Observations On the Decline of the Water Milfoil and Other Aquatic Plants, Maryland, 1962-1967

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ABSTRACT

Eurasian watermilfoil (*Myriophyllum spicatum* L.), which infested an estimated 100,000 acres of Maryland's part of Chesapeake Bay and tributaries in 1963, was reduced to a very low level by 1967. Two pathological conditions, named Lake Venice disease and Northeast disease after the areas where they were first observed, are thought to be the cause, but the responsible pathogens have not yet been identified.

There was also a great reduction of weed beds formed by a number of other species, chiefly redhead grass (*Potamogeton perfoliatus* L.), wild celery (*Vallisneria americana* Michx.), coontail (*Ceratuphyllum demersum* L.) and curly-leaf pondweed (*Potamogeton crispus* L.) but the causes are unknown. A marked reduction of problems caused by sea lettuce (*Ulva lactuca L.*) may have been due, in part, to lowered salinity.

Eel grass (*Zostera marina* L.), which had been gradually increasing in a large area on the eastern side of the Bay, became very dense in June 1967, but by September, had decreased to a quarter of its earlier level. A pathological condition—not the same one that afflicted eel grass in the 1930's—seemed to be associated with the decrease.

INTRODUCTION

Submersed aquatic plants, growing in Chesapeake Bay and tidal tributaries, have, in the last decade or so, experienced an interesting history of advance and retreat. It is the purpose of this paper to record this history as accurately as memory, photographs, data books and various reports will allow. Causes, where known, and speculations, where not, will be discussed.

The most spectacular phenomenon occurred in eurasian watermilfoil which reached its maximum abundance in 1963, then began to decline slowly in 1964 and 1965 and rapidly in 1966. Two distinct diseases are thought to be responsible.

WATER MILFOIL

Eurasian watermilfoil (*Myriophyllum spicatum* L.) differs from *M. exalbescens* Fernald, which it most closely resembles, in that its leaflets are more closely spaced on the midrib and its leaves more widely spaced on the stem, but there is little chance of misidentifying the species in Maryland for *M. exalbescence* does not occur south of Pennsylvania (6). *M. spicatum* is native to the Eurasian continent but has been known from this country since the 1880's (1), perhaps having been brought over with ship's ballast. The oldest Maryland herbarium specimens in the National Museum are dated 1895 and 1902, both from the Gunpowder River.

Apparently the plant maintained a very low population level for many years. It does not appear in a list of plants compiled in 1910 (7), the only comprehensive botanical survey ever made of the state. Beginning in the late 1930's, observations of milfoil became more frequent, and we have a fairly continuous record since then. Springer and Stewart (9) reported on the spread of the species in Maryland thus: "The Eurasian species was first reported by Francis M. Uhlner in Nanjemoy Creek, Maryland (a tributary of the Potomac) about 20 years ago. In the Potomac estuary (including adjacent fish ponds) it has since spread north to Moss Point in Charles County, Maryland (1958), and south to Piney Point, St. Mary's County, Maryland (1959) and at least to Lower Machodoc Creek, Westmoreland County, Virginia (1956). Above the tidewater portion of the Potomac, it has been found also in the Cabin John and Seven Locks area of the Chesapeake and Ohio Canal (at least as early as 1945). In 1954 it was recorded from the Gunpowder River and in 1959 from Middle River, not far below the Flats. It also occurs in New Jersey and was observed in one of the freshwater impoundments of the Pea Island National Wildlife Refuge in North Carolina during 1959. Under suitable conditions it increases rapidly in an area and within about three years forms dense mats which interfere with navigation, compete with and choke out more desirable waterfowl food plants, smother oyster grounds, curtail fishing and crabbing, discourage swimming and provide conditions that are likely to be suitable for mosquito breeding."

Milfoil became a problem for the water-oriented public sometime in the mid 1950's. The earliest documentation of this that I can find is a letter in my files answering a man complaining about the milfoil in the Wiciomico River. The letter, dated August '57, indicates that problems also existed in Port Tobacco and Nanjemoy Creeks. All these are tributary to the Potomac River. By 1958, milfoil problems had become so widespread that a meeting of interested biologists and officials was called to discuss the situation, and, if possible, do something about it. This meeting was held in 1959 and was the first of five annual meetings on the subject (8).

A survey made in 1960 estimated that milfoil had spread over 50,000 acres with very dense growth on 10,000 acres (1). By late 1962, milfoil infested 100,000 acres in "the Chesapeake Bay Region" and in 1963 was "reaching up toward the 200,000-acre mark" (10).

By mid-summer of 1967 milfoil had shrunk to an estimated one percent of its 1963 tonnage (11). An apparent resurgence occurred in the late summer of 1967 which will be discussed in the section on Northeast disease.

LAKE VENICE DISEASE

In early September of 1962, a severe pathological condition was discovered in the milfoil growing in Lake Venice, a barrier-type pond of about 22 acres located in Anne Arundel County, Maryland, near Fort Smallwood (Figure 1).

I first visited the pond just before Labor Day in 1961. At that time the milfoil covered 100 percent of the surface and was flowering profusely—so much so that the pond appeared yellow. At my next visit, exactly one year later, milfoil covered perhaps 95 percent of the area, but all of it was very brown; it was not flowering nor did it show any evidence of having flowered earlier (no seed heads). In addition, a thick (one to two) inches), dark brown, foamy scum covered about a third of the pond on the downwind side.

