NSW DEPARTMENT OF
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DIAGNOSIS, TREATMENT & PREVENTION OF THE DISEASES OF THE AUSTRALIAN FRESHWATER FISH SILVER PERCH *(Bidyanus bidyanus)*

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Phil Read • Matthew Landos • Stuart J. Rowland • Charlie Mifsud

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DISCLAIMER

The information contained in this publication is based on knowledge and understanding at the time of writing (April 2007). However, because of advances in knowledge, users are reminded of the need to ensure that information upon which they rely is up to date and to check currency of the information with the appropriate officer of New South Wales Department of Primary Industries or the user's independent adviser..

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FOR FURTHER INFORMATION

GRAFTON AQUACULTURE CENTRE
PMB2, Grafton NSW 2460
Ph 02 6640 1690, or

PORT STEPHENS FISHERIES CENTRE
Locked Bag 1, Nelson Bay NSW 2316
Ph 02 4982 1232

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INTRODUCTION

Silver perch (*Bidyanus bidyanus*) is an Australian native freshwater fish endemic to the inland Murray-Darling River System (**Fig. 1**). Hatchery techniques were developed at the Narrandera Fisheries Centre (formerly the Inland Fisheries Research Station) in the late 1970's, and transferred to a new commercial industry in 1982. The routine production of fingerlings by government and private hatcheries over the past 27 years has provided a solid foundation for the development of a grow-out industry.

Research at the Grafton Aquaculture Centre in the early 1990's demonstrated that silver perch is an excellent species for pond culture with high survival, fast growth and high production rates. An industry has been developing since the mid 1990's and is based primarily on aerated, earthen ponds. There is some production in recirculating aquaculture systems (RAS) and cages, and these intensive systems have potential for significant production in the future. Some silver perch farms have achieved high survival and production under commercial conditions; however, for success, farms must have a good supply of high quality water, a reliable power supply for aeration and pumps, be geographically located in a region that enables fast growth and good health, and be well managed.

Diseases of silver perch under culture conditions have been previously described by Ashburner (1983), Rowland (1983), Rowland and Ingram (1991) and Callinan and Rowland (1995). The expansion of the silver perch grow-out industry since the mid 1990's has seen a corresponding increase in the incidence of diseases, plus several new diseases and pathogens including a winter fungal disease, gill flukes, and various syndromes and conditions. The common diseases of silver perch are caused by protozoans, monogeneans, fungi and bacteria. Diseases have had a significant impact on commercial production through induced stress on fish, loss of growth and production, death of stock and high costs of treatments.

In 2001 – 2005, a project entitled 'Development of a Health Management Strategy for the Silver Perch Aquaculture Industry' was undertaken to study the diseases and health management of silver perch. Major outcomes were: identification of new and important diseases; development of control and preventative methods for most diseases; and a health management strategy for the industry based on three separate

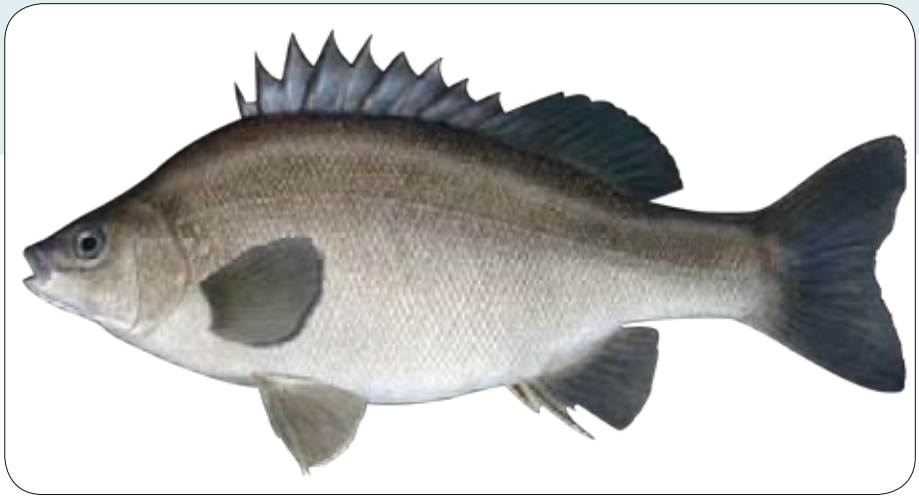

Figure 1
Silver Perch (*Bidyanus bidyanus*).
Source: Patrick Tully

publications, 'Health Management Plan for Silver Perch Culture', 'Hatchery Quality Assurance Program for Murray Cod (*Maccullochella peelii peelii*), Golden Perch (*Macquaria ambigua*) and Silver Perch (*Bidyanus bidyanus*)' and the manual 'Diagnosis, Treatment and Prevention of the Diseases of the Australian Freshwater Fish Silver Perch (*Bidyanus bidyanus*)'. This manual has been prepared as an easy-to-use publication with numerous photographs of diseased fish and pathogens that will facilitate the prompt diagnosis and appropriate treatment of silver perch diseases. Its use in conjunction with the health management plan, should reduce the incidence and severity of disease outbreaks, leading to improved survival and performance of fish, and increased production, efficiency and profitability of silver perch farms.

ANATOMY OF SILVER PERCH

Silver perch (*Bidyanus bidyanus*) belongs to the Class Osteichthyes or bony fishes. Teleosts are the highest bony fishes in evolutionary terms, and constitute the largest group within Osteichthyes. Teleosts can loosely be divided into two main groups, the soft-rayed and spiny-rayed fish. The latter group, which includes silver perch, is more advanced and typically possess bony spines in some fins, ctenoid scales and a swim bladder which lacks a connection with the oesophagus. Most of the organ systems (heart, liver, kidney, etc) of teleosts are similar to those found in mammals, with differences reflecting adaptation to an aquatic life. The cardiovascular system consists of a single circuit, with blood being pumped by the heart to the gills from where it passes under low pressure to the body and then back to the heart. Silver perch also have lymphatic, nervous, reproductive and endocrine systems. Some organs, like the pancreas are difficult to locate and are sited in mesenteric fat between the pyloric caecae (**Figs. 2** and **3**). Silver perch are poikilothermic (cold blooded) animals, i.e. their body temperature is determined by the environmental temperature. Fins are median (dorsal,

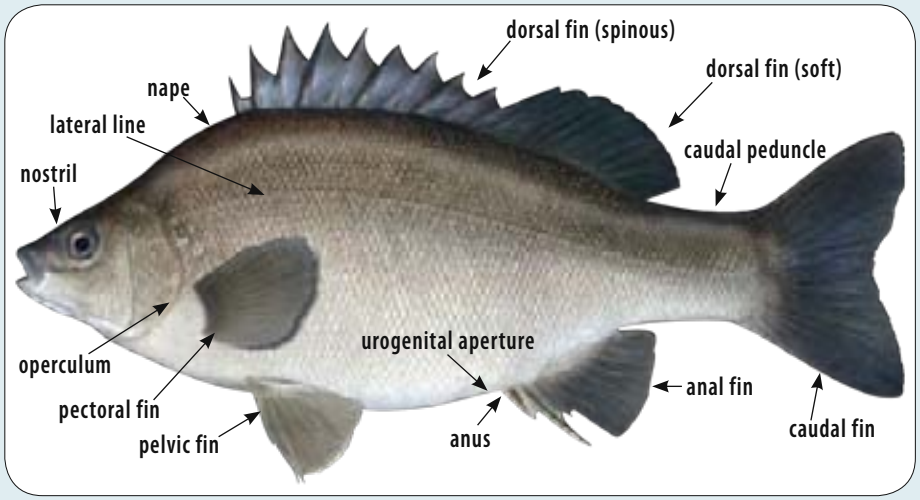

Figure 2
External features of silver perch.
Source: Patrick Tully

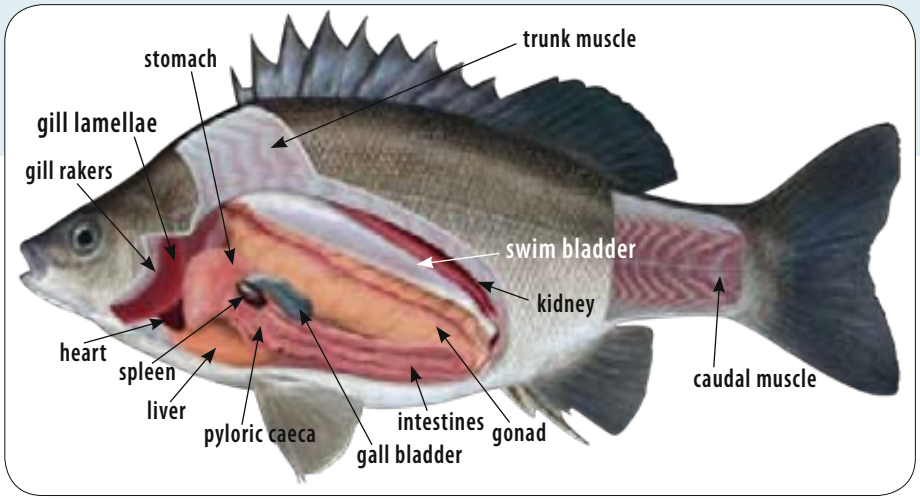

Figure 3
Gross anatomy of the viscera of silver perch.
Source: Patrick Tully and Phil Read

caudal and anal) or paired (pectoral and pelvic). The skeleton (**Fig. 4**) is made up of the skull, vertebral column and the fin skeleton. The skull has a complex arrangement of bony plates (neurocranium) which still allow flexibility and surrounds the olfactory, optic and otic areas. The lower section of the skull (branchocranium) consists of bones associated with the jaw, operculum and gill arches.

EXTERNAL FEATURES

Opercula – bony cover which serves to protect the gills and assist with respiration.

Lateral line – pressure sensory organ having epidermal pores; runs bilaterally from head to caudal fin.

Fins – bony/spiny and soft-rayed; assist with locomotion, positioning and aggressive behaviour.

Epidermis – cellular-epidermal material, sloughed cells, and mucus secreted to the surface; immuno-active properties; assist in osmoregulation and swimming; protects against abrasion; primary protection against the environment; sensory receptors; excretory, some respiratory functions; dermis contains many pigment cells containing melanin (**Fig. 5**).

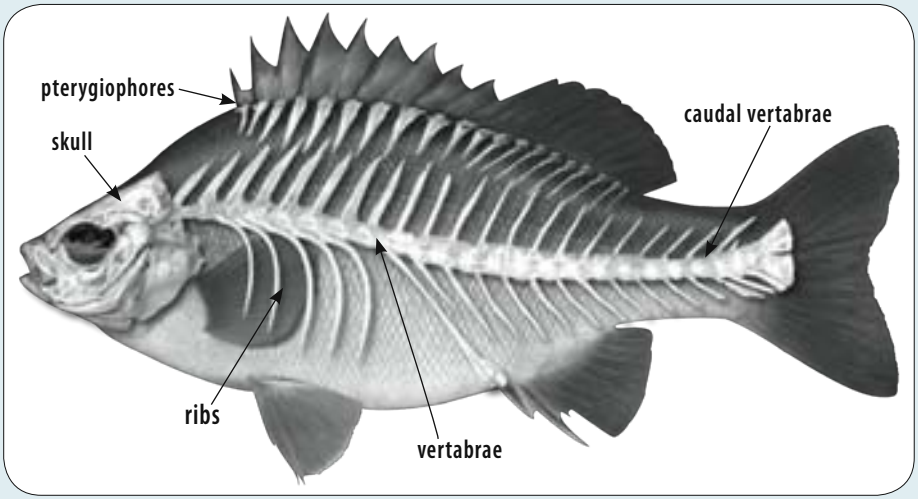

Figure 4
Main skeletal features of silver perch.
Source: Patrick Tully and Phil Read

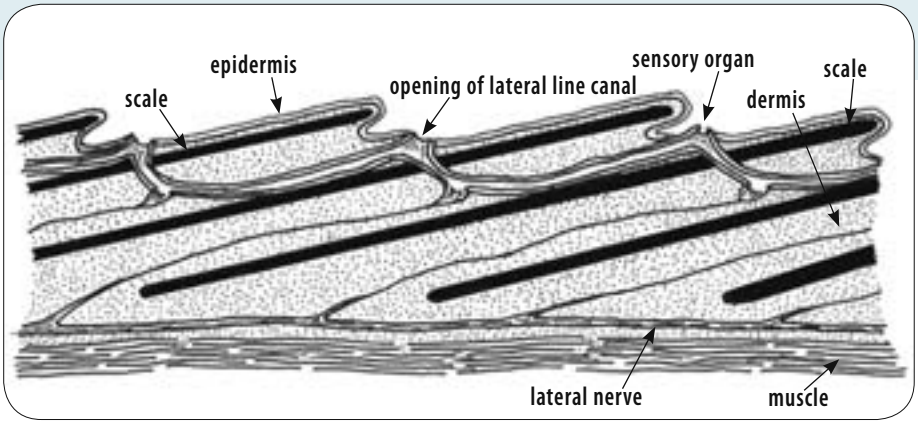

Figure 5
Diagrammatic representation of a section through fish skin.
Source: Phil Read (adapted from Storer et al. 1972)

Scales – calcified, flexible plates with growth rings; covered by an epidermal layer of cells; assists in osmotic control; physical protection (**Fig. 6**).

RESPIRATION

Gills – main respiratory organ; large surface area; uptake of oxygen and excretion of nitrogenous wastes and carbon dioxide; maintenance of osmotic and ionic balance (absorption of water, excretion of salts) (**Fig. 7**).

INTERNAL FEATURES

Swim bladder (gas bladder) – thin-walled sac filled with gases; detects water pressure changes; assists with body buoyancy and positioning; sound production and perception.

Kidney – excretion of water to maintain blood osmolarity balance; protein and ion resorption; removal of nitrogenous wastes from the blood; anterior and posterior kidney.

Heart – two chambered (atrium and ventricle); posterior to gills in separate thoracic cavity; distributes blood via arteries to the gills, organs and body.

Spleen – flat, strap-like, dark, red-coloured organ located near the stomach within the abdominal fat; circulatory system filter; capable of generating new blood cells.

Liver – large, pale tan to red in colour; produces enzymes to assist digestion; stores carbohydrates and fats; processes nutrient and toxins absorbed from gut; blood cell destruction and regulation; nitrogen excretion.

Gall bladder – dark, mottled green; bile production to assist in lipid digestion.

Stomach – firm, sac-like organ at termination of oesophagus; digestion; secretes mucus, enzymes and acid.

Pyloric caecae – finger-like pouches; digestion.

Blood – hyper-osmotic; transfer of nutrients, gases and wastes; immune response.

Intestine – digestion; osmoregulatory control; lipid and protein regulation.

Gonads – sex organs; paired; suspended from the dorsal abdominal wall; testes white/cream coloured, flattened/ angular; ovaries pink/cream coloured, rounded.

Musculoskeletal system – skeleton of true bone, skull, vertebral column, ribs pectoral girdle, accessory bones; red and white muscle utilised for aerobic activity and short anaerobic bursts of power.

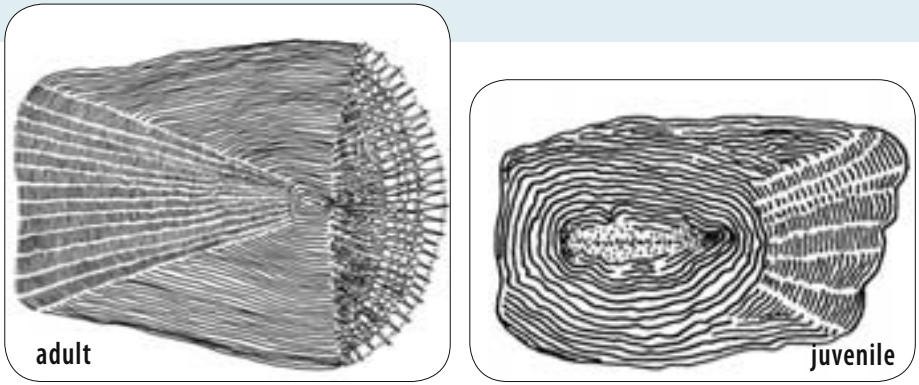

Figure 6
Ctenoid scales from the flank of a mature silver perch and the caudal peduncle of a juvenile fish.
Source: Phil Read

Figure 7
Silver perch gills (×100 mag.).
Source: Phil Read

DIAGNOSTIC TECHNIQUES

DIAGNOSTIC TOOLS

The following equipment is needed to perform on-farm, disease diagnosis.

- Binocular compound microscope with powered light source, moveable stage, 10× ocular with 4×, 10×, 20× and 40× lens magnification (final magnification, 40×, 100×, 200× and 400×).
- Dissection kit including forceps, sharp-pointed scissors, scalpel and blades, microscope slides and coverslips, cutting board and killing knife or pithing spike.
- Cast net, seine net and hand nets; buckets, airstones and airpump.

OBSERVATION

Ponds, cages and tanks should be checked daily, in particular those with the following features.

- Recently (within 2 weeks) stocked or partially harvested;
- High stocking densities (particularly fingerling ponds);
- High feeding rates (>60 kg/ha; summer temperatures);
- Poor water quality or sudden variations e.g.

  'Crash' of algal bloom

  Low dissolved oxygen (DO) (<3 mg/L)

  Very high temperatures (>30°C)

  Sudden decreases in temperature (>4°C in 24 hrs)

  High ammonia and high pH

  Heavy surface scum, blue-green algae blooms

  Recent water exchange;

- Bird predation;
- Valuable broodfish.

SIGNS

Diseased fish display many physical and behavioural signs, and these can vary between diseases, levels of infestation or infection, size, age and condition of fish, culture facility and season. In general, any unusual signs in fish or unexpected changes in the water could be indicative of disease or pending disease. Some of the more common and important signs displayed by fish are:

- Loss of appetite – reduced feeding activity by some, many or all fish;
- Fish congregating near the surface, edges, aerators (**Fig. 8**);
- Fish swimming or acting abnormally, e.g. flashing;

Figure 8
Diseased silver perch schooling in incoming water.
Source: Phil Read

- Fish with unusual colouring; may be pale or dark, eroded patches of skin, bulging eyes, red brains or enlarged and discoloured organs;
- Moribund or dead fish;
- Increasing numbers of moribund or dead fish.
- Presence or absence of feed in gut.

WATER QUALITY MONITORING

The first action after the observation of unusual signs is to immediately monitor water quality with calibrated meters. Ensure:

- DO (>3 mg/L), ammonia (<0.1 mg/L), pH (6 – 9.5) and temperature (10–30°C), these are minimum or maximum levels, or acceptable ranges.

MANAGEMENT OF SUB-OPTIMAL WATER QUALITY

- Stop feeding;
- Increase aeration;
- Exchange water;
- Add lime to increase low pH (if <6.0 pH).

SAMPLING FISH

Fish should be sampled (minimum 4 fish) (**Fig. 9**) after observation of the following conditions:

- Mortalities and/or moribund (sick) fish;
- Sudden decrease in feeding behaviour (over 1–3 days);
- Abnormal swimming behaviour ('flashing'; fish 'listless' and or 'gasping'at pond edges or surface; fish positioned high in the water column or in water currents, circling or rapid swimming, rubbing on standpipes or ropes), abnormal colour;
- Fish unsighted for 2 weeks or more, particularly during autumn, summer and spring (>15°C water temperature).

Figure 9
Sampling fish using a cast net.
Source: Phil Read

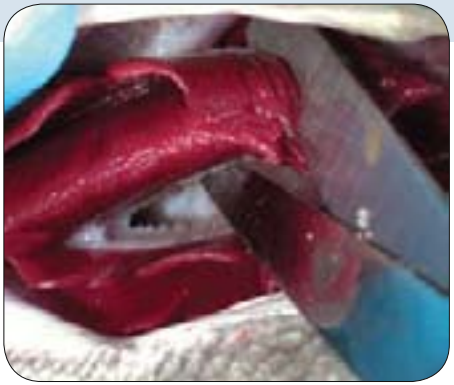

Figure 10
Severing the gill arch to remove gill tissue.
Source: Jeff Guy

EXAMINATION OF GILL AND SKIN TISSUE

Live, preferably moribund fish collected for necropsy should be held in containers having aerated pond water (use water from pond). Microscopic examination should commence immediately following euthanasia (severing of spinal cord immediately behind head or pithing brain) as some parasites are prone to detaching and/or immobilisation soon after death of fish. Using anaesthetics for euthanasia is not recommended as some ecto-parasites may be killed or detach rapidly from the fish and will therefore be unseen during the examination. The following procedures are used to diagnose ecto-parasitic and some fungal and bacterial diseases.

Gill examination – large fish:

- lift the opercular cover and using scissors, sever the white, cartilaginous gill arch (2 cuts) and remove >10 mm of gill (**Figs. 10** and **11**);
- place on a slide and using a scalpel, sever the cartilaginous gill arch from the primary gill lamellae (**Fig. 12**);
- discard the gill arch and add 2–3 drops of distilled water to the gill lamellae (do not use chlorinated tap water or pond water);
- lay a coverslip over the material and apply light pressure to encourage the lamellae to spread (**Fig. 13**);
- examine the tissue at low (40×), then higher magnification (100×).

Gill examination – small fish (<10 g):

- using scissors remove the opercular cover to reveal the gills;
- using scissors, sever a gill arch and remove a set of gills;
- separate one gill and discard the others;
- place the gill (cartilaginous gill arch attached) on a slide and add 2–3 drops of distilled water;
- lay a coverslip on the sample (the coverslip will be raised off the slide due to the thickness of the gill arch);
- add drops of water beneath the raised end of the coverslip until the tissue is 'flooded';
- examine the tissue.

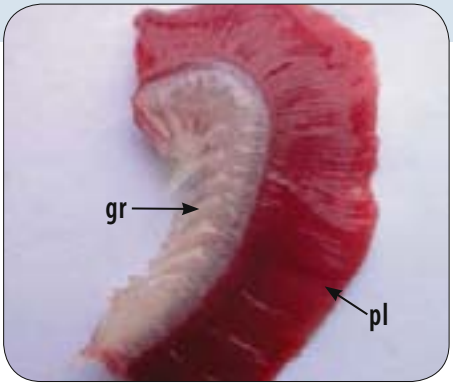

Figure 11
Single gill arch showing primary lamellae [**pl**] and gill raker [**gr**] [arrows].
Source: Phil Read

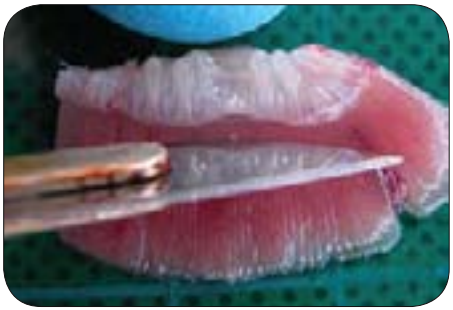

Figure 12
Separating gill lamellae from a gill arch.
Source: Jeff Guy

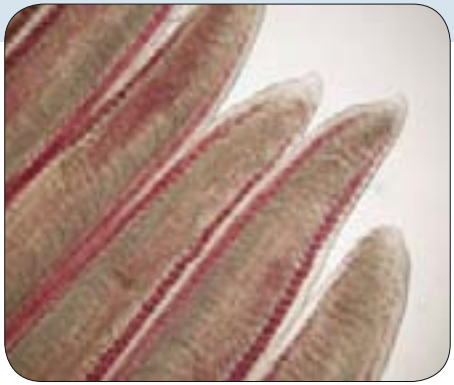

Figure 13
Healthy gill tissue beneath a cover slip.
Source: Phil Read

Skin examination:

- position the caudal fin on a slide;
- using a scalpel and light pressure, scrape along the side of the body facing upwards (**Fig. 14**);
- remove skin/mucus from the scalpel to the slide;
- add 2–3 drops water, coverslip and examine as described for gill tissue.

