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Sea urchin mass mortality associated with algal debris from ice scour

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ABSTRACT: We documented a mass mortality of about 50,000 green sea urchins in the Mingan Islands, northern Gulf of St. Lawrence, which was associated with two events, the scouring by icebergs of macroalgae from the kelp fringe and the accumulation of the algal debris (primarily *Alaria esculenta*) over a 650 m^2 area (45–70 cm deep) on an urchin barrens. The urchins appeared to have the bald-sea-urchin-disease. We also found urchins with infected lesions in other shallow water areas and the incidence varied from 0.3 % on urchin barrens to 4–9% in grazing fronts next to the kelp fringe. Our field observations and experiments with urchins placed in cages showed that the large mat of drift algae probably provided a rich bacterial source which increased the infection of mechanically abraded urchins. Presumably urchins became abraded when the urchin zone was covered with the algal debris, however, a wave-tank study did not show that urchins were damaged by 14 h of moderate algal abrasion.

1 INTRODUCTION

The major role of sea urchins in structuring nearshore benthic communities is well documented (Lawrence 1975, Harrold & Pearse 1987). Sea urchin grazing can destroy kelp beds, and urchins on overgrazed areas can persist for years (see Scheibling & Hatcher 2001 for review). Nevertheless, various mechanisms can reduce urchin grazing and permit macroalgal recolonization. Dramatic declines in urchin populations caused by mass mortalities have been reported in California (Johnson 1971, Pearse et al. 1977), the Carribean (Lessios et al. 1983), the Mediterranean (Boudouresque et al. 1981), Nova Scotia (Scheibling & Stephenson 1984) and Norway (Christie et al. 1995). Such events are followed by community changes including rapid colonization by macroalgae (Pearse & Hines 1979, Scheibling & Raymond 1990). A major example is the repeated devastations of green sea urchin populations over much of the Atlantic coast of Nova Scotia by the protozoan parasite *Paramoeba invadens* (Miller & Colodey 1983, Scheibling & Stephenson 1984, Scheibling et al. 1999). The parasite is thought to be introduced periodically with the intrusion of warm waters from the Gulf Stream (Scheibling & Hennigar 1997). Urchin populations elsewhere in eastern Canada do not appear to have been affected by epizootic diseases.

Urchins with lesions on their tests have been associated with several mass mortality events (Johnson 1971, Boudouresque et al. 1981, Lessios et al. 1983, Scheibling & Stephenson 1984). These lesions consist of necrotic areas on the test, which are devoid of spines, pedicellariae and tube feet, and may be caused by secondary bacterial infection (Scheibling & Stephenson 1984, Roberts-Regan et al. 1988). Occasionally lesions also occur on urchins in healthy populations (Gilles & Pearse 1986, Roberts-Regan et al. 1988). Such lesions commonly found in several species of echinoids are referred to as "bald-sea-urchin disease" (Maes & Jangoux 1984, see Table 5.1 in Jangoux 1984). Laboratory studies indicate that mechanical abrasion of the test is necessary for bald-urchin-disease to develop and healthy urchins can experimentally be infected when necrotic tissue from an infected urchin is added to an injured area (Maes & Jangoux 1984, Gilles & Pearse 1986, Roberts-Regan et al. 1988). The lesions develop within a few days but are rarely followed by death of infected individuals, rather tissues in the affected area usually regenerate.

Natural physical disturbances can have a major influence on urchin populations (see review by Lawrence 1996). Ice scour of intertidal and subtidal habitats is a regular event at high latitudes and can remove both benthic invertebrates and algal beds (Keats et al. 1985, Conlan et al. 1998, Gutt 2001).

In this study, we describe a mass mortality of the green sea urchin, *Strongylocentrotus droebachiensis*, associated with an accumulation of drift algae. The source of the algae was from scouring of shallower water areas by icebergs. We infer that the ice scour event indirectly caused the mass mortality by increasing the frequency of injuries that led to bald-urchin disease.

2 MATERIALS AND METHODS

We first observed the urchin mortality on 12 June 2003 at Petite Ile au Marteau, in the Mingan Islands in the northern Gulf of St. Lawrence, eastern Canada (50°13'6"N, 63°41'12"W). Initially we quantified the extent of the mortality by measuring the area where dying and dead urchins were found, and the area covered with algal debris. We also sampled two accumulations of dead urchins in crevices (two 0.25-m^2 quadrats) to examine their size structure. On 18 June, we returned to the site and measured animals in two 4-m^2 quadrats to evaluate the density of urchins as well as numbers of three carnivores which were feeding on the dead and dying urchins, the whelk *Buccinum undatum* and the sea stars *Asterias vulgaris* and *Leptasterias polaris*. We quantified the frequency of lesions on urchins in these samples. Finally, we sampled the urchins and carnivores on 22 August, this time using ten 1-m^2 quadrats.