In mid-summer of 1963, milfoil covered only about 20 percent of the surface but by Labor Day had increased to about 25 percent. There was no flowering. By mid-summer of 1964 coverage was down to about 10 percent. In 1965, mid-summer coverage was less than 10 percent, but by September had increased to 20 percent. My notes for 1966 say that on June 4, coverage was less than five percent but the plants appeared healthier than anytime since 1961. On June 30, however, coverage had increased to 50 percent but there was no flowering. On September 5, coverage was down to 25 percent with much brown scum on the water. In 1967, observations in June and September showed only a negligible amount of milfoil—coverage was perhaps about one tenth of one percent. Figure 2-a shows a badly diseased bed of about 20 percent coverage.

The pathological condition, which we call "Lake Venice disease," for obvious reasons, first appears as a light,

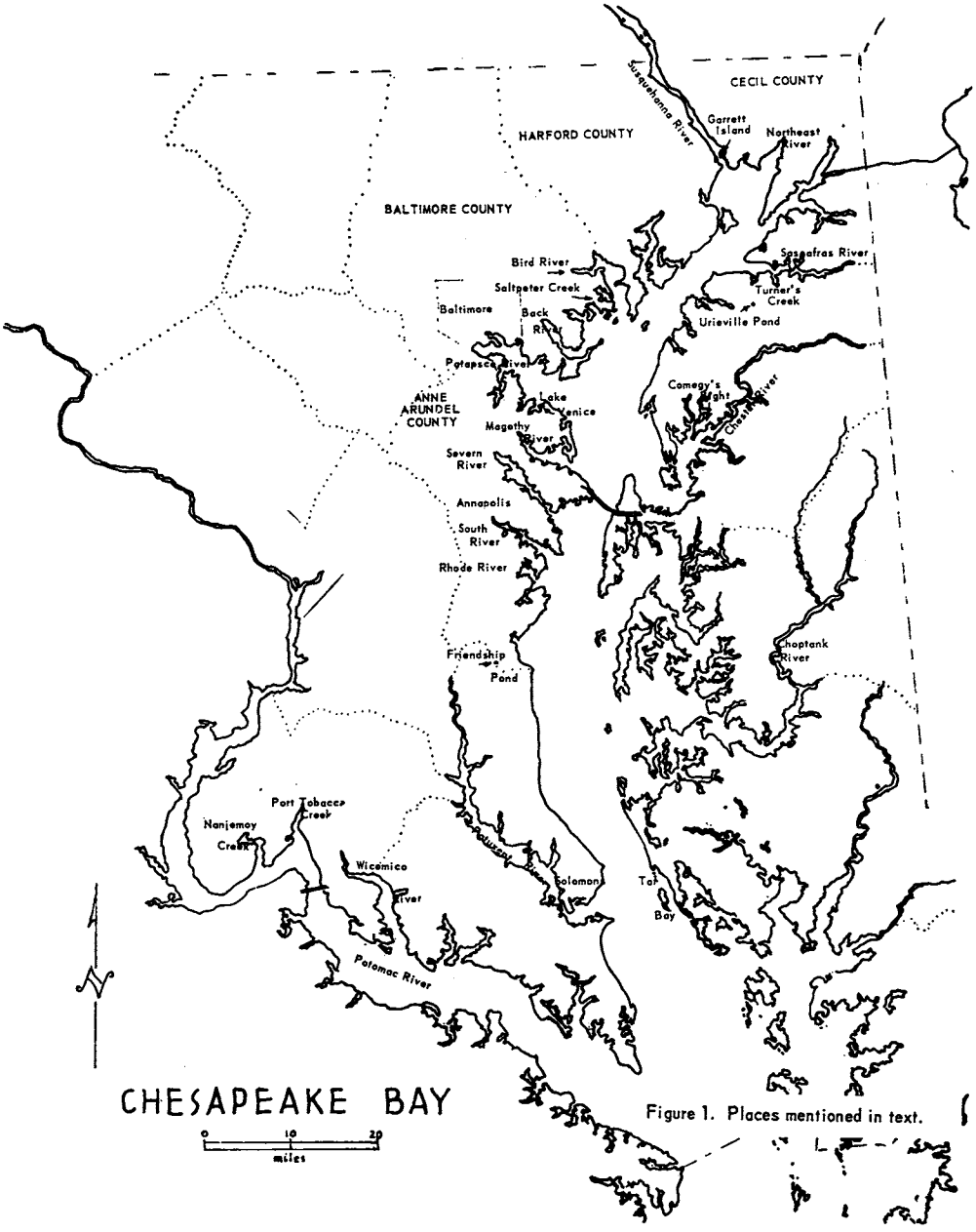


Figure 1. Places mentioned in text.

brownish coating on the leaves, as if silt had settled on the plant. This impression is strengthened if the plant is shaken in the water, for the coating tends to come off. As the disease progresses, this coating—an inelegant but more descriptive term is "crud"—becomes thicker until it entirely obscures the leaflets. The stem, too, becomes covered. The plant does not die immediately; rather, it slowly wastes away. It seldom flowers and when it does, flowering is very sparse. The dark brown scum first seen at Lake Venice has been seen in several other areas but only in those that are well protected from the wind.

Under a microscope Lake Venice-diseased plants show an amazing variety and quantity of diatoms, sessile protozoans, epiphytic algae, fungi, etc. One botany professor remarked that one could teach a course in microbiology with what could be found on one slide. Silt is visible on many specimens, but not to the degree expected from gross examination. The thickest "crud" appears to be an amorphous mass of brown slimy material. Figure 2 shows the disease in various stages. Figure 2-g shows a sprig of diseased milfoil laid out on a level surface to demonstrate its extreme flabbiness. Compare this picture with Figure 3-e and 4-d.

In June of 1964, using a large aquatic weed harvester, we collected and weighed a hopper load each of diseased and healthy milfoil from two adjacent stands in the Magothy River. The diseased load weighed 2,260 pounds, the healthy, 1,110. Both loads were allowed to drip about the same length of time. The difference seems to be due to the "crud" probably because it can hold more capillary water than the fluffier healthy plants.

