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Sea urchin pathogen: a possible tool for biological control

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ABSTRACT: A sea urchin pathogen recently exerted great impact on the Nova Scotia near-shore community by releasing large areas of habitat to seaweed colonization and growth. The pathogen has promise for use as a tool for biological control because it is virulent, apparently species specific, can be maintained and transferred in the laboratory, and is waterborne. The purpose of this study was to demonstrate that it could be transferred from the laboratory to the field. This was accomplished in a reciprocal transplant experiment. Sea urchins from 2 sites 3.5 km apart were brought to the laboratory; one group was exposed to the disease-causing agent and the other served as control. Both groups were returned to the field but released at opposite sites. Sea urchins naturally occurring near the release of the exposed sea urchins developed disease symptoms within 4 wk; and by 8 wk when the temperature became too low for transfer of disease, diseased sea urchins were found as far as 120 m from the release site. No disease occurred at the control site by 8 wk or in 5 additional areas within a few kilometers of the 2 release sites

INTRODUCTION

Sea urchin domination of shallow, rocky marine habitat to the exclusion of macroalgae (except calcareous forms) is very well documented. In St. Margarets Bay, a well-studied embayment in Nova Scotia, about 10 % of this habitat was dominated by the green sea urchin *Strongylocentrotus droebachiensis* and 90 % by macroalgae in 1968 (Mann, 1972). Sea urchin domination increased to 60 % by 1973 (Breen and Mann, 1976) and to 90 % by 1978 (Chapman, 1981). In areas where sea urchins were removed experimentally macroalgae re-established 100 % cover within a few months (Breen and Mann, 1976; Chapman, 1981). Lawrence (1975) reviewed 38 cases of sea urchin-dominated barren grounds worldwide. In several of these, as in St. Margarets Bay, macroalgae colonized the bottom after sea urchins were removed experimentally.

Macroalgae are obviously beneficial to the marine plant industry (e. g. giant kelp in California and Irish moss in Nova Scotia). By contributing primary production and habitat complexity they may also enhance finfish abundance (e. g. Quast, 1968); Simenstad et al., 1977; Leaman, 1980; Moreno and Jara, 1984) and shellfish yields (Wharton and Mann, 1981). The degree of enhancement remains a subject of discussion (Quast, 1968; Pringle et al., 1982) and ongoing research.

Artificial methods of controlling sea urchins have included the employment of SCUBA divers to break sea urchins with hammers or to spread calcium oxide over aggregations of sea urchins (North, 1974). Because these methods are expensive and must often be repeated to produce lasting effects, they are practical on only a small scale.

Documented cases of sea urchin mortality due to disease suggest this as a tool for biological control. Three localized cases of *Strongylocentrotus franciscanus* mortalities in California were of a few month's duration and a few hectares in area (Johnson, 1971; Pearse et al., 1977). In one area the canopy of *Macrocystis pyrifera* expanded by over 10 ha following the mortalities (Pearse and Hines, 1979). Three sea urchin species *(Paracentrotus lividus, Sphaerechinus granularis, Arbacia lixula)* were affected by localized mass mortalities along the European coast of the Mediterranean during 1978–1979 (L. Fenaux, Station Zoologique at Villefranche-sur-Mer, pers. comm.). Explosive growth of epiphytes on seagrass followed mortality of *P. lividus* on the Mediterranean coast of France (Boudouresque et al., 1981). Widespread mortality of *Diadema antillarum* has occurred recently

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throughout the Caribbean (Lessios et al., 1983; Bak et al., 1984). On the south coast of Nova Scotia at least 250,000 t of *S. droebachiensis* died of disease from 1980 through 1983. This near complete mortality along 1,400 km of shoreline released at least 500 km^2 of habitat to colonization by macrophytes. If this colonization leads to mature beds, algal biomass will increase by about 1.8 million t, and annual net production by about 7 million t (Miller, 1984). In none of these cases has the disease agent been positively identified.

The disease agent(s) causing mass mortalities in Nova Scotia has several characteristics which make it favourable for use in biological control. It is transferable in the laboratory; it can be maintained in the laboratory for at least one year; it is waterborne; it appears to be species specific; and sea urchins lack natural resistance to it at warm temperatures (at least in the laboratory) (Miller and Colodey, 1983; Miller, 1984, Scheibling and Stephenson, 1984). To be useful in biological control the agent must also be transferable from the laboratory to a field population of sea urchins. The purpose of this study was to demonstrate such a transfer.

