Coccidiosis of the Chicken

BY JOHN F. CHRISTENSEN AND ENA A. ALLEN

AN ACCOUNT of the life history, symptoms of infection, and methods of control of a group of internal parasites that are among the most serious enemies of poultry production.

"It is probable that coccidia cause greater economic loss among domesticated animals of the temperate zone than any other group of protozoa." Thus Becker aptly indicated the importance of coccidiosis, the specific disease caused by these protozoan parasites. It may be added that the coccidia probably cause greater economic loss among chickens alone than among all other domesticated animals combined, despite the fact that coccidiosis occurs frequently in severe outbreaks among other domesticated fowl, cattle, and sheep and occasionally among goats, pigs and dogs. Because of the preponderant significance of the disease in chickens, this article is confined to a description of the coccidia and coccidiosis of these birds. However, the life histories of the parasites, the nature of the disease, and the principles of control are essentially similar for all domesticated and semidomesticated fowl, and the information given therefore applies in general also to coccidiosis of the duck, goose, guinea fowl, pheasant, pigeon, quail, and turkey, all of which harbor characteristic kinds or species of coccidia.

THE LIFE HISTORY OF A POULTRY COCCIDIIUM

The coccidia belong to a group of Protozoa known as Sporozoa, so designated because they produce sporelike infective bodies at

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2 Becker, Elery R. Coccidia and Coccidiosis of Domesticated, Game and Laboratory Animals and of Man, 147 pp., illus. Ames, Iowa. 1834.
some stage in their life histories. Resistant cysts—enclosed, egglike forms—of these parasites are discharged from their hosts in the feces or droppings and must pass through a process of development called sporulation in the outside environment before being infective to other animals. With the exception of *Tyzzeria* of the duck, the coccidia of the chicken, and all other barnyard fowls belong to the genus *Eimeria*, the life histories of all species of which are in general similar.

The life history may be said to begin with the discharge of the microscopic, egglike, resistant forms known as oöcysts in the droppings of infected birds. Under conditions of optimum moisture, moderate temperature, and ample oxygen supply, these eliminated oöcysts sporulate within a few days. This process consists in division of the protoplasm within each oöcyst shell into four elongated bodies, each in turn secreting its own shell, within which the protoplasm again divides to form two comma-shaped or sausage-shaped sporozoites. Each sporulated oöcyst thus contains eight sporozoites, which are infective agents capable of giving rise to coccidial infection when swallowed by susceptible birds.

Sporulated oöcysts may be ingested by susceptible birds soon after development is complete, or they may lie dormant for months in litter or soil, if temperature and moisture are favorable, before being picked up by new hosts. Once eaten by a susceptible bird, the oöcysts are in some way activated in the intestine of the host so that the sporozoites are released into the intestinal canal. The liberated sporozoites are capable of slow flexing movements, and when they come in contact with the intestinal wall in certain regions of the alimentary tract they penetrate cell membranes and enter the living protoplasm.

Inside the host cells the sporozoites lose their ability to move around, and they grow at the expense of the parasitized cells. After a definite period of growth the protoplasm of each parasite divides into many small, elongated bodies known as merozoites, which are expelled from the tissue by rupture of the cell membranes. These merozoites also possess limited powers of locomotion and immediately parasitize other cells. Again there is growth and multiplication, with the production of additional swarms of merozoites. After two or more generations of such asexual reproduction, some of the merozoites become sexually differentiated after entering new cells. The female parasites are large, rounded, and inactive. The male parasite produces by segmentation vast numbers of extremely small, actively moving bodies each capable of activating or fertilizing a single female parasite. After fertilization and secretion of a resistant shell, the female parasites are extruded, or pushed out, from the damaged host tissue into the alimentary stream and discharged from the host birds in the droppings, being then designated as oöcysts.

**CECAL COCCIDIOSIS**

There are two distinct types of coccidiosis of the chicken, depending upon the site of localization and multiplication of the parasites in the digestive tracts of the infected birds. In cecal coccidiosis, the
coccidia invade the mucous membrane of the ceca, or blind guts, of chicks (fig. 1), producing as a result of rapid multiplication and extensive destruction of tissue an acute and often highly fatal disease characterized by severe cecal hemorrhage. In the intestinal disease, the parasites become localized largely in the small intestine and give rise to a serious but usually less acute infection of older, maturing birds sometimes characterized by extreme wasting of flesh and slow, insidious development.