In the summer following its discovery, 1963, the disease was not reported from anywhere except Lake Venice. It is probable that small stands were infected here and there but escaped notice. In 1964, however, the disease spread to all the Anne Arundel County Rivers, but was not reported from anywhere else in Maryland.

In 1965, Lake Venice disease was seen in all the milfoil rivers on the western side of the Bay, some of the tributaries of the Potomac and, on the east side of the Bay, the Sassafras River System. Healthy milfoil was still predominant in most rivers, even though my notes make very little mention of this (normal conditions are not likely to be recorded in field notes).

In 1966, healthy milfoil was so rare that it was recorded from only two places; one of these was a fresh water pond (Friendship Pond in southern Anne Arundel County), the other was an area in the Susquehanna River near Garrett Island. Not all of the diseased milfoil was afflicted with Lake Venice disease—about half the beds had Northeast disease, while a few had both maladies.

In 1967, no healthy milfoil was seen anywhere in Maryland; and most beds showed symptoms of both diseases.

NORTHEAST DISEASE

Early in June 1964, two biologists investigating a report of pollution near the Northeast River noticed an abnormal condition in the milfoil of the area. They found no popution, so speculated that the plants might be affected by something else. At first it was assumed they had found some previously overlooked symptoms of the disease we had been observing for two years, but the plants with the new symptoms looked somewhat different and for a while the situation was confusing. However, things began to clear up, for my field notes for July 3, say this: "Beginning to suspect we have two diseases operating. The silty stuff we might call 'Venice Vapors', the other we might call 'Northeast Disease.' "

The earliest symptom[1] that can be seen with the naked eye is broken leaflets. Very often it appears as if some creature had taken a bite out of the leaf. Because this symptom is so obscure, it seems quite possible that the disease may have appeared earlier than 1964 but went unnoticed. As the disease progresses, the stem and leaves turn a dark, brownish green and become stiff. Usually, the lower leaves begin to drop off and soon all the leaves are gone, leaving only a bare, stiff, blackened stem. These too, then disappear, probably by sinking to the bottom. The stems are not always killed as far down as the roots, for often new growth will shoot up from the lowest nodes—sometimes even before the old plant material is gone. This new growth at first appears healthy but in a week or so shows symptoms of the disease. Another symptom, which seems to occur when the plant does not die quickly (or perhaps is more resistant), is gross distortion of the petioles. In this case, the upper leaves are affected more than the lower ones, and are generally less than half the size of a healthy leaf. These symptoms are pictured in Figure 3. Perhaps the best field identification method is to lay a branch of milfoil on a level surface. If the weight of the plant can be supported on the ends of the leaves, as shown in Figure 3-e, there is no doubt the plant has Northeast disease. As in the Lake Venice disease, flowering is extremely sparse or absent.

Under a microscope, diseased plants, with an exception to be noted later, appear to be free of epiphytic organisms. However, dark brown or black spots can be seen on the stems which we suspect is the very earliest visible symptom. They differ from other spots that are sometimes seen in that the boundaries of the spots follow the cell walls. Thus each spot has edges consisting of straightlines and 90 degree angles (epidermal cells are cylindrical). Some outline a single cell, most are of a group of cells.

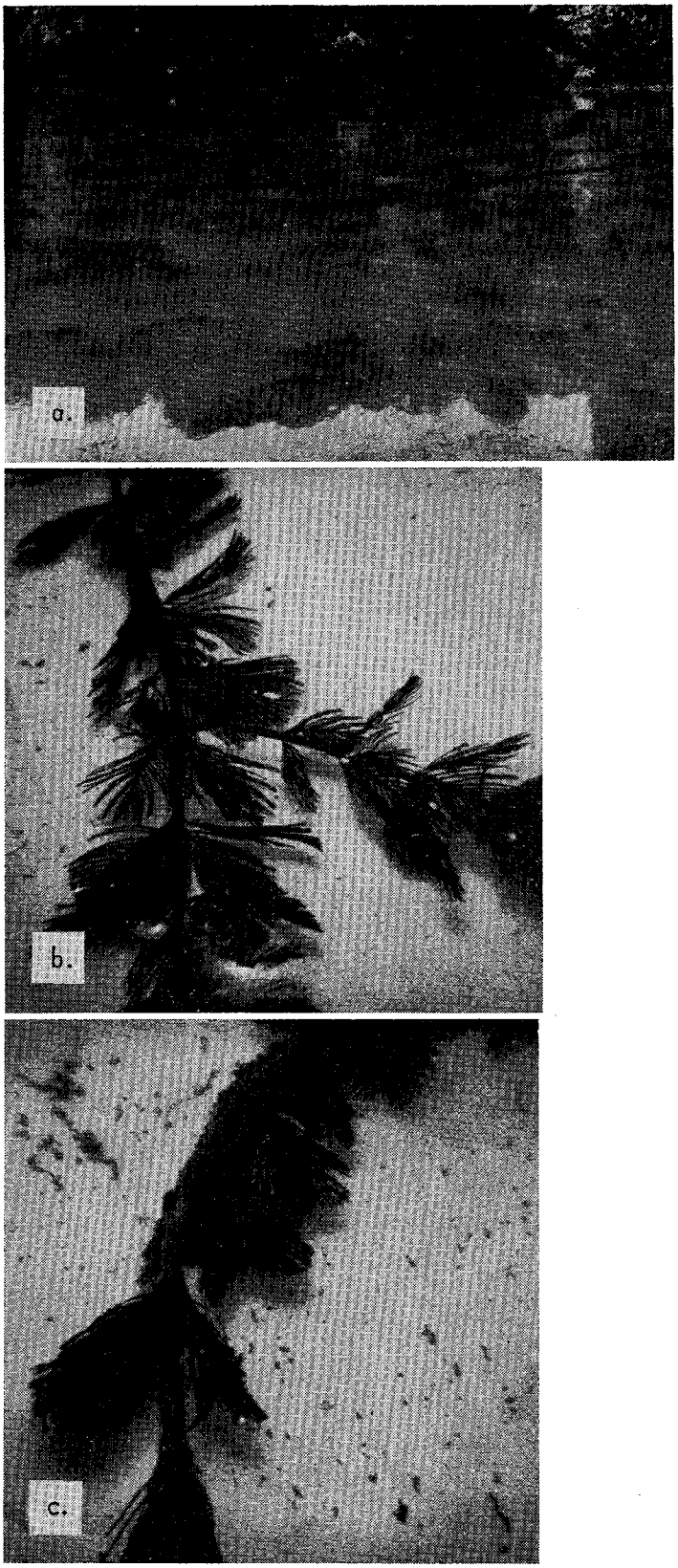
Perhaps the most dramatic of the effects of Northeast disease occurred in Saltpeter Creek in June 1964. When we worked in the area in late April there was a lush stand of milfoil almost reaching the surface. Two months later, on June 26, there were only dead, black stems to be seen. These stood rigidly out of the water about four inches. The stems were so stiff that a piece 12 inches long held only at the bottom would not bend over. These stems collected on our outboard and when the motor was lifted the propeller gave the impression of a rotary brush—stiff bristles radiating from a hub. On this same day, in shallow water, we could see new growth about three inches long arising from the lowest part of the stems.

