Wasting disease and present eelgrass condition

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Tidal Waters Division

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PREFACE

This study was carried out from June 16th to December 31st 1987, and April to September 1988 at the Laboratory of Aquatic Ecology of the Catholic University of Nymegen, the Netherlands, for the Dutch Ministry of Transport and Waterworks, under project no. DGW 896/INDICAT, titled: "Onderzoek van de in de Waddenzee voorkomende begroeiingen met *Zostera marina*, als indicator voor de toestand van het Waddenzee milieu". Work at the lab was supervised by Prof. C. den Hartog, and coordinated with the ministry via Drs. V. De Jonge. I am much indebted to both for this opportunity, and for the freedom I enjoyed in pursuing the "wasting disease" problem.
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ABSTRACT

Wasting disease of the 1930's

An attempt was made to correlate wasting disease of the 1930's with factors such as seawater temperature, salinity and sunshine, in order to establish a possible link with mechanisms behind climatic cycles, such as the sunspot cycle, and the related Russell cycle.

Europe. Salinity and seawater temperature fluctuations could not have contributed significantly to eelgrass stress, but there is a good correlation with reduced sunshine and raised turbidity levels. This correlation was verified with an existing computor simulation model for eelgrass growth. In the Netherlands, the response of eelgrass to adverse light conditions was probably much enhanced by the activities associated with the closure of the Zuyder Sea in 1932.

USA. Salinities and temperatures were above average, but certainly not uniquely or dramatically so, while sunshine was well above average. No possible causal link could be established between climatic factors and wasting disease initiation. This lends support to the suggestion by Short *et al* (1987) that the wasting disease epidemic was perhaps caused by the emergence of a pathogenic strain of *Labyrinthula*.

Present conditions

Recent reports of recurrence of wasting disease have made it desirable to re-assess present eelgrass condition.

The Netherlands. Eelgrass in the Dutch Wadden Sea has declined to the point of virtual disappearance. Probable main causes are: increased turbidity, competition with macro-algae and toxic pollution. A simulation model experiment shows that eelgrass growth is almost impossible under the present environmental conditions except, perhaps, for marginal growth of annual specimens. A die-off of eelgrass beds occurred in Lake Grevelingen in 1987, but plants did not clearly display wasting disease symptoms. Simulation experiments showed that reduced amounts of sunshine may have played an important role in this decline. The wasting disease-like symptoms did appear later in the season, and a pathogen-like strain of *Labyrinthula* (the probable wasting disease pathogen of the 1930's) was isolated from infected tissue. The situation is dormant in these winter months, but it would be very interesting to monitor the situation in the next growing season.

Roscoff, France. Eelgrass plants from Roscoff show wasting disease symptoms, and several *Labyrinthula spp.* were isolated from lesions on leaves. One of them appeared to be pathogenic. These symptoms occur in herbarium specimens from the mid-1970's onwards, but no eelgrass decline has occurred so far, due to the infection.

USA. Reports from the USA record several local eelgrass declines, and a widespread occurrence of a pathogenic strain of *Labyrinthula*. However, one cannot yet conclude that an epidemic such as that of the 1930's is at hand.

Possible cyclic nature of wasting disease ?

The data are too incomplete to be conclusive, but one may tentatively state that there is no apparent causal link between factors such as temperature, salinity and sunshine, and wasting disease, that can account for the scale of the epidemic of the 1930's. One can only state that sunshine probably played an important role in Europe. Without an apparent causal link, one can only speculate about the effects of the sunspot cycle on eelgrass. If a second wasting disease epidemic develops out of the present "first signs", one can conclude that the sunspot cycle and wasting disease are remarkably well in phase. A causal link must then be sought in another direction, such as the introduction and removal of species by encroaching, warmer, southern waters.

Part 1

INTRODUCTION

Wasting disease symptoms and history

The term "wasting disease" is applied to the epidemic that struck the North Atlantic population of the marine phanerogam *Zostera marina L.* in the early 1930's. The plants declined and wasted away, hence the name. It is the greatest known decline of this species, as it was almost wiped out simultaneously in much of its radius on both the American and European sides of the North Atlantic. The symptoms appeared to be uniform. Small brown lesions developed, even on young leaves, that gradually became larger and darker, and finally covered most of the leaf. This process took several weeks, and these dead leaves were eventually sloughed off, sinking to the sea-bed. Rhizomes were generally reported to be unaffected, at least in early stages of the disease (Van der Werff, 1934), but others report discoloration (den Hartog, 1987). In any case, rhizomes usually survived for one or more seasons, but eventually succumbed to this repeated defoliation that exhausted plant reserves.

The disease first appeared in 1930 on the east coast of North America, in Virginia (Lewis, 1932; Huntsman, 1932), and was first reported in France, on the European side of the North Atlantic, in 1931 (Fischer-Piette *et al*, 1932). It was first thought that this time difference between first recordings represented a spreading of the disease from America to Europe, but later reconstructions indicate a synchronous occurrence (Rasmussen, 1977; den Hartog, 1987). As the extent of the epidemic became evident, it received much attention from the scientific world. After initial indications that a bacteria (Fischer-Piette *et al*, 1932) or an Ascomycete fungus, *Ophiobolus halimus* (Petersen, 1936; Mounce and Diehl, 1934) was the etiologic agent, there gradually emerged a consensus that a slime mold-like protist, *Labyrinthula macrocystis* Cienkowski, was the primary pathogen (Renn, 1935, 1936; Van der Werff, 1938; Young, 1943). However, this did not suffice to explain the wasting disease phenomenon. *L. macrocystis* was found to occur as an endo-parasite or saprobic organism in many normal eelgrass stands, such as the then unaffected Pacific population (Young, 1938), and it was also found to be common as a normal secondary decomposer in other marine plants, such as algae and other seagrasses (Young, 1943; den Hartog, 1987). The question remained, what had made *L. macrocystis* virulent ? Many hypotheses were proposed, such as siltation (Milne and Milne, 1951), pollution (Milne and Milne, 1951), precipitation extremes (Martin, 1954), salinity extremes (Young, 1938, 1943), deficiency of sunlight (Tutin, 1938) and abnormally high water temperatures (Renn, 1937; Rasmussen, 1977). None of these were conclusive, however, and at best they showed a reasonable correlation with decline in eelgrass beds, albeit never being generally applicable to the entire North Atlantic region.

During the period following wasting disease, eelgrass beds showed repeated recovery and new outbreaks (Addy and Aylward, 1944; Rasmussen, 1977; den Hartog, 1987). True recovery did not seem to take place until the period 1955-1965, and even then it was not complete in many areas (Rasmussen, 1977; Short *et al*, 1988). Outlines on the history of the epidemic are given by Johnson and Sparrow (1961), Pokorny (1967), Rasmussen (1977), and more recently by Short *et al* (1986), den Hartog (1987), Whelan and Cullinane (1987) and Short *et al* (1988).

Renewed interest

Interest in wasting disease was re-awakened by recent reports of a possible recurrence of the disease, both in North America (on the east coast, from Nova Scotia down to North Carolina, and on the west coast, in Washington; Short *et al*, 1986, 1988) and Europe (Exmouth, England, and St. Efflam and Roscoff, France; Short *et al*, 1988). The situation seems to be parallel to the epidemic of the 1930's: the overall symptoms on the plant are very similar, a virulent strain of *Labyrinthula* has been isolated from infected North American plants (Short *et al*, 1987b), and there seems to be a simultaneous occurrence on both sides of the Atlantic. Whether this phenomenon will manifest itself on such a widespread and destructive scale as in the 1930's remains to be seen. Disease related die-offs of eelgrass beds have been confirmed in Maine, New Hampshire and Massachusetts (Short *et al*, 1987), but the authors are reluctant to label it an epidemic. No clearly disease-related die-offs have been recorded in Europe up to now. Infected plants were common in Roscoff, but this was apparantly not causing any die-off (Den Hartog, p.c. 1987). Eelgrass decline was reported in Lake Grevelingen, the Netherlands, in the late summer of 1987 (Van Lent, p.c. 1987; personal observation), but wasting disease-like symptoms were not clearly present at the time.

Aims of present study

The objectives of this study were:

a. An analysis of climatic and oceanographic data (esp. water temperature, sunshine, precipitation and salinity) in order to elucidate the possible relationship between eelgrass population dynamics and sunspot activity cycles, as proposed by Glémarec (1979).

b. In a broad sense, a study of the condition of *Zostera marina* stands in the Dutch Wadden Sea, whereby total area, biomass and condition of individual plants comprising these stands were to be investigated. Special attention was to be paid to possible infections with *Labyrinthula macrocystis* and epiphyte composition and densities.

Objective a) was approached in several steps, whereby the following questions were tackled:

i) What climatological mechanism may explain the occurrence of wasting disease in the 1930's ? (starting with data from the Netherlands, later expanding to Europe and the North American Atlantic region)
ii) If such a mechanism is plausible, what periodicity is to be expected, and does this coincide with past declines in eelgrass populations ?
iii) Is there a general correlation between sunspot activity cycles and eelgrass decline ?

Objective b) had to be modified, as it became evident that one could no longer speak of an eelgrass population (other than very ephemeral and transient) in the Dutch Wadden Sea (Dijkema, p.c. 1987; personal observations, see part 3). The trend of rapid deterioration that was noticed between 1972-5 (den Hartog and Polderman, 1975; Polderman and den Hartog, 1975; Schellekens, 1975) had obviously taken its toll, and all that remained were rarely encountered individual plants with an annual habit (Dijkema, p.c. 1987; Bellemakers, p.c. 1987). Eelgrass collected at Roscoff (Brittany, France), two localities in the German Wadden Sea (Jadebusen and Cnldumersiel) and at several localities in Zeeland Province, the Netherlands (Lake Grevelingen, Lake Veere, Zandkreek) were examined instead, for signs of wasting disease symptoms and occurrence of *Labyrinthula spp.*.

Part 2

CLIMATE AND WASTING DISEASE OF THE 1930'S

WHY FOCUS ON CLIMATIC FACTORS ?

Causes in retrospect. As was stated in the introduction, the question remaining in the wake of the wasting disease epidemic was what had actually caused it ? The pathogen was identified, but why hadn't it caused widespread eelgrass mortalities before, and why did it occur simultaneously on such a wide scale in the early 1930's ?

A host of mechanisms were forwarded, but many of these were either only hypothetical or only explained local die-off of eelgrass. Degradation mechanisms (those forwarded to explain wasting disease are marked with an asterix) operating at a local scale include:

- *mechanical damage*: herbivory by ducks and geese (Wolff *et al*, 1967), herbivory by turtles and fish (Kirkman, 1978), uprooting by fish (Orth, 1975), harvesting by man (Van Goor, 1921; Van der Werff, 1934), boat propellers (Clark, 1975) , silt deposition (Martinet, 1782; Milne and Milne, 1951*; Martin, 1954*; Kirkman, 1978), ice-scouring (Lewis, 1932; Tutin, 1942); storms (Lewis, 1932).

- *chemical mechanisms*: pollution by oil (Duncan and Cotton, 1933*), urban and industrial waste (Milne and Milne, 1951*), oxygen depletion and toxification of sea-bed (Nienhuis, 1983), eutrophication (Duncan and Cotton, 1933*).

- *desiccation* of exposed parts, due to tidal extremes (Van der Werff, 1934*; Tutin, 1942).

Short *et al* (1988) approach the problem of simultaneous, widespread occurrence from the theoretical end, and state that two possible mechanisms underlie wasting disease. Either the pathogen was harboured at one locality, and was spread rapidly from there, or it was universally present and some external factor operating on a widespread (North Atlantic) scale triggered the disease. Short *et al* consider that there is little evidence for the former, and propose several theoretical mechanisms for the latter:

i) The pathogen was an obligate pathogen that occurred at low densities; a widespread external influence created a stress in the eelgrass populations or increased the virulence of the pathogen, or both occurred simultaneously.

ii) The pathogen mutated from a non-aggressive secondary decomposer to a pathogenic form, under influence of a widespread external factor.

iii) A long-term life cycle exists for the pathogen that includes an inactive form, such as a cyst, and a widespread external factor initiated the disease.

From this theoretical approach we may conclude that either the disease was spread by a vector, or it was triggered by a widespread external factor (through a variety of possible mechanisms). As Short *et al* (1987a) report, there is little evidence to support the occurrence of a vector mediated epidemic, although, admittedly, there are insufficient data available from the 1930's to determine if the disease may have spread from a single location. Indeed, the records do not allow a reliable chronological reconstruction, as later recordings often simply imply a later observation and not a later occurrence of the disease. It is difficult to conceive a transport mechanism, however, that could have accomodated such a rapid dissemination.

Vectors

The possibility of vector-borne disease transmission was briefly investigated in the course of this study.

Bird species that regularly consume eelgrass and occur on both sides of the Atlantic are: *Anas acuta* (pintail duck), *A. crecca* (green-winged teal), *A. platyrhynchos* (mallard), *Branta bernicla* (brent goose) and *Cygnus olor* (mute swan) (McRoy and Helfferich, 1980; Nienhuis, 1984). Only *Branta bernicla* typically feeds on eelgrass in North America, and would have populations shared between the two continents. *Cygnus olor* occurs on both continents as a result of its introduction into North America by man, but there is no exchange between the continents. *A. acuta* and *A. platyrhynchos* only very rarely exchange back and forth, and are not profound consumers of eelgrass. *A. crecca* almost definitely does not share populations between Europe and N. America (Batt, p.c. 1987). Several arguments oppose a vector role by *B. bernicla*. Deeper stands of *Zostera marina* were affected first, whereas birds graze in shallow waters. Secondly, the eelgrass pathogen would have had to be retained over a summer cycle within the Arctic circle, with Greenland as the probable population exchange area; however, the birds converge on the northern and north-eastern coasts of Greenland (Handbook of Birds of Europe, the Middle East and North Africa, Vol.1), while eelgrass stands are off the south-western coast (den Hartog, 1970). Thirdly, there is no complete overlap between *B. bernicla* and wasting disease occurrence. For example, wasting disease occurred in western Sweden (Molander, 1933), but *B. bernicla* never occurs there; *B. bernicla* flocks were common in the Moray Firth, Scotland, up to the 1930's (Atkinson-Willes and Matthews, 1960), but wasting disease was not reported from this locality. Bird mediated dispersal of wasting disease cannot be outruled, but it is highly unlikely that it caused a rapid dissemination between Europe and N. America.

Ocean currents. These are soon outruled as a possible candidate for wasting disease dispersal across the North Atlantic, as it would take at least several months, but more probably up to several years (Thurman, 1975; Wiebe, 1982). This would be too long to explain the simultaneous occurrence. Currents do, however, explain the local dissemination of the disease, as pointed out in Danish waters by Blegvad (1934) and in the Dutch Wadden Sea by Van der Werff (1934).

Man. It is unlikely that boats transported infected tissue across the Atlantic, as large ocean-going vessels do not venture into shallow waters where eelgrass typically occurs, and it is hard to picture a mechanism by which eelgrass may remain adhered to a boat's hull in the course of a trans-Atlantic crossing.

One must conclude that a hypothesis involving a vector is somewhat far-fetched, and strains the imagination. Other available evidence also indicates that wasting disease was not an isolated phenomenon, only involving *Zostera marina*, *Labyrinthula macrocystis* and a vector. Evidence is gradually emerging that the early 1930's were quite turbulent for marine organisms of the North Atlantic, and changes seem to coincide with a northward extension of warmer, southern waters (Cushing and Dickson, 1976; Cushing, 1982; see part 3).

Climatic events may not only help explain the widescale simultaneous occurrence of wasting disease, but their study may also give a clue to possible recurrences of such epidemics. Indeed, the proposal that certain long-term cycles in eelgrass bed dynamics may be influenced by solar cycles (via effects on climate) forms one of the grounds for this study.

AUTECOLOGY OF HOST AND PARASITE

Autecological responses to specific environmental parameters, such as temperature, salinity, illumination and depth, form a handy instrument for judging the impact of climatic irregularities on host/parasite relationships. Strong aberrations may swing a balance between the two in favour of either one, and it is suggested that such an occurrence may have initiated the wasting disease epidemic of the 1930's.

The influence of environmental parameters on *Zostera marina* growth has been summarized in the past by Setchell (1929), Tutin (1938, 1942), den Hartog (1970) and Burrell and Schubel (1977). More recent are papers on light and temperature relationships, scattered throughout a number of journals (Backman and Barilotti, 1976; Bulthuis, 1987; Dennison, 1987; Dennison and Alberte, 1982, 1985; Fonseca *et al*, 1983).

Species of *Labyrinthula* have been widely studied in the past decades, but nevertheless their phylogeny remains poorly understood. Since being first described by Cienkowski in 1867, they have been variously assigned to the Protozoa, algae, Mycota and Protista (Moss, 1987), a confusion that nicely demonstrates their diverse taxonomic affiliations. In textbooks on mycology, for instance, they are often classified as a separate order of the class *Plasmodiophoromycetes* (Gams, 1979), while in zoological works they are often placed in their own phylum, *Labyrinthomorpha* (Levine *et al*, 1980). In works on wasting disease they are often referred to as mycetozoans (Renn, 1934, 1936) or slime molds (Den Hartog, 1987), but are more carefully referred to as "slime mold-like protists" (Short *et al*, 1988). General information and notes on how to culture *Labyrinthula spp.* are given in Young (1943), Vishniac (1955), Johnson and Sparrow (1961), Pokorny (1967), Sykes and Porter (1974), Moss (1987) and Porter (1987). The influence of environmental parameters on *Labyrinthula* growth has been summarized by Young (1943), Johnson and Sparrow (1961) and Pokorny (1967).

Table 1 gives the response of both *Zostera marina* and *Labyrinthula macrocystis* to a number of important abiotic parameters. For obvious reasons, the first three (temperature, salinity and illumination) are most important when assessing the effects of climatic events. With regard to temperature, *Z. marina* has a lower optimum than *L. macrocystis* (15-20°C, and 16-24°C, respectively). The same also applies to salinity, where *Z. marina's* optimum is reported to be 10-30‰, while that of *L. macrocystis* is reported to be 22-40‰ or even 30-42‰. Eelgrass growth is strongly determined by light conditions (Dennison, 1987; Dennison and Alberte, 1982, 1985), while that of *L. macrocystis* is independent of illumination. Summarizing, the following circumstances:

- water temperatures above about 20°C
- salinities above 30‰
- reduced light conditions are (each) more favourable for the parasite than the host. It is highly unlikely, however, that small shifts past these thresholds could have induced wasting disease. For such a catastrophic event to occur, the vitality of the host would have to have diminished due to stress. Stress occurs in *Z. marina* if water temperatures are above about 25 °C, salinity is above 32-42‰ or if illumination conditions are much reduced. Conditions inhibiting the proliferation of *L. macrocystis* are water temperatures below 16°C, and salinities below 12-15‰.

Table 1: Relationship between Zostera marina, Labyrinthula macrocystis and abiotic factors.

factor	Zostera marina	Labyrinthula macrocystis
1. temperature		
tolerance	0-30°C (16) -6-44°C (1) 5-27°C (4) -10-35°C (14) 25-30°C = lethal (15)	0.3-27°C (25)
growth possible	10-20°C (16) (3) under 23°C vegetative (10) under 25°C generative (10)	
optimum	15°C vegetative growth (16) 17-18°C generative growth (16) (3) 15-20°C (10)	14-24°C (25) for infection of Z. marina greater than 16°C (25) 25°C for closely related L. vitellina (29)
2. salinity		
tolerance	freshwater to 42 o/oo (two days) (10) 3 o/oo lower limit in Baltic (5) 5-32 o/oo (12)	freshwater to double normal sea salinity (25) 24 o/oo NaCl (29)
optimum	10-30 o/oo (3) less than 12-15 o/oo (15)	22-40 o/oo (25) 30-42 o/oo (14) less than 15 o/oo results in arrest of infection (17) less than 12-15 o/oo no infection occurs (15) less than 10 o/oo no infection occurs (28)
3) illumination		
compensation point	8 J/cm^2.day (22) 19 " (7) 24 " (6)	independent
saturation point	60 " (22) 185 " (8) both points are temperature dependant (2)(30)	
beds recorded to:	20% light transmission (26) 3-10 % " " (27) 8-24 % " " (13)	
4) substrate	from gravel mixed with coarse sand to almost liquid mud	Zostera marina, Rupola maritima, Zannichellia palustris + numerous algae
4) depth	upper limit tolerance = 15% exposure time (11); optimum = 5% exposure time or less (11) maximum depth recordings: 14 metres (Denmark) (13) 13 " (Ireland) (24) 18-30 " (USA Pacific) (5)	???
5) water movement	120-150 cm/s (9)	???
6) pH	7.3 - 9.0 (3) (4)	4 - 9 (25), optimum 8 (14), 7.5 (23)

1) Biebl and McRoy (1971)
2) Bulthuis (1987)
3) Burrell (1973)
4) Burrell and Schubel (1977)
5) Den Hartog (1970)
6) Dennison (1987)
7) Dennison and Alberte (1982)
8) Dennison and Alberte (1985)
9) Fonseca et al (1983)
10) Kawasaki et al (1986)
11) Keller and Harris (1966)
12) McRoy (1966)
13) Ostenfeld (1908)
14) Pokorny (1967)
15) Rasmussen (1977)
16) Setchell (1929)
17) Short et al (1987a)
18) Terawaki and Iitsuka ('86)
19) Thayer and Phillips (1977)
20) Tutin (1938)
21) Tutin (1942)
22) Verhagen and Nienhuis (1983)
23) Vishniac (1955)
24) Whelan and Cullinane (1985)
25) Young (1943)
26) Backman and Barilotti (1976)
27) Pellikaan (1980)
28) Petersen (1935)
29) Sykes and Porter (1973)
30) Marsh et al (1986)

CLIMATE AND WASTING DISEASE IN THE DUTCH WADDEN SEA IN THE 1930'S

TEMPERATURE

The exact relationship between temperature and *Zostera marina* growth remains uncertain, in spite of Setchell's (1929) often cited findings. He proposed a very specific growth range of 10-20°C, with an optimum of 15°C for vegetative growth and 17-18°C for generative growth. Temperatures above 20°C and below 10°C were assumed to be growth inhibiting. As Bulthuis (1987) points out, most investigators today dispute Setchell's absolute temperature limits. Many authors find that eelgrass growth is not, or only slightly controlled by water temperature under many circumstances (Phillips, 1974, in Bulthuis, 1987; Jacobs, 1979; Wium-Andersen and Borum, 1984). Others find that tolerance levels are well above Setchell's upper limit of 20°C. What complicates matters is that there probably is a certain amount of phenotypic variation in temperature tolerance of *Z. marina*, and that *in vitro* experiments with temperature/photosynthesis responses do not correspond with observations in the field. In the laboratory, temperature optima for photosynthesis lie well above temperature optima for plants in the field (Bulthuis, 1987).

At photosynthetically saturating light levels, the optimum temperature may be as high as 30°C, while at low light intensities it may be as low as 5°C (Marsh *et al*, 1986; Bulthuis, 1987). This phenomenon is largely determined by respiration levels (Marsh *et al*, 1986), that increase sharply with increases in temperature. What is evident is that a combination of high water temperature and low irradiation results in a reduction of net photosynthesis. One may further add that *L. macrocystis* has a higher temperature optimum than *Z. marina* (16-24°C and 15-20°C, respectively), and that temperatures between approximately 20-24°C would probably favour the parasite more than the host.

The hypothesis that abnormally high summer seawater temperatures initiated wasting disease has largely been advocated by Rasmussen (1977), who backed this with data from Denmark. Summer and winter seawater temperature data of the Dutch Wadden Sea (adapted from Van der Hoeven, 1982) were investigated for the period 1861-1982. The results are given below in table 2 and fig. 1.

Data from West-Terschelling (mid-northern Dutch Wadden Sea) show the same pattern as that of Den Helder station. One may conclude from these data that seawater temperatures of the Dutch Wadden Sea of the early 1930's do not support a hypothesis of a temperature-induced eelgrass decline. Temperatures were slightly above the long-term average, both in winter and summer months (see fig 2), but not uniquely so, and certainly not dramatically so. The highest temperature recorded in the summer months of the period 1930-35 was 19.0°C, for both Den Helder and West-Terschelling, both in August 1932. These slighly raised temperatures do not account for a physiological stress of *Z. marina*.

Slightly raised water temperatures may have aggravated an already prevailing epidemic, as this situation favours parasite growth above eelgrass growth. At the most, however, this only represents a slight shift of optima. Long-term data indicate that the then prevailing combination of high water temperatures and below average summer sunshine was fairly unique this century. An already occurring epidemic may also have been stimulated by the higher winter water temperatures, resulting in a higher survival rate of the parasite.

Table 2: **Seawater temperatures of the Dutch Wadden Sea (Den Helder station; data Van der Hoeven, 1982).**

summer (July, August, September)

long-term average 1860-1982 = 16.9°C

departures from norm:		
more than +2°C	:	1868, 1947
" " +1°C	:	1861, 1865, 1868, 1872, 1875, 1880, 1884, 1911, 1939, 1947, 1959, 1969, 1973, 1976, 1982
0°C	:	1930
-0.7°C	:	1931
0.9°C	:	1932
0.9°C	:	1933

winter (January, February, March)

long-term average 1860-1982 = 3.7°C

departures from norm:		
more than +2°C	:	1863, 1866, 1869, 1882
+1°C	:	1862, 1867, 1868, 1872, 1874, 1877, 1878, 1884, 1899, 1910, 1912, 1913, 1915, 1916, 1920, 1921, 1923, 1925, 1926, 1927, 1935, 1943, 1948, 1957, 1961, 1967, 1973, 1974, 1975
+0.8°C	:	1930
+0.1°C	:	1931
+0.9°C	:	1932
+0.2°C	:	1933

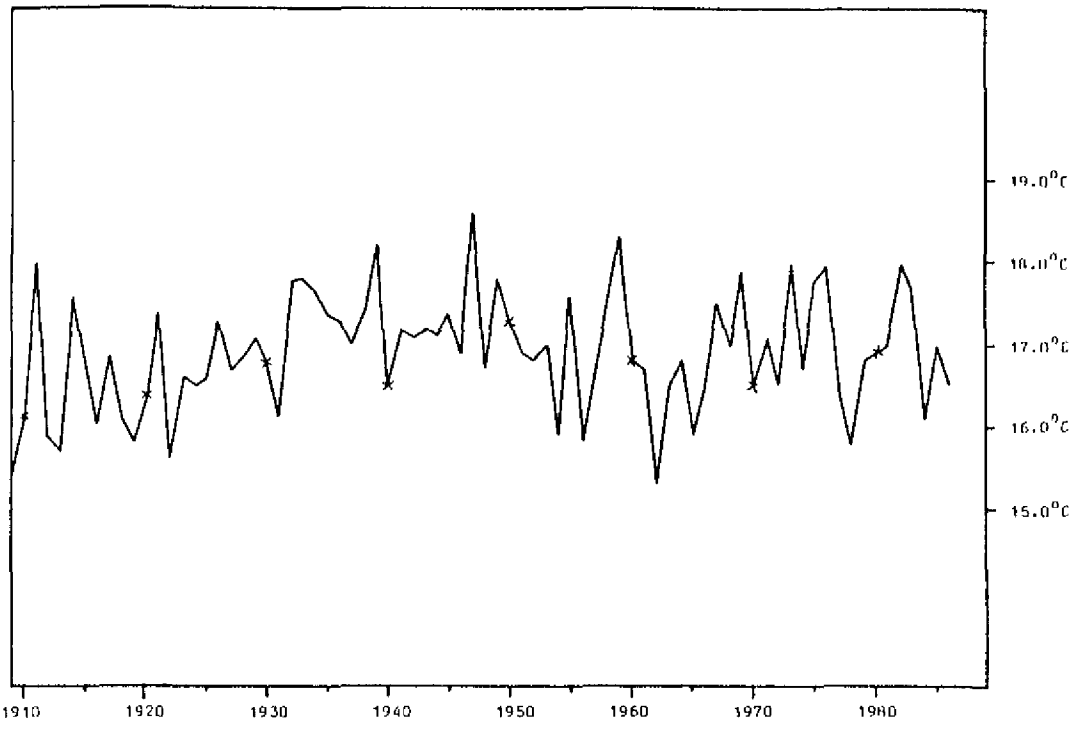

Figure 1: Surface seawater temperatures, Den Helder station, 1910-1987

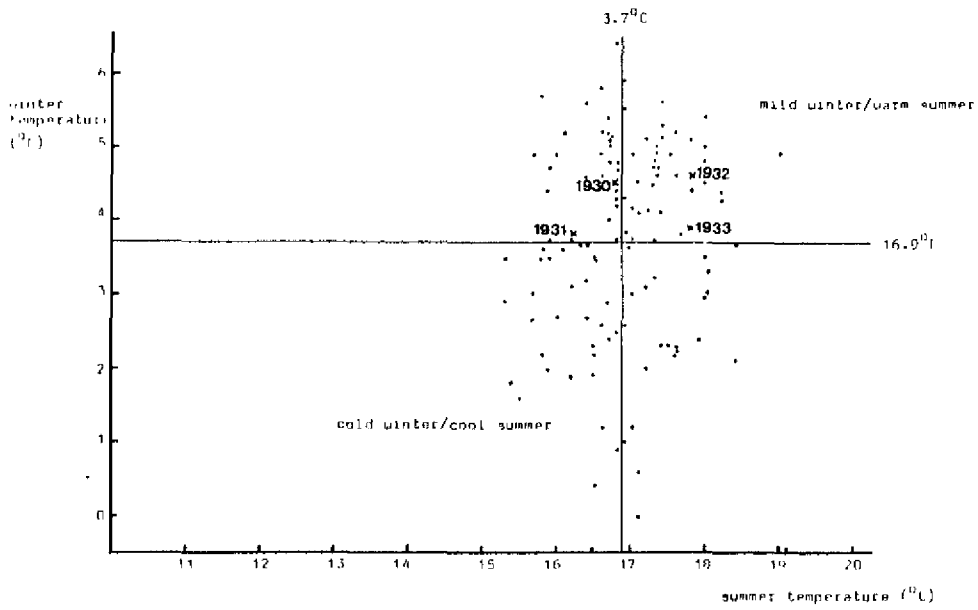

Figure 2: Summer versus winter seawater temperatures, Den Helder, 1861-1934.

SALINITY OF THE DUTCH WADDEN SEA

Salinity of coastal waters is closely correlated with precipitation (+ related river discharge) and insolation, especially in confined waters such as bays, and near estuaries, though ocean currents may play a role. As recorded in the previous chapter, *Zostera marina's* salinity optimum is about 10-30‰, while that of *Labyrinthula macrocystis* is 22 (or 30) - 42‰. The parasite is not reported to be pathogenic at salinities below 12-15‰. *Z. marina's* upper tolerance level is uncertain, probably being somewhere in the range of 32-42 ‰ (depending on phenotype, exposure time, etc ...). The hypothesis that salinity anomalies may have initiated wasting disease has been advocated by Martin (1954), who correlated wasting disease with precipitation extremes: drought in the eastern USA, and excess rainfall in western Europe.

Data on the salinity of the Dutch Wadden Sea were adapted from Van der Hoeven (1982), and are given below in table 3, for the stations Den Helder and West-Terschelling.

Table 3: Salinity of the Dutch Wadden Sea

(in ‰'s, based on monthly values, adapted from Van der Hoeven, 1982).

Den Helder long-term average, 1861-1981 = 30.1‰

year	minimum	maximum	average
1930	27.2	31.9	30.1
1931	27.3	30.1	29.1
1932	27.2	30.3	29.4
1933	29.4	31.9	30.6

West-Terschelling long-term average 1919-1981 = 30.4‰

year	minimum	maximum	average
1930	25.8	33.0	30.1
1931	26.4	31.4	29.3
1932	28.0	32.2	31.2
1933	29.1	32.9	31.3

The greatest dynamics in salinity occurred at West-Terschelling, where the salinity range for the period 1930-33 was 25.8 to 33.0‰. For Den Helder this range was 27.2 to 31.9‰. These extremes do not suggest that physiological stress occurred in the host plant due to salinity anomalies, as even the higher values (of up to 33 ‰) are by no means unique. Salinities of 33‰ or more were recorded at least a dozen times at the Den Helder station in the period 1861-1930, with no apparent adverse effects on eelgrass stands. At the West-Terschelling station, salinities of 33‰ or more were recorded four times in the period 1919-1930 (records from before 1919 are lacking), also without recorded effects.

At the lower end of the salinity range, eelgrass was much less affected by wasting disease (Van der Werff, 1934; Blegvad, 1934; Harmsen, 1936; den Hartog, 1970, 1987), which agrees with the observation that *L. macrocystis* is not pathogenic at salinities below 12-15‰. However, this relationship is further complicated by the fact that plants of high and low salinities may show great phenological differences. For example, euhaline sites may bear a broadleaved, perennial sublittoral form of eelgrass, and a mesohaline site may bear an annual, narrow-leaved form

of the littoral zone. Where mesohalinity coincides with the littoral zone, other factors such as temperature extremes, light regime and desiccation also complicate the relationship with salinity.

SUNLIGHT IN THE DUTCH WADDEN SEA

Introduction

Light limitation is probably the most important factor determining the lower end of the vertical distribution of *Zostera marina* (Backman and Barilotti, 1976; Dennison and Alberte, 1985). In spite of this, this factor received little attention in the studies that appeared in the wake of the wasting disease epidemic. Most studies focussed on pathogenic organisms (Renn, 1936,1942; Mounce and Diehl, 1934; Tutin, 1934; Young, 1943), extent of eelgrass stand damage (Cottam, 1933-1949; Butcher, 1934; Molander, 1933; Blegvad, 1934; Renn, 1934, 1936a, 1936b, 1937), or environmental factors such as salinity (Martin, 1954) and temperatures (Martin, 1954; Rasmussen, 1977). The only reference to the possible adverse effects of poor light conditions was made by Tutin (1938), who reported that the British Isles received 20% less sunshine in 1931-2, and suggested that « enfeeblement of the plant due to lack of sunshine in 1931-2 is the fundamental cause of the epidemic » . The suggestion was rejected by Atkins (1938), who demonstrated that the 20% deficiency of sunshine was by no means an abnormal phenomenon in the British Isles, and that in other years such a deficiency did not lead to noticeable mortalities in eelgrass stands. Atkins was convincing, and all later authors adhered to his conclusions, ruling out light limitation as a possible trigger mechanism for wasting disease.

The lines of argument presented by both Tutin and Atkins, however, harbour several inaccuracies that warrant closer scrutiny. Firstly, both stake their arguments on annual sunshine figures, which are far too broad to be of biological significance. Far better would be to observe sunshine data of the period of eelgrass growth; for *Z. marina* in the Netherlands this is usually in the period May to September (Verhagen and Nienhuis, 1983; Nienhuis, 1984). Furthermore, hours of sunshine should be converted to units of PAR (photosynthetically active radiation, i.e. in wavelengths 400-700nm), per unit of time and area, as the amount of PAR per hour of sunshine varies throughout the year, depending on the declination of the sun. Secondly, eelgrass depth distribution should be considered when determining the effects of reduced illumination. Light levels that are photosynthetically saturating for a stand at 1m below MLWL may be below the light compensation point for a stand at 3m below MLWL. Thirdly, factors that influence the attenuation of light in water should be regarded in combination with variations in amount of PAR. Increases in turbidity may amplify adverse effects if, for instance, they coincide with reduced sunshine levels.

Monthly hours of sunshine

Data on hours of sunshine were obtained from the annual reports of the Royal Dutch Meteorological Institute (KNMI) at De Bilt; long-term figures were obtained from Braak (1937) and Müller (1983). Monthly and annual totals are given below in table 4, for the station Den Helder (De Kooij), that lies in the western part of the Dutch Wadden Sea. Conspicuous is the apparant excess of sunshine in March (1931: +67%; 1932: +40%; 1933: +42%) and the deficiency of sunshine in May (1931: -27%; 1932: -25%; 1933: -26%) and July (1931: -14%; 1932: -11%) in the years during which wasting disease occurred. Annual total hours of sunshine, however, were slightly above average for all years 1930-1934.

Table 4: Monthly hours of sunshine, Den Helder

(data adapted from KNMI annual reports)

years	J	F	M	A	M	J	J	A	S	O	N	D	annual
1930	56	95	146	151	205	266	224	229	121	97	55	37	1680
1931	41	66	210	166	177	251	186	214	167	137	38	44	1697
1932	57	100	177	153	182	243	193	223	148	89	39	54	1657
1933	78	116	178	153	179	223	230	243	192	97	44	71	1806
1934	48	97	118	160	234	233	278	223	202	84	31	10	1717
1909-1936	46	77	125	175	244	226	217	203	151	98	52	34	1649
Müller (1983)*	54	74	127	181	227	238	217	207	151	102	48	40	1665

* Müller does not give the exact period, but states that the values are the average of 75 years.

Fig. 3 displays average monthly hours of sunshine for Den Helder. As was stated in the previous paragraph, it is PAR that is of primary importance to photosynthesizing plants, and not hours of sunshine. Hours of sunshine were converted to total radiation (i.e. radiation with wavelength 300-3000nm, expressed in $W/m^2.day$) by means of a regression equation and conversion tables given by Frantzen and Raaff (1982). Global radiation was converted to PAR (400-700nm) by multiplication with a factor 0.45 (Lüning, 1981; Colijn, 1982), and subsequently expressed in $J/cm^2.day$. These values are given in table 5.

The departures from the norm are less outspoken for PAR than they are for hours of sunshine: the above-average March values are 1931, +34%, 1932, +21%, 1933, +22%, and the below average May values are 1931, -16%, 1932, -15% and 1933, -16%.

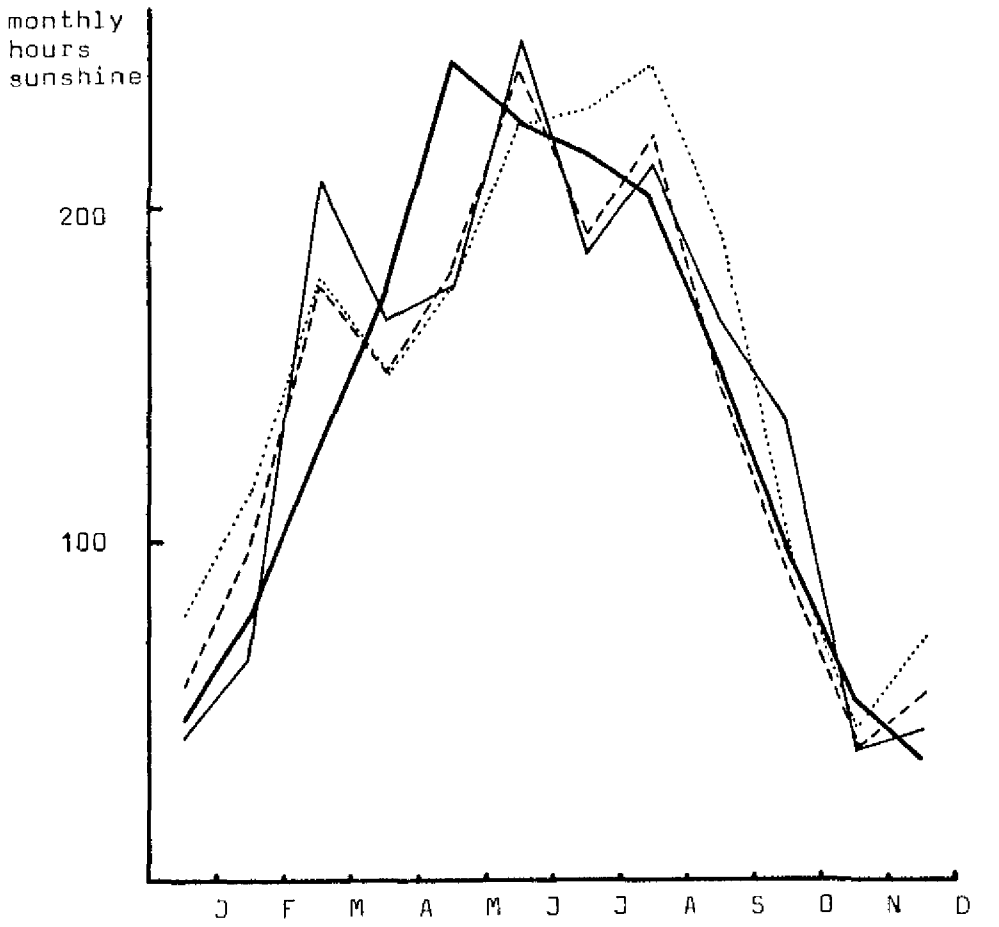

Figure 3: Hours of sunshine, Den Helder.
——— =long-term average; — =1930; - - =1931 ···· =1932

Table 5: PAR at Den Helder

(in Joules per square cm per day).

year	J	F	M	A	M	J	J	A	S	O	N	D
1931	103	208	536	626	741	958	782	754	519	335	122	87
1932	115	253	482	602	747	944	796	773	488	269	122	95
1933	133	273	488	600	744	899	875	813	564	281	128	107
1909-36	106	222	399	640	883	897	842	726	490	282	136	80

The question remains, of course, whether these departures from the norm were unusual, and could they have had adverse effects on eelgrass stands in the Dutch Wadden Sea? In fig. 4 hours of sunshine during May-August (=primary growing season for *Z. marina* in the Netherlands) are given for the period 1909-1987. Viewed at this scale, the years 1931-3 appear to have received average hours of sunshine during the growing season. May and July were decidedly dull during 1931 and 1932, as is illustrated in fig. 5. For the May months one may conclude that these were unusually dull compared to the preceding decades, though seawater temperatures were above normal. The July months of 1931 and 1932 were dull, but not uniquely so. The combination of two dull months (departure from the norm, hours sunshine, more than 10%) during the growing season in two consecutive years is fairly unique, as it occurred only once before in the period 1909-1931, during the years 1912-3. No decline in eelgrass stands off the Dutch coast are known from this date, but Cottam (1934, 1935) reports a decline for populations of the French coast for 1913.

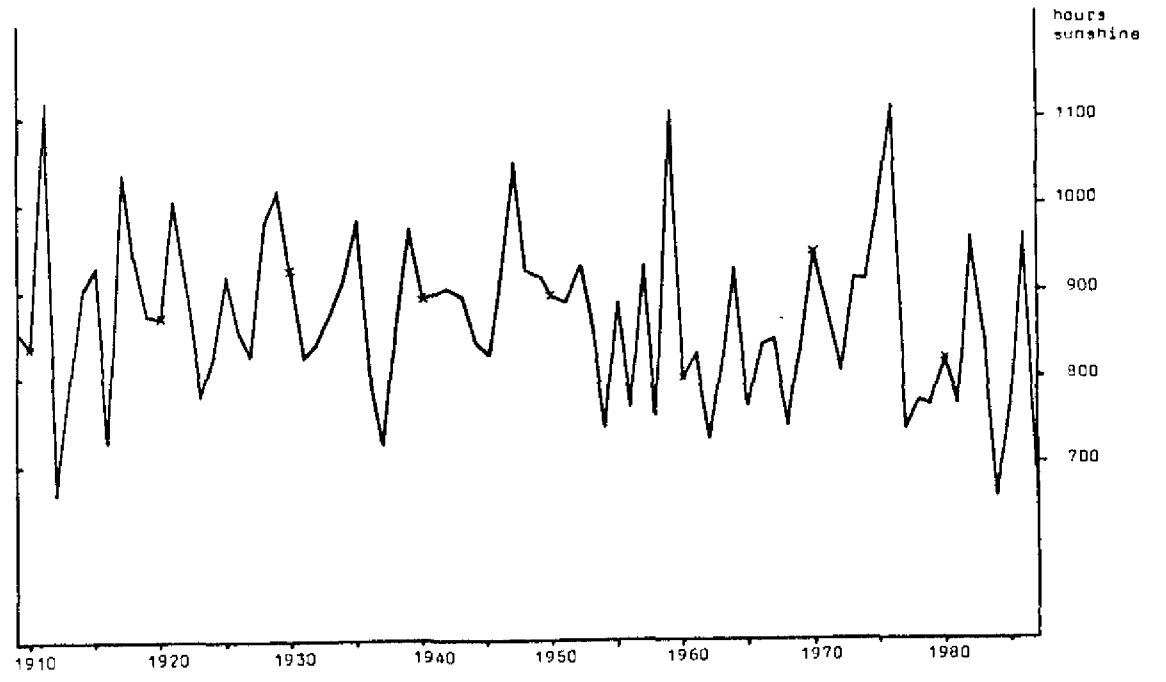

Figure 4: Hours sunshine May-August Den Helder station, 1909-1987.

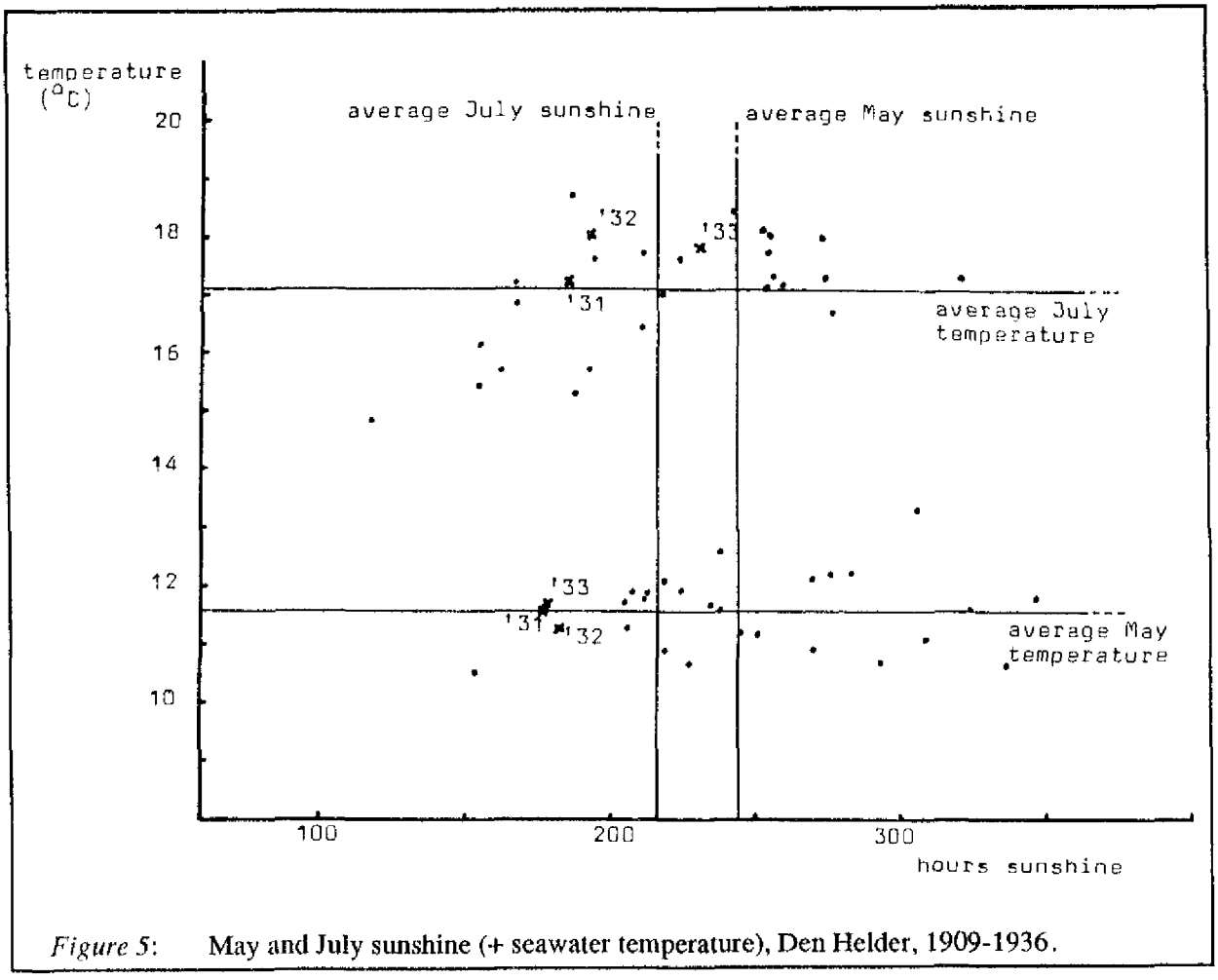

Figure 5: May and July sunshine (+ seawater temperature), Den Helder, 1909-1936.

