Hematodinium sp. - Bitter Crab Disease of Tanner Crabs

I. Causative Agent and Disease

Bitter crab disease is caused by a parasitic dinoflagellate, *Hematodinium* sp. belonging to the protozoan phylum Alveolata, subphylum Dinoflagellida, order Syndinida and family Syndiniceae. A major feature of classification for dinoflagellates is the biflagellated grooved dinospore stage. Obligate *Hematodinium* parasites have been described from several species of crustaceans, certain fishes and cephalopods. In crustaceans the type species is *H. perezi* parasitizing the European shore crab. All *Hematodinium* described from crustaceans parasitize the hemolymph causing systemic disease and mortality affecting at least 26 species of crustacean hosts in Europe, Australia and North America including many commercially important crab species and 13 species of benthic amphipods.

II. Host Species

The bitter crab *Hematodinium* is reported from Tanner and snow crabs (*C. bairdi, C. opilio*) in southeast Alaska, the Gulf of Alaska and the eastern and western Bering Sea; from deep water Tanner crabs (*C. tanneri*) in British Columbia, Canada and from snow crabs in Atlantic Canada.

III. Clinical Signs

Parasitized crabs are lethargic and die when handled, have an exaggerated red color of the carapace, flaccid chalky textured meat and white opaque hemolymph caused by the myriad numbers of dinoflagellates. The cooked meats of parasitized crabs have an astringent aspirin aftertaste and are unmarketable, hence the “bitter crab” name of the disease.

IV. Transmission

Although the natural route of transmission has not been established, there are likely to be several modes in which the parasite is transmitted horizontally via: stages of the parasite in seawater that enter breaks in the cuticle during mating and molting; cannibalism or feeding on detritis containing resting spores; sexual transmission via seminal fluids of parasitized male crabs and possibly through a reservoir host such as bottom dwelling amphipods. The parasite and disease have been transmitted in the laboratory by injection of hemolymph containing vegetative trophonts and dinospore stages. Life history stages include: trophonts which are single cells and have slow division; larger plasmodia with multiple nuclei; pre-spores with multiple nuclei and rapid division followed by sporulation of biflagellated spores with a single nucleus. Spores are of two types, a large macrospore and a small microspore, generally with only one spore type occurring per host. Spores may possibly have a disseminatory or resting stage function rather than transmission. Laboratory studies suggest that the life cycle of the parasite occurs over a 15 to 18 month period. Infestation likely occurs from the trophont stage during the spring molting period from mid-March to May. Crabs dying in the summer and fall following sporulation were likely parasitized in the spring of the previous year. After sporulation in the fall the prevalence of parasitism becomes almost undetectable (eclipse) until the new infestations of the previous spring build to detectable levels that can be observed the following late winter and early spring.

V. Diagnosis

Diagnosis is based on typical clinical signs with the occurrence of myriad numbers of dinoflagellate stages (usually non-motile trophonts) in wet mounts or stained smears of hemolymph. Trophonts are 15.4 X 20.7 µm, have an indistinct nuclear profile and foamy cytoplasm with droplets exuding from the surface pellicle in a stained hemolymph smear. A key diagnostic feature visible in many trophonts is the dinokaryon nuclear division producing condensed V-shaped pairs of chromosomes. Nonmotile plasmodia are similar in appearance but larger and multinucleated. Prespores are similar to plasmodia but each nucleus has a distinct lobed nuclear outline. Macrospores are oval 15.2 X 11.4 µm having slow motility and a beaked protrusion on one end. The nucleus is longer than it is wide with a distinct outline. The microspore is elliptical and smaller at 12.0 X 4.4 µm and has rapid motility with a refractile body at the posterior end and 2 flagella more often visible on stained smears. The microspore develops an obvious bent corkscrew shape within 6 days after sporulation. Histological examination demonstrates parasite stages within hemal sinuses throughout the tissues of the crab host. PCR primers are available for certain *Hematodinium* sp. parasitizing commercial crab species.

VI. Prognosis for Host

Virtually 100% mortality occurs in Tanner crabs naturally parasitized by *Hematodinium* sp. when maintained in the laboratory. A similar mortality of parasitized crabs is assumed to occur in wild Tanner crab populations. Significantly higher prevalences of bitter crab disease occur in new shell crabs and crabs less than 60 mm carapace width. There is a seasonal prevalence/intensity peaking in July through October followed by a reduction or eclipse until late winter and early spring. Peak mortality occurs in August through September due to sporulation. Chronic mortality occurs in weakened hosts from secondary infections by other pathogens. Options for control of bitter crab disease in southeast Alaska include: harvest Tanner crabs from October through November during the eclipse period of the disease when crab meats may be less full but of acceptable quality (requires no culling of parasitized crabs); grossly parasitized crabs should be properly disposed of (landfill; incinerated; ground and cooked or chlorinated) rather than released; discourage culling through education programs to recognize the disease and follow proper disposal; provide an economic incentive by developing alternative use products for harvested parasitized crabs (surimi, chitin products, etc.).

VII. Human Health Significance

There are no zoonotic human health concerns regarding bitter crab disease in Tanner crabs. However, parasitized crabs have an unpalatable flavor and undesirable meat texture.

