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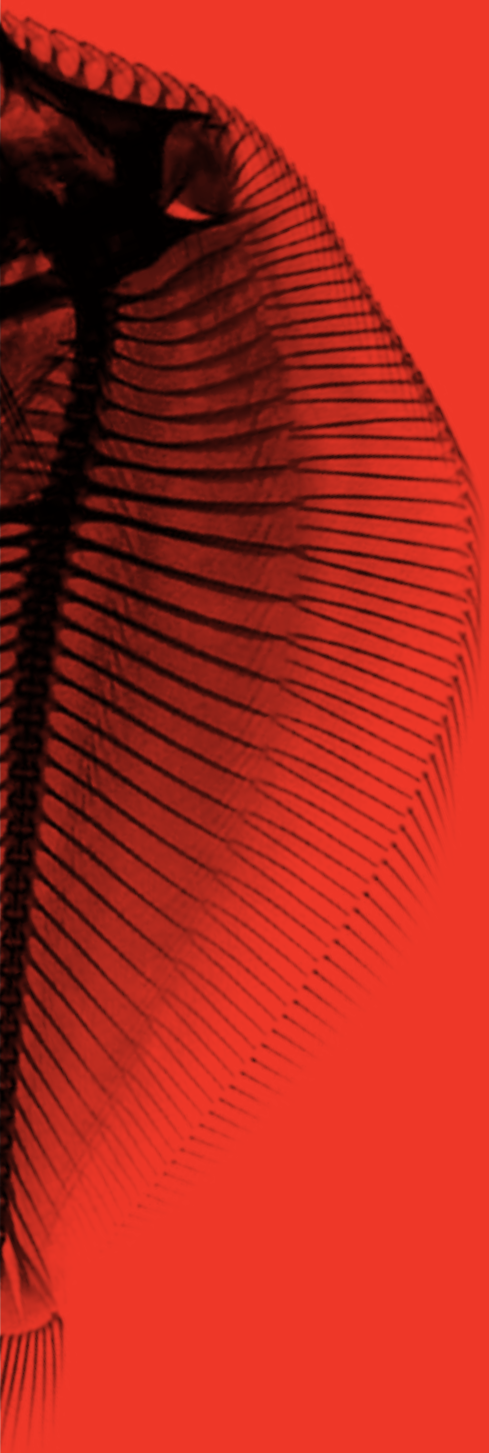

Contents

Foreword

The Australian Government Department of Agriculture, Fisheries and Forestry is pleased to introduce the fourth edition of this field guide.

The field guide aims to help people recognise diseases of significance to aquaculture and fisheries in Australia. This edition incorporates new and updated information gathered from an extensive review of the third edition. It now covers 48 aquatic animal diseases of significance to Australia that affect species of finfish, crustaceans, molluscs and, for the first time, amphibians.

Early recognition and reporting of suspected disease outbreaks is critical to protecting our unique aquatic biodiversity, and fisheries and aquaculture sectors. It can permit a rapid disease response to contain outbreaks, increasing the opportunity for disease eradication and thereby limiting potential impacts on aquatic animal industries and the environment. People who work closely with aquatic animals are usually the first to notice signs of a significant disease event. This field guide provides guidance for these people—commercial fishers, aquaculture workers, recreational fishers, quarantine staff, scientists, conservationists and students—to recognise significant aquatic animal diseases, should they occur.

Many people and institutions have contributed to the field guide, including fish health experts from industry, research organisations, state and territory governments, and government agencies of the Asia–Pacific region (including New Zealand), Canada, Denmark, Norway, the United Kingdom and the United States of America. Drawing extensively on experience and research activities in aquatic animal health management, both in Australia and abroad, the guide complements the growing body of practical knowledge published for aquaculture and fisheries in Australia. On behalf of the Australian Government, I thank all contributors to this production for their efforts and commend this field guide to you.

Dr Mark Schipp
Australian Chief Veterinary Officer

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The current publication has built upon the contributions made to previous editions of this guide and we recognise all who ensured information was accurate, current and consistent. Thanks to Dr Rob Adlard (Queensland Museum), Dr Rachel Bowater (Oonoonba Veterinary Laboratory, Queensland Department of Primary Industries and Fisheries), Dr Susan Bower (Fisheries and Oceans Canada), Dr David Bruno (Marlab, Aberdeen, Scotland), Dr Jeremy Carson, Dr Kevin Ellard and Dr Judith Handlinger (Tasmanian Department of Primary Industries, Parks, Water and Environment), Dr Supranee Chinabut (Thailand Department of Fisheries), Dr Mark Crane (CSIRO Australian Animal Health Laboratory), Dr Ben Diggles (DigsFish Pathology Services, Australia), Dr Ralph Elston (Aqua Technics Inc, United States of America), Dr AS Sahul Hameed (Abdul Hakeem College, India), Professor Brian Jones (Department of Fisheries, Western Australia), Dr Cedric Komar (Intervet Norbio Singapore), Dr Malcolm Lancaster (Primary Industries Research Victoria), Dr James Moore (California Department of Fish and Game), Dr Kazuhiro Nakajima (Fisheries Research Agency, Japan), Dr Niels Jørgen Olesen (Danish Veterinary Institute), Dr Shane Raidal and Dr Fran Stephens (Murdoch University), Dr M Sano (National Research Institute of Aquaculture, Japan), Dr Peter Walker (CSIRO Australian Animal Health Laboratory), Professor Richard Whittington (University of Sydney) and Dr Shi Zhengli (Chinese Academy of Sciences, People's Republic of China).

Members of the Australian Government Department of Agriculture, Fisheries and Forestry—-Aquatic Animal Health Program coordinated the production of this fourth edition including Alexander McLaren, Ryan Keightley, Suzanne Payne and Steve Wortley. Alistair Herfort is recognised as co-ordinator of the first three editions of the field guide and provided advice in the production of this edition.

Photographic contributors

International contributors

- Dr David Alderman (Centre for Environment, Fisheries and Aquaculture Science, United Kingdom)
- Dr Isabelle Arzul (French Research Institute for Exploration of the Sea—IFREMER)
- Dr Jerri Bartholomew (Oregon State University)
- Dr Franck Berthe (IFREMER)
- Dr Susan Bower (Fisheries and Oceans Canada)
- Dr Sandra Bravo (Universidad Austral de Chile)
- Professor Eugene Burreson (Virginia Institute of Marine Science)
- Dr Birgit Dannevig (National Veterinary Institute, Oslo)
- Dr Dianne Elliott (United States Geological Survey)
- Dr John Fryer (deceased—Oregon State University)
- Dr AS Sahul Hameed (Aquaculture Division, Department of Zoology, C Abdul Hakeem College, Tamil Nadu, India)
- Professor Larry Hanson (College of Veterinary Medicine, Mississippi State University)
- Professor Tore Håstein (National Veterinary Institute, Oslo)
- Dr Ron Hedrick (University of California, Davis)
- Dr Naoki Itoh (Tohoku University, Japan)
- Dr Erik Johnson (Private Practitioner, Johnson Veterinary Services, Georgia)
- Dr Somkiat Kanchanakhan (Department of Fisheries, Thailand)
- Dr Michael Kent (Oregon State University)
- Dr Gael Kurath (United States Geological Survey)
- Professor Don Lightner (University of Arizona)
- Professor Tor Atle Mo (National Veterinary Institute, Oslo)
- Dr James Moore (California Department of Fish and Game)
- Dr Kazuhiro Nakajima (Fisheries Research Agency, Yokohama)
- Dr Ronald Pascho (Western Fisheries Research Center, Seattle)
- Dr Trygve Poppe (Norwegian School of Veterinary Science, Oslo)
- Dr Melba Reantaso (Food and Agriculture Organization of the United Nations)
- Dr Hans Jürgen Schlotfeldt (European Association of Fish Pathologists)
- Dr Jim Winton (United States Geological Survey)
- Dr Oded Zmora (University of Maryland, Baltimore)

Australian contributors

- Dr Lee Berger (James Cook University)
- Dr Rachel Bowater (Queensland Department of Primary Industries and Fisheries)
- Dr Richard Callinan (formerly New South Wales Department of Primary Industries)
- Dr Jeffrey Go (University of Sydney)
- Dr John Humphrey (Northern Territory Department of Industry, Fisheries and Mines)
- Dr Brian Jones (Western Australia Department of Fisheries)
- Dr Clive Jones (Queensland Department of Primary Industries and Fisheries)
- Dr Matt Landos (Future Fisheries Australia)
- Professor Bob Lester (University of Queensland)
- Professor Barry Munday (deceased—University of Tasmania)
- Professor Leigh Owens (Queensland Department of Primary Industries and Fisheries)
- Dr Mark Porter (University of Tasmania)
- Dr Howard Prior (Queensland Department of Primary Industries and Fisheries)
- Dr Sarah Ridgeway (née Kleeman; formerly Australian Government Department of Agriculture, Fisheries and Forestry)
- Dr Greg Storie (Queensland Department of Primary Industries and Fisheries)
- Dr Patrick Tully (New South Wales Department of Primary Industries)
- Staff at the CSIRO Australian Animal Health Laboratory
- Staff of the Department of Microbiology and Immunology, James Cook University
- Staff at New South Wales Department of Primary Industries

How to use this field guide

The field guide begins with coverage of finfish, mollusc, crustacean and amphibian anatomy including images and illustrations to help the reader describe lesions when reporting a suspected disease. It follows with descriptions for each infectious disease present in Australia's National List of Reportable Diseases of Aquatic Animals. These are presented alphabetically and classified into infectious diseases affecting finfish, molluscs (e.g. oysters), crustaceans (e.g. prawns) and amphibians (e.g. frogs)

Each disease page describes the signs of disease (at the farm/tank/pond level and gross and microscopic pathological signs), the disease agent, host species that carry the disease agent, the presence of the disease in Australia, epidemiology of the disease, other diseases in the field guide that may have similar signs, and sample collection and reporting of disease outbreaks. Most disease pages have photographs of animals with gross signs of disease or histological images detailing the typical tissue changes present.

Diagnosing diseases of aquatic animals is a structured process which begins with making detailed observations, then asking the right questions. The primary aim of this process is to create a broad list of possible diseases which may account for the observed signs. This can be further refined by targeted questioning and diagnostic tests. Questions to ask may include; which species, age class, sizes and sex of animals are involved? What specifically have you observed—for example, behavioural changes, changes in feed intake (reduced or increased), changes in faecal output? Are there obvious gross lesions (colour changes, ulcers, spots, etc.)? Does disease result in morbidity only or are there mortalities? What level of mortality has been observed? Do animals show signs of recovery? At what rate did you observe disease (sudden death or chronic progression of disease)? Is this the first occurrence of disease with this presentation in your facility? Are any neighbouring properties or facilities reporting diseases? Have there been recent introductions to the facility or any changes in practice?

After making your initial observations, go to the 'signs of disease' section of the disease entries. Look up the diseases listed that match your observations, and you will find photographs and further information to help you narrow the search for the cause of the disease. For example, you might have observed disease in a marine fish, but then find that the listed disease with similar presenting signs affects only freshwater species—in this way, you can eliminate the unlikely, and your list of

differential diagnoses will be shorter. In all cases, however, laboratory tests would be required for a definitive diagnosis. Local authorities with responsibility for aquatic animal health can advise on further courses of action should you suspect any disease listed in the field guide.

Signs of disease

Diagnostic information based on disease signs at the farm, tank or pond level, and gross pathological signs (abnormalities that can be seen with the naked eye) is important. However, in most cases, it is impossible to arrive at a definitive diagnosis on the basis of these signs alone. Although some users will not have experience in histological examination or the equipment required for such examination in the field, the field guide also lists microscopic pathological disease signs.

Mass mortality

A fish kill involving a range of species is more likely caused by an environmental problem (such as toxicity or oxygen depletion). Deaths limited to one species (where other species are also present) is more likely a result of an infectious agent.

Behavioural changes

All species of aquatic animals have characteristic protection, food-gathering and breeding behaviours. Abnormal behaviour, such as a decrease in feeding, could indicate stress from disease.

Some behavioural changes can occur across groups of species, or even across different phyla. In molluscs, few behavioural differences are observed such as delayed closing (in oysters) and decreased feeding or adhesion to vertical surfaces (in abalone). Finfish often gather at water inlets or gasp for air at the surface if the water is depleted of oxygen. If irritated by skin parasites, they may scrape themselves on hard surfaces. Whirling or 'corkscrew' swimming could indicate disease from an infectious agent or aquatic toxins. In both finfish and crustaceans, gathering at the surface or pond edges can often be a sign of disease.

Gross signs

Gross pathological changes may indicate the presence of infectious disease. Careful observation and further laboratory investigation is required to make a definitive diagnosis because many of the signs are not singularly pathognomonic (characteristic for a specific disease) and may be common pathological changes associated with multiple infectious agents. The table below describes some of the more commonly observed visible signs and the groups of aquatic animals dealt with in this field guide for which these are most often observed.

Sign	Finfish	Molluscs	Crustaceans	Amphibians
Changes in the colour, texture and opacity of flesh	*	*	*	*
Tissue necrosis and lesions	*	*	*	*
Retraction of gill margins		*		
Pustules	*	*		
External spots	*		*	
Changes in surface colour	*		*	*
Secondary fungal or bacterial growth	*		*	*
Deformities and tumours	*		*	*
Swollen or discoloured organs or faecal castes	*	*	*	*
White midgut line	*		*	
Broken or damaged appendages			*	
Erosion of shell			*	
Lesions or ulcers of skin or gills	*			*
Haemorrhaging with associated anaemia	*			*
Granulomas	*			
Exophthalmos (popeye)	*			
Ascites (accumulation of fluid in peritoneal cavity)	*			*
Petechial haemorrhages (pinpoint bleeding in skin and mucous membranes)	*			*
Ecchymotic haemorrhages (bleeding or bruising beneath the skin or mucous membranes)	*			*
Excessive mucus on gills and skin	*	*		
Dropsy (accumulation of fluid in body tissues)	*			*
Protrusion of scales	*			

Host range

A list of species known to be susceptible to the infectious agent is provided. Species are further classified as either naturally susceptible (diseased animals have been identified in the wild) or experimentally susceptible. Lists of susceptible species reflect the information available at the time of publishing; however, with further understanding and sampling, it is expected that such lists will expand and/or require refining. Common and scientific names for hosts are provided.

Presence in Australia

Information on the national distribution of diseases listed in the field guide is based on formal reporting through the regional Quarterly Aquatic Animal Disease reporting program (managed by NACA, FAO and OIE). Australia has been an active participant since 1998.

Where a listed disease has been reported under the program to have been present, a map illustrating where it occurred in Australia is included. States or territories having reported disease are shown in orange in the distribution map. It is important to note that, although a map may identify a state or territory as having reported a disease, this neither implies that it is present at the time of publication or that it occurs across the entire state or territory.

Readers should consult the World Animal Health Information Database (WAHID) interface (www.oie.int/wahis/public.php?page=home) or the International Database on Aquatic Animal Diseases (www.collabcen.net/idaad) for current information on global distribution of diseases outside Australia.

Exotic diseases

Diseases in this field guide described as exotic are those that do not occur in Australian aquatic animal populations.

Endemic diseases

Endemic (enzootic) diseases are those that have established in Australian aquatic animal populations. They might be native to Australia or might have been introduced in the past.

Epidemiology

The field guide describes epidemiological factors that are important to each disease. The key to describing the epidemiology of a disease involves understanding the relationship between the infectious agent, the host/s and the environment. Factors relating to the infectious agent include its life cycle (direct transmission or a requirement for intermediate host stages), survival outside the host (carriage on fomites, passage in waste water) and sensitivity to certain temperatures or salinities. Host factors may include the susceptible species, ages, sexes and sizes involved. Environmental factors include seasonal and non-seasonal variations in temperature, food availability, salinity, available oxygen, species movement and exposure to different environments (for example, migrations or gathering for breeding); these factors can affect disease agent survivability and host immune competence.

Differential diagnosis

The list of similar diseases at the bottom of each disease page refers only to the diseases covered by this field guide. Gross signs observed might well be representative of a wider range of diseases not included here. Therefore, these diagnostic aids should not be used as a guide to a definitive diagnosis, but rather as a tool to help identify the diseases included in this field guide that most closely account for the observed gross signs. Further diagnostic testing will be required to confirm either presence or freedom from a suspected disease.

1 Introduction

This field guide has been designed to provide ready access to information on the aquatic animal diseases significant to Australia. These diseases have potential to cause disruption to Australia's aquatic animal biodiversity, fisheries and aquaculture productivity, and international trade. The diseases covered here are in Australia's National List of Reportable Diseases of Aquatic Animals, which includes those reportable through the regional Quarterly Aquatic Animal Disease reporting program (managed by NACA, FAO and OIE), as well as other diseases considered of national significance.

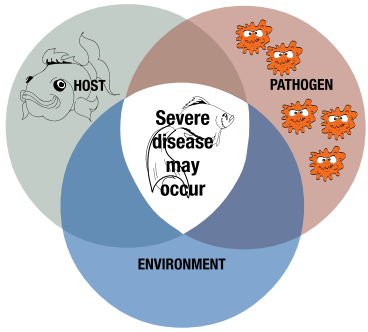
The field guide is aimed at fishery and aquaculture managers, their field staff, veterinary workers and students of aquatic animal health. The publication is also relevant to people in the seafood processing and retail industry, recreational and commercial fishers, and the general public. For people participating in national surveillance and monitoring for aquatic animal disease, the field guide is a valuable disease reference. For the casual reader, the field guide gives an informative and often graphic account of the diseases and organisms that threaten Australia's expanding aquaculture industries.

The marine and freshwater environments in Australia are rich with many types of animals. The field guide focuses on fish, molluscs (e.g. oysters), crustaceans (e.g. prawns) and amphibians (e.g. frogs). It is not possible to list every disease for every species in a publication of this type, so the emphasis is on infectious diseases found on Australia's National List of Reportable Diseases of Aquatic Animals, significant to our aquaculture industries, fisheries and environment.

Nature of disease

The diseases described in this field guide are caused by infectious agents—viruses, bacteria, fungi or parasites; diseases with non-infectious causes are outside its scope. Non-infectious causes of disease in the aquatic environment are often attributable to the environment itself: morbidity and mortality can result from natural or human-influenced events that lead to oxygen depletion, production of aquatic toxins, or changes in water temperature or salinity.

Figure 1 Relationship between the host, pathogen and environment in disease outbreaks



However, disease will not occur simply because an infectious agent is present. Rather, the likelihood of disease being expressed is determined by the specific interactions between the host (the aquatic animal), the infectious agent or pathogen, and the environment (Figure 1).

Laboratory tests and sampling

Photographs of gross disease signs, such as those in this field guide, can help an investigator to create a preliminary list of possible disease agents (differential diagnosis list) for the case under investigation. However, although gross signs narrow the search for possible agents, they are not adequate for definitive diagnosis; consequently, representative samples from the diseased animal(s) and the environment in which they live need to be taken for analysis. Information about sampling can be found in the following publication:

- Asia diagnostic guide to aquatic animal diseases, Food and Agriculture Organization of the United Nations Fisheries Technical Paper 402/2 (www.fao.org/docrep/005/y1679e/y1679e00.htm).

The website of the Australian Government Department of Sustainability, Environment, Water, Population and Communities (www.environment.gov.au/water/index.html) provides information on the management of inland water quality. The OIE Manual of diagnostic tests for aquatic animals 2011 provides standard diagnostic methods for OIE-listed diseases.

Reporting disease

Fishery and aquaculture industry managers, as well as farmers and their staff, should be aware of their responsibilities to rapidly report any suspicion of diseases on Australia's National List of Reportable Diseases of Aquatic Animals to local authorities.

In preparation for a possible disease incursion, fishery and aquaculture industry managers should develop an emergency management plan, in consultation with farmers and appropriate extension staff.

If you identify signs of disease in a culture system, contact your aquatic animal health officer. If your observation is of wild aquatic animals, contact a wildlife or fisheries officer. The contacts page at the end of this field guide provides current state and territory government contact details so that you can report your find and ask further questions on the observations you have made. You will be directed to an expert on diseases of aquatic animals within your state or territory. A national emergency animal disease watch hotline number has been established to assist early reporting of suspicious disease events. Call 1800 675 888 (free call and available 24 hours).

Follow the directions and advice provided by the officer you contact. This field guide will help you find the information the officer needs.

2 Anatomy

Finfish

The external and internal anatomy of finfish varies considerably across species. Specific adaptations to predator–prey interactions, aquatic habitat variability and dietary preferences can explain these differences. Examples include the short intestinal length of carnivorous fish compared with the relatively long intestine of herbivorous fish, and the ventrally directed mouth of bottom-feeding species such as European carp (*Cyprinus carpio*) compared with the upward oriented mouth of the surface-feeding saratoga (*Scleropages leichardti*).

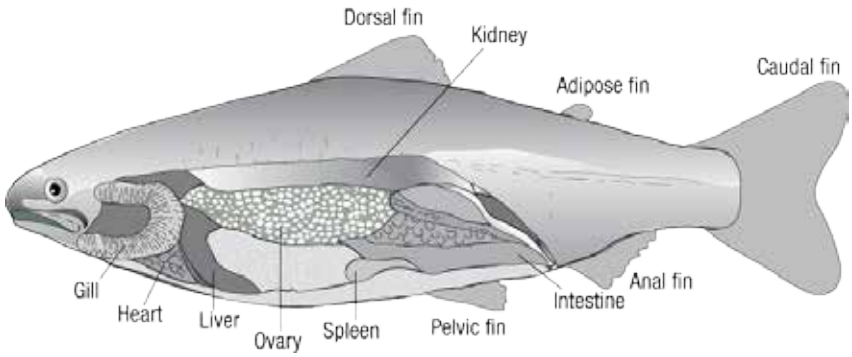
Gravid female Atlantic salmon (*Salmo salar*); note distended abdomen and protruding spawning vent

Source: M Porter

Male Atlantic salmon (*Salmo salar*) showing typical hooked mouth when mature

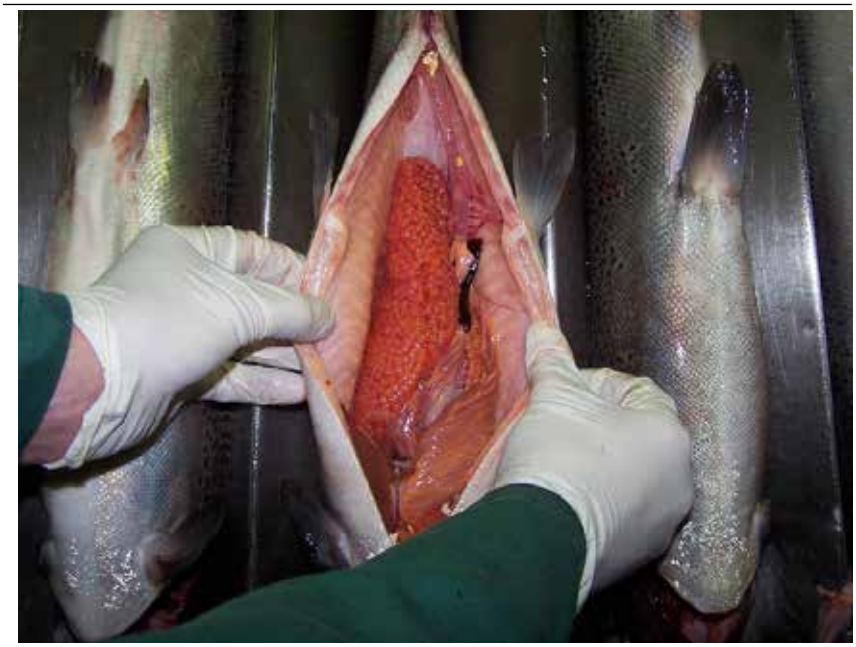
Source: M Porter

Anatomy of a juvenile salmon



Source: Aquatic Animal Health Program, Animal Health Policy, Australian Government Department of Agriculture, Fisheries and Forestry

Gravid female Atlantic salmon (*Salmo salar*). Note the stomach cavity dominated by the ovary. Compare the relative size of the ovary with the rest of the internal organs



Source: K Nelson

Gravid female Atlantic salmon (*Salmo salar*), showing location of the ovary (orange) in relation to the liver and intestines

Source: M Porter

Degenerative eggs in an old female Atlantic salmon (*Salmo salar*)

Source: M Porter

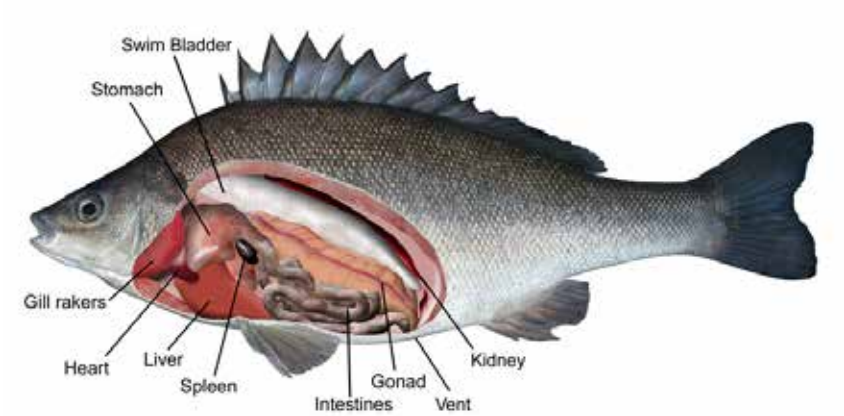
Golden perch (*Macquaria ambigua*)

Source: Illustration © State of New South Wales Department of Primary Industries 2006

Silver perch (*Bidyanus bidyanus*)

Source: Illustration © State of New South Wales Department of Primary Industries 2006

Anatomy of a silver perch



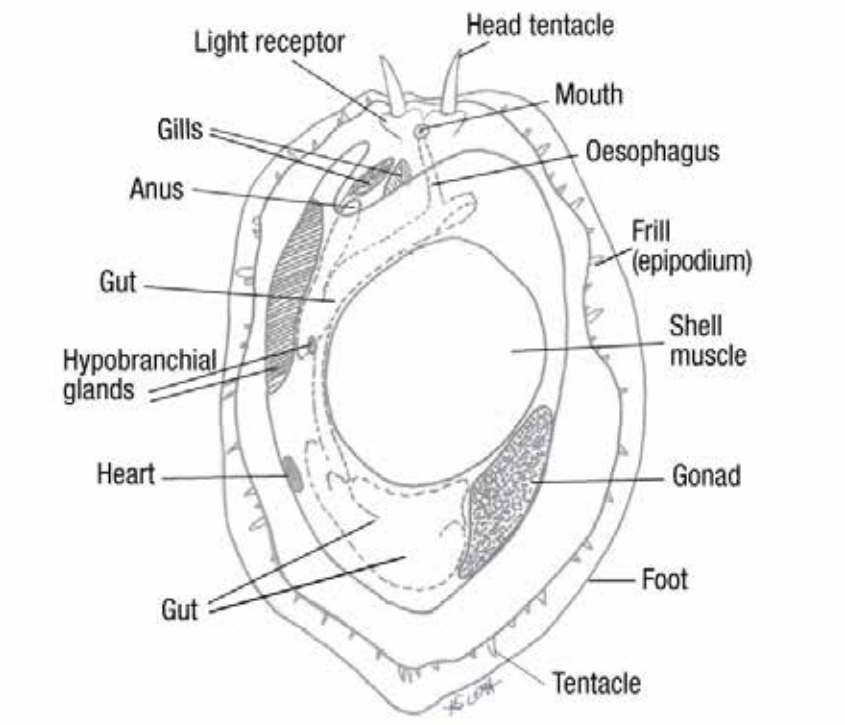
Source: Illustration © State of New South Wales Department of Primary Industries 2006

Molluscs

Abalone: top and bottom left—greenlip (*Haliotis laevigata*); top right—blacklip (*Haliotis rubra*); bottom right—hybrid tiger (*Haliotis laevigata* × *rubra*)

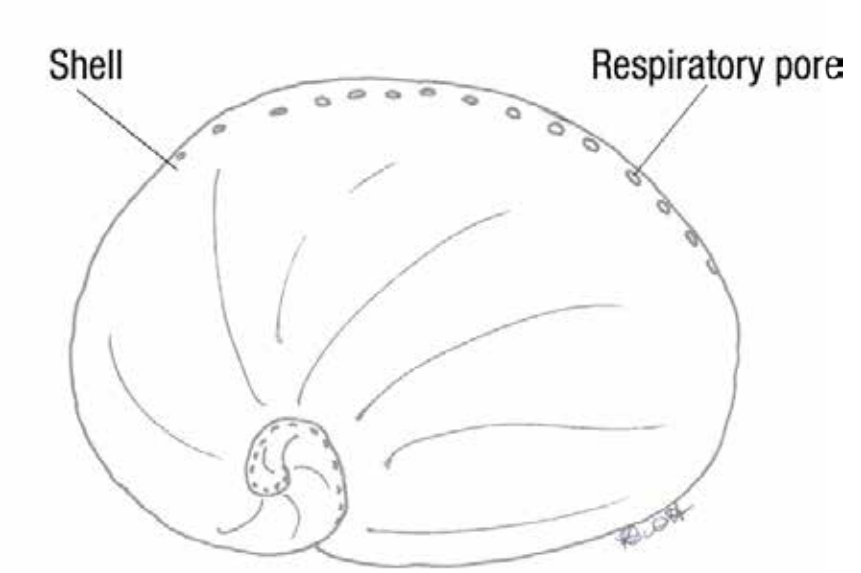
Source: K Scott

General anatomy of an abalone (*Haliotis* sp.)—ventral view



Source: K Scott

General anatomy of an abalone (*Haliotis* sp.)—dorsal view

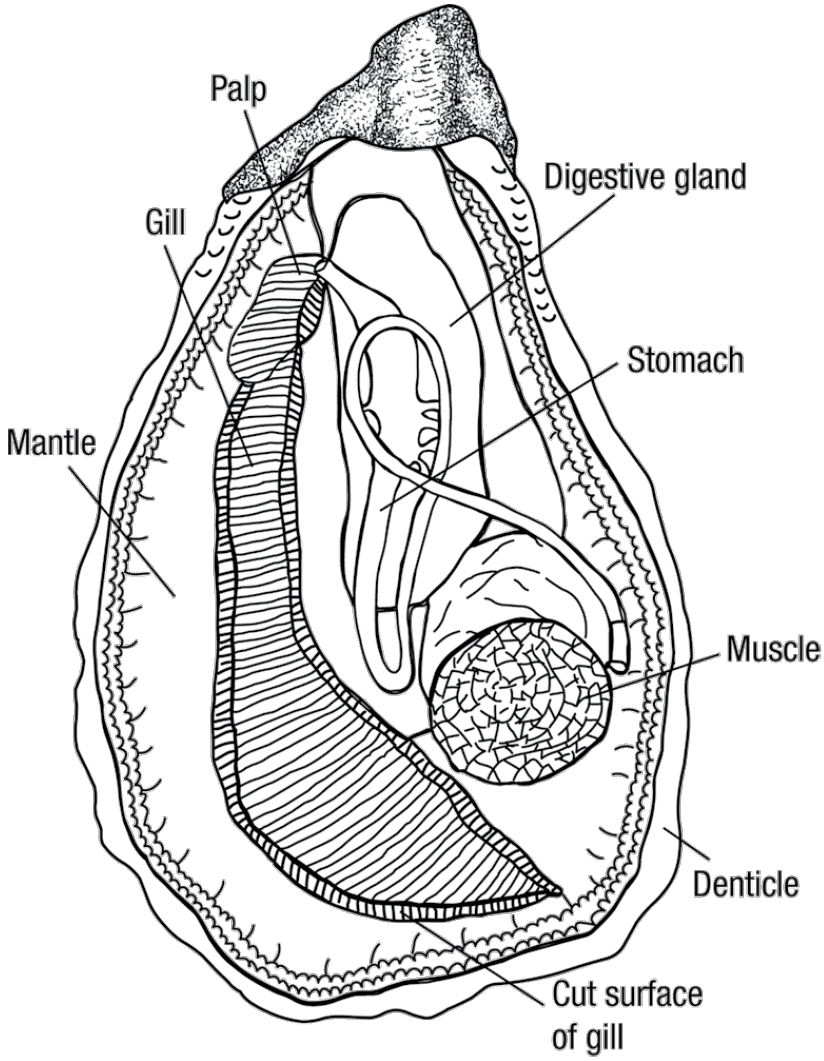


Source: K Scott

Healthy flat oyster

Source: French Research Institute for Exploration of the Sea—IFREMER

General anatomy of an oyster (*Ostrea* sp.)



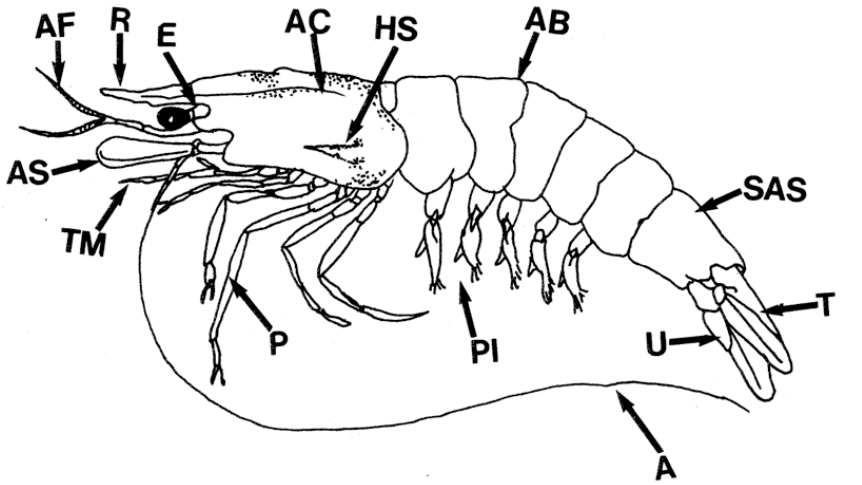
Source: Aquatic Animal Health Program, Animal Health Policy, Australian Government Department of Agriculture, Fisheries and Forestry

Crustaceans

Prawn

The common names 'prawn' and 'shrimp' are often used synonymously for the same crustaceans in different parts of the world. This field guide uses the terms most commonly used in Australia. Where Australian species of crustaceans are primarily involved in the disease, they are referred to as prawns. However, where the species of crustacean involved in the disease are exotic to Australia, they are referred to as shrimp.

External anatomy of a prawn



A = antenna; AB = abdominal segment; AC = adrostral carina; AF = antennular flagellum; AS = antennal scale; E = eyestalk; HS = hepatic spine; P = pereiopod; Pl = pleopod; R = rostrum; SAS = sixth abdominal segment; T = telson; TM = third maxilliped; U = uropod

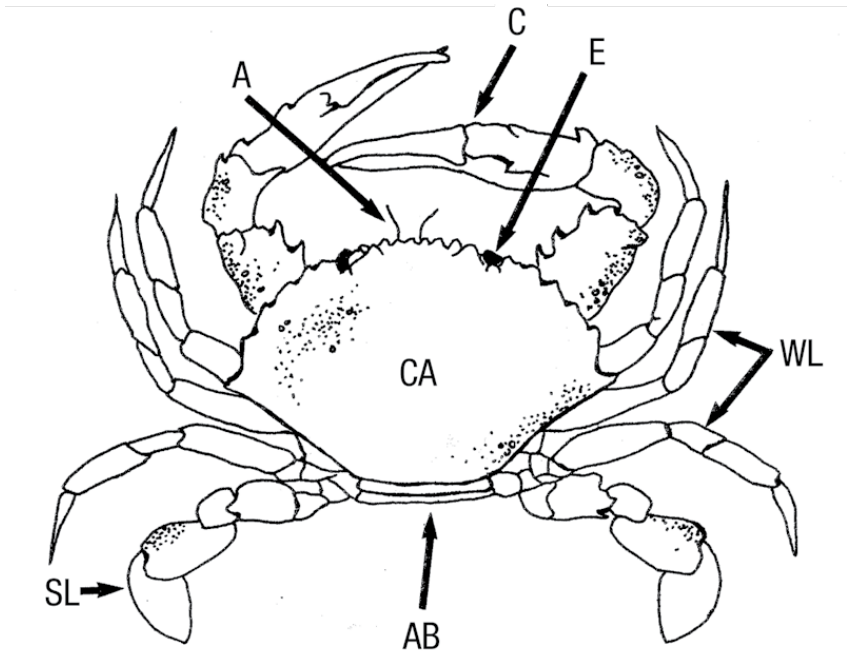
Source: Illustration © State of Queensland, Department of Primary Industries and Fisheries, 2008 (illustrator: R Bowater)

Black tiger prawn (*Penaeus monodon*)

Source: Aquatic Animal Health Program, Animal Health Policy, Australian Government Department of Agriculture, Fisheries and Forestry

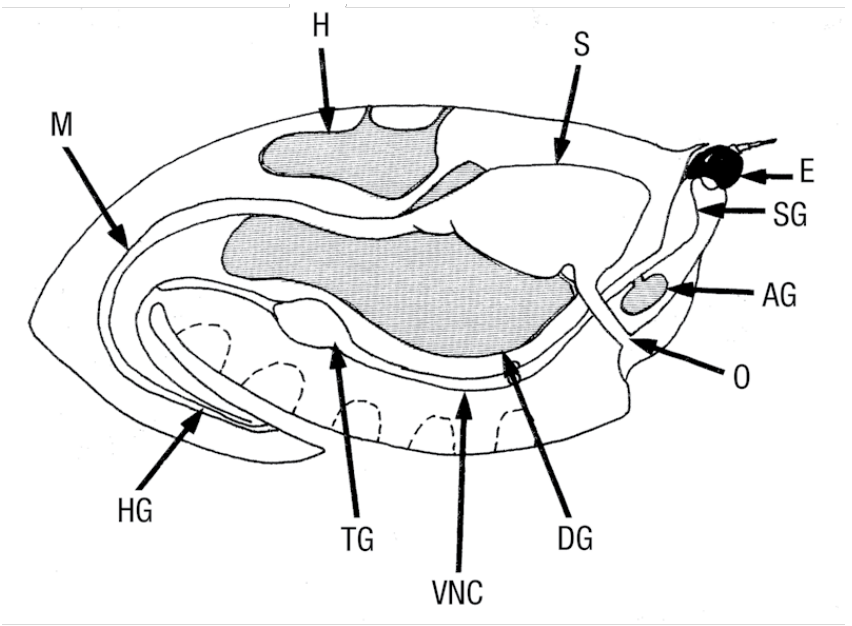
Crab

External anatomy of a crab



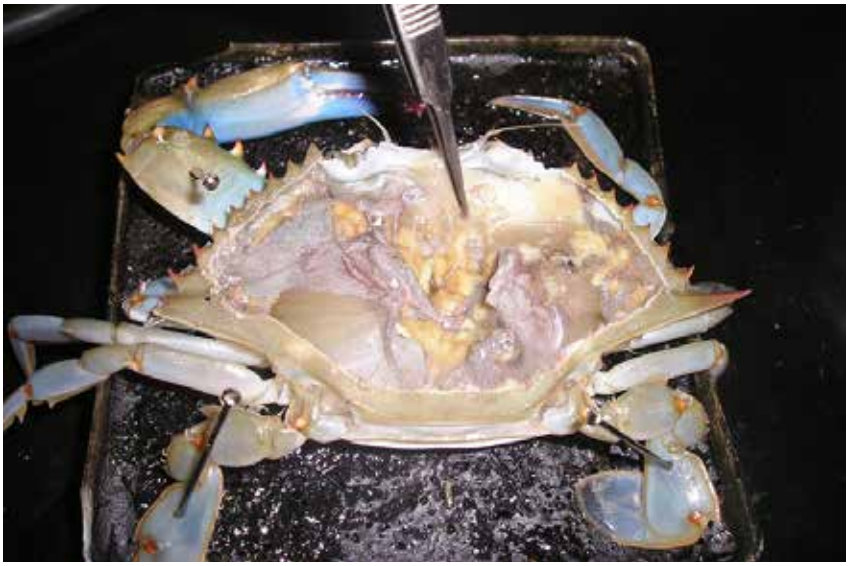
A = antenna; AB = abdomen (underneath); C = claw; CA = carapace; E = eye; SL = swimming leg; WL = walking leg
Source: Illustration © State of Queensland, Department of Primary Industries and Fisheries, 2008 (illustrator: R Bowater)

Internal anatomy of a crab: saggital section (cut down the centre and looking side on)



AG = antennal gland; DG = digestive gland; E = eye; H = heart; HG = hindgut; M = midgut; O = oesophagus; S = stomach; SG = supraoesophageal ganglion; TG = thoracic ganglion; VNC = ventral nerve cord
Source: Illustration © State of Queensland, Department of Primary Industries and Fisheries, 2008 (illustrator: R Bowater)

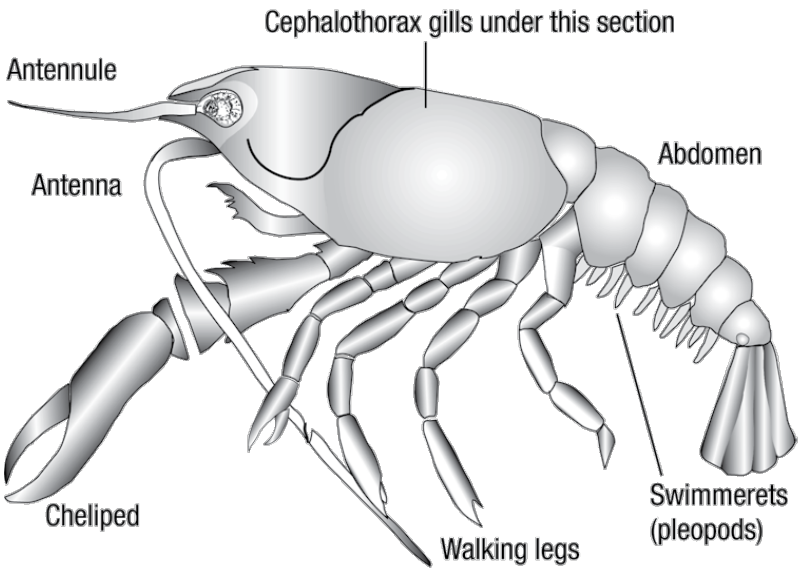
Blue swimmer crab (*Callinectes sapidus*) with carapace removed, revealing internal structure



Source: O Zmora

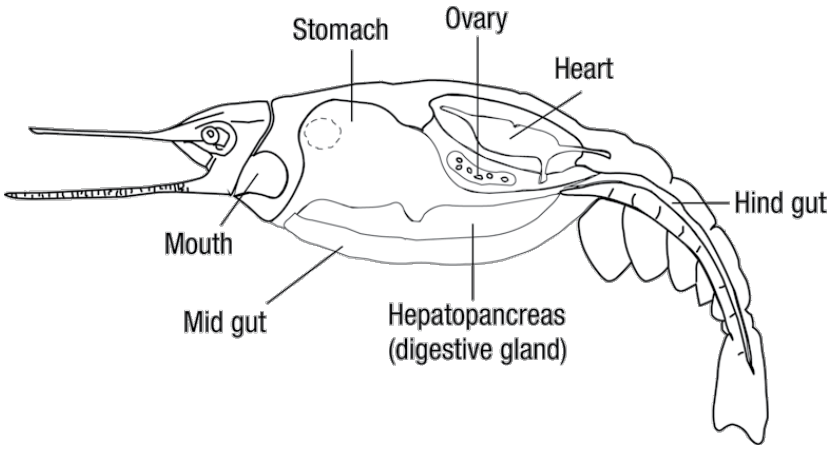
Crayfish

External anatomy of a freshwater crayfish



Source: Aquatic Animal Health Program, Animal Health Policy, Australian Government Department of Agriculture, Fisheries and Forestry

Internal organs in section



Source: Aquatic Animal Health Program, Animal Health Policy, Australian Government Department of Agriculture, Fisheries and Forestry

Redclaw crayfish (*Cherax quadricarinatus*)

Source: C Jones

Redclaw crayfish (*Cherax quadricarinatus*) with section of carapace removed, revealing location of the gills

Source: C Jones

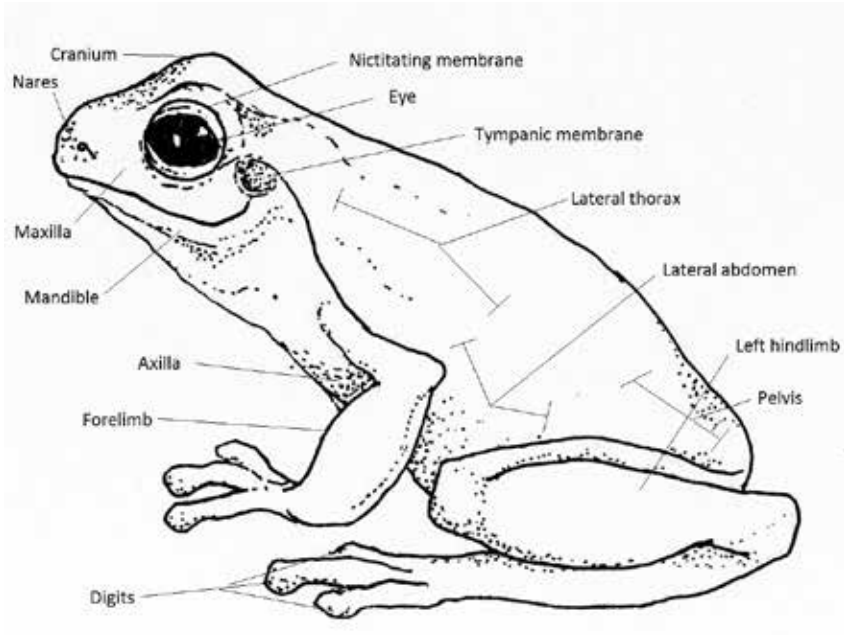
Amphibians

Amphibians are comprised of the orders Anura (frogs and toads), Caudata (including salamanders, newts and sirens) and Gymnophiona (caecilians). In Australia, we are primarily concerned with the health of anurans. Amphibians undergo a metamorphosis that is unique among vertebrates, from a juvenile, obligatory aquatic form with gills (tadpoles) to a semi-aquatic adult form with lungs. Adult amphibians also have the unique ability to breathe transdermally (through the skin); thus, changes to the skin through disease, trauma or mishandling can have significant health impacts.

Early recognition and a deep understanding of the significant diseases affecting amphibians will be crucial to halting the current decline in species biodiversity seen in Australia. Although not of great commercial interest in Australia, native amphibians act as sentinels of change in aquatic environments.

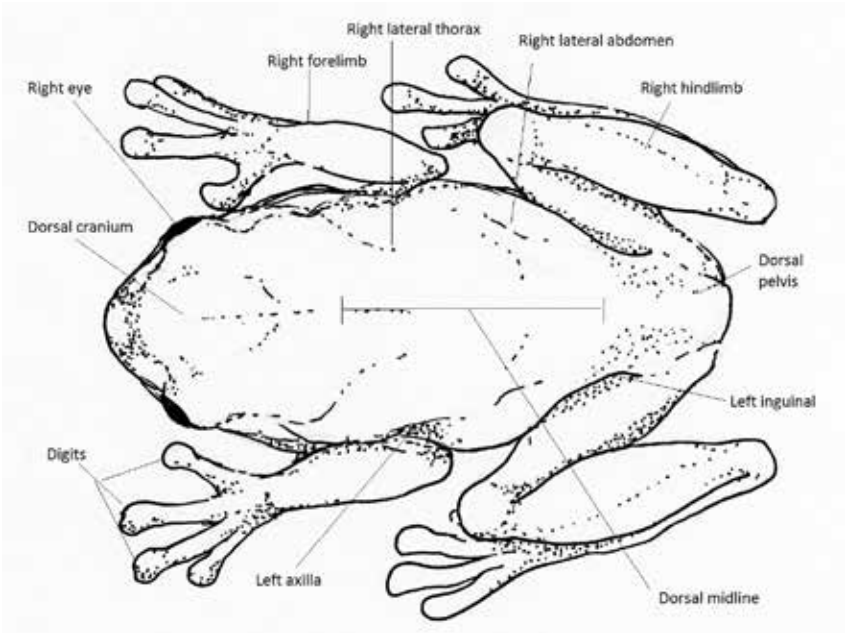
The anatomical descriptions below are not exhaustive but are intended to help field guide users communicate the location and extent of gross external anatomical changes.

External anatomical descriptions for a frog: left lateral view



Source: Aquatic Animal Health Program, Animal Health Policy, Australian Government Department of Agriculture, Fisheries and Forestry

External anatomical descriptions for a frog: dorsal view



Source: Aquatic Animal Health Program, Animal Health Policy, Australian Government Department of Agriculture, Fisheries and Forestry

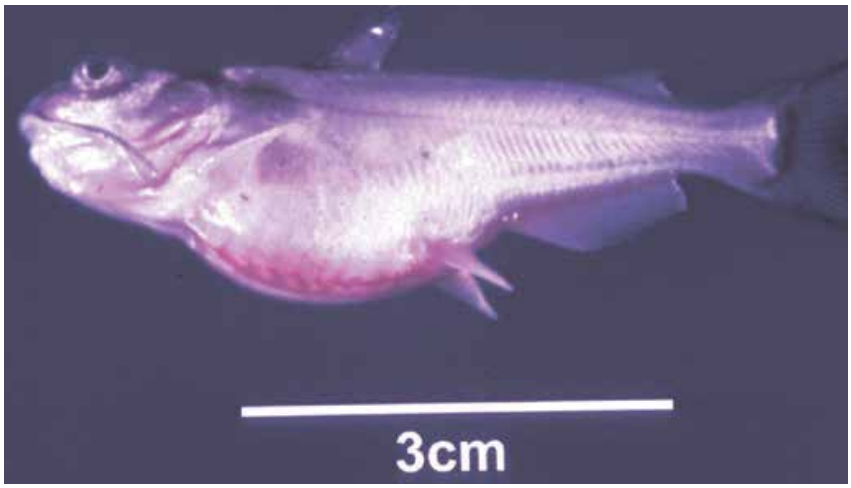
3 Diseases of finfish

Exotic disease

Viral diseases of finfish

Channel catfish virus disease (CCVD)

CCVD-affected channel catfish fingerling; note swollen stomach and 'popeye'



Source: LA Hanson

Haemorrhages present on the base of the body, gills and fins of channel catfish infected with CCV

Source: United States Department of Agriculture

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- decreased feeding activity (usually the first sign)
- high mortality in fry and juvenile ictalurid catfish
- erratic swimming
- brief episodes of hyperactivity when fish are disturbed, followed by extended periods of lethargy
- large congregations of fish at the sides of hatching troughs or ponds, motionless in a head-up, tail-down position.

Gross pathological signs are:

- swollen abdomen
- exophthalmos (popeye)
- haemorrhaging of fins and ventral abdomen
- haemorrhaging of the musculature, liver and kidneys
- dark and enlarged spleen
- fluid in the abdominal cavity
- pale, enlarged kidneys, which may be the only internal indication of disease in infected fish.

Microscopic pathological signs are:

- extensive necrosis of renal tubules and interstitial tissues of the kidney.

Disease agent

CCVD is caused by a herpesvirus (ictalurid herpesvirus 1).

Host range

Ictalurid herpesvirus 1 has been isolated from crucian carp (*Carassius carassius*) and common carp (*Cyprinus carpio*) not exhibiting disease signs. It is currently unknown if they act as reservoirs for the virus.

Species known to be susceptible to CCVD are listed below.

Common name [a]	Scientific name
Blue catfish	*Ictalurus furcatus*
Channel catfish	*Ictalurus punctatus*

a Both species listed are naturally susceptible (other species have been shown to be experimentally susceptible).

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Horizontal transmission occurs directly from virus shed in water, and from virus carried by animal vectors and on fomites.
- Vertical transmission (from parent to offspring via eggs) is believed to be common.
- Mortality rates in exposed fish can increase to greater than 95%. Survivors of CCVD may experience short-term reductions in feed conversion (reduced weight gain). Generally, these fish appear normal but become carriers of the virus.
- Susceptibility appears to vary according to the strain of the virus.
- The disease occurs almost exclusively in fish that are less than 1 year old (fry and fingerlings) or smaller than 15 cm in length. The majority of occurrences are in fish less than 4 months old.
- Mortality rates are highest where water temperature exceeds 27 °C and declines with a reduction in temperature. Mortality rates are negligible at water temperatures lower than 18 °C.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Aeromonas salmonicida—atypical strains, enteric septicaemia of catfish

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found on the Centre for Environment, Fisheries and Aquaculture Science (Cefas) International Database on Aquatic Animal Disease (IDAAD) website at www.cefas.defra.gov.uk/idaad/disocclist.aspx.

This hyperlink was correct and functioning at the time of publication.

Epizootic haematopoietic necrosis (EHN)

Mass mortality of redfin perch (*Perca fluviatilis*); note the small size of individuals affected and the swollen abdomen of the fish at the centre of the photograph

Source: J Humphrey

Note the characteristic haemorrhagic gills of the redfin perch (*Perca fluviatilis*) on the left

Source: J Humphrey

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- typically many (hundreds or thousands) small, dead fish less than 10 cm long found on the downwind bank of the water body
- large numbers of fish-eating birds (e.g. seagulls) at the water surface
- loss of appetite
- juveniles (<25 mm long) often swimming in a disorientated fashion at the surface
- occasional adults affected when the disease first arrives in an area.

Gross pathological signs are:

- swollen abdomen
- darkened skin colour
- petechial (pinpoint) haemorrhages at the base of the fins
- haemorrhaging of the gills
- dead fish
- enlargement of the kidney, liver and spleen
- focal white to yellow liver lesions.

Microscopic pathological signs are:

- coagulative or liquefactive necrosis of the liver, kidney and spleen
- necrotic lesions in the heart, pancreas, gastrointestinal tract and gills
- small numbers of basophilic intracytoplasmic inclusion bodies surrounding necrotic areas of the liver and kidney.

Disease agent

EHN in Australia is caused by epizootic haematopoietic necrosis virus (EHNV), a systemic iridovirus (*Ranavirus*).

Closely related ranaviruses cause similar systemic necrotising iridovirus syndromes in sheatfish and catfish in Europe (European sheatfish virus and European catfish virus).

Note: In this guide, only EHN disease caused by EHNV is discussed.

Host range

Species known to be susceptible to EHN are listed below.

Common name [a]	Scientific name
Macquarie perch	*Macquaria australasica*
Mosquito fish	*Gambusia affinis*
Mountain galaxias	*Galaxias olidus*
Murray cod [b]	*Maccullochella peelii peelii*
Rainbow trout [a]	*Oncorhynchus mykiss*
Redfin perch [a] or European perch	*Perca fluviatilis*
Silver perch	*Bidyanus bidyanus*

a Naturally susceptible (other species have been shown to be experimentally susceptible). **b** Demonstrated to carry EHNV.

Presence in Australia

EHN has been officially reported from the Australian Capital Territory, New South Wales, South Australia and Victoria.

Epidemiology

- EHN is usually seen in Australia as large kills of small redfin perch. High mortality can also occur among older perch in newly affected areas.
- Clinical outbreaks are typically seen in fingerlings and juvenile fish, associated with poor water quality and/or certain water temperatures (between 11 °C and 17 °C in rainbow trout and above 12 °C in redfin perch).
- Mortalities occur over a short period (several weeks) in summer, and then the disease may disappear from an area for years.
- Low mortality rates over a period of months have been reported in young, farmed rainbow trout.
- Infectivity is less severe in rainbow trout than in redfin perch, with the disease mainly affecting fingerlings <125 mm long.
- Low-grade mortalities with covert EHNV infection can also occur in juvenile fish. Therefore, care must be taken when moving redfin perch and rainbow trout from the known geographical range of EHNV to areas where it is exotic, unless freedom can be adequately demonstrated.
- EHN is a resistant virus, surviving for months in water, persisting in frozen fish tissues for more than two years and in fish carcases for at least one year.
- EHNV may be carried on equipment including nets and boats, in fish (live or dead) used for bait and via the gut, feathers, feet and bill of piscivorous birds.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not

included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Furunculosis, infectious haematopoietic necrosis, viral haemorrhagic septicaemia

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of EHN are summarised in the World Organisation for Animal Health Manual of diagnostic tests for aquatic animals 2011, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

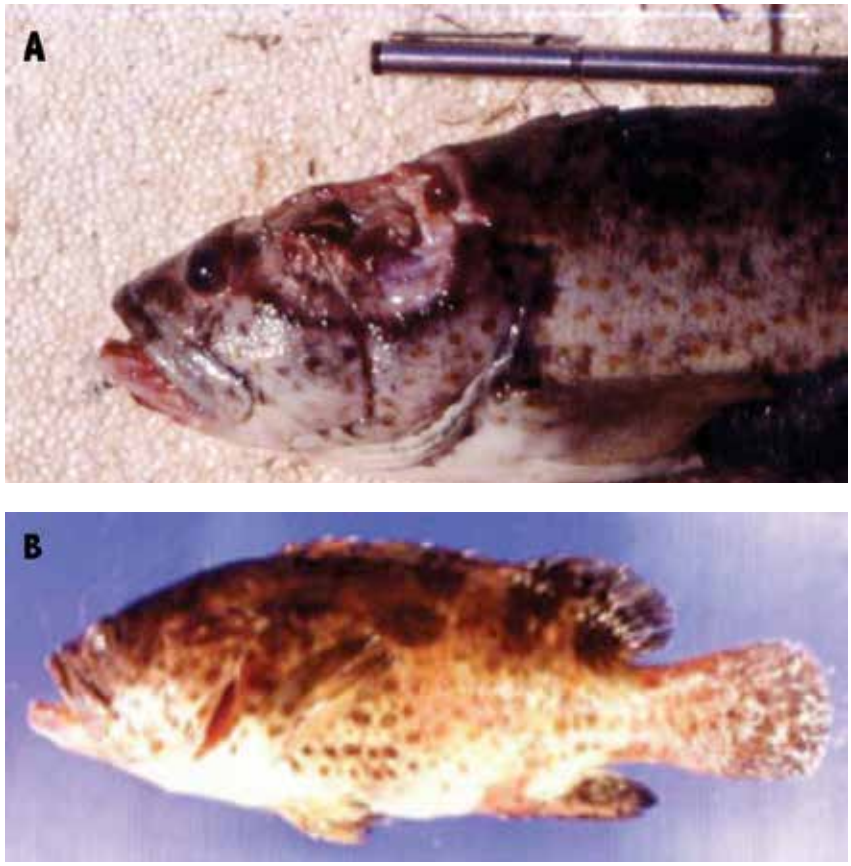
Further information can be found on the disease pages of Fisheries and Oceans Canada at www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm.

These hyperlinks were correct and functioning at the time of publication.

Exotic disease

Grouper iridoviral disease
(Also known as sleepy grouper disease)

Gross signs of secondary infection in grouper infected by grouper iridovirus, showing (a) deep ulceration in muscular tissue and (b) red boils on the body surface



Source: S Kanchanakhan

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- typically high mortalities, with many dead and moribund fish
- high mortality of young grouper
- innappetance
- lethargic swimming
- rapid opercular movements and dashing to the surface for air (in the terminal phase).

Gross pathological signs are:

- darkened body colour, pale gills and enlarged spleen
- signs of secondary infection such as deep ulceration or papular lesions.

Microscopic pathological signs are:

- necrosis of the splenic pulp and myocardium
- necrosis and reduction of haematopoietic tissue.

Disease agent

The infectious agent is grouper iridovirus (GIV). Synonyms include grouper iridovirus of Taiwan (TGIV) and Singapore grouper iridovirus (SGIV).

Host range

Fish known to be susceptible to GIV are listed below.

Common name [a]	Scientific name
Estuarine rock cod	*Epinephelus tauvina*
Malabar grouper	*Epinephelus malabaricus*
Yellow grouper	*Epinephelus awoara*

a All the listed species are naturally susceptible (other species have been shown to be experimentally susceptible).

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Grouper iridovirus disease causes losses not only in fry and juvenile grouper but also in 1–2-year-old, market-sized grouper, a highly priced product in tropical mariculture.
- Clinical disease and highest mortalities are usually seen in 3–4-month-old fish after stocking into sea cages.
- Older diseased fish typically appear lethargic due to anaemia.
- Horizontal contact and waterborne transmission appear to be the principal mechanisms for virus spread.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Viral encephalopathy and retinopathy

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found on the Network of Aquaculture Centres in Asia–Pacific website: library.enaca.org/Health/DiseaseLibrary/Disease_card_GIV_Nakajima.pdf.

This hyperlink was correct and functioning at the time of publication.

Infection with infectious spleen and kidney necrosis virus (ISKNV)-like viruses

Exotic disease

ISKNV in dwarf gourami: dwarf gourami naturally infected with an ISKNV-like iridovirus (top fish) showing pale colouration compared with unaffected fish (bottom fish)

Source: J Go

ISKNV in Murray cod fingerling: Murray cod fingerling experimentally infected with an ISKNV-like iridovirus showing discolouration around the front of the body (normal colouration evident near the tail) and signs of respiratory distress at time of death (flared opercula)

Source: J Go

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- mortality between 50% and 100%
- fish remaining on bottom of tank and not feeding well
- lethargy
- respiratory distress (rapid movement of opercula).

Gross pathological signs are:

- changes in body colour (e.g. darkening or lightening of body colour)
- exophthalmos (popeye)
- abdominal distension (due to fluid or enlargement of organs).

Microscopic pathological signs are:

- basophilic hypertrophied cells throughout numerous body tissues, but especially the haematopoietic tissues of the kidney and spleen.

Disease agent

ISKNV-like viruses are a group of viral agents in the genus *Megalocytivirus*, family *Iridoviridae*. These viruses predominantly cause disease in freshwater fish (particularly cichlids, gouramis and poeciliids). The megalocytivirus agents included in this disease grouping are:

Cichlids[1]:

- Angelfish iridovirus
- Cichlid iridovirus[2] (includes ram cichlid and chromide cichlid)
- Iridovirus in *Apistogramma* spp.
- Iridovirus in oscars
- Iridovirus in rainbow krib
- Iridovirus in curviceps

Gouramis[3]:

- Dwarf gourami iridovirus
- Pearl gourami iridovirus
- Iridovirus in thick-lipped gourami, three-spot gourami and silver gourami
- Iridovirus in paradise fish

Poeciliids[4]:

- African lampeye iridovirus[5]
- Swordtail iridovirus[6]
- Iridovirus in mollies and platys
- Iridovirus in guppies.

1 All fish belonging to the family Cichlidae.
2 Although currently uncharacterised, based on histopathology cichlid iridoviruses are considered to be megalocytiviruses.
3 Fish of the subfamilies Luciocephalinae and Macropodinae, family Osphronemidae.
4 All fish belonging to the family Poeciliidae.
5 All fish belonging to the subfamily Aplocheilichthyinae, family Poeciliidae.
6 Although currently uncharacterised, based on histopathology swordtail iridovirus and other iridoviruses identified under the family Poeciliidae are considered to be megalocytiviruses.

Host range

Species known to be susceptible to ISKNV-like viruses are listed below. The list includes some native Australian fish species known to be highly susceptible to ISKNV-like viruses.

