other pathogenic organisms. The effects of algal blooms can range from catastrophic fish mortality (such as the chinook salmon kill in Big Glory Bay, Stewart Island, in 1989 associated with a bloom of *Heterosigma* cf. *akashiwo* (see Chang et al. 1990)), to the equally important issues of food safety, public health, product marketability, and public acceptance of the aquaculture industry.

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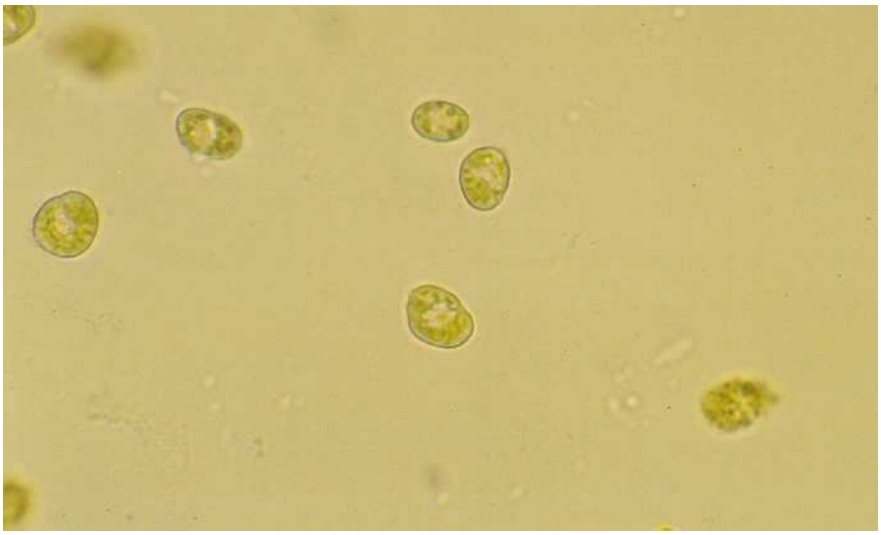
Algal blooms in sea cage culture. Photos by N. Boustead.

Above: Dead chinook salmon, *Oncorhynchus tshawytscha*, floating at the surface of a sea cage after a bloom of the dinoflagellate *Heterosigma* cf. *akashiwo*. **Below:** Live *Heterosigma* in a wet preparation.



Disease: Gas bubble disease

Species and life stage affected: All life stages of all species of marine fish, including snapper (*Pagrus auratus*), kingfish (*Seriola lalandi*), and seahorses (*Hippocampus abdominalis*). Also freshwater fish and crustaceans, including lobsters *(Jasus edwardsii*).

Gross signs: Unusual behaviour, gas bubbles evident in fins, eyes, gills, lateral line and other tissues, exophthalmia.

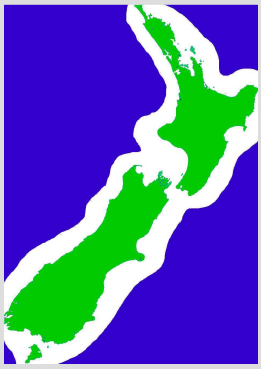
Causative agent: Oxygen supersaturation of the water.

Diagnosis: Diagnosis is through visualisation of gas bubbles throughout various tissues.

Treatment and prevention: Remove source of oxygen supersaturation (e.g., broken or cavitating pumps). Also, aerate cold water which has been heated before use to minimise the chances of supersaturation (cold water carries more oxygen than does warm water). Bubbles may be reabsorbed if fish are pressurised to simulate deeper water (Elston et al. 1997).

Distribution in New Zealand

Gas bubble disease is a husbandry related disease which can occur wherever marine fish and shellfish are cultured.



Worldwide distribution: Gas bubble disease has been recorded worldwide in fish and shellfish.

General comments: Fish can often tolerate quite high levels of supersaturation for short periods of time (minutes, hours), but disease and mortalities can occur if chronic exposure to even very low levels of supersaturation is allowed. There may be an extreme amount of variation in susceptibility to gas bubble disease, both within a given population of fish (Mesa et al. 2000) and between fish species. Seahorses appear to be particularly susceptible to a type of gas bubble disease when allowed to ingest air bubbles in heavily aerated aquaria (Woods 2000). Fish in the wild may also be exposed to human-induced supersaturated water discharged from dams and reservoirs, and photosynthetic activity of aquatic plants can be a natural source of supersaturation (Schisler 2000). See also p. 60 for this disease in freshwater fishes.

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Gas bubble disease in a juvenile snapper (*Pagrus auratus*). Photo by B. Diggles.

Note unilateral exophthalmia of the right eye with numerous gas bubbles evident (arrow). This fish was euthanased by a cut through the backbone.

Disease: Neoplasia (tumors)

Species and life stage affected: Neoplasms can occur in all species of marine and freshwater fish. In New Zealand, neoplasms have been observed in cultured snapper (*Pagrus auratus*), ornamental fish, and many wild fish.

Gross signs: Variable. Sometimes abnormal growths are visible externally. Other neoplasms may not be externally apparent, but may be associated with abnormal behaviour.

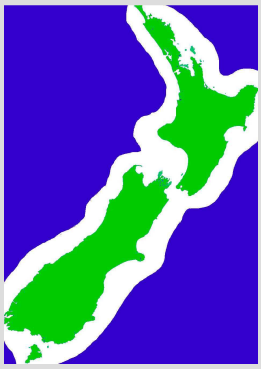
Causative agents: Some neoplasms are associated with viral infections and many are associated with environmental pollution or contamination of hatchery water supplies with substances such as chlorine (Meyers & Hendricks 1984). Some may have a genetic basis (Masahito et al. 1985).

Diagnosis: Presumptive diagnosis can be obtained when abnormal growths are grossly visible, but in all cases confirmation of neoplasm and classification of the type of neoplasm requires histology and perhaps other techniques such as immunocytochemistry.

Treatment and prevention: Usually unknown. If a high prevalence of neoplasia exists in fingerlings, suspect pollution of hatchery water or inbreeding depression in broodstock. Separation of affected fish from healthy fish is advised in case a viral aetiology is implicated.

Distribution in New Zealand

Tumours have been observed in fish throughout New Zealand.



Worldwide distribution: Neoplastic growths have been recorded throughout the world in both cultured and wild marine and freshwater fish.

General comments: Most neoplasms are non infectious, occur at very low prevalences, and hence do not cause major problems in aquaculture. However, increased prevalence of neoplasms in an aquaculture facility may indicate a predisposing virus is present, sub-optimal conditions of water quality, or genetic problems associated with inbreeding of broodstock (Hessler et al. 1997). A list of neoplasms recorded from New Zealand fish up until 1980 was produced by Boustead (1982). Most of these were from wild fish.

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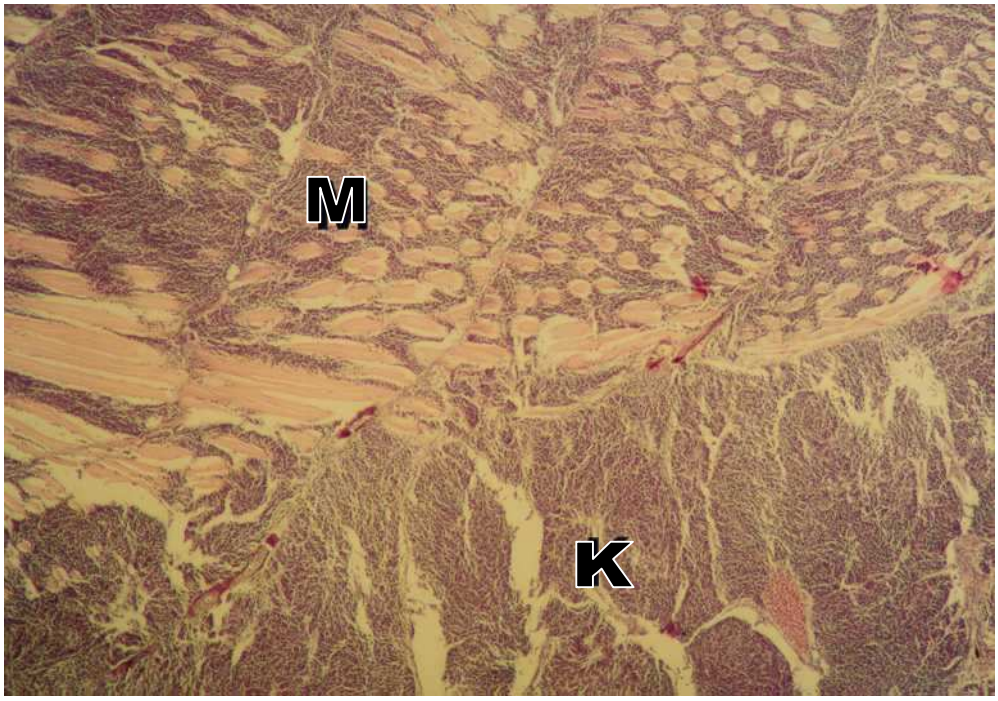
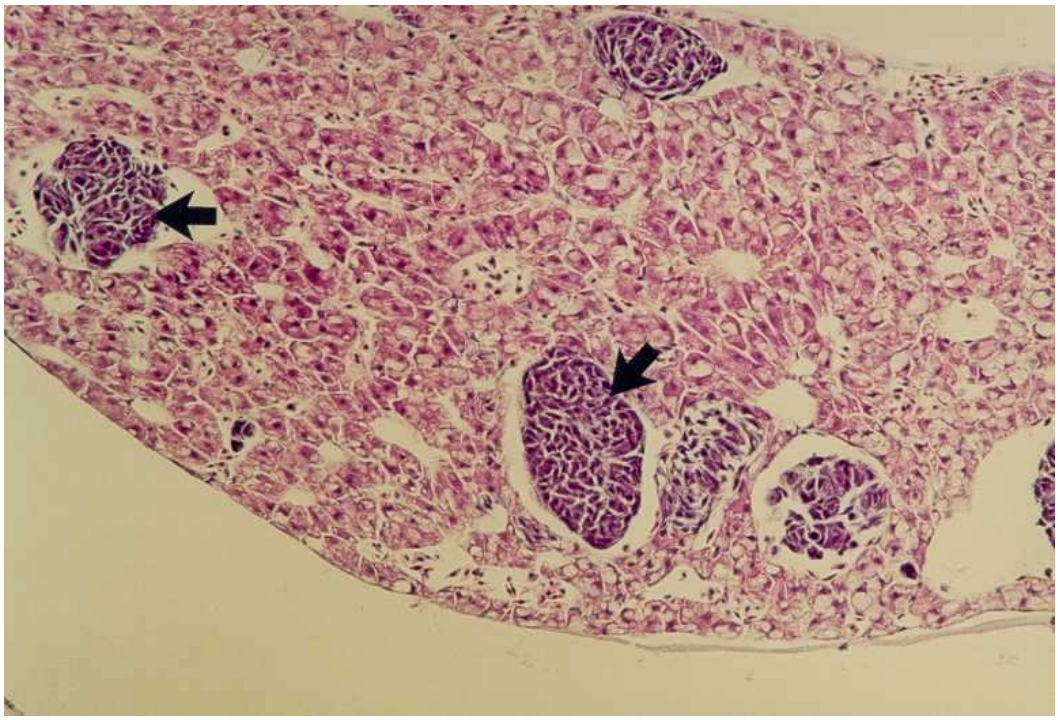


Neoplasia in marine fish: (Registry of Tumors of Lower Animals, Accession no. 6872). Photos by B. Diggles.

Above: Muscle (M) and kidney (K) of a moribund juvenile snapper which displayed massive swelling of the muscles and abdomen and protruding scales. The kidney and most of the muscle has been obliterated by an aggressive primitive neuroectodermal tumor with features of medulloblastoma.

Below: Liver of the same fish showing numerous metastatic tumours (arrows) invading the organ.



Diseases of unknown aetiology of marine fishes

Disease: Kidney cysts in snapper

Species and life stage affected: Juvenile and adult snapper (*Pagrus auratus*).

Gross signs: No behavioural signs recorded. Affected fish appear healthy and do not lose condition. Upon filleting or dissecting affected fish, numerous (up to 200, but mostly 1–5) white nodules ranging from 2 to 7 mm in diameter are evident in all parts of the kidney.

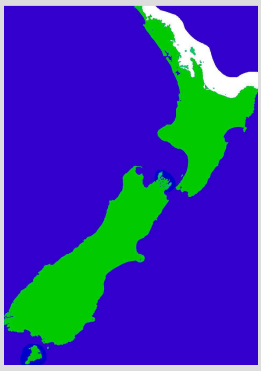
Causative agent: Unknown.

Diagnosis: Presumptive diagnosis can be obtained in instances where the nodules are grossly visible when fish are filleted or dissected. Definitive diagnosis requires histology of the kidney to demonstrate characteristic fibrous nodules with a mucoid degeneration of the centre and various degrees of peripheral infiltration by eosinophills (Hine & Anderson 1981). The nodules may comprise up to 90% of kidney volume in older fish.

Treatment and prevention: Unknown.

Distribution in New Zealand

Kidney cysts have been recorded in wild snapper from a number of locations along the northeast coast of the North Island.



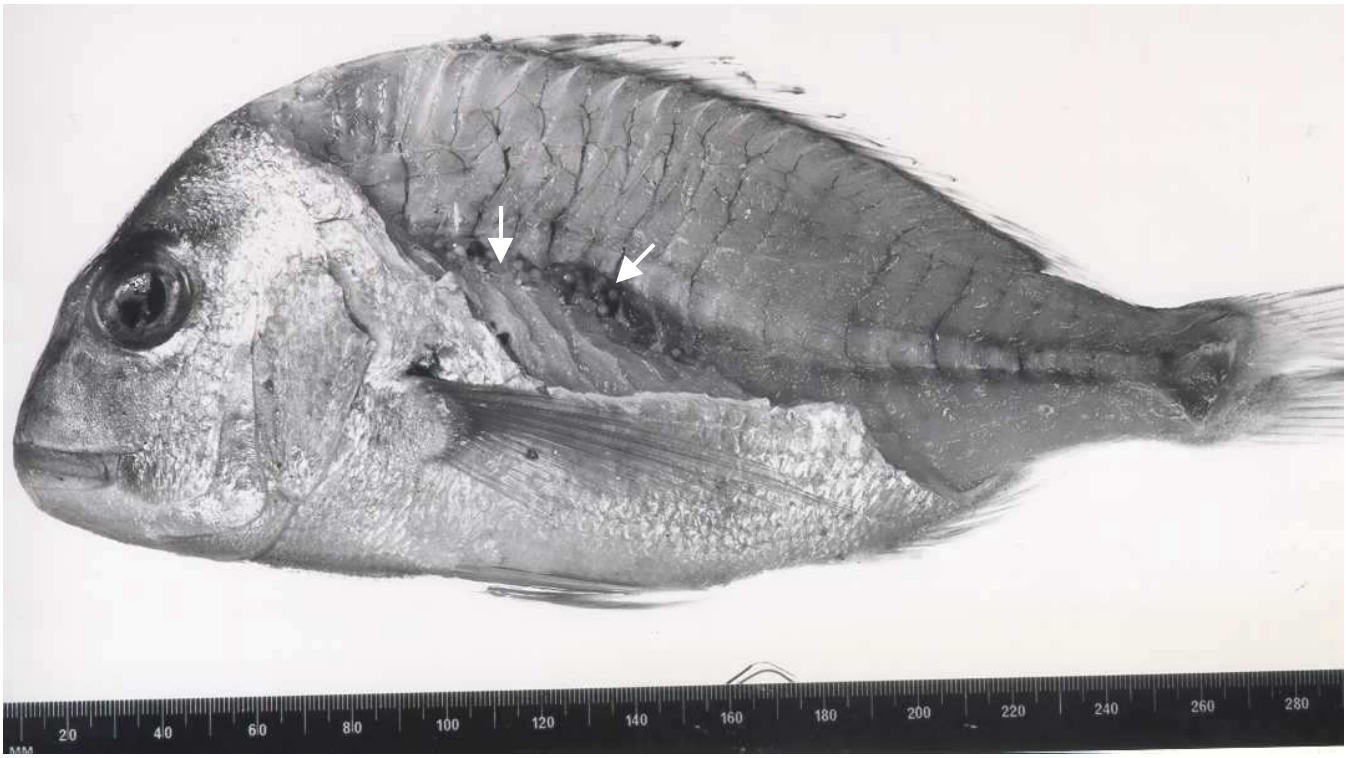
Worldwide distribution: Similar cysts have been recorded in the kidney of another sparid fish, the bream (*Acanthopagrus australis*) from Moreton Bay, Australia (Roubal 1994).

General comments: In small snapper (under 15 cm) the nodules are small (c. 2 mm) and occur at low prevalences (< 10%). Once fish reach 30 cm nearly all fish have at least 1 to 5 nodules in all parts of the kidney, and rarely in the spleen. Both sexes are equally affected (Hine & Anderson 1981). Though no signs of disease have been recorded in affected fish, heavy nodule infections must impair kidney function and hence could predispose fish to opportunistic pathogens.

References

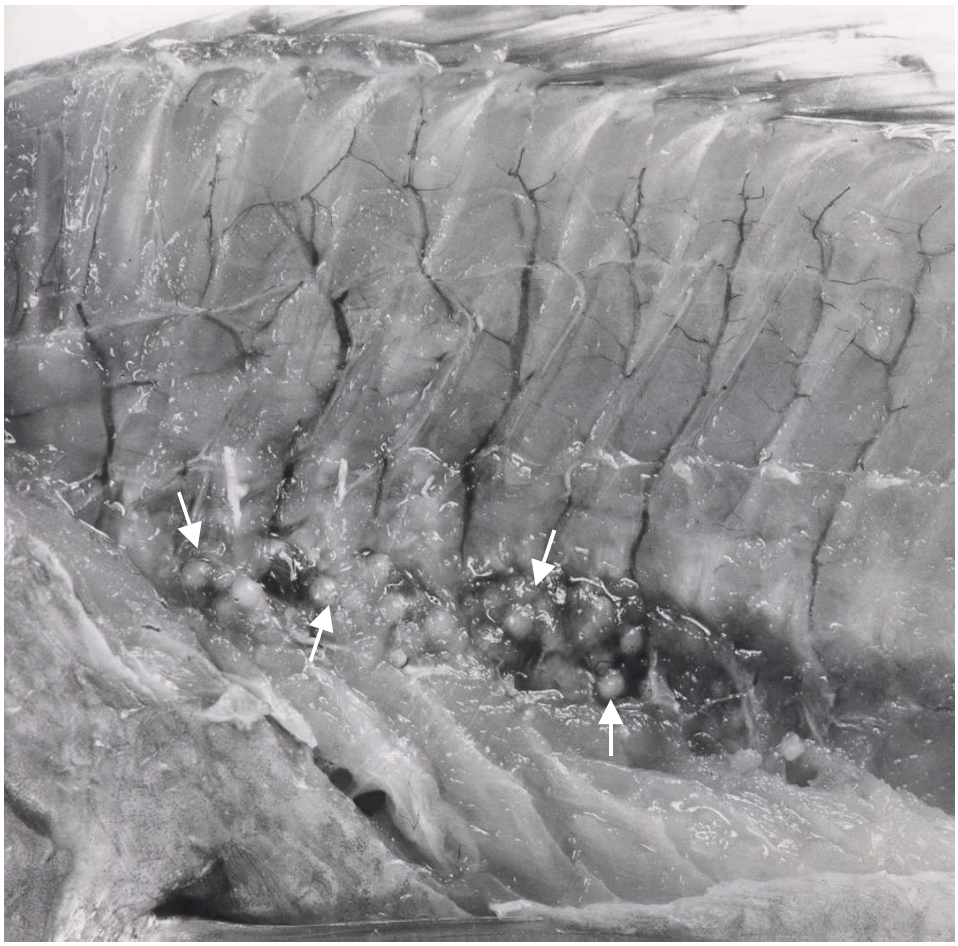
Hine, P.M.; Anderson, C.D. (1981). Diseases of the gonads and kidneys of New Zealand snapper, *Chrysophrys auratus* Forster (F. Sparidae). *In*: Fowler M.E. (ed.) Wildlife diseases of the Pacific Basin and other countries, pp. 166–170. Proceedings of the 4th International Conference of the Wildlife Diseases Association, Sydney, 25–28 August 1981.

Roubal, F.R. (1994). Infection of the kidney of *Acanthopagrus australis* (Pisces: Sparidae) with *Sphaerospora* sp. (Myxosporea), *Prosorhynchus* sp. (Digenea), and cysts of unknown origin. *Diseases of Aquatic Organisms 20*: 83–93.



Kidney cysts in snapper (*Pagrus auratus*). Photos by M. Hine.

Above: A 28 cm snapper with multiple nodules evident in the kidney (arrows).
Below: Closeup of the kidney showing many white nodules (arrows).



Diseases of Crustaceans

CRUSTACEANS

Viral diseases of crustaceans

Disease: White spot disease (WSD) (white spot baculovirus, penaeid rod shaped DNA virus, systemic ectodermal and mesodermal baculovirus, and other names)

Species and life stage affected: A wide range of crustaceans, particularly penaeid prawns, but also crabs, spiny lobsters, copepods, and other arthropods.

Gross signs: Cessation of feeding, formation of white spots (0.5–2 mm diameter) under the cuticle, loose shells followed by a rapid increase in mortality (up to 100% within 5–10 days).

Causative agents: White spot syndrome virus (WSSV) complex, a closely related group of double stranded DNA baculo-like viruses.

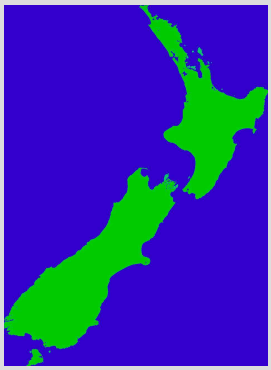
Diagnosis: Presumptive diagnosis is sometimes attempted upon viewing white spots on the cuticle, but this is unreliable as similar lesions can occur from infection by other disease agents (e.g., Wang et al. 2000), and some species do not display lesions (Rajendran et al. 1999). A minimum of histopathology is required to demonstrate the presence of characteristic basophilic inclusion bodies in the nuclei of tissues of ectodermal and mesodermal origin. Definitive diagnosis requires a combination of one or more of the following methods; PCR, ISH, western blot analysis, or TEM (Lightner 1999, OIE 2000).

Treatment and prevention: No treatment available. Once WSD is confirmed in an aquaculture facility, slaughter followed by disinfection and drying out are required. WSSV can be deactivated using UV irradiation (9×10^5 µW/ml), heat treatment (55 °C for 90 min, 70 °C for 5 min), acidity (pH 3 for 1 h, alkalinity (pH 12 for 10 min), ozone (0.5 µg/ml for 10 min) and bleach and povidone iodine (100 ppm for 10 min) (Chang et al. 1998). Avoidance is through exclusion of wild crustaceans which are potential reservoirs of infection, PCR testing of broodstock, and development of resistant stocks. The virus survives freezing and can be introduced via imports of frozen prawns and crabs originating from areas where the disease occurs.

Distribution in New Zealand

Unreported, but as WSSV infects a wide range of crustaceans, it may be a potential threat to culture of crustaceans in New Zealand.

WSD IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: WSD was first reported in farmed *Penaeus japonicus* in Japan in 1993, and has since been spread throughout China and to other prawn farming areas in Asia, India, Europe, the Middle East, the United States and South America.

General comments: WSSV has naturally or experimentally infected over 50 species of crustaceans, including most species of penaeid prawns (Lightner 1999), several genera of crabs, spiny lobsters (genus *Panulirus*) and freshwater prawns (*Macrobrachium* sp.) (Rajendran et al. 1999), and freshwater crayfish. Many of these species can act as asymptomatic reservoirs of infection. Total losses of 100% of stock and 80% of total production are common in countries where penaeid culture has been newly affected by WSD (Gillespie 2000, Nair 2000). Total financial losses to aquaculturists worldwide attributed to WSD between 1993 and 2000 are thought to approach US$ 4 to 6 billion (Lightner 1999).

References

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Gillespie, D. (2000). Peru turns to tilapia as disease devastates shrimp. *Fish Farming International 27(2)*: 4.

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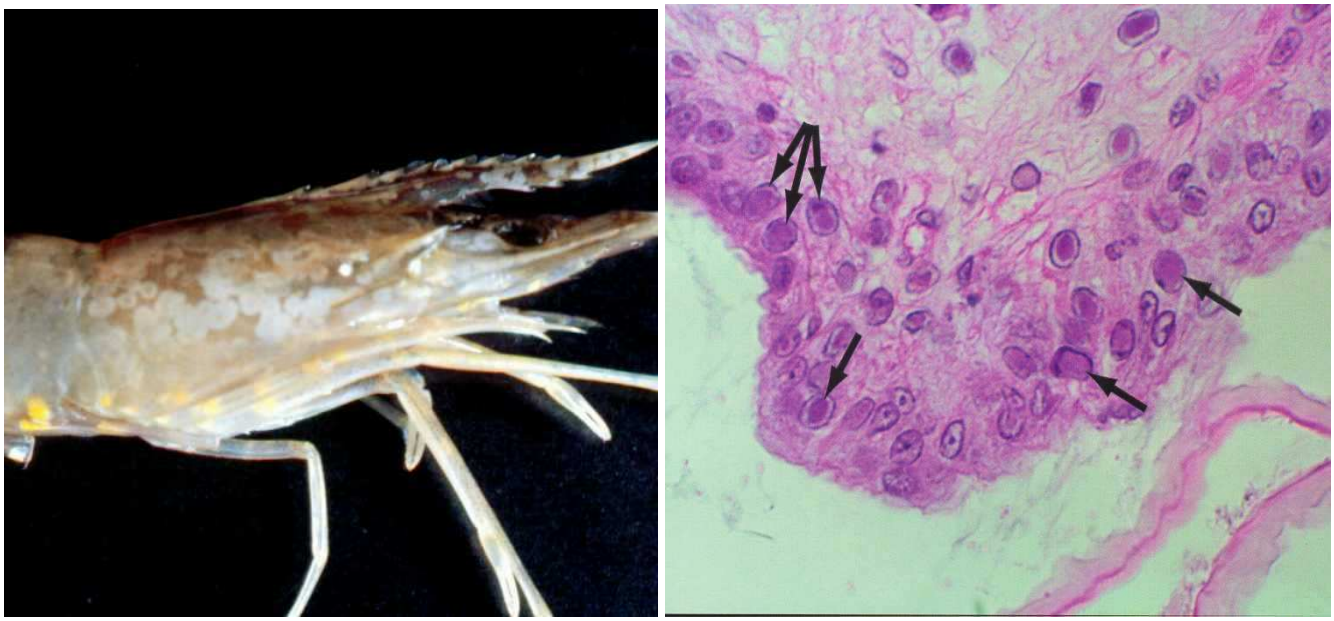
Wang, Y.G.; Lee, K.L.; Najiah, M.; Shariff, M.; Hassan, M.D. (2000). A new bacterial white spot syndrome (BWSS) in cultured tiger shrimp *Penaeus monodon* and its comparison with white spot syndrome (WSS) caused by virus. *Diseases of Aquatic Organisms 41*: 9–18.



White spot disease in cultured prawns. Photos by D. Lightner.

Left: Gross appearance of WSSV infected *Penaeus monodon* exhibiting classical WSD lesions on the carapace.

Right: Histological section through the stomach of a penaeid prawn showing the presence of WSSV intranuclear inclusion bodies (arrows) within cuticular epithelium.

Microbial diseases of crustaceans

Disease: Bacterial enteritis

Species and life stage affected: Larval, juvenile, and adult red rock lobsters (*Jasus edwardsii*).

Gross signs: No specific gross signs. Lobsters with advanced infections may stop feeding and become lethargic.

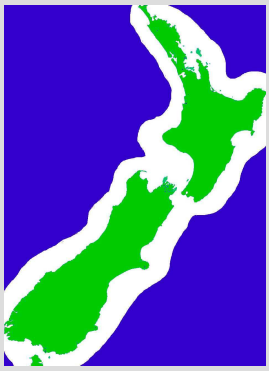
Causative agents: Probably numerous types of opportunistic marine bacteria, including *Vibrio harveyi*.

Diagnosis: Bacterial erosion of the hepatopancreas tubules, usually without septicemia, detected by histopathology. Complete erosion of the epithelium may elicit a host reaction. If culture of implicated bacteria is attempted, discriminating causative agents from the bacterial flora normally present in the hepatopancreas is difficult.

Treatment and prevention: Bacterial diseases usually result from injury or sub-optimal culture conditions, so improvement of water quality, food quality, or rearing system design may be required to improve the health of stock and prevent reoccurrence of disease. Antibiotic treatment may be possible if the bacteria involved can be identified and their antibiotic sensitivities can be determined, though problems with development of resistant strains of bacteria may occur with prolonged antibiotic use.

Distribution in New Zealand

Observed in lobsters held throughout the country. Possibly a problem wherever lobsters are cultured intensively under sub-optimal conditions.

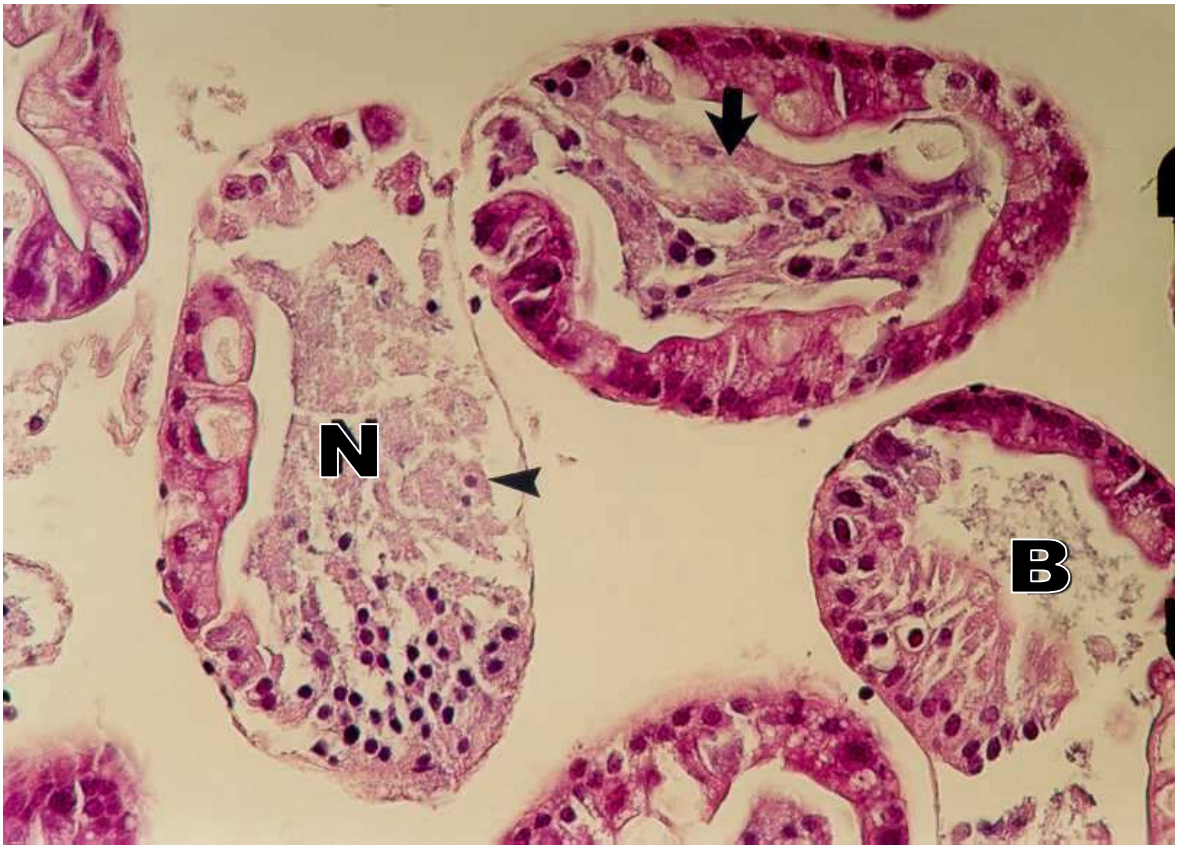


Worldwide distribution: Has also been recorded in *J. edwardsii* from Australia (Handlinger et al. 2000).

General comments: Bacterial enteritis is usually recorded in heavily fouled and/or moribund lobsters and may be related to cessation of feeding.