Experimental and control sites, labelled D (diseased) and H (healthy) respectively are located 3.5 km apart in eastern Nova Scotia (Fig. 1). Both sites are exposed

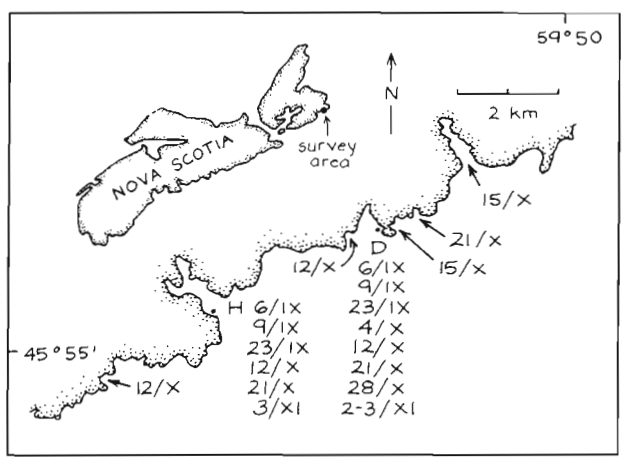

Fig. 1. Site of release of diseased (D) and healthy sea urchins (H); other sites inspected for presence of diseased sea urchins; dates each site was visited

to the open Atlantic over about a 30° arc; the substratum at both sites is a mixture of boulders and granite bedrock; and water depths are 6 m below chart datum at Site D and 3.5 m at Site H.

The choice of study sites had the following constraints. (1) Sea urchin density should be high to aid in transfer of the waterborne disease agent among individuals. The minimum required density is not known. (2) Water temperature should be high enough to allow transfer of the disease within a few weeks. In laboratory exposures Scheibling (1984) reported time to 50 % morbidity as less than 10 d for 15 to 22 °C but about 30 d at 12 °C, the minimum temperature at which disease could be transferred. In laboratory exposures at 17 °C urchins from 4 locations were 100 % moribund by 14 d (Miller, 1984). (3) Natural occurrence of the disease should be avoided; but (4) until more is known of its pathogenicity it should not be introduced far from its known area of distribution. The easternmost point where mass motalities were previously identified was Louisbourg in 1981 (Miller and Colodey, 1983), about 10 km west of Site D. At several dive sites east of Louisbourg (Fig. 1) sea urchins were large and abundant; and subtidal macroalgae were absent in 1983, suggesting that any recent sea urchin mortalities were slight. (5) Logistic considerations required that the study sites be near a safe harbour and have at least partial shelter from ocean swells so that diving was not so restricted by weather. These constraints limited the choice of experimental sites to within tens of kilometers of those chosen.

METHODS

A reciprocal transplant experiment begun on August 26, 1983 is summarized in Table 1. Apparently healthy sea urchins within the size range 3 to 6 cm diameter were collected from Sites D and H (Fig. 1) by SCUBA divers. Bottom temperature was 18 °C. On the same day these urchins were brought to Halifax and placed in tanks with running sea water maintained at 16 °C ± 1 C°. Beginning on August 29 sea urchins from Site H were exposed to diseased sea urchins taken

Table 1. Study schedule, Autumn 1983

Date	Activity
Aug 26	Sea urchins collected from Sites D and H and placed in laboratory tanks
Aug 29–Sep 6	Sea urchins from Site H exposed to diseased sea urchins from laboratory stock
Sep 6	Disease-exposed sea urchins from Site H released at Site D, and non-exposed sea urchins from Site D released at Site H
Sep 9–Oct 21	Qualitative observations on presence of disease at Sites D and H
Oct 12–21	Qualitative observations on presence of disease at five additional sites
Oct 28–Nov 3	Sites D and H sampled quantitatively for numbers of sea urchins healthy and moribund or dead

from a laboratory stock maintained since the previous autumn. This diseased stock had been maintained by continually adding healthy animals to, and removing dead animals from, a tank of running sea water kept at > 14 °C. To expose the sea urchins from Site H, stock sea urchins with obvious external symptoms of disease were placed in the tank in mesh bags. This exposure was continued, replenishing diseased sea urchins as they died, until September 6.

