Development of Resistance to *Minchinia nelsoni* (MSX) Mortality in Laboratory-Reared and Native Oyster Stocks in Delaware Bay

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Introduction

In the spring of 1957, devastating mortalities of oysters occurred in lower Delaware Bay. In 1958 and 1959, the mortalities were repeated, but this time over a much wider area of the estuary. During this period, cumulative kill reached 90-95 percent in the high-salinity planting areas and 50-70 percent on seed beds in lower-salinity regions (Fig. 1). Only in very low-salinity areas, such as the tidal rivers and creeks and on the uppermost seed beds, did oysters escape the mortality (Haskin et al., 1965). In 1959, similarly destructive kills occurred in lower Chesapeake Bay (Andrews and Wood, 1967). The disease-causing organism, a haplosporidan parasite, was identified and named *Minchinia nelsoni* (more commonly known as MSX) by Haskin et al. (1966).

Since the original outbreak of *M. nelsoni,* the Oyster Research Laboratory at Rutgers University has pursued many lines of investigation into the nature of the MSX problem. All attempts to transmit the disease under controlled laboratory conditions have failed, so these studies have been primarily field investigations concerned with various epizootiological aspects of the disease. This paper describes one part of the overall investigation: The development of resistance to MSX-caused mortality in both laboratory-reared and native oyster populations in Delaware Bay.

The Delaware Bay mortalities were reminiscent of those occurring in Malpeque Bay, Prince Edward Island, Canada, in 1915. During the epizootic, more than 90 percent of the oysters in Malpeque Bay were killed (Needler and Logie, 1947). Although an etiological agent has never been positively iden-

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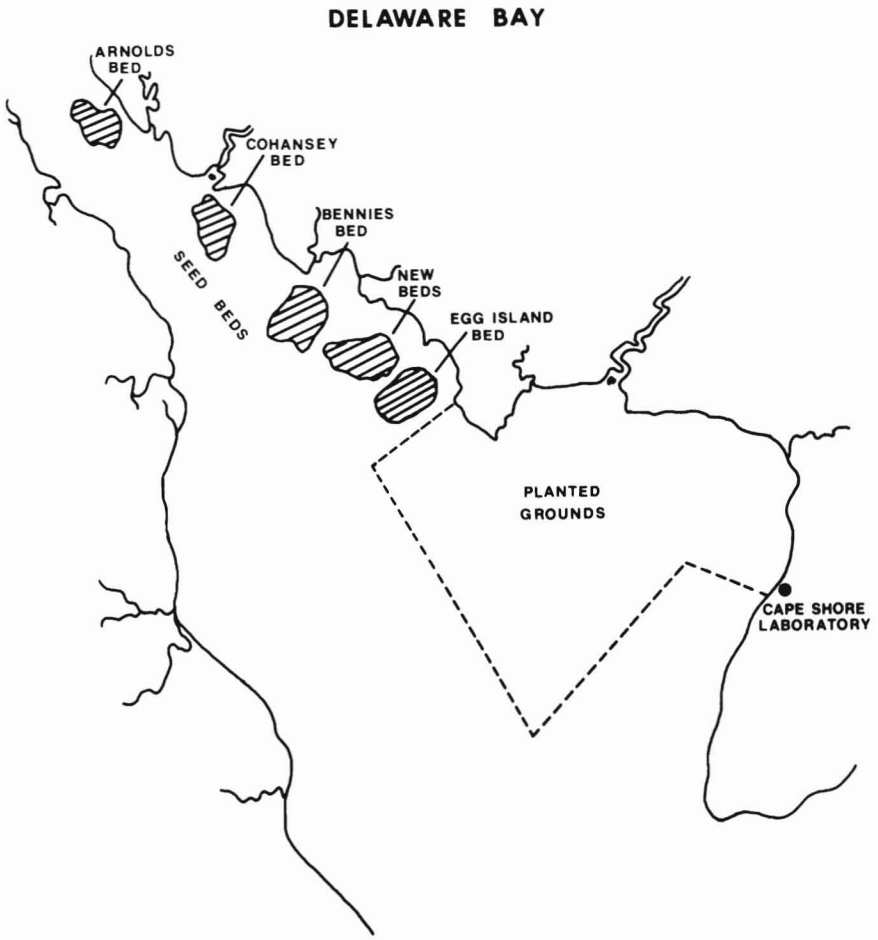


Figure 1.—Delaware Bay showing locations of New Jersey's natural seed oyster beds and planted (leased) grounds.

tified, a highly contagious pathogen is suspected (Frazer, 1937-38). Needler and Logie (1947), studying Malpeque disease in the 1930's, reported the first, and to date only, instance of a population of marine invertebrates acquiring resistance to a disease. Fifteen years after the initial kill, these investigators found that native Malpeque Bay oysters, offspring of survivors of the 1915 epizootic, were surviving normally; in contrast, oysters imported into the bay from areas which had not experienced kill suffered heavy mortalities.

In a pattern similar to the Malpeque experience, and during the years immediately following the Delaware Bay epizootic, several lines of evidence suggested that our native oysters were developing resistance to MSX: 1) From 1958 through 1960, successive year classes of native spat on the flats in front of our lower Delaware Bay Cape Shore Laboratory, all subjected to heavy disease pressure, showed increasing survival with each year class. During its first full year of exposure 1957 set had 84 percent kill, 1958 set had 48 percent kill, and 1959 set had 29 percent kill (Haskin[1]); 2) Beginning in 1961, seed oysters transplanted onto the leased grounds showed improved survival; 3) In addition, native Delaware Bay stocks from seed beds, planted grounds, and Cape Shore, when tested for survival at Cape Shore, had considerably lower mortalities than did oysters imported from MSX-free east coast locations (Haskin[2]).

In later histopathological studies, lower mortalities were correlated with lighter, more localized infections (Myhre and Haskin, 1969; Ford, 1970), suggesting that a defense mechanism involved the containment of the parasite in small, local, nonlethal lesions.