The effects of reduced illumination on eelgrass growth may be regarded in relation to specific photosynthetic responses of the plant, i.e. compensation point and saturation point. The compensation point of *Z. marina* is approximately 17 $J/cm^2.day$. (average of values given by Dennison and Alberte, 1982; Verhagen and Nienhuis, 1983 and Dennison, 1987), and its saturation point is about 123 $J/cm^2.day$ (average of values given by Verhagen and Nienhuis, 1983; Dennison and Alberte, 1985).

Light attenuation in water may be calculated via the Lambert-Beer equation:

$$I_H = I_O . e^{-k.H}$$

whereby I_H is the light intensity at depth H, I_O is the light intensity just below the water surface and k is the light attenuation factor (property of water, dependent on turbidity) (Jerlov, 1970). On the basis of monthly PAR values one may calculate the average depth at which the light intensity averaged at compensation or saturation point of Z. *marina*. These calculations are displayed graphically in fig. 6, below, for the years 1931-33, and for the average PAR values of the period 1909-36. K was regarded as a constant 0.9 (as calculated by Giesen et al, 1989b, for

pre-wasting disease Wadden Sea areas with eelgrass stands). From fig. 6 one may conclude that either reduced light conditions had little or no effect on eelgrass physiology, or a calculation based on monthly PAR averages and a constant k-value is too crude an instrument to detect these effects. In the next section, the effects of changes in turbidity on eelgrass growth are dealt with. Calculations based on daily PAR values are given in the subsequent section.

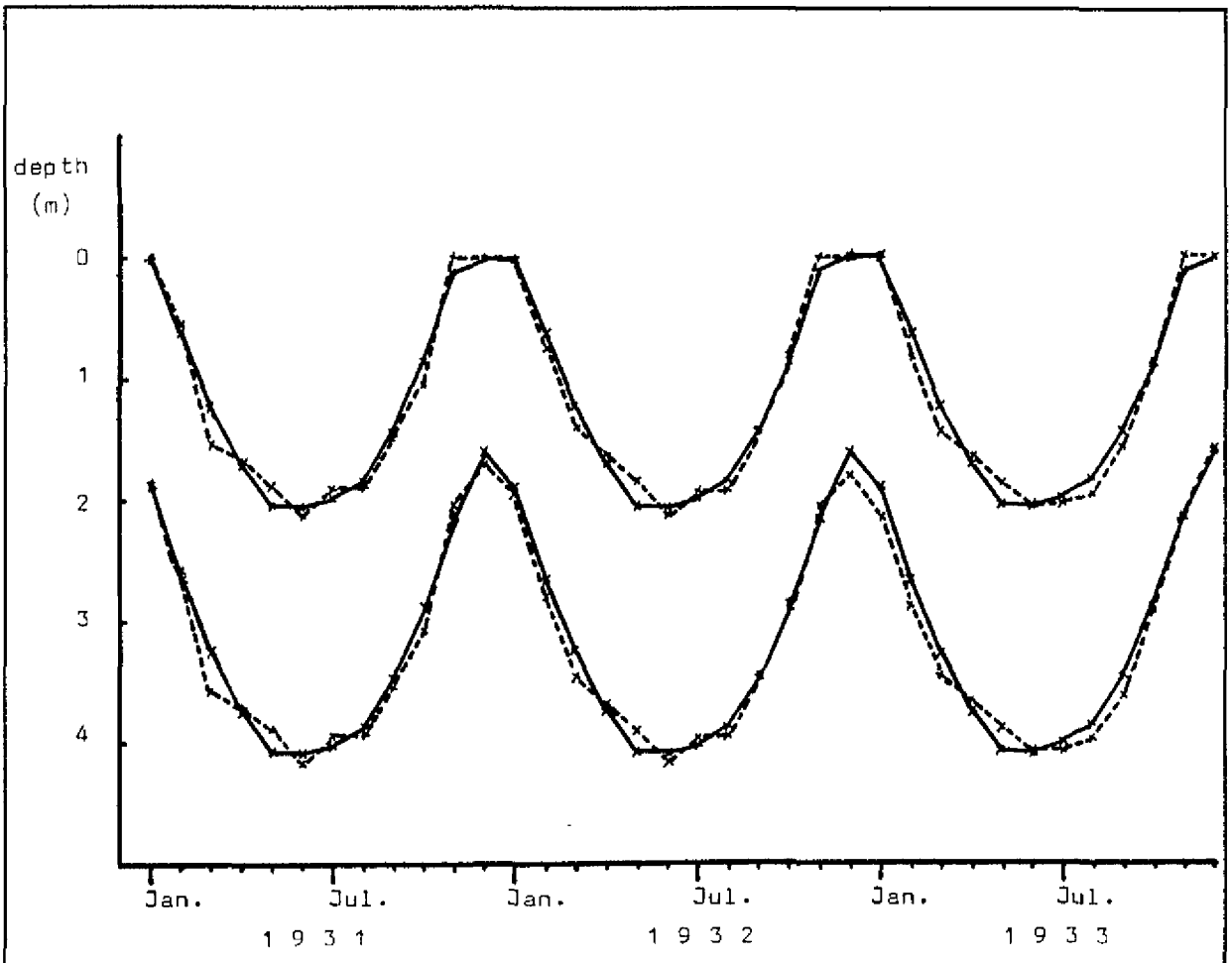

Figure 6: Saturation (top) and compensation (bottom) depths for Zostera marina, Dutch Wadden Sea, 1931-33.
bold line = with average PAR values, dashed line = with actual PAR values for these years.

Turbidity effects

Turbidity is a state of reduced clarity caused by the presence of suspended matter. Changes in turbidity thus directly influence the attenuation of light in water, and result in changes in k-values. Many factors influence turbidity of coastal waters (see below).

Tides influence k-values in shallow seas, as turbulence caused by tidal currents stirs up sediment from the bottom. K tends to fluctuate around a mean, in phase with tidal motions (Visser, 1970); the latter vary in strength, depending on the declination of both sun and moon (Thurman, 1978). The early 1930's witnessed an extreme northward declination of the moon (Stevens, 1936), which would have caused above average tidal extremes and current velocities. These phenomena may have contributed slightly to raised turbidity levels.
The construction of the "Afsluitdijk", that formed the closure of the former Zuyder Sea, resulted in stronger tidal currents, and an increase in tidal extremes by 20-50 cm (Den Hartog, 1987). This probably contributed to increases in turbidity.

The **state of the sea** (waves and swell) has an almost immediate effect on turbidity (Visser, 1970), as wave action stirs up bottom sediments. Average long-term wind speeds recorded at Den Helder (data from KNMI) are:

J	F	M	A	M	J	J	A	S	O	N	D	
15	14	14	14	13	12	13	12	12	13	15	15	(in m/s)

From the early 1930's we have estimates of wind velocities, on a Beaufort scale (not directly comparable to average wind velocities, but they give an indication). The number of days per month with a wind velocity of Beaufort 6 or more are given below, for Den Helder station:

J	F	M	A	M	J	J	A	S	O	N	D	year
10	5	5	6	1	3	8	7	5	10	4	10	1931
11	6	4	11	2	1	2	1	7	9	8	3	1932

From these data we can observe that the late growing season of 1931 (July and August), and the early growing season of 1932 (May) may have witnessed increased turbidity due to a combined wind/wave action.

Plankton abundance affects the transparency of water (Visser, 1970), and annual blooms in both phyto- and zooplankton, occurring in March and April, and to a lesser degree in August and September (Cadée, 1986; Cadée and Hegeman, 1986) have a seasonal influence on turbidity (Visser, 1970).

Rainfall was well above average in the months April, May and July of both 1931 and 1932:

1931	April = +85%	May = +45%	July = +35%
1932	April = +51%	May = +53%	July = +39%

This is also illustrated by fig. 7. Precipitation may cause an increase in turbidity of coastal waters, as river discharges increase (= delayed effect, as waters do not enter the coastal system immediately; this is illustrated in the next section). A minor contribution is also made by direct runoff along the coastline.

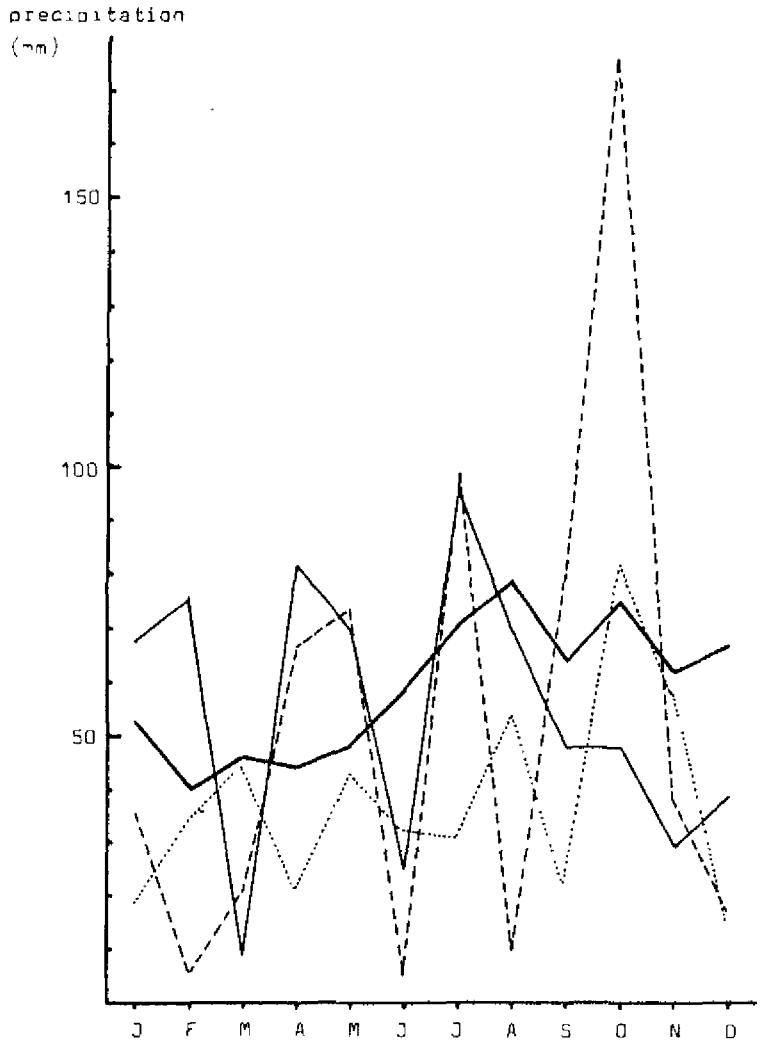

Figure 7: Precipitation at De Bilt, the Netherlands, 1931-33.
(▬ = long-term average; ── =1931; - - =1932; ···· =1933)

River discharges directly affect turbidity of coastal waters, by introducing large amounts of suspended matter into the marine environment. Coarser particles and much of the clay fraction (that form aggregations under saline conditions) tend to precipitate near the river mouth. Finer (non-clayey) and organic particles remain in suspension for longer periods, and thus affect underwater illumination over large areas. Indirectly, there is also an influence via the input of nutrients, that in turn promote plankton growth (Gieskes and Kraay, 1977; Cadée, 1986b). The Rhine River is by far the greatest contributor to river discharges in the Dutch Wadden Sea. In fig. 8, the rate of flow measured at Lobith, the Netherlands, is given for the years 1931-33, along with the long-term average for 1901-81 (adapted from Van der Hoeven, 1982). 1931 experienced flow rates in the range of 24-60% above average during the growing season, but 1932 and 1933 were generally below average. It must be mentioned, however, that the influence on turbidity is not only determined by the total volume, but also by sudden increases in volume (these tend to scour river beds and raise turbidity in the river itself). Both 1932 and 1933 witnessed a doubling of Rhine River discharges in the spring, that may have influenced turbidity in the early part of the growing season. The effect of river discharges on salinity in the Wadden Sea is dealt with in the previous chapter. Total volumes were not unusual in the early 1930's (the distribution pattern was, however). The long-term average (again, for Lobith) for 1901-81 is 2193 m^3/s (Van der Hoeven, 1982), while those of 1930, 1931, 1932 and 1933 were 2557, 2742, 2066 and 1605 m^3/s, respectively. Discharges of 2742 m^3/s or more occurred thrice between 1901-30, and rates of 1605 m^3/s or less occurred twice in the same period.

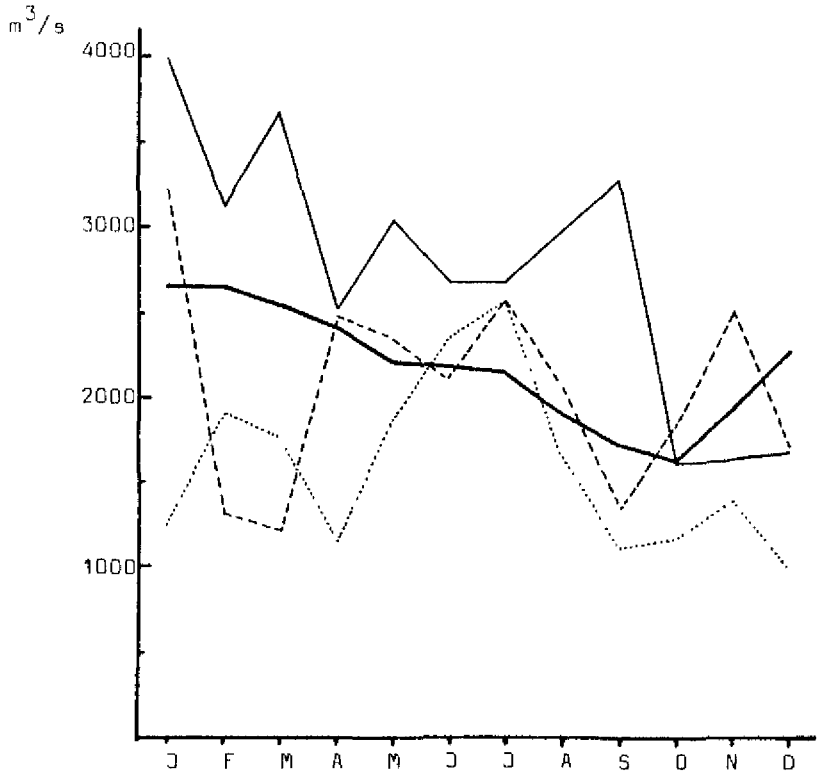

Figure 8: Rhine River discharges, measured at Lobith, the Netherlands. (adapted from Van der Hoeven, 1982); ━ =average;—=1931;--=1932;····=1933

Of the aforementioned factors, rainfall, river discharges and wind are readily quantified, but their relative contribution to turbidity remains obscure. One can only state that, in periods with much wind, a high rainfall and excessive river discharges, the turbidity is likely to be raised to above the annual average. If this coincides with the growing season, this will probably result in negative effects on eelgrass growth. Normal seasonal variations in attenuation coefficient (k) for one locality are in the range of about 0.1 for relatively clear waters (k in the range 0.2-0.3, calculated from Visser, 1970) to a range of about 0.2-0.3 for more turbid waters (k = 2.4 - 3.4; calculated from Manuels, 1978). Giesen et al (1989b) estimate that the average attenuation value of the sites where eelgrass occurred in the Dutch Wadden Sea (prior to 1932) was approximately k = 0.9. The range expected here is thus approximately k = 0.9 ± 0.2. In order to estimate the probable k value of each month in the years 1931-33, each of the abovementioned factors contributing to turbidity was subdivided into three classes, each class representing 1/3 if the total range occurring in that factor. These classes were then weighted with a value 1, 2 or 3, and a total was calculated for the combination of the three factors (rainfall, river discharge and wind). The lowest total was allocated to the lowest probable k value (=0.7), and the highest total to the highest probable k value (=1.1). All other totals were then strung out between these two extremes. These k values were then incorporated in the calculation of compensation and saturation point depths for *Zostera marina*, as was carried out in the previous chapter (then with a constant k = 0.9), and the results are presented below in fig. 9. The differences between fig.'s 6 and 9 show that there probably was an upward shift of compensation points in 1931 and 1932 from about -4.0m to -3.3m, and an upward shift of the saturation point from -2.0m to -1.5m.

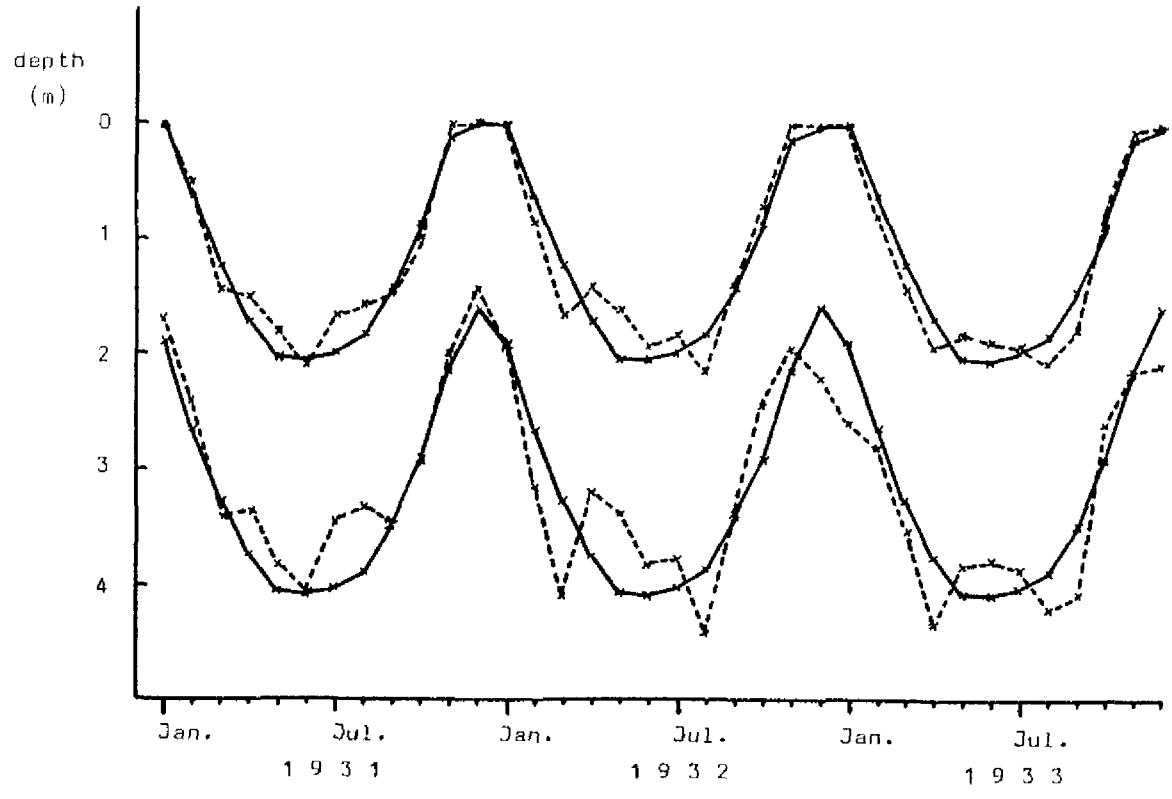

Figure 9: Compensation (bottom) and saturation (top) point depths for Zostera marina. bold line = using average PAR values for 1909-36, dashed line = using PAR values of 1931-33.

Conclusions and suggestions

On the basis of these results one may conclude that a combination of reduced amounts of sunshine, and possibly also the effects of certain factors that increased turbidity (wind, and perhaps excessive rainfall + river discharges), contributed to physiological stress in the more submerged parts of eelgrass stands in the Dutch Wadden Sea in the years 1931-33. It may be argued that the calculation of the relative contribution of the turbidity enhancing factors was an arbitrary matter. It remains, however, that turbidity was probably above average, for not only did the aforementioned three factors contribute, but also increased tidal extremes and the dredging and filling activities associated with the construction of the closure dam of the former Zuyder Sea (den Hartog, 1987; Giesen et al, 1989b).

Application of a simulation model for reconstructing eelgrass behaviour in the Dutch Wadden Sea in 1931 and 1932.

Introduction

In 1981 a computor simulation model for eelgrass growth was developed by the Delft Hydraulics Engineering Bureau and the Delta Institute, at Yerseke, for the Dutch Ministry of Transport and Public Works (Verhagen, 1981; Verhagen and Nienhuis, 1983). This model supplies information on the growth and decomposition of both above and underground parts of *Zostera marina*. The effects of light, temperature and wind are accounted for, but their variations are only incorporated to describe an average year, i.e. with sunlight describing a perfect cosine, seawater temperature rising to 10°C on 16^{th} of May, and eight storms annually, with a fixed distribution. The model describes the development of a single plant during one year, at depth 0.75m, starting with an underground (rhizome) biomass of 20 g/m^2 at the onset of the growing season.

Several experiments were carried out with this simulation model, in an attempt to reconstruct the behaviour of eelgrass stands in the Dutch Wadden Sea in the early 1930's. In the previous chapter it became evident that reduced light conditions may have played a a role in causing physiological stress in eelgrass plants, and thus have contributed to an initiation of the wasting disease epidemic. In the experiments with this simulation model, a number of factors influencing underwater illumination were altered from constants to variables; these included depth, light attenuation factor (k) and light intensity at the water surface. A second adjustment of the original model was an increase of the simulation period from one to two years, using rhizome biomass at the end of the first year as the base value for the second year.

The application of this model to the present problem involves several assumptions. Firstly, the model originally has been developed to describe the eelgrass population of Lake Grevelingen, a brackish lake in the south-western part of the Netherlands. The eelgrass population of this lake is best described as submerged littoral plants: they are perennials, but narrow-leaved and reminiscent of annual eelgrass forms. An important question is then, can one expect great differences in photosynthetic response to reduced light conditions? A comparison with other areas, however, shows that both compensation and saturation points for Lake Grevelingen eelgrass lie lower than the records from elsewhere. This is to be expected, to a certain extent, when comparing eelgrass of northern latitudes with that of more southerly latitudes, as a degree of adaptation exists (Jiménez *et al*, 1987). Verhagen and Nienhuis (1983) report a compensation point of $8 J/cm^2.day$, while Dennison and Alberte (1982) and Dennison (1987) report 19 and 24 $J/cm^2.day$, respectively. The same holds for the saturation point: Verhagen and Nienhuis (1983) report 60 $J/cm^2.day$, Dennison and Alberte (1985) report 185 $J/cm^2.day$. Maximum growth rates incorporated in the model are similar to those found by other authors. A higher saturation point means that this maximum growth rate is attained later, and, if anything, Verhagen and Nienhuis' eelgrass growth model probably underestimates the negative response to reduced light conditions.

Verhagen (1981) and Verhagen and Nienhuis (1983) report that the depth response is accurately calibrated for the depth range 0.75-2.0m. At depths of more than 3 metres, and less than 0.5 metres, eelgrass beds gradually erode and disappear if the model is run for a number of consecutive years. Possible problems caused by these limitations were overcome by running the model for only two consecutive years, by making a comparison with standard years (i.e. years without reduced light conditions) and by confining observations to the depth range 0.75-2.0m.

Methods

In Verhagen and Nienhuis' (1983) growth model, the growth ratio depends on I_H, the light intensity at depth H. I_H is classicly described via the Lambert-Beer equation (Jerlov, 1970):

$$I_H = I_O . e^{-k.H}$$

whereby I_0 is the light intensity just below the water surface and k is the light attenuation factor.

I_0 values used in the model describe a perfect cosine, with a maximum of 740 $J/cm^2.day$ in June, and a minimum of 60 $J/cm^2.day$ in December. In the following simulation experiments, actual I_0 values for 1931 and 1932 were used instead; these (1931, 1932 and cosine light values) are given in fig. 10, along with the aforementiond cosine function. Actual I_0 values were calculated from data on hours of sunshine at Den Helder station (KNMI annual reports), using the regression equation and tables given by Frantzen and Raaff (1982). In this calculation total radiation (wavelengths 300-3000nm) was converted to PAR (400-700nm) by multiplication with a factor 0.45 (Lüning, 1981; Colijn, 1982). In these simulation experiments k values of 0.9, 1.0, 1.1, 1.2, 1.3,and 1.4 (m^{-1} and depths of 0.75, 1.00, 1.25, 1.50, 1.75 and 2.00m were used. Giesen et al (1989b) calculate that the average k value of the Dutch Wadden Sea prior to the closure of the Zuyder Sea was 0.9, and in the previous chapter it was argued that turbidity was above average during 1931 and 1932. The depth range 0.75-2.00m coincides with that recorded for most eelgrass beds in the Dutch Wadden Sea before the 1930's (Martinet, 1782; Redeke, 1916; Van Goor, 1919, 1921, 1922).

Finally, it must be mentioned that in the original model rhizome biomass could attain sub-zero values; in our growth simulations these were adjusted as attaining a minimum value of 0 (=zero).

Results

In these simulation experiments it became evident that at attenuation coefficients (k) of 1.0 or more, or at depths of 1.25m or more, the underground (rhizome) biomass is strongly reduced by the end of the first growing season (see fig. 11a); light follows the idealized cosine function. If this biomass is used as the base value for the start of the second year, the reduction of rhizome biomass takes place in slightly shallower waters, and at slightly lower k values (fig 12b)). A reduction in the shallowest zone is apparent in both figures. In this model (both the original, and the modified one used in these simulations), eelgrass growth and the development of rhizomes is limited by mechanical damage, due to wave action and turbulence (Verhagen, 1981).

The same simulations were carried out for 1931 (fig. 12a + b, respectively). Compared to the simulation using the idealized cosine light function, these plots appear to be more gradual. The reduction of rhizome biomass occurs at shallower depths and in more transparent waters than during an "ideal" year, and this effect is most obvious in 1932. Highest end-of-season rhizome biomasses are achieved in the clearest waters (k = 0.9) at depths of 1.50-2.00m if the cosine function is used, and at depths of 1.00-1.50m if 1931 and 1932 light values are incorporated.

In fig. 13a the results of a simulation whereby the end-of-season rhizome biomass of 1931 is used as the base value for 1932, which is subsequently run. Rhizome biomass is greatly reduced in many k value/depth combinations, and growth would appear to be only possible in relatively shallow and clear waters.

In order to simulate the closure of the former Zuyder Sea, a depth increase of 30cm was introduced in May 1932, the date of closure (den Hartog, 1987, reports an initial increase of 20-50cm), while other circumstances were kept similar to that depicted in fig. 13a; see 13b. The depth indicated on the x-axis represents the original depth before closure. In many k-value/depth combinations rhizome biomass is zero by the end of 1932. In turbid waters with a k value of 1.3 or more, rhizome biomass is zero at all depths.

In fig. 14a and 14b two-year simulations are portrayed for above-ground and rhizome biomass, respectively, for eelgrass at a depth of 1.50m and waters with an attenuation coefficient of k = 0.9. Three simulations are given in each figure: a double (ideal) cosine light-function year, 1931 followed by 1932, and 1931 followed by 1932 together with a depth increase of 30cm to simulate the closure of the Zuyder Sea. At this (1.5m) ideal depth for eelgrass growth, and in these relatively transparent waters, no great differences can be observed in the above-ground parts. Rhizomes, however, are a different story. In the cosine and 1931/1932 functions, rhizome biomass either increases or remains fairly stable. If the increase in water depth is simulated, however, a strong reduction of rhizome biomass follows.

As the main eelgrass beds occurred in fairly shallow waters (0.5 - 2.0 m below MLW), the effect of the 1.2 - 1.5 m tides in the Dutch Wadden Sea must have been fairly marked.

The average low water and high water levels in the early 1930's (up to the closure of the former Zuyder Sea by the "Afsluitdijk" in May 1932) were normal. After closure, however, a number of changes took place. Tidal current velocities increased, along with the level of tidal fluctuations. This contributed to the instability of the existing geomorphology, and resulted in a natural re-adjustment of the creek and gully patterns to the new hydrological situation (den Hartog, 1987). Den Hartog (1987) reports an increase in tidal fluctuations by 0.2 - 0.5 metres. The changes in tides are given below in table 6, for the Dutch Wadden Sea stations Den Helder and West-Terschelling.

It is apparent that average tidal differences did not change very much (+9 cm for Den Helder, +0.6 cm for West-Terschelling), nor did average low water levels (-1.7 cm for Den Helder, -2.7 cm for West-Terschelling). Only the extremes show more variation, with +22 cm for Den Helder and +46 cm for West-Terschelling.

The effect of tidal patterns on the wasting disease epidemic of the early 1930's was considered on the basis of the timing of high water levels. If high tides are synchronous with the daily period of maximum insolation, its effects on reducing eelgrass growth are naturally greater than if high water coincides with early morning or late afternoon tides. In table 7, days of unfavourable tides (peak water level attained between 12.00 - 16.00) are given for the May - August months of 1931-33. Days of low insolation ($\leq$ 3 hours sunshine/day) are also given in table 7. From this table, it is apparent that unfavourable tides probably enhanced the effects of low insolation during a number of months in the early 1930's, especially in May '31, May '32, May and June '33. Favourable tides (low water between 12.00 - 16.00), however, may have improved illumination conditions for eelgrass on a number of dull days. The only way to properly assess the effect of tides on wasting disease in the early 1930's is an hour-by-hour calculation of underwater illumination on eelgrass beds of x metres depth. This leads to far from the present scope of this study.

At present one may conclude that:
- tides negatively affected underwater illumination in a number of months during the wasting disease epidemic
- the closure of the former Zuyder Sea affected tidal extremes, but brought only minimal changes to average tidal extremes and average LWL's.

Table 6: Tides at Den Helder and West-Terschelling in cm.

Differences between 1930-31 and 1932-34 reflect the changes brought about by the closure of the former Zuyder Sea on 19th of May, 1932.

Den Helder

period	average tide	highest HW	lowest HW	highest LW	lowest LW	average LW	max. HW	max. LW
1930-31	122.9	87.1	2.8	-28.4	-117.0	-80.4	112	-153
1932-34	131.9	99.8	0.3	-30.7	-122.5	-87.7	145	-142

West-Terschelling

period	average tide	highest HW	lowest HW	highest LW	lowest LW	average LW	max. HW	max. LW
1930-31	165.0	114.4	21.8	-40.5	-137.0	-97.9	141	-148
1932-34	165.6	126.5	13.7	-42.0	-138.3	-100.6	179	-156

Table 7: Unfavourable tides and dull weather, 1931-33.

A tide was considered unfavourable for eelgrass growth (#), if the highest waterlevel was attained in the middle of the day (i.e., between 12.00 - 16.00 hours). A day was considered dull (#) if there were ≤ 3 hours of sunshine. Former values were obtained from Rijkswaterstaat, the Hague; the latter were obtained from KNMI annual reports.

1931

		1									10										20										30
May	tide	.	.	.	.	.	.	.	.	.	.	.	#	#	.	.	.	.	.	.	.	.	.	.	#	#	#	.	.	.	.
	sun	.		#	#	.	.	#	#	.	.	#	.		.	.	#	#	#	#	.	.	#	.	.	.	.	#	.	.	#
June	tide	.	.	.	.	.	.	.	#	#	#	#	#	.	.	.	.	.	.	.	.	.	.	#	#	#	#	.	.	.	.
	sun	.	#	#	.	.	#	.	.	.	#	.	#	.	.	.	.	.	.	.	.	.	.	.	#	.	.	.	.	.	.
July	tide	.	.	.	.	.	.	.	#	#	#	#	#	.	.	.	.	.	.	.	.	.	#	#	#	#	.	.	.	.	.
	sun	.	.	.	.	.	.	#	.	#	.	.	.	.	.	#	.	#	#	.	.	.	.	.	#	.	.	.	.	#	.
August	tide	.	.	.	.	.	.	.	.	#	#	#	.	.	.	.	.	.	.	.	.	#	#	#	#	.	.	.	.	.	.
	sun	.	.	.	.	.	#	#	.	.	#	.	#	.	.	.	.	.	.	#	.	#	.	.	.	#	.	.	.	.	.

1932

		1									10										20										30
May	tide	.	.	.	.	.	.	.	.	.	.	.	.	.	#	#	.	.	.	.	.	.	.	.	.	.	.	#	#	#	.	.
	sun	.	#	#	#	.	.	.	.	#	.	.	#	.	.	.	.	#	.	.	.	.	#	#	#	.	.	.	.	.	.	#
June	tide	.	.	.	.	.	.	.	.	.	.	#	#	#	#	.	.	.	.	.	.	.	.	.	.	#	#	#	#	#	.	
	sun	#	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	
July	tide	.	.	.	.	.	.	.	.	.	#	#	#	#	.	.	.	.	.	.	.	.	.	.	.	#	#	#	#	#	.	.
	sun	.	.	.	.	.	.	.	#	.	.	.	.	#	.	.	.	.	#	.	#	#	.	.	.	.	.	.	.	.	.	.
August	tide	.	.	.	.	.	.	.	#	#	#	#	#	.	.	.	.	.	.	.	.	.	.	.	.	#	#	#	.	.	.	.
	sun	.		#	.	.	.	#	.		.	.		.	.	.	#	.	.	.	.	.	#	.	#	.	.	.	.	.	.	.

1933

		1									10										20										30
May	tide	.	.	#	#	#	.	.	.			.	.	.		.	#	#	#	.	.	.	.	.	.	.	.	.	.	.	.
	sun	#	#	.	.	.	#	#	#	.	#	.	#	#	.	.	.	.	#	.	.	.	.	.	#	#	.	#	.	.	.	.
June	tide	#	#	#	#	.	.	.		.	.	.	.	.	#	#	#	#	#	.	.	.	.	.	.	.	.	.	.	.	#	
	sun	.	.	.	.	.	.	.	.	#	#	#	#	.		.	.	.	.	.	.	.	#	#	.	#	#	#	.	.		
July	tide	#	#	#	.	.		.	.	.	.	.	.	#	#	#	#	#	#	.	.	.	.	.	.	.	.	.	#	#	#	
	sun	.	.	.	.	#	.	.	.	.	#	#	.	#	.	.	.	.	.	.	.	.	.	.	.	.	.	.	#	.	#	
August	tide	#	#	.	.	.	.	.	.	.	.	.	.	#	#	#	#	.	.	.	.	.		.	.	.	.	#	#	#	#	
	sun	.	.	.	.	.	.	.	.	.	.	#	.	.	.	.	.	.	.	.	.	.	#	.	.	.	.	.	.	.	.	

Discussion and conclusions

- The results of these simulation experiments indicate that reduced light conditions in 1931 and 1932 may indeed have played an important role in reducing the vitality of the eelgrass population of the Dutch Wadden Sea.

- An important role was probably also played by the closure of the former Zuyder Sea in May, 1932. The activities associated with this construction not only raised turbidity in the area, but also resulted in elevated maximum water levels. The construction also affected the hydrological situation in the Dutch Wadden Sea, and contributed to changes in the erosion cycle.

- To interpret the results more accurately, it would be important to carry out simulation studies with PAR data of other years than only 1931 and 1932. This because it can be visualized that any variation around a mean may result in a reduction of net biomass production, as a part of the variation may result in below-saturation-point PAR in a period that average PAR remains above saturation point. This problem has been partly eliminated by Verhagen's choice of cosine function: this lies well below the average light intensity (for both Vlissingen and Den Helder meteorological stations) as a result of calibration with field observations on eelgrass growth response.

- The effects of tides on eelgrass growth in the Dutch Wadden Sea were marginally assessed. The changes brought about by the closure of the former Zuyder Sea mainly affected the extremes, while the average tidal levels were only marginally affected. A proper assessment of the effects of tides would involve an hour-by-hour calculation, which was beyond the scope of the present study.

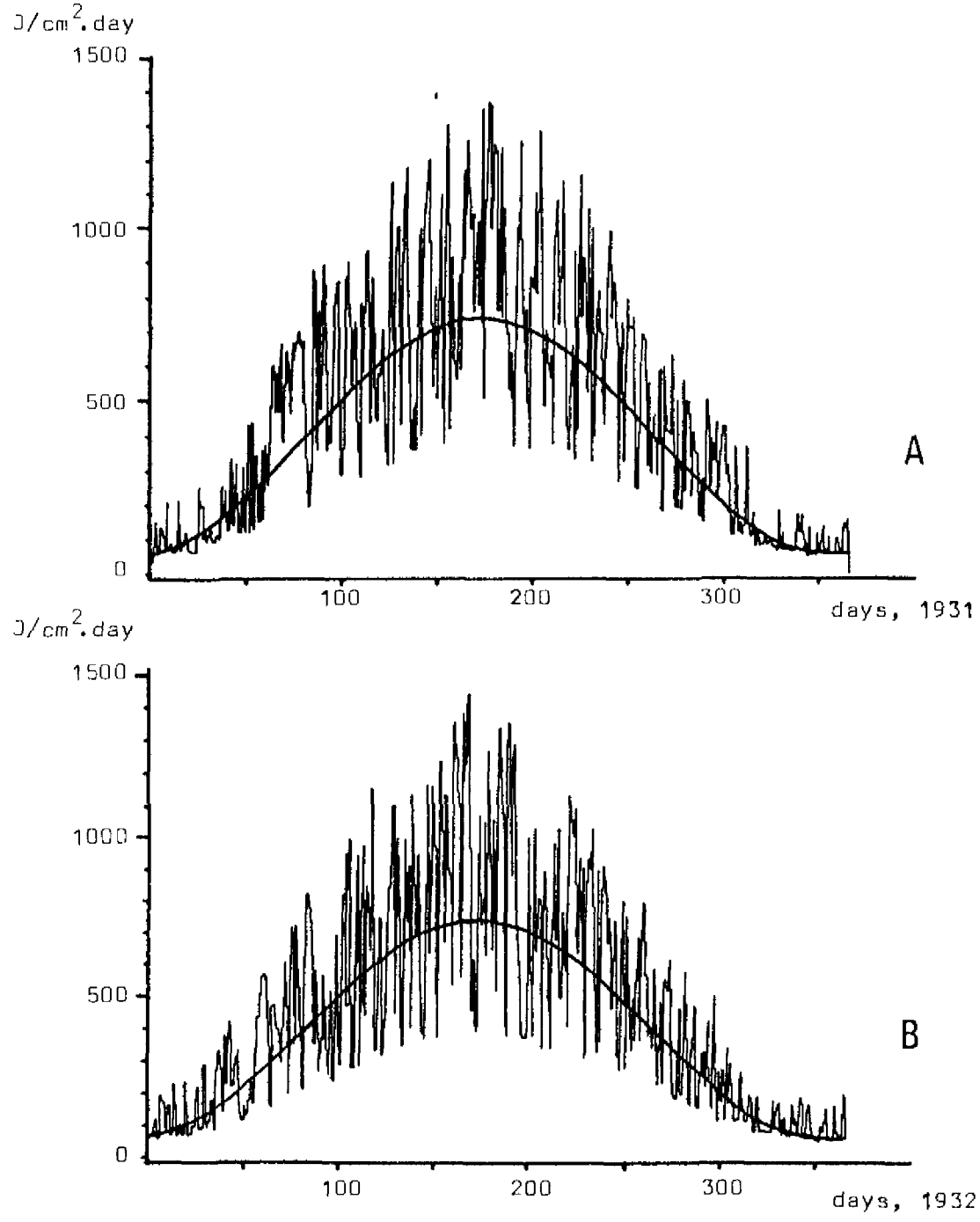

Figure 10: PAR (photosynthetically active radiation, 400-700nm), Den Helder station, 1931 and 1932.
The cosine function in both figures depicts the idealized PAR used in the original simulation model by Verhagen and Nienhuis (1983). 1931 = A; 1932 = B.

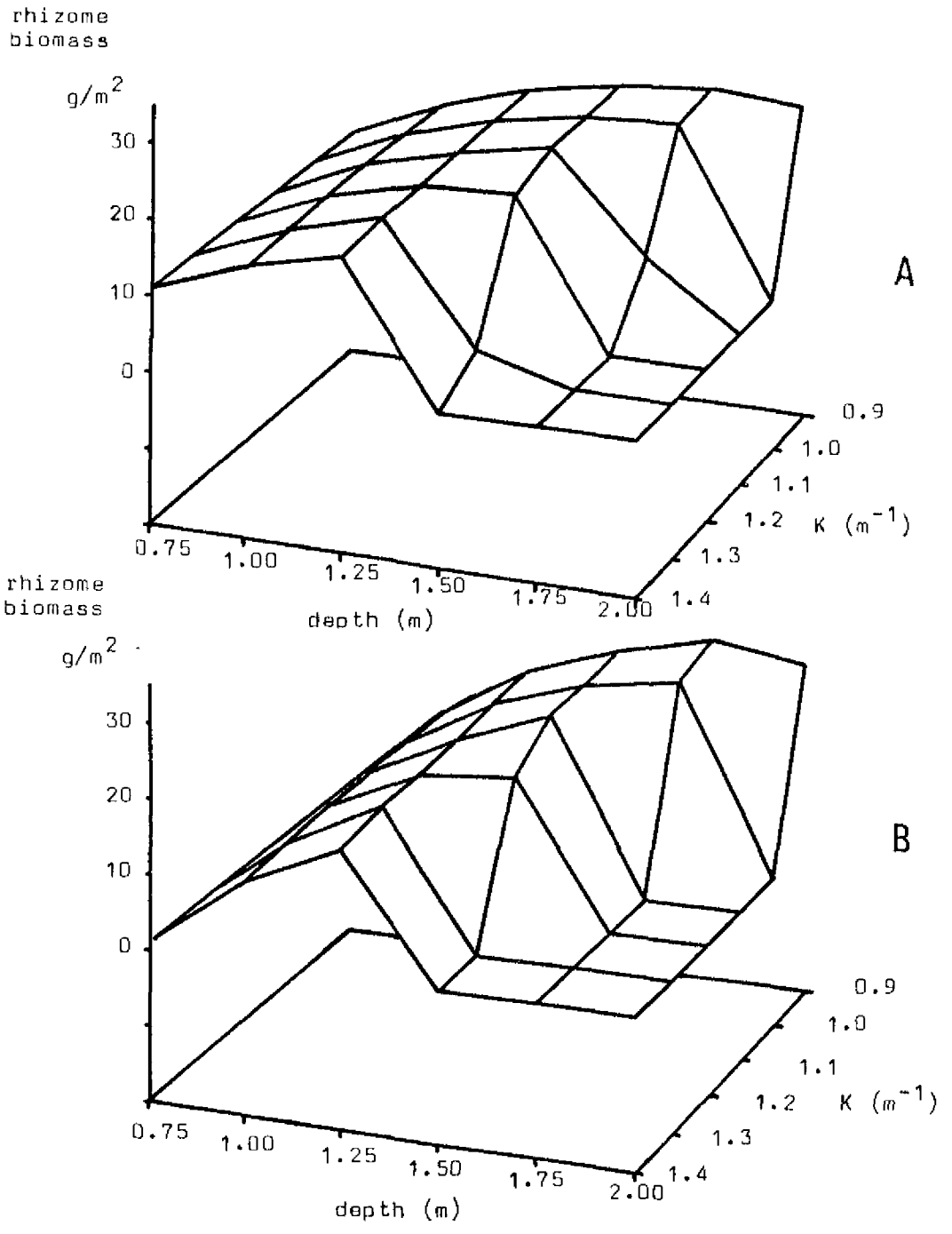

Figure 11: Simulation of rhizome biomass at the end of the growing season;input = cosine light function.
In ash-fresh dry weight, at different depths and k values. The top figure (a) gives the results after one growing season, using the idealized cosine light function as the input for PAR values. The bottom figure (b) depicts the same, but after running the model for a second season, using first season end values as the basis for the second year.

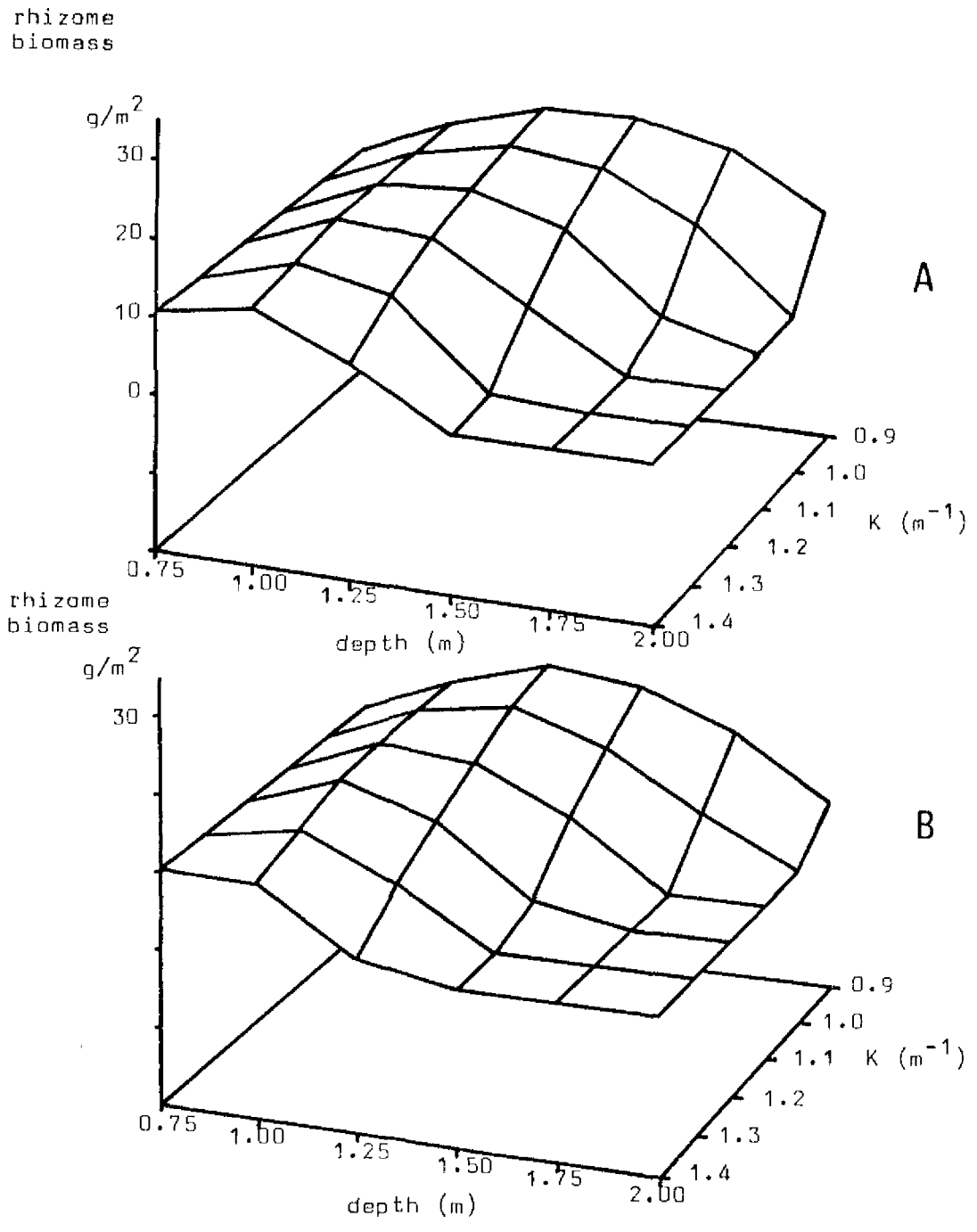

Figure 12: Simulation of rhizome biomass after one growing season, using 1931 and 1932 light values.
In ash-free dry weight, at different depths and k values. Actual light intensities of 1931 (a) and 1932 (b) were used for PAR input.

