the observations of Ransom[1] and our own,[2] artificial digestion has no apparent injurious effect upon living trichinae. One may conclude, therefore, that most of the larvae are destroyed after dissemination in the body. The manner in which they are destroyed remains unknown, since, in no instance, have we been able to find the parasite *in situ*.

Feeding experiments were not undertaken with trichinous chick muscle because of the exceedingly light infections in all birds. It is likely that our method of examination is more reliable for demonstrating the presence of larvae than the feeding of muscle to experimental animals, for with the digestion method practically all the larvae, including degenerated ones, in a given amount of muscle are made apparent.

At no time did any of the chicks show discomfort or ill effects from the infections with Trichinella.

It may be concluded that Trichinella larvae occasionally invade and develop in the musculature of fowl. They have never occurred in sufficient numbers as to be microscopically demonstrable, so that their situation in relation to the muscle fiber is unknown. In this host the life of the larvae is evidently short, for a large proportion of those found appeared lifeless or degenerated. It is doubtful that infection from this source ever occurs.

DONALD L. AUGUSTINE
MEDICAL SCHOOL AND SCHOOL
OF PUBLIC HEALTH
HARVARD UNIVERSITY

PRELIMINARY NOTE CONCERNING THE APPEARANCE OF EXPERIMENTAL DERMATITIS (PELLAGRA) IN RATS[1]

PELLAGRA, until comparatively recently, has been regarded as a food poisoning and attributed to the use of maize or Indian corn as a food.

After other theories were thought to have been the cause of pellagra, Goldberger in 1925 reported that there was concerned a heretofore unrecognized or unappreciated dietary factor which he designated as factor P–P, and which he believed played the essential rôle in the prevention of pellagra. His factor was later identified as vitamin B_2 or G.

Yeast, which is rich in this factor, proved effective in the treatment of pellagra cases and has become more or less routine treatment for the disease. However, this does not explain the high incidence of pellagra among people on maize or yellow corn.

Ethel Browning, in her monograph on the vitamin, says that "Mellanby's investigation (1930) into the toxic factors in cereals opens up a further interesting speculation, which may reconcile the conflicting results. He suggests that maize may contain a toxic factor which may be prevented from exercising its pellagra producing capacity by the presence of a protective factor of the nature of a vitamin, or possibly two vitamins—B_2 and A."

From a series of experiments carried on at Teachers College, Columbia University, it was suggested that this factor which Mellanby thinks caused toxicity might be B_1. These experiments showed that the pellagra producing capacity was not so much a question of low B_2 (G) as it was of low or subminimal amounts of B_2 (G) coupled with high or adequate B_1.

Animals were fed a B_1 and B_2 free diet. In one series the B_1 was normal, constant and the B_2 graded from subminimal to high, and in the second series the B_2 fed was normal, constant and the B_1 graded the same as above. It was found that when the B_2 was low or subminimal and B_1 adequate or high, pellagra developed more uniformly in practically all cases and with little variation in the number of days in the appearance of dermatitis.

These experiments would explain the appearance of pellagra on a diet of maize or any other of the whole cereals. They are rich in B_1 and low in B_2, and a continuous diet of such cereals supplemented with other foods containing little or no B_2 would produce pellagra if compatible with the theory.

A synthetic diet was used in the foregoing experiments. A series is now being run with an adequate diet, except that vitamin B_2 (G) has been destroyed by irradiation. Results are apparently comparable to the first series. Details of the experiment will be reported later.

MINERVA KELLOGG
WALTER H. EDDY

CONTROL OF DOWNY MILDEW DISEASE OF TOBACCO THROUGH TEMPERATURE REGULATION

THE downy mildew or blue mold disease of tobacco caused by *Peronospora hyoscyami* DBy. has in the past two years become recognized as a very serious problem to tobacco growers in this country. This disease has long been destructive in Australia and no effective means of control are known. Among many suggestions made we find the possibility of temperature regulation mentioned and since the disease attacks the plants while they are still in the beds this would be a practicable measure.

In the Farmers' Handbook, 5th edition, published in 1929 by the New South Wales (Australia) Department of Agriculture, it is stated, "Experiments conducted by the department during the past few years indicate that if the temperature of the seedlings is not allowed to fall below 45 degrees F. and the surrounding air does not become humid, blue mold does not

[1] *Jour. Agric. Res.*, 5: 819, 1916.
[2] *Amer. Jour. Hyg.*, 17: 697, 1933.
[1] From the Laboratory of Physiological Chemistry, Teachers College, Columbia University, New York City.