Lowered mortalities, nonetheless, were the earliest and certainly the most substantial evidence that Delaware Bay oysters had developed resistance to MSX. At this point, however, in the early 1960's, we were not able to demonstrate that lowered kill in native stocks was due to an inherited trait rather than to selection within the generation of oysters under test. Also, we had no means of sorting out the influence of fluctuating disease activity on mortality rates from that due to selection. To resolve these questions we began laboratory rearing of stocks of oysters with various histories of exposure to MSX. The test of their offspring for resistance to MSX began with first exposure to the disease. Our first oysters were spawned in 1961, and since 1964 numerous stocks, with known parentage and precise selection history, have been reared and tested on a routine basis each year.

A second method of investigating development of resistance to MSX has been to examine data from the large-scale monitoring of many groups of native oysters throughout Delaware Bay for trends in mortality and infection levels over the course of the past 20 years.

In this paper we present evidence that resistance to MSX-caused mortality is heritable; that it can be expressed to a high degree in laboratory-reared oysters with rigorously selected parents; and that it has developed in native populations under natural selection in Delaware Bay, although to a lesser measure than is possible using the laboratory spawning and experimental selection procedures.

Methods

Laboratory-Reared Stocks

Oysters to be spawned, other than imported susceptible stocks, are partially conditioned in trays on the tidal flats at our Cape Shore Laboratory. Groups of about 30 oysters are finally conditioned within the laboratory. Spawnings involved groups of males and females, usually from 4 to 10 each. Larvae are reared using a modified Milford technique (Hidu et al., 1969) and are fed daily, with each water change, on natural phytoplankton in Delaware Bay water. All bay water used in the larval culture is passed through 60μm plankton netting to prevent contamination by wild larvae. Spat (recently set oysters) are placed in trays and held in Cape May harbor, usually until October when the setting season for native oysters in Delaware Bay has passed. Then for each stock, duplicate trays are set up, each containing from 2,000 to 5,000 spat. Between March and November these trays are held on the flats in front of the laboratory where, being exposed on most low tides, they can be examined frequently. During periods of heavy mortality, this may be on a daily basis. In November, trays are moved from the flats to deeper water to prevent possible ice damage. They have generally been stored in a tidal creek behind the laboratory or, more recently, in Cape May harbor. Predation is normally quite low because of: 1) Frequent handling of the oysters; 2) low tide exposure; 3) the fact that the trays are raised several inches above the bottom on drainage tiles. Obvious predator mortality is tallied and subsequently excluded from disease mortality calculations.

Mortality counts are cumulated seasonally and annually and groups with an ancestral history of selection by MSX (called resistant or selected stocks) are compared with each other and with offspring of oysters with no history of selection by MSX (called susceptible or unselected stocks). Mortalities in paired trays of each stock rarely differ by more than a few percentage points and when a large difference does appear, it can usually be related to some non-MSX stress, such as mudding. In these cases, mortalities are not used in the calculations.

In assessing survival rates for laboratory-reared oysters, a standard exposure period was established at the start of the larval rearing program. This interval, which begins in October of their first year and ends in June as they complete their third year, spans a 33-month period. It was chosen because mortality rates in early studies declined sharply after the second summer-fall exposure period, indicating that most susceptibles had been weeded out and also that oysters living on the Cape Shore flats

[1]Haskin, H. H. 1961. Delaware Bay oyster mortality project. Unpublished report to U.S. Fish and Wildlife Service for period 1 Jan. - 30 June 1961.

[2]Haskin, H. H. 1960. Delaware Bay oyster mortality project. Unpublished report to U.S. Fish and Wildlife Service for period 1 July 1959 - 30 June 1960.

for this length of time begin to experience kill caused by another oyster parasite, *Labyrinthomyxa marina,* which greatly complicates interpretation of the mortalities.

From 1964 through 1977, a total of 31 resistant stocks were spawned and their offspring were subjected to the complete test exposure. Ten of these were first generation resistants, 14 were second, 5 were third, and 2 were fourth generation resistant groups. Oysters are not used as spawners unless they have survived the standard 33-month exposure to MSX. There is, therefore, a minimum of 3 years of selection for MSX resistance between generations. This means, for instance, that the total exposure time to intense MSX activity and selective mortality of the ancestors of the F_4 generation is at least 12 years. In addition to breeding oysters selected for resistance to MSX mortality, we have, each year, spawned unselected oysters imported from various locations along the east coast where there is little or no MSX activity. To date, 24 such groups have been tested. In most years, susceptible imports have come from Long Island Sound, the Navesink River in New Jersey, and the James River in Virginia. Mortalities in these stocks serve as controls, providing a base line against which survival of selected groups can be judged.

Monitoring Program

In New Jersey, the Delaware Bay oyster industry transplants oysters from public, upper bay, natural setting areas (seed beds) to individually leased growing grounds in the lower bay (Fig. 1). Shortly after the first epizootic, a program was established to monitor numerous bed populations throughout the bay for MSX prevalence and for mortality. Now in its 20th year, this monitoring program has provided detailed statistics from 5 major seed beds, and for 78 different plantings of oysters on the leased grounds. Each lower bay planting has been sampled on a regular basis for 1-5 years. On the selected seed beds, sampling has been continuous for up to 20 years. Until 1971, samples were collected on a monthly basis, weather permitting. Since then, a schedule of 7-8 sampling periods per year, designed to coincide with critical phases of the MSX cycle, has been in effect.

At each station a 1-bushel sample of oysters, gapers[3], and boxes[4], is collected with a 30-inch oyster dredge. A "recent" mortality count, based on the number of gapers and new boxes (those with little or no fouling) is made. The interval during which this mortality has occurred is estimated using knowledge of recent fouling rates in the bay. This mortality interval may be as short as 2 weeks during the summer or as long as 3 months in the winter. Interval mortalities are then cumulated to provide seasonal and annual totals. Mortality due to predation by oyster drills and mud crabs, dredge damage, mudding, etc. is distinguished from disease kill which includes that associated with MSX. All mortalities considered in this discussion, except where specified, refer to the second category only. Both predation and disease mortalities are computed separately as a function of the total sample. It is likely that predators kill some oysters which would otherwise die with MSX. Thus the disease mortalities reported here are lower than if the oysters were protected from predation as they are to some extent in the trays. In nearly all cases, a 20-oyster sample has been fixed for histological study. Most of these have been worked up so that indices of infection prevalence and intensity accompany the mortality statistics. These data will be presented in forthcoming papers.

