Chapter 3

Emerging Bacterial Zoonoses in Migratory Birds

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Additional information is available at the end of the chapter

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Abstract

The seasonal variance, global warming, and extraordinary climate conditions around the world change the physiology and behaviors of different animal species. Free ranging birds and mammals harbor some species of potentially pathogenic bacteria; however, these diseases do not result in spontaneous deaths. Being significant individuals of the ecosystem, free living immigrant birds are prone to bacterial diseases. Migratory birds are accommodated in areas located on migration routes to provide rest, food, and water. During this stay, they spread the diseases they bring with them to the poultry in the region and to the poultry farms that do not take adequate biosecurity measures—especially to the free range poultry farms. The migratory birds confront numerous health risks brought on by bacterial species that affect other livestock populace and public health. This chapter provides brief reference on bird-to-bird transmission and general aspects of emerging bacterial zoonoses of migratory birds for wildlife professionals, veterinary practitioners, and students.

Keywords: migratory birds, emerging zoonoses, bacterial diseases

1. Introduction

Numerous wild bird species exist together with people and depend on anthropogenic wellsprings of environment and nutrition. Bird migration is one of the most curious topics in humans about birds' lifetime period. This behavior is one of the most important and critical turning points for birds throughout their lives. Every year more than 50 billion birds in the world migrate, depending on the seasonal change of food resources, and 5 billion birds of 187 species leave Europe and Asia each year and migrate to Africa. Migratory birds relocate a large number of kilometers all through various mainlands and convey certain pathogens. Sixty-one percent of human pathogens are zoonotic, 60.3% of all developing diseases in people are zoonoses, and 71.8% of these initiate in natural life. The recurrence of free living fowl death occasions and the assortment of

irresistible bacterial maladies have expanded extraordinarily amid late decades. Convenient and exact identification of mortality is expected to appropriately guide disease control procedures [1–3]. This review presents brief of emerging bacterial diseases of migratory birds.

2. Emerging bacterial zoonoses in migratory birds

2.1. Avian cholera

Avian cholera is an infectious disease coming about because of contamination by *Pasteurella multocida*. A few subspecies of microscopic organisms have been proposed for *P. multocida*, and 16 distinctive strains have been described [4].

2.1.1. Transmission

Acute pasteurellosis infections are common in worldwide and they can cause bird deaths in 12 h, albeit 24–48 h is typical. Vulnerability to contamination and the formation of malady depends on various factors, including gender, age, and hereditary variety [5]. Many birds harbor the organism in nasal clefts. The presence of the bacterium is generally related to severity of upper respiratory infection in the birds. The enzootic focus of infection is healthy nasal carriers [6]. Transmission to vulnerable birds from contaminated wetlands or from direct bird-to-bird contact is the in all probability routes of transmission amid epizootics (**Figure 1**). Two field cultures were isolated from raccoons that were pathogenic for poultry. Sparrows and pigeons carried organisms without showing clinical signs, but 10% of infected rats developed acute pasteurellosis. The possibility that insects may serve as vectors of FC has been investigated. Transmission by flies, however, is probably not common, as indicated by previous studies. Although FC was maintained in two lots of chickens during the height of the fly season, no spread of the disease occurred to adjoining lots separated only by bird nesting. It was observed that larvae, nymphs, and adult ticks (*Argas persicus*) contained *P. multocida* after feeding on infected hens. A previous demonstration described that the red mite (*Dermanyssus gallinae*) became infected with *P. multocida* after feeding on infected birds, but the mite did not transmit the organism [2].

2.1.2. Clinical aspects

Birds that survive the initial acute septicemic stage may later succumb to the debilitating effects of emaciation and dehydration, may become chronically infected, or may recover. Female Common Eiders are frequently discovered dead sitting on their clutch [7]. Birds with signs suggestive of neurological involvement (unpredictable ungraceful flight, surrounding while at the same time strolling or swimming, or opisthotonos) have additionally been accounted for [8].