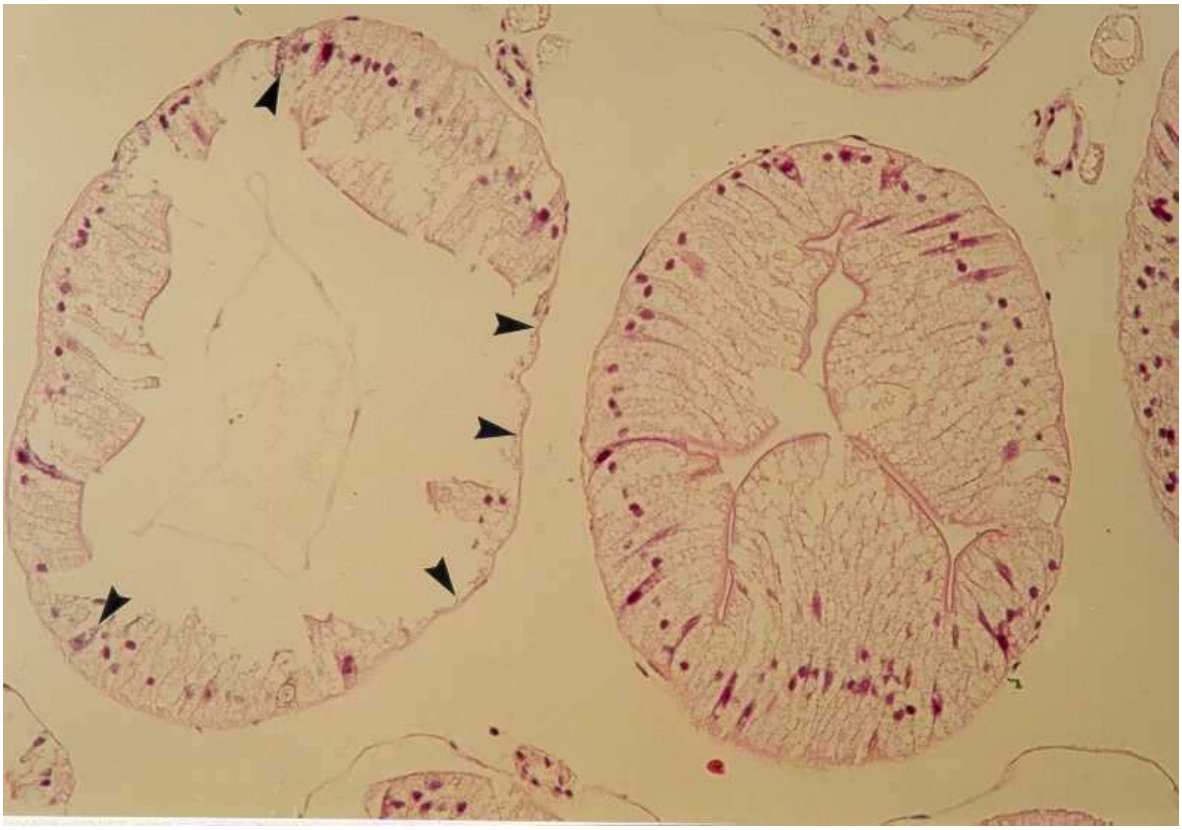
References

Handlinger, J.; Carson, J.; Ritar, A.; Crear, B., Taylor, D.; Johnson, D. (2000). Disease conditions of cultured phyllosoma larvae and juveniles of southern rock lobster (*Jasus edwardsii*). *In*: Evans, L.H.; Jones, J.B. (eds) Proceedings of the International Symposium on Lobster Health Management, pp. 75–87. Adelaide (Australia), 19–22 September 1999.



Histology of bacterial enteritis in *Jasus edwardsii*. Photos by J. Handlinger.

Above: Note generalised necrosis of hepatopancreocytes (N, arrowhead) in one hepatopancreas tubule, and bacteria (B, arrow)) in the lumen of other tubules of a juvenile lobster. **Below:** Areas of focal erosion of hepatopancreas epithelium (arrowheads) associated with bacteria in an adult lobster.



Disease: Crayfish plague (krebspest, kraftpest, crayfish aphanomyciasis)

Species and life stage affected: Most freshwater crayfish of the Families Astacidae, Cambaridae, and Parastacidae, but particularly European crayfish (*Astacus astacus*, *A. leptodactylus*, *Austropotamobius pallipes*). Most crayfish native to North America, including the signal crayfish (*Pacifastacus leniusculus*) and crawfish (*Procambarus* sp., *Orconectes* sp.), are largely resistant and can be asymptomatic carriers of the disease (Dieguez-Uribeondo & Soderhall 1993).

Gross signs: Infected crayfish show whitened necrotic musculature in the tail, often accompanied in chronic infections by melanisation (blackening) of affected exoskeleton. Most mortalities tend to occur in summer, but disease can occur at any time of the year. In European crayfish, mortality is almost always 100% and no evidence of resistance has been observed in over 100 years.

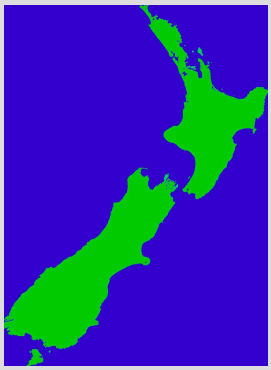
Causative agent: The oomycete fungus *Aphanomyces astaci*.

Diagnosis: Presumptive identification of the causative organism can be made from a fresh microscopic mount of a piece of infected exoskeleton to demonstrate the presence of delicate, branching, aseptate fungi 7–9 µm in diameter. Isolation and culture of *A. astaci* are required for absolute confirmation.

Treatment and prevention: *Aphanomyces astaci* has been controlled in the laboratory by baths of 0.5 mg/L malachite green or 100 mg/L hydrogen peroxide for 1 hour, and is killed in 5 minutes by bleach (100 mg/L active chlorine) (Lilley & Inglis 1997). Once natural waters are infected, however, further spread of infection is inevitable. In crayfish farms total destruction of all stock and disinfection may eliminate the pathogen. Crayfish plague is principally transmitted on infected crayfish, both susceptible and resistant. Prevention is by exclusion of crayfish sourced from areas where crayfish plague occurs. Contaminated fishing gear, boots, and equipment can also carry infection. Fish, birds, and other wildlife may act as vectors during large outbreaks.

Distribution in New Zealand

Unreported. *A. astaci* infects a wide range of freshwater crayfish, hence is a potential threat to koura in New Zealand.



CRAYFISH PLAGUE IS AN INTERNATIONALLY SIGNIFICANT DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: *Aphanomyces astaci* almost certainly originated from North America, and was probably translocated to Europe with North American crayfish in the mid 1800s. Between 1870 and 1940, European freshwater crayfish (*Astacus astacus*, *A. leptodactylus*, and *Austropotamobius pallipes*) were almost completely destroyed, mostly surviving only in small isolated water systems above 600 m elevation. Between 1960 and 1990, native crayfish populations in Spain, Britain, Norway, and Greece were also largely destroyed by crayfish plague (Alderman et al. 1990).

General comments: North American crayfish, particularly the signal crayfish, are largely resistant to *A. astaci* and therefore can act as carriers. However, in poor environmental conditions even signal crayfish can be killed by crayfish plague. Laboratory tests have shown freshwater crayfish native to Australia and Papua New Guinea are susceptible to crayfish plague (Unestam 1975), suggesting that New Zealand's native koura (*Paranephrops planifrons*, *P. zealandicus*) are probably also susceptible.

References

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Dieguez-Uribeondo, J.; Soderhall, K. (1993). *Procambarus clarkii* Giard as a vector for the crayfish plague fungus, *Aphanomyces astaci* Schikora. *Aquaculture and Fisheries Management 24*: 761–765.

Lilley, J.H.; Inglis, V. (1997). Comparative effects of various antibiotics, fungicides and disinfectants on *Aphanomyces invaderis* and other saprolegniaceous fungi. *Aquaculture Research 28*: 461–469.

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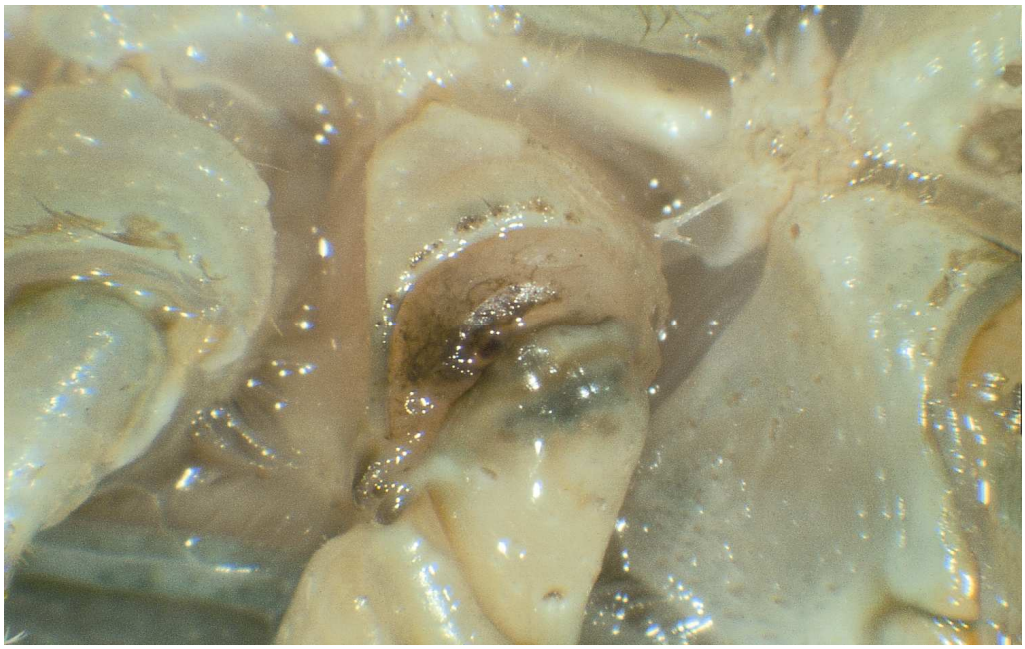
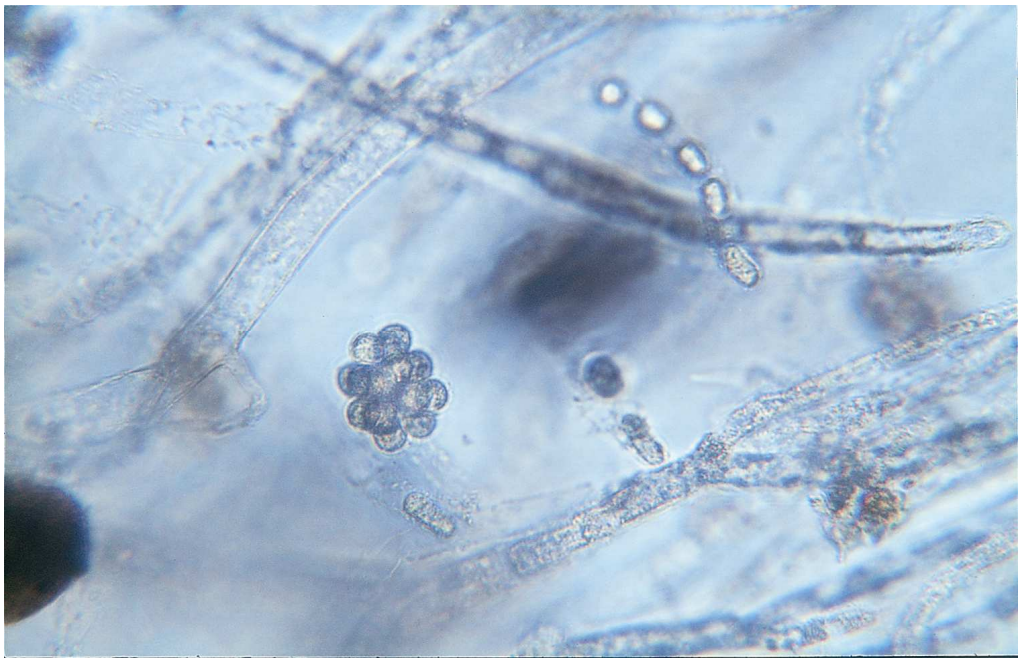


Crayfish plague in European crayfish. Photos by D. Alderman.

Above: Focal melanisation at the base of a walking leg. **Below:** Hyphae and spores of *Aphanomyces astaci*.



Disease: Epibiont fouling

Species and life stage affected: Phyllosoma larvae, puerulus larvae, and juveniles of red rock lobsters (*Jasus edwardsii*) and packhorse lobsters (*J. verreauxi*). Adult paddle crabs (*Ovalipes catharus*).

Gross signs: Fouling of external surfaces, especially the carapace and gills, with microbial epibionts and organic detritus, lethargic behaviour, low to moderate mortalities, especially during the moult.

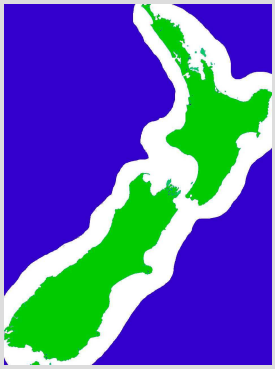
Causative agents: Various types of filamentous *Leucothrix*-like bacteria, sessile ciliates (*Carchesium* sp., *Epistylis* sp., *Zoothamnium* sp.), free living nematodes.

Diagnosis: Mixed growths of filamentous *Leucothrix*-like bacteria, rod shaped and gliding bacteria, sessile stalked ciliates, and sometimes free living nematodes and detritus are evident when whole larvae or affected areas of gills of juveniles are examined using wet squashes or histology. Heavy fouling may be associated with gill necrosis in juvenile lobsters and adult paddle crabs.

Treatment and prevention: Increasing water flow and aeration can reduce fouling problems. Chemical baths with formalin, chelated copper compounds, and algicides may also reduce epibiont loading. Prevention is via increased water flow and improved system hygiene. High temperatures (over 20 °C) increase the need for adequate water flow. Decontamination of *Artemia* used as live food for phyllosoma larvae is important to prevent *Artemia* acting as vectors for fouling organisms.

Distribution in New Zealand

Observed in samples taken from throughout the country, but particularly in the North Island where water temperatures are higher. Possibly a problem wherever lobsters and crabs are cultured intensively under sub-optimal conditions.



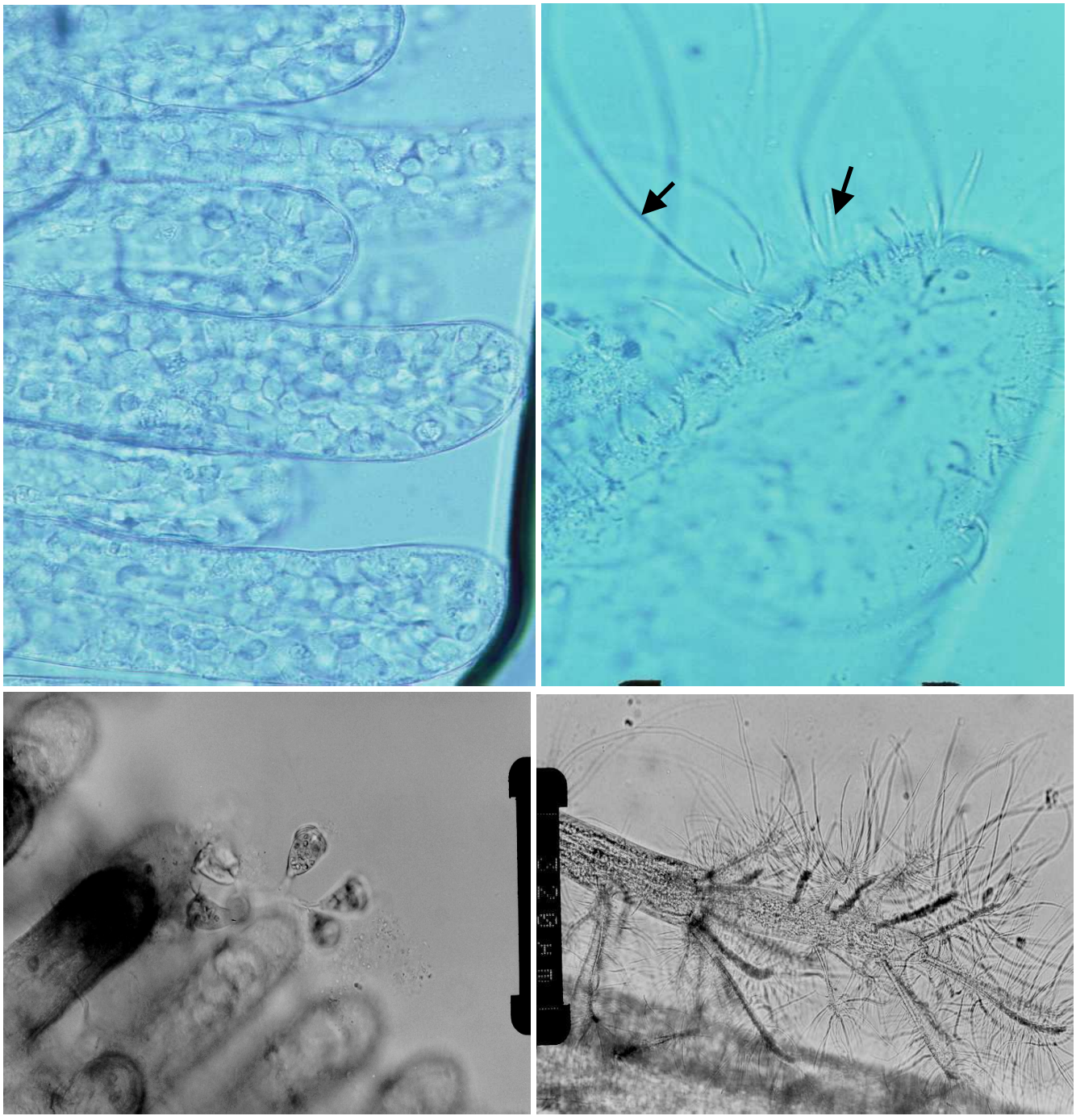
Worldwide distribution: Epibiont fouling is a ubiquitous problem in the intensive culture of crustaceans worldwide, including homarid lobsters (Fisher et al. 1978) and penaeid prawns (Lightner, 1983).

General comments: Mortalities of juvenile lobsters in rearing systems using recirculated seawater can be associated with moderate to heavy growths of epibionts. Affected animals are sluggish and exhibit brown coloration in the gills. Most deaths occur at night just before or during the moult, probably because oxygen demand increases at night, when moulting usually occurs, and the heavy epibiont growth reduces respiratory effectiveness. Epibionts appear to be gradually accumulated over time. Their presence indicates poor system hygiene or poor water flow. Heavy epibiont growth on phyllosoma larvae is common in upwelling systems using a high percentage of recirculated water.

References

Fisher, W.S.; Nilson, E.H.; Steenbergen, J.F.; Lightner, D.V. (1978). Microbial diseases of cultured lobsters: a review. *Aquaculture 14*: 115–140.

Lightner, D.V. (1983). Diseases of cultured shrimp. *In*: McVey, P.V. (ed). CRC handbook of mariculture, Vol. I. Crustacean aquaculture, pp. 289–320. CRC Press, Boca Raton.



Epibiont infestations in *Jasus* spp.. Photos by B. Diggles.

Above left: Wet preparation of normal gills of a juvenile *J. edwardsii*. **Above right:** Wet preparation of lobster gills with a moderate infestation of a *Leucothrix*-like filamentous bacteria (arrows). **Below left:** Sessile ciliate (*Carchesium* sp.) attached to gill of a juvenile lobster. **Below right:** Wet squash of the leg of a phyllosoma larva of *J. verreauxi* showing heavy fouling by a *Leucothrix*-like filamentous bacteria.

Disease: Gill mycosis

Species and life stage affected: Red rock lobster (*Jasus edwardsii*) puerulus larvae and juveniles up to 30 mm carapace length.

Gross signs: Brown/black lesions at the base of the gills near insertion of the walking legs, lethargic behaviour, low to moderate mortalities, especially during the moult.

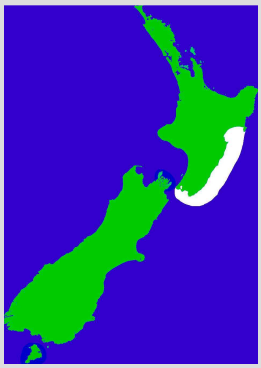
Causative agent: An invasive fungus, *Haliphthoros* sp.

Diagnosis: Fungal mycelia are easily observed under the microscope in wet preparations of the gill filaments adjacent to the blackened areas, or by using routine histopathological methods. Specific identification of the causative fungus can be obtained by inoculating infected gill filaments into marine agar 2216 (Difco) containing antibiotics to culture the fungus. Identification can then be performed using morphological and/or molecular techniques.

Treatment and prevention: The fungal spores are susceptible to a variety of chemical treatments, including trifuralin, formalin, and malachite green (Diggles 2001), but the disease can be prevented by improving husbandry such as removal of uneaten food and regular cleaning of detritus from tanks.

Distribution in New Zealand

Has been observed in onshore experimental grow-out facilities on the southeast coast of the North Island. Not known from wild lobsters.



Worldwide distribution: Invasive mycoses caused by the fungus *Haliphthoros milfordensis* have been recorded in homarid lobsters in the Northern Hemisphere (Fisher et al. 1975, Fisher & Nilson, 1977).

General comments: This disease has been associated with significant mortalities in puerulus and juvenile *J. edwardsii* held for ongrowing at water temperatures between 10 and 18 °C. Up to 30–50% of animals were affected in poorly maintained systems. The disease generally does not affect lobsters greater than 30 mm carapace length. Death of affected lobsters usually occurs just before or during the moult, perhaps from restriction of moulting due to extensive melanisation (Fisher et al. 1975, Fisher & Nilson 1977), though secondary bacterial infection may also be implicated in some cases.

References

Diggles, B.K. (2001). A mycosis of juvenile spiny rock lobster *Jasus edwardsii* (Hutton, 1875) caused by *Haliphthoros* sp., and possible methods of chemical control. *Journal of Fish Diseases 24*: 99–110.

Fisher, W.S.; Nilson, E.H.; Shleser, R.A. (1975) Effect of the fungus *Haliphthoros milfordensis* on the juvenile stages of the American lobster *Homarus americanus*. *Journal of Invertebrate Pathology 26*: 41–45.

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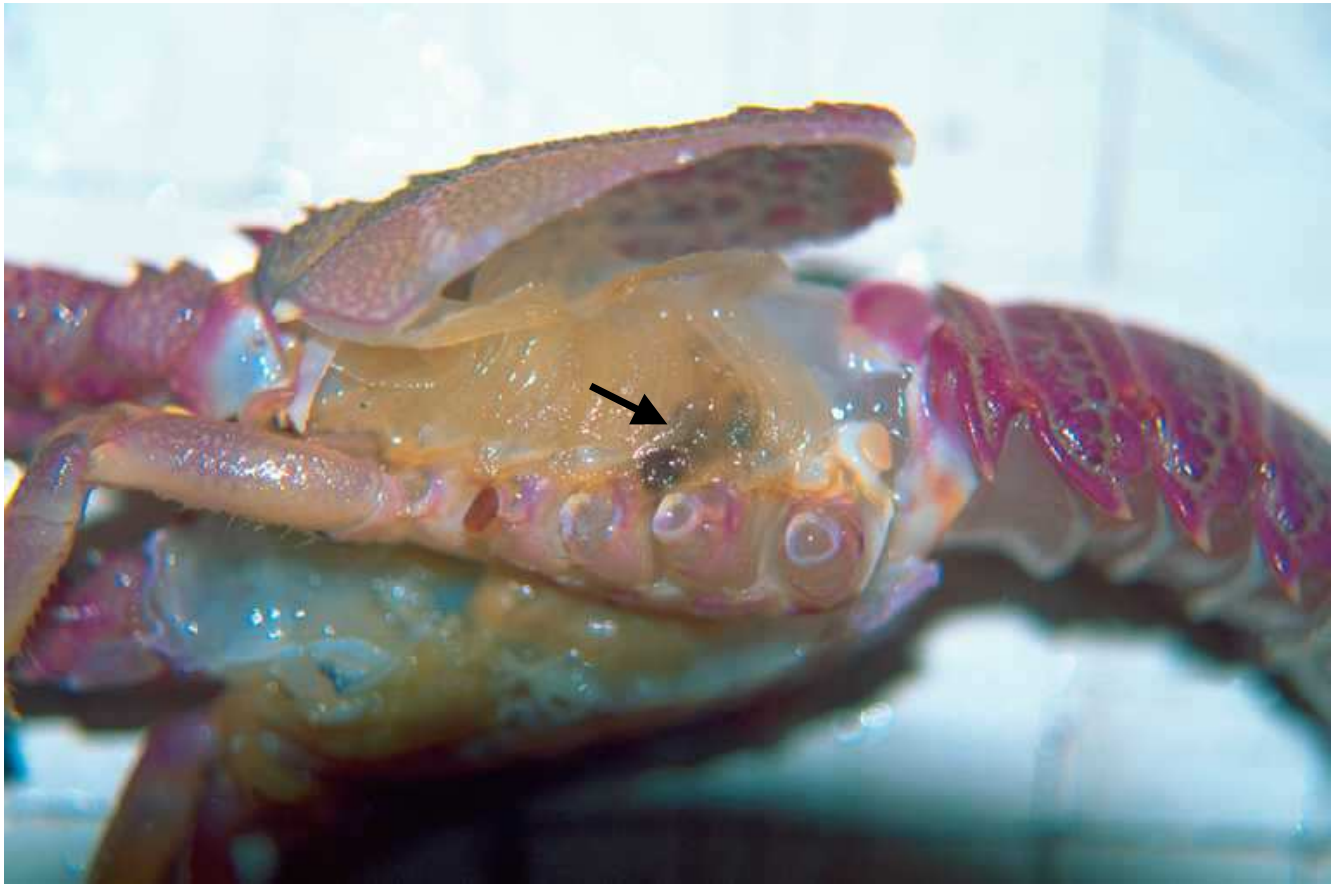
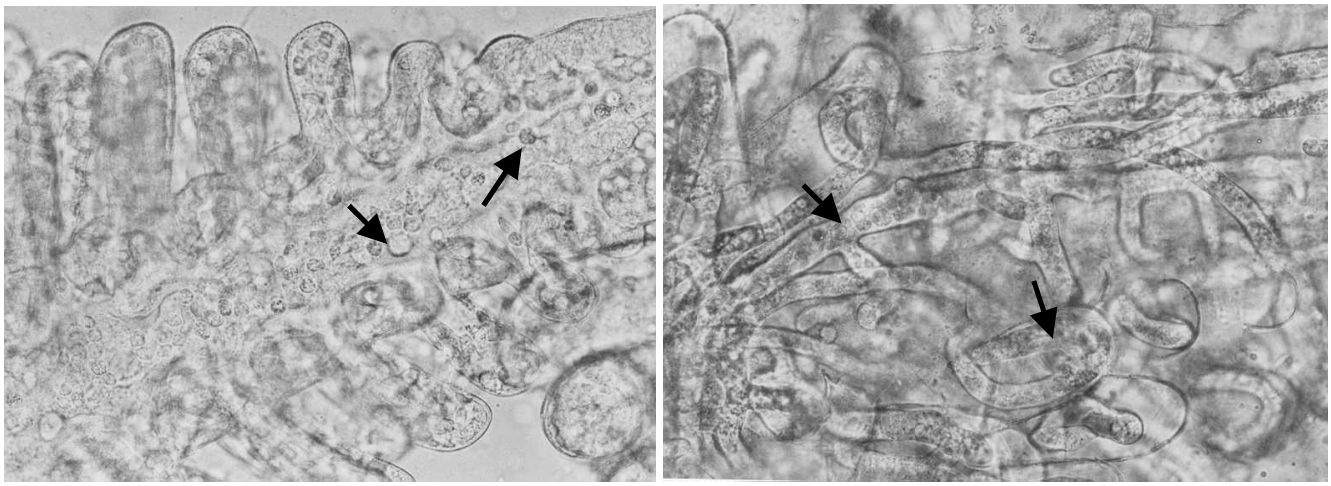


Gill mycosis in juvenile *Jasus edwardsii*. Photos by B. Diggles.

Above: A black/brown lesion in the gills (arrow) at a site of fungal infection. Melanisation is particularly apparent at the base of the walking leg. **Below left:** Wet squash of normal gill, containing haemocytes (arrows). **Below right:** Wet squash of gill from a gill lesion showing fungal hyphae (arrows) inside the gill cuticle.



Disease: Luminous vibriosis

Species and life stage affected: Phyllosoma larvae of packhorse rock lobster (*Jasus verreauxi*).

Gross signs: Larvae are luminous when viewed in the dark, moderate to heavy mortalities.

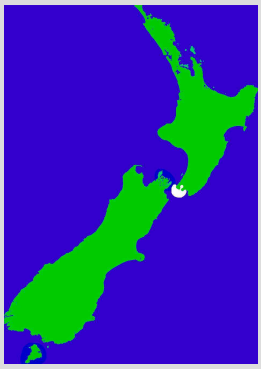
Causative agents: Luminescent marine bacteria, particularly *Vibrio harveyi.*

Diagnosis: Presumptive diagnosis can sometimes be obtained by observing larvae in complete darkness. Those affected by *V. harveyi* can be faintly luminous in the dark. Massive bacterial plaques will be evident in the lumen of hepatopancreas tubules using histopathology. Definitive diagnosis requires culture of luminescent bacteria such as *V. harveyi* from internal organs followed by biochemical or molecular characterisation.

Treatment and prevention: *Vibrio harveyi* may be introduced into larval culture tanks through the water or via live food such as *Artemia*, so thorough decontamination of water, live food, and tank surfaces is recommended to minimise bacterial growth. Any handling that may injure larvae will increase mortality rates. Infection may be transferred by cannibalism, so removal of dead larvae from affected systems is recommended. Treatment with antibiotics may be successful initially, but deformities of larvae may occur and antibiotic resistance is likely to develop over time. Conditioning of culture water and/or live food with probiotic bacteria (Moriarty 1998) are worth investigation as possible methods of prevention.

Distribution in New Zealand

Has been recorded in experimental culture of phyllosoma larvae of *Jasus verreauxi* in Wellington. Possibly a problem wherever phyllosoma larvae are cultured intensively under sub-optimal conditions.



Worldwide distribution: Luminous vibriosis is a persistent problem in rearing of larval and juvenile penaeid prawns worldwide (Lightner 1983, Lavilla-Pitogo et al. 1998). It also occurs in phyllosoma larvae of *J. edwardsii* in Tasmania (B. Crear, personal communication).

General comments: Onset of disease in *J. verreauxi* phyllosoma larvae occurs as early as the 1st instar at water temperatures of 23 °C, and can affect larvae at temperatures down to 16 °C (Diggles et al. 2000). Losses of up to 80% of stock have been attributed to the disease.

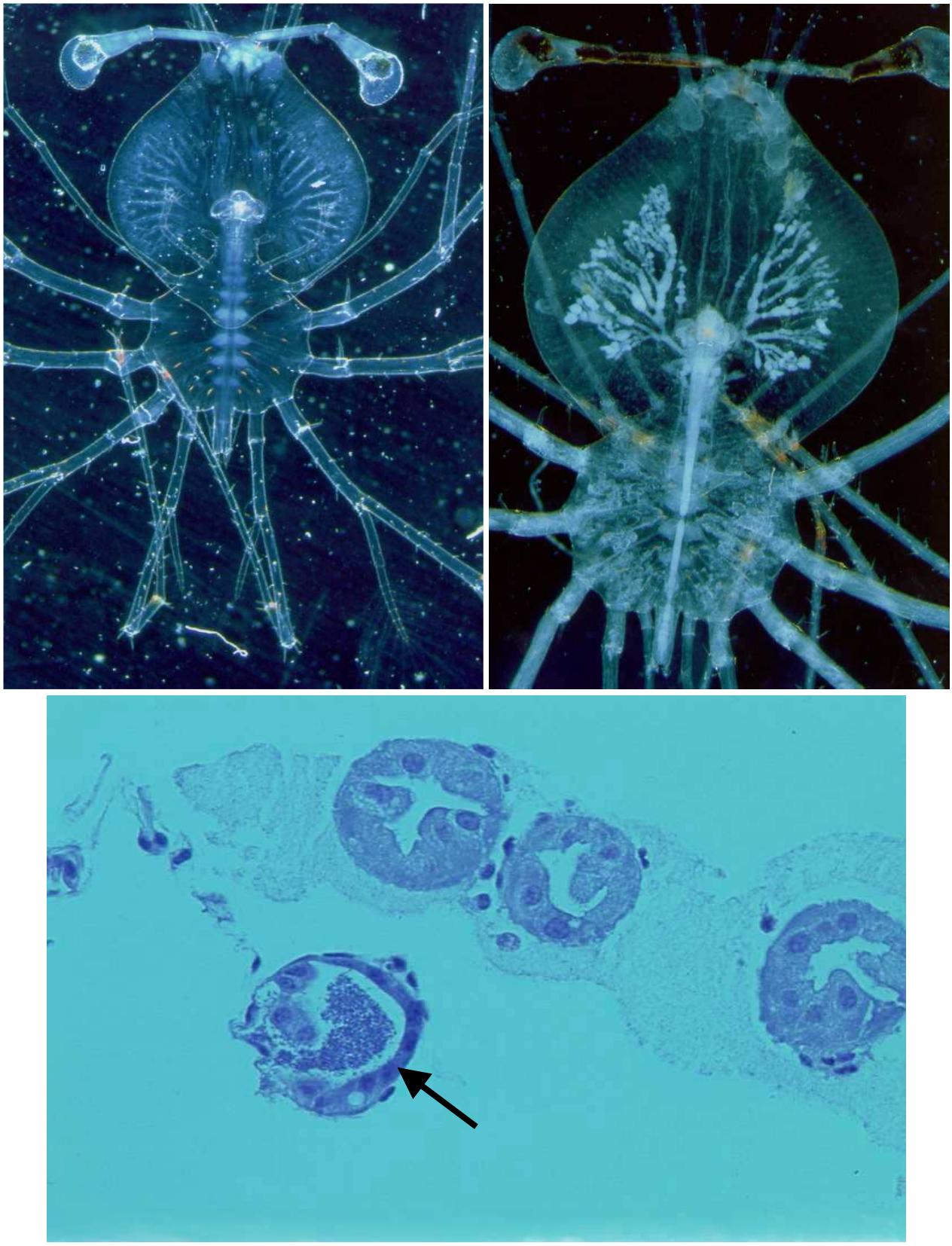
References

Diggles, B.K.; Moss, G.A.; Carson, J.; Anderson, C.D. (2000). Luminous vibriosis in rock lobster *Jasus verreauxi* (Decapoda: Palinuridae) phyllosoma larvae associated with infection by *Vibrio harveyi*. *Diseases of Aquatic Organisms 43*: 127–137.

