

QPX Susceptibility in Hard Clams Varies with Geographic Origin of Brood Stock

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he results of recent investigations by VIMS and collaborating scientists in Massachusetts and New Jersey indicate that clam strains produced from brood stocks of South Carolina and Florida origin are more susceptible to QPX (Quahog Parasite Unknown) disease than clam strains originating from Virginia, New Jersey, and Massachusetts brood stocks.

During a 3-year study clam strains produced at VIMS from brood stocks originating from Massachusetts, New Jersey, Virginia, South Carolina, and Florida were grown at sites in Massachusetts, New Jersey and Virginia and evaluated for survival, growth, condition and QPX disease susceptibility.

The clams originating from South Carolina and Florida brood stocks had significantly higher prevalence of QPX and higher mortality than clams originating from Virginia, New Jersey and Massachusetts brood stocks. At the termination of the experiment cumulative mortality was 79% in FL and 52% in SC, as compared to 36% in VA, 33% in MA, and 20% in NJ clams. Differences between stocks were highly significant with mortality in FL and SC being significantly higher than the northern stocks. QPX prevalence in the FL and SC stocks ranged from 19-21% and 27-29% respectively in the second and third year of the study, while QPX prevalence in the VA, NJ, and MA stocks was 10% or less. Mortality was significantly correlated with QPX prevalence during the second and third years of the investigation.

Given these results, growers should consider the geographic origin of clam seed as an important component of their QPX disease avoidance/management strategies. Please see the attached report for background on QPX and for more information on this recent study. For further information contact Lisa Ragone Calvo at phone number 804 684-7339 or email ragone@vims.edu.

Introduction

QPX (Quahog Parasite Unknown) is a protistan parasite of hard clams. Mercenaria mercenaria, that has caused significant mortalities of cultured clams in maritime Canada, Massachusetts, New Jersey and Virginia. The parasite was first documented in the 1950s in New Brunswick, Canada (Drinnan & Henderson 1963). The disease resulted in clam mortalities in Prince Edward Island (Whyte et al. 1994) in 1989 and since 1995 has caused high mortalities in Massachusetts (Smolowitz et al. 1998, R. Smolowitz pers. comm.). In 1996 and 1997 high prevalences and mortalities were reported in New Jersey (Ford et al. 2002). The parasite was first found in Virginia in 1996. Since 1996 QPX has been found at nine locations on the seaside of Virginia's Eastern Shore from Chincoteague Bay south to Fisherman's Island (Ragone Calvo et al. 1998). In 2001 the parasite caused extensive clam mortalities at a seaside location, which resulted in a significant economic loss to an Eastern Shore clam culturist. This summer the parasite has again been associated with extensive clam mortalities at another seaside location.

Outbreaks of QPX can be sudden and catastrophic with mortalities of 80-95% occurring in some instances (Smolowitz et al. 1998). Occurrence of the parasite has been limited to market and near-market sized clams; the pathogen has not been detected in hatchery-produced seed clams (Ford et al. 1997). QPX is believed to be an opportunistic facultative parasite that is widespread in high salinity coastal environments. This means that the QPX organism does not absolutely depend on a parasitic way of life, but easily adapts to it after infecting a host. Generally, organisms that are stressed by some factor or a number of factors are less resistant to parasitism and more apt to be negatively affected by a parasite. Stress associated with high planting densities and poor husbandry is believed to increase the risk of QPX disease problems. Another factor that appears to be an

important determinant of QPX disease is the strain of the clam host. In the mid-1990s clam growers and scientists in Massachusetts anecdotally noted significant differences in survival among clam strains originating from geographically distinct areas that were grown at the same site; New Jersey clam stocks suffered much higher mortality than local seed grown on Cape Cod. In New Jersey South Carolina clams suffered higher QPX mortality than New Jersey clams (Ford et al. 2002).

During the last three years VIMS scientists in collaboration with researchers from Rutgers University in New Jersey and from Woods Hole Oceanographic Institute and the Marine Biological Laboratory in Massachusetts conducted an investigation to test the hypothesis that host genetic origin and geographic location effect QPX disease in hard clams. The main objective of this 3-year study was to compare clam growth, condition, survival and QPX prevalence and severity of five hatchery-reared strains of hard clams at three regionally distinct (Massachusetts, New Jersey, and Virginia) locations where QPX occurs. The purpose of this report is to provide seed producers, nursery operators, growers and resource managers with up-todate information that will assist in the implementation of wise and effective QPX disease avoidance strategies.