2.1.3. Diagnosis

Similarly as with other bacterial diseases, isolation of the causative agent is required for an authoritative identification. To isolate *P. multocida*, sear the tissue or exudate with a spatula and obtain a specimen by inserting a sterile cotton swab or wire loop through the seared surface. If birds are living, squeeze mucus from the nostril or insert a cotton swab into the nasal cleft.

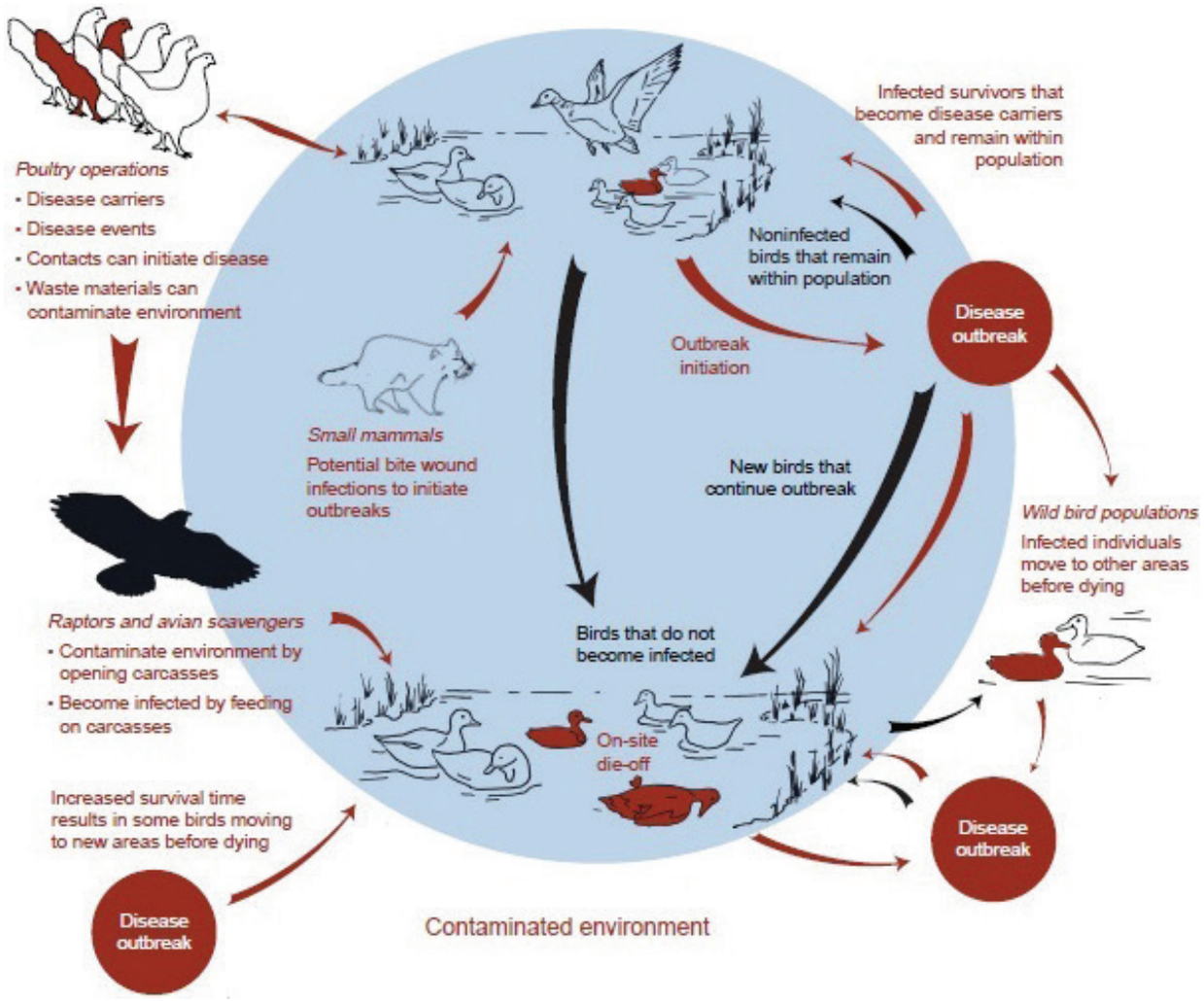

Figure 1. Disease cycle of avian pasteurellosis [1].

