Epizootiology of Perkinsus marinus disease of oysters in Chesapeake Bay, with emphasis on data since 1985.

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Abstract : Since 1987 *Perkinsus marinus* has been the most important path eastern oyster, *Crassostrea virginica*, in Chesapeake Bay, USA, because of i distribution and persistence in low salinity areas. The pathogen became est

oyster beds in the Chesapeake Bay as a result of natural spread during the
drought years from 1985 to 1988 or by movement of infected oysters durin
period. Elevated salinities resulting from drought conditions and concomitan
winters allowed *P. marinus* to proliferate in what were historically low salinit
Oyster mortality was high on most beds and landings of market oysters dec
low levels in both Maryland and Virginia during the late 1980s and early 199
seasonal periodicity of *P. marinus* is primarily controlled by temperature. Bo
and intensity of infections begin to increase in June as temperature increase
and overwintering infections begin to proliferate. Maximum values of preva
intensity occur in September immediately following maximal summer tempe
Infection regression occurs during winter and spring as temperature declin
minimum prevalence and intensity values in April and May. Prevalence and i
marinus infections in oysters from the James River, Virginia, over a 5 year pe
significantly correlated with temperature when temperature data were lagg
Temperature explained 39% of the variability in prevalence and 46% of the
intensity. The relationship between temperature and annual variability in *P.*
abundance is somewhat obscure, in part because of the difficulty in separat
temperature effects. Nonetheless, data from 1988 to 1994 from the James
that abnormally warm winters have a more significant impact on summer *P.*
abnormally cold winters. Salinity is the primary environmental factor that cor
distribution and intensity of *P. marinus* infections. Long-term oyster disease
along a salinity gradient in the James River revealed a statistically significant
between salinity and *P. marinus* prevalence and intensity. *P. marinus* infecti
light in intensity and no oyster mortality results if salinity is consistently less
However, infections may persist for years in low salinity areas. If summer/a
salinities range from 9 to 15 ppt some infections may progress to moderate
intensity, but oyster mortality is relatively low. If summer/autumn salinities a
greater than 15 ppt, moderate and heavy infections may be numerous and
mortality may be high. Field studies in the York River, Virginia, suggest that m
infections are acquired from July through early October, but peak infection a
occurs during late August and is correlated with oyster mortality. The early
process in oysters and the role of zoospores in transmission dynamics in na
poorly understood. No direct link between oyster defence mechanisms and
marinus infections has been established. If oyster defense mechanisms do
marinus infections, the components have not been identified. There is little
support the common perception that pollution is responsible for the dramat
P. marinus abundance since 1985. Pathogen abundance is clearly correlated
increases resulting from drought conditions in the late 1980s, although ther
subtle effects of toxicants of poor water quality on the host/parasite interac

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