

sation of the growth of sarcoma 37. This phenomenon is striking; whether or not the concomitant tumor necrosis was the result of a specific action of the drugs used cannot now be answered. The effects noted may also be explained as the result of the prolonged depressed state with accompanying inanition, dehydration, changes in metabolism, lowering of body temperature, and a drop in blood pressure with consequent hypoxia. All these are factors which by themselves can produce marked tumor damage and can slow tumor growth.

The appearance of fat granules in the ascitic cells similarly may be the result of the depressed state of the host. Unlike the controls in which the ascites daily increased in volume, the ascitic fluid in treated mice was very scanty, thick and viscous, but rich in cells. As a result of the progressive dehydration and other changes, the metabolic state of the ascitic cells could well have been affected, and the fat granules (which many would call "degeneration" granules) could be the consequence of the changes.

Whether the action of these drugs is specific or is mediated through the host, it is suggested that these drugs provide an additional means for study of the host-tumor relationship, particularly in conjunction with other tumor-necrotizing drugs.

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#### References and Notes

1. A. Goldin *et al.*, *Science* 125, 156 (1957).
2. We wish to thank Ira Kline for preparation of the material that was treated with the special stains used in this investigation.
3. Serpasil (Ciba).
4. Thorazine (Smith, Kline and French).

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### Effect of Gibberellic Acid on Growth of Maize Roots

Gibberellic acid has been shown to stimulate markedly stem and leaf elongation in a number of plants (1-4). The specific genetic constitution of a strain or variety appears to determine whether or not it will respond to applied gibberellic acid by increased shoot growth. So far the most notable responses have been observed in dwarf types. Phinney (2) has reported that applications of gibberellic acid to five single-gene, dwarf mutants in maize so enhanced growth that the treated plants were almost indistinguishable from plants carrying the normal alleles of the mutant genes. One other dwarf mutant made only a slight response, and another made no response. Such differential responses of shoot

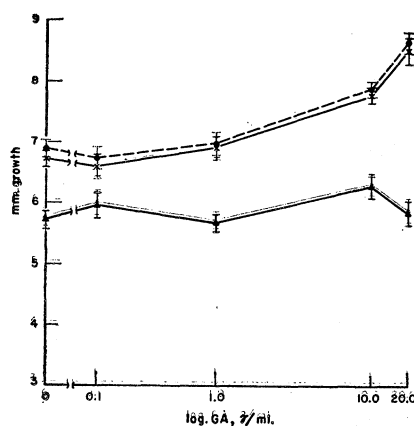


Fig. 1. Growth of excised, apical, 10-mm segments of primary roots of two maize inbreds and their hybrid as affected by gibberellic acid. The limits indicated at each graph point represent  $\pm 2$  times the standard error; x, hybrid; ●, line 854, ▲, line 857.

growth have also been reported in varieties of *Pisum*, *Phaseolus*, and *Vicia* containing dwarfism alleles (1). To date, the only experiments on root growth reported are those of Brian, Hemming, and Radley (3), who found that gibberellic acid had no significant effect on the growth of roots of cress seedlings.

In the work reported here, gibberellic acid (5) was added to White's supplemented solution (6) in which excised, apical, 10-mm segments of maize roots were grown for the 24-hour period representing the sixth to seventh day after the beginning of germination. Apical segments of both primary and adventitious seminal roots of two inbred lines of maize and their distinctly heterotic hybrid, bearing our laboratory numbers 854, 857, and 854 x 857, were used. Neither inbred line contains any dwarfism alleles, and other studies have shown that the growth rates of the inbreds beyond the very early seedling stage are comparable to the growth rate of the hybrid. The effects of gibberellic acid on growth of excised apical segments of primary roots are shown in Fig. 1. The points on the graph for 0.1, 1.0, and 10.0 µg/ml represent means of three or four replicates of at least ten roots each. The smallest number of roots represented is 35, the largest 93. The points for 20.0 µg/ml represent only single tests with 16 to 40 roots. The supply of gibberellic acid was too limited to permit repetition of the 20 µg/ml tests. The primary roots of line 857 were not affected by gibberellic acid over the range of concentrations used. Those of line 854 were significantly stimulated by concentrations of 10 µg/ml and further stimulated by 20 µg/ml. At 20 µg/ml, growth was increased on the order of 24 percent. The effect on the primary roots of the hybrid appears to be identical with that upon the roots of 854. The curves

suggest that the primary roots of 854 and the hybrid might be further stimulated by higher concentrations.