Specimens may also be streaked on MacConkey and blood agar media to aid in identification. Colonies characteristic of *P. multocida* are transferred to dextrose starch agar slants incubated 18–24 h. Tubes of phenol red broth base containing 1% glucose lactose, sucrose, mannitol, and maltose are then inoculated with growth from the slant. Fermentation of glucose, sucrose, and mannitol without gas is characteristic of *P. multocida*. Lactose usually is not fermented, but some avian isolates will ferment it. Inoculate 2% tryptose in 0.85% saline solution, incubate 24 h at 37°C, and test for indole (Kovac's test). Indole is almost always produced by *P. multocida*. There should be no hemolysis of blood and no growth on MacConkey agar [9, 10].

2.1.4. Treatment and prevention

Antibacterial chemotherapy has been used extensively in the treatment of FC with varying success, depending to a large extent on the promptness of treatment and drug used. Sensitivity testing is often advantageous, because strains of *P. multocida* vary in susceptibility to chemotherapeutic agents, and resistance to treatment may develop, especially during prolonged use of these agents. Prevention of FC can be effected by eliminating reservoirs of *P. multocida* or by preventing their access to poultry flocks. The choice of adjuvant for an autogenous vaccine

can be water-in-oil emulsion or aluminum hydroxide. Autogenous bacterins using aluminum hydroxide as the adjuvant are useful for the vaccination of turkey breeder or broiler breeder flocks that are in lay because the water-in-oil emulsion, in combination with the whole bacterial cell, results in a significant tissue response by the bird. The use of live FC vaccines stimulates an effective immune response but has the disadvantage of potentially resulting in mortality in the vaccinated birds. If the mortality post vaccination becomes excessive, it can be reduced by the administration of an antibiotic. This should be avoided, if possible, until at least 4 days post vaccination when there will be at least partial immunity induced by the vaccine [11].

2.2. Salmonellosis

The genus *Salmonella* has a broad range of conveyance and is a standout among the most widely recognized reasons for bacterial diarrhea of the bowels in human and animals. In terms of history, clinical signs, epizootiology, lesions, and control and eradication procedures, pullorum disease and fowl typhoid have many similarities. However, differences have been reported for these two diseases, which are caused by these two different serovars (i.e. *Salmonella* Pullorum and *S*. Gallinarum, respectively). These two bacterial taxa are generally regarded as separate serovars, namely *S. enterica* subsp. *enterica* serovar Gallinarum (*S*. Gallinarum) or *S*. Pullorum, but debate continues as to whether they are single or different taxa within the same serovar [12].

2.2.1. Transmission

Salmonellosis can be transmitted from multiple points of view. The relative contribution of vertical transmission in the two organisms is unclear because it is easy to establish persistent infections and egg transmission with *S*. Pullorum [13] but much less easy with *S*. Gallinarum, and it may be that horizontal transmission is more important in this highly virulent organism in which experimental infection generally either results in clinical disease and mortality or no infection depending on the genetic background of the host. It is known that *S*. Pullorum persists within macrophages in the spleen during the carrier state [14]. Although numbers gradually reduce, they are not eliminated [13], and at sexual maturity, the numbers increase as a result of reduced capacity of T cells to respond specifically and nonspecifically to antigens [15], probably following increases in sex hormones. The reduced immune responsiveness enables the bacteria to spread to the reproductive tract.

2.2.2. Clinical aspects

The birds can manifest somnolence, weakness, depressed appetite, poor growth, and adherence of chalky white material to the vent. Death ordinarily follows inside 24 hours. On the off chance that enteritis occurs, there might be diarrhea, making the vent pasted with liquid defecation and urates [12].