Fin clip examination:

- remove a small piece of caudal fin (or any portion of fins that demonstrate abnormalities such as ragged edges) and prepare it as described for skin scraping.

LABORATORY SUBMISSION OF FISH FOR DISEASE DIAGNOSIS

When the disease cannot be diagnosed on-farm, samples of fish should be delivered to a fish disease diagnostic laboratory. Moribund fish are the most suitable and reliable for diagnosis.
If delivery of live specimens is not possible, freshly euthanased specimens should be kept on wet ice and sent unfrozen provided they will arrive at the laboratory within 3 hours. If the delay is greater than 3 hours, the specimens should be sent preserved in 10% neutral buffered formalin. Tissue should not exceed 7 mm in thickness. In all cases the sample (organs or whole fish) should be representative of the population and ideally include moribund specimens.

Live specimens:

- package 4 to 6 fish in strong plastic bags; ⅓ pond water, ⅔ pure oxygen; air-tight seal; watertight container;
- preferably overnight transport or deliver samples personally;
- do not feed fish prior to transport

Iced specimens:

- wrap fish in a damp cloth or towel;
- place the sample in a plastic bag and cover with ice;
- place in a watertight container and deliver within 3 hrs.

Figure 14
Scraping the skin to obtain a sample.
Source: Jeff Guy

Preserved specimens – small fish (<30 g):

- using scissors, open fish gut cavity from the vent to the gills (do not sever organs);
- place samples in a plastic bottle with 10% neutral buffered formalin;
- use 10 times the volume of fixative to tissue.

Preserved specimens – large fish:

- remove fat and dissect out organs (anterior and posterior kidney, gill, liver, heart, intestine, stomach, spleen, brain, skin with section of lateral line); sample any skin lesions ensuring a 5 mm margin of normal tissue is included; fix tissue/organs as for small fish.

Preserved specimens are required to be packaged in three watertight containers and labelled clearly prior to submission under IATA transport regulations. Check with your courier to ensure packaging meets recommended standards.

Frozen specimens:

- recommended for toxicological analysis and some forms of virology;
- frozen tissues are of little use for histopathology or microbiology (the two major diagnostic tools used in most fish disease investigations).
- freeze samples as soon as possible following the suspected poisoning.

Information to accompany laboratory submissions:

- name; address; phone number and date
- species of fish; age and size
- water quality including pH; temperature; DO; ammonia; salinity
- size and type of production facility e.g. pond, cage, tank, raceway
- stocking density
- date mortality began and mortality rate per day
- clinical signs e.g. poor feeding; abnormal swimming; 'flashing'
- water source
- diet; size and manufacturer; ration; storage

- recent husbandry (past 2 weeks) e.g. harvesting; grading; water exchange; fish delivery
- system changes e.g. new tanks; changes in diet; chemical treatments
- environmental changes; weather conditions.

LABORATORY PROCEDURES

Specimens received at the laboratory will be processed and further examined.

- *Histopathology* – fixed or fresh tissues decalcified and individual tissues 'cut in' to assess single cell depth sections; stained; microscopically examined for pathogens and changes in tissue.
- *Bacteriology* – bacterial culture on specific media; targeted organs – kidney, spleen, brain, peritoneal fluids; also skin lesions; fresh tissue only (do not submit fixed tissue for bacterial culture); identification of bacteria; screening of bacterial colonies for antibiotic susceptibility (**Fig. 15**).
- *Virology* – viral culture on various fish cell lines; molecular diagnostics including PCR, sequencing and immunological tests to identify viruses.

LEARNING TO RECOGNISE THE 'SIGNS' AND DIAGNOSE DISEASE

Daily observation of fish, ponds and tanks, regular monitoring of water quality and a concise evaluation of skin and gill biopsies are the first steps used to investigate fish diseases. To the new or inexperienced fish farmer, interpreting physical signs and microscopic images is challenging. However, the regular sampling of healthy and diseased fish will facilitate the recognition of normal and abnormal tissues, help in identification of the common pathogens, and develop the skills required to accurately diagnose disease. Most disease outbreaks in silver perch culture involve the common diseases and pathogens – during the early stages of an investigation look for the obvious!

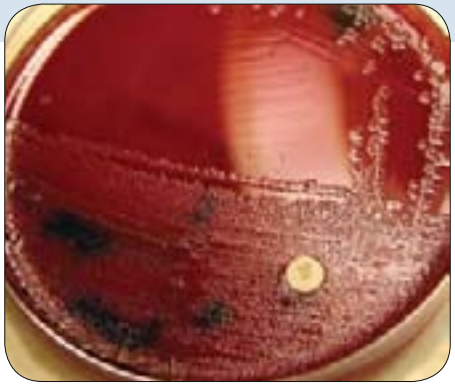

Figure 15
Bacterial colonies growing on a blood agar plate.
Source: Matt Landos

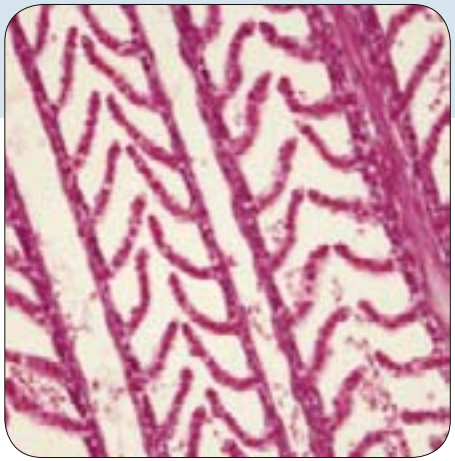

Figure 16
Histological section of normal gill structure.
Source: Matt Landos

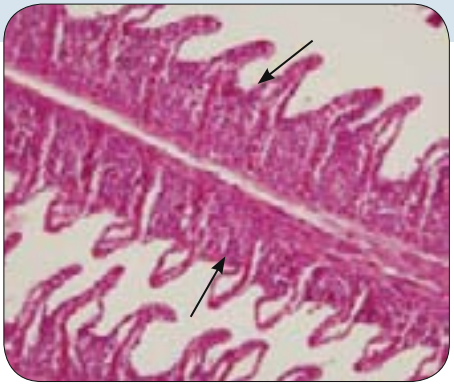

Figure 17
Histological section showing hyperblastic [arrows] silver perch gills.
Source: Matt Landos

Changes in the appearance of both skin and gill tissue are common during a disease event; however, determining the aetiological agent(s), or causes, is occasionally difficult due secondary invasion of pathogens or other organisms, or the manifestation of overt signs caused by systemic disease. Changes in skin can include hyperaemia, haemorrhaging, ulceration, erosion, changes in pigment or thickening of the epithelium. Diseased gill tissue often shows hyperplasia and hypertrophy, causing cell growth and fusion between the secondary lamellae. Skin and gill tissue can have parasites and bacteria present without causing clinical disease; the interpretation of their significance will depend upon other clinical findings (**Figs. 16, 17, 18, 19, 20, 21** and **22**).

Maintaining water quality is critical in preventing disease; however, it is not always controllable. Water quality (DO, temperature, pH and TAN) should be monitored on a regular basis using high-quality meters. Sudden changes in water quality e.g. an algae 'crash' (pond water turning tea colour), large temperature fluctuations (>5°C over 24 hrs), high TAN's and periods of low DO (<3 mg/L) can cause stress and disease. Protozoan and fungal infections are particularly common, and observation of ponds should be daily following periods of poor or changing water quality.

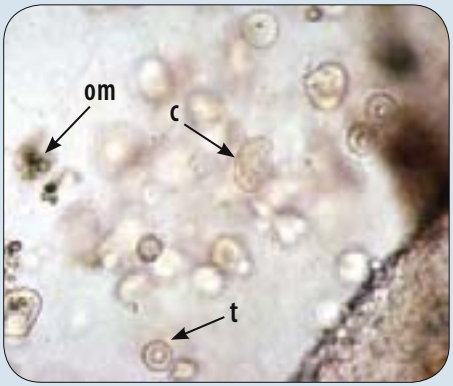

Figure 19
Wet mount of gill tissue showing [arrows] organic matter [**om**], and the parasites *Trichodina* sp [**t**] and *Chilodonella hexasticha* [**c**] (×100 mag.).
Source: Phil Read

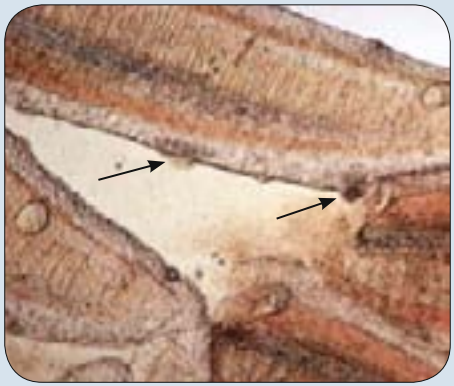

Figure 21
Wet mount of gill tissue showing abnormal changes [arrows] to cell structure.
Source: Phil Read

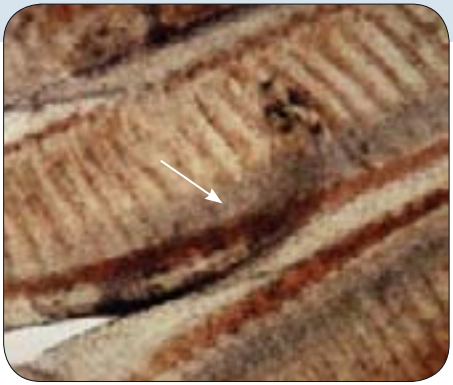

Figure 18
Gill fluke [arrow] – a large parasite.
Source: Phil Read

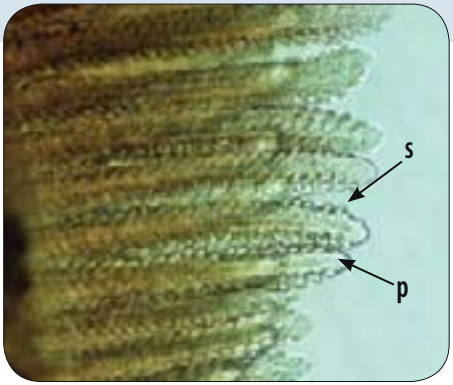

Figure 20
Silver perch gills; primary [**p**] and secondary lamellae [**s**] [arrows].
Source: unkown

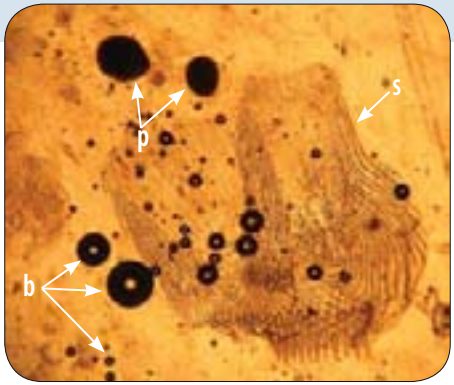

Figure 22
Wet mount of skin showing [arrows] scales [**s**], air bubbles [**b**] and parasites [**p**].
Source: Phil Read

ECTO-PARASITIC PROTOZOANS

Protozoans are usually single celled organisms that reproduce by binary fission and have specialised organelles such as cilia or flagella for locomotion. When present in large numbers, they greatly impair the epithelium, particularly of gill tissue. Some protozoans feed on the cells and mucus, while others cause physical injury, and some may produce toxins. Protozoans cause more diseases in silver perch culture than any other group of organisms.

CHILODONELLOSIS

Infestations of the protozoan *Chilodonella hexasticha* cause the disease chilodonellosis in silver perch in ponds, cages and tanks. The disease usually progresses rapidly; however, the parasite may exist in low numbers (e.g. 1–2 organisms on 5 fish) over a period of months before predisposing conditions cause a rapid increase in parasite numbers. The parasite can cause serious losses of silver perch, especially fingerlings. Outbreaks in all seasons, particularly autumn, winter and early spring, and over the temperature range of 10–30°C. Needs to be diagnosed and treated quickly to avoid large losses. May have a cyst stage.

Pathogen

Chilodonella hexasticha is a ciliate protozoan; ovoid to kidney shaped; dorso-ventrally flattened; 50–70 µm in length, 20–40 µm wide.

Signs

- Chronic to acute morbidity and/or mortality rates
- Loss of appetite
- Flashing
- Lethargy, swimming slowly, head-up position, often near surface and edges
- Ragged fins
- Emaciation
- Skin may have mottled and/or grey appearance

Diagnosis

Microscopic examination at 100× magnification. Found mainly on gills but also skin (**Figs. 23** and **24**). Infested fish may have heavy or light parasitic loads (**Fig. 25**); large variation between individual fish can sometimes make diagnosis difficult in the early stages of an outbreak; important to examine at least 4 fish from pond. The organism can quickly become immotile or detach rendering detection more difficult; prompt examination required following slide preparation. Cytoplasm often appears granular; cilia noticeable at higher magnification. Characteristic gliding motion; often slow or little movement at low temperatures; groups of individuals often clumped on gill tips.

Treatment

Tanks:

- 10 g/L salt (NaCl) for 60 min, flush and repeat following day.
- 5 g/L salt continuous (including purging) and/or formalin 25 mg/L continuous bath for at least 8 h, flush and repeat the following day; aerate well; no feeding.
- formalin 150 mg/L for 60 minutes (not larvae or fry); observe during treatment; aerate well (oxygen, if needed); flush well on completion.

Ponds/cages:

- formalin 25–30 mg/L, maintain 24 h aeration for 4–5 days; one treatment usually sufficient; re-examine pond fish to gauge treatment effectiveness; may need to re-treat ponds after 3–4 weeks.
- prophylactic for broodfish: formalin 25–30 mg/L winter/early spring (e.g. June then again in August)

Prevention

Quarantine and treat all fish prophylactically (2–5 g/L salt), including fingerlings prior to stocking, new arrivals to the farm, and fish being moved between ponds. Maintain good water quality and nutrition. Do not overstock. If possible, use source water having no trash fish and prevent bird activity on ponds.

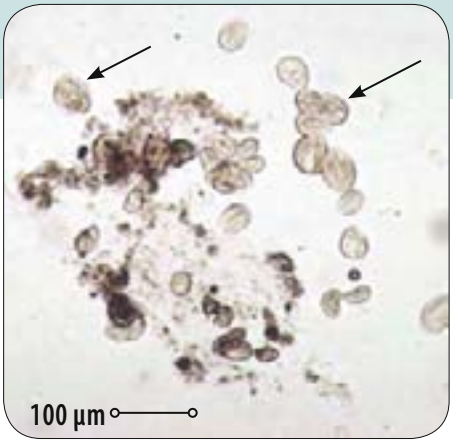

Figure 23
Chilodonella hexasticha in skin mucus [arrows] (×100 mag.).
Source: Stuart Rowland

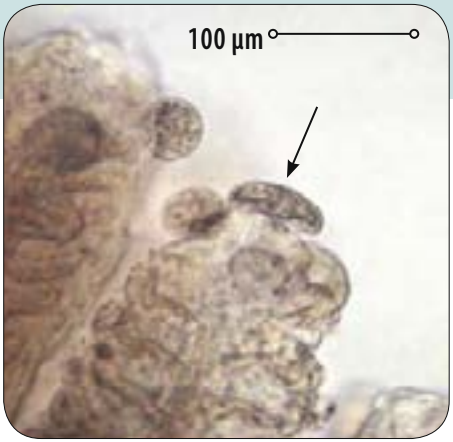

Figure 24
Chilodonella hexasticha 'grazing' [arrow] gill lamellae (×200 mag.).
Source: Phil Read

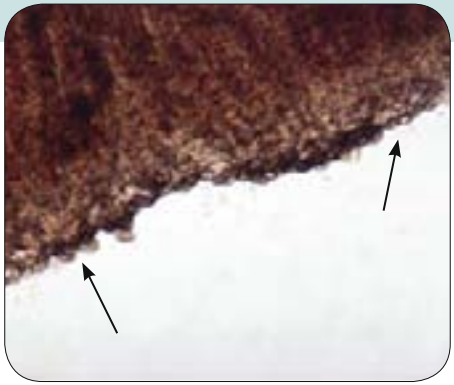

Figure 25
Heavy infestation of *Chilodonella hexasticha* on [arrows] primary gill lamellae (×100 mag.).
Source: Phil Read

ICHTHYOPHTHIRIOSIS (WHITE SPOT, ICH)

One of the most common and serious diseases of silver perch. Infestations of the ciliate protozoan, *Ichthyophthirius multifiliis*, cause the disease known as white spot or ich. Occurs in silver perch in ponds, tanks and cages. Disease can progress rapidly and cause 100% mortality. Affects fish of all sizes. There is some evidence in other species of freshwater fish that survivors of white spot develop immunity against future infestation. Occurs year round; decrease in water temperatures to/below 15°C in autumn associated with outbreaks. Needs to be quickly diagnosed and treated. Has a complex life cycle (**Fig. 26**) involving attached, encysted and free-swimming stages; stages of life-cycle temperature-dependent, less than 4 days, >24°C; more than 5 weeks, <7°C; some evidence of parasite reproducing underneath epithelium without a free-swimming stage; only free-swimming stages susceptible to chemicals.

Pathogen

Ichthyophthirius multifiliis is a ciliate protozoan; trophonts, 50–1,000 µm (1,000 µm = 1 mm) attach to fish (**Figs. 27** and **28**); mature trophonts leave fish and encyst on substrate and equipment (such as nets) as tomonts; division into numerous tomites; then released as free-swimming, infestive theronts that are oval or round shaped, granular appearance, 20–50 µm; attach to fish using a penetrating gland.

Signs

- Chronic to acute morbidity and/or mortality
- White nodules on skin (advanced cases)
- Opaque to white eyes (advanced cases)
- Positioned in water currents and/or at sides of ponds
- Lethargy and loss of appetite
- Flashing (often strong and repeated on pond bottom, standpipes, ropes) (**Figs. 29** and **30**)
- Ragged fins, skin raised and broken
- Mild skin haemorrhaging; striated skin markings or mottling
- Skin lesions and secondary bacterial and fungal infections

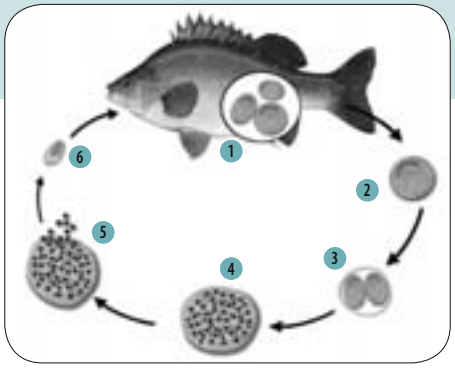

Figure 26
Diagrammatic representation of the life cycle of the parasitic protozoan *Ichthyophthirius multifiliis*; 1 and 2 – trophonts; 3 – tomont; 4 – tomites; 5 – tomites released; 6 – theronts.
Source: Patrick Tully

Figure 27
Silver perch infested with white spot.
Source: Phil Read

Figure 28
Trophonts attached to the head of a silver perch.
Source: Phil Read

Diagnosis

Microscopic examination of skin and gill tissue. Parasites readily seen at 40–100× magnification. Trophonts usually embedded under gill and skin epithelium (**Figs. 31** and **32**); can be present only on gills; cytoplasm usually dark granular with slow 'swirling' motion; horseshoe-shaped macronucleus sometimes visible in mature trophonts, spherical nucleus in immature individuals; body with uniform cilia; theronts ciliated, usually clearer cytoplasm than trophonts.

Treatment

Tanks:

- salt (NaCl) 2–5 g/L salt continuous until disease controlled, may be for up to 20 days; aerate well; no feeding;
- temperature manipulation to decrease length of life cycle–raise to 30°C for 10 days.

Ponds/cages:

- formalin 30 mg/L initially (when water temperature <25°C), then maintain levels between 25–30 mg/L until disease controlled (see chapter *Calculations, Treatments and Dose Rates*); aeration continuous, monitor DO daily and provide additional aeration if necessary; or
- copper (as copper sulfate, $CuSO_4$) 0.1–0.2 mg/L, recommend 0.2 mg/L initially, then monitor and adjust free Cu^+ ion levels daily to maintain concentration between 0.1 and 0.2 mg/L; continuous aeration during treatment until disease controlled; for copper treatment, alkalinity must be >50 mg/L (see chapter *Calculations, Treatments and Dose Rates*). Caution: copper sulfate is an algacide, the decay of algae can cause dangerously low DO.

Prevention

Quarantine and treat all fish prophylactically (2–5 g/L salt), especially fingerlings and new fish on farm prior to stocking. Maintain good water quality. Quarantine infected ponds including

Figure 29
Silver perch infested with white spot about to 'flash' on a submerged pipe.
Source: Phil Read

Figure 30
Silver perch 'flashing'.
Source: Phil Read

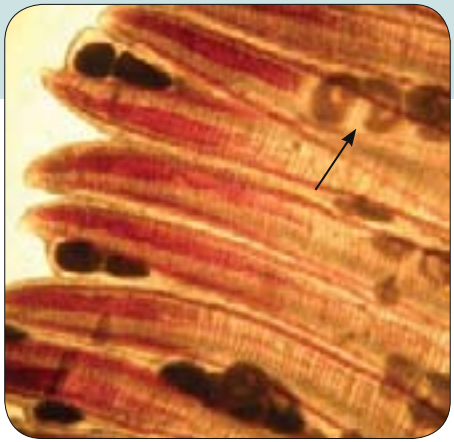

Figure 31
Heavy infestation of *Ichthyophthirius multifiliis* on silver perch gills; note 'horse-shoe' shaped macronucleus in some parasites [arrow] (×100 mag.).
Source: Phil Read

equipment, vehicles, water; prevent aquatic birds spreading disease. Nil water exchange when source water known to hold diseased fish. Closely observe/monitor fish in all ponds and tanks when white spot is diagnosed in any pond or tank. It is common for more than one pond or tank to be affected simultaneously by this parasite. Dry and de-silt ponds between crops.

ICHTHYOBODOSIS (ORIGINALLY CALLED COSTIOSIS)

Ichthyobodo necator (previously known as *Costia necatrix*) is a flagellate protozoan that causes the disease ichthyobodosis. It is one of the smallest parasites and is often overlooked during monitoring. *Ichthyobodo necator* is particularly dangerous for fingerlings and larger fish held at high stocking densities in tanks and cages. Outbreaks have been common in recirculating aquaculture systems (**Fig. 33**). *Ichthyobodo necator* exists in a detached, mobile form or an attached form; the latter usually feeding on gill tissue (**Fig. 34**). *Ichthyobodo necator* causes disease over a wide temperature range. Epizootics have been uncommon during grow-out of silver perch in ponds.