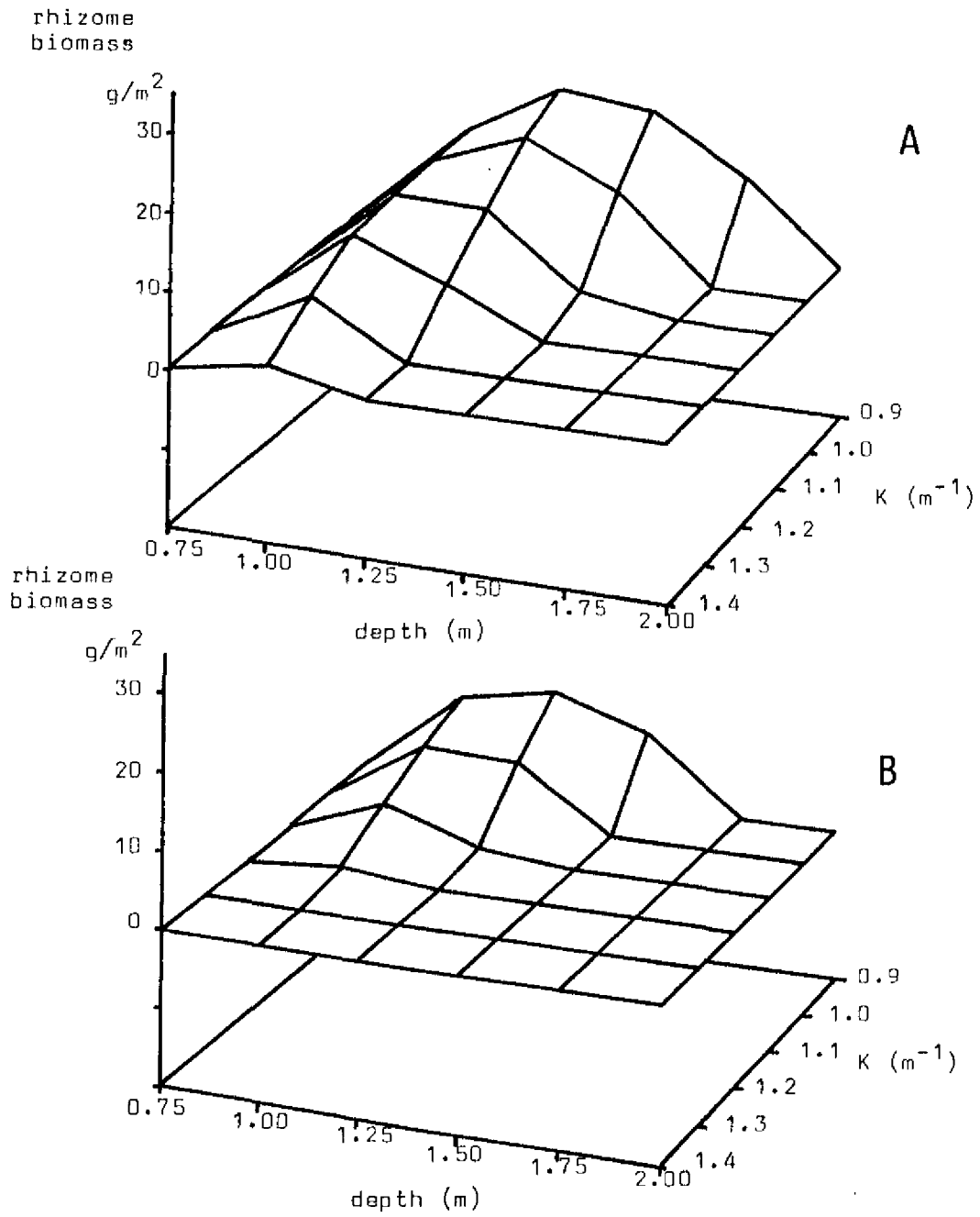

Figure 13: Simulation of rhizome biomass after two growing seasons, 1931-2, and 1931-2 + waterlevel increase.
In ash-free dry weight, and at different depths and k values. In the top figure (a), the 1931 end-of-season biomass was used as the base for 1932, which was subsequently run. The bottom figure (b) depicts the same, but incorporates a depth increase of 0.3m on 28th May, 1932 (x-axis depths are original depths).

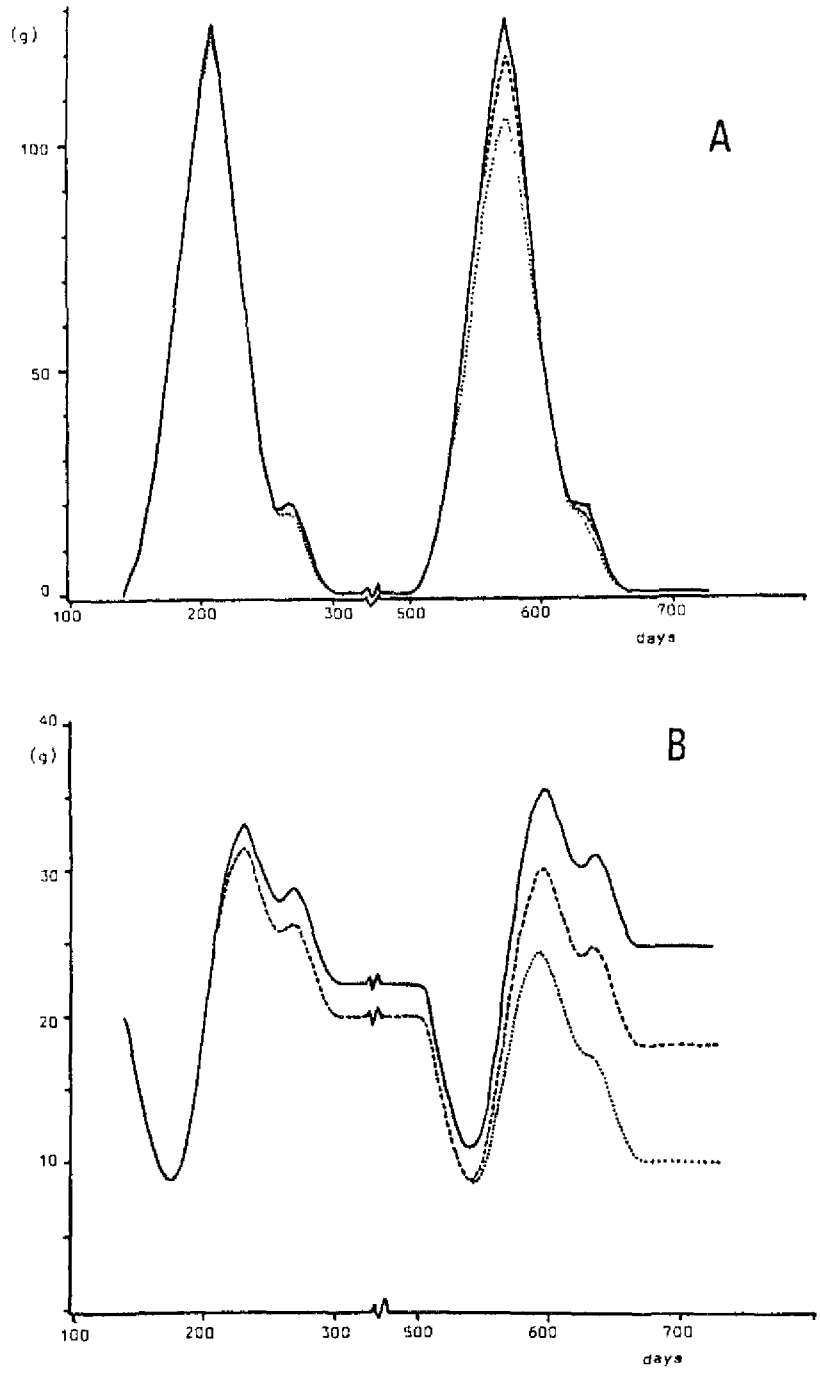

Figure 14: 2-year simulations of aboveground and rhizome biomass.
Both figures depict a two-year simulation of above-ground (a) and rhizome biomass (b) in ash-free dry weight, whereby the end-of-season rhizome biomass of the first year is used as the basis for the second year. ——= double cosine light function, – – = 1931 followed by 1932, ····· = 1931 followed by 1932 and a 30cm depth increase on 28/5/32.

CLIMATE AND WASTING DISEASE IN EUROPE IN THE 1930'S

TEMPERATURE

Rasmussen (1977) related wasting disease to high summer water temperatures in Danish waters. He found a good correlation between decline in eelgrass stands and days with water temperatures above 20°C, the temperature above which, according to Setchell (1929), heat rigor occurs in *Z. marina*. He also pointed out that winter seawater temperatures were also very mild at the time, and that this unique coincidence could account for the eelgrass decline. Quoting Rasmussen: «...the primary cause of the mass destruction of the *Zostera* in the 1930's can be found in the changed temperature conditions, so that the higher water temperature generally weakened the eelgrass, and thereby directly or indirectly contributed to its destruction... It should be noted that the severe, sudden temperature increase in the 1930's came after an unusually cool period...». His primary assumption is that within its large distribution area, *Z. marina* has a number of ecological races with different temperature sensitivity, especially with regard to the upper thermal limit.

Rasmussen's (1977) data are far from conclusive, however, even for the Danish situation. According to his data, only the mean temperature of the warmest month of 1932 is above 20°C, and even then it is only about 20.7°C. All other years from 1930-36 have averages below 20°C for the warmest months, and warmest months with temperatures of 20.7°C or more occurred thrice between 1900 and 1930 (Rasmussen's 1977 data). It would not appear that this phenomenon was as unique as Rasmussen suggests, and it is difficult to believe that one mildly warm month could have severely affected Z. *marina*. Another observation disrupting Rasmussen's hypothesis is the general consensus that deeper stands of eelgrass were affected first, while shallower stands (sites where one would expect higher temperatures) were often less affected. Blegvad (1934), for example, reports this pattern of wasting disease occurrence for Danish waters at numerous localities. This line of reasoning remains a little cloudy, however, as shallow water eelgrass stands may indeed be better adapted to higher temperatures (having a different phenotype). Furthermore, shallow environments referred to are often brackish, and it is known that *Labyrinthula macrocystis* is unable to infect eelgrass at salinities less than 12-15‰ (see chapter on autecology).

Atkins and Jenkins (1952) summary of temperature data of the English Channel for the period 1921-49 do not reveal anything unusual for the early 1930's. Both warmest and coldest months of all years between 1929-34 are about average, being surpassed by several other years in the period 1921-49. In short, their data certainly do not support a temperature based hypothesis of physiological stress in eelgrass.

On page 8 it was concluded that temperatures of Dutch coastal waters were slightly raised in the early 1930's, with a maximum of 0.9°C. This increased level was by no means unusual, nor were tolerance levels of *Z. marina* exceeded, or even approached. Cushing (1982) and Cushing and Dickson (1976) report a warming-up of the North Atlantic in the early 1930's by about 1°C. With only some data on Dutch and Danish coastal waters, and some general observations on the North Atlantic it is perhaps premature to conclude that temperature fluctuations had no effect on eelgrass stands in the early 1930's. It would be wise to support this with data from other localities and, if possible, with recordings from stations less remote from eelgrass stands. Laboratory temperature increase studies on thermally acclimatized eelgrass may also be helpful in identifying adverse temperature increase levels. Tentatively, one may conclude that it is unlikely that the slight temperature increases recorded in European coastal waters of the early 1930's had a directly adverse effect on eelgrass stands. However, this increase may have enhanced the effects of an already prevailing epidemic, by promoting parasite growth and retarding *Z. marina's* growth rate.

Surface seawater temperature data were obtained from nine European stations (Plymouth, station E1-English Channel, lightship Noordhinder-English Channel, Den Helder, West-Terschelling, lightship Helgoland-Reede, lightship Elbe-1, lightship Gedser Rev, lightship Vyl; see fig. 15 for locations). Data series from 1900 onwards were obtained for the first seven stations. Summer averages, plus averages of the warmest month of the year are given for these seven stations, together with long-term averages for both, in fig. 16a to 16g. Apart from the station Elbe-1 (fig. 16g), and perhaps station E1 (fig. 16a) , an abnormal warm-up during the early 1930's is not apparent. Temperatures are above average, but not dramatically or uniquely so.The departures from the norm are given in table 8. Stations E1 and Elbe-1 witnessed increases of 2.3 and 2.2 °C, respectively, during the warmest month of 1933 and 1932, respectively. At other stations this temperature increase of the warmest month ranged between 0.6 - 1.7 °C. Summer (July, August, September) temperatures witnessed an increase of 0.8 - 1.6 °C (average = 1.1 °C). The highest monthly average recorded at these nine stations during the period 1930 - 1933 was 19.3 °C (Elbe-1, August 1932). Average of all nine stations for the hotttest month of 1930 - 1933 is 18.2 °C. It is thus likely that temperatures above 20 °C were not witnessed at most stations during the period 1930 - 1933. At all stations except the Elbe-1 (data were lacking for Gedser Rev , at 54° 21'N/07° 40'E, and Vyl, at 55° 21'N/07° 40'E), the 1930 - 1933 maxima (for both summer and warmest month) were surpassed during other years of the period 1900 - 1987. One may conclude that, as temperatures very likely remained well below the critical temperature of 20°C (Setchell, 1929; Giesen, 1988), and as the maxima recorded in 1930 - 1933 were not unique for the period 1900 - 1987, temperature anomalies are unlikely to have initiated the wasting disease epidemic of the early 1930's. At least, not in western Europe. This is contrary to what is concluded in the often quoted paper by Rasmussen (1977), who analysed the situation for Danish waters only.

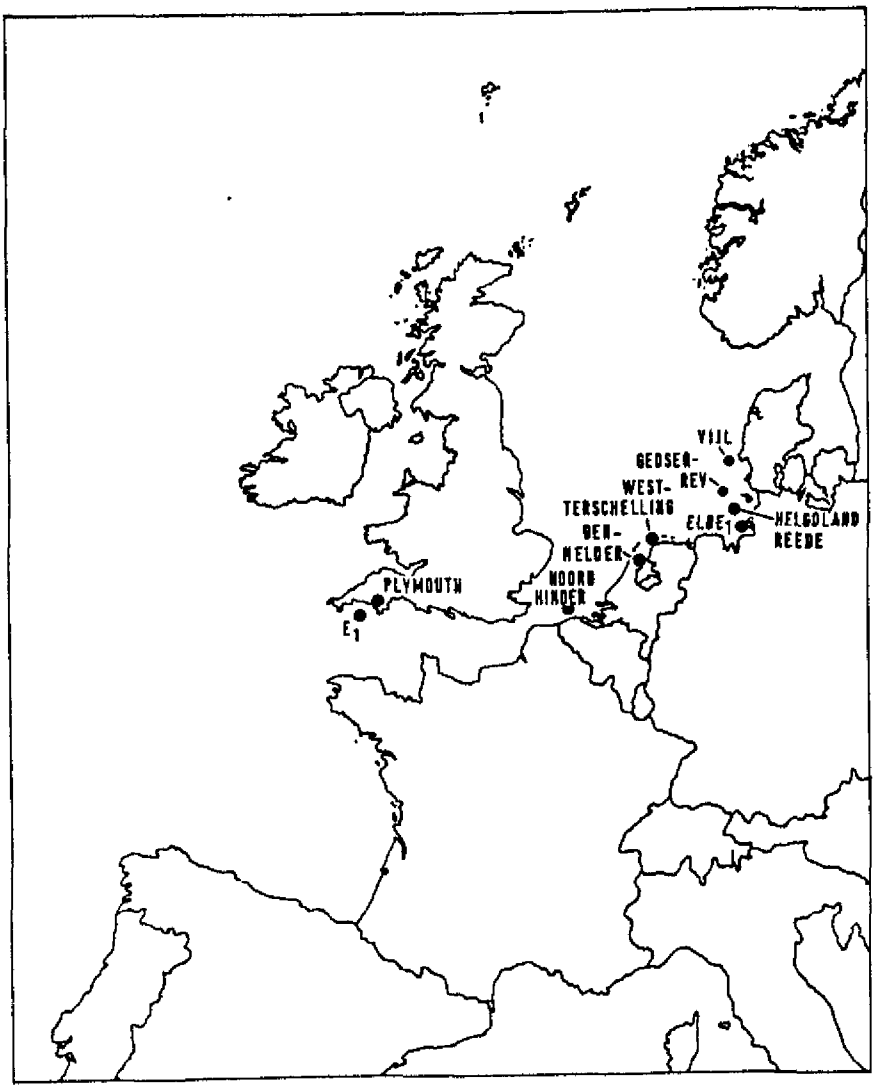

Figure 15: Location of stations.

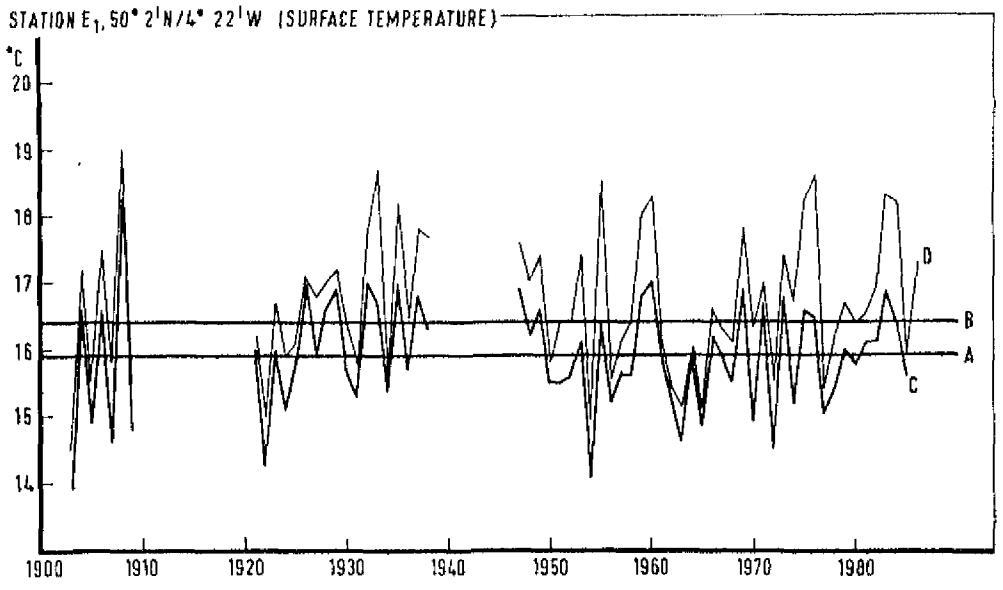

Figure 16a Seawater temperature, station E1.
A = long-term summer (J/A/S) average; B = long-term average for warmest month (August); C = summer (J/A/S) average of given month; D = average for warmest month of given year.

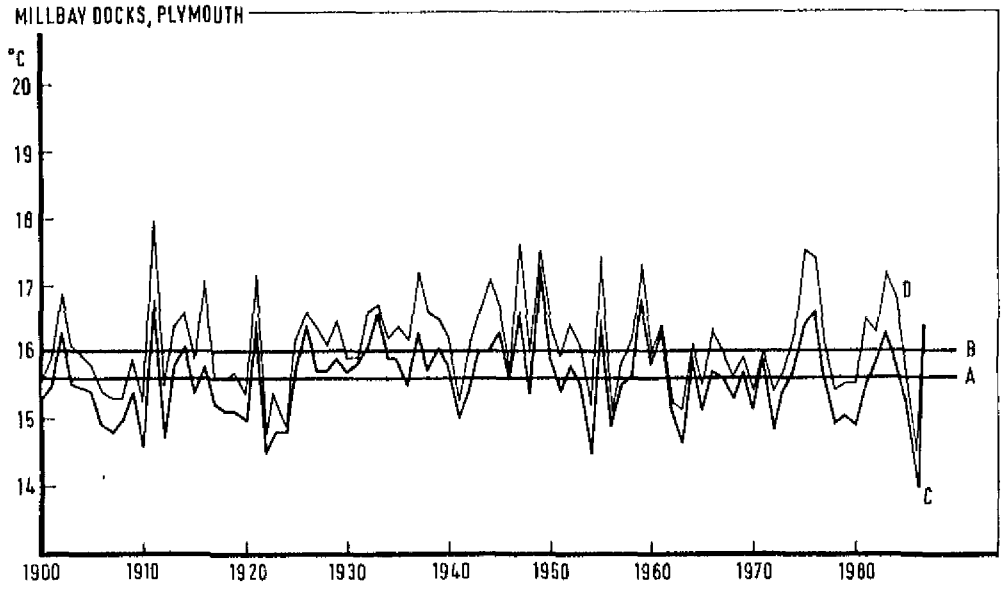

Figure 16: b: Surface seawater temperature Plymouth (Millbay Docks).
A = long-term summer (J/A/S) average; B = long-term average for warmest month (August); C = summer (J/A/S) average of given month; D = average for warmest month of given year.

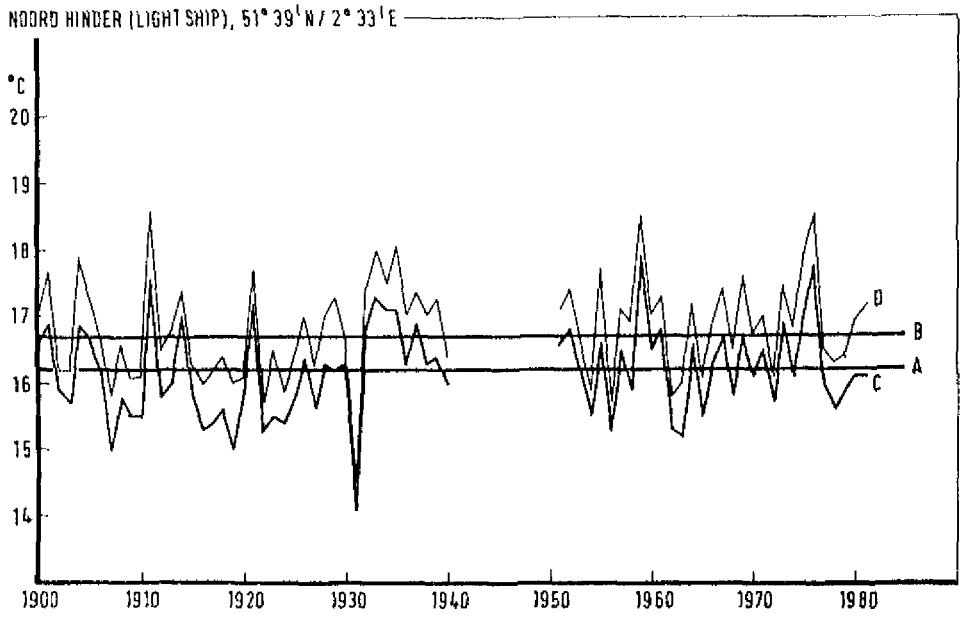

Figure 16: c: Surface seawater temperature Noordhinder lightship.
A = long-term summer (J/A/S) average; B = long-term average for warmest month (August); C = summer (J/A/S) average of given month; D = average for warmest month of given year.

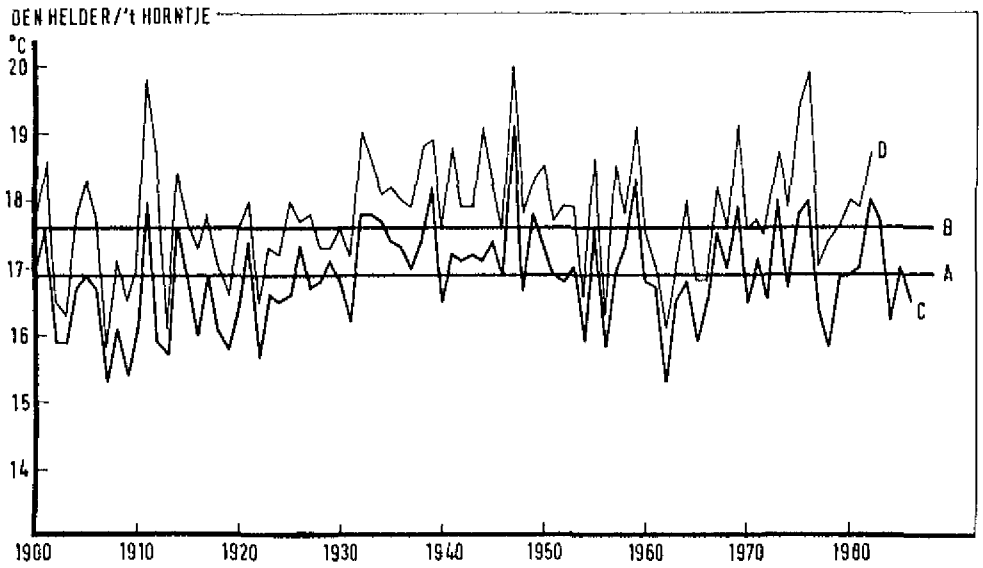

Figure 16: d: Surface seawater temperature Den Helder.
A = long-term summer (J/A/S) average; B = long-term average for warmest month (August); C = summer (J/A/S) average of given month; D = average for warmest month of given year.

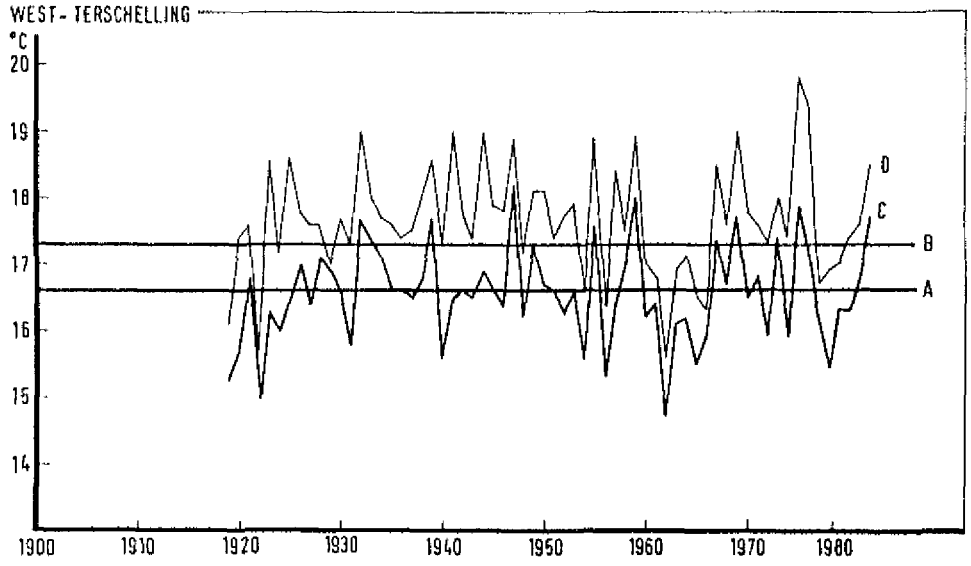

Figure 16: e: Surface seawater temperature West-Terschelling.
A = long-term summer (J/A/S) average; B = long-term average for warmest month (August); C = summer (J/A/S) average of given month; D = average for warmest month of given year.

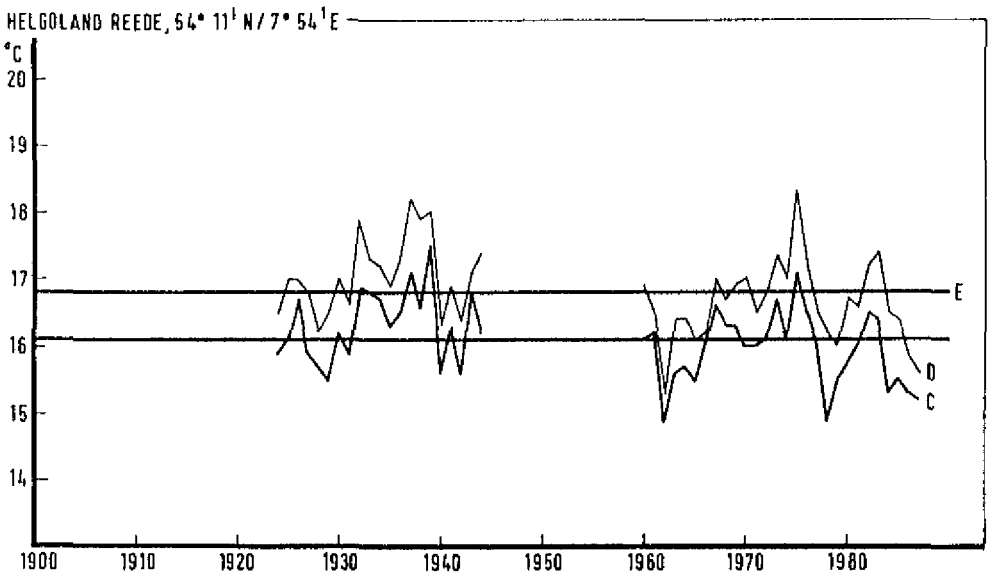

Figure 16: f: Surface seawater temperature Helgoland Reede lightship.
A = long-term summer (J/A/S) average; B = long-term average for warmest month (August); C = summer (J/A/S) average of given month; D = average for warmest month of given year.

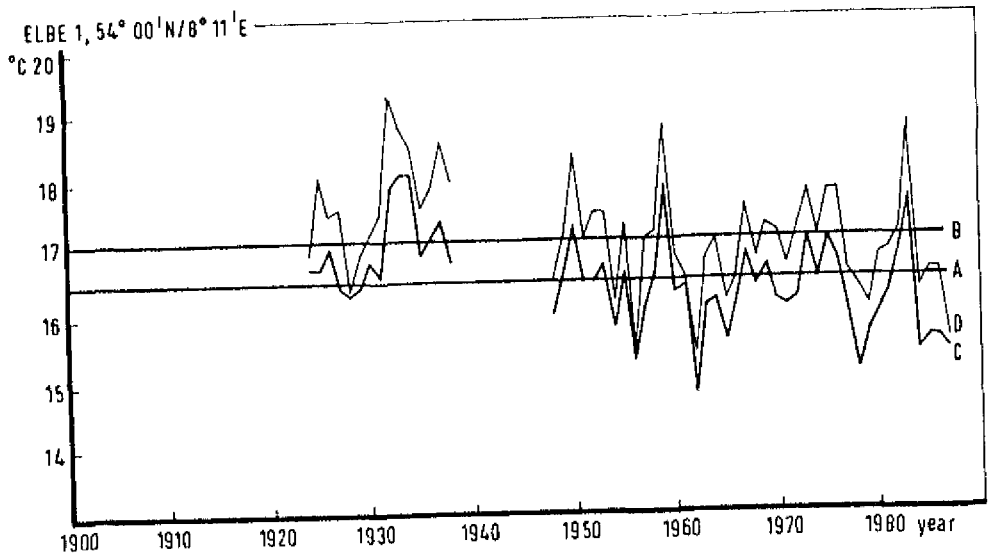

Figure 16: g: Surface seawater temperature Elbe-1.
A = long-term summer (J/A/S) average; B = long-term average for warmest month (August); C = summer (J/A/S) average of given month; D = average for warmest month of given year.

Table 8: Surface seawater temperatures, 1900 - 1987.

	A	B	C	D	E	F	G	H	I
long-term average summer temperature	16.2	16.9	16.6	15.6	15.9	16.1	16.5	16.0	16.1
long-term average warmest month	16.7	17.6	17.3	16.0	16.4	16.8	17.1	16.7	16.8
summer '30	16.3	16.8	16.6	15.7	15.7	16.2	16.8	---	---
" '31	14.1	16.2	15.8	15.8	15.3	15.9	16.6	14.8	15.4
" '32	16.8	17.8	17.7	16.1	17.0	16.9	17.9	17.0	17.1
" '33	17.3	17.8	17.4	16.6	16.7	16.8	18.1	16.9	16.6
warmest month '30	16.7	17.6	17.7	15.9	16.4	17.0	17.2	---	---
" " '31	14.5	17.2	17.3	15.9	15.8	16.6	17.5	15.5	16.4
" " '32	17.4	19.0	19.0	16.6	17.7	17.9	19.3	18.0	17.4
" " '33	18.0	18.6	18.0	16.7	18.7	17.3	18.8	17.8	17.2
extremes:									
summer:	17.9	19.1	18.2	17.3	18.3	17.5	19.3		
year:	1959	1947	1947	1949	1908	1939	1932		
warmest month:	18.6	20.0	19.8	18.0	19.0	18.3	18.1		
year:	1911	1947	1975	1911	1908	1975	1932		
years exceeding 1930-33 max.:									
summer average	2x	8x	3x	3x	1x	3x	none	---	---
warmest month	3x	6x	1x	12x	1x	3x	none	---	---
temperature above average in warmest summer 1930-33	1.1	0.9	1.1	1.0	1.1	0.8	1.6	1.0	1.0
temperature above average in warmest month 1930-33	1.3	1.4	1.7	0.7	2.3	1.1	2.2	1.3	0.6
number of years recorded in 1900-87	72	87	64	88	66	49	55	---	---

notes:
- summer = July, August, September
- A = Noordhinder, B = Den Helder, C = West-Terschelling, D = Plymouth (Millbay Docks), E = Station E1, F = Helgoland, G = Elbe-1, H = Gedser Rev, I = Vyl.

SALINITY

It was noticed during the wasting disease epidemic that eelgrass stands of brackish waters, such as the Baltic Sea, estuaries and littoral zones, often escaped infection, or did not display the typical wasting disease symptoms and mass mortality (Van der Werff, 1934; Blegvad, 1934; Rasmussen, 1977; den Hartog, 1987; Short *et al*, 1988). Petersen (1935) reports that no wasting disease symptoms occurred at salinity levels below about 10‰, but Rasmussen (1977) considers this threshold to be in the range of 12-15‰. This latter value is also supported by recent studies by Short *et al* (1988) on a wasting disease-like illness of eelgrass (also with *Labyrinthula sp.* as the primary pathogen, and with similar symptoms). They have demonstrated that salinities of 15‰ or less prevent the spreading of lesions by *Labyrinthula* to new leaves. The mechanism by which this low salinity inhibition seems to operate is probably twofold (for field situations, at least). Firstly, *L. macrocystis* is an obligate marine species, having a salinity optimum of 22-40‰ (Young, 1943) or perhaps even as high as 30-42‰ (Pokorny, 1967). It may withstand immersion in freshwater for short periods (Young, 1943), but it will succumb to longer periods of exposure. A second mechanism explaining the apparant escape of brackish water eelgrass stands is the fact that these populations often differ from the typical sublittoral stands. In these sublittoral beds, *Z. marina* is perennial and has a low generative output, whereas littoral brackish water populations are generally annuals, and have a high seedset (den Hartog, 1987; Keddy, 1987).

Martin (1954) correlated declines in eelgrass stands with precipitation extremes; wasting disease on the USA Atlantic coast correlated with drought, while that of the western European coast seemed to correlate with above average precipitation. His analysis of meteorological data of six stations (Aberdeen, Greenwich, Utrecht, Paris, Lyons and Marseilles) showed an average of 22% above-average rainfall in 1930, with other peaks shortly before and after this date. He proposed three mechanisms by which this could have had detrimental effects: excessive freshness, excessive turbidity and submergence under silt. Though precipitation was indeed above average, there is little evidence that turbidity, salinity or siltation alone may have had much effect on eelgrass vitality. The Dutch Wadden Sea is under strong influence of the Rhine River (Gieskes and Kraay, 1977); nevertheless, salinity levels were not abnormal during these years of "excessive precipitation". It is also highly unlikely that a lowering of the salinity could induce wasting disease, as *Z. marina* can withstand lower salinities much better than the parasite, *L. macrocystis*. There is furthermore no evidence of an opposite situation of raised salinity levels in west European coastal waters prior to the wasting disease epidemic. Most meteorological records show above-average rainfall and below-average insolation, a combination that does not promote salinity increases. Salinity data of surface waters were obtained for eight European stations (station E1 - English Channel, Noordhinder, Den Helder, West-Terschelling, Helgoland-Reede, Elbe-1, Gedser Rev and Vyl; see fig. 15). Of the first six, data were available for the entire period 1900-1987. These are given below in fig. 17), where salinity maximum and minimum are given for each year, along with the long-term average salinity.

In all cases there is no salinity anomaly evident in the early 1930's, as was suggested by Martin (1954). At all stations salinity was well within the normal range for that site, with greater extremes witnessed at the same station during other years. One may conclude that abnormal salinities are unlikely to have contributed to the outbreak of wasting disease in Europe during the early 1930's.

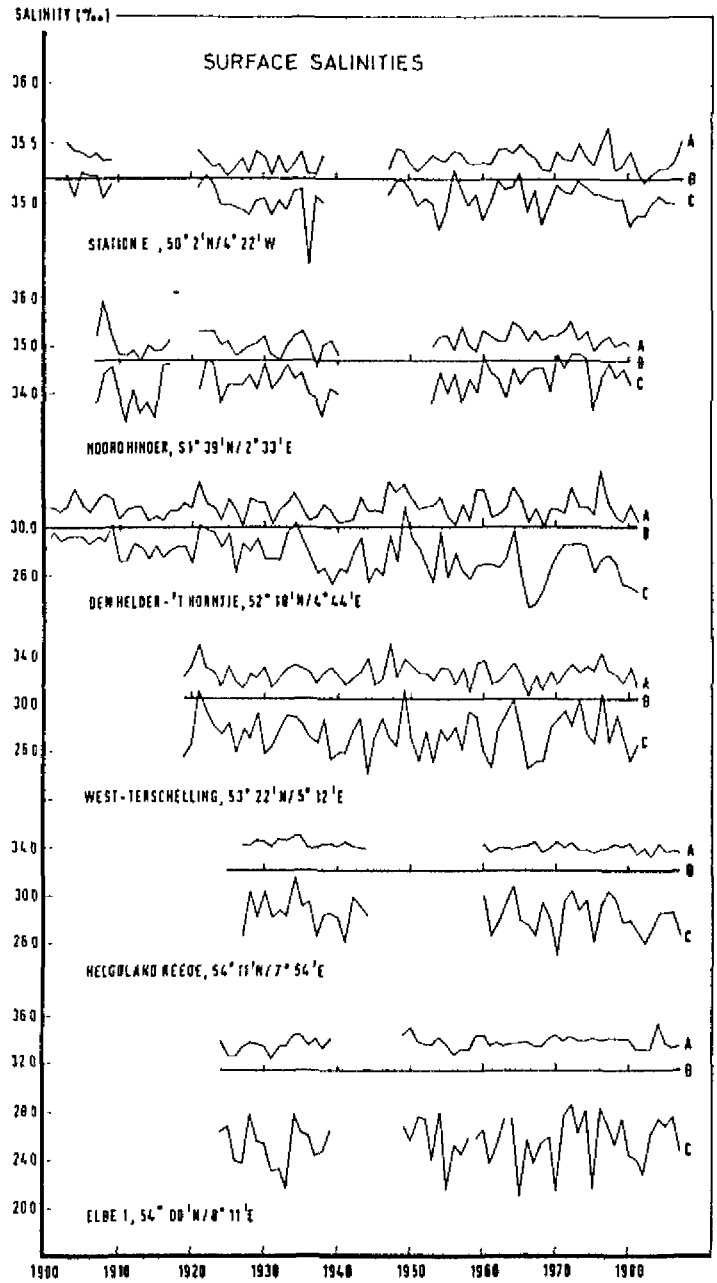

Figure 17: Surface seawater salinity for six European stations. Locations are given in fig. 8). A = highest monthly average, B = long-term average, C = lowest monthly average.

Fluctuations in salinity levels probably did not contribute to the outbreak of wasting disease either. Conditions generally lead to a slight lowering of salinity levels, a change that favours eelgrass and not *L. macrocystis*. Areas with naturally lower salinity levels (i.e. brackish waters) were less affected by wasting disease, not only due to the aforementioned mechanism, but also due to the fact that eelgrass of brackish habitats usually has an annual growth form. This form has a higher seed setting rate than the perennial, sublittoral form, and is more likely to escape infectious diseases (den Hartog, 1987).

SUNLIGHT

Lack of sunshine as a possible cause of wasting disease was first proposed by Tutin (1938) for the British Isles. He was soon refuted by Atkins (1938), and that line of thought was abandoned. However, it was shown that limited light conditions may indeed have played an important role in initiating wasting disease in the Dutch Wadden Sea. The difference in approach, compared to Tutin and Atkins, is that meteorological data in the previous chapters were analysed on the basis of daily and monthly figures, instead of annual values.

Is there evidence to support the hypothesis that sunshine deficiency may have been a widespread phenomenon in Western Europe during the growing seasons of 1931-2, and that this may have contributed significantly to the outbreak of wasting disease ? In fig. 19, 20, 21 and 22 sunshine data (monthly averages) are presented for the years 1931-33 for sixteen stations in Western Europe (for locations, see fig. 18)). Below average sunshine is evident at most stations in April, May and July, 1931, April, May and August, 1932, and April and May, 1933. Percentages above or below the norm are given for the growing season months in table 9. Averages are given below:

	April	May	June	July	Aug.	Sept.
1931	-19.8	-17.4	-11.9	-29.8	+5.2	-5.9
1932	-6.1	-18.8	+10.1	-11.4	-7.8	-11.9
1933	+0.4	-15.4	+10.5	+4.9	+15.2	+22.9

Maximum departures from the norm for Den Helder (in May) were minus 25-27% in 1931-33; other stations show even greater differences, for instance in April 1931: Rennes -52%, Bovbjerg -43%; Dundee -35%; May 1931: Rennes -42%, Bovbjerg -38%, Tylstrup -34%, Helgoland -30%; June 1931: Cork -41%, Dublin -40%, Helgoland -30%; July 1931: Bovbjerg -52%, Dundee -51%, Oban -46%, Dublin -45%, etc... . From these data one may conclude that a strong deficiency of sunshine occurred over a considerable part of Western Europe during 1931-2. This sunshine deficient area included the British Isles, Brittany (France), the Netherlands, the German North Sea coast and Denmark; it did not extend as far south as Portugal, however. It would also appear that the deficiency recorded at Den Helder station is mild compared to other stations (eg.: Rennes, Plymouth, Dundee, Dublin, Helgoland, Bovbjerg). Tentatively, one may conclude that, if lack of sunshine played an important role in the initiation of wasting disease in the Dutch Wadden Sea, this was also the case in a large part of Western Europe.

Several questions remain, however, as this hypothesis does not explain why wasting disease was not reported in Scotland, Wales and Northumberland (Butcher, 1934), and why it did seem to occur in Portugal (Henriques, 1933). Whelan and Cullinane's recent (1987) article on wasting disease in Ireland may help explain this phenomenon. Prior to their article, only one publication had been widely quoted with regard to wasting disease in Ireland (Renouf, 1934), and upon re-examination this was found not to describe wasting disease, but merely the normal seasonal changes of *Zostera*. After searching through more obscure literature and interviewing persons living on the Irish coastline, they found evidence for several hitherto unrecorded incidences of wasting disease in the early 1930's. Similarly, the fact that wasting disease was not recorded in northern England and Scotland may thus not mean that it did not occur, but that it escaped being recorded. Henriques (1933) observation of eelgrass decline in the Aveiro lagoon, near Coimbra, dates from August 1932 (quite late in the growing season), and it is not unlikely that this was a normal seasonal event.

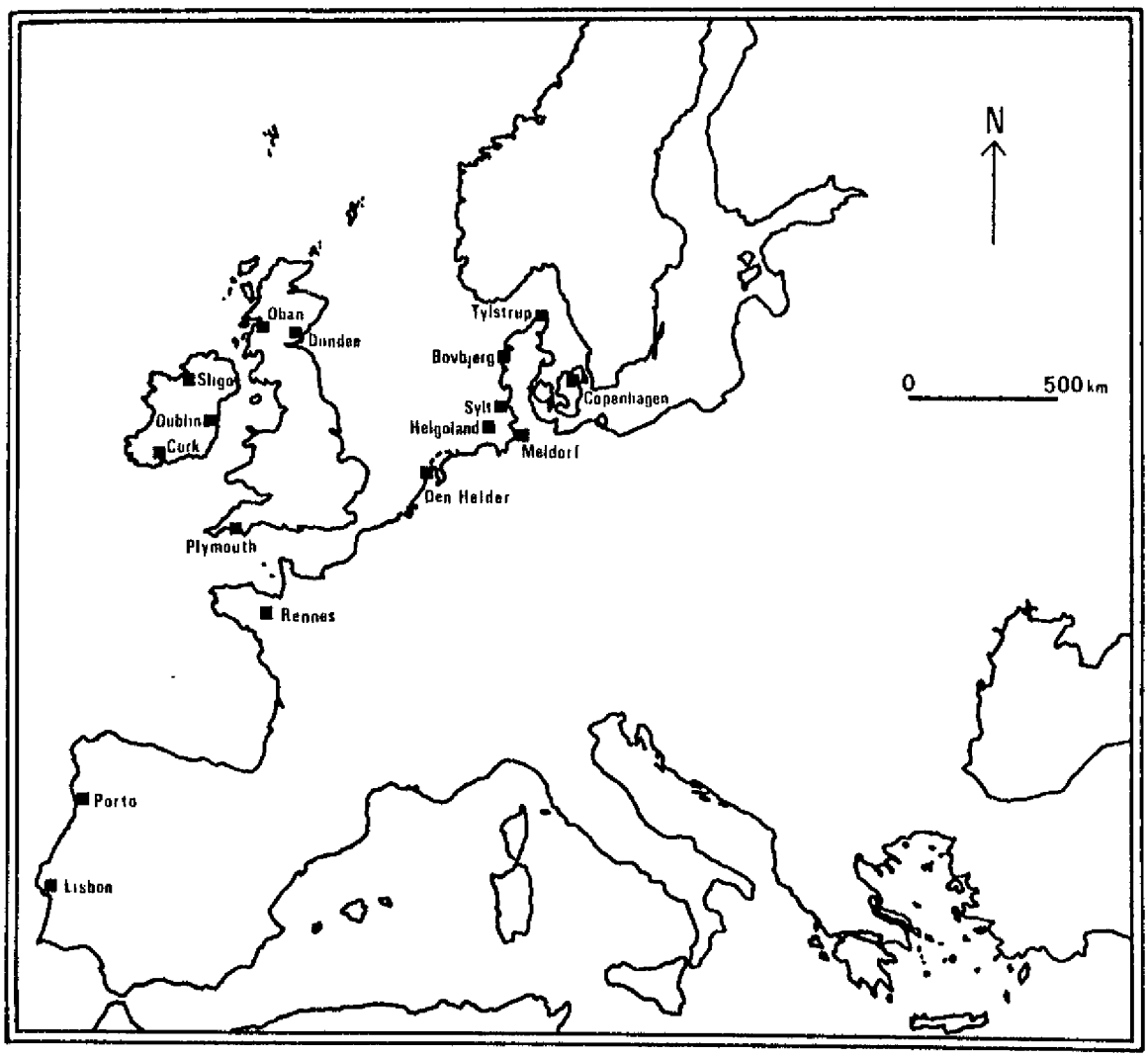

Figure 18: Location of sixteen European meteorological stations.