By August 7, the creek was again filled with a lush growth of milfoil, but the plants showed definite symptoms of Northeast disease.

In 1964, Northeast disease was seen only in the rivers of Baltimore and Harford Counties and in the Northeast River in Cecil County. In 1965, it had about the same distribution but the infected beds seemed thinner and there was almost no flowering.

In 1966, Northeast disease was observed almost every-

[1]Sometimes herbicides, especially 2,4-D, have been suspected as the cause of Northeast symptoms. However, light doses of herbicide result in a turningback of the terminal leaves as shown in Figure 4-e. This is very different from Northeast symptoms.



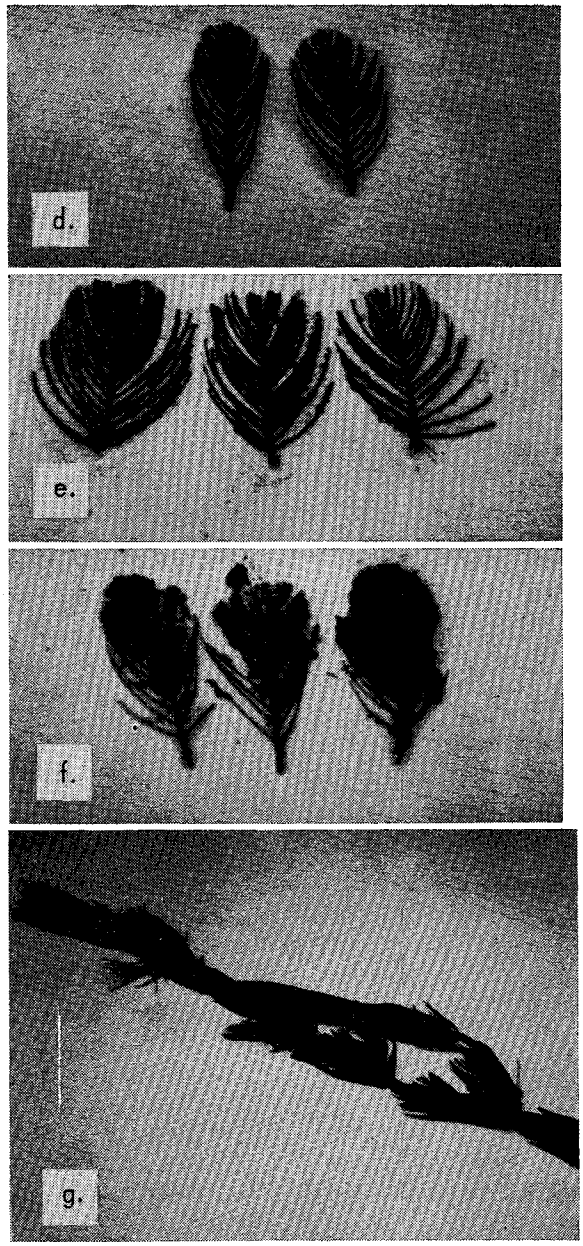
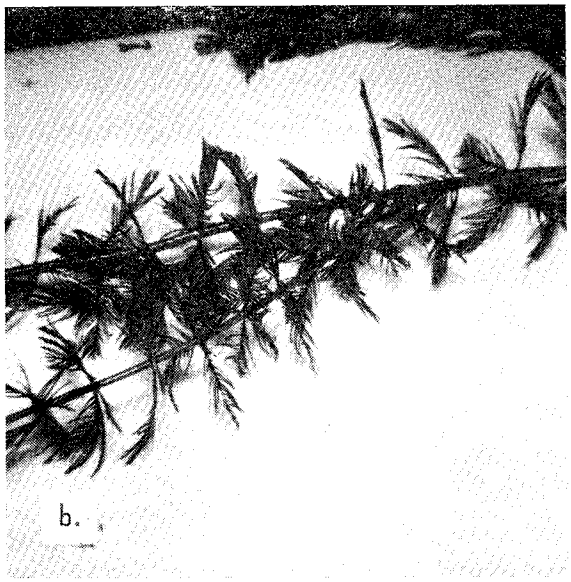