Pathogen

Ichthyobodo necator free-swimming form, kidney-shaped, dorso-ventrally flattened, 10–20 µm in size. Two flagella attached to ventral part of body; attached form, pear-shaped (**Fig. 35**). Some evidence in other fishes of salt resistance in *I. necator*.

Signs

- Chronic morbidity and/or mortality
- Mucus secretion and epidermal sloughing
- Fish may have a 'bluish' sheen to the skin
- Flashing
- Ragged fins
- Hyper-ventilation
- Lethargy and loss of appetite

Figure 32
Ichthyophthirius multifiliis trophonts [arrows] in skin mucus and scales (×100 mag.).
Source: Phil Read

Figure 33
Disease caused by the protozoan, *Ichthyobodo necator*, can be common in tank systems with poor water quality.
Source: Phil Read

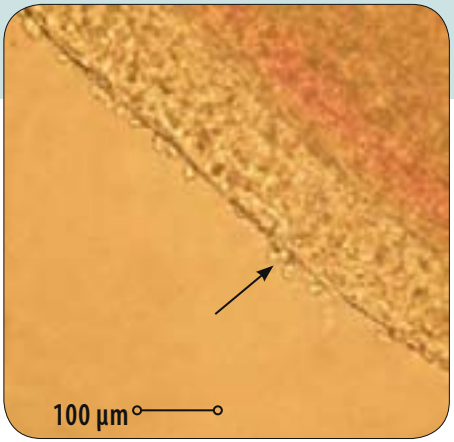

Figure 34
Ichthyobodo necator [arrow] attached to gill tissue (×400 mag.).
Source: Phil Read

Diagnosis

Microscopic examination of gill and skin; parasites often detach from host – prompt examination of fresh tissue. Free-swimming form may exhibit a flicking motion; often rapid swimming around tissue sample; flagella difficult to see; attached pear-shaped parasites seen at 100–400× magnification on gill epithelium (**Figs. 34** and **36**); gill hyperplasia, particularly between secondary lamellae.

Treatment

Tanks:

- salt (NaCl) 10–13 g/L for 60 min, flush and repeat following day; and/or
- formalin 25 mg/L, continuous bath, flush and repeat after 1 or 2 days; aerate water well; no feeding.
- formalin 150 mg/L for 60 minutes (not larvae or fry); observe during treatment; aerate well (oxygen if needed); flush well on completion

Ponds/cages:

- formalin 25–30 mg/L, may need to repeat after 1 or 2 days; maintain 24 h aeration for 4–5 days; one treatment usually sufficient in ponds; repeated treatments may be required in RAS.

Prevention

Quarantine and treat all fish prophylatically, especially fingerlings prior to stocking. Maintain good water quality and nutrition in cages and tanks. Reduce stress and overcrowding. Regularly sample, examine and monitor stock.

TRICHODINOSIS

Trichodinosis is a relatively innocuous disease caused by infestations of the ciliated protozoans, *Trichodina* spp. *Trichodina* spp. are widely distributed, but seldom cause mortalities in large (>100 g) silver perch. *Trichodina* can infect silver perch larvae and fingerlings in large numbers, but rarely cause high level mortality in the short-term. Trichodinosis is often associated with poor water quality and high organic loadings, and is commonly found on juvenile fish that are overcrowded, debilitated with other diseases or in poor condition. Common on post-larvae and fry in larval rearing ponds.

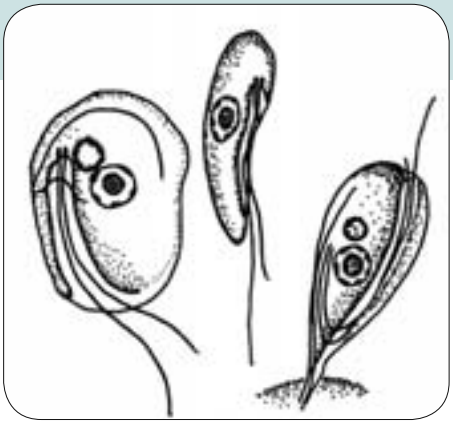

Figure 35
Diagram of *Ichthyobodo necator*.
Source: Phil Read (redrawn from Hoffman, 1967)

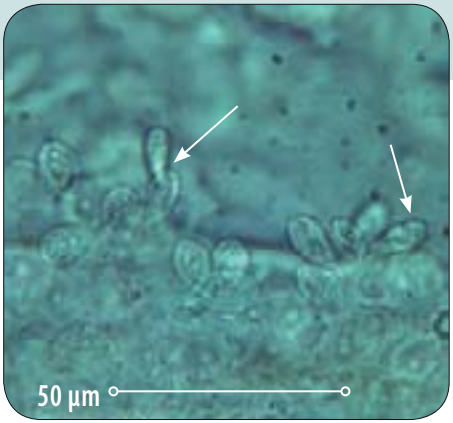

Figure 36
Ichthyobodo necator [arrows], a flagellated protozoan (×1000 mag.).
Source: Stuart Rowland

Pathogen

Trichodina spp. are circular, saucer-shaped, 25–100 µm in diameter with a fringe of cilia around the perimeter; circular arrangement of tooth-like structures within the body; browses over skin and gill surface often with a spinning motion (**Figs. 37** and **38**).

Signs

- Chronic morbidity and/or mortality
- Emaciation
- Excess mucus production
- Frayed fins, skin erosion, dark skin
- Flashing and flaring of opercula
- Lethargy
- Head-up, swimming near surface

Diagnosis

Microscopic examination of skin and gill; easily recognised at 100× magnification and common on skin and fin tissue (**Fig. 39**); observation of low numbers (e.g. 1–3 parasite/field of view) is inconsequential. Increasing levels of infestations to above 20 parasites/field of view may require treatment.

Treatment

Tanks:

- salt (NaCl) 10 g/L for 60 min;
- formalin 25 mg/L, continuous bath for at least 8 h; aerate water; no feeding.

Ponds/cages:

- formalin 15–20 mg/L, maintain 24 h aeration for 4–5 days, one treatment usually sufficient for ponds; or
- copper (as copper sulfate, $CuSO_4$) 0.2 mg/L, alkalinity must be >50 mg/L, continuous aeration during treatment, one treatment should be sufficient; monitor water quality for 4–5 days after treatment.

Prevention

Quarantine and treat all fish prophylactically (2–5 g/L salt), especially fingerlings prior to stocking. Eliminate poor water quality by reducing feeding, maintaining or increasing aeration, and implementing water exchange. Maintain well-fed larvae and fry, and reduce overcrowding.

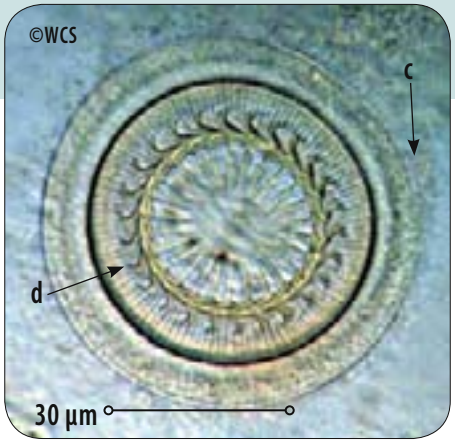

Figure 37
Trichodina sp. showing [arrows] cilia [**c**] and denticles [**d**].
Source: courtesy of www.fishdisease.net

Figure 38
Scanning electron micrograph (SEM) of *Trichodina* sp.
Source: courtesy of www.fishdisease.net

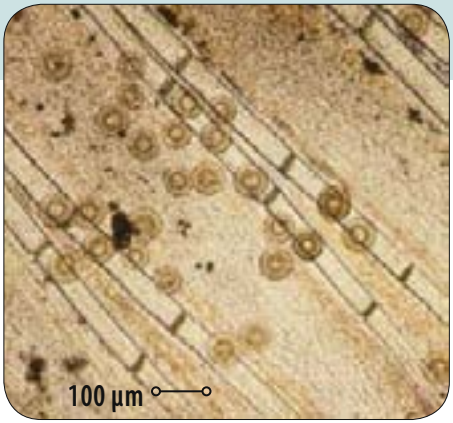

Figure 39
Trichodina sp. a parasitic ciliated protozoan (×100 mag.).
Source: Stuart Rowland

TETRAHYMENOSIS

Tetrahymena spp. are free-living saprophytic ciliates that cause the disease tetrahymenosis in silver perch; however, it is rarely a problem on farms. *Tetrahymena* spp. are similar in appearance to *Chilodonella hexasticha* but are usually more pear-shaped and often larger (up 100 µm in length) (**Fig. 40**). Severe losses have occurred in silver perch fry associated with a high organic load in the water. In advanced cases in other fish species such as carp, catfish, and salmon, the parasite can penetrate muscle tissue and organs causing swelling, necrosis and ulceration.

Pathogen

Tetrahymena is pyriform or radially symmetrical, ovoid body 30–60 µm wide, 50–100 µm long; evenly-distributed cilia; movement is often 'spiralling' compared to the 'gliding' of *C. hexasticha*. Granular appearance of cytoplasm (**Fig. 41**). May be confused with nonpathogenic ciliates.

Signs

- Chronic morbidity and/or mortality
- Excess mucus production
- Epithelial damage, e.g. localised swelling, ulceration, and necrosis
- Loss of appetite and lethargy
- White patches or spots on skin
- Emaciation

Diagnosis

Microscopic examination of skin and gill tissue; parasite recognisable at 100× magnification; relatively fast 'gliding' movement; penetration of *Tetrahymena* into muscle or organ tissues possible; however, few cases reported for silver perch.

Treatment

Tanks:

- formalin 25 mg/L, continuous bath for at least 8 h; aerate water; no feeding.

Ponds/cages:

- formalin 15–20 mg/L; maintain 24 h aeration for 4–5 days; one treatment usually sufficient for ponds; systemic infections may be difficult to treat.

Prevention

Improve water quality; reduce stress and overcrowding.

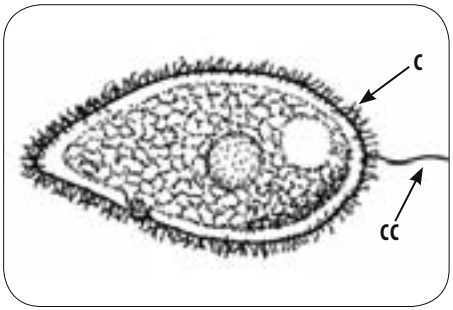

Figure 40
Tetrahymena sp. – diagram with key characteristics [arrows]; cilia [**c**], caudal cilium [**cc**].
Source: Phil Read

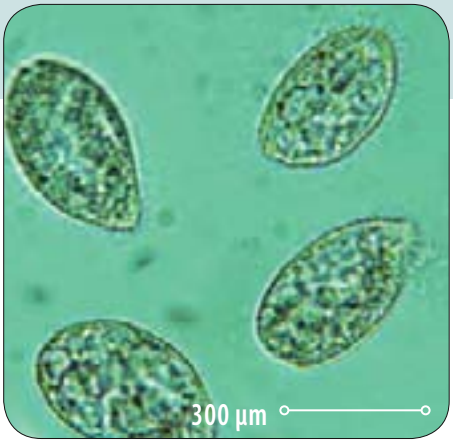

Figure 41
Tetrahymena sp., a ciliated protozoan (×200 mag.).
Source: Stuart Rowland

MYXOZOAN INFECTIONS

Many species of myxosporidians are found worldwide in a variety of fish hosts. They are obligate parasites with different life stages and intermediate hosts. There have been few records of myxosporidians causing disease in silver perch and light infections usually do not cause mortalities or affect growth. However in one epizootic, a high infection rate (>90% of fish affected) of *Henneguya* sp. on silver perch (300–450 g) resulted in chronic mortality (40/day) over two weeks before the disease was controlled. Gill function was severely compromised; most secondary lamellae were invaded by multiple spore-producing, histozoic plasmodia. There is evidence *Henneguya* spp. utilise annelids (worms) as an intermediate host in their lifecycle. The lifecycle of the species infecting silver perch it not known. Most infestations have occurred at temperatures >20°C.

Pathogen

Myxosporidian (*Henneguya* spp.), spore size can vary, 8 to15 µm × 4 to10 µm, tail 40 to 50 µm; two polar capsules and two caudal appendages (**Figs. 42, 43** and **44**).

Signs

- Chronic fish mortality
- Fish listless at pond surface; individual or small groups
- Loss of appetite
- Gills grossly thickened with nodules
- Poor growth and emaciation

Diagnosis

Grossly thickened gill lamellar tissue with mildly nodular appearance (**Fig. 45**).

Microscopic examination of gills, 100× magnification; dark brown/tan encysted plasmodium (**Fig. 46**); intra-lamellar, ~ 80–150 µm in diameter, ovoid to round in shape, inflammatory response marked in resolving plasmodia; squashed preparation of plasmodium, spores as per description above (**Fig. 47**); plasmodium could be confused with 'Ich'. No motility of spores or encysted plasmodia.

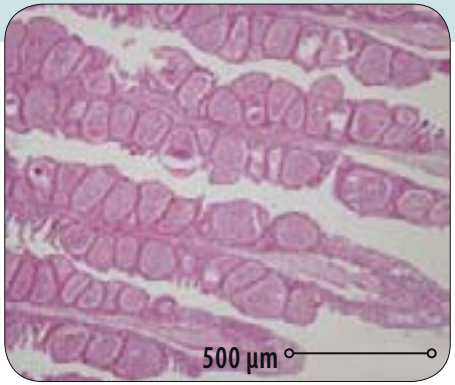

Figure 42
Silver perch gills severely compromised by *Henneguya* sp.– histological section showing the spore-producing histozoic plasmodia.
Source: Matt Landos

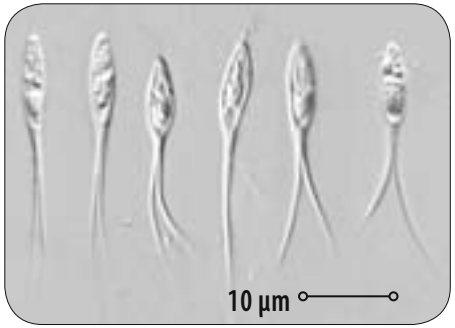

Figure 43
High magnification of *Henneguya* spores.
Source: Prof.Iva Dykova, Institute of Parasitology, Czech Republic

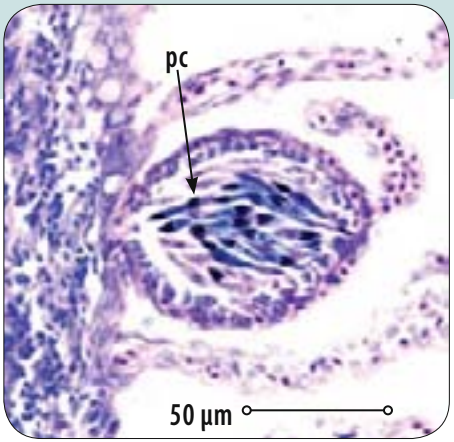

Figure 44
Histological section of *Henneguya* plasmodia showing internal spores [arrow] each with two polar capsules [**pc**].
Source: Prof.Iva Dykova, Institute of Parasitology, Czech Republic

Treatment

Ponds/cages:

Treatments not well defined;

- formalin, 25 mg/L may control outbreaks by targeting free-swimming spore stages; re-treat after 2–3 days; aerate 24 h for several days.

Prevention

Quarantine fish before stocking, examine a sub-sample microscopically. Maintain good water quality and appropriate stocking densities; dry and de-silt ponds regularly (every 1 to 2 years); control intermediate hosts. Disinfect tank systems and equipment on a regular base.

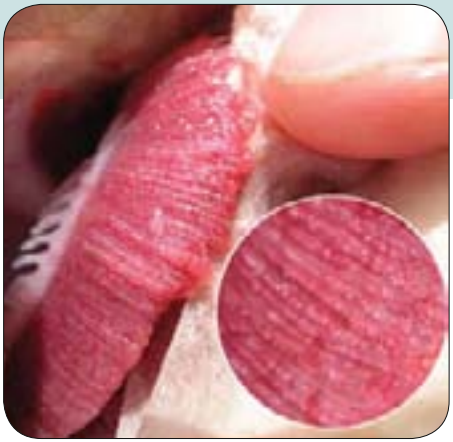

Figure 45
Silver perch gills; grossly visible changes caused by the myxosporidian, *Henneguya* sp.
Source: Phil Read

Figure 46
Henneguya plasmodia on silver perch gills (×100 mag.).
Source: Phil Read

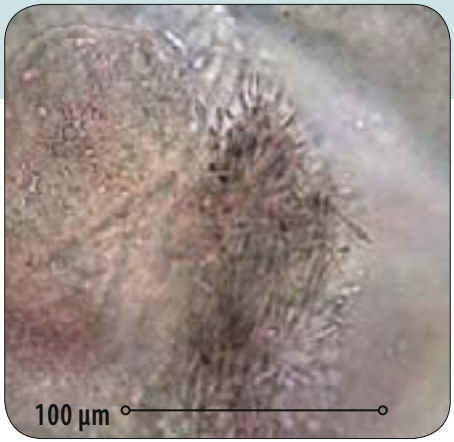

Figure 47
Wet mount of *Henneguya* spores (×400 mag.).
Source: Phil Read

ECTO-COMMENSAL CILIATE INFESTATIONS

Species of sessile, ecto-commensal ciliates have rarely been recorded on silver perch; to date all cases have been identified as *Ambiphyra* spp. Parasites attach to skin or gill tissue by a holdfast (scopula) often in colonies (**Fig. 48**). The parasites feed on bacteria and organic matter in the water and use the host primarily for attachment. There have been no reports of problems with this parasite in the silver perch industry. Large numbers on the gills could potentially impede gas exchange, metabolic function and/or offer a site for bacterial and fungal infection (**Fig. 49**).

Pathogen

Bell or goblet-shaped, elongated ciliates; attached either by stalk or scopula; spiral of cilia at posterior end (**Fig. 50**); some species having ciliary girdle mid-body; most range 40–100 µm in length; some species solitary; some colonial. No obvious motility.

Signs

- Hyperventilation (heavy infestations)
- Emaciation
- Flashing

Diagnosis

Microscopic examination of gill (mainly), fin and skin tissue; easily recognisable at 100× magnification; identification to species not needed; treatment the same for all species

Treatment

Tanks:

- formalin 25 mg/L, retreat after 2–3 days; aerate water; no feeding.
- salt 10 g/L (NaCl) for 60 min or prolonged immersion at 5 g/L; some species may be resistant.

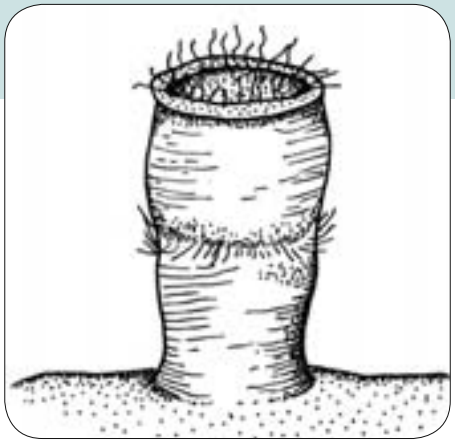

Figure 48
Diagram of key characteristics of *Ambiphrya* sp.
Source: Phil Read (redrawn from Noga, 2000 & Hoffman, 1967)

Figure 49
Ambiphrya sp., a ciliated, ecto-commensal parasite on silver perch gills (×100 mag.).
Source: Unknown

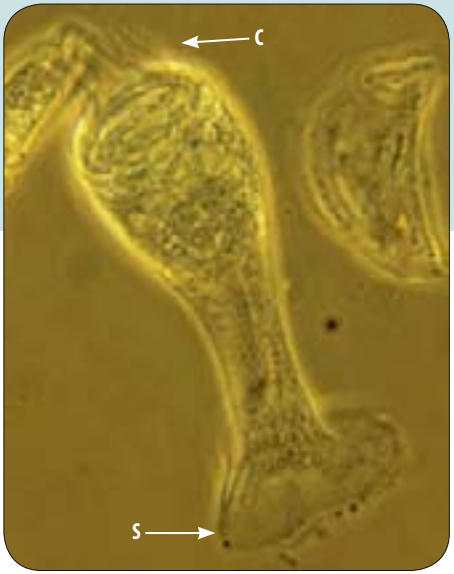

Figure 50
Ambiphrya sp., showing [arrows] scopula [**s**] and oral cilia [**c**] (×200 mag.).
Source: Brett Ingram

Ponds/cages:

- formalin 15–20 mg/L, maintain 24 h aeration for 4–5 days, repeat treatment after 2–3 days if necessary.

Prevention

Presence is indicative of organically-polluted water that could have a high concentration of bacteria; improve water quality (aeration and exchange) to alleviate the problem and minimise the risk of recurrence.

MONOGENEANS

Monogeneans (skin and gill flukes), in particular *Lepidotrema bidyana* (Dactylogyridae) are commonly found on silver perch in ponds, cages and tanks. They have become widely distributed in the industry over the last 10 years, and the parasites now occur on most farms. This rapid spread demonstrates the risk of moving pathogens with live fish and the need for stringent on-farm quarantine measures to be implemented to avoid the transfer of pathogens between farms and onto farms from wild fish. Gill flukes attach using their posterior, haptor organ to gills (adult flukes) and to skin (juveniles). Heavy infestations (>15 parasites per field of view) of flukes have not caused mortalities in silver perch; however, the parasite may cause stress, poor feeding response and growth, tissue damage and interference with gill function, predisposing the fish to fungal and bacterial diseases (**Fig. 51**). Eradication of *L. bidyana* from ponds and tanks is difficult with current therapeutic agents. Infestations of a similar parasitic group in the family Gyrodactylidae have also been recorded on silver perch (**Fig. 52**).

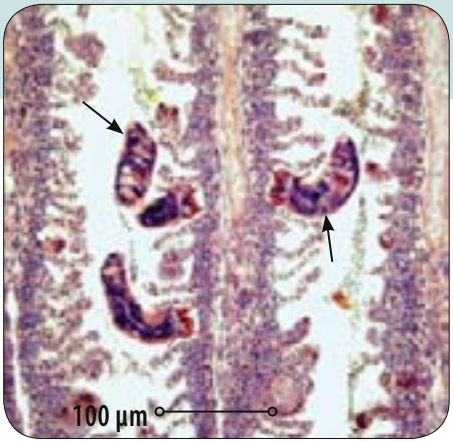

Figure 51
Histological section of gill showing the monogenean *Lepidotrema bidyana* [arrows].
Source: Prof. Iva Dykova, Institute of Parasitology, Czech Republic

Figure 52
Gyrodactylus sp. showing [arrows] haptors [**h**] on the adult and the internal larva (×100 mag.).
Source: Stuart Rowland

Pathogen

Worm-like parasite, adults up to 600 µm in length; attached posteriorly; oviparous, producing eggs which are released to the aquatic environment; eggs hatch as a free-swimming infestive stage (oncomiracidium); juveniles can develop into gravid adults within 7 days; eyespots and head organs obvious; low numbers of parasites may be present without causing problems or stress.

Signs

- Flashing
- Excessive mucus production on skin and gills
- Gill hyperplasia
- Loss of appetite
- Emaciation (heavy, prolonged infestations)

Diagnosis

Easily identified; microscopic examination of gill at 40–100× magnification (**Fig. 53**); both adults (gill) and juveniles (skin) show characteristic stretch and recoiling motion.

