RECENT INVESTIGATION ON THE EEL-GRASS PROBLEM: PRELIMINARY REPORT

EDWARD LORRAINE YOUNG, III
Harvard Medical School

During the past year several new facts have thrown light on the study of the labyrinthulan parasitism of the eel-grass, *Zostera marina*. To account for the behavior of the epidemic has been the most perplexing part of the problem. The disease struck suddenly and almost universally on both shores of the Atlantic Ocean in 1929-30 causing great wastage. But it was early noticed that the severity of infection, rate of wasting, degree of persistence of grass, and type of recovery varied locally (1). In exposed areas the grass was nearly wiped out; in more sheltered regions it showed more resistance and persistence.

A universal shift of some one or more environmental factors upsetting the physiological balance of *Zostera* or of its parasite, *Labyrinthula,* has seemed the most logical explanation both for the onset of the epidemic and for the local fluctuations in its virulence. However, attempts to correlate any such factors with the existing conditions have to date failed. Three of the most likely of these factors have been studied; pH, temperature, and salinity; and data of their effects on both the grass and the parasite have been collected. Since the coastal waters have not fluctuated beyond their normal pH range of 8.1 to 8.4 over the period in question this factor has been eliminated completely.

The effect of shifts of either temperature or salinity on *Zostera* seem to be nil. Dr. Prytherch has found dense beds of healthy grass in Milford Harbor, Conn. thriving in temperatures of minus 10 degrees centigrade to 35 degrees centigrade and in salinity changes from fresh water to 30 per mille.

Recent investigations on the physiology of *Labyrinthula,* however, have shown that certain environmental factors may alter its activity. The effects of temperature change are negligible as the parasite is healthy and active in a range between 3 degrees and 30 degrees centigrade (2). The parasite also grows in salinities ranging from pure fresh water to those of double the normal concentration (2). But the significant point here is that its apparent physiological activity is weakened. With a decrease in salinity the spindles or cell bodies lose their color; the tracks on which they glide progressively weaken; and the bond holding the spindles to the track decreases in strength. These changes are not marked as salinity increases until the higher concentrations are reached. This would seem to indicate that the range of optimum activity for the parasite is from just below the normal salinity of coastal waters to quite a bit above it (say from 15 to 22% chlorinity). From this, it follows that if the % chlorinity of any area drops below 15 the virulence of *Labyrinthula* will be reduced while *Zostera* will remain unaffected.

A study of hydrographic data tends to support this theory. Several sets of figures on the chlorinity of two sheltered eel-grass beds were obtained from the Mt. Desert Island region during July and August 1937. The most striking figures were those from Northeast Branch. This outlet drained a large fresh water pond. At its mouth, a distance of some four or five hundred yards was inundated at high tide with salt water. In this area there was a large dense bed of *Zostera marina*. During 1935 and 1936 it had been observed to be of large extent and extremely healthy during June and July, but almost completely blighted by late August. This chain of events was more pronounced in 1937. The water from the open bay of that region during 1937 had a chlorinity of 16.8 to 18.2% during July; but by August 27 it had risen to 20.1.

The first samples of water were taken from this grass bed in the early part of July. At low tide the % chlorinity was 5.5; at high tide 13.5. The grass, from gross examination, was practically unparasitized as were both *Ruppia* (reported parasitized in 1936 (2)), and various green algae growing nearby. Samples were taken again on August 27 when both the *Zostera* and the *Ruppia* and several algae were badly infected; the former two being nearly wiped out. At this date the % chlorinity had risen to 19.9 at low tide and 18.9 at high tide.

These figures and the degree of wastage at the corresponding period check closely with the observed activity of *Labyrinthula* at these chlorinity concentrations. The rapid increase in % chlorinity is easily accounted for by the exceedingly dry weeks of July and August.

A second though less complete set of figures was obtained from a similar river mouth at the head of Somes Sound. Here, however, only a small flow of fresh water emptied through an area some eight or nine hundred yards long which was inundated with salt water at high tide. The dense *Zostera* bed was unaccompanied by *Ruppia* or the green algae found in the previous region. On August 21 the chlorinity of the water in the open sound was 18.4 while that at the upper reaches of the eel-grass beds was 10.7 at low tide and 16.4 at high. Wastage was negligible.

The obvious possibility that temperature increase might be a concomitant factor in this late season wastage has not been disregarded, but it is considered but a secondary factor at best. Unfortunately absolute figures are not available, but the temperature of all coastal waters around Mt. Desert Island ran from 2 to 5 degrees higher throughout the summer of 1937 than in 1936, i.e., an increase from early July to late August of from around 12 to 17 degrees centigrade in 1936 as compared with 14 to 20 degrees centigrade in 1937. If temperature was a primary factor in wastage the beds of grass should have been badly diseased by mid-July in 1937 rather than, as usual, late August.

This theory of the effect of salinity on the wastage received unexpected support from conditions in Naniamo Bay, B.C., last summer. In July parasitism of west coast eel-grass was first conclusively

demonstrated in grass from that region collected in July 1937 (3). On consulting the hydrographic records it was found that the mean ‰ chlorinity had been increasing steadily since 1934—an increase from June through December 1934 to January through May 1937 of 13.90 to 15.70. Over the same period the temperature change was negligible.

These figures as reported do not attempt to account for the suddenness of onset and the universality of the disease, but they do indicate a possible explanation for the seemingly haphazard local variations so far observed throughout the diseased areas. Unfortunately the possible significance of this line of investigation was not realized in time to make more than a beginning. But the need for a more careful study of this relationship between chlorinity and the virulence of the disease is indicated.

REFERENCES

1. Renn, C. E., 1935, The wasting disease of eel-grass. Ph.D. Thesis, Rutgers University, New Brunswick, N.J.
2. Young, E. L., 1937, Notes on the Labyrinthulan parasite of the eel-grass, *Zostera marina*. *Bull. Mt. Desert Is. Biol. Lab.*, p. 33.
3. ———, 1937, *Labyrinthula* on Pacific coast eel-grass. *Canadian J. Res.* (in press).

THE EXCRETION OF URIC ACID BY THE CHICKEN

JAMES A. SHANNON
Department of Physiology, New York University College of Medicine

In view of the metabolic importance of uric acid in the birds, considerable interest attaches to the mechanism of its excretion. The available evidence shows that this is effected in part by glomerular filtration and in part by tubular excretion, but no observations have been made on normal, unanesthetized birds, or at plasma concentrations above the normal level. The present investigation is intended to examine the process of tubular excretion in greater detail. Methods will be described in a full report to be presented elsewhere.

The uric acid/inulin clearance ratio in the chicken at normal plasma uric acid concentrations is 7.5 to 15.8. As the plasma level is raised this ratio is depressed (due to a reduction in the uric acid clearance) until at plasma levels of 100 mgm. per cent it has a value of 1.8 to 3.2. These facts are confirmatory evidence of the tubular excretion of uric acid in the chicken and show that this process accounts for 87 to 93 per cent of the total uric acid excreted at normal plasma levels. The rate of tubular excretion of uric acid increases with increasing plasma level until it reaches a maximum value at 20 to 40 mgm. per cent plasma uric acid. At normal plasma uric acid levels the rate of tubular excretion of uric acid is more than 50 per cent of the maximum rate.

The development of a highly efficient tubular mechanism for the