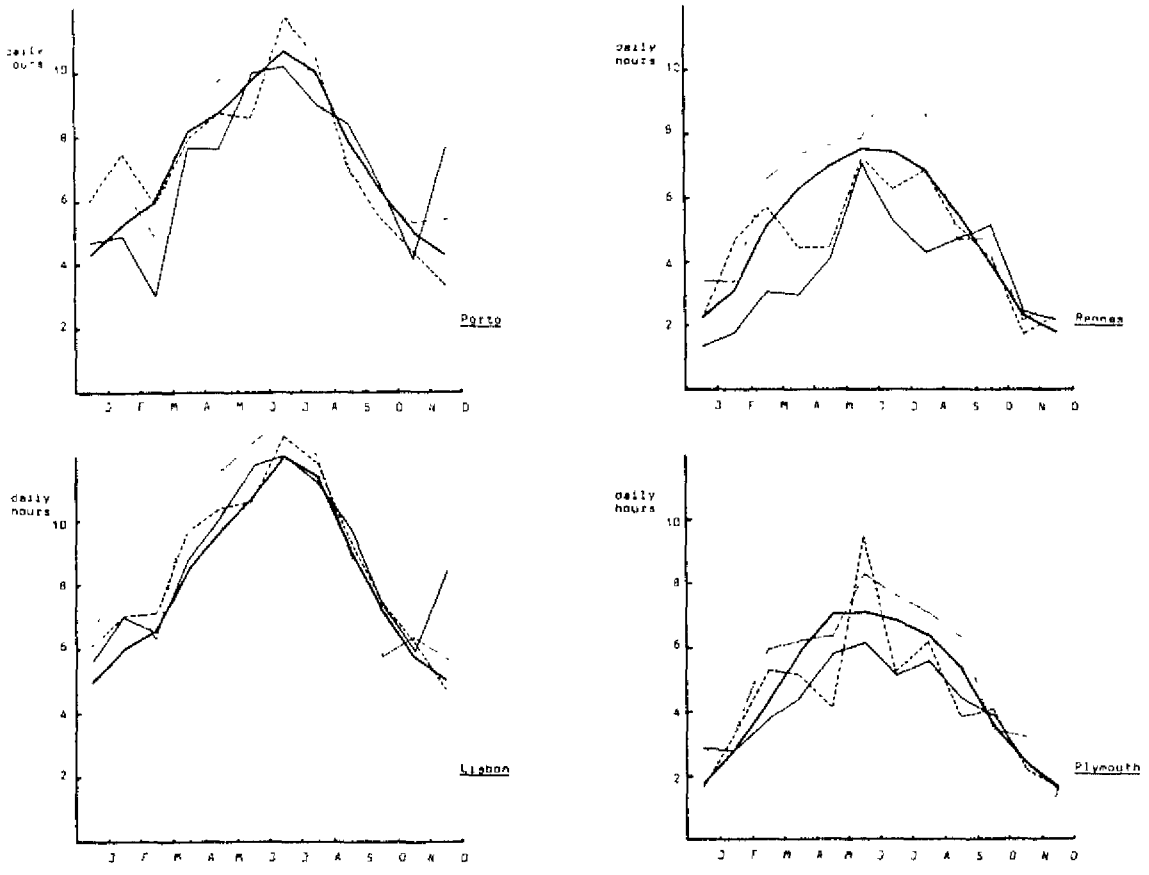

Figure 19: **Hours sunshine for Porto, Lisbon, Rennes & Plymouth..**
(in monthly averages): ▬ = long-term average; — = 1931; - - - = 1932; ····· = 1933

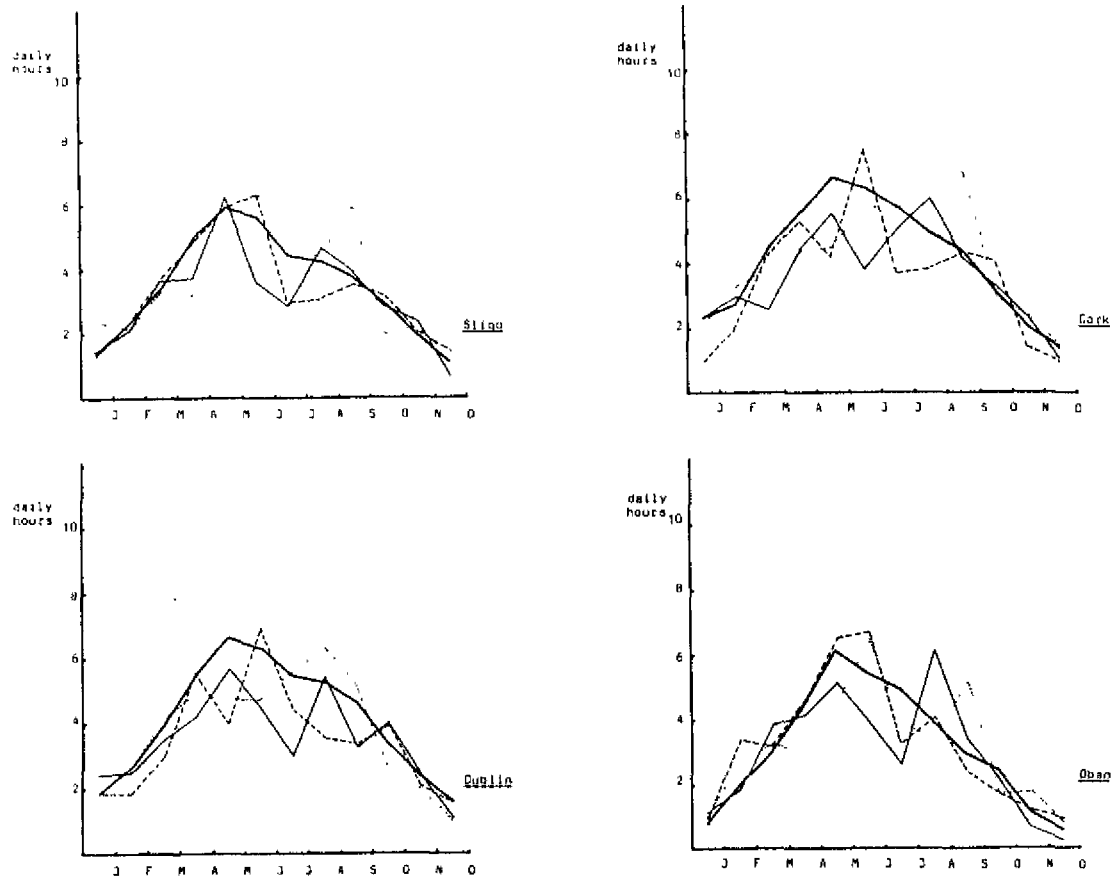

Figure 20: Hours sunshine for Sligo, Dublin, Cork & Oban..
(in monthly averages): ▬ = long-term average; —— = 1931; - - - = 1932; ····· = 1933

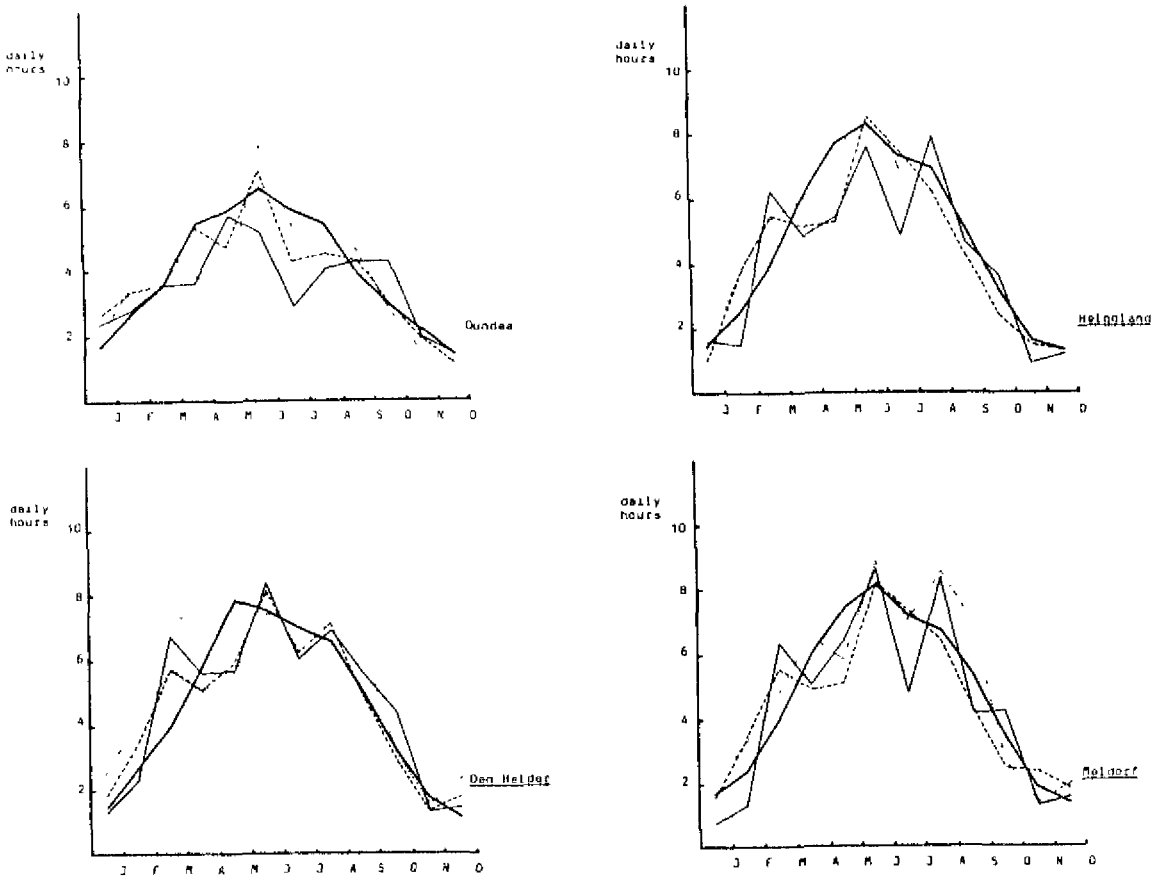

Figure 21: Hours sunshine for Dundee, Den Helder, Helgoland & Meldorf.. (in monthly averages): ▬ = long-term average; — = 1931;- - - = 1932;····· = 1933

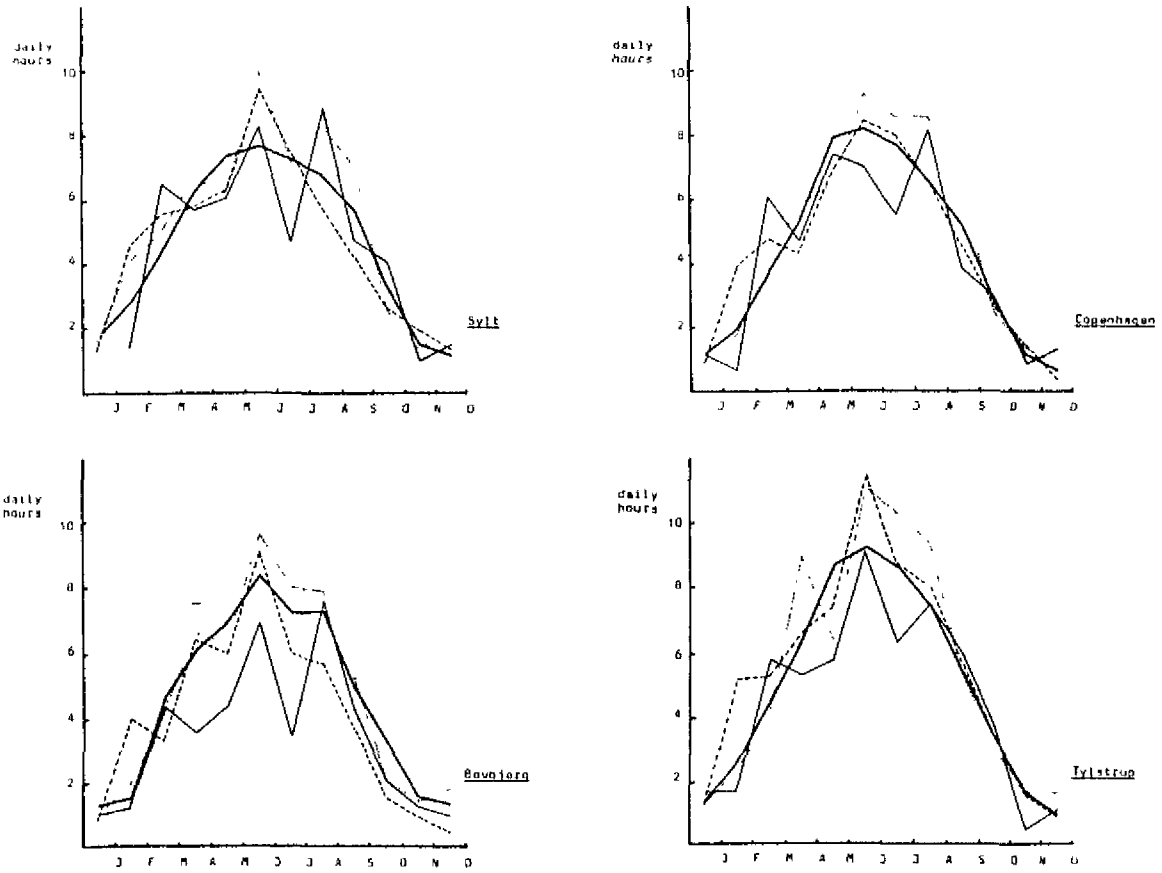

Figure 22: Hours sunshine for Sylt, Bovbjerg, Copenhagen & Tylstrup..
(in monthly averages): ▬ = long-term average; — = 1931;– – – = 1932;····· = 1933

Table 9: Monthly sunshine in Western Europe, 1931-33

Departures from the norm are given in (in %'s).

		April	May	June	July	August	September
1931	Den Helder	-5	-27	+11	-14	+5	+11
	Porto	-5	-13	+3	-5	-10	+8
	Lisbon	+3	+5	+10	0	-2	+7
	Rennes	-52	-42	-8	-30	-37	-15
	Plymouth	-26	-18	-14	-26	-14	-19
	Dundee	-35	-3	-22	-51	-26	+10
	Oban	-5	-13	-26	-46	+55	+17
	Sligo	-26	+5	-36	-35	+10	+5
	Dublin	-24	-15	-40	-45	+4	-32
	Cork	-21	-18	-41	-12	+22	-5
	Meldorf	-16	-14	+7	-34	+22	-23
	Helgoland	-21	-30	-9	-34	+14	-10
	Sylt	-10	-17	+7	-36	+14	-18
	Bovbjerg	-43	-38	-18	-52	+4	-14
	Tylstrup	-17	-34	-1	-27	0	+11
	Copenhagen	-13	-7	-14	-29	+22	-28
1932	Den Helder	-13	-25	+8	-11	+10	-2
	Porto	-2	0	-12	+10	+5	-12
	Lisbon	+14	+7	-1	+5	+4	+3
	Rennes	-29	-37	-4	-15	0	-7
	Plymouth	-12	-41	+33	-24	-5	-28
	Dundee	-4	-20	+8	-27	-17	+8
	Oban	+9	+8	+24	-34	+7	-17
	Sligo	-4	0	+13	-34	-27	-5
	Dublin	0	-40	+10	-20	-33	-28
	Cork	-5	-37	+18	-35	-22	0
	Meldorf	-18	-31	+2	+3	-4	-23
	Helgoland	-16	-31	+2	+3	-11	-18
	Sylt	-8	-13	+23	+3	-15	-26
	Bovbjerg	+5	-13	+8	-16	-23	-27
	Tylstrup	+3	-15	+25	0	+7	+4
	Copenhagen	-18	-13	+4	+12	0	-12
1933	Den Helder	-13	-27	-1	+6	+20	+34
	Porto	+12	+11	+8	+12	-5	-10
	Lisbon	+25	+19	+16	+8	+5	0
	Rennes	+16	+8	+3	+31	+25	-7
	Plymouth	+7	-10	+17	+11	+10	+17
	Dundee	-31	-41	+21	-9	+15	+21
	Oban	-32	-25	+20	-20	-13	+76
	Sligo	-35	-14	+13	+4	+5	+59
	Dublin	-11	-30	-24	+2	+17	+11
	Cork	-23	-32	-11	-2	+35	+55
	Meldorf	+9	-22	+9	-1	+27	+30
	Helgoland	-1	-15	+19	-6	--	--
	Sylt	+6	-14	+29	-2	+24	+26
	Bovbjerg	+9	-9	+14	+12	+9	+6
	Tylstrup	+40	-27	+21	+20	+24	+4
	Copenhagen	+28	-18	+14	+12	+30	+21

CLIMATE AND WASTING DISEASE IN NORTH AMERICA IN THE EARLY 1930'S

INTRODUCTION

The history of eelgrass decline along the USA Atlantic coast during the wasting disease epidemic is best recorded in a series of publications by Cottam (1933, 1934, 1935, 1937, 1938, 1940, 1945, 1947, 1949) and Renn (1934, 1936a, 1936b, 1937, 1942). The disease was first recorded in Virginia in 1930 (Lewis, 1931; Huntsman, 1931), from where it appeared to rapidly spread in a northerly direction in the course of the next two years. By 1932 most eelgrass stands between North Carolina and Nova Scotia had been devastated. Even during these years of peak eelgrass destruction, however, a degree of recovery was recorded at several localities. In most cases this was only of a temporary nature, and usually regrowth was soon followed by new mortality. The first signs of a more definite recovery was reported from low salinity areas, such as Chesapeake Bay (Virginia/Maryland), Shinnecock Bay (Long Island, New York) and the Great Bay (New Hampshire). Eelgrass had never completely disappeared from these areas, and Renn (1937) reports from these sites that, although leaves were affected, growth rates remained high.

The appearance of wasting disease in the early 1930's coincided with exceptionally dry and warm weather in the USA. Unusually high temperatures were recorded all down the east coast from Florida to Maine in 1930, from Virginia to Maine in 1931, and from North Carolina to Maine in 1932 (annual reports, USDA Weather Bureau). Severe drought prevailed in most areas in 1930 and drops of up to 50% below the norm were recorded. Several authors (Stevens, 1936; Martin, 1954) related these abnormal conditions to wasting disease, arguing that water temperatures and salinities were raised, and had probably affected eelgrass populations. Unfortunately, actual data were never consulted, and their arguments remained hypothetical. In the following, seawater temperature, salinity and light conditions are dealt with.

SEAWATER TEMPERATURES

Stevens (1936) found an apparent correlation between past periods of eelgrass decline (as reported by Cottam, 1934, 1935) and transgressions in the North Atlantic. These transgressions consist of temporary encroachments of warmer, tropical waters upon temperate waters, resulting in temporary increases in water temperature, and decreases in fish catches. However, seawater temperature data were not analysed to substantiate this assumption.

In table 10 below, seawater surface temperature data are presented for seven stations along the USA east coast (see fig. 23 for locations); data were kindly made available by Dr. J.R. Hubbard of the NOAA, Rockville, Maryland. Summer water temperatures do not depart much from the norm during the years 1929-31. The greatest deviation for this period is +1.2°C for Eastport, Maine, in 1930. When regarded on a monthly basis, the highest departure from the norm is +1.5°C for the Battery, New York, July 1931. Averages for the seven stations are, for 1929: -0.5°C; for 1930: +0.1°C; for 1931: +0.4°C. for 1932 (not presented in the table). Winter water temperatures were, at the most, only slightly above normal, the highest increase being +0.6°C for Portland and Charleston (both winter 1929).

Average increases are, for 1929: +0.2°C; for 1930: +0.1°C; for 1931: -0.4°C. When regarded on a monthly basis, the greatest departures from the norm is -1.7°C, for Charleston, March 1929.

When considering the effects on *Zostera marina* growth, one must bear in mind that this species does not occur further south on the USA east coast than Beaufort, North Carolina (den Hartog, 1970). It is unlikely that these minor increases in seawater temperature could have caused enough physiological stress in eelgrass to induce wasting disease, even though in some areas (Baltimore, Atlantic City and the Battery, NY) summer temperatures are probably above the growth optimum. Increases in summer seawater temperature of 1°C or more (in one or more months) occurred regularly along the Atlantic coast of the USA. For Charleston this occurred in 12 out of 64 recorded years, Baltimore: 20 out of 72, Atlantic City: 34 out of 73, the Battery, NY: 14 out of 59, Boston: 36 out of 65, Portland: 23 out of 54, and Eastport: 13 out of 56 (NOAA data). These departures from the norm come and go, without any apparent wasting disease effects (except, perhaps, in recent years; see part 3)). The only likely adverse effect of these slight temperature increases is that the growth of the pathogen, *L. macrocystis*, is favoured above that of *Z. marina*, as the former has a higher temperature optimum.

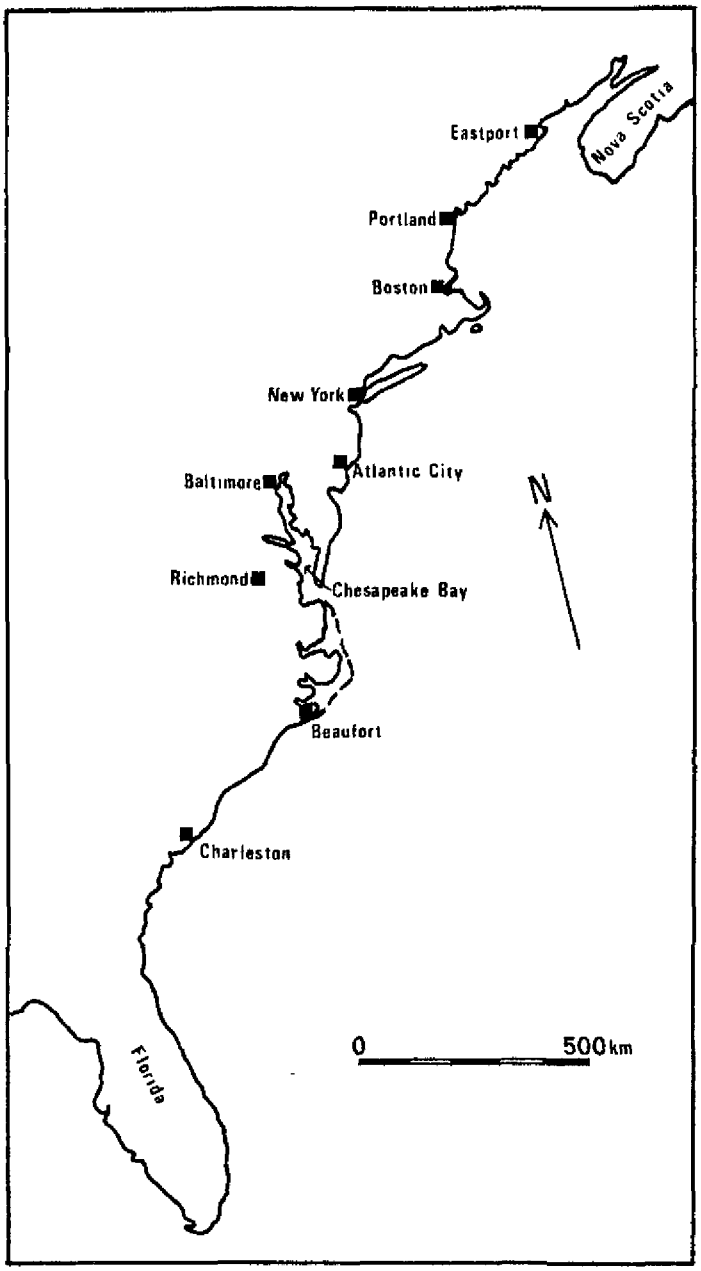

Figure 23: Station location map, east coast USA.

Table 10: Coastal seawater temperatures and salinities, east coast USA, 1929-31, in°C.

	J	F	M	A	M	J	J	A	S	O	N	D	
Charleston													
temperature average	10.9	11.4	14.1	18.7	23.2	26.9	28.4	28.5	26.7	22.2	15.8	11.9	'29-'50
1929	11.4	11.0	15.8	20.6	23.7	26.3	28.3	28.6	25.9	21.2	17.7	11.1	
1930	10.8	12.0	13.4	18.2	24.3	25.7	28.9	27.7	28.0	21.1	14.6	10.1	
1931	8.4	10.9	12.0	17.5	22.5	26.4	29.6	28.4	27.8	22.9	17.5	16.2	
salinity average	30.3	29.8	28.2	28.8	30.3	31.8	30.6	30.7	29.9	30.2	31.4	31.4	'21-'42
1929	29.7	26.0	23.3	26.7	28.5	28.5	28.1	30.3	30.1	28.6	29.4	28.6	
1930	27.5	27.1	28.4	26.5	29.5	31.0	31.*	33.1	33.2	32.9	32.5	32.1	
1931	30.7	30.8	30.6	28.9	30.8	32.7	32.5	33.6	33.3	33.5	34.1	34.2	
Baltimore													
temperature average	2.6	2.4	7.1	11.2	17.6	23.3	26.3	26.1	21.7	18.2	11.8	5.7	'14-'43
1929	2.8	1.8	6.7	12.7	17.7	23.4	25.9	25.4	23.6	16.7	12.5	4.6	
1930	3.2	3.6	6.6	10.6	18.6	23.7	26.9	26.1	25.5	18.5	11.3	4.8	
1931	2.2	1.3	5.5	11.6	17.6	23.2	27.3	27.0	25.8	19.9	14.2	9.2	
salinity average	9.8	9.6	8.1	5.9	5.6	6.0	6.7	7.6	9.3	10.5	10.6	10.5	'14-'43
1929	10.2	11.5	6.0	4.1	2.6	3.9	5.9	7.6	9.2	9.6	9.3	9.0	
1930	7.6	9.2	7.1	5.5	7.5	9.7	8.6	9.6	11.9	14.9	17.4	17.5	
1931	17.1	17.0	17.4	9.8	7.7	6.4	8.1	9.2	11.9	12.1	13.3	12.9	
Atlantic City													
temperature average	2.7	1.9	4.1	8.2	13.1	18.0	20.6	21.8	20.8	16.1	10.5	5.1	'12-'43
1929	2.9	1.7	5.1	8.6	12.2	17.5	19.8	21.0	20.3	14.5	10.8	4.5	
1930	3.1	2.5	4.7	7.9	13.8	17.4	20.5	20.9	22.2	14.6	9.8	4.2	
1931	1.8	2.0	3.9	5.5	12.6	17.8	21.4	22.1	21.6	17.2	12.5	7.5	
salinity average	31.4	31.5	31.4	31.1	31.4	31.8	31.9	31.6	31.5	31.6	31.6	32.4	'12-'43
1929	32.1	32.1	32.1	31.1	31.9	32.4	32.7	32.7	32.5	32.1	31.9	31.8	
1930	31.4	31.5	31.5	32.0	32.8	32.8	33.1	32.7	32.9	32.9	32.5	32.4	
1931	32.1	32.1	31.9	31.8	32.3	31.9	31.4	31.0	31.6	32.1	32.3	32.1	
the Battery, N.Y.													
temperature average	3.2	2.2	5.8	7.7	13.0	18.4	22.0	23.0	21.4	16.8	11.4	6.1	'27-'55
1929	2.8	1.5	5.8	7.3	11.6	18.1	21.1	21.5	20.8	15.1	11.2	4.4	
1930	3.3	2.5	4.3	7.5	13.6	18.5	21.8	23.0	22.5	16.9	11.0	5.8	
1931	2.9	2.5	4.2	8.0	12.7	18.0	23.5	23.3	22.3	17.6	12.5	8.1	
salinity average	20.8	21.0	18.0	16.5	18.7	21.0	22.7	23.5	23.7	23.4	21.8	20.9	'27-'55
1929	22.4	22.9	17.7	23.0	17.3	22.1	23.3	24.8	25.4	24.3	23.5	22.7	
1930	20.4	21.8	17.6	19.6	22.6	22.2	24.6	25.2	25.4	25.5	24.7	24.8	
1931	24.4	24.7	22.9	18.2	19.3	20.8	21.7	23.4	24.2	23.9	23.0	21.4	
Boston													
temperature average	1.4	0.7	2.9	6.8	11.6	15.6	18.0	18.6	17.3	13.2	8.7	3.8	'21-'50
1929	1.4	0.9	3.1	6.4	10.0	16.4	16.7	16.6	17.4	13.1	8.5	2.3	
1930	2.8	0.5	3.2	6.1	11.5	15.4	17.2	18.4	18.7	13.0	9.1	4.0	
1931	1.5	0.6	3.5	7.5	10.5	16.1	18.4	18.6	18.0	13.9	9.6	4.8	
salinity average	29.4	29.7	27.3	27.2	28.0	29.1	29.9	30.3	30.2	30.2	30.2	29.7	'21-'50
1929	30.1	30.1	28.4	26.4	28.5	30.1	30.8	31.4	31.2	31.2	31.1	30.6	
1930	29.9	30.2	30.2	29.5	30.2	30.8	31.0	30.8	31.5	31.1	30.6	30.7	
1931	30.3	29.3	25.5	27.6	28.1	25.4	28.6	29.7	30.1	31.0	31.0	30.7	
Portland													
temperature average	0.5	-0.1	1.1	4.4	8.7	12.6	15.5	15.9	14.4	9.8	7.0	2.8	'21-'49
1929	1.6	0.3	1.5	3.7	---	---	14.8	14.7	14.5	11.6	7.7	2.3	
1930	---	---	1.5	4.4	9.2	14.0	15.6	15.9	15.7	12.2	7.1	1.2	
1931	---	---	2.4	5.9	11.3	13.6	16.4	15.9	15.2	11.6	8.0	4.4	
salinity average	30.2	29.9	29.0	28.1	28.4	29.5	30.6	30.6	30.6	30.6	30.2	29.8	'21-'49
1929	28.6	29.0	31.1	30.3	---	---	31.6	31.9	32.0	30.6	29.1	29.4	
1930	29.1	---	28.9	29.7	30.1	31.1	31.6	31.6	31.2	30.6	30.8	29.1	
1931	30.1	29.8	28.6	28.8	30.6	29.8	31.4	31.1	31.1	30.4	30.1	30.7	
Eastport													
temperature average	3.4	1.8	1.8	3.0	4.8	7.0	9.3	10.9	11.3	10.6	8.7	6.0	'29-'50
1929	---	---	---	---	---	---	---	---	11.1	9.6	7.6	4.0	
1930	2.4	0.9	1.4	3.2	5.3	7.3	9.0	12.5	11.7	11.4	9.3	6.2	
1931	3.3	1.1	1.8	3.1	4.8	7.3	9.3	10.9	11.3	10.1	9.1	6.6	
salinity average	32.1	32.0	31.9	31.4	31.4	31.6	32.0	32.3	32.5	32.5	32.4	32.1	'29-'50
1929	---	---	---	---	---	---	---	---	32.4	32.4	32.3	32.4	
1930	31.9	31.8	31.1	31.1	31.1	31.4	31.9	32.4	32.3	32.3	31.9	32.0	
1931	32.0	31.9	31.5	30.7	30.4	30.7	31.1	31.5	31.8	31.9	32.1	32.4	

SALINITY

Martin (1954) related eelgrass decline to precipitation extremes, and suggests a correlation between drought and wasting disease off the USA east coast in the 1930's. The 1930 drought was the worst on record at the time, and Martin was able to correlate previous eelgrass declines (as reported by Cottam, 1934, 1935) to precipitation extremes, though, admittedly, « At best they constitute strong circumstantial evidence; hardly proof ». The mechanism by which drought may affect the livelihood of eelgrass is via salinity changes, and Martin argues that « salinity in bays and estuaries was above normal for nearly two years, and brackish water limits in rivers shifted far upstream ». Unfortunately, salinity data were not checked to verify this hypothesis, though a lively discussion followed the presentation of his paper at the 19th North American Wildlife Conference. In this discussion, Dr. H. Lewis of Nova Scotia argued that he could not conceive that such an increase of salinity had occurred in the St. Lawrence Gulf, where eelgrass diminution was nevertheless very marked. As the hinterland of the St. Lawrence River received above-average rainfall, instead of being subjected to drought as was much of the USA (USDA Weather Bureau, annual reports), Lewis' observation would seem to be correct.

In table 10 above, salinity data are presented for seven stations along the USA Atlantic coast (data kindly made available by Dr. J.R. Hubbard of the NOAA, Rockville, Maryland). Summer salinities are usually only slightly above the norm; for 1929: +0.3‰; 1930: +1.2‰; 1931: 0 ‰. The greatest departure from the norm on record is +2.5‰ for Baltimore, summer 1930, while on a monthly basis the greatest departure from the norm is 3.7‰, for Baltimore, June, 1930.

It is unlikely that these salinity increases caused physiological stress in eelgrass. The greatest salinity increases (both relative and absolute) during the 1930 drought occurred at the lower end of the salinity range (eg., Baltimore, with an average summer salinity of 6.8 ‰), where salinity remained well within the tolerance range of *Z. marina*. At the higher end of the scale, it may be argued, small changes may have greater effects, as eelgrass usually does not occur in hypersaline waters. Atlantic City, N.J., with summer maxima of 31.5-31.9‰, witnessed salinities of up to 33.1‰ in 1930. However, similar salinity increases also occurred in 1932, 1933, 1941, 1950, 1966 and 1980 (NOAA data), without any apparent adverse effects. It may also be argued that in brackish waters the increase in salinity may not cause physiological stress in the host plant, but nevertheless affect the plant, by no longer inhibiting the proliferation of *L. macrocystis*. This latter species has a salinity optimum of 22-40 ‰ (Young, 1943) or even 30-42‰ (Pokorny, 1967). In this way, however, salinity changes do not initiate the outbreak, but only modify the effects of an already manifest epidemic. This is further supported by the fact that similar summer increases in salinity were recorded at Baltimore (in the upper Chesapeake Bay, where vast eelgrass stands occur; den Hartog, 1970) in 1941, 1954, 1955, 1959, 1965, 1977, 1980, 1981, 1985 and 1986, without any apparent effects, except, perhaps in the past few years (see part 3). The sharp decline recorded by Orth and Moore (1983) began in the early 1970's, when no abnormal summer salinity increase occurred.

SUNLIGHT

Stevens (1939) investigated USA sunshine data following the discussion by Tutin (1938) and Atkins (1938) on the possibility of « decreased illumination weakening the host plant ». Drawing upon annual sunshine data, he concludes that sunshine deficiency was not the cause of eelgrass stress. Annual figures can be misleading, though (see previous chapters), and this warrants closer scrutiny. Light conditions on the USA Atlantic coast were unlike

those of the European coast during the early 1930's. Monthly averages for the years 1930-32 are given below in fig. 24) for four stations. All four almost constantly show excess sunshine throughout the year, and this is consistent with the general pattern recorded by the USDA Weather Bureau ("excess sunshine and temperature, lack of precipitation"). Lack of sunshine is thus unlikely to have caused stress in eelgrass stands, though, admittedly, day by day values would have to be analysed to be entirely certain of this conclusion.

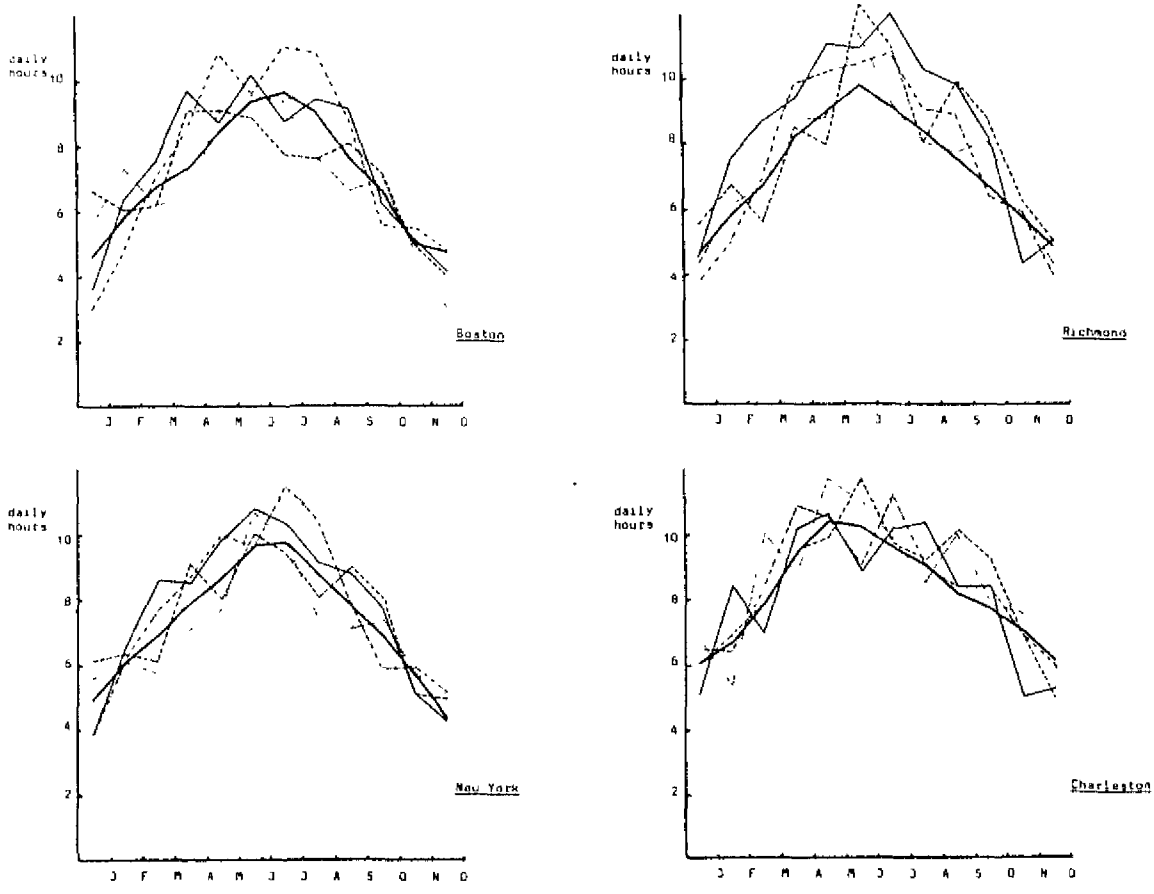

Figure 24: Sunshine USA east coast.
——— = data for the long-term average; ·—·= 1930;
·· - - - = 1931; ··· = 1932 (data USDA Weather Bureau, annual reports).

Part 3

PRESENT EELGRASS CONDITION AND WASTING DISEASE RECURRENCE

As stated in part 1, interest in wasting disease was recently re-awakened by reports of a possible recurrence of the disease, both along the Pacific and Atlantic coasts of the USA, and in Western Europe.

In the following chapters this is studied in more detail, starting with the present condition of Dutch eelgrass populations, later expanding to that of other areas in Western Europe. Reference is further made to recent USA reports on eelgrass decline, and conclusions are drawn about the possible recurrence of wasting disease.

DUTCH EELGRASS STANDS

Dutch eelgrass populations occur in two distinct geographical regions: the barrier island region of the Wadden Sea, and the estuarine area in Zeeland Province. As both eelgrass history and problems are entirely different, they are dealt with separately.

DUTCH WADDEN SEA EELGRASS

Zostera marina was once very common and widely distributed in the Dutch Wadden Sea, but its recent history, sadly enough, is one of decline and disappearance. These changes are dealt with by den Hartog and Polderman (1975), Polderman and den Hartog (1975) and Van den Hoek *et al* (1979). Eelgrass was harvested and collected for numerous uses, varying from dike construction to filling for cushions and mattresses. As it was also of economic importance, it attracted scientific interest at an early date. Martinet (1782) devoted a study to this species, dealing with anatomy, biology, and procedures and problems associated with harvesting and preparation. He was much ahead of his time, being well aware of the dynamic equilibrium of seagrass beds, and even suggesting transplantation and germination experiments for establishing new beds. This scientific interest also resulted in a repeated (albeit incomplete) mapping of Wadden Sea eelgrass beds by Oudemans *et al* (1870), Van Goor (1919), Harmsen (1936), Reigersman *et al* (1939), den Hartog and Polderman (1975) and Schellekens (1975). These maps are presented in fig. 25).

In November 1987 a number of field trips were made to sites where eelgrass beds were formerly recorded in the Dutch Wadden Sea: Balgzand (11/11/87), Schiermonnikoog (19-20/11/87) and Terschelling (21-23/11/87). At both Balgzand and Schiermonnikoog neither *Z. marina* nor *Z. noltii* were encountered on the transects covered in the field (see fig. 27b and 27c). At these former sites of seagrass occurrence a number of seaweed species abounded, esp. *Ulva spp., Chaetomorpha spp.* and *Enteromorpha sp.*. At Terschelling (fig. 27a) the situation was somewhat different; eelgrass was not encountered, but *Z. noltii* seemed to be holding itself, or even expanding in some areas. The *Z. noltii* tidal flats were frequented by several bird species, esp. brent geese (*Branta bernicla*), pied oyster-catcher (*Haematopus ostralegus*), and the algal species common at the two other locations were far less abundant, being replaced by a more varied assemblage of Phaeophyceans and Rhodophyceans. Recent reports of eelgrass at Terschelling are of several isolated annual plants (summer 1987; Bellemakers, p.c. 1987) and a small bed in Terschelling harbour (den Hartog, p.c. 1987). The former were not found, and the latter not visited, due to unfavourable tides.

On 19-21 April 1988 a field trip was made to the western part of the Dutch Wadden Sea, with the Rijkswaterstaat vessel "Bresem", and staff of DGW Haren. A number of tidal flats where *Zostera marina* formerly occurred were examined, and the occurrence and density of *Ulva spp.* and *Enteromorpha spp.* was determined along 300 m transects. These transects were located on four tidal flats, Lutjewaard, Stompe, Balgzand and Breehorn (see fig 26). No *Zostera spp.* were encountered, only the aforementioned green algae, in relatively low densities, attached to mollusc shells.

On a separate occasion, in May 1988, Mr. W. Visser of DGW Haren examined the tidal flats of the "Schorren" Nature Reserve, to the north-east of Texel island. Once again, no *Zostera spp.* were found.

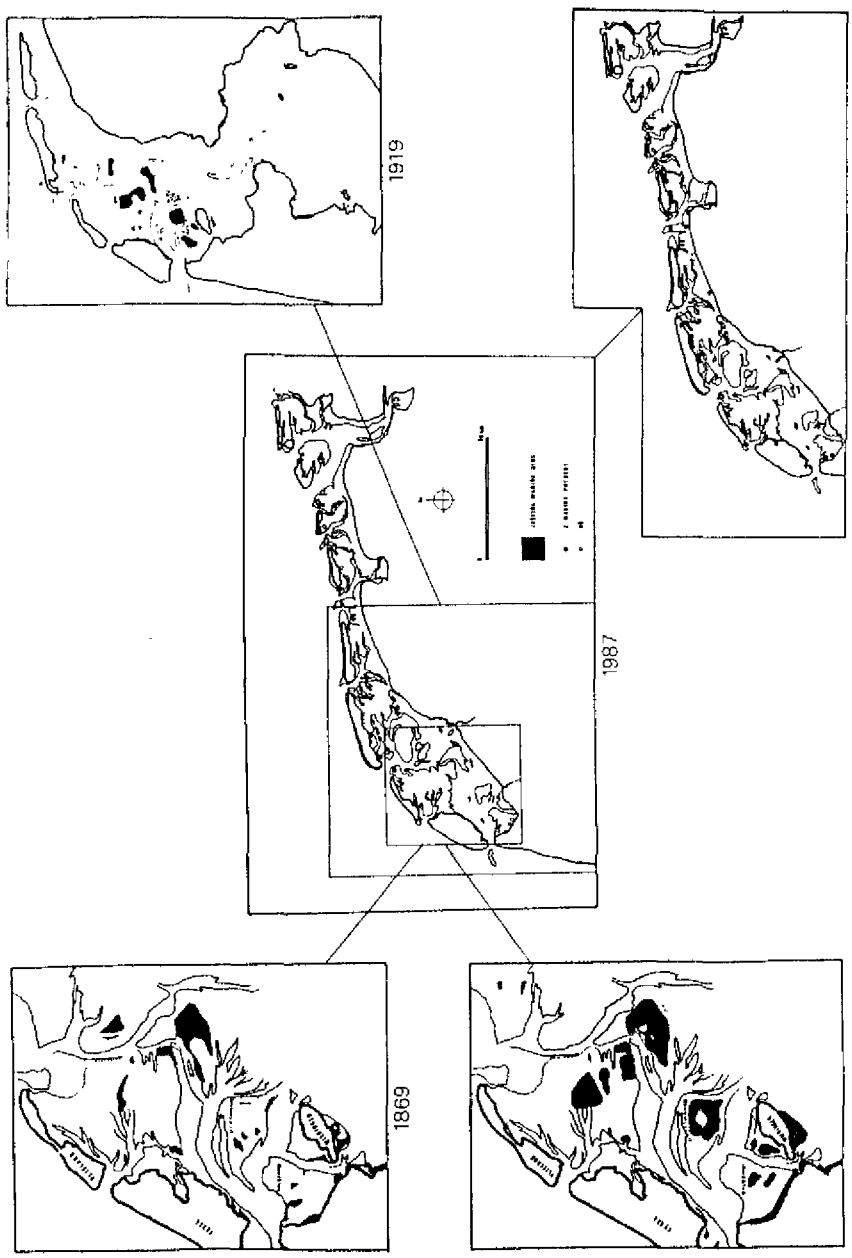

Figure 25: Zostera marina in the Dutch Wadden Sea, 1869-1987.
1869 (Oudemans, 1870), 1919 (Van Goor, 1919), 1930 (Reigersman *et al*, 1935), 1972/3 (Polderman and den Hartog, 1975), 1987 (this report).

On May 5th 1988, Mr. H. Peletier of DGW Haren found small (15-20 cm), intertidal annual specimens of *Zostera marina* on the land-reclamation flats ("landaanwinningsvakken") of the Eemshaven, near Delfzijl, at the mouth of the Ems-Dollard estuary. These appeared healthy, with no signs of wd-lesions.

Drs. M.J.S. Bellemakers of the Laboratory of Aquatic Ecology examined the tidal flat opposite the harbour of West-Terschelling (Terschelling Island) on 19 August 1988, where *Zostera marina* appears to be re-establishing itself. At present, the total area under eelgrass is estimated to be about « two or three football fields in size ». Plants collected by Drs. Bellemakers were examined at the lab in Nijmegen. They were very elongated (60-70 cm, on average), narrow-leaved (2.5 mm, on average) specimens, and virtually all bore inflorescences. Condition was poor, with wd-lesions on most plants, and many brown leaves. It is probable that these are annual plants, and this lack of vigour represents normal end-of-season die-off. A more thorough on location examination can soon verify this assumption.

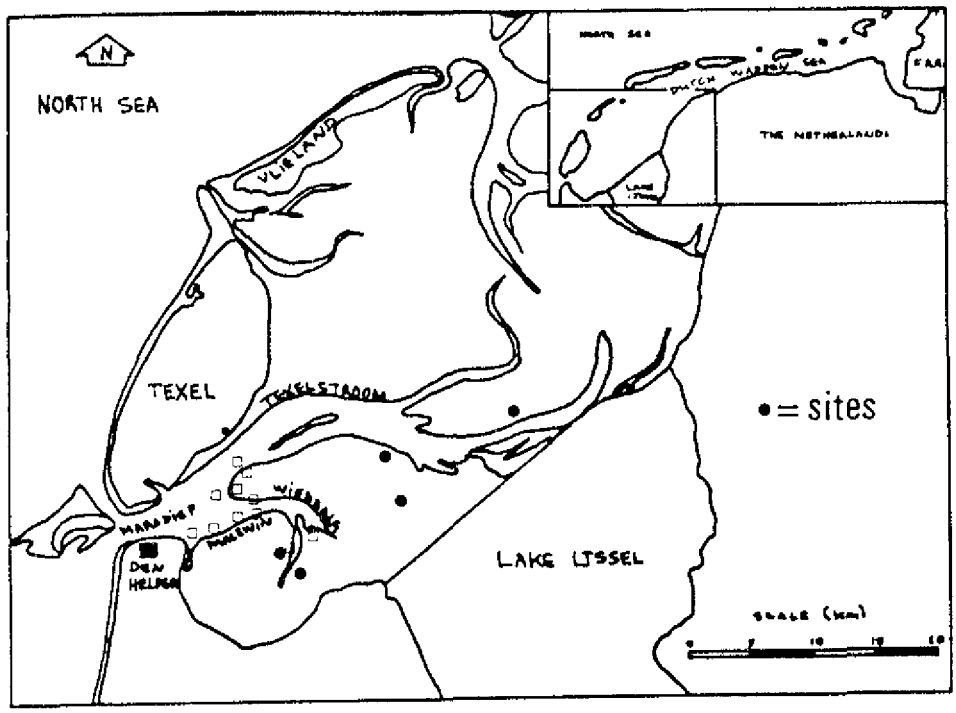

Figure 26: Dutch Wadden Sea sites.
These sites were visited on 19-21 April 1988 with Rijkswaterstaat vessel "Bresem. No eelgrass was encountered.

TERSCHELLING

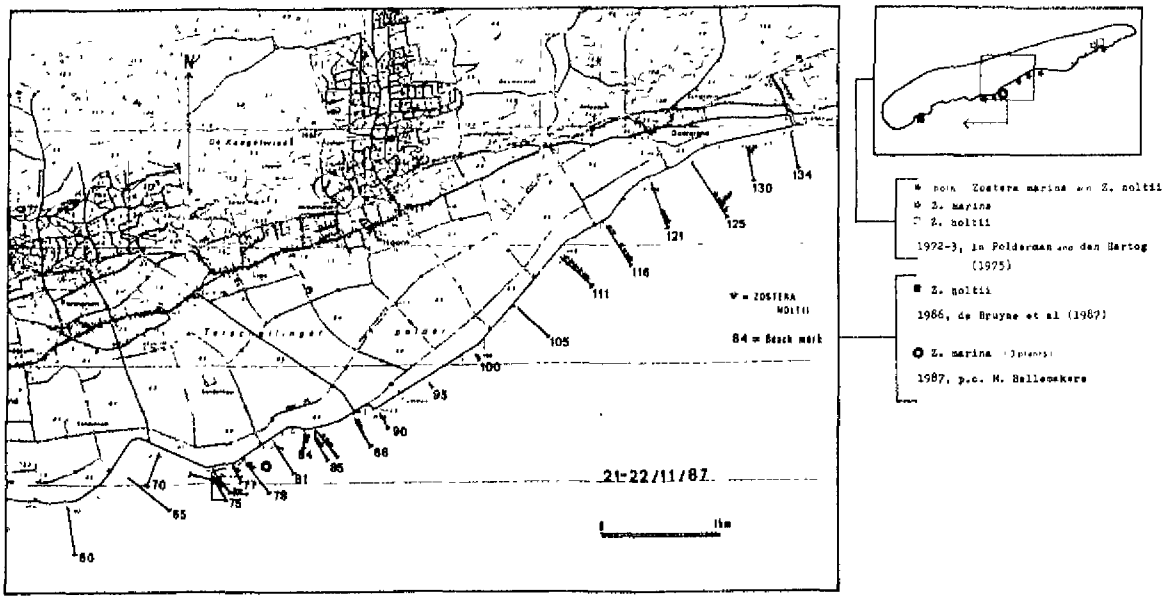

Figure 27: Seagrasses recorded in the Dutch Wadden Sea, 1987.
A. Terschelling Island.