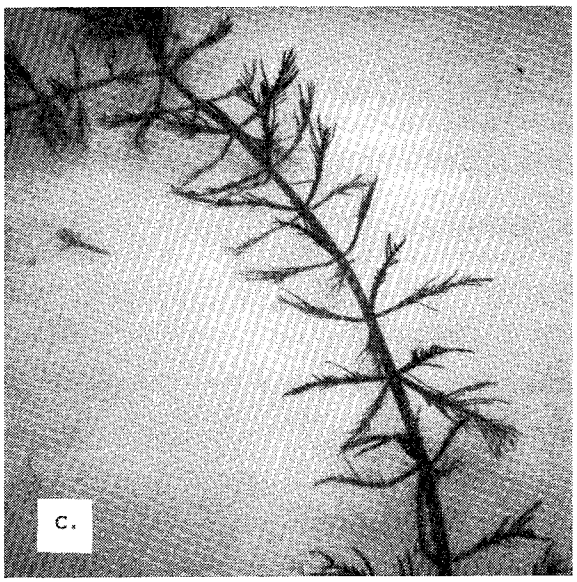
Figure 2. Lake Venice disease in milfoil.

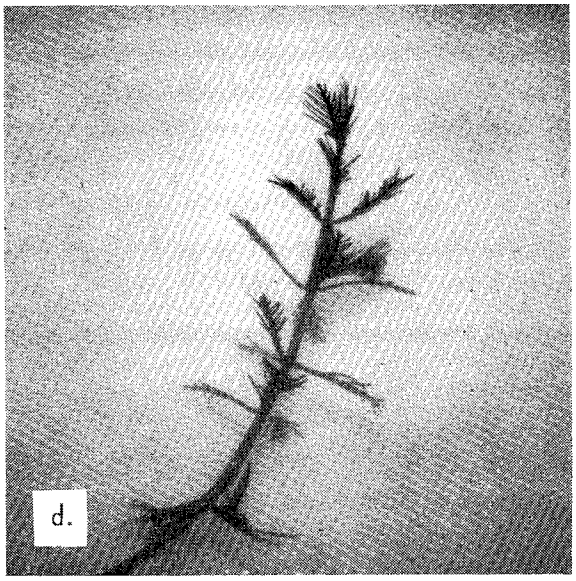
a. Overall aspect. Compare with Fig. 4-a
b. Intermediate stage. Leaflets brown but not obscured.
c. Advanced stage. Leaflets obscured.
d. Intermediate stage.
e. Intermediate stage.
f. Advanced stage.
g. Advanced stage. Laid out to demonestrate extreme flabiness. Compare with Fig. 3-e.

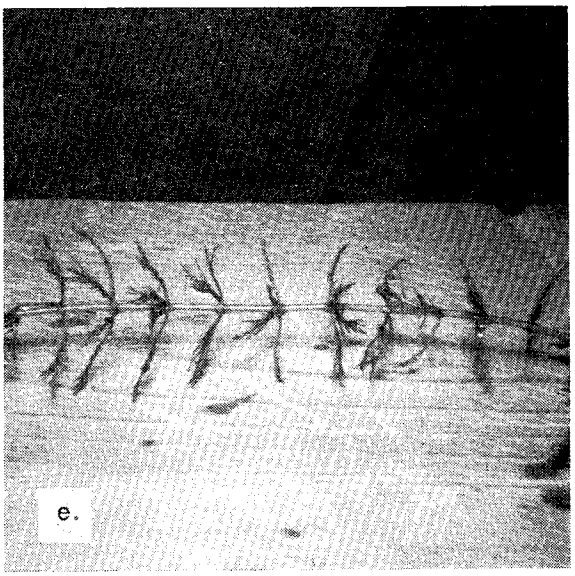
where in Maryland tidewater and the beds infected with it seemed to be about half as big as they were in 1965. Moreover, these stands were very thin—containing perhaps a quarter of their normal tonnage. The same reduction in volume could be seen in the beds infected with Lake Venice disease.

In the last week of July 1966, there was a sudden and rapid resurgence of milfoil. It was estimated that new stems, usually originating from the tips of the plants or a bud near the tip, grew as much as 12 inches in three days. They flowered immediately and had all the characteristics of healthy plants. However, in ten days to two weeks, symptoms of either diseases were evident. Only about half of the observed milfoil stands showed this sudden growth.









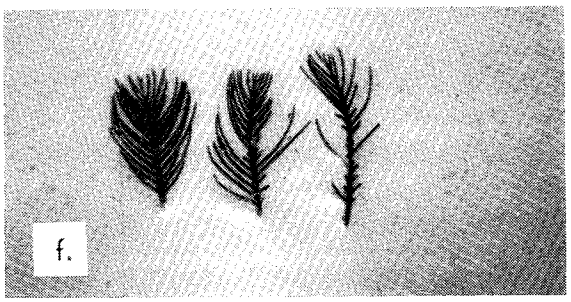


Figure 3. Northeast disease in milfoil.

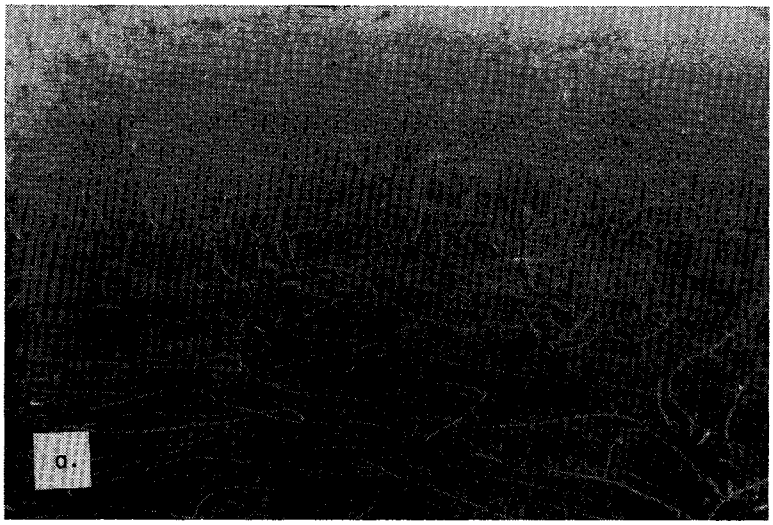
a. Early stage. Leaves not very stiff, few broken leaflets.
b. Intermediate stage. Leaves stiff, broken leaflets numerous
c. Advanced stage Leaves stiff, few unbroken leaflets, distorted petioles common.
d. Advanced stage, terminal portion.
e. Advanced stage. Laid out to demonstrate leaf stiffness. Compare with Figs. 2-g and 4-d.
f. Leaves typical of three stages.

