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Minchinia nelsoni (MSX) Disease of the American Oyster

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It has been almost 20 years since the Multinucleate Sphere X (MSX) (Fig. 1) was first recognized as the etiologic agent responsible for the sudden loss of oysters in Delaware Bay (Haskin, Canzonier, and Myhre, 1965). The first mortalities of oysters were in 1957; by the spring of 1958, massive mortalities had occurred. Losses were reported as high as 85 percent in some areas (Haskin et al., 1965). The devastation wrought by the disease was well illustrated by Sindermann and Rosenfield (1967). They indicated that oyster production in Delaware Bay prior to the epizootic was about 7.5 million pounds of shucked oyster meats. By 1960, oyster production for that area was below 100,000 pounds.

Many state, university, and Federal agencies cooperated in the search for answers to the problem. MSX was eventually identified and named *Minchinia nelsoni* (Haskin, Stauber, and Mackin, 1966). The definitive spore stage (Fig. 2) was identified and described by Couch, Farley, and Rosenfield (1966). Barrow and Taylor (1966) showed that plasmodial and spore stages shared specific antigenic properties. Ultrastructural details were described by Perkins (1968) and Rosenfield, Buchanan, and Chapman (1969). Farley (1967, 1968) proposed a life cycle for the parasite and described the disease syndrome caused by the parasite.

The disease did not remain a problem restricted to Delaware Bay. By 1959, MSX had reached the waters of Maryland and Virginia with a similar devastating impact (Andrews, 1966; Rosenfield and Sindermann, 1966; Sieling, Otto, and Rosenfield, 1969). The epizootiology of the disease in each of these areas has been thoroughly studied and reported (Haskin et al., 1965; Andrews, 1966, 1968; Rosenfield and Sindermann, 1966; Andrews and Wood, 1967; Couch and Rosenfield, 1968; Ford, 1973; Farley, 1975).

The oyster industry has made a comeback in most of the areas affected. By 1972, the disease had virtually disappeared in the Maryland portion of Chesapeake Bay, indicating a decrease in parasite pressure (Otto, Hamed, and Rosenfield, in press). In all of the areas involved, disease resistant populations have developed from the survivors of the initial epizootic (Haskin, 1974; Farley, 1967, 1975; Andrews, 1968; Haskin and Canzonier, 1969; Myhre and Haskin, 1970; Ford, 1973).

Myhre and Haskin (1970), Farley (1975), and Douglass and Haskin (In press, a) reported that the resistant oysters became infected as readily as did susceptible strains, but that resistant stocks were able to survive with the parasite and that some eventually overcome the disease. Susceptible oysters were unable to cope with the parasites and eventually died. Since Maryland oysters are no longer exposed to high parasite pressures, resistant populations could be reduced by breeding with unexposed oyster stocks, possibly rendering them more susceptible to a reoccurrence of the disease.

Farley (1968) described a hemocytic response to the parasites in susceptible oysters. There is little doubt that there is a response in susceptible oysters. The normal vesicular connective tissue is almost completely displaced by the cellular response (Fig. 3). The initial response is of the hyaline hemocyte cell type (Farley, 1967) (Fig. 4). Douglass and Haskin (In press, b) quantitatively measured the changes in the hemocyte populations due to *M. nelsoni* and found them to be from 80 to 140 percent higher than in uninfected oysters. Farley (1975) compared the hyaline hemocyte response in oysters to the T cell response in vertebrates and suggested a surveillance function for foreignness.

In resistant oysters, the infection usually involves only the epithelial cells of the gills. When the parasite does cross basement membranes and becomes established in the host, there is little host response. In later infections, the parasites appear to be moribund, some phagocytosis occurs, and the infections may eventually disappear (Farley, 1967).

The nature of the resistance mechanisms oysters have toward *M. nelsoni* is unknown. There is little doubt that resistance is involved. I have examined populations of oysters with *M. nelsoni* prevalence greater than 60 percent; however, mortalities are not comparable to those of the original epizootic. The humoral response suggested by Farley (1975) and Douglass and Haskin (In press, b) has yet to be demonstrated. Acton and Evans (1968) demonstrated enhanced nonspecific clearance of bacteriophages from the oyster. The mechanism of this enhanced memory is still in question. Tripp (1975) reviewed studies of humoral factors in molluscan immunity and indicated that there are many questions yet to be answered.

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Figure 1.—Multinucleate spherical ("X") plasmodial stage of *Minchinia nelsoni* (arrow). 1,000×.

