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COMPARISON OF A CURRENT EELGRASS DISEASE TO THE WASTING DISEASE IN THE 1930S

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ABSTRACT

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A comparison is made of the wasting disease that struck the whole Atlantic population of *Zostera marina* L. in the 1930s and a current outbreak of a rather similar disease in *Z. marina* beds along the north-eastern coasts of the U.S.A. Although the disease phenomena on the plants appear to be very similar, disease-related declines of *Z. marina* are at present still very local. In Europe, diseased plants have been found, but no declines have been observed.

The wasting disease in the 1930s was not investigated before the epidemic reached a devastating stage. Present observations may indicate that a new widespread die-off may be developing. In order to facilitate the study of the current epidemic, a scenario of disease and related decline, with several variants, has been elaborated, based on the existing knowledge of the epidemic of the 1930s, but also clearly showing the gaps in this knowledge.

INTRODUCTION

The wasting disease of eelgrass, *Zostera marina* L., in the 1930s was investigated intensively after its epidemic character had been recognized (Adams, 1933; Cotton, 1933; Lewis, 1932; Lewis and Taylor, 1933; Petersen, 1932, 1934; Renn, 1934; Taylor, 1933). The disease first appeared on the Atlantic coasts of North America in 1930 and subsequently it was discovered in 1931 on the Atlantic coast of Europe (Cotton, 1933; Cottam, 1934). Within 2 years, *Z. marina* declined to the point of almost complete disappearance in North America as well as in Europe. There followed a period in which eelgrass showed slight recovery, often followed by new outbreaks of the disease (Addy and Aylward, 1944). True recovery of eelgrass beds did not take place before the period

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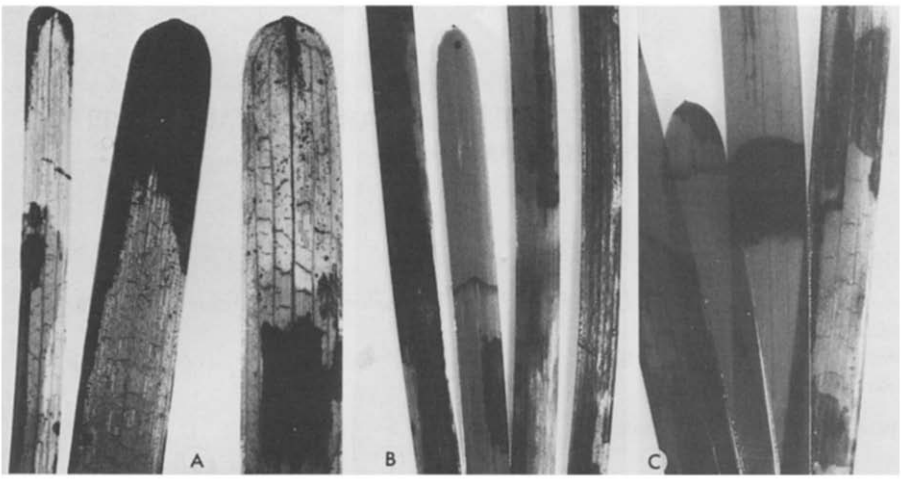

Fig. 1. (A) Leaf-tips of diseased *Zostera marina* from the Dutch Waddensea, photographed during the epidemic of the 1930s (by the courtesy of Dr. S.J. de Groot). (B–C) Part of an infected shoot; the youngest leaf (with leaf-tip) already shows symptoms, Roscoff, June 1986.

1955–1965 (Blois et al., 1961; Rasmussen, 1977). However, eelgrass populations did not re-occupy all their former habitats. In some areas, where the species had been very dominant before the wasting disease outbreak, it has failed to return, e.g., the northern shore of Long Island (New York, U.S.A.), the Dutch Waddensea and the Königshafen, Sylt (F.G.R.).