Common name [a]	Scientific name
African lampeye killifish **a**	*Aplocheilichthys normani*
Angelfish **a**	*Pterophyllum scalare*
Banggai cardinalfish **a**	*Pterapogon kauderni*
Barramundi **a**	*Lates calcarifer*
Chinese perch or mandarin fish **a**	*Siniperca chuatsi*
Curviceps **a**	*Laetacara curviceps*
Dwarf cichlids **a**	*Apistogramma* spp.
Dwarf gourami **a**	*Colisa lalia*
Flathead mullet **a**	*Mugil cephalus*
Grass carp	*Ctenopharyngodon idellus*
Grouper **a**	*Epinephelus sp.*
Guppy **a**	*Poecilia reticulata* (also known as *Lebistes reticulatus*)
Japanese parrotfish	*Oplegnathus fasciatus*
Largemouth bass	*Micropterus salmoides*
Marble goby **a**	*Oxyeleotris marmoratus*
Molly **a**	*Poecilia sphenops*
Murray cod **a**	*Maccullochella peelii peelii*
Nile tilapia **a**	*Oreochromis niloticus*
Orange chromide **a**	*Etroplus maculatus*
Oscara	*Astronotus ocellatus*
Paradise fish **a**	*Macropodus opercularis*
Pearl gourami **a**	*Trichogaster leerii*
Rainbow krib **a**	*Pelvicachromis pulcher*
Ram cichlid **a**	*Mikrogeophagus ramirezi*
Red drum **a**	*Sciaenops ocellatus*
Sailfin mollies **a**	*Poecilia latipinna*
Silver gourami **a**	*Trichogaster microlepis*
Southern platyfish or red wagtail platy **a**	*Xiphophorus maculatus*
Swordtail or green swordtail **a**	*Xiphophorus helleri*
Thick-lipped gourami **a**	*Colisa labiosa*
Three-spot gourami **a**	*Trichogaster trichopterus*
Zebrafish	*Danio rerio*

a Naturally susceptible (other species have been shown to be experimentally susceptible).

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- ISKNV-like viruses are prone to inactivation by desiccation or heat at temperatures above 50 °C, but are stable in water at 4 °C for extended periods.
- ISKNV-like viruses have been found to cause disease in a wide variety of species (i.e. may lack strict host specificity).

- Based on experimental transmission studies, horizontal transmission via cohabitation, water, ingestion of excreta or cannibalism is likely.
- There is evidence that some species may be long-term asymptomatic carriers of ISKNV-like viruses, and that prevalence in infected populations may be high.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Epizootic haematopoietic necrosis, grouper iridoviral disease, red sea bream iridoviral disease

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Biosecurity Australia 2010, *Importation of freshwater ornamental fish: review of biosecurity risks associated with gourami iridovirus and related viruses—provisional final import risk analysis report*, Biosecurity Australia, Canberra.

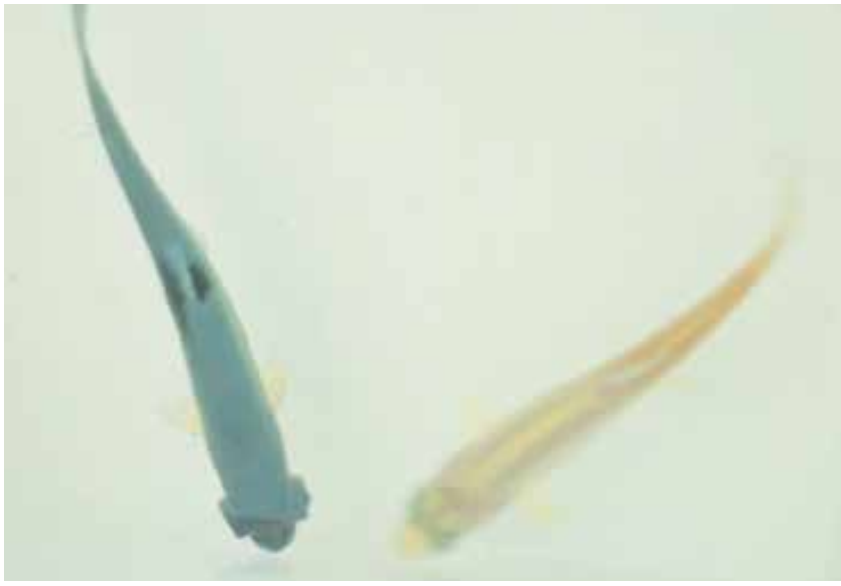
Infectious haematopoietic necrosis (IHN)

Exotic disease

Chinook salmon fry (*Oncorhynchus tshawytscha*) with IHN; note characteristic darkening from the tail region, swollen stomach and haemorrhaging at base of the fins

Source: J Fryer

Rainbow trout fry (*Oncorhynchus mykiss*) with (left) and without (right) IHN; note the darker colour of the infected fish



Source: G Kurath

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

The disease signs described below are seen only in young salmonids; infection in adults is subclinical.

Disease signs at the farm, tank or pond level are:

- mass mortality
- lethargic swimming with intermittent bouts of frenzied, abnormal activity (spiral swimming and flashing).

Gross pathological signs are:

- numerous yolk sac haemorrhages in the fry of rainbow trout and salmon
- darkening of the skin and pale gills
- haemorrhages on the abdomen and around the pupil of the eye
- exophthalmos (popeye) and a swollen abdomen
- trailing white faecal cast
- bleeding at the base of the fins
- stomach empty of food but distended with a gelatinous substance
- pale internal organs
- petechial (pinpoint) haemorrhages in the fatty tissue and muscle surrounding the organs and stomach wall
- spinal deformities in surviving fish.

Microscopic pathological signs are:

- necrosis of eosinophilic granular cells in the intestinal wall
- degenerative necrosis in haematopoietic tissues.

Disease agent

IHN is caused by infectious haematopoietic necrosis virus (IHNV), a virus in the family *Rhabdoviridae* and genus *Novirhabdovirus* that is related to viral haemorrhagic septicaemia virus.

Host range

Species known to be susceptible are listed below.

Common name [a]	Scientific name
Amago salmon **a**	*Oncorhynchus rhodurus*
Arctic char	*Salvelinus alpinus*
Atlantic cod **a**	*Gadus morhua*
Atlantic salmon **a**	*Salmo salar*
Ayu	*Plecoglossus altivelis*
Brook trout **a**	*Salvelinus fontinalis*
Brown trout **a**	*Salmo trutta*
Chinook salmon **a**	*Oncorhynchus tshawytscha*
Chum salmon **a**	*Oncorhynchus keta*
Coho salmon **a**	*Oncorhynchus kisutch*
Cutthroat trout **a**	*Oncorhynchus clarkii*
Gilt-head sea bream	*Sparus aurata*
Grayling	*Thymallus thymallus*
Lake trout	*Salvelinus namaycush*
Masu salmon **a**	*Oncorhynchus masou*
Pacific herring	*Clupea pallasii*
Pacific salmon **a**	*Oncorhynchus* spp.
Pike	*Esox lucius*
Pile perch	*Damalichthys vacca*
Pink salmon **a**	*Oncorhynchus gorbuscha*
Rainbow trout **a**	*Oncorhynchus mykiss*
Shiner perch	*Cymatogaster aggregata*
Sockeye salmon **a**	*Oncorhynchus nerka*
Tubesnout	*Aulorhynchus flavidus*
Turbot	*Scophthalmus maximus*
Whitespotted char	*Salvelinus leucomaenis*
White sturgeon	*Acipenser transmontanus*

a Naturally susceptible (other species have been shown to be experimentally susceptible).

Non-fish carriers include the species listed below.

Common name	Scientific name
Gill lice	*Salminicola* spp.
Leeches	*Piscicola* spp.
Mayfly	*Callibaetis* spp.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- IHN is typically found in young, farmed trout and salmon fry or fingerlings, and in adults during or just following spawning.
- Mass mortalities can occur in hatcheries, typically with 100% of the population infected and mortality as great as 90%.
- Mortality also occurs in wild populations of Pacific salmon infected with IHNV, and survivors can be the source of infection of farmed stock.
- Susceptibility to infection varies between individuals of the same species and appears to be largely age dependent, with younger individuals being more susceptible.
- Fish that survive IHN are potential carriers of the virus for a period that depends on environmental conditions (e.g. temperature).
- The virus is shed into the water from infected fish through faeces, urine, spawning fluids and external mucous.
- Transmission is generally horizontal, with the virus entering fish through the gills and skin, although some blood-sucking parasites may serve as vectors.
- Virus can be transferred to new areas via the movement of infected fish or eggs, and by other sources such as contaminated equipment, water or birds.
- Outbreaks are most likely to occur around the time of spawning, with increased levels of virus released into the environment with spawning fluids.
- IHN is a cold-water disease. Clinical signs typically occur at temperatures between 8 °C and 15 °C. Outbreaks rarely occur once water temperatures reach more than 15 °C.
- It is believed that the spread of IHNV from the west coast of North America to Asia and Europe has been principally via the shipment of infected fish and eggs, suggesting some degree of vertical transmission. Although the risk of such egg-associated transmission is significantly reduced by the common practice of egg surface disinfection, it is not eliminated.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Enteric red mouth disease, epizootic haematopoietic necrosis, furunculosis, infectious pancreatic necrosis, infectious salmon anaemia, viral haemorrhagic septicaemia, whirling disease

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of IHN are summarised in the World Organisation for Animal Health Manual of diagnostic tests for aquatic animals 2011, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information can be found on the Centre for Environment, Fisheries and Aquaculture Science (Cefas) International Database on Aquatic Animal Disease (IDAAD) website at www.cefas.defra.gov.uk/idaad/disocclist.aspx.

These hyperlinks were correct and functioning at the time of publication.

Exotic disease

Infectious pancreatic necrosis

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- sudden and progressive increase in mortality at first feeding of fry, particularly in faster growing individuals
- cumulative mortality rates from 10% to 90%
- low persistent mortality
- fish lying still on the bottom
- fish swimming with a spiralling, corkscrew motion.

Gross pathological signs are:

- long, thin, whitish trailing faecal casts
- swollen abdomen
- darkening body colour
- gills typically pale
- exophthalmos (popeye)
- lesions and ulcers in the pancreas, oesophagus and stomach
- haemorrhages sometimes present in ventral areas, including the ventral fins
- abnormally pale spleen, kidney, liver and heart of fry
- intestines empty or filled with clear mucus.

Infectious pancreatic necrosis in rainbow trout (*Oncorhynchus mykiss*). Note the abdominal distension and darkened body colour

Source: T Håstein

Microscopic pathological signs are:

- extensive and/or severe necrosis of acinar pancreatic cells
- focal or generalised necrosis of the liver
- sloughing of intestinal mucosa with characteristic 'McKnight' cells in the lumen (eosinophilic and hyaline epithelial cells).

Disease agent

IPN virus is the type species of the genus *Aquabirnavirus* (family *Birnaviridae*). Several serotypes have been identified and these are divided into serogroups A and B. Serotypes are described by their different geographical origins, the most frequently found and highly virulent is the Sp serotype. The signs above are representative of IPN in salmonids.

Host range

Species likely to be severely affected by IPN virus are listed below.

Common name [a]	Scientific name
Atlantic salmon	*Salmo salar*
Brook trout	*Salvelinus fontinalis*
Brown trout	*Salmo trutta*
Coho salmon [a]	*Oncorhynchus kisutch*
Pacific salmon [a]	*Oncorhynchus* spp.
Rainbow trout	*Oncorhynchus mykiss*

a All species listed are naturally susceptible (other species have been shown to be experimentally susceptible).

Presence in Australia

EXOTIC DISEASE—not present in Australia

Other aquabirnaviruses have been identified in farmed Atlantic salmon in Tasmania, Australia, and in diseased turbot and asymptomatic sea-run chinook salmon in New Zealand. The Tasmanian *Aquabirnavirus* is distinct from IPN virus, it is not associated with high mortalities and only occurs in Macquarie Harbour on the west coast of Tasmania.

Epidemiology

- IPN virus is highly contagious and fish that survive following infection are presumed to become carriers. Asymptomatic carrier fish represent a risk for introduction to healthy stocks.
- Viral transmission can occur horizontally; the virus enters fish through the gills or gastrointestinal tract, and vertically; transmitted via eggs of infected carrier broodfish.
- IPN virus is shed in faeces, urine, spawning fluids and external mucus. Spawning favours the transmission of IPN virus with increased levels of virus excreted in spawning fluids.
- Outbreaks of disease are most likely to occur when fish are stressed. Factors raising physiological stress levels include first feeding, high stocking densities, fluctuations in water temperature and salinity and management practices requiring handling of fish. Outbreaks are known to occur at water temperatures as low as 4°C and as high as 18°C.
- The disease can cause high mortalities (70%) in young trout, with cumulative mortalities varying from 10% to 90%.
- The highest mortality rates usually occur in freshwater hatcheries in fry less than 6 months of age. However, IPN is known to affect post-smolt Atlantic salmon after transfer from freshwater to seawater.
- IPN virus can survive in both fresh and salt water environments. It is quite stable and resists destruction by disinfection, thus enabling it to persist in a range of environmental conditions on equipment such as nets and containers.
- Virus may be spread and healthy stocks exposed via contaminated transport water, infected eggs and blood feeding parasites. Piscivorous birds are also known vectors of IPN virus.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infectious haematopoietic necrosis, infectious salmon anaemia, viral haemorrhagic septicaemia

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

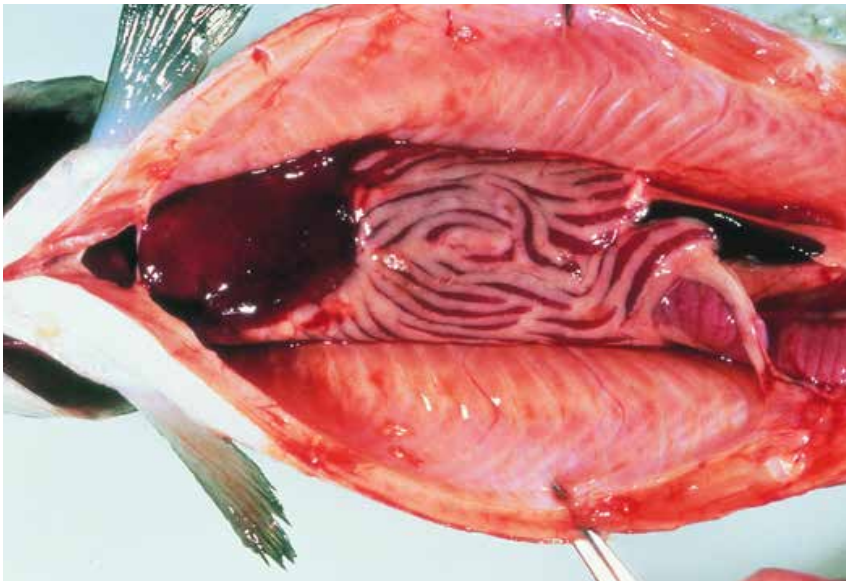
Further information can be found on the Centre for Environment, Fisheries and Aquaculture Sciences (Cefas) International Database on Aquatic Animal Disease (IDAAD) website at www.cefas.defra.gov.uk/idaad/disocclist.aspx.

This hyperlink was correct and functioning at the time of publication.

Infectious salmon anaemia (ISA)

Exotic disease

Atlantic salmon (*Salmo salar*) with ISA, showing gross lesions, dark liver, ascites and enlarged spleen



Source: T Poppe

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- mortality rate up to 100%
- fish congregating near the surface
- fish gasping at the surface
- lethargy
- loss of appetite.

Gross pathological signs are:

- pale gills
- swollen abdomen
- exophthalmos (popeye)
- bleeding eyes and fin rot
- ecchymotic (bruise-like) skin haemorrhages
- scale-pocket oedema
- swollen and dark liver, kidney and spleen (early sign); liver may be almost black
- petechial (pinpoint) haemorrhages in internal fat, peritoneum and skeletal muscle

- dark red intestinal wall mucosa
- ascites (fluid in the abdominal cavity)
- surface haemorrhages on the liver
- pale heart.

Microscopic pathological signs are:

- renal interstitial haemorrhage and tubular necrosis
- branchial lamellar and filamental congestion
- congestion of the intestine and pyloric caecae
- perivascular inflammation and focal necrosis in the liver.

Disease agent

ISA virus is in the *Orthomyxoviridae* family of viruses.

Host range

The only species known to display clinical signs of ISA is the Atlantic salmon (*Salmo salar*).

Species known to be naturally asymptomatic carriers are listed below.

Common name [a]	Scientific name
Brown trout	*Salmo trutta*
Coho trout	*Oncorhynchus kisutch*
Rainbow trout	*Oncorhynchus mykiss*

a All species listed are naturally susceptible (other species have been shown to be experimentally susceptible).

Species shown experimentally to be asymptomatic carriers include the following.

Common name	Scientific name
Arctic char	*Salvelinus alpinus*
Atlantic cod	*Gadus morhua*
Atlantic herring	*Clupea harengus*
Coalfish or pollock	*Pollachius virens*
Salmon louse	*Lepeophtheirus salmonis and Caligus elongatus*

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- ISA occurs mainly in the Northern Hemisphere in spring and early winter at water temperatures from 3°C to above 15°C.
- The disease has caused major epizootics and severely impacted Atlantic salmon aquaculture production; most recently in Chile.
- Mortality rates vary from 15% to 100%; mortality may occur over a prolonged period, not necessarily as acute outbreaks.

- ISA is mainly transmitted horizontally through the water column but also by vectors (sea lice and populations of asymptomatic wild fish carriers).
- Experimental infection models demonstrated mortalities within 15 days of exposure to ISA.
- Spread of the disease has occurred with the movement of live juvenile salmonids from one fish farm to another, with the discharge of organic waste from fish processing plants into the marine environment and via water movement.
- The majority of natural outbreaks seem to occur in salmonid post-smolts.
- ISA has been the subject of extensive eradication campaigns in several countries, including Scotland. These can be successful and require vigilance to maintain 'free' status.
- It appears that stressors such as husbandry practices (e.g. treatment against salmon lice or infectious diseases), rising or falling temperatures, and poor water quality can predispose salmon to outbreaks of ISA.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Enteric red mouth disease, infectious haematopoietic necrosis, infectious pancreatic necrosis

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

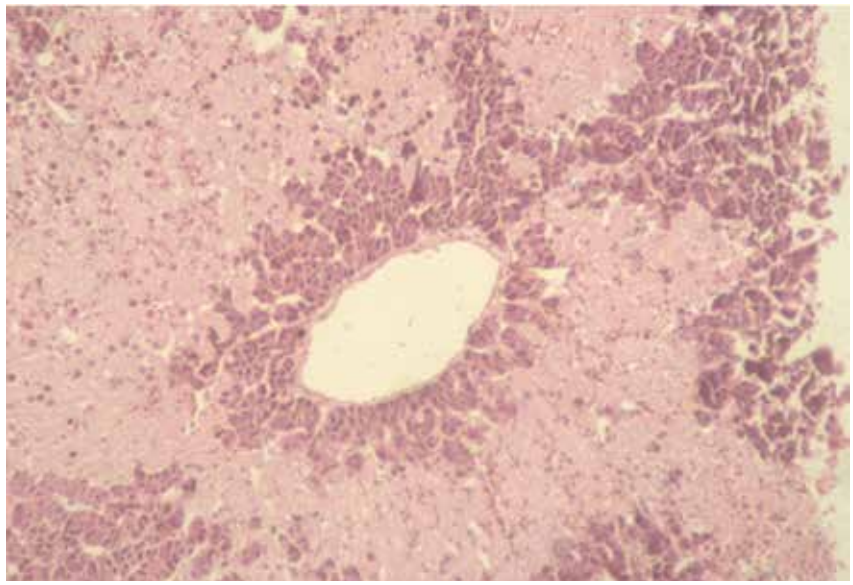
Further reading

The accepted procedures for a conclusive diagnosis of ISA are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

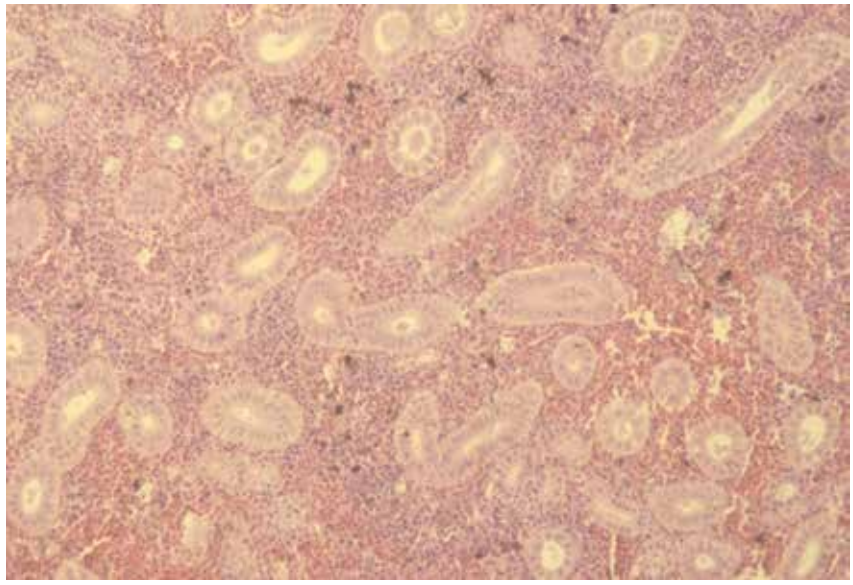
Further images

Liver from Atlantic salmon (*Salmo salar*) with ISA, showing multifocal bridging necrosis, leaving tissue around smaller veins viable



Source: T Poppe

Kidney from Atlantic salmon (*Salmo salar*) with ISA, showing renal interstitial haemorrhage



Source: T Poppe

Koi herpesvirus disease (KHV)

(Also known as carp interstitial nephritis and gill necrosis virus)

Exotic disease

Koi carp (*Cyprinus carpio*) with gross lesions associated with KHV. Operculum is removed showing mottled red (haemorrhage) and white (necrosis) gill, sunken eyes and a single ulcer on the ventral skin.

Source: E Johnson

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- mass mortalities, with many dead and moribund fish floating at the surface
- disorientation and erratic swimming behaviour (sometimes hyperactivity)
- fish coming to the surface and gasping
- fish separated from the shoal.

Gross pathological signs are:

- pale patches on the skin
- overproduction or underproduction of mucous on the skin and gills
- superficial branchial (gill) and skin haemorrhages
- enophthalmia (sunken eyes), erosion of the fins (occasionally), and blistering of the skin
- severe gill necrosis and/or erosion, seen as red and white patches
- focal or generalised loss of skin
- adhesions in the abdominal cavity and abnormal colouration of internal organs (lighter/darker or mottled)
- enlargement and surface haemorrhages of the kidney and liver.

Microscopic pathological signs are:

- inflammation and necrosis of gill tissues, and adhesion of gill filaments
- nuclear swelling, margination of chromatin and pale eosinophilic intranuclear inclusions of the epithelium
- non-specific inflammation and necrosis in other organs.

Disease agent

The causative agent of this disease is koi herpesvirus (KHV), also known as cyprinid herpesvirus 3 (CyHv3).

Host range

Species susceptible to KHV disease are listed below.

Common name [a]	Scientific name
Common carp and koi carp [a]	*Cyprinus carpio*

a Naturally susceptible (other species have been shown to be experimentally susceptible).

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- An outbreak in Japan during the spring of 2004 occurred in wild carp populations in water temperatures of 15–16°C. Most of the dead fish were adult. In the field, it appears that adult carp are more susceptible than juveniles.
- The virus may survive at low temperatures (5°C), but the temperature range for disease outbreaks is primarily between 16°C and 25°C. Mortalities commonly appear between 22°C and 25°C, with few at temperatures above 30°C.
- The disease affects all age classes of common and koi carp; the disease has occurred in fingerlings, juveniles and adults.
- Moving infected fish from cool (13°C) to warm (23°C) water results in rapid onset of mortality.
- Mortality rates can vary between 70% and 100%.
- Reservoirs of KHV are clinically infected fish and covert carriers. Virus is shed via faeces, urine, gills and skin mucus.
- Transmission of KHV is horizontal, mainly via water, but possibly via animal vectors and fomites.
- Vertical transmission cannot be excluded as a possible transmission route.
- Secondary gill infections (e.g. *Flavobacterium columnare* and *Aeromonas* spp.) are often associated with KHV infection.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Epizootic ulcerative syndrome, spring viraemia of carp

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of KHV are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

Exotic disease

Red sea bream iridoviral disease

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- low to high mortality
- lethargic swimming
- obvious opercular movement (indicating an increase in respiratory effort).

Gross pathological signs are:

- dark skin (a change in skin colour is a significant gross sign)
- petechial (pinpoint) haemorrhage of the gills
- pale gills and enlarged spleen.

Microscopic pathological signs are:

- enlarged cells, deeply Giemsa positive, in the spleen, heart, kidney, liver and gills of infected fish, which are characteristic of this disease
- small dark spots within fresh wet mounts of gill lamellae (melano–macrophage centres).

Disease agent

RSIVD is caused by a virus in the genus *Megalocytivirus*, family *Iridoviridae*.

Host range

Species known to be susceptible to RSIVD virus are listed below.

Common name [a]	Scientific name
Barramundi	*Lates calcarifer*
Black rockfish	*Sebastes schlegeli*
Black sea bream or black porgy	*Acanthopagrus schlegeli*
Brown marbled grouper	*Epinephelus fuscoguttatus*
Chicken grunt	*Parapristipoma trilineatum*
Chinese emperor	*Lethrinus haematopterus*
Chub mackerel	*Scomber japonicus*
Cobia	*Rachycentron canadum*
Crescent sweetlips	*Plectorhinchus cinctus*
Crimson sea bream	*Evynnis japonica*
Croceine croaker	*Pseudosciaena crocea*
Estuarine rockcod	*Epinephelus tauvina*
Giant grouper	*Epinephelus lanceolatus*
Girella or rudderfish	*Girella punctata*
Greater amberjack	*Seriola dumerili*
Japanese flounder	*Paralichthys olivaceus*
Japanese horse mackerel	*Trachurus japonicus*
Japanese parrotfish	*Oplegnathus fasciatus*
Japanese seabass	*Lateolabrax japonicus*
Japanese Spanish mackerel	*Scomberomorus niphonius*
Japanese yellowtail	*Seriola quinqueradiata*
Largemouth bass	*Micropterus salmoides*
Longtooth grouper	*Epinephelus bruneus*
Malabar grouper	*Epinephelus malabaricus*
Northern bluefin tuna	*Thunnus thynnus*
Orange-spotted grouper	*Epinephelus coioides*
Red sea bream	*Pagrus major*
Red-spotted grouper or Hong Kong grouper	*Epinephelus akaara*
Samson fish	*Seriola hippos*
Seabass	*Lateolabrax* spp.
Seven-band grouper	*Epinephelus septemfasciatus*
Silver trevally	*Pseudocaranx dentex*
Six-bar grouper	*Epinephelus sexfasciatus*
Snapper	*Pagrus auratus*
Snubnose dart	*Trachinotus blochii*
Spangled emperor	*Lethrinus nebulosus*
Spotted halibut	*Verasper variegatus*
Spotted knifejaw	*Oplegnathus punctatus*
Tiger puffer	*Takifugu rubripes*
Yellow grouper	*Epinephelus awoara*
Yellowfin seabream	*Acanthopagrus latus*
Yellowtail kingfish	*Seriola lalandi*

a All species are naturally susceptible (other species have been shown to be experimentally susceptible).

Presence in Australia

EXOTIC DISEASE—not present in Australia

Epidemiology

- RSIVD is highly contagious.
- Juveniles are more susceptible to disease than adults.
- Mortality is highly variable (0–100%).
- Transmission is horizontal (via the water column from other infected fish). Vertical transmission has yet to be investigated.
- Outbreaks occur at water temperatures greater than 24-25 °C.
- The virus is stable within tissue to –80 °C, and can be inactivated by ether, chloroform and formalin.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Epizootic haematopoietic necrosis, grouper iridoviral disease, infection with infectious spleen and kidney necrosis virus (ISKNV)-like viruses

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

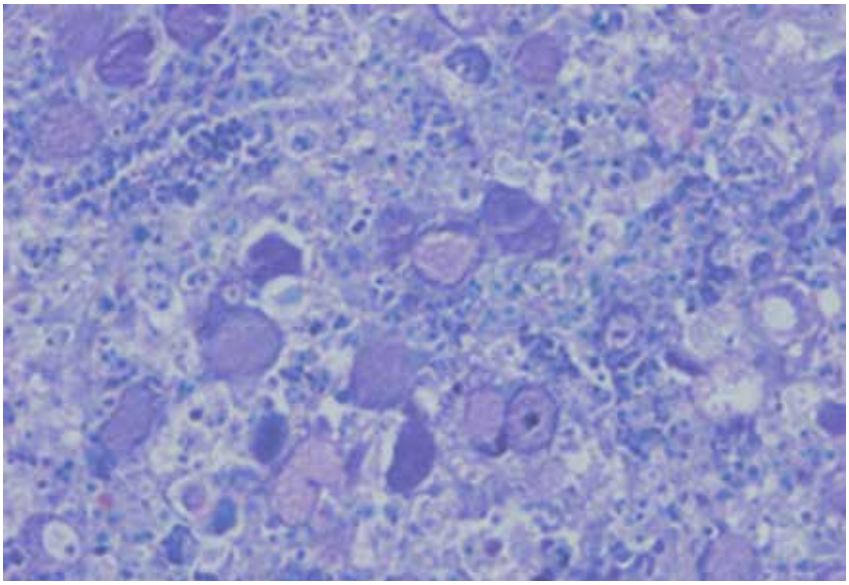
Further reading

The accepted procedures for a conclusive diagnosis of RSIVD are summarised in the World Organisation for Animal Health Manual of diagnostic tests for aquatic animals 2011, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

Further image

Giemsa positive staining enlarged cells present within tissue section of fish infected with red sea bream iridovirus



Source: K Nakajima

Exotic disease

Spring viraemia of carp (SVC)

SVC in common carp (*Cyprinus carpio*); note characteristic haemorrhagic skin, swollen stomach and exophthalmos ('popeye')

Source: HJ Schlotfeldt

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- mortality rates of 30–100%
- lethargy
- separation from shoal
- lethargic swimming
- accumulation of fish at the water inlet and sides of the pond.

Gross pathological signs are:

- exophthalmos (popeye)
- swollen abdomen and a protruding vent
- possibly a trailing white or yellowish faecal cast
- petechial (pinpoint) haemorrhages of the skin, gills and eyes
- haemorrhages on skin and base of fins and around the vent
- darker body colour, with pale gills
- diffuse swelling and haemorrhage of internal organs and degeneration of gill lamellae
- ascites (fluid in abdominal cavity)
- intestines containing mucous instead of food.

Microscopic pathological signs are:

- liver hyperaemia and oedematous perivasculitis
- pericarditis and infiltration of the myocardium
- hyaline degeneration and vacuolation of the renal tubules, which are clogged with casts
- inflammatory and hyperaemic changes in all major organs.

Disease agent

SVC virus is a rhabdovirus that is closely related to infectious haematopoietic necrosis virus and viral haemorrhagic septicaemia virus.

Host range

Species known to be susceptible to SVC are listed below.

Common name	Scientific name
Bighead carp **a**	*Aristichthys nobilis*
Common carp and koi carp **a**	*Cyprinus carpio* (species most susceptible)
Common roach	*Rutilus rutilus*
Crucian carp **a**	*Carassius carassius*
Goldfish **a**	*Carassius auratus*
Grass carp **a**	*Ctenopharyngodon idellus*
Guppy	*Poecilia reticulata* (also known as *Lebistes reticulatus)*
Ide or orfe **a**	*Leuciscus idus*
Pike **a**	*Esox lucius*
Pumpkinseed	*Lepomis gibbosus*
Rainbow trout **a**	*Oncorhynchus mykiss*
Silver carp **a**	*Hypophthalmichthys molitrix*
Tench **a**	*Tinca tinca*
Wels catfish or sheatfish **a**	*Silurus glanis*
Zebrafish	*Danio rerio*

a Naturally susceptible (other species have been shown to be experimentally susceptible).

Non-fish carriers include the species listed below.

Common name	Scientific name
Fish louse	*Argulus foliaceus*
Grey heron	*Ardea cinerea*
Leeches	*Piscicola* spp.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- SVC is very contagious among common carp.
- Clinical disease is linked closely to environmental disturbances.
- Mortality rate is usually less than 40% but can range from 5% to 100%, with younger fish (<1 year old) being more susceptible.

- Fry are susceptible to disease at temperatures up to 23 °C.
- Disease may also occur in older fish (>1 year), usually when water temperatures are between 11 °C and 17 °C (associated with the stress of an abnormally cold spring in Europe, and possibly due to cold temperatures weakening the fish's immune system during the spring).
- Fish that survive SVC are presumed to carry the virus.
- Outbreaks are most likely to occur with increased stress levels, such as around the time of spawning, and coincide with increased levels of virus excreted with spawning fluids.
- Transmission of the virus to uninfected fish is horizontal, and the virus enters fish through the gills and skin.
- The virus enters the water in faeces, urine and spawning fluids. Transport of live infected fish, contaminated water and contaminated eggs of infected fish (suggestive of vertical transmission) contributes to disease spread.
- Blood-sucking parasites such as anchor worm and leeches can transmit the virus from fish to fish.
- Stressors (e.g. overcrowding) can trigger an outbreak in apparently healthy populations.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Enteric red mouth disease, enteric septicaemia of catfish, infection with *Aeromonas salmonicida*—atypical strains, koi herpesvirus disease

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

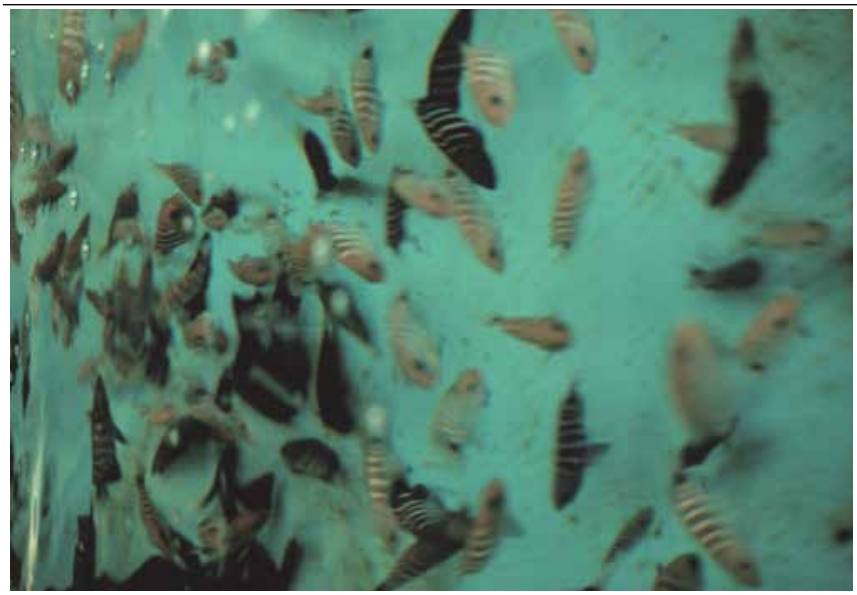
Further reading

The accepted procedures for a conclusive diagnosis of SVC are summarised in the World Organisation for Animal Health Manual of diagnostic tests for aquatic animals 2011, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication

Viral encephalopathy and retinopathy
(Also known as viral nervous necrosis)

Viral encephalopathy and retinopathy in seven-band grouper (*Epinephelus septemfasciatus*); dark fish are affected, light fish are normal. A change in colouration is an important indicator of disease. Species differ in how they are affected (e.g. barramundi show lighter colouration when affected).



Source: B Munday

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- 50–100% cumulative mortality over a period of 48 hours to several weeks
- higher mortalities in larvae and juvenile fish (9–28 days old) although older fish (>28 days) may also be affected
- anorexia
- abnormal swimming behaviours, including erratic, uncoordinated darting, spiral and/or looping swim pattern; corkscrew swimming
- fish resting belly-up (loss of equilibrium)
- hyperactivity
- sporadic protrusion of the head from the water.

Gross pathological signs are:

- colour change—larval barramundi become lighter, but groupers become darker
- blindness
- abrasions
- emaciation
- overinflated swim bladder (the only significant internal gross pathological sign).

Microscopic pathological signs are:

- vacuolation of central nervous tissues, including retina
- intracytoplasmic inclusions in brain tissues as crystalline arrays or aggregates.

Disease agent

Viral encephalopathy and retinopathy (VER) or Viral Nervous Necrosis (VNN) is a disease caused by a *Betanodavirus*, which is in the family *Nodaviridae*. VER was formerly referred to as barramundi picorna-like virus in Australia.

Host range

Species known to be susceptible to VER virus are listed below.

Common name	Scientific name
Atlantic halibut **a**	*Hippoglossus hippoglossus*
Atlantic salmon	*Salmo salar*
Australian bass **a**	*Macquaria novemaculeata*
Barcoo grunter	*Scortum barcoo*
Barfin flounder **a**	*Verasper moseri*
Barramundi **a**	*Lates calcarifer*
Brown-marbled grouper **a**	*Epinephelus fuscoguttatus*
Chinese catfish **a**	*Silurus asotus*
Cobia **a**	*Rachycentron canadum*
Convict surgeonfish	*Acanthurus triostegus*
Common sole **a**	*Solea solea*
Dusky grouper	*Epinephelus marginatus*
Estuarine rockcod **a**	*Epinephelus tauvina*
Estuary catfish	*Cnidoglanis macrocephalus*
European eel **a**	*Anguilla anguilla*
European seabass **a**	*Dicentrarchus labrax*
Flounders **a**	*Paralichthyidae*
Gilt-head sea bream **a**	*Sparus aurata*
Golden grey mullet **a**	*Liza auratus*
Golden perch	*Macquaria ambigua*
Greater amberjack **a**	*Seriola dumerili*
Grouper and estuary cod **a**	*Epinephelus* spp.
Haddock **a**	*Melanogrammus aeglefinus*
Humpback grouper **a**	*Cromileptes altivelis*
Japanese flounder **a**	*Paralichthys olivaceus*
Japanese tilefish	*Branchiostegus japonicas*
Japanese parrotfish **a**	*Oplegnathus fasciatus*
Japanese seabass **a**	*Lateolabrax japonicus*

Common name	Scientific name
Longtooth grouper **a**	*Epinephelus bruneus*
Macquarie perch	*Macquaria australasica*
Malabar grouper **a**	*Epinephelus malabaricus*
Mangrove Jack	*Lutjanus argentimaculatus*
Milkfish	*Chanos chanos*
Mullet **a**	*Mugil cephalus*
Murray cod	*Maccullochella peelii peelii*
Narrowstripe cardinalfish	*Apogon exostigma*
Orange-spotted grouper **a**	*Epinephelus coioides*
Red drum **a**	*Sciaenops ocellatus*
Red mullet	*Mullus barbatus*
Red snapper	*Lutjanus erythropterus*
Red-spotted grouper **a** or Hong Kong grouper	*Epinephelus akaara*
Russian sturgeon **a**	*Acipenser gueldenstaedtii*
Samson fish	*Seriola hippos*
Senegalese sole **a**	*Solea senegalensis*
Seven-band grouper **a**	*Epinephelus septemfasciatus*
Shi drum **a**	*Umbrina cirrosa*
Sleepy cod	*Oxyeleotris lineolatus*
Silver perch	*Bidyanus bidyanus*
Silver trevally **a**	*Pseudocaranx dentex*
Snubnose dart **a**	*Trachinotus blochii*
Spotted coralgrouper	*Plectropomus maculatus*
Spotted knifejaw **a**	*Oplegnathus punctatus*
Spotted wolffish	*Anarhichas minor*
Striped trumpeter **a**	*Latris lineata*
Thread-sail filefish **a**	*Stephanolepis cirrhifer*
Tiger puffer **a**	*Takifugu rubripes*
Tilapi **a**	*Oreochromis niloticus*
Turbot **a**	*Psetta maxima (also known as Scophthalmus maximus)*
White grouper **a**	*Epinephelus aeneus*
White seabass **a**	*Atractoscion nobilis*
Winter flounder **a**	*Pseudopleuronectes americanus*
Yellow-wax pompano **a**	*Trachinotus falcatus*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

VER has been officially reported from New South Wales, the Northern Territory, Queensland, South Australia, Tasmania and Western Australia. It is primarily reported to affect larvae or fry.

Epidemiology

- VER is a disease which has been found in at least 40 species of marine fish from 16 families, and has been diagnosed in all inhabited continents.
- Most fish are affected as larvae or juveniles; however, in recent years, mortalities have occurred in older fish up to harvest size, particularly in European seabass, groupers (*Epinephelus* spp.) and Atlantic halibut. Disease incidence in the groupers and seabass has been associated with high water temperatures.
- Susceptibility and mortality are age dependent (onset of clinical disease in younger fish of some species results in higher mortality).
- Batches of barramundi larvae for aquaculture are now routinely screened for this disease in Australia.
- The incubation period for the disease in barramundi is 4 days, with typical disease onset 9–28 days after hatching, rarely occurring in older fish (50–60 days old). In silver trevally, disease onset is 1 day after hatching.
- Transmission is believed to occur horizontally, through the water column (i.e. via mouth, gills and skin), and vertically (parent to offspring). The rate of transmission may be influenced by stressors, including handling, repeated spawning, high stocking densities, high ambient temperature and virulence of the particular *Betanodavirus* strain. Sand worms of the family *Nereidae*, genus *Nereis*, collected in proximity to an infected farm have had positive detection of *Betanodavirus*.
- Virus can survive for 1 year in the right environmental conditions (pH 2–9 and 15°C) and can persist subclinically in infected live fish. Therefore, fish products and byproducts may facilitate the spread of virus to unaffected areas.
- Cumulative mortality at 1 month is typically 50–100% in barramundi and 100% in silver trevally. In Australian hatcheries, 100% mortality in <3 days in larvae is typical.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Grouper iridoviral disease

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found at;

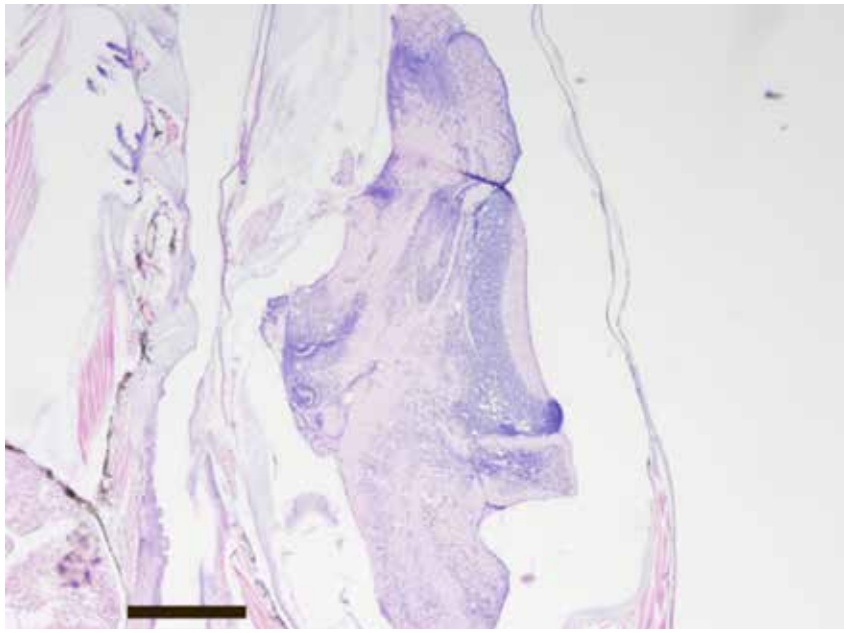
Sub-Committee on Animal Health Laboratory Standards (SCAHLS) website at www.scahls.org.au/procedures/anzsdps2

Centre for Environment, Fisheries and Aquaculture Science (Cefas) International Database on Aquatic Animal Disease (IDAAD) website at www.cefas.defra.gov.uk/idaad/disocclist.aspx.

These hyperlinks were correct and functioning at the time of publication.

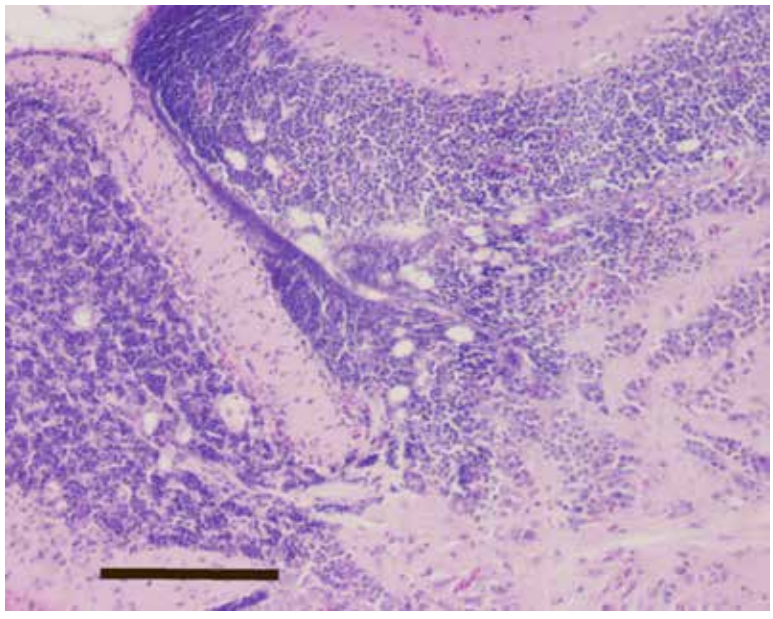
Further images

Barramundi (*Lates calcarifer*) 20 day old juvenile. Transverse section through larval head. Vacuoles associated with VER infection are readily visible in the brain even at 20x magnification. Haematoxylin and Eosin stain. Scale bar = 0.3 mm.



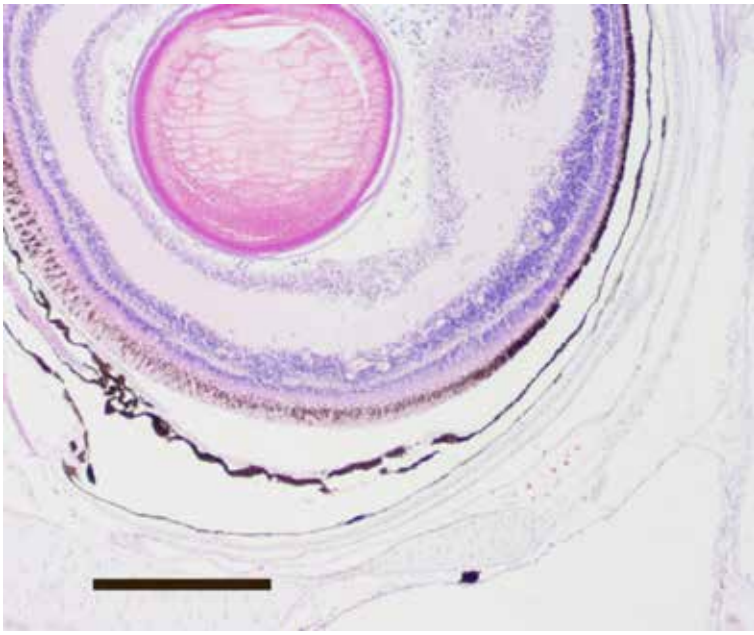
Source: B Jones

Barramundi (*Lates calcarifer*) 20 day old juvenile. Transverse section through brain. Juncture of optic lobe and mesencephalon. Vacuoles associated with VER infection are readily visible. Haematoxylin and Eosin stain. Scale bar = 0.1 mm.



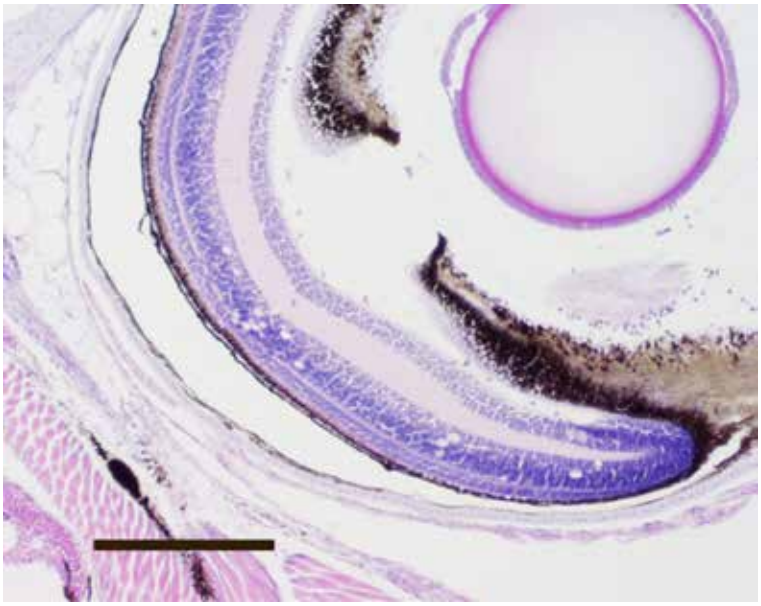
Source: B Jones

Barramundi (*Lates calcarifer*) 22 day old juvenile. Transverse section through retina. Vacuoles associated with VER infection are visible. Haematoxylin and Eosin stain. Scale bar = 0.2 mm.



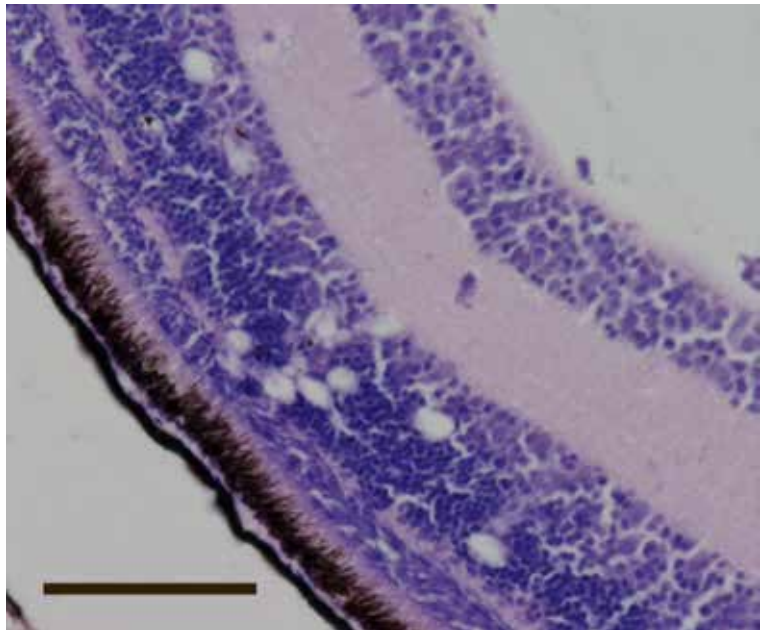
Source: B Jones

Barramundi (*Lates calcarifer*) 22 day old juvenile. Transverse section through retina. Vacuoles associated with VER infection are visible. Haematoxylin and Eosin stain. Scale bar = 0.2 mm.



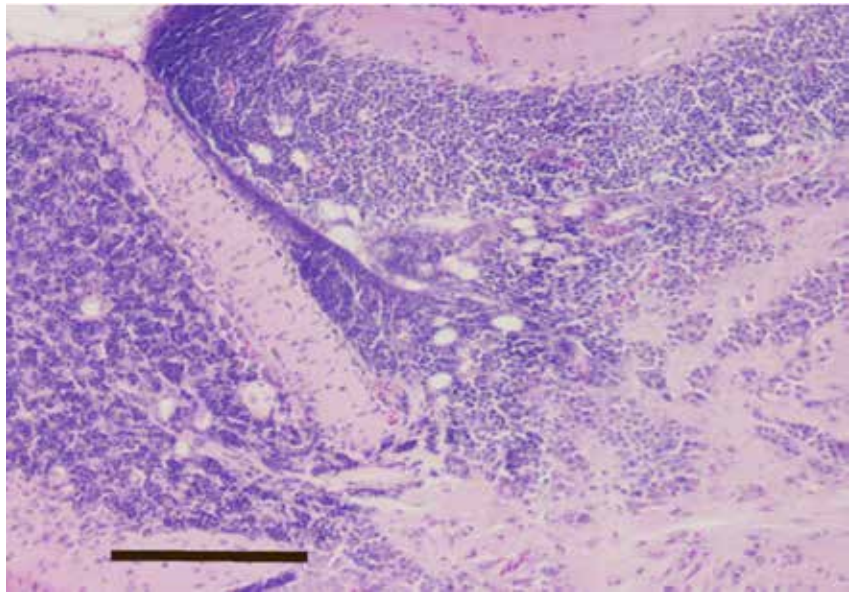
Source: B Jones

Barramundi (*Lates calcarifer*) 20 day old juvenile. Transverse section through retina. Vacuoles associated with VER infection are visible. Haematoxylin and Eosin stain. Scale bar = 0.05 mm.



Source: B Jones

Barramundi (*Lates calcarifer*) 20 day old juvenile. Transverse section through brain. Juncture of optic lobe and mesencephalon. Vacuoles associated with VER infection are readily visible. Haematoxylin and Eosin stain. Scale bar = 0.1 mm.

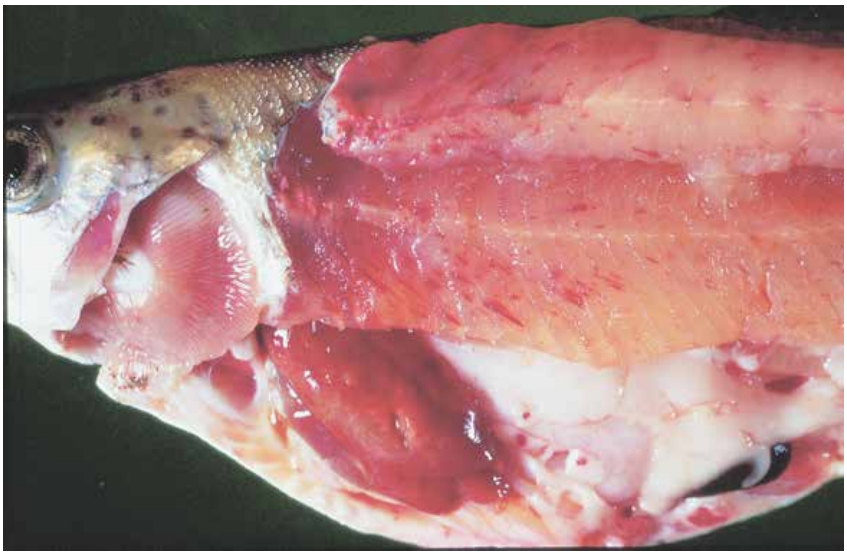


Source: B Jones

Viral haemorrhagic septicaemia (VHS)

Exotic disease

VHS in rainbow trout (*Oncorhynchus mykiss*); note pale colour of stomach region, pinpoint haemorrhages in fatty tissue and pale gills



Source: T Håstein

VHS in rainbow trout (*Oncorhynchus mykiss*); note swollen stomach and 'popeye'

Source: T Håstein

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease outbreaks are seen in farmed trout and other salmonids, as well as in farmed turbot and Japanese flounder.

Disease signs of *acute* infection at the farm, tank or pond level are:

- rapid onset of high mortality
- lethargic swimming
- separation from shoal
- loss of appetite
- crowding at pond edges.

Disease signs of *chronic* infection at the farm, tank or pond level are:

- significant cumulative mortality (protracted)
- uncoordinated swimming (ataxia) with rotating movement around body axis (i.e. spinning).

Disease signs of the *neurological* form of the disease at the farm, tank or pond level are:

- low mortality
- severe abnormal swimming behaviour (flashing and spiralling).

General gross pathological signs are:

- exophthalmos (popeye)
- haemorrhaging under the skin, around the base of pectoral and pelvic fins and in the eyes
- swollen abdomen
- pale gills, with or without petechial (pinpoint) haemorrhages.

Gross pathological signs of *acute* infection are:

- slight darkening of the body colour
- exophthalmos (popeye)
- bleeding around the eyes
- bleeding under the skin around the base of the pectoral and pelvic fins
- skin ulceration
- swollen abdomen
- pale gills with pinpoint haemorrhages
- ascites (fluid in the abdominal cavity)
- petechial (pinpoint) haemorrhages in the fatty tissue, intestine, gonads, liver, swim bladder and muscle
- dark-red kidneys.

Gross pathological signs of *chronic* infection are:

- often an absence of external signs
- intense darkening of the skin
- exophthalmos (popeye)
- pale gills (anaemic)

- pale abdominal organs
- pale and mottled liver (evidence of haemorrhages on surface)
- pale gastrointestinal tract that is empty of food.

Microscopic pathological signs are:

- accumulation of erythrocytes in skeletal muscle fibres
- extensive focal necrosis in the liver, kidney and spleen
- VHS virus-positive endothelial cells in vascular system evident from immunohistochemistry.

Disease agent

VHS virus is a rhabdovirus of the genus *Novirhabdovirus*. Several genogroups or genotypes of the virus have been identified from different environments in different parts of the world:

- type I, continental Europe—freshwater group, trout farms (highly pathogenic to rainbow trout)
- type II, European marine strain (Baltic sea)—marine strain affecting wild and cultured marine and freshwater species (has low pathogenicity in rainbow trout)
- type III, north Atlantic marine group (North sea near the British Isles)
- type IVa, west coast of north America and east Asian group—marine group affecting a range of free-living marine and cultured species (highly pathogenic in Pacific herring; however, rainbow trout appear refractory to infection with this genotype)
- type IVb, Great lakes region—significant mortalities in wild freshwater species in the Great lakes.

Host range

VHS virus has been isolated from a broad range of marine and freshwater fish in Europe and the north Pacific (including cod, sprats, herring, haddock and turbot).

Species known to be susceptible to VHS are listed below.

Common name	Scientific name
Armoured weaselfish	*Hoplobrotula armata*
Atlantic cod	*Gadus morhua*
Atlantic halibut	*Hippoglossus hippoglossus*
Atlantic herring	*Clupea harengus*
Atlantic salmon	*Salmo salar*
Black crappie	*Pomoxis nigromaculatus*
Black sea bream or black porgy	*Acanthopagrus schlegeli*
Bluegill	*Lepomis macrochirus*
Blue whiting	*Micromesistius poutassou*
Bluntnose minnow	*Pimephales notatus*
Brook trout	*Salvelinus fontinalis*
Brown bullhead	*Ictalurus nebulosus*
Brown trout	*Salmo trutta*
Burbot **a**	*Lota lota*
Channel catfish	*Ictalurus punctatus*
Chinook salmon	*Oncorhynchus tshawytscha*

Common name	Scientific name
Chub mackerel	*Scomber japonicus*
Coho salmon	*Oncorhynchus kisutch*
Dab	*Limanda limanda*
Emerald shiner	*Notropis atherinoides*
English sole	*Parophrys vetulus*
Eulachon **a**	*Thaleichthys pacificus*
European eel	*Anguilla anguilla*
European seabass	*Dicentrarchus labrax*
European sprat	*Sprattus sprattus*
Flounder	*Platichthys flesus*
Fourbeard rockling	*Enchelyopus cimbrius*
Freshwater drum **a**	*Aplodinotus grunniens*
Gilt-head sea bream	*Sparus aurata*
Gizzard shad	*Dorosoma cepedianum*
Golden trout	*Oncorhynchus aguabonita*
Grayling	*Thymallus thymallus*
Greenland halibut	*Reinhardtius hippoglossoides*
Haddock	*Melanogrammus aeglefinus*
Hairtail	*Trichiurus lepturus*
Hybrid (rainbow trout × coho salmon)	*Oncorhynchus mykiss* × *O. kisutch*
Iberian nase	*Pseudochondrostoma polylepis*
Japanese flounder **a**	*Paralichthys olivaceus*
Japanese yellowtail	*Seriola quinqueradiata*
Korean flounder	*Glyptocephalus stelleri*
Lake trout	*Salvelinus namaycush*
Lake whitefish	*Coregonus clupeaformis*
Largemouth bass	*Micropterus salmoides*
Lesser argentine	*Argentina sphyraena*
Mullet	*Mugil cephalus*
Mummichog **a**	*Fundulus heteroclitus*
Muskellunge **a**	*Esox masquinongy*
Norway pout	*Trisopterus esmarki*
Pacific cod	*Gadus macrocephalus*
Pacific hake **a**	*Merluccius productus*
Pacific herring **a**	*Clupea pallasii*
Pacific salmon	*Oncorhynchus* spp.
Pacific sand eel	*Ammodytes personatus*
Pacific sand lance	*Ammodytes hexapterus*
Pacific sardine **a**	*Sardinops sagax*
Pacific tomcod	*Microgadus proximus*
Pike **a**	*Esox lucius*
Plaice	*Pleuronectes platessa*
Poor cod	*Trisopterus minutus*
Pumpkinseed	*Lepomis gibbosus*
Rainbow trout **a**	*Oncorhynchus mykiss*
Red-spotted grouper or Hong Kong grouper	*Epinephelus akaara*
River lamprey	*Lampetra fluviatalis*

Common name	Scientific name
Rock bass	*Ambloplites rupestris*
Rockfish	*Sebastes* spp.
Round goby **a**	*Neogobius melanostomus*
Sablefish **a**	*Anoplopoma fimbria*
Sand eel	*Ammodytes* spp.
Sand goby	*Pomatoschistus minutus*
Senegalese sole	*Solea senegalensis*
Shiner perch	*Cymatogaster aggregata*
Shorthead redhorse	*Moxostoma macrolepidotum*
Silver pomfret	*Pampus argenteus*
Silver redhorse	*Moxostoma anisurum*
Smallmouth bass **a**	*Micropterus dolomieui*
Snapper	*Pagrus auratus*
Splake (lake trout × brook trout)	*Salvelinus namaycush* × *S. fontinalis*
Spottail shiner	*Notropis hudsonius*
Striped bass	*Morone saxatilis*
Surf smelt **a**	*Hypomesus pretiosus*
Three-spined stickleback	*Gasterosteus aculeatus*
Trout-perch	*Percopsis omiscomaycus*
Tubesnout	*Aulorhynchus flavidus*
Turbot **a**	*Psetta maxima (also known as Scophthalmus maximus)*
Walleye pollock or Alaska pollock **a**	*Theragra chalcogramma*
White bass	*Morone chrysops*
Whitefish	*Coregonus* spp.
White perch	*Morone americanus*
Whiting	*Merlangius merlangus*
Yellowback seabream	*Evynnis tumifrons*
Yellow perch **a**	*Perca flavescens*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Variant strains of the virus are responsible for disease in different geographical locations.
- Marine and freshwater species are susceptible to VHS virus infection. Younger fish are generally more susceptible to disease.
- Rainbow trout appear to be less susceptible to infection by marine strains of the virus.
- Water temperatures in an outbreak are generally near 10°C. At water temperatures between 15°C and 18°C, the disease generally takes a shorter course with a modest accumulated mortality, but transmission can occur at water temperatures up to 22°C. Mortality and morbidity have rarely been documented when water temperatures are above 18°C, although VHS virus genotype IV has caused at least one fish kill at 20–22°C, and some isolates can replicate in vitro at temperatures up to 25°C.

- Transmission is horizontal directly through the water, from virus shed in faeces, urine (predominantly) and sexual fluids of clinically infected or carrier fish. The virus can also be spread by birds that have consumed infected fish, via blood-feeding vectors such as leeches, and on equipment that has been in contact with water from infected fish. The virus gains entry via the gills, skin wounds, oral exposure (predation) and possibly through the skin.
- Once infected, survivors are lifelong carriers of the virus; however, shedding is intermittent.
- Stressors including overcrowding, extreme temperatures and overfeeding will greatly reduce an animal's resistance to infection.
- Mortality rate can range from 10% to 80%, depending on the VHS virus isolate, environmental variables (temperature), age, species, route of exposure and presence of additional stressors (highest mortality rates occur with acute infection, and lowest mortality rates in the neurological form).
- VHS virus is thought to have existed in the marine environment before its apparent transfer to fresh water, where it first became virulent in trout.
- It has been suggested that the European freshwater strains of VHS virus originated from fish in the northern Pacific and Atlantic oceans. The mechanism of transfer was possibly through the feeding of marine fish to cultured freshwater species.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Enteric red mouth disease, epizootic haematopoietic necrosis, epizootic ulcerative syndrome, infectious haematopoietic necrosis, infectious pancreatic necrosis, whirling disease

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of VHS are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

For more information on VHS virus isolates, refer to the European Community Reference Laboratory for Fish Disease at www.fishpathogens.eu/vhsv.