Histological and mortality data from both monitoring program and experimental tray stocks have established that the major infective period for MSX in Delaware Bay is in June, with a second period of variable, and generally lesser, activity in late summer and early fall (Haskin et al., 1965; Ford, 1970). Andrews (1966) found a similar pattern for MSX in Virginia. Oysters first exposed in June usually begin dying in late July or early August. Mortalities extend into November, tapering off as water temperatures drop. Additional kill is recorded in late winter and early spring and is associated with cold weather stresses as well as with MSX. A third mortality period occurs in June and July and is thought to be primarily the result of infections acquired late the previous year which remained subpatent over the winter, then proliferated with warming temperatures (Andrews, 1966; Ford, 1970).

[3]"Gapers" are dead or dying oysters with meat still in the shell.

[4]"Boxes" are dead oysters which no longer contain meat, but with valves still attached at the hinges.

Results

Laboratory-Reared Stocks

As of summer 1977, a total of 55 lots of laboratory-reared oysters had been carried through a 33-month testing period, and cumulative mortalities for all have been calculated. As indicated earlier, these lots have been grouped into susceptibles (24 lots) and first, second, and third generation resistants with 10, 14, and 5 lots, respectively. For each of these 55 lots, cumulative mortalities by season have been calculated and then these mortalities averaged for each group. The results are shown in Figure 2. Also included are the results for Cape Shore natives (nine year classes).

The patterns of mortality are quite clear in this summarizing figure. Newly set spat first exposed to MSX in the fall may acquire infections then. Some differential kill results the following spring, but mortalities for all groups are closely clustered through June. It is not until the oysters are exposed to a complete MSX infective period beginning in June that large mortalities are seen. By November, as death rates decline with the onset of cold weather, a clear pattern of differential total kill has been established, and this is maintained during the rest of the test period. Mean mortalities at the end of this initial kill have reached 73 percent in the susceptibles and 60 percent in Cape Shore natives. Resistant laboratory-reared oysters have distinctly less kill, at 37-42 percent, but show little difference among generations.

At the end of the test period mean

mortality for the susceptible stocks has climbed to 93 percent, while that for Cape Shore natives stands at 81 percent. Resistant groups show decreasing mortalities, from 68 percent in the first generation, to 64 percent in the second, and 56 percent in the third (Table 1).

Two fourth generation resistant stocks have also been tested, but their final mortalities (38 percent and 95 percent) were so disparate that they were not included in the figure. Additional fourth generation stocks are currently undergoing testing and may provide the data needed to establish more definite mortality rates for this generation.

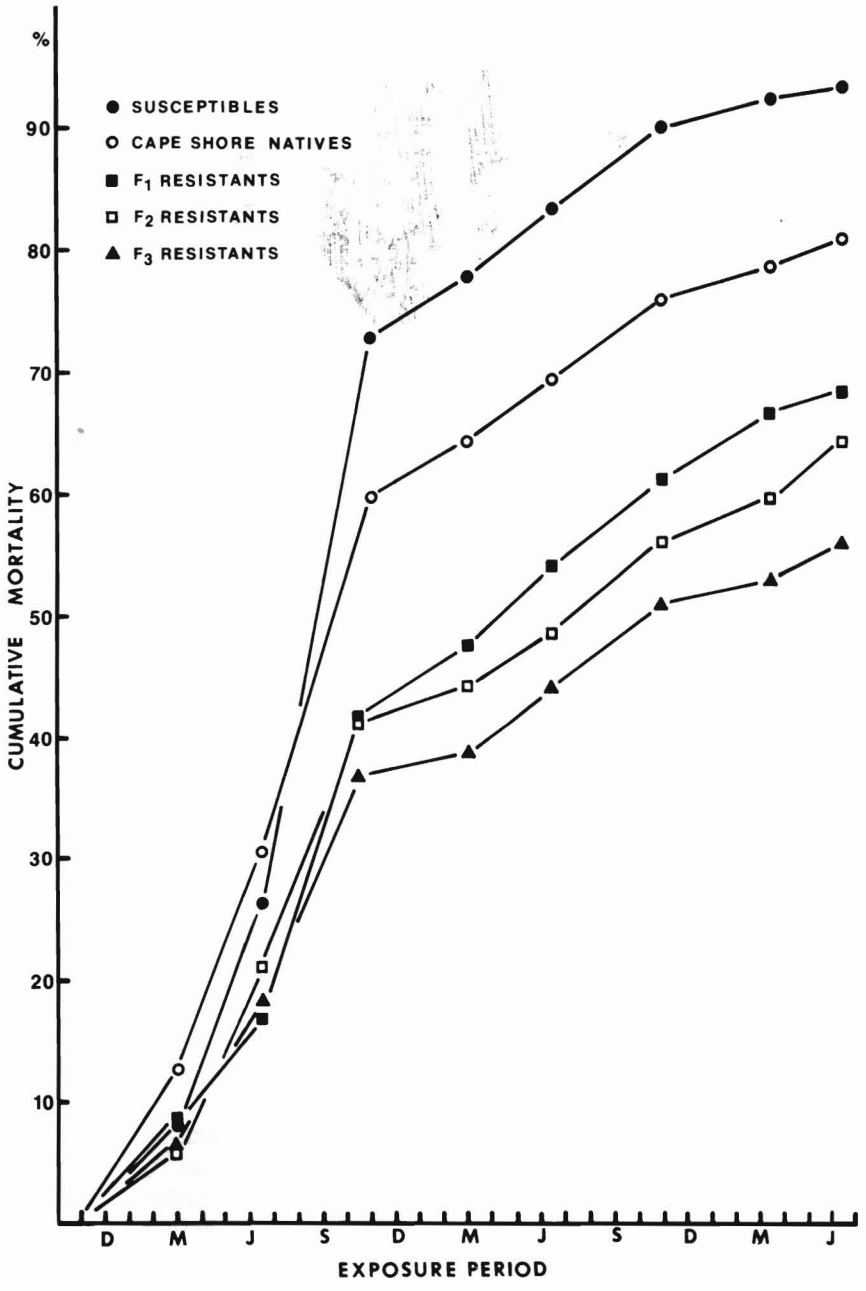
It is convenient to consider survival rather than mortality as a measure of resistance to kill in oyster populations. Using survival of susceptibles as a base line (representing survival of unselected oysters in epizootic situations), we have calculated survival ratios for the various groups of selected oysters. This is simply the ratio of the percent survival of selected stocks to the 7 percent survival of susceptibles at the end of the test exposure (Table 1). After nearly 3 years of intensive selection against MSX in each generation, first, second, and third generation resistant stocks have 4.6, 5.1, and 6.3 times as many survivors, respectively, as do unselected groups. The first selection, that operating on susceptible stocks before they produce the first generation resistants, raises the survival ratio more than do the next two selections combined. Native Cape Shore oysters have nearly three times as many survivors as do susceptible imports.