At present, a disease and related decline of eelgrass is occurring in New England, U.S.A. First recognized in the Great Bay Estuary on the Maine–New Hampshire border, the current disease presents many of the same symptoms described by researchers in the 1930s: small black patches spread on the leaves of otherwise healthy looking plants, which finally become black (Figs. 1 and 2), leading to death and detachment of the leaves (Pokorny, 1967; Short et al., 1986). Intensive infection throughout a large area leads to a decline in diseased eelgrass populations: in Great Bay Estuary, large infected beds in the Piscataqua River and Little Bay disappeared between 1981 and 1984, and in 1985 through 1987 parts of eelgrass beds in Great Bay have disappeared.

Eelgrass plants with infected leaves have been found during 1985–1987 in the U.S.A. in Penobscot Bay (ME), Maquoit Bay (ME), Great Bay (NH) (Fig. 2b–d), Rye Harbor (NH), Cape Ann (MA), Lynn Harbor (MA), Charlestown Pond (RI), Mystic River (CT) and Beaufort (NC) (F.T. Short, personal observation, 1987). Diseased plants have also been found along the coast of Nova Scotia, Canada (Paine and O'Brien, personal observation, 1986). Further, plants with disease symptoms have been observed along the Pacific coast of the U.S.A. in Puget Sound, Washington (Short et al., 1987). Similar

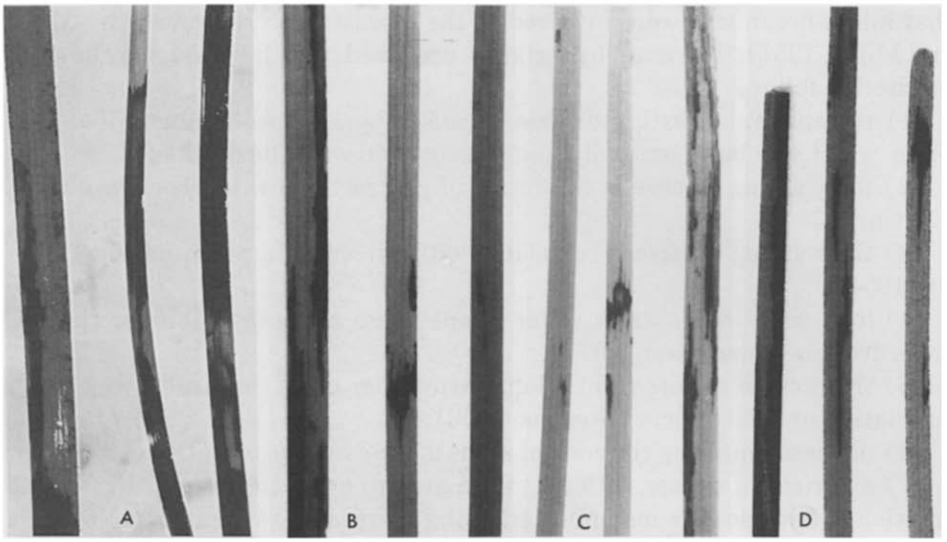

Fig. 2. Leaves with disease symptoms in various degree, (A) Roscoff, June 1987. (B–D) Great Bay estuary, New Hampshire, 1986.

symptoms of infection have been detected in 1986 in Europe in Roscoff [Finistère, France (Figs. 1b–c, 2a)], St. Efflam (Côtes du Nord, France) and in the river Exe near Exmouth (Devon, England), In 1987, the infection of plants had increased in Roscoff, no eelgrass die-off was evident. In the river Exe the eelgrass did not show any symptoms in 1987. In The Netherlands, however, a decline of eelgrass, characterized by blackening of the leaves, occurred in the Grevelingen lagoon (den Hartog, personal observation).

