Chronic respiratory disease of poultry in Western Australia

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Widespread chronic respiratory disease was revealed in a survey of respiratory infections in Perth metropolitan area poultry flocks. Most occur in early or mid-summer and cause a serious fall in egg production. Return to full lay after the infection is usually slow.

While these infections can cause production losses in flocks which are under heavy stress, none of the more serious forms of chronic respiratory disease was found. This gives encouragement for future control of the disease in Western Australia.

A disease affecting the respiratory system of fowls, which was different from any of the commonly recognised infections, was first noted after World War II, in the U.S.A.

Before this time, a number of specific respiratory diseases had been identified. These included:

- The viral infections Newcastle disease, infectious bronchitis, infectious laryngotracheitis and fowl pox;
- The bacterial diseases infectious coryza due to *Haemophilus gallinarum*, fowl cholera caused by *Pasteurella* organisms, and pullorum disease; and
- The intermediate (large virus) infections such as psittacosis and ornithosis.

This "new" disease was characterised by a fairly rapid onset and a protracted course, by mortality rates which could vary from insignificant to severe, and by a marked and often prolonged drop in egg production.

During the 1950's the emphasis in this "chronic respiratory disease" (CRD) shifted to the rapidly expanding broiler
industry and soon threatened the whole industry. Only by painstaking research has the complicated cause of CRD been worked out.

The various factors involved are now fairly well understood.

It is known that in most infections of this kind, a microorganism intermediate in size between the large viruses and the bacteria—and now called *Mycoplasma gallisepticum*, or pleuro-pneumonia-like organism (PPLO), is present. However, when pure cultures of this microorganism are inoculated into healthy, unstressed fowls, no clinical disease can be produced.

The relationship between this new microorganism and chronic respiratory disease first started to show itself in 1951, when weakened living viruses came into general use as immunising agents against Newcastle disease, infectious bronchitis and fowl pox.

It was then found that birds carrying the seemingly innocuous PPLO's would, on vaccination with the immunising agents, break down with chronic respiratory disease.

Although this was the most spectacular of the many events associated with the development of CRD, further research uncovered other factors which could precipitate the disease. These factors were identified with the changing conditions in poultry raising, such as the intensive hatching and brooding systems then coming into use, the increasing practice of rearing thousands of birds in single houses, and the over-reliance on vaccination and drug treatments to the detriment of time-honoured sanitation and hygiene.

At present, chronic respiratory disease is recognised as a typical example of a "stress" disease. Several unrelated factors may be implicated in individual outbreaks.

American Survey

In 1951-52, one of us* conducted a survey on the factors associated with outbreaks of CRD in the most intensive poultry area in the U.S.A.

Hundreds of flocks were studied in detail. At many of them, pleuro-pneumonia-like organisms were isolated but not often alone. Usually, some other disease agent was found, such as the virus of Newcastle disease, the virus of bronchitis, the virus of ILT, or other viruses. Sometimes the bacteria causing infectious coryza, or fowl cholera, were found. Secondary intestinal parasitic infestations were common.

This survey emphasised the complex nature of the disease.

Without intention, a group of birds inoculated with pleuro-pneumonia-like organisms, but showing no signs of disease, was injected with a chemical caponiser, diethylstilbestrol, and almost at once broke down with the typical signs of CRD. This was an early demonstration of the fact that almost any stress condition, including those completely unrelated to the respiratory system of fowls, might precipitate the disease.

Later we were able to show that symptomless carriers transmitted the pleuro-pneumonia-like infection through the fertile egg to their progeny and this perpetuated the disease in a closed flock.

Local Survey

In January, 1962, a study was begun on the role played by this group of microorganisms in undefined poultry disease conditions in Western Australia.

There had been little previous indication that this disease was having any significant effect on poultry health in this State, but no study of infections by P.P.L. organisms had yet been done here.

For several years a rise in incidence of respiratory conditions affecting poultry in Western Australia has been noted during the summer months, reaching a peak in January. It was, therefore, decided to do the survey during this month in as many flocks as possible in the Perth metropolitan area.

**METHODS**

During January and February, 1962, typical birds from affected flocks were brought to the Animal Health Laboratory, South Perth. Diagnostic procedures for the detection of infection by pleuro-pneumonia organisms, viruses and pathogenic disease-causing bacteria were undertaken on these birds. A blood test very similar to the pullorum agglutination test,
was used to detect infection by pleuropneumonia-like organism. This is a highly accurate test for this organism.

[Technical details of the methods and tests used are included in the appendix.]

RESULTS

Twenty flocks in the greater metropolitan district of Perth were studied. Of these, 12 had suffered from similar respiratory disease on at least one occasion in the past two or three years, while in the rest of the flocks, the disease appeared for the first time in 1962.

Typical Outbreak

In the typical outbreak, the condition makes its appearance in the last half of December, or in January or February, with the onset of upper respiratory symptoms.

There may be a mucus-like discharge from the nose, an increased respiratory rate with some mouth-breathing, and coughing. Considerable respiratory noise may be heard throughout the flock particularly at night, when the birds are at rest.

These symptoms appear very rapidly, and within several days most of the flock may be affected. There is an accompanying severe drop in egg production.

The signs of respiratory infection may begin to abate somewhat after two or three weeks, but the decline in production is usually quite prolonged and may last as long as three months.