Table 1.—Survival ratios for Cape Shore native and laboratory-reared oyster stock after 33-months exposure to MSX at the Cape Shore. The ratio of survivors has been calculated by comparing the percentage survival of each selected group with the 7 percent survival of susceptibles, both at the end of the last exposure.

Oyster stock	Number of groups	Percent Cumulative Mortality	Ratio of survivors
Susceptibles	24	93	1
Cape Shore natives	9	81	2.7
First generation resistants	10	68	4.6
Second generation resistants	14	64	5.1
Third generation resistants	5	56	6.3

After the first large selective kill, cumulative mortality curves display distinctly similar slopes (Fig. 2). This suggested to us that mortality for all stocks might be approaching a common rate. This would imply that differential selective mortality had ceased and that virtually all of the oysters susceptible to MSX kill had been weeded out of the population. To explore this possibility, mean seasonal mortality rates have been examined (Fig. 3). While there is a clear lessening of mortality differences among stocks as exposure and

Figure 2.—Cumulative mortality means for oyster stocks exposed to MSX in experimental trays on the Cape Shore tidal flats. The 33-month exposure period is shown on the abscissa.



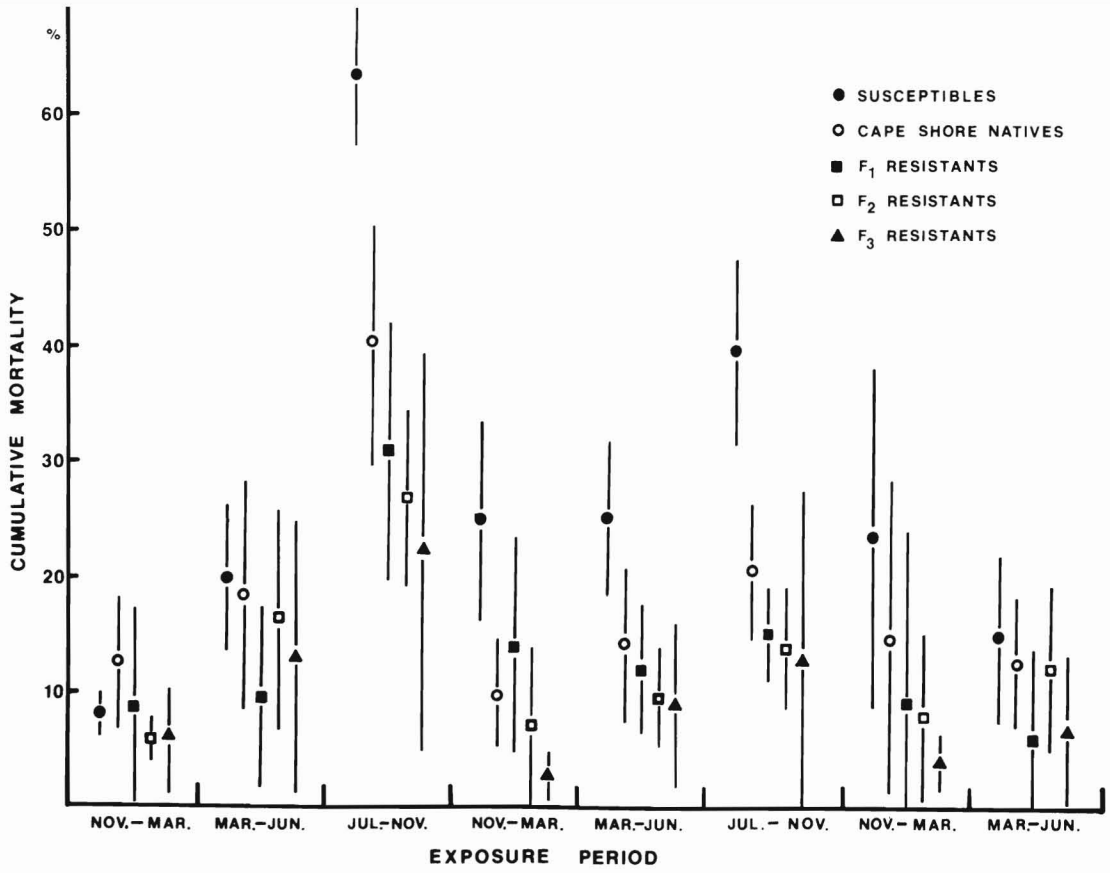


Figure 3.—Seasonal mortality means for oyster stocks exposed to MSX in experimental trays on the Cape Shore tidal flats. Successive seasons during the 33-month exposure period are shown on the abscissa. Analysis of variance was used to estimate variability due to year of exposure and to stock differences, and this in turn was used to construct 95 percent confidence intervals about each mean.

selection progress over the test period, there is, at the same time, a consistent pattern of highest kill in the susceptibles, followed by Cape Shore natives and then by laboratory-reared resistants throughout the entire 33 months. For the most part, the same pattern is followed among resistant groups, with mean seasonal mortalities progressively decreasing with increasing generation number.

