Perkinsosis

Overview

- Affected species of economic importance include the American oyster (*Crassostrea virginica*) and under experimental conditions the Pacific oyster (*C. gigas*), Suminoe oyster (*C.ariakensis*), gold lipped pearl oyster (*Pinctada maxima*), Sand Gaper clam (*Mya arenaria*), and Baltic tellia clam (*Macoma balthica*).
- Outbreaks occur as water temperature rise above 20°C
- Can cause serious economic losses in oyster fisheries
- No treatment
- Notifiable disease in the UK
- Large scale mortalities up to 95%
- Known to be widespread on the east coast of the USA, Puerto Rico, Cuba and Brazil

Introduction

Perkinsosis also known as "Dermo disease" is a disease of marine molluscs. Affected species of economic importance include the American oyster (*Crassostrea virginica*) where 95% mortalities have occurred. Under experimental conditions the Pacific oyster (*C. gigas*), Suminoe oyster (*C.ariakensis*) and gold lipped pearl oyster (*Pinctada maxima*) have also been infected, although they are all less susceptible. In addition, clams (*Mya arenaria*) and (*Macoma balthica*) have been experimentally infected.

Aetiological agent: A parasitic disease caused by *Perkinsus marinus*, a protistan parasite in the phylum Apicomplexa.

Geographical distribution

The disease is widespread on the east coast of the USA from Massachusetts to Florida, Gulf Coast to Venezuela, Puerto Rico, Cuba and Brazil. The parasite was also accidentally introduced into Pearl Harbour, Hawaii.

To date no mortalities associated to *P.marinus* have been reported within the UK.

Susceptible species

P. marinus affects *C.virginica*, and experimental infections have been recorded in *C. gigas, C.ariakensis, C. rhizophorae, Mya arebaria* and *Macoma balthica* although other species of *Perkinsus* infect a wider range of hosts.

Epizootiology & clinical signs

Outbreaks of Perkinsosis usually occur with warm summer water temperatures above $20^{\circ}C$

Clinical signs may include pale digestive glands, gaping, shrinking of the mantle away from the outer edge of the shell and stunted growth. In the later stages pus like

areas of soft tissue can be observed in the oyster. Many of these symptoms are similar to that of many shellfish diseases and they are not pathognomonic signs, which are specific to Perkinsosis.

The life cycle of *P.marinus* is relatively complex and the predominant stage takes place in the haemocytes (blood cells) of the oyster, where it exists as a trophozoite until the host dies, releasing large amounts of its progeny into the sea as zoosporangia in which bi-flagellated zoospores develop. The biflagellate zoospores then enter a new host via the gill, mantle or gut before entering the haemocytes and starting the cycle again. The parasite can spend long periods of time within the haemocyte increasing in numbers. Snails, crabs and fish that scavenge on dead oysters may also act as vectors



Diagnosis

Histology is the most commonly used diagnostic tool for surveillance and monitoring programmes, however when positive results or mortalities occur other presumptive diagnostic methods can be used, including electron microscopy and molecular probes. Tissue samples may also be grown in culture, but this is a non-specific test.



Treatment and control

There are no known treatments for this disease, however management techniques will aid in the control of the disease. As with all infectious diseases that can be horizontally transmitted, prevention is the best control method, and avoiding moving infected oysters into an uninfected area is the most effective approach.

This is especially significant with *Perkinsus marinus* as no eradication programme has proven possible to date, although some success has been achieved by reducing stocking density and moving oysters to a low salinity before water temperatures increases. However transferring already infected oysters to a low salinity may give rise to strains of *P. marinus* resistant to low salinity, and therefore caution is necessary when using low salinity as a method of control.

References:

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