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AVIAN BORRELIOSIS

KNOWLES *et al.* (415), Lesbouyries (445), and Loomis (459) presented excellent reviews of this subject.

BORRELIA ANSERINA AND ITS TRANSMISSION

The causative agent, *Borrelia anserina* Sakharoff 1891, was first seen in geese by Sakharoff in Siberia. Marchoux and Salimbeni (472) established the vector and isolated the organism from fowl in Brasil. The common fowl or "blue" tick, *Argas persicus* Oken 1818, is a vector. In Brasil, *A. miniatus* was incriminated as the transmitting agent. The pigeon tick, *Argas reflexus*, is occasionally found infected with *B. anserina* in the Old World.

Argas persicus was the vector in the outbreak of "range paralysis" of fowl in Texas between 1937 and 1939 which claimed a mortality of 27 to 91% (113). Larvae and nymphs of *A. persicus* were found infectious to White Leghorns in Texas (131) and in the U.S.S.R. (540). Transovarian transmission of *B. anserina* in *A. persicus* was emphasized (361, 415, 541). The examination of the ova of *A. persicus* for small forms of borreliae is a tedious task because granules of nonmicrobial origin are present in the ova.

Nikitina (541) stated that the saliva of *A. persicus* also contains borreliae.

The red fowl mite, *Dermanyssus gallinae*, was suggested as an alternate vector. This mite remains infectious for only 3 days, and the organisms do not multiply in it according to Knowles *et al.*

(415). Hungerford and Hart (376) also conducted experiments with *D. gallinae* and were satisfied that it can transmit *B. anserina*.

Zuelzer (747) could not find *Argas* in an outbreak along the Baltic Sea and proposed that *Culex* mosquitoes are vectors of *B. anserina*. This was not confirmed by the experiments of Nieschulz and Bos (539), who observed that while mosquitoes may take up *B. anserina* the organisms disappear from the mosquitoes in a few days. Kapur (396) had the same results with *Anopheles albopictus*. McNeil *et al.* (488) and Loomis (459), studying epidemiology of infected turkeys in California, came to the conclusion that *B. anserina* was transmitted by the feces of the infected birds, or by cannibalism as reported also from India (666).

Artificially induced transmission of *B. gallinarum* by *Ornithodoros moubata* was accomplished in the laboratory (115, 284).

The blood of the chickens is highly infectious for susceptible birds.

Borrelia anserina is 6 to 30, usually 8 to 20 μ long, and 0.2 to 0.3 μ wide, with 5 to 8 spirals of various length. It is actively motile with lashing movements. It was studied under the phase and the electron microscope (229, 673) and its fibrils were observed.

The biochemistry of this *Borrelia* was investigated (385, 626, 653, 654). *B. anserina* gives the impression of being a true anaerobic organism, but does not differ from other borreliae in its enzymatic systems.

B. anserina can be stained or observed by darkfield illumination as can other borreliae. Gross and Ball (325) successfully used fluorescein-labeled antibody to demonstrate *B. anserina*.

B. anserina appears to survive in citrated blood at 0° C for 3 weeks but disintegrates in 10% saponin or bile solutions.

The organism can be cultured in Tyrode's solution or in coagulated egg white with 10% rabbit serum (288), as well as on the chorioallantoic membrane or in the allantoic cavity of developing chick embryos (91, 487).

Kligler *et al.* (413) demonstrated several serologically different types of *B. anserina*. Saurinov and Delamater (623) used agglutination and precipitation tests for the serologic differentiation of *B. anserina*. Convalescent serum kills the organisms. The borreliocidin (borreliolysin) in the convalescent serum is complement-depen-

dent. Together with the immobilizine test the test for borreliocidin is very helpful in the diagnosis of the disease (698).*

PATHOLOGY

The organism appears to remain in the organs although only Himmelweit (359) was able to demonstrate phagocytosis of live *B. anserina*. *B. anserina* has been found in the lung, liver, spleen, kidney, and brain of infected birds but was confined to vascular and interstitial spaces. Antigen has been demonstrated in the tissues (325). Mathey and Siddle (477) studied the pathology in spontaneously infected Mongolian pheasants. There were ecchymoses under the skin, hyalin degeneration of the muscles, sometimes more extensive hemorrhages in subserosal spaces of the gizzard and the heart, eventually with necrotic foci. The spleen was small, whereas it is usually enlarged and mottled with hemorrhagic foci in turkeys afflicted with the disease. The liver is also enlarged, with hemorrhages and focal necrosis. Reddy *et al.* (601) in India found mild meningoencephalitis with perivascular infiltrates and gliosis, and necrotic foci in the kidneys as in infected chickens. The lymph nodes are often enlarged. There is catarrhal enteritis.

COURSE OF THE DISEASE

The disease caused by *B. anserina* is often called avian spirochetosis. Clinically, it begins after an incubation period of approximately 3 to 8 days, usually 4 days. The birds are cyanotic and have yellowish-greenish diarrhea, appear restless, then crouch with closed eyes. If they move, ataxia may become evident. Paralysis of the wings may be present. The body temperature reaches 43° C (109.5° F) or more in 2 to 3 days, sometimes later, and returns to 40° C (104° F) in about a week in those birds which survive the attack. The disease may last about two weeks.

Gabritschewsky in 1898 carried out a long series of experiments to demonstrate the immunologic features of avian borreliosis. Hoffman and Jackson (368) proved the identity of the disease in chickens, ducks, and turkeys. Doves, pigeons, grouse, canaries, and in

*Recently Soumrov *et al.* (Zbl. Vet. Med., *16*:328, 1969) reported excellent results with the precipitin test. Al-Hilly (Amer. J. Vet. Res., *30*:1877, 1969) recommended immunodiffusion as a practical serologic test.

the laboratory also turtledoves and sparrows acquired the infection. In some epidemics, more female than male birds become ill. Death often occurs on the 3rd or 4th day of the disease. The mortality rate may be higher in adult than in younger birds.

Young rabbits can be infected with intravenous injections of *B. anserina* but the organisms disappear in a few days. Guinea pigs, mice, rats, monkeys, and cold-blooded animals are not susceptible to *B. anserina* (86, 415, 459).

No relapses are observed. Immunity or premunition develop, lasting for several months (409) or a year (254).

B. anserina infections have been reported from Europe, Siberia, India, Africa, Australia, Indonesia, South and Central America, lately also from the Southwest and Western regions of the United States and Canada (368, 369, 399, 415, 459, 477, 718). Apparently transmission by feces of infected birds is noted more often at present than in the past.

TREATMENT AND PREVENTION

Packchanian (559) discussed chemotherapy. Penicillin is usually recommended as procaine penicillin, intramuscularly, 100,000 units. Adult chickens may be given 5,000 to 10,000 units divided in 5 to 6 doses over a period of 12 to 15 hours. Baby chicks may require much higher doses.

Diaz Ferrón (238) recommended tetracycline, 125 mg per day. In Europe, oxytetracycline, 2 mg per Kg body weight, is given in one dose, intramuscularly.

Landauer (431) recommended freshly collected blood of infected birds heated to 56° C for 30 minutes as a vaccine. Kolev (418a) in Bulgaria immunized one-year-old birds with 1 ml formolized egg-grown vaccine. Ninety-six per cent of the immunized fowl remained resistant to the disease for one year but only birds older than 4 months responded favorably.

Dickie and Barrera (239) demonstrated that after an outbreak the flock does not harbor the disease for longer than 30 days. General hygienic procedures and quarantine, coupled with antibiotic treatment, should, therefore, be successful.