calculation indicates that the peat bed has been compressed to between one-half and one-seventh of its original thickness, with the deeper and older samples showing greater compaction.

The details of the evaluation of compressed samples will be considered elsewhere, but it may be stated that their depths and ages do not contradict the evidence from the uncompacted samples. The radiocarbon dates and general stratigraphic relationships demonstrate that submergence has been continuous on the Connecticut coast for at least 7000 years and probably for over 11,000 years, with no evidence of pauses or reversals in the submergent trend. From 7000 to 3000 years ago submergence was at the rate of 0.6 foot per century; during the last 3000 years the rate has been only half as great. It is significant that not one of several hundred borings in Connecticut tidal marshes showed more than 10 feet of salt-marsh peat overlying bay mud. Apparently, sediment accumulation and salt-marsh growth have been able to keep pace with submergence only during the last 3000 years; prior to that time the more rapid submergence maintained open, although shallow, estuaries and lagoons on the sites of the present salt marshes.

It must be emphasized that Fig. 1 shows submergence, the relative movement of land and sea level. The cause of the submergence remains to be evaluated. However, the close correspondence of the curve in Fig. 1 with similar curves published for the northwestern Gulf of Mexico (*2*) and for the Netherlands (*3*) supports the hypothesis that the submergence of the last 7000 years is the result of a worldwide, or eustatic, rise of sea level. Contradictory evidence of a stillstand of the sea for the last 3000 years in southwestern Louisiana (*4*) and for the last 5000 years in southern Louisiana (*5*) may be due to unrecognized variables such as compaction, local tectonic deformation, and reworking of old shells into more recent beach ridges (*6*).

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5 November 1962

Biochemical Tests Indicative of Reaction of Castor Bean to Botrytis

Abstract. *Pectic and cellulolytic enzymes caused browning and maceration of capsules of susceptible varieties of castor bean but not of resistant varieties. Results of tests in which pericarp tissue of the capsule was treated with vanillin and H_2SO_4 and with an aqueous solution of $FeCl_3$ and $K_3Fe(CN)_6$ showed that resistance to the hydrolytic enzymes was correlated with both a low concentration of flavanols and related compounds and the presence of oxidized compounds in injured tissue. Resistance appeared to be due to inactivation of pectic, cellulolytic, and other hydrolytic enzymes by oxidation products of phenolic compounds. The biochemical tests made rapid measurement of resistance possible.*

Histological studies of castor bean capsules (*1*) failed to show stomatal or cuticular penetration by germ tubes of spores of *Botryotinia* (*Botrytis*) *ricini* (Godfrey) Whet. prior to maceration of the tissue. The pectic enzyme action of *Botrytis* has been recognized as important in pathogenesis (*2*), and the enzymatic activity of isolates from castor bean has been demonstrated (*1*). Since pathogenesis and symptom development in *Botrytis* capsule mold of castor bean appeared due to pectic or cellulolytic enzymes produced by the causal fungus on the capsule surface, we studied the reaction of capsules to these hydrolytic enzymes in relation to varietal susceptibility. The development of a biochemical test for resistance was suggested by work on Panama disease of banana (*3*).

Fully developed green capsules of resistant, moderately susceptible, and highly susceptible varieties of castor bean were sprayed with a 25-percent solution of Pectinol 59L (Rohm and Haas) in distilled water containing a wetting agent. The sprayed capsules were incubated at a relative humidity of 100 percent for 16 hours at 33°C. In six tests, each with ten or more capsules of each variety, capsules of resistant varieties remained firm and green; capsules of moderately susceptible varieties turned brown (a characteristic of oxidation and polymerization of phenolic compounds); and capsules of highly susceptible varieties turned brown and the tissue became macerated. Surface-disinfested capsules reacted similarly. The enzyme solution was effective also after being sterilized by filtration. Similar results were obtained with a 12-percent solution of Cellulase 36 (Rohm and Haas) in phosphate buffer, *p*H 4.7, with a wetting agent. Temperature, time, *p*H, and enzyme concentration were critical factors in the results. The commercial enzyme preparations were shown to contain pectin methylesterase, polygalacturonase, cellulases that hydrolyze carboxymethylcellulose solution and that degrade insoluble cellulose, and enzymes that rapidly macerate carrot tissue. Intact capsules, whether or not they had been surface-disinfested, and mechanically injured capsules of resistant or susceptible varieties remained firm and green when sprayed with water or with heat-inactivated enzyme solution.

Filtrates obtained from cultures of the causal fungus grown on sterilized capsule pericarp contained enzymes that rapidly macerated carrot tissue and that hydrolyzed 1.5-percent citrus pectin solution and 1.25-percent carboxymethylcellulose solution. These filtrates had no effect on insoluble cellulose or on castor bean capsules. This possibly was due to the formation of insufficient amounts of the hydrolytic enzymes. By growing the fungus on a synthetic medium with insoluble cellulose as the sole source of carbon we obtained a cellulase capable of degrading insoluble cellulose. This enzyme produced reactions in the capsules similar to those caused by the commercial preparations.

