boiling potash. While the floor may show rather large vesiculate structure and the walls and roof very fine vesiculations, it is only the roof that is generally reinforced by many imbedded solid particles. The roof also is joined to the top of the walls with a preformed joint that finally allows the roof to rise up and fall off, leaving the wall to stand as rounded conspicuous white line about the nearly invisible floor for months' duration, after the young have escaped. The roof also may be prolonged laterally as more or less extensive rim or eaves just above the breaking joint; the top of the wall may also be somewhat prolonged laterally under the above named eaves of the roof.

The capsules of different species present differences in size and in outline, but especially in character of the reinforcement of the roof as well in extent or absence of the rim. Moreover, in any one species the capsules vary in size and form according to size of the females making them and differ in nature and size of solid objects used for roof reinforcement according to character of substrate on which the animal feeds in different localities; or even in the same locality, whether on bottom or on floating objects.

However, these capsules of the Neritidae, which are comparable to eggshells of birds and reptiles as being secretions placed about an albuminous mass in which one or more ova have been placed, may aid in classification.

E. A. ANDREWS

JOHNS HOPKINS UNIVERSITY

RESPIRATORY INFECTION IN EQUINE ENCEPHALOMYELITIS

EQUINE encephalomyelitis has been transmitted to two horses by single instillations of 50 cc of Berkefeld filtered virus into the nostril. The stock strain of virus used was originally obtained from the brain of a horse which was killed when prostrate with encephalomyelitis during the epidemic in Western Nevada in September, 1931. This strain of virus has been maintained by routine passage through guinea-pigs and horses for the past eighteen months.

For inoculation of the horses, a one per cent. Locke's solution emulsion was prepared with the brain and liver virus from three guinea-pigs. This was allowed to extract at 5° C. for three hours, with occasional agitation. The virus emulsion was then centrifuged and filtered through Berkefeld N filters (7 to 8 lb. test) and the filtrates pooled. Aerobic and anaerobic cultures made from the filtrates did not show any bacterial growth during ten days' incubation. The pH of the pooled filtrate was 7.2.

Fifty cubic centimeters of the Berkefeld filtered virus was instilled into one nostril of a horse. Seven days later, this horse showed a temperature of 105° F. and marked congestion of the conjunctiva. Definite symptoms of encephalomyelitis were present on the tenth day; namely—depression, incoordination and receding fever. Motor paralysis and prostration occurred on the eleventh day, when the animal was sacrificed for humane reasons. The virus was subsequently demonstrated in the brain of this horse by inoculation of guinea-pigs.

The second horse was inoculated with the same strain of virus recaptured from the first horse by guinea-pig passage, guinea-pig brain virus being used. The same experimental conditions were duplicated, namely, the method of preparing the virus emulsion and filtration.

Horse number two received 50 cc of Berkefeld filtered virus in one nostril. Six days later this horse had a temperature of 104° F. On the seventh day, the temperature was 105° F. and accompanied by profuse nasal discharge and congestion of the conjunctiva. On the eighth day depression and preparalytic symptoms appeared, the temperature receding to 103.8°. Motor paralysis, amaurosis and prostration occurred on the ninth day, when the subject was destroyed. Virus was recaptured from the brain and cord by guinea-pig passage.

As controls on each of the two intranasal infection experiments, guinea-pigs and another horse were injected intracranially under anesthesia with the same Berkefeld filtered virus. All the control guinea-pigs developed characteristic symptoms of encephalomyelitis and either had died or were destroyed by the fifth day.

The two control horses injected intracranially with virus developed typical symptoms of encephalomyelitis on the sixth day after injection. One was prostrate on the eighth day and was destroyed. The other lingered until the twelfth day, became prostrate and was sacrificed. This individual, being an old horse, presumably had some natural resistance.

The incubation period of the disease in horses which received virus in the nostril was two and three days longer than that following intracranial injection.

Postmortem examination of the two horses which received virus in the nostril showed hemorrhagic infiltration at the base of the brain tending to follow the olfactory tract and extending posteriorly to the piriform lobes. The spinal fluid in one instance was very turbid and had a large mononuclear count of 680 per cmm. Polyneuclear leucocytes were not observed. Test for sugar was negative.

It appears that inanimate objects, such as feed racks and watering troughs, are not vectors of infection. During the past year one corral has been

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.

LYMAN R. VAWTER
EDWARD RECORDS
NEVADA AGRICULTURAL EXPERIMENT STATION, RENO

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.

JACOB BIELY
V. ELVIRA PALMER
I. MICHAEL LERNER
V. S. ASMUNDSON
THE UNIVERSITY OF BRITISH COLUMBIA

[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.

BOOKS RECEIVED

COLLIN, J. E. *Diptera of Patagonia and South Chile.* Part IV: *Empididae.* Pp. viii + 334. 74 figures. British Museum. Oxford University Press.

EINSTEIN, ALBERT. *On the Method of Theoretical Physics.* Pp. 20. Oxford University Press. $0.50.

GARSTANG, WALTER. *Report on the Tunicata.* Part I: *Doliolida.* Pp. 56. Illustrated. British Museum. Oxford University Press. 5s.

ORR, A. P. *Physical and Chemical Conditions in the Sea in the Neighborhood of the Great Barrier Reef.* Pp. 49. British Museum, Oxford University Press. 5s.

TILLYARD, R. J. *The Panorpoid Complex in the British Rhætic and Lias.* 31 figures. British Museum, Oxford University Press. 5s.