Native Seed

The general mortality pattern for planted oysters in lower Delaware Bay is similar to that for experimental stocks tested at the Cape Shore Laboratory. Mortalities for monitored leased-ground plantings have been calculated by season and year of exposure to MSX on the planted grounds. These have been averaged and then cumulated over a 3-year exposure period (Fig. 4). Differences from the experimental stocks at the Cape Shore Laboratory should be noted: 1) Seed oysters, which have little or no MSX, are transplanted in late May and early June so their initial lower bay exposure to the disease includes a complete summer infective period. This contrasts with the laboratory-reared groups which have undergone some selection from late fall infections before they experience a full June infection period; 2) Seasonal mortality intervals for Cape Shore stocks span somewhat different periods than do those calculated for planted oysters. However, they include essentially the same critical mortality periods (i.e., late summer-fall, winter-spring, and early summer) so that comparisons may be made without difficulty; 3) The major mortality on the planted grounds occurs in late winter and early spring, a period during which oysters die from a combination of overwintering stress as well as from MSX. In contrast, the greatest seasonal mortality in experimental stocks occurs in late summer and fall, when virtually all kill has been associated with MSX (Haskin et al., 1965).

Cumulative mortalities for all planted oysters sampled between 1960 and 1977 average 36 percent after 1 year (Fig. 4). After a second and third year of exposure, average total mortalities have risen to 50 percent and 56 percent, respectively. Neither seasonal mortality levels themselves, nor their decrease after exposure and selection are as great as for experimental stocks.

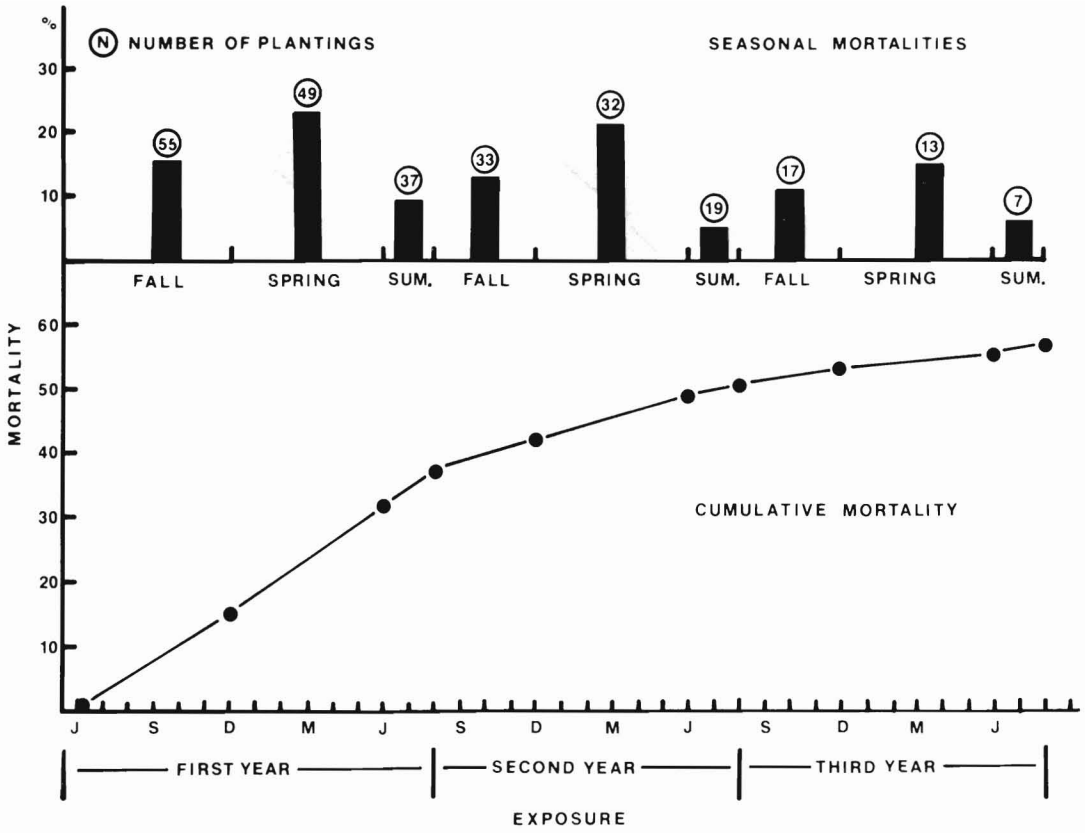


Figure 4.—Seasonal and cumulative mortality means for native seed on the planted grounds over the period 1960-77. Mortalities for all grounds sampled during a particular season and year of exposure to MSX have been averaged and are shown in the upper graph along with the number of plantings involved in each calculation. These seasonal mortalities have been cumulated over a 3-year period and are shown in the lower graph.

Calculating averages from pooled data in this situation has certain disadvantages. Mortalities are a great deal more variable among planted grounds than among experimental stocks with a common selection history under test at the Cape Shore Laboratory. This variability is due to fluctuations in MSX activity from year to year on the planted grounds, to variable disease pressure within the planted ground area, and to stress from harvest dredging (Haskin[5]). Averaging is a useful tool for demonstrating general patterns, but it tends to mask extremes which add to a more complete understanding of the data.

Four plantings of seed oysters on the leased grounds have been chosen to illustrate mortality extremes and also some more typical mortality levels, recorded after the 1957-59 epizootic. None of these groups was harvested during the sampling period. These are compared with experimental tray stocks in Figure 5. For comparison with planted grounds, Cape Shore tray mortalities have been calculated beginning in July of their first summer's exposure, disregarding kill which has taken place since the previous October, most of which is not associated with MSX. Highest kill for any group on the planted grounds since the original epizootic was suffered by James River seed oysters imported and planted experimentally in 1964. At 84 percent after 2 years, this value falls midway between laboratory-spawned susceptibles (92 percent) and native Cape Shore set (73 percent). For native seed, highest kill was recorded for a group of 1972 plants. Their 65 percent 2-year mortality equalled that for first generation laboratory-reared resistants (62 percent) and represents a doubling of survival over James River imports, despite the fact that these 1972 plants were experiencing the heaviest disease activity on record since the first epizootic (Haskin[6]). An example of a ground with relatively low mortality was one planted in 1974 which lost 39 percent over 2 years. More typical for oyster

[5]Haskin, H. H. 1972. Disease resistant oyster program - Delaware Bay 1965-1972. Unpublished report to National Marine Fisheries Service.

