In the more distant parts of the boulder train material was collected by the writer only where it was found in place in gravel pits and road cuts. Beach pebbles, which might have been brought by floating ice, were disregarded. On Cape Cod some gray and greenish-gray specimens were found whose structure is identical with that of the Hingham red felsite. Some of these when broken are purplish within and some are gray throughout. Whether or not these are weathered fragments of the Hingham red felsite is immaterial, as numerous pebbles of the typical red felsite were found in the same places.

In Hyde Park, fifteen miles west of the Hingham locality, are some exposures of red felsite that are part of the Mattapan volcanic complex. The rock found there is less strongly colored than that from Hingham and differs from it in the details of flow-structure. The two sorts of rock are easily distinguished. The boulder train extending from the Hyde Park area is rather scanty, and no specimens that could have been carried from there by the ice were found near the western margin of the Hingham boulder train.

The line of the terminal moraine in the northern part of Nantucket (about 83 miles from the source in Hingham) is usually recognized as the limit of the farthest advance of the ice in that locality. Specimens found south of the moraine were probably transported by streams flowing from the ice and deposited in the outwash plain. They are not technically in the boulder train and should not be so considered unless it can be shown that they were deposited by the ice of a previous glaciation or by a possible over-riding of the ice contact by the last glacier.

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PRELIMINARY STUDIES OF A CEREBRAL DISORDER OF YOUNG CHICKENS[1]

A DISORDER of chicks characterized by nervous involvements was observed on a number of poultry farms in Rhode Island during the past winter and spring. The disease appeared to be quite wide-spread over the state, and reports from other sections of New England would indicate that a similar difficulty was experienced by poultry growers in these sections.

The symptoms of the disorder resembled those described by Pappenheimer[2] and Dunlap[3] to a considerable extent. The only characteristic and consistent lesions were those of the brain in which the cerebellum was most often involved. This portion of the brain on gross examination showed edema and swelling with visible gross hemorrhages of the tissue. In some instances the same type of lesions was also noted in the cerebrum.

Observation showed the disease to be most prevalent between the ages of three to six weeks, although it has been observed by the writers in chicks as young as 16 days old. Since it appeared to involve the faster growing chicks of the lot, it was found more frequently in cockerell than in pullet chicks. The disease outbreak showed a rather sudden onset.

Cultures made from the brain and other tissues on various types of media remained sterile and suspensions of macerated brain tissue inoculated intravenously, subcranially and subcutaneously failed to incite symptoms of the disorder. Since no growth was produced on cultural media and attempts to produce the disease by inoculation had failed, it would appear that the condition was not one of an infectious nature but probably one of a nutritional type. It was observed in the field under a wide variation of management conditions, feed mixtures, breeds and strains of stock, and as a result presented a confused picture as to the possible cause of the disease.

Since an unusual situation existed during the year with respect to the quality of ingredients available for feeding purposes in comparison with previous years, indication tests were inaugurated with the hope that they would shed some light on the problem. The rations comprised various types of mixtures where some particular ingredient formed the major portion of the mixture and in some cases a deficiency ration was employed.

In only one group did the typical nervous symptoms and lesions develop. This group had been fed a ration composed of:

72.4	pounds	yellow corn meal
22.0	"	dried skim milk
2.2	"	calcium carbonate
1.1	"	sodium chloride
2.2	"	cod-liver oil

This ration produced a mortality in this group of chicks of 50 per cent., half of which showed the typical brain lesions upon autopsy.

The high corn ration was compounded on the basis that the 1935 corn crop seemed to be somewhat unusual from the standpoint of uniformity in view of its poor quality as evidenced by poor germination and high moisture content. A repeat trial is being inaugurated for confirmation purposes.

[1] Published by permission of the Director of Research as Contribution No. 495 of the Rhode Island Agricultural Experiment Station.

[2] A. Pappenheimer and M. Goettsch, *Jour. Exp. Medicine*, 53: p. 11, 1931.