During July we quantified the incidence of urchins with infected lesions in healthy populations at three sites, Ile au Marteau (4 July, 30 to 150 m from the mortality area), Ile au Goéland East (2 km from the mortality site) and Ile au Goéland West (2.5 km from the mortality site). At each location we collected urchins in twenty 0.25-m^2 quadrats in each of two habitats, at the grazing front (at the subtidal edge of the kelp *Alaria esculenta* fringe) and on the urchin barrens. Each urchin was carefully examined underwater for lesions on aboral, lateral and oral surfaces. We compared variations in the proportion of infected urchins in the two habitats (grazing front and barrens) using a t-test with the sampling in three sites as replicates.

As the mortality occurred in an area with much algal debris, we conducted a field experiment to determine if algal debris was a mediating factor causing the lesions and mortality. On June 27, we placed three cages (5 mm mesh) containing 20 urchins (30–50 mm in test diameter) in each of three habitats at Petite Ile au Marteau, (1) underneath the algal mat, (2) on the barrens and (3) at the grazing front. The first two situations were in the area where mortality was observed and the third in shallower water above the mortality area. Because previous studies report that injury is necessary for the formation of lesions, we pulled off spines from a portion of the aboral surface close to the madreporite for 10 of the 20 urchins in each cage. We then evaluated the condition of all urchins in each cage 4 d later (on the 7th day all of the cages were carried away by strong wave surge). We further conducted a 14-h experiment in a wave tank (Gagnon et al. 2003) to test whether urchins are injured by the movement of algae debris. The wave tank was divided longitudinally into two sections, one of which was filled with blades of *Alaria esculenta* (to a depth of about 15 cm) and the other had no algae. Twenty healthy urchins (30–50 mm) were placed in each section. The waves (23 wave cycles per minute) were greater than we observed between 12 June and 6 July at the mortality site at Petite Ile au Marteau. The urchins were carefully examined for injuries at the end of the experiment.

3 RESULTS AND DISCUSSION

Scraping of the bottom by locally formed ice occurs each winter in the Mingan Islands but icebergs rarely reach this region. However, icebergs from the Labrador coast, driven by easterly winds, did drift into the islands between 31 May and 2 June 2003.

We observed that one iceberg penetrated to 15 m in depth. Several dives along the outer (southern) side of the islands (Marteau, Goéland West, Pointe Enragée) showed that many areas of the shallow sublittoral community had been scoured by the ice. We observed areas of the kelp fringe (mainly *Alaria esculenta* but also areas of *Laminaria digitata*) measuring up 5–8 m in width, where the kelp blades had been shaved off. In some areas the kelp was broken off at the stipes whereas in other areas the holdfasts had also been removed. In the most severely scoured areas ice had scraped the crustose coralline algal cover. We also observed broken urchins and body parts of sea stars that had accumulated in crevices. These smaller accumulations of dead urchins were probably the direct result of the ice scour and were distinct from the intact urchins which accounted for most of the mass mortality.

During our first survey of the mass mortality site at Petite Ile au Marteau on 12 June (10 days after the ice scour) we found an abundance of dead urchins in an area of the urchin barrens community on a gently sloped bedrock platform at 6 to 7 m in depth. There was also a large accumulation (>1300 m^3) of kelp debris on a roughly rectangular area, 35 m in length and 18.5 m in width (650 m^2), on the urchin barrens. This material was mainly blades of *Alaria esculenta* and *Laminaria digitata* and it varied in depth from 45 to 70 cm. The kelp fringe in shallower water, which must have been the source of this material, was located 150 m from the algae debris.

We estimated there were 50,000 morbid urchins in the area surrounding the algal accumulation (based on two 0.25-m^2 quadrats sampled from the estimated

Figure 1. (A) Accumulation of dead sea urchins with intact tests at the mass mortality site located on a bedrock platform at 7 m in depth at Petite Ile au Marteau in the Mingan Islands. This mortality event was associated with a large accumulation of drift algae that resulted from scouring of the subtidal kelp fringe by icebergs. (B) A sea urchin on the algal mat showing a large lesion. Photographs reproduced from video.

surface area with morbid urchins). The mortality appeared to be recent as 77% of dead individuals still had an intact peristomial membrane and Aristotle's lantern, and 11% had some attached spines (Fig. 1a). The size structure of the dying and dead urchins (Fig. 2) indicated that all urchin sizes >20 mm in diameter had been affected by the mortality event.

