system, although Nordenskiold's name is not. By inference Nordenskiold has named the type. If a question of priority should arise 1893 should be the date —not 1924.

(5) The type must be sufficiently well described that the description does not conflict with the description of any other type.

(6) A name printed without a description should be ignored.

(7) The date of acceptance of a manuscript for publication, when printed in a journal, supersedes the date of publication of the journal in matters of priority.

(8) The name and description of a new type, to be accepted, must be printed, lithoprinted or mimeographed, and not less than fifty copies distributed to libraries of anthropological laboratories and workers in the field.

(9) As suggested by Gladwin and Gladwin (1930) when a type is named sherds and, if available, whole vessels should be designated type specimens and set aside for future comparison.

The authors of this paper would be glad to receive comments on the above rules.

HAROLD S. COLTON
LYNDON L. HARGRAVE
MUSEUM OF NORTHERN ARIZONA
FLAGSTAFF

THE INHIBITING INFLUENCE OF A VIRUS ON ONE OF ITS MUTANTS

SEVERAL years ago the writer[1] called attention to the fact that when tobacco plants showing the symptoms of the common mosaic (referred to as light-green mosaic) were reinoculated with the virus of a yellow mosaic, which is considered to be a mutant of the former, no change in the symptoms occurred.

The writer inoculated tobacco plants with virus mixtures in which the virus extract of the yellow mosaic was 999 times more concentrated than the virus extract of common mosaic. All the inoculated plants developed symptoms of yellow mosaic. However, 47 days after inoculation the young leaves were showing the symptoms of common mosaic. Other plants, inoculated at the same time with mixtures containing 499 parts and 99 parts of the extract from yellow-mosaic plants, also developed symptoms of yellow mosaic, but the common mosaic symptoms made their appearance earlier than 47 days.

Tobacco plants having yellow mosaic were reinoculated with the purest virus of common mosaic obtainable. From 5 to 20 leaves which developed after this reinoculation developed yellow mosaic, then from 8 to 25 leaves developed progressively less yellow mosaic in combination with common mosaic until the subsequent foliage developed only typical common mosaic. Suckers from such plants developed the typical symptoms of common mosaic. Some of the first few leaves which manifested only the light and dark-green mottling carried a small amount of the virus of yellow mosaic. However, subsequent leaves were free of detectable amounts of yellow-mosaic virus, except in cases where the small yellow-mosaic mutation spots occurred and in such cases the virus of yellow mosaic was confined to these spots and the adjacent tissue.

Other workers have found that certain yellow-mosaic viruses which induce necrotic lesions in suitable species will not induce these lesions if the plants have been previously infected with certain leaf-mottling viruses.

This phenomenon has been referred to as acquired immunity[2, 3] and as induced immunity.[4] On this basis the virus of common mosaic may be looked upon as an immunizing agent or "vaccine" and from the evidence presented in the second and third paragraphs it appears that we are dealing with a condition of incompatibility in which the "vaccine" virus eventually suppresses the yellow-mosaic virus in the meristematic tissues. This interpretation makes it unnecessary to assume that the "vaccine" virus induces the plant to set up a special defence mechanism which in turn combats the virus of yellow mosaic.

It appears that the virus of common mosaic represents a rather low or primitive form of "vaccine," since the disease induced by it becomes permanent and is a distinct menace to the plant during its life under normal cultural conditions. On the other hand, the virus seems to represent a uniquely high type of "vaccine," since it suppresses the development of the yellow-mosaic virus and ultimately induces what possibly may be considered a cure for yellow mosaic in those parts of the plant which are formed after the original yellow-mosaic virus has been sufficiently reduced. The *G* virus used in Salaman's[5] tests against the *L* virus in tobacco and *Datura stramonium* is a more efficient "vaccine" since it induces very slight symptoms with no appreciable effect on the health of the plants. It is possible that virus mutants may be isolated which will protect as well as "cure" and yet not survive indefinitely in an active form in the plant.

The inhibiting characteristic of the common-mosaic virus is regarded as one of the strongest lines of evidence in support of the view that the occasional small yellow-mosaic spots[1] which have been associated with common mosaic in all the 5,000 or more tobacco plants studied by the writer resulted not from viruses intro-

[1] H. H. McKinney, *Jour. Agr. Research*, 39: 557, 1929.

[2] L. O. Kunkel, *Phytopathology*, 24: 437, 1934.

[3] W. C. Price, *Phytopathology*, 25: 776, 1935.