The growth of the adventitious seminal roots was affected by gibberellic acid in the same manner as the growth of the primary roots, but the amount of stimulation of 854 and the hybrid was proportionately less than that in the primary roots, about 12 percent at 20 µg/ml. The adventitious seminal roots are of later origin than the primary roots, and they normally grow somewhat less than the primary roots under the conditions and during the experimental period used here.

More extensive experiments with the effects of gibberellic acid have been carried out (7), and it may be noted that all our results indicate that the root growth in certain genotypes of maize is significantly stimulated by gibberellic acid. The results presented in this preliminary note, showing a positive growth response of one inbred, no response by the other inbred, and a hybrid response essentially parallel to that of the first inbred, suggest direct inheritance of a growth system which can be affected by gibberellic acid.

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#### References and Notes

1. P. W. Brian and H. G. Hemming, *Physiol. Plantarum* 8, 669 (1955).
2. B. O. Phinney, *Proc. Natl. Acad. Sci. U.S.A.* 42, 185 (1956).
3. P. W. Brian, H. G. Hemming, M. Radley, *Physiol. Plantarum* 8, 899 (1955).
4. B. O. Phinney, *abstr., Plant Physiol.* 31, Suppl. 20 (1956).
5. The gibberellic acid was kindly supplied by Curt Leben, Eli Lilly and Co., through Irwin Spear.
6. P. R. White, *A Handbook of Plant Tissue Culture* (Cattell, Lancaster, Pa., 1943).
7. A paper describing our more extensive experiments is in preparation.

5 November 1956

### Radiocarbon Dates from Sandia Cave, Correction

Frank C. Hibben has reported in *Science* (1) that in 1948 the late Kirk Bryan submitted two samples of charcoal from Sandia Cave for radiocarbon age determination to W. F. Libby, who was then at the University of Chicago. Hibben reported that these samples came from fire hearths located in the Sandia level of the cave and that "From these two samples, tentative dates of 17,000-plus years ago and 20,000-plus years ago, respectively, were derived." There is no proof that these alleged dates were ever determined by radiocarbon analysis or that Bryan ever submitted any samples from Sandia Cave to any laboratory.

It is a matter of record that the dates were not determined at the University of Chicago laboratory, as Hibben, in correspondence, has recognized. No other laboratory is known to have determined radiocarbon dates until the Lamont laboratory began operations in 1951. Bryan died on 22 August 1950.

None of Bryan's intimate associates, including reputable archeologists and geologists, some of whom were deeply concerned with the development of radiocarbon dating, can recall having heard Bryan mention the samples or the dates to which Hibben refers. Furthermore, Bryan's records have been searched and no reference to the alleged Sandia samples has been found. The dates must be struck from the record before they cause further confusion.

Other confusing evidence indicates that the dates lack a proper source and record. Hugo Gross quotes "Dr. Frank C. Hibben, oral communication," as the authority for the statement that "... this method (radiocarbon dating) recently indicated an age of 11,000 and 19,000 years, respectively, for the Folsom and Sandia layers in Sandia Cave" (2). These dates, published in 1951, differ significantly from those published by Hibben in 1955—that is, 17,000-plus and 20,000-plus years ago. The latter dates were alleged to have been determined on charcoal said to have come from two fire hearths of only the Sandia level. The discrepancies in figures and the attribution to levels throw great doubt upon the statements in Hibben's article in *Science*.

The repudiation of the dates quoted by Hibben as originating with Bryan has no bearing on the dates from Sandia Cave determined by H. R. Crane (3). Such dates indicate the radiocarbon content of the samples delivered to the Michigan laboratory. Whether or not these samples are contemporaneous with the Sandia level is a completely different, unrelated question.

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#### References

1. F. C. Hibben, *Science* 122, 688 (1955).
2. H. Gross, *Bull. Texas Archeol. and Paleontol. Soc.* 22, 114 (1951).
3. H. R. Crane, *Science* 122, 689 (1955).

8 November 1956

In regard to the two samples of charcoal from Sandia Cave collected by the late Kirk Bryan, there has been a dearth of evidence as to where and how Bryan dated these samples. I am in agreement with Johnson that these dates should be removed from the record. This removal, of course, in no way invalidates either the very fine geologic work of Bryan or the dating of the Sandia deposits themselves.

