

THE DOMESTIC PIGEON AS A POSSIBLE CARRIER OF NEWCASTLE DISEASE.

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During a Newcastle disease epizootic, the question may arise whether pigeons are responsible for the spread of infection. Pigeons are frequently closely associated with poultry; yet usually enjoy unrestricted movement. Racing pigeons are often taken hundreds of miles for release and may be taken from an infected to a free area.

There are several reports in the literature unsupported by adequate evidence that the pigeon is susceptible to the natural disease. However, Doyle (1927) showed that the pigeon is highly susceptible when injected intramuscularly, but he failed to produce the disease by natural means. One spontaneous case of Newcastle disease in a pigeon has been reported by Iyer (1939).

In South Africa the pigeon was widely used as an aid to diagnosis in 1945, being used primarily because the inability to contract the disease naturally would eliminate a possible serious source of error in diagnosis (Kaschula *et al.*, 1946).

The object of this report is to record the results of an experiment to determine whether pigeons which had been dosed *per os* with virulent virus, would excrete virus in their faeces or develop an immunity.

METHODS AND MATERIALS.

The methods were similar to those used to determine that the Cape sparrow and two species of wild doves may be active carriers of virus (Kaschula, 1950).

A consignment of pigeons was obtained from Pretoria, an area free from the disease where live virus immunization had not been practised. A random selection was found to be fully susceptible to infection by intramuscular injection.

The virus used was a recently isolated virulent strain propagated in the allantoic sac of fertile hens eggs after 8 days pre-incubation at 37° C.

EXPERIMENTAL.

Four pigeons were placed in a previously sterilized cage. They were transferred daily into other sterilized cages and supplied with fresh food and water. Prior to being dosed with virus a blood sample was collected from each for an H.I. test on the serum. These were all negative. Each pigeon received *per os* 0.1 c.c. of allantoic fluid of a freshly isolated Cape virulent strain of Newcastle disease virus. The pooled faeces passed from 8.00 to 10.00 a.m. each morning were collected in a sterile container mixed with saline and centrifuged. The

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supernatant fluid was decanted and after treatment with streptomycin and penicillin (5,000 units of each per 1 c.c.), 0.1 c.c. was injected into the allantoic sac of each of six eggs containing 8 day embryos. Each egg that died was tested by the slide H.A. test for virus, and subinoculated into other eggs to confirm the presence or absence of virus. The examinations were continued daily for 18 days.

RESULTS.

No pigeon showed any clinical signs of illness, but material collected on the twelfth day killed four out of six embryos. Newcastle disease virus was isolated from the allantoic fluid of these eggs, being identified by subinoculation into other fertile eggs, by haemagglutination, and by haemagglutination-inhibition by specific immune serum.

Two weeks after the last faeces specimens were tested (i.e. on the 32nd day after dosage with virus) the four pigeons were again submitted to the H.I. test. All were negative.

It was then decided to challenge the immunity to intramuscular infection by the same strain of virus. They received 0.1 c.c. of the same virulent strain of virus intramuscularly.

Two of the four pigeons became ill on the fifth day. One died on the eighth day but the other recovered. The third and fourth pigeons did not react. The two controls developed paralysis on the fourth and fifth day and died on the fifth and seventh day respectively.

When the three surviving pigeons were tested for the presence of H.I. antibodies three weeks after the challenge, all showed high titres.

CONCLUSION AND DISCUSSION.

From this small but somewhat laborious experiment two highly significant conclusions may be drawn. Pigeons, after being dosed with virulent Newcastle disease virus—

- (1) may subsequently secrete virulent virus in the faeces;
- and (2) develop a resistance to infection which cannot be detected by the haemagglutination-inhibition test, but is demonstrable by the intramuscular injection of virulent virus shown to produce 100 per cent. mortality in fully susceptible pigeons.

Although active virus was collected from the faeces on only one day (the 12th after dosage) this single positive is of greater significance than all the other negatives. The faeces collection and testing was carried out under conditions which exclude any possibility of accidental contamination. It should also be borne in mind that only a small portion of the faeces excreted was collected, that voided between 8 and 10 o'clock each morning immediately after the test birds had been transferred to fresh sterile cages.

From the data submitted it is impossible to do more than express the opinion that multiplication of the ingested virus occurred in the pigeons. It is difficult to believe that for a period of 12 days viable virus could be retained within the alimentary canal of a bird before being excreted in viable form. It appears to be far more reasonable to postulate that multiplication of the virus took place within the pigeons but that under the conditions of the experiment it could be demonstrated in excreta on only one occasion.

The conclusion is warranted that under conditions favouring even the single ingestion of a large quantity of virus, the domestic pigeon may play an active part in the dissemination of infectoin. For this reason in any campaign aimed at the control of an outbreak of the disease, domestic pigeons should be included in the list of birds subjected to immunization. In a limited laboratory experiment, this was accomplished with safety with the use of the Roakin Strain (Beaudette *et al.* 1949). This was applied in the same manner as live virus vaccine to fowls (van Roekel *et al.* 1948). Several hundred pigeons were vaccinated in the field with complete safety.

The failure of the pigeon to develop haemagglutination-inhibiting antibodies in spite of the acquisition of at least a low grade immunity is interesting in that it indicates a limitation of the H.I. test as an index of acquired immunity.

SUMMARY.

When virus was dosed *per os* with virulent Newcastle disease virus and daily examinations were made of virus in their pooled faeces, virus was recovered on the 12th day.

No H.I antibodies were produced. The challenge caused two pigeons to sicken on the 5th day, one of which died on the 8th day while two appeared unaffected. Two controls died and previous experience was that pigeons are highly susceptible to the virulent strain. Surviving pigeons had high H.I titres.

It is concluded that the pigeons dosed *per os* with the virulent virus developed an increased resistance to the challenge.

A method of live virus immunization in the pigeon is mentioned.

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REFERENCES.

- BEAUDETTE, F. R., BIVINS, J. A. AND MILLER, B. R. (1949). Newcastle disease immunization with live virus. *Cornell Vet.*, Vol. 39, No. 3, pp. 302-334.
- DOYLE, T. M. (1927). A hitherto unrecorded disease of fowls due to a filter passing virus. *Jnl. of Comp. Path. and Therap.* Vol. 40, No. 2, pp. 144-169.
- IYER, S. G. (1939). Paralysis in a pigeon due to Doyle's (Raniket) disease virus. *Indian Jnl. of Vet. Sc. and Anim. Husb.*, Vol. 9, No. 4, pp. 379-382.
- KASCHULA, V. R., CANHAM, A. S., DIESEL, A. M. AND COLES, D. (1946). Newcastle disease in Natal. *Jnl. of S.A.V.M.A.*, Vol. 17, No. 1, pp. 1-14.
- KASCHULA, V. R. (1950). The epizootology of Newcastle disease and its control by vaccination. *Jnl. of S.A.V.M.A.*, Vol. 21, No. 4, pp. 134-140.
- VAN ROEKEL, H., SPERLING, F. G., BULLIS, K., OLESINK, O. (1948). Immunization of chickens against Newcastle disease. *Jnl. Am. Vet. Med. Ass.*, Vol. 112, No. 851, pp. 131-132.