

## Blue Comb

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BLUE COMB, a disease of young laying fowls, is known also as pullet disease, avian monocytosis, new wheat disease, X-disease, summer disease, mud fever of turkeys, and contagious indigestion. It has occurred in the Northeastern, Middle Atlantic, North Central, and Southern States and California.

It occurs more commonly in the warm months, June to November, in chickens 4 weeks to 2 years old, and in poults of all ages. Mortality from blue comb may range from zero to 50 percent and the morbidity to 20 percent in chickens.

Research workers have tried for a long time to discover the cause of blue comb. The disease resembles an infection in its spread through the flock, but in most cases it is a metabolic disturbance without an infectious cause. A virus has been suspected.

D. E. Stover, of the California Department of Agriculture, Sacramento, Calif., found the virus of Newcastle disease in outbreaks of blue comb; the association of this virus with explosive outbreaks of blue comb is common. Birds in a subclinical stage of leukosis often develop blue comb before the lesions of leukosis are well developed.

Other infections, among them staphylococcal and streptococcal infections and trichomoniasis of the intestinal tract, can be involved in outbreaks of blue comb, but those diseases are secondary to the injury and dysfunction of tissues incidental to blue comb.

Poultrymen's suspicion that wheat was involved was confirmed by G. D. Quigley, of the University of Maryland, who in further studies considered the possibility that blue comb is due to a mineral imbalance.

The disease cannot be entirely prevented in susceptible flocks, because as

R. K. Cole, at Cornell University, discovered, genetic makeup is a factor.

Poultrymen in localities where blue comb is prevalent have found that birds brought into production rapidly are more susceptible, that losses in the spring are more likely in high-producing flocks, and that blue comb is most common 3 or 4 days after a hot spell or when temperatures are above 85° F.

Symptoms of blue comb vary according to the stage of the disease and may include depression; loss of appetite; cyanosis of the head (bluish-red comb) and skin because of poor circulation; fever; sunken eyes; and dehydration, with a shriveling of the skin of shanks, comb, and wattles. The crop may be distended and sour. A sudden drop in egg laying and a slow return to normal are usual. Poults have a fetid diarrhea and tend to pile in the brooder house. Blue comb lasts 1 to 2 weeks.

A high proportion of chickens recover, but mortality may be 25 to 100 percent in young poults.

Sometimes birds are found dead before symptoms are noted. Sudden deaths may produce few or no lesions (tissue changes). The more prolonged cases on autopsy may show a focal necrosis of the liver, hemorrhages on the serous membranes, chalkiness of the pancreas, and tenacious mucus in the intestines. Renal (kidney) damage is a prominent lesion in the subacute and a constant lesion in the chronic form of the disease.

Chronic blue comb is indistinguishable from visceral gout, a disease characterized by marked paleness and swelling of the kidneys and ureters due to accumulations of uric acid salts.

Blue comb must be differentiated from fowl cholera and fowl typhoid. The latter diseases may be identified by culture of the dead bird and iso-

lation of the respective causative organisms, but in blue comb disease no specific causative micro-organism has been isolated.

Good management will do much to alleviate the impact of stress factors on young pullets just coming into production and may prevent the onset of the disease. These stress factors include an unbalanced laying diet, forcing for higher production, sudden changes in diet, unfavorable weather (especially excessive heat and humidity), inadequate supplies of fresh water, rough handling or excitement of the birds, and changes in environment.

After the onset of an outbreak, such management procedures as the provision of an ample supply of fresh water, restriction of grain consump-

tion, and cool quarters are advised. Also recommended is the administration of molasses and some form of potassium, such as muriate of potash, fertilizer grade.

Antibiotics, such as Aurcomycin, Terramycin, streptomycin, and penicillin, are used by some in place of molasses or potash.

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## Fowlpox

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FOWLPOX, also called avianpox, birdpox, avian diphtheria (no relation to human diphtheria), sorehead, and canker, is a widespread disease.

The filterable virus that causes it occurs as inclusion bodies in the cells of the skin or mucous membranes. These bodies may be viewed by sectioning (slicing) the skin cells, staining them, and observing them under a powerful microscope.

One inclusion body may contain as many as 20 thousand elementary bodies, each of which can cause fowlpox. These bodies are thought to be the real virus carriers, and, when liberated from the inclusion body and stained, can be seen under the microscope.

The late J. R. Beach, after research at the University of California, in 1939 classified avianpox as fowlpox, turkeypox, pigeonpox, and canarypox.

Fowlpox and turkeypox cross-immunize; that is, fowlpox will immunize both chickens and turkeys, although some strains differ.

The lesions—tissue changes—produced by an attack of fowlpox occur on the unfeathered parts of the body, comb, wattles, face, and eyelids and sometimes on the feet and around the cloaca. Lesions in turkeys may occur on the breast.

Pock lesions begin as a small, round blister, which contains a serous—watery—fluid. As the pock, or pimple, enlarges, the center becomes yellowish from an accumulation of virus and pus. Later the pock ulcerates, ruptures, and discharges a sticky fluid. The ulcer dries in a few days, and a dark-red or black scab forms over the raw lesion. The scab sticks for some time before it drops off. The pocks develop