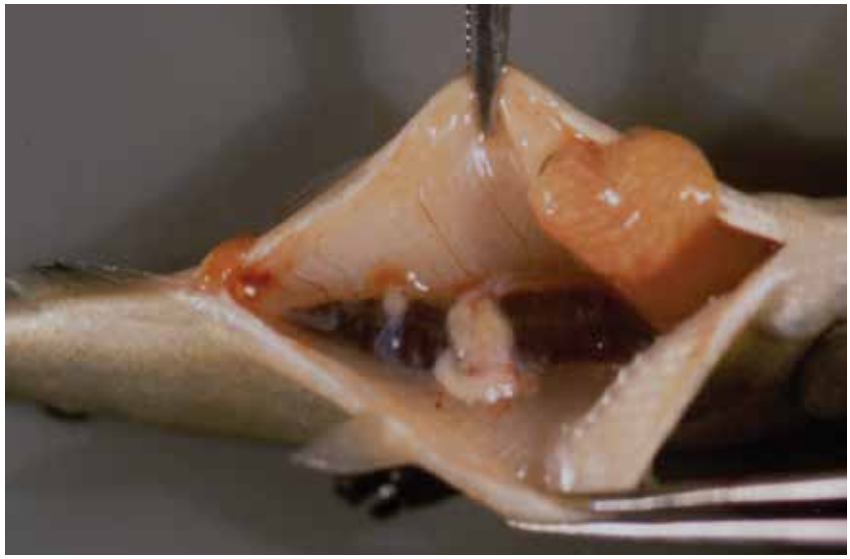
These hyperlinks were correct and functioning at the time of publication

Exotic disease

Bacterial diseases of finfish

Bacterial kidney disease

Kidney lesions in a juvenile chinook salmon (*Oncorhynchus tshawytscha*) affected by bacterial kidney disease



Source: R Pascho and C O'Farrell

Ventral view (bottom) of an adult chinook salmon (*Oncorhynchus tshawytscha*) with dermatitis ('spawning rash') caused by *Renibacterium salmoninarum*

Source: R Pascho and D Elliott

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- lethargy
- increasing mortality.

Gross pathological signs are:

- exophthalmos (popeye)
- swollen abdomen and skin blisters or shallow ulcers (remnants of ruptured blisters)
- darkening of skin and pale gills
- haemorrhages at the base of the fins or at the vent
- creamy-white, granulomatous, nodular lesions in the kidney and sometimes in the liver and spleen, which may be encapsulated
- ascites (fluid in the abdominal cavity)
- haemorrhages on the abdominal wall and in the viscera
- diffuse, white membranous layer on one or more internal organs
- enlarged spleen
- cystic cavities in skeletal muscle.

Microscopic pathological signs are:

- focal or diffuse granulomatous reaction in the kidneys, liver and spleen
- small, rod-shaped bacteria (*Renibacterium salmoninarum*) in histological sections of skin lesions.

Disease agent

Bacterial kidney disease is caused by the bacterium *Renibacterium salmoninarum*. It is a slowly progressive and frequently fatal infection of cultured and wild salmonids in both fresh and marine waters.

Host range

Salmonid species known to be susceptible to bacterial kidney disease are listed below.

Common name	Scientific name
Arctic char	*Salvelinus alpinus*
Atlantic salmon **a**	*Salmo salar*
Black sea salmon	*Salmo trutta labrax*
Brook trout **a**	*Salvelinus fontinalis*
Brown trout **a**	*Salmo trutta*
Chinook salmon **a**	*Oncorhynchus tshawytscha*
Chum salmon	*Oncorhynchus keta*
Coho salmon **a**	*Oncorhynchus kisutch*
Cutthroat trout **a**	*Salmo clarki*
Dunube salmon **a**	*Salmo hucho*
Masu salmon **a**	*Oncorhynchus masou*
Pink salmon **a**	*Oncorhynchus gorbuscha*
Rainbow trout **a**	*Oncorhynchus mykiss*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Susceptible non-salmonid species include the following.

Common name **a**	Scientific name
Ayua	*Plecoglossus altivelis*
Burbot	*Lota lota*
Common shiner	*Notropis cornutus*
Fathead minnow	*Pimephales promelas*
Grayling **a**	*Thymallus thymallus*
Pacific herring	*Clupea pallasii*
Sablefish	*Anoplopoma fimbria*
Sea lamprey **a**	*Petromyzon marinus*
Shiner perch	*Cymatogaster aggregata*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Non-salmonid carriers include the following species.

Common name	Scientific name
Bartail flathead	*Platycephalus indicus*
Greenling	*Hexagrammos otakii*
Japanese scallop	*Patinopecten yessoensis*

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- The causative bacterium is likely to persist only within salmonids and not in the environment. However, as *R. salmoninarum* is often endemic in wild salmon populations, hatcheries can be constantly exposed to bacteria shed into the water by wild fish upstream.
- Other non-salmonid species have been demonstrated susceptible to infection with *R. salmonarium*, but only when raised in proximity to highly infected salmonids.
- The bacterium is transmitted both horizontally (between fish via the water) and vertically (parent to offspring via eggs). Surface disinfection of eggs does not prevent vertical transmission.
- Advanced infection becomes apparent only after the first year of the fish's life.
- Coho (*Oncorhynchus kisutch*) and chinook (*O. tshawytscha*) salmon are the most important worldwide sources of infection.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to bacterial kidney disease.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

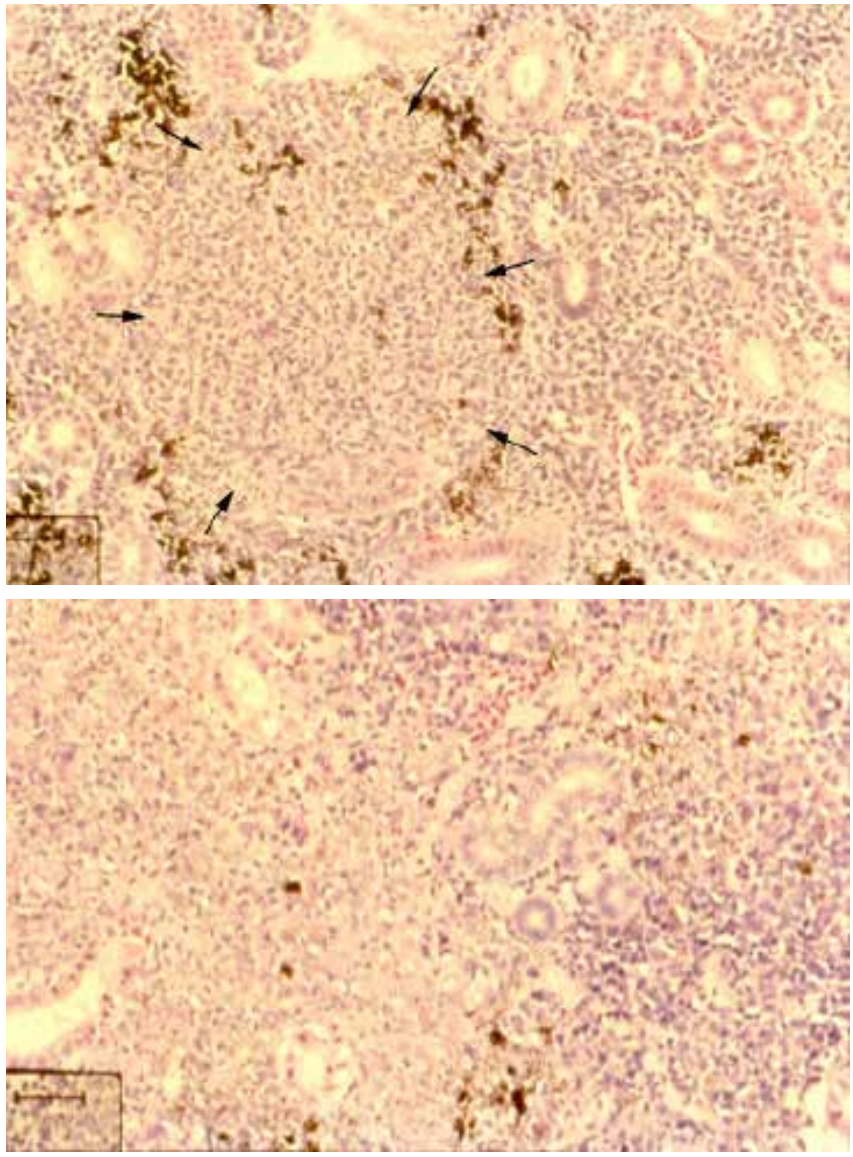
Further reading

Further information can be found on the Centre for Environment, Fisheries and Aquaculture Science (Cefas) International Database on Aquatic Animal Disease (IDAAD) website at www.cefas.defra.gov.uk/idaad/disocclist.aspx.

This hyperlink was correct and functioning at the time of publication.

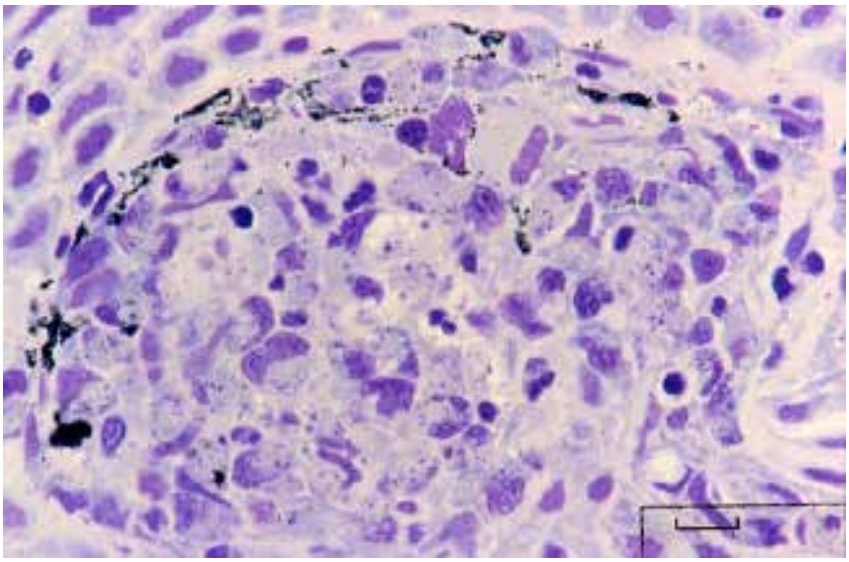
Further images

Focal (top photo, arrows) and diffuse (bottom photo) granulomas in the posterior kidney of juvenile chinook salmon (*Oncorhynchus tshawytscha*) with bacterial kidney disease. (Haematoxylin and eosin stain; scale bars = 50 µm)



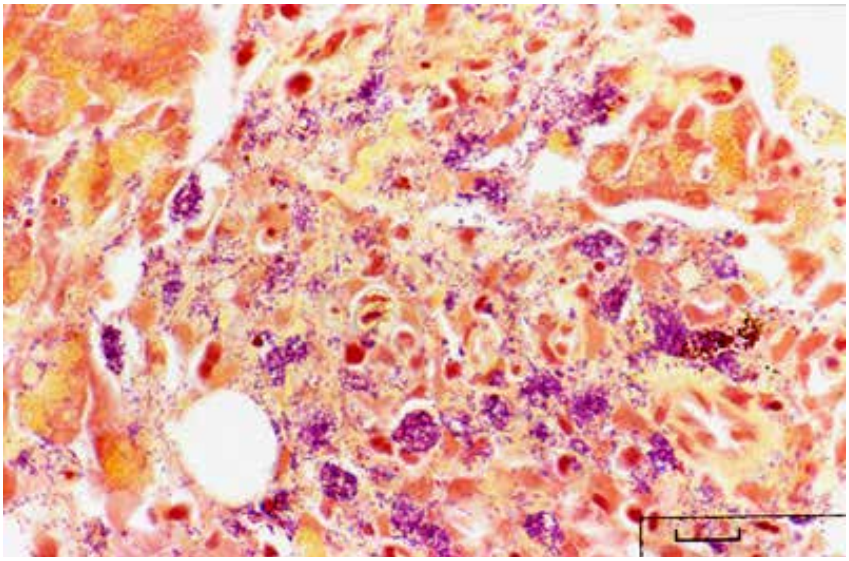
Source: R Pascho and C O'Farrell

Histological section of a skin lesion of a juvenile chinook salmon (*Oncorhynchus tshawytscha*) infected with *R. salmoninarum*. Most of the small, rod-shaped *R. salmoninarum* are visible within the cytoplasm of macrophages. Note that the bacteria are purple-blue in this Giemsa-stained preparation, in contrast to the black melanin granules.



Source: R Pascho

Gram-stained histological section of pancreatic tissue of a juvenile chinook salmon (*Oncorhynchus tshawytscha*) with systemic bacterial kidney disease. *R. salmoninarum* cells are present extracellularly and intracellularly within macrophages. Note the colour difference between the gram-positive (purple–blue) bacteria and the brown–black melanin granules.



Source: R Pascho

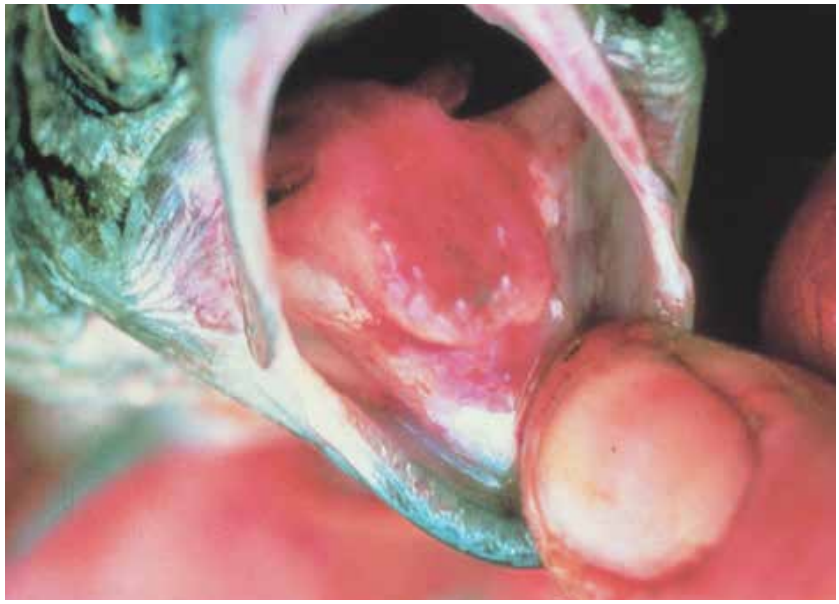
Exotic disease

Enteric red mouth disease

Enteric red mouth disease in rainbow trout; note skin and eye haemorrhages, and swollen abdomen

Source: HJ Schlotfeldt

Enteric red mouth disease in rainbow trout; note reddened mouth and tongue



Source: HJ Schlotfeldt

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- separation from other fish
- occasional nervous signs
- acute infections in fingerlings, although fish of all ages are affected usually with sustained low level mortalities observed

Gross pathological signs are:

- dark body colour (seen in atypical infections)
- haemorrhages at base of paired fins and vent
- reddening (subcutaneous haemorrhages) of the gill cover, corners of mouth, gums, palate and tongue
- exophthalmos (popeye) and orbital haemorrhages
- loss of appetite
- swollen abdomen
- ascites (fluid in the abdominal cavity)
- pinpoint haemorrhages on the liver, pancreas, pyloric caecae, swim bladder and lateral musculature surfaces
- enlarged, friable black spleen
- inflamed lower intestine containing thick yellow fluid.

Microscopic pathological signs are:

- generalised haemorrhagic septicaemic inflammatory response of all tissues
- bacterial colonisation of well-vascularised tissues (spleen, liver, heart, gills, kidney)
- necrosis of haematopoietic tissue in the kidney and spleen.

Disease agent

The causative agent of enteric red mouth disease is the bacterium *Yersinia ruckeri*. There are several serotypes of the bacterium, and classification systems can be based upon whole-cell typing as well as individual cell-wall antigen groupings. The serotype responsible for enteric red mouth disease is the Hagerman strain, serotype O1a, which is considered to be the most virulent.

Although the enteric red mouth strain (serotype O1a) is exotic to Australia, a virulent form of *Y. ruckeri* (serotype O1b) is endemic in Australia. It produces a septicaemic condition in Atlantic salmon (*Salmo salar*) known as yersiniosis. A characteristic of this form of the disease is exophthalmos (popeye) and the formation of pronounced haemorrhages in the eye that give rise to the description of 'blood spot' disease. The prominent reddening of the corners of the mouth, gums and palate typical of classical enteric red mouth disease does not normally occur in yersiniosis.

Host range

Species known to be susceptible to enteric red mouth disease are listed below.

Common name	Scientific name
Atlantic salmon	*Salmo salar*
Brown trout	*Salmo trutta*
Common carp	*Cyprinus carpio*
Goldfish	*Carassius auratus*
Rainbow trout	*Oncorhynchus mykiss*

Species not commonly found in Australia but known to be susceptible to infection are listed below.

Common name	Scientific name
Salmonid	
Arctic char	*Salvelinus alpinus*
Brook trout	*Salvelinus fontinalis*
Chinook salmon	*Oncorhynchus tschawytscha*
Coho salmon	*Oncorhynchus kisutch*
Cutthroat trout	*Salmo clarkii*
Sockeye salmon	*Oncorhynchus nerka*
Non-salmonid	
Bighead carp	*Aristichthys nobilis*
Burbot	*Lota lota*
Channel catfish	*Ictalurus punctatus*
Cisco	*Coregonus artedi*
Common sole	*Solea solea*
European eel	*Anguilla anguilla*
Emerald shiner	*Notemigonus atherinoides*
Fathead minnow	*Pimephales promelas*
Siberian sturgeon	*Acipenser baeri*
Silver carp	*Hypophthalmichthys molitrix*
Turbot	*Scophthalmus maximus*
Whitefish	*Coregonus peled and C. muksun*

It should be assumed that all species in the host range are naturally susceptible.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

(A related disease known as 'blood spot' occurs in Australia).

Epidemiology

- Many other aquatic species are potential carriers but show no signs (e.g. some crustaceans, including freshwater crayfish).
- Transmission can be horizontal, via direct contact with infected fish or carriers. Carriers are particularly important sources of infection under stressful situations (e.g. increasing temperature).
- The organism can survive in the environment, with some strains able to form biofilms.
- Vertical transmission (fish to egg) is suspected but is yet to be proven.
- This disease causes septicaemia (bacteria are spread through the body via the blood).
- Fish of all ages are affected, and outbreaks usually begin with low mortalities that slowly escalate. The severity of the outbreak depends on the strain and presence of stressors.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infectious haematopoietic necrosis, infectious salmon anaemia, spring viraemia of carp, viral haemorrhagic septicaemia

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

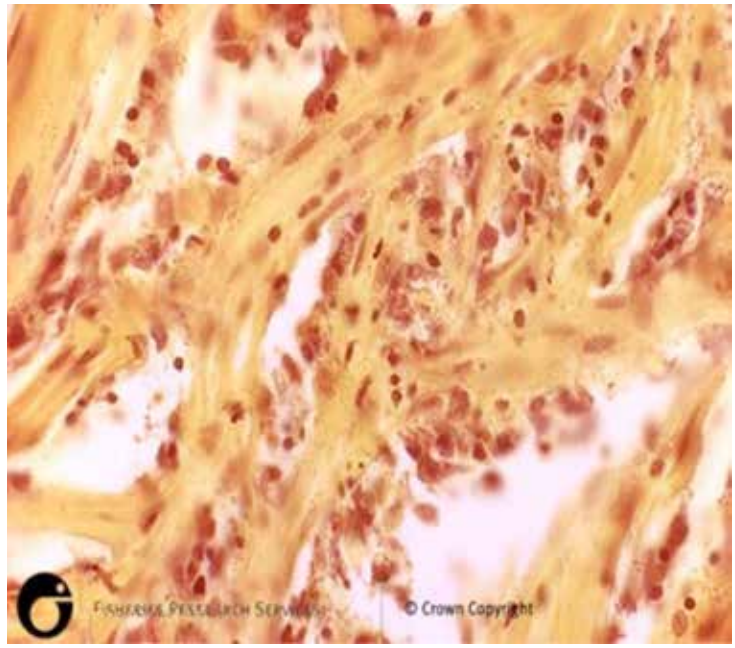
The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found on the Centre for Environment, Fisheries and Aquaculture Science (Cefas) International Database on Aquatic Animal Disease (IDAAD) website at www.cefas.defra.gov.uk/idaad/disocclist.aspx.

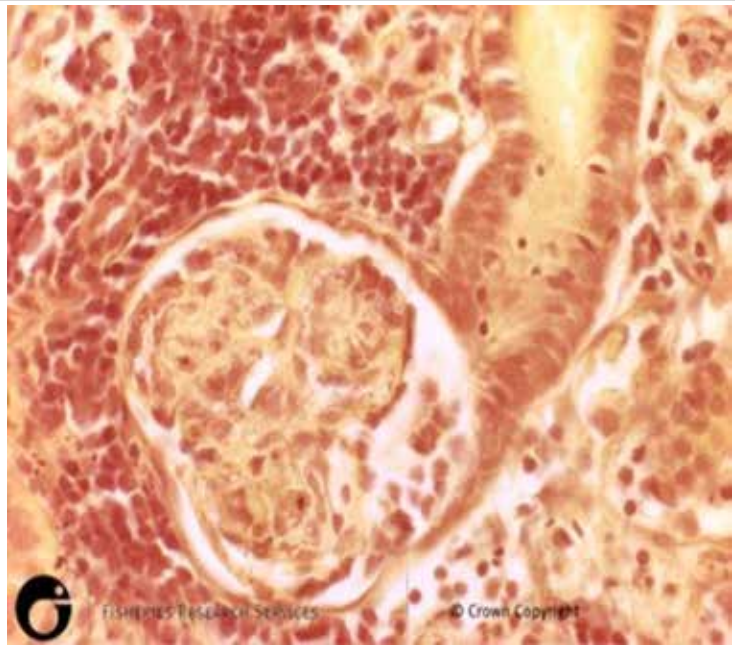
This hyperlink was correct and functioning at the time of publication.

Diffuse haemorrhage evident on tissue section taken from a rainbow trout naturally infected with *Yersinia ruckeri*



Source: Scottish Government

Cross section of a kidney nephron from a rainbow trout naturally infected with *Yersinia ruckeri*

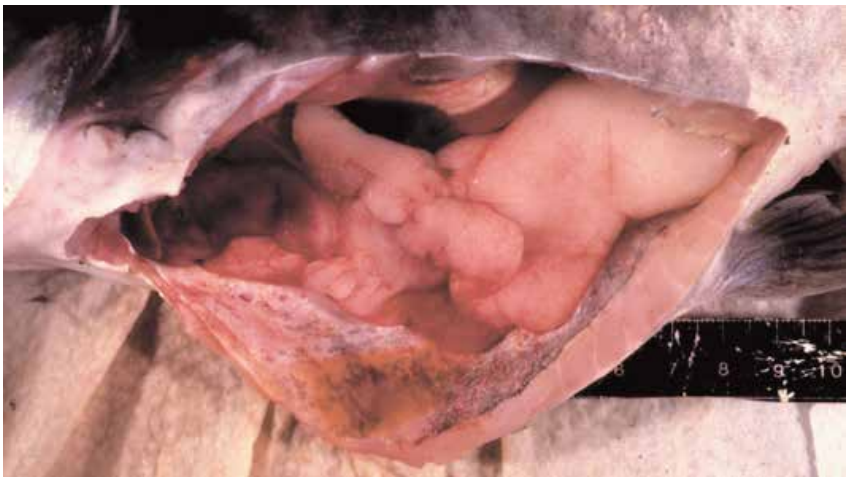


Source: Scottish Government

Enteric septicaemia of catfish

(Also known as edwardsiellosis or hole-in-the-head disease)

Fluid resulting from ascites in enteric septicaemia of catfish

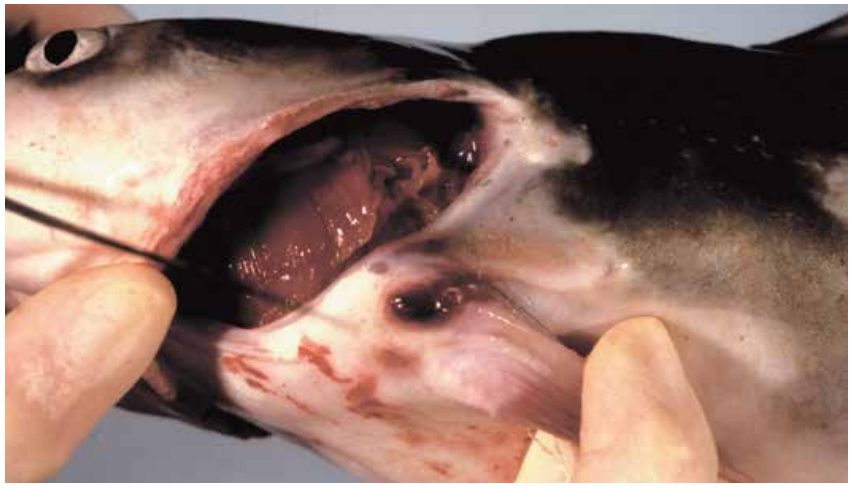


Source: LA Hanson

Cranial ulcers common in chronic enteric septicaemia of catfish

Source: LA Hanson

In chronic infections, lesions occasionally occur in the joints of the pectoral or dorsal spines.



Source: LA Hanson

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- lethargic swimming
- abnormal behaviour—alternating listlessness and chaotic swimming
- disorientation and swimming in spirals
- loss of appetite
- 'surfing', with head protruding from water.

Gross pathological signs in the *chronic* encephalitic form are:

- swelling on top of the head, occasionally progressing to the erosion of connective tissue and exposure of the brain (a 'hole in the head')
- granulomatous inflammation of the brain.

Gross pathological signs in the *acute* septicaemic form are:

- pale gills
- darkening of the skin (observed in species other than channel catfish)
- multiple small white spots on the skin
- raised skin patches progressing to shallow ulcers on the flanks and head
- haemorrhage at the base of the fins, around the mouth, and on the throat, operculum (gill cover) and abdomen
- exophthalmos (popeye)
- swollen abdomen ('pot-belly')
- ascites (fluid in the abdominal cavity)

- lesions on the liver and other internal organs
- intestine frequently bloody, but may be transparent or clear yellow
- red, swollen anal region with trailing faecal casts (has been observed in infected barramundi)
- soft and pale spleen, anterior kidney or posterior kidney, with petechial (pinpoint) haemorrhages.

Microscopic pathological signs are:

- gram-negative rods in histological sections (muscle, kidney)
- locally extensive cellulitis in the head region
- necrotising myositis.

Disease agent

Enteric septicaemia of catfish is caused by the bacterium *Edwardsiella ictaluri*, which belongs to the family *Enterobacteriaceae*.

Host range

Enteric septicaemia of catfish is a highly infectious bacterial disease of the catfish families Ictaluridae, Plotosidae, Clariidae, Siluridae, Pangasiidae and Ariidae, but especially the ictalurids. Several non-catfish species are also susceptible and are listed below.

Common name	Scientific name
Ayu **a**	*Plecoglossus altivelis*
Barramundi or Asian seabass	*Lates calcarifer*
Berney's catfish	*Neoarius berneyi*
Black bullhead **a**	*Ameiurus melas*
Black catfish	*Neosilurus ater*
Blue catfish **a**	*Ictalurus furcatus*
Brown bullhead **a**	*Ameiurus nebulosus*
Channel catfish **a**	*Ictalurus punctatus*
Chinook salmon	*Oncorhynchus tshawytscha*
Glass knifefish **a**	*Eigenmannia virescens*
Rainbow trout	*Oncorhynchus mykiss*
Rosy barb **a**	*Puntius conchonius*
Sind danio **a**	*Danio devario*
Sutchi catfish **a**	*Pangasius hypophthalmus*
Tadpole madtom **a**	*Noturus gyrinus*
Toothless catfish	*Anodontiglanis dahlia*
Walking catfish **a**	*Clarias batrachus*
White catfish **a**	*Ameiurus catus*
Yellow bullhead **a**	*Ameiurus natalis*
Yellow catfish **a**	*Pelteobagrus fulvidraco*
Zebrafish **a**	*Danio rerio*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

Enteric septicaemia of catfish has been officially reported in Australia from imported aquarium fish (danios, rosy barbs) held in contained facilities. In 2010 and 2011, native black catfish, toothless catfish and Berney's catfish held in a facility also holding imported aquarium fish became infected, resulting in mortalities. Targeted surveillance and testing has not provided evidence of persistent infection with *E. ictaluri* in wild or farmed fish populations in Australia.

Epidemiology

- *E. ictaluri* has also been linked to disease outbreaks in barramundi (also known as Asian seabass) and Pangasius.
- Enteric septicaemia of catfish has primarily been a disease of channel catfish; however, it has been reported from other catfish and non-catfish species. Most of the epidemiological knowledge about the disease is based on its occurrence in channel catfish.
- Horizontal transmission is via the faecal–oral route, cannibalism, and contact with contaminated water and materials used in handling infected fish.
- Faeces are the main source of shedding and dissemination, although dead fish and equipment such as fishing nets and sorting devices may be contaminated and spread the disease.
- The intestinal tract is the primary site of infection for the acute septicaemic form. The chronic encephalitic form is thought to establish after entering fish via the olfactory sacs.
- Surviving catfish carry the bacterium, which also seems to be able to survive in the intestinal tracts of other fish species.
- Disease occurs primarily at water temperatures between 18°C and 28°C, making spring and autumn the most common times for outbreaks. Stress is often a predisposing factor.
- The bacterium can survive 3–4 months in pond water, mud and vegetation.

- Environmental stressors can influence the expression of clinical signs and extend the incubation period.
- Morbidity and mortality can be high in heavily stocked ponds.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Channel catfish virus disease, spring viraemia of carp

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

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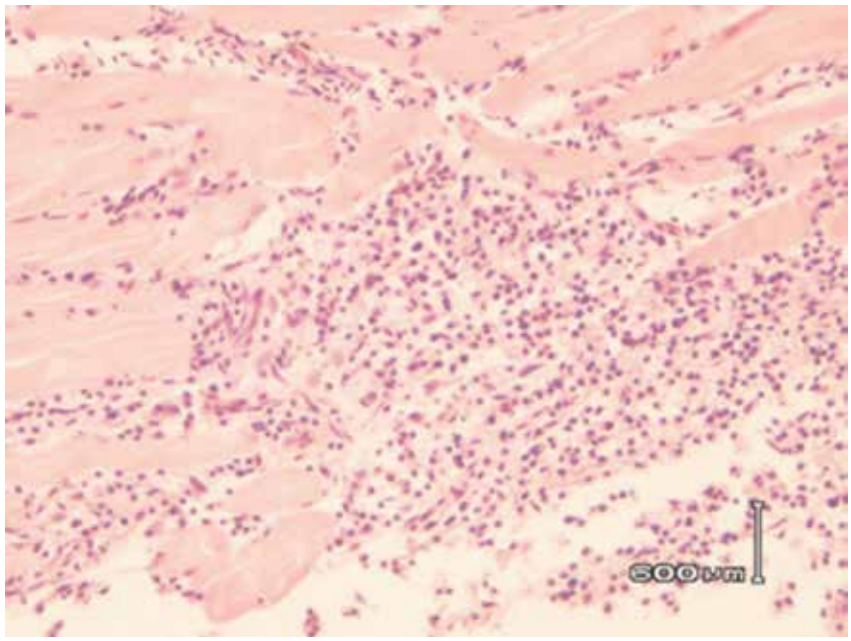
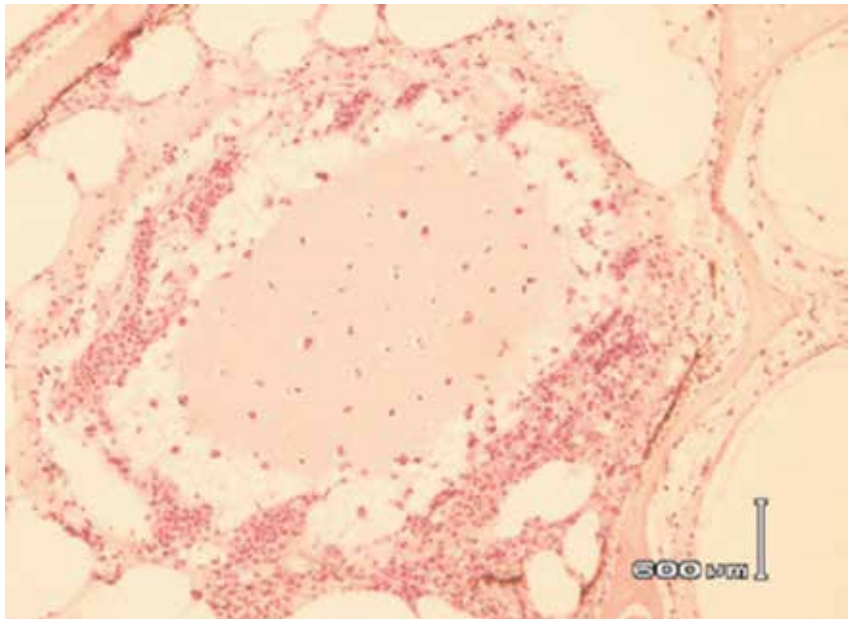
Further images

Necrotising myositis



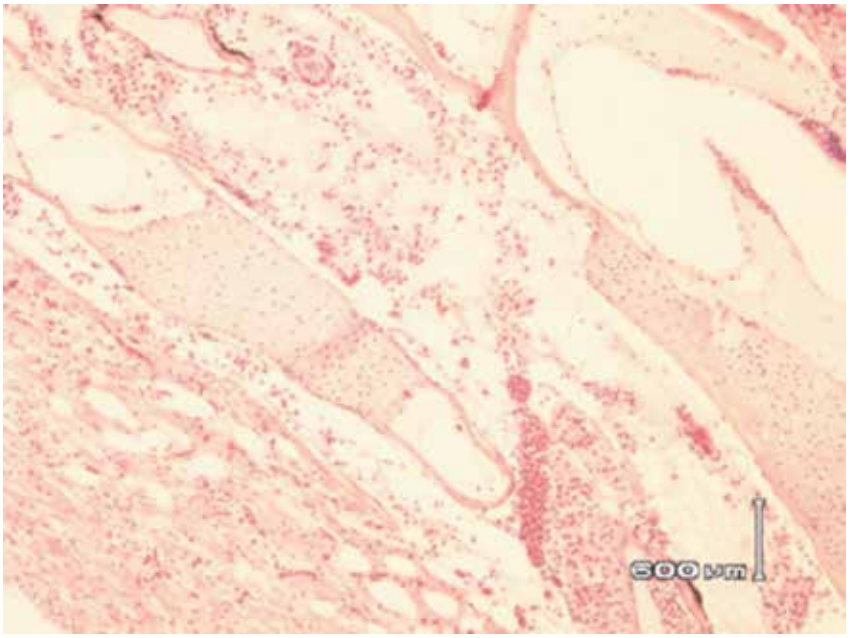
*Source:*G Storie and H Prior

Locally extensive cellulitis in the head, involving connective tissue surrounding cranial bones and cartilage



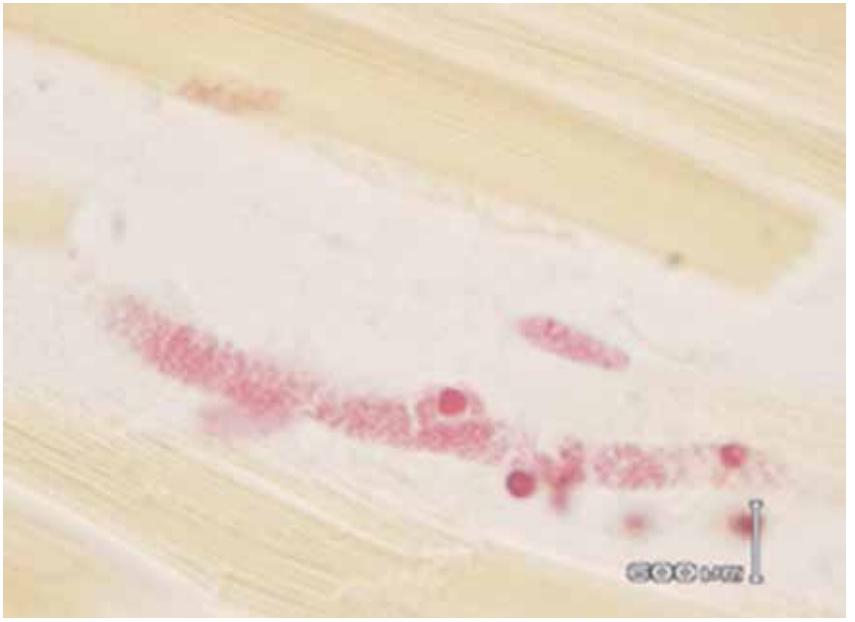
*Source:*G Storie and H Prior

Locally extensive cellulitis in the head, involving connective tissue surrounding cranial bones and cartilage



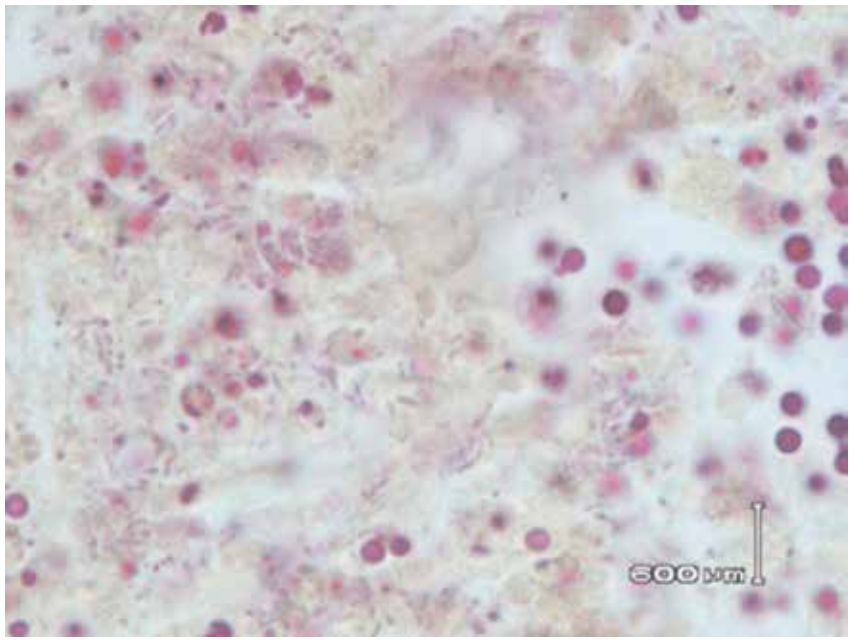
Source: G Storie and H Prior

Gram-negative rods (*Edwardsiella*) in skeletal muscle



Source: G Storie and H Prior

Gram-negative rods (*Edwardsiella*) in kidney



Source: G Storie and H Prior

Furunculosis

(Also known as infection with *Aeromonas salmonicida* subsp. *salmonicida*)

Exotic disease

Furunculosis in Atlantic salmon (*Salmo salar*); note the large furuncle (boil) on the side of the fish

Source: T Håstein

Furunculosis in Atlantic salmon (*Salmo salar*); note the furuncle cut away to show the underlying necrotic tissue

Source: T Håstein

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- sudden death, perhaps with slight exophthalmos (popeye)
- often no clinical signs in young fish with a peracute form of the disease
- death in a matter of days following initial clinical signs of disease in growing fish with acute furunculosis
- high mortality
- lethargic swimming or swimming just below the surface
- loss of appetite
- respiratory distress and jumping from the water immediately before an outbreak.

Gross pathological signs are:

- furuncles (or boils) involving skin and/or muscle, progressing to crater lesions (usually restricted to the subacute or chronic phase in adult salmon)
- haemorrhages on the skin, mouth and fin bases (mainly of paired fins)
- darkening of body colour and pale gills
- bloody discharge from nares and/or vent
- exophthalmos (popeye)
- haemorrhages in muscle and internal organs
- enlarged spleen and focal necrosis of the liver
- stomach filled with mucus, blood and sloughed epithelial cells
- congested intestine
- death without any clinical signs other than darkening of the skin, which can occur in peracute infections in juvenile salmon.

Microscopic pathological signs are:

- fusion of gill lamellae, with necrosis of the epithelium
- eosinophilic inflammatory changes in gills
- bacterial colonies in many tissues
- sloughing of renal tubular cells into the renal tubular lumen
- sloughing of intestinal epithelial cells into the intestinal lumen.

Disease agent

Furunculosis is caused by the bacterium *Aeromonas salmonicida* subsp. *salmonicida*. This bacterium is intracellular and so may avoid some host immune defences after infection. During infection, the bacterium produces extracellular toxins that may play a significant role in the pathogenesis of the disease. There are four other subspecies of *A. salmonicida*; these 'atypical' strains are considered to cause a different disease syndrome, infection with *Aeromonas salmonicida*—atypical strains, which is also discussed in this guide.

Host range

All salmonids are believed to be susceptible to *A. salmonicida* subsp. *salmonicida*. Species known to be susceptible are listed below.

Common name	Scientific name
Atlantic cod **a**	*Gadus morhua*
Atlantic salmon **a**	*Salmo salar*
Brook trout **a**	*Salvelinus fontinalis* (particularly susceptible)
Brown trout **a**	*Salmo trutta* (particularly susceptible)
Bullhead	*Cottus gobio*
Common shiner	*Notropis cornutus*
Eel **a**	*Anguilla rostrata*
Gilt-head seabream **a**	*Sparus aurata*
Halibut **a**	*Hippoglossus stenolepis*
Minnows	*Galaxiidae*
Pike	*Esox lucius*
Pike perch	*Sander lucioperca*
Rainbow trout **a**	*Oncorhynchus mykiss*
Salmonids (all presumed susceptible)	*Salmonidae*
Sea lamprey **a**	*Petromyzon marinus*
Turbot	*Psetta maxima* (also known as Scophthalmus maximus)
Wolffish **a**	*Anarhichas lupus*
Wrasse	*Labridae*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

The strain of *Aeromonas* causing furunculosis in salmonids overseas (i.e. the typical strain) is not present in Australia. However, atypical strains are present (see 'Infection with *Aeromonas salmonicida*—atypical strains').

Epidemiology

- Furunculosis is highly contagious and affects fish of all ages.
- The disease one of the most commercially significant salmonid diseases, occurring in freshwater and marine salmonid aquaculture in all countries except Australia and New Zealand.
- Overseas, this disease must be controlled on farms by medication or vaccination.
- Outbreaks typically occur at temperatures above 10°C, however outbreaks can occur in very young fish and at temperatures as low as 2–4°C. Disease may be precipitated by endogenous (e.g. smoltification, spawning) and exogenous (e.g. temperature fluctuations, poor water quality) stressors.
- Australia's relatively warm water temperatures may favour the establishment of *A. salmonicida* subsp. *salmonicida*.
- Rainbow trout are relatively resistant to the disease, but are still considered susceptible.
- Horizontal transmission occurs via the water column, but also through direct fish-to-fish contact and animal vectors (birds and invertebrates such as sea lice). *A. salmonicida* subsp. *salmonicida* has been detected on the surface of fertilised eggs; surface decontamination using iodine is effective in preventing vertical transmission (passage of infection from parent to offspring).
- *A. salmonicida* subsp. *salmonicida* can survive for months in some environments.

- Non-salmonids may become infected by ingesting tissue of infected salmonids. Similarly, transmission to non-salmonids can occur where fish caught for feed are taken from waters near an outbreak.
- Susceptibility to the disease increases with damaged mucous and skin, such as occurs when fish are handled with nets.
- Fish surviving disease outbreaks are recognised as carriers of the disease and may continue to infect the remaining population without themselves showing any outward signs of infection. Stress may precipitate disease in subclinical carriers.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with *Aeromonas salmonicida*—atypical strains, epizootic haematopoietic necrosis, infectious haematopoietic necrosis

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found on the Australian Government Department of Agriculture, Fisheries and Forestry website at www.daff.gov.au/animal-plant-health/aquatic/aquavetplan/furunculosis.

This hyperlink was correct and functioning at the time of publication.

Infection with *Aeromonas Salmonicida*—atypical strains

(Disease resulting from infection with atypical strains of *Aeromonas salmonicida* is known by such names as goldfish ulcer disease; carp erythrodermatitis; ulcer disease of flounder, eel and salmon; or, in Tasmania, marine aeromonas disease of salmonids [MAS])

Goldfish ulcer disease in goldfish; note characteristic ulcers on the body

Source: J Carson

Atypical *A. salmonicida* in silver perch (*Bidyanus bidyanus*)

Source: M Landos

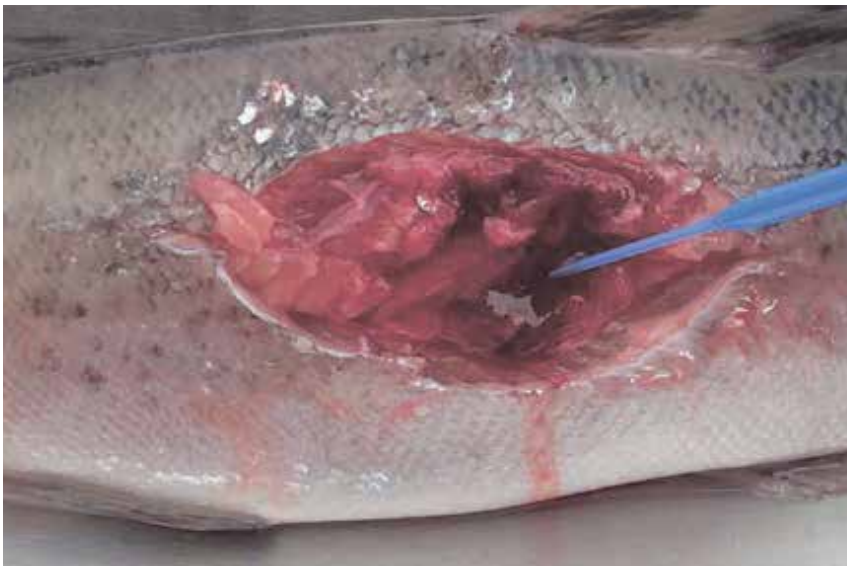
A. salmonicida biovar Acheron in Atlantic salmon (*Salmo salar*), known as marine aeromonid disease of salmonids

Source: K Ellard

The blood-filled furuncles are specific to this condition, but are rarely noted because they rupture easily, resulting in ulcerations extending into the muscle

K Ellard

Ulcers develop below the surface of the skin, extending into muscle

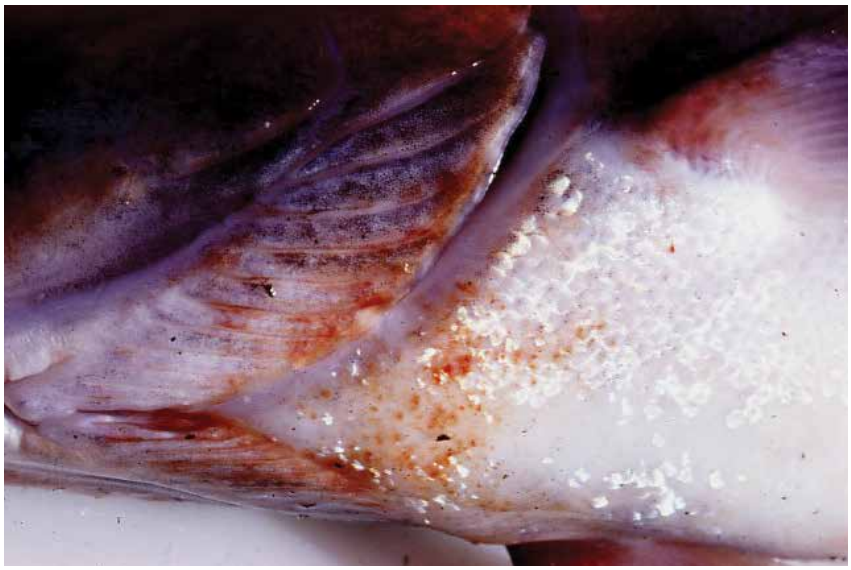


Source: K Ellard

Ventral side of greenback flounder (*Rhombosolea tapirina*), with lesion caused by the greenback flounder strain of *A. salmonicida*

J Carson

Atlantic salmon (*Salmo salar*) infected with the greenback flounder strain of atypical *A. salmonicida* after cohabitation with infected flounder



Source: J Carson

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- lethargic swimming
- abnormal swimming and disorientation
- loss of appetite
- increased mortality.

Gross pathological signs are:

- white raised patches on the skin that progress to ragged-edged red ulcers
- haemorrhages on the skin and fin bases (usually the paired fins)
- fingernail-sized ulcers found anywhere on the fish but most often on the upper side of the lateral line behind the head or at the base of the tail fin
- pale gills with petechial (pinpoint) haemorrhages
- intestinal protrusion through the abdominal wall following severe ulceration
- haemorrhages in muscle and internal organs
- swollen kidneys and spleen.

Microscopic pathological signs are:

- hyperplasia of the gills, which may contain bacterial colonies
- ulcerated areas that show oedema, hyperaemia, leukocyte infiltration and considerable degenerative changes

- hyperaemia and haemorrhage in the spleen and kidneys
- fibroblast-like cells, which may produce granulomas in the dermis, spleen and kidney.

Disease agent

Atypical strains of the *A. salmonicida* bacterium differ from the typical strain causing furunculosis in salmonids. Atypical strains affect mainly non-salmonids (wild and cultured, marine and freshwater). Infection with atypical *A. salmonicida* does not necessarily result in the acute mortality and septicaemia that are characteristic of the typical furunculosis strain, but manifests more as external lesions and ulceration, often involving secondary infection. An exception to this is infection by MAS in Tasmania; in such cases, Atlantic salmon are most commonly affected, and clinical presentation is similar to furunculosis.

There are five subspecies of *A. salmonicida*, only one of which is known as a typical strain and causes furunculosis (*A. salmonicida salmonicida*). The other four subspecies (*achromogenes*, *masoucida*, *smithia* and *pectinolytica*) are referred to as atypical *A. salmonicida* and are recognised worldwide. Of these, all except *A. salmonicida pectinolytica* cause disease in fish. A new atypical strain that has recently been described in Australia, *A. salmonicida* biovar Acheron, causes MAS in Atlantic salmon.

Host range

Species known to be susceptible to atypical *A. salmonicida* are listed below.

Common name	Scientific name
American plaice	*Hippoglossoides platessoides*
Arctic char	*Salvelinus alpinus*
Atlantic cod **a**	*Gadus morhua*
Atlantic herring **a**	*Clupea harengus*
Atlantic salmon **a**	*Salmo salar*
Black rockfish **a**	*Sebastes schlegeli*
Brook trout	*Salvelinus fontinalis*
Brown trout **a**	*Salmo trutta*
European carp **a**	*Cyprinus carpio*
Chub **a**	*Leuciscus cephalus*
Chum salmon **a**	*Oncorhynchus keta*
Common roach **a**	*Rutilus rutilus*
Crucian carp **a**	*Carassius carassius*
Dab **a**	*Limanda limanda*
Dace **a**	*Leuciscus leuciscus*
Eel **a**	*Anguilla* spp.
Flounder **a**	*Platichthys flesus*
Fourbeard rockling **a**	*Enchelyopus cimbrius*
Freshwater bream **a**	*Abramis brama*
Goldfish **a**	*Carassius auratus*
Goldsinny **a**	*Ctenolabrus rupestris*
Grayling **a**	*Thymallus thymallus*
Great sandeel **a**	*Hyperoplus lanceolatus*
Greenback flounder **a**	*Rhombosolea tapirina*
Haddock **a**	*Melanogrammus aeglefinus*
Halibut **a**	*Hippoglossus stenolepis*

Common name	Scientific name
Japanese flounder **a**	*Paralichthys olivaceus*
Lesser sand eel	*Ammodytes lancea*
Masu salmon **a**	*Oncorhynchus masou*
Minnow **a**	*Phoxinus phoxinus*
Pike	*Esox lucius*
Pink salmon	*Oncorhynchus gorbuscha*
Plaice **a**	*Pleuronectes platessa*
Rainbow trout **a**	*Oncorhynchus mykiss*
Redfin perch or European perch	*Perca fluviatilis*
Rudd **a**	*Scardinius erythrophthalmus*
Sablefish	*Anoplopoma fimbria*
Shotted halibut **a**	*Eopsetta grigorjewi*
Shububkin **a**	*Carassius* sp.
Silver bream **a**	*Blicca bjoerkna*
Silver perch **a**	*Bidyanus bidyanus*
Smallmouth bass **a**	*Micropterus dolomieui*
Sockeye salmon **a**	*Oncorhynchus nerka*
Spotted wolffish **a**	*Anarhichas minor*
Striped trumpeter **a**	*Latris lineata*
Tomcoda	*Gadus microgadus*
Turbota	*Psetta maxima* (also known as *Scophthalmus maximus*)
Viviparous blenny **a**	*Zoarces viviparus*
Whitefish	*Coregonus* spp.
Whiting **a**	*Merlangius merlangus*
Wrasse **a**	*Labrus bergylta*
Yellow bass **a**	*Morone mississippiensis*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Laboratory experiments indicate that all trout and salmon species, as well as many non-salmonids, are potentially susceptible to atypical strains of *A. salmonicida*. For example, Atlantic salmon and striped trumpeter can be infected by cohabitation with infected flounder. Rainbow trout are relatively resistant to atypical strains.

Presence in Australia

Atypical *A. salmonicida* has been officially reported from New South Wales, Queensland, South Australia, Victoria (goldfish ulcer disease) and Tasmania (greenback flounder biovar and Acheron biovar). Movement controls are in place to prevent the spread of goldfish ulcer disease to Western Australia and Tasmania. The Acheron biovar has been reported only from Tasmania and is limited to an isolated production area.

Epidemiology

- Transmission occurs horizontally (between fish via the water).
- Susceptibility to the disease increases with damaged mucus and skin, which occurs when fish are handled with nets.
- Outbreaks are expected to occur at water temperatures above 10°C (i.e. summer months in southern waters of Australia) and may be precipitated by stress (i.e. handling, overpopulation and rapid temperature fluctuations).
- Secondary infection with other bacteria often results.
- Fish that survive disease outbreaks are recognised as carriers of the disease and may continue to infect the remaining population without themselves exhibiting signs of infection.
- Diagnosis based on clinical or external signs of disease is difficult because clinical signs vary and skin ulcers are often infected with opportunistic bacteria and fungi. A definitive diagnosis requires laboratory examination.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Channel catfish virus disease, epizootic ulcerative syndrome, furunculosis, spring viraemia of carp

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

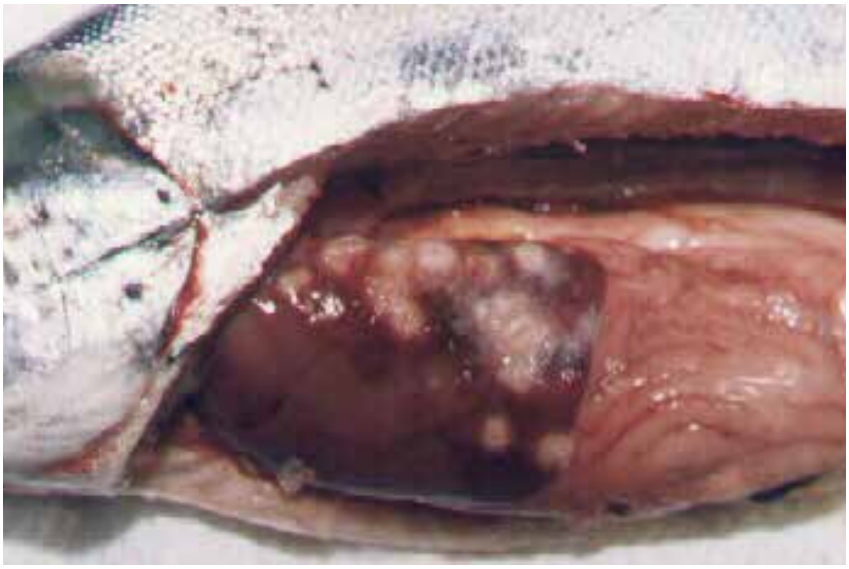
BK Guðmundsdóttir (undated), *The fish pathogen Aeromonas salmonicida subsp.* achromogenes, available at www.nfmikro.net/Vintermotet04/Foredrag/Gudmundsdottir.htm.

This hyperlink was correct and functioning at the time of publication.

Piscirickettsiosis

Exotic disease

Piscirickettsiosis in Atlantic salmon (*Salmo salar*); note pale circular granulomas in liver and pinpoint haemorrhaging in pyloric caeca



Source: S Bravo

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- increasing mortality
- loss of appetite
- emaciation
- lethargic swimming
- circling
- swimming near the surface or at the sides of the net or pens.

Gross pathological signs are:

- progressive skin lesions that range from areas of raised scales, to white raised plaques, to shallow ulcers on flanks and head
- darkening of skin and pale gills
- swollen abdomen
- grey, swollen spleen and kidney
- mottled liver (due to diffuse haemorrhages) or ring-shaped white to pale-yellow lesions (granulomas and areas of necrosis)

- ascites (fluid in the abdominal cavity)
- signs of peritonitis, including generalised diffuse inflammation of abdominal organs, adhesions and increased volume of free abdominal fluid
- petechial (pinpoint) haemorrhages of the gastrointestinal tract, swim bladder and visceral fat.

Microscopic pathological signs are:

- vasculitis and necrosis of the liver and kidney; inflammatory macrophage infiltration
- *Rickettsia*-like organisms in macrophages and epithelial cells.

Disease agent

Piscirickettsiosis is caused by the bacterium *Piscirickettsia salmonis*, which has recently been classified within the gammaproteobacteria, family *Piscirickettsiaceae*.

Host range

Fish known to be susceptible to piscirickettsiosis are listed below.

Common name [a]	Scientific name
Atlantic salmon	*Salmo salar*
Chinook salmon	*Oncorhynchus tshawytscha*
Coho salmon	*Oncorhynchus kisutch* (most susceptible)
European seabass	*Dicentrarchus labrax*
Masu salmon	*Oncorhynchus masou*
Pink salmon	*Oncorhynchus gorbuscha*
Rainbow trout	*Oncorhynchus mykiss*

a All species listed are naturally susceptible (other species have been shown to be experimentally susceptible).

Piscirickettsiosis is only known to affect aquaculture stock and has not been recorded in fish from the wild.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Salmonid rickettsial septicaemia is a term used to describe diseases of salmonids caused by *Rickettsia*-like organisms, including *P. salmonis*. Piscirickettsiosis refers to the disease specifically caused by the bacterium *P. salmonis*.
- Outbreaks usually occur after fish are introduced to saltwater pens at water temperatures between 12°C and 18°C.
- Transmission is mainly horizontal (fish to fish). Although *P. salmonis* has been isolated in reproductive organs of salmonids, vertical transmission has not been definitively demonstrated.
- Bacteria are assumed to gain entry by breaching the physical barriers of the skin or gills. The invading bacteria then spread throughout the body via the blood (haematogenous spread), resulting in septicaemia.
- Mortality rates typically range between 10% and 30%, but have been recorded at 90% in seawater-raised coho salmon from Chile.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to piscirickettsiosis.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

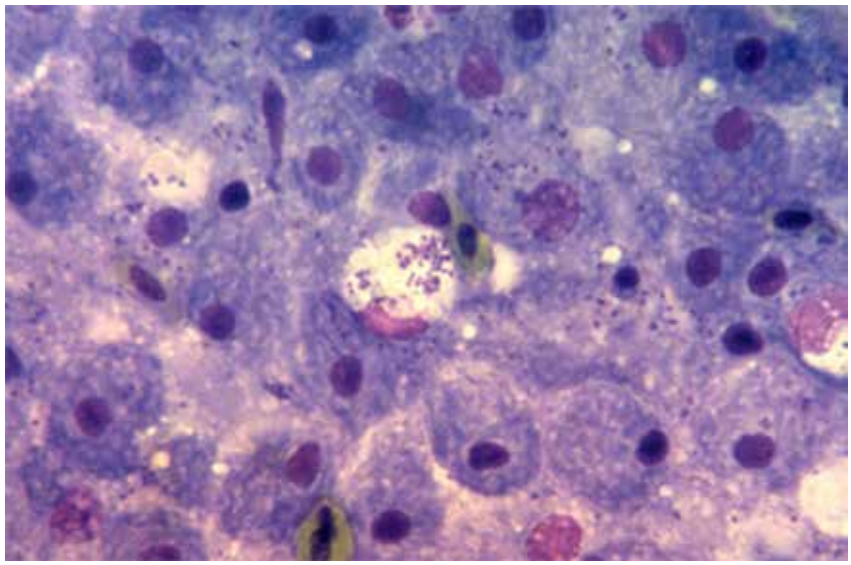
Further reading

Further information can be found on the Centre for Environment, Fisheries and Aquaculture Science (Cefas) International Database on Aquatic Animal Disease (IDAAD) website at www.cefas.defra.gov.uk/idaad/disocclist.aspx.

This hyperlink was correct and functioning at the time of publication.

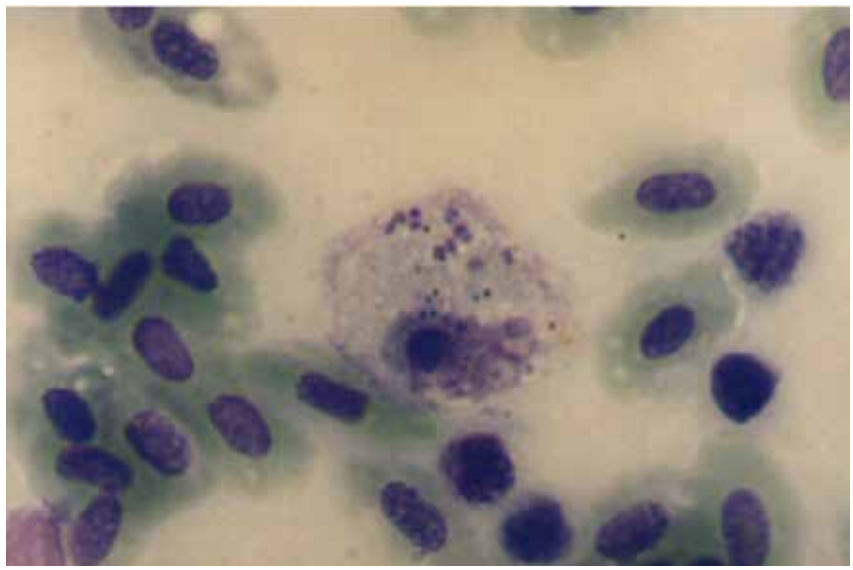
Further images

P. salmonis detected in tissue imprint



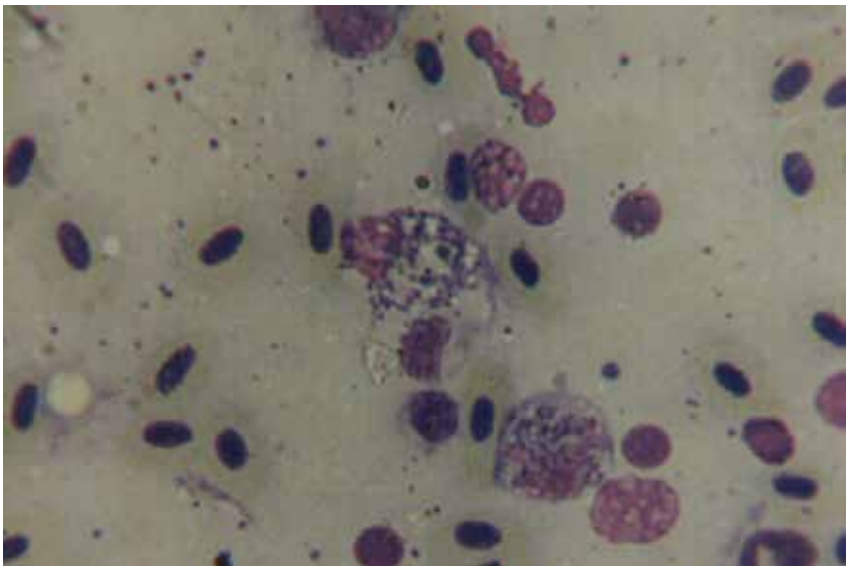
Source: European Association of Fish Pathologists

P. salmonis detected in tissue imprint



*Source:*S Bravo

P. salmonis detected in tissue imprint



Source: S Bravo

Exotic disease

Parasitic diseases of finfish

Gyrodactylosis

Gyrodactylosis in Atlantic salmon (*Salmo salar*); note excessive mucous and peeling of skin, especially around the tail

Source: T Håstein

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- lethargy
- high mortality in Atlantic salmon (*Salmo salar*)
- scrubbing (rubbing against objects in response to skin irritation) and flashing (darting and twisting of fish and erratic swimming)
- gathering in low-current waters when heavily infected.

