leaves of *N. glutinosa*. The results of five other experiments showed a similar inactivation.

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THE ETIOLOGY OF A RESPIRATORY DISEASE OF CHICKENS

THE occurrence of a severe type of respiratory infection in chickens, which was believed to be distinct and previously undifferentiated, was observed by Pistor, Hoffman, Beach and Schalm[1] in California early in 1933.

The disease is easily transmitted either by inoculation with exudative material or by contact exposure. In either case a nasal discharge develops which is followed by an edematous inflammation of the periorbital tissues, a purulent conjunctivitis and often a distention of the sinuses of the head with exudate. The infection may spread to the trachea, bronchii and lungs, causing rales, dyspnea, coughing, and sometimes death from suffocation. Infection by contact exposure may start as a tracheitis and bronchitis. The appetite diminishes or ceases with the onset of symptoms, resulting in a rapid and extreme emaciation. If the affected fowl does not die in the early stages of the disease, the edematous swellings of the head and the distention of the sinuses soon disappear, but a nasal discharge and a mild conjunctivitis and tracheitis may persist for many days, and finally the fowl may die in a greatly emaciated state or recovery may slowly occur.

Smears of the various exudates stained by Giemsa's or by Gram's method have quite consistently shown the presence of small bacilli, which vary in length and stain, in the majority of cases, only at the poles. This organism is not typical of the pasteurella group. Another organism, a vibrio, is also consistently found, sometimes in great profusion, in the nasal, conjunctival and tracheal exudates but not as yet in the edematous exudate.

The various exudates have been streaked on plates of the common culture media, including horse-blood agar plates which have been incubated aerobically, in an atmosphere containing 10 per cent. CO_2 and also sealed with modeling clay, the method which Nelson[2] found suitable for cultivating the causative organism of a coryza of chickens. Growth of an organism pathogenic for chickens has been obtained only on the blood agar plates incubated in an atmosphere at 10 per cent. CO_2. On such plates there has appeared, often in pure culture, very small (pinpoint) discrete colonies, which, when washed off and inoculated intranasally and intratracheally into susceptible chickens, from 6 weeks to six months old, have in 45 out of 48 cases produced symptoms varying from a slight nasal discharge to all those observed in fowls inoculated with exudate.

The incubation period of the culture-induced disease varies from 15 to 48 hours, which is the same as observed in the exudate-induced disease. Smears of the various exudates of the culture-induced disease have consistently shown the presence of the bacillus but never the vibrio. The duration of the disease induced by culture has varied between 2 and 22 days, a much less prolonged course than in exudate-induced infection.

The organism appears to belong to the class of hemophilic bacteria, for attempts to grow it in the absence of hemoglobin have been unsuccessful, and the influence of the "V" factor on its growth is readily observed on contaminated plates by the larger size and greater opacity (satellite phenomenon) of the colonies nearest the contaminant. In smears made after 24 hours incubation, the organism is found to be a small Gram negative rod, which has a tendency to form long filaments; after 48 hours' incubation, a few fragments and single rods, but rarely filaments, are observed and, after incubation for 60 or more hours, only fragments of indefinite shape, which stain faintly, are found. Transfers made after the organism has fragmented give rise to rods and filaments again in the transplant. The pathogenicity of the organism is not affected by the fragmentation. Polar staining of the organism in cultures has rarely been observed, but lightly stained areas are commonly seen in the filaments. The organism has grown aerobically in horse serum at the base of blood agar slants, and after prolonged incubation slight colonization has occurred on the surface of the slant.

The vibrio has grown in the horse serum at the base of blood agar slants, but up to the present time has not been isolated in pure culture. Until this is accomplished, it will be impossible to conclude whether it is concerned in the etiology of the disease.

Fowls after recovery from exudate-induced disease have reacted in a variable manner to subsequent inoculation with exudate; some have been refractory, while others have shown no resistance other than that the disease produced was less severe than that in the controls. Fowls that have recovered from culture-induced disease have, in 8 of 10 trials, proved refractory to a subsequent inoculation with cultures. Such fowls, however, have been susceptible to inoculation with exudate, although the resultant disease has been less severe than that in the controls, and, with one exception, edema of the periorbital region has not occurred.