The paucity of <20 mm urchins in our samples was likely due to the rapid decomposition of smaller individuals (many small urchins were present but fell apart as we tried to collect them). During this period, decomposition was likely slow due to the low water temperatures (3 to 5°C). The greatest numbers of dead urchins were found on open areas on the upper side of the algal mat, suggesting that the mortality may have occurred as the algae passed over this area. Over the following two weeks we observed that the algal mat moved upslope and covered the accumulation of dead urchins. A storm with strong wave surge occurred on 7 July and observations the following day showed that

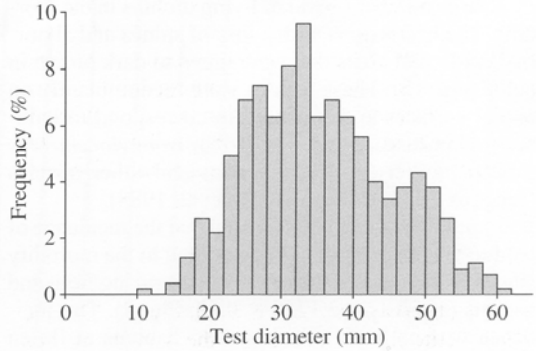

Figure 2. Size distribution of morbid sea urchins in the mortality site at Petite Ile au Marteau (n = 447).

the entire algae accumulation had disappeared and only a few broken urchin tests remained.

The dead and dying urchins attracted large numbers of scavengers which were eating the urchins. Sampling on 18 June showed that there were high densities of the whelk *Buccinum undatum* (3.4 ind.m^{-2}), and of the sea stars *Asterias vulgaris* (0.8 ind.m^{-2}) and *Leptasterias polaris* (1.1 ind.m^{-2}) compared to the sampling on 22 August (no dead urchins remained), where the density of scavengers had decreased (0.2 ind.m^{-2}, 0.1 ind.m^{-2} and 0.3 ind.m^{-2}, respectively). Most were actively devouring tissues from the urchins (some *A. vulgaris* fed in aggregations on individual urchins). The strong chemosensory and locomotory abilities of the whelk make it well suited for exploiting animal remains (Himmelman, 1988). Distance chemodetection is also used by *L. polaris* (Rochette et al. 1994) and *A. vulgaris* (Zafiriou 1972, David Drolet, Université Laval, pers. comm.) in locating food resources. In addition, we observed healthy urchins feeding on damaged (but living) conspecifics. Cannibalism by green sea urchins has been previously reported (Himmelman & Steele 1971, Propp 1977, Hagen 1987). On 2 July, we transplanted 17 urchins showing large areas of bald-urchin-disease (see below) from the mortality area to the grazing front where other urchins were healthy and 11 (65%) of these were being cannibalized when we returned 24 h later.

The quadrat sampling on 18 June indicated an urchin density of 12 ind.m^{-2}, whereas the sampling on 22 August, two months after the mortality event, indicated a density of 35 ind.m^{-2}. Densities outside the mortality site were much higher (e.g. 90 ind.m^{-2} in July). We observed no macroalgal colonization of the mortality site during the summer, presumably because the grazing of the urchins that were there (and had presumably immigrated into the site) was sufficient to prevent algal growth. This agrees with Chapman's (1981) observation that densities of 20 urchins.m^{-2} are sufficient to prevent the macroalgae colonization.

A large number (36%) of living urchins in the mortality site had lesions with a loss of spines and abnormal epidermal areas that were green to dark brown in color (Fig. 1b). These lesions were predominantly on aboral surfaces and appeared to correspond to bald-sea-urchin-disease as described for *trongylocentrotus droebachiensis* (in Nova Scotia) and other species (Jangoux 1984, Roberts-Regan et al. 1988).

Sampling at three sites showed that the incidence of bald-urchin disease was low compared to the mortality site. Nevertheless, it varied between grazing front and barrens (t-test, $t_2 = 3.17$, $p = 0.02$; Fig. 3). The incidence was no higher in these two habitats at Ile au Marteau, near the mortality site. Roberts-Regan et al. (1988) also report a low incidence of infected urchins (7%) for healthy urchin populations in Nova Scotia. Bald-urchin lesions do not usually cause death, except when they cover >30% of the surface of the test (Maes & Jangoux 1984, Gilles & Pearse 1986). Urchins with expansive lesions (Fig. 1b) were common at the mortality site in early June.

