used for horses inoculated with virus. Twenty inoculated horses have developed symptoms of encephalomyelitis and either died or were destroyed while in this corral. All new horses were purposely placed in this corral without any sanitary precautions whatsoever, other than removal of manure. The same feed racks and watering troughs were used. No spontaneous cases of encephalomyelitis have occurred and all the presumably exposed horses proved susceptible to intranasal or intracerebral inoculation of virus one to four months later.

The existence of virus carriers among otherwise healthy horses is suspected. In several instances during the past two years, previously unexposed horses have developed typical cases of encephalomyelitis when brought to ranches having a history of encephalomyelitis among the horse population from six to fifteen months prior to the arrival of the new stock, the old horse stock remaining on the ranch not showing any clinical evidence of the disease during the previous outbreak or during the intercurrent period. In one instance all the new arrivals developed the disease in seven to ten days after arrival, while on other ranches one or more new additions developed the disease soon after arrival on the premises.

The period of incubation in experimental horses following intranasal exposure to virus agrees with the reports from practising veterinarians that additional cases of equine encephalomyelitis usually occur in seven to ten days after the first case came to clinical notice.

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INHERITANCE OF RESISTANCE TO FOWL PARALYSIS (NEUROLYMPHOMATOSIS GALLINARUM)

ALTHOUGH a number of investigators have reported that fowl paralysis can be transmitted by inoculation, only a comparatively small percentage of the inoculated birds develop clinical symptoms of the disease. Moreover, a certain percentage of the non-inoculated controls also develop fowl paralysis. These facts have rendered the experimental study of this disease very difficult.

In a previous report,[1] data obtained from a spontaneous outbreak of fowl paralysis have been presented. It was found that some families were entirely free from paralysis, while others showed a high incidence of the disease, thus indicating the existence in the domestic fowl of hereditary differences in resistance to fowl paralysis.

In order to obtain further data on this phase of the problem, inoculation experiments were started with chicks from known "susceptible" lines. Approximately one half of the chicks were inoculated at 1 to 7 days of age with emulsions of tissue from paralyzed birds. The inoculated and non-inoculated chicks were kept under identical conditions in the same house. Sixty-three out of 202 chicks, or 31.4 per cent., developed paralysis. There was no difference between the inoculated and non-inoculated groups of chicks in the incidence of fowl paralysis. The post-mortem observations made on some of the paralyzed birds have been described elsewhere.[2]

When the chicks were grouped into families the following results were obtained: four families, comprising 27 chicks, were entirely free from paralysis; eight families, which showed a close approximation to a 1:1 ratio, gave 49 normal to 42 paralyzed chicks; and five families showed a 3:1 ratio (62 normal to 16 paralyzed). No large family of chicks, all of which became paralyzed, was obtained, but in one family of 6 individuals 5 chicks developed paralysis. The chick which did not develop paralysis was killed at 147 days of age.

These results indicate that resistance to fowl paralysis depends upon a dominant gene, and points to the conclusion that in the stock used only one pair of genes is involved. No evidence of sex linkage was observed.

It appears that the proportion of paralyzed to normal birds in any transmission experiment would depend on the genetic constitution of the chicks as regards resistance or susceptibility to fowl paralysis. Hence a genetic analysis of the material is necessary in evaluating the results of fowl paralysis transmission experiments.

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[1] V. S. Asmundson and Jacob Biely, *Can. Jour. Research*, 6: 171–176, 1932.

[2] Jacob Biely, V. Elvira Palmer and I. Michael Lerner, *Can. Jour. Research*. In press.

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