Lavilla-Pitogo, C.R.; Leano, E.M.; Paner, M.G. (1998). Mortalities of pond-cultured juvenile shrimp, *Penaeus monodon*, associated with dominance of luminescent vibrios in the rearing environment. *Aquaculture 164*: 337–349.

Moriarty, D.W. (1998). Control of luminous *Vibrio* species in penaeid aquaculture ponds. *Aquaculture 164*: 351–358.

Lightner, D.V. (1983). Diseases of cultured shrimp. *In*: McVey, P.V. (ed), CRC handbook of mariculture, Vol. I. Crustacean aquaculture, pp. 289–320. CRC Press, Boca Raton.



Luminous vibriosis of phyllosoma larvae of *Jasus verreauxi*. Photos by B. Diggles.

Above left: Uninfected larvae, note transparent appearance. **Above right:** Fourth instar phyllosoma infected with *Vibrio harveyi*. Note the opaque hepatopancreas and appendages filled with luminescent bacteria, and melanised base of the eye stalks. **Below:** Histopathology of hepatopancreas tubules of a luminous phyllosoma. Note atrophy of the lower tubule (arrow) which contains masses of bacteria.

Disease: Shell disease

Species and life stage affected: Juvenile and adult red rock lobsters (*Jasus edwardsii*), adult packhorse lobsters (*J. verreauxi*), and adult paddle crabs (*Ovalipes catharus*).

Gross signs: Erosion and blackening of the carapace, tail fan, and walking legs. Blister-like lesions on the tail fan may also occur, but their cause may be distinct from classical shell disease.

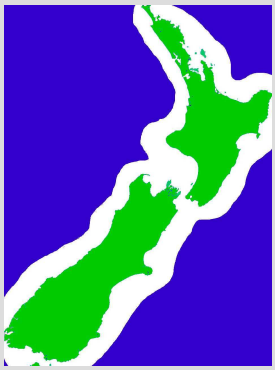
Causative agents: Presumably chitinoclastic bacteria (*Vibrio* sp., *Pseudomonas* sp., *Aeromonas* sp.), and/or unidentified fungi.

Diagnosis: Erosion and/or blackening of affected areas of the cuticle are easily observed with the naked eye. Microscopic examination of the affected areas can be used to attempt to determine whether disease is associated with bacterial or fungal invaders. Both may be visible in wet preparations. Culturing causative agents and demonstrating their involvement is usually difficult due to the high numbers of naturally occurring heterotrophic bacteria present on the carapace.

Treatment and prevention: Occurrence of shell disease in cultured crustaceans indicates that improvement of water quality and/or holding conditions may be required. Elimination of potential sources of injury should assist in reducing the prevalence of shell disease and also tail blisters.

Distribution in New Zealand

Occurs throughout New Zealand.



Worldwide distribution: Shell disease is ubiquitous and occurs in both marine and freshwater crustaceans worldwide (Getchell 1989, Sindermann 1990, Noga et al. 2000). Tail blistering apparently has been reported only from *Jasus edwardsii* in Australia and New Zealand.

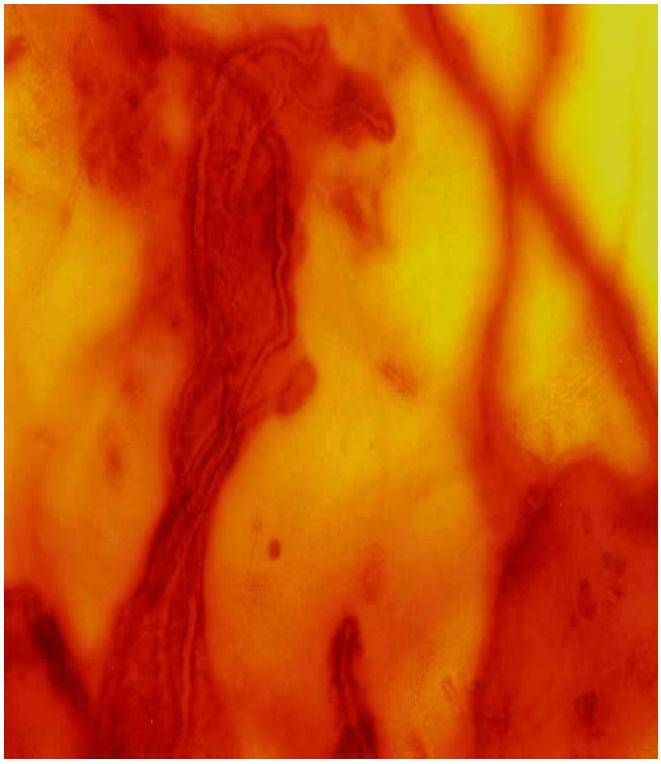
General comments: Shell disease has been recorded in both wild and captive *Jasus edwardsii* and *J. verreauxi*. In these species, shell disease lesions are most obvious on the ventral part of the tail fan and other areas of the carapace in contact with bottom surfaces or subject to injury. Superficial lesions are usually eliminated during moulting. The cause is assumed to be infection of wounds or abrasions by chitinoclastic bacteria. However, fungi are occasionally implicated. Blistering of the tail fan may be a syndrome distinct from classical shell disease and its cause requires further investigation.

References

Getchell, R.G. (1989). Bacterial shell disease in crustaceans: a review. *Journal of Shellfish Research 8*: 1–6.

Noga, E.J.; Smolowitz, R.; Khoo, L.H. (2000). Pathology of shell disease in the blue crab, *Callinectes sapidus* Rathburn (Decapoda: Portunidae). *Journal of Fish Diseases 23*: 389–399.

Sindermann, C.J. (1990). Bacterial diseases of crustaceans. *In*. Principal diseases of marine fish and shellfish. Vol. 2, 2nd edition, pp. 48–70. Academic Press, San Diego.



Shell disease and tail blistering in lobsters. Photos by B. Diggles and N. Raethke.

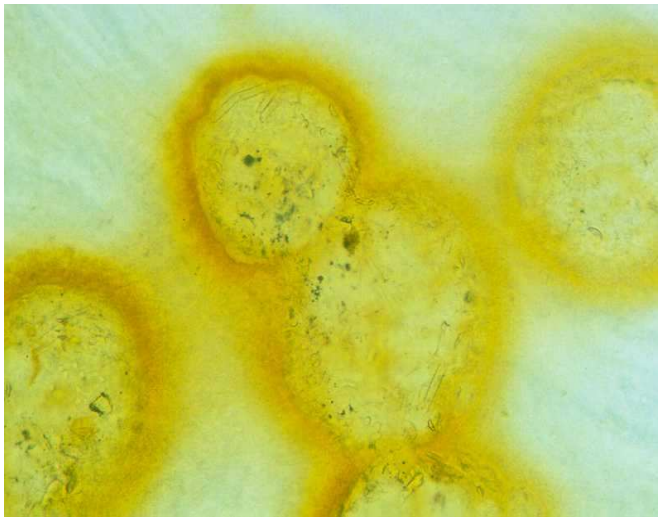
Above left: Tail blisters (arrows) in *J. edwardsii* held in a concrete tank.

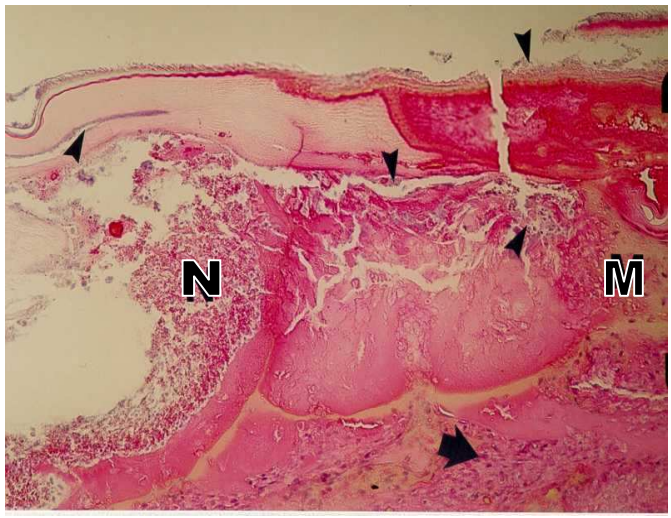
Above right: Erosion of the tail fan of a *J. edwardsii* held in a concrete tank.

Left: Wet preparation of a shell disease lesion in *J. verreauxi* showing melanised tracks caused by fungal hyphae.

Below left: Wet preparation of carapace of *J. verreauxi* showing melanisation at edges of circular lesions caused by chitinoclastic bacteria.

Below right: Histopathology of a tail blister lesion in *J. edwardsii* showing numerous bacteria (arrowheads) inside the cuticle and areas of necrosis (N), melanisation (M), and haemocyte infiltration (arrow).





Disease: Vibriosis (septicaemia)

Species and life stage affected: All life stages of red rock lobsters (*Jasus edwardsii*), packhorse rock lobster (*J. verreauxi*), and paddle crabs (*Ovalipes catharus*).

Gross signs: Can vary between species. Phyllosoma larvae may become opaque or exhibit blackened areas on the carapace. Affected juvenile and adult lobsters and crabs are usually lethargic and stop feeding. The haemolymph may become turbid and body musculature may become opaque. A reddening of the haemolymph has been observed in moribund paddle crabs.

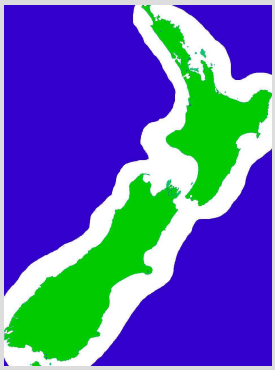
Causative agents: Opportunistic bacteria of the genus *Vibrio*. Both *Vibrio splendidus* I and *Vibrio harveyi* have been isolated from the haemolymph of adult *J. edwardsii* in New Zealand.

Diagnosis: Septicaemia is defined by an infection of the bloodstream, hence diagnosis is based on isolation of bacteria from the haemolymph. Once bacteria are isolated in culture on bacteriological media, they can be identified using biochemical or molecular methods.

Treatment and prevention: Occurrence of bacterial disease usually results from injury and/or sub-optimal culture conditions. Elimination of potential sources of injury, and improvement of water quality, food quality, or rearing system design may be required to improve the health of affected stock and prevent reoccurrence of septicaemia. Antibiotic treatment may be possible if the bacteria involved can be identified and their antibiotic sensitivities can be determined, but routine use of antibiotics will promote development of resistant strains of bacteria and is not a viable long-term alternative to good husbandry practices.

Distribution in New Zealand

Opportunistic bacteria of the genus *Vibrio* are ubiquitous in the marine environment and can infect crustaceans throughout New Zealand if they are held or cultured under sub-optimal conditions.



Worldwide distribution: Opportunistic bacteria of the genus *Vibrio* are ubiquitous in the marine environment hence vibriosis is a problem worldwide wherever crustaceans are injured, stressed or held under sub-optimal conditions (Brinkley et al. 1976, Lightner 1983, Sindermann 1990).

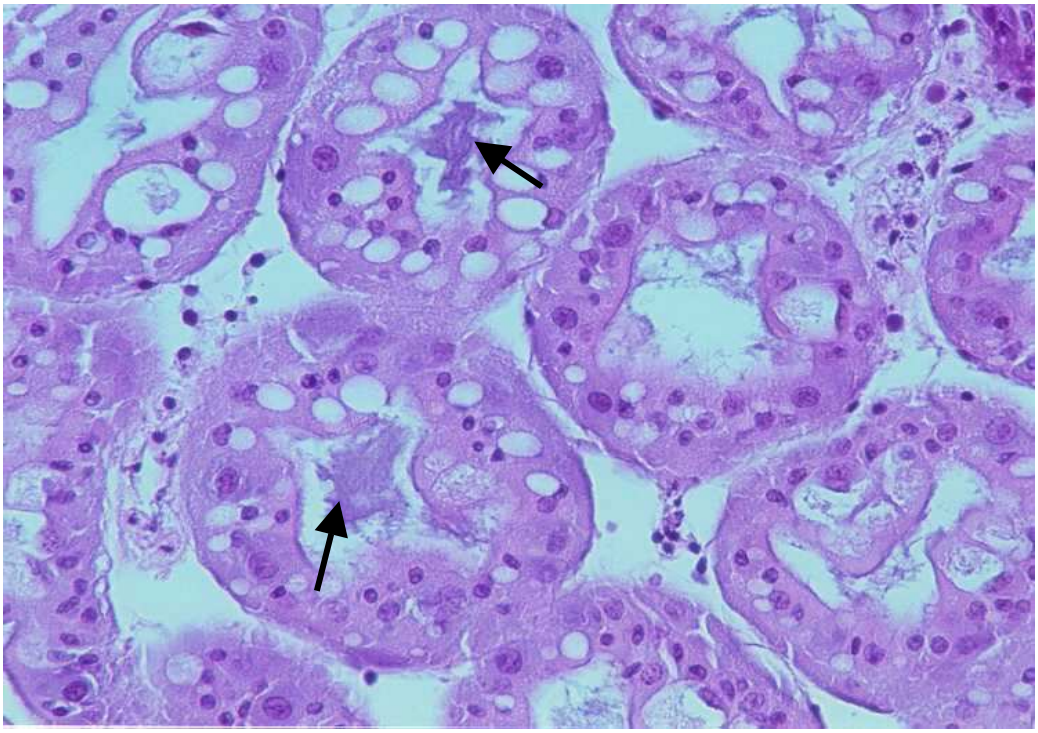
General comments: Vibriosis, like other bacterial diseases of crustaceans, is caused by opportunistic pathogens which become invasive and destructive when host defences are breached by injury or lowered due to abnormal environmental conditions (Sindermann 1990).

References

Brinkley, A,W.; Rommel, F.A.; Huber, T.W. (1976). The isolation of *Vibrio parahaemolyticus* and related vibrios from moribund aquarium lobsters. *Canadian Journal of Microbiology 22*: 315–317.

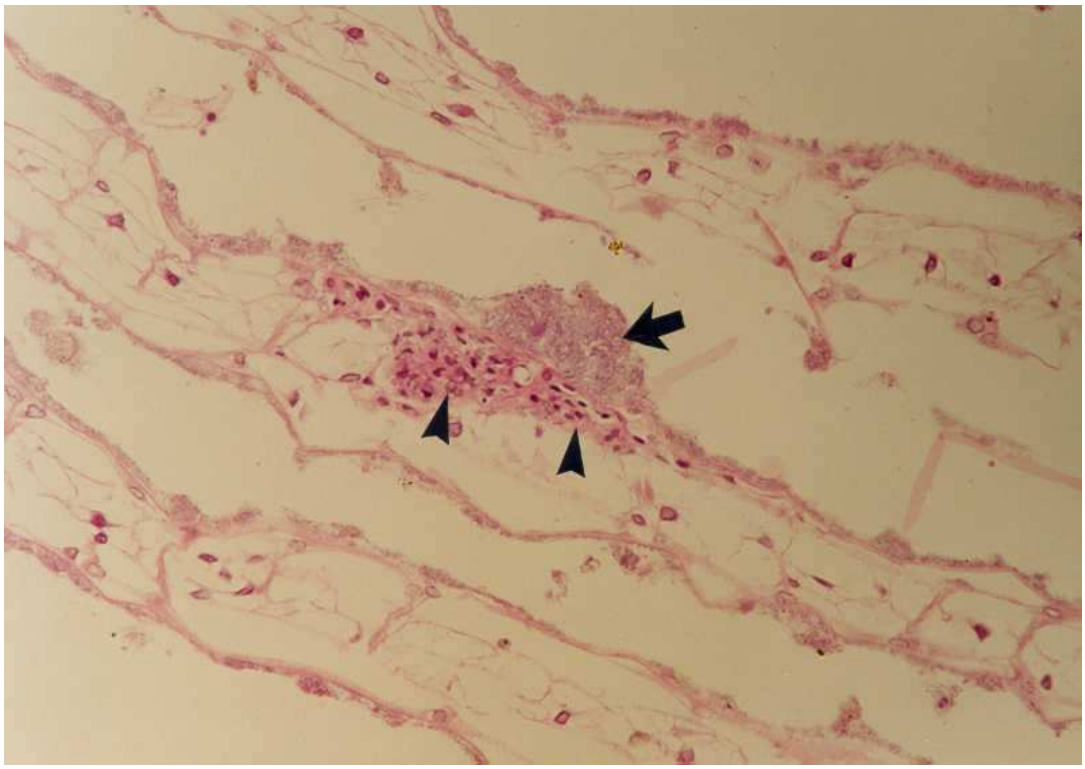
Lightner, D.V. (1983). Diseases of cultured shrimp. *In*: McVey, P.V. (ed), CRC handbook of mariculture, Vol. I. Crustacean aquaculture. pp. 289–320. CRC Press, Boca Raton.

Sindermann, C. J. (1990). Bacterial diseases of crustaceans. *In*. Principal diseases of marine fish and shellfish. Vol. 2, 2nd edition, pp. 48–70. Academic Press, San Diego.



Vibriosis in lobsters and crabs. Photos by B. Diggles.

Above: Vibriosis in juvenile *Jasus edwardsii*. Histology of the hepatopancreas showing large plaques of bacteria (arrows) inside the tubule lumens. **Below:** Vibriosis in adult paddle crabs (*Ovalipes catharus*). Histology of the gill showing that bacteria on the outside of the gill cuticle (arrow) have penetrated the gill cuticle in one area. The presence of the bacteria inside the gill has elicited a host response consisting of clumping of haemocytes (arrowheads) to prevent bacteria entering the haemolymph spaces.



Diseases of unknown aetiology of crustaceans

Disease: Black hepatopancreas disease

Species and life stage affected: Red rock lobster (*Jasus edwardsii*) sub-adults.

Gross signs: Lethargy; upon dissection large, hard, blackened necrotic lesions in the hepatopancreas.

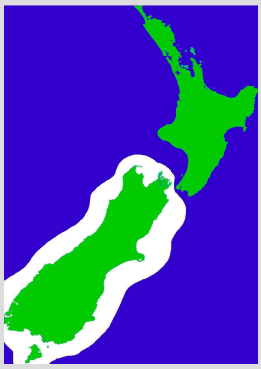
Causative agent: Unknown, possibly related to diet and/or infection with bacteria and/or unknown protozoa.

Diagnosis: Affected lobsters are reportedly lethargic, stop feeding, and eventually die. Presumptive diagnosis can be obtained by dissecting lobsters and observing hardened, black, necrotic lumps in the hepatopancreas. Bacteria and protozoan-like cells can be observed in histological sections.

Treatment and prevention: Unknown. Perhaps provision of an adequate diet will prevent this condition from occurring.

Distribution in New Zealand

Known only from one instance in lobsters used in a dietary experiment in an experimental grow-out facility in the South Island.

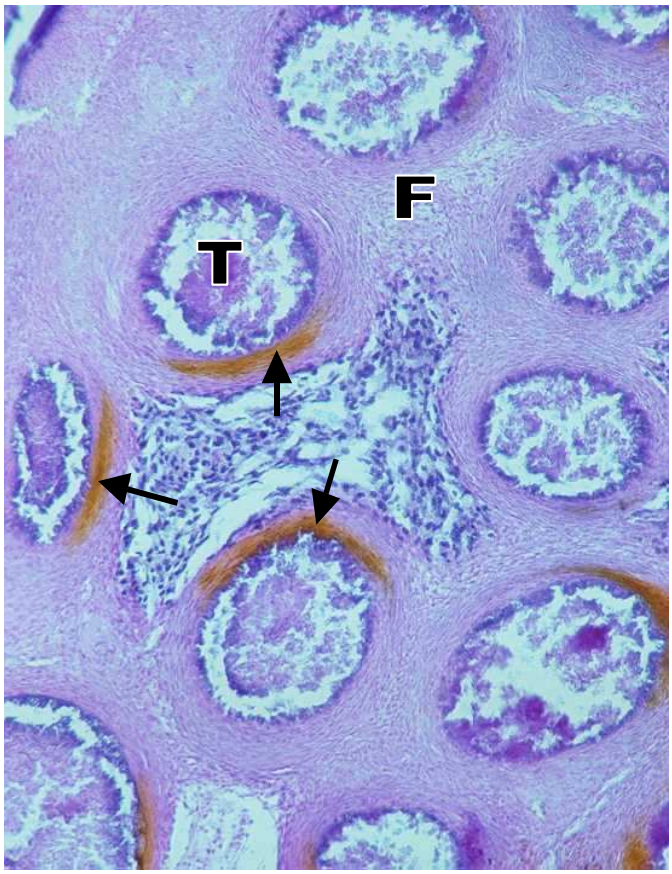


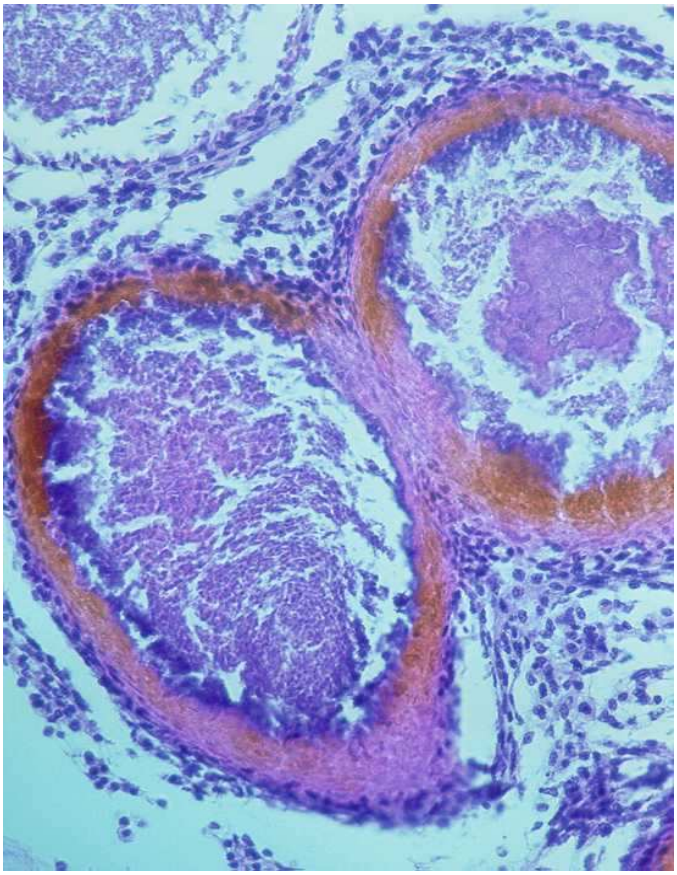
Worldwide distribution: Currently only recorded from lobsters in New Zealand. This disease resembles bacterial necrosis and mummification of hepatopancreas tubules of marron, *Cherax tenuimanus*, in Australia. Disease in marron was thought to be caused by failure to digest an inadequate diet (Langdon et al. 1992).

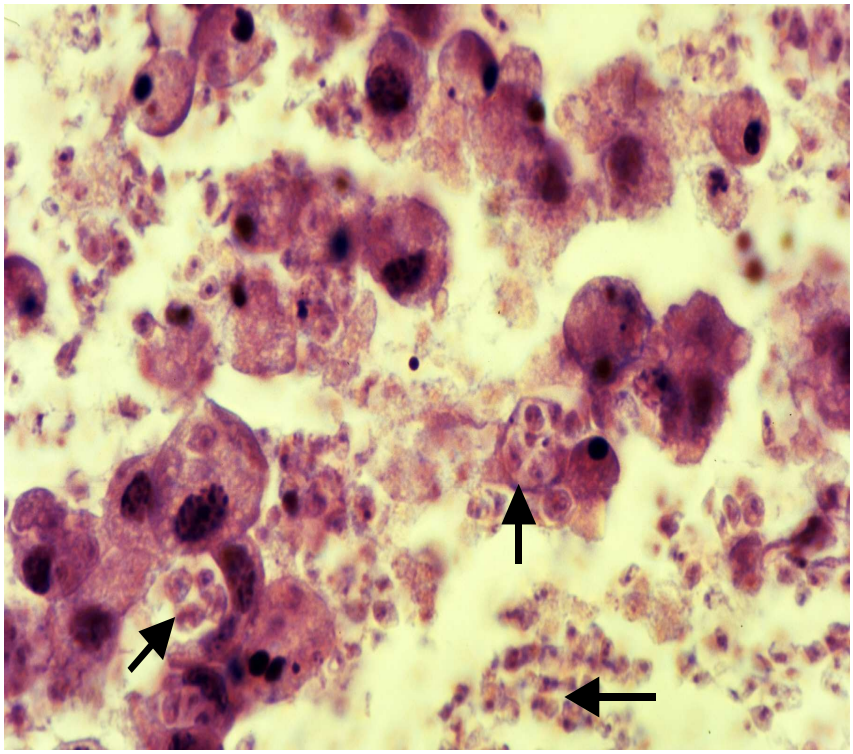
General comments: This disease has been observed only once in sub-adult lobsters fed an experimental food containing a high percentage of abalone (*Haliotis* sp.) viscera. Histopathology showed complete necrosis of affected hepatopancreas tubules which were surrounded by a melanised layer with accompanying fibrosis. Some tubules contained a mass of gram-negative bacteria and also protozoan-like cells. Whether the presumptive protozoa or bacteria play a primary or secondary role in the disease is at this stage unclear.

Reference

Langdon, J.S.; Buller, N.; Ostle, C.; Thorne, T. (1992). Bacterial necrosis and mummification of the digestive gland associated with feeding peas, *Pisum sativum*, to freshwater crayfish, *Cherax tenuimanus*. *In*: Diseases in Asian aquaculture I. Shariff, M.; Subasinghe, R.P.; Arthur, J.R. (eds),), pp. 199–205. Fish Health Section, Asian Fisheries Society, Manila.







Histopathology of black hepatopancreas disease in *Jasus edwardsii*. Photos by B. Diggles and J. Handlinger.

Above left: Necrotic hepatopancreas tubules showing necrosis of tubules (T), melanin deposition (arrows), and extensive fibrosis (F).

Above right: Necrotic tubule at higher power showing brown layer of melanin and cell debris inside remains of the tubule.

Left: High power view of cell debris inside tubule showing clumps of protozoan-like cells (arrows).

Disease: Turgid lobster syndrome (TLS)

Species and life stage affected: Juvenile and adult red rock lobsters (*Jasus edwardsii*).

Gross signs: Affected lobsters exhibit fluid-filled, swollen arthrodial membranes apparently caused by an increase in haemolymph volume.

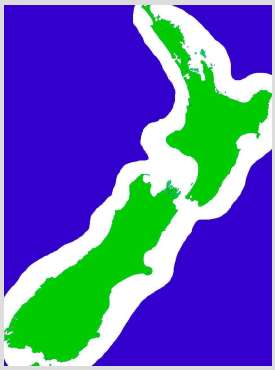
Causative agent: Unknown. May be a nonspecific response to a variety of stressors.

Diagnosis: TLS is associated with abnormal protrusion of the arthrodial membranes, especially between the carapace and abdomen. Haemolymph is expelled under pressure if the membrane is punctured. An increase in the number of circulating granulocytes and pre-granulocytes is sometimes noted. In early stages of TLS the affected lobsters stop feeding, show limited swelling, and become lethargic, while in later stages they cannot flex their abdomens and mortalities may occur. Bacteria such as *Vibrio harveyi* and *V. splendidus* I are occasionally, but not always, isolated from the haemolymph.

Treatment and prevention: Specific methods of treatment and prevention are unknown, but reduction or elimination of possible sources of stress on affected lobsters is recommended. In particular check water quality parameters, especially ammonia and salinity. Some lobsters appear to spontaneously recover without intervention.

Distribution in New Zealand

Has been observed in lobster holding facilities throughout New Zealand.



Worldwide distribution: TLS-like symptoms have also been reported in *J. edwardsii* and western rock lobster (*Panulirus longipes*) in holding facilities in Australia (A. Brown, W. Hosking, personal communication).

General comments: The swelling may be a nonspecific response to a variety of stressors. Possible causes may include starvation, as this causes increased haemolymph volume (Dall 1974), poor nutrition, salinity variation, osmotic imbalance, or exposure to toxicants such as pesticides (perhaps at levels below detection limits). Lobsters appear more likely to be affected just before the moult. In one case TLS was associated with hypersalinity (44‰) and the swelling was reversed when salinity was reduced to normal (36‰) (A. Brown, personal communication). In many cases, however, no salinity variation from normal can be detected.

Reference

Dall, W. (1974). Indices of nutritional state in the western rock lobster, *Panulirus longipes* (Milne Edwards). I. Blood and tissue constituents and water content. *Journal of Experimental Marine Biology and Ecology 16*: 167–180.

Turgid lobster syndrome (TLS) in *Jasus edwardsii*. Photos by B. Diggles.

Above: Normal appearance of the arthrodial membrane between the carapace and abdomen in an adult *Jasus edwardsii*. **Below:** Characteristic protrusion of the arthrodial membrane between the carapace and abdomen (arrow) associated with TLS.

Diseases of Molluscs

DISEASES OF MOLLUSCS

Viral diseases of molluscs

Disease: Amyotrophia of abalone

Species and life stage affected: Juvenile Japanese black abalone (*Nordotis discus discus*).

Gross signs: Unusually high mortalities in juvenile abalone associated with wasting of the foot.

Causative agent: Unknown. Possibly a virus (Nakatsugawa et al. 1999).

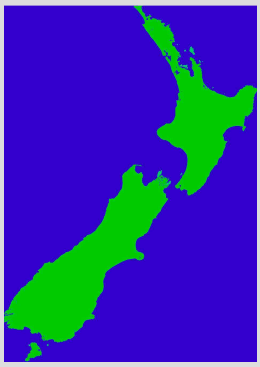
Diagnosis: Detection of multiple tumours surrounding the nerves of the foot by histology in the absence of other potential disease agents.

Treatment and prevention: No known treatment. The disease agent is transmitted horizontally through the water, but filtration to 0.22 µm can exclude the disease agent (Nakatsugawa et al. 2000).

Distribution in New Zealand

Unrecorded.

AMYOTROPHIA IS AN EXOTIC DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Currently known only from cultured juvenile abalone in Japan.

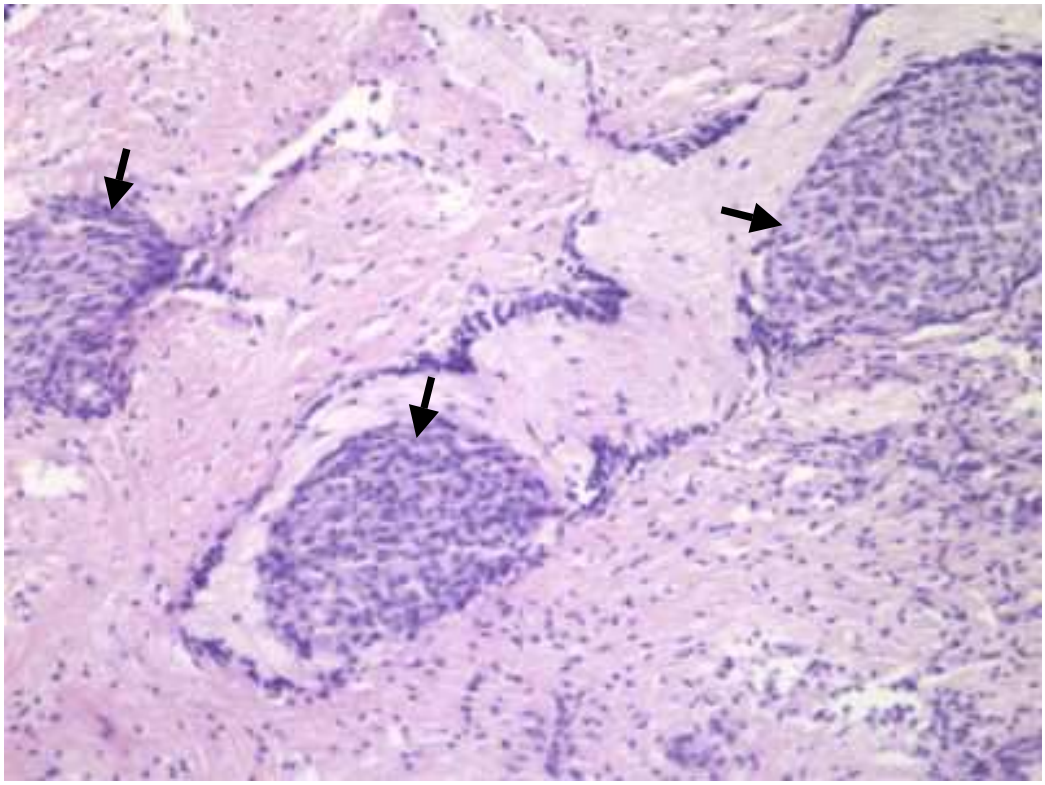
General comments: Granulomatous, tumour-like lesions have been detected in wild-caught adult paua (*Haliotis iris*) in New Zealand (Diggles, unpublished), but they were found in various organs and were not limited to the nerves. Their cause is unknown.

References

Nakatsugawa, T. (1990). Infectious nature of a disease in cultured abalone with muscular atrophy. *Fish Pathology 25*: 207–211.

Nakatsugawa, T.; Nagai, T.; Hiya, K.; Nishizawa, T.; Muroga K. (1999). A virus isolated from juvenile black abalone *Nordotis discus discus* affected with amyotrophia. *Diseases of Aquatic Organisms 36*: 159–161.

Nakatsugawa, T.; Okabe, M.; Muroga, K. (2000). Horizontal transmission of amyotrophia in Japanese black abalone. *Fish Pathology 35*: 11–14.



Histopathology of amyotrophia in Japanese black abalone (*Nordotis discus discus*). 200x magnification.
Photo by C. Friedman.

