## FURTHER INVESTIGATIONS ON THE EGG DROP SYNDROME

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This condition, first described in 1976, is characterised either by a sudden increase in the production of thin shelled, soft shelled or shell-less eggs or by a failure to achieve predicted production levels. In either case the birds remain apparently healthy.

The etiological agent is a duck adenovirus. This virus only spreads from infected ducks to fowl if they are in very close contact. The spread appears to be due to contact with faeces and aerosol spread is minimal.

In Northern Ireland the disease was eradicated from an infected basic breeding organisation. Eradication was based on the following premises:

- a) Birds infected through the embryo quite often failed to develop detectable antibody.
- b) These infected birds would show EDS around peak production. Following EDS antibody was detectable.
- c) Birds over 40 weeks of age, even if infected, were unlikely to excrete virus.
- d) The viruses infecting fowl in 1976-1977 at least had poor lateral spreading ability.

Therefore using these findings, chicks were hatched from flocks over 40 weeks of age. They were segregated from infected birds in the hatchery and were reared in semi isolation. These flocks were tested by HI at regular intervals. If a large number of reactors were found, these were removed. After 40 weeks, if the flock passed a 100% HI test, eggs for breeding were then collected.

## RECENT RESEARCH ON EGG DROP SYNDROME '76 (EDS '76)

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## SUMMARY

EDS '76 has become widespread in the fowl population and it is a cause of considerable economic loss. Eradication of the disease may be possible from the breeding and elite stock as vertical transmission is an important means of the spread of the virus. Screening individual birds for antibodies and removing them before lay may eliminate carriers. Where resources are limited, vaccination with an inactivated vaccine that induces high antibody

levels may be attempted. The results reported here would indicate that it is likely that both vertical and horizontal infection are reduced.

Of interest is the recent observation (Baxendale, unpublished) that vaccination of chicks previously experimentally infected as 1 day old embryos and which had developed only low H1 antibody, titres resulted in a considerable boost in antibody titre. If such birds were potential carriers, an antibody boost may reduce the chance of these birds excreting virus.

DETECTION OF ANTIBODIES AGAINST EDS - VIRUS (BC - 14 STRAIN) IN DOMESTIC HENS IN MEXICO

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One thousand one hundred blood samples were obtained from 67 chicken flocks throughout Mexico. Four hundred and six samples were positive to Adenovirus antibodies using the agar gel precipitin (AGP) test.

Hemagglutination inhibition (HI) tests were performed on the 406 AGP-positive samples: 318 were negative, 36 had titers of less than 1/40, and 52 were considered positive (titers of 1/40 or higher).

Most of the positive samples came from commercial medium-size layers and heavy breeders.