Gross pathological signs are:

- ulcers
- sloughing of the skin
- greyish appearance; as disease progresses, dorsal and pectoral fins may have a whitish appearance due to thickening of the epidermis
- excess mucus on skin
- frayed fins.

Disease agent

The causative agent for gyrodactylosis is an obligate freshwater ectoparasite, *Gyrodactylus salaris*, which is a flatworm (Platyhelminthes) belonging to the family Gyrodactylidae (class Monogenea). *G. salaris* occurs in Europe, mainly around the Baltic Sea and Norway.

Most waters have many types of flukes that are parasitic on fish gills and skin. Any evidence of infestation with these parasites beyond what is visible to the naked eye (as described above) requires identification by a parasitologist experienced in identifying *Gyrodactylus* species.

Host range

All species of salmonid should be considered potentially susceptible to *G. salaris* unless testing finds them to be not susceptible. Species which have been demonstrated susceptible to gyrodactylosis are listed below.

Common name [a]	Scientific name
Arctic char	*Salvelinus alpinus*
Atlantic salmon	*Salmo salar*
Brook trout	*Salvelinus fontinalis*
Brown trout [b]	*Salmo trutta*
Grayling	*Thymallus thymallus*
Lake trout	*Salvelinus namaycush*
Rainbow trout	*Oncorhynchus mykiss*

a All the species listed are naturally susceptible (other species have been shown to be experimentally susceptible). **b** Susceptibility of brown trout is very low; G. salaris will usually only establish in a brown trout population that inhabits the same area as a population of infected salmonids.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- *G. salaris* may be present for years in farmed salmonids, especially rainbow trout, without the fish showing any clinical signs of disease.
- *G. salaris* is a freshwater parasite that cannot survive in seawater; however, it can survive a few days at salinity of up to 20 parts per thousand.
- The parasite can survive 5–6 days detached from the host but cannot survive drying out.
- Transmission is horizontal (directly via the water column) by contact between infected and uninfected fish, or by contact between host fish and detached parasites on the substrate.
- Mortality in susceptible farmed Atlantic salmon can reach 100%.
- The parasite is readily spread between farms and countries through the transport of infected fish.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Many diseases listed in this field guide appear similar to gyrodactylosis. Further laboratory diagnosis is required for any presumptive diagnosis.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

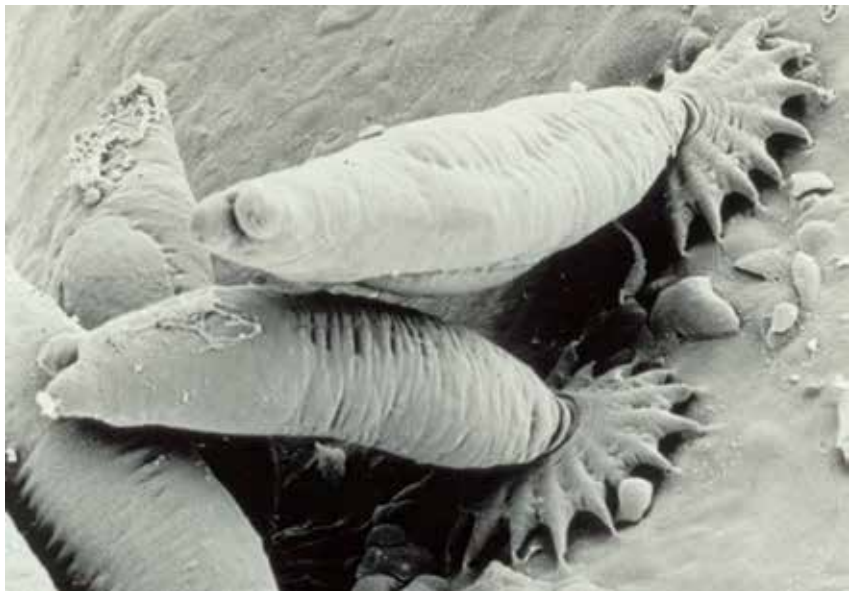
Further reading

The accepted procedures for a conclusive diagnosis of gyrodactylosis are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

Further image

Scanning electron micrograph of *G. salaris* attached to the skin of an Atlantic salmon (*Salmo salar*) parr



Source: TA Mo

Whirling disease

Exotic disease

Whirling disease in trout; note deformed skull resulting from long-term infection

Source: T Håstein

Whirling disease in trout; note classic darkening of the skin from vent to tail

T Poppe

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- mass mortalities in fry
- convulsive movements
- increased rate of breathing
- jerking backwards movements
- swimming with a whirling motion ('tail chasing')
- erratic, nervous darting movements until exhausted.

Gross pathological signs are:

- darkening of the skin from the vent to the tail ('blacktail')
- spinal curvature
- skull deformation and shortened gill plates.

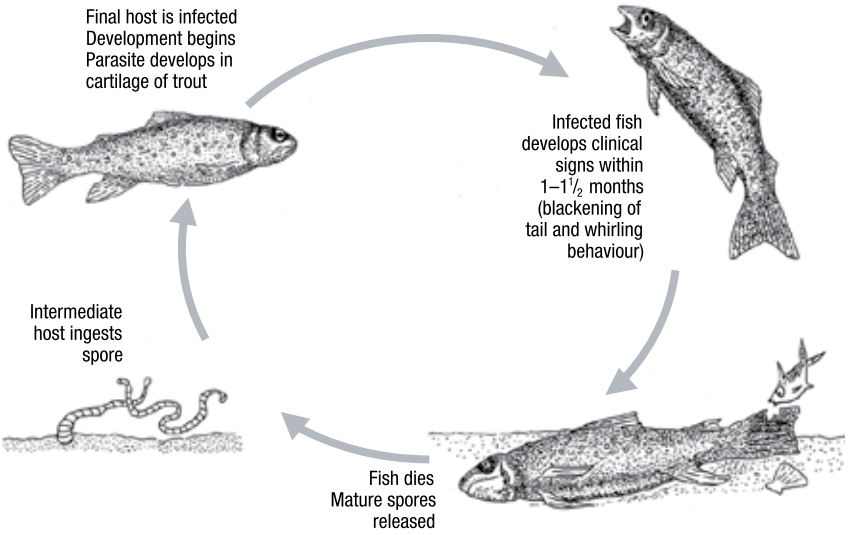
Disease agent

Whirling disease is caused by *Myxobolus cerebralis*, a parasitic protozoan that affects salmonids.

Life cycle

- *M. cerebralis* has a complex life cycle involving two hosts: fish (trout or salmon species) and an intermediate host, the tiny bottom-dwelling tubifex mud worm (*Tubifex tubifex*).
- Myxobolus spores (myxospores) develop in infected fish and are released into the environment when the fish dies. The myxospores are very persistent and can survive in moist environments for many years.
- Myxospores are ingested from the environment by tubifex mud worms. The spores then rapidly multiply in the worm's intestine.
- A fragile waterborne spore stage of the parasite (triactinomyxon stage) is released from an infected intermediate host and attaches itself to a definitive host (i.e. fish). This must occur within a few days for survival of the spore stage. The parasite then migrates through the fish skin into the central nervous system, and ultimately into the cartilage.
- Following death and decomposition of infected fish, myxospores are released into the environment and the life cycle continues.
- Spores survive passing through the digestive tract of predators (e.g. birds) and can be transferred from place to place on muddy boots and equipment.

Life cycle of *Myxobolus cerebralis*



Source: Australian Government Department of Agriculture, Fisheries and Forestry

Host range

Species known to be susceptible to whirling disease are listed below.

Common name [a]	Scientific name
Atlantic salmon	*Salmo salar*
Brook trout	*Salvelinus fontinalis*
Brown trout	*Salmo trutta*
Bull trout	*Salvelinus confluentus*
Chinook salmon	*Oncorhynchus tshawytscha*
Cutthroat trout	*Oncorhynchus clarki*
Golden trout	*Oncorhynchus aguabonita*
Rainbow trout	*Oncorhynchus mykiss* (most susceptible)
Sockeye salmon	*Oncorhynchus nerka*

a All the species listed are naturally susceptible (other species have been shown to be experimentally susceptible).

The presence of clinical signs in all the species listed here can depend on many factors, particularly the age of the primary host of the initial infection. For example, infected rainbow trout older than 9 weeks will generally show no clinical signs; chinook salmon do not exhibit clinical signs of disease beyond the age of 3 weeks.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Whirling disease is chronic and primarily affects young fish.
- Susceptibility is influenced by water temperature, age and species.
- Young fish are highly susceptible because the parasite attacks their soft cartilage, resulting in nerve damage, skeletal deformities and sometimes death.
- Clinical signs of the disease are not evident until fish are approximately 7 cm long.
- When fish are 8–10 cm long, cartilage forms into bone and they are no longer susceptible to disease; however they remain carriers of the parasite.
- The intermediate host, the worm *Tubifex tubifex*, is found in Australia.
- Because tubifex worms live in mud, the disease can be partly controlled in trout farms by growing young fish in concrete raceways.
- The parasite spreads mainly through the stocking of infected fish and also through the alimentary tracts of fish-eating migratory birds.
- *M. cerebralis* is highly resistant in the myxospore stage. The myxospore released from the fish can withstand freezing and desiccation, and may survive in a stream for 20–30 years with appropriate environmental conditions. However, the spore stage released from the worm is short lived and susceptible to standard disinfection procedures.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infectious haematopoietic necrosis, viral haemorrhagic septicaemia

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

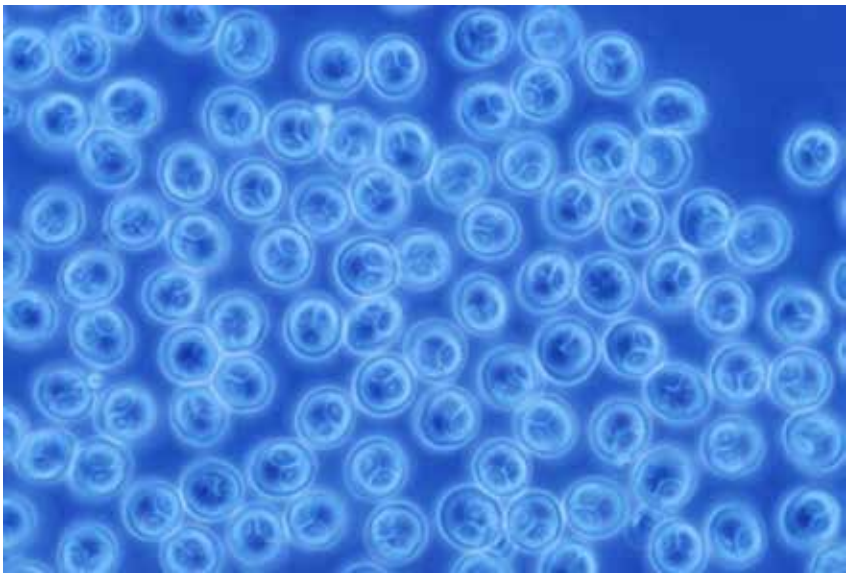
Further reading

Further information can be found on the Colorado Department of Natural Resources website at wildlife.state.co.us/Fishing/Management/Pages/WhirlingDisease.aspx.

This hyperlink was correct and functioning at the time of publication.

Further images

Phase micrograph of *M. cerebralis* myxospores extracted from infected rainbow trout cranial cartilage by the pepsin trypsin digest method. The spores measure approximately 10 µm in diameter



Source: J Bartholomew

Whirling disease in trout; note classic darkening of the skin from anus to tail (i)

Source: J Bartholomew

Whirling disease in trout; note classic darkening of the skin from anus to tail (ii)

Source: J Bartholomew

Trout infected with *M. cerebralis* displaying typical deformity

Source: J Bartholomew

Other diseases of finfish

Epizootic ulcerative syndrome (EUS)

(Also known as red spot disease, mycotic granulomatosis and ulcerative mycosis)

EUS in sand whiting; note progression of red lesion (top) to deep ulcer (bottom) and classic red sores on the body

Source: New South Wales Department of Primary Industries

EUS in a juvenile silver perch; note classic red ulcer on the body

Source: New South Wales Department of Primary Industries

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- loss of appetite
- dark body colour
- mass mortality
- erratic swimming
- 'rubbing' on the surfaces of tanks
- increased respiration and respiratory effort.

Gross pathological signs are:

- lesions on the body—red spots, black burn-like marks, or deeper ulcers with red centres and white rims
- progressive lesions—lesions start as reddening under a single scale but quickly spread to involve adjacent scales. Lesions continue to widen and deepen, forming ulcers that erode underlying tissues to expose (depending on location) skeletal musculature, vertebrae, brain or visceral organs
- unilateral or bilateral clouding of the eye (particularly in barramundi)

Microscopic pathological signs are:

- erythematous dermatitis
- hyphae (threads) associated with granulomatous tissue, sometimes extending into visceral organs
- liquefactive necrosis of muscle tissue.

Disease agent

EUS is caused by infection with the oomycete *Aphanomyces invadans*. Although previously regarded as a fungus, the genus *Aphanomyces* is now classified with diatoms and brown algae in a group called Stramenopiles or Chromista.

Host range

More than 100 estuarine and freshwater species of fish are known to be susceptible to EUS. Known susceptible species likely to be encountered in Australia are listed on the next page (b).

Common name	Scientific name
Australian bass	*Macquaria novemaculeata*
Archer fish **a**	*Toxotes chatareus*
Barcoo grunter	*Scortum barcoo*
Barramundi **a**	*Lates calcarifer*
Bony bream **a**	*Nematalosa erebi*
Chanda perch **a**	*Ambassis agassizii*
Cichlids **a**	*Cichlidae*
Climbing perch **a**	*Anabas testudineus*
Cyprinids **a**	*Cyprinidae*
Dusky flathead	*Platycephalus fuscus*
Eastern freshwater cod	*Maccullochella ikei*
Eel **a**	*Anguilla australis*
Estuarine rockcod **a**	*Epinephelus tauvina*
Flathead goby **a**	*Glossogobius giuris*
Fork-tailed catfish	*Arius spp.*
Froggatt's catfish	*Cinetodus froggatti*
Giant glassfish **a**	*Parambassis gulliveri*
Giant gudgeon **a**	*Oxyeleotris selheimi*
Goldfish	*Carassius auratus auratus*
Golden perch	*Macquaria ambigua*
Long tom **a**	*Strongylura krefftii*
Mangrove jack **a**	*Lutjanus argentimaculatus*
Mouth almighty **a**	*Glossamia aprion*
Mullet **a**	*Mugil cephalus*
Mullets **a**	*Mugilidae*
Murray cod	*Maccullochella peelii*
Nurseryfish	*Kurtus gulliveri*
Primitive archer fish **a**	*Toxotes lorentzi*
Rainbow fish **a**	*Melanotaenia splendida*
Rainbow trout	*Oncorhynchus mykiss*
Saratoga **a**	*Scleropages jardini*
Scat **a**	*Scatophagus argus*
Silver perch **a**	*Bidyanus bidyanus*
Silver trevally **a**	*Pseudocaranx dentex*
Sleepy cod **a**	*Oxyeleotris lineolatus*
Striped grunter **a**	*Amniataba percoides*
Spangled perch **a**	*Leiopotherapon unicolor*
Triangular shield catfish **a**	*Arius leptaspis*
Tropical two-winged flying fish **a**	*Exocoetus volitans*
Whiting **a**	*Sillago ciliata*
Yellowfin bream **a**	*Acanthopagrus australis*

a Naturally susceptible (other species have been shown to be experimentally susceptible). **b** A current list of known susceptible species found both within and outside of Australia can be accessed at the address below (see further reading).

Presence in Australia

EUS is endemic in many freshwater catchments and estuaries in Australia and has been officially reported from New South Wales, the Northern Territory, Queensland, Victoria, South Australia and Western Australia.

Epidemiology

- The disease is seen in more than 100 freshwater and estuarine species of fish and infection has spread rapidly throughout Asia, associated with the movement of ornamental fish.
- Predisposing factors leading to skin damage, such as parasites, bacterial or viral infection, physical trauma or acidic water are normally required to allow the *Aphanomyces* oomycete to initiate clinical signs of EUS. Secondary opportunistic infections are also a common finding.
- Disease is often associated with acid water run-off and can appear after heavy rains (particularly after a long, dry period) with water temperatures of 18–22°C favouring sporulation.
- Disease transmission is through zoospore transfer in water, direct contact between fish and skin contamination (penetration assisted by damage to skin).

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Aeromonas salmonicida—atypical strains, koi herpesvirus disease, viral haemorrhagic septicaemia

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found on the following websites:

Network of Aquaculture Centres in Asia–Pacific: library.enaca.org/Health/DiseaseLibrary/EpizooticUlcerativeSyndrome.pdf

Northern Territory Government: www.nt.gov.au/d/Content/File/p/Fishnote/FN01.pdf.

The accepted procedures for a conclusive diagnosis of EUS and a full list of susceptible species are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online. These hyperlinks were correct and functioning at the time of publication.

4 Diseases of molluscs

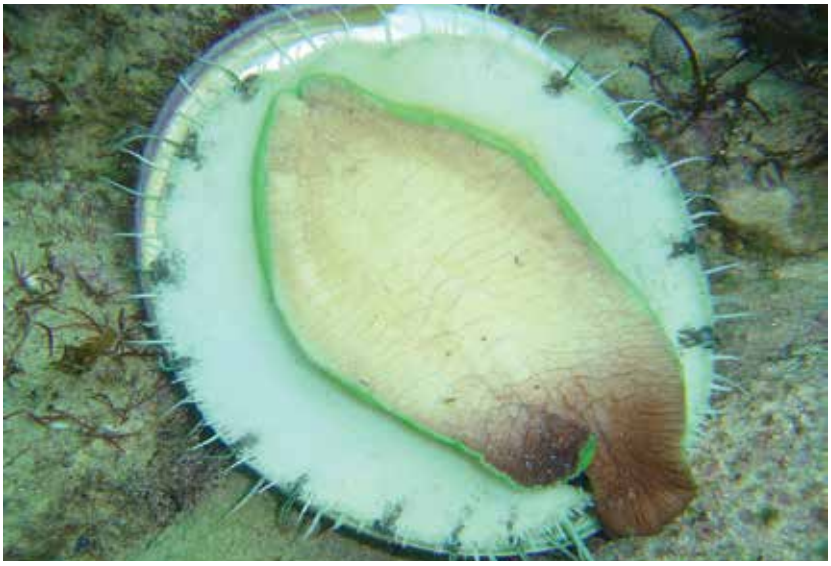
Viral diseases of molluscs

Abalone viral ganglioneuritis (AVG)
(Listed by the OIE as infection with abalone herpes virus [AbHV])

Abalone with AVG. Note the swollen and protruding mouth parts, particularly the prominent radula (toothed, chitinous, ribbon part of the mouth). Note also the retracted (curled) foot margins exposing bare shell beneath

Source: Victorian Department of Primary Industries

Healthy abalone; there is no evidence of any protruding mouth parts or foot curl



Source: Victorian Department of Primary Industries

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level (or in the wild) are:

- rapid and high cumulative mortality of up to 90%
- clean (empty) shells on substrate due to predation of moribund and dead abalone
- inability to adhere to the substrate
- inability to right when placed upside down.

Gross pathological signs are:

- swollen and protruding mouth parts
- reduced activity of the pedal muscle
- edges of the foot curled inwards, leading to exposure of clean, shiny shell
- tetany or 'hard foot'
- excessive mucous production
- abnormal spawning and bloating.

Microscopic pathological signs are:

- inflammation and necrosis of neural tissue.

Disease agent

AVG is caused by abalone herpes virus.

Host range

Species known to be susceptible to AVG are listed below.

Common name	Scientific name
Blacklip abalone **a**	*Haliotis rubra*
Diversicolor or jiukong abalone **a**	*Haliotis diversicolor*
Greenlip abalone **a**	*Haliotis laevigata*
Tiger abalone **a**	*Haliotis rubra* × *Haliotis laevigata*

a Naturally susceptible.

Presence in Australia

AVG has been officially reported from Victoria and Tasmania. In Victoria, AVG has been observed in farmed and wild abalone. In Tasmania, the disease has not been observed in wild abalone populations. However, AbHV has been detected in wild abalone and disease has been observed in abalone following capture.

Epidemiology

- AVG affects the nervous system of abalone.
- AVG affects all ages of abalone.
- Horizontal transmission has been demonstrated by:
  - exposing healthy abalone to water containing diseased abalone in the same tank without direct contact between the diseased and healthy abalone
  - placing healthy abalone in water that was previously inhabited by diseased abalone
  - intramuscular injection of healthy abalone with a filtered tissue homogenate from diseased abalone.
- Mortality can occur within 4 days of infection and within 1–2 days following the onset of clinical signs.
- It is unknown whether a carrier state exists.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to AVG.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of infection with abalone herpes virus are summarised in the World Organisation for Animal Health Manual of diagnostic tests for aquatic animals 2011, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information can be found on the following websites:

Tasmanian Department of Primary Industries, Parks, Water and Environment: www.dpiw.tas.gov.au

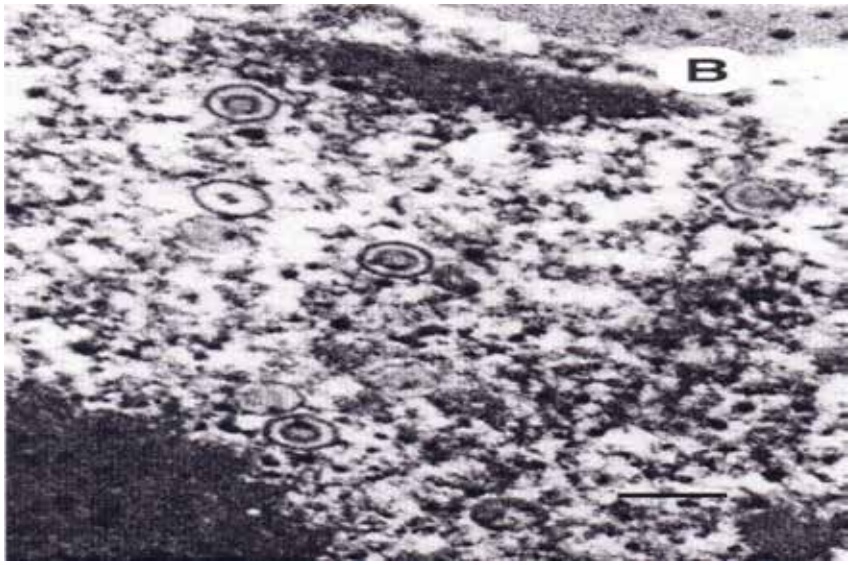
Victorian Department of Primary Industries: www.dpi.vic.gov.au/fisheries/pests-weeds-diseases/abalone-disease

Victorian Abalone Divers Association: www.vada.com.au.

These hyperlinks were correct and functioning at the time of publication.

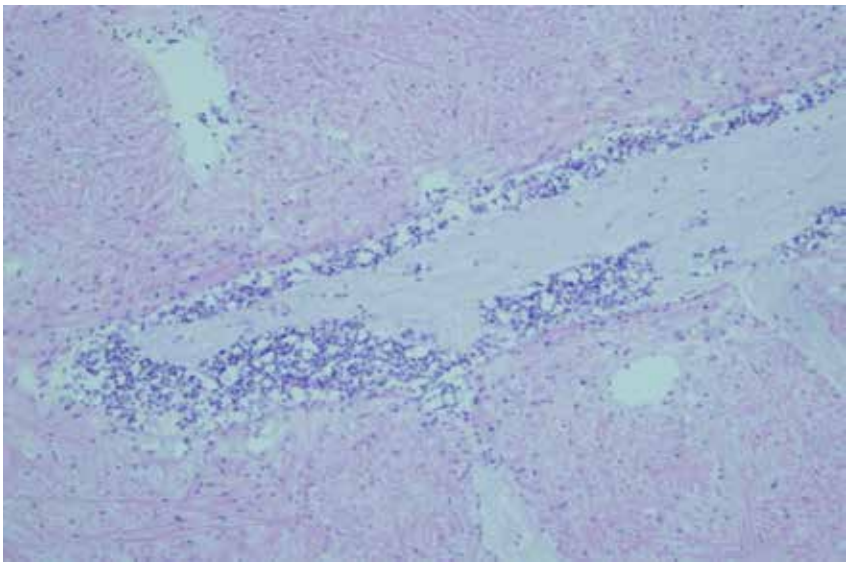
Further images

(1) Electron micrograph of the herpes virus responsible for causing AVG



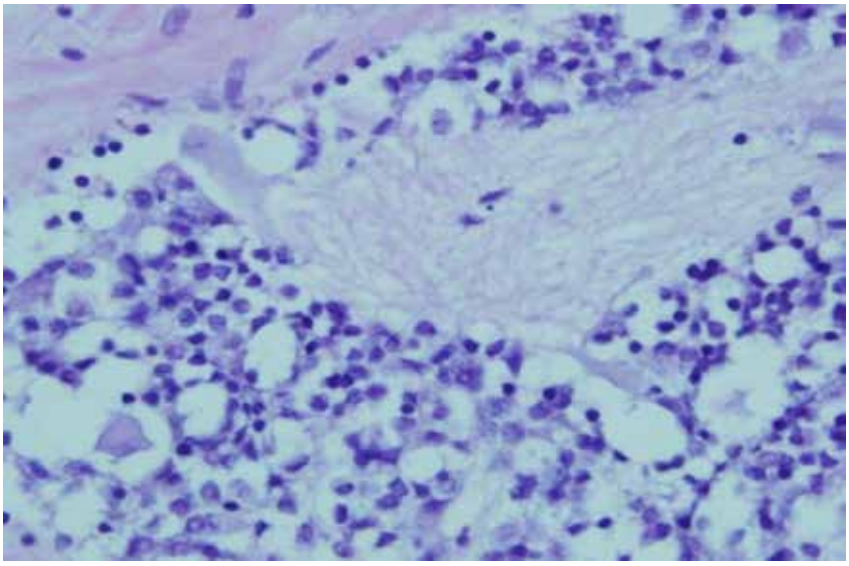
Source: CSIRO Australian Animal Health Laboratory

(2) Electron micrograph of the florid inflammatory response around the ganglia. The blue spots highlight the inflammatory cells typical of infection with AVG



Source: Victorian Department of Primary Industries

(3) Higher resolution image showing inflamed cells surrounding the ganglia



Source: Victorian Department of Primary Industries

Infection with Ostreid herpesvirus-1 microvariant (OsHV-1 µvar)

(Also known as Pacific oyster mortality syndrome [POMS])

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- cumulative mortalities in Pacific oysters approaching 100% within 8–10 days of infection.

Gross pathological signs are:

- cessation of feeding and swimming by larvae, which exhibit velar lesions
- gaping in adults
- pale digestive gland in spat and older oysters.

Microscopic pathological signs are:

- ulcerative and erosive lesions in the connective tissue of mantle, gills, labial palps and digestive tissue
- nuclear hypertrophy, nuclear chromatin margination and pyknosis
- inflammatory changes ranging from mild and localised, to severe and extensive.

Disease agent

Ostreid herpesvirus-1 (OsHV-1) is the only member of the genus *Ostreavirus* (family *Malacoherpesviridae*, order *Herpesvirales*). OsHV-1 µvar is a genotype of this virus.

Host range

Species known to be susceptible to OsHV-1 µvar are listed below.

Multiple bivalve species are known to be susceptible to other genotypes of OsHV-1.

Common name	Scientific name
Pacific oyster **a**	*Crassostrea gigas*

a Naturally susceptible (other species have been shown to be experimentally susceptible).

Presence in Australia

OsHV-1 µvar has been officially reported from only two estuaries in New South Wales.

Epidemiology

- The disease can affect all age groups of oysters, and there may be higher mortality in the younger life stages.
- Higher mortality appears to be associated with higher water temperature and crowding.
- Infected adults may be a source of infection for larvae or spat. However, it is not certain if true vertical transmission occurs. Horizontal transmission has been demonstrated.
- Some adults may survive with subclinical OsHV-1 infections, and act as carriers of the disease.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to infection with OsHV-1 µvar.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of infection with OsHV-1 µvar are summarised in the World Organisation for Animal Health Manual of diagnostic tests for aquatic animals 2011, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information on OsHV-1 µvar can be found on the following websites:

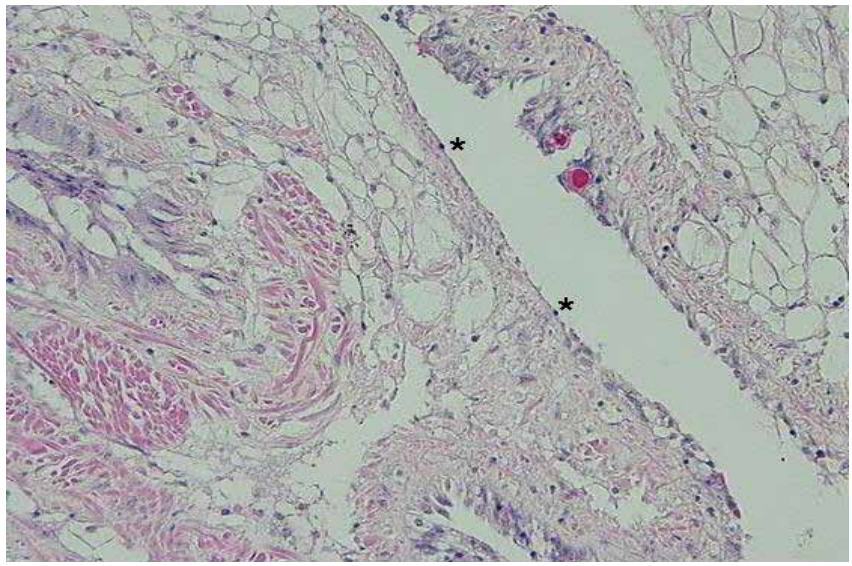
disease pages of Fisheries and Oceans Canada: www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm

EUROPA, the European Commission: ec.europa.eu/food/animal/liveanimals/aquaculture/oyster_mortalities_en.htm.

These hyperlinks were correct and functioning at the time of publication.

Further image

OsHV-1 µvar, 20×. High viral load causing multifocal to coalescing ulceration with attenuation of epithelium and pyknotic nuclei (*)



Source: M Gabor

Iridovirosis (gill necrosis virus)

Exotic disease

Iridovirosis (gill necrosis virus) in oysters. Note scarring of gill tissue

Source: D Alderman

Signs of disease

Important: animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- high mortality.

Gross pathological signs are:

- yellow or green pustules on mantle or adductor muscle
- yellow spots on gills and labial palps that spread as the disease progresses
- spots that increase in size and develop brown centres as the tissue dies, leaving a hole in the gill structure.

Microscopic pathological signs are:

- necrosis of gill or labial palp tissue
- massive haemocytic cellular infiltration around lesions
- basophilic, cytoplasmic inclusions found in most lesions.

Disease agent

Gill necrosis virus is an icosahedral DNA (deoxyribonucleic acid) virus.

Host range

Species known to be susceptible to iridovirosis are listed below.

Common name [a]	Scientific name
European flat oyster	*Ostrea edulis*
Pacific oyster	*Crassostrea gigas*
Portuguese oyster	*Crassostrea angulata*

a All species above are considered naturally susceptible

It is not known if the Sydney rock oyster *(Saccostrea glomerata)* is susceptible.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- A number of iridoviruses causing disease in oysters have been identified, but not all are associated with gill necrosis virus disease, and some affect oysters at different life stages.
- Horizontal transmission occurs directly via the water column through the surface of the gills.
- Little is known about the distribution of the organism responsible for this condition, but molluscan iridoviruses are generally considered to be distributed in oceans worldwide.
- A protist, *Thankatostrea polymorpha* in the phylum Sarcomastigophora, has also been associated with this disease.
- Outbreaks usually occur in spring and sometimes in summer.
- Surviving oysters do not repair perforated gill structures and are potential carriers of the virus.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to iridovirosis (gill necrosis virus) in oysters.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found on the Fisheries and Oceans Canada website at www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm.

This hyperlink was correct and functioning at the time of publication

Exotic disease

Bacterial diseases of molluscs

Infection with *Xenohaliotis californiensis*

(Also known as withering syndrome of abalone)

Abalone on right has withering syndrome. Note the severe atrophy (withering) of foot muscle. Specimen on left is healthy

Source: J Moore

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- reduced feeding
- inability of individuals to right themselves when upside down
- weakness and lethargy (clinging to horizontal surfaces rather than vertical or inverted)
- inability to adhere to the substrate.

Gross pathological signs are:

- wasting of body mass
- retraction of mantle
- atrophy of the foot muscle
- decreased response to tactile stimuli
- diminished reproductive output
- mottling of digestive gland (dark brown with small foci of tan-coloured tissue).

Microscopic pathological signs are:

- presence of intracellular bacteria in the cells of the digestive epithelia
- atrophy of digestive tubules
- increase in connective tissue, inflammation and metaplasia of the digestive gland.

Disease agent

Infection with *Xenohaliotis californiensis* is caused by the intracellular bacterium *X. californiensis* of the family *Rickettsiaceae*.

Host range

Five Haliotis species native to the Californian coast are susceptible to infection with *X. californiensis.* It is assumed that all species of Haliotis are susceptible. There have been no infection experiments on species of abalone that are wild or cultured in Australia, so susceptibility to *X. californiensis* is not confirmed in these species.

Species known to be susceptible to infection with *X. californiensis* are listed below.

Common name	Scientific name
Blacklip abalone **a**	*Haliotis rubra*
European abalone **a**	*Haliotis tuberculata*
Flat abalone	*Haliotis wallalensis*
Green abalone **a**	*Haliotis fulgens*
Japanese abalone	*Haliotis* discus hannai
Pink abalone **a**	*Haliotis corrugata*
Red abalone **a**	*Haliotis rufescens*
Small abalone **a**	*Haliotis diversicolor supertexta*
White abalone **a**	*Haliotis sorenseni*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- The pathogen *X. californiensis* occurs in marine waters.
- The bacterium attacks the lining of the digestive tract, apparently obstructing the production of digestive enzymes. As a result, the abalone starve and expend their own body mass reserves, causing 'withering' of the foot, which impairs their ability to adhere to substrates and makes them vulnerable to predation.
- Abalone not eaten by predators usually die from starvation.
- Susceptibility varies between species of abalone (99% cumulative decline in black abalone and 30% in red abalone since the disease was first observed in 1986).
- Abalone can be infected with the bacterium without developing the disease.
- Transmission is known to occur horizontally, by cohabitation with infected abalone, and is via the faecal–oral route.
- Environmental stressors, such as elevated water temperature, may predispose carriers of the bacterium to disease. Survivors can remain carriers of the bacterium.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to infection with *X. californiensis*.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state/territory agency.

Further reading

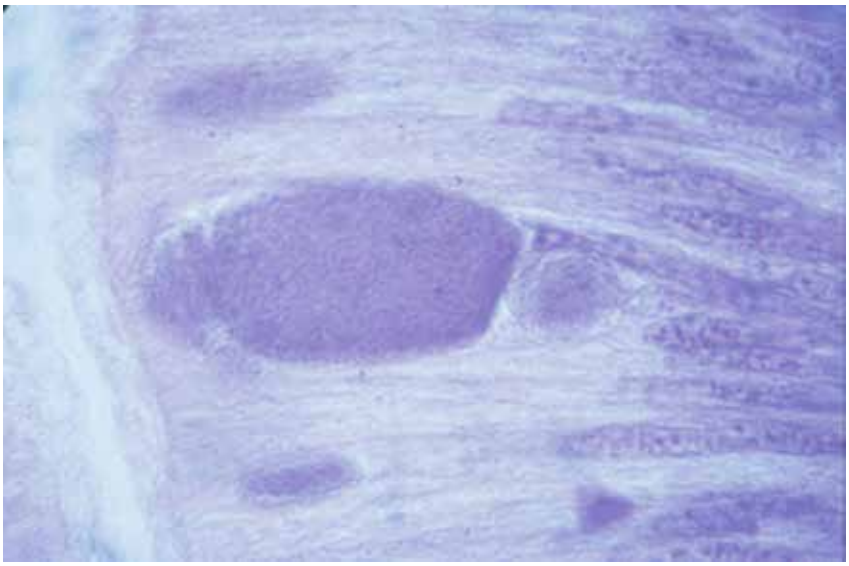
The accepted procedures for a conclusive diagnosis of infection with *X. californiensis* are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information can be found on the Australian Government Department of Agriculture, Fisheries and Forestry website at www.daff.gov.au/animal-plant-health/aquatic/aquavetplan.

These hyperlinks were correct and functioning at the time of publication.

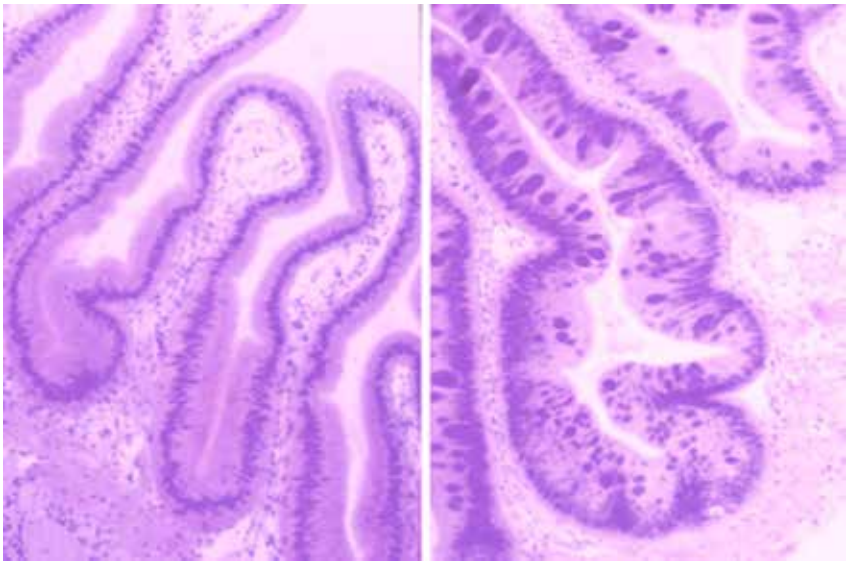
Further images

Close-up of an inclusion in the postoesophagus of a red abalone (*Haliotis rufescens*) positive for infection with *X. californiensis*



Source: J Moore

Postoesophagous tissue from healthy, farmed red abalone (*Haliotis rufescens*) on left; similar tissue on right from an abalone infected with *X. californiensis*, showing basophilic inclusions in epithelial cells, each containing thousands of individual bacteria



Source: J Moore

Parasitic diseases of molluscs

Infection with *Bonamia exitiosa*

New Zealand flat oyster infected with *Bonamia exitiosa*; note typical gaping

Source: B Diggles

New Zealand dredge oyster (*Ostrea chilensis*) infected with *Bonamia exitiosa*

Source: B Jones

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Diseases caused by any of the microcell species are similar, with few or no clinical or gross signs present with light infection. Identification of the Bonamia or Mikrocytos species requires histological laboratory examination and molecular diagnostic techniques.

Disease signs at the farm, tank or pond level are:

- increased mortality.

Gross pathological signs are:

- stunted growth and poor condition
- weakened shell closure, leading to slight gaping
- watery flesh
- algae-covered shell lips after the mantle shrinks and no longer reaches the edges
- deformities of the gill margins.

Infection with species of *Bonamia exitiosa* rarely results in gross pathological signs of disease in oysters; often the only sign is increased mortality.

Microscopic pathological signs are:

- microcell parasites, usually found in or near epithelia
- microcell parasites within haemocytes and connective tissues (especially the gill and mantle)
- basophilic, spherical or ovoid parasites, 2–3 µm in diameter.

Disease agent

B. exitiosa is an intrahaemocytic protist in the phylum Haplosporidia that causes lethal infection of certain oysters. Some evidence suggests that *B. exitiosa* in New Zealand is similar but not identical to the species of Bonamia known to infect southern mud oysters in Australia. Based on current information they are considered to be separate species.

Host range

Species known to be susceptible to infection with B. exitiosa are listed below.

Common name	Scientific name
European flat oyster **a**	*Ostrea edulis*
New Zealand dredge oyster **a**	*Ostrea chilensis*
Southern mud oyster or Australian flat oyster **a**	*Ostrea angasi*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Mortalities can occur all year, with highest prevalence in mid to late summer.
- The disease dynamics of *B. exitiosa* in the New Zealand dredge oyster can be affected by exposure to temperature extremes (below 7 °C or above 26 °C), high salinity (40‰), starvation (prolonged holding in filtered seawater), handling (vigorous stirring four times per day) or heavy coinfection with apicomplexan protists.
- Cohabitation of infected and uninfected oysters in holding tanks appears to facilitate transmission of infection to the uninfected oysters.
- A seasonal pattern of disease has been observed in New Zealand since 1964, with separate epizootics in 1985–1991 and 1998–2003 resulting in cumulative mortality of wild populations of New Zealand dredge oysters of more than 90%.
- Transmission is thought to occur from host to host via infective stages being carried along water currents between oyster beds.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with B. ostreae, *Bonamia* spp. and *Mikrocytos mackini*

There are few or no visual cues to the presence of this disease other than poor condition, shell gaping and increased mortality. Consequently, it is impossible to differentiate between Bonamia species based on gross signs alone; any presumptive diagnosis requires further laboratory examination.

Light microscopy can contribute diagnostic information, but further laboratory examination and molecular diagnostic techniques are required for a definitive diagnosis.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

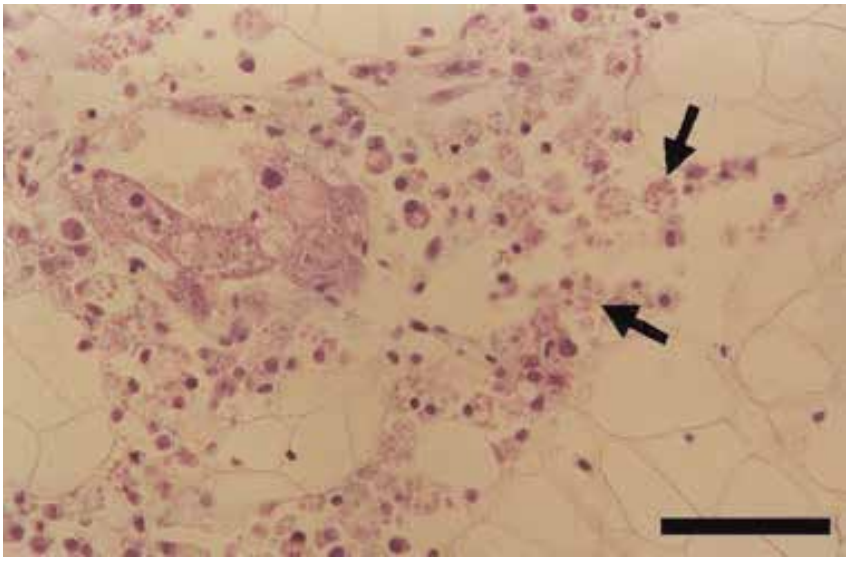
The accepted procedures for a conclusive diagnosis of infection with B. exitiosa are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information can be found on the website of the Sub-Committee on Animal Health Laboratory Standards at www.scahls.org.au/procedures/anzsdps2.

These hyperlinks were correct and functioning at the time of publication.

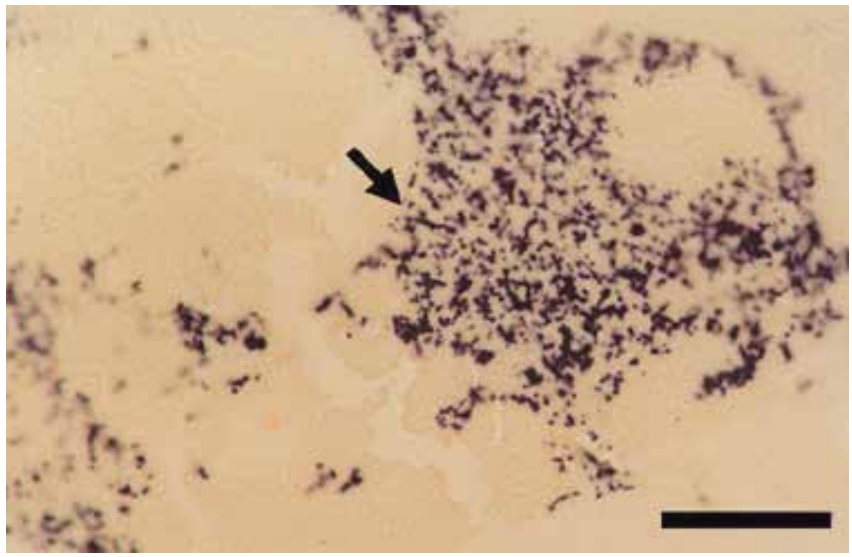
Further images

1) Heavy *B. exitiosa* infection in New Zealand dredge oyster (*Ostrea chilensis*), by histology



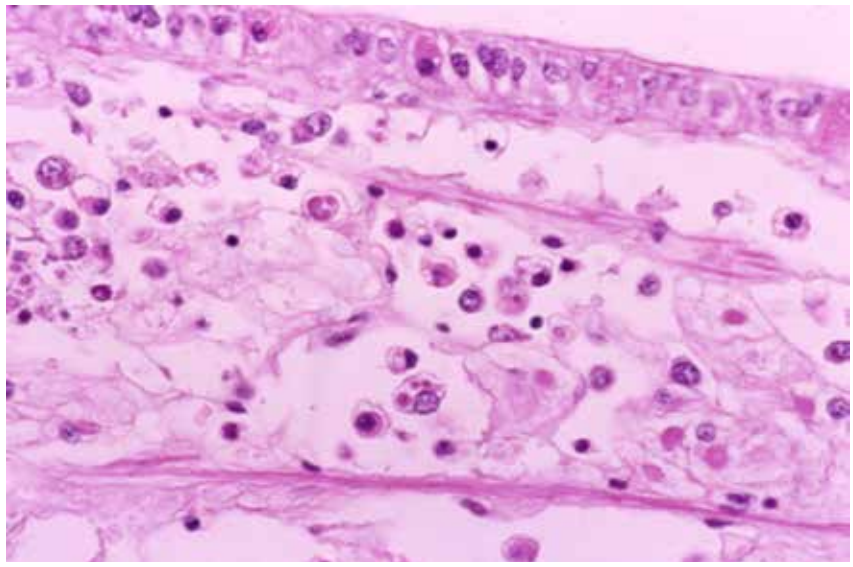
Source: B Diggles

(2) Heavy B. *exitiosa* infection in New Zealand dredge oyster (*Ostrea chilensis*), by in situ hybridisation with a molecular probe



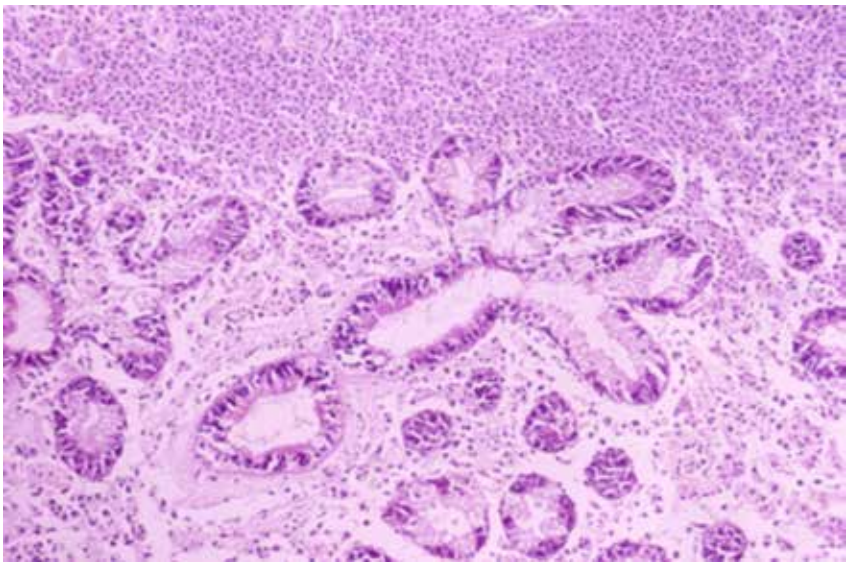
Source: B Diggles

(3) Leydig tissue with circulating haemocytes, many of which have *Bonamia exitiosa* in them (the small pink spheres in the haemocyte cytoplasm, smaller than a nucleus)



Source: B Jones

(4) Inflammatory response in a section through the digestive gland of New Zealand dredge oyster (*Ostrea chilensis*)



Source: B Jones

Exotic disease

Infection with *Bonamia ostreae*
(Also known as bonamiosis)

Oysters infected with *Bonamia ostreae*, illustrating classic gaping

Source: D Alderman

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Diseases caused by any of the microcell species are similar, with few or no clinical or gross signs present with light infection. Identification of the Bonamia or Mikrocytos species requires histological laboratory examination and molecular diagnostic techniques.

Disease signs at the farm, tank or pond level are:

- dead or gaping oysters
- increased mortality.

Gross pathological signs are:

- generally poor condition
- gills appearing eroded
- yellow discolouration of the gills and mantle.

Infection with *Bonamia ostreae* rarely results in gross pathological signs of disease in oysters; often the only sign is increased mortality.

Microscopic pathological signs are:

- dense infiltrations of haemocytes, some containing microcell parasites, in the connective tissue of the gill and mantle, and in the vascular sinuses around the stomach and intestine
- extensive lesions, including perforated ulcers in the connective tissues of the gills, mantle and digestive gland.

Disease agent

B. ostreae is an intrahaemocytic protist belonging to the phylum Haplosporidia.

Host range

Species known to be susceptible to infection with *B. ostreae* are listed below.

Common name	Scientific name
Argentinian flat oyster **a**	*Ostrea puelchana*
European flat oyster **a**	*Ostrea edulis*
New Zealand dredge oyster **a**	*Ostrea chilensis*
Southern mud oyster or Australian flat oyster **a**	*Ostrea angasi*

a Naturally susceptible

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- The critical host age for development of disease appears to be 2 years; however, other age classes are susceptible to infection.
- Significant mortalities usually occur at water temperatures of 12–20 °C.
- Systemic infection of haemocytes effectively starves the oyster of energy required for survival. As it fights the infection, the animal eventually dies from exhaustion and starvation.
- Some studies suggest that prevalence and intensity of infection increase during late winter and autumn, but the disease may occur at all times of the year.
- The pre-patent period is up to 5 months.
- Transmission of the parasite can occur directly from host to host and indirectly between oyster beds via the water.
- The disease is thought to have spread from California to Europe by human movement of infected oysters.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with *B. exitiosa*, *Bonamia* spp. and *Mikrocytos mackini*

There are few or no visual cues to the presence of this disease other than poor condition, shell gaping and increased mortality. Consequently, it is impossible to differentiate between Bonamia species based on gross signs alone; any presumptive diagnosis requires further laboratory examination.

Light microscopy can contribute diagnostic information, but further laboratory examination and molecular diagnostic techniques are required for a definitive diagnosis.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of infection with B. ostreae are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information can be found at:

- the disease pages of Fisheries and Oceans Canada: www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/pages/bonostoy-eng.htm
- the Sub-Committee on Animal Health Laboratory Standards (SCAHLS) website: www.scahls.org.au/procedures/anzsdps2.

These hyperlinks were correct and functioning at the time of publication.v

Infection with *Bonamia spp*
(Also known as bonamiosis)

Infection with *Bonamia* sp. in Australian flat oysters (*Ostrea angasi*); note the smaller size of infected oysters on right compared with normal ones on left

Source: CSIRO Australian Animal Health Laboratory

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Diseases caused by any of the microcell species are similar, with few or no clinical or gross signs present with light infection. Identification of the *Bonamia or Mikrocytos* species requires histological laboratory examination and molecular diagnostic techniques.

Disease signs at the farm, tank or pond level are:

- increased mortality.

Gross pathological signs are:

- stunted growth and poor condition
- weakened shell closure, leading to slight gaping
- watery flesh
- algae-covered shell lips after the mantle shrinks and no longer reaches the edges
- deformities to the gill margins.

Infection with *Bonamia* spp. rarely results in gross pathological signs of disease in oysters; often the only visual cue is increased mortality.

Microscopic pathological signs are:

- microcell parasites in or near epithelia—may be in very low numbers
- in Australian flat oyster (*Ostrea angasi*)apparently low numbers of parasites causing, massive focal haemocyte aggregation with necrotic foci.

Disease agent

Bonamia species are intrahaemocytic protists in the phylum Haplosporidia that cause lethal infection of certain oysters. This disease listing includes *B. perspora* and unidentified *Bonamia* spp., but excludes *B. exitiosa* and *B. ostreae*, which are addressed in separate disease listings.

Host range

Species known to be susceptible to infection with *Bonamia spp.* are listed below.

Common name	Scientific name
Argentinian flat oyster **a**	*Ostrea puelchana*
Crested oyster **a**	*Ostrea equestris*
New Zealand dredge oyster **a**	*Ostrea chilensis*
Southern mud oyster or Australian flat oyster **a**	*Ostrea angasi*
Suminoe oyster **a**	*Crassostrea ariakensis*

a Naturally susceptible

Presence in Australia

Infection with *Bonamia spp.* has been officially reported from New South Wales, South Australia, Tasmania, Victoria and Western Australia in flat oysters.

Epidemiology

- Mortalities can occur all year but usually happen in spring and summer.
- Significant mortalities usually occur at water temperatures of 12–20 °C.
- Animals may be infected without mortalities; the factors that contribute to outbreaks are unclear.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with *B. ostreae*, *B. exitiosa* and *Mikrocytos mackini*

There are few or no visual cues to the presence of this disease other than poor condition, shell gaping and increased mortality. Consequently, it is impossible to differentiate between Bonamia and Mikrocytos species based on gross signs alone; any presumptive diagnosis requires further laboratory examination.

Light microscopy can contribute diagnostic information, but further laboratory examination and molecular diagnostic techniques are required for a definitive diagnosis.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found at the following websites:

Centre for Environment, Fisheries and Aquaculture Science (Cefas) International Database on Aquatic Animal Disease (IDAAD): www.cefas.defra.gov.uk/idaad/disocclist.aspx

disease pages of Fisheries and Oceans Canada: www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm

Sub-Committee on Animal Health Laboratory Standards: www.scahls.org.au/procedures/anzsdps2.

These hyperlinks were correct and functioning at the time of publication.

Exotic disease

Infection with *Marteilia refringens*
(Also known as Aber disease, digestive gland disease or marteiliosis)

Healthy flat oyster

Source: French Research Institute for Exploration of the Sea—IFREMER

Flat oyster infected with *Marteilia refringens*

Source: French Research Institute for Exploration of the Sea—IFREMER

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- high mortality
- reduced growth rate
- gaping.

Gross pathological signs are:

- poor condition and emaciation
- pale digestive gland
- inhibited gonad development.

Microscopic pathological signs are:

- tissue necrosis.

Disease agent

Marteilia refringens is a protozoan parasite (phylum Cercozoa, order Paramyxida) that affects the digestive system of multiple bivalve species, including oysters, mussels, cockles and clams.

Host range

Species known to be susceptible to infection with *M. refringens* are listed below.

Common name	Scientific name
American oyster **a**	*Crassostrea virginica*
Argentinian flat oyster	*Ostrea puelchana*
Asiatic oyster **a**	*Ostrea denselammellosa*
Blue mussel **a**	*Mytilus edulis*
Calico scallop	*Argopecten gibbus*
Common cockle **a**	*Cardium edule*
Dwarf oyster	*Ostrea stentina*
European flat oyster **a**	*Ostrea edulis*
European razor clam **a**	*Solen marginatus*
Mediterranean mussel **a**	*Mytilus galloprovincialis*
New Zealand dredge oyster **a**	*Ostrea chilensis*
Olympia oyster **a**	*Ostrea conchaphila*
Pacific oyster	*Crassostrea gigas*
Rock oyster **a**	*Saccostrea cucullata*
Small brown mussel	*Xenostrobus securis*
Southern mud oyster or Australian flat oyster	*Ostrea angasi*
Striped venus clam **a**	*Chamelea gallina*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- *M. refringens* infections result in high cumulative mortality (50–90%), associated with sporulation of the parasite in the epithelial cells of the digestive tubules. Highest cumulative mortalities usually occur during summer and autumn.
- Earlier stages of sporulation occur in epithelia of the digestive ducts and possibly the gills.
- Several intermediate hosts or a free-living stage are thought to be required during the lifecycle of *M. refringens*. The copepod *Paracartia grani* is one intermediate host and may be involved in transmission of *M. refringens* between bivalves.
- *M. refringens* can exist in a carrier state in some oysters, which can be potential reservoirs of infection.
- Factors triggering a pathogenic host response are not clearly established, but may include environmental stresses and differences in susceptibility to disease between stock.
- The temperature threshold for parasite sporulation and transmission is 17 °C; however, this is thought to vary with other environmental factors.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with *Marteilia sydneyi*

The clinical signs of infection with *M. refringens* are almost identical to those of infection with other paramyxea (i.e. high mortalities associated with colourless and translucent tissues, poor condition, pale digestive gland and a shrunken body). Therefore, any presumptive diagnosis requires further laboratory examination.

Light microscopy can contribute diagnostic information, but further laboratory examination and molecular diagnostic techniques are required for a definitive diagnosis.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

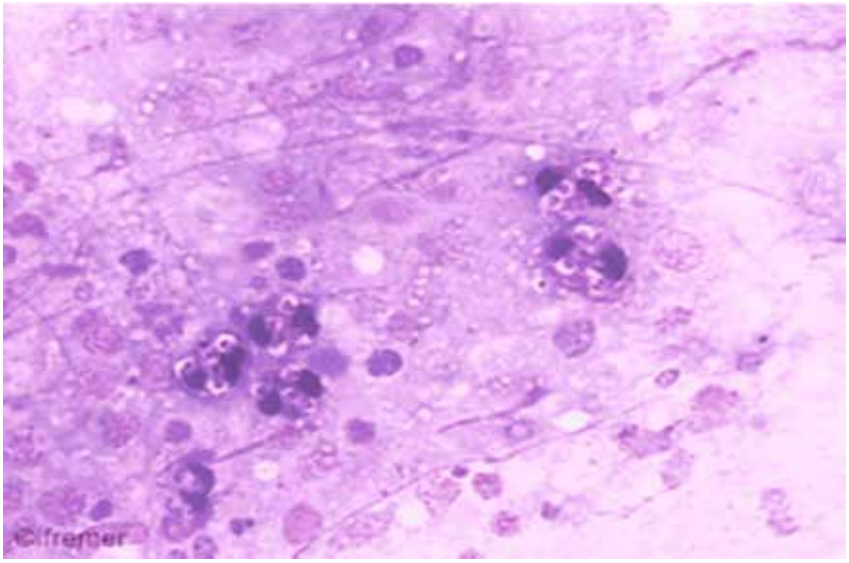
The accepted procedures for a conclusive diagnosis of infection with M. refringens are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information is also available on the disease pages of Fisheries and Oceans Canada at www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm.

These hyperlinks were correct and functioning at the time of publication.

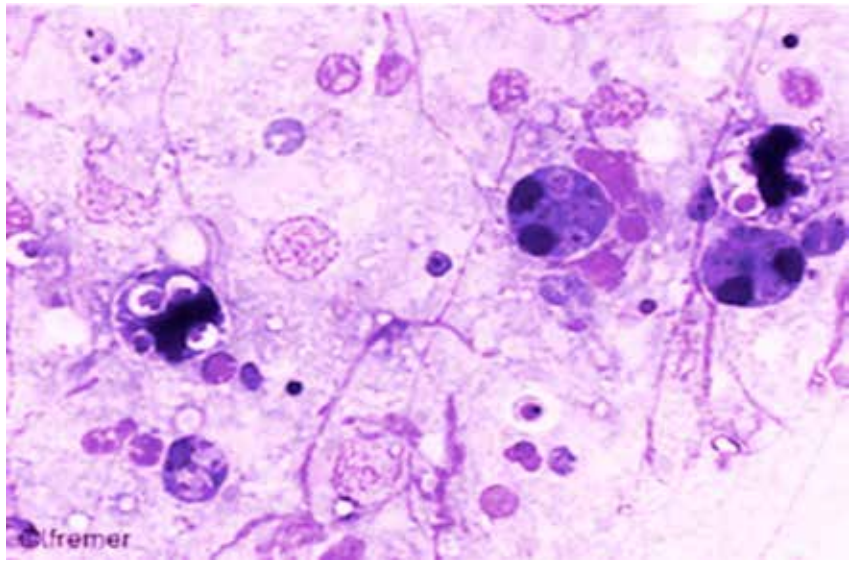
Further images

(1) *Marteilia refringens* tissue imprint (80×)



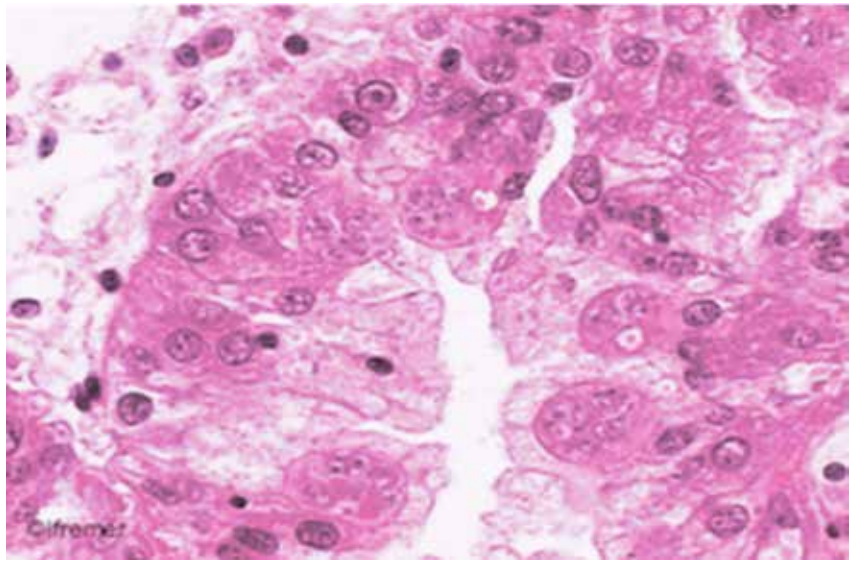
Source: French Research Institute for Exploration of the Sea—IFREMER

(1) *Marteilia refringens* tissue imprint (120×)



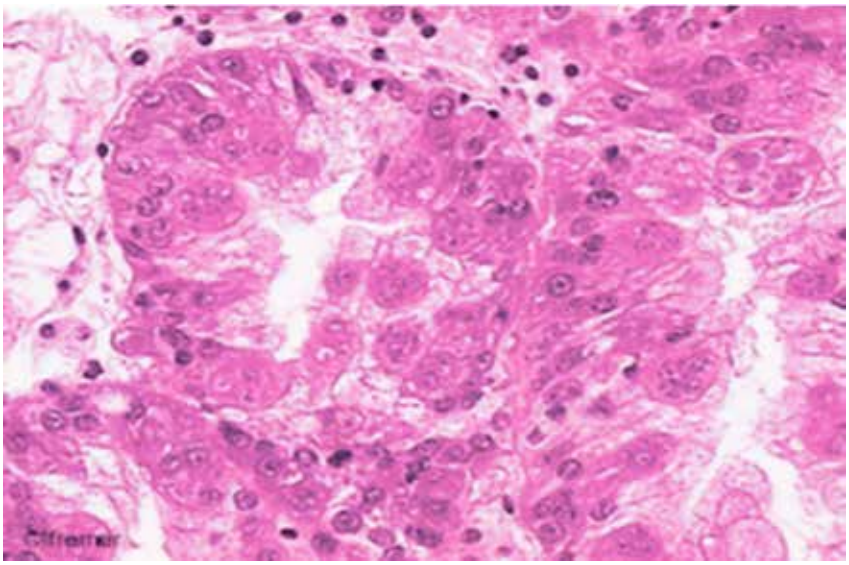
Source: French Research Institute for Exploration of the Sea—IFREMER

(1) *Marteilia refringens* tissue imprint (120×)



Source: French Research Institute for Exploration of the Sea—IFREMER

(1) *Marteilia refringens* tissue imprint (80×)



Source: French Research Institute for Exploration of the Sea—IFREMER

Infection with *Marteilia sydneyi*
(Also known as QX disease and marteiliosis)

Infected Sydney rock oyster (*Saccostrea glomerata*) on right, showing a yellowish, watery body ('pale sick'); Sydney rock oyster on left is normal

Source: R Adlard

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- high cumulative mortalities of up to 100%.