Figure 2.—Acid-fast spores of *M. nelsoni* in tubule of digestive diverticula. Arrow indicates operculate spore (1,000×).

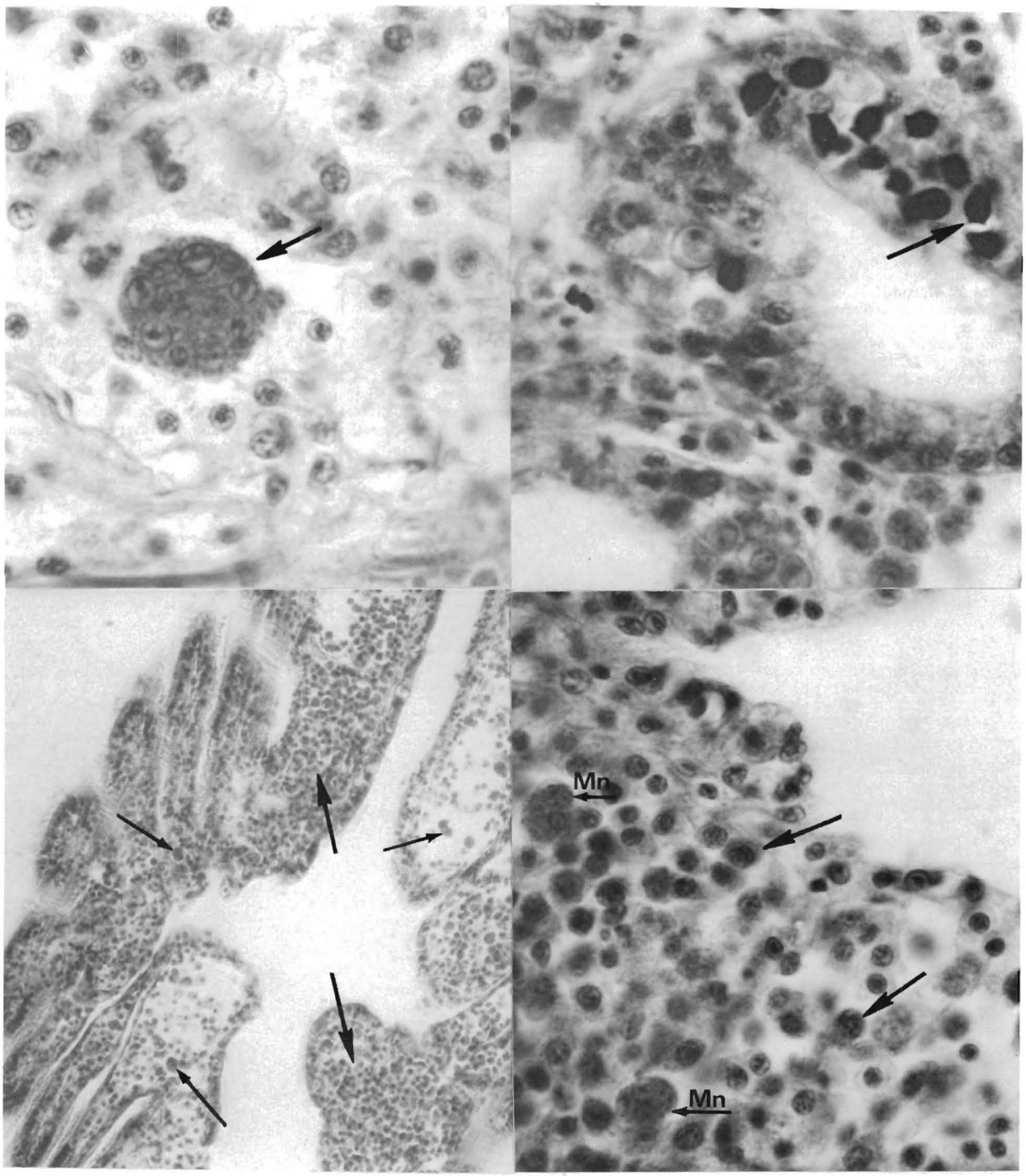

Figure 3.—Gill filament infected with *M. nelsoni* (small arrows) showing massive infiltration of hemocytes (large arrows). 200×.

Figure 4.—Higher magnification of gill lesion showing hyaline hemocyte (arrows) in response to *M. nelsoni* (Mn) infection. 1,000×.

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