ian transmissions were on extremely high frequencies producing the so-called ultra-short waves. It is again curious that we should have closed a cycle. The first waves used for communication were hundreds or thousands of times longer than those which Hertz produced. Gradually long-distance communication came back to the short waves. And to-day television conditions require our return to ultra-short waves. From Hertz to television seems a long cycle, and yet this is another instance where pure research first produced the agency which, generations later, was both needed and available.

Mr. Dunlap properly emphasizes the position of Marconi as a sort of bridge between the rudimentary foundations of electromagnetic wave technique and our present evolved and complicated radio art. He makes it clear, however, that Marconi was far from a passive medium. His personal bravery and tenacity, combined with scientific thoroughness and faith in technical development, led Marconi step by step from the rôle of the world's first radio amateur to a position as its leading radio engineer.

As Mr. Dunlap indicates, it is difficult for us to evaluate fairly the contributions of Marconi, since we are too close to some of them and have only inklings of others. So far as radio telegraphy and telephony are concerned, we have a fair idea of the outstanding character of Marconi's contribution. In the nascent arts of radio facsimile and television transmissions, we can not clearly view the vortex of progress in which we whirl. In more remote applications of radio technique, as, for example, meteorological studies, we have only a foreshadowing of what Marconi's work on short-wave transmission may ultimately mean.

We have learned much of the electrical constitution of the upper atmosphere by radio methods—and as Mr. Dunlap points out, the short wave experiments of Marconi here blazed a trail the ultimate significance of which few have appreciated.

It may be added that Marconi is presented as a prepossessing modest personality, never losing touch with humanity and the common things of everyday life. The major premise of the book, which is fully proven, is that men of the type of Marconi and his predecessors are beacons lighting the world toward what may be brighter days and constituting constructive forces in an age too prone to the destruction of human values.

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SPECIAL ARTICLES

JAPANESE B ENCEPHALITIS VIRUS: ITS DIFFERENTIATION FROM ST. LOUIS ENCEPHALITIS VIRUS AND RELATIONSHIP TO LOUPING-ILL VIRUS

WHEN St. Louis encephalitis was first recognized in 1933, it was likened both clinically and epidemiologically to summer encephalitis B of Japan.[1] Later, however, when the St. Louis virus agent was discovered and established in mice,[2] it proved to be neutralized specifically by sera of St. Louis but not Japanese convalescents.[3] Now, within the past two years, the virus agent of the Japanese disease has been established in mice[4] and found by repeated cross serological and resistance tests to be immunologically distinct from the St. Louis virus.[5, 6]

Moreover, the reactions of the two viruses in animal species prove to be readily distinguishable. The St. Louis virus is pathogenic apparently only for mice and *Macacus rhesus* monkeys. Mice injected with the virus usually show as a first sign of disease tremors and convulsions, fail to show virus regularly in the blood stream, and are resistant to intraperitoneal or subcutaneous injections of all save maximum doses. Monkeys injected intracerebrally with massive doses are relatively resistant, less than 50 per cent. showing a mild, non-fatal encephalitis. Japanese virus, on the other hand, is pathogenic for mice, *Macacus rhesus* monkeys and sheep. Mice injected with the virus generally show paralysis of extremities as a first sign of disease, carry virus in the blood stream during the early stages of infection, and are relatively susceptible to intraperitoneal or subcutaneous injections. Monkeys given an intracerebral injection of a small quantity of virus develop an acute, fatal encephalitis characterized by cerebellar incoordination and specific necrosis of the Purkinje cells. Sheep injected intracerebrally or intranasally develop an acute, fatal encephalitis but appear resistant to subcutaneous injection.

[1] J. P. Leake, *Jour. Am. Med. Assn.*, 101: 928, 1933; 103: 728, 1934.

[2] L. T. Webster and G. L. Fite, SCIENCE, 78: 463, 1933; *Jour. Exp. Med.*, 61: 103, 411. 1935.

[3] L. T. Webster and G. L. Fite, SCIENCE, 79: 254, 1934; *Jour. Exp. Med.*, 62: 827, 1935.

[4] The first reports in Japanese journals are cited in later publications by S. Kasahara *et al.*, Kitasato Arch. *Exp. Med.*, 13: 48, 248, 1936; H. Hashimoto *et al.*, *Jour. Am. Med. Assn.*, 106: 1266, 1936; T. Taniguchi *et al.*, *Jap. Jour. Exp. Med.*, 14: 185, 1936; R. Kaneko *et al.*, *Klin. Woch.*, 15: 674, 1936; R. Kawamura *et al.*, *Kitasato Arch. Exp. Med.*, 13: 281, 1936.

[5] R. Kawamura *et al.*, *Kitasato Arch. Exp. Med.*, 13: 281, 1936; *Arch. Path.*, 22: 510, 1936; M. Kudo *et al.*, *Jour. Immunol.*, 32: 129, 1937; Y. Kawakita, personal communication.

[6] Unpublished tests on six Japanese strains by the author.