Note the three tumour-like lesions (arrows) adjacent to nerves in the foot.

Disease: Digestive epithelial virosis

Species and life stage affected: Greenshell™ mussels (*Perna canaliculus*), scallops (*Pecten novaezelandiae*), rock oysters (*Saccostrea glomerata*), toheroa (*Paphies ventricosa*), and probably other bivalves.

Gross signs: Unusual mortalities of between 50 and 100% of reseeded mussel spat during the summer months. Other species may not show any gross signs of infection.

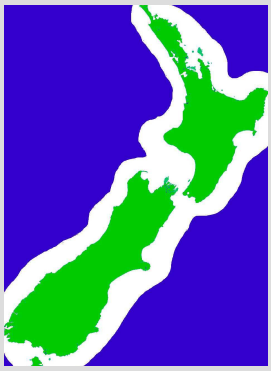
Causative agents: Small unenveloped RNA viruses.

Diagnosis: Presumptive diagnosis may be obtained using histology to show extensive haemocytosis and liquefactive necrosis of digestive tubule epithelial cells. Definitive diagnosis is by electron microscopy to demonstrate electron-dense, uncoated virus-like particles 25–45 nm in diameter in digestive tubule epithelium.

Treatment and prevention: No known treatment. However improvements in husbandry may reduce the impact of the disease (Jones et al. 1996).

Distribution in New Zealand

Probably ubiquitous.



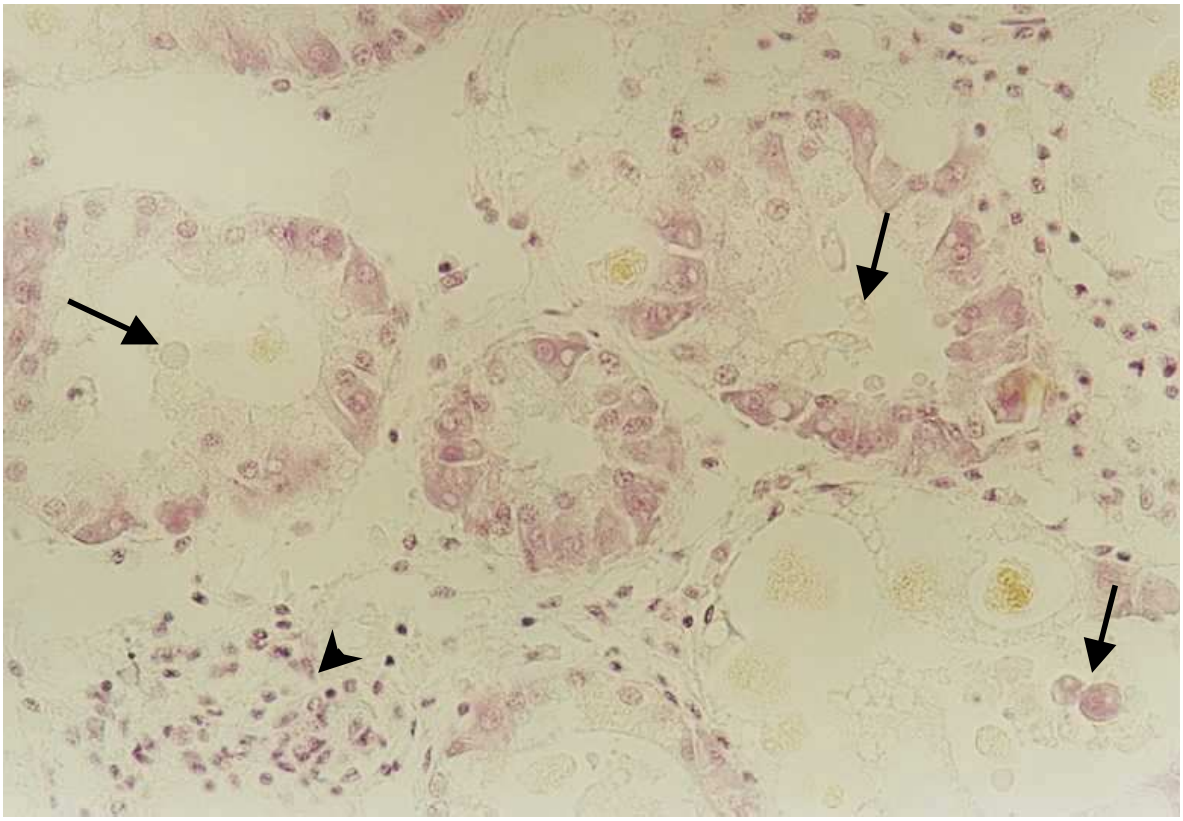
Worldwide distribution: Although reported only from Greenshell™ mussels (Jones et al. 1996), scallops, and toheroa (Hine & Wesney 1997) in New Zealand, examination of scallops (*Pecten alba*) from Port Phillip Bay, Australia, and scallops (*Pecten maximus*) from Loch Fyne, Scotland, has shown the disease to be equally prevalent there (Hine : unpublished data).

General comments: The viruses are associated with sloughing of the gut lining (epithelium), but their actual role in this "disease" is unclear. The sloughing and renewal of the gut lining occurs in many species of bivalves, and it appears to be a natural process in healthy animals. Study has shown that all scallops examined from around New Zealand have these viruses. It appears, however, that high levels of these viruses occur in lethargic and dying scallops, and therefore the viruses may be associated with the natural process of renewal of the gut lining, but if they reach high levels, they may cause disease.

References

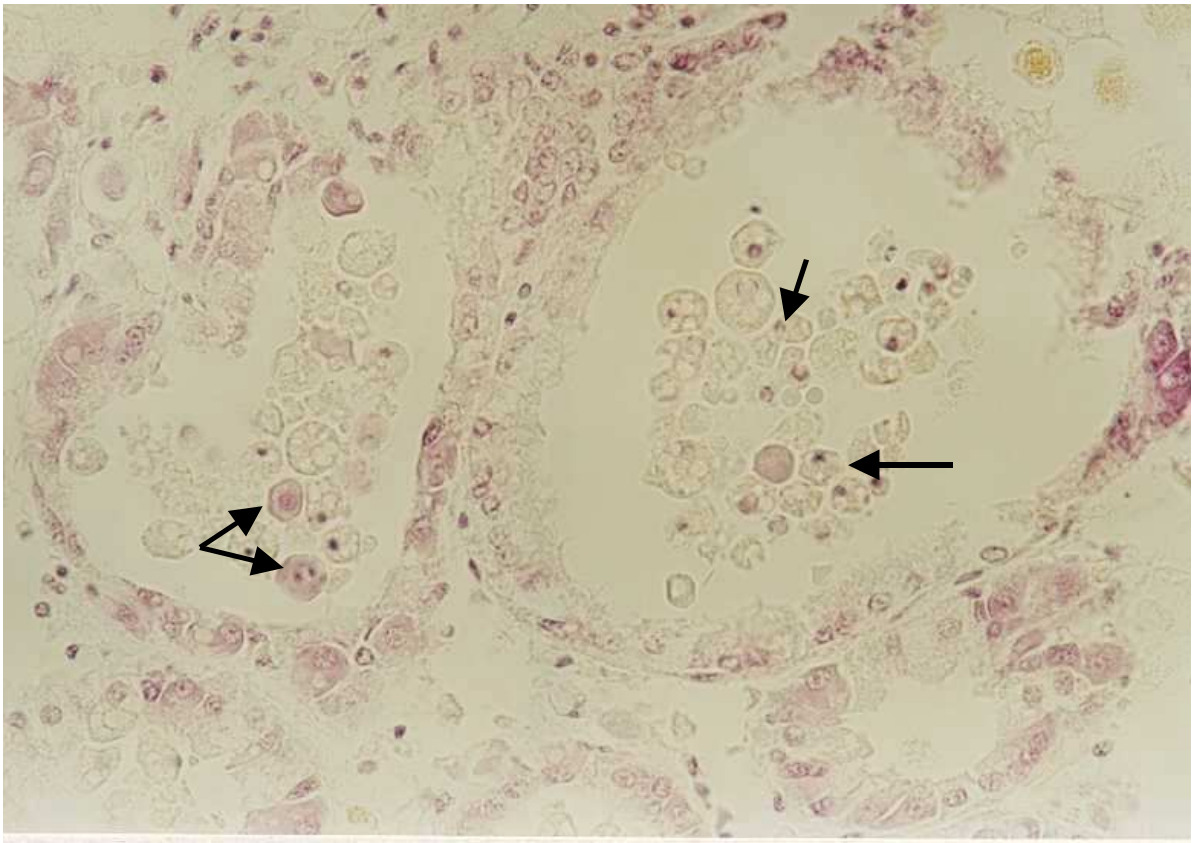
Hine, P.M.; Wesney, B. (1997). Virus-like particles associated with cytopathology in the digestive gland epithelium of scallops *Pecten novaezelandiae* Reeve, 1853 and toheroa *Paphies ventricosum* (Gray, 1843). *Diseases of Aquatic Organisms 29*: 197–204.

Jones, J.B.; Scotti, P.D.; Dearing, S.C.; Wesney, B. (1996). Virus-like particles associated with marine mussel mortalities in New Zealand. *Diseases of Aquatic Organisms 25*: 143–149.



Histology of digestive epithelial virosis in scallops (*Pecten novaezelandiae*). Photos by B. Diggles.

Above: Light to moderate sloughing of epithelial cells of the digestive gland (arrows) with a focus of haemocytosis (arrowhead). **Below:** Extensive sloughing and necrosis of the digestive gland tubule epithelium with numerous necrotic epithelial cells evident in the tubule lumen (arrows).



Disease: Gill necrosis virus disease (GNV) (haemocytic infection virus disease (HIV))

Species and life stage affected: Juveniles and adults of Pacific oysters (*Crassostrea gigas*).

Gross signs: Gill erosion, yellow to brownish spots on the gills, sometimes yellow or green pustules on the mantle or in the adductor muscle.

Causative agent: An iridovirus-like virus, 380 nm in diameter.

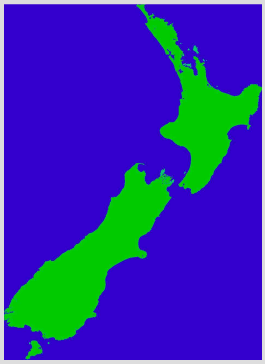
Diagnosis: Presumptive diagnosis is by examination of tissues using histology to demonstrate massive haemocytic infiltration, polymorphic hypertrophic cells, and hypertrophic globular cells containing basophilic inclusions. Definitive diagnosis is by electron microscopy to demonstrate virus particles inside basophilic inclusions.

Treatment and prevention: No known treatment. Prevention is by slaughter and disinfection.

Distribution in New Zealand

Unreported.

GNV AND HIV ARE EXOTIC DISEASES. IF YOU SUSPECT THAT YOUR STOCK HAVE THESE DISEASES, RING MAF ON 0800-809-966



Worldwide distribution: France, Portugal, Spain, and the U.K.

General comments: This disease was first reported in France in November 1966 and caused epizootic mortalities until 1970 when the similar disease HIV replaced it. Subsequently, HIV also disappeared, possibly because a sub-species of *Crassostrea gigas*, then called *Crassostrea angulata*, was particularly susceptible, and it was virtually wiped out by the disease. Subsequently, it was replaced by the less susceptible *C. gigas*.

Reference

Comps, M. (1988). Epizootic diseases of oysters associated with viral infections. *American Fisheries Society Special Publication 18*: 23–37.

No photographs currently available

Disease: Herpesvirus infection of oysters

Species and life stage affected: Larvae and spat of Pacific oysters (*Crassostrea gigas*). Adults, and probably larvae and spat, of Bluff oysters (*Ostrea chilensis*).

Gross signs: Heavy mortalities in oyster larvae associated with abnormal swimming behaviour. Adult Bluff oysters may be asymptomatic carriers.

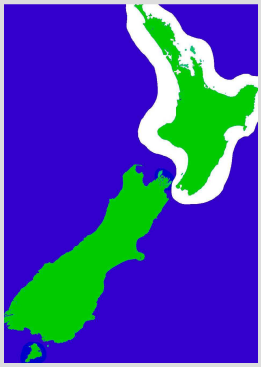
Causative agents: Herpesviruses.

Diagnosis: Definitive diagnosis requires electron microscopy to demonstrate the presence of enveloped, herpesvirus-like virus particles in affected organs. PCR based molecular probes are also available.

Treatment and prevention: This disease is particularly problematic during hot summers, and in Pacific oysters it can be avoided by lowering water temperatures.

Distribution in New Zealand

In Pacific oysters around Northland, and in flat oysters from Wellington Harbour. Probably ubiquitous, but more common in the northern parts of the country.



Worldwide distribution: First reported from oysters (*Crassostrea virginica)* on the east coast of the U.S. (Farley et al. 1972). Subsequently reported from New Zealand Pacific oysters (Hine et al. 1992), French Pacific oysters (Renault et al. 1994) and flat oysters (Renault et al. 2000), Australian flat oysters (Hine & Thorne 1997), and New Zealand flat oysters (Hine et al. 1998). These viruses also occur in clams (*Ruditapes decussatus*) in France, Sydney rock oysters, and Pacific oysters in Mexico.

General comments: The virus is probably vertically transmitted and molecular evidence suggests that the viruses from different hosts in one region are more closely related to each other than the viruses from the same host in different regions. One theory to account for this is that the natural host of the virus is the Pacific oyster, and that with the spread of Pacific oysters for aquaculture, the virus has been spread to new regions. In those regions the virus has started to infect other host species.

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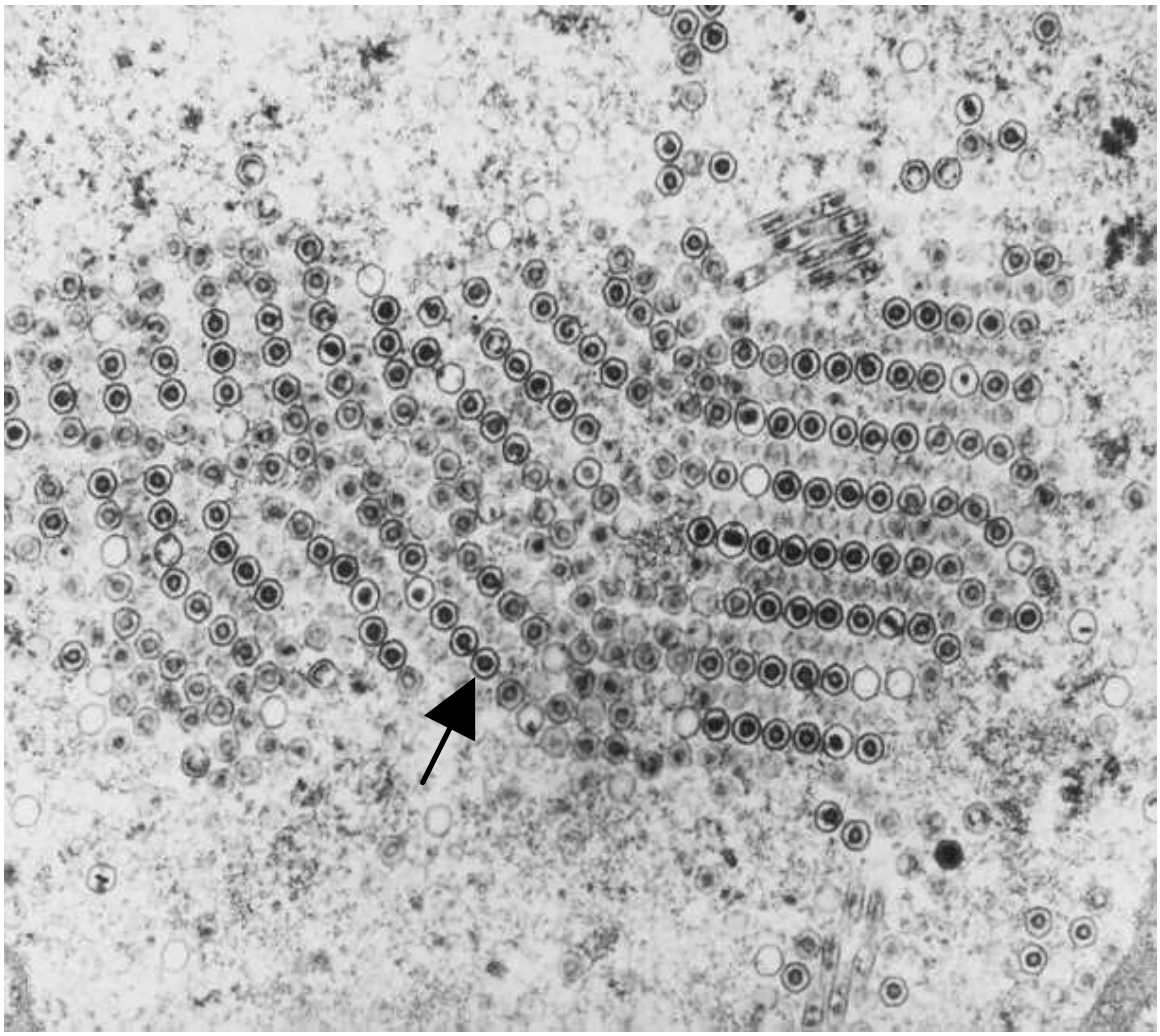
Hine, P.M.; Wesney, B.; Besant, P. (1998) Replication of herpes-like viruses in larvae of the flat oyster *Tiostrea chilensis* at ambient temperatures. *Diseases of Aquatic Organisms 32*: 161–171.

Hine, P.M.; Wesney, B.; Hay, B.E. (1992). Herpesviruses associated with mortalities among hatchery-reared larval Pacific oysters *Crassostrea gigas*. *Diseases of Aquatic Organisms 12*: 135–142.

Renault, T.; Le Deuff, R-M.; Chollet, B.; Cochennec, N.; Gérard, A. (2000). Concomitant herpes-like virus infections in hatchery-reared larvae and nursery-cultured spat *Crassostrea gigas* and *Ostrea edulis*. *Diseases of Aquatic Organisms 42*: 173–183.

Renault, T.; Le Deuff, R.M.; Cochennec, N.; Maffart, P. (1994). Herpesviruses associated with mortalities among Pacific oyster, *Crassostrea gigas*, in France – comparative study. *Revue de Médecine Véerinairet 145*: 735–742.



Herpesvirus in Bluff oysters (*Ostrea chilensis*). Photo by M. Hine.

TEM micrograph of a paracrystalline array of enveloped herpes-like viruses (arrow) inside the nucleus of an epithelial cell in the mantle.

Disease: Oyster velar virus disease (OVVD)

Species and life stage affected: Larvae of Pacific oysters (*Crassostrea gigas*).

Gross signs: Heavy mortalities in oyster larvae associated with abnormal swimming behaviour and detachment of the larval velum.

Causative agent: An iridovirus.

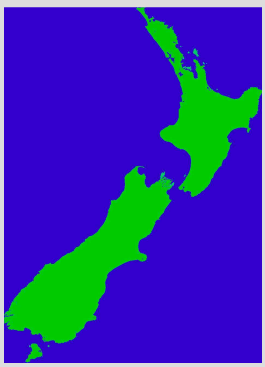
Diagnosis: Necrosis and detachment of the larval velum (without bacterial infection) may be observed by light microscopy (Elston & Wilkinson 1985). Electron microscopy demonstrating the iridovirus for confirmation (Elston 1979, 1993).

Treatment and prevention: No known treatment. Prevention is by destruction and safe disposal of infected stocks, and disinfection of tanks and gear.

Distribution in New Zealand

Unreported.

OVVD IS AN EXOTIC DISEASE. IF YOU SUSPECT THAT YOUR STOCKS HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Washington State, U.S.

General comments: First signs are that the larvae have problems in swimming, and they gradually sink to the bottom of the tank. However, larvae infected with herpesviruses show the same behaviour, and electron microscopic examination is needed to distinguish these two diseases. Once either virus disease occurs, killing stock and disinfection are recommended, but subsequent mortalities may be avoided by lowering temperatures in the case of herpesvirus infections.

References

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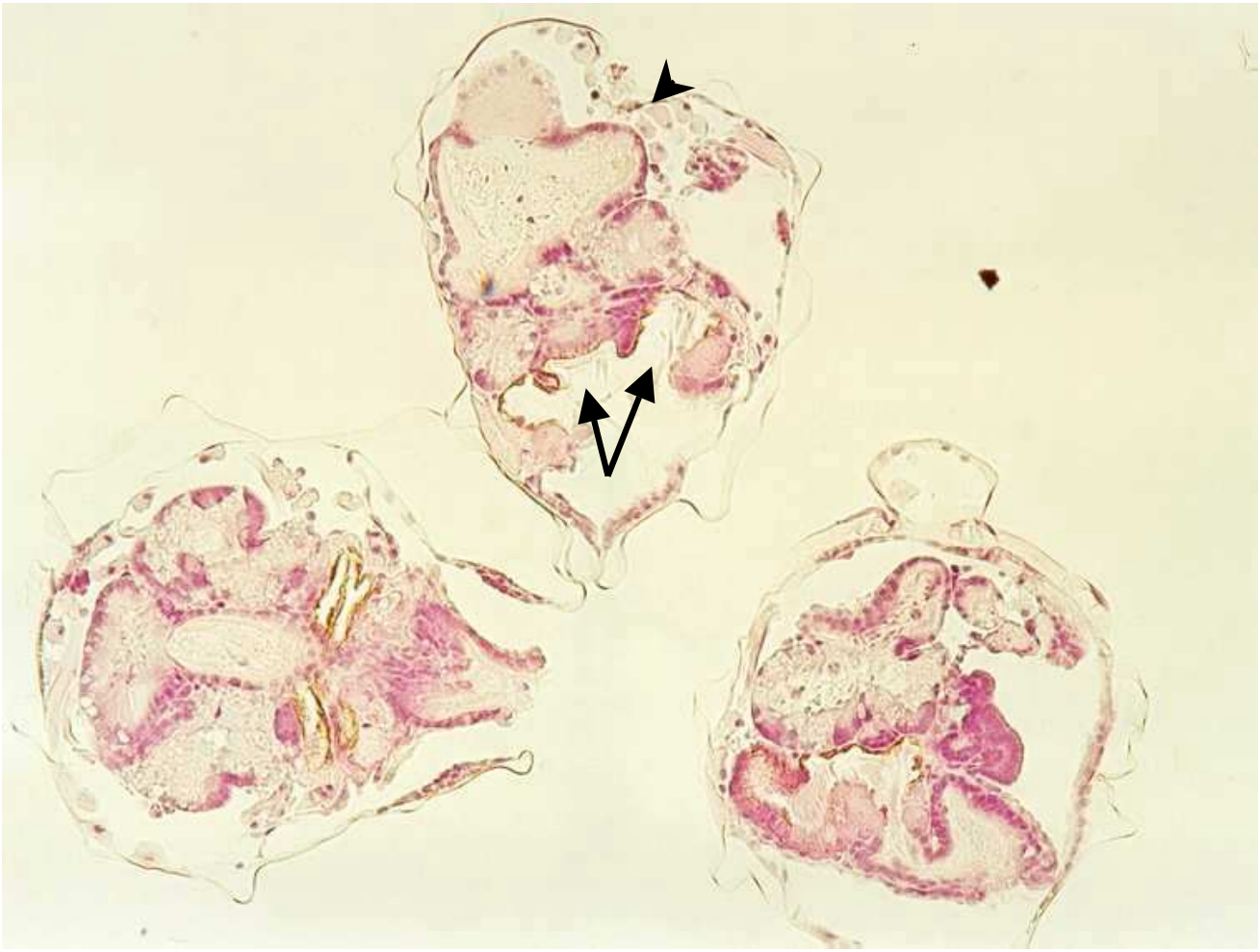


Histological section of OVVD affected larvae of a Pacific oyster (*Crassostrea gigas*). Photo by B. Diggles.

Note absence of bacterial infection in all three larvae, necrotic cells (arrowhead), and detachment and fragmentation of the velum (arrows) in one larva. TEM was required to confirm this as OVVD.

Microbial diseases of molluscs

Disease: Brown ring disease of clams (BRD) (vibriosis)

Species and life stage affected: Spat, juveniles, and adults of Manila clams (*Ruditapes philippinarum*) and carpet-shell clams (*R. decussatus*). Vibriosis occurs in many species of cultured molluscs, including pearl oysters (*Pinctada* sp.).

Gross signs: An abnormal brown deposits in the nacre between the mantle and the edge of the shell.

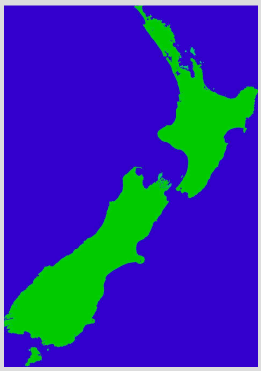
Causative agents: Gram-negative bacteria of the genus *Vibrio*. BRD is caused by *Vibrio tapetis* (also known as *Vibrio* P1); brown conchiolin deposits in other species of mollusc can be associated with a variety of species of *Vibrio*, particularly *Vibrio harveyi*.

Diagnosis: It may be difficult to detect bacteria by light microscopy in early infections. Confirmatory diagnosis requires isolation and identification of the causative bacterium from shell lesions and haemolymph using standard microbiological methods. A colony-blot ELISA is available for BRD (Noel et al. 1996).

Treatment and prevention: Vibriosis is usually associated with high stocking densities and poor water quality. BRD may be controlled by raising water temperatures, as the disease does not occur along coasts that have warm summer temperatures, and although 100% infection occurs at 8–14 °C, only 20% infection occurs at 21 °C (Paillard et al. 1999). Infections may be treated with antibiotics, though modification of husbandry practices, particularly reduction in stocking density, are the preferred method of control.

Distribution in New Zealand

Vibriosis may occur throughout New Zealand wherever molluscs are reared at high densities or under conditions of poor water quality. However, BRD has not been reported.



BRD IS AN EXOTIC DISEASE AND CANNOT BE DISTINGUISHED FROM VIBRIOSIS EXCEPT IN A LABORATORY. IF YOU SUSPECT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: Vibriosis has been recorded worldwide in cultured molluscs, particularly in the warm waters of the tropics and subtropics. BRD caused by *Vibrio tapetis* is restricted to the Atlantic coasts of Europe.

General comments: BRD is particularly problematic in clams cultured under crowded conditions. *Vibrio harveyi* is an ubiquitous marine bacterium that can cause disease in crustaceans, molluscs, and less frequently in fishes, particularly when they are cultured under crowded conditions. The anomalous brown nacre in BRD occurs as a thin ring around the perimeter of the shell (Paillard et al. 1994). In contrast, molluscs with vibriosis caused by *Vibrio harveyi* usually display broad based conchiolin deposits in the nacre (Dybdahl & Pass 1985, Pass et al. 1987).

References

Dybdahl, R.; Pass, D.A. (1985). An investigation of mortality of the pearl oyster, *Pinctada maxima*, in Western Australia. *Western Australian Fisheries Department Report No. 71*. 78 p.

Noel, T.; Nicolas, J.L.; Boulo, V.; Mialhe, E.; Roch, P. (1996). Development of a colony-blot ELISA assay using monoclonal antibodies to identify *Vibrio* P1 responsible for "brown ring disease" in the clam *Tapes philippinarum. Aquaculture 146*: 171–178.

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Paillard, C.; Allam, B.; Oubella, R.; Ford, S.E. (1999). Temperature effects on brown ring disease susceptibility and defense-related activities in the Manila clam, *Ruditapes philippinarum. Journal of Shellfish Research 18*: 298.

Pass, D.A.; Dybdahl, R.; Mannion, M.M. (1987). Investigations into the causes of mortality of the pearl oyster, *Pinctada maxima* (Jamson), in Western Australia. *Aquaculture 65*: 149–169.

Vibriosis in pearl oyster *Pinctada* sp. associated with infection by *Vibrio harveyi*. Photo by B. Diggles.

The abnormal brown coloration in the nacre (arrow) is broad based, which is different from the gross signs of BRD, in which the brown stain occurs as a thin ring on the nacre around the perimeter of the shell.

Disease: Mycoplasmosis of scallops

Species and life stage affected: Adult scallops (*Pecten novaezelandiae*).

Gross signs: There may be pinkish-orange pustules in the adductor muscle.

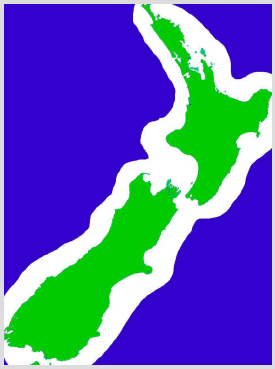
Causative agent: An unidentified mycoplasma (intracellular bacterium).

Diagnosis: Electron microscopy.

Treatment and prevention: None known.

Distribution in New Zealand

Probably ubiquitous.

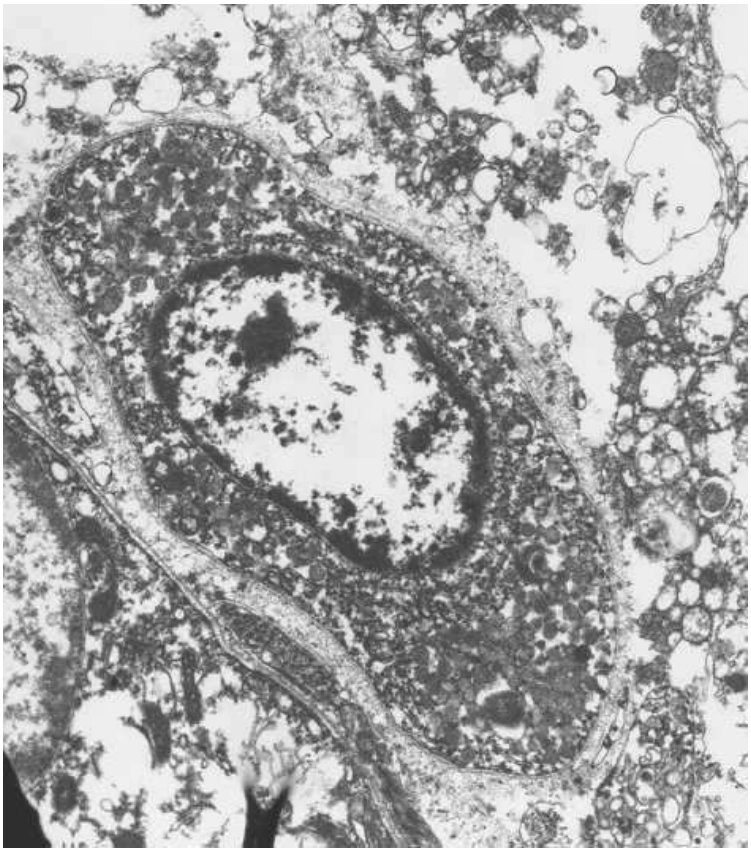


Worldwide distribution: Mycoplasmas are known from scallops in New Zealand, and in *Patinopecten yessoensis* from the west coast of Canada.

General comments: The causative mycoplasmas occur in haemocytes around lesions, but their role in this minor disease is unclear (Bower & Meyer 1991). Mycoplasmas are a group of procaryote organisms that resemble bacteria, but do not possess a cell wall and thus have variable morphology.

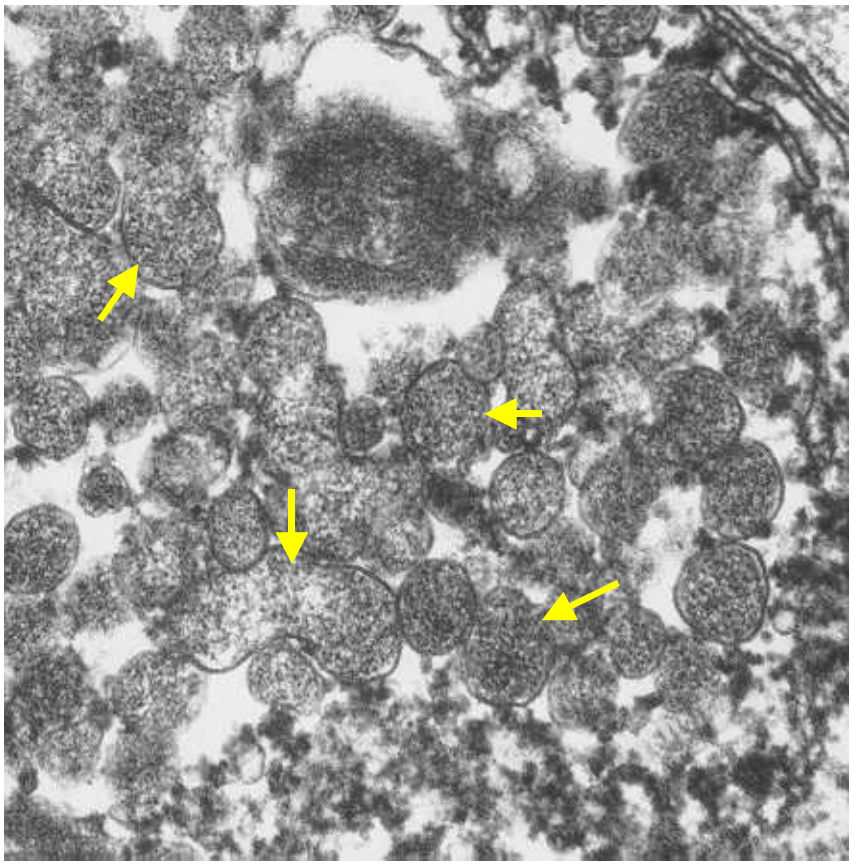
Reference

Bower, S.M.; Meyer, G.R. (1991). Disease of Japanese scallops (*Patinopecten yessoensis*) caused by an intracellular bacterium. *Journal of Shellfish Research 10*: 513.



