

ACUTE HEPATOPANCREATIC NECROSIS DISEASE

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Acute hepatopancreatic necrosis disease (AHPND) emerged as a new disease of shrimp in 2010. The disease is sometimes referred to as early mortality syndrome, which is a broader term used to describe a range of shrimp health problems that lead to early mortality. The information presented in this technical fact sheet reflects the epidemiological observations and research information available to date (December 2013), together with additional data on *Vibrio parahaemolyticus*.

AETIOLOGY

Classification of the causative agent

AHPND has a bacterial aetiology. A bacterial isolate identified as a member of the *Vibrio harveyi* clade, most closely related to *Vibrio parahaemolyticus*, has been found to cause AHPND experimentally. Further studies to characterise the causative agent, including genetic characterisation, are continuing.

Resistance to physical and chemical action

Data are from transmission experiments for AHPND and strains of *V. parahaemolyticus* occurring in seafood:

Temperature:	Freezing – Attempts to transmit AHPND using infected frozen shrimp have been unsuccessful. Strains of <i>V. parahaemolyticus</i> in seafood are known to be sensitive to freezing (–18 to –24°C) and reductions in culturable cells to non-detectable levels have been reported after freezing for several weeks. Time of total inactivation depends on the initial number of bacteria and temperature. Refrigeration – Strains of <i>V. parahaemolyticus</i> in seafood are known to be sensitive to refrigeration (4°C) and reductions in culturable cells have been reported, but not to non-detectable levels during the storage life of seafood products at this temperature. Heating – Strains of <i>V. parahaemolyticus</i> in seafood are known to be sensitive to heating and reductions in culturable cells to non-detectable levels have been reported at 55°C for 5 minutes and 80°C for 1 minute.
Chemicals/ Disinfectants:	Susceptible to common disinfectants. No culturable cells after exposure to pH 5 for 15 minutes.
Survival:	Survival to 9 and 18 days in filtered estuarine water and filtered seawater respectively. Densities of <i>V. parahaemolyticus</i> in seawater are known to be temperature dependant.

EPIDEMIOLOGY

Hosts

AHPND has been reported from farmed populations of the following shrimp species:

- *Penaeus vannamei*
- *Penaeus monodon*
- *Penaeus chinensis*

Transmission

AHPND has been transmitted experimentally by immersion and reverse gavage. Based on these experiments, transmission by oral routes and cohabitation should be expected.

Occurrence

AHPND has been officially reported in China (People's Rep. of) and Vietnam (2010), Malaysia (2011) and Thailand (2012) and Mexico (2013).

AHPND is not listed by the OIE. However the disease does meet the definition of an emerging disease in the OIE *Aquatic Animal Health Code (Aquatic Code)*. Member countries should consider their obligation to report any occurrence of the disease in accordance with Chapter 1.1 of the *Aquatic Code*.

AHPND is listed in the Quarterly Aquatic Animal Disease (QAAD) Reporting Program for Asia and the Pacific. Information on the occurrence of this disease in the Asia-Pacific region is available in QAAD reports commencing from 2013 at the following websites: <http://www.enaca.org/> and <http://www.rr-asia.oie.int/>

Food safety

Consumption of raw or undercooked seafood contaminated with *V. parahaemolyticus* can lead to development of acute gastroenteritis in humans. Results of preliminary investigations of AHPND did not detect the human pathogenic strains producing thermostable direct haemolysin (TDH) and TDH-related haemolysin (TRH), in the *V. parahaemolyticus* causing AHPND. There has been no report of human-related disease (e.g. gastroenteritis) linked to the consumption of affected shrimp from any of the affected countries since the emergence of AHPND (FAO, 2013).

DIAGNOSIS

A disease card for AHPND is available from the Network of Aquaculture Centres in Asia Pacific (NACA: see references).

Clinical diagnosis

Typical signs of AHPND begin within 10–30 days after stocking of post larvae into a newly prepared pond. The following signs may be observed:

- Hepatopancreas (HP) often pale to white 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 and finger
- Onset of clinical signs and mortality starting as early as 10 days post-stocking
- Moribund shrimp sink to the bottom

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
- The sloughed HP cells provide a substrate for intense bacterial growth, resulting in massive secondary bacterial infection 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 haemocytic aggregation and haemocyte encapsulation of necrotic HP tubules and melanisation of the more proximal portions of HP tubules in some shrimp

Laboratory diagnosis

Samples

For culture of bacteria:

- Live shrimp

For histopathology:

- Whole juvenile shrimp fixed in Davidsons fixative

For molecular tests:

- Whole juvenile shrimp fixed in 100% ethanol or frozen

Procedures

Identification of the agent

- Bacterial culture
- Biochemical methods
- PCR and sequencing

Histopathology

- Demonstration of characteristic histopathology

PREVENTION AND CONTROL

Commodity	Relevant knowledge	Likelihood of transmission to farmed or wild shrimp populations
Live shrimp for aquaculture	AHPND has been transmitted experimentally by immersion. Transmission by cohabitation is expected. <i>Penaeus monodon</i> , <i>P. chinensis</i> and <i>Penaeus vannamei</i> are known to be susceptible.	High
Fresh dead shrimp for human consumption	AHPND has been transmitted experimentally by immersion and reverse gavage, and transmission by the oral route is likely. <i>Vibrio parahaemolyticus</i> is sensitive to refrigeration but will remain viable for several weeks in chilled aquatic animal products.	Low Transmission would require the existence of pathways for exposure of susceptible populations.
Frozen shrimp for human consumption	Attempts to experimentally transmit AHPND from frozen shrimp tissues have failed. Freezing has been shown to reduce the number of culturable <i>V. parahaemolyticus</i> but may not eliminate bacteria entirely. Time to total inactivation depends on the initial number of bacteria and temperature.	Negligible Transmission would require the existence of pathways for exposure of susceptible populations.
Live shrimp feed	AHPND has been transmitted experimentally by immersion and reverse gavage, and transmission by the oral route is likely. Live feeds (e.g. for hatchery brood stock) may present a transmission pathway if sourced from populations in AHPND endemic areas.	High
Manufactured shrimp feeds – extruded	AHPND has been transmitted experimentally by immersion and reverse gavage, and transmission by the oral route is likely. <i>Vibrio parahaemolyticus</i> does not tolerate heating and is not expected to survive commercial feed manufacturing processes where temperatures reach 100°C for at least 1 minute.	Negligible

REFERENCES AND OTHER INFORMATION

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The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (scientific.dept@oie.int). Last updated December 2013.