Gross pathological signs are:

- shrunken body
- generally poor condition
- death within 60 days of infection
- colourless and translucent tissues because the gonad is completely resorbed
- digestive gland (usually a deep green or brown colour) becomes pale yellow–brown.

Microscopic pathological signs are:

- focal haemocytic inflammation of the gills
- epithelial and connective tissue hyperplasia of the gill epithelium, with fusion of gill filaments.

Disease agent

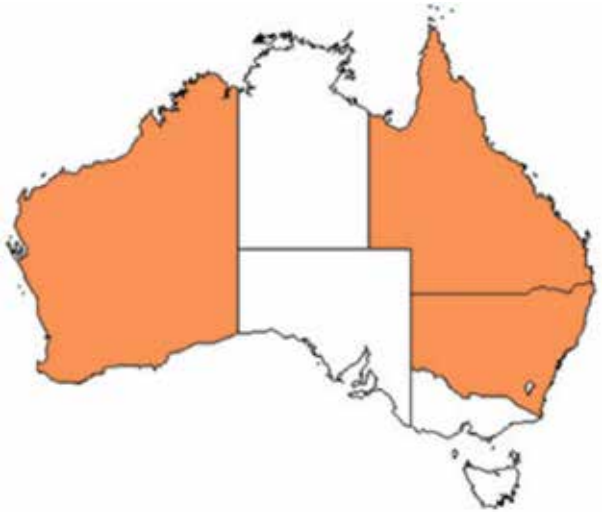
Marteilia sydneyi is a protozoan parasite of the phylum Paramyxea.

Host range

Species known to be susceptible to infection with *M. sydneyi* are listed below.

Common name	Scientific name
Sydney rock oyster	*Saccostrea glomerata*

Presence in Australia



Infection with *M. sydneyi* is responsible for losses in the Sydney rock oyster industry on the east coast of Australia. The parasite and disease are present in Queensland and New South Wales. A related species of *Marteilia* has been associated with disease in the coral rock oyster (*Saccostrea cucullata*) in Western Australia and was officially reported in 1994. It has not been reported since.

Epidemiology

- Despite the presence of the pathogen, disease has not been observed in some eastern Australian estuaries.
- Oysters may be subjected to infection for a period of only 2 weeks per year.
- Outbreaks occur in summer and autumn. Warm temperatures favour parasite development, leading to greatest mortality at the end of summer.
- The disease is associated with low salinity and high water temperature.
- Transmission is horizontal; the pathogen passes from the environment into the epithelium of the palps and gills where it proliferates without forming spores.
- The life cycle of *M. sydneyi* is not fully understood. Intermediate hosts are required to complete the life cycle; most recent studies confirm that a benthic polychaete is an intermediate host.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with *Marteilia refringens*

The clinical signs of infection with *M. sydneyi* are almost identical to those of infection with Haplosporidia (i.e. high mortalities associated with colourless and translucent tissues, poor condition, pale digestive gland and a shrunken body). Therefore, any presumptive diagnosis requires further laboratory examination.

Light microscopy can contribute diagnostic information, but further laboratory examination and molecular diagnostic techniques are required for a definitive diagnosis.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found on the following websites:

New South Wales Department of Primary Industries: www.dpi.nsw.gov.au/fisheries/pests-diseases/animal-health/aquaculture/qx-oyster-disease

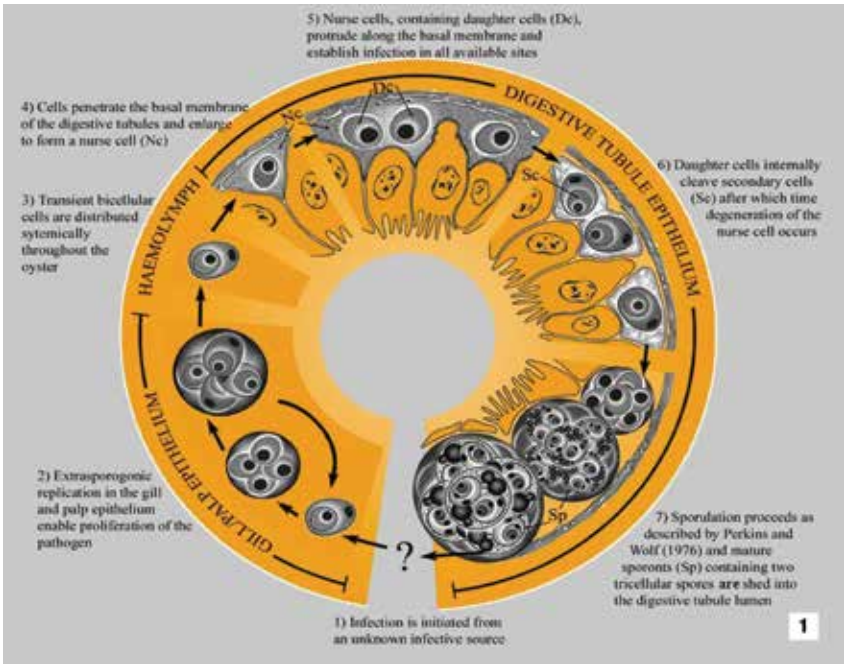
Department of Fisheries, Western Australia: www.fish.wa.gov.au/docs/pub/FHSlideofQuarter/200609.php?0408

Disease pages of Fisheries and Oceans Canada: www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm.

These hyperlinks were correct and functioning at the time of publication.

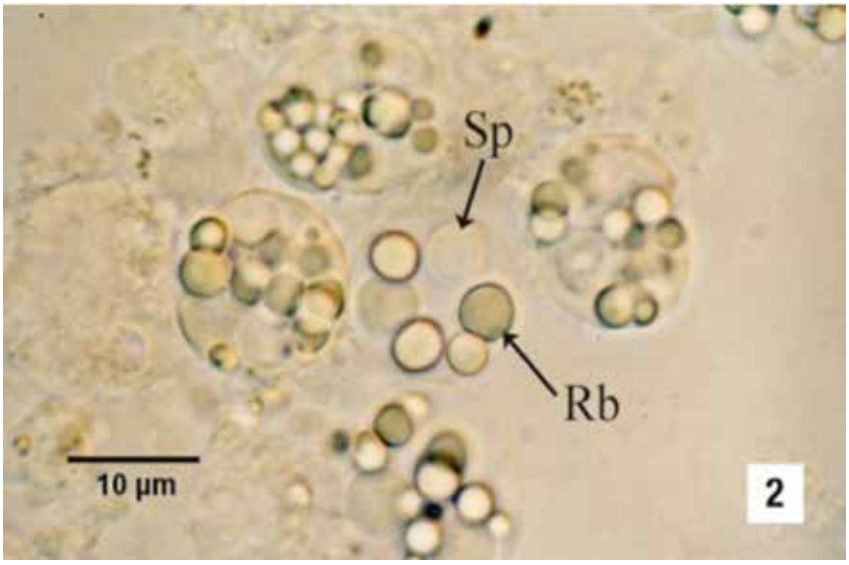
Further images

(1) Hypothetical development of *Marteilia sydneyi* in Sydney rock oyster (Saccostrea glomerata)



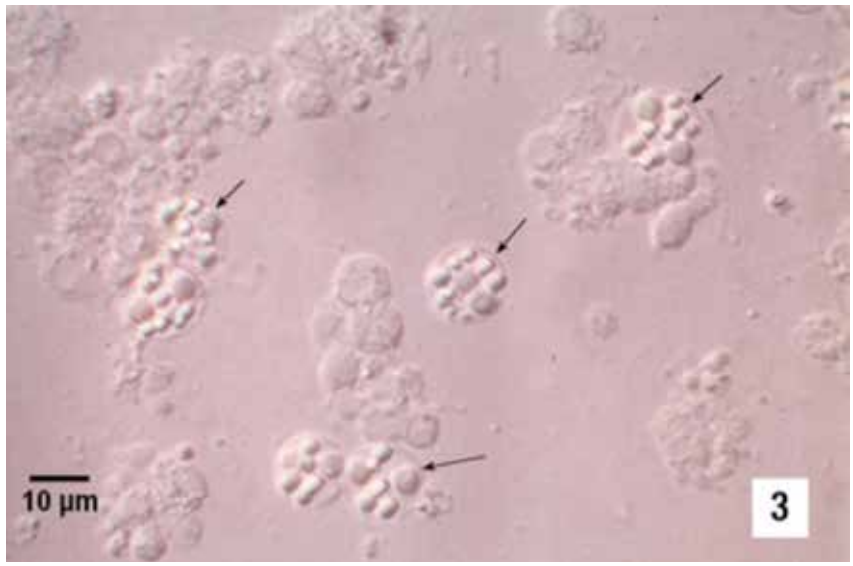
Source: S Kleeman

(2) Sporonts of *Marteilia sydneyi* containing refractile bodies (Rb) and spores (Sp)



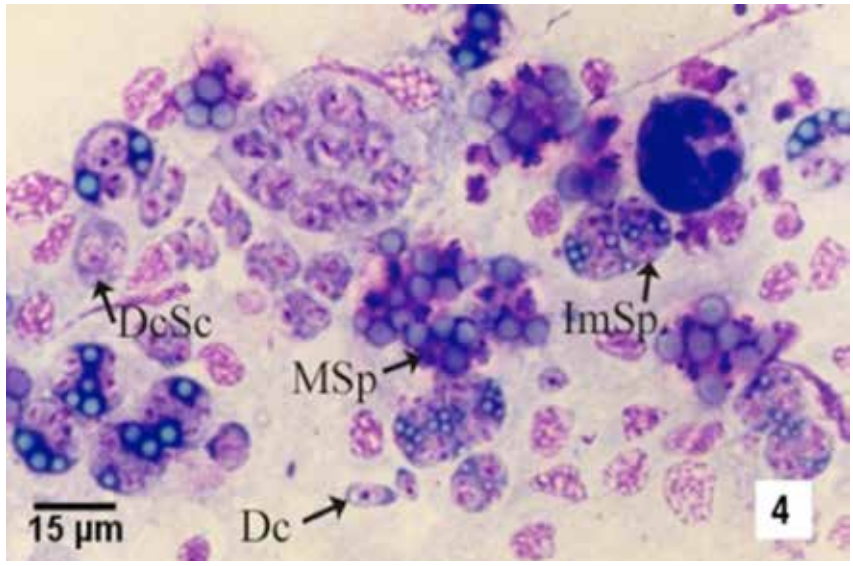
Source: S Kleeman

(3) Sporonts of *Marteilia sydneyi* (arrows) viewed under interference contrast optics



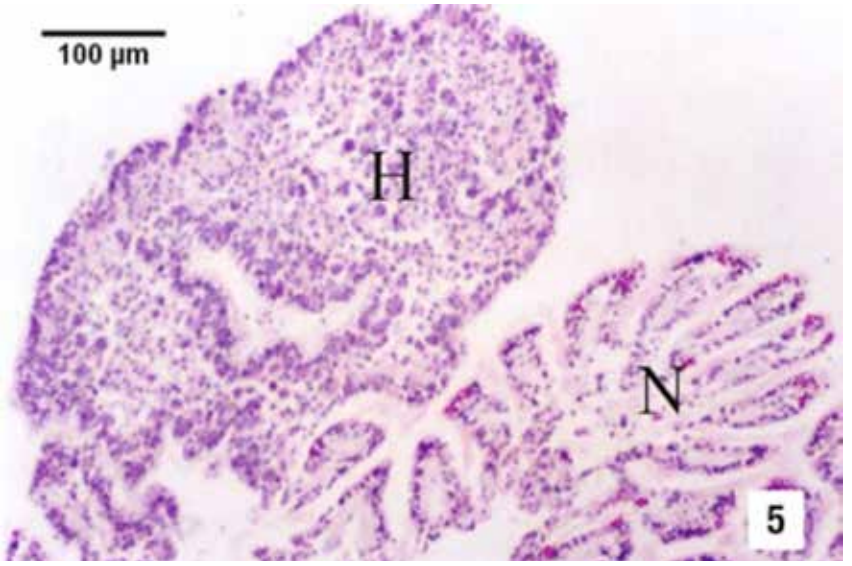
Source: S Kleeman

(4) Hemacolor (Merck)-stained tissue imprint of the digestive gland of Sydney rock oyster (*Saccostrea glomerata*) infected with *Marteilia sydneyi*, showing various life cycle stages, including daughter cells (Dc), daughter cells containing secondary cells (DcSc), immature sporonts (ImSp) and mature sporonts (MSp). Note that the various stages observed are often ruptured from their enclosing cells (i.e. the nurse cells or sporangiosori).



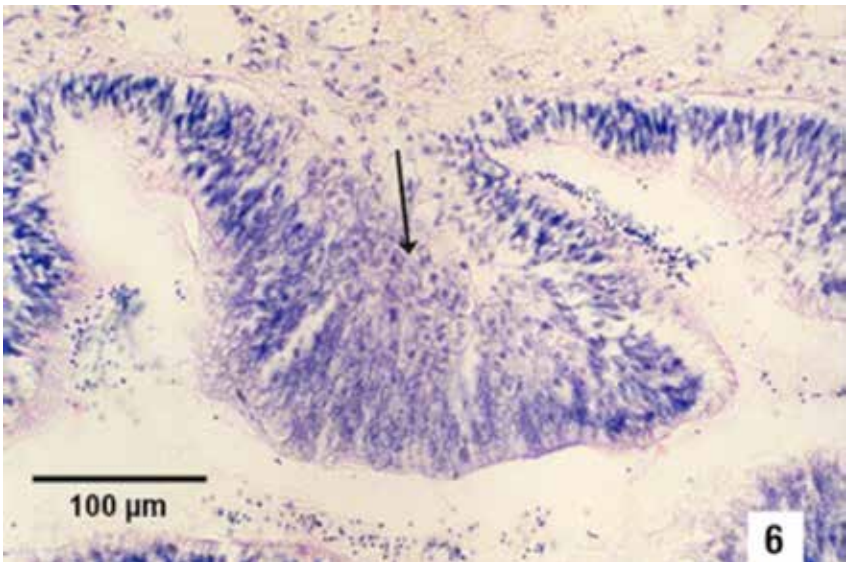
Source: S Kleeman

(5) Oyster reaction to the presence of numerous extrasporogonic stages in the epithelium of the gills consisting of epithelial and connective tissue hyperplasia (H), and fusion of filaments, in contrast with relatively normal-looking gill tissue (N)



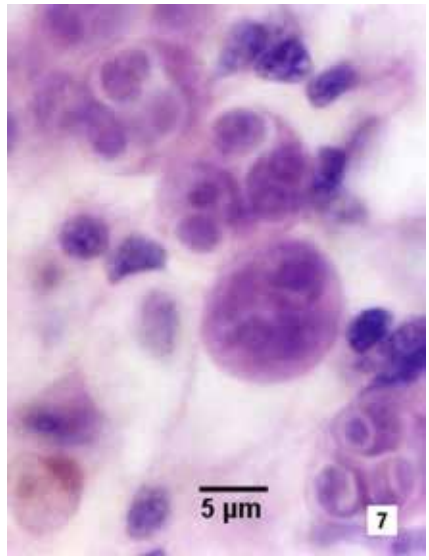
Source: S Kleeman

(6) Replicating stages in the palp epithelium; note the hypertrophy of the epithelial cells in the presence of proliferating parasites (arrow) in infected areas



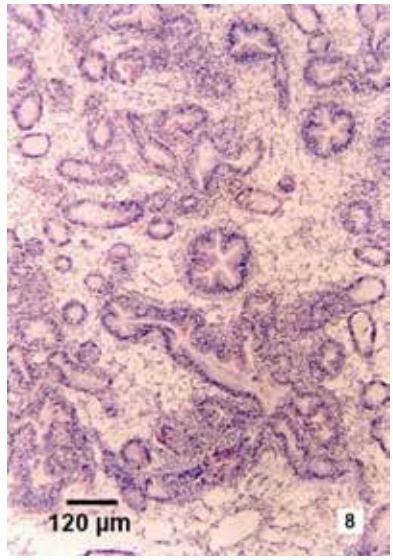
Source: S Kleeman

(7) Higher magnification of extrasporogonic stages in the epithelium of the gills (see phase 2 in Figure 1)



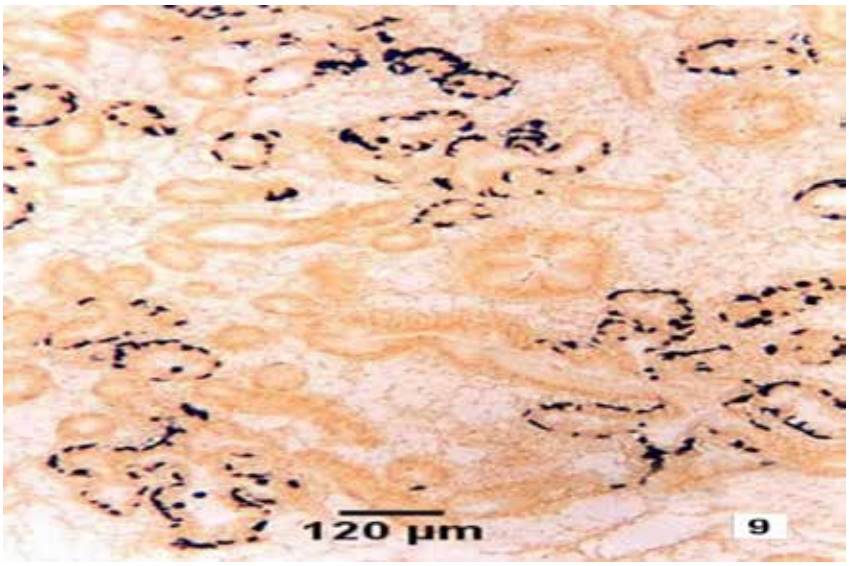
Source: S Kleeman

(8) Haematoxylin and eosin stained section, showing haemocytic infiltration of the connective tissue surrounding infected digestive gland tubules



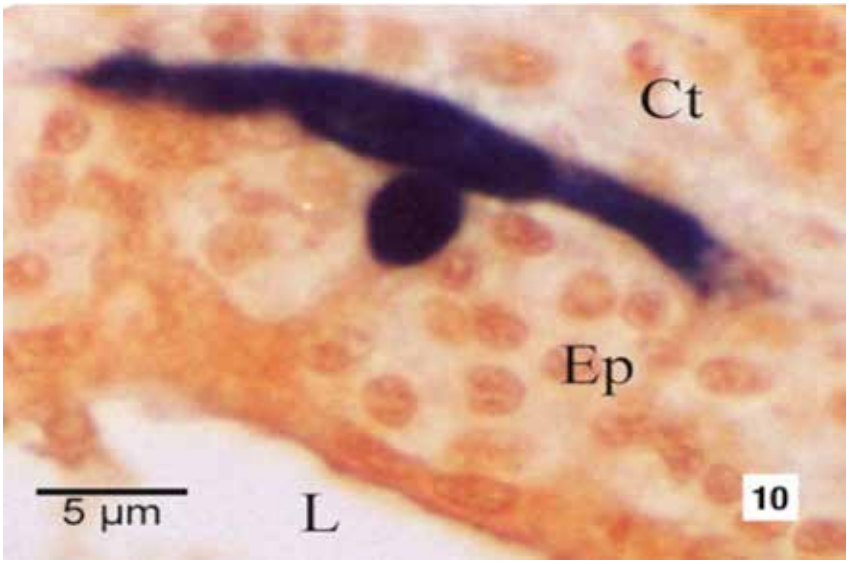
Source: S Kleeman

(9) Tissue section, showing the location of presporulating nurse cell stages (stained black) in digestive gland tubule epithelia



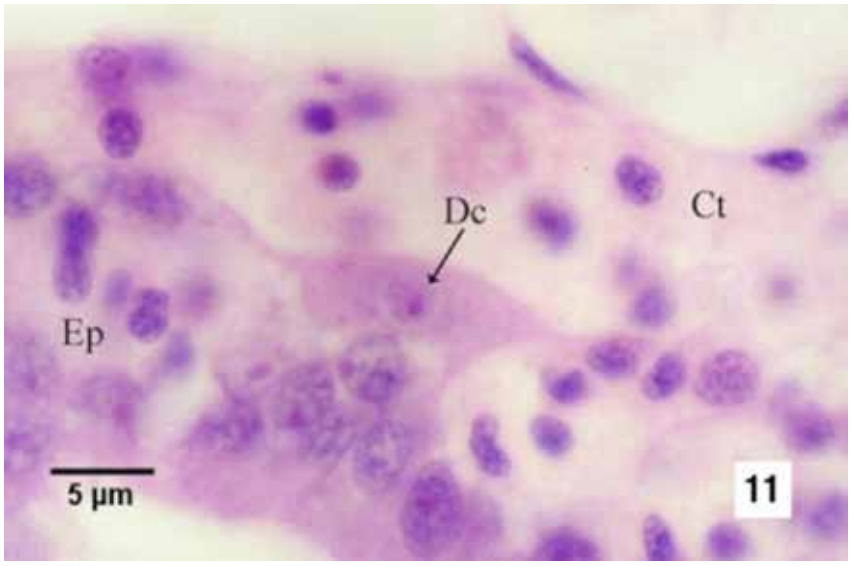
Source: S Kleeman

(10) Nurse cell (stained black by in situ hybridisation) demonstrating the extent of the pseudopodial extensions along the basal membrane of the digestive tubule epithelium (Ep). This feature is not evident with haematoxylin and eosin staining. Other labelled features are the connective tissue (Ct) that surrounds the tubule and the lumen (L) of the tubule.



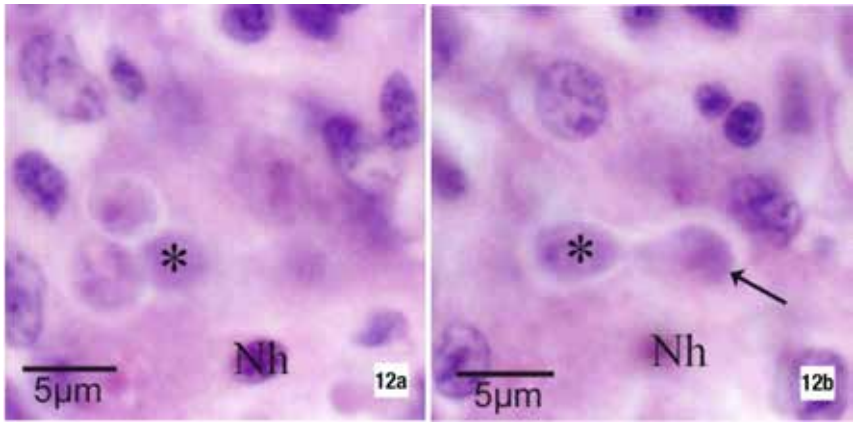
Source: S Kleeman

(11) Nurse cell containing one daughter cell (Dc) along the basal membrane of the tubule between the connective tissue (Ct) surrounding the tubules and the tubule epithelium (Ep)



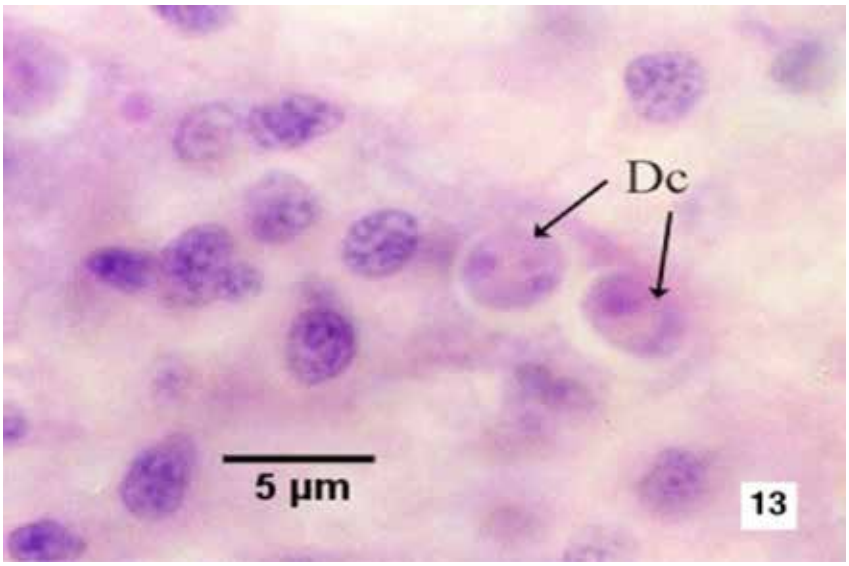
Source: S Kleeman

(12a & b) The same tissue section but at different focal planes, demonstrating the budding of a daughter cell (arrow in Figure 12b) within the nurse cell. An asterisk marks the same daughter cell, and Nh denotes the same host cell nucleus in each figure. There are two additional daughter cells within the nurse cell (Figure 12a).



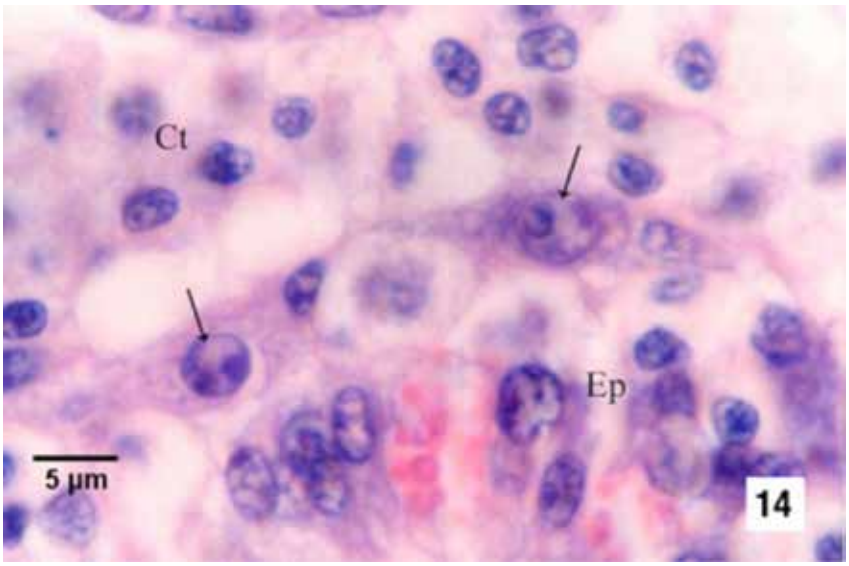
Source: S Kleeman

(13) Nurse cell containing two daughter cells (Dc, see phase 5 in Figure 1)



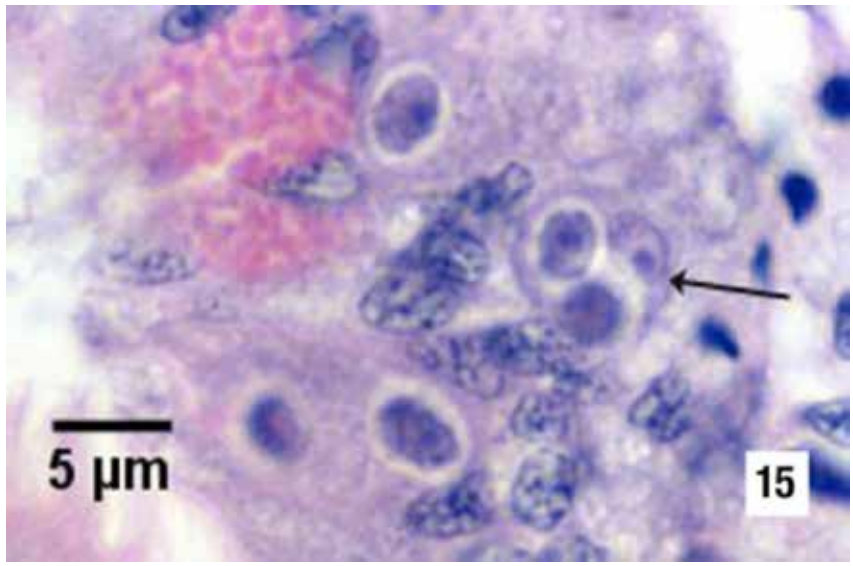
Source: S Kleeman

(14) Nurse cells containing bicellular daughter cells (arrows) along the basal membrane between the tubule epithelium (Ep) and the connective tissue that contains many infiltrating haemocytes (see phase 6 in Figure 1)



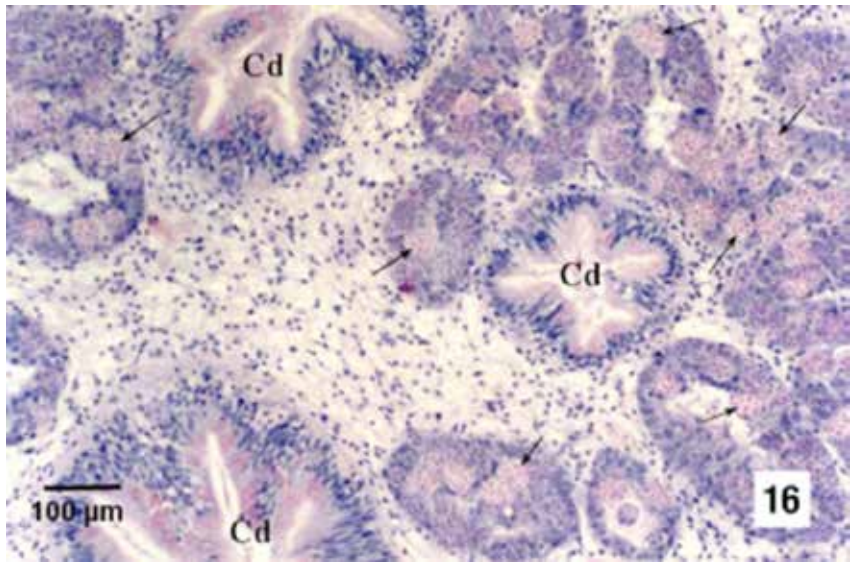
Source: S Kleeman

(15) Primary cell (arrow) containing two secondary cells (sporont primordia) just before sporulation (see initiation of phase 7 in Figure 1)



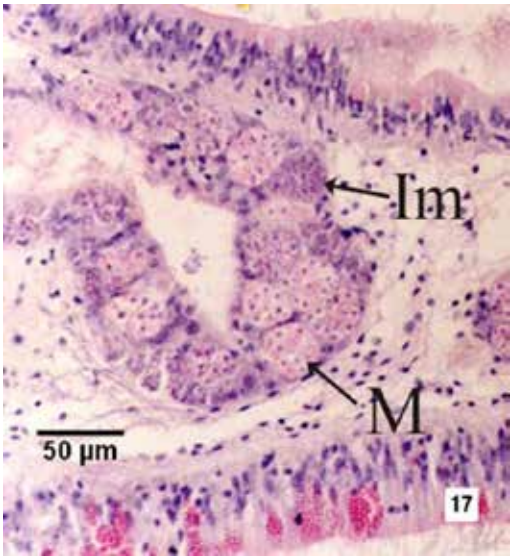
Source: S Kleeman

(16) Numerous sporulating stages (arrows) in the digestive gland tubules; note that sporulation does not occur in the ciliated ducts (Cd) of the digestive gland



Source: S Kleeman

(17) Immature sporonts (Im) and mature sporonts (M) within sporangiosori in a digestive gland tubule; note that the epithelium of the tubule is almost completely replaced by *Marteilia sydneyi*



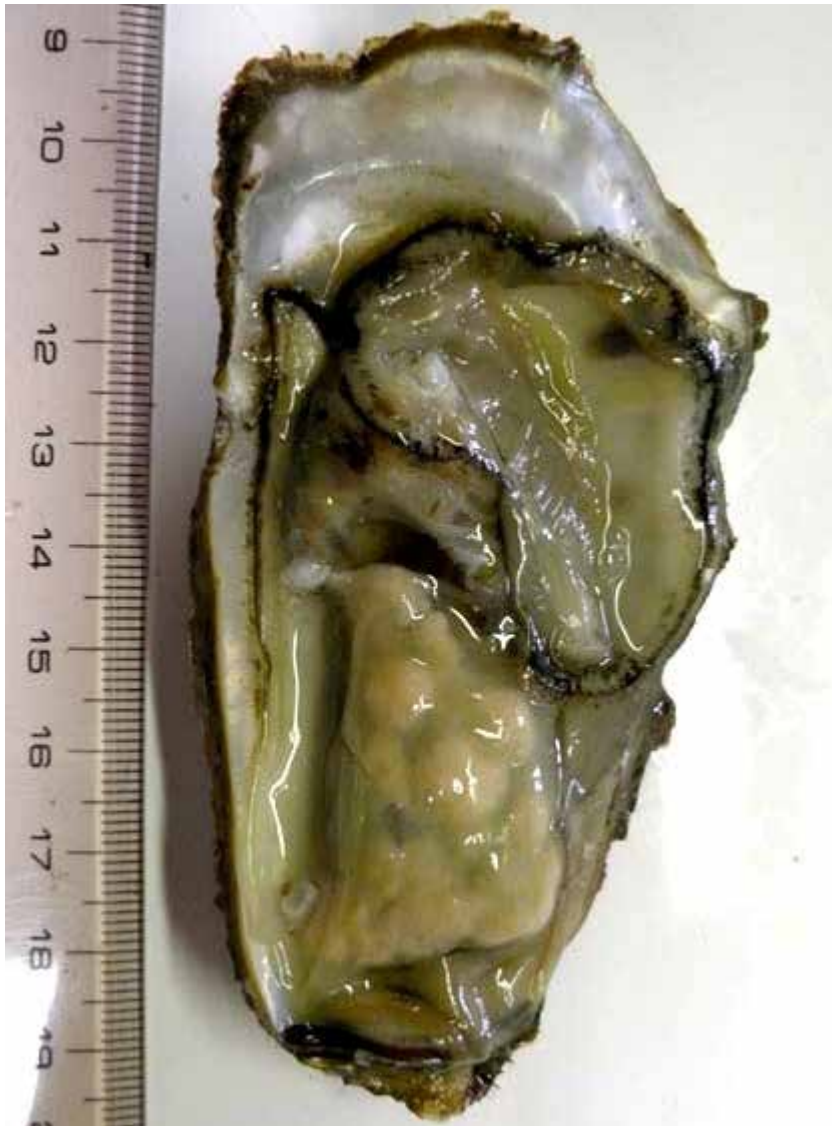
Source: S Kleeman

Exotic disease

Infection with *Marteilioides chungmuensis*

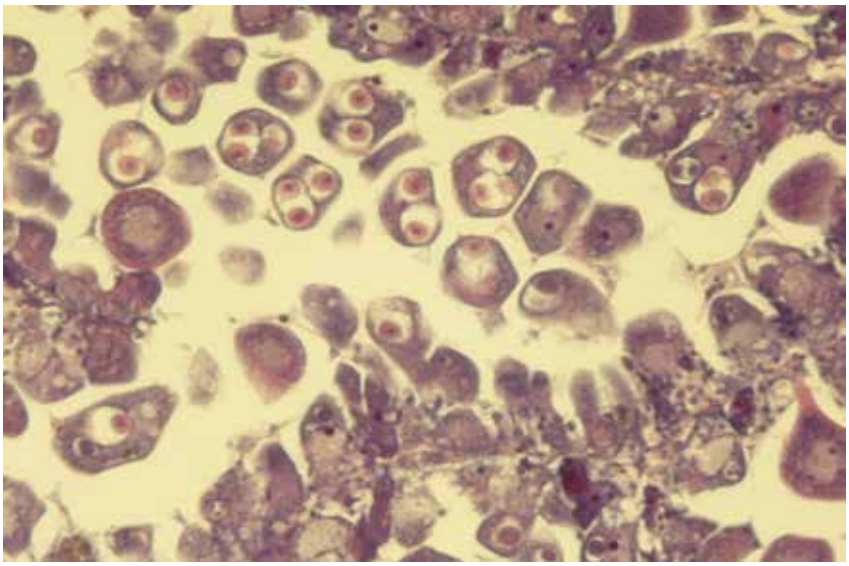
(Also known as marteilioidosis)

Pacific oyster (*Crassostrea gigas*) infected with *Marteilioides chungmuensis*



Source: N Itoh

Histological section showing intracellular (within oocytes) *Marteilioides chungmuensis*



Source: N Itoh

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- spawning failure
- high mortalities.

Gross pathological signs are:

- visible distension of the mantle surface due to infected eggs retained within the follicle
- nodule-like structures on the gonad surface.

Microscopic pathological signs are:

- parasites within oocytes.

Disease agent

Marteilioides chungmuensis is a protozoan parasite of the phylum Paramyxea, and is responsible for infection of Pacific oyster oocytes.

Host range

Species known to be susceptible to infection with *M. chungmuensis* are listed below.

Common name	Scientific name
Pacific oyster **a**	*Crassostrea gigas*
Iwagaki oyster	*Crassostrea nippona*

a Naturally susceptible

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- *M. chungmuensis* infects the cytoplasm of mature oocytes and can affect a substantial proportion of eggs.
- Prolonged spawning activity of infected oysters has been observed, resulting in nutritional wasting and mortality.
- Prevalence of infection increases during spawning in summer and decreases after spawning in winter.
- Infected oysters lose their marketability, due to their abnormal appearance.
- Mode of transmission is unknown; however, intermediate hosts may be involved in the life cycle of the disease.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to infection with *M. chungmuensis*.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

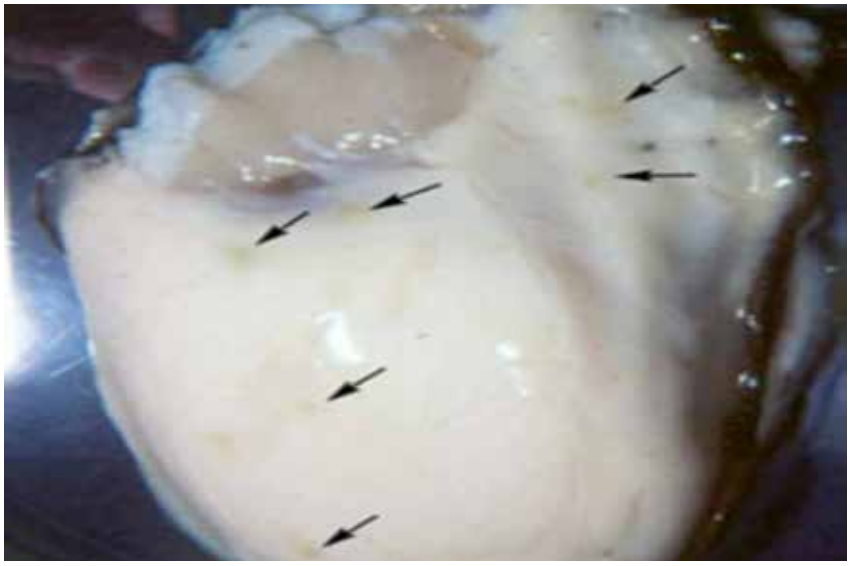
Further information can be found on the disease pages of Fisheries and Oceans Canada at www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm.

This hyperlink was correct and functioning at the time of publication

Infection with Mikrocytos mackini

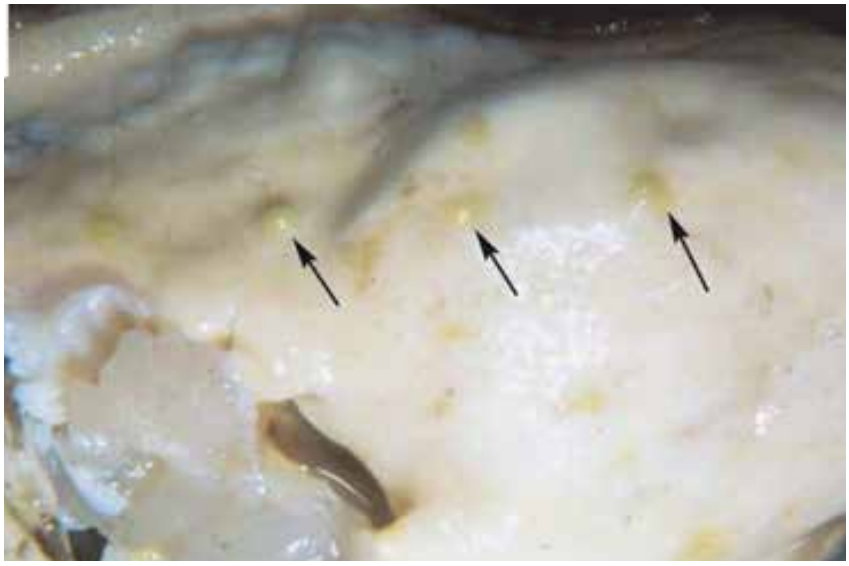
Exotic disease

Pacific oyster (*Crassostrea gigas*) removed from shell and showing lesions (arrowed) characteristic of infection with *Mikrocytos mackini*.



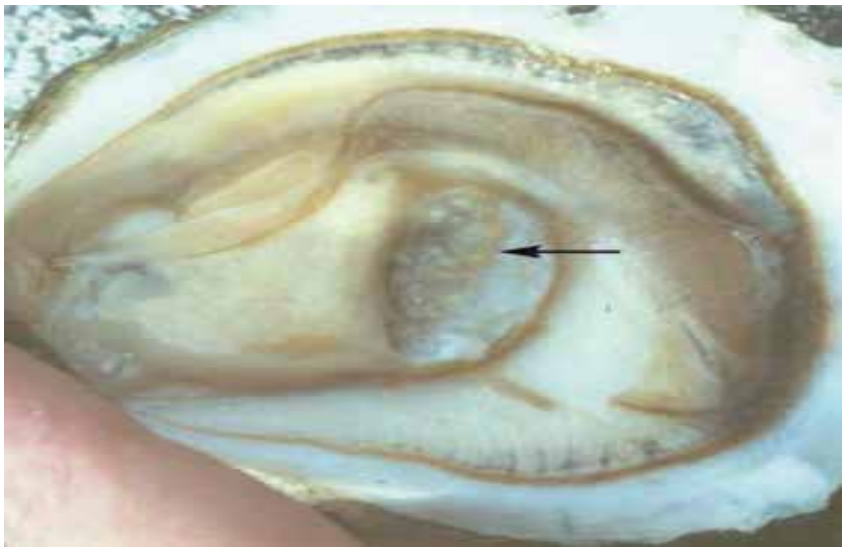
Source: S Bower

European flat oyster (*Ostrea* edulis), with top valve removed, showing numerous lesions in the adductor muscle (arrowed) caused by *Mikrocytos mackini* (experimental infection)



Source: S Bower

Pacific oyster (*Crassostrea gigas*) removed from shell, showing lesions (arrowed) seen during later stages of infection with *Mikrocytos mackini*. Typically, *M. mackini* can no longer be found in oysters at this advanced stage of the disease.



Source: S Bower

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Diseases caused by any of the microcell species are similar, with few or no clinical or gross signs present with light infection. Identification of the *Bonamia or Mikrocytos* species requires histological laboratory examination and molecular diagnostic techniques.

Disease signs at the farm, tank or pond level are:

- mortalities.

Gross pathological signs are:

- focal yellow–green lesions up to 5 mm in diameter within the body wall or on surfaces of the gonad, labial palps, gills or mantle
- brown scars on the shell adjacent to lesions on the mantle surface
- gaping oysters (due to impaired adductor muscle contraction).

Microscopic pathological signs are:

- focal intracellular infection, mainly of vesicular connective tissue cells, resulting in haemocyte infiltration and tissue necrosis
- intracellular microcell protozoa, 2–3 µm in diameter, in vesicular connective tissue cells immediately adjacent to lesions.

Disease agent

Mikrocytos mackini is an intracellular protozoan parasite that causes lethal infection of certain oysters. *M. mackini* is the only species described in the genus and is unrelated to *Bonamia* spp.

Host range

Species known to be susceptible to infection with *M. mackini* are listed below.

Common name	Scientific name
American oyster	*Crassostrea virginica*
European flat oyster	*Ostrea edulis*
Olympia oyster **a**	*Ostrea conchaphila*
Pacific oyster **a**	*Crassostrea gigas*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Severe infections appear to be restricted to oysters over 2 years old.
- The disease is associated with low temperature and high salinity. Most mortalities occur during April–May (spring in the Northern Hemisphere). There is a 3–4 month pre-patent period when temperatures are less than 10 °C.
- The Pacific oyster appears to be more resistant to the disease than other species challenged experimentally under laboratory and field conditions.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with *Bonamia* spp., *B. ostreae* and *B. exitiosa*

There may be few or no visual cues to the presence of this disease other than poor condition, shell gaping and increased mortality. Any presumptive diagnosis requires further laboratory examination.

Light microscopy can contribute diagnostic information, but further laboratory examination and molecular diagnostic techniques are required for a definitive diagnosis.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained.

If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

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Further reading

Further information can be found on the following websites:

disease pages of Fisheries and Oceans Canada: www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm

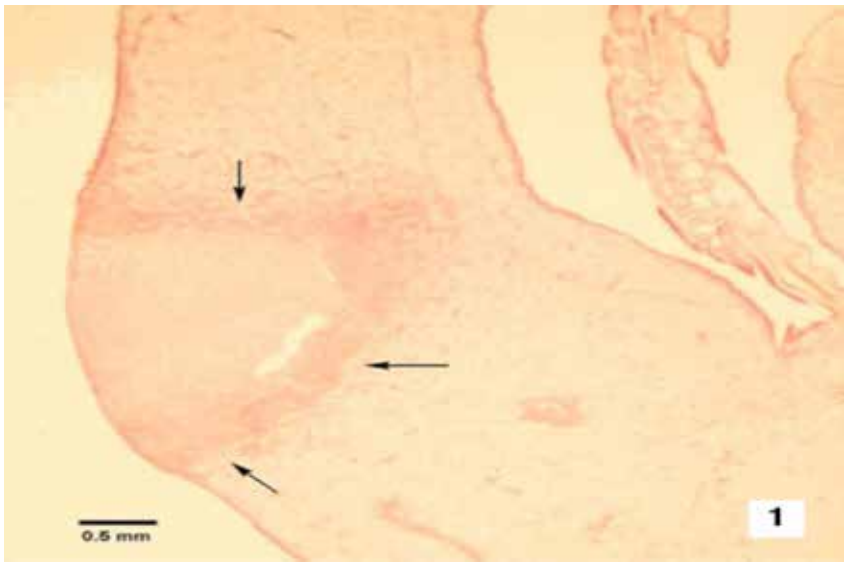
Centre for Environment, Fisheries and Aquaculture Science (Cefas) International Database on Aquatic Animal Disease (IDAAD): www.cefas.defra.gov.uk/idaad/disocclist.aspx

European Union Reference Laboratory for Molluscs Diseases website (at the French Research Institute for Exploration of the Sea—IFREMER): wwz.ifremer.fr/crlmollusc/Main-activities/Tutorials/Mikrocytos-mackini.

These hyperlinks were correct and functioning at the time of publication.

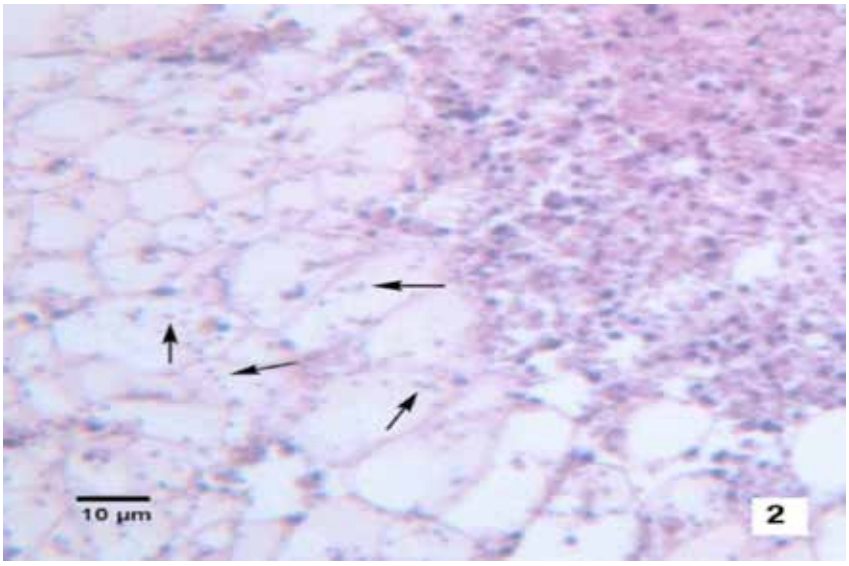
Further images

(1) Haematoxylin and eosin stained section through a lesion caused by *M. mackini* on the mantle of a Pacific oyster (*Crassostrea gigas*). This intracellular protozoan (not visible at this magnification) usually occurs in the intact vesicular connective tissue cells immediately surrounding the periphery of the lesion (arrows).



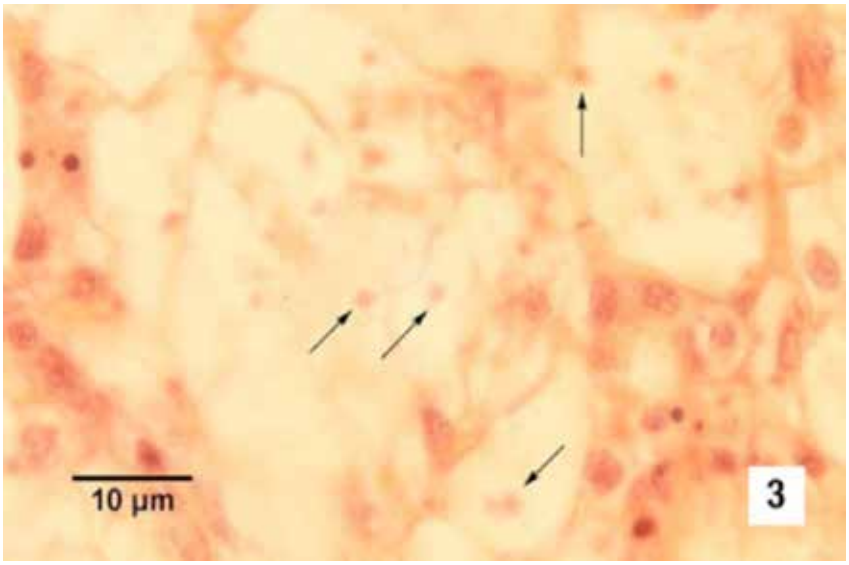
Source: S Bower

(2) Many *M. mackini* (arrows) within vesicular connective tissue cells next to a lesion characterised by an accumulation of haemocytes and necrotic cells (haematoxylin and eosin stain)



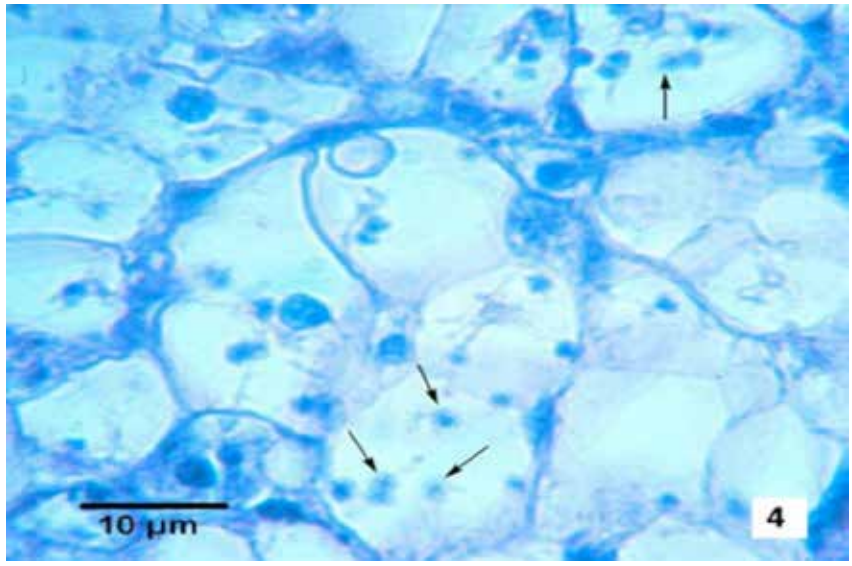
Source: S Bower

(3) Oil immersion magnification of *M. mackini* (arrows) within the cytoplasm of vesicular connective tissue cells of a Pacific oyster (*Crassostrea gigas*) (haematoxylin and eosin stain)



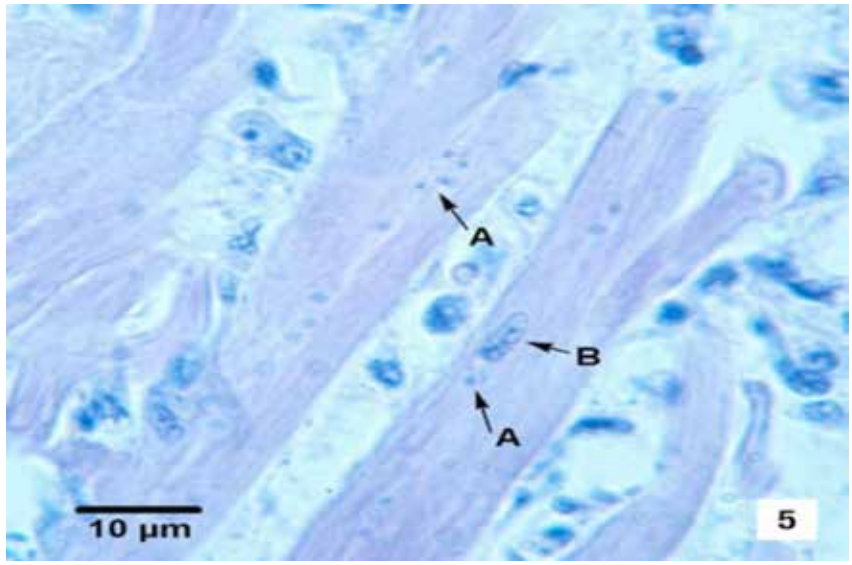
Source: S Bower

(4) As for Figure 3 but from a different specimen. Because of the small size of this parasite, it is very difficult to visualise and photograph in histological preparations. (haematoxylin and eosin stain)



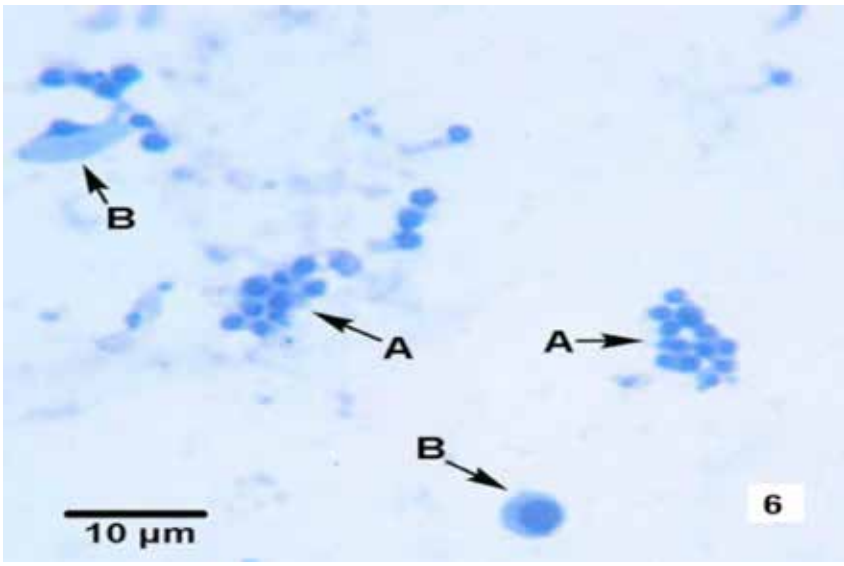
Source: S Bower

(5) *M. mackini* (A) within fibres of the adductor muscle of a Pacific oyster (*Crassostrea gigas*). One *M. mackini* is located close to the nucleus (B) of a muscle cell. (haematoxylin and eosin stain)



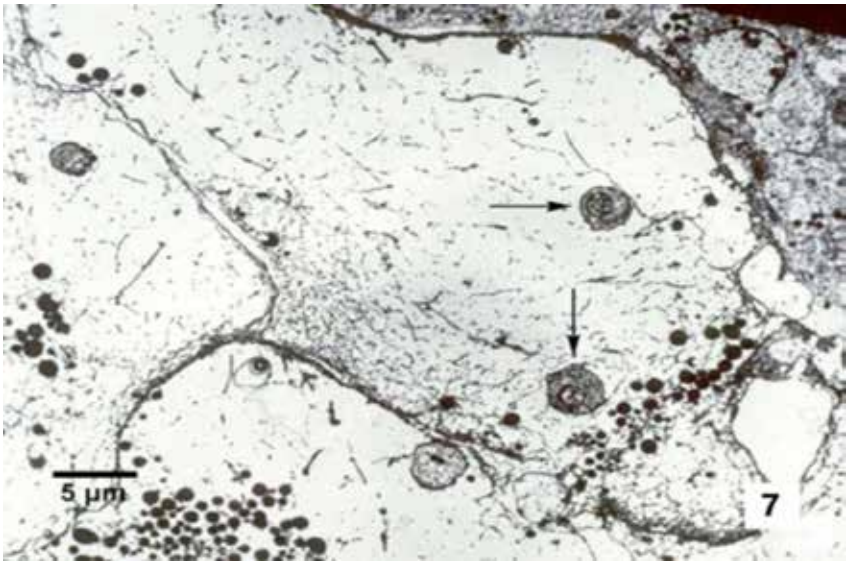
Source: S Bower

(6) Clusters of *M. mackini* (A), isolated and partially purified from a heavily infected Pacific oyster (*Crassostrea gigas*), among host cell debris (B) (Hemacolor® stain)



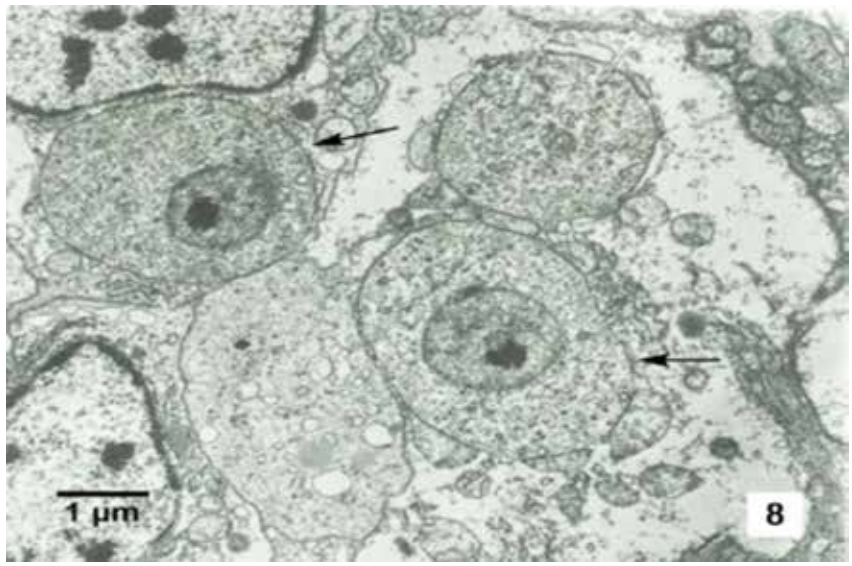
Source: S Bower

(7) Electron micrograph of a Pacific oyster vesicular connective tissue cell containing *M. mackini* (arrows) from a Pacific oyster (*Crassostrea gigas*) (uranyl acetate and lead citrate stain)



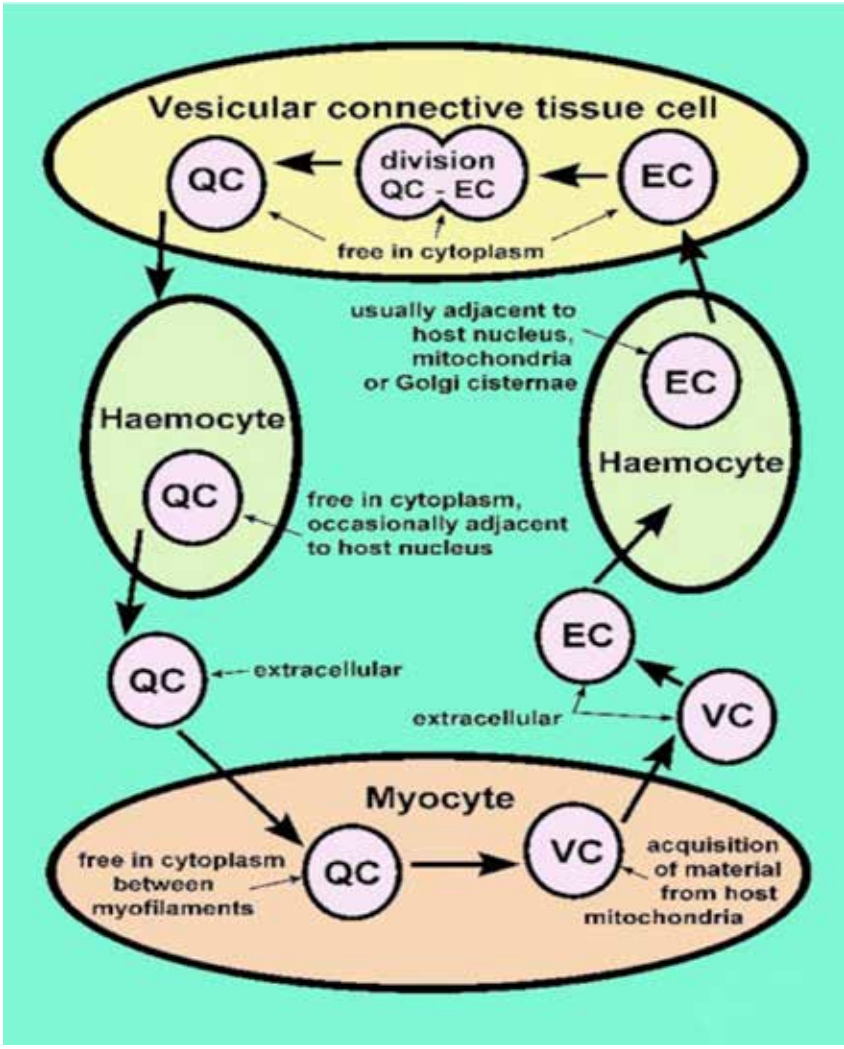
Source: S Bower

8) *M. mackini* (arrows), each containing a nucleus with a pronounced nucleolus and lacking mitochondria (uranyl acetate and lead citrate stain)



Source: S Bower

(7) Electron micrograph of a Pacific oyster vesicular connective tissue cell containing *M. mackini* (arrows) from a Pacific oyster (*Crassostrea gigas*) (uranyl acetate and lead citrate stain)



Source: S Bower

Exotic disease

Infection with *Perkinsus marinus*

American oyster (*Crassostrea virginica*) on right, showing typical gross signs of infection with *Perkinsus marinus*; specimen on left is healthy

Source: E Burreson

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- gaping
- retarded growth
- poor condition or emaciation.