At the point when the esophagus is cut open, the nodules might be viewed as huge, diffuse plaque-like nodules or as discrete, nodular regions inside the esophagus (**Figure 2**). Occasional cases of PD can be subclinical, even though the disease may originate by egg transmission. Mortality usually peaks during the second or third week of life. In these situations, the birds exhibit lassitude and

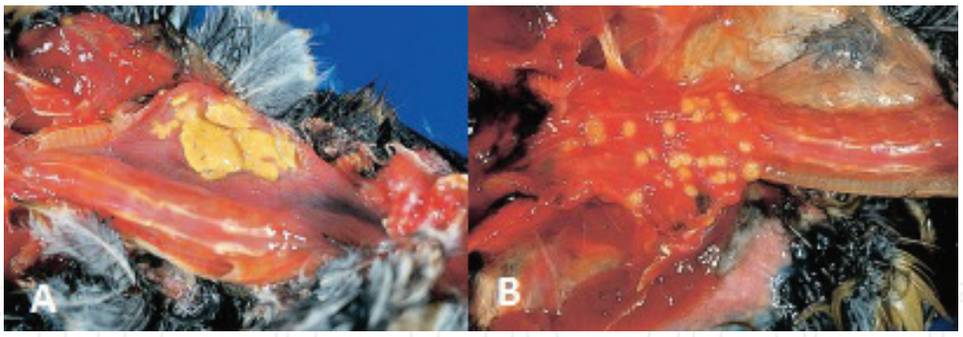

Figure 2. (A) Diffuse plaque-like nodules. (B) discrete, nodular regions inside the esophagus [1].

an inclination to huddle together under heaters, having droopy wings and distorted body appearance. Labored breathing or gasping may be observed as a result of extensive involvement of the lungs due to PD. Survivors may be greatly retarded in their growth and appear underdeveloped and poorly feathered. In certain instances, a relatively high incidence of infection in the joints, which can produce lameness and obvious joint enlargement, can occur in juveniles. In acute to subacute cases, there is multifocal necrosis of hepatocytes. In chronic cases, especially in cases in which there are large nodules in the heart, the liver will have chronic passive congestion with interstitial fibrosis. The spleen may have severe congestion or fibrin exudation of vascular sinuses in acute stages and severe hyperplasia of the mononuclear phagocytic system cells in later stages. The ceca in young chicks may have extensive necrosis of the mucosa and submucosa, with an accumulation of necrotic debris mixed with fibrin and heterophils in the lumen [1].

2.2.3. Diagnosis

The clinical signs and lesions produced by PD or FT are not pathognomonic. Other *Salmonella* infections may produce similar lesions in the liver, spleen, and intestine, which cannot be distinguished grossly or microscopically from those produced by PD or FT. Aspergillus or other fungi may produce similar lesions in the lungs. *S*. Pullorum and *S*. Gallinarum can localize in major joints and tendon sheaths of chicks. Such signs and lesions resemble those produced by organisms such as *Mycoplasma synoviae*, *Staphylococcus aureus*, *Pasteurella multocida*, or *Erysipelothrix rhusiopathiae*. Local infections with *S*. Pullorum and *S*. Gallinarum in adult carriers, particularly of the ovary, may appear identical to those produced by other bacterial infections such as coliforms, staphylococci, *P. multocida*, streptococci, and other *Salmonellae*. Birds of any age may be infected with *S*. Pullorum or *S*. Gallinarum but fail to show grossly discernible lesions. A definitive diagnosis of PD and FT can be made only following the isolation and identification of *S*. Pullorum and *S*. Gallinarum, respectively [2]. The bacteria can be identified by molecular procedures also [16, 17].

2.2.4. Treatment and prevention

The disinfection must be done daily with hypochlorite-type solutions in feeding points of free-ranging birds in consequence of sudden outbreaks. Reasonably effective prophylactic and therapeutic drugs for poultry production have been developed against PD and FT. Treatment is

generally neither feasible nor desired. Sulfonamides, in particular, frequently suppress growth and may interfere with feed and water intake and egg production. Sulfonamides that have been used in the treatment of PD and FT include sulfadiazine, sulfamerazine, sulfathiazole, sulfamethazine, and sulfaquinoxaline. Transmission through shell penetration and feed contamination by *S*. Pullorum has been reported can be partially prevented only by formaldehyde fumigation [18].