It is still unclear whether the current disease is identical to that of the 1930s and whether it will produce a widespread loss of eelgrass populations. However, the similarities are such that the current infection presents a unique opportunity for the study of a disease and epidemic process from early in the event. Although the wasting disease of the 1930s is probably a classic example of a marine epidemic (Andrews, 1976), the fact that most investigations into its cause started after the epidemic had exercized its greatest impact may account in part for the lack of agreement among researchers concerning the etiology of the disease. In reviews, numerous authors have proposed various hypotheses concerning the cause of the wasting disease (Tutin, 1938; Young, 1943; Johnson and Sparrow, 1970; Rasmussen, 1977; den Hartog, 1987). The only conclusion to be reached from these differing hypotheses is that the cause of the 1930s wasting disease has never been conclusively determined (den Hartog, 1987). Many of the early investigations lead to the conclusion that *Labyrinthula macrocystis* Cienkowski, an infectious slimemold-like protist was the organism responsible for the epidemic. Other investigators, however, suggested that the decline was induced by environmental causes, although not denying

that micro-organisms were involved in the disease process (review by Milne and Milne, 1951). The main hypotheses, proposed over the years, may be summarised as follows:

(1) the epidemic wasting disease is caused by a micro-organism, *Labyrinthula*, which has been generally found associated with the decline;

(2) the wasting disease is the result of protracted low levels of insolation (Tutin, 1938);

(3) the wasting disease is correlated with extremes in precipitation (Martin, 1954);

(4) long-term increases in water temperature are responsible for the eelgrass decline (Rasmussen, 1977);

(5) the disease results from a long-term lunar cycle that influences ocean circulation or tidal effects (Stevens, 1936);

(6) oil waste entering the coastal areas is the causal agent (Duncan, 1933);

(7) a variety of causes, differing from area to area, are responsible for the creation of adverse circumstances, enabling the disease to break out. No single phenomenon seems widespread enough to explain the almost simultaneous occurrence of wasting disease in the whole North Atlantic (den Hartog, 1987).

The idea that the 1930s decline of eelgrass resulted from the action of a biotic pathogen combined with environmental influences that would favour the disease has particularly been worked out by Young (1938, 1943) for salinity and by Renn (1937) for temperature. Studies by Renn (1936) demonstrated the contagious nature of the wasting disease, but proof of the causal agent was never completed. *Labyrinthula macrocystis* was consistently associated with the 1930s disease, but its pathogeny was not demonstrated. Now, a species of *Labyrinthula* has been shown conclusively, via Koch's postulates, to be the cause of the current eelgrass epidemic on the Atlantic and Pacific coasts of North Ameria (Short et al., 1987).

No conclusive work on disease pathogens was carried out during the 1930s wasting disease. Our work on the current disease is in its preliminary stages, and an epidemic of the proportions of the 1930s, if there is to be one, has yet to manifest itself. Therefore, in establishing a scenario for the current disease, we are comparing two sets of incomplete observations, albeit incomplete for different reasons. However, many similarities exist between descriptions of the 1930s phenomena of both disease and decline and what we are currently seeing on the coasts of North America and Europe. It is therefore useful to compare what we see now with the symptomatology and epidemiology as noticed in the 1930s for what may be revealed about the ecology of a disease in a widespread and important macrophyte.

THE CURRENT DISEASE AND DECLINE

The current infection of eelgrass plants is distinct and easily recognizable; it appears as black (or dark brown) lesions on otherwise healthy and rapidly

growing leaf tissue (Figs. 1 and 2). Although easily overlooked by the casual observer, careful examination of diseased shoots shows that the lesions appear on even the new, young leaves of a shoot (Fig. 1b–c). It is this characteristic that provides the best evidence of the disease, since even under senescing conditions, necrotic areas on the youngest leaves of healthy eelgrass plants are rarely found. Dark brown longitudinal lesions that spread along young eelgrass leaves were described by observers of the 1930s wasting disease (Fig. 1a).