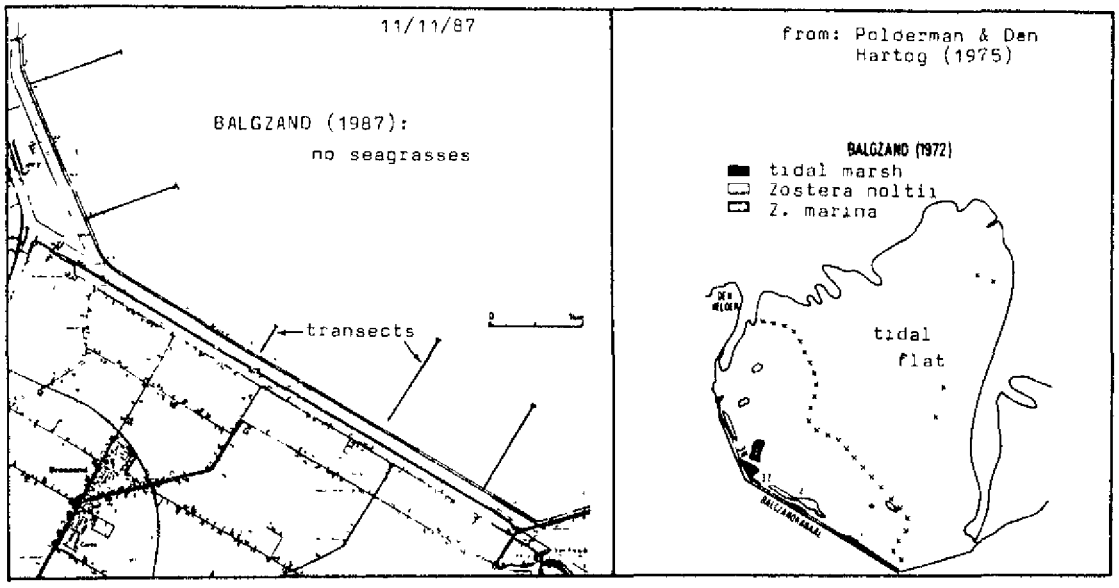

Figure 27: b: Balgzand tidal flat.

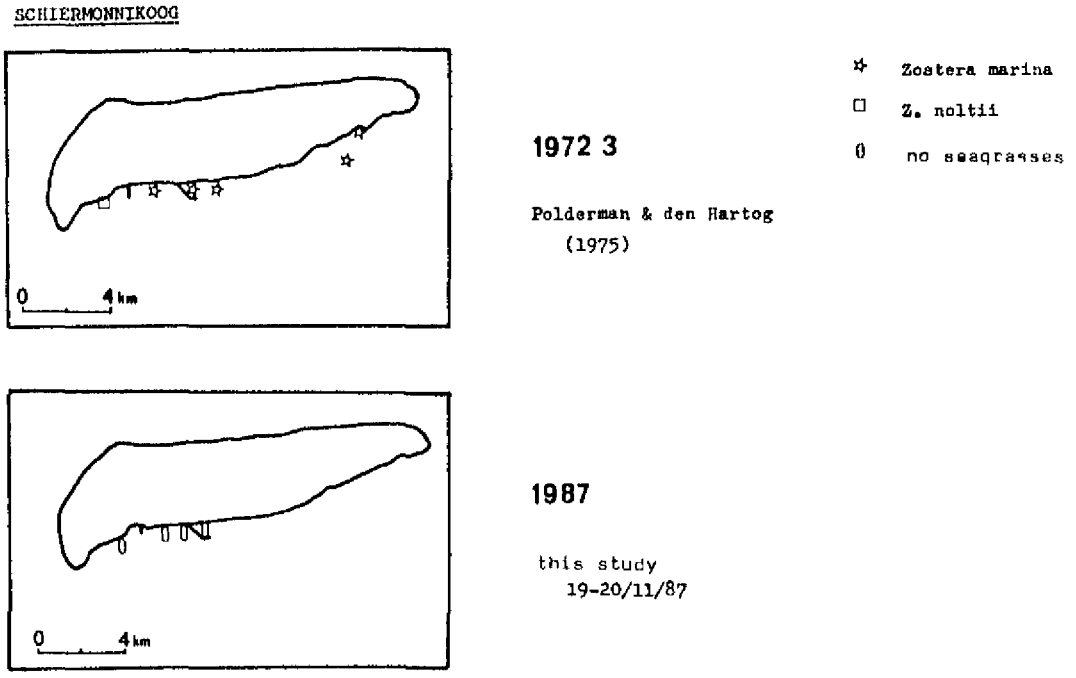

Figure 27: c: Schiermonnikoog Island.

According to den Hartog and Polderman (1975), eelgrass history in the Dutch Wadden Sea up to 1930 was one of constant changes, but these are to be regarded as normal long-term fluctuations within the large scale pattern of the dynamics of the Wadden Sea ecosystem. Wasting disease of the 1930's decimated the population, as occurred elsewhere in Western Europe. In the late 1930's, however, eelgrass beds began to recover in most parts of its former distribtion range, but not in the Dutch Wadden Sea. It was concluded that the construction of the "Afsluitdijk", the dike separating the former Zuyder Sea from the Wadden Sea, had altered conditions to the extent that the Wadden Sea was no longer very favourable for eelgrass growth (Harmsen, 1936; Reigersman *et al*, 1939). The silty substratum had eroded due to changing current patterns, tidal fluctuations had increased, and the hydrological pattern had altered (den Hartog and Polderman, 1975; den Hartog, 1987). After the 1930's a slight recovery was shown by littoral eelgrass stands, but compared to the vast, sublittoral population of former times (15,000 ha in the early 20th century; van Goor, 1919), this was never more than a shadow of what it was (den Hartog and Polderman, 1975). Recovery was not a gradual process, as is demonstrated by the following history of the Balgzand tidal flats (adapted from den Hartog and Polderman, (1975). In the early 1930's the Balgzand eelgrass population was decimated by the wasting disease epidemic. Following this disease, a second period of decline set in between 1936-47, followed by an increase up to 1965, when the greatest (post-1932) development was achieved. After that date another period of decline set in, and in the years 1971-73 alone, den Hartog and Polderman (1975) estimated a decrease of 30-60% of the remaining littoral eelgrass population. In the course of this study, late 1987, sites where *Z. marina* was reported to occur in the early 1970's were re-examined but unfortunately no specimens were encountered. It was concluded that eelgrass had all but disappeared from the western Wadden Sea.

Causes of the virtual disappearance

The causes of dynamics in the Dutch Wadden Sea eelgrass populations can be summarized as follows:

1) Changes up to 1930. Normal long-term fluctuations within the large-scale pattern of dynamics in the Wadden Sea ecosystem; some beds erode, new ones appear elsewhere (Martinet, 1782; Van Goor, 1919; den Hartog and Polderman, 1975).

2) Early 1930's: decimation of eelgrass populations due to wasting disease (Van der Werff, 1934; Harmsen, 1936; den Hartog, 1987).

3) Period between 1936-47: decline of the remaining littoral populations, perhaps due to the severity of the winters of 1940, 1941, 1942 and 1947 (den Hartog and Polderman, 1975).

4) Period between 1947-65: recovery of littoral eelgrass populations (den Hartog and Polderman, 1975).

5) Period after 1965: rapid decline of remaining littoral eelgrass population. Den Hartog and Polderman (1975) suggest that pollution (esp. toxins, such as heavy metals, PCB's and detergents) entering the Wadden Sea from the Rhine River may be the cause of this more recent decline.

Van den Hoek *et al* (1979) find it difficult to believe that erosion of substrate, changes in currents and rise in tidal fluctuations could have prevented re-establishment of *Z. marina*, and suggest that an increase in turbidity may have been the cause. It is suggested here, in this study, that increased turbidity did indeed prevent the recovery of sublittoral beds after 1932, and that it may have also played a role in the disappearance of the remaining littoral population in the late sixties/early seventies, though other factors, such as toxic pollution and competition with macro-algae (esp. *Ulva spp.* and *Chaetomorpha spp.*) may have played a role (De Jonge, p.c. 1987). Giesen et al (1989b) give approximate histories of turbidity in the Dutch Wadden Sea for both channels and tidal flats. These two geomorphological units were approached separately, as turbidity levels may differ greatly between the two, as is illustrated below (from Giesen et al, 1989b):

channels		k-value	D_{sd}
	pre-1932	0.6	2.6
	1950's	1.0	1.6
	1970's	1.2	1.4
	1978-80	1.4	1.2
tidal flats			
	pre-1932	0.9	1.8
	1950's	1.5	1.1
	1970's	2.8	0.6

The response of an eelgrass population to these changes in turbidity was simulated by using the growth simulation model developed by Verhagen and Nienhuis (1983), as described on page 24. For the tidal flat simulation, depth was allowed to vary between 0.5-3.0m, and the attenuation coefficient k from 0.9-2.8 m^{-1}; results are given in fig 28). For the channels, depth was varied from 2-7m, and k from 0.5-1.4 m^{-1}; results are given in fig. 29). In both simulations, rhizome and above-ground biomass were determined after two years of average sunshine (according to the cosine function used in Verhagen and Nienhuis' original simulation model, 1983).

The channels. Both above-ground and rhizome biomass plots show that at least from the 1950's onwards, no eelgrass growth was possible in the (deeper) channels. The peaks date from 1930 simulations; the above-ground biomass remains zero from the late 1950's onwards, and the rhizome biomass either drops to zero, or remains a constant 20 $g.m^{-2}$ (the base value of rhizome biomass in the simulation model). The latter is an artifact, as in reality rhizomes could not survive for two years without shoot growth.

The tidal flats. The shoot biomass plot shows that above-ground growth was possible in shallow waters of 0.5-1.5m depth in the 1950's, but that this had virtually disappeared by the 1970's. The rhizome biomass plot, however, shows that only at 1m depth rhizome biomass remains constant (or increases slightly) in the 1950's. At depths above or below this level rhizome biomass decreases, and populations at these depths may be described as unstable. The same artifact as noted above, under channels, is present, namely that rhizome biomass remains constant at 20 $g.m^{-2}$ if shoot growth is zero. In reality these rhizomes would perish, and their biomass drop to zero as well.

As no data on turbidity were available between 1930 and the 1950's, one can only speculate on the rate of increase in turbidity between these dates. Giesen et al (1989b) list the primary causes for turbidity increase: erosion of silt substrates during the wasting disease epidemic, activities connected with the closure of the former Zuyder Sea in 1932, Rhine River pollution (more suspended matter, and eutrophication leading to increased plankton numbers) and deposit extraction/dredging activities. The first two causes operated in the early 1930's, and their effect must have been fairly immediate, within the course of several years at the most. The second two causes are of a later date. Gieskes and Kraay (1977) report that in the first years following the Second World War the Rhine only enriched coastal waters to a minor degree, and that eutrophication set in in the early 1960's with the introduction of phosphate containing detergents. De Jonge (1986) reports that extraction and dredging has been going on in the Malzwin-Wierbalg channel system (Western Dutch Wadden Sea) since 1961. It would thus appear that we have an initial increase in turbidity in the 1930's, followed by a more gradual increase from the 1960's onwards.

Is this supported by observations in the 1930's ? Mr. P. Krijnen of Oudeschild (Texel Island), a former eelgrass fisherman, reports (in an interview with Mr. B. Koning of the "Maritiem en Jutters" Museum in Oudeschild in July, 1988) that a general increase in turbidity directly due to the closure of the "Afsluitdijk" was not noticed. He does recall, however, that as the currents were now a lot stronger, the water was naturally somewhat "thicker" (i.e. more turbid) than before. Van der Werff (1934) reports of much more sand and silt being deposited, « covering the shoots with a thick layer », though he considers it to occur as a consequence of the increased volume of water above

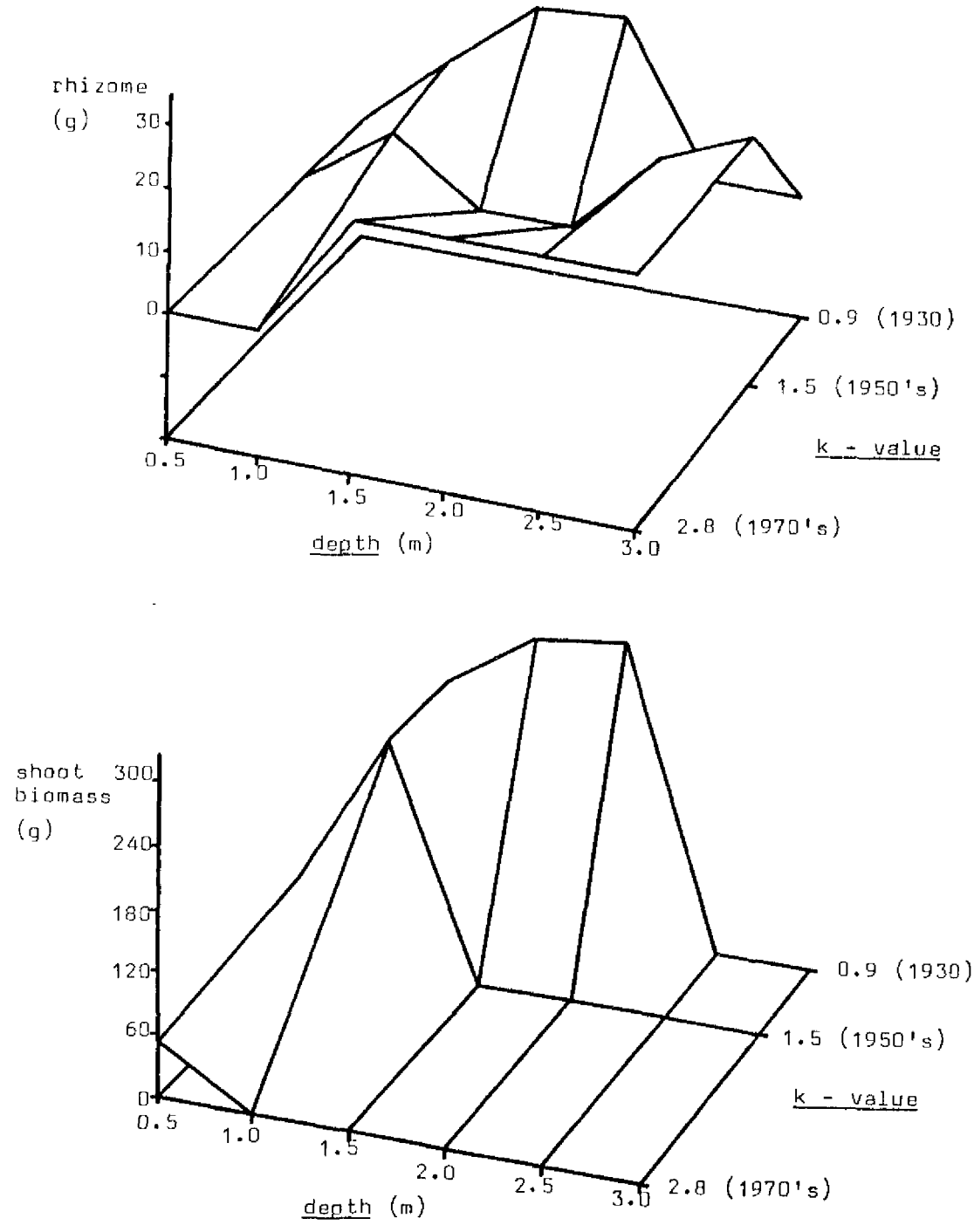

Figure 28: Simulation of tidal flat eelgrass rhizome growth.
tidal flat growth simulation of end- of- season eelgrass rhizome biomass (top) and maximum above-ground shoot biomass (bottom), in ash-free dry weight.

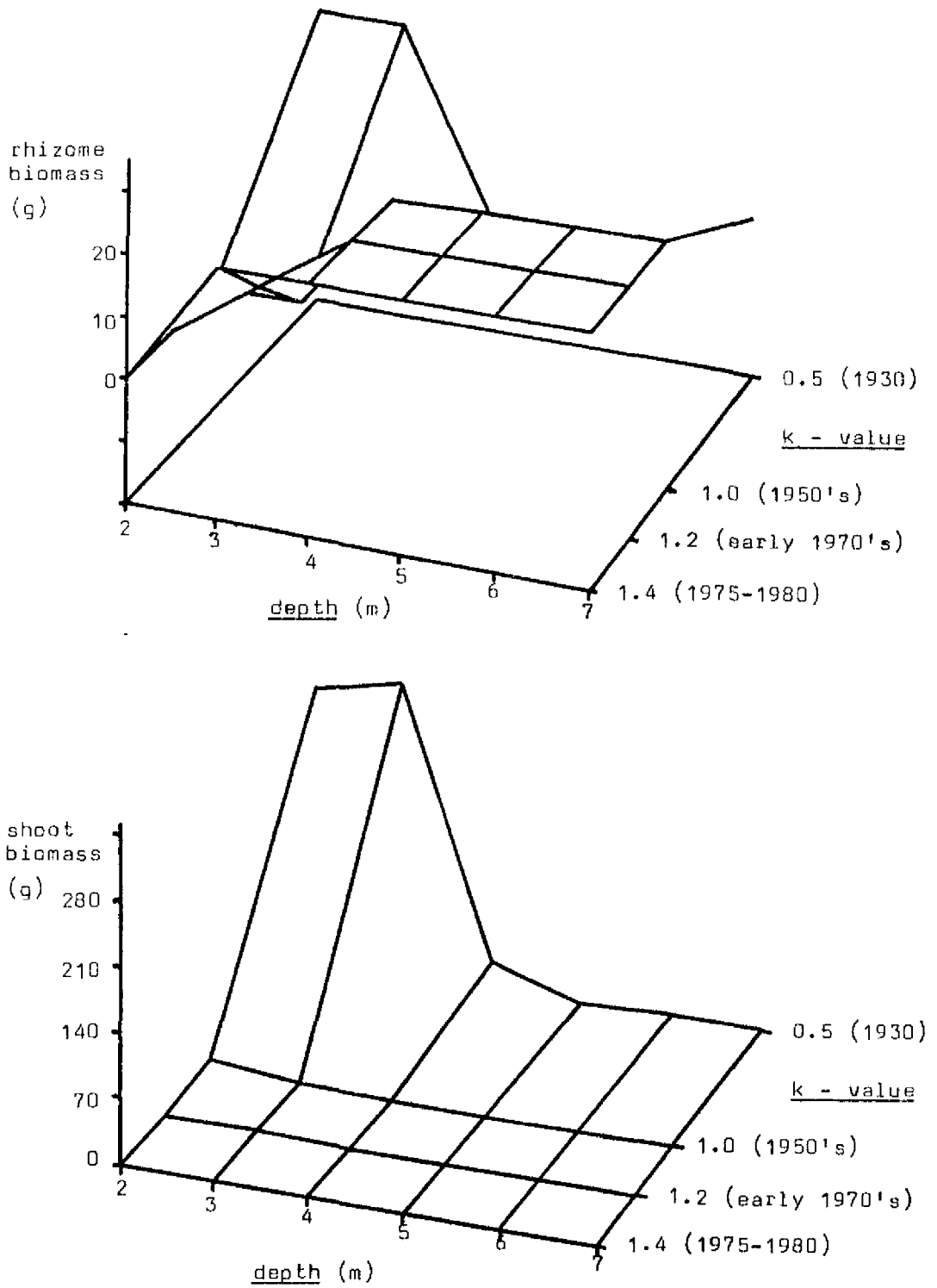

Figure 29: Simulation of channel eelgrass rhizome growth.
channel growth simulation of end-of-season eelgrass rhizome biomass (top) and maximum above-ground shoot biomass (bottom), in ash-free dry weight.

the eelgrass beds (caused by the dam closure). Many reports exist on the erosion of silt substrate after eelgrass disappearance (Wohlenberg, 1935; Defoer *et al*, 1935; Reigersman *et al*, 1939; Wilson, 1949; Rasmussen, 1977; den Hartog, 1987); see fig.30. This erosion could not have occurred without influencing turbidity.

ZEELAND PROVINCE EELGRASS

Prior to the wasting disease epidemic, *Zostera marina* stands in the estuarine area of Zeeland Province were never as vast as those of the Dutch Wadden Sea. Before the Delta Works began, after the 1953 floods, small intertidal populations occurred in several sheltered areas, such as in parts of the Grevelingen estuary (Nienhuis, 1983). After the closure of the Grevelingen estuary in 1971, a rapid colonization of the thus formed brackish lake followed (Nienhuis and De Bree, 1977). The population grew from a small, intertidal stand to a number of vast underwater "meadows", with a maximum area of about 4,000ha in 1978 (Nienhuis, 1983). These eelgrass plants are best regarded as submerged intertidal plants: they have narrow leaves (2-4mm width), an annual above-ground biomass, but are nevertheless perrenials (Nienhuis, 1983), and are found to a depth of 6-7 metres (Pellikaan, 1980). After 1978 a rapid decline set in, and by 1980 the total eelgrass area had dropped to 60% of its 1978 level (Nienhuis, 1983, 1984). The most plausible explanation for this mass decline was that an increase in organic matter deposition on the lake bottom, following a change in nutrient loading, had taken place, causing rapid de-oxygenation and toxification of the sediments, and a die-back of rhizomes and roots (Nienhuis, 1983). From 1981-86 the situation seemed to be fairly stable, only to be followed by a new die-off in 1987 (personal observations and p.c. Van Lent, 1987). This new decline became evident during the late summer months, especially in the deeper populations (at 3m depth or more), but shallower populations were also affected. By late 1987 all deeper populations seemed to have been affected. It was speculated that the cause might have been a change in the currents of the lake, as the regime of sluice opening had been altered and currents were generally slightly stronger (Van Lent, p.c. 1987).

Field observations

Fig. 31 depicts the estuarine region of Zeeland Province. The main *Zostera* locations are indicated, along with the sites that were visited and sampled in the course of this study. On the first of July, 1987, two sites were visited on the northern shores of Lake Grevelingen: Herkingen and Battenoord. At Herkingen the population west of the yacht harbour was investigated; eelgrass beds here occur at depths of about 0.5-1.2 metres. Plants appeared healthy, but air-lacunae perforations were common on most plants (see fig. 32). These are visible as dark green lines, that appear where sections of leaf air-lacunæ are filled with water. At Battenoord, the population occurred in slightly shallower water, and the general appearance was one of reasonable health. A possible indication of some form of disturbance was the presence of numerous dead crabs; dozens of dead specimens were encountered on a transect of about 400m, along with very few live ones. Another sign of possible stress was that fish were found to be sluggish, and could easily be touched by hand. *Chaetomorpha spp.* were common at both sites, found draping many eelgrass "tussocks".

On the 21st of July 1987, a second field trip was carried out, together with other participants of the International Seagrass Workshop held at the Delta Institute at Yerseke, Zeeland Province. At Lake Grevelingen, an excursion was made to a site about 1km west of Bruinisse yacht harbour, on the lake's southern shore. The eelgrass population here was located at about 1.2-1.4m depth, and many specimens possessed inflorescences. A section of several tens of square metres that had formerly borne eelgrass, now consisted of mud, with occasional tangles of brown rhizomes still partially inbedded. The remaining plants at this location often had small, brown lesions, and it was speculated

a

b

Figure 30: Erosion of silt substrate after wasting disease elimination of eelgrass bed. A): Tubes of the marine polychaete worm, *Lanice conchilega*, are exposed after 7cm of sediment has eroded. B): exposed rhizomes; both adapted from Wohlenberg, (1935).

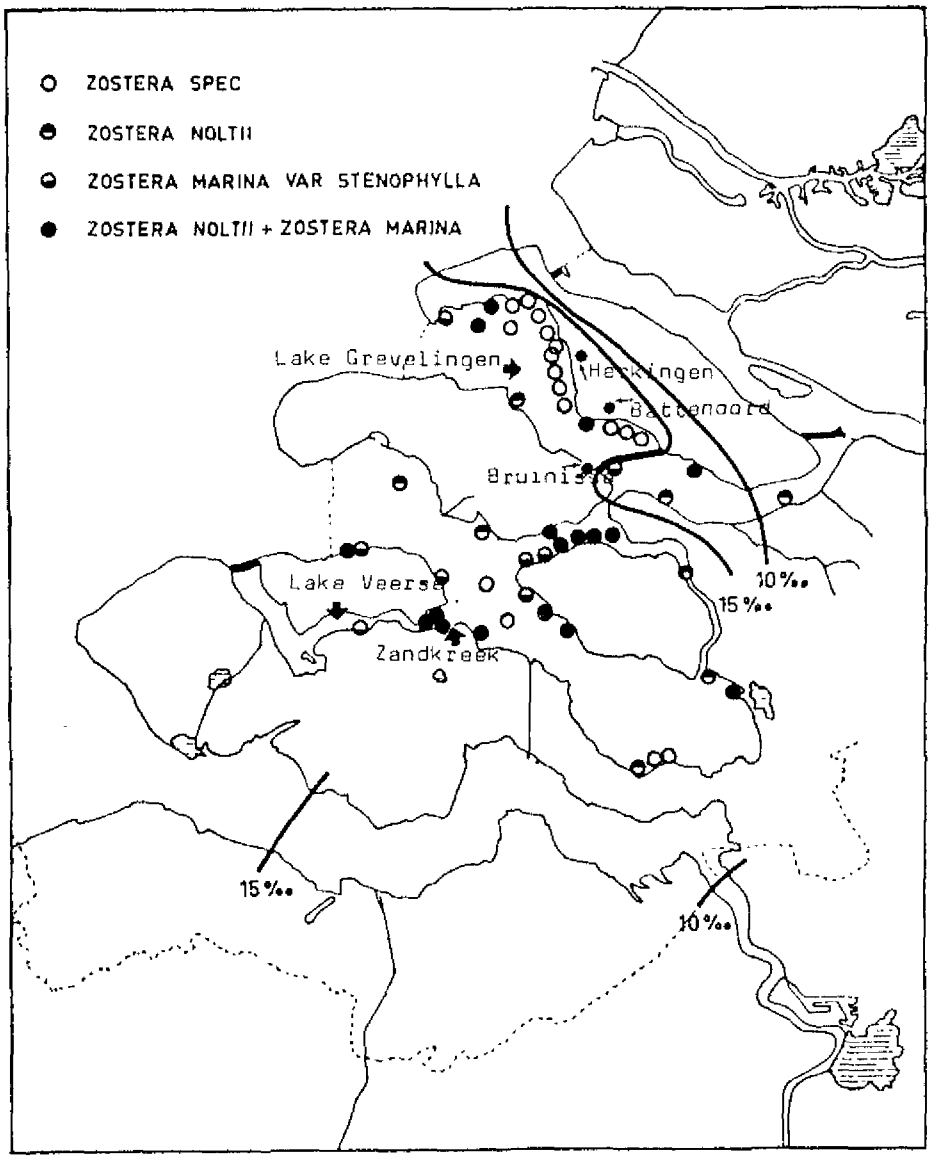

Figure 31: Eelgrass in the estuarine region of Zeeland Province (adapted from Wolff et al, 1967).

that these might indeed be symptoms of wasting disease. Opinions were unequivocal, however, and no-one seemed entirely convinced. A second site was visited on July 21st, at the Zandkreek location, where intertidal *Z. marina* occurs together with *Z. noltii*. The eelgrass plants were confined to small depressions on the tidal flat, and consisted of large, annual plants. These did not exhibit the lesions witnessed at Bruinisse, and appeared to be healthy.

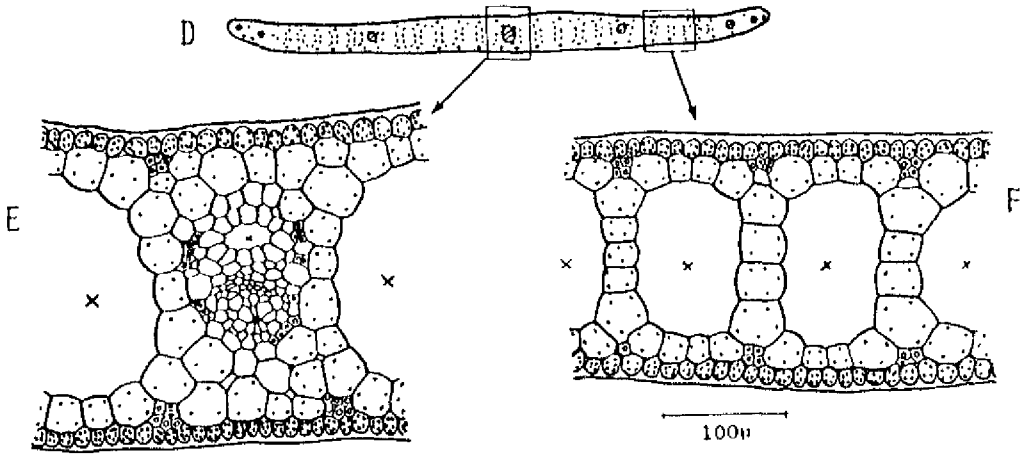

Figure 32: Cross-section of Zostera marina leaves.
(after Tomlinson, 1980); D = whole leaf, E = vein, flanked by air lacunæ, F = leaf section, with air lacunæ marked with x's.

The Herkingen site was revisited on a third field trip to Lake Grevelingen on the 27th October 1987. In contrast to the previous visit, small, dark lesions were fairly common, and a count was made of their occurrence and size, on a total of 100 plants. In this count a distinction was made between youngest leaves and the rest; the results are given below:

youngest leaves

no lesions	-	81% of the plants
lesions cover 1% of leaf	-	12% " " "
" " 2-9% " "	-	6% " " "
" " 10-30% " "	-	1% " " "
" " >30%" " "	-	0% " " "

other leaves

no lesions	-	72% of the plants
lesions cover 1% of leaf	-	11% " " "
" " 2-9% " "	-	14% " " "
" " 10-30% " "	-	2% " " "
" " >30% " "	-	1% " " "

These lesions appeared much like those described for wasting disease, but considering the season, it is also quite possible that these form part of the normal end-of-season senescence pattern. Rasmussen (1977), for instance, also reports that it is difficult to distinguish between wasting disease symptoms and end-of-season decay. A preliminary

search in records on eelgrass decomposition (Fenchel, 1970; Harrison and Mann, 1975; Thayer *et al*, 1977; Fenchel, 1977; Pellikaan, 1982; Kenworthy and Thayer, 1984), however, revealed no reports of late season eelgrass appearance. Such observations may be very useful if distinctions are to be made in the future.

Eelgrass beds at Battenoord were visited on a regular basis as a part of the 1988 study. A scale was developed for determining the degree of wd-lesion presence (= lesion density factor, or ld-factor), and this was used throughout the growing season of 1988. The ld-factor was determined as follows:

- Firstly, 40-100 plants were collected at random at a site, after which each specimen was examined separately, and the density of wd-lesions was estimated for each leaf. This resulted in lists, describing plant 1-leaf 1, plant 1-leaf 2, etc... up to plant n-leaf 6.
- The ld-factor was then calculated for each plant separately, by multiplication with the following value per density class:

Table 11: "Weight" per lesion density and leaf class.

lesion density	≤1%	2-5%	6-10%	11-30%	≥31%
weight per leaf class:					
leaf 1	0.2	0.4	0.6	0.8	1.0
leaf 2	0.1	0.2	0.4	0.6	0.8
leaf 3	---	0.1	0.2	0.4	0.6

≥leaf 4 not included in calculation.

- The average ld-factor of all plants per site = ld-factor for that site. No wd-lesions gives a ld-factor of 0 (zero), and the maximum a heavily infected plant may achieve is an ld-factor of 2.8.

It must be noted that older leaves (≥ leaf 4) were excluded from calculations. This was because some plants possessed only three leaves, while others had up to six. Furthermore, normal end-of-season die-off is thus excluded to some extent. Younger leaves were given a greater "weight" for any lesion present, as the duration of their exposure to possible infectious agents is less than that of older leaves.

Eelgrass ld-factor changes in time are given in figure 33, below, together with average and 1988 sunshine. At the time of the first observations (15 June 1988), wd-lesions were virtually absent, and the ld-factor is subsequently = 0. After this there was a steady increase, reaching a peak early in August, that was followed by a decrease towards early September. It may be argued that this represents the normal end-of-season die-off, and regrowth of vegetative shoots. This agrees with observations in the field, as shoots with inflorescences decayed late in July, and seemed to be replaced by vegetative shoots. However, if vegetative shoots alone are regarded, the same pattern emerges. One must bear in mind that leaves with wd-lesions do not recover; any improvement in the ld-factor is always a result of an appearance of new, healthy leaves. The sunshine data suggest that a lowered insolation level may be involved, as the wd-lesions appeared after a six-week dull spell, with 20% below average sunshine (in terms of $J/(cm^2.day)$). However, shading experiments did not show a very significant increase in ld-factor, indicating that the relationship is more complicated than it seems at a first glance. Salinity and temperature fluctuations do no seem to be involved

in the inducement of wdl-lesions. Salinity remained constant, as little or no freshwater was allowed to enter the lake during the growing season, and temperatures are unlikely to have increased dramatically during the dull June and July months. However, one cannot outrule other possible sources of influence, such as pollution entering the area from Battenoord yachting harbour. This remains unlikely, though, as this harbour is very small, and similar increases were noted simultaneously in Herkingen (personal communication, F. van Lent).

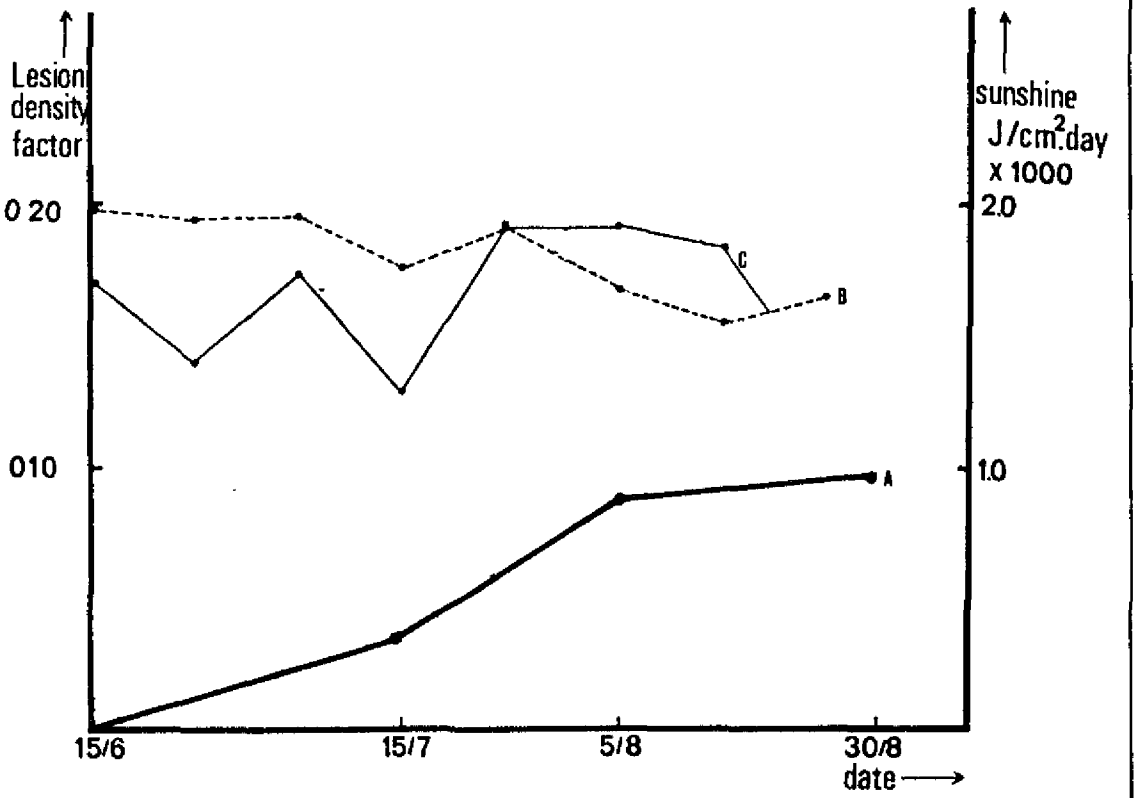

Figure 33: Lesion density factor of Lake Grevelingen eelgrass.
Site = Battenoord. Line a)= ld-factor; b)= average sunshine Vlissingen (average per "decade"; each month is divided into three "decades" by the Royal Dutch Meteorological Institute, KNMI); c)= 1988 sunshine Vlissingen.

Aquarium experiments

Ld-factor and condition.

The following aquarium experiment, carried out at the Laboratory of Aquatic Ecology, was designed to test the effects of temperature and light on eelgrass condition, especially regarding the presence of wasting disease-like lesions. Eelgrass plants (5cm sods) were collected at Lake Grevelingen on June 15th, 1988, and placed in twelve aquaria. Artificial marine-mix salt was used (Wimex, of Wiegandt GmbH, Krefeld, West Germany), at a salinity of 20.0‰, that was pumped through the aquaria at a turnover rate of once every second day. Six aquaria were kept at 18°C, and six at 21.0° Of each temperature class, two aquaria were illuminated with 178 $\mu E/(m^2.s)$, two with 75 $\mu E/(m^2.s)$ and two with 28 $\mu E/(m^2.s)$ (measured with a newly calibrated Licor-b light meter). According to Dennison and Alberte (1982, 1985), Dennison (1987) and Verhagen and Nienhuis (1983), these values are equivalent to: > saturation point, ± saturation point and << saturation point, for eelgrass photosynthesis. Daylight simulation lamps were used (Philips, 400 W, type HPI-t150), and reduction was achieved by shading. Air was bubbled continuously through all twelve aquaria. At regular intervals, a number of parameters, such as lesion density factor, browning of leaves, leaf transparency and water clarity were noted.

Results of the ld-factor measurements are given below in fig. 37, and general observations are given in table 12. Table 12 shows that wasting disease-like symptoms developed rapidly in eelgrass of five out of six aquaria kept at 21°C, while plants kept at 18°C did not develop these symptoms. The *'s of aquaria 1,2 and 4 on the 5th of August represent a blackening of leaf tips of old leaves. These wd-lesions did not develop on the youngest three leaves in plants of these aquariums. Isolation experiments revealed an association of *Labyrinthula spp.* with the wd-lesions, in all trials undertaken. Lesion density factors of the duplicate aquariums are given in fig. 12). The difference between the 18°C and the 21°C series is striking. Wd-lesions are far more pronounced on the latter. The effect of shading is less pronounced, but nevertheless still obvious. It would appear, from this experiment, that a combination of warm water and reduced light conditions leads to (a quicker) appearance of wd-lesions in *Zostera marina.*

Photographs are given in for aquaria 3 and 9 (fig. 34 and 35). Both were kept under dark conditions ($28\mu E/(m^2.s)$), but aquarium 3 was maintained at 18°C, and 9 at 21°C. No. 3 shows the development of transparent patches, but no wd-lesions. No. 9 leaves are yellowed, with many wd-lesions.

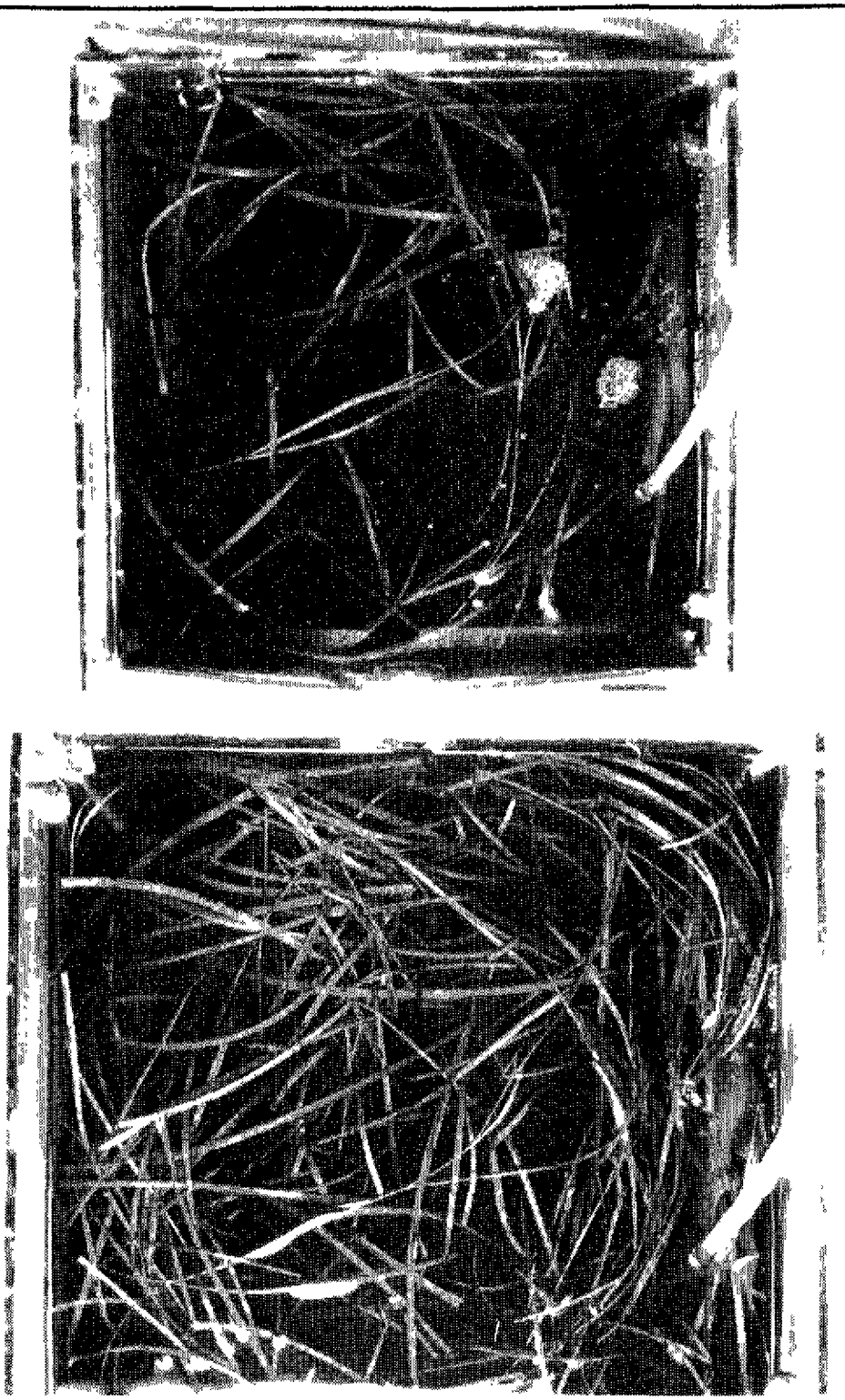

Figure 34: Photographs of aquarium 3 eelgrass.
Top = 5th July, 1988; bottom = 19th July, 1988.

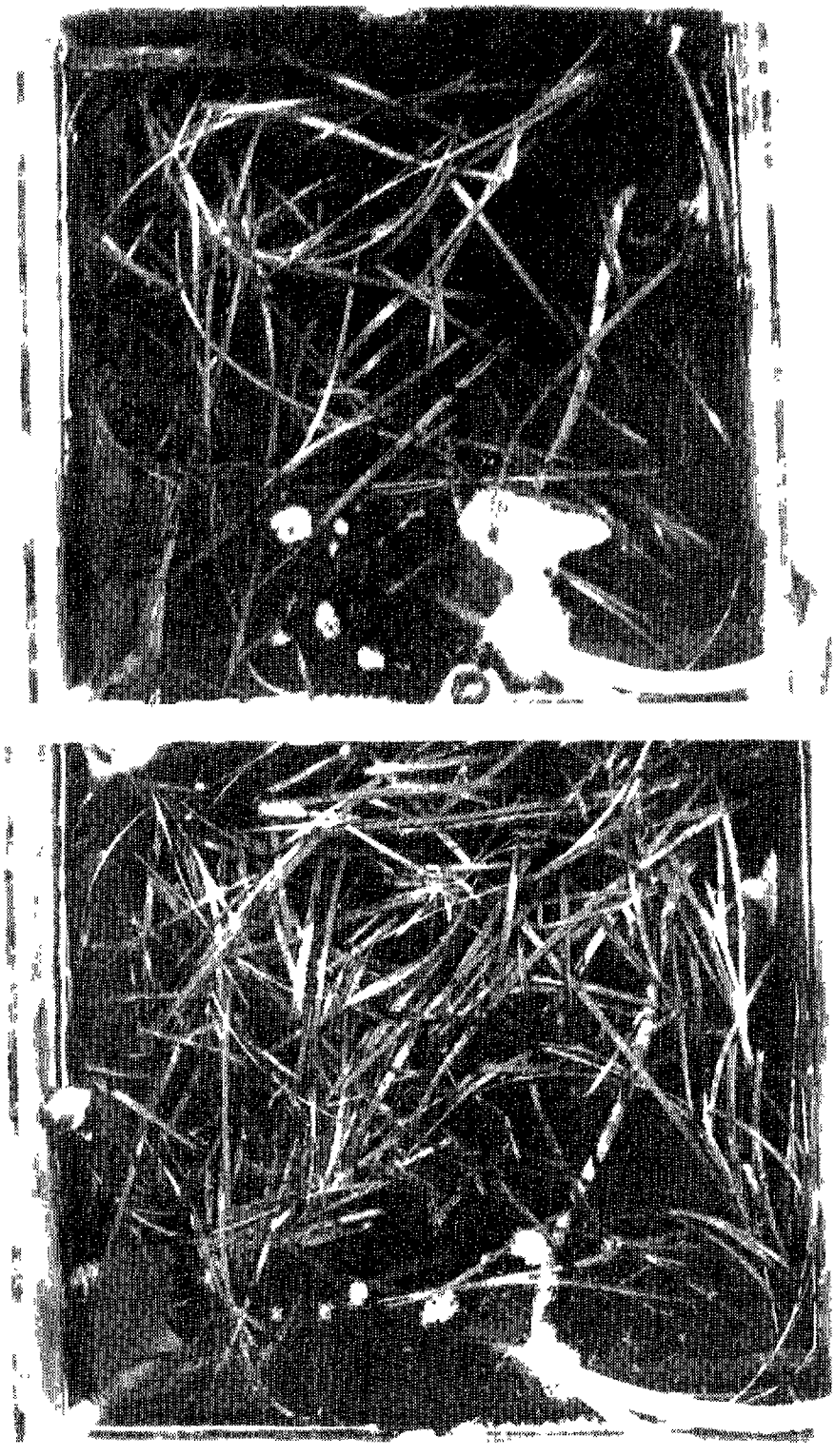

Figure 35: Photographs of aquarium 9 eelgrass.
Top = 5th July, 1988; bottom = 19th July, 1988.

Table 12: Eelgrass condition under different temperature/light regimes.

no.	C	E	T	B	W	C	E	T	B	W	C	E	T	B	W	C	E	T	B	W
1	*	*	*	+	-	*	+	*	+	*	+	+	+	+	-	+	+	+	+	-
4	-	+	*	+	-	*	#	+	#	*	-	+	#	+	-	*	+	+	+	-
2	+	-	-	*	-	*	-	*	*	*	+	*	*	*	-	+	*	+	+	-
5	+	-	*	*	-	+	-	*	#	-	*	*	+	+	-	*	*	+	+	-
3	+	-	+	*	-	+	-	#	*	-	+	*	+	+	-	+	-	+	+	-
6	*	-	*	*	-	+	+	+	+	-	+	+	+	+	-	+	+	+	+	-
7	-	+	*	#	-	*	*	+	#	-	-	#	+	+	-	-	+	+	#	-
10	*	+	+	*	-	*	*	+	+	*	*	*	+	#	+	+	*	+	#	#
8	+	*	*	*	-	*	+	+	+	*	+	*	+	+	*	*	*	+	+	+
11	*	*	+	*	-	+	-	+	*	*	+	*	+	+	+	+	*	+	+	#
9	+	-	*	*	-	+	-	+	*	*	+	+	+	+	+	+	+	+	+	+
12	+	-	*	*	-	+	-	+	*	*	+	+	+	+	+	+	+	+	+	+
	29 June					5 August					18 August					29 August				

aquariums 1 + 4 = 18°C, light
aquariums 2 + 5 = 18°C, shaded
aquariums 3 + 6 = 18°C, heavily shaded
aquariums 7 + 10 = 21°C, light
aquariums 8 + 11 = 21°C, shaded
aquariums 9 + 12 = 21°C, heavily shaded

C = clarity; + = clear, * = relatively clear, - = turbid
E = epiphytes; # = very many, + = many, * = relatively few, - = few
T = transparent patches on leaves; # = very many, + = many,
* = relatively few, - = absent
B = browning of leaves; # = very much, + = much,
* = relatively little, - = absent
W = wd-lesions; # = very many, + = many, * = present, - = absent

Figure 36: Aquarium experiment setup.
15 l. aquaria were cooled in a large tank, in which 18°C water flowed. Screens reduced ambient light levels (Philips daylight lamps) to the required levels.