Gross pathological signs are:

- shrinkage of mantle away from the outer edge of the shell
- occasional lesions in soft tissue
- pale digestive gland
- thin, watery tissues.

Microscopic pathological signs are:

- large, multifocal lesions in the gut epithelium or connective tissue of any organ containing *P. marinus* cells
- haemocyte infiltration and phagocytosis of *P. marinus* cells
- destruction of the gut epithelium.

Disease agent

P. marinus is an alveolate protist nominally in the Dinoflagellida, but there is ongoing scientific debate about its higher taxonomy.

Host range

Species known to be susceptible to infection with *P. marinus* are listed below.

Common name	Scientific name
American oyster **a**	*Crassostrea virginica*
Baltic macom **a**	*Macoma balthica*
Blue mussel	*Mytilus edulis*
Cortez oyster **a**	*Crassostrea corteziensis*
Mangrove oyster **a**	*Crassostrea rhizophorae*
Pacific oyster **a**	*Crassostrea gigas*
Sand gaper mussel	*Mya arenaria*
Suminoe oyster **a**	*Crassostrea ariakensis*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Water temperatures above 20 °C encourage proliferation of the parasite, resulting in systemic disruption of connective tissue and epithelial cells, leading to high mortalities.
- Reduced salinity levels (below 9‰) can prevent clinical disease resulting from *P. marinus* infection, even in warm water (above 20 °C).
- Cumulative American oyster mortalities of up to 95% have been observed in the first summer following transfer of naive stock to an area where the disease is known to be present.
- Transmission is horizontal.
- Infection levels increase during spawning and under the stress of oxygen depletion.
- Prevalence and intensity of infection are greatest in oysters more than 1 year old and at depths greater than 90 cm.
- Exposure to pollutants will increase the prevalence of infection.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with *Perkinsus olseni*

The clinical signs of infection with *P. marinus* are similar to those of infection with other species of *Perkinsus* (i.e. occasional pustules in soft tissue, pale digestive gland, poor condition, emaciation, shrinkage of mantle and retarded growth). It is therefore difficult to make a presumptive diagnosis based on gross signs alone; any presumptive diagnosis requires further laboratory examination.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

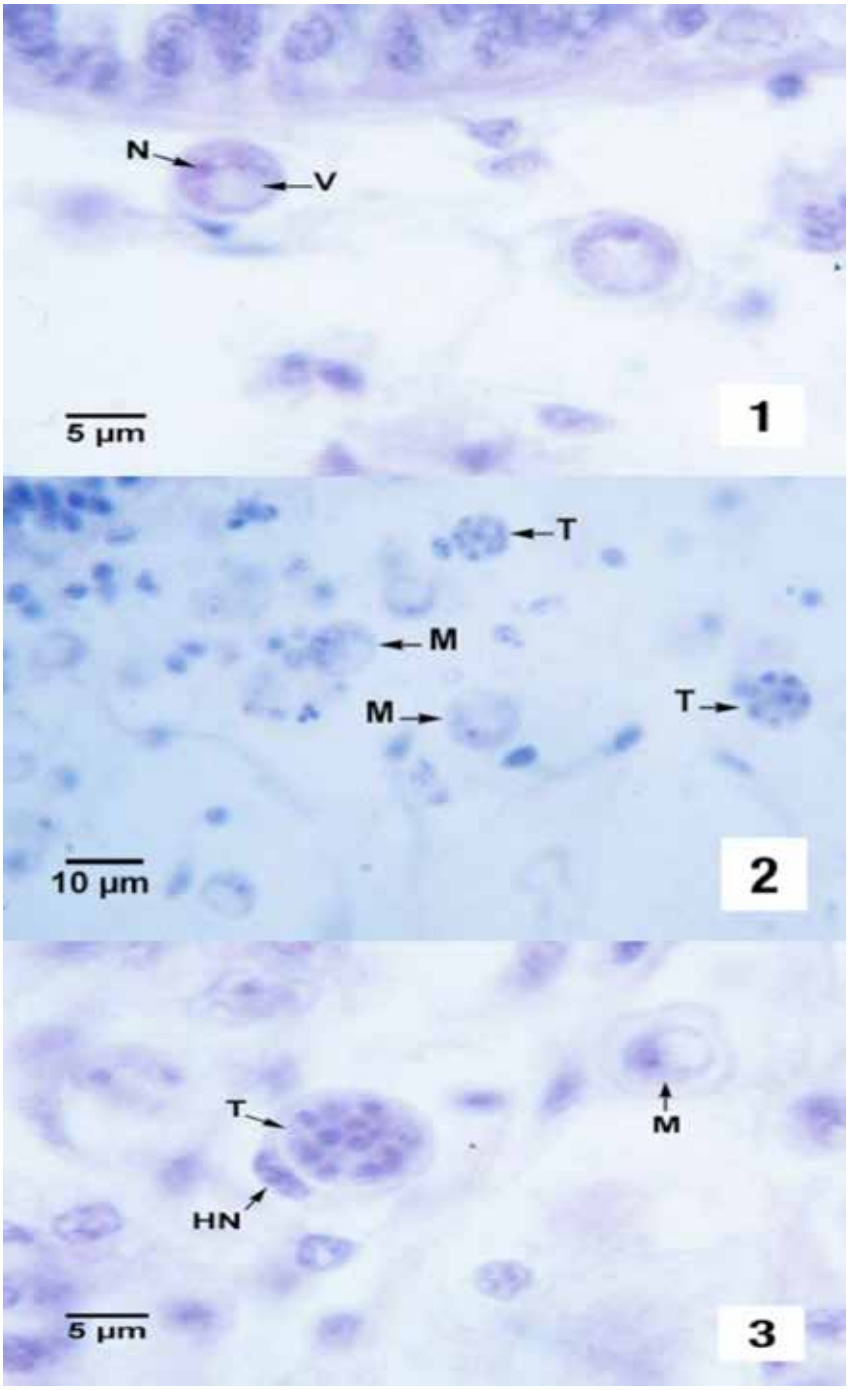
The accepted procedures for a conclusive diagnosis of infection with *P. marinus* are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011,* available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information is available on the disease pages of Fisheries and Oceans Canada: www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm.

These hyperlinks were correct and functioning at the time of publication

Further images

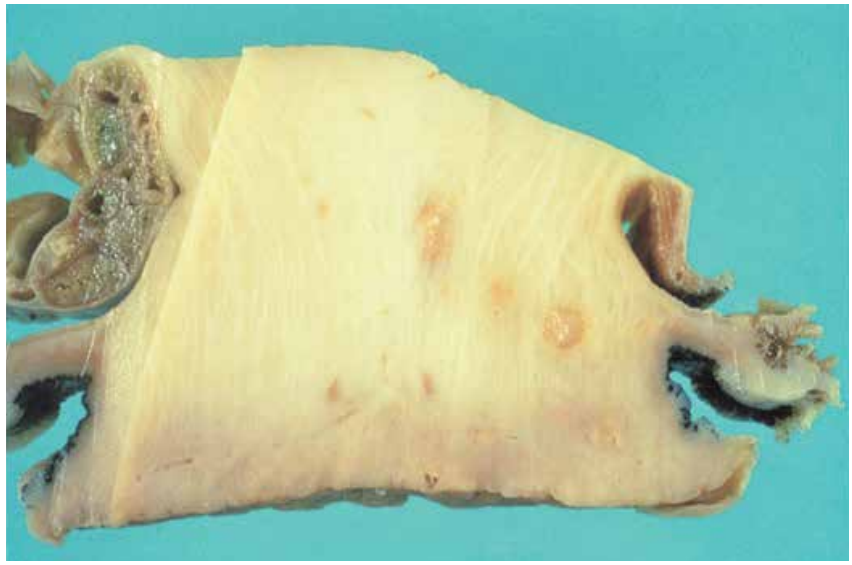
(1,2 & 3) A 16-cell tomont (T) containing developing trophozoites. This tomont is contained within a haemocyte (HN indicates the nucleus of the phagocytic cell), and a maturing trophozoite (M) is nearby.



Source: S Bower

Infection with *Perkinsus olseni*

Infection with *Perkinsus olseni* in abalone; note blisters on body tissue



Source: New South Wales Department of Primary Industries

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- morbidity observed in cultured greenlip (*Haliotis laevigata*) and blacklip (*H. rubra)* abalone
- gaping in bivalve species.

Gross pathological signs are:

- spherical brown abscesses up to 8 mm in diameter containing a caseous creamy-brown deposit in the foot and mantle of blacklip and greenlip abalone (these gross pathological changes impact on the marketability of abalone)
- thin, watery tissues with a pale digestive gland
- nodules in the mantle and gills.

Microscopic pathological signs are:

- large, multifocal lesions in connective tissue, containing haemocyte aggregations around *Perkinsus* cells.

Disease agent

Several species of the genus *Perkinsus* are responsible for causing perkinsosis in molluscs such as oysters, mussels, clams and abalone worldwide. *P. olseni* is the only species known to cause this disease in the Asia–Pacific region and is responsible for perkinsosis in abalone, clams and pearl oysters. *P. atlanticus* is a junior synonym of *P. olseni*.

Host range

Species known to be susceptible to infection with *P. olseni* are listed below.

Common name	Scientific name
Asian littleneck clam **a**	*Venerupis philippinarum*
Blacklip abalone **a**	*Haliotis rubra*
Crocus clam **a**	*Tridacna crocea*
Elongated giant clam or rugose giant clam **a**	*Tridacna maxima*
European aurora venus clam **a**	*Venerupis aurea*
Giant clam **a**	*Tridacna gigas*
Greenlip abalone **a**	*Haliotis laevigata*
Grooved carpet shell or venerid clam **a**	*Ruditapes decussatus*, *R. semidecussatus*
Kumamoto oyster	*Crassostrea sikamea*
Manila clam	*Venerupis philippinarum*
New Zealand cockle **a**	*Austrovenus stutchburyi*, *Macomona liliana* and *Barbatia novae-zelandiae*
Pacific oyster **a**	*Crassostrea gigas*
Pearl oyster	*Pinctada sugillata*, *P. margaritifera* and *P. martensii*
Pullet carpet shell	*Venerupis pullastra*
Sand cockle	*Katelysia rhytiphora*
Silverlip pearl oyster	*Pinctada maxima*
Staircase abalone **a**	*Haliotis scalaris*
Sydney cockle	*Anadara trapezia*
Venerid commercial clam **a**	*Pitar rostrata*
Whirling abalone **a**	*Haliotis cyclobates*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

P. olseni has been officially reported from New South Wales, South Australia and Western Australia. Although *P. olseni* was originally reported from abalone, recent studies suggest that, in Australia, a single species of *Perkinsus* occurs in a wide variety of molluscs, including clams and pearl oysters.

Epidemiology

- *P. olseni* has been associated with mass mortality of *Haliotis* spp. (both blacklip and greenlip abalone) in the Gulf of St Vincent, South Australia, and coastal New South Wales (mostly blacklip abalone).
- Horizontal transmission occurs directly from host to host. Some environmental conditions (temperature and salinity) can promote a lifelong carrier state.
- Infection intensity increases with the age of the host.
- Prezoosporangia that escape from necrotic pustules or decaying dead abalone undergo further development to zoosporangia in sea water.
- Within 9 days at 20 °C and 3 days at 28 °C, hundreds of motile, biflagellated zoospores (about 3 µm × 5 µm) exit from the zoosporangium. The zoospores are infective to abalone as well as to other molluscs.
- *P. olseni* can survive in salt water for several weeks at –20 °C, however fresh water is lethal to the pathogen.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infection with *Perkinsus marinus*

The clinical signs of infection with *P. olseni* are similar to those of infection with other species of *Perkinsus* (i.e. occasional pustules in soft tissue, pale digestive gland, poor condition, emaciation, shrinkage of the mantle and retarded growth). It is difficult to make a presumptive diagnosis based on gross signs alone; any presumptive diagnosis requires further laboratory examination.

Sample collection

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Emergency disease hotline

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Further reading

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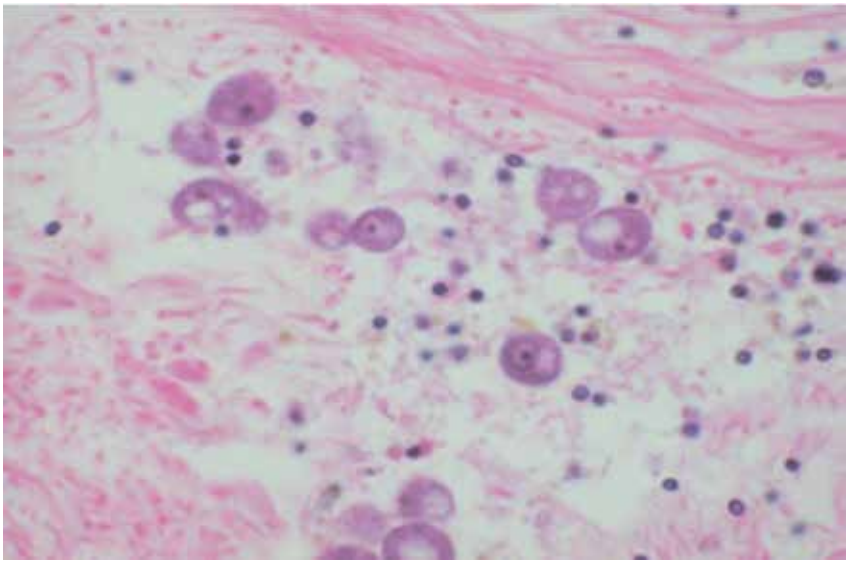
These hyperlinks were correct and functioning at the time of publication.

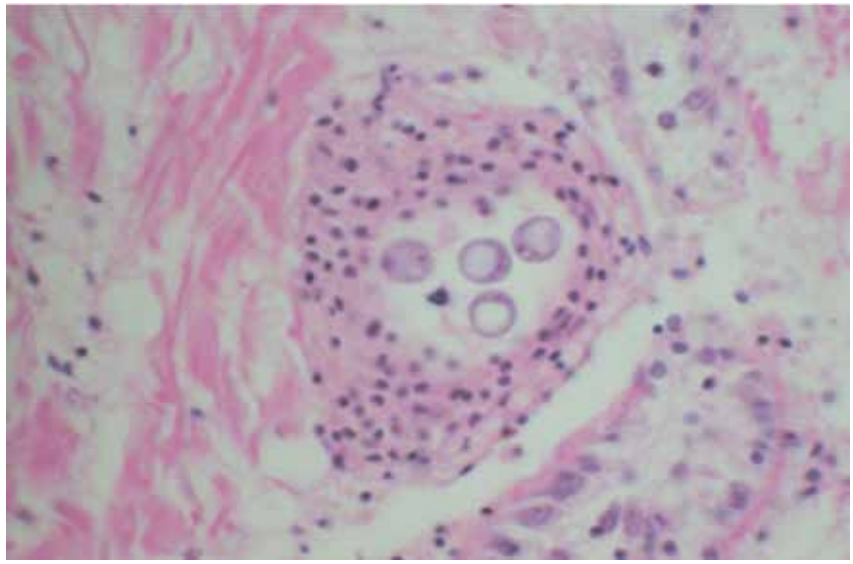
Further images

(1) *P. olseni* in greenlip abalone (*Haliotis laevigata*)



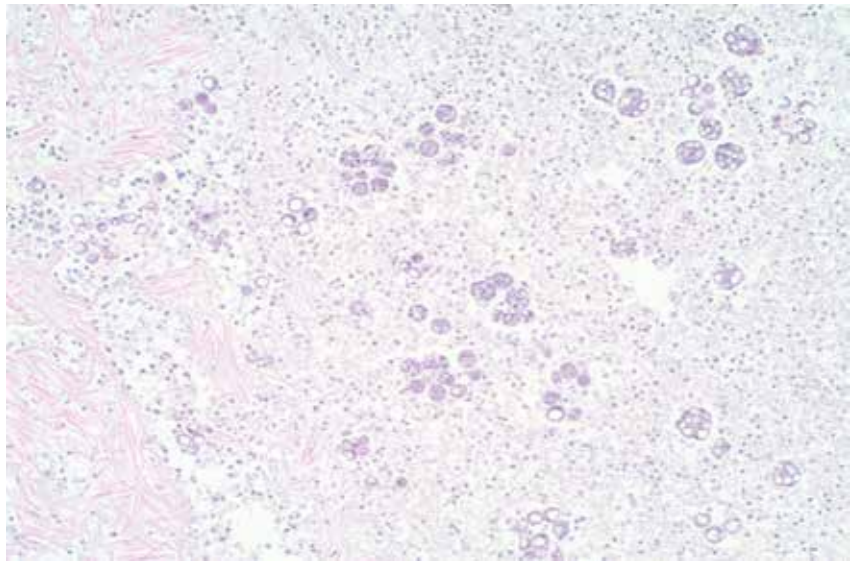
Source: E Burreson

(2) *P. olseni* in a clam (*Ruditapes* sp.)



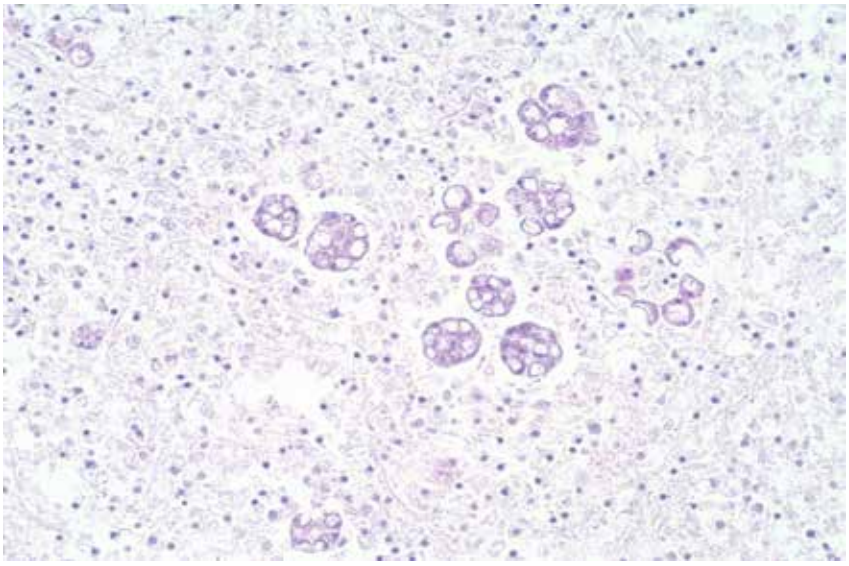
Source: E Burreson

(3) Pedal tissue with the edge of the cyst containing multilocular *Perkinsus* organisms among haemocytes and floccular debris (haematoxylin and eosin stain; 100×)



Source: Stella Bastianello/Gribbles VetLab

(4) Cyst with multilocular *Perkinsus* organisms, as well as some more mature crescent-shaped organisms among haemocytes and floccular debris (haematoxylin and eosin stain; 200×)



Source: Stella Bastianello/Gribbles VetLab

Other diseases of molluscs

Exotic disease

Akoya oyster disease

(Also known as mass mortality of Japanese pearl oyster or red adductor disease of pearl oyster)

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- mass mortalities.

Gross pathological signs are:

- stunted growth
- slow closing of valves
- atrophied and shrunken soft tissue
- watery appearance, due to lack of nutrient storage
- red–brown adductor muscle (as opposed to creamy white in healthy oysters)
- decreased quality of pearls, including dark spots, gross pearl deformities, dark colouration and reduced lustre.

Microscopic pathological signs are:

- degenerated loose connective tissue in the mantle and an increased number of host cells
- necrosis, atrophy, swelling and vacuolisation of muscle fibres, with increased connective tissue supporting framework.

Disease agent

The causative agent of Akoya oyster disease has not been determined. Based on some infection experiments, a viral aetiology is suspected.

Host range

Species known to be susceptible to Akoya oyster disease are listed below.

Common name	Scientific name
Japanese pearl oyster or Akoya oyster **a**	*Pinctada fucata martensii*

a Naturally susceptible

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Akoya oyster disease affects oysters more than 1 year old, including juvenile, adult and seeded oysters.
- The disease results in high cumulative mortality (reported up to 80%), particularly during summer and autumn when water temperature is higher (above 20 °C).
- Susceptibility may vary with strains and subspecies of the Akoya oyster.
- Transmission is thought to be horizontal.
- The only effective control methods appear to be avoidance by restricting movement around high-risk areas and moving oysters to cooler water during the summer.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are considered similar to Akoya oyster disease. This disease should be suspected when mass mortality occurs in Akoya oysters.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found on the website of the Network of Aquaculture Centres in Asia–Pacific at library.enaca.org/Health/DiseaseLibrary/AkoyaDisease.pdf.

This hyperlink was correct and functioning at the time of publication.

5 Diseases of crustaceans

Viral diseases of crustaceans

Gill-associated virus disease

Black tiger prawn (*Penaeus monodon*) infected with gill-associated virus; note red colouration

Source: D Callinan

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- high mortality (up to 80%)
- moribund prawns aggregating near the surface at pond edges
- initial increase in feeding at an abnormally high rate, followed by a sudden decline.

Gross pathological signs are:

- reddening of body and appendages
- biofouling with exoparasites
- emaciation
- pink to yellow colouration of the gills.

Microscopic pathological signs are:

- lymphoid organ necrosis (hypertrophied nuclei, marginated chromatin and vacuolation).

Disease agent

The causative agent is gill-associated virus (GAV), genotype 2 of six distinguished genotypes in the yellowhead complex of viruses. GAV is the type species of the genus *okavirus* in the family *Roniviridae* and order *Nidovirales*. Comparison of DNA sequences indicates that GAV and yellowhead virus are closely related, but have distinctly different genotypes, sharing 85% of their genetic material. Natural genetic recombination between GAV and other genotypes in the yellowhead complex has been reported outside Australia.

Host range

Species known to be susceptible to infection with GAV are listed below.

Common name	Scientific name
Black tiger prawn **a**	*Penaeus monodon*
Brown tiger prawn	*Penaeus esculentus*
Gulf banana prawn	*Fenneropenaeus merguiensis*
Kuruma prawn	*Marsupenaeus japonicus*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

GAV has been officially reported from New South Wales, Queensland, the Northern Territory and Western Australia.

Epidemiology

The epidemiology of GAV is thought to be very similar to that of yellowhead virus:

- Transmission can be horizontal, directly from the water column and through ingestion of infected material.
- Vertical transmission can occur via surface contamination or infection of tissue surrounding the fertilised egg.
- Viral multiplication and disease appear to be induced by environmental stress.
- Mortality usually occurs among early to late juvenile stages in rearing ponds.
- Experimental infections with GAV indicate that larger (~20 g) kuruma prawns are less susceptible to disease than smaller (~6–13 g) prawns of the same species.
- GAV has been associated with mortalities of up to 80% in black tiger prawn ponds in Australia.

GAV occurs commonly as a chronic infection in healthy broodstock and farmed black tiger prawns in eastern Australia. It has also been associated with acute infections and disease outbreaks in ponds, causing high mortality, but produces gross signs and patterns of tissue tropism different from those for yellowhead virus.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Yellowhead disease

As GAV and yellowhead are closely related viruses, molecular testing is required to discriminate between the two diseases.

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

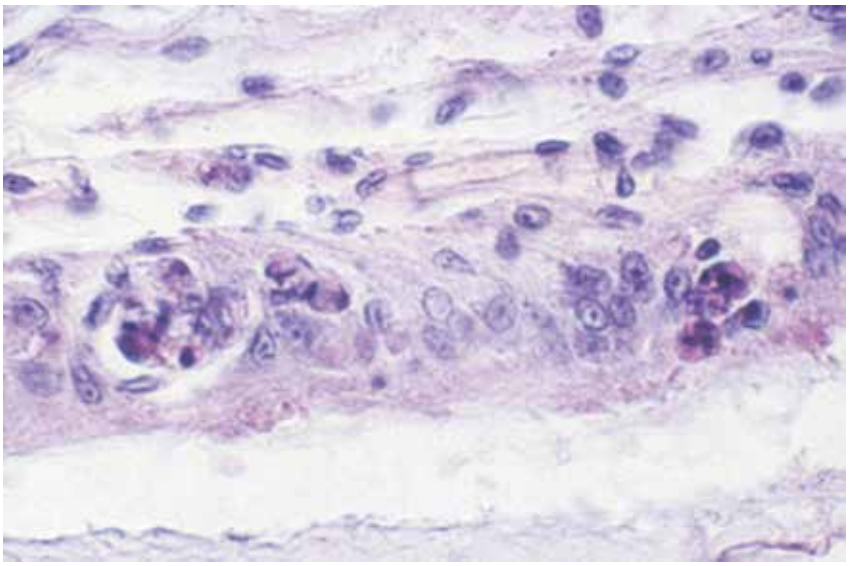
Further reading

The accepted procedures for a conclusive diagnosis of GAV are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

Further image

Histological changes present with gill-associated virus. Lymphoid necrosis includes signs such as hypertrophied nuclei, marginated chromatin and vacuolation

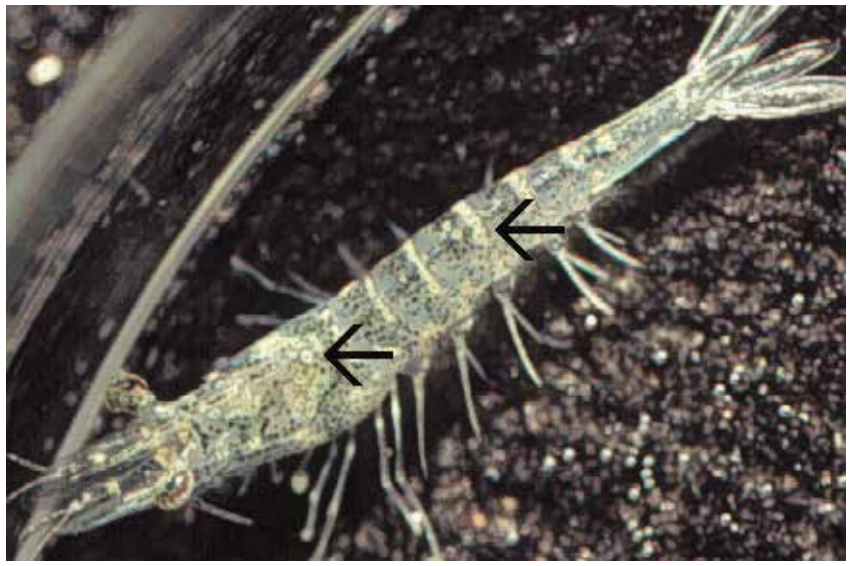


Source: L Owens

Infectious hypodermal and haematopoietic necrosis (IHHN)

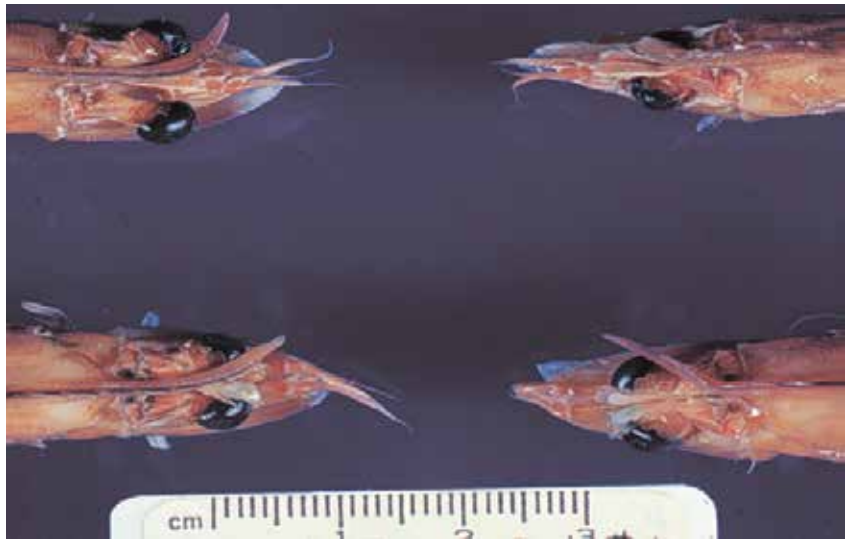
(Also known as infection with *Penaeus* stylirostris densovirus [PstDNV])

IHHN in juvenile Pacific blue shrimp (*Litopenaeus stylirostris*); note white to buff lesions under the shell (arrows)



Source: DV Lightner

IHHN in juvenile Pacific blue shrimp (*Litopenaeus stylirostris*); note classic rostrum deformation



Source: DV Lightner

IHHN in juvenile Pacific blue shrimp (*Litopenaeus stylirostris*); note deformed tail fan and sixth abdominal segment

Source: DV Lightner

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- reduced food consumption
- cannibalism
- repeatedly floating slowly to the water surface, rolling over and then sinking to the bottom
- morbidity or mortality
- poor hatching success of eggs
- poor survival of larvae and postlarvae.

Gross pathological signs are:

- opaque abdominal musculature.

In Pacific blue shrimp, additional gross pathological signs are:

- cuticular roughness
- cuticular deformities
- white to buff mottling of the shell, especially at the junction of abdominal shell plates.

In Pacific blue shrimp, Pacific white shrimp and black tiger prawns, additional gross pathological signs are:

- blue appearance of moribund prawns
- runt-deformity syndrome, the effects of which include
  - reduced and irregular growth in juveniles and subadults
  - deformed rostrums growing to one side
  - deformed sixth abdominal segment.

Microscopic pathological signs are:

- eosinophilic to pale basophilic intranuclear inclusion bodies (Cowdry type A inclusions) within tissues of ectodermal and mesodermal origin. Note that the inclusion bodies may be easily confused with developing intranuclear inclusion bodies caused by white spot disease.

Disease agent

IHHN and runt-deformity syndrome are caused by a *Densovirus* belonging to the family *Parvoviridae* (subfamily Densovirinae). At least three distinct genotypes of IHHN virus have been identified, but only two are known to be infectious to either Pacific white shrimp or black tiger prawns. Homologues of portions of the IHHN viral genome have been reported from the genomes of host prawns

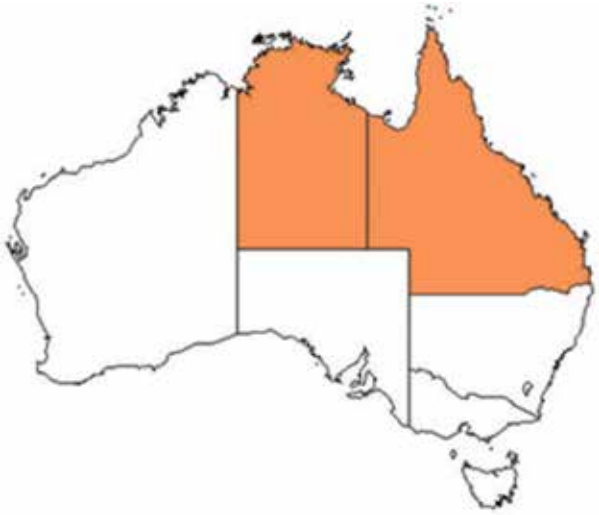
Host range

Host species of IHHN are listed below.

Common name	Scientific name
Banana prawn	*Fenneropenaeus merguiensis*
Black tiger prawn **ab**	*Penaeus monodon*
Pacific blue shrimp **a**	*Litopenaeus stylirostris*
Chinese white shrimp	*Fenneropenaeus chinensis*
Green tiger prawn or grooved tiger prawn **a**	*Penaeus semisulcatus*
Indian banana prawn	*Fenneropenaeus indicus*
Kuruma prawn	*Marsupenaeus japonicus*
Northern brown shrimp	*Farfantepenaeus aztecus*
Northern pink shrimp	*Farfantepenaeus duorarum*
Northern white shrimp	*Litopenaeus setiferus*
Pacific white shrimp **a**	*Litopenaeus vannamei*
Southern white shrimp **a**	*Litopenaeus schmitti*
Western white shrimp **a**	*Litopenaeus occidentalis*
Yellow-leg shrimp **a**	*Fenneropenaeus californiensis*

a Naturally susceptible (other species have been shown to be experimentally susceptible)
b Hybrids of the black tiger prawn and the brown tiger prawn (*P. esculentus*) are considered susceptible

Presence in Australia



IHHN virus in the black tiger prawn has been officially reported from the Northern Territory and Queensland.

Epidemiology

- Gross signs of disease in an infected animal become evident from about 35 days of postlarval development.
- IHHN virus-resistant prawns and early life stages are carriers, and may transfer the virus to more susceptible species and life stages.
- The typical gross signs of runt-deformity syndrome may be observed in juveniles and subadults.
- Mortality events seldom occur in infected adult prawns.
- IHHN suppresses the prawns' immune system, allowing infection by other disease agents.
- Infected tissue remains infectious after 5 years of storage at –20 °C, after 10 years at –80 °C, and after storage in 50% glycerine.
- IHHN infection can result in cumulative mortalities as high as 90% in postlarvae and juveniles.
- Transmission of IHHN virus can be via horizontal or vertical routes. Horizontal transmission has been demonstrated by cannibalism or through contaminated water, and vertical transmission has been demonstrated via infected eggs.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

The microscopic signs of disease may also be similar to the early stages of white spot disease. Further laboratory examination is needed for a definitive diagnosis.

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

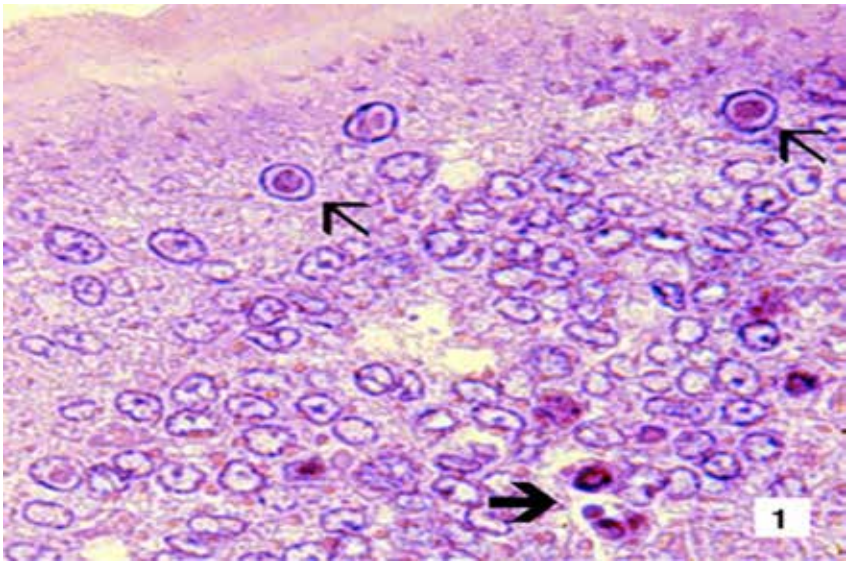
The accepted procedures for a conclusive diagnosis of IHHN are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information can also be found on the disease pages of Fisheries and Oceans Canada: www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm.

These hyperlinks were correct and functioning at the time of publication.

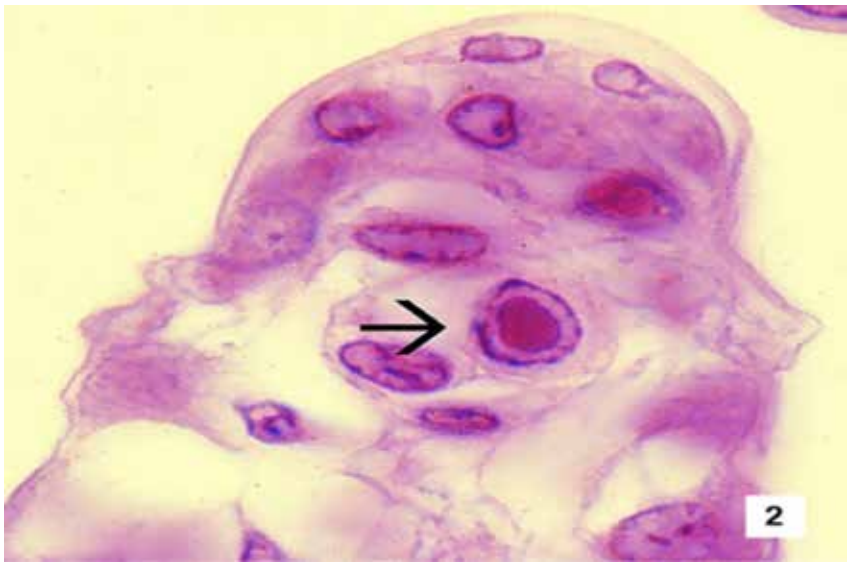
Further images

(1) Low magnification (830×) light micrograph of a haematoxylin and eosin stained section of a juvenile Pacific blue shrimp (*Litopenaeus stylirostris*) with severe acute IHHN disease. The section is through the cuticular epithelium and subcuticular connective tissues just dorsal and posterior to the heart. Numerous necrotic cells with pyknotic nuclei or with pathognomonic eosinophilic intranuclear inclusion bodies (Cowdry type A) are present (arrows).



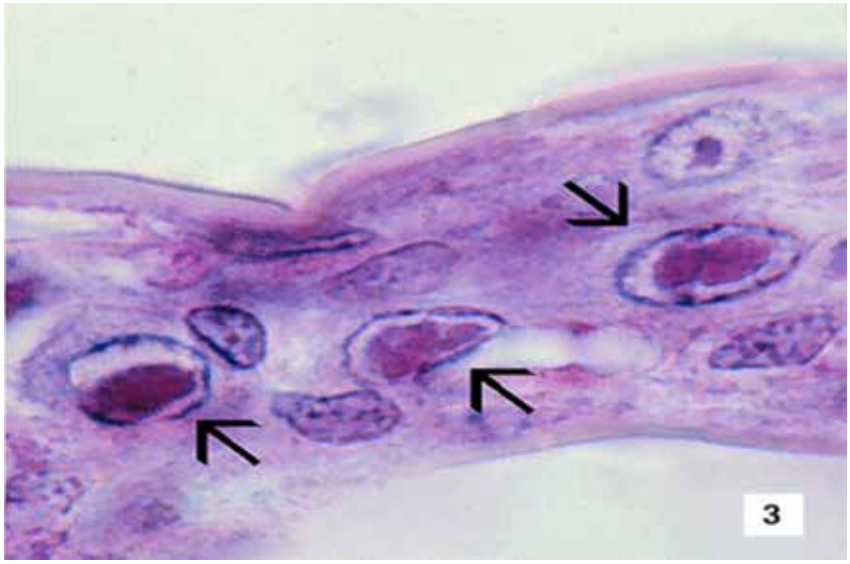
Source: DV Lightner

(2) High magnification (1800×) light micrograph of gills, showing eosinophilic intranuclear inclusions (Cowdry type A) that are pathognomonic for IHHN virus infection



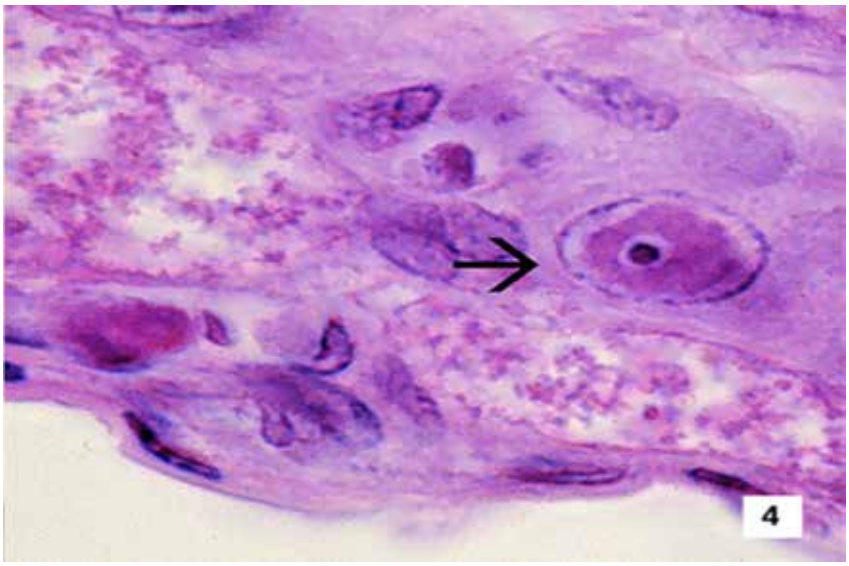
Source: DV Lightner

(3) A high magnification (1800×) light micrograph of a gill lamella, showing three adjacent cells with diagnostic IHHN virus Cowdry type A inclusions in their hypertrophied nuclei



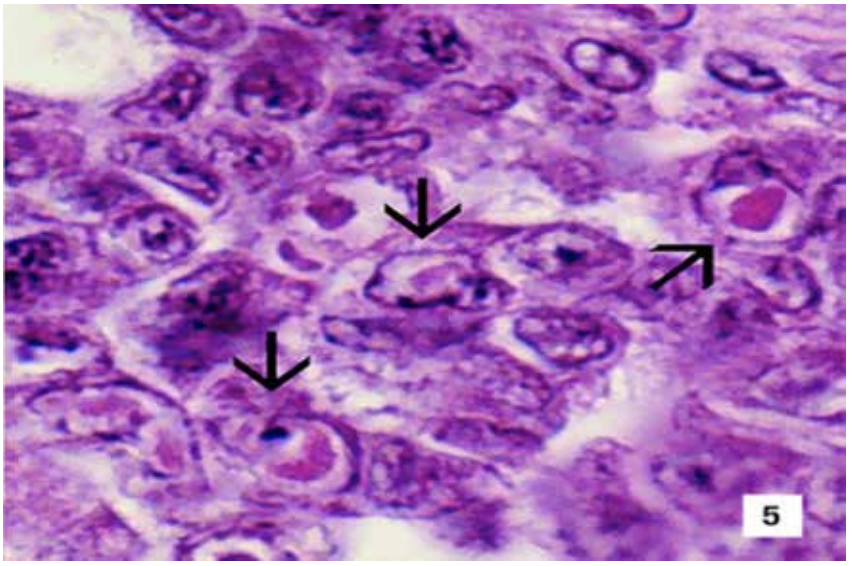
Source: DV Lightner

(4) An IHHN virus Cowdry type A inclusion in the nucleus of a gill epithelial cell, showing a chromatin process within the inclusion body (1800×)



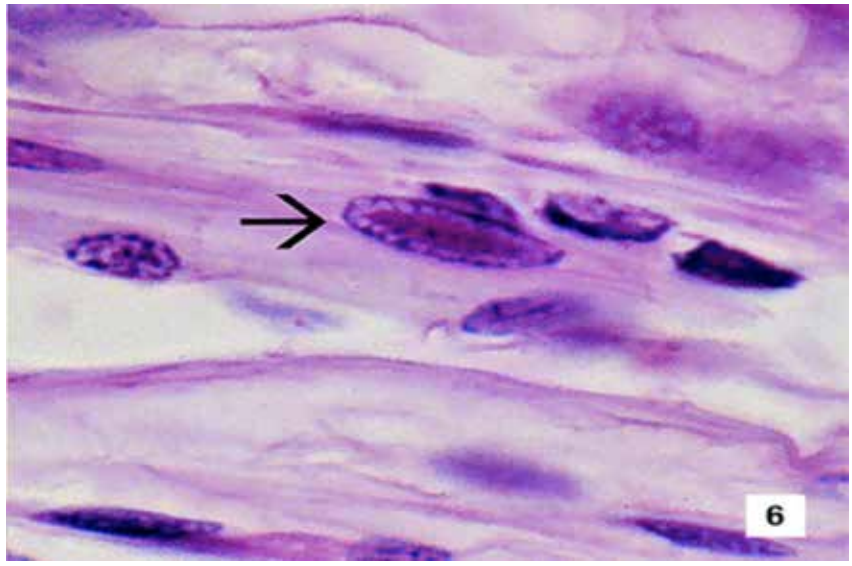
Source: DV Lightner

(5) Section through a haematopoietic nodule showing several cells with IHHN virus Cowdry type A inclusions (1800×)



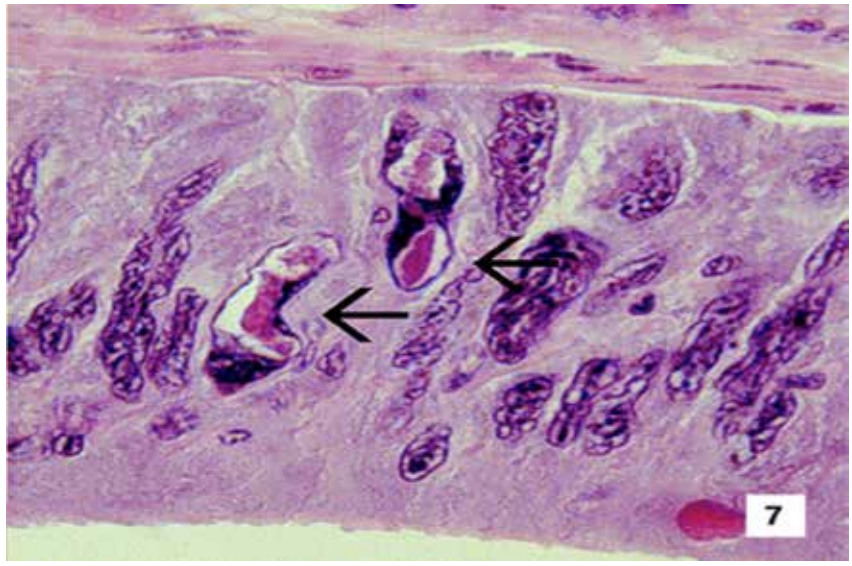
Source: DV Lightner

(6) Midsagittal section of the ventral nerve cord of a juvenile Pacific white shrimp (*Litopenaeus vannamei*) with runt-deformity syndrome. Note that Cowdry type A inclusions take the shape of the host cell nucleus, and so in the nerve cord they are often elliptical and appear slightly different from Cowdry type A inclusions in other tissues (1800×).



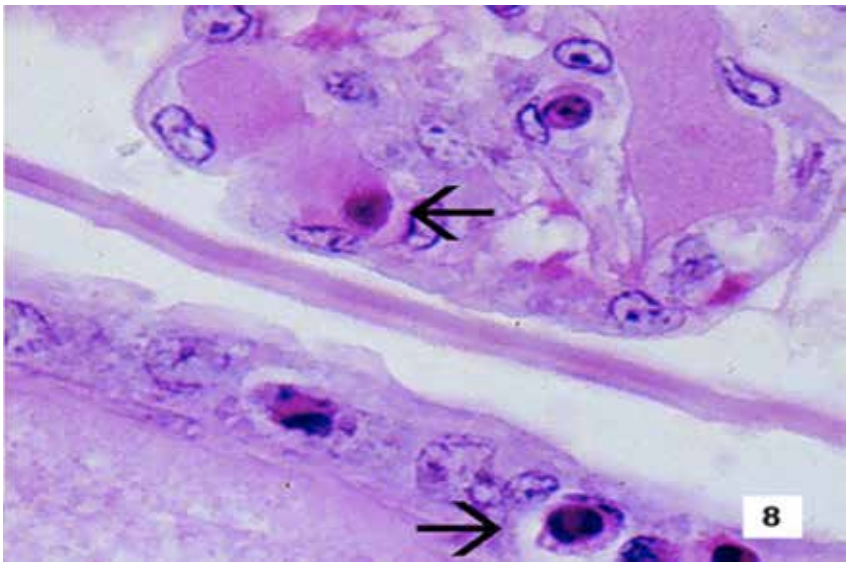
Source: DV Lightner

(7) Section of the vas deferens of an adult Pacific white shrimp (*Litopenaeus vannamei*) with IHHN. Bizarrely shaped Cowdry type A inclusions (which take the shape of the nuclei of this tissue) are illustrated (700×).



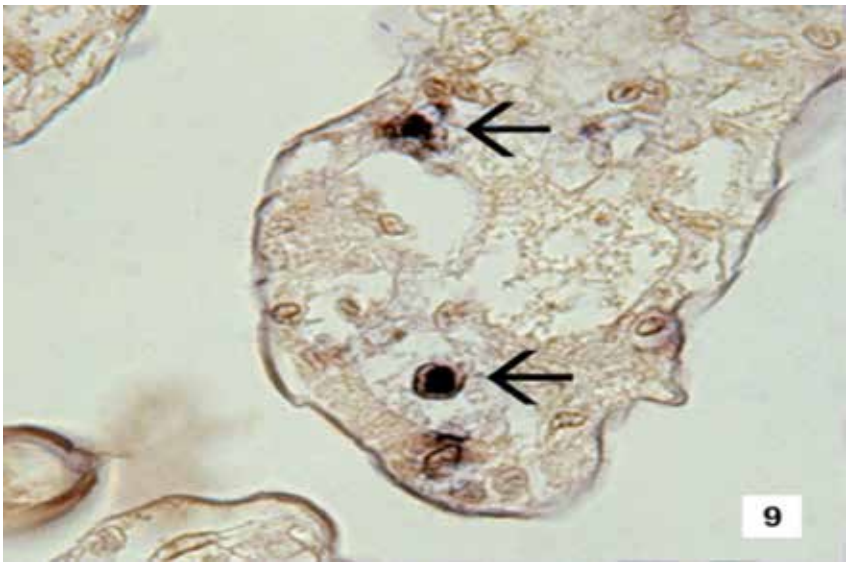
Source: DV Lightner

(8) Section of the gills of a juvenile Pacific blue shrimp (*Litopenaeus stylirostris*) with G4 IHHN. Although necrotic cells with pyknotic nuclei are numerous, no diagnostic Cowdry type A inclusions are apparent (700×).



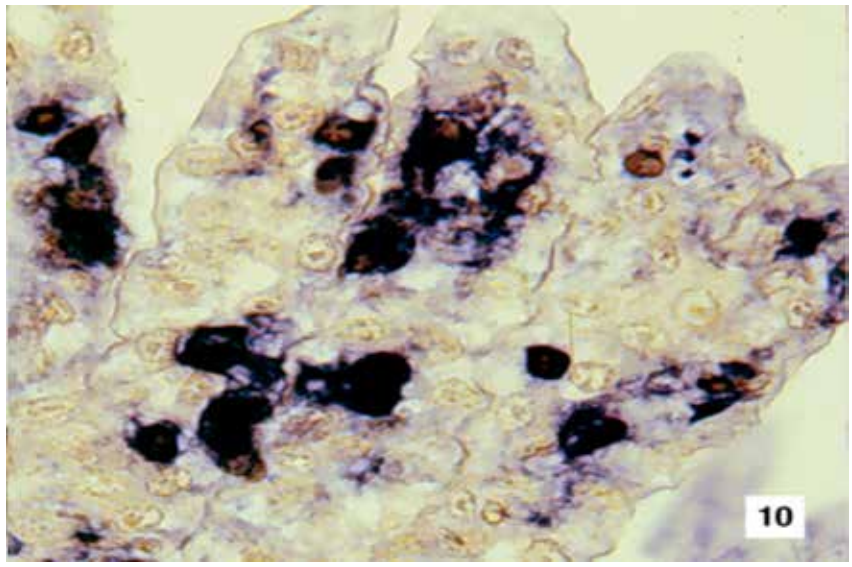
Source: DV Lightner

(9) A section of gills has reacted with a digoxygenin (DIG)-labelled DNA gene probe for IHHN virus. Several IHHN virus-infected cell nuclei have reacted with the probe. Viral DNA is stained dark blue to black by the detection reaction for DIG-labelled probe. DIG-labelled probe and Bismarck Brown (700×).



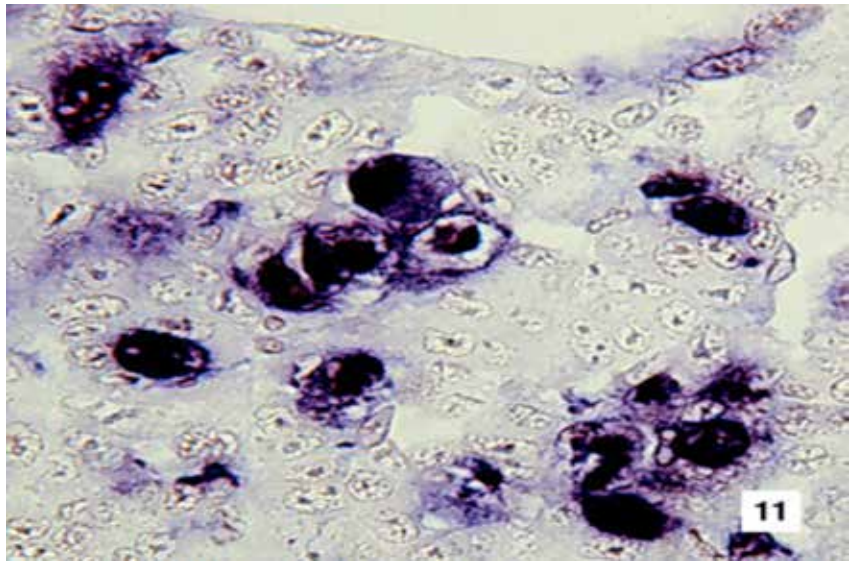
Source: DV Lightner

(10) Section of gills from a juvenile *L. stylirostris* with G4 IHHN. Probe positive IHHN virus-infected cells are abundant. DIG-labelled probe and Bismarck Brown (700×).



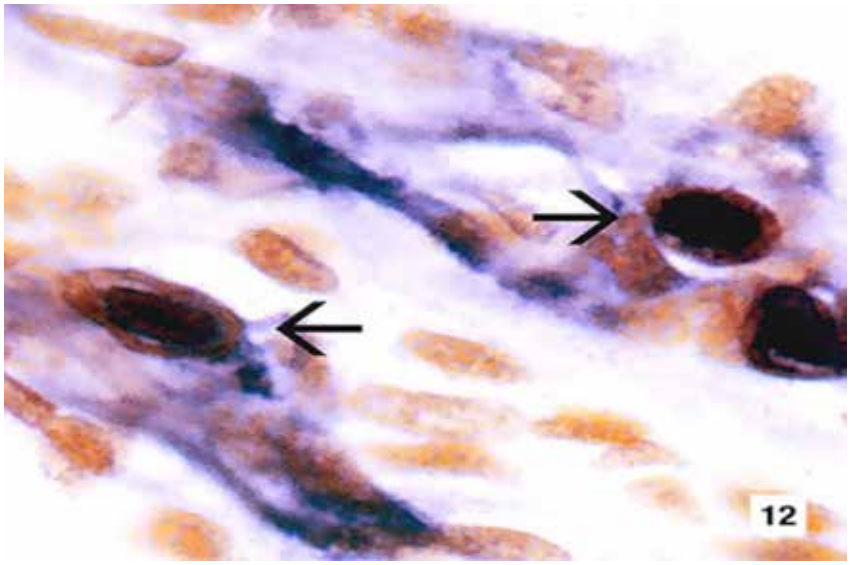
Source: DV Lightner

(11) Section of haematopoietic tissue from a juvenile blue shrimp with IHHN. Probe-positive cells are abundant. DIG-labelled probe and Bismarck Brown (700×).



Source: DV Lightner

(12) Mid-sagittal section from a juvenile white shrimp with runt-deformity syndrome. The probe has reacted with several Cowdry type A inclusions and with cellular debris or haemolymph with a high content of IHHN virus. DIG-labelled probe and Bismarck Brown (600×).



Source: DV Lightner

Infectious myonecrosis

Gross signs of infectious myonecrosis in naturally infected farmed Pacific white prawns (*Litopenaeus vannamei*), exhibiting various degrees of skeletal muscle necrosis, visible as an opaque, whitish discolouration of the abdomen.

Source: DV Lightner

Signs of disease

Important: animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- lethargy
- large numbers of moribund animals and significant mortalities (up to 70%), specifically during or following stressful events.

The acute form of the disease produces gross signs and elevated mortalities, but disease progresses to a chronic phase with persistent low-level mortalities

Gross pathological signs are:

- focal to extensive white necrotic areas in the striated muscle commonly observed in distal abdominal segments
- necrotic and reddened tail fan
- lymphoid organs increased to 3–4 times normal size
- moribund prawns with a full stomach as affected individuals may continue to feed until death.

Microscopic pathological signs are:

- coagulative necrosis of skeletal muscle by haemolytic infiltration and fibrosis.

Disease agent

The causative agent is infectious myonecrosis virus (IMNV), a putative *totivirus*. Phylogenetic analysis based on its RNA-dependent RNA polymerase gene coding sequence groups IMNV most closely with *Giardia lamblia* virus, a member of the family *Totiviridae*.

Host range

Species known to be susceptible to IMNV are listed below.

Common name	Scientific name
Black tiger prawn	*Penaeus monodon*
Pacific shrimp	*Litopenaeus stylirostris*
Pacific white shrimp **a**	*Litopenaeus vannamei*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- IMNV was originally identified in north-eastern Brazil in cultured *L. vannamei*; however, the virus has now been reported in South-East Asia, including the Indonesian provinces of West Nusa Tenggara, East Java and Bali.
- Clinical signs may have sudden onset following stressful events (e.g. capture by net, reduced feeding, sudden changes in temperature or salinity).
- Affected life stages include juveniles and subadults. Significant mortalities occur in juvenile and subadult pond-reared populations.
- Horizontal transmission has been demonstrated via cannibalism. Vertical transmission (direct passage from parents to offspring via eggs or sperm) is likely, but not confirmed.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

White tail disease

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

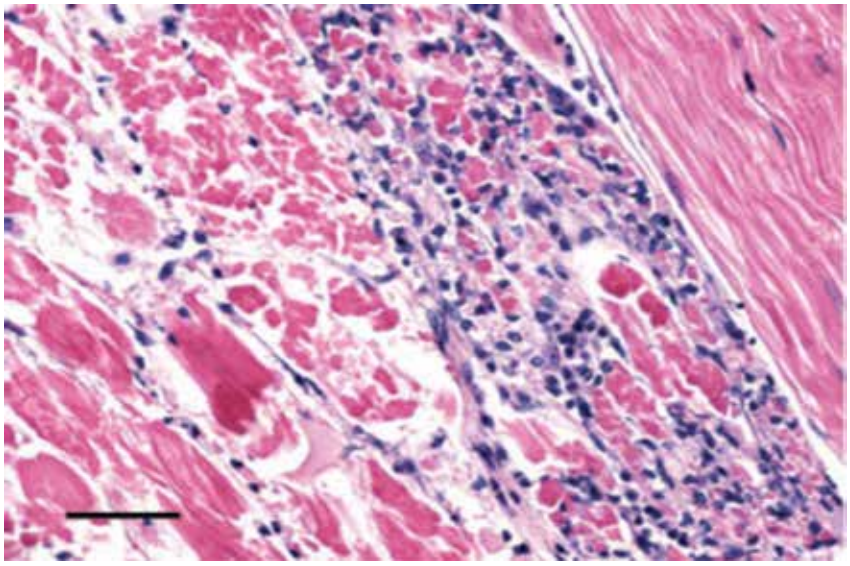
The accepted procedures for a conclusive diagnosis of infectious myonecrosis are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals*. The latest edition of the manual is available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

Further images

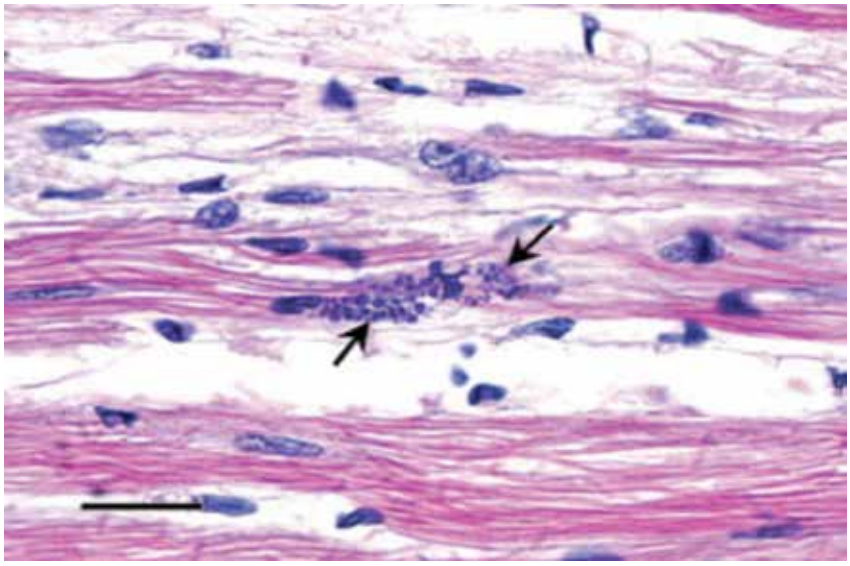
Myonecrosis due to infection of *Litopenaeus vannamei* with purified virions (1–3).

(1) Coagulative necrosis of skeletal muscle accompanied by haemocytic infiltration and fibrosis. Normal skeletal muscle can be observed in the upper right corner. Haematoxylin and eosin stain; bar, 50 µm.



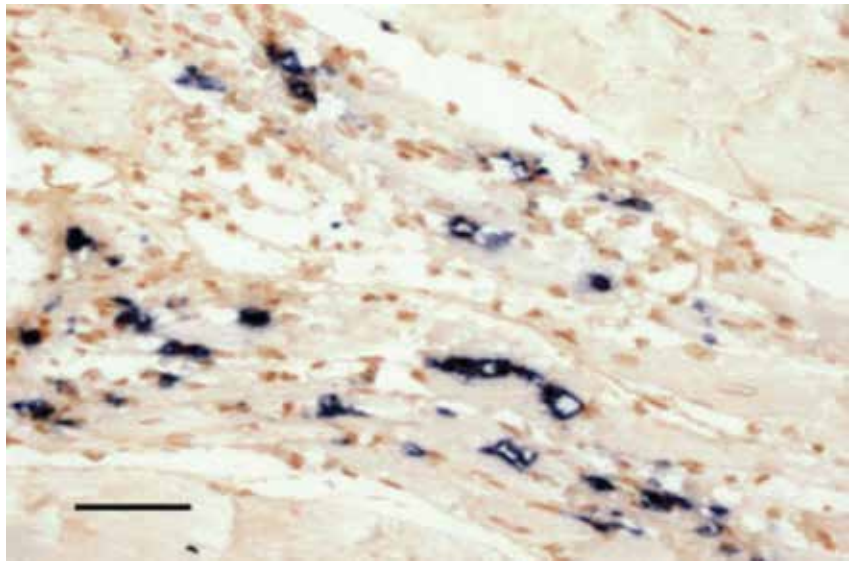
Source: DV Lightner

(2) Perinuclear pale basophilic to dark basophilic inclusion bodies are evident in this group of muscle cells (arrows point at some examples). Haematoxylin and eosin stain; bar, 20 µm.



Source: DV Lightner

(3) In situ hybridisation of skeletal muscle tissue using a digoxigenin-labelled infectious myonecrosis virus probe. A black precipitate is present in areas where the probe has hybridised with target virus. Bismarck brown counterstain; bar, 50 µm.

