The principle of operation is very simple. When at rest the unstoppered leveling bulb B_2 is in the lower position. When suction is turned on, mercury is pulled up into the stoppered bulb B_1 until the weight of this bulb becomes greater than that of the lower bulb. The upper bulb then descends, but does not reach a position as low as the highest position of the lower bulb. The partial rotation of the arm caused by the change in weights brings the valve to such a position that room air is free to flow into the stoppered bulb, and as a result of the increased pressure, mercury flows downward into the lower bulb. The shift in weight now pulls the lower bulb to its bottom position and changes the relative positions of the valve openings so that they are again in the same position in which they were at the beginning of the cycle. One set of valve openings actuates the pulsator itself, while the other produces changes in pressure in the tubing connected with whatever apparatus the pulsator is being used to run.

The pulsator can be easily and cheaply constructed, and most of the materials used are available in any laboratory. It is clean, quiet in operation, and can be placed anywhere out of the way. During the past few months the one now in use has been in operation almost daily and has required almost no care aside from weekly lubrications with vaseline.

The pulsator is adjustable through a wide range of speeds, ranging from very rapid to very slow pulsations. For use with the gas analysis apparatus the optimum speed has been found to be about four pulsations per minute.

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SPECIAL ARTICLES

EXPERIMENTAL TRICHINOSIS IN CHICKS

WHILE it is generally known that *Trichinella spiralis* will develop to the adult stage in the intestine of birds, the development of muscle larvae from oral infection, so far as we are aware, has never been observed.

In the present study mature Trichinella larvae have been demonstrated consistently in the musculature of chicks fed infective material. It is believed that failure heretofore to demonstrate this stage of the parasite in this host is due to the method of search employed (that of direct microscopic examination).

In the first of the present experiments, eight two-day-old chicks were fed thousands of isolated infective Trichinella larvae. On the first day after infection, one of the chicks was killed and a thorough search of the intestine was made for the parasite. A few young adults were found in the lower portion of the small intestine. Forty-five days after the infective feeding, two of the chicks died of a coccidial infection (diagnosed *Eimeria tenella* by Dr. E. Elizabeth Jones). A careful microscopic search of muscles failed to reveal any evidence of trichinosis. The white and dark meat were removed separately, then ground and treated by artificial peptic digestion. In the concentrated sediment from each, about two hundred fully developed Trichinella larvae were found, many of which were inactive and appeared lifeless. The remaining five chicks were killed a few days later. None showed infection by direct microscopic examination, but a few dead larvae were obtained from each chick by the digestion method.

It was thought that the lightness of the infections might be due to a mechanical destruction of a large number of larvae during their passage through the gizzard. To determine whether ingested larvae may thus be destroyed, a ten-week-old chick was fed thousands of isolated Trichinella larvae, and its droppings during the following three hours were collected for examination. In the intestinal discharges, numerous broken and mangled larvae were found, as well as many coiled larvae, which responded sluggishly to warmth. The chick was then killed and the entire intestinal tract examined. Active, uninjured larvae were found in the crop and proventriculus. In the gizzard and throughout the small and large intestine, however, many crushed forms and pieces of larvae were found along with others which were apparently unharmed. It is evident from these results that a large number of Trichinella larvae are destroyed in the gizzard of birds.

In the following experiments, it was hoped to establish a sufficiently heavy muscle infection whereby its nature could be studied. Four eleven-day-old chicks were etherized, and well over a thousand Trichinella larvae liberated from their cysts by the ingestion method were injected directly into the lumen of the jejunum of each. Three days later one of the chicks was killed. Numerous adult males and females with eggs in the uterus were found in the middle portion of the small intestine. The three remaining chicks were killed and examined fifty-four days after injection of larvae. Direct microscopic examinations of the muscles were negative. A few brownish dead larvae were obtained from each by the digestion method. From these results it is evident that the protective mechanism of chickens against trichinosis is not centered solely in the gizzard. According to

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.