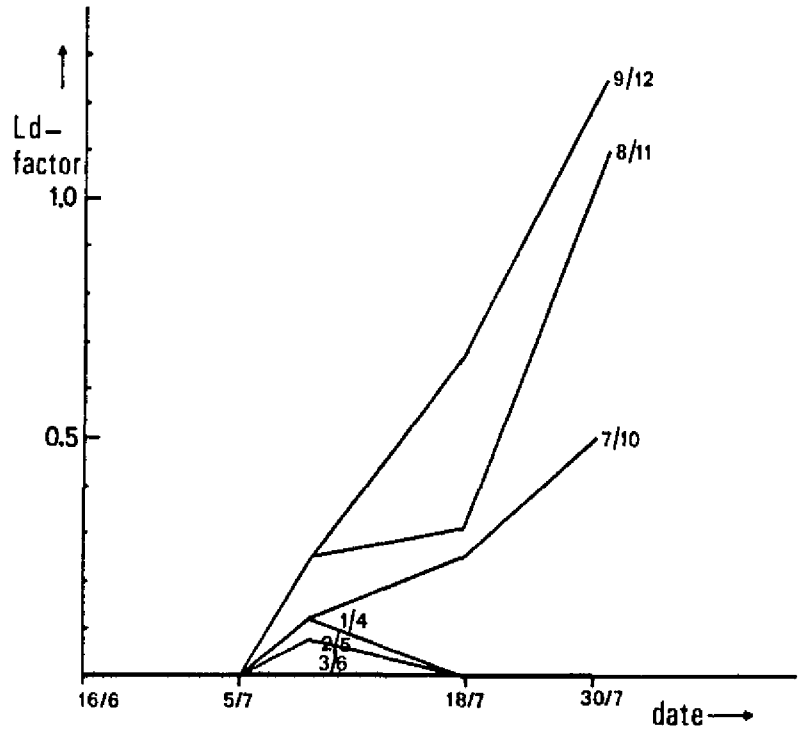

Figure 37: Eelgrass ld-factor and aquarium light/temperature variations.
1-6 = 18°C; 7-12 = 21°C.
1,4,7,10 = light; 2,5,8,11 = shaded; 3,6,9,12 = heavily shaded.

Quantification of lesions and decay.

This experiment was similar to the previous one, outlined above, with the exception that wasting disease-like lesions and leaf browning were quantified in a different manner. The same experimental setup was maintained, with two temperature classes (17.0°C and 19.5°C) and three illumination classes (178, 75 and 28 $\mu E/(m^2.s)$), in duplicate. At regular intervals, shoot samples were collected at random in each aquarium (with the exception that both generative and vegetative shoots were collected each time). These shoots were rinsed (to remove salt), and dissected into morphological parts: stems (and sheaths), leaves and inflorescences (roots and rhizomes were not collected). The parts were thereupon divided into three classes: green, brown (decaying) and wasting disease-like lesions, oven-dried for 24 hours at 80°C, and weighed.

The results for the leaves are given below in fig. 38 to 41. No regular pattern seems to emerge, unfortunately. The vegetative shoots (fig. 38) show no marked difference between 17.0°C and 19.5°C classes, or between the different light regime classes. In all, there is a steady increase in the incidence of brown leaves as time goes on, while wd-lesions remain at a low level. The generative shoots (fig. 39) show a different pattern, but once again there is no difference here between temperature and light regime classes. Browning is far more advanced than in the vegetative shoots by the end of the experiment (i.e., after three weeks). This is hardly surprising, as senescence normally sets in quite rapidly under natural conditions (personal observations in Lake Grevelingen). In fig. 40 and 41, temperature classes, light regime classes and vegetative/generative classes are viewed separately. There is no significant difference between the results of the 17.0°C class and the 19.5°C class, both in browning and wd-lesion incidence. The light regime classes (fig. 41), surprisingly enough, show a lower incidence of browning of leaves in the *most shaded* class, while one would expect deterioration to set in sooner under these conditions. The only clear difference that meets expectations is that between vegetative and generative shoots (fig. 40). Deterioration appears to be far more rapid in the latter.

The results do not comply with the expectations. The reason for this is probably twofold:

- The experiments began with plants of differing condition. In 7A they were healthy, unaffected, and consisted mainly of vegetative shoots. In the present experiment they consisted of both generative and vegetative shoots, that were already partially affected with wasting disease-like lesions (collected in this state in the field).

- Normal end-of-season senescence may be programmed in a plant, regardless of ambient light conditions. This pattern may mask the effects of light and temperature that would have effect earlier in the season.

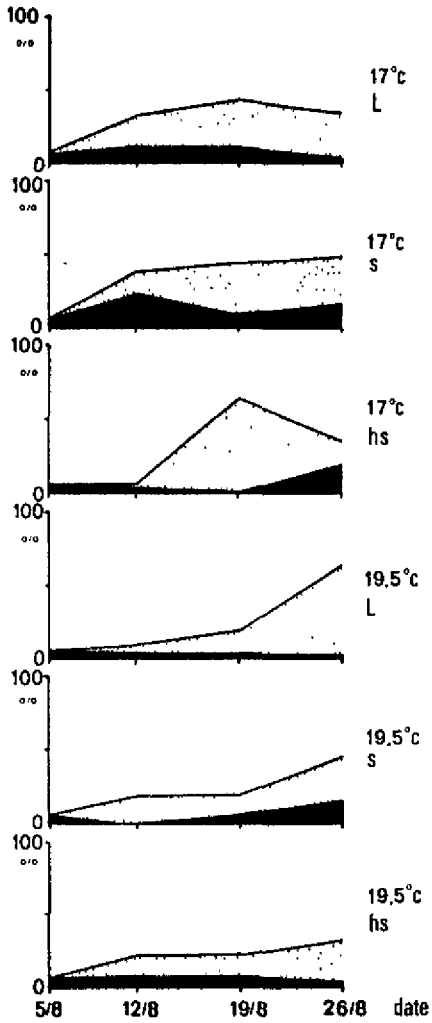

Figure 38: Decay and lesions of vegetative shoots.
Decay (shaded) and lesions (black) are given for vegetative shoots of the different temperature and light classes. L = light (above saturation point), S = shaded (approximately saturation point), HS = heavily shaded (between saturation and compensation point).

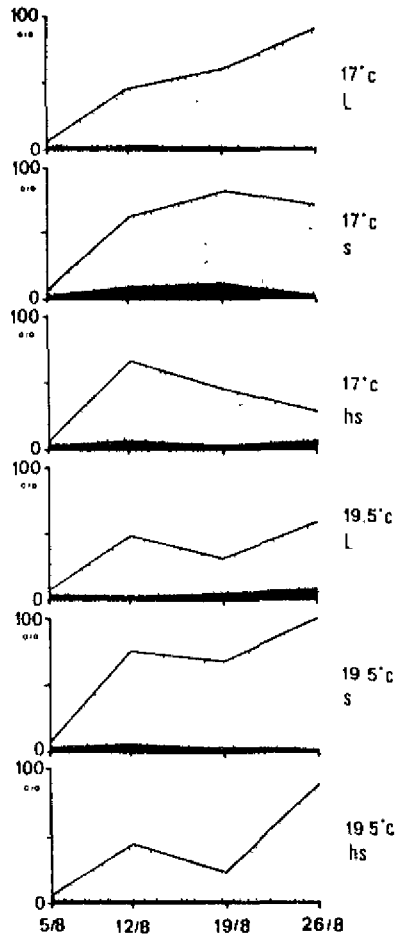

Figure 39: Decay and lesions of generative shoots.
Decay (shaded) and lesions (black) are given for generative shoots of the different temperature and light classes. L = light (above saturation point), S = shaded (approximately saturation point), HS = heavily shaded (between saturation and compensation point).

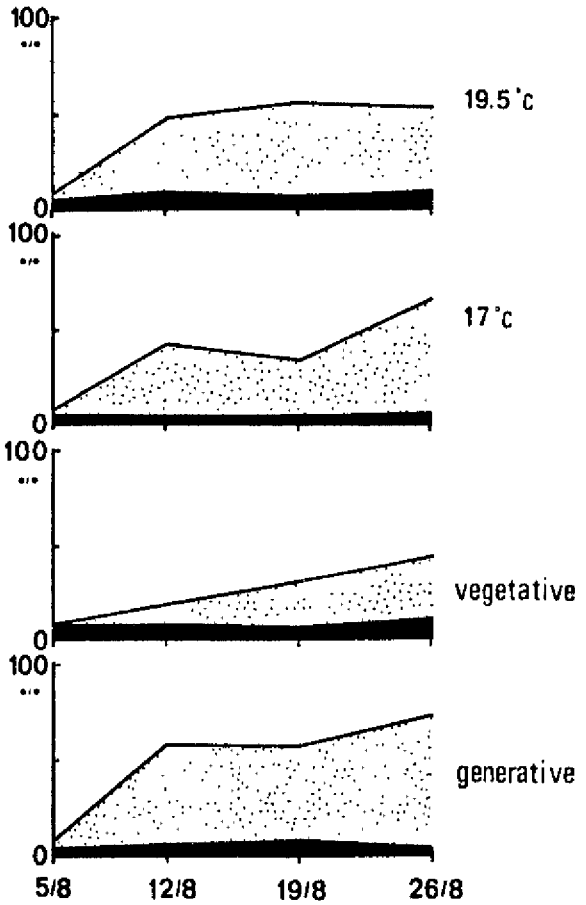

Figure 40: Decay and lesions in temperature, vegetative and generative classes.
In all, categories have been lumped together on the basis of one parameter. Decay is shaded, lesions are indicated in black.

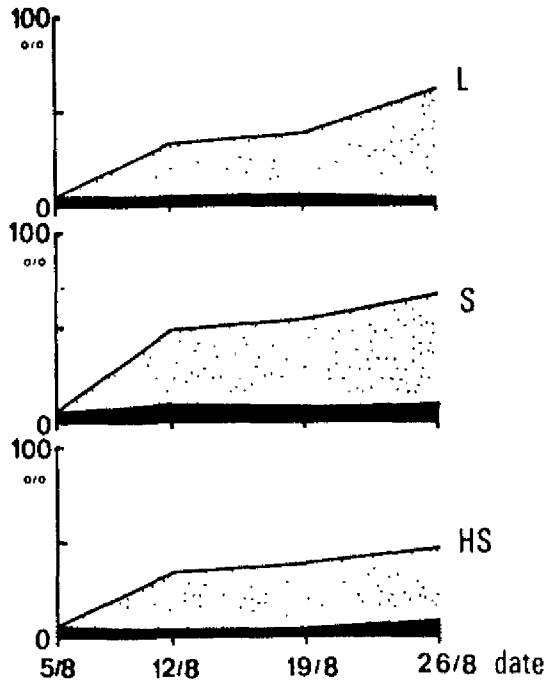

Figure 41: Decay and lesions in light regime classes.
Decay is shaded, lesions are given in black. Categories have been lumped together on the basis of light regime alone. L = light (= above saturation point for photosynthesis; S = shaded (= approximately saturation point) and HS = heavily shaded (between compensation and saturation point).

Microscopic preparations

Samples taken during the field trips to Zeeland were supplemented by further samples, kindly collected by Frances Van Lent and Patty Brouwer, both of the lab of Aquatic Ecology, working at the Delta Institute at Yerseke. Plants were kept in 15 litre aquaria (12 in all), filled with artificial seawater (15, 20 and 30‰ salinity), aerated, and maintained at 18°C (conditions similar to those described by De Cock, 1977). Tanks were replenished with fresh seawater every second week. Fresh samples of these specimens were examined under a normal light microscope. Other material was fixed in 3% formalin, inbedded in parafin and microtome-cut to 10 μm sections, that were mounted and stained with acridine orange and aniline blue. Acridine orange more typically stains bacteria, while aniline blue strongly stains fungi (Newell, 1981).

Plants of all locations (Herkingen, Battenoord and Bruinisse, in Lake Grevelingen; Lake Veere; Zandkreek) had developed dark lesions by the end of the growing season (September-October). Examination of these lesions, however, did not reveal the distinct strands of spindle-shaped *Labyrinthula* observed by Renn (1934, 1936), Van der Werff (1938) and Armiger (1964), among others. In fresh material, amorphous brown strands could be observed (fig 42), and it was later concluded from isolation experiments that these were probably *Labyrinthulæ*. In fresh material, jerked movements could occasionally be observed in these strands, at magnifications of 400-1000x. Stained material also failed to reveal typical spindle-shaped *Labyrinthulæ* , but amorphous brown strands, ascomycete fungi, bacteria and diatoms could readily be identified. A number of conclusions could be drawn from observations of microscopic preparations:

- Examination of infected material from Bruinisse, where eelgrass decline was well under way when this sample was collected, only revealed a bacterial infection, in combination with a minor occurrence of ascomycete fungi. It is probable that infection had already progressed to a degree that these organisms had overshadowed *Labyrinthulæ*.

- From material with differing degrees of lesion development it could be concluded that a succession of infecting organisms occurred in green, still attached leaves (including the youngest leaves): the *Labyrinthulæ* (amorphous, brown strands) were present first, followed by bacteria, joined at a later stage of decay by ascomycete fungi. This compares with the detritus "community" described by Fenchel (1970) for *Thalassia testudinum*: decomposition by bacteria and fungi, the former of which are consumed by flagellates and ciliates, while fungi may be eaten by nematodes.

- Low magnification (20-40x) shows that the lesions that developed on these leaves are very similar to wasting disease symptoms (fig. 43 and 44). Lesion patterns indicate that the primary infection mainly spreads longitudinally via air-lacunæ. Sieve plates partitioning these lacunæ serve as temporary barriers, and accumulations of micro-organisms may be observed on these plates (fig. 45). Lesions are usually asociated with leakages of the air-lacunæ, that are subsequently flooded (fig. 46), reducing the buoyancy of the leaves. This contrasts with observations by Spierenburg (1934), who reports of finding affected partitions between air-lacunæ (fig. 47), resulting in deflation.

- Lateral spread of infection occurs both via intercellular cavities and directly from infected cell to adjacent cells. Epidermis, mesophyll and vein cells are infected alike, though the typical discoloration (dark brown to black to the naked eye, brown when magnified) is strongest in the epidermis sites of infection.

- No difference could be observed in disease symptoms and rate of spreading between plants kept in 15, 20 or 30‰ tanks. It would appear that the salinity inhibition of *Labyrinthula* infection is not a gradual process, but begins rather abruptly at salinities below 15‰, as found by Short *et al* (1987).

- Photographic plates made of both fresh and fixed-and-stained eelgrass tissue (lesioned material) were examined by Dr. A. van der Werff in June, 1988. In his opinion, the symptoms and parasitic organisms were identical with what he had observed in the 1930's.

A number of formalin (3%) fixed leaf samples were freeze-dried, gold sprayed and examined under a scanning electron microscope. It was hoped that intercellularly occurring micro-organisms might be thus observed but, unfortunately, no distinction could be made between possible artifacts and infectious organisms. Transmission electron-microscopy could offer better results, as it is often applied to identify *Labyrinthula* ultrastructure (Porter, 1969, 1987). This technique, however, is far more time consuming, and could not be applied in the course of this study.

Isolation experiments

Trials were carried out with various isolation media and surface sterilization methods. The best general isolation medium for *Labyrinthula* was found to be a semi-solid agar (1.2% agar), on sterilized seawater basis (artificial seawater, 30‰), 1% blood or serum (Porter, 1987), to which 300 mg/l streptomycin and 200 mg/l penicillin were added (Johnson and Sparrow, 1961). The latter were added to check bacterial growth; blood has the advantage of being readily available, while serum-based medium is far more transparent. The best surface sterilization of leaves was attained by a 2 minute immersion in 0.5% Sodium hypochlorite (diluted bleach), followed by a 2 minute rinse in distilled water, and a subsequent 2 minutes in sterile seawater (Newell and Sell, 1982). Ethanol (96%) was not effective, as short rinses did not control epiflora growth, and longer immersions (>1 minute) inhibited *Labyrinthula* growth. Three 1cm pieces of leaf tissue were placed in petri dishes (9cm diameter) on 20-25ml of medium, and stored in the dark at room temperature (20-25°C). Additions of live yeast (as bait) to the medium surface was also unsuccessful, as the growing yeast masked *Labyrinthula* growth.

Early trials (August 1987) were unsuccessful as far as isolation of *Labyrinthula* was concerned. This was probably because plants were kept in aquaria for several weeks before isolation trials, and infections were probably too advanced. Later trials demonstrated the presence of *Labyrinthula* in all fresh (albeit with lesions) *Z. marina* plants from all sites in Zeeland Province. See fig. 48 and 49. Later the same *Labyrinthula sp.* was isolated from *Z. noltii* plants collected at Terschelling. The frequency of *Labyrinthula* occurrence in *Z. marina* (with small lesions) was: Herkingen, 28 out of 36 trials; Battenoord, 14 out of 19; Lake Veere, 12 out of 20; Zandkreek, 5 out of 12.

Several distinct growth forms of *Labyrinthula* were observed in culture dishes (fig. 50a - f). These may represent *Labyrinthula's* response to changes in micro-environment, as drying of the petri-dish medium may cause the transition from growth on the surface (2 dimensional, lobe- or net-shaped) to growth in the agar itself (branched or feather-like). Fig. 50e - f show the transition from 2 dimensional to branched growth. At higher magnifications (400-1000x) no distinction can be made between individual spindle cells of the thus formed branches, though this is easily done when these cells form threads of a net-like 2-dimensional growth form. This may explain why only amorphous, brown strands and not spindle cells were observed in cells exhibiting wasting disease-like lesions. This is further supported by the fact that no micro-organisms other than *Labyrinthulæ* were readily isolated from leaves with small lesions (and amorphous, brown strands, when observed at higher magnifications).

Vishniac's medium (Vishniac, 1955), consisting of mineral salts, thiamin and a carbon source, was also tried in the search for a more defined medium. Glycerol (1%) was used as a carbon source, and positive results were achieved with most isolates, though growth rates were slow. The size of the isolated *Labyrinthula spp.* was approximately 3-4 μm width, and 8-10 μm length. This compares with sizes given for numerous *Labyrinthula spp.* by Pokorny (1967, table 13).

Labyrinthula spp. were kindly isolated and identified by Dr. David Porter (University of Georgia, USA). Isolates from Herkingen, Lake Veere and Zandkreek were found to be identical, and similar to the pathogenic *Labyrinthula* strain isolated from locations in the USA, where this organism seems to be responsible for recent eelgrass decline (Short *et al*, 1986, 1987, 1988). The genus *Labyrinthula* is furthermore undergoing taxonomic revision (by Porter), and the former classification (*L. macrocystis, L. vitellina, L. roscoffensis,...*) is invalidated by many inconsistencies. Porter (p.c., 1988) reports that he has over 100 isolates at present, but believes that there are less than 10 species. At least five different "species" of *Labyrinthula* have been identified in *Zostera marina* in the past (from Johnson and Sparrow, 1961): *L. minuta* (USA), *L. vitellina v. pacifica* (USA), *L. vitellina v. vitellina* (Japan), *L. macrocystis* (USA, the Netherlands, UK), *L. sp. type LX* Watson (USA). Porter's preliminary conclusion, however, is that the typical wasting disease-like lesions in *Zostera marina* are generally associated with one pathogenic strain of *Labyrinthula*, which he has named provisionally *Labyrinthula* P.

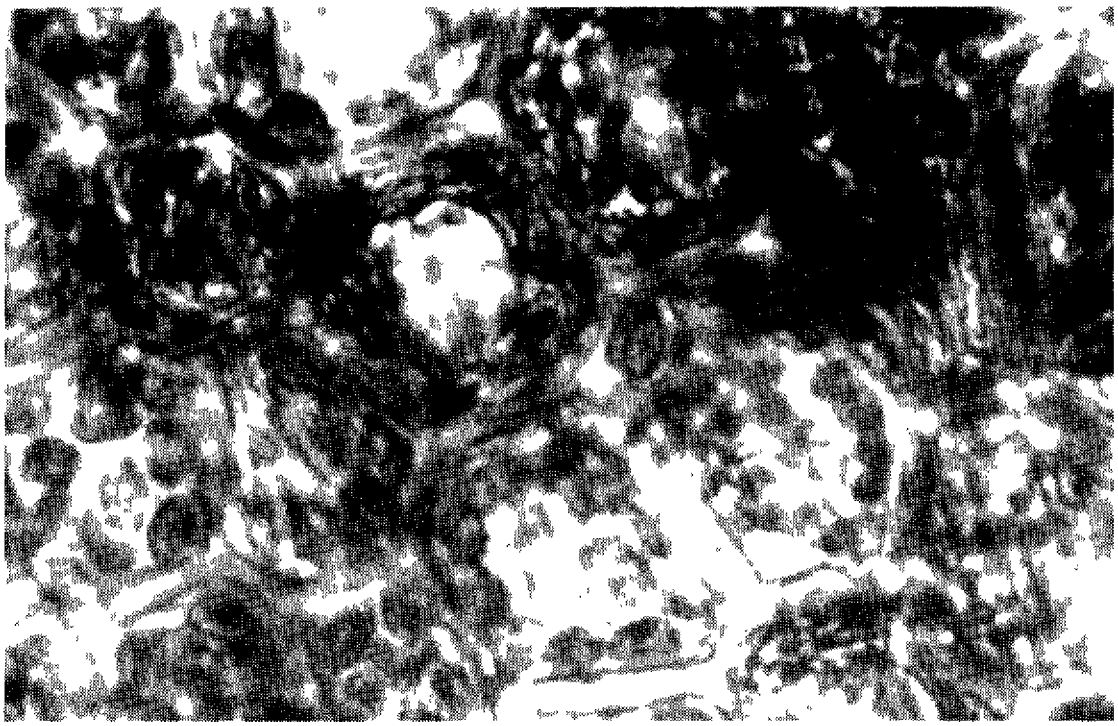

Figure 42: Amorphous, brown strands in infected Zostera marina (fresh) leaf epidermis cells.

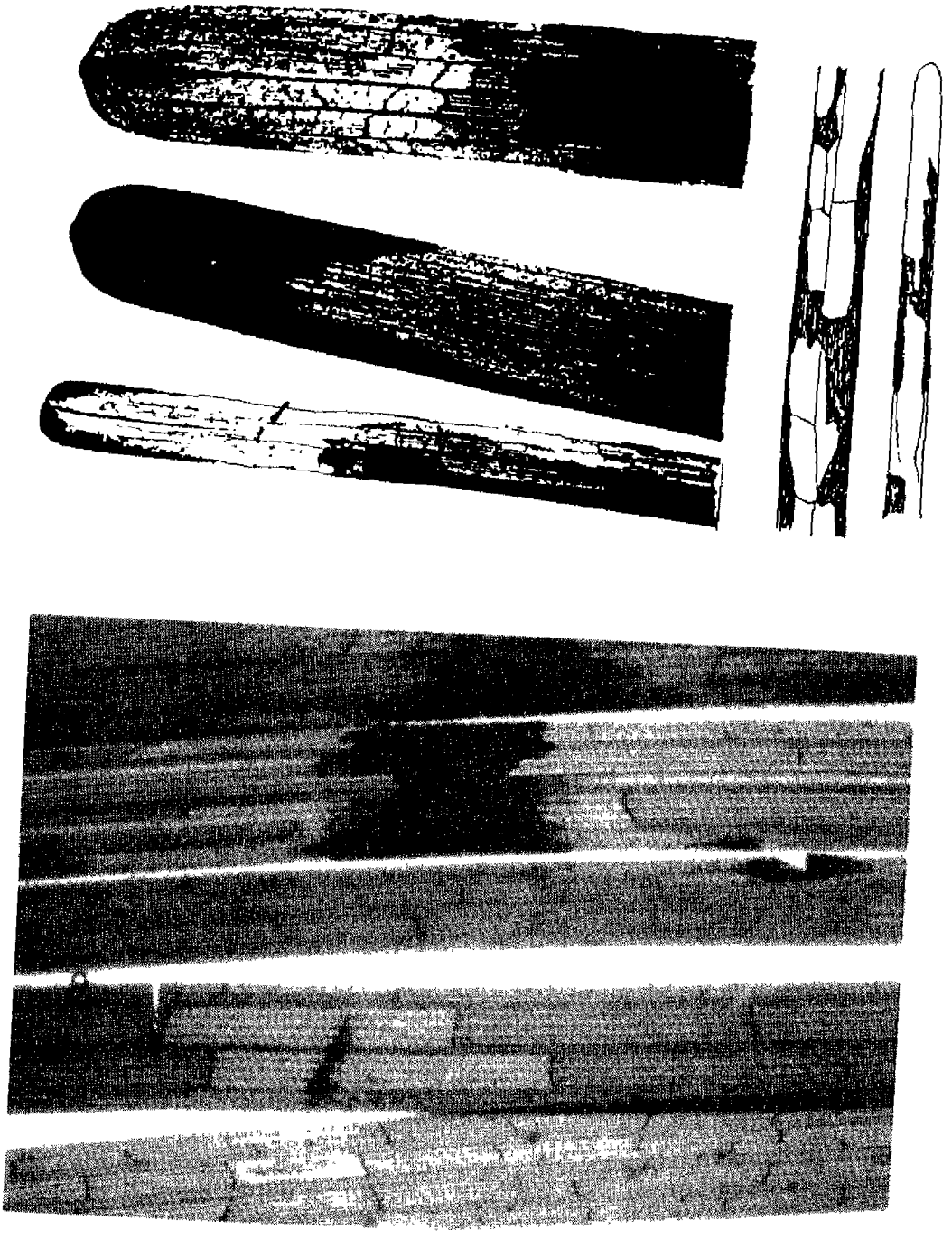

Figure 43: Lesions on leaves of Zostera marina.
top left = RIVO archive (1933); top right = Petersen (1935); bottom = leaf from Lake Grevelingen, 1987.

Figure 44: Wasting disease symptoms on epidermis of fresh leaf.
Infected cells are brown; note the discoloration of chloroplasts and the appearance of amorphous brown strands.

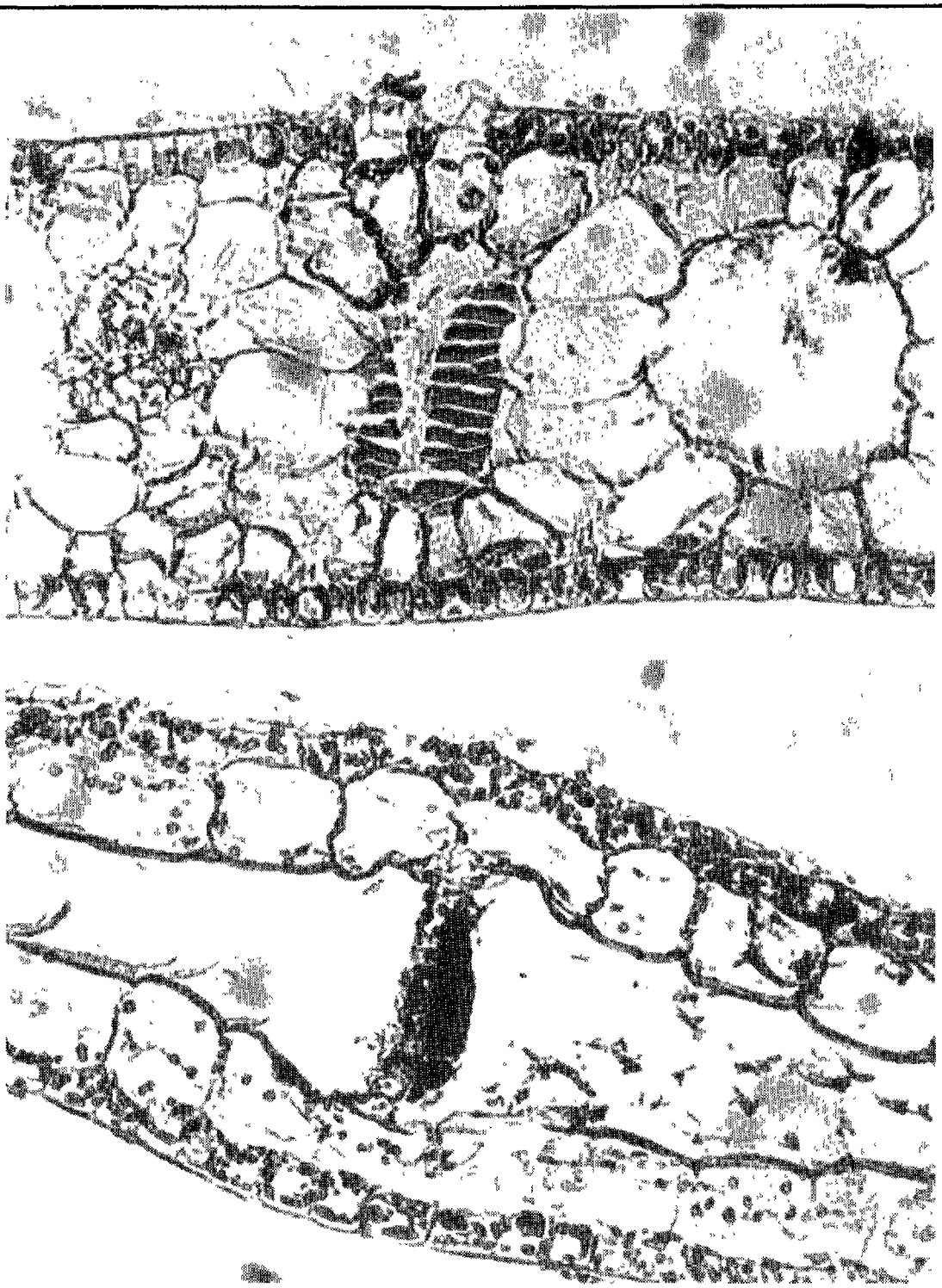

Figure 45: Accumulations of micro-organisms on air-lacuna sieve plates.
top = cross-section, bottom = longitudinal-section

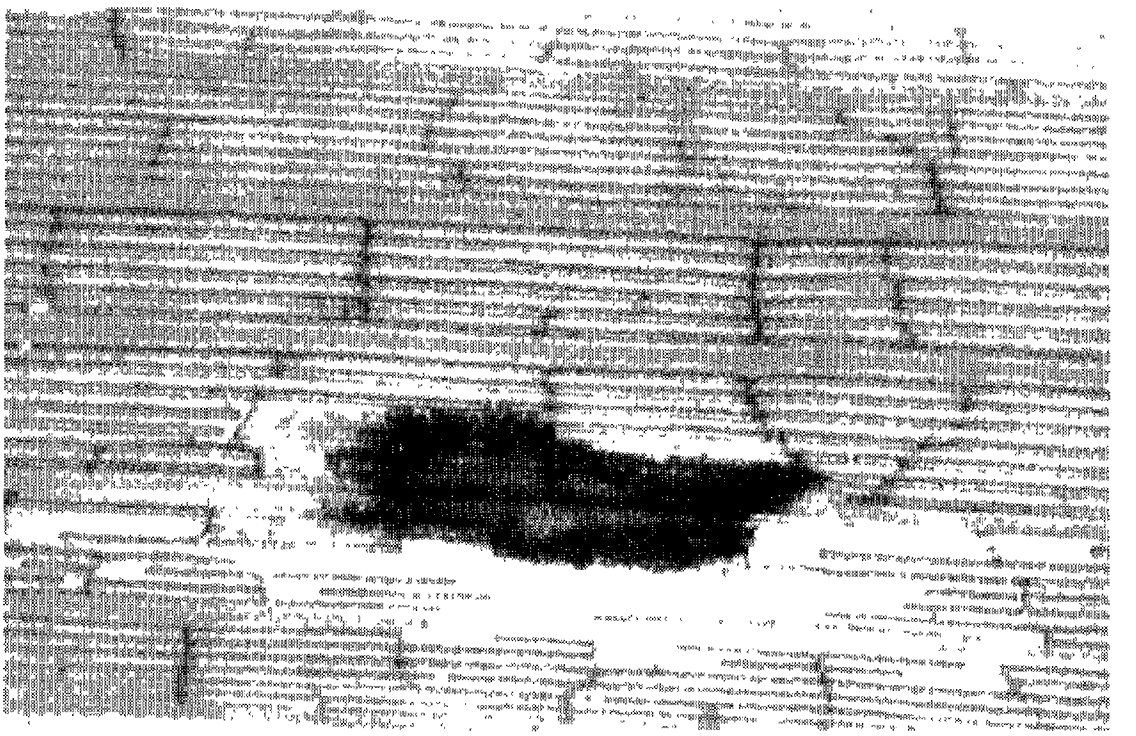

Figure 46: **Leakage associated with lesion on fresh Zostera marina leaf**

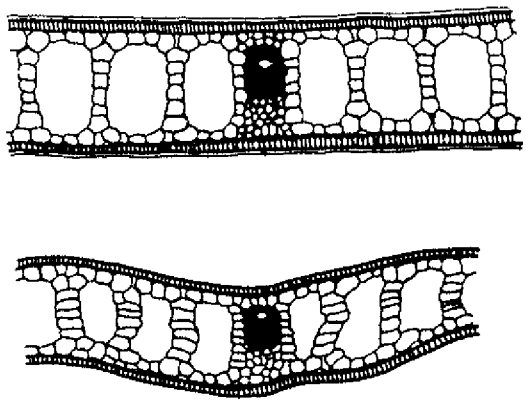

Figure 47: Partitions between air-lacunæ affected by wasting disease.
adapted from Spierenburg (1934), in Polderman and den Hartog (1975), top = healthy, bottom = affected

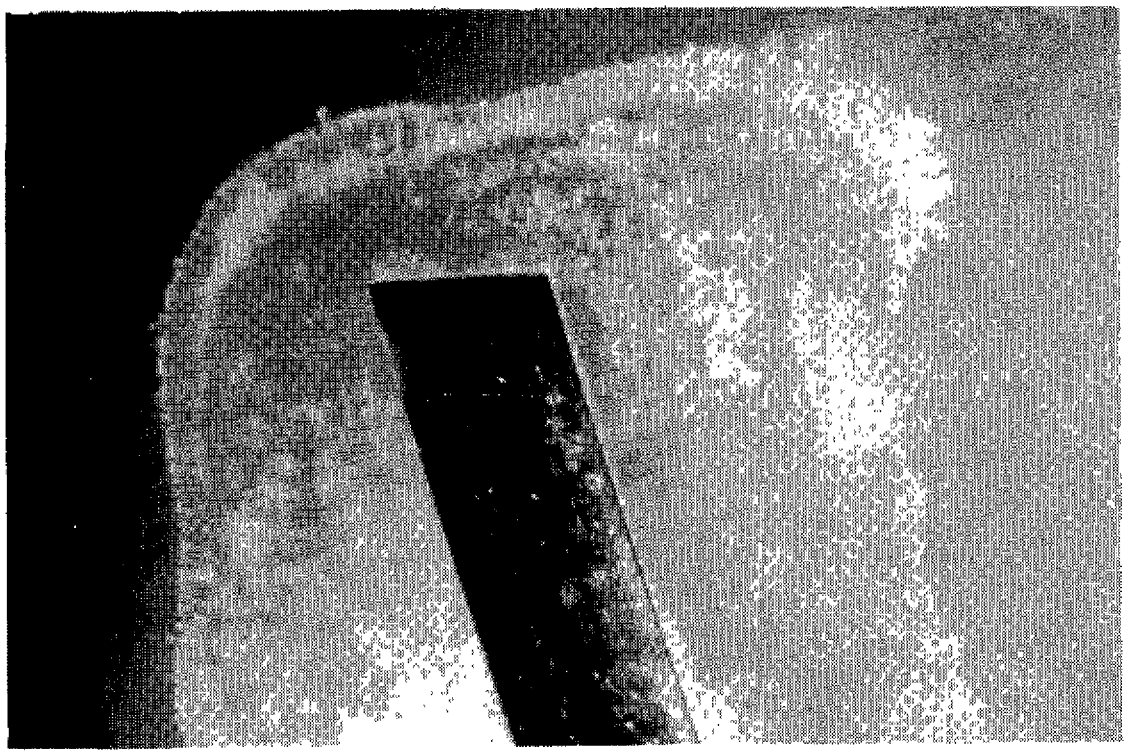

Figure 48: Leaf section on isolation medium, with *Labyrinthula* colony emerging. (leaf width = 3mm)

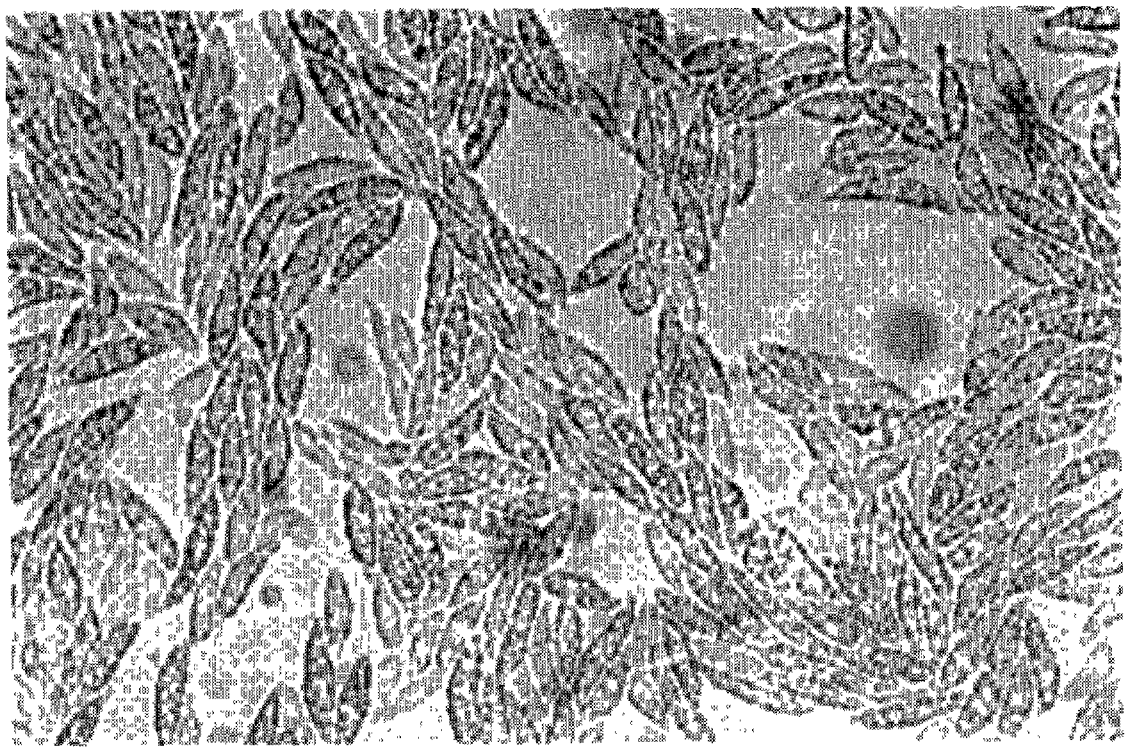

Figure 49: *Labyrinthula* sp..
length of individual spindle cells = ±8-10 µm

Table 13: Sizes of Labyrinthula spp.
adapted from Pokorny (1967)

TABLE 2. *Reported dimensions of* Labyrinthula *spp.*

Species	Length x Width		Investigator	Year
L. macrocystis	18-25 μ	4-5 μ	Cienkowski	1867
"	10-20 μ	5-8 μ	Renn	1936
"	8.5-30 μ	1.5-7 μ	Young	1943
"	15-18 μ	4-5 μ	Watson	1957
L. vitellina var. *vitellina*	15-17 μ	3-4 μ	Cienkowski	1867
L. vitellina var. *pacifica*	15-18 μ	4-5 μ	Watson	1957
L. cienkowskii	11-20 μ	—	Zopf	1892
L. zopfii	—	—	Dangeard	1910
L. valkanovii	≤8 μ	—	Valkanov	1929
L. chattonii	—	—	Dangeard	1932
L. algeriensis	8 μ	3 μ	Hollande & Enjumet	1955
L. roscoffensis	15-30 μ	—	Chadefaud	1956
L. minuta	5-10 μ	3-5 μ	Watson & Raper	1957
L. spp. Vishniac (FPLX, Slb, WH-69, WH-43)	8-10 μ	3-4 μ	Watson	1957
L. sp. Type LX	15-18 μ	4-5 μ	Watson	1957
L. sp. Strain SELX	12 μ	3-4 μ	Watson	1957
L. coenocystis	10-15 μ	3-6 μ	Schmoller	1960

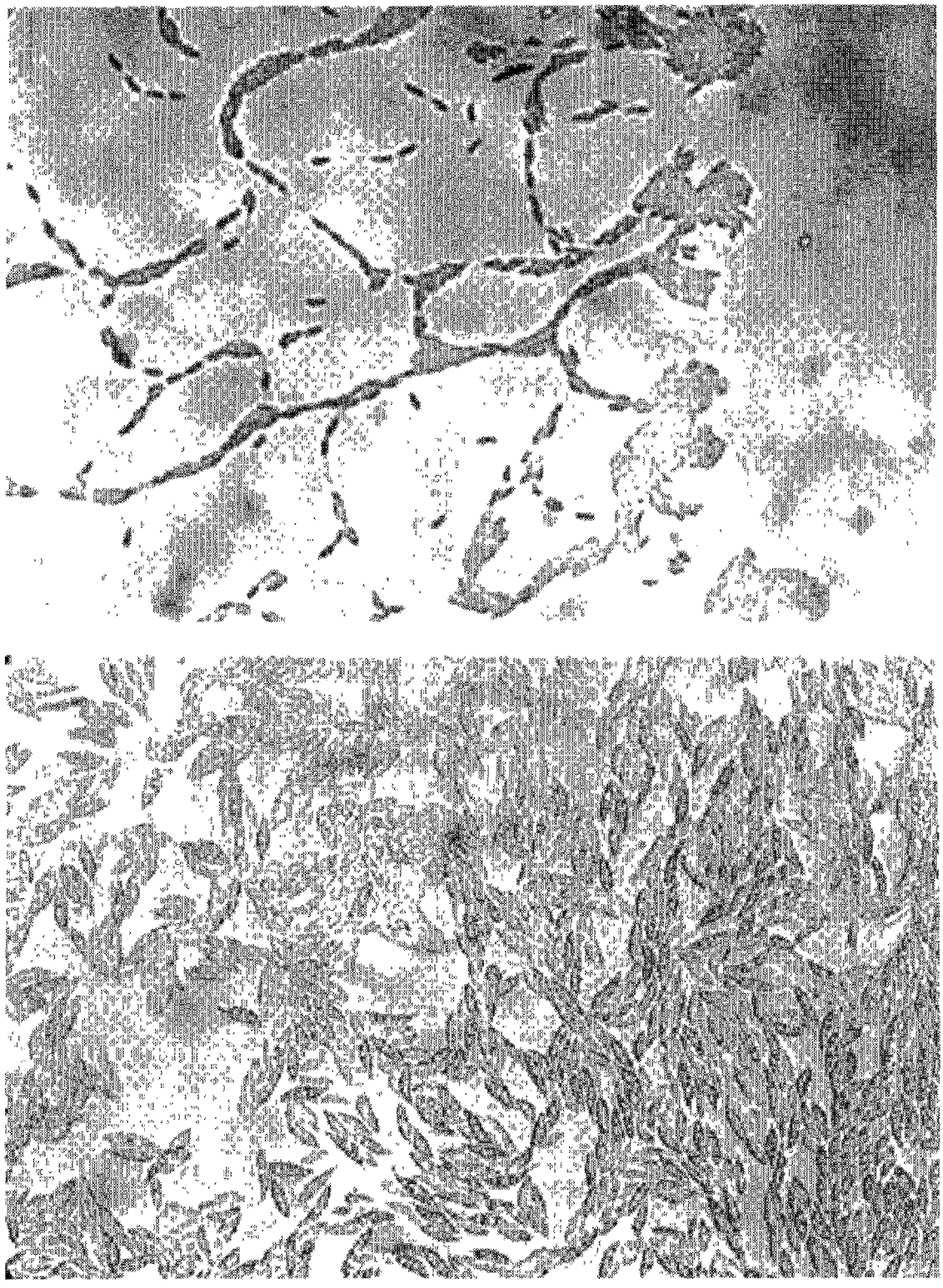

Figure 50: Growth forms of *Labyrinthula* spp..
a) = lobe-shaped, b) = 2-dimensional plate
note: lobe and plate forming colonies grow on top of the agar

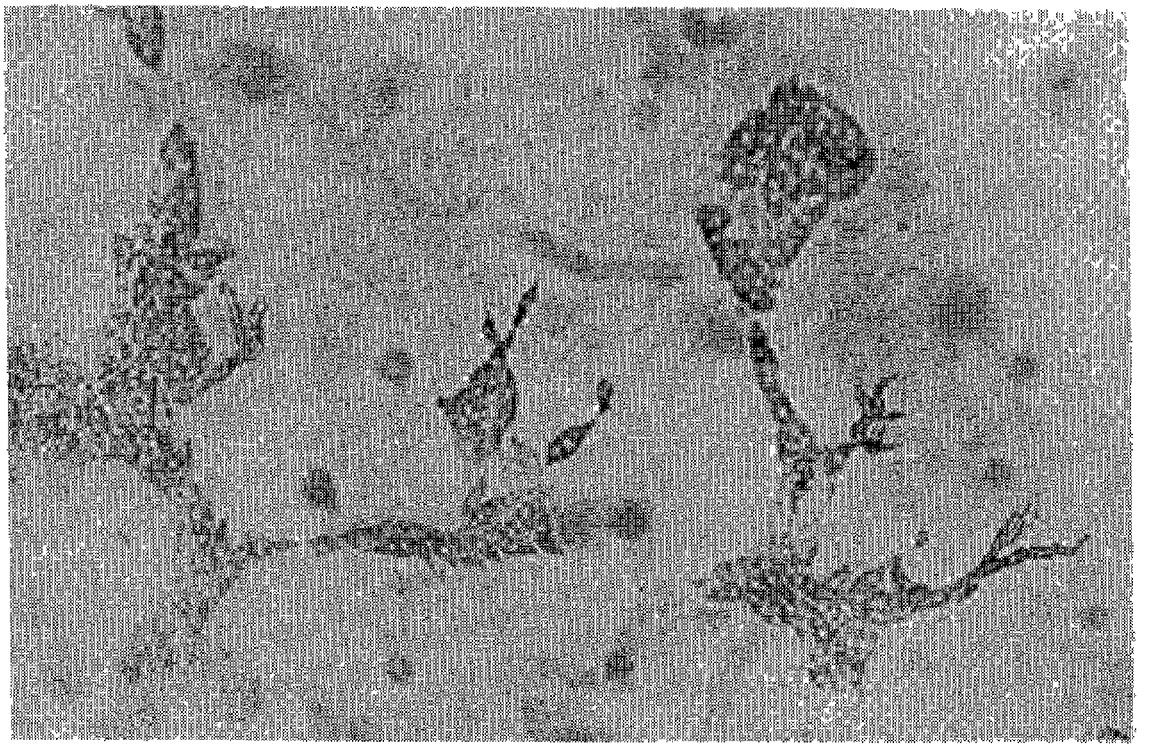

Figure 50: c: Branched form emerging from 2-dimensional plate growth-form of *Labyrinthula*.
Note: the plate growth form grows on top of the agar, the dendritic form penetrates into the agar.

