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.

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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.

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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.

make its appearance." This conclusion, however, was discredited by Angel,[1] who found that infection could be secured at temperatures as high as 31 degrees C., and he concludes, "It is, therefore, useless to attempt to control the disease by temperature modification only, as the plants are susceptible even at those temperatures that are much higher than the optimal for growth."

In our experimental approach to this problem it was recognized that at least three phases of the disease might be affected by temperature: (1) The production of conidia, (2) spore germination and infection, (3) subsequent development of the fungus in the host tissues.

In studying the influence of temperature on sporulation it was first established that this occurs in the early morning, and that this was not associated with light, since leaves held in complete darkness also sporulated about the same time. The conidiophores begin to grow out through the stomata on the under leaf surface just before dawn, and mature conidia can be gathered between 5 and 6 A. M. Sporulation, however, was not observed when the minimum temperature remained above 70° F., regardless of the maximum temperature, and in controlled temperature experiments good sporulation was only secured with temperatures ranging from 50 to 65° F., with a well defined optimum around 60–62° F. Furthermore, the temperature of the day previous was found to have an effect, and abundant sporulation was not secured with maximum day temperatures of 85° F. and above. Destructive outbreaks of the disease were invariably associated with periods of abundant sporulation.

Our studies of spore germination and infection yielded results comparable to those reported by Angel and indicated that infection was possible over a range of temperatures from 35 to 88° F.

Studies of disease development subsequent to infection, however, also yielded results of interest. With day temperatures rising above 85° F. and dropping to 70° or above at night, the fungus invaded only a small leaf area and quickly caused round, white lesions up to ¼ inch in diameter. These lesions made no further development and plants of fair size usually suffered no injury of consequence from such infections. However, when the day temperature did not go above 80° F. and the night temperature dropped to around 60° F., the fungus, following penetration, continued to spread through the leaf tissues, but without any destruction of tissues until about a week after inoculation. Then sporulation would occur over large portions of the leaf area, to be followed by the death of these parts. High temperatures consequently, while they do not inhibit infection, do prevent disease spread by preventing sporulation and also check the development of the fungus in the tissues of the host. The inhibiting effect of high temperatures has frequently been observed in the field, and periods of 5 or 6 days of hot weather have often so effectively checked the disease that it failed to renew its activity during a subsequent period of favorable weather conditions.

Proof of the practical efficiency of high temperature in controlling mildew has been obtained by the authors both in plant bed and greenhouse tests. In these experiments minimum temperatures of 70° F. or above were usually maintained, while high temperatures during the day were favored by the use of glass sash in place of cloth.

The temperature results have also solved the problem of how to keep the fungus in culture through the summer months, since it is an obligate parasite and can not be found in the field after warm weather begins. Plants growing in a chamber so constructed as to maintain a maximum and minimum of 80° and 60° F., however, have continued to provide all stages of the disease throughout the summer.

In conclusion it is to be pointed out that despite the effective disease control secured in our plant bed experiments this year it is not to be assumed that such satisfactory results would be secured under all conditions. The mildew, as it has occurred in this country during the past three years, has rarely caused severe damage until the latter part of the plant bed period, frequently at a time when the plants are almost large enough to set out. It is under these conditions that temperature regulation has proven most effective. On the other hand, our experiments indicate that small seedlings, if exposed to mildew attack, would not be so effectively protected, a fact that appears to be associated with the extreme susceptibility of the very young plants. Also our experiments have been conducted with flue-cured varieties, and there is one variety, White Burley, that is more susceptible to the mildew disease. It is probable that this increased susceptibility will make control of the disease more difficult with Burley.

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[1] Australian Council for Scientific and Industrial Research, Bul. 65, 1932.

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