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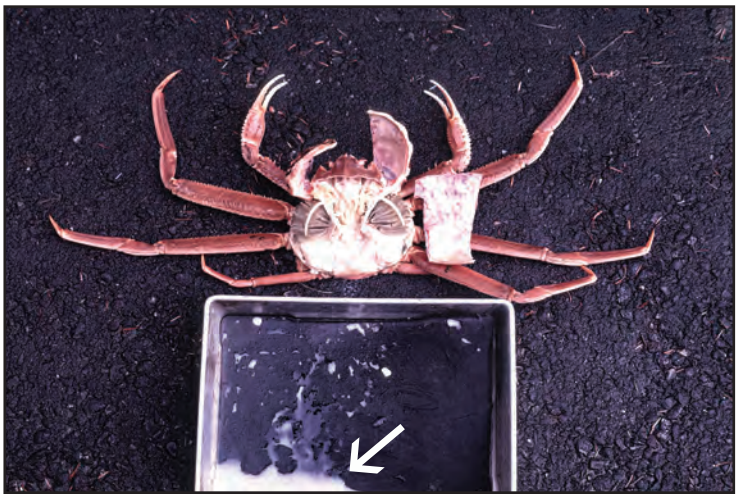
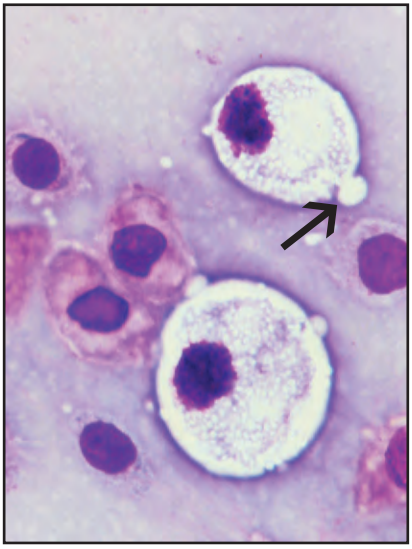
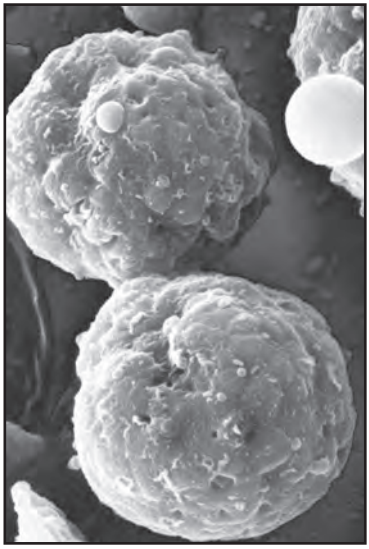
Exaggerated reddening of the crab carapace (bottom) typical of bitter crab disease



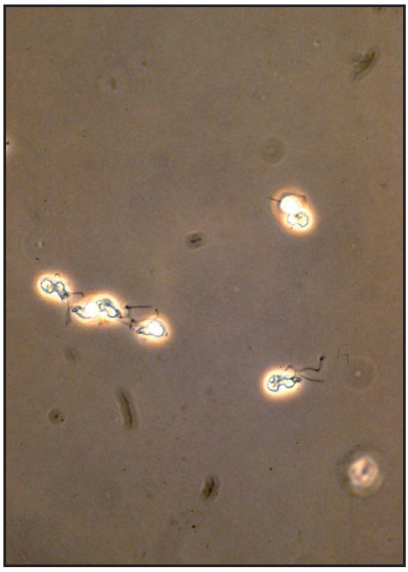
White viscera with milky hemolymph (arrow)



Vegetative stages in a hemolymph smear with irregular nuclear profile and droplets (arrow) exuding from pellicle



SEM showing droplet exuding from pellicle of vegetative stage



Twisted microspores with flagella visible in phase contrast microscopy

SEM showing the smooth surface of a microspore and two flagella

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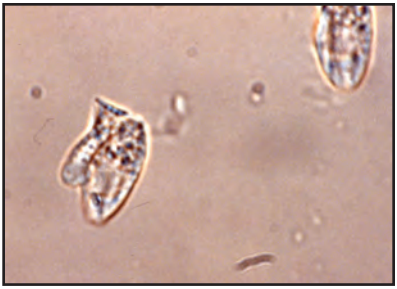
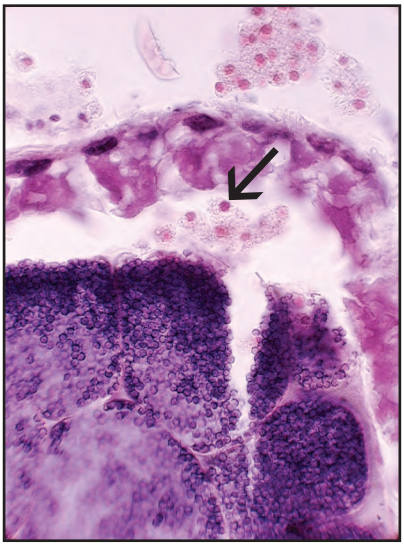
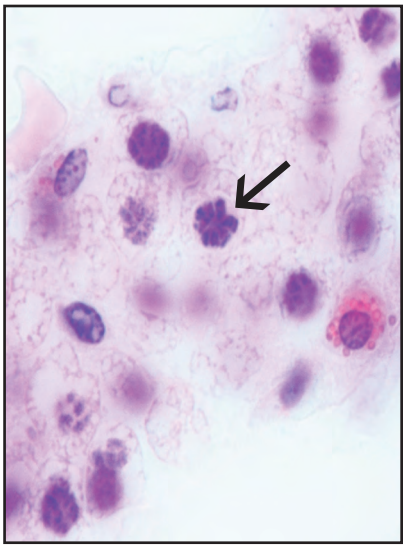
Beaked macrospore (SEM) showing warty surface and one of the two flagella



Wet mount of beaked macrospores



Histological section showing vegetative stages (arrow) inside spermatophore



Histological section showing typical dinokaryon nuclear division (arrow)

Possible *Hematodinium* Life Cycle