# Diseases of crustaceans Viral diseases—**White spot disease**

## 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.

## Disease signs at the farm level

- characterised by high and rapid mass mortality of rapid onset, mainly in farmed penaeid prawns
- can occur at any stage of the grow-out period

## Disease signs at the tank and pond level

- prawns with white spot disease often do not show distinctive clinical signs
- lethargy
- cessation of feeding
- a few days later, moribund prawns near surface at edge of rearing pond

# Clinical signs of disease in an infected animal

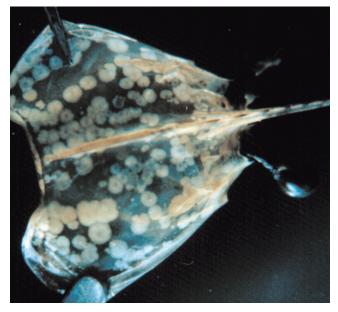
- loose shell
- white calcium deposits embedded in shell, causing white spots 0.5–2.0 mm in diameter for which the disease is named (but white spot disease can occur without these signs)
- darkened (red or pink) body surface and appendages
- heavy surface and gill fouling by external parasites
- white midgut line through abdomen of severely affected larvae and postlarvae

The shell lesions range from minute spots to discs several millimetres in diameter, and may coalesce into larger plates. They are most easily observed by removing the cuticle over the cephalothorax, scraping away any attached tissue with the thumbnail and holding the cuticle up to the light.



White spot disease in giant black tiger prawn (*Penaeus monodon*). Prawns at top and right of main photo show pink body colour typical of acute phase of infection. Those at bottom and to left show classic white spots following acute phase

Source: DV Lightner



White spot disease in giant black tiger prawn, showing classic white spots on carapace

Source: DV Lightner



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White spots in the cuticle are unreliable even for preliminary diagnosis of white spot disease, because similar spots can be produced by some bacteria, high alkalinity and other infectious or environmental conditions.

## **Disease agent**

The causative agent of white spot disease is white spot syndrome virus, a large DNA virus assigned to the new genus *Whispovirus* (family Nimaviridae). The virus infects only crustaceans and appears not to be related to any other known viruses. It is not involved in the parasitic disease, common in finfish, also known as white spot.

## **Host range**

All decapod crustaceans (order Decapoda), including prawns, lobsters and crabs from marine, brackish or freshwater environments, are considered susceptible to infection. However, the disease has mainly been a problem in farmed penaeid (family Penaeidae) prawns.

Crustaceans known to be susceptible white spot disease:

black tiger prawn*	(Penaeus monodon)
Chinese white shrimp*	(Penaeus chinensis)
Gulf banana prawn*	(Penaeus merguiensis)
Indian banana prawn*	(Penaeus indicus)
Kuruma prawn*	(Penaeus japonicus)
Pacific white shrimp*	(Penaeus vannamei)
red claw freshwater crayfish*	(Cherax quadricarinatus)
blue shrimp	(Penaeus stylirostris)
green tiger prawn	(Penaeus semisulcatus)

White spot syndrome virus also occurs naturally in many other decapods, including:

mud crabs\*(Scylla serrata, Charybdis feriatus, Portunus pelagicus, P. sanguinolentus)sand shrimp\*(Metapenaeus spp)and other arthropods(Metapenaeus spp)

# **Presence in Asia–Pacific**

White spot disease has been officially reported from Bangladesh, Burma (Myanmar), Cambodia, China, Hong Kong, India, Indonesia, Iran, Japan, Malaysia, the Philippines, the Republic of Korea, Singapore, Thailand and Vietnam.



<sup>\*</sup> naturally susceptible (other species have been shown to be experimentally susceptible)



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## **Epidemiology**

- Although many species of crustaceans are susceptible to infection, white spot disease is mainly a disease of farmed penaeid prawns.
- High mortalities have been reported in many countries.
- Experience has shown the production of prawn farms to fall to about 40% of normal for two years, and then recover to about 70% over the long term.
- Resistance to white spot syndrome virus has not been reported for any of the penaeid species listed above.
- Infection may be low level and chronic in healthy crustaceans, or acute with disease and mortalities.
- Viral multiplication and disease appear to be induced by environmental stress.
- Vertical transmission occurs from infected broodstock, causing chronic infection in postlarvae.
- Transmission of disease is usually via cannibalism of sick or dying prawns, or directly through contaminated water.
- Many other crustaceans (such as crabs, krill, lobsters and possibly copepods) and insects are potential carriers of infection while not suffering from the disease.
- Birds can transmit the disease from pond to pond by releasing caught prawns over neighbouring ponds.
- White spot syndrome virus can persist and retain infectivity in seawater for 4–7 days.
- White spots in the cuticle are unreliable even for preliminary diagnosis of white spot disease, as similar lesions can be produced by some bacteria, high alkalinity and other infectious or environmental conditions

