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Coccidia

The life cycle, pathology and host-specificity of coccidia in general.

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Several years ago there appeared in a well-known poultry journal an article on coccidiosis of chickens entitled "Poultry Enemy No. 1." It is not improbable that the author was deservedly subject to a charge of over-emphasis, but on the other hand, it is undeniable that three or four coccidian infections of poultry together constitute a serious menace to poultrymen. The annual monetary loss in the United States from this cause has never been accurately calculated, perhaps it is indeterminable, but it must amount to many hundreds of thousands of dollars. While it is true that the major losses from coccidial infections occur in the feathered tribe, domesticated mammals have by no means escaped their depredations. There are localities where, owing to favorable ecologic conditions, coccidiosis or "red diarrhea" attacks grazing cattle. Feeder cattle and lambs often become subject to intestinal infection to the great discouragement of stock-feeders. The clinician is familiar with a typical serious canine ailment in which oocysts of coccidia appear in great numbers in liquid stools. Many rabbit raisers are accustomed to losses from a liver and several intestinal types of coccidia. It would be a mistake not to recognize the possibilities of loss and disease attributable to these animal microorganisms.

It is a fact not generally recognized that one of the most brilliant chapters in the history of science is that concerned with the life histories of animal parasites. To Fritz Schandinn, the German zoologist who discovered the spirochete of syphilis, belongs the everlasting credit for first deciphering the complete life history of a coccidium, although it is now recognized that he made certain minor errors in interpretation. The species studied by him was *Eimeria schubergi*, and the host employed was a little centipede (*Lithobius forficatus*). At this point the zoologist is tempted to do a little moralizing, for Schandinn's important study was made on a "non-economic" species of parasite living in a "non-economic" host. The importance of knowing the cycle of a parasite for an understanding of its effect on the host and the manner of its dissemination, hence of its control, needs no further comment.

Life Cycle

In fact, the life cycle of a coccidium is quite unlike that of most of the microorganisms. *Eimeria tenella*, the etiological agent in bloody coccidiosis of chicks, for example, regularly undergoes a part of its cycle (endogenous) in the caeca and adjoining regions of the digestive tract of the chick and part of it (exogenous) in the moist droppings outside the body. It is perhaps most convenient to begin with the exogenous cycle, which commences with the freshly passed oocyst. The latter consists simply of a roughly spherical, nucleated mass of protoplasm separated somewhat from an ellipsoidal cyst wall measuring about 23 x 19 microns. If the cyst is kept at room temperature and in sufficient moisture for about 48 hours the protoplasmic mass becomes transformed

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into four pear-shaped spores, or sporocysts. Careful study with the microscope will reveal the presence of two uninucleate, banana-shaped bodies within each spore, the sporozoites. Each oocyst, then, comes to contain in all, eight sporozoites, the real infective stages. There is no further development of the parasite unless it is eaten.

**Infestation**

In some manner, very often through contamination of the food and drink, sporulated oocysts find their way into the digestive tract of chicks. In the crop, and also in the intestine, excystation or liberation of the sporozoites occurs, sometimes as soon as five minutes after ingestion of the oocysts. The sporozoites are able to move about, although they have no flagella or other organellae of locomotion. By bending and twisting movements they make their way to the cells of the epithelium of the caeca and immediately adjacent regions of the small and large intestines, which they succeed in penetrating. Once within the cytoplasm the sporozoite commences to round up, and becomes what is now termed a schizont, i.e., a stage capable of growing and eventually splitting up into a number of nucleated banana-shaped bodies, or merozoites. The merozoites escape from the cell, find their way into other cells, become schizonts, and eventually another generation of merozoites is liberated. These merozoites in turn enter other cells, and so they come to be third generation schizonts and third generation merozoites. The latter apparently re-enter cells after escaping, but in this case some develop into male sexual cells, microgametes, and others into female sexual cells, macrogametes. The union of a microgamete and a macrogamete results in a zygote, but because the protoplasmic part is surrounded by a wall it is called an oocyst. Great numbers of the oocysts are discharged in the evacuations from the caeca. Thus the intrinsic cycle winds up with the formation of oocysts.