On September 6 both exposed and unexposed lots were counted, weighed, boxed, and taken to the release sites. Before the releases the center of each site was marked with a subsurface buoy and most of the resident sea urchins were removed from a 3 m diameter circle. At Site D, 580 sea urchins totalling 22 kg which had been collected from Site H and exposed to disease were released in a 2 to 3 m diameter circle. At Site H, 330 sea urchins totalling 10 kg that had been collected from Site D and not exposed to disease were also released in a 2 to 3 m diameter circle. If the disease subsequently appeared at Site H it would be either because it occurred there naturally or because it was introduced with the unexposed urchins from Site D.

During the next 7 wk, Sites H and D were visited 4 and 5 times respectively to record the presence or absence of diseased urchins. During these visits the release sites were examined carefully with a more cursory look up to 40 m distance.

After the disease was noted to be spreading from Site D, 5 neighboring areas were also visited. In each area divers swam a few hundred meters within depths of 1 and 10 m. Sea urchins were very abundant and the substratum was bedrock and boulders in all areas.

After the water temperature had fallen to 10 °C, below the temperature at which the disease was expected to spread (Scheibling, 1984), sea urchins near Sites D and H were sampled quantitatively. Four radial transects were laid out at approximately right angles. Samples were taken at 5 and 20 m from Site H, and at 5, 10, 20, and succeeding 20 m intervals from Site D. The numbers of sea urchins ≥ 2 cm diameter were counted in each of 4 quadrats, 0.5 by 1.5 m, giving a total sample area of 3 m^2 at each location. The counts were partitioned into 2 categories: live without disease symptoms, and moribund or dead. Dead sea urchins were counted only if more than one-half the test was intact. The mean percentage moribund or dead was compared statistically among locations on a transect using one-way analysis of variance. The percentage data were first transformed to logits (Y),

$$Y = \log_e[(g+½)/(n-g+½)]$$

where n = total number of sea urchins in a quadrat; g = number moribund or dead. This transformation tends to normalize distributions of proportions even when they span a large range of values and is superior to the more common arc-sine transformation when n includes some small values (Snedecor and Cochran, 1967).

Temperatures were taken with a stem thermometer; they ranged from 14 to 16 °C until early October, then gradually declined to 9 °C by early November.

The occurrence of sea urchin disease was determined by the presence of external symptoms. Failure to hold on to the substratum is the most obvious. This is accompanied by gaping lantern teeth, retracted tube feet, and later by drooping and missing spines (Miller and Colodey, 1983). A few bare, intact tests and broken tests with or without spines are common in most field aggregations; however, a larger number of bare tests or even a few whole unattached urchins with spines are evidence of the disease. The distinction between moribund and dead sea urchins is not easy to determine in the field and will not be made in this paper.

A test was conducted in Halifax Harbour during October, November, and December 1983 to measure the time required for dead sea urchins to lose their spines and for their tests to break apart. Fifty apparently healthy sea urchins of 4 to 6 cm diameter were killed by immersing them in sea water at 50 °C for 30 s. They were immediately placed in a net-enclosed cage to protect them from large scavengers and suspended from a wharf in a sheltered location. They were observed frequently over the next 55 d at temperatures of 12 to 4 °C.

RESULTS

In the cage experiment in Halifax Harbour spine loss from dead sea urchins was abrupt: from slight at 6 d, to greater than 50 % at 8 d, to near total at 9 d. Tests broke apart at a more gradual rate: from 10 % at 19 d, to 50 % at 30 d, to 90 % at 48 d. Thus, the presence of spines on tests indicates recent mortality while denuded and broken tests indicate older mortalities.

On September 9, 3 d after the sea urchins were released, newly broken tests with spines still attached were common at both Sites D and H. The type of damage was indicative of rock crab *Cancer irroratus* and lobster *Homarus americanus* predation (Himmelman and Steele, 1971). Both predators were common at both sites. The sea urchins were weak and probably unable to attach firmly to the substratum after being out of water for about 9 h during transport from Halifax. Thus, they would have been vulnerable to predation. A few unattached (moribund or dead) sea urchins remained intact at Site D; however, most individuals at both sites were firmly attached.

On September 23 no unattached sea urchins were present at Site H; but at least 50 were unattached and intact at Site D, most with spines. These were assumed to be released sea urchins which succumbed to disease.

By October 4 the disease appeared to be spreading at Site D. Sea urchins with disease symptoms were found up to 10 m from the release site.