#### **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

Infectious hypodermal and haematopoietic necrosis, yellowhead disease, Taura syndrome



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## **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

The currently accepted procedures for a conclusive diagnosis of white spot disease are summarised at http://www.oie.int/eng/normes/fmanual/A\_00049.htm

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

# **Further images**



A juvenile giant black tiger prawn (*Penaeus monodon*) that is displaying the distinctive white spots of white spot disease (WSD). White spots are especially visible on the carapace and the rostrum. While providing a tentative diagnosis of WSD infection, white spots are not always visible in shrimp with acute phase white spot syndrome, and may develop in the subacute to chronic or recovery phases of the infection

Source: DV Lightner



Four juvenile black tiger prawns, including the one shown in Fig 1 (at bottom), with different gross signs of white spot syndrome. The top and right shrimp show few, if any, white spots, but show a pink to red-brown discolouration due to expansion of the subcuticular chromatophores. This reddish appearance may be a gross sign that is more apparent in the acute phase of the disease. The shrimp on the left and bottom display diagnostic white spots that develop after the acute phase of the disease

Source: DV Lightner



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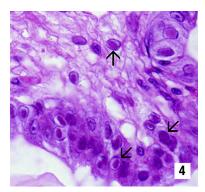
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# **Histological images**



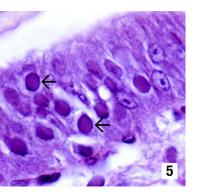
The carapace from a juvenile black tiger prawn with WSD. Calcareous deposits on the underside of the shell account for the white spots. Photo: P Saibaba, SKBR College, Amalapuram, India

Source: DV Lightner

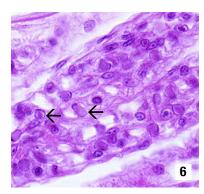


Photomicrograph (900x) of a histological section from the stomach of a juvenile black tiger prawn infected with WSD. Prominent intranuclear inclusion bodies are abundant in the cuticular epithelium and subcuticular connective tissue of the organ (arrows). Cells in different phases of infection by WSD display intranuclear inclusion bodies. The early phase inclusion bodies that predominate in this section are centronuclear, eosinophilic, and separated from the nuclear membrane and marginated chromatin by an artifactual halo (resembling infectious hypodermal and haematopoietic necrosis inclusion bodies)

Source: DV Lightner



Histological section (1300x) of the stomach of a juvenile Chinese white shrimp (*P. chinensis*) with an advanced WSD infection. Fully developed WSD intranuclear inclusion bodies (arrows) are more basophilic, appear granular in texture, and nearly fill the affected hypertrophied nucleus. Occlusion bodies are not present Source: DV Lightner



Section of the gills from a juvenile Chinese white shrimp with WSD (900x). Nearly one-quarter of the cells present are infected, as indicated by the presence of developing and fully developed intranuclear inclusion bodies of WSD (arrows)

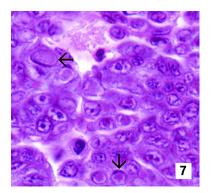
Source: DV Lightner



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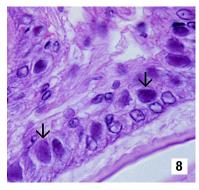


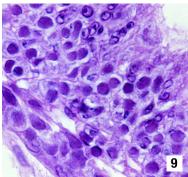
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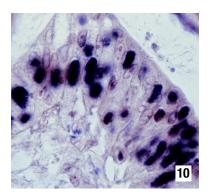
Section of a WSD-infected haematopoietic nodule (1300x) from a juvenile Chinese white shrimp. As in Fig 6, nearly one-fourth of the cells present in the section display intranuclear inclusions bodies of WSD (arrows) in various stages of development

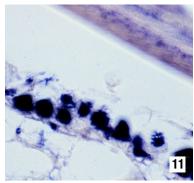
Source: DV Lightner

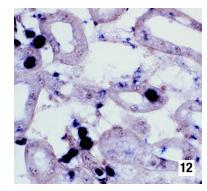




**Histological sections** (900x) of the stomachs of blue shrimp (P. stylirostris, Fig 8) and white shrimp (P. vannamei, Fig 9) experimentally infected with WSD. **Both species display** severe (grade 4) infections by WSD, with classic WSD intranuclear inclusion bodies (arrows) that are identical to those illustrated in Figs 3-7 Source: DV Lightner







Sections of various tissues from a WSD-infected iuvenile white shrimp reacted by in situ hybridisation with a DIG-labelled DNA probe to the virus. The probe has reacted strongly with intranuclear inclusion bodies containing WSD in the various tissues of this shrimp, including the cuticular epithelium of the stomach (Fig 10, 900x), the cuticular epithelium and connective tissues of the carapace (Fig 11, 900x), and epithelial cells in the antennal gland (Fig 12, 450x) Source: DV Lightner



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