Environmental influences on the current disease are being observed in the field and tested in mesocosm tanks. Preliminary experiments on the present eelgrass infection in mesocosm tanks at the Jackson Estuarine Laboratory in New Hampshire, U.S.A. have conclusively demonstrated the impact of salinity in controlling the infection, i.e., when infected plants were grown for a year in a tank having salinities of $<10‰$ the infection did not spread to new leaf tissue. In an adjacent tank having salinities between 15 and 30‰, the infection spread to the entire plant. Thus, under current conditions in the field, it is anticipated that the infection of eelgrass would be greatest at the seaward end of estuaries. This finding is confirmed by observations of the major eelgrass loss in the higher salinity end of the Great Bay Estuary (Short et al., 1986). It was observed in the 1930s that eelgrass survived in the low salinity areas of N. American estuaries (Pokorny, 1967). In Europe, the disease struck the *Z. marina* populations in Denmark, but the populations in the brackish Baltic Sea remained free of disease. In England, France and The Netherlands, brackish-water populations and eulittoral populations of *Z. marina* survived (Wilson, 1949; Rasmussen, 1977; den Hartog, 1987).

The effects of temperature and light on the spread of the infection appear to be related to the direct influence of these factors on plant physiological processes and the plants' metabolic balance. The infection in the mesocosm tanks spreads most rapidly in the late summer and autumn, at the time of maximum water temperatures when light levels are diminishing.

Infected tissue is generally found on the plants throughout the year, although the expansion of necrotic spots is slow during the cold winter season. The lesions expand rapidly in the summer, but the rapid eelgrass growth during this period maintains healthy plants. However, in late summer the shorter daylength, lower light intensity and still high water temperatures affect the physiological balance of the eelgrass as there is less time for photosynthesis, while respiration remains the same. This results in a gradual reduction of net eelgrass growth. The changing metabolism, in conjunction with the energy required to maintain a large number of leaves, initiates the metabolic stress that may create an opportunity for the disease. We can consider the current disease as a host–parasite interaction, where under normal environmental circumstances the parasite infection weakens the host, i.e., the eelgrass, but does not destroy it. This is not to say that the infection is present in eelgrass populations at all times. In fact, examination of extensive eelgrass collections from

New Hampshire and Rhode Island in the 1970s revealed no necrotic or infected areas (F.T. Short, personal observation, 1986).

SCENARIO FOR DISEASE AND DECLINE

The situation that initiates the disease-related declines is not yet known. In spite of this, a scenario with several variants can be formulated as a working hypothesis. There are two possibilities for the widespread occurrence of the disease. First, the pathogen is harboured in one location, spreads widely and the disease symptoms begin to appear. Second, the pathogenic organism is in fact always present in eelgrass, either as an asymptomatic obligate form, as a secondary decomposer, as a resistant structure, or as a mutation of a pathogenic form.

Evidence for a central locus for the current disease is suggested by the initial observations of symptoms and die-off in New Hampshire and Massachusetts. However, insufficient evidence is available from the 1930s to determine if that disease might have spread from a single location, nor is there evidence of the current disease spreading from New Hampshire, where it was first discovered, to other locations. In fact, although mechanisms can be postulated for spread of the disease along the Atlantic coast, it is difficult to hypothesize a reasonable mechanism for transport of the pathogen from the Atlantic to the Pacific in such a short time period.

A more likely situation is that the pathogen is widespread and that for some reason, in the 1930s and again in the 1980s, it has parasitized and decimated eelgrass populations. If the pathogen producing the wasting disease persists in eelgrass beds, then it must exist in a less aggressive form for long periods between outbreaks. Possible mechanisms for the pathogen's continuous presence in the environment include the organism being an obligate parasite, which can exist at low population densities, producing only a rare occurrence of symptoms, or it could be asymptomatic. In the case of an obligate parasite, a population boom of the pathogenic organism could be brought on by some external condition creating a stress in *Zostera* populations, thus making the plant more susceptible to the pathogen, or environmental conditions could alter the virulence of the micro-organism. An unlikely mechanism for an organism's continuous, but asymptomatic presence in the environment is the possibility of the disease organism mutating from a non-aggressive secondary decomposer to a pathogenic form, i.e., from a saprobic to a parasitic organism. Again, widespread environmental conditions favourable to the microorganism or unfavourable to *Zostera* could initiate an epidemic. A further possibility is that a long-term life cycle exists for the organism, a cycle which includes an inactive period or the formation of a resistant structure (cyst). In this case, either the life cycle of the pathogen or an environmental trigger could initiate the disease.