Treatment

Monogeneans can re-infest silver perch between 5 and 30 days after treatment with formalin or trichlorfon (an organophosphate), depending on water temperatures and degree of pond contamination with eggs. Up to three treatments, 21 days apart may be required to eradicate or reduce monogenean numbers to an insignificant level.

Tanks:

- formalin 30 mg/L, aerate water, no feeding; or
- formalin 150 mg/L, 30 minute bath; or
- trichlorfon 0.25 mg/L active ingredient indefinite bath; or
- salt 15 g/L, 1 h bath, repeat following day; or

Ponds/cages:

- trichlorfon 0.5 mg/L active ingredient, indefinite bath;
- formalin 30 mg/L (when water <25°C), maintain 24 h aeration for 4–5 days; monitor DO daily.

Prevention

Quarantine and treat all incoming fish prophylactically (2–5 g/L plus 30 mg/L formalin), especially fingerlings prior to stocking. Gill flukes are usually indicators of poor water quality, improve water quality. Frequently monitor gill fluke numbers. Monogeneans can be persistent in tank systems necessitating regular treatments and periodic drying. Dry ponds regularly and use calcium hydroxide [$Ca(OH)_2$] or calcium oxide (CaO) liberally on any persistently, damp areas.

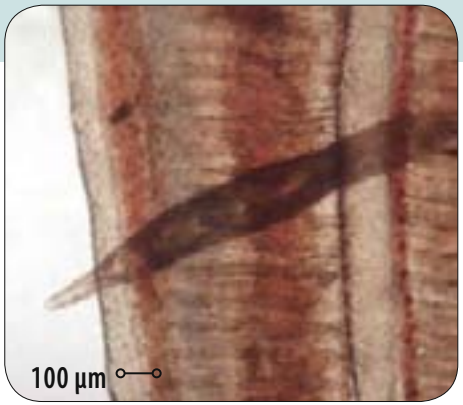

Figure 53
Lepidotrema bidyana, a large mongenean gill fluke attached to silver perch gill tissue (×100 mag.).
Source: Phil Read

COPEPODS

Copepods are crustaceans with a complex life cycle, developing through egg, nauplii and copepodid larval stages before attaching and maturing as adults on the host. The freshwater, parasitic copepods, *Lernaea* spp. and *Ergasilus* spp. have been recorded on silver perch.

LERNAEA SPP. (ANCHOR WORM)

Anchor worms possess anchor-like processes for securing themselves to the host (**Fig. 54**). Silver perch farms in the Murray-Darling Basin may have a high incidence of anchor worm because common carp (*Cyprinus carpio*) are often carriers of the parasite. However the parasite has also been recorded on silver perch (and other freshwater species) in the eastern drainage. Parasites can infest individual fish in high numbers (100's) without causing mortality; however, poor feeding response and growth has been recorded. Attachment sites are often areas for secondary bacterial or fungal infections. Numbers of parasites may increase in slow-flowing or static water sources when carp and native fish become concentrated. Anchor worm is more common in summer, but the parasite can occur year round. Marketability of fish infested with *Lernaea* is compromised due to the presence of small, red lesions.

Pathogen

Anterior end of adult female buried in flesh of fish (**Fig. 55**); body cylindrical, wormlike; cephalic segment with two to four soft horns; adult female up to 20 mm in length; paired egg sacs greenish, conical or ovoid at posterior end (**Fig. 56**); eggs hatch 1–3 days, nauplius metamorphose into copepodids 4–16 days, completion of several developmental stages prior to copulation, female attaches; male disappears, presumably dying; life cycle temperature dependant.

Figure 54
An anchor worm head showing organs use to attach below the epidermis.
Source: Phil Read

Figure 55
Lernaea sp. [arrow] attached to the soft tissue of a silver perch dorsal fin.
Source: Parri & Craig Anderson

Signs

- Anchor worms clearly visible to naked eye
- Haemorrhaging and red lesions at site of attachment (**Fig. 57**)
- Emaciation and poor growth
- Flashing

Diagnosis

Macroscopic examination of external features of fish; gravid females easily recognisable by eye attached to gills and skin, often on areas having 'softer' scale cover such as soft ray tissue, mouth and nares; Microscopic and macroscopic examination of gills; small immature stages, such as copepodids may not be grossly visible.

Treatment

Tanks:

- removal of individual parasites with forceps;
- trichlorfon 0.25 mg/L active ingredient, indefinite bath;
- salt 10 g/L, 1 h bath, repeat daily.

Ponds/cages:

- trichlorfon 0.5 mg/L active ingredient, indefinite bath; repeat every 7 days for 28 days.
- repeated treatments required to prevent re-infestation by emerging larval stages of *Lernaea*.

Prevention

Quarantine and prophylactic treatment prior to stocking; lowering of stocking density; improvement in water quality; ensure water source and storage reservoir free of carp and other fish.

Figure 56
Lernaea sp., a parasitic copepod with egg sacs [**es**] [arrow].
Source: Phil Read

Figure 57
Haemorrhaging caused by the copepod *Lernaea* sp.
Source: Ian Charles

ERGASILUS SP.

Infestations of the copepod, *Ergasilus* sp., have rarely been recorded on silver perch farms. Ergasilids are often described as 'gill maggots' due to the appearance of white egg sacs attached to the adult females. The parasite's clasping attachment causes severe gill damage and interference with gill function (**Fig. 58**). In one case, pond-reared, silver perch (>500 g) suffered a heavy ergasilid infestation (species unknown). Fish were nutritionally challenged, displayed long periods of inappetence, recorded relatively poor growth and were easily stressed during harvest. Parasites were attached to the gills. No mortalities were recorded in ponds. Damage to gill tissue caused by ergasilids can lead to secondary bacterial or fungal infections.

Pathogen

Distinct cephalothorax and abdomen, 1 mm length; large, paired, claw-like antennae (used for locomotion), length, 1 mm; female having posteriorly attached white egg sacs, length,1–2 mm; eggs ~100/sac, 0.05 mm diameter, colour darkens on maturity (**Fig. 59**). Life cycle, egg hatching to free-swimming nauplius; several copepodid developmental stages via moulting; copulation at free-swimming stage; females enter gill cavity and attach to rakers and gills.

Signs

- Flashing
- Loss of appetite
- Poor growth
- Haemorrhaging of fins
- Patchy/blotchy, dark skin
- Stress following handling
- Strong reaction to 'light' anaesthesia
- Gill hyperplasia

Diagnosis

Macroscopic examination of gills, 'gill maggots'; easily recognisable on gill tissue at 40× magnification; immature forms may not be grossly visible.

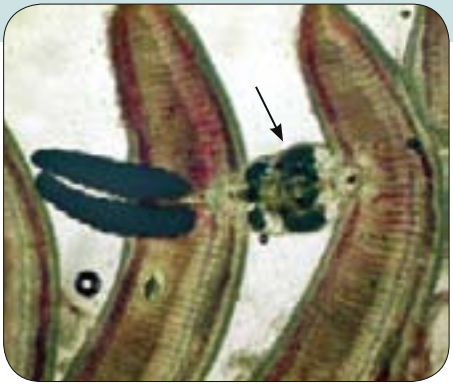

Figure 58
Ergasilid sp. [arrow] attached to gills.
Source: courtesy of www.fishdisease.net

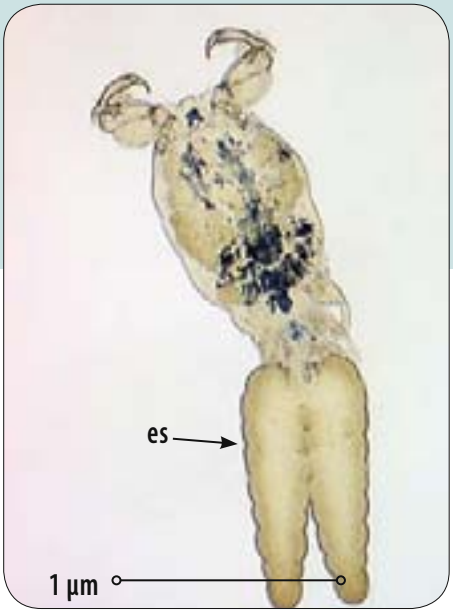

Figure 59
Ergasilid sp. showing body with grasping antennae, head, thorax, abdomen and egg sacs [**es** arrow].
Source: courtesy of www.fishdisease.net

Treatment

Tanks:

- trichlorfon 0.25 mg/L active ingredient; or
- salt (NaCl), 10 g/L continuous for 3 days.

Ponds/cages:

- trichlorfon 0.5 mg/L active ingredient, prolonged immersion.

Prevention

Quarantine and prophylactic treatment prior to stocking; lowering of stocking density; improvement in water quality; use of high quality feeds.

CESTODES AND NEMATODES

Cestodes and nematodes (tapeworms and roundworms respectively) are endoparasites inhabiting the intestine of many vertebrates including fish. Both parasites have similar life cycles with at least one intermediate host (e.g. copepod, insect nymphs, worms). The final host can be piscivorous birds or predatory fish. Some cestode and nematode larval stages (plerocercoids) are commonly found in the viscera or musculature of some fish. Long, white, ribbon-like cestodes (species unknown) have been reported protruding from the anus of silver perch held in purging systems (**Fig. 60**). Comparatively, nematodes are characterised by a slender and cylindrical body having tapered ends and no true segmentation. Nematodes (species unknown) have been found in the digestive tract of silver perch and a species of the genus *Camallanus*, has parasitised the gut of Barcoo grunter (*Scortum barcoo*) (**Figs. 61** and **62**). Normal behaviour and growth was reported for silver perch parasitised by cestodes and nematodes.

Pathogen

Cestodes: up to 40 cm; body long, ribbon-like; white to cream colour; scolex (head) not pronounced having adhesive grooves (bothria). Nematodes: slender, cylindrical body, covered with cuticle; up to 10 mm length; red in colour.

Figure 60
Large (~ 40 cm) cestode (species unknown) found in the intestine of silver perch.
Source: Phil Read

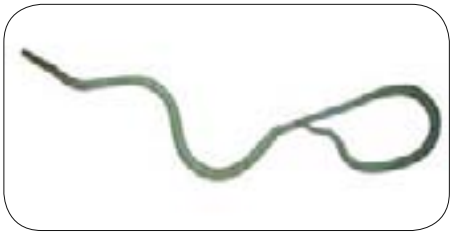

Figure 61
Intestinal (~ 2 cm) nematode (species unknown).
Source: Phil Read

Signs

- Worms protruding from vent (**Fig. 63**)
- Swollen abdomen

Diagnosis

Large worms identified grossly; wet mounts of faecal matter may be useful in determining the presence of eggs or larvae. Microscopic examination of physiological features (scolex, shape, size, and segmentation) required to identify species. Samples should be sent to a reference laboratory for definitive identification.

Treatment

There are currently no treatments which are registered, or the subject of an Australian Pesticides and Veterinary Medicines Authority (APVMA) permit. Chemicals which have registration in other food producing species of animals (non-fish) may be used under an off-label prescription from a registered Veterinary Surgeon.

Prevention

Regularly dry, de-silt and disinfect ponds using calcium hydroxide or calcium oxide. Minimise access of animals that could be carrying parasites, including birds and turtles.

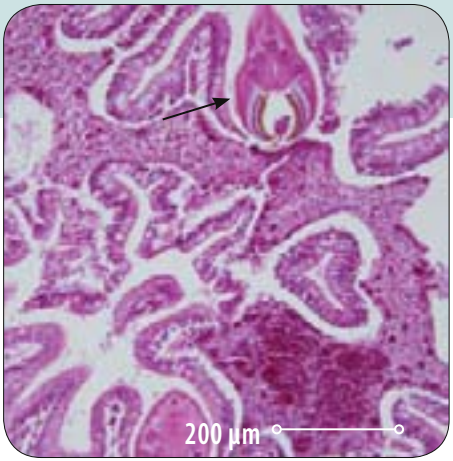

Figure 62
Histological section of the intestine of Barcoo grunter (*Scortum barcoo*) showing the nematode *Camallanus* sp. [arrow] attached to the gut wall.
Source: Matt Landos

Figure 63
Nematode (species unknown) protruding from the anus of a silver perch.
Source: Matt Landos

FUNGAL DISEASES

Fungi or water moulds belong to the Class Oomycetes and are ubiquitous in freshwaters. They form a large group of saprophytic organisms that feed opportunistically on dead organic matter. Fungi are important pathogens of fish. They are generally considered to be secondary pathogens, with infections often induced by stress, physical damage and/or immuno-suppression. Fungal infections are inherently difficult to treat because of the complex biology of aquatic fungi.

SAPROLEGNIOSIS

The fungus, *Saprolegnia parasitica*, causes the disease saprolegniosis in silver perch. Fungal infections in silver perch are common after rough handling, removal of mucus and damage to the epidermis. A new, important disease in silver perch, winter saprolegniosis, usually affects larger (>250 g) fish (**Fig. 64**) and is prevalent at temperatures <16°C, usually following rapid decreases in water temperatures (April – August) associated with cold fronts in winter; 100% mortality has been recorded in some ponds on farms, and epizootics can be severe and rapid (4–6 days). *Saprolegnia parasitica* has been recorded on silver perch fingerlings (25 mm length) held in tanks following a temperature decrease (12 to 8°C) over two days. It is associated with superficial fungal infections of the skin and gills.

Pathogen

Opportunistic water mould; white mycelial growth on skin and gills (**Fig. 65**); often coloured by trapped organic matter (**Fig. 66**); club-shaped hyphae (20 µm diameter) release zoospores (sporulation); spores motile in water; hooked, hair-like projections may assist spores attachment to host skin and gills of live fish (**Figs. 67** and **68**); pathogenicity appears to increase with decreasing water temperatures (<16°C).

Figure 64
The fungus, *Saprolegnia parasitica*, growing on large silver perch.
Source: Phil Read

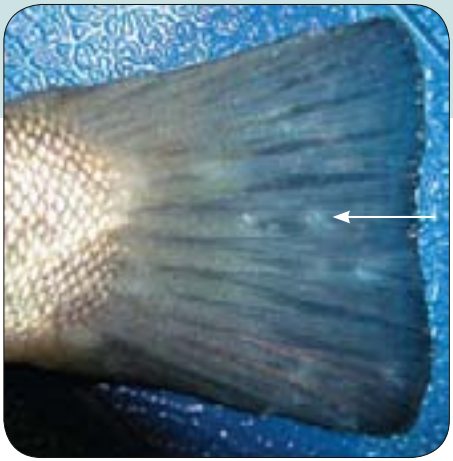

Figure 65
Early stage of saprolegniosis [arrow] on the caudal fin of silver perch.
Source: Phil Read

Signs

- Abnormal swimming and behaviour (fish at pond edges, in currents, on surface, isolated)
- Focal areas of pale skin (early stages)
- Haemorrhaging on underside of abdomen (early stages) (**Fig. 69**)
- Light fungal growth on caudal fin (early stages)
- White 'cotton wool-like' patches on skin or gills (advanced stages)
- Fungal plaques attached to the soft tissue on the distal end of the opercula (early stages)

Diagnosis

Macroscopic examination of skin and gills; pale and/or haemorrhagic areas, white to green-brown coloured fungal growth. Microscopic examination of skin and gills; fungal hyphae of variable widths (10–30 µm); sporangium often with zoospores; easily diagnosed at 100× magnification; fungal growth (mycelium) not so obvious in early stages.

Treatment

Treatment difficult, many fungal infections show resistance to chemotherapy; recovery related to the amount of skin and gill infected by fungus; gill infections are usually terminal.

Tanks:

- salt (NaCl) 2–5 g/L preventative treatment, continuous, indefinite bath (adults, juveniles, larvae); elevated temperature may assist, maintain >20°C;
- eggs, larvae, salt 2 g/L preventative treatment, continuous, indefinite bath;
- eggs, formalin 1,000 mg/L for 15 mins daily (used to control saprolegniosis on Murray cod eggs).

Figure 66
Saprolegniosis of the gill tissue; mycelium contaminated with organic matter.
Source: Phil Read

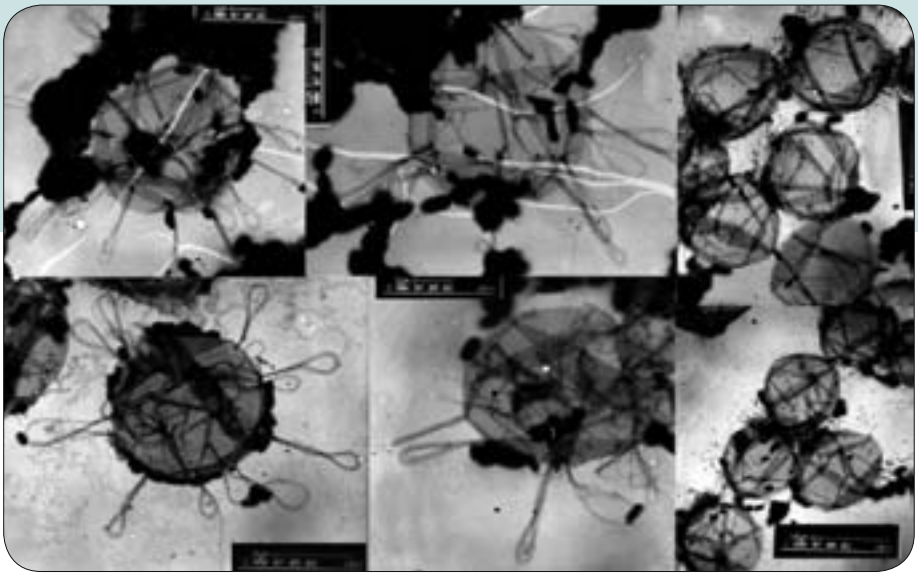

Figure 67
Electron micrograph of *Saprolegnia parasitica* spores.
Source: Dr Gordon Beakes (University of Newcastle upon Tyne, UK)

Ponds/cages:

- emergency harvest is often the only way to avoid a major loss from saprolegniosis. Fish need to be dip bathed in a high concentration of formalin 100 mg/L for 30 minutes, prior to being held in a very clean recirculation system with at least 2–5 g/L salt.
- formalin 30 mg/L initially (when water temperature <25°C), then maintaining levels between 25–30 mg/L daily (see chapter *Calculations, Treatments and Dose Rates*), until disease controlled; aeration continuous and supplemented; or
- copper (as copper sulfate, $CuSO_4$) 0.1–0.2 mg/L, recommend 0.2 mg/L initially, then monitor and adjust free Cu^+ ion levels daily to maintain copper concentrations between 0.1 and 0.2 mg/L until disease controlled; continuous aeration during treatment; alkalinity must be > 50 mg/L. Copper may provide some degree of prevention if used regularly up to and throughout the high risk period of winter.

Prevention

Salt bath (2–5 g/L) prevents or slows rate of infection in tanks. Eliminate or reduce predisposing stressors late summer/autumn when water temperatures start to fall < 18°C; ensure fish free of ecto-parasites; treat parasitic diseases; reduce feeding; reduce high organic load in ponds (dead fish and organic matter, including feed are fertile substrates for colonisation of the fungus) (**Fig. 70**); reduce biomass especially large (>500 g) fish; maintain good water quality, especially following crashes of algal blooms; avoid handling and skin damage; on farms where winter saprolegniosis is a problem, weekly applications of formalin or copper may be beneficial after temperatures decline to 16°C in late autumn/winter; remove daily any dead fish (or eggs); dry and clean ponds between crops.

Figure 68
Saprolegnia parasitica oogonium.
Source: Matt Landos

Figure 69
Mild haemorrhaging on the lower abdomen of silver perch – early stages of the disease saprolegniosis.
Source: Phil Read

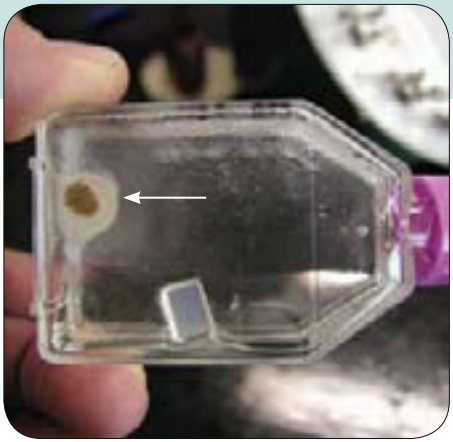

Figure 70
Saprolegnia parasitica growing [arrow] on fish feed.
Source: Matt Landos

EPIZOOTIC ULCERATIVE SYNDROME (EUS, RED SPOT DISEASE)

The aquatic fungus, *Aphanomyces invadans*, causes EUS on silver perch farms located in coastal regions in north-eastern NSW and south-eastern Queensland. The fungus is known to cause serious losses of farmed fish in Asia and estuarine fish in Australia; the latter usually following periods of high rainfall and subsequent decline of water quality (low salinity, low pH, high organic load). The disease can occur periodically on silver perch farms that have a surface water supply. Epizootics can cause high morbidity, particularly in juvenile (<100 g) fish; 100% infection rates are common. Mortalities in juvenile silver perch can be significant when the fish develop large, deep, necrotic ulcers. The disease is less of a problem in larger silver perch that often recover from infection, with whorls of scales a hallmark over the repaired area.

Pathogen

Atypical water mould, *Aphanomyces invadans*; differs from typical mould *Saprolegnia parasitica*, in having hyphae that penetrate skin and musculature; causes small to large red lesions and ulcers (4–25 mm diameter); severe and chronic inflammatory reaction; generally on body and caudal regions (**Figs. 71** and **72**); sloughing of necrotic tissue often leaves a deep, crater-shaped cavity; significant potential for secondary bacterial infections.

Signs

- Focal areas of pale skin, raised scales (early signs)
- Small red lesions and ulcers (10 mm), superficial, causing muscle tissue inflammation (myositis)
- Deep, red ulcers
- Fish mortality and/or morbidity
- Abnormal swimming (fish at pond edges, in currents, on surface, isolated)
- Loss of appetite (heavy infections)

Figure 71
The fungus, *Aphanomyces invadans*, causing EUS (red spot disease) on silver perch.
Source: Phil Read

Figure 72
Deep, necrotic ulcer – EUS on a juvenile silver perch.
Source: Phil Read

Diagnosis

Initially, macroscopic examination; pale patches of skin, small, single scale-sized red lesions, small ulcers to large, deep (exposing skeletal muscle) necrotic ulcers in advanced cases. Microscopic examination (x100 mag.) of 'squashed' muscle tissue underlying ulcers; presence of fungal hyphae, 12–18 µm in diameter, amongst muscle fibre (**Fig. 73**).

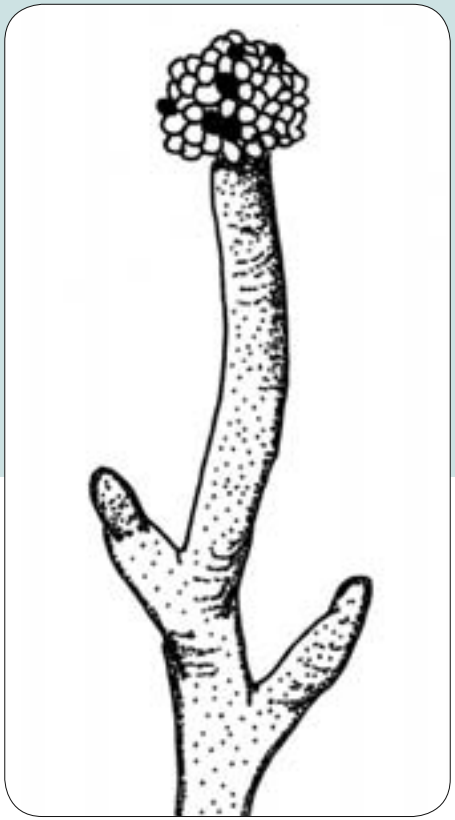

Figure 73
Diagrammatic representation of *Aphanomyces invadans* zoosporangia.
Source: Phil Read

Treatment

No known treatment of infected fish. Recovery has been recorded in large silver perch after improvement in water quality.

