Diseases of Crustaceans — **Acute Hepatopancreatic Necrosis Syndrome (AHPNS)**

**Signs of Disease**

In the absence of identified biotic or abiotic cause(s) of the syndrome, the following disease signs can be used for presumptive (pond level) and confirmative (animal level) diagnosis of the disease.

**Disease signs at pond level**

- Often pale to white hepatopancreas (HP) due to pigment loss in the connective tissue capsule.
- Significant atrophy (shrinkage) of HP.
- Often soft shells and guts with discontinuous contents or no content.
- Black spots or streaks sometimes visible within the HP.
- HP does not squash easily between thumb & finger.
- Onset of clinical signs and mortality starting as early as 10 days post stocking.
- Moribund shrimp sink to bottom.

**Disease signs at animal level by histopathology**

- Acute progressive degeneration of the HP accompanied initially by a decrease of R, B and F-cells followed last by a marked reduction of mitotic activity in E-cells.
- Progress of lesion development is proximal-to-distal with dysfunction of R, B, F, and lastly E-cells, with affected HP tubule mucosal cells presenting prominent karyomegaly (enlarged nuclei), and rounding and sloughing into the HP tubule lumens.
Acute Hepatopancreatic Necrosis Syndrome continued

Normal shrimp hepatopancreas with distinct R, B, F and E (inset) cells.
Source: C Lavilla-Pitogo

Proximal-to-distal progression of lesion in the hepatopancreas with dysfunction of R, B, F and E cells.
Source: D Lightner

Sloughing of HP cells
Lack of E-cell mitosis
Lack of B, F and R cells

Enlarged HP nuclei
Hemocytic infiltration
Secondary bacterial infection

Histopathology of *Penaeus vannamei* hepatopancreas from Thailand affected by AHPNS.
Source: T Flegel
Acute Hepatopancreatic Necrosis Syndrome continued

- The sloughed HP cells provide a substrate for intense bacterial growth, resulting in massive secondary bacterial infection (putative *Vibrio* spp.) and complete destruction of HP at the terminal phase of the disease.
- Accompanying the initial sloughing of HP tubule epithelial cells and the development of a secondary bacterial infection is intense intertubular hemocytic aggregation and hemocyte encapsulation of necrotic HP tubules and melanization of the more proximal portions of HP tubules in some shrimp.

**Disease Agent**

Idiopathic – no specific disease causing agent (infectious or toxic) has been identified.

**Host Range**

AHPNS affects both *Penaeus monodon* and *P. vannamei* (*P. chinensis* was also reported to be affected in China).

**Presence in Asia-Pacific**

AHPNS has been officially reported in China and Vietnam (2010), Malaysia (2011) and Thailand (2012).

**Important Notes**

While the apparent spread of AHPNS throughout the region suggests an infectious or at least biological agent may be involved, thus far, laboratory challenge tests have failed to demonstrate that the disease is transmissible and no infectious agent or toxin has been identified. The histopathology is suggestive of toxicity. Testing of feeds from affected farms and two crustaceacides including cypermethrin have similarly failed to reproduce the disease. The disease is not related to any of the known shrimp pathogens like WSSV, YHV, IMNV, TSV. At this stage the cause is unknown, and the possibility of an infectious agent and/or toxin cannot be discounted.
Acute Hepatopancreatic Necrosis Syndrome continued

Further information

The AHPNS news story, final report and audio recordings of 19 technical presentations during the Asia Pacific Regional Emergency Consultation held in Bangkok, Thailand (9-10 August 2012) are available at the following links for wider dissemination of scientific information about this emerging shrimp disease:

http://www.enaca.org/modules/podcast/programme.php

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This disease card was prepared based on the outcomes of the Asia Pacific Emergency Regional Consultation on Early Mortality Syndrome (EMS)/Acute Hepatopancreatic Necrosis Syndrome (AHPNS) held in Bangkok, Thailand on 9-10 August 2012. The workshop was co-organized by the Australian Government Department of Agriculture, Fisheries and Forestry and the Network of Aquaculture Centres in Asia-Pacific.