

Signs of disease

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

Clinical signs of disease in an infected animal

- lethargy
- emaciation
- soft shells
- heavy fouling from external parasites
- black gills
- reduced growth

Gross signs of disease in an infected animal

- digestive gland (hepatopancreas) degenerated — appears pale to white

Disease agent

Necrotising hepatopancreatitis is caused by a species of alpha-proteobacterium that infects the hepatopancreas of prawns, also referred to as NHP bacteria.

Host range

Crustaceans known to be susceptible to the disease:

blue shrimp*	(<i>Penaeus stylirostris</i>)
northern brown shrimp*	(<i>Penaeus aztecus</i>)
northern white shrimp*	(<i>Penaeus setiferus</i>)
white shrimp*	(<i>Penaeus vannamei</i>)
yellowleg shrimp*	(<i>Penaeus californiensis</i>)

* naturally susceptible



White shrimp (*Penaeus vannamei*) with necrotising hepatopancreatitis. Note darkening at base of swimmerets, giving fouled, 'dirty' appearance

Source: DV Lightner



White shrimp with necrotising hepatopancreatitis. Note marked reduction in size of hepatopancreas

Source: DV Lightner



Necrotising hepatopancreatitis continued

Presence in Asia–Pacific

Necrotising hepatopancreatitis has been officially reported from Vietnam.



Epidemiology

- Necrotising hepatopancreatitis requires lengthy periods of high air temperature (29°–31°C) and elevated salinity (20–40 ppt).
- Mortality can be 90–95% within 30 days of an outbreak.
- Mortalities usually occur midway through the grow-out phase.
- Necrotising hepatopancreatitis appears to be transmitted by direct ingestion of an unidentified carrier (a reservoir host).
- The disease is not transmitted either vertically (from parent to offspring) or through cannibalism.

Differential diagnosis

The differential diagnostic table and the list of similar diseases appearing at the bottom of each disease page refer only to the diseases covered by this field guide. Gross signs observed might well be representative of a wider range of diseases not included here. Therefore, these diagnostic aids should not be read as a guide to a definitive diagnosis, but rather as a tool to help identify the listed diseases that most closely account for the gross signs.

Similar diseases

The clinical signs described and shown here may also be symptomatic of other bacterial or viral infections, or poor water quality in rearing ponds (high ammonia, low oxygen, high and low pH). Further laboratory examination is required for a definitive diagnosis.

Sample collection

Because of uncertainty in differentiating diseases using only gross signs, and because some aquatic animal disease agents might pose a risk to humans, you should not try to collect samples unless you have been trained. Instead, you should phone your national hotline number and report your observations. If samples have to be collected, the agency taking the call will advise you on what you need to do. Local or district fisheries/veterinary authorities could advise you on sampling.

Emergency disease hotline

For your national emergency disease hotline number, see Whom to contact if you suspect a disease.

Further reading

http://www.oie.int/aac/eng/cards/en_diseasecard.htm

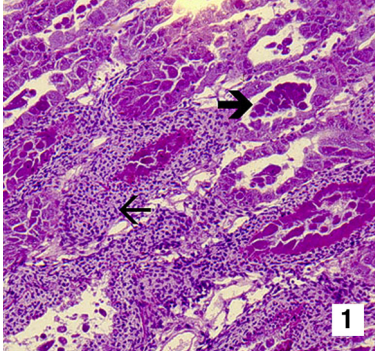
http://www.pac.dfo-mpo.gc.ca/sci/shelldis/pages/nechepsp_e.htm

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



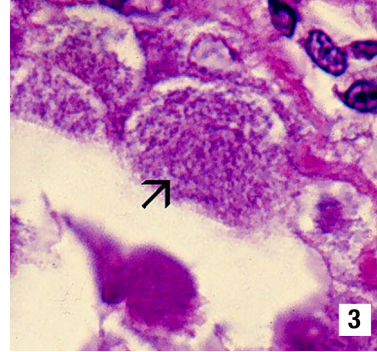
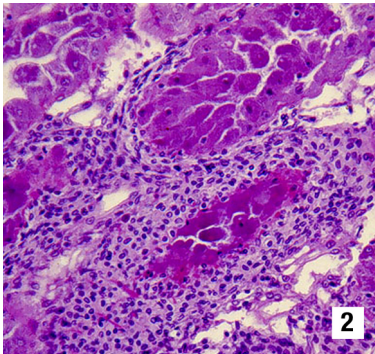
Necrotising hepatopancreatitis continued