Mycoplasmosis in New Zealand scallops (*Pecten novaezelandiae*). Photos by M. Hine.

Above: TEM of a scallop haemocyte with its cytoplasm packed with mycoplasmas. **Below:** Higher power view of mycoplasmas (arrows) of variable morphology in the cytoplasm of the haemocyte. 49 000 x magnification.



Disease: Pacific oyster nocardiosis (PON) (summer mortality)

Species and life stage affected: Adult Pacific oysters (*Crassostrea gigas*), possibly also blue mussels (*Mytilus edulis*).

Gross signs: Round yellow-green pustules up to 1 cm in diameter on the surface of the mantle, gill, adductor muscle, and heart.

Causative agent: *Nocardia crassostreae*, a gram-positive bacterium.

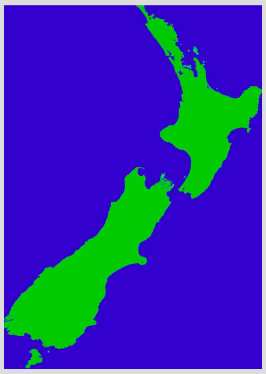
Diagnosis: Visualisation of groups of the bacteria, resembling fungal hyphae, in the connective tissue by light microscopy. PCR based molecular probes are also available (Gee & Elston 1997).

Treatment and prevention: Culturing oysters off the seafloor, and avoiding shallow embayments. *N. crassostreae* is an opportunist that lives in sediment, and infects oysters when water temperatures are elevated in mid summer.

Distribution in New Zealand

Unreported. However, the shallow embayments around Northland would be ideal for this bacterium to flourish if it were introduced into New Zealand.

PON IS AN EXOTIC DISEASE. IF YOU SUSPECT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: West coast of the U.S., Japan, and possibly other Pacific-rim countries.

General comments: The gross signs may be confused with those of Denman Island disease (see p. 174), but the latter occurs only at cold temperatures, and nocardiosis at elevated temperatures.

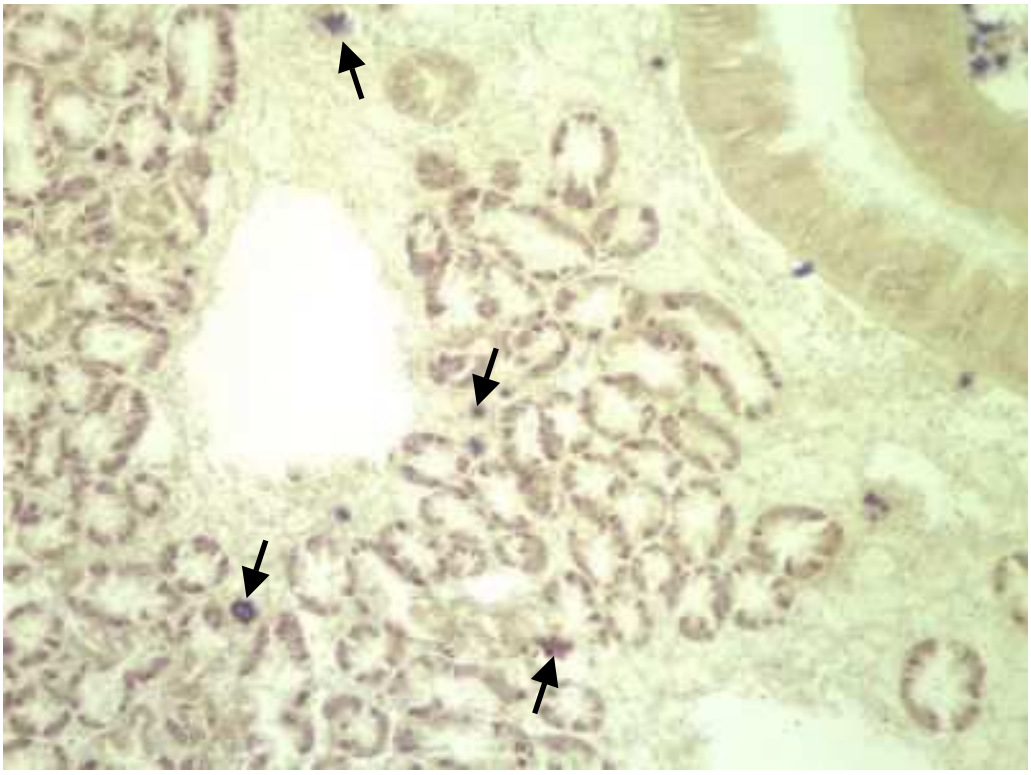
References

Friedman, C.S.; Beaman, B.L.; Chun, J.; Goodfellow, M.; Gee, A.; Hedrick, R.P. (1998). *Nocardia crassostreae* sp. nov., the causal agent of nocardiosis in Pacific oysters. *International Journal of Systematic Bacteriology 48*: 237–246.

Friedman, C.S.; Beattie, J.H.; Elston, R.A.; Hedrick, R.P. (1991). Investigation of the relationship between a gram-positive bacterial infection and summer mortality of the Pacific oyster, *Crassostrea gigas* Thunberg. *Aquaculture 94*: 1–15.

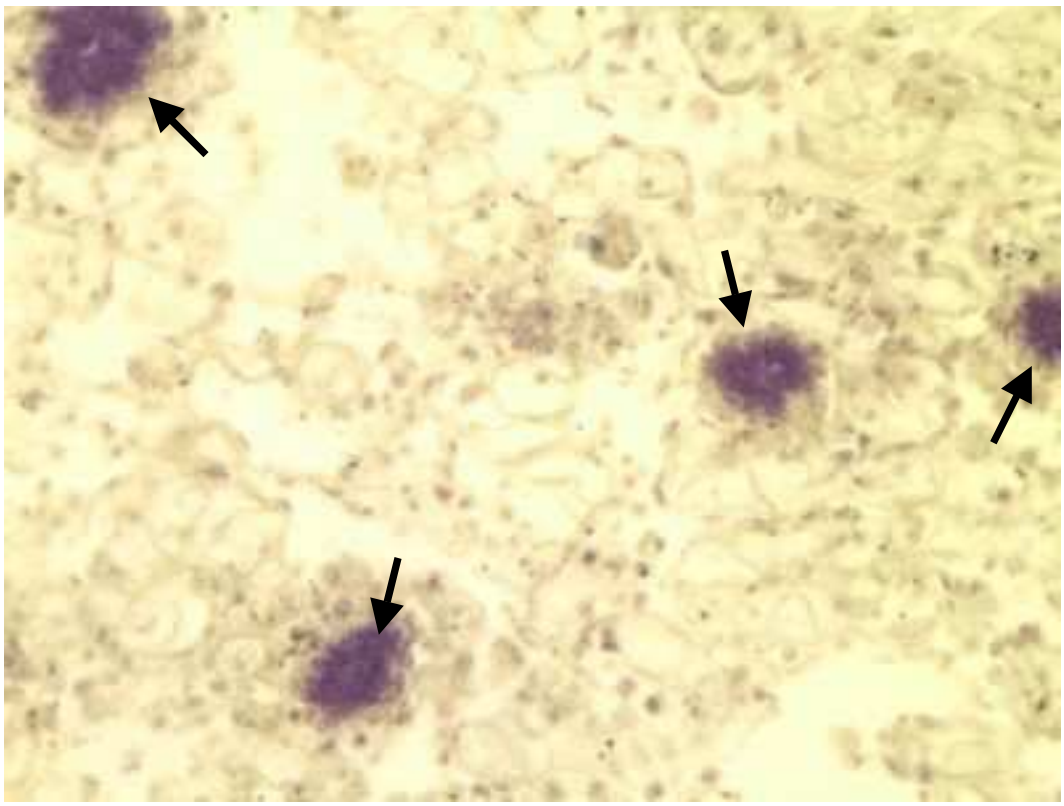
Friedman, C.S.; Hedrick, R.P. (1991). Pacific oyster nocardiosis: isolation of the bacterium and induction of laboratory infections. *Journal of Invertebrate Pathology 57*: 109–120.

Gee, A.; Elston, R.A. (1997). PCR detection of the bacterial pathogen in oyster nocardiosis. *Journal of Shellfish Research 16*: 264.



Histopathology of nocardiosis in Pacific oysters (*Crassostrea gigas*). Photos by C. Friedman.

Above: Low power view (100 x magnification) of atrophic digestive gland tubules with multiple foci of bacteria (arrows).
Below: Higher power view (200 x magnification) of four deeply stained bacterial colonies (arrows) in necrotic connective tissue, illustrating their superficial resemblance to fungal hyphae.



Disease: Paua epithelial erosion

Species and life stage affected: Juvenile paua (*Haliotis iris*).

Gross signs: Affected paua exhibit a distinct posture with a raised shell, and white patches on the epipodium, mantle, and foot. Some paua may trail thin strands of sloughed, black epithelium, lose adhesion to tank surfaces, or try to crawl out of the water.

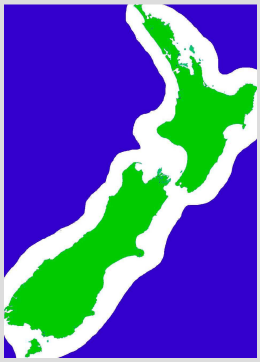
Causative agents: Opportunistic bacteria (*Flavobacterium*-like gliding bacteria, *Vibrio* sp., and perhaps others) and both motile and sessile ectocommensal ciliates (*Scyphidia* sp.).

Diagnosis: Histopathology is required to demonstrate erosion of the epithelium of the foot and mantle associated with bacteria and epicommensal ciliates, sometimes with invasion of the underlying muscle. Culturing causative agents and demonstrating their involvement is usually difficult due to the high numbers of opportunistic bacteria which quickly colonise the lesions.

Treatment and prevention: This disease is associated with poor water quality, handling damage, and/or stress. Improvement of water quality by improving water flow and removing possible sources of stress (e.g., excessive handling, elevated water temperatures) should limit spread of infections. Treatment with 50 ppm formalin for 1 hour killed ciliates but did not halt the progression of the bacterial infection (Diggles, unpublished). Antibiotic therapy may be an option if antimicrobial sensitivities of opportunistic bacteria are known, but modification of husbandry techniques is the preferred method of control.

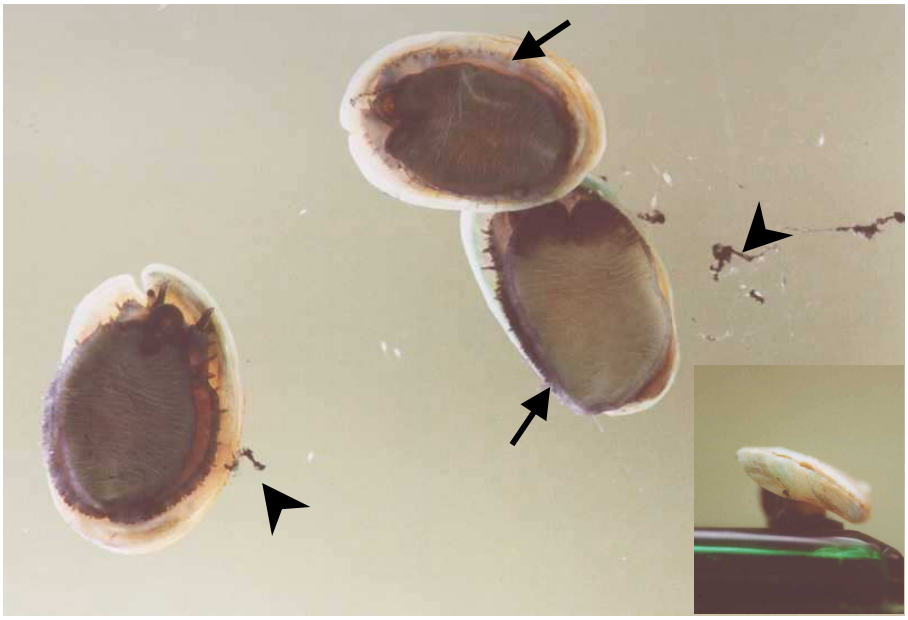
Distribution in New Zealand

Can occur in any batch of cultured paua if handled poorly or cultured under conditions of poor water quality.



Worldwide distribution: A *Flexibacter*-like infection associated with epithelial erosion has also been reported in abalone in Australia (J. Handlinger, personal communication).

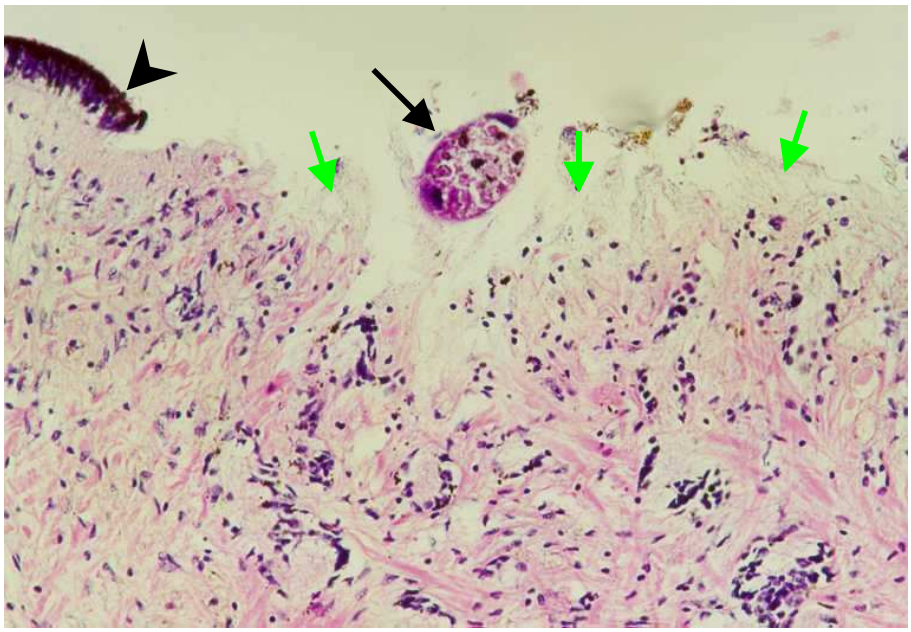
General comments: The presence of large numbers of opportunistic bacteria may result from poor water quality or infection of a wound. Once the bacteria invade the epithelium, ciliate infection appears common, usually including both motile (genera undetermined) and sessile (e.g., *Scyphidia* sp.) ciliates. Motile ciliates may not be detected by histology. It is assumed that the ciliates feed on bacteria and cellular debris in sloughing epithelium.



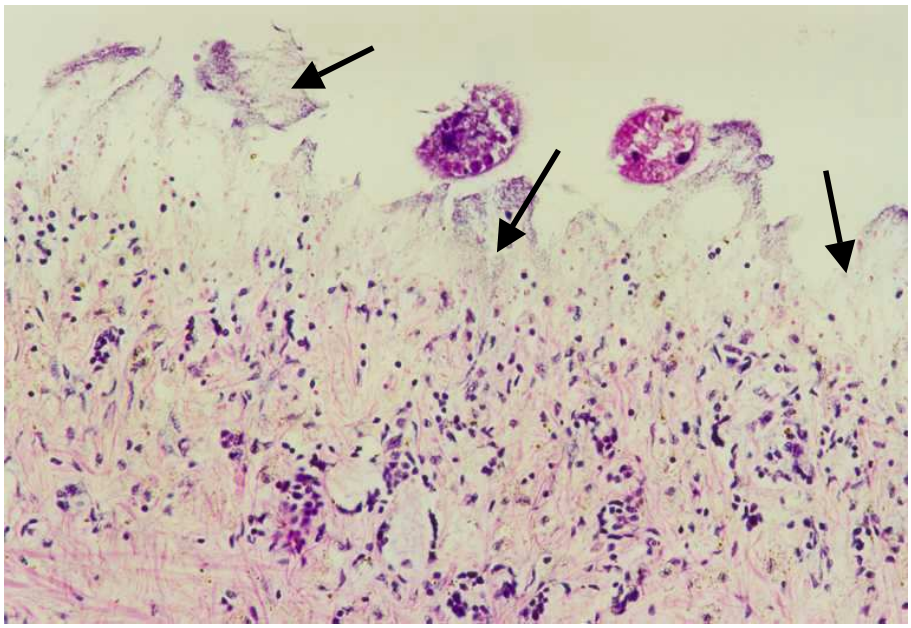
Epithelial erosion in paua (*Haliotis iris*). Photos by B. Diggles and A. Blacklock.

Above: Three juvenile paua with erosion of the epithelium of the epipodium, mantle and foot associated with bacterial infection. The whitening of the epipodium (arrows) is due to loss of black pigment (arrowheads) which has sloughed away and is adhering to the side of the aquarium.

Above (inset): Characteristic raised shell posture of affected paua.



Middle: Histology of the edge of a lesion on the epipodium, showing loss of pigment (arrowhead) due to necrosis and erosion of the epithelium associated with the presence of a ciliate (black arrow) and high numbers of bacteria (green arrows).



Below: A section of eroded, necrotic epithelium with two ciliates and large numbers of bacteria (arrows).

Disease: Paua shell mycosis

Species and life stage affected: Juvenile and adult paua (*Haliotis iris, H. australis, H. virginea*).

Gross signs: Brown, jelly-like lesions on the inner surface of the shell composed of conchiolin and fungal hyphae.

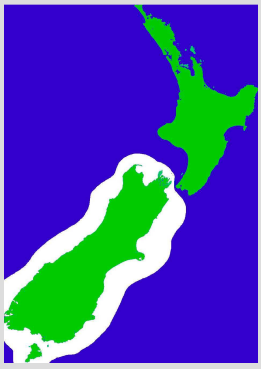
Causative agent: Opportunistic fungi.

Diagnosis: A presumptive diagnosis can be obtained by observing the presence of brown lesions on the inside of the shell, usually near the apex (Grindley et al. 1998). Definitive diagnosis involves detecting fungal hyphae using routine histopathology, or culturing fungi for identification.

Treatment and prevention: None recorded.

Distribution in New Zealand

Has been reported in wild paua throughout the South Island, and probably occurs throughout the natural range of paua.



Worldwide distribution: Fungal diseases are common in abalone worldwide. The opportunistic fungus *Haliphthoros milfordensis* causes tubercle-like lesions in the foot, mantle, and epipodium of *Haliotis sieboldii* in Japan (Hatai 1982). *Atkinsiella awabi* causes similar lesions in both greenlip (*Haliotis laevigata*) and blacklip (*Haliotis rubra*) abalone in Australia (J. Handlinger, personal communication).

General comments: In severe cases of this disease, the brown lesion may undercut the adductor muscle attachment to the shell, resulting in shell loss (Grindley et al. 1998).

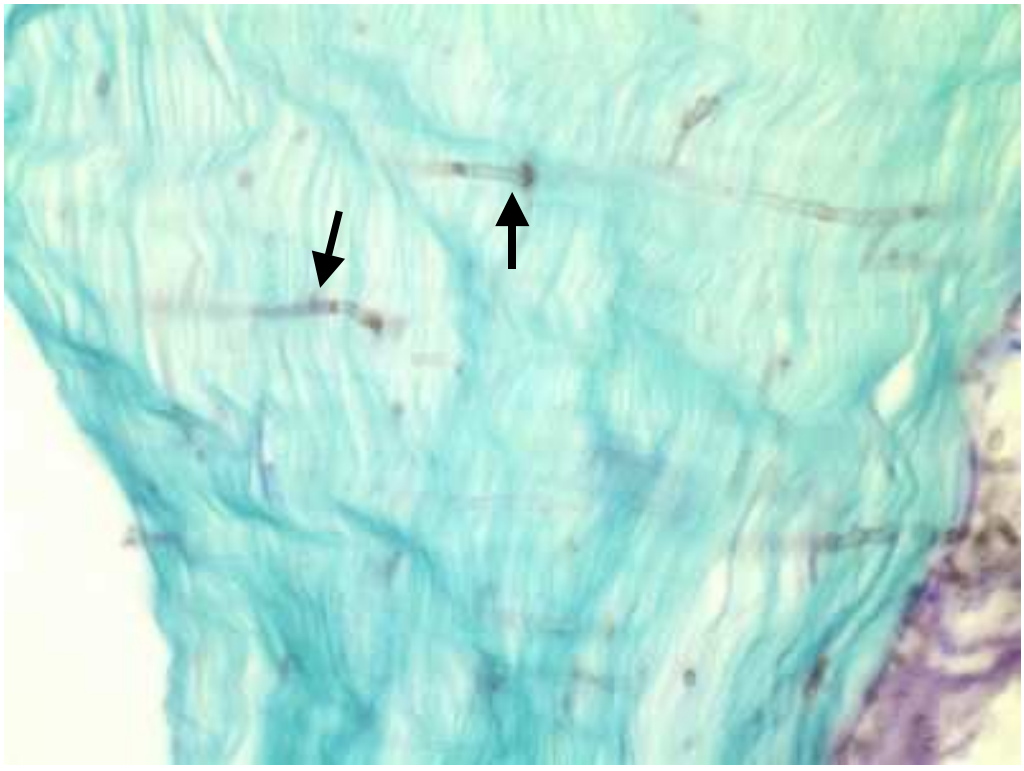
References

Grindley, R.M.; Keogh, J.A.; Friedman, C.S. (1998). Shell lesions in New Zealand *Haliotis* spp. (Mollusca, Gastropoda). *Journal of Shellfish Research 17*: 805–811.

Hatai, K. (1982). On the fungus *Haliphthoros milfordensis* isolated from temporarily held abalone (*Haliotis sieboldii*). *Fish Pathology 17*: 199–204.

Shell mycosis in paua (*Haliotis iris*).

Above: A large brown lesion near the apex of the shell of a paua, associated with infection by fungi. Photo by R. Grindley.
Below: Histology of an affected paua showing fungal hyphae (arrows) in the shell nacre staining darkly with PAS stain. 1000 x magnification. Photo by C. Friedman.



Disease: Pustule disease of abalone

Species and life stage affected: Juvenile paua (*Haliotis iris*, *H. australis*) and other species of abalone worldwide.

Gross signs: Light coloured, sometimes yellowish pustules or abscesses on the base of the foot.

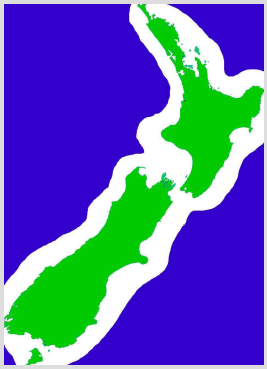
Causative agents: Wound invasion by opportunistic bacteria, usually of the genus *Vibrio*.

Diagnosis: Bacteria can be visualised using routine histopathology, or cultured from pustules using aseptic techniques for identification.

Treatment and prevention: Prevention probably by eliminating mechanical damage to foot when removing paua from raceways and tanks, possibly by using anaesthetics such as nembutal, benzocaine, or MS222 (Edwards et al. 2000, Aquilina & Roberts 2000) to relax paua before handling.

Distribution in New Zealand

Can occur in any batch of cultured paua if handled poorly during harvesting or grading.



Worldwide distribution: Diseases characterised by pustule-like lesions in the foot are probably ubiquitous and would be expected to occur whenever abalone are damaged when reared under sub-optimal conditions. Usually disease does not occur unless an open wound is present (Li et al. 1998).

General comments: Pustule disease in abalone in China is caused by infection with *Vibrio fluvialis*-II (see Liu et al. 1995, Li et al. 1998), while *V. harveyi* is more commonly associated with pustules in Australia (J. Handlinger, personal communication). Cases of pustule disease in New Zealand paua have been associated with mixed infections of *Vibrio campbellii*, *V. splendidus*-I, and an unidentified *Vibrio* sp. (B. Diggles & J. Carson, unpublished data).

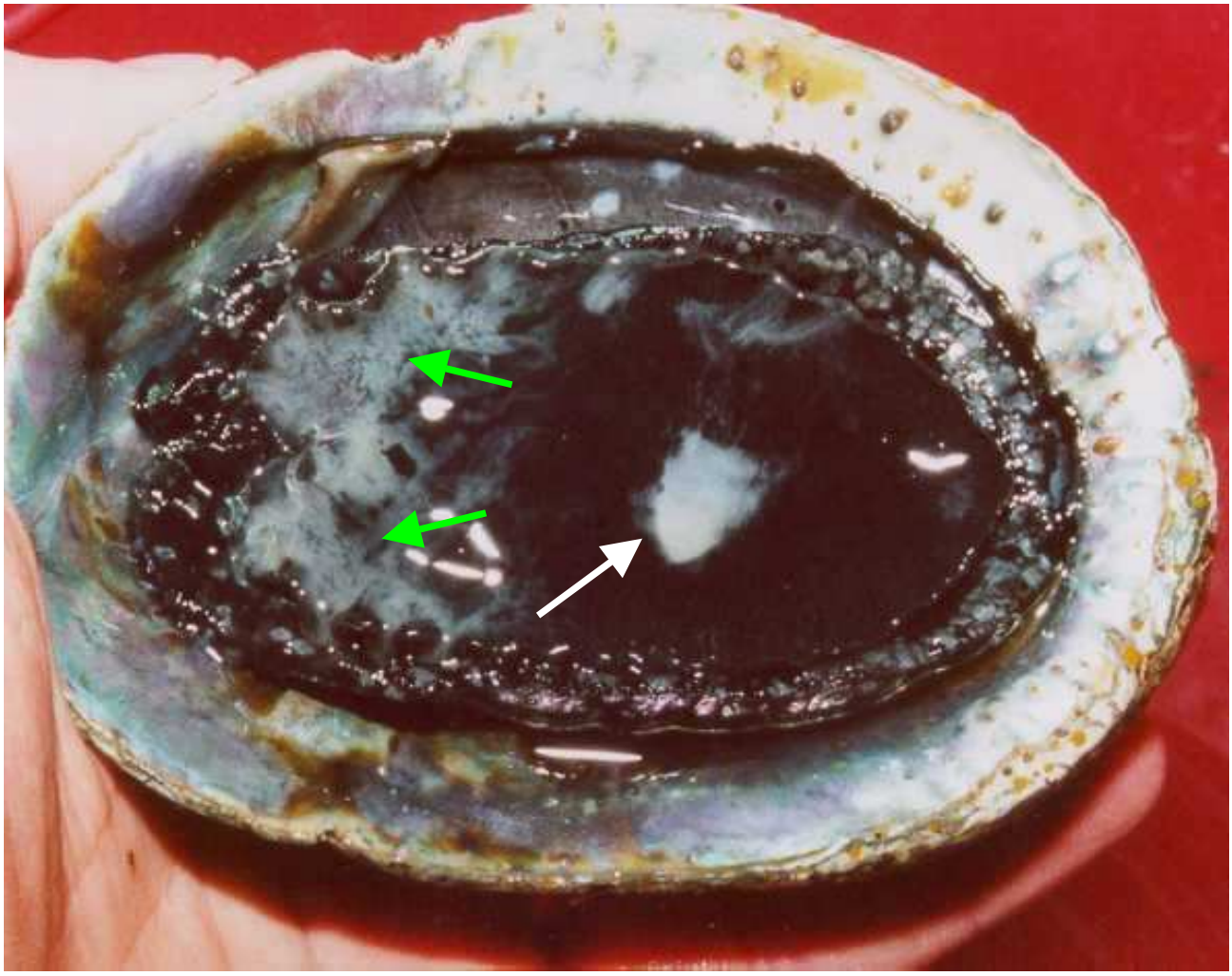
References

Aquilina, B.; Roberts, R. (2000). A method for inducing muscle relaxation in the abalone, *Haliotis iris*. *Aquaculture 190*: 403–408.

Edwards, S.; Vurke, C.; Hindrum, S.; Johns, D. (2000). Recovery and growth effects of anaesthetic and mechanical removal on greenlip (*Haliotis laevigata*) and blacklip (*Haliotis rubra*) abalone. *Journal of Shellfish Research 19*: 510.

Li, T.; Ding, M.; Zhang, J.; Xiang, J.; Liu, R. (1998). Studies of pustule disease of abalone (*Haliotis discus*) on the Dalian coast. *Journal of Shellfish Research 17*: 707–711.

Liu, J.; Nie, L.; Li, T.; Ding, M.; Mingjin, S.; Xieming, Z.R. (1995). A study of pustule disease of *Haliotis discus*. *Journal of Fisheries Science, China 2*: 78–84.



Pustule disease in paua *Haliotis iris*. Photo by B. Diggles.

On the right is a pustule caused by infection of a knife wound (white arrow); on the left is a large pustular area associated with multifocal epithelial erosion (green arrows).

Disease: Rickettsiosis

Species and life stage affected: Bluff oysters (*Ostrea chilensis*), Pacific oysters (*Crassostrea gigas*), rock oysters (*Saccostrea glomerata*), scallops (*Pecten novaezelandiae*), and cockles (*Austrovenus stutchburyi*), but not Greenshell™ or blue mussels.

Gross signs: None in oysters and cockles. Heavily affected scallops may be gaping and in poor condition.

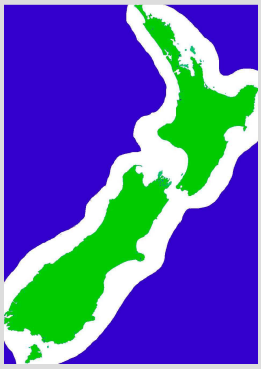
Causative agent: Unidentified species of rickettsia-like organisms (RLOs).

Diagnosis: Presumptive by histology showing basophilic inclusions in hypertrophied gill, mantle, or digestive tubule epithelium; confirmatory by electron microscopy.

Treatment and prevention: These infections usually do not cause overt disease in oysters and cockles, and neither treatment nor prevention are required in these species. However, heavy RLO infections in the gills are associated with mass mortalities in wild scallops (Diggles et al. 2000). There are no known methods for treatment and prevention of disease in scallops, as disease may be triggered by environmental stressors.

Distribution in New Zealand

Ubiquitous in flat oysters, Pacific oysters, rock oysters, scallops, and cockles, but not mussels.



Worldwide distribution: Ubiquitous worldwide.

General comments: Rickettsiae are distinguished from other bacteria in that they are obligate intracellular parasites, which usually cause hypertrophy of the host cell. Their most common site is the epithelial cells lining the digestive ducts and tubules. They do not cause overt disease in oysters and cockles, but in scallops heavy RLO infections are associated with mass mortalities (Gulka et al. 1983, Leibovitz et al. 1984, Le Gall et al. 1988, 1991, Diggles et al. 2000). Mortalities are particularly associated with gill infections, but such infections also occur at high prevalence (up to 100%) in apparently healthy scallops. It appears that it is not the presence of the bacteria that determines disease, but the intensity of infection, which may be related to environmental stressors. This pattern of high prevalence, and often high intensity, of RLO infection occurs in *P. novazelandiae*. As 23–39% of New Zealand wild scallops may experience mortalities each year (Bull 1976), these mortalities may be associated with RLO infection.

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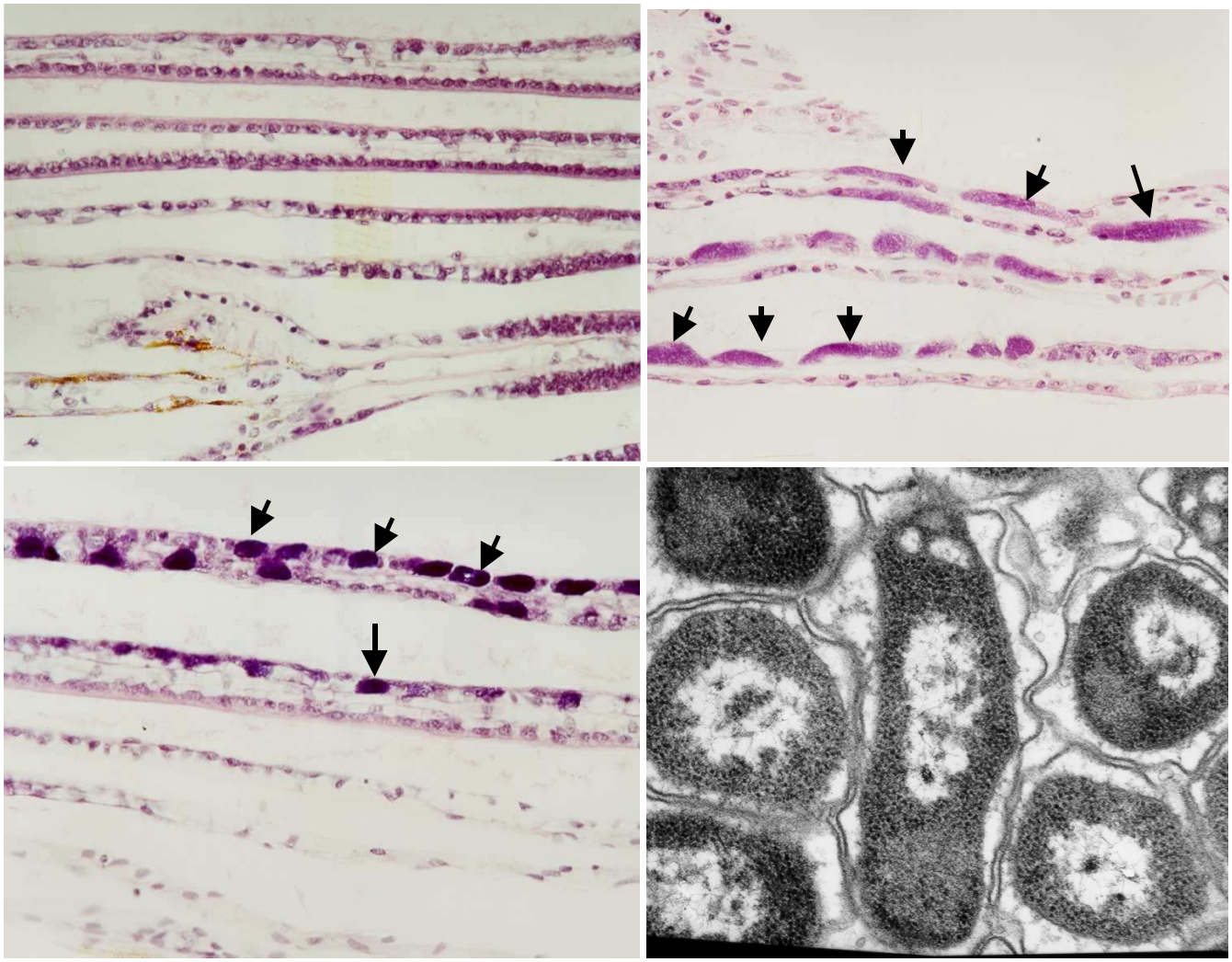


Rickettsiosis in scallops (*Pecten novaezelandiae*). Photos by B. Diggles.

