A new disease appeared in shrimp farms located in southern China and Hainan Island in 2010. By early 2011, "early mortality syndrome" (EMS) was also detected in Vietnam and Malaysia. The disease appears within 20 to 30 days of stocking ponds with postlarvae. Both black tiger shrimp, *Penaeus monodon*, and Pacific white shrimp, *Litopenaeus vannamei*, are affected by the disease. Mortalities can approach 100% in severely affected ponds, where diseased shrimp become lethargic and anorexic. Upon simple dissection, the hepatopancreas organs of the shrimp may appear atrophied and whitish with black streaks. Other signs include a soft, generally darker shell and mottling of the carapace.

**Pathology**

Both *P. monodon* and *L. vannamei* with EMS present the same pathology. Samples of shrimp preserved for histology have shown the effects of EMS appear to be limited to the hepatopancreas. In the terminal stages of the highly fatal disease, secondary bacterial infection further damages the hepatopancreas. The cause of EMS is under investigation.

**Etiology**

This degenerative pathology of the hepatopancreas is highly suggestive of a toxic etiology. Similar lesions have been reported in the H.P.s of shrimp exposed to aflatoxin B1 and the mitosis inhibitor benomyl, which supports this theory. Studies to determine the etiology of EMS run at the University of Arizona Aquaculture Pathology Laboratory have not been successful. The laboratory has tested commercial feeds collected at shrimp farms with EMS, and frozen samples of shrimp with EMS from affected farms were used in infectivity studies. A crustacide commonly used in the region to kill vectors of white spot syndrome prior to stocking has also been tested.

To date, the University of Arizona lab has not experimentally induced lesions of the hepatopancreas consistent with those observed in shrimp with EMS.

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**Summary:**

Early mortality syndrome is a new disease that has been detected at shrimp farms in Asia. It appears within 30 days of stocking and causes symptoms that include lethargy, soft, darkened shells and mottling of the carapace. The physiological effects of EMS appear to be limited to the hepatopancreas. In the terminal stages of the highly fatal disease, secondary bacterial infection further damages the hepatopancreas. The cause of EMS is under investigation.