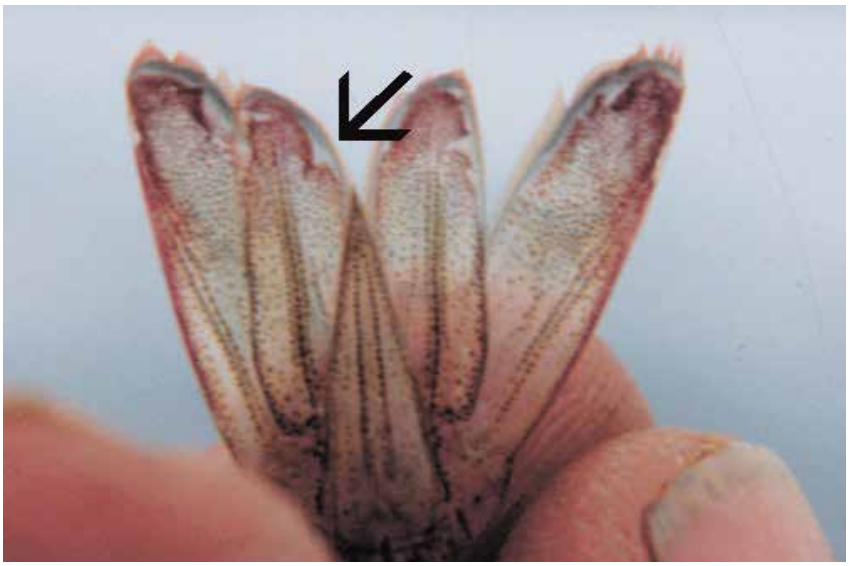


Source: DV Lightner

Taura syndrome

Exotic disease

Taura syndrome in Pacific white shrimp (*Litopenaeus vannamei*); note distinctive red tail fan of Taura syndrome. Rough edges around tail fin are also common.



Source: DV Lightner

Taura syndrome in Pacific white shrimp (*Litopenaeus vannamei*); note darkening of body from infection

Source: DV Lightner

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- lethargy
- cessation of feeding
- animals gathering at the pond edge when moribund
- sudden increase in presence of seabirds 'fishing' in ponds
- sudden onset of high mortalities in late postlarvae, juvenile or subadult prawns.

Taura syndrome has three distinguishable phases: acute, transition and chronic.

Gross pathological signs in the acute phase are:

- empty stomach and pale-red body surface and appendages
- red tail fan and pleopods due to the expansion of red chromatophores
- soft shell.

Gross pathological signs in the transition phase are:

- multiple, irregularly shaped and randomly distributed melanised (dark) cuticular lesions
- death, usually at moulting.

There are no obvious gross pathological signs of disease in the chronic phase.

Microscopic pathological signs are:

- necrosis of the cuticular epithelium of appendages
- multifocal lesions in the cuticular epithelium (evident during the transition phase).

Disease agent

Taura syndrome is caused by Taura syndrome virus, a small picorna-like RNA virus that belongs to the genus *Aparavirus* in the family *Dicistroviridae*.

Host range

Species known to be susceptible to Taura syndrome virus are listed below.

Common name	Scientific name
Black tiger prawn	*Penaeus monodon*
Chinese white shrimp	*Fenneropenaeus chinensis*
Kuruma prawn	*Marsupenaeus japonicus*
Northern brown shrimp	*Farfantepenaeus aztecus*
Northern pink shrimp	*Farfantepenaeus duorarum*
Northern white shrimp **a**	*Litopenaeus setiferus*
Pacific blue shrimp **a**	*Litopenaeus stylirostris*
Pacific white shrimp **a**	*Litopenaeus vannamei*
Red endeavour prawn	*Metapenaeus ensis*
Southern white shrimp	*Litopenaeus schmitti*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Taura syndrome is a disease of the nursery phase of the Pacific white shrimp. It usually occurs within 14–40 days of stocking postlarvae into grow-out ponds or tanks, with mortality from 40% to more than 90%.
- Taura syndrome virus has been documented in postlarvae, juvenile and adult life stages.
- Survivors of infection with Taura syndrome virus may become lifelong carriers.
- Transmission is horizontal through ingestion. Although vertical transmission is suspected, it has not been experimentally confirmed.
- Migratory birds, aquatic insects and humans are likely mechanical vectors of the disease. Individuals surviving the chronic phase of Taura syndrome are thought to be carriers of the virus.
- Resistance of the black tiger prawn and the kuruma prawn to Taura syndrome virus is unclear, but they appear to be more resistant than the Pacific white shrimp.
- Taura syndrome–resistant stocks of the Pacific white shrimp and Pacific blue shrimp are commercially available which have shown survival rates up to 100% following laboratory challenge.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

White spot disease, yellowhead disease

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of Taura syndrome are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

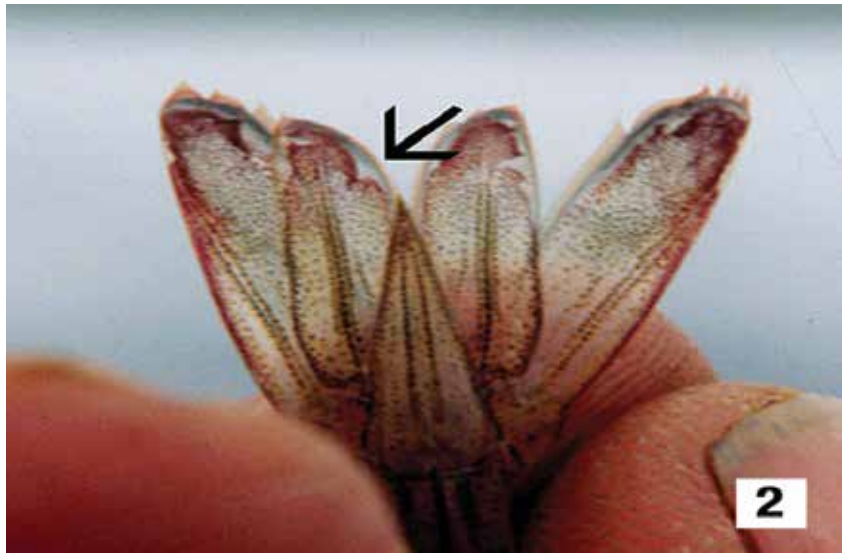
This hyperlink was correct and functioning at the time of publication.

Further images

(1) Moribund, juvenile, pond-reared Pacific white shrimp (*Litopenaeus vannamei*) from Ecuador in the peracute phase of Taura syndrome. The shrimp are lethargic, and have soft shells and a distinct red tail fan.

Source: DV Lightner

(2) A higher magnification (10×) view of the tail fan of one of the two shrimp shown in Figure 1. Use of a hand lens (or the close-up lens on a camera) shows rough edges of the cuticular epithelium in the uropods that are suggestive of focal necrosis of the epithelium at those sites (arrow).



Source: DV Lightner

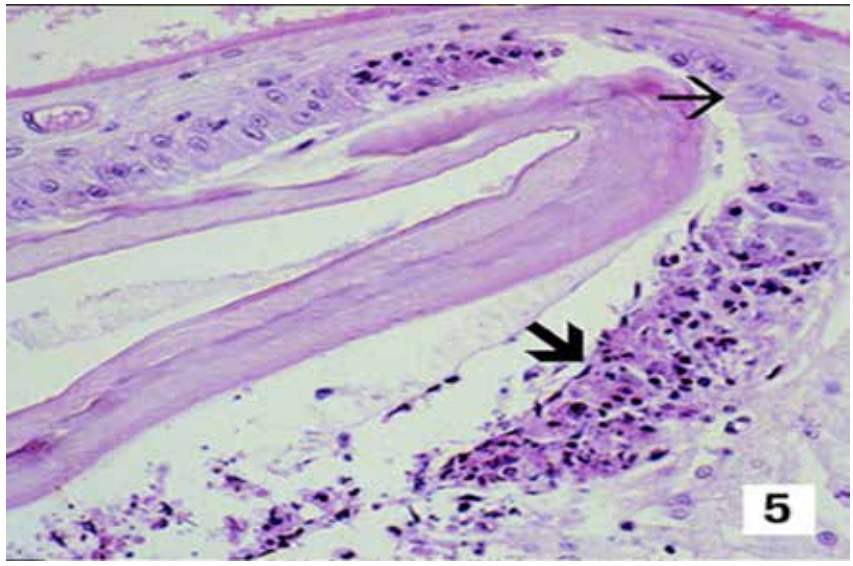
(3) Juvenile, pond-reared Pacific white shrimp (*Litopenaeus vannamei*) from Ecuador in the chronic or recovery phase of Taura syndrome. Multiple melanised foci mark sites of resolving cuticular epithelium necrosis due to Taura syndrome virus infection.

Source: DV Lightner

(4) Juvenile, pond-reared Pacific white shrimp (*Litopenaeus vannamei*) from Texas in the chronic or recovery phase of Taura syndrome. Multiple melanised foci mark sites of resolving cuticular epithelium necrosis due to Taura syndrome virus infection.

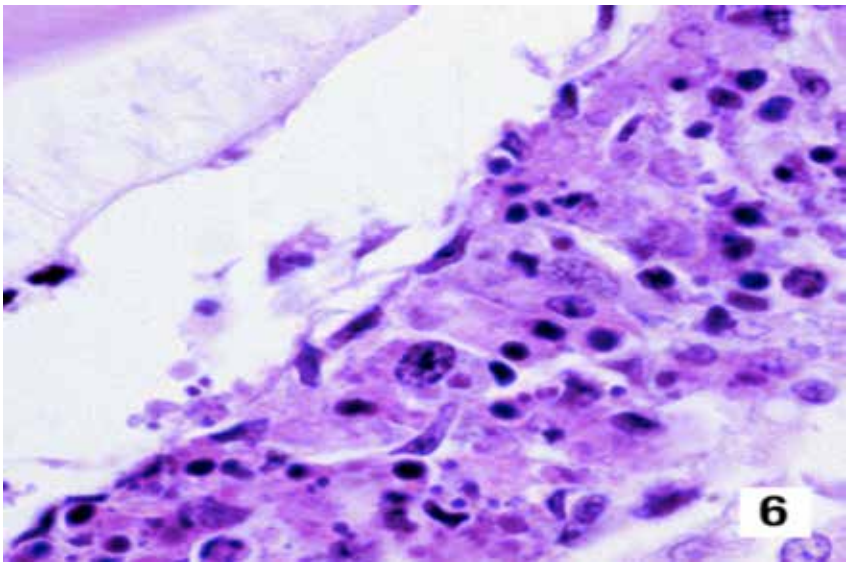
Source: DV Lightner

(5) A histological section through the stomach of a juvenile Pacific white shrimp (*Litopenaeus vannamei*) with peracute Taura syndrome. Prominent areas of necrosis in the cuticular epithelium (large arrow), which secretes the overlying acellular cuticle, are apparent. Adjacent to the focal lesions are normal-looking epithelial cells (small arrow). (300×)



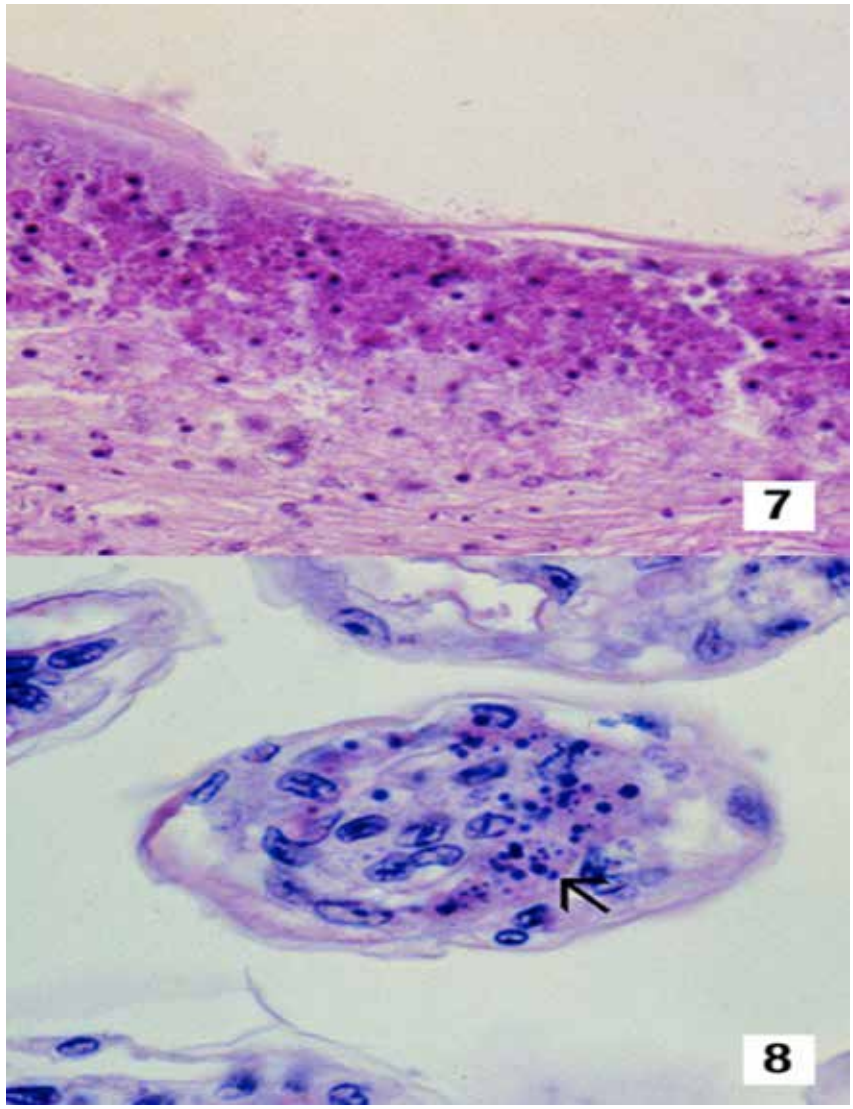
Source: DV Lightner

(6) A higher magnification (900×) of one of the classic peracute phase Taura syndrome lesions near the centre of Figure 5. Classic Taura syndrome lesions consist of necrotic cuticular epithelial and subcuticular connective tissue cells with pyknotic and karyorrhectic nuclei, a generally increased cytoplasmic eosinophilia and very numerous, variably staining, cytoplasmic inclusions. The cytoplasmic inclusions and pyknotic and karyorrhectic nuclei give the lesion a pathodiagnostic 'peppered' or 'buckshot-riddled' appearance. The peracute nature of the lesion is suggested by the absence of haemocytes in or near the lesion.



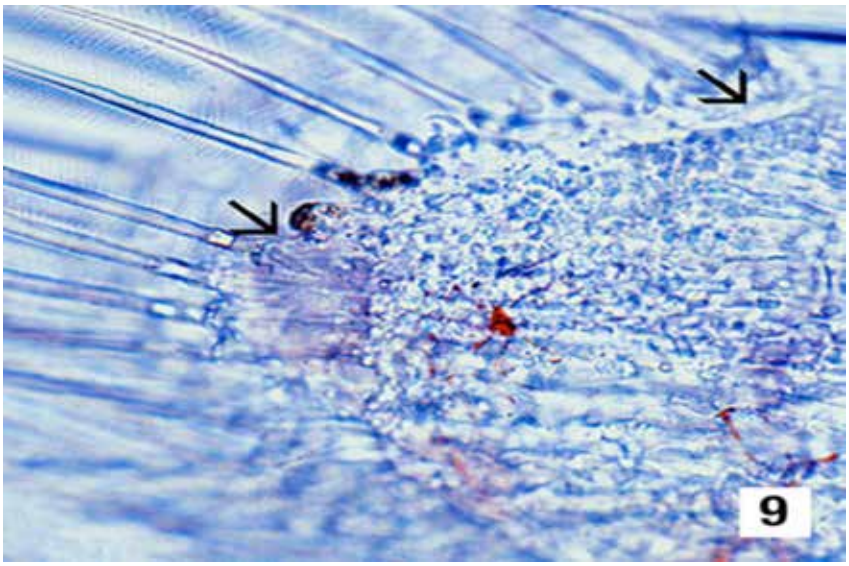
Source: DV Lightner

(7 & 8) Pathognomonic focal Taura syndrome virus lesions in other tissues (other than those shown in Figures 5 and 6) of a juvenile Pacific white shrimp (*Litopenaeus vannamei*) with peracute Taura syndrome. Figure 7 (450×) is a lesion in the cuticular epithelium and subcutis of the carapace; Figure 8 (900×) is in the gills (arrow). Nuclear pyknosis and karyorrhexis, increased cytoplasmic eosinophilia, and an abundance of variably staining, generally spherical cytoplasmic inclusions are distinguishing characteristics of the lesions.



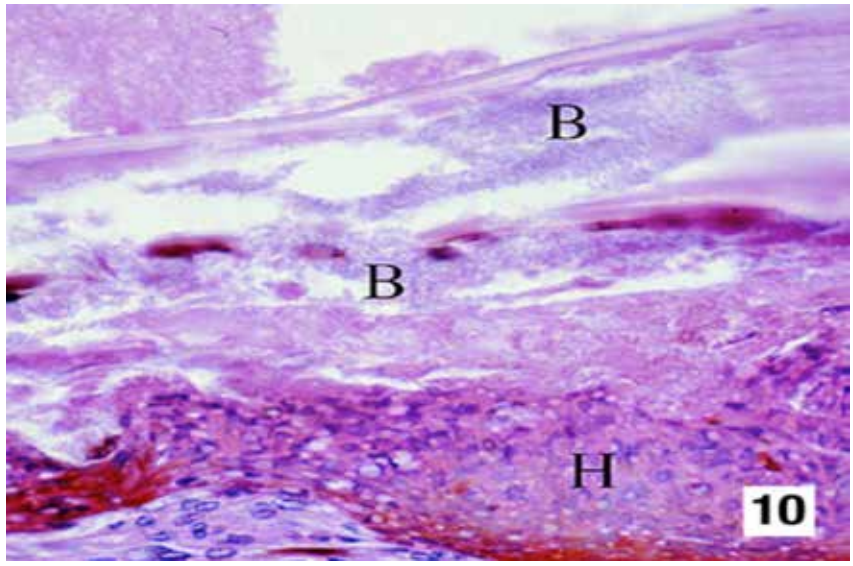
Source: DV Lightner

(9) Unstained wet mount of a uropod of an experimentally infected postlarval Pacific white shrimp (*Litopenaeus vannamei*) in the peracute phase of Taura syndrome. The postlarva was in the D4 stage of its moult cycle, as shown by the presence of the 'old' cuticle separated from the 'new' cuticle by a space. The arrows mark the approximate margins of a focal area of necrosis in the cuticular epithelium. The area of necrosis is evidenced by the presence of a vacant zone just under the cuticular epithelium (where the cuticular epithelium should be) and by the presence of refractile spheres (which are pyknotic and karyorrhectic nuclei) near the periphery of the lesion. A few expanded red chromatophores are also apparent in the subcuticular connective tissues of the uropod. (300×)



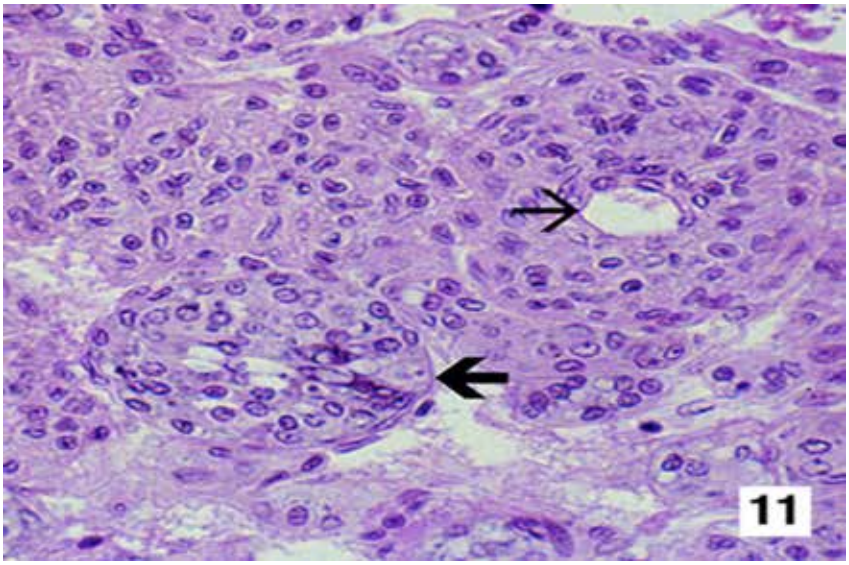
Source: DV Lightner

(10) Histological section (600×) of a resolving cuticular lesion in a juvenile Pacific white shrimp (*Litopenaeus vannamei*). A perforated cuticle that is heavily colonised with masses of bacteria (B) is at the top of the micrograph. A thick, melanised, haemocytic 'plug' (H) has formed basal to the cuticular epithelium to temporarily close the 'wound' from the outside. Basal to the haemocyte plug (H), connective tissue elements, and additional infiltrating haemocytes, provide the basal support for the regeneration of the cuticular epithelium. Pathognomonic Taura syndrome lesions in the recovery/chronic phase of Taura syndrome are usually few, relative to the resolving lesions shown here, and are often entirely absent.



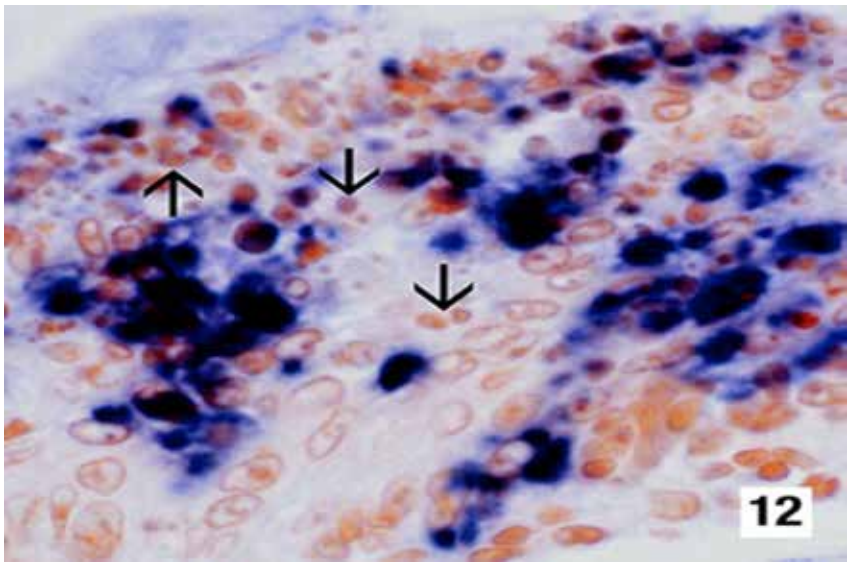
Source: DV Lightner

(11) Mid-sagittal section (450×) of the lymphoid organ (LO) of an experimentally infected juvenile Pacific white shrimp (*Litopenaeus vannamei*) in the chronic or recovery phase of Taura syndrome. Although pathognomonic Taura syndrome lesions of the type seen in the cuticular epithelium never occur in the LO, Taura syndrome virus does induce some significant lesions in this organ. Interspersed among normal-looking LO cords or tissue, which is characterised by multiple layers of sheath cells around a central haemolymph vessel (small arrow), are accumulations of disorganised LO cells that form LO 'spheroids' (LOS). LOS lack a central vessel and consist of cells that show karyomegaly and large, prominent cytoplasmic vacuoles and other cytoplasmic inclusions (large arrow).



Source: DV Lightner

(12) Histological section (900×) of an appendage from a postlarval Pacific white shrimp (*Litopenaeus vannamei*) in the peracute phase of Taura syndrome that has been reacted with a digoxygenin (DIG)-labelled cDNA probe to Taura syndrome virus. The probe has reacted intensely with Taura syndrome virus–infected cells, staining the cytoplasm of infected cuticular epithelial cells and subcuticular connective tissue cells positive for the virus. The probe does not react with the pyknotic and karyorrhectic nuclei (arrows), because Taura syndrome virus is only cytoplasmic. These nuclear remnants contribute to the 'peppered' or 'buckshot-riddled' appearance of TS lesions.



Source: DV Lightner

White spot disease

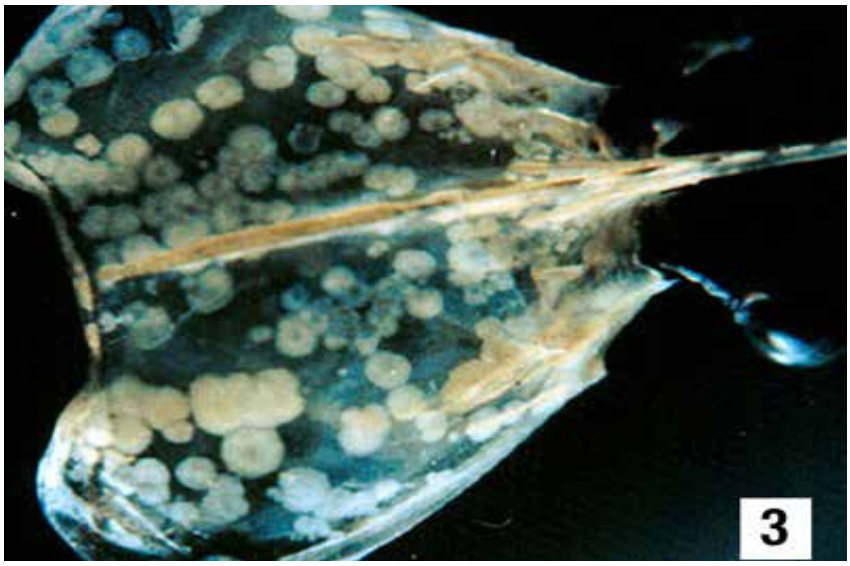
(Also known as infection with white spot syndrome virus)

Exotic disease

White spot disease in giant black tiger prawn (*Penaeus monodon*). Prawns at top and right of main photo show pink body colour typical of the acute phase of infection, while those at the bottom and to the left show classic white spots following the acute phase.

Source: DV Lightner

White spot disease in giant black tiger prawn (*Penaeus monodon*), showing classic white spots on the carapace



Source: DV Lightner

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank and pond level are:

- rapid onset of mass mortality (80% or more) in farmed penaeid prawns during the grow-out period
- lethargy
- cessation of feeding
- aggregations of moribund prawns near the water surface at the edge of the rearing pond or tank.

Gross pathological signs are:

- loose carapace
- high degrees of colour variation, with a predominance of darkened (red-brown or pink) body surface and appendages
- heavy fouling of the surface and gills by external parasites
- white midgut line through the abdomen of severely affected larvae and postlarvae
- white calcium deposits embedded in the shell, causing white spots 0.5–3.0 mm in diameter
- delayed (or sometimes completely absent) clotting reaction of the haemolymph of infected shrimp.

Prawns with white spot disease may not show distinctive clinical signs. If present, shell lesions can range from minute spots to discs that are several millimetres in diameter, and may coalesce into larger plates. They are most easily observed by removing the cuticle over the cephalothorax, scraping away any attached tissue with the thumbnail and holding the cuticle up to the light. White spots in the cuticle are unreliable, even for preliminary diagnosis of white spot disease because similar spots can be produced by some bacteria, high alkalinity and other infectious or environmental conditions.

Microscopic pathological signs are:

- hypertrophied nuclei in gills and/or cuticular epithelium
- viral aggregates (shown as small reflective spots) in unstained smear preparations of the haemolymph by dark-field microscopy
- pathognomonic inclusion bodies in histological sections in target tissues.

Disease agent

The causative agent of white spot disease is white spot syndrome virus, a large DNA virus assigned as the only member of the genus *Whispovirus* (family *Nimaviridae*). The virus infects only crustaceans and appears not to be related to any other known viruses. It is not involved in the parasitic disease, common in finfish, also known as white spot. The virus is known to occur in fresh, brackish and marine water.

Host range

All decapod crustaceans (order Decapoda), including prawns, lobsters and crabs from marine, brackish or freshwater environments, are considered susceptible to infection. However, the disease has mainly been a problem in farmed penaeid (family Penaeidae) prawns.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Although many species of crustaceans are susceptible to infection, white spot syndrome virus is mainly a disease of farmed penaeid prawns.
- Rapid mortalities have been reported in many countries of up to 80% or more within 3–10 days.
- Experience has shown that prawn farm productivity falls to about 40% of normal for 2 years, and then recovers to about 70% over the long term.
- Resistance to white spot syndrome virus has not been reported for any of the penaeid species listed above.
- Infection may be low level and chronic (lifelong carriers are possible) in healthy crustaceans, or acute with disease and mortalities.
- Viral multiplication and disease appears to be induced by environmental and handling stress such as eye-stalk ablation, spawning, moulting, changes in salinity, temperature and pH and during plankton blooms. Imposing such stressors on suspect populations can a useful method to increase the probability of detecting virus.
- All life stages are potentially susceptible, from eggs to broodstock.
- Vertical transmission occurs from infected broodstock.
- Horizontal transmission of disease is usually via cannibalism of sick or dying prawns, or directly through contaminated water.
- Although not required for transmission, vectors of the virus include rotifers, marine molluscs, polychaete worms and non-decapodal crustaceans including *Artemia salina*, the copepods, non-crustacean arthropods and insect larvae.
- Birds can transmit the disease from pond to pond by releasing caught prawns over neighbouring ponds.
- White spot syndrome virus can persist and retain infectivity in seawater at 30ºC for at least 30 days (under laboratory conditions) and for at least 4 days in ponds.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infectious hypodermal and haematopoietic necrosis virus, necrotising hepatopancreatitis, Taura syndrome, yellowhead disease

The microscopic signs described and shown here may also be symptomatic of infectious hypodermal haematopoietic necrosis. Further laboratory examination is needed for a definitive diagnosis.

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

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Further reading

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This hyperlink was correct and functioning at the time of publication.

Further images

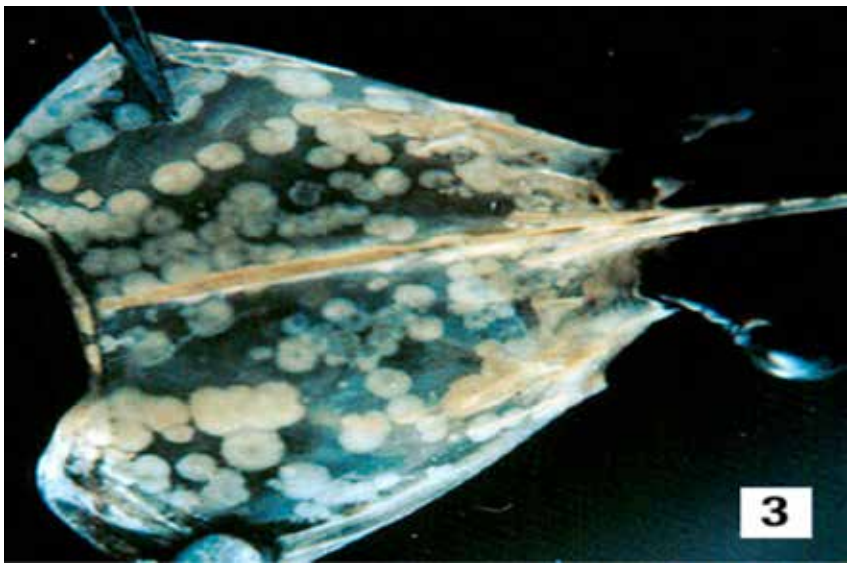
(1) A juvenile giant black tiger prawn (*Penaeus monodon*) that is displaying the distinctive white spots of white spot disease. White spots are especially visible on the carapace and the rostrum. Although providing a tentative diagnosis of white spot disease infection, white spots are not always visible in shrimp in the acute phase of the disease, and may develop in the subacute to chronic or recovery phases of the infection.

Source: DV Lightner

(2) Four juvenile black tiger prawns (*Penaeus monodon*), including the one shown in Figure 1 (at bottom), with different gross signs of white spot disease. The top and right shrimp show few, if any, white spots, but show a pink to red-brown discolouration due to expansion of the subcuticular chromatophores. This reddish appearance may be a gross sign that is more apparent in the acute phase of the disease. The shrimp on the left and bottom display diagnostic white spots that develop after the acute phase of the disease.

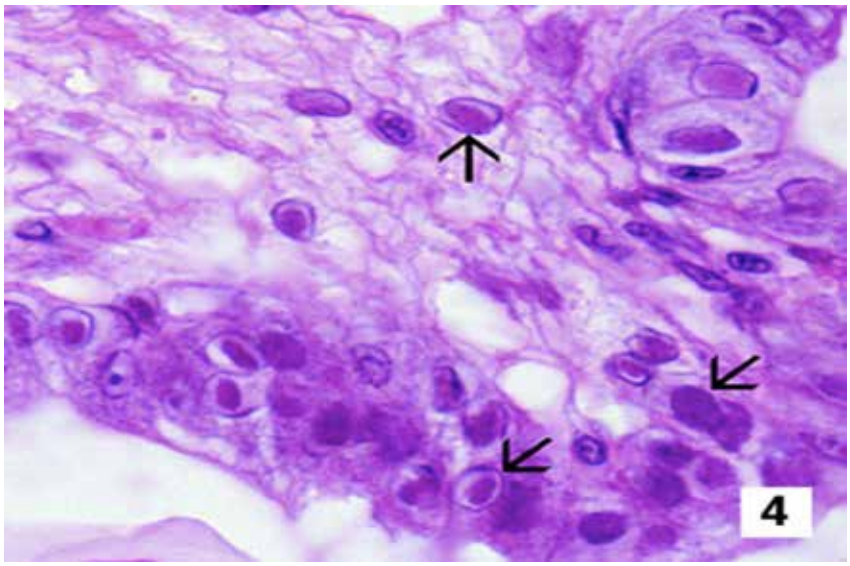
Source: DV Lightner

(3) The carapace from a juvenile black tiger prawn (*Penaeus monodon*) with white spot disease. White spots on the underside of the shell are caused by calcareous deposits. Photo: P Saibaba, SKBR College, Amalapuram, India



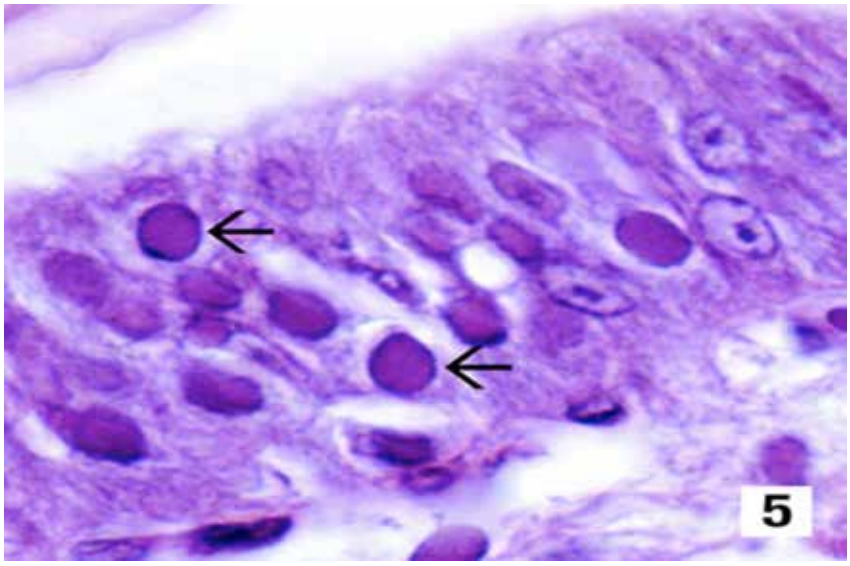
Source: DV Lightner

(4) Photomicrograph (900×) of a histological section from the stomach of a juvenile black tiger prawn (*Penaeus monodon*) infected with white spot disease. Prominent intranuclear inclusion bodies are abundant in the cuticular epithelium and subcuticular connective tissue of the organ (arrows). Cells in different phases of infection display intranuclear inclusion bodies. The early-phase inclusion bodies that predominate in this section are centronuclear and eosinophilic, and are separated from the nuclear membrane and marginated chromatin by an artifactual halo (resembling infectious hypodermal and haematopoietic necrosis inclusion bodies).



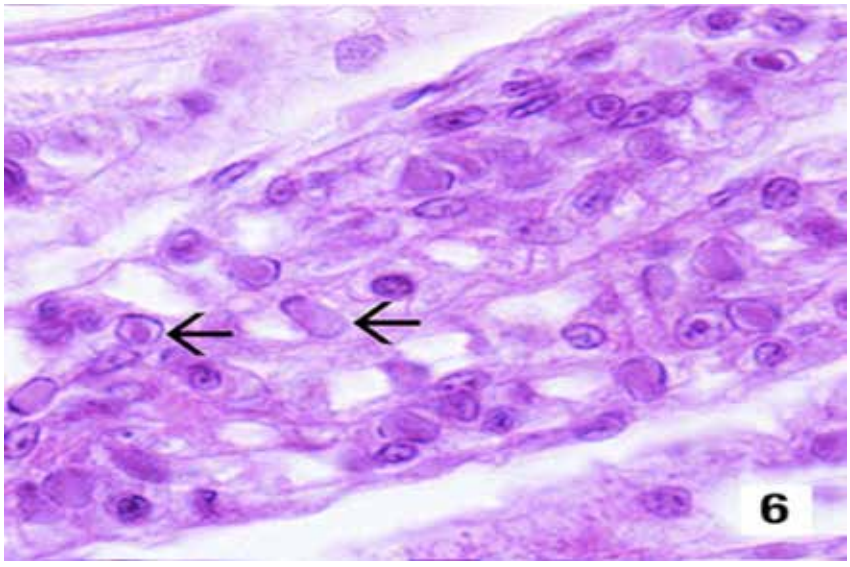
Source: DV Lightner

(5) Histological section (1300×) of the stomach of a juvenile Chinese white shrimp (*Fenneropenaeus chinensis*) with an advanced white spot disease infection. Fully developed white spot disease intranuclear inclusion bodies (arrows) are more basophilic, appear granular in texture and nearly fill the affected hypertrophied nucleus. Inclusion bodies are absent.



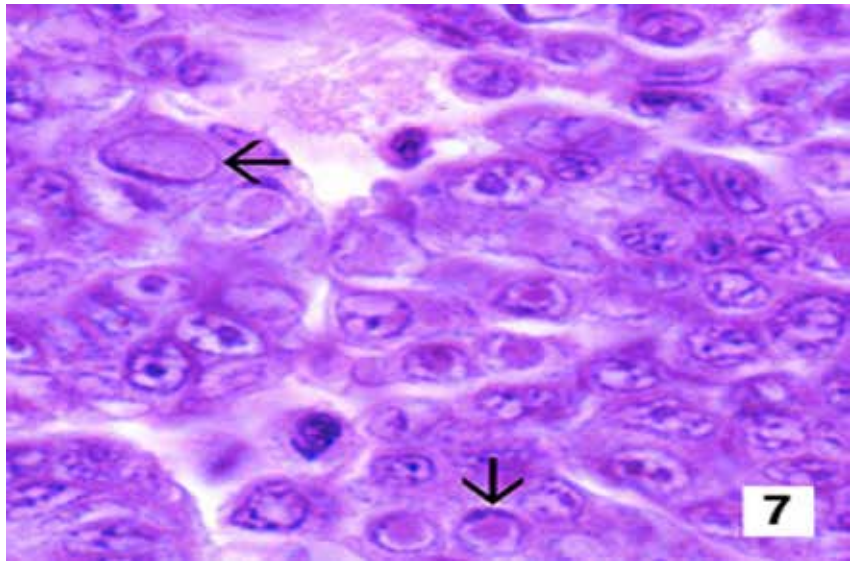
Source: DV Lightner

(6) Section of the gills from a juvenile Chinese white shrimp (*Fenneropenaeus chinensis*) with white spot disease (900×). Nearly one-quarter of the cells are infected, as indicated by the developing and fully developed intranuclear inclusion bodies of white spot disease (arrows).



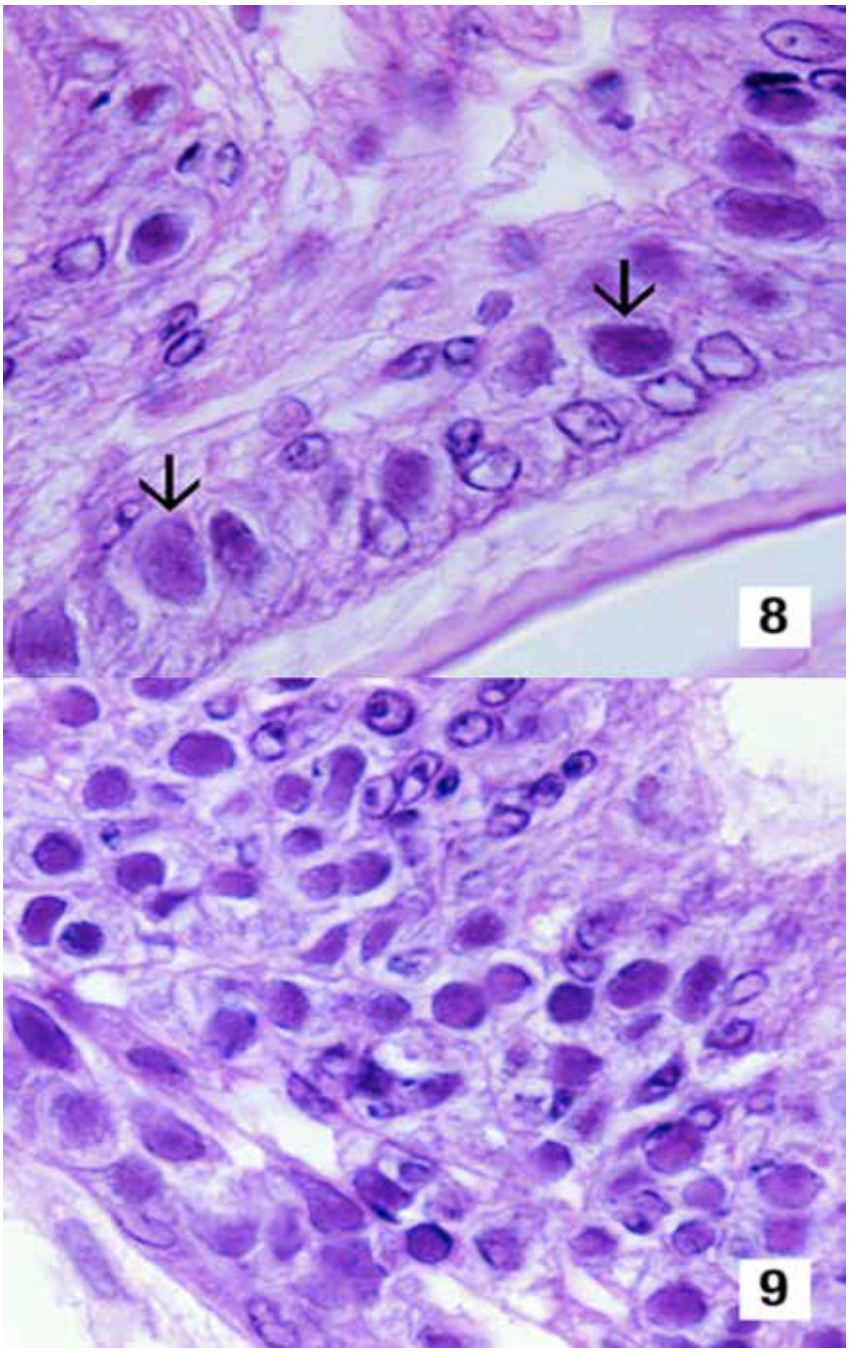
Source: DV Lightner

(7) Section of a white spot disease–infected haematopoietic nodule (1300×) from a juvenile Chinese white shrimp (*Fenneropenaeus chinensis*). As in Figure 6, nearly one-quarter of the cells in the section display intranuclear inclusions bodies of white spot disease (arrows) in various stages of development.



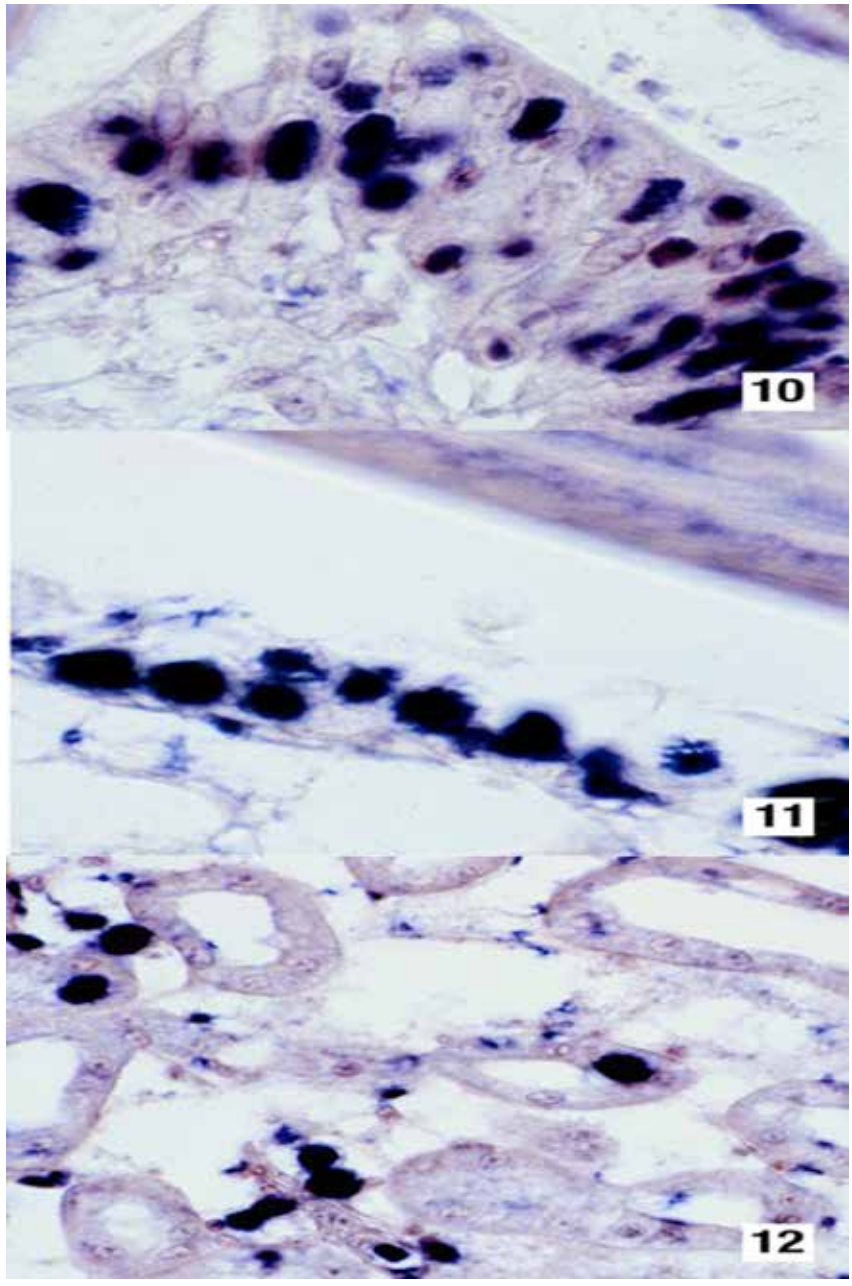
Source: DV Lightner

(8 & 9) Histological sections (900×) of the stomachs of Pacific blue shrimp (*Litopenaeus stylirostris*, Figure 8) and Pacific white shrimp (*L. vannamei*, Figure 9) experimentally infected with white spot disease. Both species display severe (grade 4) infections, with classic white spot disease intranuclear inclusion bodies (arrows) that are identical to those illustrated in Figures 3–7.



Source: DV Lightner

(10, 11 & 12) Sections of various tissues from a white spot disease–infected juvenile Pacific white shrimp (*Litopenaeus stylirostris*) reacted by in situ hybridisation with a digoxygenin (DIG)-labelled DNA probe to the virus. The probe has reacted strongly with intranuclear inclusion bodies containing the disease in the various tissues of this shrimp, including the cuticular epithelium of the stomach (Figure 10, 900×), the cuticular epithelium and connective tissues of the carapace (Figure 11, 900×), and epithelial cells in the antennal gland (Figure 12, 450×).



Source: DV Lightner

White tail disease
(Also known as white muscle disease [WMD])

Giant freshwater prawn (*Macrobrachium rosenbergii*) postlarvae showing white tail disease

Source: AS Sahul Hameed

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- presence of whitish postlarvae, followed by mortality 2–3 days after the conversion of first postlarva in larval rearing tanks
- lethargy
- mortality of up to 95% within 5 days after the appearance of the first gross signs.

Gross pathological signs are:

- particularly milky and opaque abdomen (tail), starting at the tail extremity (telson region) and gradually progressing towards the headabnormal appearance of floating moults in the tanks, which resemble 'mica flakes'
- degeneration of telson and uropods (in severe cases)
- white colouration of abdominal muscle.

Microscopic pathological signs are:

- acute Zenker's necrosis of striated muscles, characterised by severe hyaline degeneration, necrosis and muscular lysis
- pathognomonic basophilic intracytoplasmic inclusion bodies in infected muscle tissues.

Disease agent

The causative agent is *Macrobrachium rosenbergii* nodavirus (*Mr*NV) and extra small virus (XSV).

Both these viruses are associated with the disease, but their respective roles are uncertain. These viruses are known to occur in fresh and brackish water.

Host range

Species known to be susceptible to white tail disease are listed below:

Common name	Scientific name
Giant freshwater prawn **a**	*Macrobrachium rosenbergii*

a Naturally susceptible

Presence in Australia

White tail disease has been officially reported from Queensland.

Epidemiology

- Very few postlarvae showing the clinical signs of white tail disease survive. Survivors seem to grow normally in grow-out ponds.
- Outbreaks most commonly occur in larvae, postlarvae and early juveniles. Adult life stages are resistant, and act as carriers.
- Transmission is both vertical (trans-ovum) and horizontal (from virus present in the water surrounding susceptible prawns or direct contact with an infected prawn).
- Mortality rates are variable and reach up to 95%.
- Some penaeid shrimp, *Artemia* and aquatic insects are vectors of white tail disease.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Infectious myonecrosis

The clinical signs described and shown here may also be symptomatic of other bacterial or viral infections, or poor water quality. Further laboratory examination is needed for a definitive diagnosis.

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The currently accepted procedures for a conclusive diagnosis of white tail disease are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

Yellowhead disease
(Also known as infection with yellowhead virus)

Yellowhead disease in giant black tiger prawn (*Penaeus monodon*); note yellow heads of infected prawns on the left. Prawns on the right are normal.

Source: DV Lightner

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- aggregation of moribund prawns near the surface at pond edges
- abnormally high feeding rate of infected 5–15-gram prawns for several days and then cessation of feeding
- mass mortality (up to 100%) 2–4 days after cessation of feeding.

Gross pathological signs are:

- yellowing of the cephalothorax and general bleaching of the body
- white, yellow or brown gills
- exceptionally soft digestive gland compared with normal
- yellow, swollen digestive gland, making the head appear yellow.

Microscopic pathological signs are:

- moderate to large numbers of deeply basophilic, evenly stained, spherical, cytoplasmic inclusions within tissues of ectodermal and mesodermal origin.

Disease agent

The causative agent of yellowhead disease is yellowhead virus, a corona-like RNA virus in the genus *okavirus*, family *Roniviridae* and order Nidovirales. Yellowhead virus (genotype 1) is one of six known genotypes in the yellowhead complex of viruses and is the only known agent of yellowhead disease. Gill-associated virus is designated as genotype 2 (covered separately within the field guide). Four other known genotypes in the complex (genotypes 3–6) occur commonly in black tiger prawn (*Penaeus monodon*) in East Africa, Asia and Australia, and are rarely or never associated with disease.

Host range

Yellowhead disease is highly infectious for most known species of cultivated penaeid prawns. Natural and/or experimental infections have been reported to occur in the following species of penaeid and palaemonid shrimps, prawns and krill.

Common name	Scientific name
Giant freshwater prawn **a**	*Macrobrachium rosenbergii*
Barred estuarine shrimp	*Palaemon serrifer*
Black tiger prawn **a**	*Penaeus monodon*
Brown tiger prawn	*Penaeus esculentus*
Greentail prawn	*Metapenaeus bennettae*
Kuruma prawn **a**	*Metapenaeus japonicus*
Mysid shrimp **a**	*Palaemon styliferus*
Northern brown shrimp	*Farfantepenaeus aztecus*
Northern pink shrimp	*Farfantepenaeus duorarum*
Northern white shrimp **a**	*Litopenaeus setiferus*
Pacific blue shrimp	*Litopenaeus stylirostris*
Pacific white shrimp	*Litopenaeus vannamei*
Paste prawn **a**	*Ascetes* spp.
Red endeavour prawn **a**	*Metapenaeus ensis*
Sunda river prawn	*Macrobrachium sintangene*
White banana prawn **a**	*Fenneropenaeus merguiensis*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

There are variations in the susceptibility of different species to disease. Laboratory trials have shown that yellowhead virus can cause high mortality in black tiger prawns, Pacific white shrimp, Pacific blue shrimp, northern brown shrimp, northern pink shrimp, Sunda river prawns, mysid shrimp and barred estuarine shrimp. Until proven otherwise, it should be assumed that most penaeid prawns worldwide are susceptible to infection with yellowhead virus.

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- The tiger prawn suffers acute epidemics, with mortality reaching 100% within 3–5 days of the first appearance of the gross signs.
- Yellowhead virus can be transmitted horizontally by injection, ingestion of infected tissue, immersion in membrane-filtered tissue extracts or cohabitation with infected shrimp. Transmission has also been demonstrated by injection of extracts of paste prawns (*Ascetes* sp.) collected from infected ponds.
- Vertical transmission occurs from both male and female parents, possibly via surface infection or contamination of tissue surrounding fertilised eggs.
- Tiger prawns younger than 15 days postlarvae are resistant to yellowhead virus in comparison with those from postlarval days 20–25 to subadult, which are highly susceptible.
- Mortalities usually occur during the early to late juvenile stages in rearing ponds.
- There appear to be at least four genotypes of virus in the yellowhead virus group. Genotype 1 is the causative agent of yellowhead disease.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

Necrotising hepatopancreatitis, Taura syndrome, white spot disease

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

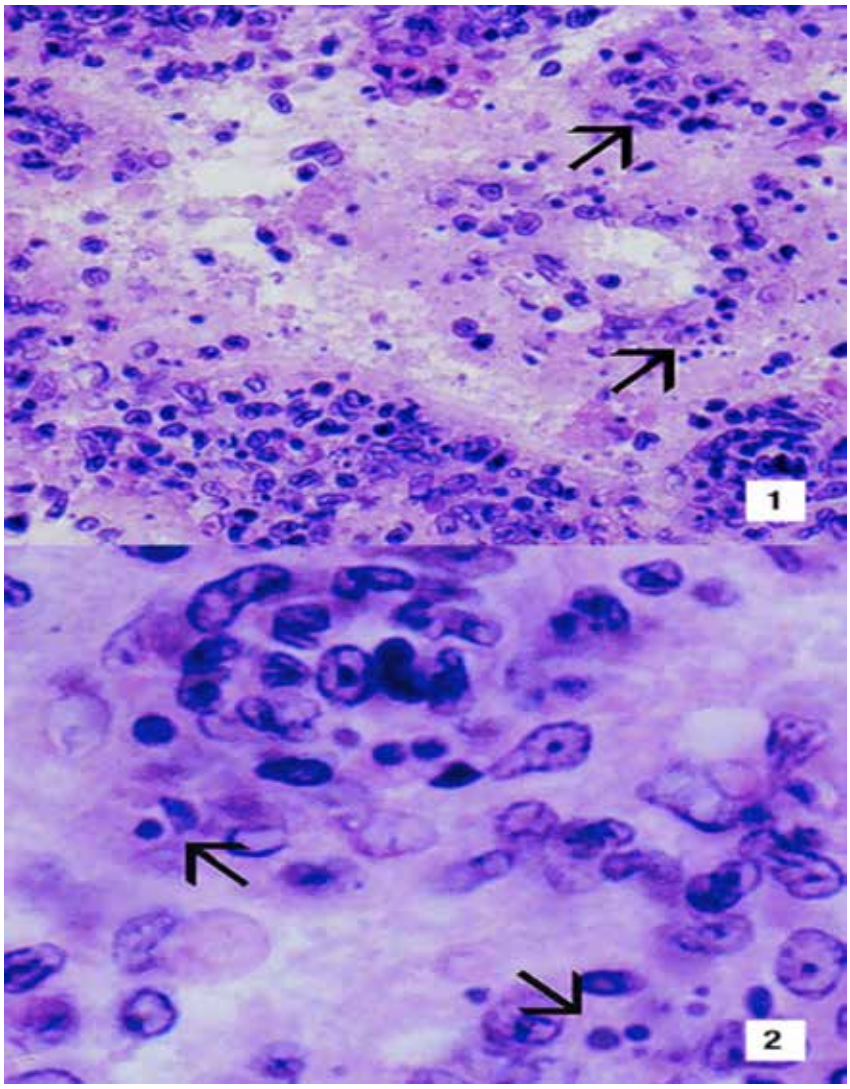
Further reading

The accepted procedures for a conclusive diagnosis of yellowhead disease are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

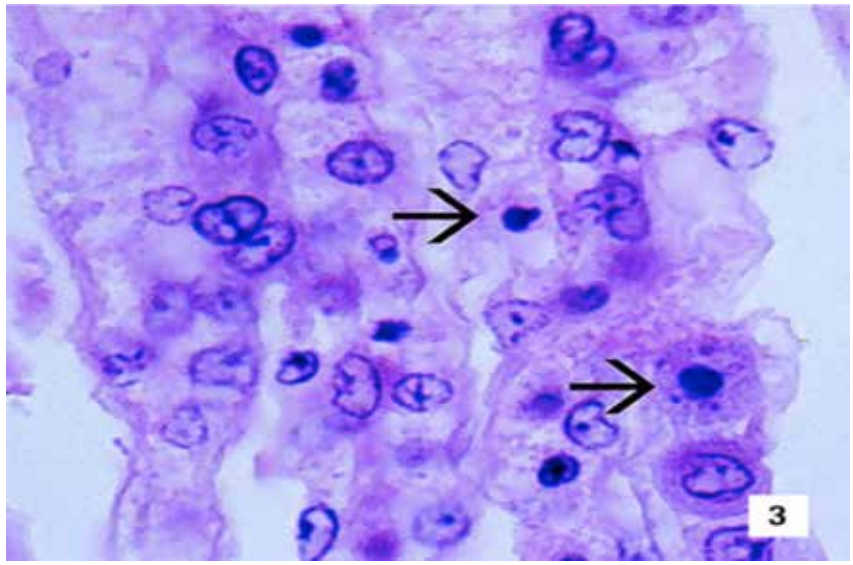
Further images

(1 & 2) Histological section of the lymphoid organ (LO) of a juvenile giant black tiger prawn (*Penaeus monodon*) with severe acute yellowhead disease at low (525×, Figure 1) and high (1700×, Figure 2) magnification. A generalised, diffuse necrosis of LO cells is shown. Affected cells display pyknotic and karyorrhectic nuclei. Single or multiple perinuclear inclusion bodies, ranging from pale to darkly basophilic, are apparent in some affected cells (arrows). This marked necrosis in acute yellowhead disease distinguishes it from infection due to Taura syndrome virus, which produces similar cytopathology in other target tissues, but not in the LO.



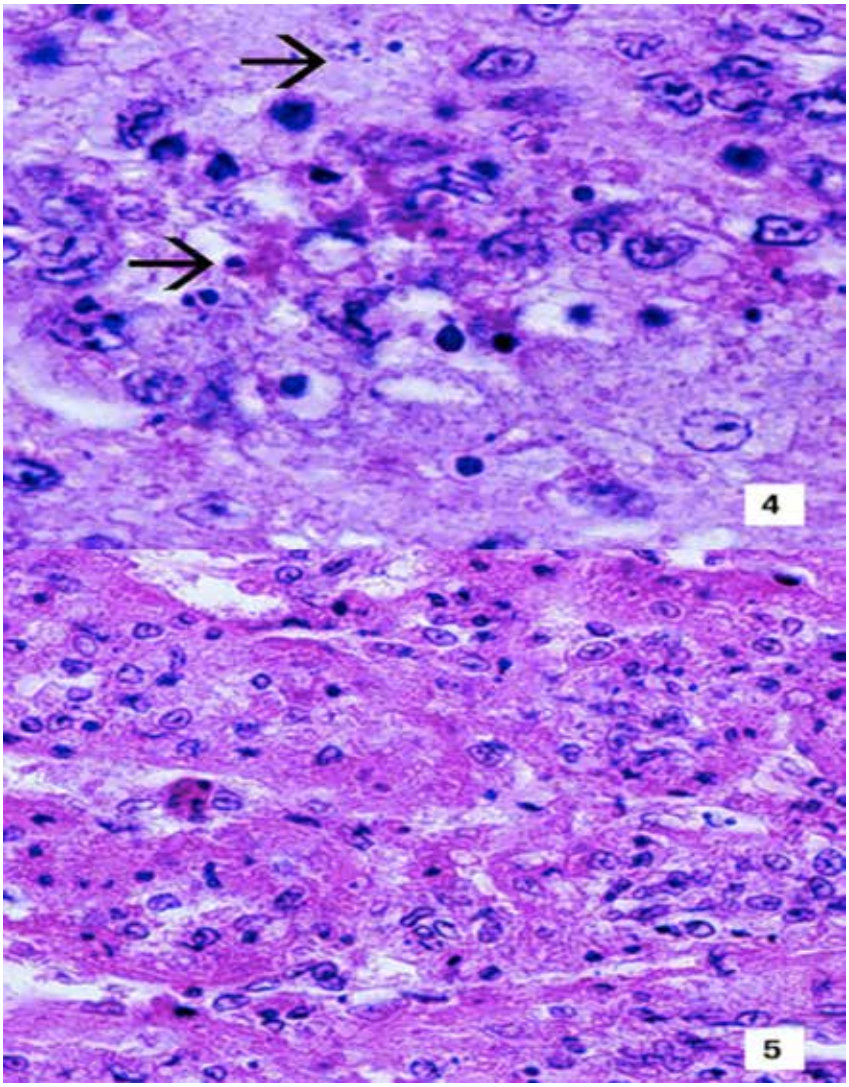
Source: DV Lightner

(3) Histological section (1000×) of the gills from a juvenile black tiger prawn (*Penaeus monodon*) with yellowhead disease. A generalised, diffuse necrosis of cells in the gill lamellae is shown, and affected cells display pyknotic and karyorrhectic nuclei (arrows). A few large, conspicuous, generally spherical cells with basophilic cytoplasm are present in the section. These cells may be immature haemocytes, released prematurely in response to a yellowhead virus–induced haemocytopaenia.