[6]Haskin, H. H. 1975. Control of disease in oyster populations of Delaware Bay. Unpublished report to National Marine Fisheries Service for period 1 June 1973 - 31 May 1974.

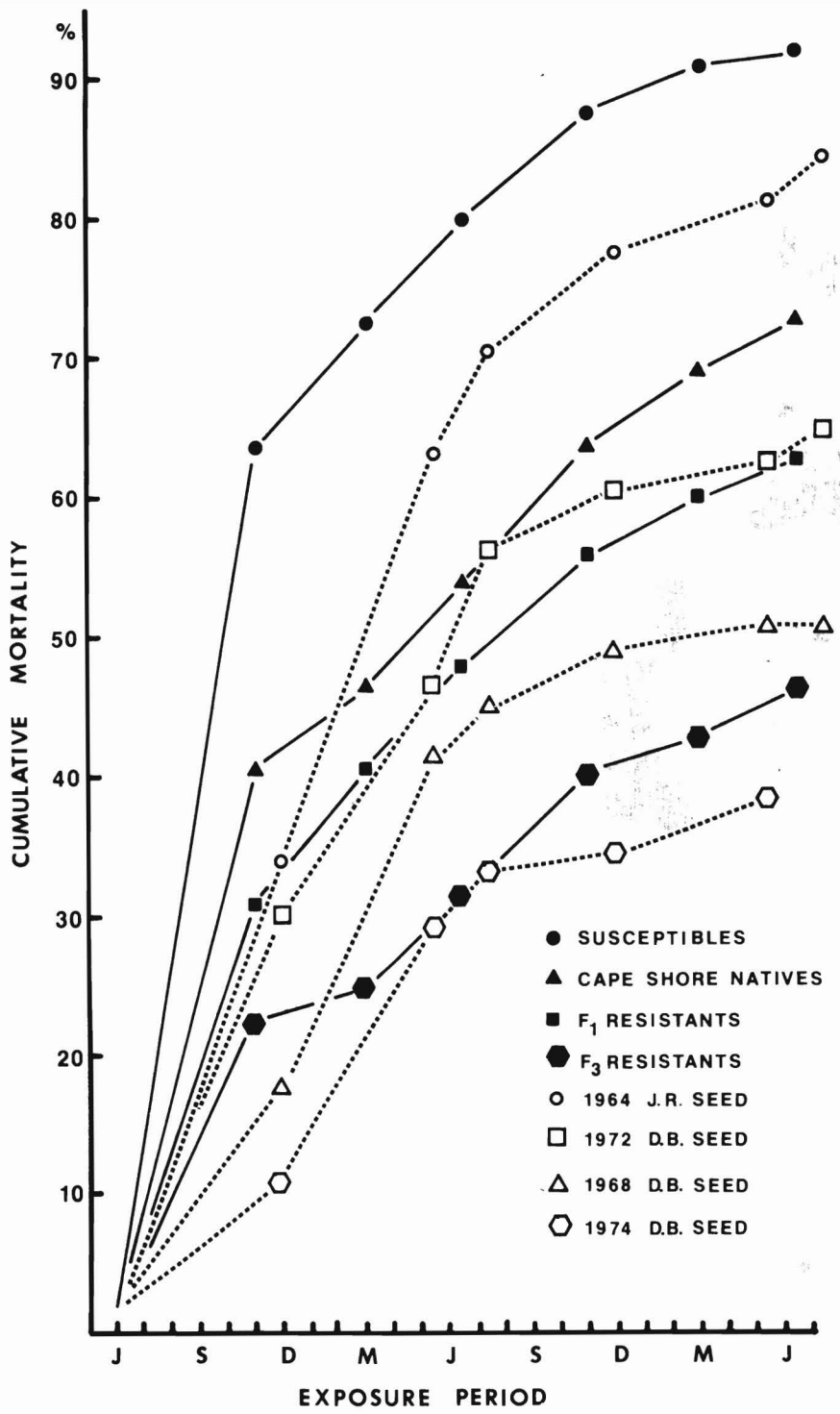


Figure 5.—Comparison of cumulative mortality means for experimental stocks tested at the Cape Shore with Delaware Bay native seed on the planted grounds. Mortalities have been calculated for a 2-year period following exposure to initial June infective period. J. R. and D. B. indicate James River and Delaware Bay seed, respectively.

plantings is the 51 percent loss shown by a ground planted in 1968. This value falls close to the 47 percent for experimental third generation resistants at Cape Shore.

When Delaware Bay seed bed oysters are tested at Cape Shore with experimental tray stocks, a different picture is seen (Fig. 6). Since 1966, 15 native groups have been thus tested. Eight have come from upper bay seed beds in the vicinity of Arnolds bed (Fig. 1), and they have shown a mean 2-year mortality of 75 percent. Nine groups spanning the distance between Egg Island and Cohansey Beds have had an average kill of 70 percent after 2 years. These figures are almost identical to the 72 percent for Cape Shore natives, and all fall between the 92 percent for laboratory-reared susceptibles and the 62 percent for first generation resistants.

When oysters in lower Delaware Bay experienced their first MSX kill, losses were as high as 85 percent during a 6-week period in the spring of 1957. During the 2 years after the initial outbreak, total cumulative mortalities reached 90-95 percent over most of the planted area, a level which compares well with the 92 percent 2-year loss for laboratory-reared susceptibles whose parents have been imported annually from areas with little or no MSX pressure, and are thus comparable with Delaware Bay natives before the epizootic in 1957.