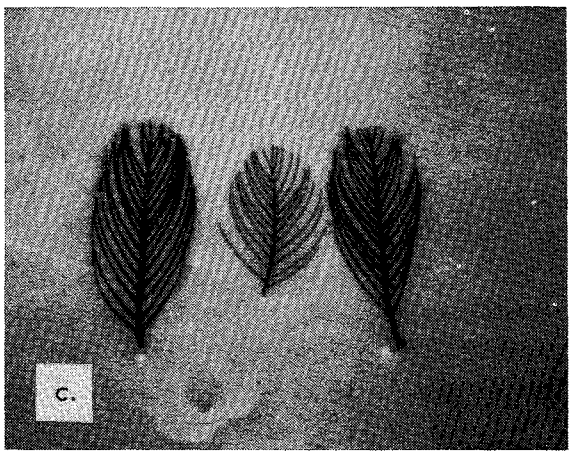
Figure 4-a shows one of these revitalized beds. The picture was taken August 3, 1966—a week earlier, it resembled the bed shown in Figure 2-a. In 1967, this bed was entirely gone.

The first observation of both diseases in the same stand came in 1966, in Turners Creek, a tributary of the Sassafras River. Upon closer inspection, a number of individual plants seemed to have symptoms of both diseases. Their leaves were small and stiff, had distorted petioles and were covered with a brown coating. This doubly diseased condition was highly unusual in 1966, but was the rule in 1967.

Estimates early in the summer of 1967 placed the total tonnage of milfoil in Maryland tidewater at less than one percent of the 1963 level. Many, if not most, of the stands had disappeared, and those that were left were very thin. A resurgence like that of the previous year occurred in the uppermost part of the Bay, but not until September. These revitalized beds later became diseased.

It is probably unsafe to ascribe this great decline of milfoil entirely to the two diseases, as a third phenomenon appeared which so complicated the situation that it is now almost impossible to separate their effects.





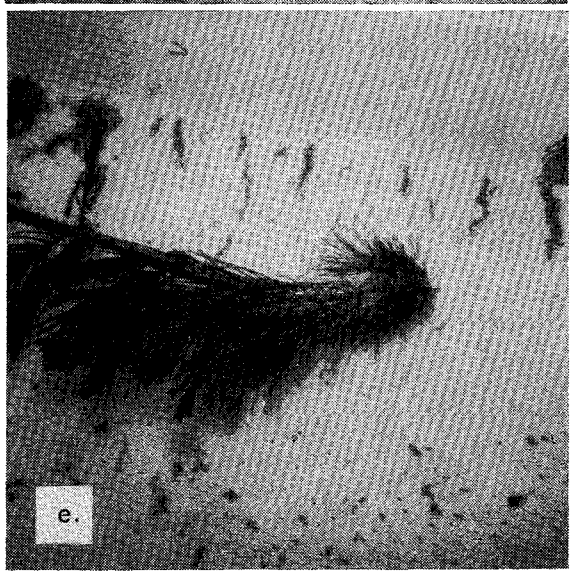


Figure 4. Healthy milfoil.

a. Overall aspect. Note numerous flowers. Compare with Fig. 2-a.
b. Terminal portion of plant.
c. Typical leaves.
d. Laid out to de monstrate moderate flabbiness.
e. Effects of light dose of herbicide. Note that terminal leaves are turned back on stems. Compare with b. above.

RHODE RIVER EVANESCENCE

This third phenomenon I call the "Rhode River Evanescence" because that is where the first of a number of mysterious disappearances, or partial disappearances of stands of other species was observed. There is no certainty that these disappearances are due to similar causes; all we know is that the results are similar.

1. The Rhode River formerly supported a rather heavy weed load, dominated, in 1964 at least, by milfoil. There were also large beds of redhead grass (*Potamogeton perfoliatus* L.) and some smaller beds of elodea (*Elodea canadensis* Michx.). Appearing as subdominant species were sago pondweed (*Potamogeton pectinatus* L.), widgeon grass (*Ruppia maritima* L.) and horned pondweed (*Zanichellia palustris* Mich.); all five species could often be found in the same bed.

In 1965, all stands of submersed aquatics seemed to be developing normally until mid-June when they almost reached the surface. Then they began a slow decline and by mid-August had disappeared from much of the river. No symptoms, except for Lake Venice disease in milfoil, were noticed. In 1966, coverage was perhaps ten percent of its previous level, and in 1967 only a few, very small and thin beds of redhead grass could be found.

2. The Sassafras River and tributaries, in early 1960's, contained extensive stands of aquatic vegetation. These beds were dominated by milfoil, but had a heavy admixture of coontail (*Ceratophyllum demersum* L.) wild celery (*Vallisneria americana* Michx.), curly leaf pondweed (*Potamogeton crispus* L.), bushy pondweed (*Najas flexilis* Willd) and elodea. In 1966, vegetation did not appear in the upper parts of any of the tributaries or from the shore out to a distance of 20 to 50 feet. This left only the mouths and the lower central areas occupied with submersed vegetation. Previously, the weed beds occupied all of the areas including the inter-tidal zones, but now we found we could travel unimpeded with our outboards in the weed-free zone adjacent to the shores and anywhere in the upper parts of the tributaries. In 1967, the few weed beds to be found were very thin and much smaller than the year before. Wild celery and coontail became much more evident as the milfoil disappeared, but still did not seem to be as abundant as when they were subdominant.