The following assist in reducing new infections through killing the infectious zoospore stage.

Tanks:

- formalin, 25 mg/L continuous bath and 50 mg/L, 1 h bath; or
- salt (NaCl) 10 g/L 1 h bath, then 5 g/L indefinite bath.
- transfer of fish to clean tank, lower stocking density (chlorinate de-stocked tank).

Ponds:

- emergency harvest, treatment (as above) and re-stocking to new ponds proven beneficial in recovery of advanced cases in fingerlings; re-stocking of large fish having early signs (small lesions to medium sized ulcers) has helped fish heal naturally; improve water quality.

Prevention

Avoid excessively high stocking densities and high biomasses. Outbreaks known to occur under one or combinations of the following predisposing conditions; infestations of ecto-parasites, poor water quality, especially high or low pH, high organic matter, days of extreme hot weather, high rainfall leading to a 'fresh' in source water. Do not pump water from rivers and creeks with a 'fresh', in flood or containing infected fish. Maintain good water quality; dry ponds regularly. Spelling surface water in a fish-free environment for 7–10 days is likely to be sufficient to eliminate infectious zoospore stages of the fungus.

BACTERIAL DISEASES

Bacteria form a large, diverse group of small, ubiquitous organisms that play a key role in nature. Bacterial cells are simple, do not contain complex organelles and can divide rapidly. Some bacteria cause common and damaging diseases in fish. Most bacterial diseases result directly from environmental stress such as poor water quality and/or rough handling. To date, seven bacterial diseases have been reported in silver perch. However, other than conditions related to poor handling, silver perch appear to be robust and largely resistant to bacterial infections. Systemic infections are very uncommon in silver perch.

COLUMNARIS

The bacterium, *Flavobacterium columnare*, causes the disease columnaris. *Flavobacterium columnare* has a worldwide distribution, is common in the pond environment and can rapidly infect fish causing high mortalities. The disease is not common in silver perch, although it has caused mortalities in fingerling and mature silver perch, but usually following stressful precursors such as physical injury, other disease and crowded conditions associated with low DO and high organic loads. *Flavobacterium columnare* can be invasive once established, through production of a range of tissue toxins; pathogenicity is usually higher at temperatures >20°C.

Pathogen

Slender, filamentous gram negative rod; 0.4 µm diameter, 4 to 10 µm length; lacking flagella, motile by gliding mechanism; preference for aerobic conditions colonising gills and skin; prevalent at temperatures >20°C.

Signs

- Chronic to acute fish morbidity or mortality
- Erosive and necrotic skin lesions on head and caudal regions (**Figs. 74** and **75**)
- Lesions 'whitish' colour, with inflamed, red periphery
- Frayed and eroded fins
- Gill infection, eroded lamellae with yellow to white necrotic edge
- Ulcers with 'yellowish' pigmentation

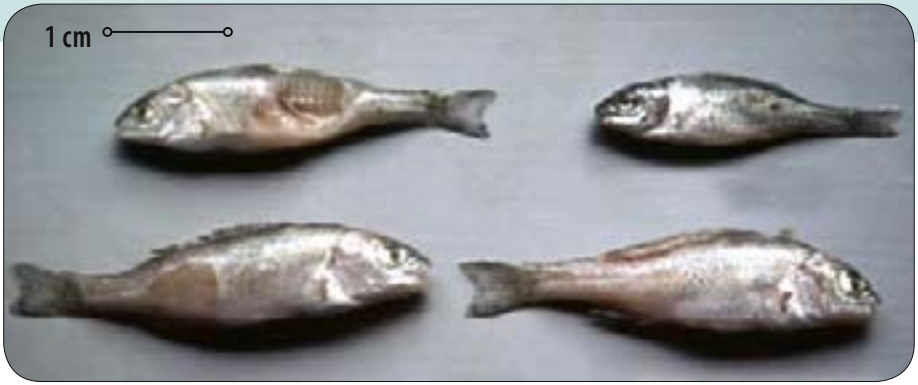

Figure 74
Columnaris bacterial infection in silver perch fry.
Source: Phil Read

Diagnosis

Microscopic examination of wet mounts of lesions (skin and gill); 1,000× magnification; bacteria as slender rods having characteristic gliding motion; bacteria usually congregate into colonies giving a 'haystack' appearance hence the name columnare; lesions may have secondary fungal infection.

Treatment

Tanks:

- oxytetracycline 20 mg/L active ingredient, 7 days at 20–30°C or 10 days at <20°C, continuous bath, maintain low light levels, good aeration, water exchange to 'dilute' bacterial load and re-treat daily; This organism is commonly sensitive to this antibiotic; however, laboratory sensitivity testing is recommended.
- salt (NaCl) 2 g/L continuous bath may assist in recovery by preventing fungal infections.
- lesions on individual fish can be swabbed with 20% iodine solution.

Ponds:

- potassium permanganate ($KMnO_4$) 2 mg/L a.i., higher levels may be required in ponds with high organic matter; total $KMnO_4$ not to exceed 6 to 8 mg/L, $KMnO_4$ becomes inactive when water colourless or light brown; in systemic infections, oxytetracycline as feed additive for 10 days, 50–100 mg/kg fish/day.
- copper (as copper sulfate) 0.2 mg/L prolonged immersion, maintain level; higher $CuSO_4$ dosages dependant upon total alkalinity, alkalinity must be >50 mg/L; continuous aeration during treatment.

Prevention

Avoid poor handling of fish, especially exposure to any periods of low DO; avoid harvesting at high temperatures (>28°C); avoid overstocking/high organic loading during harvest procedures; maintain good water quality. Ensure rapid solids removal in tank systems and maintain appropriate stocking levels (e.g. up to 40 kg/m^3 <18°C; up to 30 kg/m^3 >18°C).

Figure 75
Deep ulceration on the caudal peduncle of silver perch caused by the bacterium, *Flavobacterium columnare.*
Source: Phil Read

'TAIL ROT SYNDROME'

Tail rot syndrome or fin rot, is a relatively common bacterial disease in juvenile silver perch. It occurs mid to late summer when fingerlings are harvested and transported from ponds to quarantine tanks, or when fingerlings are transported from hatcheries and grow-out farms. The disease is characterised by necrosis of the caudal fin and distal part of the caudal peduncle and usually occurs as a result of rough handling, over-crowding, low DO, high temperatures, high organic loading and inappropriate quarantine procedures between harvest and transport periods. Mixed infections by the bacteria belonging to the Flavobacterium, Pseudomonad, Vibrio or Aeromonad bacterial groups most likely cause the disease.

Pathogen

Flavobacterium spp. as for columnaris; *Aeromonas* spp. (e.g. *A. hydrophila*), 0.8 to 1.0 µm diameter and 1.0 to 3.5 µm long, short, motile rods by a single polar flagellum, ubiquitous found in most freshwaters; *Pseudomonas* spp. (e.g. *P. fluorescens*); similar to *Aeromonas*; these bacteria are often natural inhabitants of fish mucus.

Signs

- Eroded tail fin
- Necrotic tissue on caudal peduncle
- Dark colour, sometimes blotchy – fry/fingerings
- Lethargic, often near tank surface or facing current or airstones
- Chronic mortality following harvest/ transport

Diagnosis

Gross appearance; fish with frayed edges and reddened caudal fin in early stages (**Fig. 76**); skin of the distal caudal peduncle having pale, mucoid appearance with focal haemorrhages; more advanced cases, caudal fin eroded to peduncle, ulcerated tissue often exposing skeletal structure (**Fig. 77**); secondary fungal infection common. Definitive diagnosis by fish necropsy, cultures and isolation of bacteria. Mortality increases significantly once bacteria enter circulatory system. Fish having mild cases can completely regrow the soft fin ray tissue if infection resolves.

Figure 76
Early tail rot on the distal end of a caudal fin.
Source: Phil Read

Figure 77
Silver perch fingerlings with tail rot.
Source: Stuart Rowland

Treatment

Tanks:

- oxytetracycline bath, 20 mg/L active ingredient, 7 days at 20–30°C or 10 days at <20°C; maintain low light levels, good aeration; water exchange to 'dilute' bacterial load and re-treat; this syndrome often responds to this antibiotic; however, laboratory cultures and sensitivity results are recommended to ensure appropriate antibiotic usage, plus
- salt (NaCl) 2 g/L continuous bath may assist in recovery by preventing fungal infection; – remove infected, moribund fish promptly, particularly advanced cases.
- restock into recently-filled pond with good water quality – fish can recover once restocked into the low-stress environment of a pond.

Prevention

Minimise stress during harvest procedures; use only knotless nets; avoid harvesting during hot weather, when water quality is poor and DO low; avoid over-crowding; always use bottled oxygen during harvest procedures (pond and fish transporter). Quarantine fingerlings 4–5 days post harvest and prior to transport; provide salt bath (2–5 g/L) for 24–48 hrs post-harvest, clean water and aeration.

ULCERATION CAUSED BY AEROMONAS SALMONICIDA NOVA; ('GOLDFISH ULCER DISEASE', GUD)

An atypical strain of the bacterium, *Aeromonas salmonicida*, the subspecies *nova* causes this ulcerative disease. There has been only one reported outbreak involving silver perch in an earthen pond on a commercial farm that also produced ornamental goldfish (*Carassius auratus*). The typical strain of this bacterium, *Aeromonas salmonicida* subspecies *salmonicida*, causes the serious disease, furunculosis, in salmonids. GUD was probably introduced into Australia by the importation of infected goldfish (**Fig. 78**). *A. salmonicida* has been identified in common carp (*Cyprinus carpio*) and the pathogen can be carried by infected fish without showing symptoms. The disease expresses itself as skin and/or fin ulcers and systemic infections are probably

Figure 78
Goldfish (*Carassius auratus*) with GUD.
Source: Stuart Rowland

likely late in the epizootic; mortality rates in the absence of secondary infections are usually low. Murray cod have shown resistance to GUD, but may act as asymptomatic carriers.

Pathogen

Aeromonas salmonicida nova; non-motile, gram-negative bacterium; isolates grown on agar media are typically grey, small and circular colonies; pathogen may survive long periods off the host fish.

Signs

- Chronic fish morbidity/mortality
- Lethargic swimming and inappetence
- Lesions and ulcers on skin and fins, ranging from superficial, reddened lesions to crater-like ulcers, may have pale or red centres with a peripheral 'pale' halo (**Fig. 79**)
- Fluids in abdominal cavity (ascites)

Diagnosis

Definitive diagnosis requires laboratory examination of infected fish; necropsy, culture and bacteria isolation; isolation of bacteria from internal organs (kidney and spleen) in systemic infections is more revealing than sampling superficial ulcers.

Treatment

Tanks:

- oxytetracycline (pure), bath, 20 mg/L active ingredient, 7 days, at 20–30°C or 10 days at <20°C; maintain low light levels, good aeration; water exchange to 'dilute' bacterial load and re-treat daily;
- antibiotic choice should be based on laboratory sensitivity testing

Ponds:

- prompt oral administration of appropriate antibiotic, determined by laboratory sensitivity testing; oxytetracycline, 75 mg/kg fish for 10 days; antibiotics can only be used under the supervision and prescription of a registered veterinarian; Withdrawal times apply prior to sale of fish.

Prevention

Prevention requires elimination of *A. salmonicida* from the farm and its water supply; goldfish and carp are possible sources of infection and should be eliminated from ponds, drains and water supplies; maintain good water quality; reduce stresses; regularly dry and lime ponds with $Ca(OH)_2$ or CaO (calcium hydroxide or calcium oxide).

Figure 79
Silver perch with GUD, caused by the bacterium *Aeromonas salmonicida* subspecies *nova*.
Source: Stuart Rowland

STREPTOCOCCOSIS

The bacterium, *Streptococcus iniae*, causes streptococcosis in silver perch. The disease has been rarely reported, although losses in the few outbreaks were significant. *Streptococcus* spp. are responsible for disease in many cultured fish worldwide, including rainbow trout, eels, channel catfish, tilapia, yellowtail and striped bass. The disease typically takes the form of septicaemia, and clinically fish may exhibit exophthalmia ('pop-eye') (**Fig. 80**) or fluid in the abdominal cavity and intestine (ascites). Transmission of *Streptococcus* spp. is thought to be mainly by contact and is likely to be enhanced by injury to epithelium or by stressful environmental conditions. Natural epizootics have been recorded in populations of wild fish. Silver perch reared in RAS were infected following fluctuating water quality (18 to 11°C over 12 h); however, other factors may have contributed to the epizootic.

Pathogen

Gram-positive, ovoid to elongate cocci; immotile; single or paired but rarely form chains in infected fish; β-haemolytic; many species can grow anaerobically, in a wide temperature range (10–45°C).

Signs

- Chronic fish mortality/morbidity
- Fish darkly pigmented
- Exophthalmia and abdominal distension
- Lethargy; fish near the surface and at pond edges
- Loss of appetite and nervous behaviour
- Diffuse haemorrhages around operculum, base of fins and skin, and inside of abdominal cavity (**Fig. 81**)
- Survivors may develop spinal deformity (scoliosis)

Diagnosis

Bacterial isolation from a variety of organs (spleen, liver, brain, eye and kidney); positive, bacterial culture produces dull, yellowish/grey, slightly raised and rounded, 1 to 2 mm colonies at 48 hours on blood agar media.

Treatment

Tanks:

- selection of antibiotic should be based on laboratory sensitivity testing as *S. iniae* has variable sensitivity. Whilst awaiting culture results fish can be started on

Figure 80
Silver perch infected by *Streptococcosis iniae*; note exophthalmia.
Source: Matt Landos

Figure 81
Haemorrhaging from the vent can sometimes indicate systemic bacterial infections.
Source: Phil Read

oxytetracycline, 20 mg/L active ingredient, 7 days at 20–30°C or 10 days at <20°C; maintain low light levels, good aeration; water exchange to 'dilute' bacterial load and re-treat.

Ponds:

- oral administration via feed: oxytetracycline, 75 mg/kg fish for 10 days.

Prevention

Avoid stress due to poor water quality, over-crowding, over-feeding, feeding old feed or unnecessary handling during critical periods; infected fish or carcasses should be removed; dry and lime ponds regularly; disinfect tanks, floors and equipment, sodium hypochlorite (500 mg/L for 2 mins), benzalkonium chloride (1,000 mg/L for 20 mins).

MYCOBACTERIOSIS

Mycobacteriosis is a chronic bacterial disease that can be persistent in recirculating systems such as those used for barramundi, Murray cod and aquarium fish (**Figs. 82** and **83**). The disease has been recorded in over 150 species of marine and freshwater fish worldwide. The common strains most frequently isolated include *Mycobacterium marinum*, *M. chelonae* and *M. fortuitum* although other environmental strains have been isolated from infected fish; all have a worldwide distribution. Mycobacteriosis has been recorded in pond-reared silver perch; however, the disease isn't a significant threat to the industry at present. Large silver perch (500 g) with mycobacteriosis exhibited typical white nodules (granulomas) on the viscera, particularly the spleen; approximately 20% mortality was recorded over a 2 month period. The infection propagated in the pond with prevalence increasing from approximately 20% to 60% over 8 weeks. *Mycobacterium* spp. can infect humans (zoonotic); however, the risk is low; gloves should be worn when handling fish suspected of having any bacterial diseases. Cuts, spike wounds and abrasions should be rapidly cleaned and treated with a topical disinfectant to minimise the risk of infection.

Pathogen

Straight or slightly curved, immotile rods, 0.4 × 1.0 to 4.0 µm long; gram positive, acid- fast; often staining unevenly; are slow to grow (up to 10 weeks required) and can be difficult to culture.

Figure 82
Ulceration of skin on a barramundi (*Lates calcarifer*) caused by mycobacteriosis.
Source: Phil Read

Figure 83
Barramundi (*Lates calcarifer*) with mycobacteriosis; note fin erosion.
Source: Phil Read

Signs

- Chronic fish mortality/morbidity
- Irregular swimming, fish at pond edges and surface
- Loss of appetite
- Emaciation and poor growth
- Abnormal dark colour of skin
- Shallow to deep ulcers
- Fin erosion
- Granulomas in viscera (kidney, spleen, heart), 1 to 4 mm diameter (**Fig. 84**)
- Exophthalmia and abdominal swelling (ascites)

Diagnosis

Laboratory culture of bacterium; diagnosed by acid-fast and gram staining (Ziehl-Nielson stained) of smears; histological sections of tissue granulomas (**Fig. 85**); microscopic examination, 200×, of wet, squashed mounts of kidney and spleen tissue for granulomas; the latter may include peripheral, lighter coloured, inflammatory cells.

Treatment

Once established, can be difficult to control or eradicate.

Tanks:

- remove infected fish; clean contaminated tanks, pipes and equipment; disinfect equipment using sodium hypochlorite (10,000 mg/L chlorine reported level required to kill mycobacteria).

Ponds:

- remove infected batch of fish; dry, de-silt and lime pond.

Prevention

Mycobacterium spp. can survive long periods in the environment and many strains are natural soil organisms; transmission probably via the shedding of bacteria from infected skin ulcers and 'bullying' of infected fish; dry ponds between crops; lime ponds with CaO or $Ca(OH)_2$; disinfect tanks and equipment; utilise UV and/or ozone units; remove infected and dead fish; maintain good water quality and husbandry practices; minimise damage to fish to prevent superficial infections.

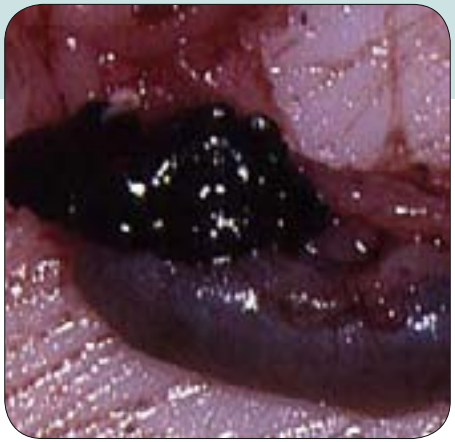

Figure 84
Silver perch spleen; note irregular margins of organ due to mycobacterial granulomas.
Source: Matt Landos

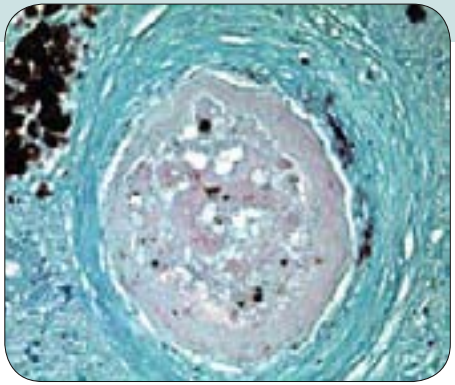

Figure 85
Histological section of a granuloma caused by mycobacteria in the kidney of an American bluefish.
Source: www.fishdisease.net

AEROMONAD DERMATITIS (MOTILE AEROMONAD INFECTION, MAI)

Aeromonad infections are one of the most common bacterial diseases of freshwater fish. Many species are susceptible, including silver perch. Aeromonad bacteria are ubiquitous and found in most freshwater ponds, rivers and bottom mud, utilising organic material as a nutrient source. The main species infecting fish are *Aeromonas hydrophila*, *A. sobria*, and *A. caviae*. The disease in silver perch is observed post-harvest during late spring and summer (causing 'summer spots') particularly when stocking densities in purging systems are increased. Clinical signs begin as de-pigmentation of areas of the epidermis on the caudal peduncle, top of the head, flanks and base of the pectoral fins (**Fig. 86**). If left untreated, 'spots' can develop into more advanced, red, necrotic skin lesions. Mild lesions can progress rapidly, with significant scale loss and haemorrhaging around the affected area from stress associated with live transportation. The disease has not caused significant mortalities in silver perch, but can render fish unsightly and unmarketable if sold whole or live.

Pathogen

Aeromonas hydrophila and *A. sobria*; gram-negative, short, motile rod; 0.8 to 0.9 µm × 1.5 to 3.5 µm; motile, singular polar flagellum.

Signs

- Loss of appetite
- Lethargy
- Loss of equilibrium
- Superficial, de-pigmented areas on flanks, fins, head or abdomen (early)
- Ulcerated lesions, margins whitish or haemorrhaging (advanced)
- Lesions may have secondary fungal infection
- Exophthalmia, opaqueness of eyes
- Distended abdomen, clear fluids; haemorrhagic, swollen intestine/vent

Diagnosis

Laboratory necropsy of diseased fish; bacterial culture, isolation and identification with accompanying histopathology examination.

Treatment

Tanks:

- treatment should be based on the laboratory antibiotic sensitivity results as this bacterium does not display a regular pattern of sensitivity (antibiotic-resistance is common).

Figure 86
Early MAI infection ('summer spots') caused by periods of low DO during harvest.
Source: Phil Read

- oxytetracycline 20 mg/L active ingredient, 7 days, continuous bath; maintain low light levels, good aeration; water exchange to 'dilute' bacterial load and re-treat;
- salt (NaCl) 2 g/L prolonged immersion or 10 g/L hourly baths daily.
- returning fish to the pond will often result in spontaneous resolution of lesions – this should not be done when water temperatures are under 18°C as super-infection of *Saprolegnia* can complicate the healing process.

Ponds:

- appropriate antibiotic; oral administration via feed; oxytetracycline, 75 mg/kg fish for 10 days; resistance to anti-microbials a problem.

Prevention

MAI is a stress-borne disease. When harvesting, avoid overcrowding, hypoxic conditions, high suspended solids and contact with bottom muds/organics; purge in good quality water; avoid high ammonia and overcrowding; regularly clean pipes and tanks; backwash filters; use UV/ozone and salt; water exchange.

EPITHELIOCYSTIS

Epitheliocystis is a widespread chronic bacterial disease affecting freshwater and marine fish. The causative agent is an intracellular bacterium, most probably Chlamydia-like organism. It has been recorded in silver perch fingerlings held in aquaria; however, the disease remains relatively insignificant under commercial pond conditions. Epitheliocystis is usually benign and non-pathogenic, but in high concentrations it has caused considerable mortalities in juvenile fish of several species in both fresh and marine environments. The organism affects mainly gills, causing gill hypertrophy and the formation of distinct transparent capsules. The

gills swell and lose their lamellae structure leading to impairment of the gills respiratory capacity; evidence of immunity developing in some species, the disease resolving without treatment.

Pathogen

Intracellular, non-motile, gram negative, coccoid bacterium; rod-shaped; visible under scanning electron microscopy; gill cysts (transparent to yellow capsules) round to oval shaped; 10–87 µm diameter; peripheral trilaminar membrane; up to 96% gill filaments affected in silver perch juveniles (**Figs. 87**, **88** and **89**).