In some outbreaks quite a few birds die, and in a few the death rate may be fairly high.

Diagnosis

The results of the diagnostic tests are briefly summarised below:

In 18 flocks, definite evidence of infection was found by the blood test. No complicating infections by viruses or by bacteria occurred in any of the birds examined from these flocks and the respiratory condition was considered to be a "pure" pleuropneumonia-like infection.

One flock showing no clinical signs of disease was included in the survey and all the fowls examined had negative blood tests.

The twentieth flock was negative to the blood test when first studied in late January, at which time the flock was in good health. A few days later respiratory symptoms developed rapidly and birds tested then showed evidence of infection by the pleuro-pneumonia organism.

Although no complicating viruses or bacteria were found in any of the outbreaks, several disease factors were recognised as predisposing causes in some of them.

Without going into details, it was clear that the following conditions could either predispose to the respiratory infection, or could make it much more serious:

1. Some degree of Vitamin A deficiency caused by lack of green feed, or by deterioration of Vitamin A supplements.
2. Previous vaccination for fowl pox. Although the vaccine of fowl pox is made from relatively harmless strains of the virus, there is little doubt that vaccination delayed until the early summer can pave the way for a subsequent infection by the pleuro-pneumonia-like organisms.
3. Infestation by the large poultry roundworm or by red mites.
4. Overcrowding and under-ventilation during periods of considerable heat stress.

DISCUSSION

The more serious forms of chronic respiratory disease were entirely absent in the flocks surveyed.

In particular, the "air sac" infections so common overseas were not found, nor was the "coryza" manifested by swollen faces and wattles, seen. Both of these complicating forms are caused by bacterial species acting along with the pleuropneumonia-like organisms, and their absence from the flocks included in the survey gives considerable encouragement for the future control of chronic respiratory disease in Western Australia.

There was, however, a pattern of disease running throughout the investigation.

Most of the outbreaks occurred in the early or mid-summer months, were rapid
in onset, and were prolonged in their course. Similar outbreaks had appeared on many of the properties during the same period in previous years.

All outbreaks were associated with a serious fall in egg production and a return to full production was usually slow.

It is known that pleuro-pneumonia-like organisms are easily transmitted to the newly hatched chickens by way of infected eggs laid by “carrier” hens. The infection may then be carried in turn by the growing birds in their upper respiratory tracts, in a dormant form, until the stress factors mentioned above are brought to bear on the flock.

Some flocks acquire their infections from “carrier” birds introduced into the flock from outside sources or from recovered birds retained on the property. There is little doubt that once a property is affected, chronic respiratory organisms acquire permanent residence in the recovered birds and are a source of danger to growing birds which may come from clean breeding flocks as day-old chickens. Only a vigorous programme of depopulation can then offer any hope of control.

**RECOMMENDATIONS**

Based upon the findings in this survey and on work carried out elsewhere, the following procedures are recommended in the control of chronic respiratory disease:

1. Hatching eggs and day-old chicks should come from flocks known to be free of the infection.
2. Brooder flocks should be reared in isolation away from all other fowls on the property and throughout their productive life should be maintained as closed units.
3. Depopulating infected houses and restocking with clean birds after a rest period should be carried out in any serious control programme. This may be done in stages without much economic consequence if such newly stocked houses, or sheds, are at least 40 feet away from infected houses and serviced first in the daily routine of feeding, cleaning and so on.
4. Fowl pox vaccination should be carried out in the early spring, before 9th or 10th week of life, or preferably, at one or two days old.
5. A potent source of Vitamin A such as green feed, or a stabilised form of the vitamin should be provided at all times.
6. Cooling sprays to reduce heat and lay dust should be provided for summer use.
7. The newer antibiotics and antibacterial agents such as the broad spectrum antibiotics and Furazolidone may be used to help control outbreaks if they occur. However, it must be emphasised that these drugs are only occasionally of great help and in many outbreaks are not effective, although they may lower mortality rates.
8. Parasitic infestations should be prevented or treated with appropriate drugs.

**APPENDIX**

**Technical Details**

Two or three typically affected birds from each of 20 poultry flocks showing signs of upper respiratory infections from January 8th, 1962, to February 1st, 1962, were brought to the Animal Health Laboratory for study.

Blood was drawn from the wing vein for the whole blood plate agglutination test using PPLO antigen supplied by Mr. R. B. Cuming, B.V.Sc., of the University of New England.

The birds were destroyed by intracerebral injections of 70 per cent. alcohol. The entire bird (except for the head) was then dipped in a detergent solution and then in running water. The feathers were plucked from the neck and breast and the entire trachea exposed and collected. This was then opened with sterile scissors and examined, then ground up in a sterile mortar for culture procedures. The thorax and abdomen were then opened and the air sacs and lungs examined. The mouth, oesophagus, nasal cavities, eyes and crop were also carefully examined. Air sacs were added to the mortar for the preparation of the inocula.

The tracheal suspension was inoculated onto plates of PPLO medium containing 10 per cent. horse serum and on to bovine blood agar plates. It was also inoculated on to the chorio-allantoic membranes of two or three, 10-day day embryo-nated eggs.

The objectives in the cultural procedures were the isolation of *Haemophilus gallinarum*, *Mycoplasma gallisepticum*, and the viruses of infectious laryngo-tracheitis and fowl pox.