Large Black dam by a pedigree Tamworth sire. When first examined she had produced two litters by a Tamworth boar (Boar *A*), and has since given birth to a third litter by another Tamworth (Boar *B*) which is a half-brother, on the sire's side, to Boar *A*. Such details as can be obtained on the occurrence of offspring with pigmented bones in these three litters are given in the following table :

| Litter | No. in litter | Pigmented | Normal | No information | Sire |
|---|---|---|---|---|---|
| 1 | 10 | 1 | – | 9 | Boar *A* |
| 2 | 10 | 2 | 3 | 5 | Boar *A* |
| 3 | 7 | 2 | 5 | 0 | Boar *B* |

A full sister to the affected sow, mated to Boar *B*, produced 14 pigs none of which had coloured bones. Both boars appear to be clinically normal.

The porphyria sow shows no symptoms of the photosensitivity which often accompanies congenital porphyria ; but as she is black all over, this is not unexpected.

The sow and Boar *A* have both been purchased by this Research Station for breeding experiments and chemical studies.

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## Treatment of a Virus Disease of Chickens with Sulphonamides

BLAKEMORE has published[1] an account of a disease produced in young chicks by the inoculation of suspensions of tissues from typical cases of fowl paralysis (neurolymphomatosis). While the lesions were superficially unlike those of typical fowl paralysis, the course and pathology suggested that the experimentally produced disease was an acute form of fowl paralysis. Glover[2] produced a similar disease in chickens by the inoculation of tissue suspensions from a turkey affected with neurolymphomatosis. It was shown that the infective agent was filterable[3]. During the course of experiments which have been carried out in this Laboratory, a number of strains of virus which originated from paralysed fowls have been studied. Intraperitoneal inoculation of young chicks with virus results in the production of lesions which are primarily necrotic and are most conspicuous in the liver and heart. Visceral lesions become visible to the naked eye 48–72 hours after inoculation. They increase in extent and frequently reach maximum severity 6–8 days after infection. In the experiments recorded here, all chicks were killed 5–10 days after infection. Diagnosis was based upon the presence or absence of macroscopic lesions.

The influence of sulphonamides in the treatment of this disease has been striking. For ease of administration the sulphonamides have been mixed into the food or dissolved in the drinking water. Chickens treated continuously from the time of inoculation until the time of killing failed to develop lesions and their tissues proved to be non-infective. Treatment commenced at the time gross lesions were established resulted in the arrest and resolution of the disease process. A total of 138 chicks inoculated with eighteen strains of virus have been treated with sulphadiazine mixed in the food at a level of $\frac{1}{24}$–$\frac{1}{8}$ gm. per oz. None has developed macroscopic lesions. Of 117 control infected untreated chicks, 101 showed gross lesions on post-mortem examination.

The disease is sensitive to treatment with most of the sulphonamides in common use. As judged by the smallest dose necessary to prevent the development of gross lesions, the value in descending order is sulphadiazine, sulphamezathine, sulphathiazole, sulphapyridine, sulphaguanidine. The toxicity of sulphanilamide to chicks is such that it proved valueless.

The action of sulphonamides in this condition appears to be similar to that in bacterial infections. It is neutralized by *p*-aminobenzoic acid. A virus-containing suspension remained infective for more than twenty-four hours when prepared in a saturated aqueous solution of sulphathiazole and kept at room temperature.

The observation that this virus disease responds to sulphonamide treatment is of special interest, since apart from lymphogranuloma inguinale and possibly trachoma, virus diseases have proved refractory to chemotherapy.

The effect of sulphonamide treatment on the clinical condition and infectivity of fowls affected with typical fowl paralysis is being studied. It is clear that much careful work will be required in order to determine if means exist whereby sulphonamides might be employed in an attempt to control fowl paralysis. The low economic value of the individual fowl imposes obvious limitations upon the medical treatment of the diseases of poultry.

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## Urease Activity and Ascorbic Acid

IN a recent note, Elson[1] has shown that ascorbic acid at low concentrations inhibits urease activity, and that this inhibition is prevented by addition of cysteine. Quastel[2] suggests that the inhibition of the activity by ascorbic acid is due to the oxidized form of ascorbic acid, namely, the diketone (dehydroascorbic acid) and not to the ascorbic acid itself. The disappearance of the inhibition on the addition of cysteine has been attributed to the reduction of the dehydroascorbic acid to the inert ascorbic acid. It seemed to us more probable that the action of ascorbic acid is not connected with either ascorbic acid or dehydroascorbic acid, but with the oxidation of the vitamin by traces of heavy metals like copper present in the reaction mixture. For several years we have been investigating the influence of ascorbic acid on enzymes, and we have obtained results showing that the vitamin as such or the dehydroascorbic acid has very little influence on the enzymes (phosphatases and amylase) ; but when the vitamin is oxidized by traces of copper present in the reaction mixture the enzyme is inhibited to a considerable extent. It was also found that substances which inhibit the oxidation of ascorbic acid annul the inhibition by the vitamin. In the light of these results we have now investigated the influence of ascorbic acid,