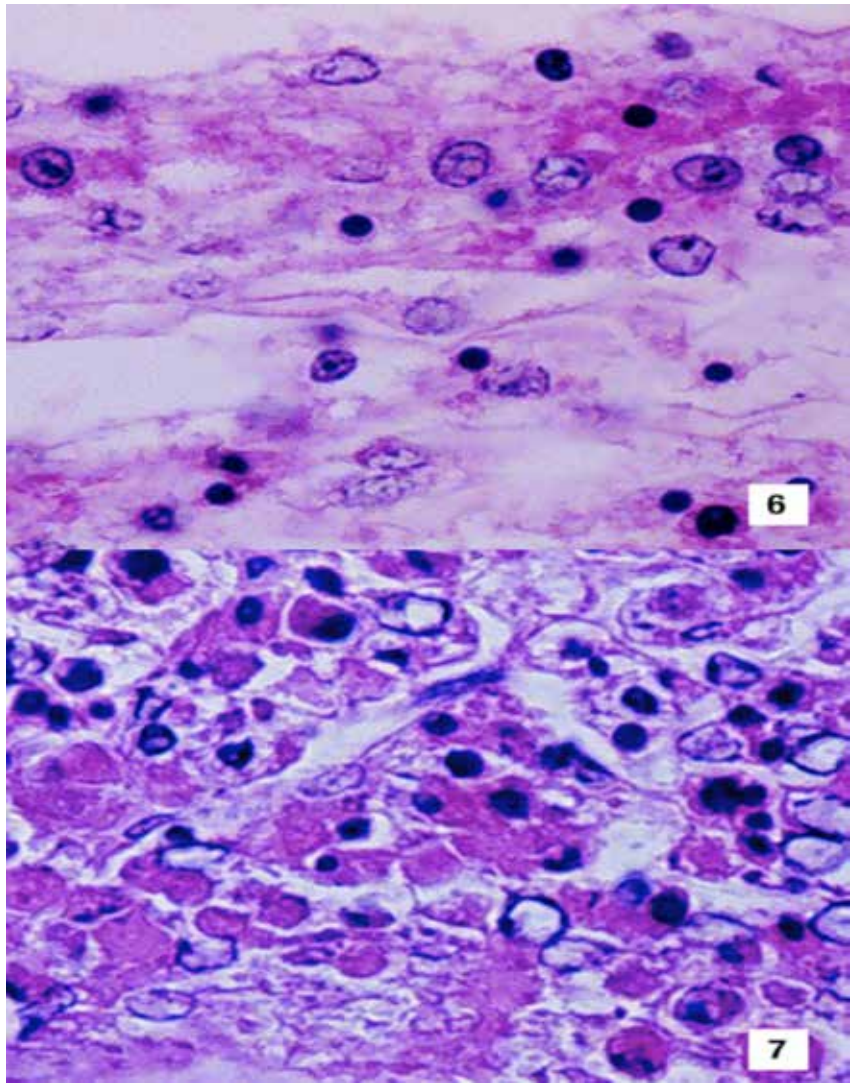
Source: DV Lightner

(4 & 5) Histological sections of the lymphoid organ (LO) of juvenile Pacific white shrimp (*Litopenaeus vannamei*) (Figure 4, 1000×) and northern brown shrimp (*Farfantepenaeus aztecus*) (Figure 5, 525×) experimentally infected with yellowhead virus. Severe (grade 3–4) diffuse to multifocal necrosis, characterised by cells with increased eosinophilic cytoplasm, pyknotic or karyorrhectic nuclei (arrows) and pale to densely basophilic perinuclear inclusions, is present.



Source: DV Lightner

(6 & 7) Histological sections (1000×) of the gills of a juvenile northern pink shrimp (*Farfantepenaeus duorarum*) (Figure 6) and the oesophagus of a Pacific white shrimp (*Litopenaeus vannamei*) (Figure 7) experimentally infected with yellowhead virus. Severe (grade 4) diffuse to multifocal necrosis, characterised by cells with increased eosinophilic cytoplasm, pyknotic or karyorrhectic nuclei, and pale to densely basophilic perinuclear inclusions, is present.



Source: DV Lightner

Bacterial diseases of crustaceans

Exotic disease

Milky haemolymph disease of spiny lobster (*Panulirus* spp.)
(Also known as milky haemolymph syndrome [MHS])

MHS in a *Panulirus ornatus*

Source: DV Lightner

MHS in a *Panulirus ornatus*

Source: DV Lightner

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- lethargy
- anorexia or cessation of feeding
- mortality soon after clinical and gross pathological signs appear.

Gross pathological signs are:

- milky haemolymph exuding from wounds or visible under swollen abdominal pleura of the exoskeleton (visible on the ventral side; if drawn into a syringe, may appear turbid or milky in severely affected specimens)
- white hypertrophied connective tissues of all major organs and tissues.

Microscopic pathological signs are:

- basophilic cytoplasmic masses of bacteria in haemocytes, fixed phagocytes and connective tissue cells.

Disease agent

Milky haemolymph disease of spiny lobster is caused by a rickettsia-like bacterium. Four distinct rickettsia-like bacteria have been found, one of which is known to be associated with the disease.

Host range

Species known to be susceptible to milky haemolymph disease of spiny lobster are listed below.

Common name	Scientific name
Black tiger prawn **a**	*Penaeus monodon*
European shore crab **a**	*Carcinus maenas*
Tropical spiny lobsters **ab**	*Panulirus* spp.

a Naturally susceptible. **b** Species primarily susceptible to milky *haemolymph* disease of spiny lobster include the ornate rock lobster (*P. ornatus*), scalloped spiny lobster (*P. homarus*) and Chinese spiny lobster (*P. stimpsoni*).

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- It is suspected that horizontal transmission occurs by direct contact with infected lobsters in the same net-pens, or indirectly by contaminated water from adjacent net-pens.
- The disease has been experimentally transmitted among lobsters by cohabitation and by injection of unfiltered haemolymph from diseased lobsters into healthy lobsters.
- Haemolymph exuding from wounds or when drawn into a syringe is turbid, appearing milky in severely affected specimens.
- The haemolymph from severely affected individuals does not clot.
- The disease affects three-month-old or older juveniles and adult lobsters.

- It is suspected that fresh foods (fishery bycatch, molluscs and decapod crustaceans) fed to net-pen-reared lobsters in Vietnam are the source of the rickettsia-like bacterial agent of the disease.
- Injection of oxytetracycline has been effective in treating and preventing the disease.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to milky haemolymph disease of spiny lobster.

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information (a disease information card) can be found on the World Organisation for Animal Health website at www.oie.int/en/international-standard-setting/specialists-commissions-groups/aquatic-animal-commission-reports/disease-information-cards.

This hyperlink was correct and functioning at the time of publication.

Exotic disease

Necrotising hepatopancreatitis (NHP)

(Also known as infection with necrotising hepatobacterium or NHP bacterium)

Pacific white shrimp (*Litopenaeus vannamei*) with NHP; note darkening at base of swimmerets, giving a fouled, 'dirty' appearance

Source: DV Lightner

Pacific white shrimp (*Litopenaeus vannamei*) with NHP; note marked reduction in size of hepatopancreas

Source: DV Lightner

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- lethargy
- emaciation
- heavy protozoan or bacterial fouling
- reduced growth rate.

Gross pathological signs are:

- soft shell
- flaccid body
- black gills
- empty intestinal tract
- degenerated or atrophied digestive gland (hepatopancreas), which appears pale to white
- black (melanised) streaks in the hepatopancreas.

Microscopic pathological signs are:

- multifocal granulomatous lesions in hepatopancreatic tubules, with atrophy of adjacent hepatopancreatic tubule epithelial cells
- tubular cells within the granulomatous lesions that can be hypertrophied and contain basophilic organisms within the cytoplasm
- sloughing of tubule epithelial cells
- severe haemocytic inflammation of the intratubular spaces.

Four distinct phases of infection have been described: initial, acute, transition and chronic. Acute and transition phases are identifiable by the presence of pathognomonic lesions in the hepatopancreas. Molecular techniques are required for positive diagnosis of NHP bacterium infected individuals in the initial or chronic phase of infection.

Disease agent

NHP is caused by infection with a Gram-negative, intracytoplasmic species of alpha-proteobacterium that infects the hepatopancreas of prawns, also referred to as NHP bacterium. The NHP bacterium exists in two morphological forms: a rod-shaped, non-flagellated, rickettsia-like organism; and a helical, flagellated form.

Host range

Species known to be susceptible to NHP are listed below.

Common name	Scientific name
Northern brown shrimp **a**	*Farfantepenaeus aztecus*
Northern white shrimp **a**	*Litopenaeus setiferus*
Pacific blue shrimp **a**	*Litopenaeus stylirostris*
Pacific white shrimp **a**	*Litopenaeus vannamei*
Yellow-leg shrimp **a**	*Fenneropenaeus californiensis*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- NHP outbreaks are often preceded by lengthy periods of high water temperatures (29–31 °C) and elevated salinity (up to 40 parts per thousand).
- Mortality can be 90–95% within 30 days of an outbreak.
- Mortalities usually occur midway through the grow-out phase.
- NHP appears to be transmitted by direct ingestion of carrier prawns (survivors of NHP bacterial infection may carry the bacteria for life) and through contaminated water.
- NHP bacteria may also be shed in faeces and contribute to disease transmission.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to NHP. Any presumptive diagnosis requires further laboratory diagnosis for confirmation.

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

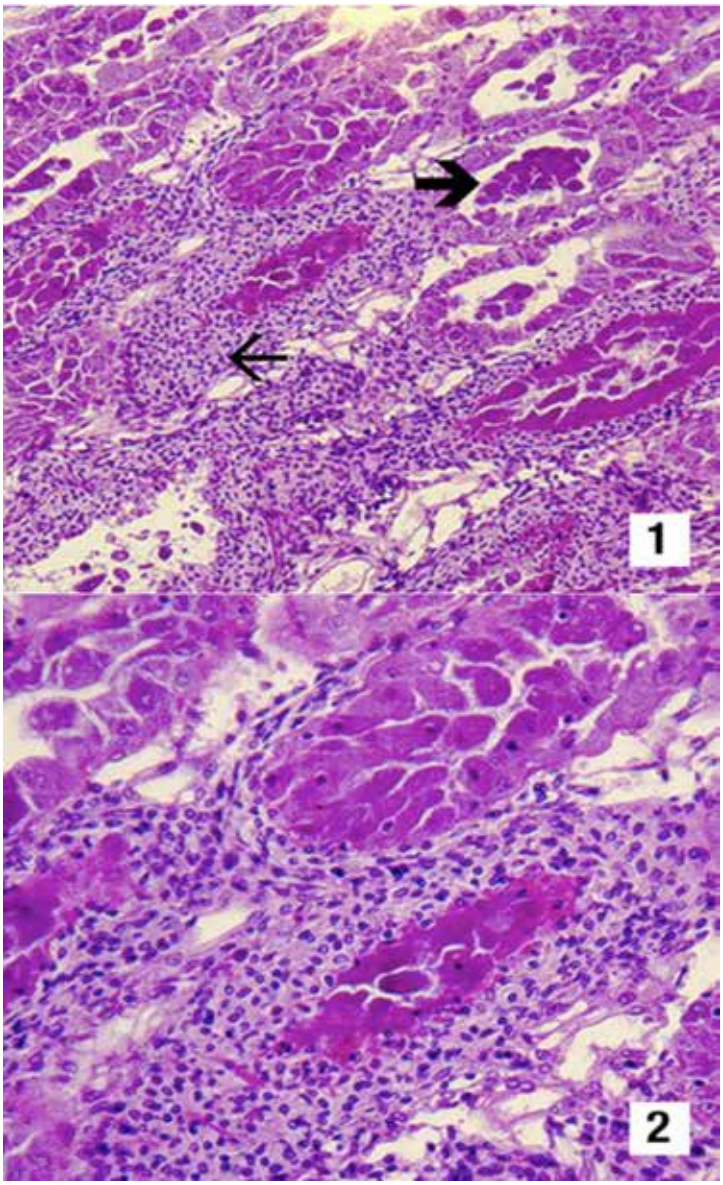
Further reading

Further information can be found on the disease pages of Fisheries and Oceans Canada at www.pac.dfo-mpo.gc.ca/science/species-especes/shellfish-coquillages/diseases-maladies/index-eng.htm.

This hyperlink was correct and functioning at the time of publication.

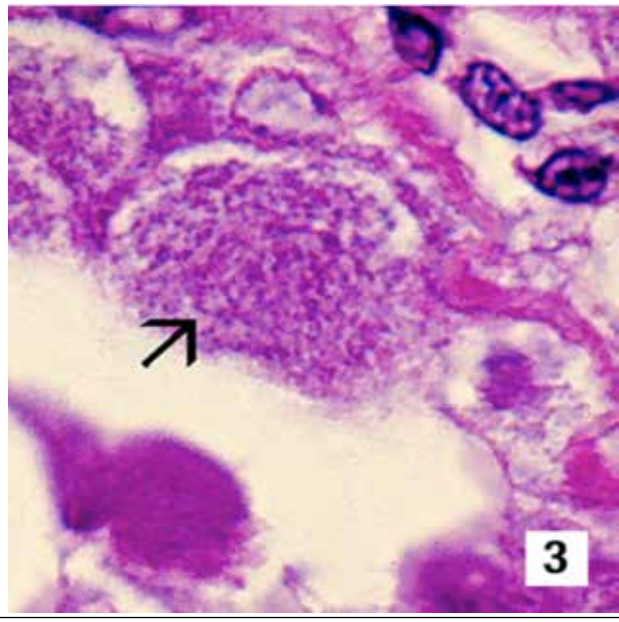
Further images

(1 & 2) Low-magnification (Figure 1, 150×) and mid-magnification (Figure 2, 300×) photomicrographs of the hepatopancreas of a juvenile Pacific white shrimp (*Litopenaeus vannamei*) with severe, subacute (grade 3–4) NHP. Severe haemocytic inflammation (with some melanised foci) of the intratubular spaces (small arrow) in response to necrosis, cytolysis and sloughing of hepatopancreas tubule epithelial cells (large arrow) are among the principal histopathological changes due to the disease.



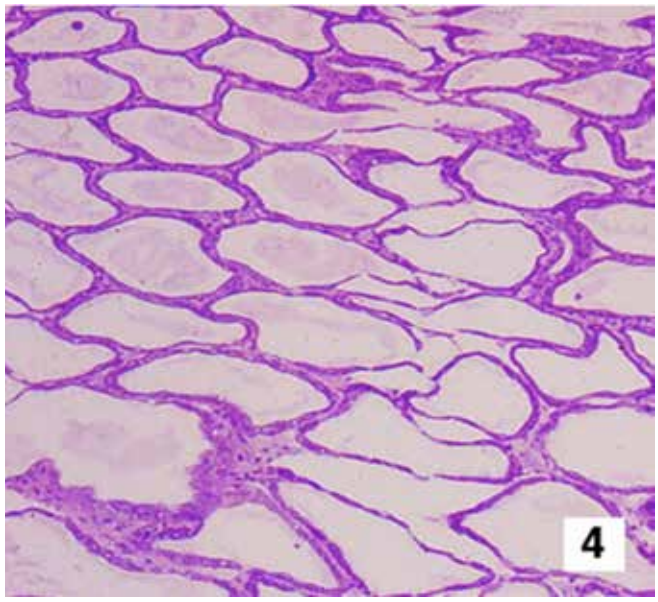
Source: DV Lightner

(3) A high-magnification (1700×) view of a portion of the hepatopancreas from Figures 1 and 2. The tubule epithelial cells show no cytoplasmic lipid droplets, but instead contain masses of the tiny, non–membrane bound, intracytoplasmic NHP bacteria (arrow).



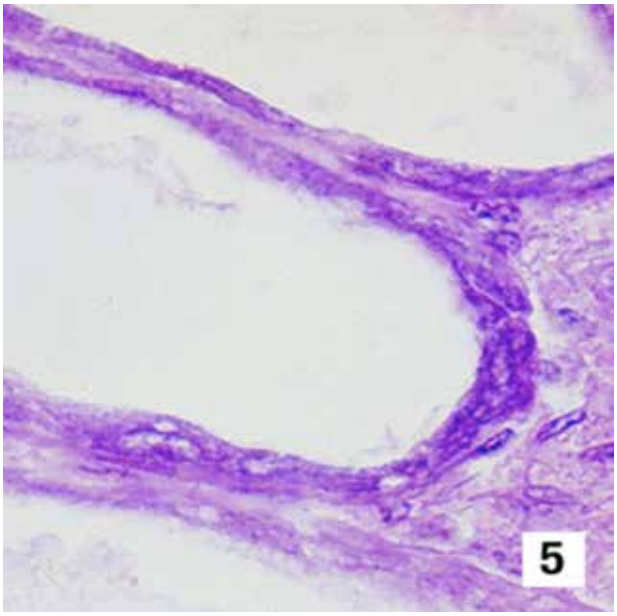
Source: DV Lightner

(4) Low-magnification (100×) view of the hepatopancreas of a juvenile Pacific white shrimp (*Litopenaeus vannamei*) with severe, chronic NHP. The hepatopancreas tubule epithelium is markedly atrophied, resulting in the formation of large oedematous (fluid filled or 'watery') areas.



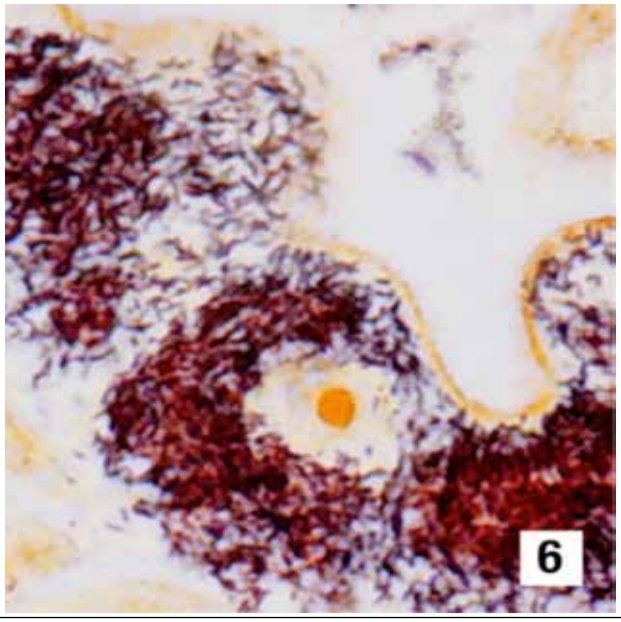
Source: DV Lightner

(5) A higher magnification (900×) photomicrograph of the atrophied hepatopancreas from a juvenile Pacific white shrimp (*Litopenaeus vannamei*) with chronic NHP. In contrast to the subacute phase of NHP, chronic-phase NHP shows no, or only occasional, foci of haemocytic inflammation of the necrotic or degenerating hepatopancreatic tubules. NHP bacteria may be found in the cytoplasm of an occasional hepatopancreatic cell.



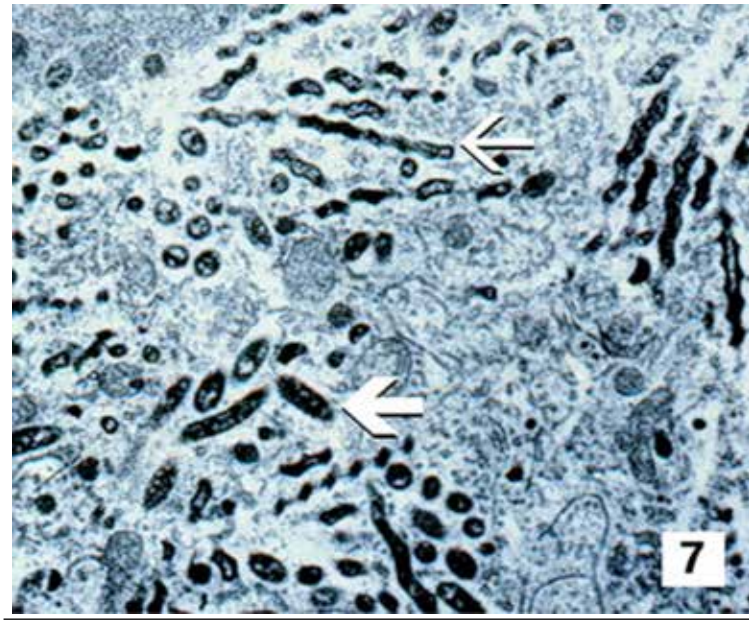
Source: DV Lightner

(6) Section of the hepatopancreas of a juvenile Pacific white shrimp (*Litopenaeus vannamei*) that is similar to that shown in Figure 3. Cytoplasmic masses of the NHP bacterium are silver stained and appear brown to black with the modified Steiner stain. Unaffected cells and nuclei are pale brown (1600×).



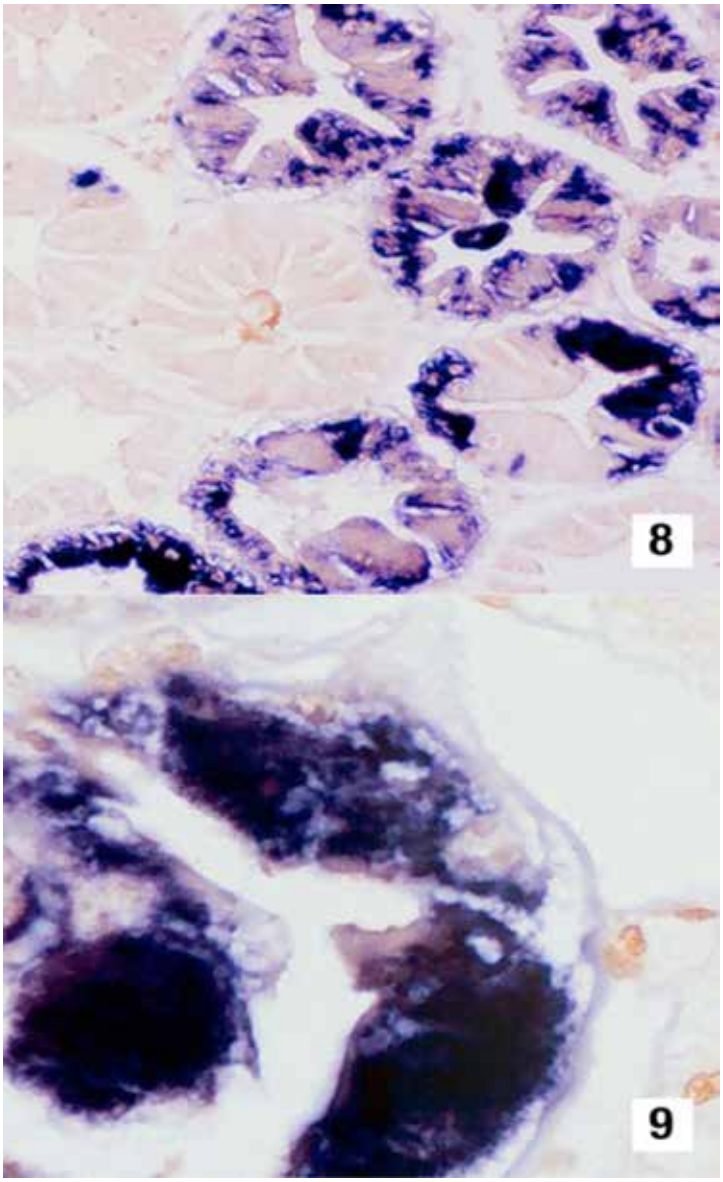
Source: DV Lightner

(7) Transmission electron micrograph (10 000×) of a hepatopancreatocyte from a juvenile Pacific white shrimp (*Litopenaeus vannamei*) with NHP. Profiles of intracellular rod-shaped forms (large arrow) and helical forms (small arrow) of the NHP bacterium are abundant in the cytoplasm.



Source: DV Lightner

(8 & 9) Above and right: Low-magnification (Figure 8, 250×) and high-magnification (Figure 9, 1000×) views of sections of the hepatopancreas of a juvenile Pacific white shrimp with NHP. This section has been assayed for NHP using a digoxygenin (DIG)-labelled DNA probe. Cytoplasmic masses of the NHP bacterium are marked blue to blue-black by the probe. Unaffected cells and host cell nuclei take the brown counter stain. DIG-labelled NHP probe and Bismarck Brown.



Source: DV Lightner

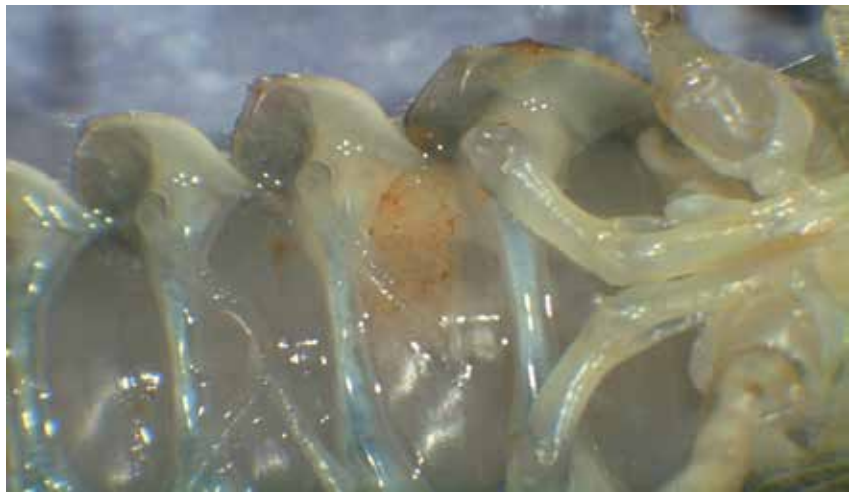
Exotic disease

Other diseases of crustaceans

Crayfish plague

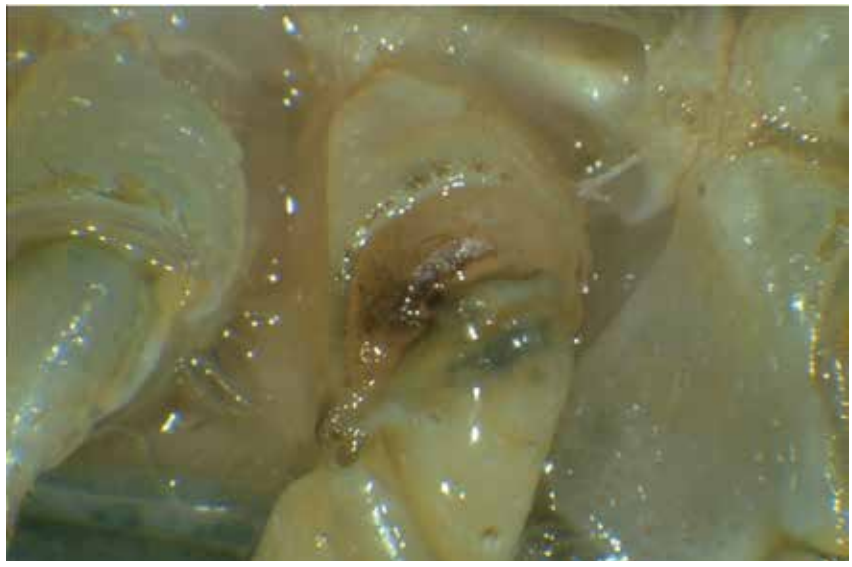
(Also known as infection with *Aphanomyces astaci*)

Crayfish plague, showing typical brown markings on a segment; healthy muscle tissue is present on either side of the affected segment



Source: D Alderman

Crayfish plague, showing classic darkening at base of walking legs



Source: D Alderman

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, tank or pond level are:

- high mortality at the time of the initial outbreak
- many dead or weak crayfish floating or lying in watercourses or ponds (mortalities may go unnoticed in the wild)
- crayfish in open water during daylight hours
- unsteady and raised gait ('walking on stilts')
- weakened, rapid tail escape response
- crayfish unable to remain upright (more evident when out of water)
- progressive paralysis
- crayfish trying to scratch or pinch themselves (occasionally seen).

Gross pathological signs are:

- growth on soft, non-calcified parts of the carapace
- brown or black spots on the carapace, where hyphae proliferate
- fine black lines on the soft shell underneath the tail
- melanised (black) shell in chronically infected individuals
- death occurring within weeks of the initial infection (particularly in European crayfish, *Astacus* sp.)
- white and necrotic musculature in the tail.

Microscopic pathological signs are:

- aseptate hyphae on the cuticle.

Disease agent

Crayfish plague is caused by infection with the oomycete *Aphanomyces astaci*. Although previously regarded as a fungus, the genus *Aphanomyces* is now classified with diatoms and brown algae in a group called Stramenopiles or Chromista.

This pathogen is known to occur in freshwater only.

Host range

It is believed that all species of freshwater crayfish are susceptible to crayfish plague. Species known to be susceptible are listed below.

Common name	Scientific name
Chinese mitten crab	*Eriocheir sinensis*
Crayfish (native to the eastern states of America)	*Orconectes* spp.
Freshwater crayfish	*Cherax* spp.
Giant Tasmanian crayfish	*Astacopsis gouldi*
Gippsland spiny crayfish	*Euastacus kershawi*
Japanese crayfish **a**	*Cambaroides japonicus*
Louisiana swamp crayfish **a**	*Procambarus clarkii*
Noble crayfish **a**	*Astacus astacus*
Signal crayfish **a**	*Pacifastacus leniusculus*
Stone crayfish **a**	*Austropotamobius torrentium*
Turkish crayfish **a**	*Astacus leptodactylus*
White-clawed crayfish **a**	*Austropotamobius pallipes*

a Naturally susceptible (other species have been shown to be experimentally susceptible)

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- Mortalities of up to 100% have occurred in Europe, with local loss of susceptible populations.
- North American crayfish (signal crayfish, Louisiana swamp crayfish, *Orconectes* sp.) can be infected without showing clinical signs or succumbing to the disease, but can become carriers of the disease agent and a source of transmission to less resistant species of crayfish.
- The disease was introduced into Europe in American freshwater crayfish and has decimated European crayfish stocks (both wild and cultured). There has been no evidence of developing resistance to the disease among European species since its introduction.
- Crayfish plague can occur at any time of year, but is more likely in the summer months.
- Death occurs between 5 and 50 days (or more) from initial infection, depending on water temperature and the initial number of zoospores.
- *A. astaci* releases motile zoospores directly to the water column when crayfish die. This is the primary transmission mechanism whereby motile zoospores of *A. astaci* are able to swim actively in the water column and show positive movement towards other crayfish.
- Translocation and migration of fish, birds and other wildlife can allow them to act as vectors, transporting the disease to previously unexposed waters.
- *A. astaci* can be introduced to a new susceptible population on contaminated ropes, traps, fishing gear, boots, nets and other equipment.
- Infection with *A. astaci* may be suspected when mortalities are observed to be limited to highly susceptible species of freshwater crayfish (where all other flora and fauna, particularly other crustaceans, are normal and healthy).

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

None of the other diseases in this field guide result in the rapid mortality or other gross signs of crayfish plague. In Australia, infection with the microbial parasite *Thelohania* (or porcelain disease) may cause similar gross signs.

Initial misdiagnosis has occurred when pollution has resulted in mortality of aquatic crustaceans where other species have survived.

In a few cases, examination by light microscopy can further define a diagnosis; however, further laboratory examination is always required for a definitive diagnosis.

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of crayfish plague are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information can be found on the Australian Government Department of Agriculture, Fisheries and Forestry website at www.daff.gov.au/animal-plant-health/aquatic/aquavetplan.

These hyperlinks were correct and functioning at the time of publication.

Exotic disease

Monodon slow growth syndrome (MSGS)

Penaeus monodon with monodon slow growth syndrome

Source: E Burreson

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the farm, pond or tank level are:

- abnormally slow growth, resulting in irregularly sized prawns
- low average daily weight gain of less than 0.1 g/day at 4 months of age.

Gross pathological signs are:

- unusually dark colour
- unusually bright yellow markings
- brittle antennae
- bamboo-shaped abdominal segments

There are no definitive microscopic pathological signs.

Disease agent

The complete aetiology for MSGS is uncertain and there isn't a clear case definition for this syndrome. A working case definition for surveillance and data gathering purposes is listed under epidemiology. A key component is the positive detection of Laem-Singh virus (LSNV) by RT-PCR (LSNV is considered a necessary but insufficient component cause). Known pathogens are unlikely to be the cause of MSGS and previous trials have indicated that a filterable infectious agent is involved.

Host range

Species known to be susceptible to MSGS are listed below.

Common name	Scientific name
Black tiger prawn **a**	*Penaeus monodon*

a Naturally susceptible

Presence in Australia

EXOTIC DISEASE—not present in Australia.

Epidemiology

- As the cause of MSGS is uncertain, a working case definition allows for suspected case data to be collected if the population:
  - test positive for Laem-Singh virus (LSNV)
  - has a coefficient of variation of more than 35% by weight
  - is free from hepatopancreatic parvovirus infection or any other hepatopancreatic infection
  - shows three of the signs of disease listed previously.
- In countries where the Pacific white shrimp (*Litopenaeus vannamei*) has already been introduced, Pacific white shrimp and black tiger prawns should be reared separately, particularly at the maturation and hatchery phase.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases listed in this field guide are similar to monodon slow growth syndrome.

Sample collection

Due to the uncertainty associated with differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Further information can be found by searching 'monodon slow growth syndrome' on the Network of Aquaculture Centres in Asia–Pacific website at www.enaca.org.

This hyperlink was correct and functioning at the time of publication.

6 Diseases of amphibians

Viral diseases of amphibians

Infection with *Ranavirus*

Ornate burrowing frog (*Limnodynastes ornatus*) metamorphs with ascites due to infection with Bohle iridovirus, a species of *Ranavirus*

Source: R Speare

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the tank or pond level (or in the wild) are:

- erratic swimming
- lethargy
- loss of equilibrium
- buoyancy problems
- morbidity
- rapid severe mortality events.

Note that behavioural changes differ between species, life stages and severity of disease.

Gross pathological signs are:

- poor body condition
- lordosis (abnormal forward curvature of the spine).

In tadpoles and metamorphs, additional gross pathological signs are:

- ascites (free fluid in the abdominal cavity)
- generalised oedema (fluid swelling) of tissues
- focal haemorrhages.

In adults, additional gross pathological signs are:

- skin ulcers
- focal and systematic haemorrhages
- oedema of subcutaneous tissue (particularly around the jaw and head, with the tongue protruding).

Microscopic pathological signs are:

- severe renal, pulmonary, hepatic, splenic and haematopoietic necroses and haemorrhages
- basophilic intracytoplasmic inclusion bodies
- erosion, ulceration and hyperplasia of epithelial epidermal cells.

Disease agent

The disease is caused by infection with members of the genus *Ranavirus* (family *Iridoviridae*). Species include frog virus 3 (FV-3), Bohle iridovirus (BIV), epizootic haematopoietic necrosis virus (EHNV), European catfish virus (ECV), European sheatfish virus (ESV) and Santee-Cooper ranavirus. There are many other tentative species in this genus. Bohle iridovirus is known from northern Australia in native frogs and cane toads.

Host range

Amphibians (all members of the class Amphibia) are considered to be susceptible to infection with ranavirus. Natural infections are known from most of the major families of the orders Anura (frogs and toads) and Caudata (including salamanders, newts and sirens). Infection with ranaviruses in fish and reptiles can result in asymptomatic infections through to epizootics, depending on species, stress and environmental factors.

In challenge experiments, juvenile green tree frogs (*Litoria caerulea*), striped burrowing frogs (*L. alboguttata*), short-footed frogs (*Cyclorana brevipes*) and red-backed toadlets (*Pseudophryne corieacea*) were acutely susceptible to Bohle iridovirus.

Adult red tree frogs (*Litoria rubella*), bumpy rocketfrog (*L. inermis*), green tree frogs, ornate nursery frogs (*Cophixalus ornatus*), sharp-snouted day frogs (*Taudactylus acutirostris*) and cane toads (*Bufo marinus*) were less susceptible in trials (Cullen & Owens 2002).

Presence in Australia

Infection with ranavirus (species type Bohle iridovirus) has been officially reported in Australia. The disease was originally isolated from tadpoles of ornate burrowing frogs (*Limnodynastes ornatus*) in far north Queensland. It has since been isolated from moribund green tree frogs and captive juvenile red-backed toadlets. There is serological evidence of ranavirus infection in cane toads across northern Australia.

Epidemiology

- Two syndromes in frogs are associated with ranavirus infection: ulcerative syndrome and haemorrhagic syndrome.
- The most common presentation is a rapid, severe mortality event with death due to peracute systemic haemorrhagic disease, although in Australia morbidity is more common.
- Mortality and morbidity vary from species to species (0–100%), and may be variable depending on virus type, and age and health status of the host.
- Horizontal transmission occurs via contaminated water, animal-to-animal contact and cannibalism.
- The virus can be spread between widely separated river systems and impoundments, which suggests viral persistence and the existence of transmission mechanisms other than direct horizontal transmission.

- Vertical transmission is considered likely, but has not been experimentally documented.
- Possible vectors include nets, boats and other equipment, or amphibians used for bait by recreational fishers. Birds are potential mechanical vectors.
- Temperature is considered a likely factor influencing disease outbreaks, with the prevalence or severity of outbreaks greater during warmer months.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases in this field guide are similar to infection with ranavirus.

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

Cullen, BR & Owens, L 2002, 'Experimental challenge and clinical cases of Bohle iridovirus (BIV) in native Australian anurans', *Diseases of Aquatic Organisms*, vol. 49, no. 2, pp. 83–92.

The currently accepted procedures for a conclusive diagnosis of infection with ranavirus are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011,* available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

This hyperlink was correct and functioning at the time of publication.

Fungal diseases of amphibians

Infection with *Batrachochytrium dendrobatidis*

(Also known as chytridiomycosis, cutaneous chytridiomycosis and amphibian chytrid fungus)

Great barred frog (*Mixophyes fasciolatus*) with severe infection with *Batrachochytrium dendrobatidis*; note pieces of shedding skin on the body

Source: L Berger

Signs of disease

Important: Animals with disease may show one or more of the signs below, but the pathogen may still be present in the absence of any signs.

Disease signs at the tank or pond level (or in the wild) are:

- lethargy
- ataxia
- paralysis
- loss of flee response
- loss of righting reflex
- abnormal sitting posture
- tetanic spasms
- nocturnal species emerging during daylight
- burrowing species remaining outside of burrows.

Gross pathological signs are:

- erythema (redness) of the ventral surface
- lesions ranging from no obvious change to sloughing (as small flakes of skin).

Note that gross changes to the skin may be seen in severe infections; however, these are not specific to the disease.

Microscopic pathological signs are:

- loss of pigmented jaw sheaths and teeth rows in tadpoles' mouthparts
- zoosporangia in the outer epidermal layers that are seen on fresh pieces of shed skin and in histological sections
- hyperkeratosis of the epidermis in areas where zoosporangia occur.

Disease agent

The disease is caused by infection with the parasitic chytrid fungus, *Batrachochytrium dendrobatidis*, of the class Chytridiomycota, order Rhizophydiales.

Host range

Most, if not all, amphibians, including all members of the orders Anura (frogs and toads), Caudata (including salamanders, newts and sirens) and Gymnophiona (caecilians), appear to be susceptible to infection with *B. dendrobatidis*. Amphibian species differ in degree of susceptibility; some are naturally resistant.

Presence in Australia

Infection with *B. dendrobatidis* has been officially reported across Australia in Queensland, New South Wales, the Australian Capital Territory, Victoria, South Australia, Tasmania and Western Australia. It does not occur in arid inland areas.

Epidemiology

- All age classes, except eggs, are known to be susceptible to infection. Mortality has only rarely been reported in tadpoles.
- The pathogen infects only keratinised tissues (skin of metamorphosed amphibians or the mouthparts of tadpoles).
- Some species are more resistant to *B. dendrobatidis* than others; some do not develop disease following infection, while others are susceptible to the disease.
- Incubation times vary from about 14 to more than 70 days, with mortalities usually occurring within 2–3 days of the first clinical signs. Mortalities have approached 100% in some Australian amphibians.

- Horizontal transmission is via waterborne, motile zoospores and is likely to be by direct animal-to-animal contact. Vertical transmission via eggs has not been demonstrated.
- Outbreaks may be associated with seasons (cooler months), altitude (most declines are generally restricted to high-altitude populations) and breeding habitat.
- Temperature affects the survival and growth of *B. dendrobatidis*; maximum growth occurs between 17 °C and 25 °C. The sporangia die in temperatures of 32 °C or higher. The pathogen can persist in very low host densities.
- Large-scale mortality of newly metamorphosed amphibians may indicate infection, as some species appear to be most susceptible at this time.

Differential diagnosis

The list of similar diseases below refers only to the diseases covered by this field guide. Gross pathological signs may be representative of a number of diseases not included in this guide, which therefore should not be used to provide a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

No diseases in this field guide are similar to infection with *B. dendrobatidis.*

Sample collection

Due to the uncertainty in differentiating diseases using only gross pathological signs, and because some aquatic animal disease agents might pose a risk to humans, only trained personnel should collect samples. You should phone your state or territory hotline number and report your observations if you are not appropriately trained. If samples have to be collected, the state or territory agency taking your call will provide advice on the appropriate course of action. Local or district fisheries or veterinary authorities may also provide advice regarding sampling.

Emergency disease hotline

The national disease hotline number is 1800 675 888. This number will put you in contact with the appropriate state or territory agency.

Further reading

The accepted procedures for a conclusive diagnosis of infection with *B. dendrobatidis* are summarised in the World Organisation for Animal Health *Manual of diagnostic tests for aquatic animals 2011*, available at www.oie.int/en/international-standard-setting/aquatic-manual/access-online.

Further information can be found on the following websites:

Australian Government Department of Sustainability, Environment, Water, Population and Communities: www.environment.gov.au/biodiversity/threatened/publications/tap/chytrid.html

James Cook University's Amphibian Diseases: www.jcu.edu.au/school/phtm/PHTM/frogs/batrachochytrium.htm

Global *Bd*-mapping project: http://www.spatialepidemiology.net/bd-maps.

These hyperlinks were correct and functioning at the time of publication.

7 Scientific Names

Finfish

Scientific name	Common name/s
Abramis brama	Freshwater bream
Acanthopagrus australis	Yellowfin bream
Acanthopagrus latus	Western yellowfin bream
Acanthopagrus schlegeli	Black seabream or black porgy
Acipenser baeri	Siberian sturgeon
Acipenser gueldenstaedtii	Russian sturgeon
Acipenser transmontanus	White sturgeon
Ambassis agassizii	Chanda perch
Ambloplites rupestris	Rock bass
Ameiurus catus	White catfish
Ameiurus melas	Black bullhead
Ameiurus natalis	Yellow bullhead
Ameiurus nebulosus	Brown bullhead
Ammodytes hexapterus	Pacific sand lance
Ammodytes lancea	Lesser sand eel
Ammodytes personatus	Pacific sand eel
Ammodytes spp.	Sand eel
Amniataba percoides	Barred grunter
Anabas testudineus	Climbing perch
Anarhichas lupus	Wolffish or Atlantic wolffish
Anarhichas minor	Spotted wolffish

Scientific name	Common name/s
Acanthurus triostegus	Convict surgeonfish
Anguilla anguilla	European eel
Anguilla australis	Eel
Anguilla japonica	Japanese eel
Anguilla rostrata	American eel
Anguilla spp.	Eels
Anodontiglanis dahlia	Toothless catfish
Anoplopoma fimbria	Sablefish
Apistogramma spp.	Dwarf cichlids
Aplocheilichthys normani	African lampeye killifish
Aplodinotus grunniens	Freshwater drum
Apogon exostigma	Narrowstripe cardinalfish
Argentina sphyraena	Lesser argentine
Aristichthys nobilis	Bighead carp
Arius leptaspis	Triangular shield catfish
Arius spp.	Fork-tailed catfish
Astronotus ocellatus	Oscar
Atherinidae	Silversides
Atractoscion nobilis	White seabass
Aulorhynchus flavidus	Tubesnout
Bagridae	Bagrid catfishes
Barbus graellsi	Spanish barbel
Bidyanus bidyanus	Silver perch
Blicca bjoerkna	Silver bream
Bothidae	Left-eye flounders
Brachydanio rerio	Zebra danio
Branchiostegus japonicus	Japanese or red tilefish
Brevoortia tyrannus	Atlantic menhadden
Carangidae	Trevally and amberjacks
Carassius auratus	Goldfish
Carassius carassius	Crucian carp
Carassius sp.	Shububkin
Catostomus commersoni	White sucker
Channa marulius	Bullseye snakehead
Channa striatus	Striped snakehead
Chanos chanos	Milkfish
Cichlidae	Cichlids

Scientific name	Common name/s
Cinetodus froggatti	Froggatt's catfish
Cirrhinus cirrhinus	Mrigal
Clarias batrachus	Walking catfish
Clarius spp.	Torpedo-shaped catfishes
Clupea harengus	Atlantic herring
Clupea pallasii	Pacific herring
Clupeidae	Herrings and sardines
Cnidoglanis macrocephalus	Estuary catfish
Cobitidae	Loaches
Colisa lalia	Dwarf gourami
Colisa spp.	Gourami
Coregonidae	Whitefish
Coregonus artedi	Cisco
Coregonus clupeaformis	Lake whitefish
Coregonus peled	Whitefish
Coregonus muksun	Whitefish
Coregonus spp.	Whitefish
Cotostomidae	Suckers
Cottos gobio	Bullhead
Cromileptes altivelis	Humpback grouper
Ctenolabrus rupestris	Goldsinny
Ctenopharyngodon idellus	Grass carp
Cymatogaster aggregata	Shiner perch
Cyprinidae	Cyprinids
Cyprinus carpio	Common carp and koi carp
Damalichthys vacca	Pile perch
Danio devario	Sind danio
Danio rerio	Zebrafish
Dicentrarchus labrax	European seabass
Dorosoma cepedianum	Gizzard shad
Eigenmannia virescens	Glass knifefish
Enchelyopus cimbrius	Fourbeard rockling
Eopsetta grigorjewi	Shotted halibut
Epinephelus aeneus	White grouper
Epinephelus akaara	Red-spotted grouper or Hong Kong grouper
Epinephelus awoara	Yellow grouper
Epinephelus bruneus	Longtooth grouper

Scientific name	Common name/s
Epinephelus coioides	Orange-spotted grouper
Epinephelus fuscoguttatus	Brown-marbled grouper
Epinephelus lanceolatus	Giant grouper
Epinephelus malabaricus	Malabar grouper
Epinephelus marginatus	Dusky grouper
Epinephelus septemfasciatus	Seven-band grouper
Epinephelus sexfasciatus	Six-bar grouper
Epinephelus spp.	Grouper and estuary cod
Epinephelus tauvina	Estuarine rockcod
Esocidae	Pikes
Esox lucius	Pike
Esox masquinongy	Muskellunge
Etroplus maculatus	Orange chromides
Evynnis japonica	Crimson seabream
Evynnis tumifrons	Yellowback seabream
Exocoetus volitans	Tropical two-winged flying fish
Fundulus heteroclitus	Mummichog
Gadus macrocephalus	Pacific cod
Gadus microgadus	Tomcod
Gadus morhua	Atlantic cod
Galaxias olidus	Mountain galaxias
Galaxiidae	Minnows
Gambusia affinis	Mosquito fish
Gasterosteus aculeatus	Three-spined stickleback
Girella punctata	Girella or rudderfish
Glossamia aprion	Mouth almighty
Glossogobius giuris	Flathead goby
Glyptocephalus stelleri	Korean flounder
Hexagrammos otakii	Greenling
Hippoglossoides platessoides	American plaice
Hippoglossus hippoglossus	Atlantic halibut
Hippoglossus stenolepis	Halibut
Hoplobrotula armata	Armoured weaselfish
Hucho hucho	Danube salmon
Hyperoplus lanceolatus	Great sandeel
Hypomesus pretiosus	Surf smelt
Hypophthalmichthys molitrix	Silver carp

Scientific name	Common name/s
Ictalurus catus	White catfish
Ictalurus furcatus	Blue catfish
Ictalurus nebulosus	Brown bullhead
Ictalurus punctatus	Channel catfish
Kurtus gulliveri	Nurseryfish
Labeo spp.	Rhinofishes
Labridae	Wrasse
Labrus bergylta	Wrasse
Laetacara curviceps	Curviceps
Lampetra fluviatalis	River lamprey
Lateolabrax japonicus	Japanese seabass
Lateolabrax spp.	Seabass
Lates calcarifer	Barramundi
Latris lineata	Striped trumpeter
Leiopotherapon unicolor	Spangled perch
Lepomis gibbosus	Pumpkinseed
Lepomis macrochirus	Bluegill
Lethrinus haematopterus	Chinese emperor
Lethrinus nebulosus	Spangled emperor
Leuciscus cephalus	Chub
Leuciscus idus	Ide or orfe
Leuciscus leuciscus	Dace
Limanda limanda	Dab
Liza auratus	Golden grey mullet
Lota lota	Burbot
Lutjanus argentimaculatus	Mangrove jack
Lutjanus erythropterus	Red snapper
Maccullochella peelii peelii	Murray cod
Macquaria ambigua	Golden perch
Macquaria australasica	Macquarie perch
Macquaria novemaculata	Australian bass
Macropodus opercularis	Paradise fish
Melanogrammus aeglefinus	Haddock
Melanotaenia splendida	Rainbow fish
Merlangius merlangus	Whiting
Merlucciidae	Hake
Merluccius productus	Pacific hake

Scientific name	Common name/s
Microgadus proximus	Pacific tomcod
Micromesistius poutassou	Blue whiting
Micropterus dolomieui	Smallmouth bass
Micropterus salmoides	Largemouth bass
Mikrogeophagus ramirezi	Ram cichlid
Misgurnus anguillicaudatus	Loach
Morone americanus	White perch
Morone chrysops	White bass
Morone mississippiensis	Yellow bass
Morone saxatilis	Striped bass
Moronidae	White seabass
Moxostoma anisurum	Silver redhorse
Moxostoma macrolepidotum	Shorthead redhorse
Mugil cephalus	Mullet
Mugilidae	Mullets
Mullus barbatus	Red mullet
Nematolosa erebi	Bony bream
Neoarius berneyi	Berney's catfish
Neogobius melanostomus	Round goby
Neosilurus ater	Black catfish
Notemigonus atherinoides	Emerald shiner
Notropis atherinoides	Emerald shiner
Notropis cornutus	Common shiner
Notropis hudsonius	Spottail shiner
Noturus gyrinus	Tadpole madtom
Oncorhynchus aguabonita	Golden trout
Oncorhynchus clarkii	Cutthroat trout
Oncorhynchus gorbuscha	Pink salmon
Oncorhynchus keta	Chum salmon
Oncorhynchus kisutch	Coho salmon
Oncorhynchus masou	Masu salmon
Oncorhynchus mykiss	Rainbow trout
Oncorhynchus mykiss × *O. kisutch*	Hybrid rainbow trout × coho salmon
Oncorhynchus nerka	Sockeye salmon
Oncorhynchus rhodurus	Amago salmon
Oncorhynchus spp.	Pacific salmon
Oncorhynchus tschawytscha	Chinook salmon

Scientific name	Common name/s
Oplegnathus fasciatus	Japanese parrotfish
Oplegnathus punctatus	Spotted knifejaw
Oreochromis niloticus	Nile tilapia
Oxyeleotris lineolatus	Sleepy cod
Oxyeleotris marmoratus	Marble goby
Oxyeleotris selheimi	Giant gudgeon
Pagrus auratus	Snapper
Pagrus auriga	Redbanded seabream
Pagrus major	Red seabream
Pampus argenteus	Silver pomfret
Pangasius hypophthalmus	Sutchi catfish
Parachondrostoma toxostoma	Southwest European nase
Paralichthyidae	Flounders
Paralichthys dentatus	Summer flounder
Paralichthys lethostigma	Southern flounder
Paralichthys olivaceus	Japanese flounder
Parambassis gulliveri	Giant glassfish
Parapristipoma trilineatum	Chicken grunt
Parophrys vetulus	English sole
Pelteobagrus fulvidraco	Yellow catfish
Pelvicachromis pulcher	Rainbow krib
Perca flavescens	Yellow perch
Perca fluviatilis	Redfin perch or European perch
Percidae	Perches
Percopsis omiscomaycus	Trout-perch
Petromyzon marinus	Sea lamprey
Petromyzontyidae	Lampreys
Phoxinus phoxinus	Minnow
Pimephales notatus	Bluntnose minnow
Pimephales promelas	Fathead minnow
Platichthys flesus	Flounder
Platycephalus fuscus	Dusky flathead
Platycephalus indicus	Bartail flathead
Plecoglossus altivelis	Ayu
Plectorhinchus cinctus	Crescent sweetlips
Plectropomus maculatus	Spotted coralgrouper
Pleuronectes platessa	Plaice

Scientific name	Common name/s
Poecilia latipinna	Sailfin mollies
Poecilia reticulata or *Lebistes reticulatus*	Guppy
Poecilia sphenops	Molly
Pollachius virens	Coalfish or pollock
Pomatoschistus minutus	Sand goby
Pomoxis nigromaculatus	Black crappie
Psetta maxima	Turbot
Pseudocaranx dentex	Silver trevally
Pseudochondrostoma polylepis	Iberian nase
Pseudopleuronectes americanus	Winter flounder
Pseudosciaena crocea	Croceine croaker
Pterapogon kauderni	Banggai cardinalfish
Pterophyllum scalare	Angelfish
Puntius conchonius	Rosy barb
Puntius sophore	Pool barb
Rachycentron canadum	Cobia
Reinhardtius hippoglossoides	Greenland halibut
Rhombosolea tapirina	Greenback flounder
Rutilus rutilus	Common roach
Salmo clarki	Cutthroat trout
Salmo hucho	Dunube salmon
Salmonidae	Salmonids
Salmo salar	Atlantic salmon
Salmo trutta	Brown trout
Salmo trutta labrax	Black sea salmon
Salvelinus alpinus	Arctic char
Salvelinus confluentus	Bull trout
Salvelinus fontinalis	Brook trout
Salvelinus leucomaenis	Whitespotted char
Salvelinus namaycush	Lake trout
Salvelinus namaycush × *S. fontinalis*	Lake trout × brook trout
Sander lucioperca	Pike perch
Sardinops sagax	Pacific sardine
Scardinius erythrophthalmus	Rudd
Scatophagus argus	Scat
Sciaenidae	Drums and croakers
Sciaenidae spp.	Croakers

Scientific name	Common name/s
Sciaenops ocellatus	Red drum
Scleropages jardini	Saratoga
Scomber japonicus	Chub mackerel
Scomberomorus niphonius	Japanese Spanish mackerel
Scophthalmus maximus	Turbot
Scortum barcoo	Barcoo grunter
Sebastes schlegeli	Black rockfish
Sebastes spp.	Rockfish
Seriola dumerili	Greater amberjack
Seriola hippos	Samson fish
Seriola lalandi	Yellowtail kingfish
Seriola quinqueradiata	Japanese yellowtail
Sillago ciliata	Whiting
Silurus asotus	Chinese catfish
Silurus glanis	Wels catfish or sheatfish
Siniperca chautsi	Chinese perch or mandarin fish
Solea senegalensis	Senegalese sole
Solea solea	Common sole
Soleidae	Soles
Sparus aurata	Gilt-head seabream
Sprattus sprattus	European sprat
Stephanolepis cirrhifer	Thread-sail filefish
Strongylura krefftii	Long tom
Symphysodon discus	Discus fish
Takifugu rubripes	Tiger puffer
Thaleichthys pacificus	Eulachon
Theragra chalcogramma	Walleye pollock or Alaska pollock
Thunnus thynnus	Northern bluefin tuna
Thymallus thymallus	Grayling
Tinca tinca	Tench
Toxotes chatareus	Archer fish
Toxotes lorentzi	Primitive archer fish
Trachinotus blochii	Snubnose dart
Trachinotus falcatus	Yellow-wax pompano
Trachurus japonicus	Japanese horse mackerel
Trichiurus lepturus	Hairtail
Trichogaster leerii	Pearl gourami

Scientific name	Common name/s
Trichogaster microlepis	Silver gourami
Trichogaster pectoralis	Snakeskin gourami
Trichogaster trichopterus	Three-spot gourami
Trisopterus esmarki	Norway pout
Trisopterus minutus	Poor cod
Umbrina cirrosa	Shi drum
Verasper moseri	Barfin flounder
Verasper variegatus	Spotted halibut
Xiphophorus helleri	Swordtail or green swordtail
Xiphophorus maculatus	Southern platyfish or red wagtail platy
Zoarces viviparus	Viviparous blenny

Molluscs

Scientific name	Common name/s
Anadara trapezia	Sydney cockle
Argopecten gibbus	Calico scallop
Austrovenus stutchburyi	New Zealand cockle
Barbatia novae-zelandiae	New Zealand cockle
Cardium edule	Common cockle
Chamelea gallina	Striped venus clam
Crassostrea ariakensis	Suminoe oyster
Crassostrea corteziensis	Cortez oyster
Crassostrea gigas	Pacific oyster
Crassostrea nippona	Iwagaki oyster
Crassostrea rhizophorae	Mangrove oyster
Crassostrea sikamea	Kumamoto oyster
Crassostrea virginica	American oyster
Haliotis corrugata	Pink abalone
Haliotis cracherodii	Black abalone
Haliotis cyclobates	Whirling abalone
Haliotis discus hannai	Japanese abalone
Haliotis diversicolor supertexta	Small abalone
Haliotis diversicolor	Diversicolor or jiukong abalone
Haliotis fulgens	Green abalone
Haliotis laevigata	Greenlip abalone
Haliotis rubra	Blacklip abalone
Haliotis rubra × *Haliotis laevigata*	Tiger abalone
Haliotis rufescens	Red abalone

Scientific name	Common name/s
Haliotis scalaris	Staircase abalone
Haliotis sorenseni	White abalone
Haliotis tuberculata	European abalone
Haliotis wallalensis	Flat abalone
Katelysia rhytiphora	Sand cockle
Macoma balthica	Baltic macoma
Macomona liliana	New Zealand cockle
Mya arenaria	Sand gaper mussel
Mytilus edulis	Blue mussel
Mytilus galloprovincialis	Mediterranean mussel
Ostrea angasi	Southern mud oyster or Australian flat oyster
Ostrea chilensis	New Zealand dredge oyster
Ostrea conchaphila	Olympia oyster
Ostrea denselammellosa	Asiatic oyster
Ostrea edulis	European flat oyster
Ostrea equestris	Crested oyster
Ostrea puelchana	Argentinian flat oyster
Ostrea stentina	Dwarf oyster
Patinopecten yessoensis	Japanese scallop
Pecten maximus	Common scallop
Pinctada fucata martensii	Japanese pearl oyster or Akoya oyster
Pinctada margaritifera	Pearl oyster
Pinctada maxima	Silverlip pearl oyster
Pinctada sugillata	Pearl oyster
Pitar rostrata	Venerid commercial clam
Ruditapes decussatus	Groove-shelled clam
Saccostrea cuccullata	Rock oyster
Saccostrea glomerata	Sydney rock oyster
Solen marginatus	European razor clam
Tridacna crocea	Crocus clam
Tridacna gigas	Giant clam
Tridacna maxima	Elongated giant clam or rugose giant clam
Venerupis aurea	European aurora venus clam
Venerupis philippinarum	Asian littleneck clam or Manila clam
Venerupis pullastra	Pullet carpet shell
Xenostrobus securis	Small brown mussel

Crustaceans

Scientific name	Common name/s
Ascetes spp.	Paste prawn
Astacopsis gouldi	Giant Tasmanian crayfish
Astacus astacus	Noble crayfish
Astacus leptodactylus	Turkish crayfish
Austropotamobius pallipes	White-clawed crayfish
Austropotamobius torrentium	Stone crayfish
Camabroides japonicus	Japanese crayfish
Carcinus maenas	European shore crab
Charybdis feriatus	Mud crab
Cherax quadricarinatus	Red-claw freshwater crayfish
Cherax spp.	Freshwater crayfish
Eriocheir sinensis	Chinese mitten crab
Euastacus kershawi	Gippsland spiny crayfish
Farfantepenaeus aztecus	Northern brown shrimp
Farfantepenaeus duorarum	Northern pink shrimp
Fenneropenaeus californiensis	Yellow-leg shrimp
Fenneropenaeus chinensis	Chinese white shrimp
Fenneropenaeus indicus	Indian banana prawn
Fenneropenaeus merguiensis	Banana prawn, gulf banana prawn or white banana prawn
Litopenaeus occidentalis	Western white shrimp
Litopenaeus schmitti	Southern white shrimp
Litopenaeus setiferus	Northern white shrimp
Litopenaeus stylirostris	Pacific blue shrimp
Litopenaeus vannamei	Pacific white shrimp
Macrobrachium rosenbergii	Giant freshwater prawn
Macrobrachium sintangene	Sunda river prawn
Marsupenaeus japonicus	Kuruma prawn
Metapenaeus bennettae	Greentail prawn
Metapenaeusz ensis	Red endeavour prawn
Orconectes sp.	Crayfish (native to the eastern states of America)
Palaemon serrifer	Barred estuarine shrimp
Palaemon styliferus	Mysid shrimp
Panulirus spp.	Tropical spiny lobsters
Pasifastacus leniusculus	Signal crayfish
Penaeus esculentus	Brown tiger prawn
Penaeus monodon	Black tiger prawn
Penaeus semisulcatus	Green tiger prawn or grooved tiger prawn

Scientific name	Common name/s
Penaeus spp.	Sand shrimp
Portunus pelagicus	Mud crab
Portunus sanguinolentus	Mud crab
Procambarus clarkii	Louisiana swamp crayfish
Scylla serrata	Mud crab

Amphibians

Scientific name	Common name
Anura (order)	Frogs and toads
Bufo marinus	Cane toad
Caudata (order)	Salamanders, newts and sirens
Cophixalus ornatus	Ornate nursery frog
Cyclorana brevipes	Short-footed frog
Gymnophiona (order)	Caecilians
Limnodynastes ornatus	Ornate burrowing frog
Litoria alboguttata	Striped burrowing frog
Litoria caerulea	Green tree frog
Litoria inermis	Bumpy rocket frog
Litoria rubella	Red tree frog
Mixophyes fasciolatus	Great barred frog
Pseudophryne corieacea	Red-backed toadlet
Taudactylus acutirostris	Sharp-snouted day frog

Other species

Scientific name	Common name
Ardea cinerea	Grey heron
Argulus foliaceus	Fish louse
Callibaetis spp.	Mayflies
Lepeophtheirus salmonis and *Caligus elongatus*	Salmon louse
Piscicola spp.	Leeches
Salminicola spp.	Gill lice

8 Who to contact if you suspect a disease

If you see any unusual symptoms in wild or farmed aquatic animals, play it safe and **report it immediately**. Don't worry about how insignificant it may be—small signs may be an early indication of a serious disease problem.

The national 24 hour Emergency Animal Disease Watch Hotline is 1800 675 888 (freecall)

Each state or territory can also be contacted if you suspect a disease. The table below shows the state and territory government agencies responsible for aquatic health, and provides points of contact.

State or territory	Government agency	Internet site	State telephone
Australian Capital Territory	Environment and Sustainable Development Directorate	www.environment.act.gov.au	132 281
New South Wales	Department of Primary Industries	www.dpi.nsw.gov.au	1800 043 536
Northern Territory	Department of Resources—Primary Industries	www.nt.gov.au	0413 381 094 (fish kills) or 08 8999 2126
Queensland	Queensland Department of Agriculture, Fisheries and Forestry	www.dpi.qld.gov.au	132 523 or 07 3404 6999
South Australia	Primary Industries and Regions SA	www.pir.sa.gov.au	Fishwatch (24 hours) 1800 065 522 or 08 8463 3000
Tasmania	Department of Primary Industries, Parks, Water and Environment	www.dpiw.tas.gov.au	1300 368 550
Victoria	Department of Primary Industries	www.dpi.vic.gov.au	136 186
Western Australia	Department of Fisheries	www.fish.wa.gov.au	1800 815 507

Contact details and hyperlinks were correct at the time of publication.

9 Further reading and weblinks

Further reading

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Aquatic animal health websites

Australian Government Department of Agriculture, Fisheries and Forestry—Aquatic Animal Health program: www.daff.gov.au/animal-plant-health/aquatic

Fisheries and Oceans Canada—Synopsis of infectious diseases and parasites of commercially exploited shellfish: www.pac.dfo-mpo.gc.ca/sci/shelldis/title_e.htm

Centre for Environment, Fisheries and Aquaculture Science—Aquatic animal disease: www.cefas.defra.gov.uk/our-science/animal-health-and-food-safety/aquatic-animal-disease.aspx

European Union Reference Laboratory for Fish Diseases: www.crl-fish.eu

FishBase: www.fishbase.org

International Database on Aquatic Animal Diseases: www.cefas.defra.gov.uk/idaad

Marine Scotland Science: www.scotland.gov.uk/Topics/marine/Fish-Shellfish/18364/18610/diseases

Network of Aquaculture Centres in Asia–Pacific: www.enaca.org

Sub-Committee on Animal Health Laboratory Standards (SCAHLS) website at www.scahls.org.au/procedures/anzsdps2

United States Department of Agriculture, Animal and Plant Health Inspection Service—Aquaculture disease information: www.aphis.usda.gov/animal_health/animal_dis_spec/aquaculture

World Organisation for Animal Health: www.oie.int

All hyperlinks were correct at the time of publication.