In many of these possible cases some external factor, acting on a global scale,

initiates the epidemic. Such an external factor could act in two possible ways. First, the epidemic disease could be activated by still unknown environmental circumstances that deregulate the metabolism of eelgrass, predisposing it to infection. The second possibility is that some environmental circumstance could activate a population expansion of an aggressive form of the pathogen. In either case, a synchronous outbreak of the disease and a subsequent decline could take place over a very large area. It seems apparent that the unknown environmental circumstances which enable the pathogen to strike its host relate to long-term cosmic cycles which exercise their influence on *Zostera* and the pathogen by changing global climatic conditions and oceanic current and tidal patterns.

Since the infection of eelgrass becomes most severe in the late summer and autumn, i.e., the time of maximum water temperature and decreasing light levels and, therefore, the time of maximum metabolic stress to the plants, an unusually warm or dark year could enhance the infection to the point of decline. A number of combinations of environmental and climatic conditions could set the stage for this decline scenario: (1) an extended period of cloud cover heavy enough to decrease ambient light levels while allowing heating from long-wave radiation and back-radiation; (2) a dry summer and autumn causing an increase in coastal water temperatures. These or other combinations of conditions favouring the disease may additionally stress infected populations during the time of the year when their metabolic processes render them most vulnerable.

Stevens (1936) related the eelgrass decline to a long-term lunar cycle, which influences oceanic current patterns. Glémarec (1979) correlated the pattern of decline and increase in eelgrass with a 55-year solar cycle, also known as the cycle of Russell (Southward et al., 1975; Cushing, 1982). The exact nature of the relationship between these cycles and the *Zostera* decline is not known. However, the timing of the current outbreak fits within these previously described cyclic patterns.

DISCUSSION

Our scenario presents a hypothetical reconstruction of the mechanisms that might have led to the outbreak of the past and present disease in eelgrass. It is based on numerous reports and observations of the wasting disease of the 1930s, supplemented with new data and views in light of the current epidemic.

Now it appears that most of the hypotheses listed in the introduction are not exclusive and competing explanations of the wasting disease, but reconcilable components of a larger scenario. The proposed direct causes of the 1930s epidemic, viz. *Labyrinthula*, as the pathogenic organism (Hypothesis 1), low

levels of insolation (Hypothesis 2), extremes in precipitation (Hypothesis 3) and long-term increases in water temperature (Hypothesis 4) fit easily within the framework of the basic scenario described above. The hypothesis that long-term cycles of ocean circulation influence the general climate and particularly the temperature of coastal waters, and so can push the disease from infection to decline (Hypothesis 5), also fits within the scenario. Hypothesis 7 is in fact already reconciled, as it states that disease-related declines can be triggered by various local causes. It can be fitted within the basic scenario, if it is interpreted in the sense that local causes can create the circumstances for an outbreak of the disease, and may enlarge its effects. Only Hypothesis 6 does not fit within these scenarios: oil causes quite different damage patterns to eelgrass and is not known to trigger disease (den Hartog, 1986).

In the proposed scenario, the similarities between the wasting disease of the 1930s and the current disease are stressed. However, there are some differences. In the 1930s, the decline of eelgrass took place simultaneously on both sides of the Atlantic. The current disease has led to local declines in some New England eelgrass populations of North America. In Roscoff, France, the disease was found in declining eelgrass beds, but was certainly not the cause of the decline (den Hartog, personal observation, 1987). However, nothing is known about the initial situation of the eelgrass beds of the 1930s. The disease was discovered when it was already in a devastating stage. Disease-related declines on the scale of the 1930s have not yet been observed anywhere in North America or Europe. A further difference is that Pacific eelgrass populations were struck by the wasting disease in the 1940s (Moffitt and Cottam, 1941), after the 1930s decline in the Atlantic. However, diseased plants have now been found on the Pacific coast in Puget Sound, Washington, U.S.A. (Short et al., 1987), simultaneously with our findings of the Atlantic eelgrass disease. Whether eelgrass declines will develop on both coasts at the same time remains to be seen.

