

Figure 4. Mean gravimetric condition index (see methods) ( $\pm$ SE) of large and small oyster cohorts cultured at 2 stocking densities in Oyster Bay. Mean shell heights are indicated at each sampling date; asterisks indicate significant differences ( $p < 0.05$ ) in mean condition between density treatments.

#### Shell Anomalies

Although most juvenile oysters examined had mats of bacteria and other microorganisms on the external shell surface, gross and histological examination of the shell indicated no evidence of penetration by fungus or other shell-boring organism.

Microscopically, the most consistent correlate with the juvenile oyster mortality syndrome, in both living and dead animals, was a layer of abnormal conchiolin deposited on the inner surface of one or both valves, but primarily on the left valve. It was frequently raised into a ridge several millimeters from the edge of the shell (Fig. 6, right valve). Most often, the ridge formed a completely closed ring (Fig. 6, right valve) on only one valve; however, rings were found on both valves of some oysters. It was not unusual to find the ridge juxtaposed to the adductor muscle along the dorsal to posterior margin of the muscle (Fig. 6, right valve). In some cases, the conchiolin layer was deposited between the adductor muscle and the shell, causing the muscle to detach. Tissues of live oysters were usually found contracted within the bounds of the ridge; however, in some cases the ridge was present inside the free edge of the mantle. Portions of the shell external to the conchiolin ridge were frequently covered by mud and fouling organisms. Some oysters, apparently in early stages of the syndrome, were found with the thin conchiolin sheet covering all or only small portions of the shell surface, but with no ridge. The left valve of affected oysters was often deeply cupped, its edge extending beyond that of the right valve.

Prevalence of abnormal conchiolin increased markedly during July in concert with the increase in mortalities (Fig. 7). Small oysters in both density treatments showed similar patterns, such that 43-48% of living oysters exhibited the syndrome by late July,

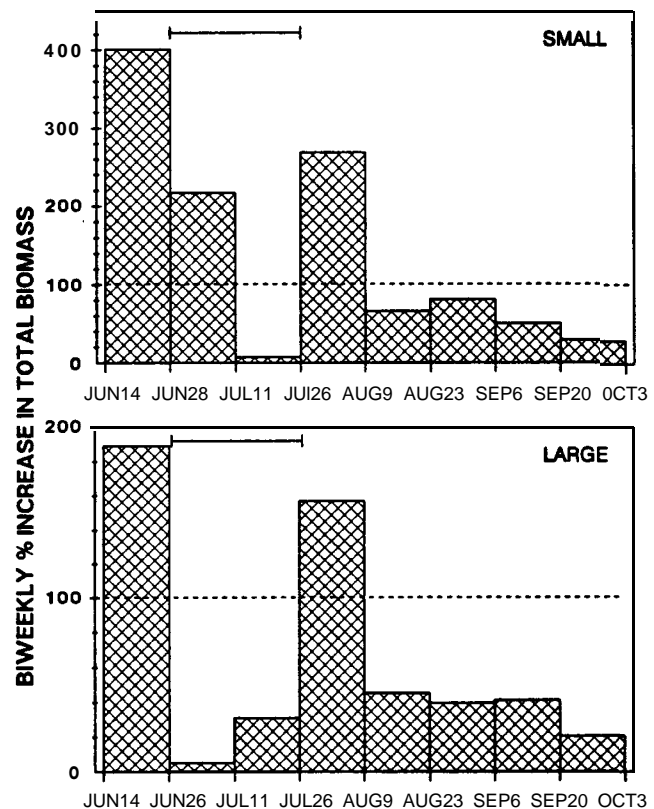


Figure 5. Percent biweekly change in total live biomass of small and large oyster cohorts held at the high stocking density in Oyster Bay, calculated as:  $[(n_2W_2 - n_1W_1)/n_1W_1] \times (t_2 - t_1)/15 \times 100$ , where  $n_1$  and  $n_2$  = numbers of survivors for each sampling interval ( $t_2 - t_1$ ) = 13 to 15 days,  $W_1$  and  $W_2$  = mean whole body weight of live oysters (pooled data from 2 replicate experimental groups,  $n$  as in Fig. 2). Horizontal dashed lines indicate the level corresponding to a doubling of biomass over a 2-week period, and solid horizontal bars mark the period of mortalities. Initial  $n$  was arbitrarily selected as that initially deployed in one experimental tray (35,000 and 7,200 oysters for small and large cohorts respectively); 100% survival was assumed after the July 26 sampling date.

after which prevalence decreased to nearly zero. In contrast, LC oysters showed a relatively high prevalence (24-34%) as early as July 11. The presence of abnormal conchiolin decreased thereafter in the low density treatment, but remained high (40%) in the high density animals through the end of July, after which it declined (Fig. 7). Prevalence of abnormal conchiolin was also high in the "late cohort," with increasing values of 21%, 40% and 52% on August 9, 16 and 23 respectively.

Longitudinal cross-sections of the lower (cupped) valve of 15 survivors from the SC collected on September 20 were examined for evidence of past alteration in the pattern of shell deposition. No anomalous deposition was apparent on the external surface of the shell or in cross-section, suggesting that survivors were relatively unaffected at the time of mass mortalities.

#### Histopathology

Light microscopy of samples collected during peak mortality showed that oysters depositing abnormal conchiolin possessed lesions of the mantle characterized by degeneration and sloughing of epithelial cells, infiltration of hemocytes into epitheliums and un-