Scope of Work

In the spring of 1999 clam seed was produced at the Virginia Institute of Marine Science from five genetically distinct brood stocks, which originated from Massachusetts (MA), New Jersey (NJ), Virginia (VA), South Carolina (SC) and Florida (FL). Five thousand seed

of each clam strain were planted in October 1999 at each of three locations where QPX is known to be present—Provincetown, Massachusetts; Tuckerton, New Jersey; and Wachapreague Inlet, Virginia. At each location an array of 20 plots, 4 plots for each clam strain, were planted at a clam density of 50 per sq. ft. The plots were sampled in

spring, summer, and fall 2000 and 2001 and again in spring 2002. On each sample date clam cumulative mortality was estimated by counting live and dead clams in a series of randomly selected cores of each plot and samples were removed for the assessment of growth, condition, and QPX prevalence. On the final sample date all live and dead clams remaining in the experimental plots were collected and quantified for the determination of final cumulative mortality. This total count of all live and dead clams yielded a more accurate assessment of mortality than random core sub-sampling.

Results and Discussion

Examination of the data from this study is still in progress. This report includes only the results from the Virginia site. However, similar trends have been noted at the New Jersey site. The Massachusetts site experienced significant losses of seed clams during the first winter thereby precluding the complete execution of the study at that location.

Growth and Condition Index. Average shell height of seed clams ranged from 8.6 to 9.6 mm at the time of planting in October 1999 (Figure 1). Florida clams were larger than all other stocks at planting; however, they only differed significantly from the Virginia stock, which had the lowest mean shell height of the five stocks. From August 2000 through the termination of the investigation shell height did not significantly differ among stocks. On the final sample date, April 2002, mean shell heights of the five stocks ranged from 41.6 to 45 mm.

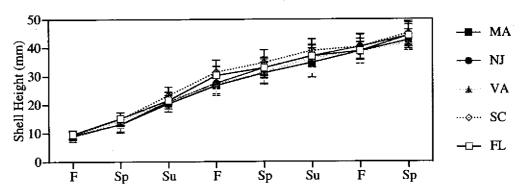


Figure 1. Mean shell height of hard clams sampled from the Virginia site in fall (F) 1999; spring (S), summer (S) and fall (F) 2000 and 2001; and spring (S) 2002. Shell height is contrasted for the five clam stocks that were tested: Massachusetts (MA), New Jersey (NJ), Virginia (VA), South Carolina (SC), and Florida (FL). Error bars represent standard deviation.

Generally SC and NJ clams exhibited higher condition index, a ratio of dry meat weight to shell width, than the other three groups; however statistically significant differences were only noted in October 2000 and June 2001 (Figure 2). On both dates SC clams were found to have a significantly higher condition index than the VA and MA stocks. No other statistically significant differences among stocks were found.

In terms of growth and condition, these results suggest that there is no advantage of growing one clam strain over another. SC clams generally exhibited slightly higher shell heights and condition indices than the other groups, but overall these differences were not significant. Mortality. Differences among strains with respect to survival and QPX disease susceptibility were much more striking than for growth and condition, particularly in the second and third year of the investigation. The first estimate of mortality was made in June 2000, eight months after planting. Mean mortality of the five stocks at this time ranged from 18 to 32% and no significant differences among stocks were found (Figure 3). Similar mean mortality estimates were determined for July 2000 through August 2001. Though there was quite a bit of variation in estimates of mean mortality, in general these differences were not significant. Mean mortality in the FL clams increased from 32% in August to 60% in November 2001. In contrast mortality in the other four stocks ranged from

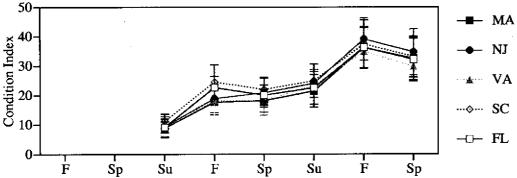


Figure 2. Mean condition index of hard clams sampled from the Virginia site in fall (F) 1999; spring (S), summer (S) and fall (F) 2000 and 2001; and spring (S) 2002. Condition index is contrasted for the five clam stocks that were tested: Massachusetts (MA), New Jersey (NJ), Virginia (VA), South Carolina (SC), and Florida (FL). Error bars represent standard deviation. Condition indices were not determined in F 1999 and S 2000.

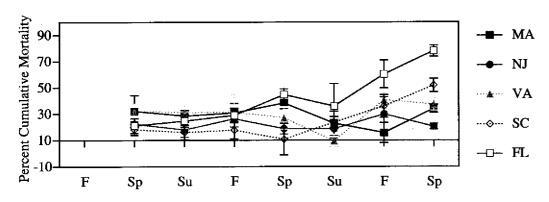


Figure 3. Mean percent cumulative mortality of hard clams at the Virginia site in spring (S), summer (S) and fall (F) 2000 and 2001; and spring (S) 2002. Mortality is contrasted for the five clam stocks that were tested: Massachusetts (MA), New Jersey (NJ), Virginia (VA), South Carolina (SC), and Florida (FL). Error bars represent standard error. In spring 2002 mortality was estimated based on total counts of live and dead in the entire plot; on all other dates mortality was estimated based on random core subsamples.