Above left: Histology of normal gill epithelium of a scallop *Pecten novaezelandiae*. **Above right:** Histology of gill epithelium of a scallop heavily infected with RLOs, which occur inside markedly hypertrophied cells (arrows). **Below left:** Another type of RLO inside intensely basophilic epithelial cells (arrows) in scallop gill. **Below right:** TEM of RLOs in scallop gill.

Disease: Withering syndrome of abalone

Species and life stage affected: Juveniles and adult abalone, including *Haliotis cracherodii, H. rufescens,* and *H. fulgens, H. corrugata*.

Gross signs: Shrinkage of the foot, retraction of the visceral tissues, and inability to adhere to the substrate.

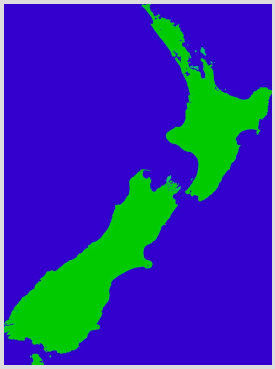
Causative agent*:* "*Candidatus* Xenohaliotis californiensis", a rickettsial organism (Friedman et al. 2000).

Diagnosis: Light microscopy shows large numbers of rickettsial organisms in epithelial cells lining the gut. Confirmation of identity by PCR (Andree et al. 2000) or ISH (Antonio et al. 2000).

Treatment and prevention: Slaughter and disinfection is standard procedure when infected stocks are detected, but treatment with oxytetracycline can halt the disease (Shields & Friedman 1999). Prevention by treatment of intake water is recommended in areas where withering syndrome is endemic.

Distribution in New Zealand

Withering syndrome has not been reported from New Zealand, but rickettsial inclusions are occasionally recorded in the gut epithelium of paua.



WITHERING SYNDROME IS AN EXOTIC DISEASE. IF YOU SUSPECT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: West coast of the United States.

General comments: Withering syndrome is a chronic disease. Affected abalone waste away through impairment of digestion due to the abundance of organisms in cells lining the gut. Since 1995 it has been accepted that the rickettsial organisms are the cause of the disease (Gardner et al. 1995), but why the disease occurred is unclear. It is generally thought that climate change, particularly El Niño events, may have favoured the emergence of this disease (Martinez et al. 2000). Rickettsia-like inclusions have been observed in the gut epithelium of paua in New Zealand, but to date have not been associated with disease.

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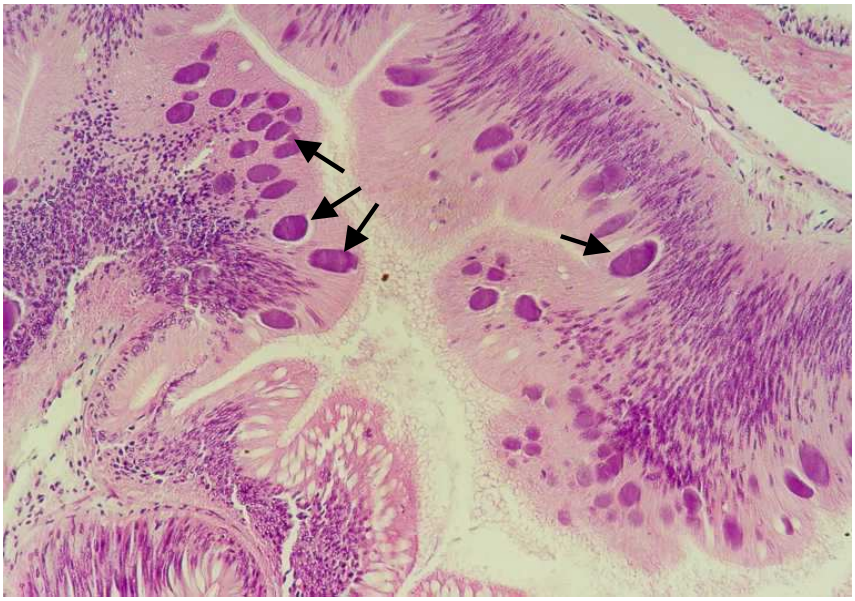
Withering syndrome in abalone (*Haliotis* sp.) from theU.S.

Above: The red abalone (*H. rufescens*) on the right is normal; the one on the left has withering syndrome. Photo by J. Moore. **Below:** Histopathology of the gut of a black abalone (*H. cracherodii*) with withering syndrome. Note numerous large basophilic inclusions (arrows) filled with rickettsial organisms in the epithelial cells lining the gut. Photo by B. Diggles.



Disease: APX (Apicomplexan Parasite X)

Species and life stage affected: Adults and juveniles of Bluff oysters (*Ostrea chilensis*) and Greenshell™ mussels (*Perna canaliculus*).

Gross signs: Severely infected Bluff oysters may gape, and are small, shrunken, pale and translucent due to lack of gametes in the gonads.

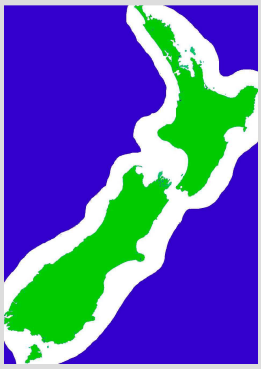
Causative agent: An undescribed apicomplexan.

Diagnosis: Histology, or whole body imprints in heavy infections.

Treatment and prevention: No treatment available. As only one stage of the parasite life-cycle occurs in bivalves, it probably uses another host, possibly a terebellid polychaete worm. If an intermediate host can be identified, it may be possible to break the life-cycle by separating the hosts.

Distribution in New Zealand

APX occurs in oysters all around New Zealand, with the exception of the Chatham Islands. In mussels, it has been found only on two farms in the Marlborough Sounds.



Worldwide distribution: Known only from New Zealand. Apicomplexans similar to APX have not been reported from any other host.

General comments: After *Bonamia*, this is possibly the most serious disease of Bluff oysters in New Zealand. APX occurs in virtually all Bluff oysters around the South Island, particularly in Foveaux Strait, and in the south of the North Island, but normally at very low infection intensities (few organisms per oyster). Disease is related to intensity of infection rather than presence or absence of APX. Heavy infections may occur in otherwise apparently healthy oysters, and may predispose those oysters to bonamiosis. *Bonamia* does not appear to predispose the oysters to APX. The disease course appears to be chronic, and heavily infected oysters may gape for a long time before death, resulting in fouling of the insides of the valves.

Reference

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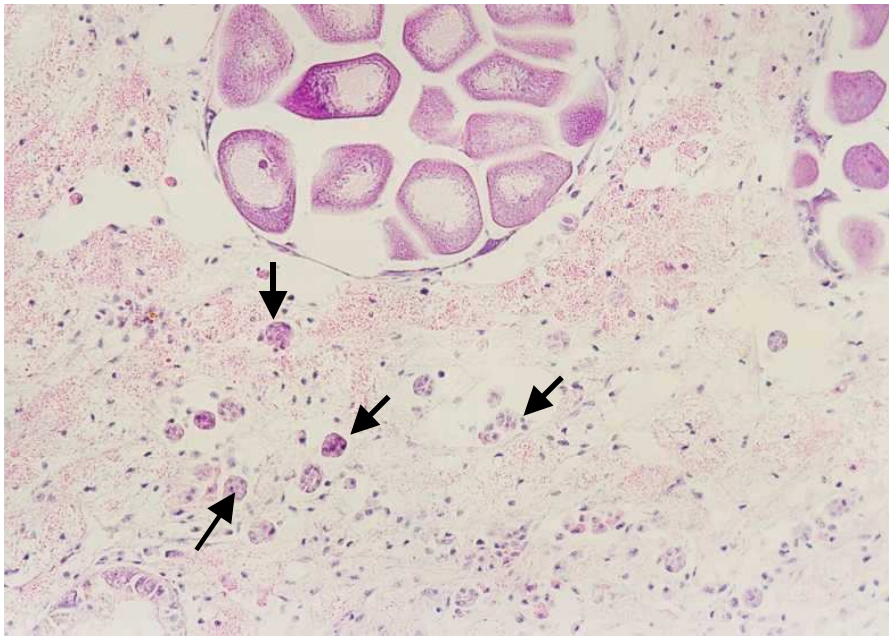
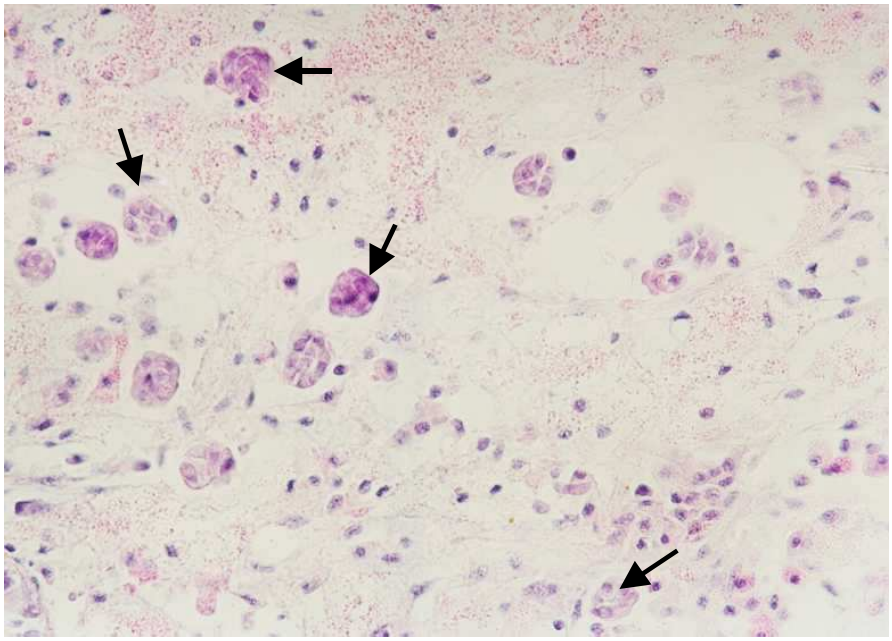
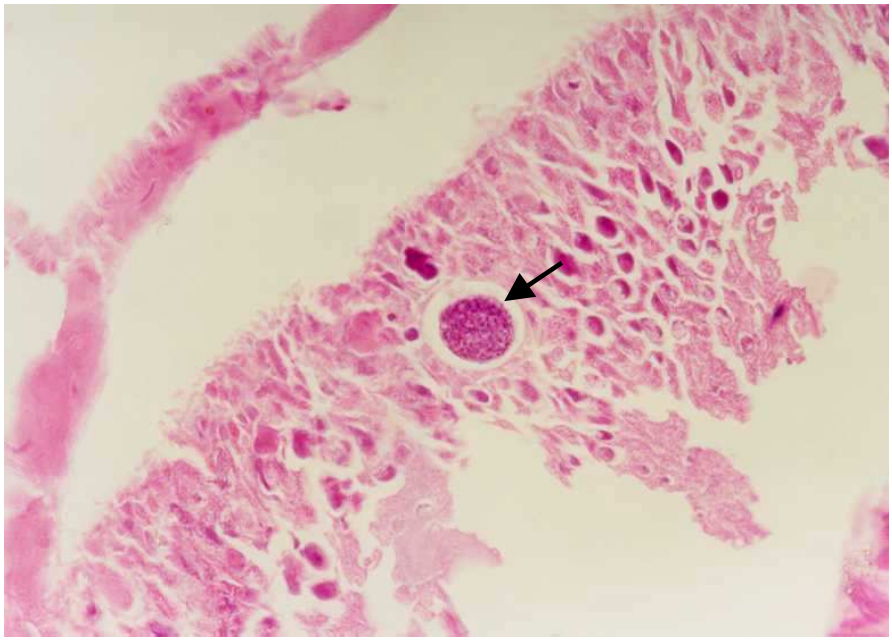






APX infections in Greenshell™ mussels (*Perna canaliculus*).
Photos by B. Diggles.

Above: Histological section through the gonad with a number of APX zoites present (arrows).

Middle: Higher power view of the gonad showing a number of paired zoites of the APX parasite

Below: A oocyst-like body (arrow) in the gut epithelium of a terebellid worm, a possible intermediate host for the APX parasite.

Disease: Bonamiosis

Species and life stage affected: Flat oysters (genus *Ostrea*), including Bluff oysters (*Ostrea chilensis*).

Gross signs: Gaping, loss of condition, gill lesions, swelling of the heart, pale, shrunken digestive gland, chronic mortalities.

Causative agent: *Bonamia exitiosus* Hine et al. (2001) in New Zealand, *Bonamia ostreae* in the Northern Hemisphere.

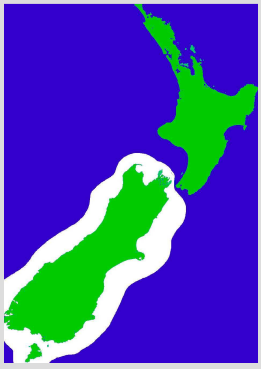
Diagnosis: Heavy to moderate infections can be diagnosed using light microscopy, by examining stained heart smears or tissue sections for small (2–3 µm) protozoans. Light infections require PCR (Carnegie et al. 2000) or ISH (Cochennec et al. 2000).

Treatment and prevention: No known treatment. Slaughter, safe disposal, and disinfection required to prevent spread of infections.

Distribution in New Zealand

In oysters all around the South Island, Stewart Island, and in Wellington Harbour. Not in the Chatham Islands.

BONAMIOSIS IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Bonamiosis affects juveniles and adults of all flat oysters, including *Ostrea angasi* in southern Australia and *Ostrea edulis* in the U.S. and Europe. *Bonamia ostreae* has destroyed the flat oyster farming industries of Europe, but is thought to have originated on the east coast of the U.S. From there it was probably moved to the west coast of the U.S. with movement of oysters. In the late 1970s infected oysters were moved from the west coast of the U.S. to France. From there it spread to the Netherlands, the U.K., Spain, Portugal, Italy, and the Adriatic Sea.

General comments: *Bonamia* sp. are parasites of the haemocytes of oysters. The parasite transmits directly, oyster to oyster. When *Bonamia* enters an uninfected oyster, the haemocytes recognise it as foreign and engulf it. Once inside the haemocyte, *Bonamia* grows and feeds on the cytoplasm of the cell. It divides many times and up to 24 *Bonamia* may be found in one haemocyte. The haemocyte bursts, the *Bonamia* are released, and they are engulfed by other haemocytes and carry on the cycle. The oyster stops producing eggs or sperm, and instead puts energy into producing more haemocytes, further favouring the parasite. Eventually the oyster dies of exhaustion. *Bonamia exitiosus* appears to have an annual pattern of infection. In Foveaux Strait it is usually hard to detect in late winter/early spring, and numbers increase with the spawning of predominantly male oysters in November–January. However, *B. exitiosus* appears to rely heavily on the lipid reserves in oyster eggs, and it is during absorption of unspawned eggs in the 25% of female oysters that do not spawn, that it uses host lipid and reaches its highest levels.

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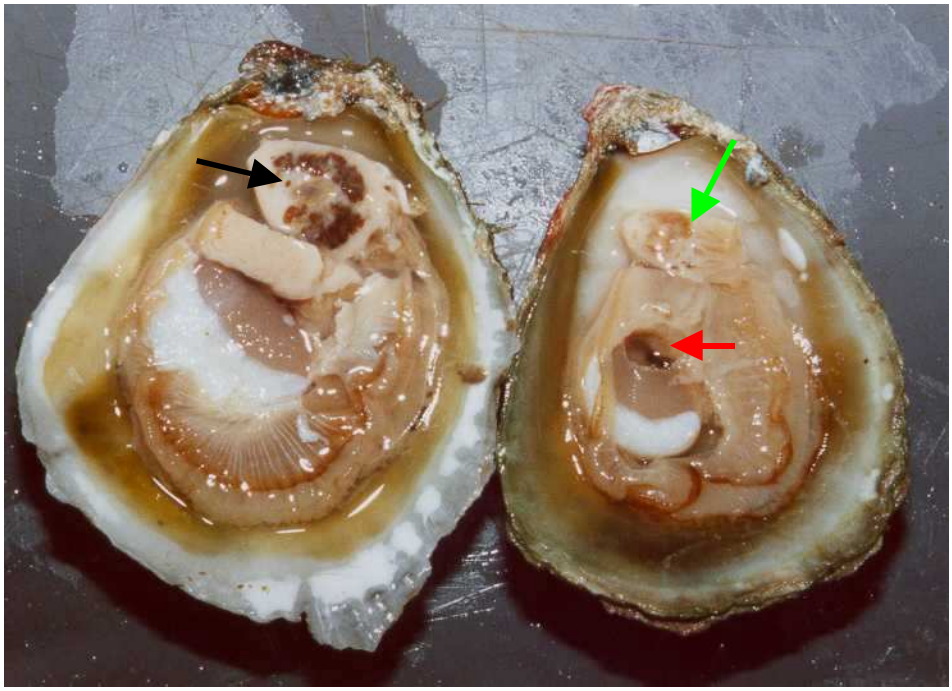
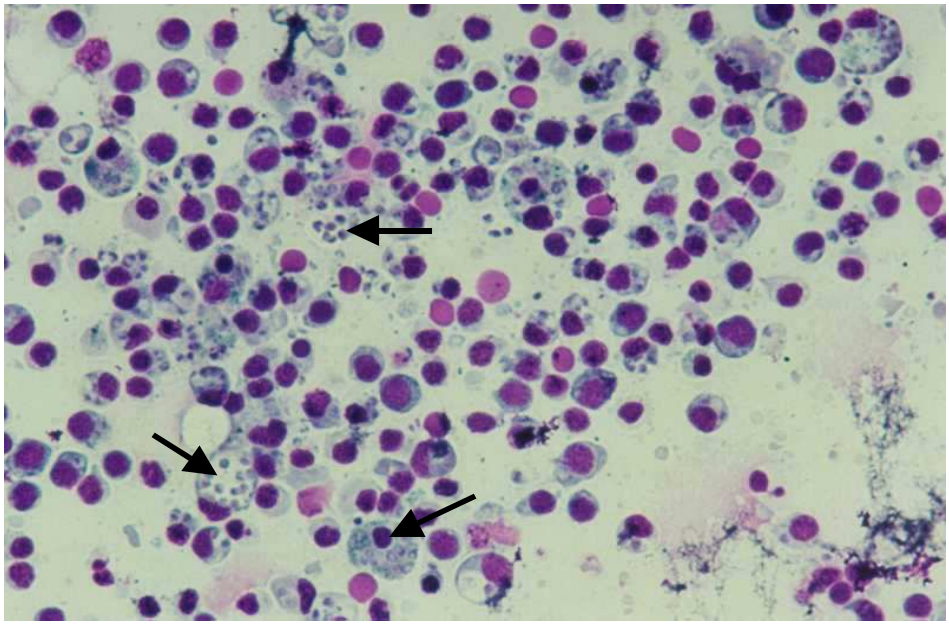


Bonamiosis in Bluff oysters (*Ostrea chilensis*).

Above: The oyster on the left appears healthy with a dark digestive gland and normal sized gonad (black arrow). The oyster on the right is infected with *Bonamia exitiosus*. It has a gonad and digestive gland which are comparatively small and pale (green arrow), and an enlarged heart (red arrow). Photo by B. Diggles.



Below: Stained heart imprint showing heavy infection of haemocytes with numerous *B. exitiosus* (arrows). Photo by M. Hine.

Disease: Haplosporidiosis (MSX, SSO)

Species and life stage affected: Juveniles and adults of eastern oysters (*Crassostrea virginica*) and Pacific oysters (*C. gigas*).

Gross signs: Weakened condition, large scale mortalities during the summer months.

Causative agent: *Haplosporidium nelsoni* (in both hosts), and *H. costale* (in *C. virginica*), both belonging to the Haplosporidia.

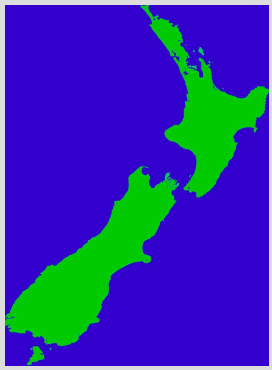
Diagnosis: Histology in moderate to heavy infections, PCR-ELISA and ISH using specific DNA probes or multiplex PCR (Russell et al. 2000) for both species.

Treatment and prevention: No treatment available. Controls on movements, slaughter and disinfection of infected stock and use of resistant oysters minimise losses. Can be excluded from hatcheries by filtration and UV sterilisation of incoming water (Ford et al. 2001).

Distribution in New Zealand

Unreported, despite examination of large numbers of Pacific oysters.

HAPLOSPORIDIOSIS IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: *H. nelsoni* originated in Japan (Friedman 1996), but was moved to the west coast of the U.S. with movement of Pacific oysters. It was then moved to the east coast of the U.S. where it jumped host into *C. virginica* and began killing oysters in 1957 (Burreson et al. 2000). More recently *H. nelsoni* has been reported from Pacific oysters in France (Renault et al. 2000), and it may have been introduced there with the importation of Pacific oysters into France to replace *Ostrea edulis* which had been devastated by *Marteilia refringens* and *Bonamia ostreae*. *H. costale* appears to be a long-standing parasite of *C. virginica* on the east coast of the U.S.

General comments: Haplosporidiosis is an OIE internationally notifiable disease, because of its devastating impact on the eastern U.S. oyster fishery and introduction to new areas with movements of Pacific oysters for aquaculture. As Pacific oysters are the natural host of *H. nelsoni*, it may occur at low prevalence and intensity levels, making it difficult to detect. Once the *Haplosporidium* spp. have infected the host, they move between the cells of the connective tissue until sporulation begins. For *H. nelsoni*, sporulation occurs in the epithelium of the digestive tract, particularly in small oysters; for *H. costale*, sporulation occurs in the connective tissue. Neither species transmits directly oyster to oyster, and therefore it is suspected they have an intermediate host. Attempts to identify those hosts have so far been unsuccessful. Currently it is thought that they may be planktonic.

References

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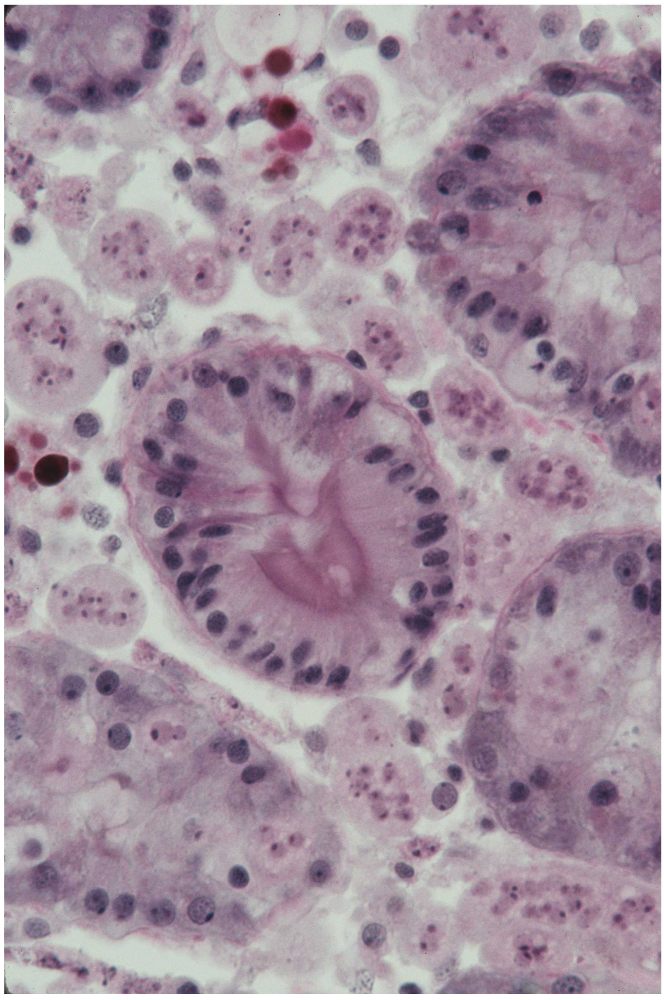
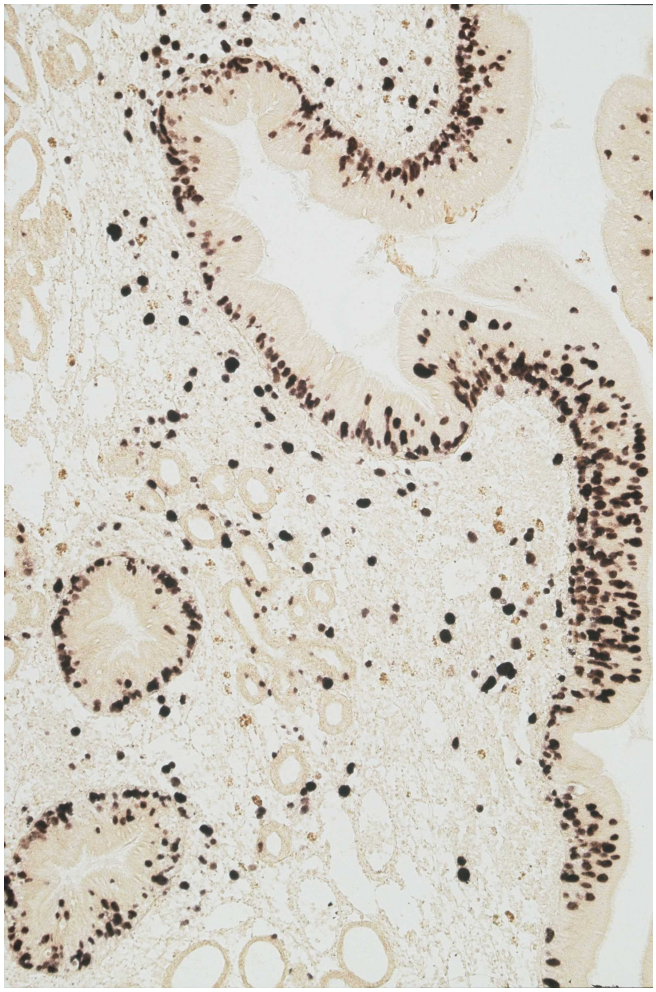
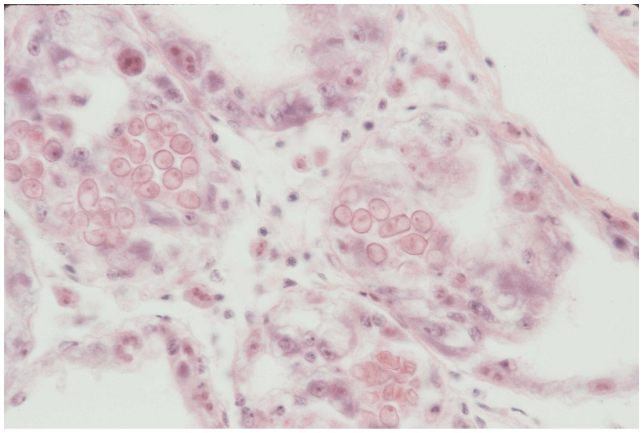






MSX disease in eastern oyster (*Crassostrea virginica*) from theU.S. Photos by E. Burreson.

Above left: Plastic histological section showing numerous MSX plasmodia between digestive gland tubules.

Above right: ISH of a heavily infected oyster. Each black signal is a MSX plasmodium.

Left: Spores of MSX in the digestive gland epithelium.

Disease: *Marteilioides* infection of oyster eggs

Species and life stage affected: The parasite infects the ova of adult Pacific oysters (*Crassostrea gigas*) and *Saccostrea echinata*.

Gross signs: Failure to spawn. In heavy infections there may be enlargement of the ovarian follicles.

Causative agent: *Marteilioides chungmuensis*, a paramyxean parasite.

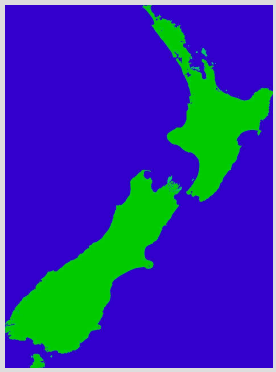
Diagnosis: The parasite is readily detectable in histological sections of the gonad by light microscopy.

Treatment and prevention: No known treatment. Prevention is by controls on movements of infected oysters, destruction of infected stocks, and disinfection of affected facilities.

Distribution in New Zealand

Unreported.

MARTEILIOIDES IS AN EXOTIC PATHOGEN. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Japan, Korea, China, northern Australia, and the west coast of the U.S.

General comments: This parasite may kill its host (Imai et al. 1968), but its greatest impact is on the fecundity of the host, as it causes spawning failure (Matsuzato et al. 1977). It may be spread by shipping, possibly by oysters on ships' hulls, because in northern Australia it is restricted to the wharves in the port of Darwin (Hine & Thorne 2000), and in the U.S. to the port of Eureka (Becker & Pauley 1968).

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No photographs currently available

Disease: Marteiliosis (Aber disease (*Marteilia refringens*), QX disease (*Marteilia sydneyi*))

Species and life stage affected: *Marteilia refringens* infects flat oysters (*Ostrea edulis, O. angasi, O. chilensis*) and blue mussels (*Mytilus edulis*). *Marteilia sydneyi* infects Sydney rock oysters (*Saccostrea glomerulata*).

Gross signs: Poor condition with a translucent emaciated digestive gland, cessation of growth, tissue necrosis, and mortalities.

Causative agent: *Marteilia* spp. belong to a group called the Paramyxea, which also infect other marine invertebrates. The Paramyxea are not closely related to any other group (Berthe et al. 2000).

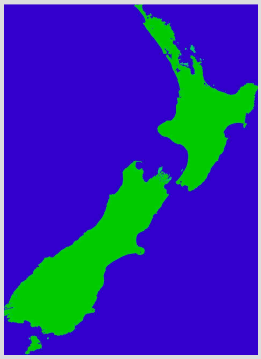
Diagnosis: Light microscopy (histology) is normally sufficient to diagnose these diseases, as the causative organisms are relatively large. Very light or early infections may be missed using light microscopy, but molecular techniques based on PCR and ISH have been developed to detect light infections (Anderson et al. 1995, Le Roux et al. 1999, Kleeman & Adlard 2000).

Treatment and prevention: Restrictions on movements, and for *M. refringens*, avoidance of planting seed during the transmission time. These parasites do not thrive at high salinities (35–37‰), and this can be used to inhibit disease.

Distribution in New Zealand

Unreported. As these parasites are relatively easy to diagnose, New Zealand is almost certainly free of them.

MARTEILIOSIS IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: *Marteilia refringens* occurs in France, Greece, Italy, Morocco, Portugal, and Spain, and *M. sydneyi* in Australia (New South Wales, Queensland, Western Australia).

General comments: Other species of *Marteilia* may also cause disease, but as the classification of these organisms is confused, their relationship to the two species presented here is uncertain. Marteiliosis caused by *M. refringens* and *M. sydneyi*, is an OIE internationally notifiable disease. The life-cycles of *Marteilia* spp. are unknown, but the diseases cannot be transmitted directly animal to animal (Roubal et al. 1989, Berthe et al. 1998), suggesting that the parasite may have an intermediate host.