A more extensive search on October 12 revealed no evidence of disease at or near Site H but widespread occurrence near Site D. At least 50 % were moribund or dead within a 5 m radius of the release site. The proportion decreased to zero at about 40 m in each of 2 directions. Most unattached urchins still had spines.

On October 21 the extent of dead and moribund sea urchins at Site D was similar to that on October 12; but a larger fraction, approximately 40 %, had lost their spines. Dr. J. H. Himmelman (Laval University), participated in this site visit and recorded a decrease in the fraction of diseased animals at increasing distance from the release site. Diseased urchins were found to a maximum distance of 50 m. There was again no evidence of disease at Site H.

During October 12 to 21, 3 to 17 d after the disease was first noticed to be spreading from Site D, 5 neighboring areas, indicated in Fig 1, were examined. The closest of these were 0.6 km to the east and 0.6 km to the west of Site D. No evidence of disease was observed at any of these 5 areas.

A severe storm passed over the study area on October 24 and 25, delaying the quantitative sampling. Sampling was commenced on October 28, but the presence of large ocean swells further delayed its completion until November 2 and 3. The storm almost certainly carried away and broke into pieces many of the dead and moribund sea urchins; however, the impact of disease was still quite apparent.

Near Site D moribund and dead sea urchins comprised a large proportion of the population, but this proportion decreased sharply with increasing distance (Fig. 2). On the northward transect the decrease was from 57 to 6 % ($P < 0.01$) over the first 40 m. Beyond 40 m there was no evidence of disease, including stations 20 m east and west of the transect. On a 20 m transect ending in a small cove eastward of the release site the percentage moribund and dead also decreased