The question naturally arises—what causes the bloody diarrhoea in *E. tenella* infection? Naturally it is to be expected that the extensive development undergone in the intestinal epithelium would destroy many cells, but this loss in itself is the cause of only a minor edema and the destroyed cells are easily replaced. Dr. E. E. Tyzzer of Harvard, who is the world's greatest authority on poultry coccidiosis, has supplied us with a most plausible explanation for the seriousness of the disorder. He discovered that the epithelial cell which harbors the second generation schizont becomes stimulated to wander from its accustomed position into the connective tissue immediately below. Not only does it wander, but it increases tremendously in size so that there is produced a pressure necrosis, resulting first in a serous exudate from the caecal wall, then in numerous minute hemorrhages. The chick literally bleeds to death. Hence, in this case, the cause of the disease is mostly mechanical. In most other coccidian infections other explanations must be sought, but toxins have not yet been definitely demonstrated.

**Host-specificity**

Another concept that should be helpful to the veterinary scientist and clinician concerns host-specificity of coccidia, or their specific host affiliations. In many European countries rabbits and chickens were kept with the cows and there was a tendency to blame the rabbits for bovine and avian coccidiosis, and vice versa. It is now known that rabbit coccidia grow only in rabbits and hares, that avian coccidia grow only in birds and that bovine coccidia grow only in members of that family. Among birds a rather strict host-specificity seems also to prevail. Chickens do not become infected from sparrows, turkeys from chickens, etc., although there are possibly some inter-specific transfers among gallinaceous birds. The writer succeeded in infecting cottontails with one species from the tame rabbit. This species was of European origin.

The control of coccidiosis constitutes a topic difficult from many angles. We do

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not know all the conditions that lead to outbreaks; for example, why do feeder cattle and sheep sometimes experience severe outbreaks? Is it the concentration of the animals and the resulting increase in chances of heavy infection? Or is it partly the metabolic changes attributable to heavier feeding of concentrates that makes the hosts more susceptible? Of one thing we are certain—conditions that make for the accumulation of large amounts of feces, efficient sporulation of the oocysts and easy contamination of food and drink are favorable for coccidian infection. Whatever measures will retard the creation of such conditions will reduce hazards from coccidiosis in the case of both domesticated birds and mammals.

The writer is tempted to take a little jibe at some of those practitioners who find in coccidiosis an escape from their inability to determine the cause of death or disease in particular instances. Too often the finding of a few oocysts suggests loss from coccidiosis. As a matter of fact, many species of coccidia are not severe pathogens. Then too, light infections of otherwise highly pathogenic species are usually practically innocuous. Still further, animals recovered from clinical attacks may continue to harbor a so-called chronic infection. These are concepts that may prove helpful.

Pregnancy Tests

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and congested. The undeveloped follicles, which normally are very small, become distended and some of the follicles project like cysts. These are filled with blood and are recognized as corpora hemorrhagica, so characteristic of pregnancy. Many of the very young follicles are unchanged, but the more mature follicles are extremely dilated. The next change seen is the transformation into lutein calls. This striking change is the basis of diagnosis in determining the presence of pregnancy. After taking a glance at a few gross specimens, it becomes an easy matter to distinguish between a positive and a negative case.

The technic consists of isolating each female rabbit in a separate cage and so having her remain until eight days have passed. Rabbits between three and five months of age and weighing at least four pounds are to be preferred. An uncatherized morning specimen of urine from the patient is collected in a clean bottle, and if not used immediately a pinch of boric acid is added as a preservative. The urine is then tested with litmus paper and if found alkaline is acidified with acetic acid. Ten cc. of urine are then injected slowly into the posterior marginal vein of the rabbit's ear. Forty-eight hours later the rabbit is anesthetized and an abdominal incision is made. A gross examination is made to determine if the ovaries contain fresh corpora lutea or bulging corpora hemorrhagica, and if so found, the patient is diagnosed as pregnant. If the ovaries contain neither corpora lutea nor hemorrhagica but only contain clear unruptured follicles, regardless of their size, the assumption is that the woman is not pregnant. The same rabbits may be used for subsequent tests on all negative cases.

The outstanding values of this test may be summarized as follows:

1. To differentiate pelvic tumors and pregnancy.
2. To aid in determining presence of extra-uterine pregnancy.
3. To terminate pregnancy early where organic disease exists which would threaten the mother's life.
4. To determine illegitimate pregnancy early.
5. To differentiate primary and secondary amenorrhea.
6. To aid in determining viability in threatened miscarriage.
7. And finally, there are a great many women who simply want to know whether they are pregnant, and by this test they can be informed within 48 hours instead of waiting 2-3 months for accurate information.

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