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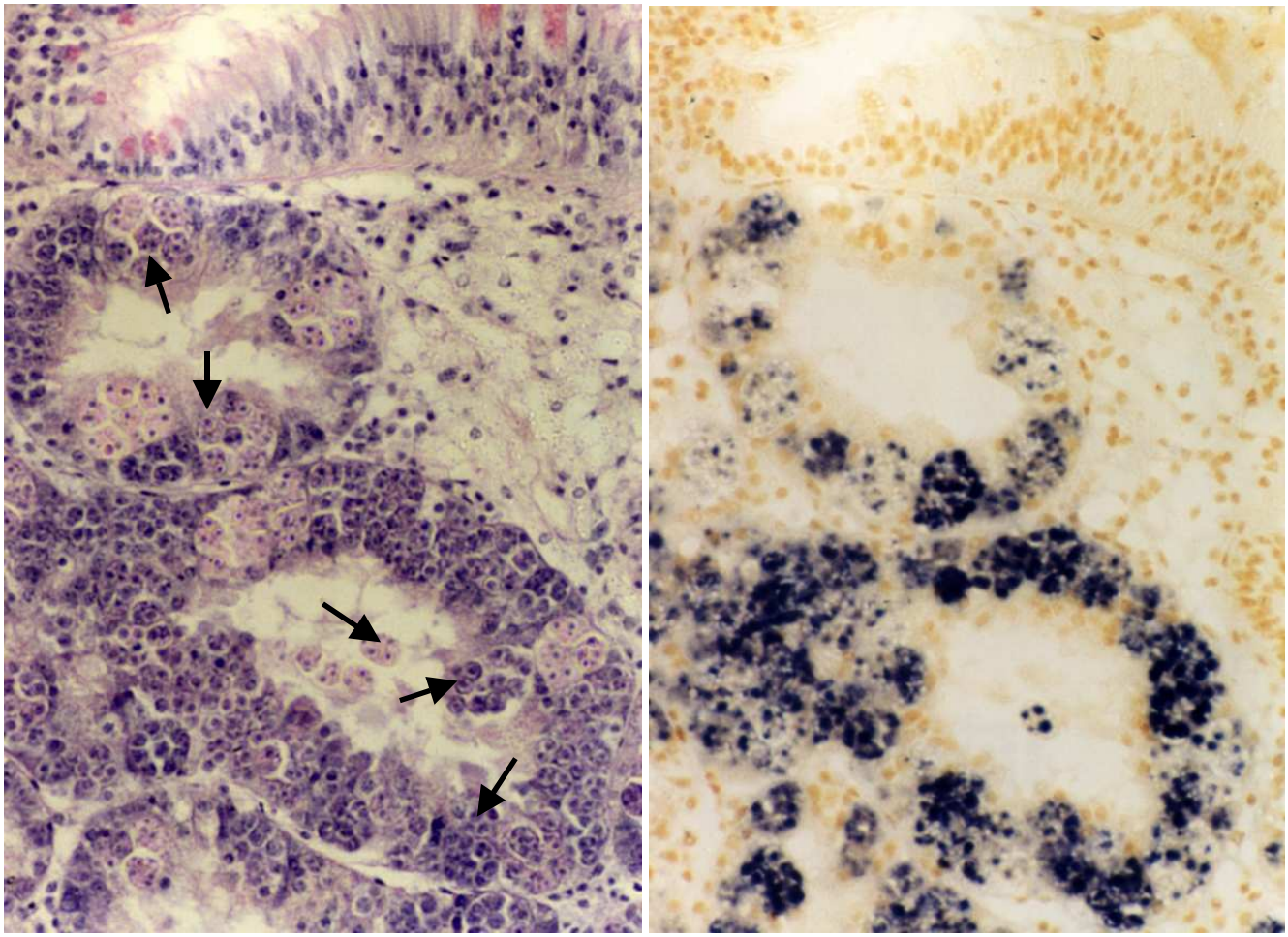


QX disease in the Sydney rock oyster, *Saccostrea glomerulata*. Photos by S. Kleeman.

Left: Histological section of the remains of the epithelium of a digestive gland tubule heavily infected with various developmental stages of QX (arrows). **Right:** The appearance of heavily infected tubules using an ISH method specific for QX. Each black spot is a QX developmental stage.

Disease: Mikrocytosis (Denman Island disease)

Species and life stage affected: Adults of Pacific oysters (*Crassostrea gigas*), and possibly *Ostrea edulis, O. conchaphila*, and eastern oysters (*C. virginica*).

Gross signs: Green pustules under 5 mm in diameter within the body wall or on the surface of the labial palps and mantle. Often there is a brown scar on the shell adjacent to the lesion.

Causative agent: *Mikrocytos mackini,* a protozoan parasite of uncertain affinity.

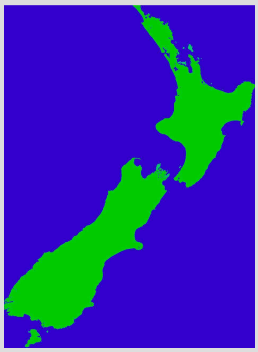
Diagnosis: In heavy infections, histology of sections through lesions shows oyster blood cells to be infected with small round unicellular parasites. Light infections are hard to detect with certainty, and problems with purifying the parasite have prevented development of molecular tools.

Treatment and prevention: The parasite is restricted to temperatures below 15 °C, so maintenance of oysters above 15 °C can be used to control or eliminate the disease.

Distribution in New Zealand

Unreported.

MIKROCYTOSIS IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966



Worldwide distribution: Around islands off the west coast of Canada.

General comments: Gross signs of mikrocytosis may be confused with those of Pacific oyster nocardiosis (see p. 152), but the latter is a warm water pathogen. Recently developed techniques which have allowed purification of *Mikrocytos mackini* (see Joly et al. 2001) should speed the development of sensitive molecular diagnostic tools for this disease.

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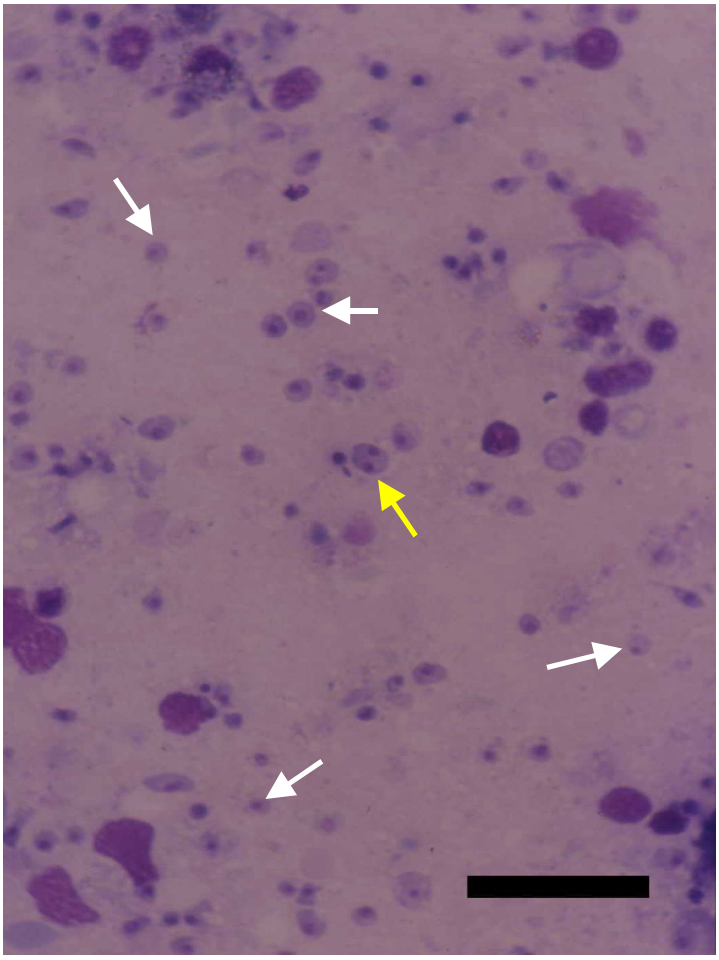
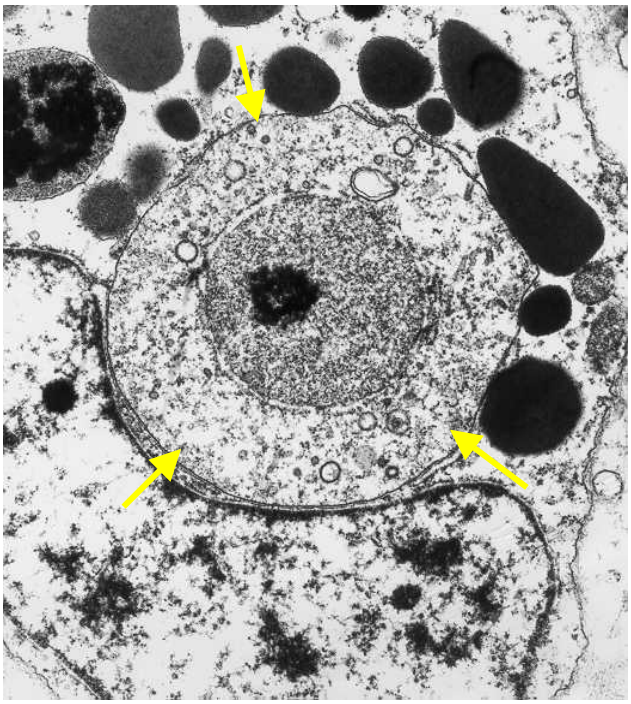


Mikrocytosis (Denman Island disease) in Pacific oysters, *Crassostrea gigas*.

Above: A stained imprint from an experimentally infected oyster, showing numerous *M. mackini* microcells (white arrows), including dividing forms (yellow arrow). Scale bar = 10 µm. Photo by R. Adlard.
Below: TEM of a microcell of *M. mackini* (arrows) inside a leydig cell. Photo by M. Hine.



Disease: Mikrocytosis (Winter mortality)

Species and life stage affected: Juveniles and adults of Sydney rock oysters (*Saccostrea glomerulata*).

Gross signs: Focal abscess-like lesions in the gills, connective tissue, gonads and alimentary tract.

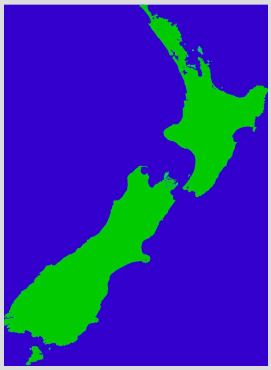
Causative agent: *Mikrocytos roughleyi*, a protozoan parasite belonging to the Haplosporidia.

Diagnosis: Heavy infections may be diagnosed from gross signs and identification by histology of microcells, (1–2 µm in diameter) with a spherical nucleus 1 µm in diameter inside haemocytes. Light infections are extremely difficult to diagnose, but molecular tools are being developed (Adlard & Lester 1995).

Treatment and prevention: *Mikrocytos roughleyi* thrives at temperatures below 15 °C and high salinities (30–35‰), and holding oysters at higher temperatures and reduced salinity will contain or destroy the parasite.

Distribution in New Zealand

Unreported, even though surveys of New Zealand *S. glomerata* have been carried out specifically to look for this parasite.



MIKROCYTOSIS IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: New South Wales, Australia.

General comments: Relatively little is known about this parasite, and its ultrastructure is as yet unpublished. However, preliminary observations confirm that it is a haplosporidian (the same group as *Haplosporidium* and *Bonamia*), and molecular data suggest that it is probably another species of *Bonamia*. *Mikrocytos mackini* (Denman Island disease) is not a haplosporidian, and it is therefore not closely related to *M. roughleyi*. As *M. mackini* was described first (Farley et al. 1988), it will retain that name and *M. roughleyi* will be reclassified.

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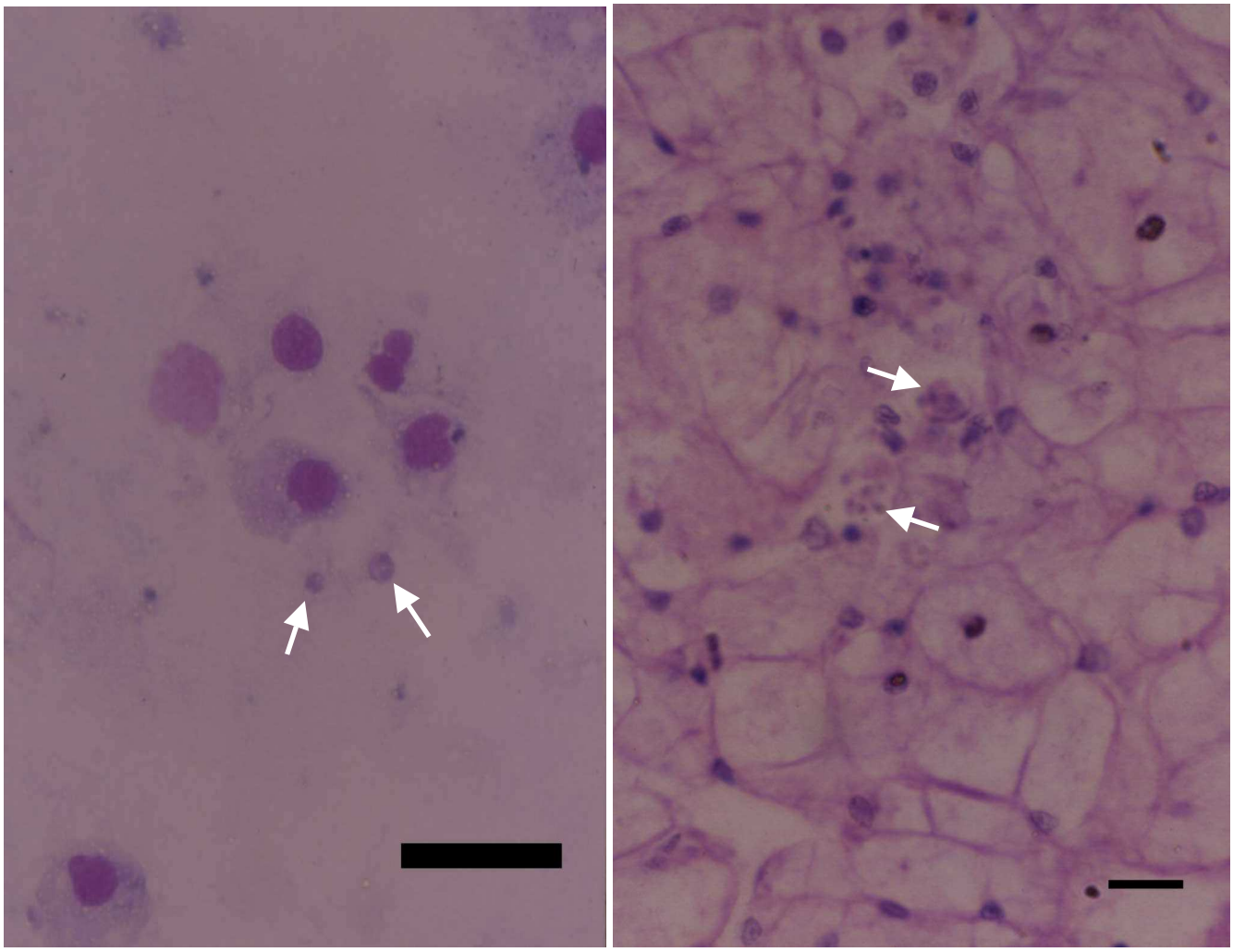


Winter mortality in Sydney rock oysters (*Saccostrea glomerulata*). Photos by R. Adlard.

Left: *Mikrocytos roughleyi* microcells (arrows) in a stained imprint.
Right: Histology showing microcells of *M. roughleyi* (arrows) in host gonad. Scale bars = 10 µm.

Disease: Paua haplosporidosis

Species and life stage affected: Juvenile and adult paua (*Haliotis iris*).

Gross signs: Lethargy, loss of righting reflex, poor adherence to surfaces, wasting of the foot, pale areas in the epipodium in heavy infections, chronic mortalities of juveniles during the summer months.

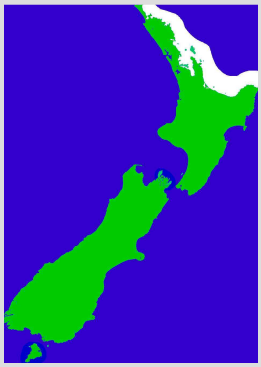
Causative agent: An unnamed haplosporidian parasite with intracellular rickettsia-like organisms.

Diagnosis: Multinucleate plasmodia of the parasite are relatively large and easy to detect using histology as they normally are surrounded by a clear space or halo in tissue sections. Spores occur in the right kidney of adult paua and are variably acid fast using Zeihl Neelsen stain. Plasmodia are detectable in wet smears of haemolymph in advanced infections using light microscopy.

Treatment and prevention: No known effective method of treatment. Chlorine and freshwater baths increased losses, because diseased animals were weaker than healthy individuals. Wild-caught adult paua may act as reservoirs of infection, hence separation of broodstock paua from juveniles and filtration plus UV sterilisation of incoming seawater to prevent entry of infective stages (Ford et al. 2001) is recommended.

Distribution in New Zealand

Known only from the northeast coast of the North Island.



Worldwide distribution: New Zealand only.

General comments: A severe chronic disease to which juvenile paua are particularly susceptible. Losses of 80–90% of juvenile paua have been associated with this disease in affected culture facilities. Wild-caught adult paua used as broodstock may be asymptomatic carriers of the disease and could be a possible source of infection of juveniles in culture facilities. Whether transmission of the disease is direct from paua to paua, or requires an intermediate host, remains unknown.

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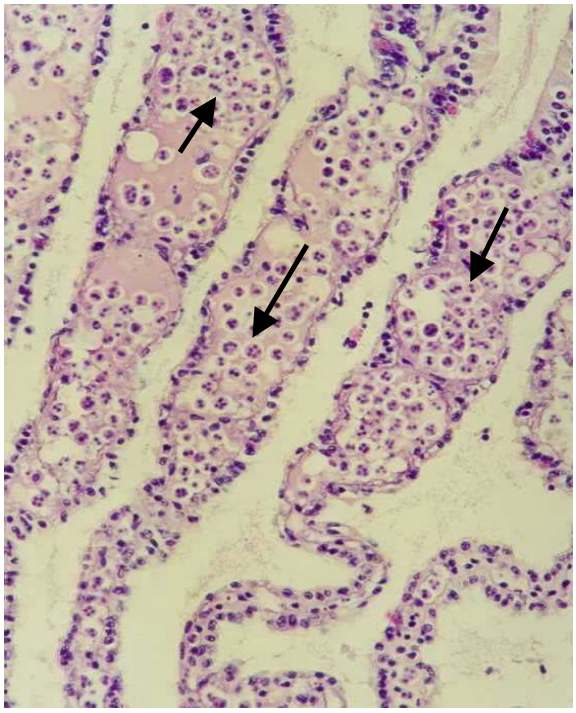
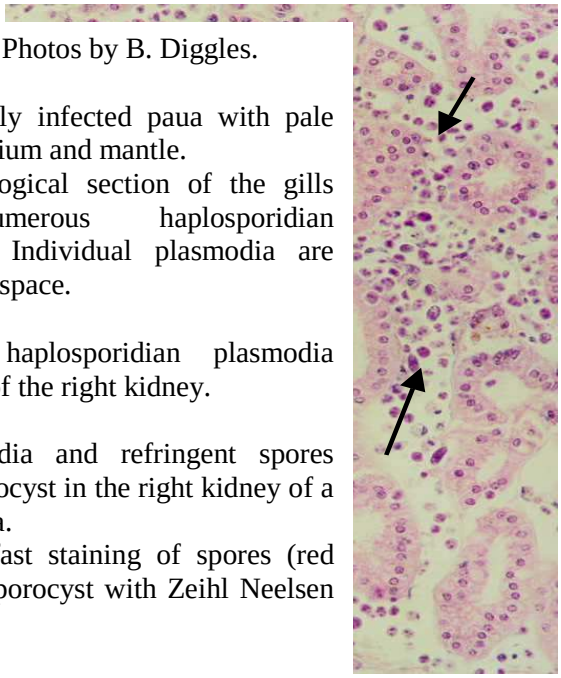


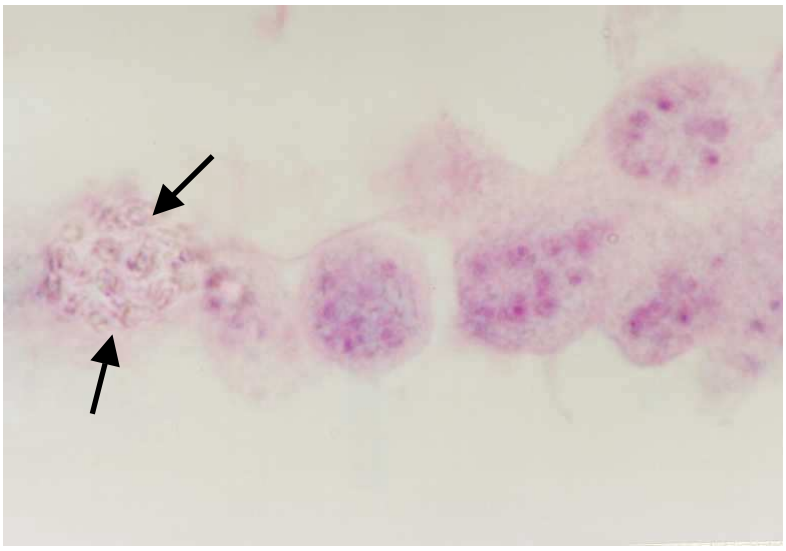


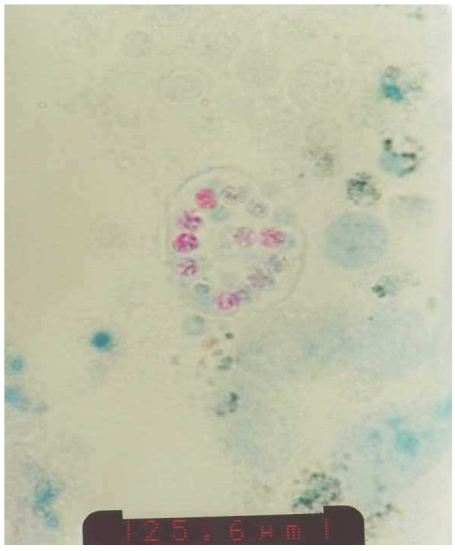
Paua haplosporidosis. Photos by B. Diggles.

Above left: A heavily infected paua with pale blotches in the epipodium and mantle.
Above right: Histological section of the gills demonstrating numerous haplosporidian plasmodia (arrows). Individual plasmodia are surrounded by a clear space.

Right: Numerous haplosporidian plasmodia (arrows) in a section of the right kidney.

Below left: Plasmodia and refringent spores (arrows) inside a sporocyst in the right kidney of a wild caught adult paua.
Below right: Acid fast staining of spores (red coloration) inside a sporocyst with Zeihl Neelsen stain.





Disease: Perkinsosis (*Perkinsus olseni/atlanticus*)

Species and life stage affected: Cockles (*Austrovenus stutchburyi*), wedge shells (*Macomona liliana*), pipi (*Paphies australis*), and *Barbatia novaezelandiae*. Also Australian abalone, *Haliotis ruber*.

Gross signs: Sometimes soft yellow abscesses are evident in the flesh, but mostly no gross signs.

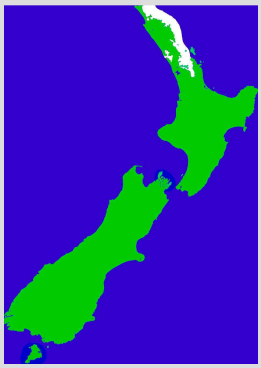
Causative agent: *Perkinsus olseni*, a parasitic dinoflagellate-like protozoan.

Diagnosis: Incubation in Ray's fluid thioglycollate medium (RFTM) followed by staining with lugols iodine to detect enlarged spherical hypnospores is the most effective means of diagnosis (Bushek et al. 1994). *Perkinsus* is also easy to detect with histology using Periodic Acid-Schiff (PAS) stain to demonstrate the PAS-positive amorphous matrix formed around developing trophozoites.

Treatment and prevention: *Perkinsus olseni* survives at least one day at 0–4 °C, and at least 197 days at –60 °C. Free-living stages are killed in less than 30 minutes in 6 mg/L chlorine, while those within tissues last over 2 hours. Free-living stages survive for less than 6 hours in distilled water, over 6 hours in 7‰ seawater, and less than 10 minutes in 120‰ seawater (Goggin et al. 1990). Infective stages can be excluded from hatcheries by filtration and UV sterilisation of incoming water (Ford et al. 2001).

Distribution in New Zealand

A recent New Zealand wide survey found that *P. olseni* occurs mainly in cockles around the Waitemata Harbour, along the east coast north of Auckland, and in Kaipara Harbour.



PERKINSOSIS IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: *Perkinsus olseni* and *P. atlanticus* appear to be the same species (Robledo et al. 2000). *P. olseni* was originally described from abalone in Australia (Lester & Davis 1981), and probably occurs in a wide range of bivalves from the Pacific Islands, northern New Zealand, around Australia (Goggin & Lester 1995), and Southeast Asia. *Perkinsus atlanticus* causes large-scale mortalities in clams (*Ruditapes philippinarum*) in Korea and Japan (Hamaguchi et al. 1998, Park et al. 1999), and in clams (*Ruditapes decussatus, R. philippinarum*) in Spain, Portugal, and Tunisia (Figueras et al. 1992). It is thought that *P. olseni* was taken to Europe in *R. philippinarum* introduced from Southeast Asia for trialling in aquaculture.

General comments: Although pathogenic to venerid clams (*Ruditapes, Austrovenus*) and abalone kept under elevated temperatures, *P. olseni* is more frequently isolated from apparently healthy molluscs.

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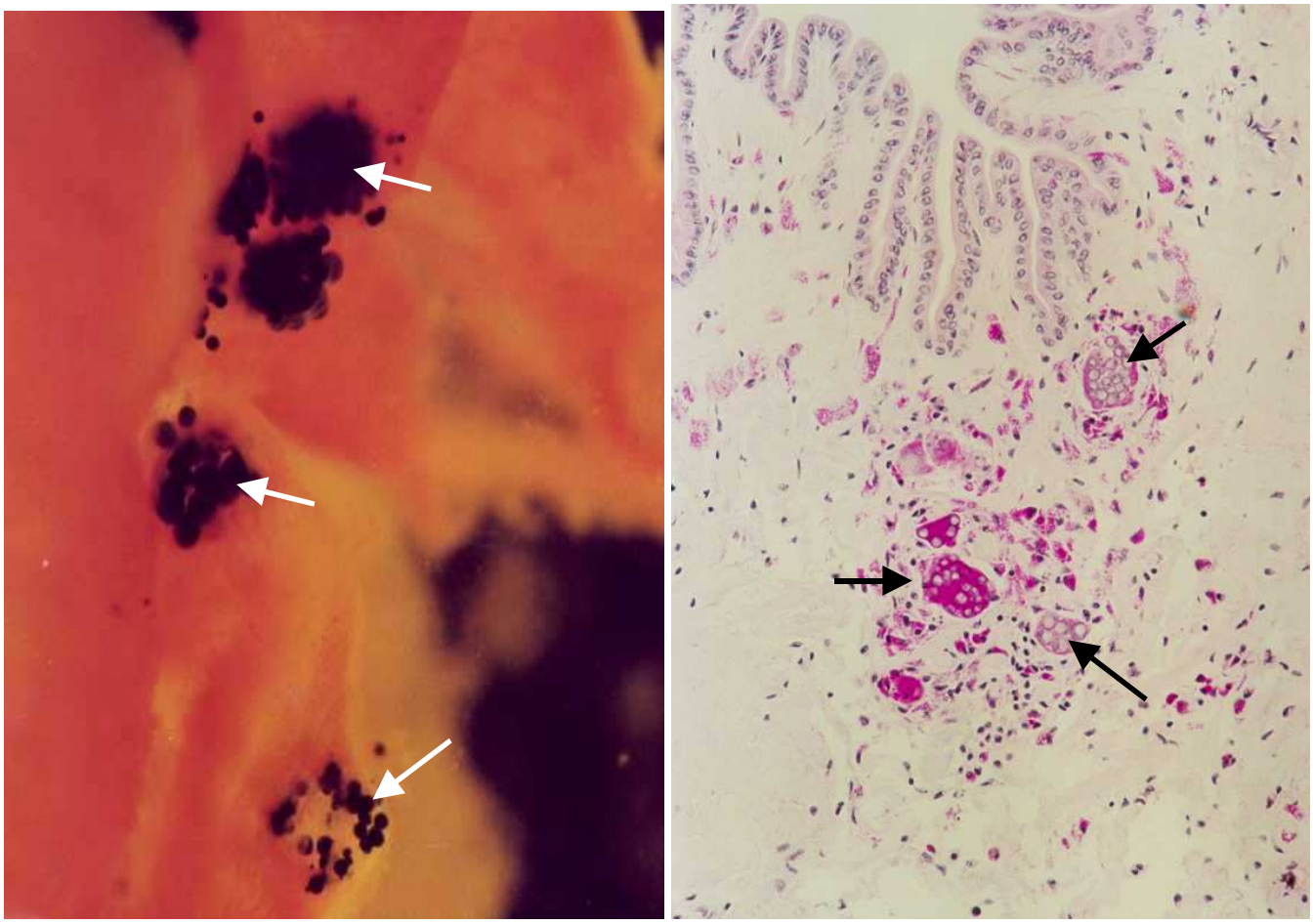


Perkinsus olseni in cockles (*Austrovenus stutchburyi*) from northern New Zealand. Photos by B. Diggles.

Left: The groups of enlarged black spheres (arrows) are *Perkinsus* hypnospores in the gills after incubation in Rays fluid thioglycollate medium and staining with lugols iodine. **Right:** Histology of a heavily infected cockle stained with PAS showing bright pink/red staining of developmental stages of *P. olseni* (arrows).

Disease: Perkinsosis (*Perkinsus marinus*)

Species and life stage affected: *Perkinsus marinus* infects juveniles and adults of eastern oysters (*Crassostrea virginica*). *P. marinus* also infects clams (*Mercenaria mercenaria*, *Mya arenaria*) and Pacific oysters (*Crassostrea gigas*) in experimental challenges (Barber & Mann 1994).

Gross signs: Severe emaciation, gaping, pale translucent digestive gland, shrinkage of mantle, loss of reproductive products, retarded growth, occasionally abscesses.

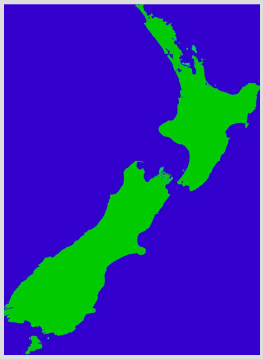
Causative agent: *Perkinsus marinus* is most closely related to dinoflagellates, but *Perkinsus* spp. form a group separate from them.

Diagnosis: Incubation in Ray's fluid thioglycollate medium (RFTM) (Bushek et al. 1994) is the most effective means of diagnosis, but the parasite is easy to detect histologically using Periodic Acid-Schiff (PAS) stain. PCR based molecular assays have been developed (Marsh et al. 1995, Robledo et al. 1998). A microplate ELISA can detect *P. marinus* at low levels, even in samples heavily contaminated with oyster protein (Dungan & Hamilton 1997). Sensitive immunoassays have also been developed for the detection of *P. marinus* in environmental samples (Dungan 1997).

Treatment and prevention: Controls on movement of oysters from contaminated areas are only partially effective as the parasite occurs in many other aquatic animals (Ford 1992). *P. marinus* can survive at 4–28 °C (Chu & Greene 1989), and at 3‰ salinity (Chu et al. 1993). Cyclohexamide may be effective in killing the parasite (Calvo & Burreson 1994). Can be excluded from hatcheries by filtration and UV sterilisation of incoming water (Ford et al. 2001).

Distribution in New Zealand

Unreported in New Zealand. As *P. marinus* is host specific to *C. virginica*, it is unlikely that it is present here.



PERKINSOSIS IS AN INTERNATIONALLY NOTIFIABLE DISEASE. IF YOU SUSPECT THAT YOUR STOCK HAVE THIS DISEASE, RING MAF ON 0800-809-966

Worldwide distribution: *P. marinus* occurs in *C. virginica* on the east coast of the U.S. from Maine to Venezuela.

General comments: After invasion through surface epithelia, *P. marinus* goes through division stages and forms zoospores in presporangia, which develop to sporangia. The zoospores are released, but it appears the meront is more effective in establishing infections (Chu 1996). The minimum infectious dose under experimental conditions is 100 meronts. After entry the pathogen secretes several substances that cause widespread tissue damage (La Peyre et al. 1996). As infection progresses, oyster growth is slowed (Barber & Mann 1994, Paynter 1996), and reproduction reduced (Kennedy et al. 1995). Death is probably due to massive tissue damage.

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Perkinsus marinus is virtually indistinguishable from *Perkinsus olseni* using light microscopy. Please refer to the previous section on *P. olseni* (p. 181) for photographs which are representative of *P. marinus*.

Metazoan diseases of molluscs

Disease: Flatworm infestation

Species and life stage affected: Pacific oysters (*Crassostrea gigas*), Greenshell™ mussels (*Perna canaliculus*), and scallops (*Pecten novaezelandiae*).

Gross signs: Gaping empty valves, flatworms attached to meat or hiding in empty shells.

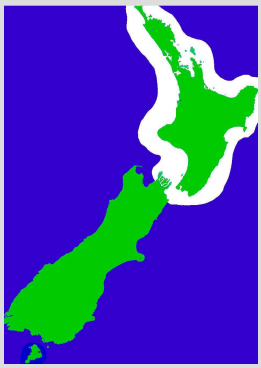
Causative agent: Flatworms (phylum Platyhelminthes), including *Enterogonia orbicularis* (Schmarda 1859) and a member of the family Planoceridae (provisional identifications).

Diagnosis: Detection of slow moving, well camouflaged flatworms inside shellfish. The flatworms avoid light and heat and produce mucus if handled.

Treatment and prevention: Freshwater, saturated salt, and chlorine solutions have only minimal effect. Heat treatment (80 °C) for short periods is effective, but can also kill spat. Treatment options are usually far more expensive than prevention. To avoid infestations, shellfish spat should be transported to growing areas as soon as practicable and not left to become overcrowded. Spat and juveniles should be grown at acceptable densities in areas of good current flow, adequate food availability and low sedimentation. Allowing access by fish to eat flatworms is also likely to prevent infestations. Oyster spat may be stored at levels above 0.5 m extreme low water neap.

Distribution in New Zealand

These two species have been recorded in most Pacific oyster growing areas of the North Island that have in the past sourced spat from the Kaipara Harbour. Flatworms have also been recorded predating oysters, scallops, and mussels in the Nelson/Marlborough region.



Worldwide distribution: Flatworms occur in molluscs worldwide.

General comments: Factors associated with flatworm infestations in Pacific oysters include overcrowding, intertidal oyster spat being left too long in bundles, sedimentation, translocation from the Kaipara Harbour, and poor farm management. In Greenshell™ mussels they are associated with overcrowded spat trays, and mussels seeded too densely, especially on the tie-off area of farm droppers. Flatworms are hermaphrodites, can lay many thousands of eggs, and have the potential to multiply rapidly, even after long periods of starvation.