2.3. Avian botulism

Synonyms for botulism are "Limberneck" and "Western duck sickness." Free-ranging and confinement-reared poultry and feral birds can be affected. Most avian cases are caused by *C. botulinum* type C or mosaic type C/D, although outbreaks due to other toxin types have been described [19, 20]. Nonhuman primates, however, have succumbed to type C botulism [2].

2.3.1. Transmission

Laboratory examinations exhibited that decomposing flesh contaminated with botulinum cells or spores can support the production of high amounts of toxin. Waterbirds and different vertebrates unintentionally digest bacteria spores while feeding and convey them in their tissues. Upon death, the subsequent anaerobic condition and rich protein source of cadaver are ideal for vegetation of spores and toxin formation [21, 22].

Avian type C botulism can be caused by ingestion of preformed toxin or by toxico-infection. More than 2000 minimum lethal doses (MLD) of type C toxin/gram of carcass tissue of intoxicated birds have been found. Birds scavenging such carcasses can readily obtain enough toxins to become affected. Fly-blown carcasses may have maggots containing 104–105 MLD of neurotoxin. In aquatic environments, small crustaceans and insect larvae may contain *C. botulinum* in their gut. If large numbers die due to oxygen depletion, toxin can be produced within these invertebrates. Ingestion of toxin-laden invertebrates has been proposed as the cause of type C botulism in ducks [23].

2.3.2. Clinical aspects

Clinical signs of botulism in chickens, turkeys, pheasants, and ducks are similar. In chickens, flaccid paralysis of legs, wings, neck, and eyelids are predominant features of the disease. Wings droop when paralyzed. Limberneck, the original and common name for botulism, precisely describes the paralysis of the neck. Because of eyelid paralysis, birds appear comatose and may seem dead. Gasping has been reported when birds are handled. Death results from cardiac and respiratory failure. Affected chickens have ruffled feathers, which may fall out with handling. Quivering of certain feather tracts has been observed. Broiler chickens showing signs of botulism may have diarrhea with excess urates in the loose droppings [2].

2.3.3. Diagnosis

Isolation of *C. botulinum* is of little help in diagnosis. However, detection and isolation of the organism in clinical samples from animals or feed or environmental samples may prove

useful in epidemiologic studies. The organism can be cultivated anaerobically at 30–42°C in cooked meat medium or trypticase-peptone-glucose-yeast (TPGY) medium [19, 24]. After 2 days of incubation, the sample can be analyzed for the presence of the neurotoxin or the neurotoxin gene. Real-time PCR assays have been developed for detection of the BoNT gene of types A–F [25, 26].

2.3.4. Treatment and prevention

Antitoxins can be applied to sick captive birds in early stage of the malady. The most widely recognized strategy for preventing disease is by evacuation of cadavers before development of flies in order to counteract distribution of toxin to different bird populations. In problem areas, removal of contaminated litter and thorough disinfection using calcium hypochlorite or formalin may help reduce spore numbers in the environment. Disinfection of areas around poultry houses has been recommended because spores may be located in the soil outside of the poultry facility and can be transported back into houses. Fly control may be another means of reducing the risk of toxic maggots in the environment. Two cases of type C botulism were reported in commercial broilers and were associated with elevated intake of iron from water and feed sources. However, the relationship between iron and toxicoinfectious type C botulism needs to be experimentally confirmed [27].

2.4. Avian tuberculosis

Mycobacterium avium complex (MAC) which along with *M. genavense* is responsible for most cases of avian mycobacteriosis. Less generally, tuberculosis in fowls is caused by *M. intracellulare*, *M. fortuitum*, *M. tuberculosis*, *M. gordonae*, and *M. nonchromogenicum* [28, 29].