Signs

- Moribund fish, mortalities
- Lethargy
- Swimming near the surface
- Distended opercular cover; rapid respiration
- Declining body condition
- Gaping mouth (mortalities)

Diagnosis

Initially, microscopic examination, 200× of gill tissue; can infect skin epithelial cells; hypertrophied cells in gill filament displaying round to oval shaped, transparent cysts; electron microscopy required to observe coccoid bodies within cysts.

Treatment

No known treatment, except disinfection and quarantine

Prevention

Little known about the organism; disease possibly transmitted by contamination of nets and other fish culture appliances; maintain cleanliness and disinfect gear and tanks regularly using chlorine baths, 1 mL/L 10–12% available free chlorine, for 60 mins. Maintain minimal stress in fish. Use of UV irradiation on intake water and isolation of infected batches of fish is recommended.

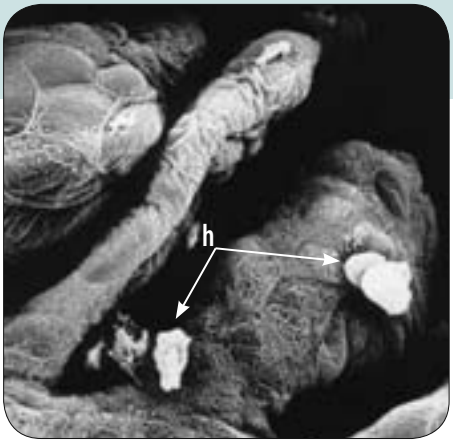

Figure 87
Electron micrograph showing gill filament cells of a fish with epitheliocystis; note swelling of the middle lamella: [**h**] hypertrophied [arrows] cell.
Source: Dr Barbara Nowak, Assoc Prof. School of Aquaculture, UTAS

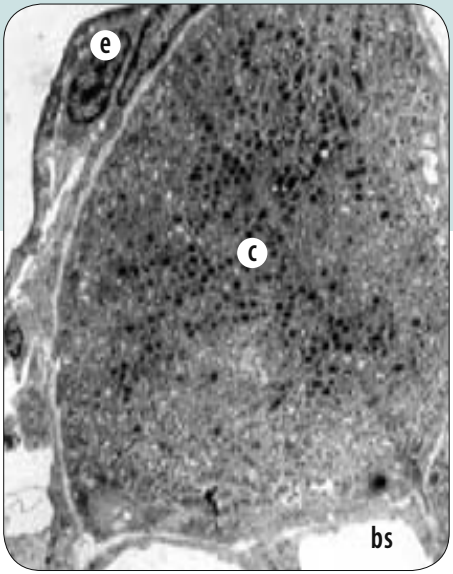

Figure 88
Electron micrograph of cyst containing pathogens: [**c**] cyst; [**e**] epithelial cells; [**bs**] blood space.
Source: Robert Tennent, Assoc. Lect., School of Medicine, UTAS

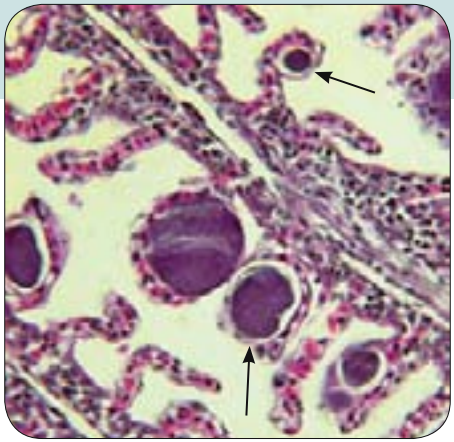

Figure 89
Histological section of a gill of a silver perch with epitheliocystis showing [arrows] basophilic inclusions.
Source: Dr Barbara Nowak, Assoc Prof. Dept of Aquaculture, UTAS

VIRAL DISEASES

Viruses are extremely small agents comprised of DNA or RNA surrounded by protein. Viruses must invade living cells in other organisms to replicate. The study of viral diseases in fish is relatively new, and a majority of fish viruses that have been reported are from economically-important species in which the diseases can cause high mortalities and severe economic losses. There are no known viral diseases affecting silver perch under culture conditions. One disease, epizootic haematopoietic necrosis, has been shown to affect the species under experimental conditions and is potentially a serious threat to silver perch culture.

EPIZOOTIC HAEMATOPOIETIC NECROSIS (EHN)

EHN is a highly infectious disease caused by the iridovirus epizootic haematopoietic necrosis virus EHNV. The virus is geographically limited to south-eastern Australia. Redfin perch (*Perca fluviatilis*) are very sensitive to the virus, with 100% mortalities a common outcome following exposure. It is known to also affect rainbow trout (*Oncorhynchus mykiss*) although mortalities in trout are usually less than 4%. Under experimental conditions in the laboratory, it has been shown that exposure to EHNV seriously affects the native fish silver perch, Macquarie perch (*Macquaria australasica*) and golden perch (*Macquaria ambigua*). Outbreaks in redfin and trout typically occur when water temperatures are above 12°C in spring/summer, often involving juveniles in both species. The disease is internationally notifiable and must be reported to disease surveillance agencies. It is known from studies on redfin perch, that EHNV is readily spread in water, with infected fish shedding viral particles via body fluids and from decomposing carcasses. The virus is highly resistant to environmental degradation, likely to remain in sediments for prolonged periods, possibly utilising insects or amphibians. Iridoviruses are known for their lack of host specificity; hence many fish species may be susceptible.

Pathogen

Virus a member of the Iridoviridae family and genus *Ranavirus*; polyhedral virus, 150–170 nm in diameter.

Signs (in redfin and trout)

- Mortalities
- Slow or rapid spiralling to the surface

- Erosion of fins; skin discolouration
- Weak swimming; 'head-standing' above the bottom
- Loss of appetite
- Cutaneous haemorrhages (terminal stages, redfin)
- Swelling of spleen and liver
- Peritoneal fluid; white foci of necrosis in liver

Diagnosis

Collection of fresh and fixed liver, kidney and spleen samples; fixing of tissue for virus isolation; cell culture; ELISA followed by histopathology, immuno-fluorescent staining, polymerase chain reaction (PCR), electron microscopy and/or other tests.

Treatment

No known treatment; strict quarantine to prevent spread; cleaning and disinfection (200 mg/L sodium hypochlorite for 2 h); proper carcass disposal.

Prevention

Exclusion of wild fish and ornamental fish, particularly redfin perch from ponds, drains and the farm's water source; reduction in stocking levels; maintain good water quality (particularly trout); regular cleaning and disinfection of gear; exclusion of piscivorous birds.

MISCELLANEOUS NON-INFECTIOUS DISEASES, DISORDERS AND CONDITIONS

HYPOXIA (LOW DO)

DO is the most limiting water quality variable in intensive aquaculture, and is closely linked to fish health. There have been significant losses of silver perch following acute hypoxia in ponds, as well as tanks and in purging and transport systems. Fish mortalities associated with low concentrations of DO have occurred following inadequate or failed aeration (particularly during summer), 'crashes' of algal blooms, and chemical treatment using formalin or copper sulfate in ponds. Generally, silver perch 'gasp' at the surface when DO decreases to 2 mg/L or less. Subjecting fish to prolonged chronic hypoxic conditions (<3 mg/L DO) will cause stress, reduce feeding and feed

assimilation, and result in poor growth. Chronic hypoxia can predispose fish to opportunistic infections. Even relatively short periods (minutes) of low DO can cause skin necrosis and secondary bacterial infections in subsequent days or weeks.

Signs

- Fish 'gasping' at water surface, particularly early morning
- Fish congregating at water inlets or near edges
- Largest fish dying first
- Acute mortality
- Dead fish having pale appearance and blotchy skin (**Figs. 90**, **91** and **92**)
- Rapid decline of feeding response (particularly tanks)
- Darkening of skin and inactivity (tanks)
- Open mouth and flared opercular

Diagnosis

Regular observation of fish and ponds. Routine daily monitoring of DO (early morning) using a high quality calibrated meter.

Treatment

Provide emergency water exchange (including from other ponds having higher DO levels) and supplemental aeration (2nd aerator and/or tractor power-take-off aerator)

Prevention

Provide adequate aeration (maintain DO >4 mg/L early morning) and emergency power back-up; avoid formalin or algacide treatments above 25°C water temperature and provide continuous 24 h aeration for 4–5 days following treatments; avoid over-crowding, excessive stocking densities and wasteful feeding; reduce feeding during periods of high water temperature (>28°C); provide oxygen (bottled) during harvest procedures and transport and in RAS.

Figure 90
Dead silver perch caused by low DO and high suspended solids during a drain harvest; note lack of pigmentation.
Source: Phil Read

Figure 91
Open mouth and flared operculum – signs of death by hypoxia.
Source: Phil Read

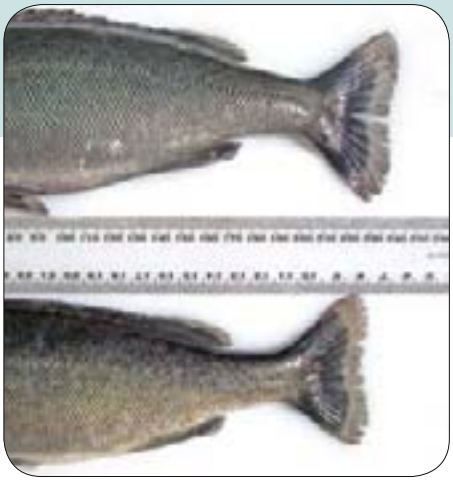

Figure 92
De-pigmentation of silver perch tails – a sign of hypoxia during harvest or transport.
Source: Phil Read

HYDROGEN SULPHIDE POISONING

Hydrogen sulphide (H_2S) is a gas that is produced during the anaerobic breakdown of organic matter by bacteria. It is commonly known as 'rotten egg gas' due to its strong, distinctive and pungent smell. There have been several cases of high mortalities of silver perch in ponds and tanks, where H_2S poisoning has been suspected. H_2S is toxic at low concentrations (e.g. levels should be <0.003 mg/L) and can cause rapid death with few, if any, diagnostic signs. Problems have occurred in poorly aerated ponds following the disturbance of bottom sediments during harvest procedures (**Fig. 93**). Some sources of bore water are naturally high in H_2S. The use of bore water that has been lying stagnant and/or the use of poorly flushed water delivery lines have also caused fish mortality in tanks. H_2S interferes with fish respiratory mechanisms causing hypoxia.

Signs

- Acute mortality following sediment disturbance or water exchange
- Erratic swimming around pond edges
- Rapid breathing, then listlessness and death
- Schooling at water inflows
- Redness in fins and tails

Diagnosis

Smell of 'rotten egg gas' may be indicative of a problem. Water sample collection (2 litres, sealed underwater) and testing by a qualified laboratory; sample can be refrigerated and stored following preservation with zinc acetate and sodium hydroxide (to >pH 9)

Treatment

Ponds:

- vigorous aeration of water using surface-spray type aerators; rapid water exchange; the addition of potassium permanganate to oxidise H_2S.

Tanks:

- use packed column degassers; lowering temperature and increasing pH will decrease H_2S toxicity.

Prevention

Maintain aerobic conditions using appropriate aerators and aeration regimes; use of aerators that provide both oxygen and create currents will reduce stagnant areas of water; remove objects (e.g. rocks, tyres, cages) from ponds that cause sediment build-up and poor water flow; regularly dry, de-silt and till ponds to oxidise sediments; flush water lines and position footvalves of pumps well above bottom sediments.

Figure 93
Black, anaerobic mud on the bottom of a pond.
Source: Stuart Rowland

GAS SUPERSATURATION (GAS BUBBLE DISEASE)

There have been several reports of losses due to gas supersaturation, but it does not represent a significant problem in the silver perch industry. There are no confirmed reports of gas bubble disease associated with nitrogen, but super-saturated levels of DO (160%) have been associated with the formation of gas emboli in the tails and gills of silver perch (**Fig. 94**). Large blooms of unicellular algae or extensive growth of macrophytes in ponds in summer may result in supersaturated levels of DO through excessive photosynthesis at high water temperatures and still weather conditions.

Signs

- Fish surfacing then diving (particularly in shallow water)
- Gas emboli in fins and/or eyes
- Oedema of secondary gill lamellae
- Exophthalmia ('pop-eye')
- Formation of bubbles on objects placed in water

Diagnosis

Macroscopic examination of fish; gas emboli in fins; ability to squeeze gas bubbles from the skin while the fish is held underwater; microscopic examination of gill, 100×; presence of gas emboli (**Fig. 95**).

Treatment

Ponds:

- aeration of pond for >1 h each day for several days using surface aerators (e.g. paddlewheels) will allow the gas to equilibrate with the atmosphere; water exchange.

Tanks:

- most likely nitrogen gas (not oxygen) caused by saturation of bore water or ingress of air under pressure via faulty pumps; excess gas can be stripped using a packed column degasser.

Prevention

Maintain daily aeration using paddlewheel or other surface splashing aerators; in smaller ponds, troublesome algal blooms in smaller ponds can be diluted using water exchange or use of an algacide (e.g. copper sulfate). Ensure pumps and associated plumbing have no leaks; use packed column degassers to strip excess gas; maintain fish transport tanks around 100% oxygen.

Figure 94
Gas emboli [arrows] in the tail of silver perch.
Source: Ian Charles

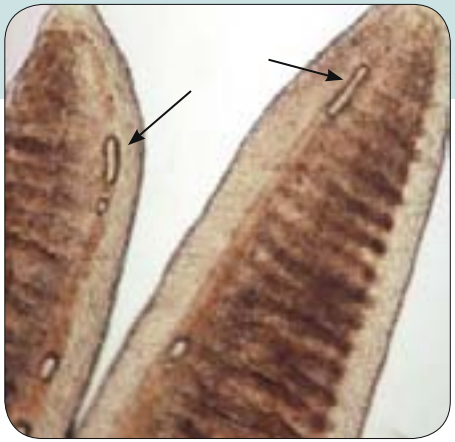

Figure 95
Gas emboli [arrows] in the gills of a silver perch (this level usually asymptomatic) (×100 mag.).
Source: Phil Read

NOXIOUS ALGAE

Noxious phytoplankton has been implicated in fish kills worldwide with the majority of problems occurring in marine systems. Noxious algae have not caused serious losses on silver perch farms, but some unexplained fish mortalities have been associated with algal blooms. In one instance, large silver perch suffered 100% mortality following the 'crash' of a large algal bloom containing the blue-green algae *Microcystis flos-aquae* and *Anabaena circinalis*; both species are known to be potentially toxic (**Fig. 96**). The crash was preceded by extreme pond water temperatures (>30°C) followed by heavy rain. Fish displayed erratic behaviour, schooling at inflowing water and those fish that were harvested recovered in tanks within 36–48 hours. Other water quality variables, except the very high temperatures were at acceptable levels throughout the event. It is possible that the algae released toxins after death and decay. However, the evidence linking fish morbidity/mortality and algal toxins under aquaculture conditions remains limited.

Signs

- Fish swimming around pond edges
- Erratic swimming (jumping, jerky movements, swimming in circles)
- Acute mortality
- Skin and fin redness

Diagnosis

Definitive diagnosis can only be done at a toxicology laboratory; fish need to be transported frozen and a water sample (2 litres) should be collected for algae identification (examination within 24 hrs or following preservation in Lugol's iodine solution and refrigeration); elimination of another aetiology (parasites, bacterial, viral and water quality) necessary to support diagnosis of algae toxicosis.

Treatment

Provide water exchange and 24 h aeration immediately; undertake emergency harvest when losses are thought to be imminent.

Prevention

Algacides can reduce noxious algae in ponds, but care must be taken to avoid oxygen depletion; maintain good water quality; reduce feeding during high water temperatures and heavy blooms of blue-green algae (**Fig. 97**); dry and clean ponds on a regular basis (every 2 to 3 years).

Figure 96
Suspected algae poisoning of silver perch; haemorrhaging around the mouth, pelvic and pectoral fins.
Source: Phil Read

Figure 97
Thick, blue-green algae scum on the surface of a silver perch pond.
Source: Phil Read

'CLOUDY' EYES AND RED TAILS

Cloudy eyes and haemorrhaging of the fins (and skin) are usually the result of poor husbandry techniques (**Figs. 98** and **99**). The problem often manifests in silver perch in tank-based quarantine and purging systems following harvesting or confinement in transport containers. The problem is more prevalent at warmer water temperatures (>20°C) and when fish are confined in waters having high organic load. The aetiology of the disease is unknown; the cause is most likely a combination of abrasive, rough handling, crowding in nets and harvest bins, and poor water quality (low DO; high solids, movement of fish between waters having significant differences in pH) associated with excessive stocking densities and stress. Transporting fish, even for short periods, in small vessels such as buckets or bins and the over loading of fish in hand nets has caused eye damage. The problem is exacerbated when using coarse netting or makeshift nets using plastics or other similar materials. Silver perch will recover in 3–5 days if held in sanitary conditions (clean water; high DO), using salt and at normal stocking densities.

Signs

- Opaqueness of one or both eyes
- Haemorrhaging of fins particularly caudal fin
- Scale loss
- Redness of skin, particularly below the lateral line

Diagnosis

Presumptive diagnosis is based on observation of the clinical signs during or soon after handling procedures.

Treatment

Tanks:

- mild clinical signs: hold fish at stocking densities <30 kg/m^3; provide salt (NaCl) at 2 g/L indefinitely; flush and retreat salt every 24 hrs for first 2–3 days; maintain clean water and high aeration (DO >4 mg/L). More severe cases: as above with daily salt baths at 10 g/L for 1 hour; secondary bacterial infections may require antibiotic treatment but generally improvement in water quality, good husbandry and hygiene will help lesions resolve spontaneously.

Figure 98
Eye damage caused by poor handling.
Source: Phil Read

Figure 99
Haemorrhaging and damaged tail fin caused by handling stress and poor water quality.
Source: Phil Read

Prevention

Avoid harvesting in poor water quality and at high temperatures (>28°C); use anaesthetic prior to hand netting fish; provide bottled oxygen through a high quality ceramic airstone during post-harvest transportation from ponds; avoid over-crowding in harvest bags and transporters; avoid handling fish compromised by disease or nutritional disorders unless emergency harvest indicated; use knotless nets.

PHYSICAL ABNORMALITIES

The incidence of abnormalities has increased in the silver perch industry over the last 5 years, with the most common being curvature of the spine (scoliosis and lordosis), deformed heads called 'axeheads' or 'snub-nosed', deformities of the opercula bones, reduced or missing pelvic fins, and 'paintbrush' caudal fins (**Figs. 100**, **101**, **102**, **103**, **104** and **105**). Jaw deformities have also been reported on some farms. The causes of the abnormalities are uncertain, but most are probably a result

Figure 100
Poorly developed 'paintbrush' tail.
Source: Phil Read

Figure 102
Abnormality of the skull ('snub-nosed').
Source: Jeff Guy

Figure 101
Poorly developed operculum [arrow] permanently exposing the gill lamellae.
Source: Phil Read

Figure 103
A juvenile fish with a deformed ('axehead') skull.
Source: Phil Read

of inappropriate hatchery practices including inbreeding and the use of poor husbandry during egg incubation, hatching and larvae rearing stages. During the hatchery phase, broodfish, eggs and larvae should be handled with great care to minimise physical damage, and maintain the highest quality water and optimum nutrition; water should be well filtered and oxygenated, and be free of pathogens and any heavy metals. Eggs and larvae should not be overstocked and any artificial diets used for post-larvae and juveniles should contain the appropriate ingredients, particularly vitamins and minerals.

Signs

- Spinal curvature
- Deformed or missing opercula
- Deformed fins
- Jaw and head anomalies

Diagnosis

Many possible causes for developmental anomalies; determining genetic link often accomplished by ruling out other possible causes.

Prevention

Use recommended hatchery and larval rearing techniques; follow recommended breeding programs; do not use broodfish suspected of being closely related or with unknown origin (e.g. caught in farm dams); replace broodfish suspected of carrying deleterious genes; use genetic strains known to be suitable for intensive culture; tag all broodfish and keep records of fish performance (weight, breeding performance, matings, etc); use high quality artificial feeds. Cull all abnormal fry prior to stocking.

ABDOMINAL SWELLING ('BLOAT')

There have been several reports of silver perch affected with gases in the abdominal cavity. Fish with this syndrome have a greatly distended abdomen, float inverted on the pond surface and are unable to feed or function normally; most eventually die. The prevalence of the disease within the pond, in the few-recorded outbreaks has been relatively low (<1% fish affected) and the cause remains unknown. Possible causes may include systemic bacterial infection, ingestion of contaminated feeds or ingestion of noxious algae. Some episodes have followed the recommencement of feeding after feeding was terminated for 1–3 days due to water quality issues or feed availability. Abdominal distension has been recorded in other species caused by swim bladder malformation and/or dysfunction; fish exhibiting abdominal swelling should be dissected to determine the site of the problem (organs or body cavity) and if the swelling is caused by fluids or gas.

Figure 104
A juvenile fish with a deformed ('axehead') skull.
Source: Phil Read

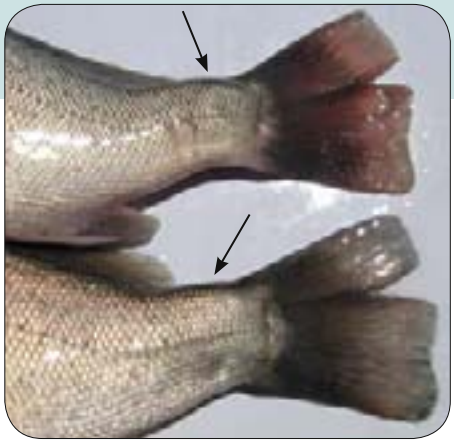

Figure 105
Fish showing curvature (lordosis) of the spine [arrows] around the caudal peduncle region.
Source: Phil Read

Signs

- Distended abdomen (**Fig. 106**)
- Organs or body cavity inflated with gases or fluids
- Fish inverted at pond surface, inability to swim normally

Diagnosis

Observation of typical abdominal distension and abnormal behaviour.

Treatment

Unknown.

Prevention

Aetiology unknown; ensure feed is fresh, mould-free and correctly stored (cool, low humidity); maintain good water quality with aeration.

NUTRITIONAL DISORDERS

Poor feeding practices such as overfeeding, underfeeding, feeding incorrect pellet sizes, the use of unsuitable or unpalatable diets and the feeding of contaminated or poor quality feed can have detrimental effects on fish health. Overfeeding fish can lead to water quality problems in particular low DO and high ammonia, and underfeeding or feeding the wrong sized pellets results in inadequate nutrition, poor performance and increased susceptibility to disease. Most diets are formulated for specific species or species groups, e.g. for optimal performance and health, carnivorous fish require higher levels of protein and energy than omnivorous fish. Adequate levels of proteins, and vitamins and minerals are essential for growth. The use of inappropriate diets can lead to health problems such as poor performance, excessive fat deposition, necrosis of the liver, parasitic diseases (see Ergasilids) or skeletal deformities (**Figs. 107**, **108** and **109**). Feeds high in fat can oxidise and become rancid affecting mineral and vitamin availability and shelf-life. Vitamin C and E deficiency and rancid food ingestion can cause immune depression, deformity, hepatic degeneration and ulceration. There have been reports of high mortalities of native fish in recirculating tank systems following the feeding of food contaminated with moulds and suspected aflatoxin. Severe disease has also been observed in association with feeding very old feed (>12 months since manufacture). Fish in ponds are at less risk of nutritional problems, particularly those farmed at lower stocking densities where natural feed may be available.