Since the early and mid-1960's, when lowered mortalities of native stocks became obvious, no further decline in mortalities has been evidenced. Kill has tended, instead, to vary with fluctuations in MSX activity. But even when disease pressure has been extremely heavy, as it has been since 1972, losses have never equalled those of the late 1950's. As indicated above, maximum 2-year kill for native seed since the 1957-59 epizootic was 65 percent on a ground planted in 1972 (Fig. 5). While histological data for the initial epizootic are scarce, all available evidence indicates that the MSX pressure experiencd by the 1972 plants was equal to that of the first kill, as measured by numbers of oysters infected,

yet 4-7 times as many oysters survived as during the early epizootic.

Discussion

Laboratory-reared stocks, whose pattern of kill upon exposure to MSX is one in which each succeeding generation has less mortality than did its parents, provide convincing evidence that resistance to MSX is, indeed, heritable. Offspring display better survival because susceptible individuals in their parents' generation die from MSX before that generation spawns the next. As each generation is exposed to the disease, its gene pool is altered by "weeding out" of susceptibles, and it is this new genetic make-up with a higher proportion of resistant genes, which is passed into the next generation.

Disease pressure at Cape Shore, where experimental oysters are exposed to MSX, is very high. Infection prevalences commonly reach 100 percent prior to heavy mortalities[7]. Any oyster which survives nearly 3 years exposed to this level of disease activity should be extremely resistant. In fact, it seems reasonable to believe that all oysters which survive such pressure should be almost equally resistant. It is puzzling, therefore, to find that differential kill continues after oysters have been exposed to, and selected by, MSX for nearly 3 years. Stocks which are most susceptible at the start of the test period continue to show highest mortality rates at its conclusion.

We propose the following explanation for our findings. A population of oysters which has never been exposed to MSX contains a random distribution of those genes which will determine its capacity to deal with the disease. These genes present a continuum of abilities ranging from total incapability in highly susceptible individuals, through stages of increasing ability to control infections, to some highly resistant oysters well-equipped to deal with intense MSX pressure.

Upon first exposure to MSX, the highly susceptible individuals, which comprise most of the population, die.

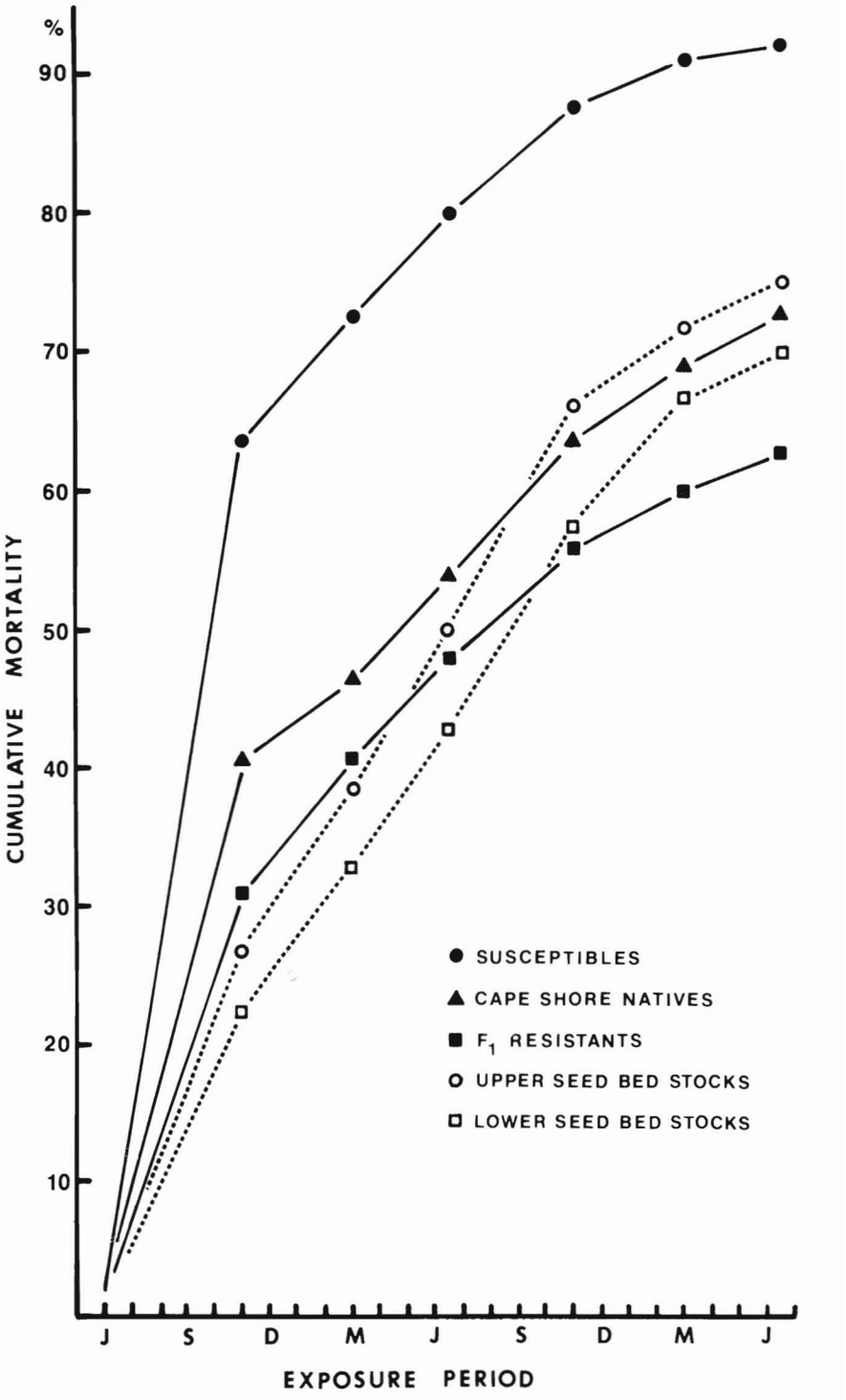


Figure 6.—Comparison of cumulative mortality means for experimental stocks and Delaware Bay native seed, both tested at the Cape Shore. Mortalities have been calculated for a 2-year period following exposure to initial June infective period.