Our comparison of the rate of infection of healthy urchins that were placed in cages in three habitats showed no infection 4 days later. However, we did observe infection of urchins that we had damaged by removing spines from part of the aboral surface. A 1-way ANOVA showed differences ($F_{2,6} = 10.75$, $p = 0.01$, SNK test, $p < 0.05$, Table 1) with habitat (grazing front, algal mat and barrens) for the damaged urchins. The infection rate was highest (67%) for urchins placed underneath the algal mat at the mortality site, least (0%) for urchins placed on the barrens near the mortality site, and intermediate (30%, but not different from the barrens) for urchins placed at the grazing front. These observations suggest that the macroalgal mat increased the likelihood of infection and probably contributed to the mass mortality.

No apparent injuries to the epidermis were observed for the urchins maintained for 14 h in the wave tank, in either the treatments with or without algal abrasion. All of the urchins in the treatment with algal abrasion stayed attached to the bottom under the algae during the trial, however, they moved, to the top of the algae once the wave action was stopped. Thus, urchins seem to be able to tolerate a considerable amount of algal abrasion. However, we cannot state what the result would be if such abrasion occurred over longer periods (days to weeks).

We observed a second mortality event on 12 August at another location, Goéland West. In this case we estimated that nearly 600 urchins were dying (quadrat sampling) and we counted three dying *Leptasterias polaris* and one dying *Asterias vulgaris*. In this case strong wave action 2 days earlier detached brown algae (mainly the filamentous alga *Chordaria flagelliformis* which grows near low tide level) and the algae accumulated in shallow water (2 m). Our previous sampling (18 July) showed that this area had supported a high density of urchins (219 urchins.m^2). We suspect that these mortalities, this time of sea stars and urchins, were due to anoxic conditions, as anoxia was indicated by the dark organic matter under the algal mat (no such indications of anoxia were observed at the mass mortality site at Ile au Marteau). Mortality of invertebrates due to oxygen deficiency caused by an accumulation of drift algae has also been reported by Scheibling & Raymond (1990) and Diaz & Rosenberg (1995). The mortalities did not appear to be propagated outside of the mat.

The development of bald-urchin-disease appears to require both mechanical abrasion (of the surface of the urchin) and the presence of water-borne pathogens. Abrasion of the test undoubtedly occurs in the field, from physical perturbations and also from interactions with other organisms, including attacks by conspecifics. Also, the occurrence of low levels of infection in barrens and grazing fronts demonstrates the presence of the pathogen in the subitdal zone. Our observations of infection rates of urchins in different habitats, indicate that damaged urchins were more

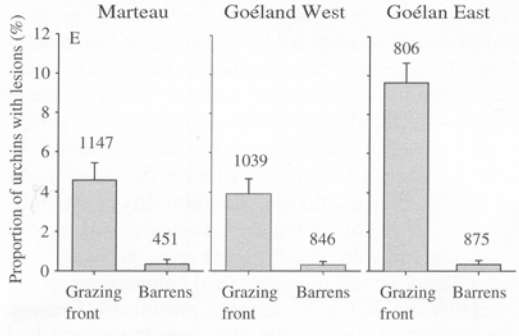

Figure 3. Proportion of sea urchins with lesions on the outer body wall in two habitats, the grazing front and urchin barrens, at Petite Ile au Marteau, Goéland West and Goéland.

Table 1. Percentage (Standard Error) of undamaged urchins and damaged urchins (spines removed from an area of the aboral surface) that became infected when placed in cages for 4 d in three habitats, the urchin-barrens, the grazing front and the algal mat. The infection rates for damaged urchins in habitats sharing the same letter are not different (SNK test, $p > 0.05$).

	Proportion infected	
Habitat	Undamaged urchins	Damaged urchins
Barrens	0.0 (0)	0.0 (0)[a]
Grazing front	0.0 (0)	30.0 (15.3)[a]
Algal mat	0.0 (0)	66.7 (8.8)[b]

likely to become diseased if they were close to macroalgal blades (infections were least on barrens, increased at the grazing sites next to the algal fringe, and very high under the macroalgal mat; Table 1, Fig. 3). This pattern suggests that macroalgal blades provide a substrate for the pathogen and that the pathogen was most abundant near the mat of algal debris. The estimated volume of the mat was $>1300\,m^3$ and the decaying algae could have been a rich source of the pathogen. Once the mortality began, infected urchins likely provided an additional source of the pathogen.

In conclusion, we documented a mass mortality of urchins on an urchin barrens in the Mingan Islands which was associated with two events, the scouring by icebergs of macroalgae from the kelp fringe and the accumulation of the algal debris on the barrens. The urchins appeared to have the bald-sea-urchin disease, which is caused by bacterial pathogens that are commonly found in the subtidal environment. The combination of background levels of physical abrasion, possibly additional abrasion and anoxia from prolonged exposure to a large macroalgal mat, and a rich bacterial flora associated with the algal mat and dying urchins likely contributed to the localized necrosis on the surface of urchins and the mass mortality event.

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