The failure of infection with the organism to immunize against the exudate is not regarded as indi-

[1] W. J. Pistor, A. H. Hoffman, J. R. Beach and O. W. Schalm, *Nulaid News*, 11: 7, 1933.
[2] J. B. Nelson, *Jour. Exp. Med.*, 58: 289, 1933.

cating that the organism is not the etiological factor in the exudate, because the infection with the exudate itself did not always protect against reinjection. It seems more logical that the pathogenicity of the organism has been reduced by cultivation so that it is incapable of producing a high degree of immunity.

The clinical manifestations of the disease under discussion closely resemble those described by Delaplane, Stuart and Bunyea[3] of a similar disease of chickens in Rhode Island and it seems not unlikely that the two may be identical. There are also points of similarity between the disease and the coryza studied by Nelson,[2] particularly his type III. The organism that has been isolated and which is believed to be an etiological factor of the disease appears to differ in some aspects from the one identified by Nelson[2] as the cause of a coryza of chickens. Further studies, however, may show that the two belong to the same species.

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THE TREATMENT OF DECIDUOUS FRUIT TREES AND NUT TREES INFECTED BY PHYMATOTRICHUM OMNIVORUM WITH AMMONIUM COMPOUNDS

THE disease known as cotton (or Texas) root-rot causes serious losses in most of the dicotyledonous crop plants grown throughout its rather restricted range in the semi-arid Southwest. One of the most serious aspects of its depredations is the destruction of many valuable long-lived perennials, such as deciduous fruit trees, nut trees and ornamental trees and shrubs. The root-rot fungus persists in the soil and, starting from one or more points in the orchard, advances in ever-widening circles until the whole planting has been destroyed. Rotation with resistant or immune species, the most successful method of control in case of field crops, is not practical in the case of tree crops.

For the past five years the author has been experimenting with visibly infected trees, using a variety of chemical and other treatments. Many of the early results were not particularly encouraging, as the root system of attacked trees is usually very seriously damaged before symptoms are clearly visible in the foliage. Some promising treatments have proven disappointing in the field because the chemicals used were too active when applied to our soils and quickly became converted to relatively insoluble and innocuous forms.

Results of the past two years, however, indicate that two of the treatments will prove successful in checking and overcoming the ravages of root-rot in some tree crops. Whether they will be effective in case of our most susceptible trees remains to be seen. The two treatments which have proven most successful are rather heavy applications of ammonium sulfate or ammonium hydrate diluted to a safe concentration with water. Ammonium hydrate was reported by Neal *et al.*[1] to be successful in killing the root-rot fungus without killing the host plant (cotton). Various workers have tried different nitrogenous commercial fertilizers in moderate amounts without finding any definite benefit.

Since both chemicals, especially the hydrate, are very toxic to the trees if applied in too great a concentration, a special method was evolved for the safe and accurate applications of these chemicals under field conditions. The choice of these two chemicals seems particularly fortunate, as they are relatively inexpensive and easily obtained, and both leave a large residue of quickly available nitrogen in the soil, for nitrification of the ammonia in either case is well advanced in three or four weeks. The strong stimulating effect of the nitrogen appears to be essential to recovery, as trees visibly affected by root-rot have greatly weakened and decayed root systems, which must be quickly replaced if the tree is to survive. The ammonium hydrate has proven somewhat disagreeable to handle on account of its volatile nature, but it is perhaps somewhat better for treating very badly diseased trees.

The outstanding success has been in treating affected pecan trees in the Yuma Valley. Pecan trees are usually visibly affected a year before they die from root-rot, while deciduous fruit trees often succumb without warning. The largest number of the treated trees were in two orchards. In the first orchard no treated trees were lost; in the second grove about 40 per cent. of the treated trees died, but many of the others showed great improvement. The higher mortality is accounted for by the fact that the very worst trees were chosen for treatment and the general condition of the grove was less favorable. Experiments on deciduous fruit trees in Yavapai County (altitude 3,000 to 3,500 feet) have been complicated by the great variability of the orchard soils of that district and the difficulty of treating a sufficient number of affected trees to secure reliable data, but results have been mainly encouraging.

While experiments have been conducted largely with badly diseased trees, it is obvious that the effect of treatment should be much more valuable to the commercial grower if applied to trees only slightly affected or standing in the path of the advancing

[1] D. C. Neal, R. E. Wester and K. C. Gunn, "Treatment of Cotton Root-rot with Ammonia," SCIENCE, n. s., 75: 139–140, January 29, 1932.

[3] J. P. Delaplane, H. O. Stuart and H. Bunyea, *Jour. Amer. Vet. Med. Assoc.*, 82: 772, 1933.