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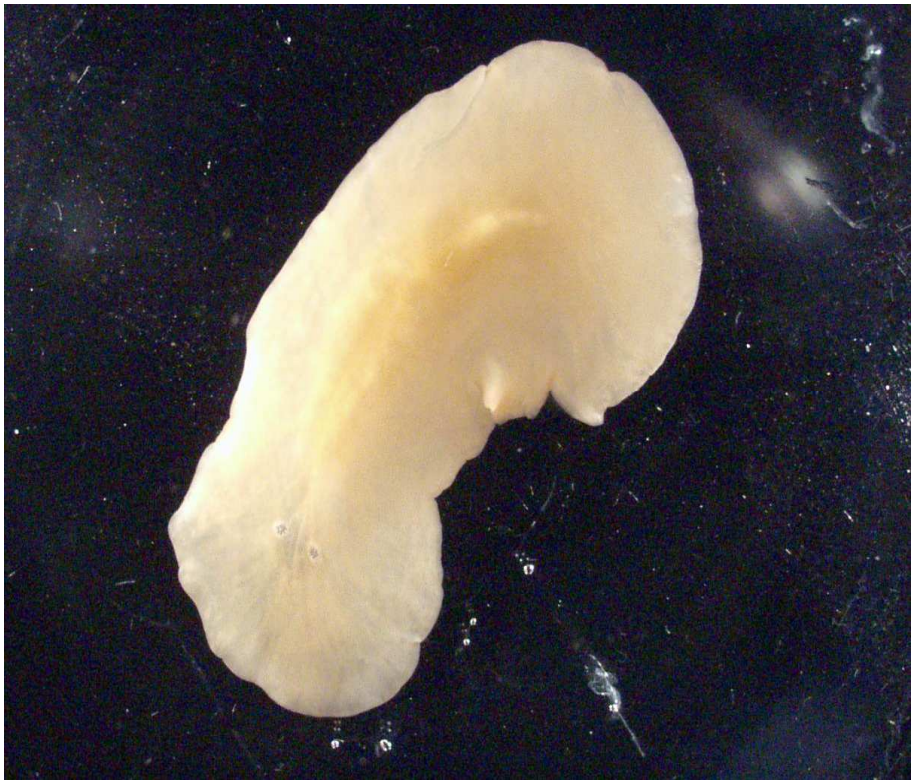


Flatworms collected from Pacific oyster spat in Croisilles Harbour, Marlborough. Photos by S. Handley.

Above: A specimen of *Enterogonia orbicularis* (provisional identification)
Below: A member of the Family Planoceridae.

Disease: Mudworm infestation (*Boccardia* sp.)

Species and life stage affected: Cultured and wild shellfish: Bluff oysters (*Ostrea chilensis*), Pacific oysters (*Crassostrea gigas*), rock oysters (*Saccostrea glomerata*), scallops (*Pecten novaezelandiae*), cockles (*Austrovenus stutchburyi*), Greenshell™ mussels (*Perna canaliculus*), and paua (*Haliotis iris*).

Gross signs: Burrows within the shell, brittle shell, shell blistering (mud blisters) in extreme cases.

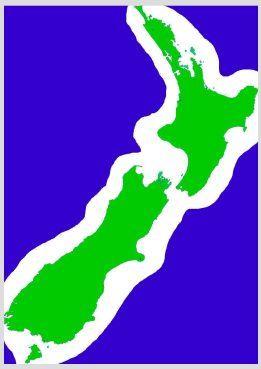
Causative agent: Polychaete worms (Family Spionidae), including *Boccardia acus*, *B. knoxi*, and *B. chilensis*.

Diagnosis: Gross signs are visible to the naked eye. Shallow burrows evident inside shell matrix, blisters in the shell that may or may not contain sediment. Mud chimneys can be seen on the outside surfaces of the shell, especially in the vicinity of the exhalant current of the feeding shellfish. Scallops can show small black dots inside the upper shell surface or inside their adductor muscle tissue. *Boccardia* sp. have two types of setiger 5 spines, in two rows. The gills begin on setiger 2 and are absent on setiger 5 (Blake & Kudenov 1978).

Treatment and prevention: Soaking in freshwater with detergent for a minimum of 24 hours for early stages of infestation. Drying shellfish out for 24 hours can kill some species. Heat treatment (80 °C) for short periods can be effective, but can kill spat. Avoiding infestations is recommended as treatment is expensive. Grow intertidal oysters above extreme low water neap, prevent overcrowding on farms, remove "rail" or "wild" oysters during harvest. For longline culture species, test growing environment for infestation levels. Cultivate shellfish outside of dispersal season.

Distribution in New Zealand

Various *Boccardia* species are found throughout New Zealand.



Worldwide distribution: *Boccardia knoxi* is also found in Tasmania.

General comments: *Boccardia* species are predominantly commensals that burrow into calcareous and coralline algae encrusted surfaces. They can induce shell blistering if their burrows penetrate through the shell of the host, especially in Pacific oysters grown on longlines. *Boccardia* species brood their eggs inside blisters, and may produce planktonic or fully developed larvae at different times of year. *Boccardia* species are generally smaller than larger *Polydora* species.

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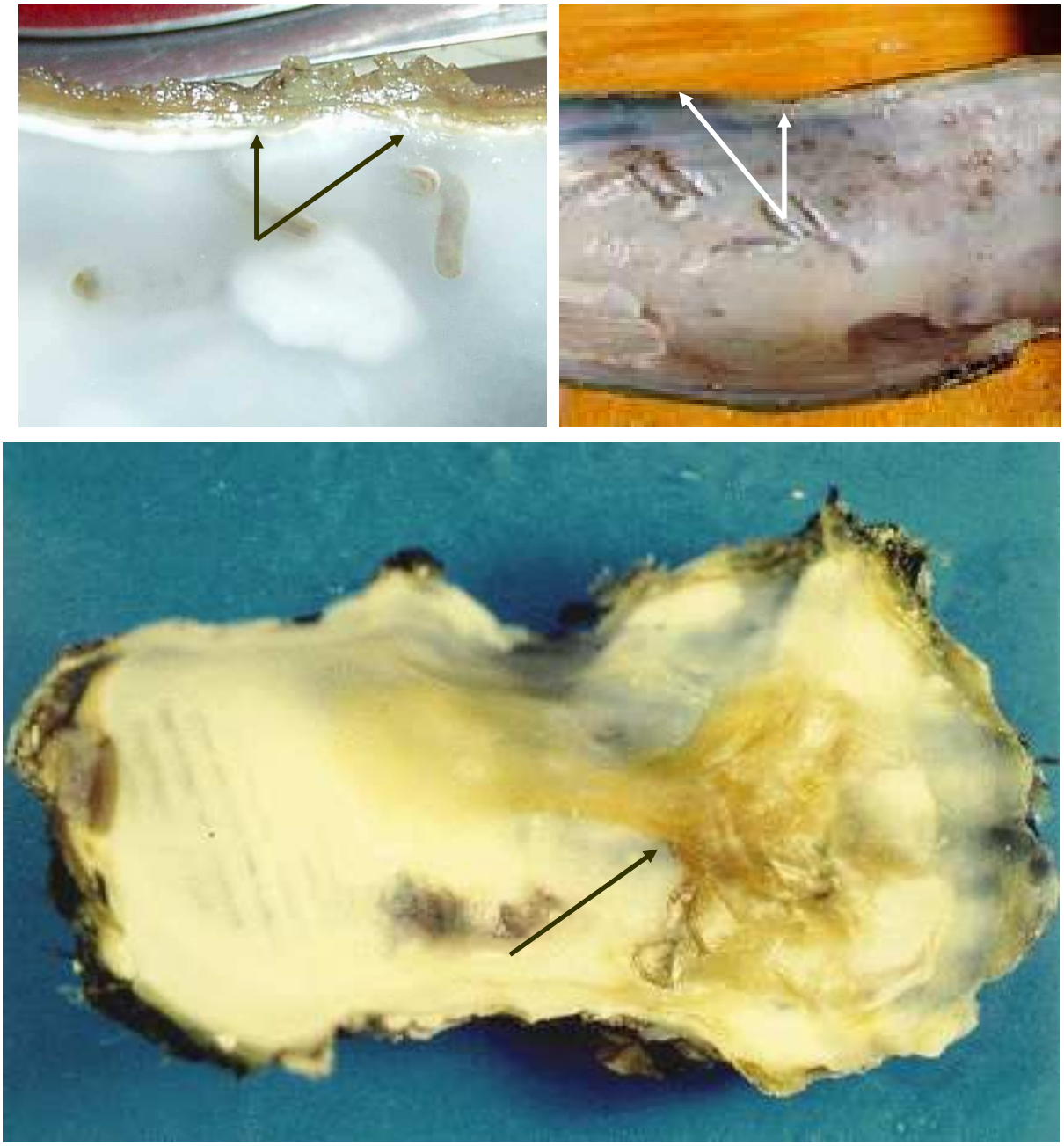


Boccardia knoxi infections in molluscs. Photos by S. Handley.

Above left: Burrows beneath the shell surface of a Pacific oyster. **Above right:** Ridge-like blisters resulting from *Boccardia* infestation of a Greenshell™ mussel. **Below:** Clear blister containing foul smelling seawater resulting from *B. knoxi* infestation in a Pacific oyster.

Disease: Mudworm infestation (*Polydora* sp.)

Species and life stage affected: Bluff oysters (*Ostrea chilensis*), Pacific oysters (*Crassostrea gigas*), rock oysters (*Saccostrea glomerata*), scallops (*Pecten novaezelandiae*), paua (*Haliotis iris*).

Gross signs: Shell blistering (mud blisters), burrows within the shell structure, brittle shell.

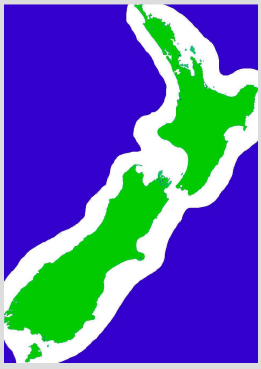
Causative agent: Polychaete worms (Family Spionidae) of the genus *Polydora*, including *P. websteri* and *P. hoplura* settling as larvae from the plankton.

Diagnosis: Blisters in the shell that usually contain sediment, hydrogen sulphide in blisters. Mud chimneys can be seen on the outside surface of the shell, especially near the exhalant current of the feeding shellfish. *Polydora* sp. have setiger 5 spines of one type only, and their gills begin posterior of setiger 5 (Blake & Kudenov 1978).

Treatment and prevention: Soaking in freshwater with detergent for minimum of 24 hours for early larval stages of infestation. Drying shellfish out for several days may be needed to kill some larger adult *Polydora* worms. Heat treatment (80 °C) for short periods can be effective, but can kill oyster spat. Avoiding infestations is recommended as treatment is expensive. Grow intertidal oysters above extreme low water neap, prevent overcrowding on farms, and remove "rail" or "wild" oysters during harvest. For longline culture species, test growing environment for infestation levels. Dispersal of larvae likely to occur at temperatures above 18 °C.

Distribution in New Zealand

Polydora hoplura and *P. websteri* have been found in most intertidal oyster farming areas of the North Island, and also in the Nelson/Marlborough region. They probably occur throughout New Zealand.



Worldwide distribution: Found worldwide.

General comments: *Polydora* species are predominantly commensals that burrow into calcareous surfaces but can settle as larvae inside the mantle edge of the host. Larval settlement inside the shell surface can result in shell blistering, especially if the worms can accumulate sediment from the outer shell surface. *Polydora* can also induce shell blistering if the worms expand their burrows and re-penetrate the shell of the host. *Polydora* species mostly brood their eggs inside blisters and produce fully developed larvae that re-infest locally. However, planktonic larvae may also be produced in some populations.

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Mudworm infestations of Pacific oysters caused by *Polydora websteri*. Photos by S. Handley.

Above left: Close up of *Polydora* feeding tubes composed of mucus-bound sediment on the outside edge of the shell. **Above right:** Long feeding tubes formed in the absence of wave action. **Below left:** A blister joined to the edge of a shell most likely formed after the settlement of a *Polydora* larva. **Below right:** Overlapping blisters resulting from multiple settlement or burrowing adult worms, the darker blisters may contain foul smelling sulphides if the worms are not aerating the blisters.

Species/disease cross reference for New Zealand aquaculture

Freshwater fishes	Diseases recorded in New Zealand
Brook trout (*Salvelinius fontinalis*)	Flavobacterial disease, whirling disease, white spot disease
Brown trout (*Salmo trutta*)	Flavobacterial disease, haemorrhagic septicaemia, neoplasm, vibriosis (*V. anguillarum*), whirling disease, white spot disease
Chinook (or Quinnat) salmon (*Oncorhynchus tshawytscha*)	Algal blooms, amoebic gill disease, ERM, flavobacterial disease, gas bubble disease, GDAS, IPN, neoplasm, nephrocalcinosis, pinhead syndrome, sunburn, vibriosis (*V. anguillarum, V. ordalii*), whirling disease, white spot disease
Carp/goldfish (*Cyprinus* sp., *Carassius* sp.)	Amoebic granulomatosis, flavobacterial disease, *Gyrodactylus ctenopharyngodontis*, haemorrhagic septicaemia, vibriosis, white spot disease
Eels (*Anguilla australis*, *A. dieffenbachii*)	Copepod infestation (*Abergasilus*), flavobacterial disease, gas bubble disease, haemorrhagic septicaemia, myxozoan infections, neoplasm, sunburn, vibriosis (*V. anguillarum*), white spot disease
Ornamental fish	Flavobacterial disease, gas bubble disease, mycobacteriosis, neoplasm, white spot disease
Rainbow trout (*Oncorhynchus mykiss*)	Flavobacterial disease, gas bubble disease, neoplasm, nephrocalcinosis, pinhead syndrome, sunburn, vibriosis (*V. anguillarum*), whirling disease, white spot disease
Sockeye salmon (*Oncorhynchus nerka*)	Copepod infestation, flavobacterial disease, nephrocalcinosis, vibriosis (*V. ordalii*), whirling disease, white spot disease

Marine fishes	Diseases recorded in New Zealand
Flounder (*Rhombosolea* sp.)	Copepod infestation (*Abergasilus*), trichodiniasis, vibriosis
Kahawai (*Arripis trutta*)	Algal blooms, copepod infestation (*Abergasilus*), neoplasm, vibriosis
Kingfish (*Seriola lalandi*)	Algal blooms, copepod infestation, ectoparasitic worms (benedenisis, zeuxaptisis), gas bubble disease, pinhead syndrome, vibriosis
Seahorses (*Hippocampus abdominalis*)	Algal blooms, gas bubble disease, vibriosis
Snapper (*Pagrus auratus*)	Algal blooms, ectoparasitic worms (anoplodiscisis, benedenisis, bivaginisis), epitheliocystis, flavobacterial disease, gas bubble disease, kidney cysts, *Myxidium* disease, neoplasm, pinhead syndrome, trichodiniasis, vibriosis, white spot disease (cryptocaryonosis)
Turbot/Brill (*Colistium* sp.)	Algal blooms, flavobacterial disease, pinhead syndrome, trichodiniasis, vibriosis

Crustaceans	**Diseases recorded in New Zealand**
Packhorse rock lobster (*Jasus verreauxi*)	Algal blooms, epibiont fouling, luminous vibriosis, shell disease, vibriosis
Paddle crab (*Ovalipes catharus*)	Algal blooms, epibiont fouling, shell disease, vibriosis
Red rock lobster (*Jasus verreauxi*)	Algal blooms, bacterial enteritis, black hepatopancreas disease, epibiont fouling, gas bubble disease, gill mycosis, shell disease, TLS, vibriosis

Molluscs	**Diseases recorded in New Zealand**
Bluff oysters (*Ostrea chilensis*)	APX, bonamiosis, herpesvirus, mudworm infestation, rickettsiosis
Cockles (*Austrovenus stuchburyi*)	Algal blooms, mudworm infection, perkinsosis, rickettsiosis
Greenshell™ mussels (*Perna canaliculus*)	Algal blooms, APX, digestive epithelial virosis, flatworm infestation, mudworm infestation
Pacific oysters (*Crassostrea gigas*)	Algal blooms, flatworm infestation, herpesvirus, mudworm infestation, rickettsiosis, vibriosis
Paua (*Haliotis* sp.)	Algal blooms, epithelial erosion, haplosporidosis, mudworm infestation, pustule disease/vibriosis, shell mycosis
Pipi (*Paphies australis*)	Perkinsosis
Rock oysters (*Saccostrea glomerulata*)	Digestive epithelial virosis, mudworm infection, rickettsiosis
Scallops (*Pecten novaezelandiae*)	Algal blooms, digestive epithelial virosis, flatworm infestation, mudworm infestation, mycoplasmosis, rickettsiosis
Toheroa (*Paphies ventricosa*)	Algal blooms, digestive epithelial virosis
Wedge shells (*Macomona liliana*)	Perkinsosis

Methods for decontaminating aquaculture facilities

Once the diagnosis of an exotic or serious disease is confirmed, decontamination of the affected facility should be an immediate priority to minimise the risk of spreading infections to other areas. In New Zealand, the recommended procedures for decontamination of aquaculture facilities are based on operational protocols outlined by MAF Biosecurity. MAF guidelines contain a list of sanitising agents approved for disinfecting aquaculture facilities. Sanitising agents are chemical substances or physical processes which can prevent infection by inactivation of microorganisms. Below are brief descriptions of some of the substances and processes commonly used for disinfection and/or decontamination of aquaculture facilities. Measures of concentration such as mg/L (milligrams per litre) are equivalent to ppm (parts per million). Much of this information has been obtained from Torgersen & Håstein (1995), Johnson (2000), and OIE (2000).

Chlorine and bleach
Chlorine is a gas which, in disinfectant form, is usually purchased as either chlorinated lime (calcium hypochlorite, or pool chlorine), which is usually 50–70% active ingredient, or bleach (sodium hypochlorite) which is an aqueous solution (usually 25–40% active ingredient) prepared from sodium hydroxide and chlorine. When chlorine dissolves in water, it reacts to form hypochlorous acid (the main disinfectant), hydrogen, and chloride. The alkaline nature of seawater reduces the effectiveness of this reaction. Chlorine is quickly inactivated by organic material and will dissipate in a few days when exposed to sunlight. All chlorine compounds are potent irritants which pose a risk to human safety and the natural environment. Before disposal, chlorine should be neutralised with sodium thiosulphate. Five moles of thiosulphate are required to neutralise 4 moles of chlorine, which means that the weight of thiosulphate required should be 2.85 times the weight of the chlorine being neutralised.

Formalin
Concentrated formalin is water containing 37% concentrated formaldehyde gas, but when used formalin is generally assumed to be composed of 100% active ingredient. Formalin is commonly used at low concentrations (50–250 mg/L (or 0.5–2.5 ml per 10 litres of water)) in bath treatments for ectoparasites, and is sometimes used at higher concentrations (8–10% or 80–100 ml formalin per litre of water) for disinfection. Whenever used as a treatment, the treated water must be adequately aerated as formalin strips oxygen from the water as it dissipates. Formalin is a highly toxic irritant and a possible carcinogen (cancer causing agent), hence care should be taken with use and disposal, especially to always provide adequate ventilation.

Heat/desiccation
Drying out or fallowing is an effective method of decontaminating aquaculture facilities as desiccation deactivates most aquatic and marine microorganisms. The time required for effective decontamination when using natural sunlight depends on the type of surface being decontaminated, type of pathogen, ambient temperature, and intensity of UV radiation in sunlight. For example, up to 3 months is required to deactivate viruses from earthen ponds at 18 °C. The process can be accelerated by using chemical disinfectants or by increasing the temperature of the heat source using tools such as flame throwers or blow lamps (dry heat), or by raising the water temperature to 80 or 100 °C (damp heat).

Iodine
Iodophors (e.g., betadine®) are generally available in 1% iodine solutions and are useful for disinfection of viruses and bacteria. Iodine is less sensitive than chlorine compounds to the presence of organic material, hence is more useful for hand washing and footbaths (at about 200 mg/L) and other applications where soil and other organic contaminants are present. However, as for chlorine, the alkalinity of seawater reduces the effectiveness of iodine. Like chlorine, iodine is also highly toxic and should be neutralised with sodium thiosulphate before disposal. The weight of thiosulphate required should be 0.78 times the weight of the iodine being neutralised.

Ozone
Ozone is a three atom form of oxygen. It is produced from air by electrical discharges or UV irradiation. Ozone has strong oxidative properties and is an effective disinfectant, inactivating bacteria and viruses at concentrations of 1–3 mg/L. However, ozone is costly to produce and use, and toxic residual ozone is difficult to measure in seawater. Ozone is also quickly inactivated by organic material.

Quaternary ammonium compounds
Quaternary ammonium compounds (ammonia) are widely available disinfectants which are very useful for disinfection of equipment. They are usually available in concentrations of about 1200 mg/L and are used as disinfectants against viruses and bacteria at concentrations of 2 mg/L for up to 1 hour.

Sodium hydroxide
Sodium hydroxide is an alkaline compound which relies on high pH to inactivate bacteria and viruses. It is the recommended disinfectant for footbaths (1% active ingredient + 0.1% detergent to aid penetration), earthen ponds and other surfaces where large amounts of organic material may reduce the effectiveness of other disinfectants. Solutions should be replaced once the pH drops below 11 and the discarded solution neutralised with acid before disposal. Care must be taken to ensure that footwear is resistant to the high pH when used in footbaths.

Ultraviolet radiation
Ultraviolet (UV) irradiation of wavelengths between 2500 and 2650 Å effectively destroys many pathogens. Because of this, UV is one of the preferred methods of disinfection of water supplies, but practicality, output and cost usually limit its use to small systems such as hatcheries. UV light does not penetrate very far into water, which must be taken into account when designing UV treatment units. Some pretreatment of water by particle filtration is recommended to minimise shadowing which may otherwise reduce the effectiveness of a UV unit. Using the UV which occurs naturally in sunlight to disinfect dried out earthen ponds requires about 3 months fallowing at temperatures around 18 °C.

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Disinfectants applicable to aquaculture and methods of use (Torgersen & Håstein 1995).

Process	Situation	Method	Comments
Physical			
Desiccation/light	Pathogens in the bottom of earthen ponds	Dry for 3 months at an average temperature of 18 °C	Drying period can be reduced by using a chemical disinfectant
Dry heat	Pathogens on concrete, stone, metal, plastic surfaces	Flame thrower, blow lamp	
Damp heat	Pathogens in transportation vehicle tanks	Steam at 100 °C or higher for 5 min	
	Waste water	60 °C 10 min, 80 °C 4 min	
Ultraviolet radiation	Viruses and bacteria in water	10 mJ/cm^2	Minimum lethal dose
	Protozoans in water	35 mJ/cm^2	
	IPN virus in water	200 mJ/cm^2	
Chemical			
Quaternary ammonium compounds	Viruses and bacteria on hands	1 mg/L for 1 min	IPN virus is resistant
	Gill bacteria on plastic surfaces	2 mg/L for 15 min	
Calcium oxide (lime)	Pathogens on dry earth	0.5 kg/m^2 for 1 month	Replace water and empty disinfected pools, keeping the effluent at pH < 8.5
Calcium hypochlorite* (chlorine)	Bacteria and viruses on all clean surfaces and in water	200 mg/L* for 30 min 100 mg/L for 1 hour 30 mg/L* several days	Can be neutralised with sodium thiosulphate **
Calcium cyanamide	Spores on earthen base	3000 kg/ha on dry surfaces, leave in contact for 1 month	
Formalin	Pathogens in sealed premises	Gas released from formgenic substances	Follow manufacturer's instructions
	Disinfect water	10 ml per litre water	
Iodine (iodophors)	Bacteria, viruses on hands and smooth surfaces	200 mg/L for a few seconds	Can be neutralised with sodium thiosulphate **
	Eyed eggs	100 mg/L for 10 min	
	Gametes during fertilisation	25 mg/L several hours	
Ozone	Pathogens, water	1 mg/L for 1 minute	Costly
Sodium hydroxide	Pathogens on resistant surfaces with cracks	1% solution, 0.1L/ m^2 for 48 hours	May stain or bleach treated surfaces
	Earth ponds	1% solution, 2 L/ m^2 for >2 weeks	
Sodium hypochlorite* (bleach)	Bacteria and viruses on all surfaces and in water	180 mg/L 20–30 s	Neutralise with sodium thiosulphate as for chlorine**
	Nets, boots, clothing, hands	30 mg/L for several days	

* Most swimming pool chlorine is approximately 65% active calcium hypochlorite, hence to obtain 200 mg/L active calcium hypochlorite requires 300 mg/L (0.3 g/L) pool chlorine. Bleach is usually 35% active sodium hypochlorite so to obtain 30 mg/L requires 0.1 ml/L bleach.

** 5 moles of sodium thiosulphate will neutralise 4 moles of chlorine/bleach or iodine. If a 1% thiosulphate solution is used, the volume (ml) required is:

for chlorine: 28.5 x (number of litres of chlorine solution x concentration in mg/L)/100. (e.g., to neutralise 50 litres of 200 mg/L chlorine, requires 28.5x (50 x 200)/100 = 2850 ml of 1% sodium thiosulphate).

for iodine: 7.8 x (number of litres of iodine solution x concentration in mg/L)/100. (e.g., to neutralise 50 litres of 100 mg/L iodine, requires 7.8 x (50 x 100)/100 = 390 ml of 1% sodium thiosulphate).

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Glossary

Acute	A rapid onset of disease with a short, but severe, course.
Aetiology	Cause
Ameliorate	Cause to become better, resolve.
Anaemia	A deficiency in the number of red blood cells, or haemoglobin.
Anorexia	Severely underweight.
Apicomplexan	A group of obligate pathogens including *Toxoplasma gondii* (causes coccidiosis) and *Plasmodium* (causes malaria).
Asymptomatic carrier	An individual infected with a disease agent, but not exhibiting any signs of disease.
Ataxia	Imperfect control of voluntary body functions.
Atrophy	Abnormally small size of cells or tissues.
Bacteria	Unicellular (rarely multicellular) organisms which lack a membrane bound nucleus (i.e. prokaryotes).
Benign	Harmless.
Chronic	Lingering, long lasting.
CL	Carapace length.
Commensal	Living in close association with another species without an apparent effect on each other.
Definitive diagnosis	A diagnosis which confirms the identity of the causative agent responsible for a disease.
Detritus	Organic debris.
ELISA	Enzyme linked immunosorbent assay.
Emaciation	Becoming lean, wasting away.
Enteritis	An infection of one or more parts of the gut.
Exophthalmia	"pop eye", a condition characterised by protrusion of the eye in fishes.
Exotic	Of foreign origin, not native or endemic.
Focal	Restricted in area.
Granulomatous	A type of cellular reaction associated with a chronic lesion.
Haematopoietic	Tissues that produce red blood cells .
Haemorrhage	Bleeding.
Haemocyte	A blood cell of an invertebrate.
Haemolymph	Blood of an invertebrate.
Hepatopancreas	(or digestive gland) An organ of the gut of crustaceans made up of numerous tubules. It is a major site of digestion and energy storage.
Histology	The study of tissues.
Horizontal transmission	Transmission of disease from animal to animal by cohabitation or via water.
Hyperplasia	Increase in the number of cells or tissues.
Hypertrophy	Increase in the size of cells or tissues.
IFAT	Indirect fluorescent antibody test.
Inflammation	A local protective response to injury or damage which serves to destroy, dilute, or wall off both the injurious agent and the injured tissue.
ISH	In situ hybridisation.
Iodophors	Iodine based disinfectants.
Latent infection	An infection that does not produce visible or clinical signs of disease.
Lesion	A localised area of pathological change in structure of an organ, tissue, or cell.
Meninges	Membranes which cover the nervous tissues of vertebrates.
Metazoan	A multicellular organism.
Moribund	Near death.
Necrosis	Cell or tissue death.
OIE	Office International des Epizooties, the World Animal Health Organization, based in Paris, France.
Opisthaptor	The posterior attachment organ of a monogenean worm (phylum Platyhelminthes), usually armed with various hooks, suckers and/or clamps.
Pallor	Pale or faded.
Parasite	An organism which lives on or inside another organism (the host), deriving nutrition from the host to the detriment of that host.
PAS	Periodic Acid-Schiff stain, a histological stain which gives positive reactions to polysaccharides (e.g., glycogen), mucopolysaccharides and glycoproteins.

Pathognomonic	Characteristic of a particular disease.
Pathology	The study of structural and functional changes caused by disease.
PCR	Polymerase chain reaction.
Peracute	Extremely rapid onset (of disease).
Pericardium	The area surrounding the heart.
Peritonitis	An infection of the peritoneum (body cavity).
Petechiae	Pin point haemorrhages.
Polymorphic	Occurs in more than one morphological form.
Prepatent	Period early in a disease process when disease cannot be detected.
Presumptive diagnosis	A tentative or provisional identification of the cause of a disease based on limited information.
Protozoan	A unicellular organism with a membrane bound nucleus.
RFTM	Ray's fluid thioglycollate medium, an anaerobic culture medium for microorganisms.
SEM	Scanning electron microscopy.
Septicaemia	An infection of the bloodstream.
Subacute	Onset and course of disease over a time span intermediate between acute and chronic.
Symbiont	An organism which lives on or in another organism in a relationship from which both organisms derive benefit from the association.
TEM	Transmission electron microscopy.
Vertical transmission	Transmission of disease from adults to offspring through the egg or sexual fluids.
Virus	Tiny organisms consisting of nucleic acid (either RNA or DNA) surrounded by a protein or protein/lipid coat, which infect cells of bacteria, plants and animals, using the host cell machinery for replication.
Zoonotic	Diseases of animals which can also infect humans.

Appendix 1. List of diseases of aquatic animals notifiable to the OIE, and other internationally significant diseases (from OIE (2001). International Aquatic Animal Health Code, 4th edition).

Diseases notifiable to the OIE (List B diseases, article 1.1.2.1.)

1. Diseases of fish

Epizootic haematopoietic necrosis
Infectious haematopoietic necrosis
Oncorhynchus masou virus disease
Spring viraemia of carp
Viral haemorrhagic septicaemia

2. Diseases of molluscs

Bonamiosis
Haplosporidiosis
Marteiliosis
Mikrocytosis
Perkinsosis

3. Diseases of crustaceans

Taura syndrome
White spot disease
Yellowhead disease

Other significant diseases (article 1.1.2.2.)

1. Diseases of fish

Channel catfish virus disease
Viral encephalopathy and retinopathy
Infectious pancreatic necrosis
Infectious salmon anaemia
Epizootic ulcerative syndrome
Bacterial kidney disease *(Renibacterium salmoninarum)*
Enteric septicaemia of catfish *(Edwardsiella ictaluri)*
Piscirickettsiosis *(Piscirickettsia salmonis)*
Gyrodactylosis *(Gyrodactylus salaris)*
Red sea bream iridoviral disease
White sturgeon iridoviral disease

2. Diseases of molluscs

None at present

3. Diseases of crustaceans

Baculoviral midgut gland necrosis
Nuclear polyhedrosis baculoviroses (*Baculovirus penaei* and *Penaeus monodon*-type baculovirus)
Infectious hypodermal and haematopoietic necrosis
Crayfish plague
Spawner-isolated mortality virus disease

Appendix 2. New Zealand's listed diseases for fish, mollusks, and crustaceans

New Zealand category 1 diseases include highly contagious exotic diseases of animals of unknown aetiology, other specified diseases from the current OIE List A and any disease that the Chief Veterinary Officer may add to this category. List A diseases are transmissible diseases that have the potential for very serious and rapid spread, irrespective of national borders, are of serious socio-economic or public health consequence, and are of major importance in the international trade of animals and animal products. An example of a List A disease is Foot and Mouth Virus of farm animals. At present no diseases of aquatic animals are on the OIE List A.

New Zealand category 2 diseases include specified diseases from the current OIE List A and List B diseases and any disease that the Chief Veterinary Officer may add to this category but excluding any OIE List B diseases that are endemic to New Zealand. List B diseases are transmissible diseases that are considered to be of socio-economic and/or public health importance within countries and that are significant in the international trade of animals and animal products.

New Zealand category 3 diseases include all diseases in the New Zealand lists of notifiable and unwanted organisms except those that are included in OIE lists A or B or are endemic to New Zealand. This category also includes any additional diseases that the Chief Veterinary Officer may add to it.

	OIE LIST B DISEASES: Organisms affecting fish	
	Disease	New Zealand category of exotic disease
	Epizootic haematopoietic necrosis virus	2
	Infectious haematopoietic necrosis virus	2
	Oncorhynchus masou virus disease	2
	Spring viraemia of carp virus	2
	Viral haemorrhagic septicaemia virus	2

	Other significant organisms affecting fish	
	Disease	New Zealand category of exotic disease
	Bacterial kidney disease (*Renibacterium salmoninarum*)	3
	Epizootic ulcerative syndrome	3
	Furunculosis (*Aeromonas salmonicida*)	2
	Gyrodactylosis (*Gyrodactylus salaris*)	3
	Infectious pancreatic necrosis virus (exotic strains)	2
	Infectious salmon anaemia virus	2
	Viral encephalopathy and retinopathy virus	3

	Organisms affecting molluscs	
	Disease	New Zealand category of exotic disease
	Bonamiosis (*Bonamia ostreae*)	2
	Haplosporidiosis (*Haplosporidium* species)	2
	Marteiliosis (*M. refringens, M. sydneyi*)	2
	Mykrocytosis (*Mikrocytos mackini, M. roughleyi*)	2
	Perkinsosis (*Perkinsus marinus*)	2

	Organisms affecting crustacea	
	Disease	New Zealand category of exotic disease
	Baculoviral midgut gland necrosis virus	3
	Spawner isolated mortality syndrome	3
	Whitespot disease	2
	Yellowhead disease	2