These reactions of Japanese virus in animal species, while differing sharply from those of St. Louis virus, are indistinguishable from those of louping-ill virus.[7] Serologically, however, Japanese B and louping-ill viruses appear unrelated.

Present knowledge indicates, therefore, that St. Louis and Japanese B encephalitis viruses, and hence the two diseases, are distinct. Japanese B and louping-ill viruses, however, are very similar to each other and also to the virus of Australian X disease as described.[8] Accordingly, possible relationships among louping-ill encephalitis of sheep in Scotland, apparently transferable to man,[9] Japanese summer encephalitis of man and Australian X disease should be further explored.

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DIETARY CONTROL IN EXPERIMENTAL COCCIDIOSIS

THERE is no effective control known for coccidiosis except stringent sanitary measures based upon an understanding of the life cycle of the protozoon. For a number of years the writer has been investigating the possible relation of diet to certain aspects of coccidian infection, particularly in the white rat. Recently efforts have been made to construct rations for the chick that would ameliorate the severity of caecal coccidiosis occasioned by *Eimeria tenella,* which seems to be by far the most virulent of the types found in this bird. Only three of the most striking experiments will be reported here in brief, but a completer report of the experiments will appear later.

One lot of control chicks was kept on a commercial ration that it had received from time of hatching. The ration was 40 per cent. yellow corn-meal, 30 per cent. wheat flour middlings, 5 per cent. ground hulled oats, 5 per cent. powdered skim-milk, 5 per cent. wheat bran and 15 per cent. various amounts of salt, cod liver oil, oyster shell, meat and bone meal, alfalfa meal, bone meal and charcoal. Another lot was given the same mixture until it was fourteen days old, when it commenced to receive the following test mixture: yellow corn-meal, 25 parts; meat and bone meal, 6; soybean meal (with nutty flavor), 22.5; hulled oats, 20; ground wheat, 10; wheat bran, 5; ground oyster shell, 1; salt, 1; charcoal, 1; beet sugar, 6.5; cod liver oil, 2. After nine days on the test ration, both controls and tests were infected with 50,000 sporulated oocysts of *Eimeria tenella* per bird. As a result of this infection nine out of eighteen controls succumbed, but only one out of thirteen in the test series.

One lot of chicks was maintained throughout on the control ration previously described. Another lot received the same until it was eight days of age, when its diet was changed to the following, by parts: yellow corn-meal, 32; hulled oats, 20; soybean meal (the kind with nutty flavor), 16; fish meal, 65 per cent. protein, 4; meat and bone meal, 4; wheat bran, 5; wheat flour middlings, 10; oyster shell, 2; alfalfa meal, 2; commercial casein, 1; salt, 1; charcoal, 1; cod liver oil, 2. After the second lot had been on the test diet for fourteen days, each bird in both lots received 60,000 *Eimeria tenella* oocysts. As a direct result of this infection nineteen out of twenty-four control birds succumbed, but only one out of twenty-four in the test series.

The two preceding test rations were constructed with the idea of eliminating powdered skim-milk and butter-milk altogether from the ration and reducing the amount of wheat flour middlings well below that in the control diet. Certain previous experiments had led us to suspect that these materials in certain combinations, particularly the skim-milk and butter-milk, were the chief inciters of ill effects in infected chicks. The chicks made good gains on both test diets, but four on the second test diet developed a partial paralysis of the type in which the toes turn inwards. They recovered within two days when placed on the control ration again. Since skim-milk has been shown to be a preventative for this type of paralysis, and since the second ration was shown to be slightly deficient in the paralysis-causing factor, a ration was constructed in which a small amount of skim-milk was included and the grain element somewhat varied. It was, by parts, as follows: yellow corn-meal, 40; hulled oats, 10; ground whole oats, 5.5; barley, 5; ground whole wheat, 5; wheat bran, 5; wheat flour middlings, 5; soybean meal, 10.5; meat and bone meal, 4; fish meal (65 per cent. protein), 2; oyster shell, 2; salt, 1; skim-milk, 2; alfalfa meal, 3.

The chicks put on the latter ration at the age of thirteen days made excellent growth during the next fourteen days. At the end of this time twenty-eight chicks on the control ration and thirty-three on the test ration were each given a forced feeding of 80,000 sporulated oocysts of *Eimeria tenella.* As a result of this infection seventeen chicks on the commercial or control ration succumbed, or about 60.7 per cent. There were only six fatalities in the group on the test diet, a mortality of only 18.7 per cent. No paralysis appeared in this group.

These and other experiments by the writer prove that coccidiosis is a disease controllable through the diet. It appears that the extreme severity manifested

[7] W. A. Pool, *Proc. Roy. Soc. Med.*, 27: 707, 1934; E. W. Hurst, *Jour. Comp. Path.*, 44: 231, 1931.

[8] J. R. Perdrau, *Jour. Path. and Bact.*, 42: 59, 1936.

[9] T. M. Rivers and F. F. Schwentker, *Jour. Exp. Med.*, 59: 669, 1934.