[3] Glen L. Dunlap, *Jour. Amer. Vet. Med. Assoc.*, 80, n. s. Vol. 33, No. 6, pp. 880–885.

SUMMARY

Chicks fed a diet in which corn was the principal ingredient, in contrast to other rations employed, were the only ones in which the typical nervous disorders were noted and typical brain lesions observed upon autopsy.

The preliminary data at hand, although not of a definite nature, would seem to indicate that some factor or factors of the corn used were responsible or at least contributory to this disorder.

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THE INSECT VECTOR FOR THE NATURAL TRANSMISSION OF EPERYTHROZOON COCCOIDES IN MICE

IT has been recognized since 1930[1] that the white mice used for experimental purposes in this country may harbor a latent blood infection which is distinct from *Bartonella muris* but which like it is activated by splenectomy. The infecting organism, *Eperythrozoon coccoides,* was first described two years previously in Berlin.[2] It is a small ring-shaped body, usually less than 1 μ in diameter, staining reddish blue by Giemsa or Wright's stain. It appears in great numbers on the red cells as well as in the plasma within one to several days following splenectomy of the carrier animal. The fact that normal carrier mice are apparently little affected by the organism and that (unlike *Bartonella muris* infections) the multiplication of the parasite following splenectomy results in no marked pathological change, has led to a rather general oversight of the possible influence of the *Eperythrozoon* on experimental results. Certain changes in the blood picture and in the size and histology of the spleen in the infected animal have recently been demonstrated.[3,4] These deviations from normal may be sufficiently great at times to be significant when exact studies on the relation of the spleen to disease and resistance are under investigation.

The presence or absence of the latent *Eperythrozoon* infection can be demonstrated by splenectomy. The uninfected mice when kept isolated from other stock will remain free of the organism. The intraperitoneal injection of blood from a carrier mouse or from one showing active infection serves as a simple method for laboratory transmission. The means for the natural transmisison of the parasite from mouse to mouse has not been recorded up to the present time. The fact that the *Eperythrozoon* is a blood parasite and that it spreads gradually but surely through a colony of mice kept under the usual laboratory conditions points to the rôle of an insect vector in its natural transmission. Negative results have been reported with the rat louse and with fleas.[1,5,6] By analogy with the natural transmission of *Bartonella muris* this vector might be suspected to be the mouse louse.[7] A series of simple experiments revealed that the louse *Polyplax serrata* does indeed serve as the insect vector of *Eperythrozoon coccoides* from mouse to mouse.

A group of mice known to be free from latent *Eperythrozoon* infection was splenectomized and kept in rigid quarantine. These served as the susceptible hosts for the transmission tests. The more commonly occurring ectoparasites in an infected colony of mice were identified and used for the transmission experiments. There were no fleas in this infected colony. The two species of mites tested, *Myobia musculi* and *Mycoptes musculinus,* failed to transmit the *Eperythrozoon* by feeding on the test host.

The experience with the louse *Polyplax serrata* was quite different. In each of eleven experiments the adults and nymphs were shown to be capable of transmitting *Eperythrozoon coccoides* to the uninfected test host by feeding on it. The organisms appeared in the blood of these splenectomized animals in from nine to seventeen days, depending on the conditions of the experiment. In two other trials in which the adult lice were kept away from the host for several hours, transmission failed to take place. The nymphs from the same host, however, that were starved for the same length of time were capable of transmitting *Eperythrozoon.* These results suggest that the strong digestive fluids of the adult louse destroy the organism, while the less active alimentary juices of the nymph permit longer survival. The details of these and other experiments are to be reported later.

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VISCERAL LEISHMANIASIS IN BRAZIL

THE viscerotomy service of The Rockefeller Foundation detected, between March, 1932, and July, 1936, eighty-five specimens of liver containing leishmania bodies morphologically identical with those of *Leishmania donovani,* which produces Indian and Mediterranean kala-azar. These bodies and the liver lesions

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