Not all declines of seagrass beds are caused by disease. Recently, declines resulting from environmental pollution and human activities in the coastal zone have been reported from all over the world (den Hartog and Polderman, 1975; Péres and Picard, 1975; Jones and Tippie, 1983; Kemp et al., 1983; Orth and Moore, 1983; Cambridge and McComb, 1984). The loss of eelgrass in the Chesapeake Bay has been variously attributed to eutrophication, herbicides from runoff and increased turbidity from development (Jones and Tippie, 1983). A seagrass decline in southwestern Australia was linked to industrial expansion and alteration of harbour circulation (Cambridge and McComb, 1984). Analysis of the eelgrass decline within the non-tidal Grevelingen lagoon in The Netherlands suggests that increased nutrient loading produced toxic sediment conditions (Nienhuis, 1983).

The process of plant die-off in these pollution-related declines is very distinct from the wasting disease decline of the 1930s and the present disease

phenomenon. However, eelgrass populations under stress of pollution may be also predisposed to the disease. As pollution is usually most massive in the low-salinity upper reaches of estuaries and embayments, and the disease is found in the saline lower reaches, the joint effect of pollution and disease may be the total elimination of eelgrass. In an unpolluted embayment, the upper reaches could function as a refuge from where, in time, recolonization could take place; this was the case in the 1930s.

The importance of eelgrass to the health of estuarine and coastal waters is well established. Now this important macrophyte is facing the threat of disease as well as pollution. Further studies are needed to clarify the yet unknown aspects of the disease and the ability of eelgrass to recover under current conditions.

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REFERENCES

Adams, J., 1933. *Zostera marina* on Anticosti Island. Nature (London), 132: 277.

Addy, C.E. and Aylward, D.A., 1944. Status of eelgrass in Massachusetts during 1943. J. Wildl. Manage., 8: 269-275.

Andrews, J.H., 1976. The pathology of marine algae. Biol. Rev., 51: 211-253.

Blois, J.C., Francaz, J.M., Gaudichon, M., Gaudichon, S. and LeBris, L., 1961. Observations sur les herbiers à Zostères de la région de Roscoff. Cah. Biol. Mar., 2: 223-262.

Cambridge, M.L. and McComb, A.J., 1984. The loss of seagrasses in Cockburn Sound, Western Australia. I. The time course and magnitude of seagrass decline in relation to industrial development. Aquat. Bot., 20: 229-243.

Cottam, C., 1934. Past periods of eelgrass scarcity. Rhodora, 36: 261-264.

Cotton, A.D., 1933. Disappearance of *Zostera marina*. Nature (London), 132: 277.

Cushing, D.H., 1982. Climate and Fisheries. Academic Press, London, New York, 373 pp.

Den Hartog, C., 1986. Effects of oil pollution on seagrass beds. Proceedings of the First International Conference on the Impact of Oil Spill in the Persian Gulf, 20-27 May 1984, at The University of Tehran, pp. 255-268.

Den Hartog, C., 1987. "Wasting disease" and other dynamic phenomena in *Zostera* beds. Aquat. Bot., 27: 3-14.

Den Hartog, C. and Polderman, P.J.G., 1975. Changes in the seagrass populations of the Dutch Waddenzee. Aquat. Bot., 1: 141-147.

Duncan, F.M., 1933. Disappearance of *Zostera marina*. Nature (London), 132: 483.

Glémarac, M., 1979. Les fluctuations temporelle des peuplements benthiques liées aux fluctuations climatiques. Oceanol. Acta, 2: 365-371.

Johnson, T.W., Jr. and Sparrow, F.K., 1961. Fungi in Oceans and Estuaries. J. Cramer, Weinheim, selected pages.

Jones, G.A. and Tippie, V.K., 1983. Chesapeake Bay program: Findings and Recommendations (USEPA), p. 48.