[4] John Caldwell, *Proc. Roy. Soc.*, Ser. B 117: 120, 1935.

[5] Redcliffe N. Salaman, *Nature*, 131: 468, 1933.

duced from the outside, but from viruses which originated as mutants in the tissues involved in the spots.

It was pointed out in an earlier paper[1] that plants having pure yellow mosaic are rarely observed in commercial tobacco and tomato fields, and they were rarely seen among the many mosaic-diseased *Nicotiana glauca* plants growing in the Canary Islands. These observations are now explained on the basis that the common-mosaic virus greatly restricts the size of the yellow-mosaic mutation spots and also the amount of the yellow-mosaic virus, thus decreasing the chances for its spread to other plants. A relatively large amount of the common-mosaic virus is thus maintained in the infected plants and this virus prevents the establishment of tobacco yellow-mosaic virus which might enter these plants from the outside.

H. H. McKINNEY

BUREAU OF PLANT INDUSTRY
U. S. DEPT. OF AGRICULTURE

IODINE THERAPY AND GOITRE

MAY I add a mite[1] with respect to the early history in the use of iodine in the successful treatment of (endemic) goiter. I refer to Moses Gunn's (1822–1891) famous prescription, which every Rush Medical College graduate is quite familiar with.

Dr. Moses Gunn went to Rush Medical College Chicago, Ill., (from Ann Arbor, Michigan), in 1867, as head of the department of surgery and as the successor to Dr. Daniel Brainard.[2] Finding a high incidence of goiter in the Chicago area he prescribed his now famous "three eight" mixture, as follows:

℞

Iodi	gr. viii
Potassii iodidi	ʒ viii
Syrupi sarsaparillae ad f	℥ viii

No doubt the supposedly "alterative" action of iodine was uppermost in the mind of Dr. Moses Gunn. At any rate, this early practitioner noted the beneficial effects of iodine therapy in the treatment of hyperplastic goiter. And, it should be noted, long before Marine (1907) put iodine therapy for this condition on a truly scientific basis!

ARNO B. LUCKHARDT

UNIVERSITY OF CHICAGO

SPECIAL ARTICLES

THE EFFECT OF ALTERATION ON THE LEAD-URANIUM RATIO AND THE CALCULATED AGE OF WILBERFORCE, ONTARIO, URANINITE

IN order to determine the effect of leaching on the lead-uranium ratio of uraninite, a determination of the lead, uranium and thorium content of three zones of a single crystal of Wilberforce, Ontario, uraninite has been made. Instead of grinding a large specimen of the mineral to uniformity and analyzing the then homogeneous material, as is usually done in age determinations, a method of carefully removing successive layers from the crystal was used, followed by analysis of the outermost layer, middle layer and innermost layer or core.

A crystal weighing about twenty-four grams was selected. It was apparently altered to some extent, as the color was reddish brown, although the cubo-octahedral structure of the crystal was still intact, this in spite of the great age of the mineral and the large amount of lead which had been generated in the crystal since its formation.

The crystal, which had previously been cleaned from any superfluous material, was weighed, treated with dilute nitric acid until approximately one third of the mass was removed, washed, dried and reweighed. The remainder of the crystal was gently treated with acid until another third was dissolved. Finally, the core was dissolved. Aliquot portions of each of these resulting solutions were analyzed for lead, uranium and thorium by methods which will be described in a later paper.

The outside section included all of a shell, perhaps from one to two millimeters thick, of highly altered material which covered the entire surface of the crystal and some of the black material which constituted the remainder of the crystal. All the middle layer and core appeared homogeneous and pure black in color.

From the data obtained from these analyses, the "lead ratio" and the corresponding age of each section of the crystal could be calculated by means of the formula:

$$\text{Approximate age} = \frac{\text{Pb}}{\text{U} + 0.36\ \text{Th}} \times 7600 \text{ million years}$$

The age as calculated by this approximate formula is somewhat higher than that given by the more exact logarithmic formula, but is used here in order that it may be compared with the results of earlier investigations of the age of this mineral.

The average results of several analyses of each section are given in the following table:

[1] McCay, SCIENCE, 82: 2128, 350, 1935; McClure, SCIENCE, 82: 2129, 370, 1935.

[2] "History of Medicine and Surgery and Physicians and Surgeons of Chicago, 1803–1922," p. 61. The Biographical Publishing Co., 133 W. Washington Street Chicago, Ill., 1922.