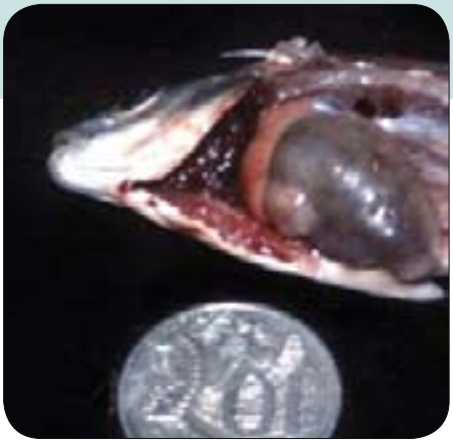

Figure 106
Silver perch fingerlings (≈ 70 g) with a gas-filled stomach.
Source: Unknown

Figure 107
Silver perch suffering poor nutrition and showing harvest stress.
Source: Phil Read

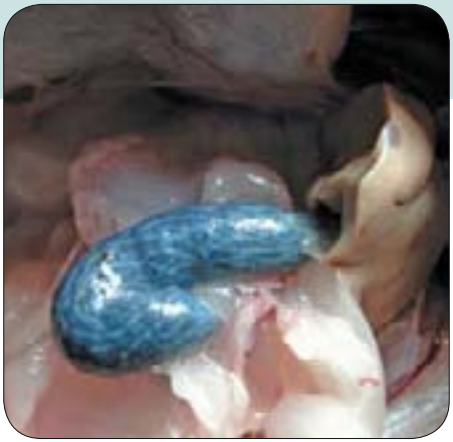

Figure 108
Starved silver perch with an enlarged gall bladder.
Source: Phil Read

Signs

- Poor growth, emaciation, starvation
- Skeletal abnormalities
- Acute mortality following the feeding of contaminated feed
- Fin erosion
- Fatty liver
- Poor response to normal handling procedures
- Concomitant disease such as bacterial or fungal infections

Diagnosis

Problems arising in species having well defined nutritional requirements are usually the result of poor feeding practices, or inappropriate storage or formulation of feeds. Suspected deficiencies in the diet require analyses by qualified nutritionists or biochemists. Collection of an accurate history of events, including feed manufacture dates, delivery and storage, and records of feeding, can provide information to help diagnose a nutritional problem.

Prevention

Use recommended feeding regimes and silver perch diets; handle and store feed in rooms having low humidity and temperatures <14°C; only order enough feed for approximately 3–4 months feeding. Ensure feed is recently manufactured.

AGGRESSION

Silver perch in small cages and tanks can display aggressive behaviour. Research has shown the problem more prevalent in smaller cages and tanks having moderate stocking densities (25–50 fish/m^3) rather than lower and higher densities. The cause of the behaviour is unknown, but is probably linked to the establishment of hierarchies involving aggressive and subordinate fish. Aggressive silver perch nip fins (mainly caudal) and flanks of submissive fish causing skin damage and necrosis, and fraying of the soft fin rays (**Fig. 110**). Submissive fish position themselves near the water surface on the sides of the tanks and cages; these fish do not feed, grow poorly and eventually become moribund and die. Secondary bacterial and fungal infections are

Figure 109
Feeds high in fat can cause excessively 'fatty' fish.
Source: Phil Read

Figure 110
Silver perch fingerling showing skin de-pigmentation and tail fin damage due to trauma and aggression.
Source: Phil Read

common. Silver perch held in purging systems for shorter periods (<14 days) and at higher stocking densities (>25 kg/m^3) behave normally.

Signs

- Moribund fish and/or mortality in tanks and cages
- Individual fish stationary near surface and corners of cages/tanks
- Skin necrosis, scale loss, blotchy appearance
- Sporadic commotion in tanks/cages

Diagnosis

Observation of aggressive behaviour between individuals particularly in fish held in small cages or tanks (e.g. 1m^3) at moderate, rather than light or heavy stocking densities; observation of clinical signs in the absence of other supportive disease diagnosis.

Treatment

Removal of affected fish to other tanks/cages; culling of aggressive individual fish; lowering or increasing stocking densities. Underfeeding of fish in tanks/cages can exacerbate the problem. Use salt and/or anti-microbial on fish having scale loss, fungal or bacterial infections.

Prevention

Avoid containment of fish in small tanks/cages over long periods (months); stock fish at low (<15 fish/m^3) or high densities (>100 fish/m^3, grow-out); hold broodfish in cages (up to 8 fish/m^3). Feed fish to satiation using semi-floating feeds and use feeding rings in cages.

DISEASE PROBLEMS IN PURGING SYSTEMS

Diseases are common in purging and tank systems in the silver perch industry. Purging (called 'conditioning' in the Silver Perch Quality Assurance Program), or the removal of off-flavours from fish, ensures a uniform, high quality product for the market. Purging requires harvested fish to be held in tanks for up to 7 days in clean, well-aerated water. Three management options are generally used: (i) a static system using periodic, 'batch,' water exchange; (ii) a recirculating system using tanks, filtration and UV units; (iii) a combination of the two. Generally, disease problems are more prevalent in recirculating systems under the following conditions: (i) where there is minimal or zero water exchange, particularly in the first 24 hours after harvesting; (ii) when poor husbandry practices have been used during the harvest; (iii) the use of poorly designed purging systems; (iv) where the system has not been 'conditioned' to assimilate sudden increases in ammonia and nitrite caused by the stocking of large numbers of harvested fish. Bacterial and fungal diseases are the most common. Heavy infestations of gill flukes are also common in purging systems and are difficult to eradicate due to the resistance of the egg stage to salt and other chemicals. It is important to recognise that when fingerlings are held in purging tanks, they can be readily infested. Treatment of fish to eliminate all parasites before stocking ponds and tanks is strongly recommended.

Signs

Clinical signs include chronic mortality (5–10 fish/day, starting 2–3 days post-harvest) frayed fins, blotchy skin, haemorrhaging, white patches on the head and caudal peduncle, dark skin, swimming into currents and lethargy.

Prevention of disease in purging systems

- Do not harvest diseased fish.
- Do not harvest fish from ponds with poor water quality.
- Do not feed fish for 2–3 days prior to harvest (prevents fouling of water and high ammonia post-harvest).
- Implement good harvest procedures (use oxygen, low stocking densities and anaesthetics in transport; measure water quality in pond, transport and purging system).
- Stock fish at rates < 30 kg/m^3 in clean, well-aerated water post-harvest.
- Exchange water in tanks constantly during harvest, then ~80% of water (in static systems) the day after harvest, and then periodically (>30%;

every 2nd to 3rd day) to remove organic matter, dilute bacterial loads and flush ammonia.

- Hold fish in continuous bath (>48 h) of 2–5 g/L salt to prevent fungal infections, reduce stress and prevent nitrite poisoning; re-salt following water exchange to ensure a level of 2–5 g/L is maintained.
- Measure water quality regularly (daily); DO, pH, salinity and ammonia.
- Do not feed fish in the purging system.
- Regularly clean/scour purging system including pipes, tanks and bio-filters.
- Leave water circulating permanently through bio-filtration units (desiccation of the system will kill nitrification bacteria).
- Periodically treat purging system with trichlorfon to lower fluke levels.

Holding some fish permanently in recirculating systems will help 'condition' or keep a load on the bio-filtration units; however, the filter's nitrification potential (the process of changing ammonia to nitrite then nitrate), is severely compromised following the introduction of large biomasses of fish (and subsequently ammonia) and any other sudden alterations to water quality (e.g. adding salt). Nitrification is further compromised when the bio-filter's specific surface area (SSA) is undersized and is unable to assimilate the ammonia load, and/or the bio-filter has been contaminated with organic matter (mucus, scales, grass, and suspended solids) due to inadequate pre-biofilter filtration or water exchange.

AQUATIC PLANTS AND FISH HEALTH

The presence of large, aquatic plants (macrophytes) in aquaculture ponds is undesirable. Plant species including milfoil (*Myriophyllum* sp.), cumbungi (*Typha* sp.) (**Fig.111**), ribbonweed (*Vallisneria* sp.) and water thyme (*Hydrilla* spp.) (**Figs.112** and **113**) have been problematic in some silver perch ponds. While some plant species are restricted to shallow areas of ponds, submerged species such as ribbonweed and water thyme are capable of colonising the entire pond including deeper sections. Some species of duckweed (Lemnaceae) are capable of rapid propagation, budding from adult plants and covering entire surfaces of ponds in a few days. Macrophytes can have a significant effect on the maintenance of fish health and the treatment and control of disease in the following ways.

- Interfere with pond management and feeding.
- Impact on water quality (clear water, compete with phytoplankton for nutrients, cause abnormally high pH levels).
- Crowd the fish and enhance disease transfer, particularly ecto-parasites.
- Restrict water flow and therapeutic chemical distribution and concentrations.
- Interfere with chemical concentrations due to high organic load.
- Provide havens for fish and parasites which may remain untreated.
- Contribute to oxygen depletion and high TAN levels when they decompose.
- Contribute to water loss through evapotranspiration.
- Hinder or prevent sampling and harvesting.

Management

- Dry and de-silt ponds every 1–2 years.
- Harvest problem weeds as soon as they appear especially prior to their seeding.
- Increase phytoplankton turbidity through correct feeding regimes and/or application of fertiliser.
- Use a herbicide (contact Extension Service for advice).

Prevention

Avoid contamination from waters introduced from other locations (e.g. fish transportation water); dissuade aquatic birds; exclude farm stock such as cattle from ponds; use bore or well waters and prevent run-off into surface supplies and ponds; quarantine all fish coming onto the property.

Figure 111
The cumbungi plant colonising pond edges.
Source: Phil Read

Figure 112
Hydrilla sp. 'choking' a silver perch pond.
Source: Phil Read

Figure 113
Extensive growth of *Hydrilla* sp. and filamentous algae; note the water clarity.
Source: Phil Read

CHEMICAL USE AND LEGISLATION (JANUARY 2007)

Chemicals play an important role in aquaculture in the management of fish diseases, the improvement of water quality and the control of aquatic vegetation.

In Australia, the Australian Pesticides and Veterinary Medicines Authority (APVMA) regulates the supply of chemicals used in agriculture, including aquaculture. In NSW other relevant legislation relative to chemical use includes the *Stock Medicine Act 1989*, the *Pesticides Act 1999* and the *Food Act 2003*. Several agencies [e.g. NSW DPI, NSW Food Authority, and Food Standards Australia New Zealand (FSANZ)] work closely with the APVMA administering issues dealing with chemical evaluation and chemical residues in food.

Further information can be obtained at **www.apvma.gov.au**, **www.foodstandards.gov.au** and **www.dpi.nsw.gov.au**.

Fish farmers need to familiarise themselves with the rules and regulations of these Acts in order to use chemicals legally.

Few chemicals are registered in Australia for aquaculture use. To be approved for use in food animals a drug must generally undergo rigorous testing of its efficacy for treating specific diseases in each species at specific dosages and routes of administration. Data must be obtained on residue dynamics, safety for the operator and consumer, and effects on the environment. This can require years of experimental trials and high costs. At the end of the process registered drugs must be used only in accordance with the label to treat the species on the label at the directed dose rates.

The APVMA may allow the use of unregistered chemicals, or registered chemicals off-label, under a minor use permit (MUP). MUPs are a 'temporary' approval system for the use of chemicals when no alternative treatment is registered. The APVMA still carries out a risk assessment before issuing an MUP.

It is important to verify with the APVMA the applicability and validity of any MUP (this can be done using their web site) and/or to verify with NSW DPI under what conditions a chemical can be used. Some chemicals registered for use on other food-producing animals (e.g. chickens, sheep or cattle) may be able to be used off-label with written veterinary directions (prescription). Some chemicals used only for water treatment and with no residue concerns are exempt from registration (see some specific examples in **Table 1**).

Table 1. Status of chemicals in the aquaculture industry (Nov 2005)

REGISTERED	MINOR USE PERMITS[a]	EXEMPT
AQUI-S (Isoeugenol)	Formalin	Zeolite
Buserelin (GRH)	Benzocaine	Calcium carbonate
Chorulon (Chorionic Gonadotrophin)	Hormones (Ovaprim, OvaRH, LHRHa)	Calcium hydroxide
Anguillvac C – (Vibriosis C vaccine)	Oxytetracycline	Inorganic fertilisers
	Copper sulfate	Organic fertilisers
	Trichlorfon	Propionic acid
	Sodium chloride	
	Potassium permanganate	

[a]Check with the APVMA for expiry dates

Registered chemicals and MUPs must be used according to label instructions. Before using any chemical to treat aquatic animals confirm that it is approved for use with the APVMA or your NSW DPI advisor to avoid illegal use and possible residue contamination.

IMPORTANT INFORMATION REQUIRED TO PREVENT, DIAGNOSE, TREAT AND CONTROL DISEASE

To keep fish losses to a minimum and maintain fish health, specific design features, strategies and a degree of preparedness must be in place (**Table 2**).

Silver perch farms adopting these practices operate efficiently and have fewer stock losses and costs.

Table 2. Information to be recorded and essential design criteria to assist in the prevention, diagnosis, treatment and control of silver perch diseases.

INFORMATION/DESIGN		COMMENT
Known tank and pond volumes at specific water levels		• Application of accurate chemical concentrations; prevention of under dosing causing ineffective treatment and poor disease control; prevention of overdosing, dangerous to fish health; ability to stock eggs and fish at correct densities.
Chemicals stored on site		• Prompt disease treatment; white spot and chilodonellosis capable of rapid spread and high mortalities over 2–3 days.
Known fish size, numbers and biomass in each pond/tank		• Assists in disease diagnosis; recording history of disease event (prevalence, mortality rate). • Systemic disease control through the application of medicated feeds based on % body weight (biomass). • Optimum nutrition via accurate feeding schedules (rations, pellet size).
Superior water quality meters and kits (DO, TAN, pH, alkalinity, salinity, $N0_2$, NO_3, formalin, copper)		• Accurate records facilitate confident interpretations; integral process in disease diagnosis and/or elimination of other possible aetiological agents; diagnosis of water quality problems in tanks and purging systems; possession of two DO meters advisable. • Safe use of $CuSO_4$ dependent upon alkalinity levels. • Need to accurately determine and maintain concentrations of formalin or copper during treatment.

INFORMATION/DESIGN		COMMENT
Aeration		• Oxygen is essential for fish health particularly during periods of disease and poor water quality. • Ability to provide additional aeration during episodes of low DO following chemical use (formalin, $CuSO_4$), phytoplankton die-offs, hot weather or overcast conditions. • Failsafe measures are mandatory – back-up generators; additional paddles; PTO aeration
Bottled oxygen, lines and airstones		• Emergency aeration in tanks. • Maintenance of fish health during harvest and transportation; prevention of bacterial disease due to periods of low DO.
Microscope, sampling and dissection equipment		• Essential for disease diagnosis, treatment and control.
Regular water quality and disease monitoring; daily observation of tanks and ponds		• Early detection/treatment and prevention of major losses. • Observation of behavioural irregularities or changes in water colour. • Discouragement of bird predation; stress can lead to poor growth and disease.
Ability to provide rapid water exchange in tanks/ponds; daily flushing of bores, bio-filters and water lines		• Essential during emergencies to secure stock particularly during episodes of poor water quality. • Rapid dilution of chemicals (over-dosing). • Flushing of high levels of ammonia/nitrite and H_2S. • Prevention of organics in bio-filters and poor nitrification or 'collapse' of the bio-filter.
Water source – bore or screened surface water		• Bore water is pathogen-free. • Surface water (rivers, dams) filtered to <500 µm will prevent introduction of trash fish potentially harbouring disease.

INFORMATION/DESIGN		COMMENT
Power generation; weekly testing of generator		• Automated during 'mains' failure; prevents major losses due to poor water quality in tanks and ponds.
Significant tank capacity and tank numbers		• To provide the capacity to hold/treat fish following emergency harvests • Essential for quarantine and prevention of disease introduction to the farm and between ponds on the farm.
Veterinarian, Association and Extension contacts		• Information and permits to obtain chemicals, investigate, diagnose and treat disease; avenues for laboratory sample submissions; additional testing of water quality.
Knowledge sharing with staff/family		• Farm operational procedures, water reticulation plan and stock inventory known by more than one person. • Written and readily accessible health strategy plan outlining standard operating procedures, volumes, treatment procedures, emergency power configuration.
Knowledge of OH&S and zoonoses		• Provide training and appropriate equipment for health and safety of staff

CALCULATIONS, TREATMENTS AND DOSE RATES

Treatments can be of great value when used properly but when incorrectly applied, can cause large losses of fish. In order to properly apply chemicals to the water or feed, it is important to accurately determine the dosage and the best application methods.

VOLUME CONVERSIONS
1,000 mL (millilitres) = 1 L (litre)
1,000 L = 1 m^3 (cubic metre)
1,000,000 L = 1 ML (megalitre)
Volume of water (round tank) = 3.142 × r^2 (radius) × d (depth)
Volume of water (square tank) = l (length) × w (width) × d (depth)
Volume of pond = length (m) × width (m) × mean depth (m)
WEIGHT CONVERSIONS
1,000 µg (micrograms) = 1 mg (milligram)
1,000 mg = 1 g (gram) = 1 mL (millilitre) of water
1,000 g = 1 kg (kilogram) = 1 L (litre) of water
1,000 kg = 1 t (tonne) = 1 m^3 of water
LENGTH CONVERSIONS
1,000 µm (microns) = 1 mm (millimetre)
10 mm = 1 cm (centimetre)
1,000 mm = 100 cm = 1 m (metre)
AREA CONVERSIONS
1 ha (hectare) = 10,000 m^2 = 2.5 acres
1 acre = 0.4 ha = 4,000 m^2 (e.g. a pond measuring 50 × 80 metres)

CONCENTRATIONS	
ppm = part per million, **ppt** = part per thousand	
LIQUID CHEMICAL FORMS	**SOLID CHEMICAL FORMS**
1 ppm = 1 L (litre) in 1,000,000 L = 1 mL in 1,000 L **20 ppm** = 20 L in 1,000,000 L = 20 mL in 1,000 L **1 ppt** = 1 mL in 1 L	**1 ppm** = 1 mg in 1 L = 1 g in 1,000 L **20 ppm** = 20 mg in 1,000 L **1 ppt** = 20 g in 1 L **5 ppt** = 5 kg in 1,000 L
ACTIVE INGREDIENT (a.i.)	
Treatment rates unless otherwise stated are based on the active ingredient (a.i.) only. **Dose** (g or mL) = Volume (m^3) × Concentration of Therapy (ppm) × 100 ÷ % a.i.	
EXAMPLE CALCULATION	
A pond (3.2 ML) is to be treated at 0.5 ppm with a liquid chemical having 40% a.i. Dose (mL) = 3,200 m^3 × 0.5 × 100 ÷ 40 = 4,000 mL = 4 L	

FORMALIN

Formaldehyde solution, also known as formalin, contains 37% to 40% dissolved formaldehyde gas; however, for application it is used as if it was a 100% pure compound. Formalin is depleted rapidly in ponds and the rate can vary according to factors such as temperature, aeration, fish biomass and organic matter. In general, formalin reaches ineffective concentrations within 48 hours and most of the formalin is lost within 4 days of application to ponds. To prevent under or over-dosing and to maintain effective concentrations, a formaldehyde test kit should be used to accurately monitor formalin concentrations.

Precautionary measures when using formalin.

- **Oxygen**

Each 5 mg/L of formalin added to a pond can remove 1 mg/L of DO from the water at temperatures over about 20°C. Formalin kills some algae and micro-organisms, which then decay using oxygen.

- **Aeration**

Ponds and tanks must be continually aerated during formalin treatment. Oxygen levels must be regularly monitored. Additional aeration may be needed during formalin treatment.

- **Paraformaldehyde**

Formalin should be stored in the dark and above 4°C to inhibit paraformaldehyde formation, a toxic white precipitate; if present, decant and use clear liquid only.

- **Water quality**

Formalin is more toxic in soft, acid water and at high temperatures. Formalin undergoes a series of chemical reactions and breaks down quickly in pond water (within 4 days).

- **Fish**

Some fish are more sensitive to formalin than others.

- **Human health**

Formalin is volatile and can cause respiratory problems. Handle and use in well-ventilated areas. Protective clothing should be worn during handling.

MEDICATED FEED

Many bacterial diseases of fish can be successfully treated with medicated feed that contains an antibiotic. Accurate diagnosis of the causative agent of the disease will ensure the correct antibiotic is selected.

EXAMPLE CALCULATION – ORAL MEDICATION

A pond of 2,000 fish, mean weight 350 g (700 kg of fish) is to be fed medicated diet. The commercial premix contains 5% of the active ingredient (antibiotic). The fish are feeding at 1% of their body weight per day. The recommended treatment level for the antibiotic is 75 mg active ingredient / kg of fish for 10 days. How much antibiotic must be added to the feed to provide the correct dosage?

1. At 1% body weight feed rate, 700 kg fish require 7 kg feed / day
   = 70 kg in 10 days.
2. Antibiotic treatment rate is 75 mg/kg fish per day
   = 75 mg × 700 kg × 10 days
   = 525,000 mg
   = 525 g / 70 kg feed
3. The premix has 5% active ingredient therefore 525 g ÷ 0.05
   = 10.5 kg of antibiotic premix is to be added to the feed.
4. The daily ration of treated feed = 7 kg / day

In practice the feeding rate when using medicated feed will often be 0.5% or 1% of the body weight per day because sick fish generally have reduced appetites. Any additional feed that is given should be non-medicated.

Important points when using medicated feed.

- **Diagnosis**

To ensure the correct antibiotic is used in the feed, the causative agent of the disease needs identification and antibiotic sensitivity testing at a veterinary diagnostic laboratory.

- **Treatment**

Treatments should always be to the maximum recommended dose and should be fed for the total number of days recommended, even if the fish appear to have recovered. Appropriate dosing and duration of treatment help prevent the development of antibiotic resistance. Overdosing and under dosing are equally unproductive and are likely to result in worse outcomes.

- **Fasting**

Withholding food for 12 to 24 hours may increase the acceptance of medicated feed and is useful to assist recovery of fish when being challenged by a disease.

- **Storage**

Medicated feed should be stored in a cool, dry place. Medicated feed should be made up as fresh as possible – ideally daily. Storage beyond 10 days is not appropriate as the chemicals breakdown and become inactivated.

- **Target diseases**

Antibiotic medicated feeds do not control parasites, fungus or viruses.

- **Human safety**

Always wear protective clothing including gloves and a face mask when handling antibiotic premixes and medicated feeds.

- **Gelatine**

Use gelatine to bind the antibiotic to the feed. This assists in avoiding loss of antibiotic into the water, prior to being ingested by the fish.

HOMEMADE MEDICATED FEEDS (Piper et al. 1982)

Gelatine

Makes 45 kg of feed for large fish.

Slowly dissolve 125 g of gelatine in 2.8 litres of hot water.

Stir the antibiotic into the gelatine until dissolved.