Infestations of water chestnut (*Trapa natans* L.) in these tributaries did not seem to be affected.

3. Comegy's Bight is a deeply indented bay on the north side of the Chester River. Its weed load was heavy and consisted mostly of milfoil and redhead grass. In the first week of August 1966, many tons of milfoil stems collected on a beach at the northernmost shore of the bight, creating a severe problem for a local resident. There was far more fragmented milfoil on the beach than could be accounted for by boat traffic. Most of the fragments appeared to be in early stages of Northeast disease, but I have never seen diseased milfoil break up to such an extent.

4. The South, Severn and Magothy Rivers in Anne Arundel County, had, until 1966, weed beds formed of the same species as in the Rhode River, except that redhead grass was much more prominent, occupying perhaps as much area as the milfoil. In late August 1966, almost all the weed beds in the South River disappearad. This was not called to my attention until after it happened so I have no observations on possible symptoms.

5. In mid-September, there appeared in the Severn River what I believe to be an earlier stage of the same phenomenon. At this time there were no aquatic weeds in any of the Severn tributaries, although formerly they were extremely abundant in these places. In the main river there were extensive beds of redhead grass which were holding huge rafts of floating redhead in place. Many of the floating plants had roots attached. Apparently there had been little wind during the previous week or this mass of fragmented plants probably would have piled up on shore and created problems. Neither milfoil nor elodea were seen anywhere in the river.

6. Also in mid-September 1966, what appeared to be a still earlier stage of the phenomenon appeared in the Magothy River. Here the tributaries had weed beds but they were much thinner and smaller than usual. In the main river, there were beds of redhead grass holding rafts of plant fragments, but these rafts were not as large nor as dense as those in the Severn.

In 1967 there were only a few small beds of milfoil and redhead grass in any of the Anne Arundel rivers.

7. The Bird River, in 1965, had very large beds of milfoil with wild celery and coontail subdominant. In 1966, milfoil retreated and wild celery and coontail occupied the vacated areas. In 1967, the area was practically bare; only a few, small, very sparse beds of these plants could be found.

8. Urieville Pond in Kent County was so choked with weeds, mostly milfoil, from 1963 through 1966, that fishing was impossible after the first of June. In 1967, no submersed weed beds of any kind appeared. Even a small stand of spatterdock (*Nuphar luteum*), which had been growing near the dam for five or six years, failed to appear. This pond has a dam about eight feet high, so there is no exchange of water with the Bay.

In all these disappearances, no warning symptoms were observed. Where the vegetation was under close observation, as in the Rhode River, the plants, except for milfoil, appeared perfectly normal. Of course, had I suspected they would disappear, I would have looked harder for symptoms. Where the plants simply didn't appear in the spring, as in Urieville Pond, there was nothing to indicate anything was wrong, again, except for milfoil.

If these phenomena have a common cause, it is unknown. There has been some speculation that rapid changes in salinity might be a factor, for the Bay had been getting progressively more salty from 1962 through 1966. However, milfoil, redhead grass and widgeon grass are known to tolerate higher salinities than they experienced in any of these rivers. Besides, salinities stayed low in the upper part of the Bay, and Urieville Pond is entirely fresh water. Carp has been suggested as a factor, but it seems that if these fish had suddenly become extremely abundant, their population increase would have been noticed. Reduced light, due to plankton or silt turbidity, has been considered as a possible cause, but to produce the observd effects, the turbidity would have to be to such an extreme degree over such a protracted period that it would be readily apparent upon the most casual observation. Disease could be a factor, but it seems unlikely that a single disease could affect so many species. There could be more than one disease, of course, but it seems that two concurrent diseases in milfoil is about all the laws of probability will allow. Pollution can be ruled out because, although the

Anne Arundel and Baltimore County rivers receive some pollution, the Sassafrass River remains comparatively clean. Whatever the cause, it must be something that can affect isolated ponds as well as tidewater.

Eutrophication is almost certainly a factor in this situation, but theoretically at least, eutrophication creates favorable rather than adverse conditions for plant growth. Casual observation suggests that plankton blooms have been increasing in the last few years, and filamentous algae has been approaching nuisance abundance in some places—which argues for eutrophication. On the other hand, Ulva, which is reputed to be one of the best indicators of enrichment, has become less abundant.

ULVA

Sea lettuce (*Ulva lactuca* L.) is a marine, thalliform, green algae which at times becomes quite abundant in Chesapeake Bay. It creates severe local problems when it piles up on beaches and rots. In the process of decay, hydrogen sulphide is produced which can be so concentrated as to discolor paint on water-front houses, tarnish silverware and copper kettles and blacken copper paint on boat bottoms.

Although sea lettuce problems have been known in Maryland for any years, they were few and far between and seemed to be confined to the Bay south of the Choptank River. However, starting about 1962, there was a gradual increase in the number of problems as evidenced by the number of complaints from water-front residents. There was also an increase in the number of residents which would account for part of the increased complaints. I did not begin to record these problems until 1965 when 34 were reported (I inspected 28 of these). In 1966, only 13 complaints came in and in 1967, only four, one of which turned out to more imagination than problem. It is likely that many problems were not reported but I believe the trend of complaints parallels that of the problems.