Kemp, W.M., Twilley, R.T.R., Stevenson, J.C., Boynton, W.R. and Means, J.C., 1983. The decline of submerged vascular plants in the upper Chesapeake Bay: Summary of results concerning possible causes. Mar. Technol. Soc. J., 17 (2): 78-89.

Lewis, H.F., 1932. The eelgrass situation on the Atlantic coast. Am. Game Conf. Trans., 19: 411-423.

Lewis, H.F. and Taylor, W.R., 1933. Disappearance of *Zostera* in 1932. Rhodora, 35: 152-154.

Martin, A.C., 1954. A clue to the eelgrass mystery. Transactions of the 19th North Amerian Wildlife Conference, Washington, DC, pp. 441-449.

Milne, L.J. and Milne, M.J., 1951. The eelgrass catastrophe. Sci. Am., 184 (1): 52-55.

Moffitt, J. and Cottam, C., 1941. Eelgrass depletion on the Pacific coast and its effect upon the Black Brant, U.S. Dep. Interior Fish. Wildl. Serv., Wildl. Leaflet 204, 26 pp.

Nienhuis, P.H., 1983. Temporal and spatial patterns of eelgrass (*Zostera marina* L.) in a former estuary in The Netherlands, dominated by human activities. Mar. Technol. Soc. J., 17 (2): 69-77.

Orth, R.J. and Moore, K.A., 1983. Chesapeake Bay: An unprecedented decline in submerged aquatic vegetation. Science, 222: 51-52.

Péres, J.M. and Picard, J., 1975. Causes de la raréfaction et de la disparition des herbiers de *Posidonia oceanica* sur les côtes françaises de la Méditerranée. Aquat. Bot., 1: 133-139.

Petersen, H.E., 1932. Wasting disease of eelgrass (*Zostera marina*). Nature (London), 132: 1004.

Petersen, H.E., 1934. Wasting disease of eelgrass (*Zostera marina*). Nature (London), 134: 143-144.

Pokorny, K.S., 1967. *Labyrinthula*. J. Protozool., 14: 697-708.

Rasmussen, E., 1977. The wasting disease of eelgrass (*Zostera marina*) and its effects on environmental factors and fauna. In: C.P. McRoy and C. Helfferich (Editors), Seagrass Ecosystems: A Scientific Perspective. Marcel Dekker, New York, pp. 1-52.

Renn, C.E., 1934. Wasting disease of *Zostera* in American waters. Nature (London), 134: 416.

Renn, C.E., 1936. The wasting disease of *Zostera marina*. A phytological investigation of the diseased plant. Biol. Bull., 70: 148-158.

Renn, C.E., 1937. The eel-grass situation along the middle Atlantic coast. Ecology, 18: 323-325.

Short, F.T., Mathieson, A.C. and Nelson, J.I., 1986. Recurrence of the eelgrass wasting disease at the border of New Hampshire and Maine. U.S.A. Mar. Ecol. Prog. Ser., 29: 88-92.

Short, F.T., Muehlstein, L.K. and Porter, D., 1987. Eelgrass wasting disease: cause and recurrence of a marine epidemic. Biol. Bull., 173: 557-562.

Stevens, N.E., 1936. Environmental conditions and the wasting disease of eelgrass. Science, 84: 87-89.

Southward, A.J., Butler, E.I. and Pennycuyck, L., 1975. Recent cyclic changes in climate and in abundance of marine life. Nature (London), 253: 710-715.

Taylor, W.R., 1933. Epidemic among *Zostera* colonies. Rhodora, 35: 186.

Tutin, T.G., 1938. The autecology of *Zostera marina* in relation to its wasting disease. New Phytol., 37: 50-71.

Wilson, D.P., 1949. The decline of *Zostera marina* L. at Salcombe and its effects on the shore. J. Mar. Biol. Assoc. U.K., 28: 395-412.

Young, E.L., III, 1938. *Labyrinthula* on Pacific coast eelgrass. Can. J. Res., 16: 115-117.

Young, E.L., 1943. Studies on *Labyrinthula*. The etiologic agent of the wasting disease of eelgrass. Am. J. Bot., 30: 586-593.