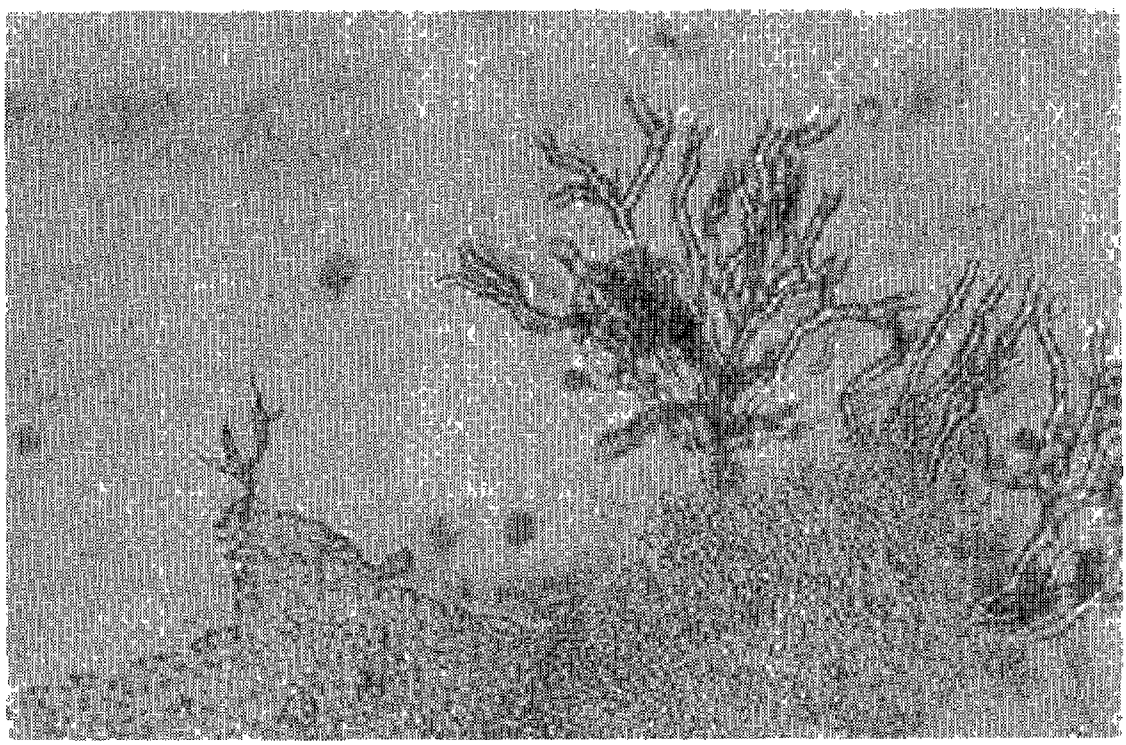

Figure 50: d: As above, but further advanced:

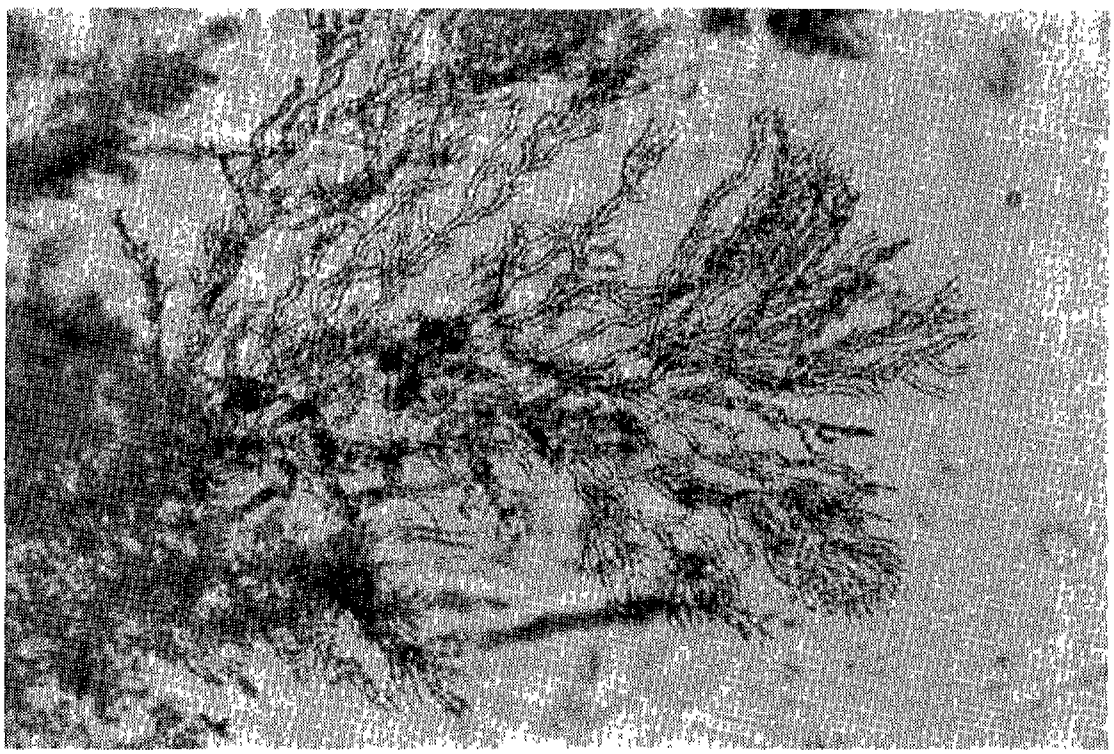

Figure 50: e: Branched form of *Labyrinthula* can attain a random, dendritic form. Note: the dendritic growth form penetrates into the agar.

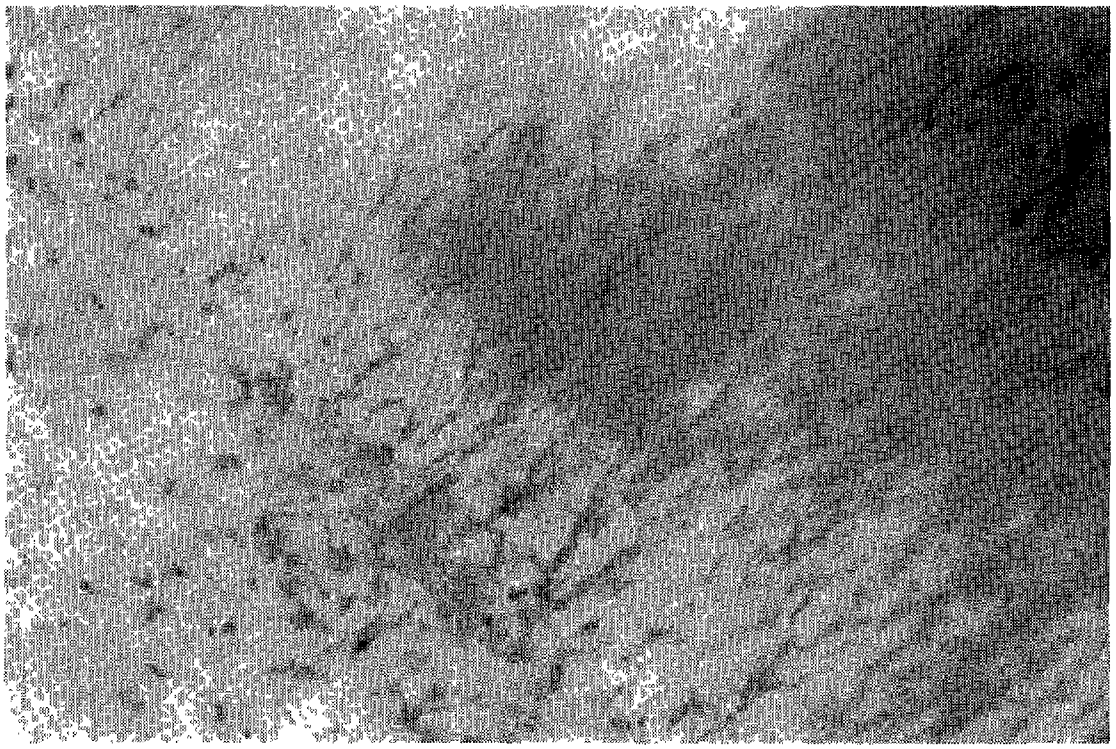

Figure 50: f: Branched form of *Labyrinthula* can attain, a dendritic form with parallel branches.
Note: the dendritic form penetrates into the agar, the plate-like form grows on top of the agar.

OTHER EUROPEAN EELGRASS STANDS

Roscoff, France

Zostera marina beds of Roscoff are the most extensive in Brittany, covering a total area of 12.7 km^2 in 1976, of which almost 22% was sublittoral (Jacobs, 1979). In 1909 their total area was 11.6 km^2, but this dropped drastically during the wasting disease epidemic of the 1930's, and recovery was still far from complete by 1957. After 1960, however, a strong recovery took place, though erosion and recolonization cycles have strongly modified the original area (Jacobs, 1979). Den Hartog reports sighting wasting disease symptoms in Roscoff eelgrass in 1986 (in Short *et al*, 1988). Though this eelgrass appeared to be infected, there was no indication that this was causing any decline (Short *et al*, 1988).

Live plants were kindly collected at Roscoff by the staff of the Laboratory of Aquatic Ecology, on 26th June 1987, and by Frances Van Lent, on the 28th August 1987. These were kept in aquaria; preparations of both live and fixed material were made, and isolation experiments carried out as given in the previous chapter. Observations are given below.

These plants exhibited many lesions, similar to wasting disease symptoms (see fig. 51). Both fresh material and fixed preparations abounded with fruiting Ascomycete fungi (fig. 52), and it was evident that the infection was well advanced. Not surprisingly, isolation experiments failed to reveal *Labyrinthulæ*, but showed a wealth of fungi and diatoms (leaf surface sterilization was not yet successful). The second set of plant specimens (August 28th) were in a far better condition upon arrival, though small, dark lesions were common on most plants, and both microscopic examination and isolation experiments were successful in revealing the presence of *Labyrinthula spp.*. Two different *Labyrinthula spp.* were isolated, one appeared to be generally similar to the species found in Zeeland Province eelgrass, the other differed in nutrient requirements, and could not grow on the Vishniac-glycerol medium. Dr. David Porter of the University of Georgia, USA, also kindly isolated and identified two *Labyrinthula spp* from Roscoff eelgrass. The first-mentioned type turned out to be the pathogenic type.

Fixed and stained preparations of eelgrass leaves gave better results than the Zeeland eelgrass preparations, perhaps because leaves were robuster and more congenial to microtome slicing. Individual *Labyrinthula* spindle cells were once again difficult to identify, but nevertheless observable without having to stretch the imagination fig. 53. In preparations of lesions of fresh leaves, brown, amorphous strands could be seen, especially in epidermis cells where whole cells were observed to be filled with these strands (fig. 54). As with preparations of Zeeland material, jerked motions could be detected in these strands at greater magnifications (400-1000x).

Herbarium specimens collected by den Hartog during annual excursions to Roscoff were examined for possible wasting disease-like lesions. These were found in about 5% of the specimens, from 1975 through to 1986, all with a typical wasting disease-like appearance. Fig. 55 shows lesions of a fresh (1987) and a herbarium (1977) specimen: these appear very similar.

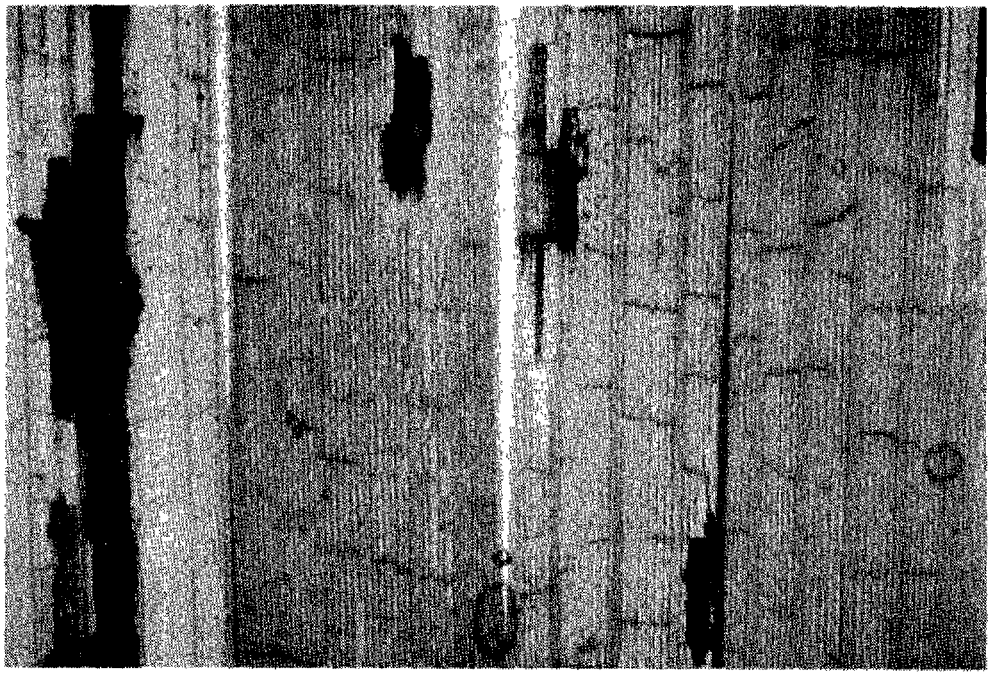

Figure 51: Lesions on fresh Zostera marina leaves from Roscoff, France. collected on August 28, 1987.

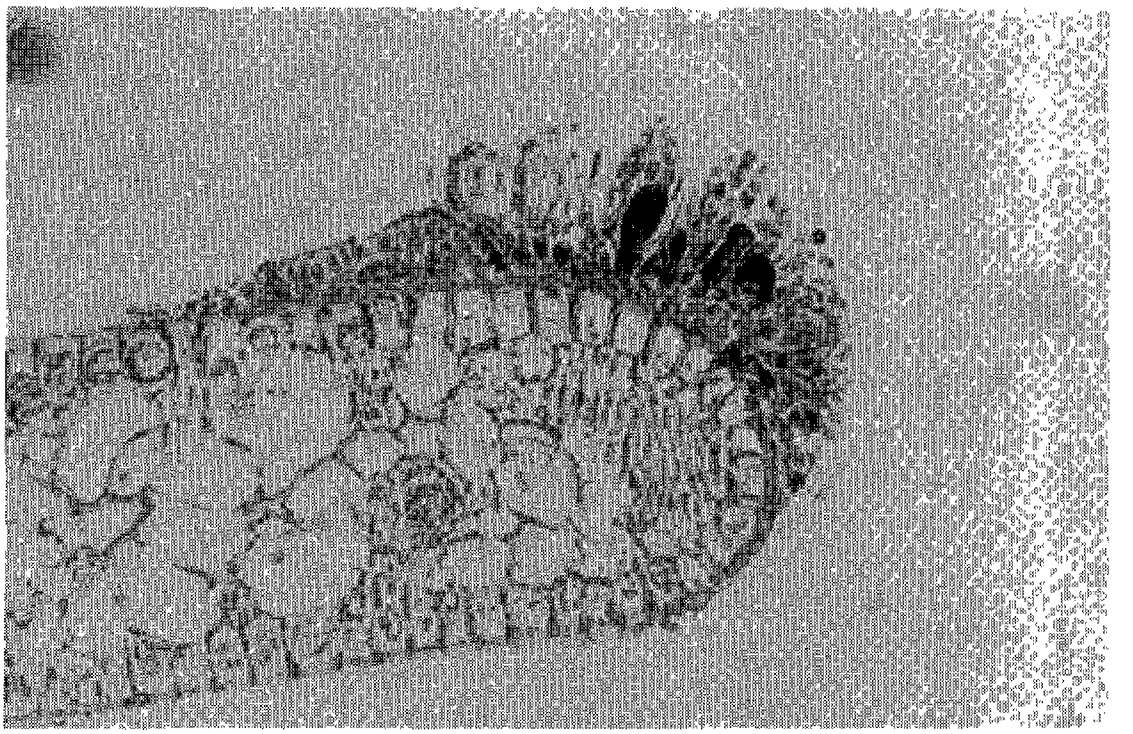

Figure 52: Fruiting ascomycete fungus on Zostera marina leaf from Roscoff, France.

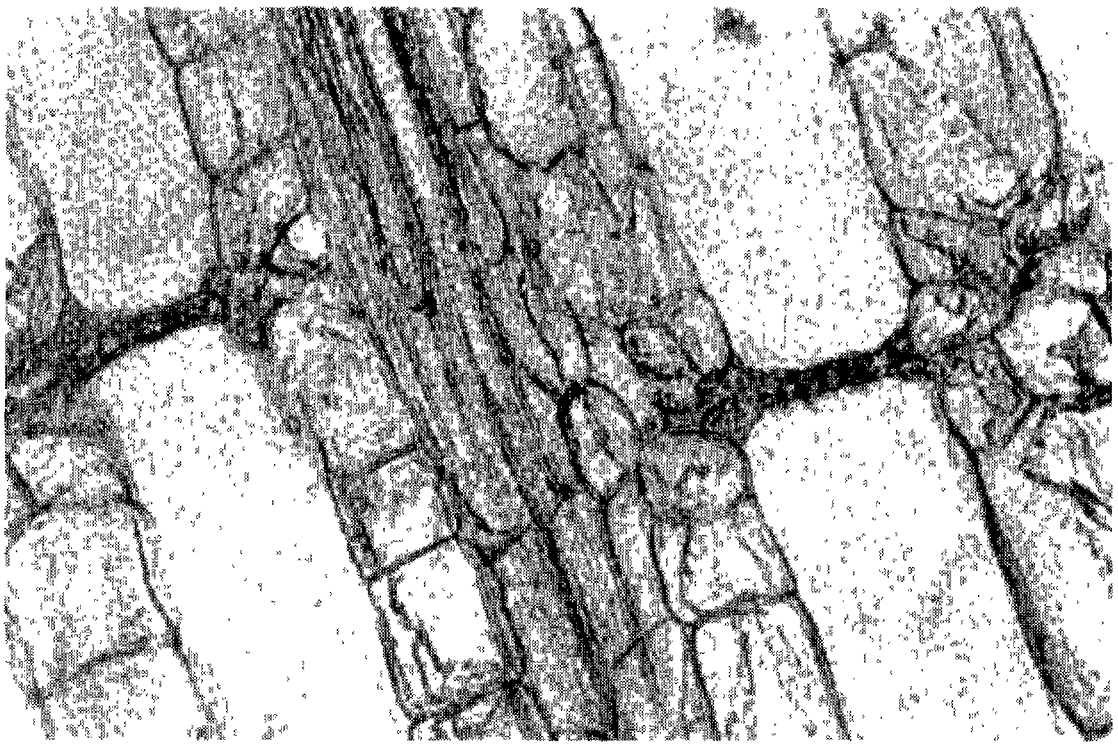

Figure 53: Strands of Labyrinthulæ in infected (fresh) leaf tissue of Zostera marina, Roscoff, France.

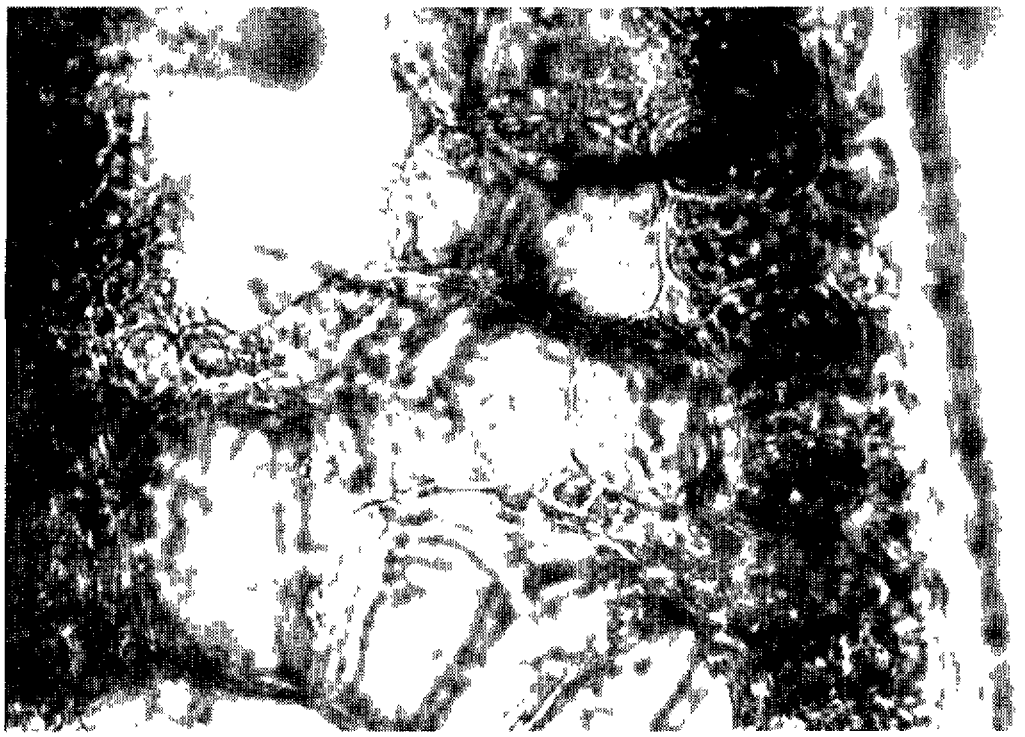

Figure 54: Amorphous brown strands of Labyrinthulæ in epidermis cells of a fresh Zostera marina leaf, Roscoff, France.

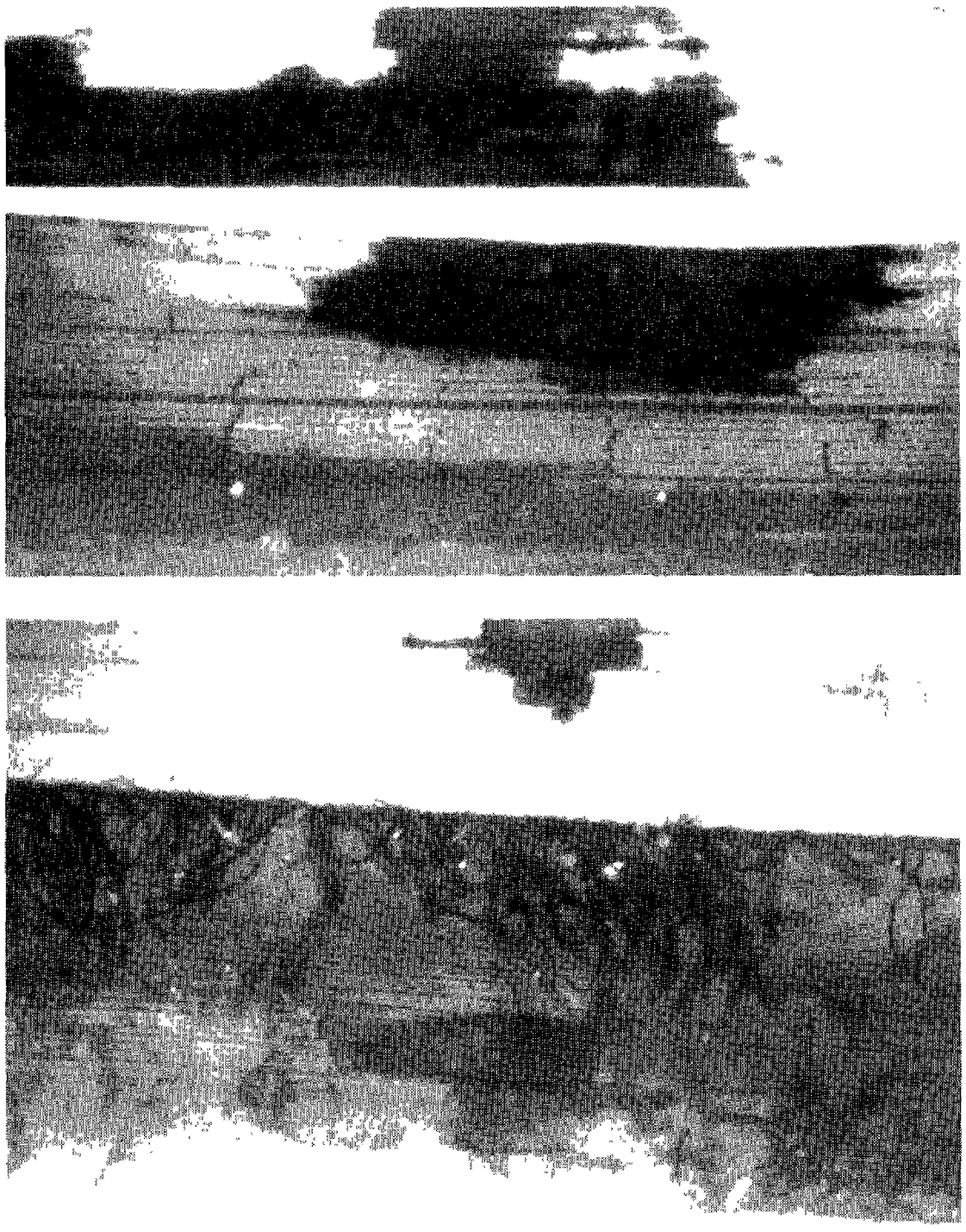

Figure 55: Wasting disease-like lesions on fresh Zostera marina leaves 1987 (small), and herbarium specimen, 1977 (large).
Note that bottom *Z. marina* leaf from Roscoff is well covered with epiphytic Rhodophyceans.

Eelgrass near Wilhelmshaven, German Wadden Sea

Eelgrass stands of the Jade and Jadebusen tidal creeks have been mapped by Linke (1939), Ragutzki (1980) and Michaelis (1987). In 1935-7 a seagrass zone of 5.3 km^2 (*Z. noltii* dominated, *Z. marina* present) encompassed most of the Jadebusen; by 1975-7 this fringe had virtually disappeared. A fairly large patch of 2.75 km^2 of *Z. marina* remained in the southwest, near Dangast, along with sparse remnants in the southeast (Michaelis, 1987). Michaelis (1987) concludes that part of the changes in the Jadebusen are natural dynamics, but also partly may be ascribed to a combination of eutrophication, toxic pollution and artificial increases in salinity. A second small stand occurred on the western shore of the Jade creek, between Crildumersiel and Horumersiel (p.c. Michaelis, 1987). Both sites were visited on the 12^{th} and 13^{th} of November 1987.

At both sites, only *Z. marina* was encountered in small depressions on the tidal flats. All specimens were small (8-15cm length) and narrow-leaved (3-4 mm width), being mainly annuals, but some had a weakly developed rhizome. Macroscopic epiphytes were absent, and many plants had flowered in the previous season, as remnants of inflorescences were common. Many birds fouraged on these tidal flats, especially ducks (*Anas platyrhynchos, Tadorna tadorna*) and gulls (*Larus argentatus* and *L. ridibundus*). Eelgrass density was very low, with averages of 1-3 plants per $10m^2$ for the densest areas encountered on the transects; this low density can partly be ascribed to the lateness of the season, and bird herbivory. Small lesions were present on several plants, but isolation experiments failed to reveal the presence of *Labyrinthula spp.*. It is possible that salinity at both sites may be too low to allow *Labyrinthula* growth, as water volumes left in these intertidal depressions are small, and soon diluted by rainfall.

Ireland

Whelan (p.c. 1987), who has recently carried out an extensive survey on *Zostera spp.* in Ireland (Whelan, 1986), including a study of wasting disease (Whelan and Cullinane, 1987), reports that he did not come across wasting disease symptoms in Irish eelgrass, in spite of dissecting vast numbers of plants from Cork Harbour, Kinsale and Ventry Bay between 1980 and 1984. He does report observations of changes in eelgrass populations:

- Ventry Bay. Lower limit of *Z. marina* was 13m in 1977/8; this had changed to 10m by 1980-2. By June 1982 the plants at Whelan's 9m sampling station had also become affected, and shoot density was much reduced. The shallowest population occurred at 1-2m below MLW.

- Carna, Galway. Beds located off Mweenish Island had reduced to about 10% of their 1960 size by 1982-3. Mainly shallow populations had disappeared, and what remained was a deeper patch at 4-6m depth.

- Kilcrohane, West Cork. Beds extend between 1-4m below MWL (are never exposed), in a small bay (300m maximum diameter). Area of this population had doubled between 1980-6, and it still appears to be expanding in 1987.

In short: no sign of wasting disease-like symptoms, but plenty of dynamics in eelgrass stands, that seem to fit in with the general concept of a dynamic equlibrium (den Hartog, 1987).

BRITTANY

A field trip was undertaken on 5-12 June, 1988, to investigate the occurrence of wasting disease-like symptoms already noticed in these eelgrass beds during the preceding two seasons (1986 and 1987) (personal communication den Hartog; laboratory examination of plant material collected at these sites).
A description of the beds at **Roscoff** is given by Jacobs (1979), who compared the 1909 situation with that of 1976. In 1976, the extent of the beds was greater than that before the wasting disease epidemic of the early 1930's, though their original shape had modified greatly. Over the past few years the beds appear to be diminishing in size, slowly-but-surely, and the general impression is that macroscopic green algae, such as *Ulva spp.* and *Enteromorpha spp.*, now outrank macroscopic brown algae that once dominated the littoral zone (personal communication den Hartog, 1988). In spite of a widespread occurrence of wasting disease-like symptoms in these eelgrass beds, den Hartog (1987) is reluctant to state that this may be the cause of the eelgrass decline. Eight sites were visited (see fig. 56); results are given below:

- site 1) Shallow tidal pool: north-east side with small eelgrass plants (30-35 cm length) and few wasting disease-like (wd-)lesions; southern and central part with large plants (45-60 cm length), with many wd-lesions.

- site 2) Small, littoral plants (30-35 cm length) with many wd-lesions.

- site 3) Very small and narrow-leaved plants (15-20 cm length), at edge of small tidal pool. No wd-lesions present.

- sites 4) and 5) (identical) Relatively small littoral plants, 30-40 cm length, wd-lesions common.

- site 6) Extensive beds of relatively large littoral plants, 40-50 cm length, interspersed with smaller plants (20-30 cm length) on raised beds. Wd-lesions are common, but not as much as at site 1), for instance.

- site 7) Same mosaic of small and large plants as site 6), but the whole is fragmentary, consisting of many small patches. Wd-lesions fairly common.

- site 8) Extensive littoral beds mapped by Jacobs (1979) between the ends of the two moles a + b (see fig. 56), at the entrance of the port, had largely disappeared. Only a heavily epiphytized small patch remained at the western edge.

- site 9) Subtidal beds, with large, broad leaved plants (over 75 cm length), interspersed with *Laminaria spp.*. Wd-lesions common, but the general appearance is healthy.

A short visit was made to the coarse sand flats at **St. Efflam**, (Côtes-du-Nord). Eelgrass at this site appeared to have increased since 1987 (personal communication, den Hartog), in spite of the fact that many plants appeared to be affected with wd-lesions, (see fig. 57). Infection was most evident in eelgrass occurring highest in the tidal zone, and was scarcely present in plants at the lower end. The beds are band-like in structure, in lines running parallel to the coastline.

Isolation experiments for *Labyrinthula spp.* were carried out on eelgrass leaf material from the higher littoral, lower littoral and sublittoral zones. *Labyrinthula spp.* were present throughout, but were not further identified. Isolation experiments carried out on *Fucus serratus, F. vesiculosus, Ulva lactuca* and *Enteromorpha intestinalis* revealed no *Labyrinthula* presence in lesioned material.

One may conclude that wd-lesions (and probably also the associated infection with *Labyrinthula spp.*) are endemic to most eelgrass beds in this part of Brittany. However, the high degree of infection does not seem to be the cause of any major decline.

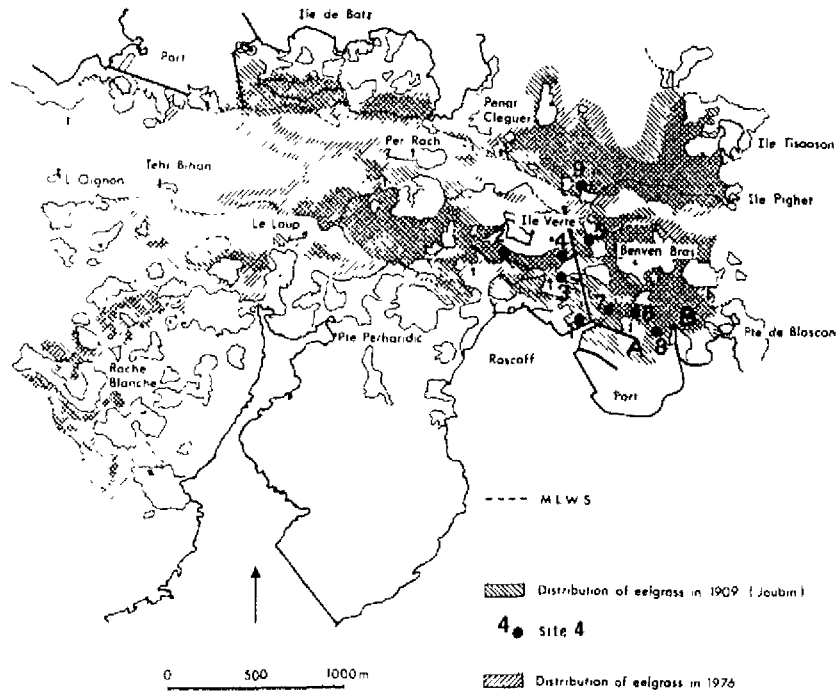

Figure 56: Eelgrass beds at Roscoff.
Adapted from Jacobs (1979); sites visited in 1988 are numbered 1-8.
A and B = moles mentioned in the text.

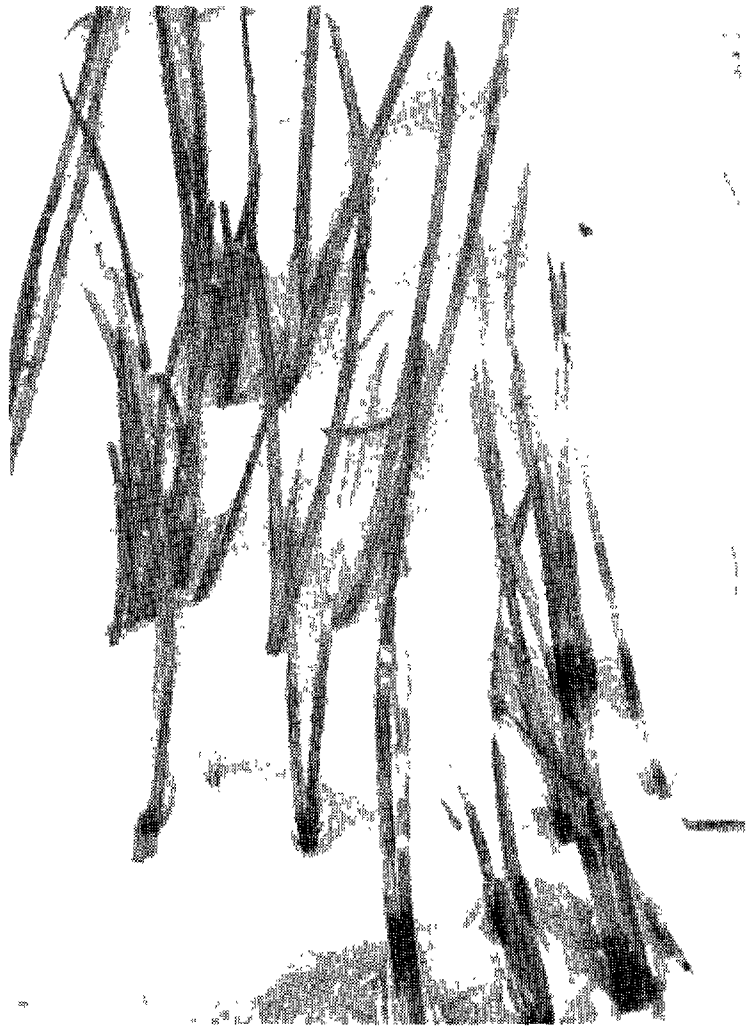

Figure 57: Eelgrass from St. Efflam.
These plants from the intertidal zone display wasting disease-like symptoms.

CORNWALL AND DEVON

A fieldtrip to southwest England was undertaken on 6-14 July 1988, following reports on the decline of *Zostera marina* in the Helford River, Cornwall by Dr. N.A. Holme (p.c. to Den Hartog, April + May, 1988). Additional sites were also visited at St. Michael's Mount, Marazion (Cornwall) and at Millbay, Kingsbridge estuary (Devon) (see fig. 58).

1) Helford River

The Helford River is a sheltered sea inlet or ria (= drowned river valley) with only a little freshwater input. It has long been recognised as having a rich and varied marine life, probably due to a combination of its sheltered environment and the small annual temperature variations (7 - 15 °C). The Helford River was designated a "Voluntary Marine Conservation Area" in July 1987. Past studies in the area are reviewed by Holme and Turk (1986), and an account of a survey undertaken in 1949 is given by Spooner and Holme (1986). Two recent general accounts of marine life in the ria are given by Covey and Hocking (1987) and Rostron (1987). *Zostera spp.* distribution on the Helford River in 1987 is given in fig 59 (adapted from Covey and Hocking, 1987).

On the basis of this map, plus additional information from Rostron (1987) and R. Covey (p.c. to N.A. Holme, 1988), seven sites were investigated (numbered 1 - 7 in map). *Zostera marina* was not encountered, neither subtidally nor intertidally. These seven sites are described below.

site 1) Calamansack wood. Depth (on admiralty chart): ± 1m below lowest astronomical tide. Investigated a zone of about 150 x 10m parallel to the coastline. Bottom: sand and fine shell grit, interspersed with small rocks. Secchi-disk visibility ± 3.5m. Bottom covered with *Ulva spp., Enteromorpha spp., Chorda filum, Gracilaria verrucosa,* .... Small epiphytic algae (not identified) very abundant, forming a dense layer around most specimens of *Chorda filum.*

site 2) Helford passage. Depth: 0 - 1m below lowest astronomical tide (admiralty chart). Bottom: sand, gravel and fine shell fragments. Secchi-disk visibility: ± 3.5m. Investigated two transects of about 150m parallel to the coastline. Growth: much *Enteromorpha spp.*, some *Ulva spp.*, and *Fucus vesiculosus* on the occasional rock. At the deeper end *Chorda filum* and *Desmaresta aculeata* were common.

site 3) Bosahan Point. Depth: (12.30 on 8/7/88) 5m. Bottom: occasional rock, with coarse sand + shell fragments. Covered about 100m parallel to the coastline, in two transects (each 100m). Growth: apparently healthy kelp beds (*Laminaria saccharina, L. hyperborea, Sacchorhiza polyschides*), with *Ulva spp., Enteromorpha spp.* present. *Sargassum muticum* common, with long (4m) fronds.

site 4) Penarvon Cove. Depth: 0-1m on admiralty chart. Bottom: soft mud. Covered: three transects of about 100m each midway between Helford Point and the point to the west of Penarvon Cove. Growth: *Enteromorpha spp.* (very common), *Ulva spp., Chorda filum, Sargassum muticum, Desmarestia aculeata* (present). Small epiphytic algae (undetermined) abundant.

site 5) Prisk Cove. Depth: 0.5 - 3.0m below lowest astronomical tide, on admiralty chart. Visibility: at least 15m, horizontally. Bottom: rocks, with sand pockets, giving way to coarse sand at greater depth, interspersed with rocks. Covered: three transects parallel to the coast, about 100m each. Growth: *Fucus vesiculosus, F. serratus* in top zone, *Laminaria hyperborea, L. ochroleuca* in deeper waters. These kelp beds appear quite healthy, and abundant in anemones, fish, etc. *Ulva and Enteromorpha* were uncommon.

site 6) Parbean Cove. Depth: 3-7m below lowest astronomical tide (admiralty chart). Bottom: fine silt, sand, shell fragments. Covered: three transects, each 150m length. Growth: (at shallower end:) *Fucus spp., Laminaria spp. Enteromorpha spp.*, occasional *Cystoseira sp., Himanthalia elongata* and dense mats of *Ulva spp.*; (at deeper end:) occasional *Laminaria spp., Sargassum muticum* and much bare substrate. Epiphyte growth very dense on some large algal species (*Himanthalia*).

site 7) Gillan Harbour. Depth: (14.30, on 9/7/88) 4-6m. Bottom: mud, sand, shell fragments, shells. Secchi-disk visibility: ± 4-5m. Covered: transect from point halfway between Gillan Cove and Dennis Head, to yellow buoy outside of the harbour. Growth: (inner harbour:) much *Enteromorpha spp., Ulva spp.*, some *Chorda filum, Cystoseris sp., Fucus vesiculosus*, (outer harbour:) much bare substrate, with occasional *Sargassum muticum, Laminaria spp.*. Small epiphytes on macroalgae are dense.

Conclusions: *Zostera marina* was not encountered at any of the seven sites investigated. At four sites (1,3,5,7) it is possible that the beds were missed because of their small size, and the patchy nature of the substrate. At the three other sites (2,4,6) it is unlikely that *Z. marina* still occurs. In general one may conclude that either the species has become very scarce in the Helford River, or it has disappeared altogether.

What could have caused the disappearance of *Zostera marina* in the Helford River ? At most of the sites investigated, green algae such as *Ulva spp.* and *Enteromorpha spp.* seem to have replaced the former eelgrass growth. This is an indication that pollution/eutrophication may have been involved. An argument against this is that *Chorda filum* is still quite common, in spite of it being recorded as sensitive to eutrophication (Grenager, 1957; Den Hartog, 1959). Nevertheless, it may be less common than it was in the past (no dense meadows were encountered, as are described in the references), and epiphyte growth on its fronds was often quite dense. A further complication is that measures have been taken to reduce input of nutrients into the ria, and less is entering the Helford than it was several years ago (p.c. N. Davies, warden of the Lizard Peninsula). It is possible that a process of accumulation has taken place, however. In any case, it would be interesting to monitor any (further) changes in the occurrence of *Chorda filum*. Another possible cause of *Zostera* disappearance is a disease outbreak. Recent reports from the USA and Brittany indicate that a new outbreak of wasting disease may be occurring in North Atlantic eelgrass beds (Short *et al*, 1986; 1988). Plants exhibiting wasting disease-like symptoms were found at Marazion (Cornwall) and Millbay (Devon).

2) Marazion

The intertidal *Zostera marina* bed occurring at Marazion is recorded by Powell *et al* (1978) and Turk (1986). This is located east of the causeway leading to St. Michael's Mount from Marazion village. The site was visited with Miss S. Davis on July 11th at 10.30-11.30. No live plants were encountered, but detached leaves and shoots were common. All displayed black lesions, resembling the wasting disease symptoms (Short *et al*, 1988). *Labyrinthula spp.* were isolated from lesioned tissue, and have been sent to Dr. David Porter (Univ. of Georgia, USA) for further identification. It is possible that the eelgrass bed was located at a greater depth, further away from the causeway than was expected (no map was available); in any case, it would appear that these *Zostera* beds are affected by *Labyrinthula spp.*.

3) Millbay, Kingsbridge Estuary

The Kingsbridge (or Salcombe) Estuary (fig. 60), in Devon, is a drowned river valley or ria and, as the Helford River, it receives little freshwater input. Because of its amenable environment (sheltered, with annual temperature ranges between 8.5 - 16 °C) it provides a habitat for a rich assemblage of marine organisms. The marine biology of the area is briefly dealt with by Powell *et al* (1978), and in more detail by Hiscock (1986). A history of the *Zostera marina* beds at Millbay is given by Wilson (1949).

The Millbay eelgrass beds were visited on July 13th. The southern part of the bed was fragmented and patchy, but this gradually coalesced into one large bed towards the northern part of the bay. The plants were quite large for intertidal specimens, with a vegetative shoot measuring up to 65cm length, and with a width of up to 6.5mm. Virtually all plants exhibited wasting disease-like symptoms, with the typical black lesions, and quite often even the youngest leaves were affected. In spite of this, the bed on the whole does not appear to be declining (there was no indication of mass sloughing-off of leaves), but a further monitoring would be very interesting. Isolation experiments indicate that the lesions are indeed associated with *Labyrinthula spp.*, and Dr. Porter identified them with *Labyrinthula* P.

4) Other locations in south-west England

Eelgrass leaf material from the Scilly Isles and the Fal Estuary was forwarded to the Laboratory of Aquatic Ecology by Dr. N. Holme of Yelverton. The samples from the Scillies were browned, probably by handling/transport, but did not bear wd-lesions, apart from perhaps one small spot on an older leaf. Leaf samples from the Fal Estuary, however, appeared to be much affected, and wd-lesions were very abundant on all leaves.

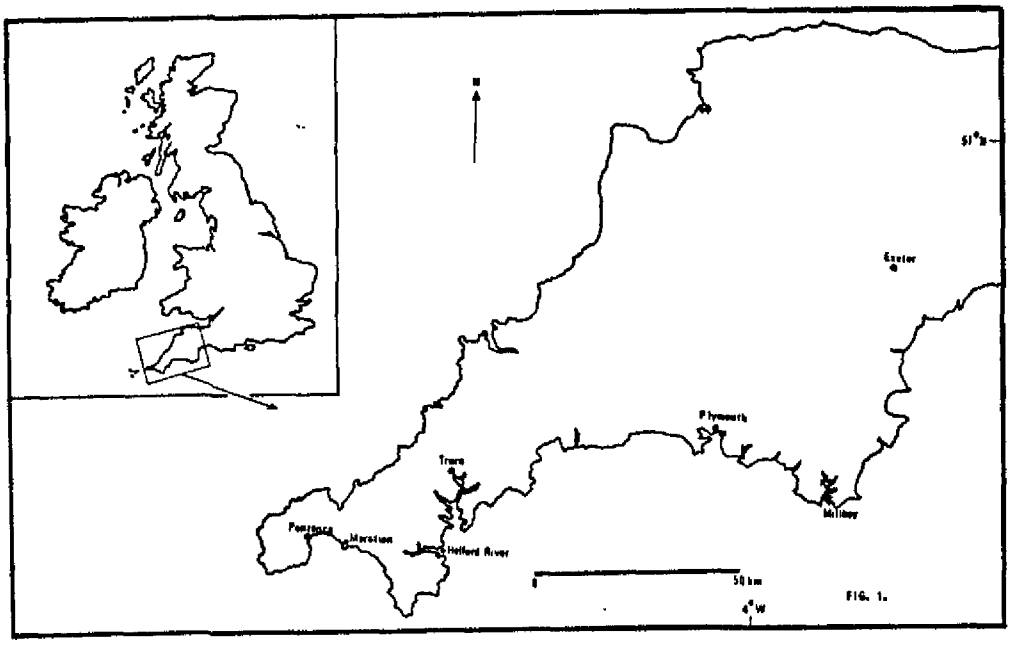

Figure 58: Locations in south-west England.

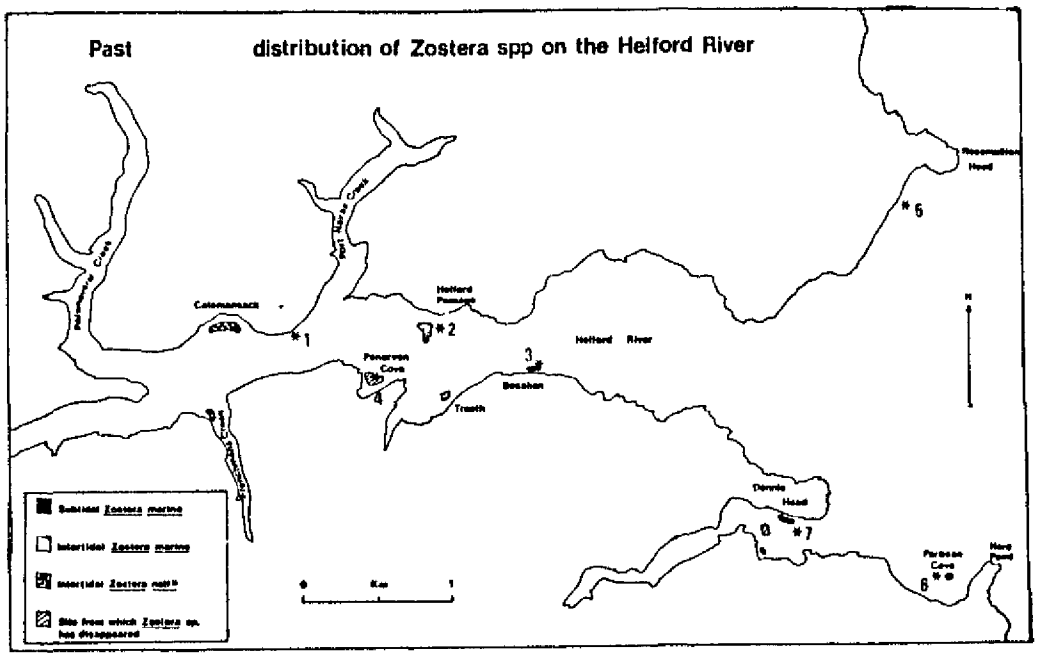

Figure 59: Helford River, Cornwall.
Adapted from Covey and Hocking (1987).