Histological images



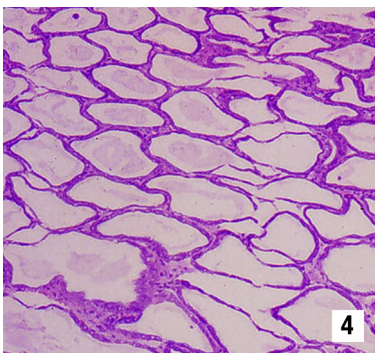
Low (Fig 1, 150x) and mid (Fig 2, 300x) magnification photomicrographs of the hepatopancreas (HP) of a juvenile white shrimp (*Penaeus vannamei*) with severe, subacute (grade 3–4) necrotising hepatopancreatitis (NHP). Severe haemocytic inflammation (with some melanised foci) of the intratubular spaces (small arrow) in response to necrosis, cytolysis, and sloughing of HP tubule epithelial cells (large arrow) are among the principal histopathological changes due to NHP

Source: DV Lightner



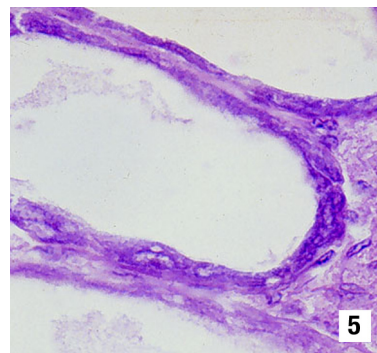
A high magnification (1700x) view of a portion of the HP from Figs 1 and 2. The HP tubule epithelial cells show no cytoplasmic lipid droplets, but instead contain masses of the tiny, non-membrane bound, intracytoplasmic NHP bacteria (arrow)

Source: DV Lightner



Low magnification (100x) view of the HP of a juvenile white shrimp with severe, chronic NHP. The HP tubule epithelium is markedly atrophied, resulting in the formation of large oedematous (fluid filled or 'watery') areas in the HP

Source: DV Lightner



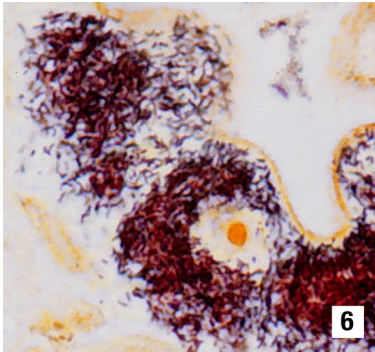
A higher magnification (900x) photomicrograph of the atrophied HP from a juvenile white shrimp with chronic NHP. In contrast to the subacute phase of NHP, chronic phase HPs shown no, or only occasional, foci of haemocytic inflammation (large arrow) of the necrotic or degenerating HP tubules. NHP bacteria (small arrow) may be found in the cytoplasm of an occasional HP cell

Source: DV Lightner



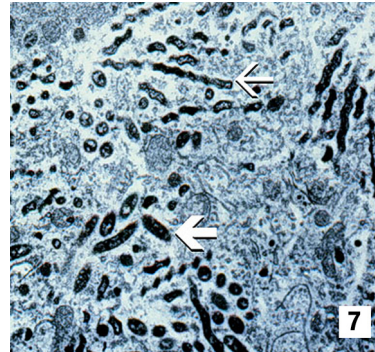
Necrotising hepatopancreatitis continued

Histological images



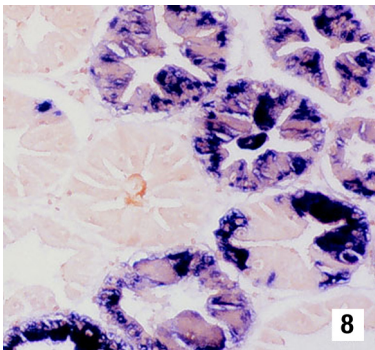
Section of the HP of a juvenile white shrimp that is similar to that shown in Fig 3. Cytoplasmic masses of the NHP bacterium are silver stained and appear brown to black with the modified Steiner stain. Unaffected cells and nuclei are pale brown (1600x)

Source: DV Lightner



TEM (10 000x) of a hepatopancreatocyte from a juvenile white shrimp with NHP. Profiles of intracellular rod-shaped forms (large arrow) and helical forms (small arrow) of the NHP bacterium are abundant in the cytoplasm

Source: DV Lightner



Low (Fig 8, 250x) and high (Fig 9, 1000x) magnification views of sections of the HP of a juvenile white shrimp with NHP. This section has been assayed for NHP using a DIG-labelled DNA probe. Cytoplasmic masses of the NHP bacterium are marked blue to blue-black by the probe. Unaffected cells and host cell nuclei take the brown counter stain. DIG-labelled NHP probe and Bismarck Brown

Source: DV Lightner

