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"FATIGUE" IN CAGED LAYERS

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POULTRY

Pullets found immobilised on the floors of their laying cages may only be suffering from the easily-treated "cage layer fatigue" and should be checked for this before being disposed of as affected with the incurable fowl paralysis.

MOST cases of leg paralysis in laying pullets are caused by fowl paralysis. As there is no effective treatment for this disease, affected birds are usually disposed of fairly quickly.

However, at this time of the year it is possible to confuse a condition known as cage layer fatigue with fowl paralysis and because cage layer fatigue can be treated simply and effectively it is important to distinguish between the two.

Cage layer fatigue is an abnormal condition which occurs in pullets housed in laying cages. Affected birds develop a leg weakness and are unable to move about the cage. The onset of this symptom is usually rapid; it is not uncommon for a bird to be in apparently perfect health and production on one day and by the next day it has lost the use of its legs.

The incidence of cage layer fatigue is not primarily seasonal. In overseas cage units where continuous pullet replacement is practiced, the condition has been found to occur throughout the year. It has been fairly well established that cage layer fatigue occurs most typically in high producing pullets at about the time that they reach their initial peak in production. This is normally about six to eight weeks after the pullets come into lay, although one farm in this State has had the disease diagnosed for the past two years in pullets before they came into production.

Cage layer fatigue is one of the minor disease problems in poultry, but it increases in importance as the number of birds housed in cages increases. Before 1960, there were few cage units in Western Australia and the condition was not well known except in our Poultry Research Station cage flock.

It is now estimated that there are about 170,000 layers housed in cages on some 75 farms in W.A. and in the past two years field officers have reported that the condition has been noticed on nearly every farm where floor-reared birds have been placed in cages.

Usually only a few birds in a flock are affected but on several farms the incidence has been near the 5 per cent. mark. An incidence in excess of 5 per cent. has never been reported in any of the literature dealing with the condition. Also there have been no reports of cage layer fatigue in second year birds or flocks housed on the floor. Some pathologists have suggested that the condition may occur in birds on litter, but may not have been observed.

Treatment

Pullets affected with cage layer fatigue will usually recover after being taken out of the cages and placed on the floor, with food and water provided so that the crippled bird can eat and drink. Recovery depends on the early removal of affected birds. Usually after a few days the bird can be returned to the cage and recovered birds are never affected again.

The cause of cage layer fatigue is not known. Some scientists believe it may be
caused by a nutritional deficiency while others claim that a management factor is involved. The first known report of the disease was from the Southern States of America in 1955. Here replacements are usually grown on litter or range before they are transferred to cages, whereas in California, where cage layer fatigue is not known, replacements are usually raised on wire from day old. In Western Australia also there has been no sign of the syndrome in cage flocks raised on wire from day old.

This lends support to the theory that some floor-raised pullets have difficulty in adjusting to wire floors, with the result that after some weeks of heavy production the pullet, deprived of exercises and changed to a different ration, cannot make the required metabolic and environmental adjustments and becomes affected with cage layer fatigue.

Cage layer fatigue has been reported from England, America and the Eastern States of Australia. Most of our knowledge on the condition, apart from local practical experience, comes from work carried out in the United States.

In 1956, Davis of New Mexico found a strain difference in the incidence of fatigue in a flock of 12,000 pullets from five strains. The incidence of the condition varied from 0.65 to 3.95 per cent. between strains. Overall recovery of affected birds after removal from their cages was 80 per cent. but in one strain there was 100 per cent. recovery.

No symptoms of fowl paralysis or leucosis have been found on post mortem of affected birds. Blood calcium and phosphorus levels have been found normal which rules out the possibility of rickets. Extra vitamin D or calcium added to the feed neither prevented nor relieved cage layer fatigue in investigations in the United States.

Post mortem results usually show that the leg bones appear to be friable and have lower bone ash values. Fractures of the leg or wing bones are not uncommon. Egg shell quality is not adversely affected, which is one of the features distinguishing this condition from rickets.

Many nutritional factors have been tested. These include increased levels of the B vitamins, vitamin D₃, calcium phosphorus, antibiotics, trace minerals and hormones, but no association with the disease was found.

Subcutaneous or intramuscular injections of therapeutic levels of hormones and vitamin C have given some favourable response, which suggests that the condition could be associated with stress factors.

At this time of the year, cage farmers with odd pullets that lie immobilised on the floor of the cage, should check whether the simply-treated condition of cage layer fatigue could be the cause before marketing the birds as being affected with fowl paralysis.

—From an A.B.C. Radio Talk.