The dates referred to by Hugo Gross were extracted from a series of lectures one of which was given by me at the University of Erlangen, Germany. These dates were derived from Bryan's geologic work and were extremely tentative. Dating by geologic means and carbon-14 were undoubtedly confused. Gross is in error in his dates of 11,000 and 19,000 years, respectively, for the Folsom and Sandia layers of Sandia Cave. The original dates given in the Erlangen lecture were 11,000 B.C. for a Yuma site, 9000 B.C. for Folsom layers in Sandia Cave, and 17,000 B.C. for Sandia level in Sandia Cave. All these dates were derived by stratigraphy and not by the radiocarbon method. As yet no carbon-14 date has been derived from the Folsom level of Sandia Cave.

Removal from the record of the radiocarbon dates attributed to Bryan does not invalidate the dates determined by H. C. Crane from mammoth ivory from Sandia Cave. These are substantiated by other evidence.

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### Theory of Ice Ages

The theory of glacial and interglacial periods during the Pleistocene offered by Ewing and Donn in a recent issue of *Science* (1) is very provoking. It is certainly true that the melting of an Arctic Ocean ice sheet such as exists at present would, by making the Arctic Ocean ice-free, provide an increased source of moisture for the polar atmosphere, and it is true that this could have been accomplished through a greater interchange of water between the Atlantic and Arctic oceans. Whether this greater interchange would be associated with a cooling of the Atlantic, as the authors maintain, and also whether the open Arctic Ocean with its new moisture supply would favor the growth of glaciers over the areas to the south, is, however, a matter for further consideration.

While all the evidence as far back as the 1930's points to a warming of the Arctic Ocean, the more recent evidence shows a simultaneous though less marked warming of the Atlantic as well (2). Equally, the extensive deglaciation that has occurred within the past 50 years or so, particularly in the 1930's, coincided with a sharp decrease in the thickness of the Arctic ice pack from a value of approximately 3.6 m, which was measured by the Fram expedition, to a value of only 2 m, which was obtained by the North Pole expedition in 1937 and also coincides with a shrinkage of its area

by more than 10 percent from the earlier to the more recent period. Consequently, if the process were to continue, an open Arctic Ocean would be associated with increasing deglaciation and eventually with no ice whatsoever.

It is acknowledged that a very substantial lowering of sea level would, by restricting the interchange of water between the Arctic and Atlantic oceans, make it possible for the Arctic Ocean to freeze over. However, a frozen Arctic Ocean would, according to the authors' view, only stop the glaciation from growing through the cutting off of the new moisture supply. For a waning of the glaciation itself, to make the cycle complete, the authors assume a starvation of the ice. This might be true of the inland ice in a very limited measure, but not of the glaciers whose waning is accompanied by a recession.

Rather, I think that the same agency that is responsible for the simultaneous warming of the Arctic and Atlantic oceans and for the shrinkage of the arctic ice pack is also responsible for the deglaciation, and that this is due primarily to a rise in temperature. In accepting the authors' claim that an open Arctic Ocean provides a new supply of moisture for the polar atmosphere and for an increase in precipitation, I suggest that this precipitation is in the form of rain, not only over the adjacent lands to the south, but also over the Arctic Ocean.

The fact that within the recent period the temperature of the South Atlantic Ocean has also increased suggests that the agency responsible operated on a broad scale and from outside the earth.

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#### References

1. M. Ewing and W. L. Donn, *Science* 123, 1061 (1956).
2. J. Smed, *Cons. perm. intern. Explor. Mer. Rapp. proc. Verb.* 125, 21 (1949); I. I. Schell, *J. Cons. perm. intern. Explor. Mer.* 18, 1 (1952); H. J. Bullig, *Deut. Wetterd., Seewetteramt.* No. 5, 1 (1954); H. Riehl, "Sea-surface temperatures of the North Atlantic, 1887-1936" (Dept. of Meteorology, Univ. of Chicago, 1956).

10 October 1956

### Presence of Serotonin in Lung and Its Implication in the Anaphylactic Reaction

Serotonin (5-hydroxytryptamine) is known to be widely distributed in the animal and plant kingdoms. In animals it has been reported to be present in gastrointestinal tract, blood platelets, spleen, and brain (1, 2), and possible functions for serotonin in each of these tissues have been suggested. There has been speculation for some time whether serotonin may