

TABLE 3.

A) Surface water temperature and mean percent cumulative mortalities (SD= standard deviation, n = 2 replicates) of large and small oyster cohorts cultured at Fishers Island at high and low stocking densities (see Table 1). B) Mean shell height (H, mm) and dry soft tissue weight (DW, mg) obtained by pooling data from two replicate experimental groups at the high-density treatment [data only available for some of the sampling dates (see text)]. ND = not determined; SE = standard error, n = sample size.

A) % Cumulative Mortality Mean (SD)					
Date	T (°C)	Small Cohort		Large Cohort	
		High	Low	High	Low
6/12	18	0.9	0.9	8.8	8.8
6/127	18	0	0	5.4 (5.4)	5.2 (0.6)
7/10	18	15.6 (11.6)	9.0 (5.6)	2.8 (1.9)	1.8 (2.6)
7/125	20	96.4 (1.1)	100	6.8 (6.0)	4.2 (1.2)
8/8	22			6.0 (1.9)	6.2 (1.2)
8/29	ND			6.8 (1.5)	3.3 (2.0)
9/13	21			7.1 (1.0)	3.6 (0.3)
9/126	18.5			10.8 (2.0)	8.2 (3.0)

B) Mean Shell Height (H and Tissue Dry Weight (DW))								
Date	Small Cohort				Large Cohort			
	H	(SE, n)	DW	(SE, n)	H	(SE, n)	DW	(SE, n)
6/12	8.9	(0.2,49)	2.54	(0.13,49)	31.7	(5.0,30)	75.40	(4.32,30)
6/127	12.2	(0.3,60)	7.26	(0.46,59)	33.4	(0.7,60)	120.89	(5.23,60)
7/10	12.0	(0.3,56)	5.28	(0.44,39)	39.8	(0.7,60)	229.24	(12.01,60)
7/125					38.9	(1.0,60)	283.98	(15.98,60)
8/8					44.2	(0.8,98)	280.38	(13.28,98)
9/13					53.9	(1.1,56)		ND

Several differences in response were observed, however, between the two cohorts. It is noteworthy for example, that the LC showed detrimental effects on growth, and appearance of the abnormal conchiolin syndrome and mantle lesions 2 weeks earlier than the SC. This correlates with the appearance of mortalities 2 weeks earlier than in the SC. The drop in condition index, coincident with mortalities, however, was much more pronounced in the SC (53% compared to only 9% in high-density treatments), yet mortalities peaked at similar levels in both cohorts by July 26. Galtsoff (1964) suggested that young *Crassostrea virginica* have flatter valves and therefore a higher CI than larger oysters (Galtsoff 1964). Rainer and Mann (1992) found no size-dependency of the volumetric CI in *C. virginica*, but tested this only for oysters 36 to 96 mm in height. The difference in initial CI values observed between cohorts in the present study cannot be attributed solely to size-dependency, however, since SC oysters had lower values even when they attained the same size as LC oysters (compare the mean condition index for the SC on July 11 with that of the LC on June 26 in Fig. 4). Difference in mortalities, growth and condition between the 2 cohorts are thus not clearly correlated with size, and may simply reflect inter-batch variability.

A decline in condition represents a sensitive but generalized stress response, of limited value in inferring specific causes of mortality. Reductions in the condition index of oysters have been related to reproductive condition (e.g. Nascimento and Pereira 1980), nutritional stress (Wright and Hetzel 1985), as well as disease (Newell 1985, Paynter and Burreson 1991). Interpretation of changes in the condition index can be confounded if rates of growth in shell and soft tissues are uncoupled (Hilbish 1986). In the present study, however, reductions in growth coincidentally affected both shell and soft tissues.

Mean tissue weight and condition index were generally greater at the lower stocking density, although these differences were not always statistically significant, especially for the SC. Differences in tissue weight between density treatments averaged only 21-24% over the study period in both cohorts, and were more pronounced during the early part of the experiment (through July 26). Mortalities, however, were reduced by as much as 39-45% at the low stocking density. Thus, reduced densities had a greater effect in reducing mortalities (especially in the small cohort), than in increasing growth rates, suggesting that the former largely resulted from reduced incidental anoxia, associated with fewer total numbers of dead animals within growing trays. These results suggest that thinning of cultured oysters may provide a management alternative to partially mitigate losses during the critical period of summer mortalities.

Histopathology

Both gross pathology and histopathological evidence suggests an irritant or toxin affecting the epithelial cells of the mantle, causing retraction of that organ and an attempt to "wall off" the oyster's soft tissues from the irritant. We found no evidence to support the contention that juvenile oyster mortality is associated with a newly described protozoan of uncertain affinity (Farley et al. 1992). The coccoid bodies present in many lesions appear to be remnants of degenerated oyster cells, not a protist, and the ciliates seen in some lesions are undoubtedly opportunistic invaders. We conclude that if a microbe is the causative agent of the "disease," it is either not found consistently or in significant numbers in tissues of affected oysters and induces lesions from a distance by production of an exotoxin, or it is easily washed free of the tissues