16 to 41%. At the termination of the experiment in April 2002, based on total counts of all live and dead clams remaining in the plots final cumulative mortality was determined to be 79% in FL, 52% in SC, 36% in VA, 33% in MA, and 20% in NJ. Differences between stocks were highly significant with mortality in FL and SC clams being significantly higher than all other stocks and significantly different from each other. Additionally, mortality in the NJ stock was significantly lower than that in all other stocks. Mortality did not significantly differ between the VA and MA stocks.

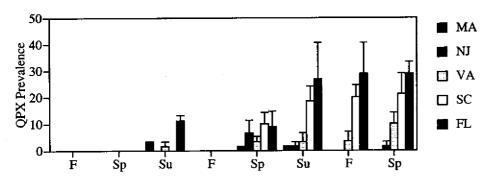


Figure 4. Mean QPX prevalence in hard clams sampled from the Virginia site in fall (F) 1999; spring (S), summer (S) and fall (F) 2000 and 2001; and spring (S) 2002. QPX prevalence is contrasted for the five clam stocks that were tested: Massachusetts (MA), New Jersey (NJ), Virginia (VA), South Carolina (SC), and Florida (FL). The absence of a bar denotes 0% prevalence. Error bars represent standard error.

QPX Prevalence. QPX was not detected in clams sampled at the initiation of the experiment in October 1999, nor in May 2000. Clams began to exhibit detectable infections in August 2000, less than one year after planting (Figure 4). Mean prevalence in FL clams, 11%, was significantly higher than in the other four stocks, which had prevalences ranging from 0-3%. No infections were detected in clams sampled in fall 2000; however, in May 2001 infections were observed in all clam stocks; prevalence was 10% in SC, 9% in FL, 7% in NJ, 3% in VA, and 2% in MA. In August 2001 and November 2001, prevalence remained low in MA, NJ and VA clams (0-4%), but significantly increased in SC and FL clams, which had 19-20% and 27-29% prevalence respectively. On both sample dates prevalence in SC and FL stocks was significantly higher than in MA, NJ, and VA stocks. Prevalence in the SC and FL clams remained high (21-29%) and differed significantly from MA and NJ clams, which had very low or no QPX infections. Prevalence in VA increased to 10% in April 2002, but this did not differ significantly from that in the other stocks.

QPX prevalence significantly correlated with mortality on all sample dates from August 2001 through the termination of the study in April 2002. This significant correlation strongly suggests that a significant proportion of the clam mortality observed in the FL and SC clams was caused by QPX.

FL and SC clams had higher QPX prevalences and showed poorer survival than the more northern clam stocks. The reasons for the association of higher QPX susceptibility and clams of southern origin can't be determined from the present study. Possibly FL and SC clams grown in northern climates exhibit a general stress associated with exposure to colder temperatures than that which they have been acclimated to, or possibly their immune response capabilities are suppressed under such conditions.

The results of this study agree with anecdotal and published (Ford et al. 2002) evidence that suggests that clams of southern origin are more susceptible to QPX than local Virginia, New Jersey and Massachusetts stocks; however, while we believe this to be a general phenomenon, this study has not demonstrated that all clam stocks of southern origin would exhibit the same elevated risk of QPX disease. Never the less, hard clam culturists should pay close attention to the impact of clam seed origin as they implement effective and rational QPX avoidance strategies.

Acknowledgements

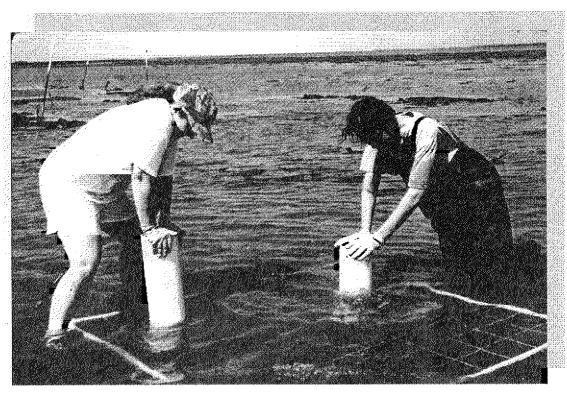
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Core sampling of clam plots.

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