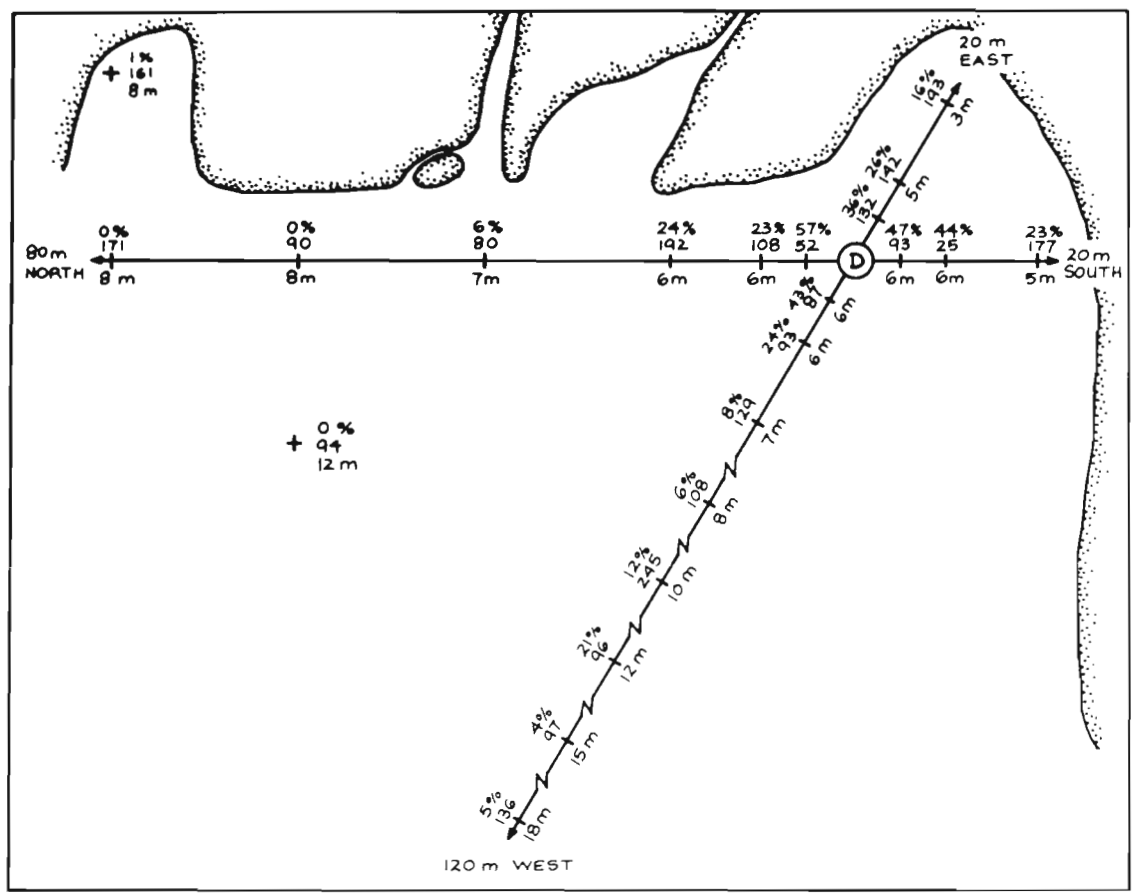

Fig. 2. Results of quadrat sampling near the release site of diseased sea urchins (D). Top number: mean percentage moribund or dead; middle number: total number of sea urchins counted; bottom number: water depth (m)

significantly ($P < 0.05$). The decrease in percentage moribund and dead was not statistically significant ($P > 0.05$) along a 20 m transect which ran southward, ending near the base of a wall forming the side of the cove. The westward transect ran away from shore and ended where the bottom changed from bedrock to sand. There, moribund and dead individuals decreased from 43 to 6 % ($P < 0.01$) over the first 40 m, increased to 21 % at 80 m, then decreased again to 6 % at 120 m ($P < 0.05$). The increase at 80 m may reflect the pattern of transfer of the disease or may represent an accumulation of the unattached animals resulting from the wave patterns of the late-October storm.

Near Site H quantitative sampling provided no evidence of disease. For the 4 sampling locations 5 m from the release site, 3 of 409 sea urchins were dead; at the 4 locations 20 m from the release site, 9 of 295 sea urchins were dead. The absence of disease at Site H indicates that it had not occurred naturally there and that it had not occurred naturally at Site D, the source of the sea urchins planted at Site H.

DISCUSSION

Considering that the disease is waterborne, the pattern of dispersion from an epicenter was remarkably clear. Sea urchins with disease symptoms were confined to less than 60 m to the north and were rare 120 m to the west. They were not found 600 m or further to the east and west.

According to Debach (1974) a natural enemy (pathogen, parasite, or predator) well suited for biological control: (1) is a good searcher for its host, (2) has higher reproductive capacity than its host, (3) responds rapidly to an increase in host density, (4) has high host specificity, and (5) tolerates as broad a range of environmental conditions as its host. Given the rapid spread of mass mortalities from 1980 through 1983 the sea urching pathogen can be considered a good searcher and to have a high reproductive capacity. Response to host increase is not well documented in the field, although in 1983 it was responsible for a kill of sea urchins that appeared following the 1980 and 1981 mortalities (Miller, 1984). No unusual mortalities of other species have been observed during field surveys over the 4 yr when urchin mass mortalities have occurred. Thus, it appears to be host specific, at least among benthic macrofauna. Casual observations in the laboratory on several benthic species *(Cancer irroratus, Carcinus maenas, Homarus americanus, Mytilus edulis, Modiolus modiolus, Crassostrea virginica, Littorina littorea, Asterias* sp., *Echinarechinus parma)* placed in tanks with diseased sea urchins in 1981–82 support this conclusion. The pathogen may not tolerate as broad a range of temperatures as the host. As mentioned earlier the pathogen is active only near the seasonal maximum temperature, and Scheibling and Stephenson (1984) have hypothesized that its appearance is related to unusually warm years.

The potential impact of controlling sea urchins is considerable, especially in areas where the green sea urchin still dominates large areas of the subtidal, e. g. eastern Nova Scotia (own obs.), Newfoundland (Himmelman, 1980; Hooper, 1980), the St. Lawrence River estuary (Himmelman et al., 1983), Maine, USA (pers. comm., R. L. Vadas, University of Maine), and northern Norway (Hagan, 1983). With the above properties plus its virulence, our ability to maintain it in the laboratory, and our ability to transfer it from the laboratory to the field, the sea urchin disease has considerable potential for use as a biological control agent.

The experimental procedure could be changed by introducing the diseased sea urchins to the field earlier in the summer and in large cages. If the disease were introduced earlier in the summer it could pass through more generations and spread over a larger area before being stopped by falling temperatures. The cages would protect sea urchins weakened by transport or disease from attack by large predators, and the first conspecifics found outside the cages would indicate the first stages of disease transfer to the natural population. However, cages were not used in this study to avoid the risk of losing the entire experiment to curious fishermen or to a severe storm.

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