Among the polyphenols shown to be effective as enzyme inactivators are compounds related to flavanol (*4*). The presence of flavanols and related compounds in fresh pericarp tissue of the capsule was determined by gently crushing small pieces of pericarp in six drops of a 10-percent solution of va-

nillin in ethanol and then acidifying the mixture with three drops of concentrated H_2SO_4. Resistant and highly susceptible varieties gave no color. Moderately susceptible varieties gave dark purple, indicative of flavanol (*5*). The presence of oxidized or reduced compounds in mechanically injured pericarp tissue was determined by thoroughly crushing pieces of pericarp in five drops of an aqueous solution of $FeCl_3$ and $K_3Fe(CN)_6$ (each 0.3 percent). Resistant varieties gave a greenish-black color; moderately susceptible varieties gave a bluish-black color; and capsules of highly susceptible varieties gave a clear blue color (Prussian blue). The development of blue or green colors with the reagent occurs with reduction or oxidation of iron ions. The black color is characteristic of many oxidized phenolic compounds.

The relation of the reactions with the two reagents to the resistance to the hydrolytic enzymes was determined from tests with capsules of 228 plants segregating for resistance. Ten or more capsules of each plant were tested with Pectinol 59L for enzyme susceptibility, and four or more capsules of each plant were tested with the two reagents. A correlation coefficient of .79 was found for resistance to the enzymes and the "resistance" reactions with the reagents. Although capsules of many plants showed intermediate reactions with the two reagents and the enzyme, it is significant that none of the capsules that showed resistance reactions with the reagents were susceptible to the enzyme.

The experiments indicate that the resistance of castor bean capsules to *Botrytis* can be determined easily by the enzymatic tests and by the reactions with vanillin and H_2SO_4 and with $FeCl_3$ and $K_3Fe(CN)_6$. Chromatographs show the vanillin-reacting flavanol to be present only in moderately susceptible tissue. Other phenols appear to be present in tissues of all levels of resistance. Oxidation of water extracts of resistant tissue in the presence of pectic and cellulolytic enzymes results in considerable inactivation of the enzymes. Resistance, as reported for certain other diseases (*4*), appears to be due to enzyme inactivation by oxidation products of polyphenols. Phenols differ in their ability to inactivate enzymes (*4*), and differences in polyphenols in the capsules of varieties of castor bean may be important in determining differences in varietal susceptibility. However, oxidation of the phenolic compounds by polyphenoloxidase in injured tissue also is involved in resistance (*4*), and this may be involved in the reactions of the crushed tissue in $FeCl_3$ and ferricyanide. In preliminary experiments, it appears that the peroxidase activity of resistant capsule pericarp is significantly greater than that of susceptible tissue (*6*).

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13 December 1962

Age and Accumulation Rate of Dolomite-Bearing Carbonate Sediments in South Australia

Recent studies of the sediment forming in shallow, ephemeral lakes and in the Coorong, a shallow arm of the sea, in the southeastern part of South Australia indicate that a calcian dolomite and a magnesian calcite are forming today by direct precipitation from saline waters (*1*). The waters range in salinity from half to about four times the salinity of sea water, but the ionic balance is very close to that of sea water. Further studies in the same general area (*2*) have shown that other ephemeral lakes with a different water composition precipitate aragonite, magnesite, and hydromagnesite. The season during which the carbonates have been observed to precipitate is a short period in early summer (October and November) when water accumulates to a depth of 1 to 2 feet in the lakes and vigorous plant growth takes place.

One of the ephemeral lakes (Lagoon M, 36°40'S, 139°54'E) in which precipitation has been observed on several occasions (*3*) was studied intensively by one of us (H.C.W.S.). A bore hole put down in the center of the lake in 1957 revealed carbonate sediment 30 inches thick. A 10-g sample taken 1 to 2 inches below the surface contained equal amounts of calcite and dolomite, with traces of quartz and clay minerals. A 20-g sample taken 19 to 20 inches below the surface consisted predominantly of calcite but contained minor amounts of dolomite and traces of quartz and clay minerals. Both samples were subjected to radiometric analysis to determine their absolute ages by the carbon-14 method. The sample obtained 1 to 2 inches below the surface (field sample No. S4-2; laboratory sample No. W-1100) was too small to be dated any more precisely than "less than 600 years" ago. It is unfortunate that a pure dolomite fraction could not be separated for testing; since this sample contained equal amounts of dolomite and calcite, some of the carbon-14 activity must reside in the dolomite. The maximum possible age of the dolomite is 1200 years; this makes these South Australian deposits the first authenticated modern dolomite locality.

The age obtained for the sample from a depth of 19 to 20 inches (field sample No. S4-20; laboratory sample No. W-1101) was 3000 ± 600 years, indicating an age difference between the two samples of approximately 2500 years. Thus, during an interval of approximately 2500 years, 457 mm (18 in.) of carbonate sediments accumulated, indicating an average rate of accumulation of approximately 0.2 mm per year. The computation requires the assumption that there has been neither a radical change in water depth nor a significant change of climate that would cause a prolonged break in the sedimentation record.

Two other estimates of the rate of accumulation of carbonate sediment have been made. Alderman (*4*) calculated a possible sedimentation rate from the composition and depth of water in one of the ephemeral lakes. His figure of 1 foot per 700 years corresponds to a rate of approximately 0.44 mm/yr. C. von der Borsch (*5*) arrived at a similar figure by counting laminae, presumably annual, in the aragonite-hydromagnesite sediments.

Alderman and Skinner (*1*) reported that a water sample with fresh precipitate still suspended had a sediment content of 0.05 g per 100 ml of lake water,