Slowly add the drug-gelatine mixture to the pellets stirring by hand or using a cement mixer.

To avoid pellet breakage, stir gently and only long enough to ensure an even drug coating.

Soy oil

Use a wt:wt ratio of 2 to 3 parts oil: 100 parts feed.

Heat soybean oil to ~ 40°C.

Quickly mix the drug evenly into the warm oil.

Pour or spray the drug-oil mixture over the pellets.

ALKALINITY AND COPPER SULFATE ($CuSO_4$)

Copper (as copper sulfate) is applied to ponds to control algae, including filamentous algae, or to control diseases caused by protozoans such as *Ichthyophthirius multifiliis* and *Chilodonella hexasticha*. It is also reported as having some bactericidal value. Copper sulfate contains approximately 24.5% copper and calculations should be based on this figure when determining the dosage (see '*Active ingredient*' above). Not all fish species are equally tolerant to copper, e.g. salmonids are highly sensitive, and concentrations of 0.25 mg/l and higher are toxic to silver perch. The toxic forms of copper are cupric ion and copper hydroxide complexes; cupric ion is most abundant at low pH, while the copper hydroxide complexes (CuOH and $Cu(OH)_2$ predominate at pH's above neutral.

Alkalinity is regarded as the main modifier of copper toxicity; however, research has shown specific levels of calcium hardness and salinity can reduce copper toxicity in some species when alkalinity remained below recommended levels. The increased bases, associated with higher alkalinity reduce toxicity by combining chemically with the copper to form less toxic compounds. In waters of low alkalinity, special care must be taken when applying copper sulfate because of the higher presence of the toxic forms. Toxic levels of copper can cause gill damage and disrupt the fish's ion regulation; clinical signs of copper toxicity in silver perch include inappetence, fish swimming at pond edges, and dark skin colour. Concentrations of copper of 0.25 mg/L and above are toxic to silver perch even at high alkalinities of 80–100 mg/L. The recommended treatment regime when using copper to treat diseases in silver perch is 0.2 mg/L (active ingredient) initially, followed by daily treatments to maintain the level between 0.1–0.2 mg/L until the disease is controlled. Alkalinity should be accurately measured prior to copper sulfate application and should be >50 mg/L as $CaCO_3$; alkalinity can be increased with the addition of lime (as limestone, $CaCO_3$; dolomite,

$CaMg(CO_3)_2$) or sodium bicarbonate ($NaHCO_3$); calcium hardness and salinity can be increased using gypsum. The copper sulfate should be thoroughly dissolved before applying to ponds (or tanks) and care should be taken to disperse the copper over the entire pond area to avoid the creation of 'hot spots'. A reliable instrument should be used daily to measure the ponds/tank copper concentration. Aeration should be maintained during the duration of the treatment and DO measured daily.

Some precautionary measures should be observed when using copper sulfate. Copper treatments in ponds containing algae, may cause oxygen concentrations to drop to dangerously low levels following the death of the algae; additional and continuous aeration should be provided during the treatment. Contact of copper compounds with skin and eyes may cause irritation; agricultural stock (sheep, cattle etc) should not drink water treated with copper and containers holding copper, particularly steel, should be well rinsed to prevent corrosion. Copper is also toxic to most pond zooplankton and should not be used in ponds where larvae and small fingerlings are reliant on plankton as a food source.

POTASSIUM PERMANGANATE ($KMnO_4$)

Potassium permanganate ($KMnO_4$) also known as 'permanganate of potash' and 'Condy's crystals', is a chemical compound that can control some fish diseases, including those caused by protozoans and monogeneans. It is a crystalline powder that is easily dissolved in water. Its application to tanks following harvest of fish has proven beneficial in controlling some bacterial diseases in silver perch. Potassium permanganate can also be used to remove iron and hydrogen sulphide from water.

The use of potassium permanganate requires careful observation and effort to achieve treatment regimes which are both safe and effective in ponds and tanks. The chemical may be rapidly lost from solution due to the oxidisation of organic matter (e.g. algae, detritus, and dissolved organic compounds). Effective treatments require a prolonged immersion in at least 2 mg/L of active chemical; higher concentrations are required if the organic content of the culture water is high. Once applied, the permanganate ion is reduced to manganese dioxide (MnO_2) rendering the chemical inactive; the water colour changing from a pink hue to a light tan. The reduction rate can vary due to differences in organic content and water quality in ponds. To determine whether a re-treatment is required, water colour should be inspected regularly by splashing water into the air or observing the water in a clear glass container. Water which has changed colour within 8 hours should be re-treated using increments of 2 mg/L until the light pink colour is restored. It is recommended not more than 6 to 8 mg/L total potassium permanganate should be added to a pond. To determine the amount of potassium permanganate needed, a simple 15 minute demand test can be done. The chemical is added to five glass containers (or clear plastic bags) each having 1 litre of culture water to achieve concentrations of 1, 2, 3, 4 and 6 mg/L, and observed for 15 minutes. The lowest concentration in which the pink hue remains is considered the endpoint. This concentration is multiplied by a factor of 2.5 to calculate the concentration (mg/L) needed for a potassium permanganate treatment.

It is advisable to adopt a precautionary approach when applying potassium permanganate (and other chemicals) to recirculating aquaculture systems (RAS).

Potassium permanganate can have inhibitory effects on nitrification, but the effects may vary with the design and operation of each RAS.

Potassium permanganate may be toxic to fish in water having high pH due to deposition of insoluble manganese dioxide on the gills. Potassium permanganate should not be mixed with formalin and care should be taken during its handling (protective clothing, dust mask, gloves). The chemical should be stored in a tightly closed container, in a cool dry area, out of direct sunlight and away from flammable and combustible materials including strong acids.

SALT (NaCl)

Salt or sodium chloride (NaCl) is used to treat infestations of some ectoparasites, relieve stress during handling and transport, and prevent fungal infections of fish in tanks. It can also be used to prevent nitrite toxicity in RAS. Salt should not be used in ponds because of the large quantities required and adverse environmental effects.

Salt can be added directly to tanks; it then dissolves slowly over the following 15–30 mins. Alternatively, the salt can be dissolved in a separate container and added slowly to the tank. Noniodized salt should be used. Water quality parameters (DO, TAN, salinity and nitrite) should be monitored during salt treatments and tanks should be flushed and re-treated if water quality deteriorates. Salt treatments can cause foam to form at the surface of tanks; however, this is usually of no concern and can easily be decanted.

Concentrations of 2–5 g/L (2–5 ppt; 0.2–0.5%; 2,000–5,000 mg/L) are recommended to relieve osmotic stress and prevent fungal infections following handling and transporting. Infestations of ectoparasites are treated with short-term (1 h) or long-term, static baths (i.e. no flow). For example, chilodonellosis is effectively treated with 10 g/L for 1 h (the treatment should be repeated after 24 h), whereas control of ichthyophthiriosis (white spot) requires a bath of 2 g/L until the pathogen is eradicated (may be up to 4 weeks at low water temperatures). Some larger parasites (e.g. gill flukes) may require higher dosages for effective control (e.g. 15 g/L, 1 h bath). Nitrite toxicity ('brown blood' disease) in RAS and purging systems can be controlled by adding salt. Generally 5 to 6 parts of chloride will protect fish from 1 part nitrite; therefore 2 g/L should prevent nitrite toxicity problems in silver perch. Rapid addition of salt to RAS can decrease biofilter efficiency; salt should be added in small increments (<2 g/L every few days) to allow the biofilter to adjust. Alternatively, tanks can be isolated from the biofilter, treated at recommended salt rates and then flushed prior to re-joining normal water recirculation.

Salt is usually purchased in 25 kg bags and should be stored in a dry area with low humidity and away from metal surfaces. Recently-packed salt is less likely to have 'sweated' which often causes the salt to cake rendering it more difficult to handle and dissolve. Farms should have a specific effluent pond to store saline water to prevent a build up of salt levels in the farm's water system.

TRICHLORFON

Trichlorfon is an organophosphate insecticide that can be used to treat infestations of monogenean and crustacean ectoparasites such as gill flukes and anchor worms. The chemical acts by interfering with cholinesterase, an essential nervous system enzyme of the parasites. When added to water, trichlorfon degrades to the more toxic dichlorvos; the chemical reaction is

accelerated by factors such as light, aeration, and high pH and temperature. Ponds with high pH (>8.5) and temperatures in the afternoon should be treated in the early morning so as to maintain an effective concentration for as long as possible.

Trichlorfon can be highly toxic to fish, and adversely affect the fish's immune system. Pond and tank water volumes need to be precisely calculated in order to apply the appropriate concentration. To avoid 'hotspots', the dose should be mixed with water prior to application particularly when treating small tanks or cages. The major breakdown product, dichlorvos, is more toxic and can be persistent, degrading more slowly than trichlorfon; therefore some caution is required when using repeated trichlorfon treatments to avoid toxicity and/or resistance problems. Tanks should be well aerated and flushed between treatments. It is advisable to allow 2–3 weeks between treatments. Fish gasping, rolling or shaking indicate trichlorfon and/or dichlorvos toxicity.

Trichlorfon is also toxic to some crustaceans and zooplankton. Gill flukes may become resistant to trichlorfon with long-term use on a farm. A strategy of alternating trichlorfon and formalin to treat infestations of gill flukes may prevent the development of resistance and reduce adverse effects on silver perch and ponds.

Care must be taken when handling and storing trichlorfon. As with all organophosphates, trichlorfon is readily absorbed through the skin. Protective clothing including respirator, gloves and overalls must be worn when handling the chemical. Trichlorfon will also decompose in the presence of alkalis, and is incompatible with strong oxidizing agents. It should be kept well packed in a cool, dry place. Heat may cause the decomposition of trichlorfon and the release of dichlorvos and other highly toxic fumes. As with other organophosphate pesticides, it is advisable to stay upwind from trichlorfon treatment areas.

WITHDRAWAL PERIODS

It is the legal responsibility of the person prescribing the treatment and the farmer to ensure illegal residues are not present in food fish harvested for human consumption. The excretion of a drug can vary greatly with environmental conditions, especially temperature. Because of this, the term 'degree days' has been advocated for estimating the required withdrawal time. Degree-days are calculated by adding the mean daily water temperatures for the total number of days measured. Thus if the mean temperature were 25°C for the 20 days immediately after stopping the treatment, the degree days would be 500 (25×20). When specific withdrawal times are not given for a chemical, a good rule of thumb is 600-degree days. Always seek advice from a veterinarian regarding withdrawal periods.
Residue tests are available to ensure your withholding periods have been sufficient.

NOTIFIABLE FISH DISEASES

Within Australia, lists of notifiable or reportable fish diseases are defined by each state. The lists include all relevant *Office International des Epizooties* (OIE) listed notifiable diseases that represent Australia's international surveillance, monitoring, notification and reporting obligations. These diseases are considered important for social, economic, international trade and public health reasons.

In NSW declared diseases are listed in the *Fisheries Management (Aquaculture) Regulation 2007*.

In NSW it is a condition of the Aquaculture Permit that all significant disease outbreaks are reported to a Fisheries Officer. This will lead to an accurate diagnosis of the disease and the implementation of any control and/or containment measures that may be required. This information greatly assists in the understanding and management of diseases and helps protect other aquaculture industries.

Two of these diseases, Epizootic Ulcerative Syndrome (EUS) caused by the fungal pathogen *Aphanomyces invadans*, and Goldfish Ulcer Disease (GUD) caused by the bacterium *Aeromonas salmonicida nova* have been reported in silver perch.

The current NSW Notifiable Diseases list and other relevant information can be viewed at *www.legislation.nsw.gov.au* under the *Fisheries Management (Aquaculture) Regulation 2007*.

GLOSSARY

Acid fast
A laboratory staining technique using carbol-fuchsin (red dye), heat and alcohol wash

Aetiology
The science that deals with the cause of disease

Ascites
An effusion and accumulation of serous fluid in the abdominal cavity

Asymptomatic
Showing no symptoms; no visible signs of a disease condition

Basophilic
Applied to a cell, its components, or products that can be stained by a basic dye

Binary fission
Asexual reproduction where cells divide after which each daughter cell grow to the original form

Cilia
Hairlike locomotor organelles of ciliated protozoa

Ciliate
Protozoan bearing peripheral cilia

Caudal peduncle
The region of body to which the caudal fin is attached

Cephalothorax
The combined head and thorax region of crustaceans

Chronic
Occurring over a long period of time, with gradual or consistent mortality rate; also recurring

Cytoplasm
Part of cell outside of the nucleus and within the cell membrane

Distal
Away from the point of reference or attachment

Ecto-commensal
Organisms on the host's surface; living together with no harm to either

ELISA
The Enzyme-Linked ImmunoSorbent Assay is a biochemical technique used mainly in immunology to detect the presence of an antibody or an antigen in a sample.

Epithelium
The cellular covering of external and internal body surfaces

Epizootic
A disease affecting a population

Emaciation
Wasting of the body

Eutrophication
The enrichment of a body of water by the addition of nutrients

Exophthalmos
Abnormal protrusion of the globe of the eye from the orbit

Flagella
Whiplike organelle that is used for locomotion of some protozoans

Foci
The size or distribution of changes to a tissue as limited to a small area, pertaining to, or emanating from a focus

Gram negative (Gram positive)
Staining characteristics of bacteria dependent on the cell walls constituents

Granuloma
A chronic inflammatory response with nodular aggregations of macrophages (phagocytic cells)

Haemorrhage
The escape of blood from vessels

Histopathology
The study of microscopically visible changes in diseased tissue

Histozoic
Tissue invading; usually pertaining to parasites

Hyperaemia
An excess of blood in a body part

Hyperplasia
Abnormal increase in the number of normal cells in tissue or an organ

Hyperventilating
Rapid and abnormal increased respiratory activity

Hyper-osmotic
High concentration of salts/compounds in the body compared to the environment

Hypoxia
Deficiency of oxygen

Idiopathic
Occurring without known cause

Moribund
In a dying state

Morphology
The science of the form and structure of organisms

Mycotic
Pertaining to fungi

Necropsy
A medical examination of the fish after death involving dissection

Necrosis
The death of tissues or cells within a living body

Obligate
Characterised by the ability to survive only in a particular environment

Oedema
The excessive accumulation of fluids in tissue spaces

Operculum
Bony covering of the gill

Osmoregulatory
Control of ionic concentrations to facilitate normal cellular function

Oviparous
Egg laying, with hatching outside the mother's body

Peritoneal cavity
Abdominal cavity which contains various organs

Phagocyte
An inflammatory cell capable of ingesting bacteria, foreign particles and other cells

Piping
The act of fish gulping or gasping at the water surface

Poikilothermic
Having a body temperature that varies with the temperature of the environment

Prophylactic
Defending or protection from disease

Pyriform
Pear-shaped

Septicemia
Systemic disease in the blood associated with pathogenic organisms or their toxins; often bacterial toxin

Scoliosis
Lateral, abnormal curvature of the spine

Sessile
Attached, not mobile

Systemic
Affecting the body as a whole (versus only affecting skin or gills)

TAN
Total Ammonia Nitrogen; the sum of un-ionised and ionised ammonia

Theront
The free-swimming, infestive stage of I. multifiliis

Tomite
The small ciliated stage that is released from the cyst stage in I. multifiliis

Trophont
The feeding stage of I. multifiliis *under gill and skin epithelium*

Tubercles
Small nodule or raised area on a surface, a chronic inflammatory response to a pathogen or a foreign body

Ulcer
A local defect on the surface of an organ or tissue caused by damage and loss of dead tissue; can be haemorrhagic

Zoonosis
A disease of animals that can be transmitted to humans

REFERENCES AND FURTHER READING

Aquaculture and Aquatic Animal Health (2002), Proceedings 347, 17-19 April, 2002, Published by Post Graduate Foundation in Veterinary Science, University of Sydney.

Ashburner, L.D. (1983). Disease problems in fish culture. In, *Proceedings of the First Freshwater Aquaculture Workshop*, February 1983. (Editor L.F. Reynolds), pp. 154-180. Department of Agriculture New South Wales, Sydney.

Brown, L. (Editor) (1993). *Aquaculture for Veterinarians*. Pergamon Press Ltd.

Callinan, R.B. and Rowland S.J. (1995). Diseases of silver perch. In, *Silver Perch Culture* (Eds S.J. Rowland and C. Bryant), pp. 67-75. Austasia Aquaculture, Sandy Bay, Australia.

Frances, J., Tennent, R. and Nowak, B.F. (1997). Epitheliocystis in silver perch, *Bidyanus bidyanus* (Mitchell). *Journal of Fish Diseases* **20**, 453-457.

Hoffman, G.L. (1967). *Parasites of North American Freshwater Fishes*, University of California Press, Berkeley. pp 486.

Hoffman, G.L. (1999). *Parasites of North American Freshwater Fishes*, 2nd ed, Cornell University Press.

Ingram, B.A., Gavine, F. and Lawson, P. (2005). *Fish Health Management Guidelines for Farmed Murray Cod*. Fisheries Victoria Research Report Series No. 32. Department of Primary Industries, Melbourne.

Landos, M., Rowland, S.J., Nixon, M., Mifsud, C., Read, P.A. (2007). Evaluation of trichlorfon to treat infestations of the monogenean gill fluke, *Lepidotrema bidyana*, on silver perch (*Bidyanus bidyanus*). In, *Development of a Health Management Strategy for the Silver Perch Aquaculture Industry*, Report to Fisheries Research and Development Corporation on Projects 2000/267 and 2004/089. NSW Department of Primary Industries.

Langdon, J.S. (1987). Epizootic haematopoietic necrosis, a new viral disease in redfin perch, *Perca fluviatilis* L., in Australia. *Journal of Fish Diseases* **10**, 289-297.

Langdon, J.S. and Humphrey, J.D. (1989). Experimental transmission and pathogenicity of epizootic haematopoietic necrosis virus (EHNV) in redfin perch and 11 other teleosts. *Journal of Fish Diseases* **12**, 295-310.

Langdon, J.S. (1992). Major protozoan and metazoan parasitic diseases of Australian finfish. In *Finfish Workshop*. pp 1-26, Proceedings 182, Post Graduate Committee in Veterinary Science, University of Sydney, Australia.

Mifsud, C. and Rowland, S.J. (2007). Use of salt to control ichthyophthiriosis and prevent saprolegniosis in silver perch (*Bidyanus bidyanus*). In, *Development of a Health Management Strategy for the Silver Perch Aquaculture Industry*, Report to Fisheries Research and Development Corporation on Projects 2000/267 and 2004/089. NSW Department of Primary Industries.

Noga, E.J. (2000). *Fish Disease –Diagnosis and Treatment*. Iowa State University Press, Iowa.

Piper, R.G., McElwain, I.B., Orme, L.E., McCraren, J.P., Fowler, L.G. and Leonard, J.R. (1982). *Fish Hatchery Management*. United States Dept. of the Interior, Fish and Wildlife Service, Washington, DC, pp 517.

Plumb, J.A. (1994). *Health Maintenance of Cultured Fishes – Principal Microbial Diseases*. CRC Press, Boca Raton.

Rowland, S.J. (1983). Design and operation of an extensive aquaculture system fro breeding warmwater fishes. In, *Proceedings of the First Freshwater Aquaculture Workshop, February 1983*. (Editor L.F. Reynolds), pp. 121-144. Department of Agriculture New South Wales, Sydney.

Rowland S.J. (1995). Water quality in the intensive pond culture of silver perch. In, *Silver Perch Culture* (Eds S.J. Rowland and C. Bryant), pp. 51-65. Austasia Aquaculture, Sandy Bay, Australia

Rowland, S.J. and Bryant, C. (Editors) (1995). *Silver Perch Culture. Proceedings of Silver Perch Workshops, Grafton and Narrandera, April, 1994*. Austasia Aquaculture, Sandy Bay, Australia.

Rowland, S.J. and Ingram, B.A. (1991). Diseases of Australian Native Freshwater Fishes with Particular Emphasis on the Ectoparasitic and Fungal Diseases of Murray Cod *(Maccullochella peeli),* Golden Perch (*Macquaria ambigua*) and Silver Perch (*Bidyanus bidyanus*). Fisheries Bulletin **4**, NSW Fisheries, Sydney.

Rowland, S.J., Ingram, B.A. and Prokop, F.B. (1991). Suspected cysts of the protozoan parasite *Chilodonella hexasticha*. *Bulletin of the European Association of Fish Pathologists* **11**, 159-161.

Rowland, S.J., Landos, M., Callinan, R. B., Mifsud, C., Read, P.A. and Nixon, M. (2007). Review of the infectious diseases of the Australian freshwater fish silver perch (*Bidyanus bidyanus*) in aquaculture. In, *Development of a Health Management Strategy for the Silver Perch Aquaculture Industry*, Report to Fisheries Research and Development Corporation on Projects 2000/267 and 2004/089. NSW Department of Primary Industries.

Rowland, S.J., Mifsud, C., Nixon, M., Read, P.A. and Landos, M. (2007). Use of formalin and copper to treat ichthyophthiriosis in silver perch. In, *Development of a Health Management Strategy for the Silver Perch Aquaculture Industry*, Report to Fisheries Research and Development Corporation on Projects 2000/267 and 2004/089. NSW Department of Primary Industries.

Rowland, S.J., Read, P.A., Landos, M., Mifsud, C., Nixon, M., Tully, P.A. and Callinan, R.B. (2007). Health Management Plan for Silver Perch Culture. In, *Development of a Health Management Strategy for the Silver Perch Aquaculture Industry*, Report to Fisheries Research and Development Corporation on Projects 2000/267 and 2004/089. NSW Department of Primary Industries.

Rowland, S.J. and Tully, P. (2004). Hatchery Quality Assurance Program for Murray Cod (*Maccullochella peelii peelii*), Golden Perch (*Macquaria ambigua*) and Silver Perch (*Bidyanus bidyanus*). NSW Department of Primary Industries, Sydney.

Rowland, S.J., Mifsud, C., Nixon, M. and Boyd, P. (2006). Effects of stocking density on the performance of the Australian freshwater silver perch (*Bidyanus bidyanus*) in cages. Aquaculture **253**, 301-308.

Rowland, S.J., Nixon, M., Landos, M., Mifsud, C., Read, P. and Boyd, P. (2006). Effects of formalin on water quality and parasitic monogeneans on silver perch (*Bidyanus bidyanus* Mitchell) in earthen ponds. *Aquaculture Research* **37**, 869-876.

Schlotfeldt, H.-J., Alderman, D.J., Baudin-Laurencin, F., Bernoth, E.M., Bruno, D.W., Daelman, W., Kleingeld, D., Lorenzen, E. and Thorud, K. (1995). What should I do? A practical guide for the fresh water fish farmer. Supplement to *Bulletin of the European Association of Fish Pathologists* **15** (4) 1-60.

Selosse, P.M. and Rowland, S.J. (1990). Use of common salt to treat ichthyophthiriasis in Australian warmwater fishes. *The Progressive Fish-Culturist* **52**, 124-127.

Whittington, R.J., Djordjevic, S.P., Carson, J. and Callinan, R.B. (1995) Restriction endonuclease analysis of atypical *Aeromonas salmonicida* isolates from goldfish, *Carassius auratus*, silver perch *Bidyanus bidyanus*, and greenback flounder *Rhombosolea taparina* in Australia. *Diseases of Aquatic Organisms* **22**,185-191.