[7]Unpublished data. New Jersey Oyster Research Laboratory, P.O. Box 1059, Piscataway, N.J.

Some survivors of the initial kill also contain susceptible characteristics, but these are masked to varying degrees by resistant ones. Such individuals are infected with MSX, however, and are weakened by lingering infections, renewed infections, and non-MSX stresses. There is, following the initial massive kill, a progressive "weeding out" in which oysters with the lowest proportion of susceptible genes survive longest. At the end of the 33-month test period, survivors with a preponderance of resistant genes still have some masked susceptible qualities.

In reproduction, genes are recombined and some offspring acquire a greater porportion of susceptible characters than their selected parents had. Upon first exposure to MSX, these die and there follows the same gradual selection of less susceptible individuals as in the parent generation.

The argument for such a recombination is strengthened by seasonal mortality patterns (Fig. 3). There is a consistently higher mortality during first exposure in any given generation than there was in the parents during their last season of exposure.

With each succeeding generation, fewer susceptible traits remain to be passed along, and MSX-caused mortalities decrease accordingly. But even after three generations of rigorous selection and breeding, MSX still kills oysters in the F_3 generation. At least as far as this generation, then, there is evidence that resistance to MSX kill has not levelled off, but is continuing to develop in laboratory-reared stocks.

Two important aspects of an oyster's ability to deal with MSX are the dosage of infective particles it receives and the kinds and numbers of additional stresses with which it must cope. The failure of native oysters from widely different areas in Delaware Bay to show differential mortality, when tested at the Cape Shore Laboratory, suggests that there may be enough mixing of larvae within the estuary so that oysters setting in all areas are equally resistant. The lack of differential mortality may, however, result from overwhelming doses of infective particles at Cape Shore. A quite different result was obtained in a recent study. In each of 3 years, oysters from three different seed beds, Arnolds, Cohansey, and New Beds, were placed in trays and exposed to MSX on a planted ground (Fig. 1). In each of the years, mortalities followed a consistent pattern: Arnolds seed suffered heaviest kill, followed closely by that from Cohansey Bed; New Beds oysters always had the least mortality (Haskin, see footnote 6). This is the expected pattern, since MSX selection pressure on the seed beds diminishes in any upbay direction along the decreasing salinity gradient. The differential in resistance of these stocks, demonstrated on the planted ground, has been completely masked under the testing conditions on the Cape Shore flats.

Working at this laboratory, Valiulis and Haskin (1972) showed conclusively that the demonstration of resistance in oysters to the pathogen *Labyrinthomyxa marina* was dose-dependent. We tested stocks with differing resistances to MSX mortality to see if those oysters most resistant to MSX were also most resistant to *L. marina* mortality. Unlike MSX, *L. marina* can be transmitted in known dosage under laboratory conditions. Valiulis and Haskin (1972) found that, with heavy parasite dosages, all of the stocks died in equally great numbers. With lower doses, however, oysters resistant to MSX kill were shown to be also more resistant to *L. marina* than were MSX-susceptible stocks. This work may indicate that the mechanism of resistance to MSX in the selected laboratory-reared stocks is not specific for MSX. In any event, whatever the mechanism may be, it is suggested that resistance to MSX mortality may also be overwhelmed by increased dosage of that parasite.

A second reason for higher kill, and perhaps for higher prevalences, at Cape Shore is that it is a much less stable and probably harsher environment than are the planted grounds. Oysters are stacked in trays and may suffer from crowding. They are exposed at low tide, are subjected to temperature extremes, and are periodically infested with heavy accumulations of tube-building worms, particularly *Polydora*. Some of these stresses may become especially critical in the case of oysters which survive initial exposure but remain infected with MSX. We know that infected oysters which are removed to MSX-free areas can support chronic infections for at least 3 years (Haskin and Ford[8]). If MSX-infected oysters are surviving marginally under disease pressure, the additional stress of unfavorable ambient conditions may result in death.

There has been very little MSX-associated kill in the upper bay except for the first years of the epizootic and again during severe drought in the mid-1960's, when elevated salinities permitted upbay MSX intrusion. Some infected oysters are usually present on the lower seed beds, but even when infections are present, disease-related mortality is very low. Long-term monitoring shows that normal river flows will maintain a salinity regime in which MSX selection is effectively prevented over most of the seed area, even when the pathogen is flourishing in the higher-salinity waters of the planted grounds (Haskin and Ford[9]). Lack of selection over such a vast area means that there is little likelihood of further measurable increase in resistance of native seed. In fact, it is probable that the present level of resistance to kill, 3-4 times that of unselected stocks, was reached within a few years of the original epizootic.

The MSX epizootic in Delaware Bay compounded an already serious problem for the oyster industry. A lengthy period of set failures had resulted in a serious shortage of seed for the local planters. They had been relying heavily on imported seed, but MSX precipitated a ban on imports, forcing them to rely on scarce native seed. The shortage continued into the late 1960's, well after MSX kill had subsided, and hampered the industry's recovery from the

[8]Haskin, H. H., and S. E. Ford. 1977. Control of disease in oyster populations of Delaware Bay Unpublished report to National Marine Fisheries Service for period 1 July 1975 to 30 June 1976.
[9]Haskin, H. H., and S. E. Ford. 1978. Control of disease in oyster populations of Delaware Bay. Unpublished report to National Marine Fisheries Service for period 1 July 1976 to 30 June 1977.

early kills. A series of very good sets occurring throughout the bay between 1968 and 1973 has recently provided planters with an ample supply of seed oysters. It is this readily available, relatively resistant native seed, easily and economically planted, which has enabled oystermen to remain in business despite substantial losses to MSX. Since the disease shows no signs of diminishing, and native oysters will probably not become much more resistant, a continuing supply of inexpensive seed is an absolute necessity for the industry. Only naturally produced oysters are abundant and inexpensive enough to meet these needs. It is imperative, then, that natural seed beds and the water over them be protected from any and all degrading influences.

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