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Haplosporidian And Haplosporidian-Like Diseases Of Shellfish

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Introductory Remarks

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In the last two decades we have become aware that there are numerous haplosporidian and haplosporidian-like diseases of the world's shellfish. Some of these pathogens, such as *Minchinia nelsoni* (Andrews, 1966) and *Marteilia refringens* (Grizel et al., 1974), are responsible for highly destructive diseases of commercially important shellfish populations, especially oysters. Since the list of these pathogens has been growing in the last few years, it is very appropriate and timely that we hold this symposium. It is hoped that from our presentations and discussions there will be derived a better understanding of: 1) The degrees of phylogenetic and taxonomic interrelationships of the pathogens; 2) life cycles; 3) infection sources; and 4) ecological conditions influencing activity of the diseases.

If the taxonomy of the organisms is well known and based on a strong knowledge of structure then judgments can be made as to the probability that ecological or any other information gained from studying one species can be applied to another species. Obviously, the species which are more closely related phylogenetically will have biological characteristics which are more nearly similar than those species which are not so closely related. The characteristics will include more than just morphological ones. Also involved will be life cycles and response to environmental conditions. Such knowledge can be of great practical significance in efforts to control diseases or temper their impact.

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The haplosporidian first recognized to cause significant mortalities of a commercially important shellfish was *Minchinia nelsoni*. It was detected in 1957 as causing high mortalities of *Crassostrea virginica* in the Delaware Bay on the U.S. east coast. The pathogen then appeared in the Chesapeake Bay, and in those two estuaries has heavily damaged the oyster industry. Another species, *Minchinia costalis,* found in oysters along the Eastern Shore of Virginia (Delmarva Peninsula), was described in 1962. As with *M. nelsoni,* high mortalities occur. The complete life cycles are not known for either pathogen.

In 1969 it became apparent that a previously undescribed disease organism was causing oyster, *Ostrea edulis,* mortalities along the western Brittany coast of France (Alderman, 1974). Since then the disease, described as *Marteilia refringens* by Grizel et al. (1974), has spread to most Atlantic estuaries of France and into Atlantic waters of northern Spain (see Alderman, 1979; Balouet, 1979; and Grizel, 1979 in this symposium). Van

Banning (1979), in this symposium, considers the results of importing the disease into Dutch waters.

By 1972 a similar disease was recognized by Wolf (1972) to be active in eastern Australian waters where heavy mortalities of *Crassostrea commercialis* were recorded. Perkins and Wolf (1976) then described the ultrastructure of the pathogen and named it *Marteilia sydneyi,* noting its very close similarity to *M. refringens* on the other side of the world. Since *Crassostrea gigas* was imported and established in both regions, one wonders if there is any correlation between the presence of *C. gigas* and the appearance of the *Marteilia* species. Cahour (1979) describes in this symposium her search for *Marteilia* spp. in *C. gigas*. Alderman (1974) has rightly warned, as have others, against the dangers of international shipment of oysters for culturing purposes. Certainly, careful studies of the kind presented in this symposium need to be made on all populations of shellfish which are used in international shipments for culture purposes.

There will undoubtedly be disagreement over whether *Marteilia* spp. can be considered to be a haplosporidian. Possibly we can reach agreement during this symposium as to its true taxonomic position. As already stated, such considerations have significance beyond the specialized field of taxonomy. We need to know degrees of interrelatedness if informed decisions are to be made in interpreting available data and designing future experiments and surveys.

Of less direct importance to the health of commercially significant shellfish, but of commercial significance, are the hyperparasites of parasitic worms found in shellfish. When sporulation of the haplosporidians occurs, the worms become black and, therefore, are highly visible in the shellfish thus making the meat less marketable. For example, *Urosporidium crescens* parasitizes the metacercariae of *Carneophallus* sp. and causes them to become black in the tissues of *Callinectes sapidus,* the blue crab of eastern U.S. estuaries. This results in the condition known as "pepper crab disease" since the encysted trematode worms resemble peppercorns (Perkins, 1971). Likewise, the immature anisakid nematode worms found in the surf clam, *Spisula solidissima,* on the eastern U.S. continental shelf become black when the hyperparasite, *Urosporidium spisuli,* sporulates. The resulting highly visible worms cause serious marketing problems (Perkins et al., 1975).

Despite numerous studies of the Haplosporidea, life cycles of none of the species are known, nor has transmission of infections been accomplished under laboratory conditions. Transmission has been accomplished in the field by importing susceptible individuals into an endemic area, but the origins of the infective cell elements are not known despite excellent and extensive epizootiological studies such as those of Andrews (1966), Andrews and Frierman (1974), and Haskin et al. (1965). Likewise, similar difficulties are being experienced by Alderman, Balouet, Grizel, and van Banning[1] in trying to understand the life cycle of *Marteilia refringens*. Obviously, if we are to advance our knowledge of the ecology of the diseases and reach the point of being able to make suggestions to shellfish industries as to what control measures can be utilized, we must answer the very basic questions of how transmissions of infections occur, what are the life cycles of the pathogens, and where are the reservoirs of infective cells? If any of these questions could be answered for any of the haplosporidians, whether the species is commercially important or not, possibly the resultant information will be of significance toward understanding other species. Therein lies the greatest potential for success of this symposium beyond the excellent specialized information which will undoubtedly be contributed on each species.

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