Cecal coccidiosis is primarily a disease of chicks 3 to 5 weeks old, although infection may occur in birds of any age. The disease usually strikes rather suddenly, producing illness or death in a considerable number of chicks at about the same time. In severe infections, many birds may die suddenly without showing visible symptoms, but they usually have pale combs and some blood on the feathers surrounding the vent, and when the birds are opened for examination, the ceca are found to be bulging with blood. Other birds have a ruffled, droopy appearance (fig. 2), lose appetite, fall off rapidly in weight, have a subnormal temperature, show pale skin and mucous membranes as a result of cecal hemmor-
rhage, and discharge bloody droppings. Some of these sick birds die during the first few days of illness, while others linger for several days to a week or more before finally succumbing to emaciation or wasting of flesh and loss of blood. With good care, many sick chicks gradually overcome the pallor, regain flesh, and recover.

Tyzzer demonstrated that cecal coccidiosis is caused by a single species of coccidium, *Eimeria tenella*. The information he obtained from study with experimentally infected birds provided the basis for a complete understanding of the natural disease. On the fourth day after inoculation with heavy doses of sporulated oöcysts of *E. tenella*, birds show symptoms of severe cecal coccidiosis, and when they are killed for examination, bleeding from the cecal walls has begun. The greatest number of deaths occur on the fifth and sixth days of infection, when cecal hemorrhage reaches its peak, the birds show pale skin and mucous membranes from loss of blood, and the droppings contain blood. Chicks that die during the period of greatest hemorrhage have ceca bulging with fluid blood (fig. 1). When death occurs later in the infection, the blood and tissue debris in the ceca have formed clotted or cheesy cores, while the cecal walls appear thickened and mottled from hemorrhage and the tissues and organs are pale from loss of blood. It was demonstrated that the widespread leakage of blood from the cecal mucous membrane during the infection results from the destruction of tissue by the maturing second generation parasites and the liberation of enormous numbers of second generation merozoites.

**INTESTINAL COCCIDIOSIS**

Although usually less acute and spectacular in its onset than the cecal infection, intestinal coccidiosis causes a tremendous economic loss to poultry farmers by making heavy inroads on flocks of maturing chickens at a time when considerable cash outlay is represented. Of the six well-established species of coccidia that become localized in the small intestine of chickens, *Eimeria necatrix* is the one responsible for severe clinical intestinal coccidiosis, although other species have been demonstrated by experimental infections to produce less severe clinical symptoms. Thus, *E. acervulina* may produce coccidiosis of long duration characterized by extreme emaciation, and *E. maxima* causes thickening of the intestinal wall and produces a slight hemorrhage from the damaged mucous membrane in very heavy infections.

This type of coccidiosis is primarily a disease of maturing birds and frequently appears soon after pullets are confined to laying houses. The infection is first recognized in only a few birds of a flock, but additional individuals develop symptoms from day to day until considerable numbers are affected. The symptoms are in general similar to those noted for chicks with cecal coccidiosis, consisting in droopiness, a ruffled appearance, loss of appetite, increasing

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emaciation and weakness, and pallor resulting from intestinal hemorrhage. Death from the disease may occur during the early stages of the attack, but usually only after several days to 2 or 3 weeks of illness. Many sick birds gradually regain their appetite and strength and recover from the attack.

As Tyzzer had done previously for *Eimeria tenella*, Tyzzer, Theiler, and Jones worked out the intricate details of the life history of *E. necatrix* and the course of experimental infection in the chicken in order to obtain the information essential for an accurate conception of the natural disease. They demonstrated that susceptible chickens experimentally infected with large single dosages of sporulated oocysts of *E. necatrix* show the first symptoms of coccidiosis at the

end of 4 days of infection. The heaviest death losses occur on the sixth and seventh days of infection as a result of the destruction of tissue and the bleeding produced by the action of the parasites in the intestinal mucous membrane. The unopened small intestine of birds that die at the height of infection appears heavily peppered with small opaque spots and hemorrhagic blotches and streaks, especially in the middle portion (fig. 3). Microscopic study reveals that these opaque spots or lesions are large colonies of maturing second-generation parasites located deep in the mucous membrane and that the bleeding commences at the centers of these lesions in the tissue destroyed by the parasites. The affected intestine is congested with blood and is flabby, distended, and easily ruptured; it is filled with material varying from a clear, jellylike, yellowish exudate to clotted blood, which often forms a solid, fibrinous cylinder completely blocking the intestinal canal. During the sixth day, the muscular function of the intestine is usually completely lost as a result of obstruction with these masses of clotted tissue, exudate, and blood. Birds that survive the seventh day of these heavy experimental infections usually recover. Within a day or two the intestine resumes functioning, and the recovering chickens begin discharging watery droppings containing blood, mucus, and occasional cylindrical masses of clotted material. Gradually the droppings become normal, appetite returns, and paleness disappears, but the birds may remain extremely emaciated from malnutrition.