There was also an expansion and reduction in the range of these problems. The northernmost occurred in 1965 near the mouth of Back River in Baltimore County. In 1966, the northernmost was in the Magothy River, and in 1967 in the South River. One hypothesis is that the advance and retreat of Ulva was due to changes in salinity, as the Bay gradually became saltier, starting in 1962, and then started a freshening trend in 1966. However, the peak period for salinity was late fall of 1966, more than a year after Ulva had begun its retreat.

EELGRASS

Eelgrass (*Zostera marina* L.) is a marine angiosperm which can thrive in salinities down to about 12 parts per thousand. At one time, there were extensive beds along the Atlantic seacoast in both North America and Europe, but in 1930-33, a malady, which came to be known as "wasting disease," attacked the plant over its entire Atlantic range. Causes of the disease were never satisfactorily determined, but a fungus and a bacterium were under suspicion (14). By 1933 there was little eelgrass to be found anywhere, but a slow recovery began and it now has become a problem in some places (13). In Chesapeake Bay, its disappearance apparently was not as spectacular as along the coast, for Cottam (2) wrote: ". . . there are local sections, such as estuaries and the mouths of some of the rivers, in which sea-water is markedly diluted by fresh water, where there still exist dense stands of apparently normal and healthy eelgrass. Th upper Chesapeake south of Baltimore and Annapolis is an example. Only time can tell whether these areas will be affected."

Sometime before World War II there was an attempt by the U. S. Fish and Wildlife Service botanists to plant eelgrass (6) in Chesapeake and Chincoteague Bays. Some of the planting stock came from the west coast, but most was from the few halthy stands that could be found in the area. Apparently, these attempts resulted in failure.

In the late 1940's, when I first came to Chesapeake Bay, there was a small stand of apparently healthy eelgrass at Solomons and another larger stand near Tar Bay. There must have been others, of course, but there seems to be no way to document this. As far as I know, these beds have flourished continously since then.

Early in July 1967, I received a report that the eelgrass in Tar Bay had "all turned white." When I visited the area on July 6, I found an extensive and extremely dense stand of eelgrass, so large it must have occupied several thousand acres. Widgeon grass was mixed with it as a subdominant. Most of the eelgrass appeared normal but there were many small patches in which the leaves lying on the surface were a silvery whitish color, and from a distance, the area looked mottled. There was also a large amount of dead eelgrass leaves washed up on shore—somewhat more than seemed normal.

During the rest of the month there was little change in the pattern of whitish patches, but there was a large increase in the amount of dead plant material on shore. This was rotting in many places and producing hydrogen sulphide. My field notes for July 29 say this: "A great deal of eelgrass washed up on shore. Much paint damage to many houses. Washed-up eelgrass has patches that are dull rose in color. Other patches (drier) are white. Most of material is green or brown. Very little widgeon grass on shore." I assumed that the dull rose and white patches on the beached material was due to various bacteria.

During August, eelgrass became less dominant and widgeon grass more. By mid-September, widgeon grass was the dominant plant and eelgrass subdominant. There were only a few small areas where eelgrass leaves were floating at the surface; most of it was lying on the bottom.

Close examination of the whitish leaves revealed that the lack of color was due to an absence of chlorophyll. Typically, the leaves are completely chlorotic and very pale at the tip. Proceeding down the leaf, this whitishness graded imperceptibly to tan, then to brown, then to greenish brown and finally to a healthy green color near the base. Many of the leaves were broken but this did not seem to be associated with the pathological condition, for most of the chlorotic leaves still had their original tips. There were no spots as has been described for wasting disease, nor did any of the leaves appear wilted. Leaves dredged from the bottom in September were dark brown and dead, like those washed up on shore.

Perhaps the best description of wasting disease symptoms is: ". . . Those plants most severely attacked appeared wilted and contained little chlorophyll. The entire plant seemed to be affected and disintegrating. More commonly the disease begins as a grayish brown spot or a lesion on the margin of the leaf. The outermost leaves and sheaths are usually affected. Each leaf affected turns brown, dies

and decays. Frequently, the death and disintegration is progressive from tip to base so that it is not uncommon to see leaves with the proximal portions still green and apparently healthy while their distal portions are entirely missing. Often a number of diseased areas are noted on the same leaf, and frequently the affected portion follows a considerable space down one margin or larger vein before extending across the leaf" (2).

It seems that the symptoms noted in Tar Bay are not quite the same as those of the wasting disease of the 1930's. This does not necessarily mean that we have a different disease, for conceivably, different strains of plants might react differently to the same pathogen.

DISCUSSION

All the phenomena described in this report may be related, that is, have a common cause. However, there is such a wide variety of species and environments involved that a common cause seems very unlikely. On the other hand, it seems equally unlikely that such a long list of disappearances and retreats could occur in such a short time and not be related. Perhaps Chesapeake Bay is undergoing, or about to undergo, a violent change of some sort. One can accept an epidemic or a disappearance of a single species without suspecting a severe disturbance in the ecology, because these things are normal in nature. It is when too many of them crowd into a relatively short space of time that one begins to feel uncomfortable with the overall picture.

Unfortunately, no one is at present making a serious investigation of these phenomena—with one notable exception.

This exception is the work being done at Johns Hopkins by Miss Suzanne Bayley, a graduate student working under Dr. Charles H. Southwick of the Department of Pathobiology.[2] In less than a year, she has ben able to determine that the pathogen of Northeast disease is a filterable agent, probably a virus. At this writing (December 1967), a few more steps remain before certainty is reached. It is expected that Miss Bayley will soon submit a paper on her very interesting work.

The future of submersed aquatics in Maryland tidewater is uncertain. The trend of milfoil abundance is definitely downward but a disease-resistant strain could develop at anytime which would reverse the trend. No predictions or even guesses can be made about the trend in other species because the factors involved are completely unknown. We shall have to wait until much more data is available.

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