

during specimen preparation for microscopy. It is highly unlikely, however, that a whole population of microbes would be washed from a histological preparation. Furthermore, enough material has been examined at three laboratories (Battelle, HSRL, and VIMS) to make it most unlikely that even a small population of highly virulent microbes would be overlooked.

Juvenile oyster mortality syndrome, including the formation of conchiolin deposits around the contracted oyster tissues, resembles "brown ring disease" of cultured Manila clams, *Tapes philippinarum*, in western Europe (Paillard et al. 1989, Paillard and Maes 1990). The disease was first reported in 1987 when it caused heavy mortalities in cultured clams. Nearly all of the moribund clams exhibited abnormal deposits of organic material on the inner shell. Neither protozoan nor metazoan parasites were detected in histological sections, but a bacterium of the genus *Vibrio* was isolated from diseased clams and caused the brown ring syndrome when injected into healthy clams (Paillard and Maes 1990). Similarly, anomalous conchiolin deposits, generally around the posterior edge of the shell margin, are associated with mortalities of the golden lip pearl oyster, *Pinctada maxima*, in Western Australia. Pass et al. (1987) suggested that *Vibrio harveyi*, isolated from affected pearl oysters, was involved in causing the disease. In both *P. maxima* and *T. philippinarum*, the conchiolin deposits differed somewhat from those of *C. virginica* in that the former were not consolidated into a distinct thin ridge, but rather were spread into a wider band with more irregular borders.

The bacterial etiology of "brown ring disease" in Europe and the fact that histopathological lesions in affected oysters are similar to those found by Dungan and Elston (1988) in association with bacterial destruction of the hinge ligament in juvenile Pacific oysters, *C. gigas*, indicates that a bacterium cannot be ruled out as the cause of juvenile oyster mortality. Whether the bacteria which we found in tissues and in chambers within the anomalous conchiolin deposits are one or several species of opportunistic bacteria or are the causative agent(s) of the mortality remains to be determined. It should be noted that deposition of conchiolin can be affected by other stressors, including handling (e.g. in bay scallops, Palmer 1980) and exposure to anthropogenic contaminants (Hillman unpubl. observations).

In conclusion, the results obtained suggest that oysters may

have been affected by a toxin-producing agent (most likely of bacterial or microalgal origin), or by a chemical contaminant which caused mantle retraction and secretion of an abnormal conchiolin layer as a defense mechanism. Death presumably occurred when the muscle became detached from the valve due to conchiolin deposition between the muscle and the shell, and/or degeneration of myoepithelial cells accompanied by bleeding. Mortalities were probably aggravated by entry of secondary invaders into lesions, and by the development of anoxic conditions (in turn aggravated by high summer temperatures) within the trays as oysters began dying. Future work should further investigate site-specificity of mortalities, the influence of rearing temperature, and age/size of affected oysters, the potential role of bacteria, and especially the association of mortalities with the occurrence of blooms of dinoflagellates or other potentially toxic phytoplankton species.

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