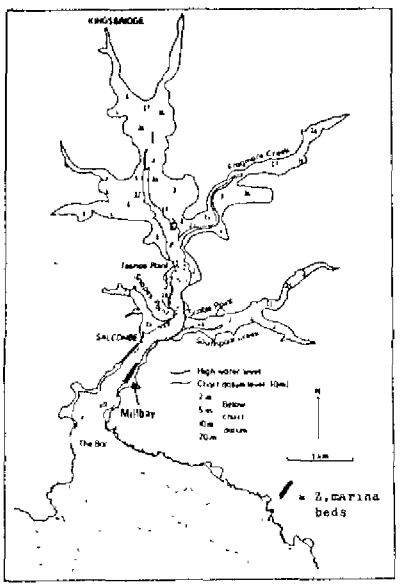

Figure 60: Millbay (Salcombe), Kingsbridge Estuary, Devon. Adapted from Powell et al (1978).

RECENT REPORTS OF WASTING DISEASE IN NORTH AMERICAN EELGRASS POPULATIONS

The first report of a possible recurrence of wasting disease in North American waters was that of Short *et al* (1986). Their studies on the strong decline of *Z. marina* populations in the Great Bay Estuary (New Hampshire-Maine border) between 1981 and 1984 indicated that the decline was not associated with pollution, but that apparantly healthy leaf tissue was infected by *Labyrinthula*, and displayed wasting disease-like symptoms. Decline was not general in the Great Bay area, being pronounced in high salinity areas, populations being stable or even increasing in low salinity waters. Experiments showed that at 30‰ salinity, 100% infection with 75% mortality took place, while at 10‰ salinity, plants grew in spite of the presence of lesions.

By 1987 the eelgrass decline had progressed further in the Great Bay, and infected plants were also found in Penobscot Bay (ME), Maquoit Bay (ME), Rye Harbor (NH), Cape Anne (MA), Lynn Harbor (MA), Charleston Pond (RI), Mystic River (CT) and Beaufort (NC) and along the Nova Scotia coast (Canada) (Short *et al*, 1988). Plants with wasting disease-like symptoms were also observed along the Pacific coast of the USA, in Puget Sound, Washington (Short *et al*, 1988). Mesocosm experiments further demonstrated that at salinities of 15‰ or less, the infection did not spread to healthy leaves (Short *et al*, 1988). In a later study (Short *et al*, 1987) it was demonstrated with tests of Koch's postulates that *Labyrinthula* from diseased plants could subsequently re-infect healthy plants, that thereupon developed the same symptoms. They also demonstrated that a pathogenic strain of *Labyrinthula* exists, giving 100% infection in these disease tests. Several other *Labyrinthula* strains were also tested, but these were not capable of evoking wasting disease-like symptoms.

Short *et al* (1988) report that there are some differences with the wasting disease of the 1930's. The current disease has lead to local declines in some New England eelgrass populations in North America, but this is certainly not of the magnitude of the 1930's. Also, the simultaneous occurrence of diseased plants on the Pacific and Atlantic coast of North America is unlike the situation of the 1930's.

POSSIBLE CYCLIC NATURE OF WASTING DISEASE

Cottam (1934, 1935) reports of past periods of eelgrass decline, occurring in 1854, 1889, 1894, 1908, 1913, 1915, 1917 and 1920-2, but all these are fairly local phenomena. The 1913 decline, for instance, is based on a French report, and may only refer to a lapse in the market situation instead of eelgrass decline. As stated in the previous chapters, the present signs of wasting disease recurrence are only indications that wasting disease may occur. Even if this disease does occur, and causes widescale havoc in eelgrass populations, we then only have two points on a time-scale on which to base a discussion on the possible cyclic nature of wasting disease occurrence. As conclusive evidence for other occurrences of wasting disease (other than that of the 1930's) is lacking, this subject remains somewhat speculative.

Wasting disease of *Zostera marina* in 1931-3 coincided with several other phenomena in the North Atlantic, that all appeared to be connected with a northward extension of warmer, southern waters (Cushing and Dickson, 1976; Cushing, 1982). These include a general paucity in plankton numbers (Southward *et al*, 1975), a northward extension of southern species, especially fish, but also other species, such as octopus in the English Channel (Cushing, 1982), *Ichthyosporidium* disease of herring and mackerel (Fish, 1934; Sproston, 1944; Johnson and Sparrow, 1961), paucity of macroplankton and winter phosphorus (Cushing and Dickson, 1976), a drastic decline in fish catches in the English Channel and near Newfoundland (Cushing, 1982), abnormal dinoflagellate bloom (Pokorny, 1967) and the appearance of boreal invaders in high latitudes in the North-eastern Atlantic. Because of these events, Cushing (1982) refers to the period as "the dramatic decade".

This dramatic decade was subsequently followed by a period of cooling and a reversal of the changes, only to be followed by a renewed "dramatic decade" (though not as outspoken as that of 1925-35) in the late 1960's/early 1970's. It was hypothesized that these events form part of a large-scale cycle, termed the Russell Cycle (Cushing and Dickson, 1976), that appears to be subsequently linked to changes in current patterns in the North Atlantic. Five prominent components describe this cycle (Cushing, 1982): the magnitude of the winter phosphorus maximum; the quantity of fish larvae; the quantity of macroplankton; the presence or absence of arrow-worms (*Sagitta elegans* Verrill and *S. setosa* J. Muller); and the appearance of a pilchard population.

Glémarec (1979) observed that eelgrass beds in the Gulf of Morbihan (France) exhibited dynamics that appeared to be in phase with the Russell cycle. A period of erosion came to an end in 1915, and was followed by a period of rapid extension in 1915-25. During the wasting disease epidemic the beds were lost, but this was followed by a gradual, rather inconspicuous recovery, that did not pick up momentum, however, until about 1965. In 1975 the maximum rate of extension was observed. Glémarec (1979) remarked on similarities between the cycles, but does not propose an explanation for general eelgrass decline. It is highly interesting, of course, that this proposed mechanism, with a cycle of 50-55 years, predicts another major eelgrass decline in the period 1980-85.

A clue to the mechanism underlying the Russell Cycle is given by Southward *et al* (1975), who describe the correlation between cyclic climatic changes and abundance of marine life. The basis of these cyclic events is formed by the sunspot cycle of 10-11 years (and greater cycles of 50-55 and 100-110 years), that was observed to be positively correlated with surface seawater temperatures, without any apparent lag phase. The latter indicates that temperatures of inshore waters are probably more directly linked through atmospheric events, than through changes in ocean currents (Southward *et al*, 1975). The abundance of various marine biota was found to be correlated in a similar manner, but with a lag in phase of 1-4 years.

Although the Russell Cycle and the sunspot cycle help show relationships between different large-scale events, they do not explain the occurrence (and possible recurrence) of wasting disease. To do this, their impact on widescale environmental factors must be regarded. A number of studies exist on the correlation between sunspot activity and environmental factors. King (1973) found a strong positive correlation between the length of the growing season and temperature, and sunspot numbers. He also found that minima and maxima in sunspot numbers coincided with extreme winters and summers, and that sunspot activity correlated negatively with rainfall in northern Africa.

Southward *et al* (1975) found a strong, positive correlation between surface seawater temperatures and sunspot activity, and linked changes with marine biota. Muir (1977) and Colebrook (1977) demonstrate a strong, positive correlation between seawater temperature and sunspot numbers, and suggest that sunspot activity influences the atmospheric effects on the Gulf Stream.

Summarizing, wasting disease occurred simultaneously with a number of other biological phenomena, that appear to be correlated to, and in phase with, the Russell Cycle. This cycle appears to be controlled by the sunspot activity cycle, that in turn has profound, but as yet imperfectly understood, effects on certain environmental factors, such as temperature, precipitation and ocean currents.

In Europe, wasting disease of the 1930's seemed to be correlated to a combination of elevated seawater temperatures and reduced light conditions (see part 2). In what way does this relate to the sunspot activity cycle? Fig. 61 gives the sunspot cycle (line a), together with average summer (July-September) seawater temperatures (line b) and the average growing season (May-August) hours of sunshine (line c), for Den Helder, in the western part of the Dutch Wadden Sea. Though there is a certain amount of agreement between a on the one hand, and b + c on the other hand, it is obvious (and hardly surprising) that sunspot numbers alone do not determine hours of sunshine and seawater temperature. There is a good correlation between b and c, however, but this shows a number of interesting exceptions. Especially the years 1931-3 show a surprisingly high summer seawater temperature, relative to summer sunshine, which may be a further indication of an influx of warmer waters.

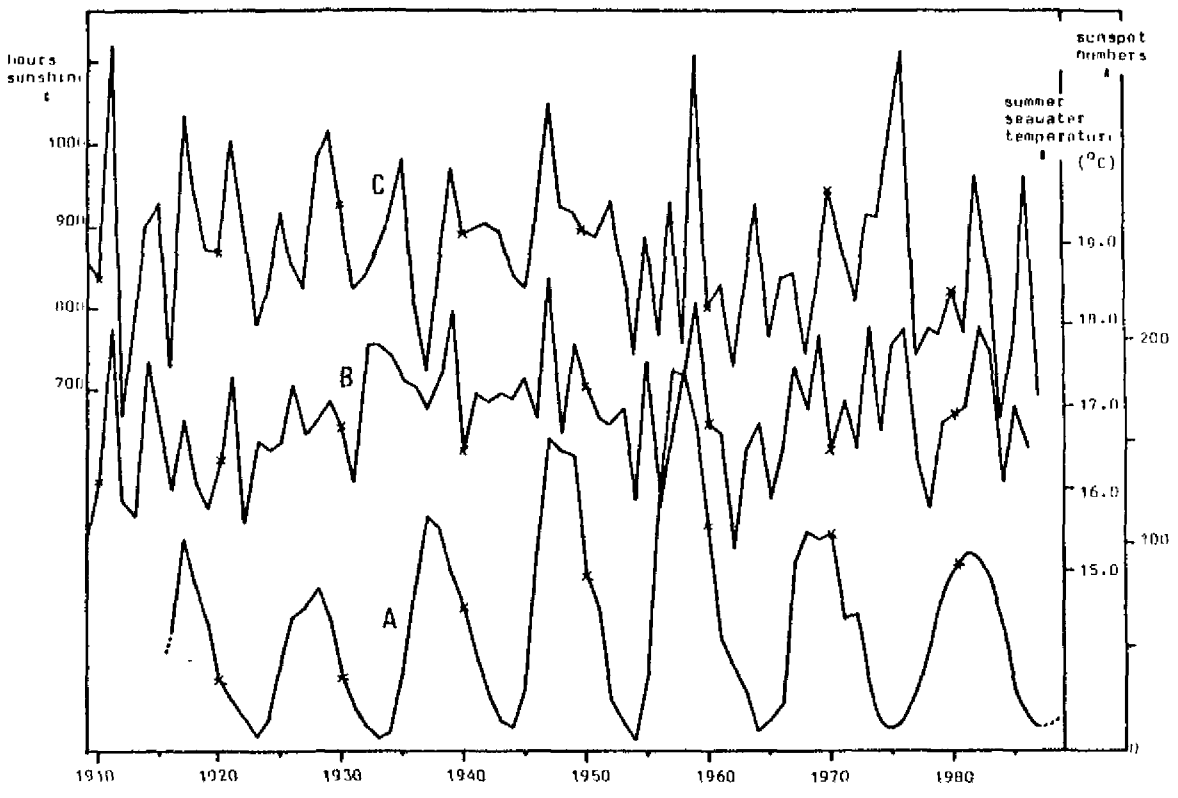

Figure 61: Sunspot numbers (A), seawater temperatures (B) and hours of sunshine (C), Den Helder, the Netherlands, 1909-1987.

Contrary to what may be expected according to Glémarec's hypothesis, no completely similar pattern of sunspot/temperature/sunshine has occurred in the past decade, though there was a combination of relatively high temperature and low irradiation between 1978-81. It would thus appear that present (European) data cannot substantiate Glémarec's hypothesis, but it must be admitted that the present coverage is far from complete.

Part 4

CONCLUSIONS AND DESIDERATA

Conclusions on climate and wasting disease in the Dutch Wadden Sea of the early 1930's

Seawater temperatures were slighlty raised (max. +0.9°C) in the early 1930's, both in the summer and winter months. These increases were by no means unusual, nor do they approach limits inhibiting *Z. marina* growth. It would thus appear that raised seawater temperatures played no role in initiating wasting disease. They may have slightly enhanced the effects of an already prevailing epidemic, as higher winter temperatures increase eelgrass respiration (thus reducing net photosynthesis) and ensure a higher survival rate of pathogenic organisms. The slightly elevated summer temperature represents a slight shift from the eelgrass growth optimum towards the optimum for *Labyrinthula* growth.

The variations found in Dutch Wadden Sea salinities of the early 1930's do not suggest that they were the cause of physiological stress in eelgrass stands. As with seawater temperatures, these variations were by no means unique, nor did they exceed tolerance limits of *Z. marina*. Salinities were slightly below average in 1931, and slightly above average in 1932. At most, the latter may have slightly enhanced the effects of an already prevalent epidemic.

Reduced light conditions appear to have affected the vitality of eelgrass stands of the Dutch Wadden Sea in the early 1930's. Reduced light levels are not evident in 1931 and 1932, if only annual sunshine figures are regarded, but are revealed by the monthly values. On this basis, the reductions experienced in 1931-2 are unusual, compared to the preceding decades. If the effects of this lack of sunshine on eelgrass photosynthesis are calculated on the basis of monthly sunshine figures, however, no dramatic effects are noted. These effects are only evident if the elevated turbidity levels coinciding with this lack of sunshine is also introduced in the calculation. Even more illuminating are the results of the eelgrass growth simulation, based on daily sunshine figures: these clearly show the adverse effects of reduced light conditions. The computor simulation model also demonstrates that the closure of the Zuyder Sea had adverse effects on the eelgrass population in 1932, and may have amplified the effects of the epidemic.

A combination of dull weather and conditions leading to increased turbidity of coastal waters (heavy rainfall, bright early spring, above-average river discharges, strong winds) probably lead to an enfeeblement of *Z. marina* stands in the Dutch Wadden Sea of 1931-2. This effect was probably much enhanced by the closure of the former Zuyder Sea, as this resulted in an elevation of maximum waterlevels, stronger currents, and the dredging and filling activities furthermore directly added to ambient suspended matter levels.

The simulation model suggest that much of the existing eelgrass stands would have succumbed as a result of the poor environmental conditions (lack of sunshine, raised turbidity of Dutch Wadden Sea waters), with or without the presence of a pathogen. This supports suggestions that the wasting disease pathogen, *Labyrinthula macrocystis*, is no more than an endoparasite or saprophyte that finished off an already enfeebled plant.

Desiderata with respect to the cyclic nature of wasting disease

We may conclude that, at present, and using available data on sunshine, salinity and temperature, that these are no firm grounds to assume that wasting disease may have a cyclic nature, dependent on sunspot activity cycles. In Europe, there does appear to be a correlative link between wasting disease of the 1930's and reduced amounts of sunshine. In the USA, no similar relationship was found. A direct link between climatic factors and the present possible recurrence of wasting disease remains vague. If wasting disease does occur on the same magnitude as in the 1930's, we can conclude that it is in phase with the sunspot activity cycle (and the Russell Cycle). As the link via the aforementioned climatic factors is vague (or absent), we must conclude that, either the data are too inaccurate (no

daily *in situ* recordings in existing eelgrass beds), or the relationship runs through a hitherto unknown mechanism. If the latter is the case, the effects of encroachment of warmer, southern waters should be regarded in more detail, especially with regard to the appearance and disappearance of species. At present it is impossible to disprove or conclude that wasting disease is recurring in European eelgrass stands, as information is scanty and seems contradictory. General, apparently not disease-related declines have occurred in the past decades in the Dutch Wadden Sea, parts of the German Wadden Sea and at several localities in Ireland, and more recently in Lake Grevelingen in the Dutch province of Zeeland. Wasting disease-like symptoms were recently found on plants from Roscoff (France), but this does not seem to be causing decline of the eelgrass population, and subsequent examination of herbarium collections showed that these lesions have been present since the mid-1970's. Isolation of the infectious agent, and subsequent disease tests (by Dr. Porter) revealed the occurrence of pathogenic *Labyrinthula's*. Wasting disease-like symptoms also developed on eelgrass collected at several localities in Zeeland Province in October and Novermber 1987; isolation experiments and disease tests (again by Dr. Porter) revealed a pathogenic strain of *Labyrinthula*. The latter is quite puzzling, as an eelgrass decline was observed in Lake Grevelingen in the summer of 1987, possibly partly caused by adverse light conditions, but without clear wasting disease-like symptoms. Why was the pathogen apparently not activated by the stressed state of the eelgrass population (the symptoms did not clearly appear until late in the year) ? Wasting disease-like symptoms were recorded in Zeeland Province eelgrass in autumn, but these were not limited to *Z. marina* populations of Lake Grevelingen. They also occurred in intertidal eelgrass populations that were not affected in the summer of 1987 (Lake Veere, Zandkreek), and appear to be part of the normal end-of-season senescence pattern. It is desireable that further data on recent European eelgrass population dynamics be gathered in order to adequately assess the possible recurrence of wasting disease.

The wasting disease-like symptoms recently witnessed in Europe and North America may indicate that eelgrass populations are on the verge of another large-scale epidemic, such as occurred in the early 1930's. However, wasting disease does not consist of symptoms alone, and no widespread decline of eelgrass due to infection with a pathogenic *Labyrinthula* has been reported up to now. What at present appears to be symptoms of wasting disease may be the result of local causes of eelgrass stress, leading to subsequent *Labyrinthula* infection. Another possibility is that in some of the observations (at least those of material from Zeeland Province) the symptoms are part of the normal end-of-season senescence, that have hitherto escaped attention.

A proper assessment of the possible recurrence of wasting disease is only possible if:

- additional data on recent eelgrass population dynamics are gathered and their causes are assessed

- beds are monitored, and both disease-related and normal senescence patterns are described, as only then can one discern between wasting disease symptoms and normal decay

- herbarium material is examined to assess the occurrence of lesions, both before the 1930's and between the wasting disease epidemic and recent declines

- *Labyrinthula* are isolated, identified and tested from samples of different localities, and the distribution of the pathogenic strain is established

REFERENCES

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

- **Armiger, L.C.** (1964)
  An occurrence of *Labyrinthula* in New Zealand *Zostera*. New Zealand J. Bot., 2:3-9.

- **Atkins, W.R.G.** (1938)
  The disappearance of *Zostera marina*. J. Mar. Biol. Ass. UK, 23:207-210.

- **Atkins,W.R.G. and Jenkins, P.G.** (1952)
  Note on sea temperatures in the English Channel, 1921 to 1949, and Plymouth sunshine and light. J. Mar. Biol. Ass. UK, 31:327-333.

- **Atkinson-Willes, G.L. and Matthews, G.V.T.** (1960)
  The past status of the Brent Goose. British Birds, 53:352-357.

- **Backman, T.W. and Barilotti, D.C.** (1976)
  Irradiance reduction effects on standing crops of the eelgrass *Zostera marina* in a coastal lagoon. Mar. Biol., 34:33-40.

- **Biebl, R. and McRoy, C.P.** (1971)
  Plasmatic resistance and rate of respiration and photosynthesis of *Zostera marina* at different salinities and temperatures. Mar. Biol., 8:48-56.

- **Blegvad, H.** (1934)
  An epidemic disease of the Eel-grass (*Zostera marina L.*). Rep. Danish Biol. Sta., 39:3-8.

- **Boonenburg, K.** (1961)
  Het verdwijnen van het zeegras rond Wieringen. "Uit het Peperhuis", Med. Rijksmuseum Het Zuiderzeemuseum Enkhuizen, p. 229-234.

- **Braak, C.** (1937)
  The climate of the Netherlands. F. Sunshine and cloudiness. Royal Dutch Meteorological Institute (KNMI), Rep. 102, no.40, 51 pp..

- **Bulthuis, D.A.** (1987)
  Effects of temperature on photosynthesis and growth of seagrasses. Aquat. Bot., 27:27-40.

- **Burrell, D.C.** (1973)
  Review paper for the system oceanography working group International Seagrass Workshop, Leiden, the Netherlands, 22-26th October 1973, 21 pp..

- **Burrell, D.C. and Schubel, J.R.** (1977)
  Seagrass ecosystem oceanography. In: Seagrass ecosystems, a scientific perspective. Marine Science, vol.4, ed.'s C.P. McRoy and C. Helfferich. Dekker Publ., N.Y., 314 pp., p.195-232.

- **Butcher, R.W.** (1934)
  Zostera, report on the present condition of eelgrass on the coasts of England, based on a survey during August-October 1933. J. Cons. perm. int. Explor. Mer., 9:49-65.

- **Butcher, R.W.** (1935)
  Wasting disease of *Zostera marina*. Nature, 135:545.

- **Cadée, G.C.** (1982)
  Tidal and seasonal variation in particulate and dissolved organic carbon in the western Dutch Wadden Sea and Marsdiep tidal inlet. Neth. J. Sea Res., 15:228-249.

- **Cadée, G.C.** (1984)
  Has input of organic matter into the western part of the Dutch Wadden Sea increased during the last decades ? Neth. J. Sea Res., Publ. Series, 10:71-82.

- **Cadée, G.C.** (1986a)
  Recurrent and changing seasonal patterns in phytoplankton of the westernmost inlet of the Dutch Wadden Sea from 1969-1985. Mar. Biol., 93:281-289.

- **Cadée, G.C.** (1986b)
  Increased phytoplankton primary production in the Marsdiep area (western Dutch Wadden Sea). Neth. J. Sea Res., 20:285-290.

- **Cadée, G.C. and Hegeman, J.** (1974)
  Primary production of phytoplankton in the Dutch Wadden Sea. Neth. J. Sea Res., 8:240-259.

- **Cadée, G.C. and Hegeman, J.** (1979)
  Phytoplankton primary production, chlorophyll and composition in an inlet of the western Wadden Sea (Marsdiep). Neth. J. Sea Res., 13:224-241.

- **Cadée, G.C. and Hegeman, J.** (1986)
  Seasonal and annual variation in Phaeocystic pouchetii(Haptophyceae) in the westernmost inlet of the Wadden Sea during the 1973 to 1985 period. Neth. J. Sea Res., 20:29-36.

- **Clark, J.R.** (ed.) (1977)
  Coastal ecosystem management. Wiley-Interscience Publ., N.Y./London, 928 pp..

- **Colebrook, J.M.** (1977)
  Possible solar control of North Atlantic oceanic climate. Science, 266:476.

- **Colijn, F.** (1982)
  Light absorption in the waters of the Ems-Dollard estuary and its consequences for the growth of phytoplankton and microplankton. Neth. J. Sea Res., 15:196-216.

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

- **Cottam, C.** (1935a)
  Further notes on past periods of eelgrass scarcity. Rhodora, 37:269-271.

- **Cottam, C.** (1935b)
  The present eelgrass situation along the American Atlantic coast. Plant. Dis. Reptr., 19:230-231.

- **Cottam, C.** (1937)
  Status of eelgrass on the North Atlantic coast, January 1937. Wildl. Res. and Mgmt. Leafl., 35-94.

- **Cottam, C.** (1938)
  Status of eelgrass on the North Atlantic coast, February, 1938. Wildl. Res. and Mgmt. Leafl., 35-110.

- **Cottam, C.** (1941)
The eelgrass situation, fall 1940. Plant. Dis. Reptr., 25:46-52.

- **Cottam, C.** (1945)
Eelgrass conditions along the Atlantic seaboard of North America. Plant. Dis. Reptr., 29:302-310.

- **Covey, R. and Hocking, S.** (1987)
Helford River Survey. A report to the Helford River Steering Group. 121 pp.

- **Cushing, D.H.** (1982)
Climate and fisheries. Academic Press, N.Y./London, 363 pp..

- **Cushing, D.H. and Dickson, R.R.** (1976)
The biological response in the sea to climatic changes. Adv. mar. Biol., 14:1-122.

- **De Cock, A.W.A.M.** (1977)
Culture of *Zostera marina L.* in the laboratory. Aquaculture, 12:279-281.

- **Defoer, H.B.** (1935)
Rapport van de commissie tot onderzoek naar mogelijk verband tussen de afsluiting van de Zuiderzee en den toestand van de wiervisscherij benoorden de Afsluitdijk. stencilled report.

- **De Jonge, V.N.** (1986)
Extraction of deposits in the Dutch Wadden Sea: a possible cause of increased turbidity. Min. of Transport and Waterworks (Min. v. Verkeer en Waterstaat, Rijkswaterstaat Dienst Getijdewateren) Rep. GWAO-86.161, 5 pp..

- **den Hartog, C.** (1959)
The epilithic algal communities occurring along the coast of the Netherlands. North Holland Publ. Co., Amsterdam, 241 pp.

- **den Hartog, C.** (1970)
The seagrasses of the world. Verh. Koninkl. Ned. Akad. v. Wetenschappen, afd. Natuurkunde. 2^{nd} series, 59-1. North Holland Publ., Amsterdam/London, 275 pp..

- **den Hartog, C.** (1987)
"Wasting disease" and other dynamic phenomena in *Zostera* beds. Aquat. Bot., 27:3-13.

- **den Hartog, C. and Polderman, P.J.G.** (1975)
Changes in the seagrass populations of the Dutch Wadden Sea. Aquat. Bot., 1:141-147.

- **Dennison, W.C.** (1987)
Effects of light on seagrass photosynthesis, growth and depth distribution. Aquat. Bot., 27:15-26.

- **Dennison, W.C. and Alberte, R.S.** (1982)
Photosynthetic responses of *Zostera marina L.* (Eelgrass) to *in situ* manipulation of light intensity. Oecologia (Berl.), 55:137-144.

- **Dennison, W.C. and Alberte, R.S.** (1985)
Role of daily light period in the depth distribution of *Zostera marina* (eelgrass). Mar. Ecol. Prog. Ser., 25:51-61.

- **Dennison, W.C. and Alberte, R.S.** (1986)
Photo-adaptation and growth of *Zostera marina L.*(eelgrass) transplants along a depth gradient. J. Exp. Mar. Biol. Ecol., 98:265-282.

- **Duncan, F.M. and Cotton, A.D.** (1933)
  Disappearance of *Zostera marina*. Nature, 132:483.

- **Fenchel, T.** (1970)
  Studies on the decomposition of organic detritus derived from the turtle grass Thalassia testudinum. Limnol. Oceanogr., 15:14-20.

- **Fenchel, T.** (1977)
  Aspects of the decomposition of seagrasses. In: Seagrass ecosystems, a scientific perspective. Marine Science, vol.4, ed.'s C.P. McRoy and C. Helfferich. Dekker Publ., N.Y./Basel, 314 pp., p.'s 123-145.

- **Fischer-Piette, E., Heim, R. and Lami, R.** (1932)
  Note préliminaire sur une maladie bactérienne des *Zostères* C.R. Acad. Sci. (Paris), 195:1420-1422.

- **Fish, F.F.** (1934)
  A fungus disease of fishes of the Gulf of Maine. Parasitology, 26:1-16.

- **Fonseca, M.S., Zieman, J.C., Thayer, G.W. and Fisher, J.S.** (1983)
  The role of current velocities in structuring eelgrass (*Zostera marina L.*) meadows. Estuarine, Coastal and Shelf Sci., 17:367-380.

- **Frantzen, A.J. and Raaff, W.R.** (1982)
  De relatie tussen de globale straling en de relatieve zonneschijnduur in Nederland. Sci. Rep., Royal Dutch Met. Inst. (KNMI), W.R.82-5, 22 pp. + tables.

- **Gall, M.H.W.** (1949)
  Measurements to determine extinction coefficients and temperature gradients in the North Sea and English Channel. J. mar. biol. Ass. UK, 28:757-780.

- **Gams, W.** (1979)
  De fungi van Nederland. Het systeem van de fungi; tabel voor de hoofdgroepen en verwijzingen naar determinatie-werken. Wet. Med. K.N.N.V. No. 136, 115 pp..

- **Gieskes, W.W.C. and Kraay, G.W.** (1977)
  Continuous plankton records: changes in the plankton of the North Sea and its eutrophic southern bight from 1948 to 1975. Neth. J. Sea Res., 11:334-364.

- **Glémarec, M.** (1979)
  Les fluctuations temporelles des peuplements benthiques liées aux fluctuations climatiques. Oceanologica Acta, 2:365-371.

- **Graham, J.J.** (1966)
  Secchi-disk observations and extinction coefficients in the central and eastern North Pacific Ocean. Limnol. Oceanogr., 11:184-190.

- **Grenager, B.** (1957)
  Algological observations from the polluted area of the Oslofjord. Nytt Magasin for Botanik, 5:41-60.

- **Harmsen, G.W.** (1936)
  Systematische Beobachtungen der Nordwest-Europaeischen Seegrasformen. Ned. Kruidk. Archief, 46:852-877.

- **Harrison, P.G. and Mann, K.H.** (1975)
  Chemical changes during the seasonal cycle of growth and decay in eelgrass (*Zostera marina*) on the Atlantic coast of Canada. J. Fish. Res. Board Can., 32:615-621.

- **Hiscock, K.** (1986)
  Surveys of harbours, rias and estuaries in southern Britain. Salcombe Harbour and the Kingsbridge Estuary. Vol. 1, report. Field Studies Council (FSC) and Oil Pollution Res. Unit (OPRU), FSC/OPRU/40/85. March 1986, 69 pp.

- **Holme, N.A. and S.M. Turk** (1986)
  Studies on the marine life of the Helford River: fauna records up to 1910. Cornish Biol. Records. No. 9/1986, 26 pp.

- **Holmes, R.W.** (1970)
  The Secchi disk in turbid coastal waters. Limnol. Oceanogr., 15:688-694.

- **Huntsman, A.G.** (1932)
  Disease in Eel Grass. Progress reports of the Atlantic Biol. Station and Fisheries Exp. Station, No. 5.

- **Ibelings, B.** (1987)
  De wasting disease bij *Zostera marina* L., een overzicht van de historie, de oorzaken en de gevolgen. Doctoraalscriptie nr.79 (student's doctoral report no. 79), 76 pp., Laboratory of Aquatic Ecology, Catholic University of Nijmegen.

- **Idso, S.B. and Gilbert, R.G.** (1974)
  On the universality of the Poole and Atkins Secchi disk-light extinction equation. J. appl. Ecol., 11:399-401.

- **Jacobs, R.P.W.M.** (1979)
  Distribution and aspects of the production and biomass of eelgrass, *Zostera marina L.*, at Roscoff, France. Aquat. Bot., 7:151-172.

- **Jerlov, N,G.** (1970)
  Light: general introduction. In: Marine ecology, environmental factors, ed.: O.Kinne. Wiley Interscience, London, p. 95-102.

- **Jiménez, C., Niell, F.X. and Algarra, P.** (1987)
  Photosynthetic adaptation of *Zostera noltii* Hornem. Aquat. Bot., 29:217-226.

- **Johnson, T.W. and Sparrow, F.K.** (1961)
  Fungi in oceans and estuaries. Cramer Publ., N.Y., 668 pp..

- **Jong, J.** (1976)
  Kern geografie nr.4. Kern der fysische geografie. 21st ed., Vershuys Publ. Amsterdam, 175 pp..

- **Kawasaki, Y.** (1986)
  Effects of temperature on *Zostera marina L.* II. Vegetative growth and reproduction. Denryoku Chuo Kenkyusho Hokoku 0(0486019): i-iv, 1-23.

- **Keddy, C.J.** (1987)
  Reproduction of annual eelgrass: variation among habitats and comparison with perennial eelgrass (*Zostera marina L.*). Aquat. Bot., 27:243-256.

- **Keddy, C.J. and Patriquin, D.G.** (1978)
  An annual form of eelgrass in Nova Scotia. Aquat. Bot., 5:163-170.

- **Keller, M. and Harris, S.W.** (1966)
  The growth of eelgrass in relation to tidal depth. J. of Wildl. Mgmt., 30:280-285.

- **Kemp, W.M., Twilley, R.R., Stevenson, J.C., Boynton, W.R. and Means, J.C.** (1983)
  The decline of submerged vascular plants in upper Chesapeake Bay: Summary of results concerning possible causes. Mar. Technol. Soc. J., 17(2):76-89.

- **King, J.W.** (1973)
  Solar radiation changes and the weather. Nature, 245:443-446.

- **Kirkman, H.** (1978)
  Decline of seagrasses in northern areas of Moreton Bay, Queensland. Aquat. Bot., 5:63-76.

- **Levine, N.D.** (1980)
  A newly revised classification of the Protozoa. J. Protozool., 27:37-58.

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

- **Linke, O.** (1939)
  Die Biota der Jadebusenwatten. Helgol. wiss. Meeresuntersuchungen: 201-348.

- **Lüning, K.** (1981)
  Light. Chapter 9 in: The biology of seaweeds. Bot. Monographs, vol. 17, ed.'s C.S. Lobban and M.J. Wynne. Blackwell Publ.: 326-364.

- **Manuels, R.** (ed.) (1978)
  Water-, stof- en warmtehuishouding van het Balgzand. Internal report NIOZ (Netherlands Inst. Sea Res.), 1978-2, 112 pp..

- **Marsh, J.A., Dennison, W.S. and Alberte, R.S.** (1986)
  Effects of temperature on photosynthesis and respiration in eelgrass (*Zostera marina L*.). J. Exp. Mar. Biol. Ecol., 101:257-267.

- **Martin, A.C.** (1954)
  A clue to the eelgrass mystery. Trans. 19th North American Wildl. Conf., p. 441-449.

- **Martinet, J.F.** (1782)
  Verhandeling over het wier der Zuiderzee. Verh. Hollandsche Mij. der Wetenschappen (Haarlem), 20(2):54-119 + plates I-VI.

- **Mazzella, L.** (1979)
  Photosynthetic characteristics of *Zostera marina L.* (Eelgrass). Biol. Bull., 157:382.

- **McRoy, C.P.** (1966)
  The standing stock and ecology of eelgrass, *Zostera marina* ,Izembek Lagoon, Alaska. MSc. thesis, University of Washington, Seattle, 138pp..

- **McRoy, C.P. and Helfferich, C.** (1980)
  Applied aspects of seagrasses. In: Handbook of seagrass biology: an ecosystem perspective, eds. Phillips and C.P. McRoy. Garland Press, N.Y./London: 297-343.

- **Michaelis, H.** (1987)
  Strukturveränderungen der Wattenfauna am Beispiel des Jadebusens. In: Umwelt Vorsorge Nordsee, - Belastungen, Gütesituation, Massnahmen; Min. of Environment, Hannover, Niedersachsen, August 1987.

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

- **Molander, A.R.** (1933)
Anteckninger till *Zostera*vegetationens förekomst vid västkusten. Ny Svensk Fiskeritidskrift, 18:207-208.

- **Moss, S.T.** (1987)
Biology and phylogeny of the *Labyrinthulales* and Thraustochytriales. In: The biology of marine fungi, ed. S.T. Moss. Cambridge Univ. Press, Cambridge: 105-129.

- **Mounce, I. and Diehl, W.W.** (1934)
A new Ophiobolus on eelgrass. Canadian J. Res., 11:242-246.

- **Muir, M.S.** (1977)
Possible solar control of North Atlantic oceanic climate. Nature, 266:475-476.

- **Müller, M.J.** (1983)
Handbuch ausgewählter Klimastationen der Erde. Univ. of Trier, W. Germany. Forschungstelle Bodenerosion, Mertsdorf (Ruwertal), Trier, 346 pp..

- **Newell, N.D.** (1963)
Crises in the history of life. Sci. Am., 208:76-92.

- **Newell, S.Y.** (1981)
Fungi and bacteria in or on leaves of eelgrass (*Zostera marina* L.) from Chesapeake Bay.Appl. Environm. Biol., 41:1219-1224.

- **Newell, S.Y. and Fell, J.W.** (1982)
Surface sterilization and the active mycoflora of leaves of seagrass. Bot. Mar., 25:339-346.

- **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., 17:69-77.

- **Nienhuis, P.H.** (1984)
Zeegras, mysterieuze opkomst en ondergang van een waterplant. Natuur en Techniek, 54:414-433.

- **Nienhuis, P.H. and De Bree, B.H.H.** (1977)
Production and ecology of eelgrass (*Zostera marina* L.) in the Grevelingen estuary, the Netherlands, before and after the closure. Hydrobiologia, 52:55-66.

- **Orth, R.J.** (1975)
Destruction of eelgrass, *Zostera marina*, by the cownose ray, *Rhinoptera bonasus*, in the Chesapeake Bay. Chesapeake Sci., 16:205-208.

- **Orth, R.J. and Moore, K.A.** (1983)
Chesapeake Bay: An unprecedented decline in submerged aquatic vegetation. Science, 222:51-53.

- **Ostenfeld, C.H.** (1908)
On the ecology and distribution of the grass wrack (*Zostera marina*) in Danish waters. Rept. Danish Biol.Sta., 16:62-.

- **Oudemans, C.A.J.A.** (1870)
Verslag der Staatscommissie inzake de wiervisscherij. typescript, the Hague.

- **Pellikaan, G.C.** (1980)
De verspreiding en de groei van zeegras, *Zostera marina* L., in relatie tot de instraling. Delta Institute for Hydrobiol. Res., Yerseke, the Netherlands. Students Rep. no. D8-1980, 103 pp..

- **Pellikaan, G.C.** (1982)
Decomposition processes of eelgrass, *Zostera marina* L.. Hydrobiol. Bull., 16:83-92.

- **Petersen, H.E.** (1935)
Preliminary report on the disease of the eelgrass (*Zostera marina* L.).Rep. Danish Biol. Sta., 40:1-8.

- **Petersen, H.E.** (1936)
Studies on a parasitic fungus in the eelgrass, *Zostera marina* L.. L. Bot. Tidsskr., 43:1-9.

- **Phillips, R.C.** (1980)
Transplanting methods. In: Handbook of Seagrass Biology, An Ecosystem Perspective. Ed.'s R.C. Phillips and C.P. McRoy. Garland Press, New York/London, pp. 41-56.

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

- **Polderman, P.J.G. and den Hartog, C.** (1975)
De zeegrassen in de Waddenzee. K.N.N.V. Wet.Med. nr. 107, May 1975, 32 pp..

- **Poole, H.H. and Atkins, W.R.G.** (1929)
Photo-electric measurements of submarine illumination throughout the year. J. mar. biol. Assoc. UK, 16:297-324.

- **Porter, D.** (1969)
Ultrastructure of *Labyrinthula*. Protoplasma, 67:1-19.

- **Porter, D.** (1974)
Phylogenetic considerations of the *Thraustochytriaceae* and *Labyrinthulaceae*. Veröff. Inst. Meeresforsch. Bremerh. Suppl., 5:19-44.

- **Porter, D.** (1987)
Labyrinthulomycetes. In: Zoosporic fungi in teaching and research, ed.'s M.S. Fuller and A. Jaworski. Southeastern Publ. Corp., Athens, GA. ISBN 0-935460-02-0, p.'s 110-113.

- **Postma, H.** (1961)
Suspended matter and secchi-disc visibility in coastal waters. Neth. J. Sea Res., 1:359-390.

- **Powell, H.T., Holme, N.A., Knight, S.J.T. and Harvey** (1978)
Survey of the littoral zone of the coast of Great Britain. Report on the shores of Devon and Cornwall. S.M.B.A./M.B.A. intertidal survey unit, Plymouth, Feb. 1978, 124 p.'s + 11 maps.

- **Ragutzki, G.** (1980)
Verteilung der Oberflächensedimente auf den Niedersächsischen Watten. Forsch. Stelle f. Insell u. Küstenschutz, 32:55-67.

- **Rasmussen, E.** (1977)
  The wasting disease of eelgrass (*Zostera marina*) and its effects on environmental factors and fauna. In: Seagrass ecosystems, a scientific perspective, Marine Science vol.4, ed.'s C.P. McRoy and C. Helfferich.Dekker Publ., N.Y./Basel, p.'s 1-51.

- **Redeke, H.C.** (1916)
  Over het wier en de wiervisscherij.Meded.Visscherij, 22:77-81.

- **Reigersman, C.A.J.** (1939)
  Rapport omtrent de invloed van de wierziekte op de achteruitgang van de wierbedrijven. stencilled report, the Hague.

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

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

- **Renn, C.E.** (1936)
  Persistence of the eelgrass disease and parasite on the American Atlantic coast. Nature, 138:507-8.

- **Renn, C.E.** (1937)
  The eelgrass situation along the middle-Atlantic coast. Ecology, 18:323-325.

- **Renn, C.E.** (1942)
  Demonstration of *Labyrinthula* parasite in eelgrass from the coast of California. Science, 95:122.

- **Renouf, L.P.W.** (1934)
  Zostera decline on the coast of County Cork, I,F.S.. Nature, 133:912.

- **Rostron, D.** (1987)
  Surveys of harbours, rias and estuaries in southern Britain. The Helford River. Vol. 1, report. Field Studies Council (FSC) and Oil Pollution Res. Unit (OPRU), FSC/OPRU/17/87, 69 p.'s.

- **Schellekens, A.W.H.J.** (1975)
  Zeegrasonderzoek Terschelling. Lab. of Aquatic Ecology, student's report no. 17, 26 pp. + maps.

- **Setchell, W.A.** (1929)
  Morphological and phenological notes on *Zostera marina* L.. Univ. Calif. Publ.Bot., 14:389-452.

- **Short, F.T., Mathieson, A.C. and Nelson, D.** (1986)
  Recurrence of the eelgrass wasting disease at the border of New Hampshire and Maine, U.S.A.. Mar. Ecol. Prog. Ser., 29:89-92.

- **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.

- **Short, F.T., Ibelings, B. and Den Hartog, C.** (1988)
  Comparison of a current eelgrass disease to the wasting disease in the 1930's. Aquat. Bot., 30:295-304.

- **Southward, A.J., Butler, E.I. and Pennycuick, L.** (1975)
  Recent cyclic changes in climate and in abundance of marine life. Nature, 253:714-717.

- **Spierenburg, D.** (1933)
  Een ziekte in het zeegras (*Zostera marina* L.). Tijdschr. over Plantenziekten, 39:193-199.

- **Spooner, G.M. and Holme, N.H.** (1986)
  Studies on the marine life of the Helford River: 2. Results of a survey in September 1949. Cornish Biol. Records. No. 10/1986, 29 pp.

- **Sproston, N.G.** (1944)
  Ichthyosporidium hoferi (Plehn and Mulsow, 1911), an intertidal fungoid parasite of the mackerel. J. Mar. Biol. Assoc., 26:72-98.

- **Stevens, N.E.** (1936)
  Environmental conditions and the wasting disease of eel-grass. Science, 84:87-89.

- **Stevens, N.E.** (1939)
  Environmental factors and the wasting disease of eelgrass. Rhodora, 41:260-263.

- **Sykes, E.E. and Porter, D.** (1973)
  Nutritional studies of *Labyrinthula sp.*. Mycologia, 65:1302-1311.

- **Terawaki, T. and Iitsuka, T.** (1986)
  Development of techniques for marine macrophyte bed creation in the coastal zone near a power plant: 3. Effects of bed soil, planting number and light transmission on the growth of *Zostera marina*. Denryoku Chuo Kenkyusho Hokoku, 0(485030) i-iii, 1-15.

- **Thayer, G.W. and Phillips, R.C.** (1977)
  Importance of eelgrass beds in Puget Sound. Mar. Fisheries Rev. Paper 1271:18-22.

- **Thayer, G.W.** (1977)
  Seasonal distribution and changes in the nutritive quality of living, dead and detrital fractions of *Zostera marina* L.. J. Exp. Mar. Biol. Ecol., 30:109-127.

- **Tomlinson, P.B.** (1980)
  Leaf morphology and anatomy in seagrasses. In: Handbook of seagrass biology, ed.'s R.C. Phillips and C.P. McRoy. Garland Press, N.Y./London: 7-40.

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

- **Tutin, T.G.** (1942)
  Zostera L.. J. Ecol., 30:217-226.

- **Van den Hoek, C., Admiraal, W., Colijn, F. and De Jonge, V.N.** (1979)
  The role of algae and seagrasses in the ecosystem of the Wadden Sea, a review. In: Flora and vegetation of the Wadden Sea, ed. W.J. Wolff. Final Report of the section "Marine Botany" of the Wadden Sea Working Group, 3:9-118.

- **Van der Hoeven, P.C.T.** (1982)
  Observations of surface water temperature and salinity, State Office of Fishery Research (RIVO), 1860-1981. Scientific Rep. WR 82-8, Royal Dutch Meteorological Inst. (KNMI), De Bilt, the Netherlands, 118 pp..

- **Van der Werff, A.** (1934)
  Rapport van het onderzoek inzake de afsterving van het zeegras (*Zostera marina* L.) in de Waddenzee gedurende 1932 en 1933. Abcoude, the Netherlands, Feb. 1934, unpublished, 12 pp.. Archives State Office of Fishery Res. (RIVO), Ijmuiden, the Netherlands.

- **Van der Werff, A.** (1938)
  A new parasitic organism in *Zostera marina*. Chronica Bot., 4:498-9.

- **Van der Werff, A.** (1961)
  Over een tot dusver onbekend organisme in *Zostera marina* L. (written in 1936). "Uit het Peperhuis", Med. Rijksmuseum Het Zuiderzee museum, Enkhuizen, p.235-241.

- **Van Goor, A.C.J.** (1919)
  Het zeegras (*Zostera marina*L.) en zijn betekenis voor het leven der visschen. Rapp. Verh. Rijksinst. Visscherij, I(4):415-498.

- **Van Goor, A.C.J.** (1921)
  Die *Zostera*-Assoziation des holländischen Wattenmeeres. Trav. bot. néerl., 18:103-123.

- **Van Goor, A.C.J.** (1922)
  De halophyten en de submerse phanerogamen. In: Flora en fauna der Zuiderzee, p.'s 48-53.

- **Verhagen, J.H.G.** (1981)
  Een model voor de zeegrasgroei in het Grevelingenmeer. Waterloopkundig Lab./Delft Hydraulics Lab., Rep. no. R1310-9, 42 pp. + appendices.

- **Verhagen, J.H.G. and Nienhuis, P.H.** (1983)
  A simulation model of production, seasonal changes in biomass and distribution of eelgrass (*Zostera marina*) in Lake Grevelingen. Mar. Ecol. Prog. Ser., 10:187-195.

- **Vishniac, H.S.** (1955)
  The nutritional requirements of isolates of *Labyrinthula spp.*. J. Gen. Microbiol., 12:455-463.

- **Visser, M.** (1970)
  The turbidity of the southern North Sea. Deutschen Hydrographischen Zeitschrift, 23:97-117.

- **Weinberg, S.** (1976)
  Submarine daylight and ecology. Mar. Biol., 37:291-304.

- **Whelan, P.M.** (1986)
  The genus *Zostera* in Ireland. PhD thesis, Univ. College Cork, Ireland.

- **Whelan, P.M. and Cullinane, J.P.** (1985)
  The algal flora of a sublittoral *Zostera* bed in Ventry Bay, southwest Ireland. Aquat. Bot., 23:41-51.

- **Whelan, P.M. and Cullinane, J.P.** (1987)
  The occurrence of "wasting disease" of *Zostera marina* in Ireland in the 1930's. Aquat. Bot., 27:285-289.

- **Wiebe, P.H.** (1982)
  Rings of the Gulf Stream. Sci. Am., 246:50-60.

- **Wilson, D.P.** (1949)
  The decline of *Zostera marina* L. at Salcombe and its effects on the shore. J. mar. biol. Assoc. UK, 28:395-412.

- **Wium-Andersen, S. and Borum, J.** (1984)
  Biomass variation and autotrophic production of an epiphyte-macrophyte community in a coastal Danish area: I. Eelgrass (*Zostera marina* L.) biomass and nett production. Ophelia, 23:33-46.

- **Wohlenberg, E.** (1935)
  Beobachtungen über das Seegrass, *Zostera marina* L., und seine Erkrankung im nordfriesischen Wattenmeer. Beiträge zur Heimatforschung im Schleswig-Holstein, Hamburg und Lubeck, 2:1-19.

- **Wolff, W.J., De Koeyer, P., Sandee, A.J.J. and De Wolf, L.** (1967)
  De verspreiding van rotganzen in het Deltagebied in relatie tot de verspreiding van hun voedsel. Limosa, 40:163-174.

- **Young, E.L.** (1938)
  Labyrinthula on Pacific coast eel-grass. Canadian 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.