CONTROL OF COCCIDIOSIS OF THE CHICKEN

Unfortunately, losses from coccidiosis do not cease with the deaths and sickness that result from an outbreak. According to Tyzzer, the damage resulting from severe infections to the ceca and intestine sometimes results in the permanent loss of function of considerable areas of mucous membrane in recovered chickens, and this contributes to the chronic un thriftiness of many of these birds. Such fowls not only mature more slowly but produce fewer eggs during maturity than similar chickens that have had no early attack of clinical coccidiosis. These after effects of the disease emphasize the wisdom of prevention as a means of control.

It is well established that if reinfection is prevented, the coccidial parasites run fairly rapid, definite courses of multiplication within the host birds and are soon eliminated as oocysts; that birds surviving severe or continued mild infections are subsequently resistant or immune to severe reinfections with the same species of coccidia; and that such immune adult birds are healthy carriers of coccidia and thus possible sources of infection to young stock. Prevention is based on these facts, which at once suggest that the supply of infective oocysts should be kept as low as possible by application of rigid sanitation and that young susceptible birds should be kept strictly isolated from adult fowls.


Coccidiosis of the Chicken

Floors, walls, windows, and equipment of brooder houses should be cleaned thoroughly with soap and hot water before the birds are admitted. After that the floors should be thoroughly dry-cleaned with a scuffle hoe and a stiff broom at least twice weekly until the birds are 10 or 12 weeks old, then weekly until they are transferred to laying houses. Dry cleaning prevents the accumulation of moisture, which favors the sporulation of oöcysts. Fresh, clean litter should be put in after each cleaning. If chicks are kept in cages, cleaning should be done daily, if possible. Feeding and watering equipment should be cleaned frequently and should be designed to prevent accumulations of moisture on floors or contamination of feed and drinking water with infective material.

If there is enough land available, it is wise to use movable brooder houses that can be transferred occasionally to new ground. Stationary brooder houses should be located on well-drained, preferably sandy soil, which provides the quick-drying terrain least favorable for oöcyst development. It has been recommended that stationary brooder houses be provided with sloping concrete runs, which can be covered lightly with sand and cleaned thoroughly at the same time the houses are cleaned. During the period of confinement to laying houses special care must be taken to prevent conditions that would favor the development of intestinal coccidiosis among the pullets. The laying houses should be cleaned thoroughly before occupancy and frequently thereafter, in order that accumulations of moisture and litter, which favor oöcyst development, may be avoided. The same attention to sanitation should be given to the feeding and watering equipment for these older birds as to those for the brooding stock. If considerable range is available, clean ground should be provided for each group of pullets. At all times, the feeding, watering, and cleaning equipment for young and adult stock should be kept separated in order to prevent any significant transfer of infective material to susceptible birds. Though strict application of these sanitary measures in the handling of young birds cannot be expected to eliminate coccidial infection completely, it should permit successful rearing.

Even when excellent care is provided, outbreaks of coccidiosis occur occasionally among young birds, and at these times there is a demand for an immediate remedy. It is the general opinion among qualified authorities on poultry coccidiosis that there is little value in medicinal treatment of sick birds during an outbreak and that effort is applied more profitably in proper management. Early diagnosis and prompt adjustments in the feeding and care of birds are important. It is wise to segregate sick chickens, provide warm quarters, avoid overcrowding, supply appetizing, easily digested, nutritious feed, and redouble efforts at sanitation in order to remove oöcysts, which are shed in the droppings of sick birds in enormous numbers. Meticulous attention to all details of good feeding and care of sick birds may be expected to minimize losses during an outbreak, but treatment “can be recommended only as a means of making the best of an already bad situation, not as a routine preventive.”

See reference in footnote 6.