2.4.1. Transmission

Nontuberculous mycobacteria, including MAC, are ubiquitous in the environment and are commonly isolated from soil and water. Humans become infected by ingestion or inhalation of MAC organisms from the environment. Infected animals and birds commonly shed mycobacteria in their feces, but are not considered to be an important source of human infections. The contaminated environment, especially soil and litter, is the most important source for the transmission of the bacilli to uninfected animals. The longer the premises have been occupied by infected birds and the more concentrated the poultry population, the more prevalent the infection is likely to be [30, 31].

2.4.2. Clinical aspects

The liver (**Figure 3A**) generally contains similar nodules, but intestines (**Figure 3B**), spleen (**Figure 3C**), and lung can present such nodules. Aggregations of these nodules may appear as firm, fleshy, grape-like clusters. Abscesses and nodular growths (**Figure 4**) have been reported on the skin of birds in the same locations where pox lesions are frequently seen around the eyes, at the wing joints, on the legs, side of the face, and base of the beak [1].

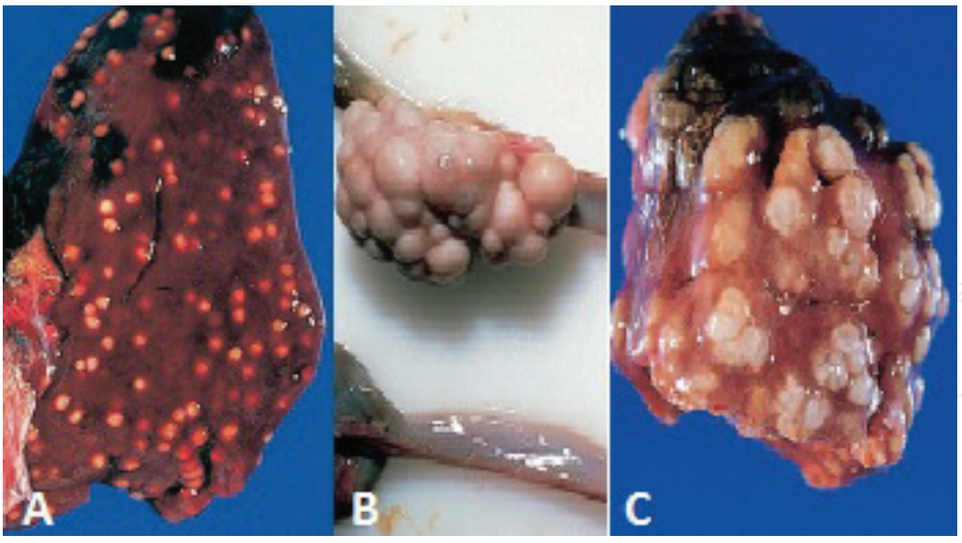

Figure 3. The raised, firm nodules in these organs are typical lesions of avian tuberculosis. (A) Liver; (B) intestine; and (C) spleen [1].

Figure 4. Nodular lesion, which was caused by avian tuberculosis, on the skin of a canvasback [1].

2.4.3. Diagnosis

Demonstration of acid-fast bacilli in smears or histologic sections of liver, spleen, or other organs strengthens the diagnosis and is sufficient for most diagnostic cases. In live, suspected infected birds, fecal smears for culture, staining, and/or PCR may be attempted but these tests are not reliable due to intermittent or no fecal shedding of bacilli [32]. Fecal positivity increases as the disease course progresses [33]. PCR has been used to detect mycobacteria, including *M. avium* and *M. genavense,* in formalin-fixed tissue, which may further aid diagnostic considerations [34]. PCR may also be used to detect mycobacteria in organ samples as well as further differentiate isolates [35].

2.4.4. Treatment and prevention

Control of avian tuberculosis in free-living birds is not viewed as plausible on the field since the bacteria perseveres in the field, is resistant to numerous tuberculosis medications and detergents, and it is hard to isolate from sick birds. Treatment with antituberculosis drugs is impractical; however, combinations of isoniazid (30 mg/kg), ethambutol (30 mg/kg), and rifampicin (45 mg/kg) may be applied to captive birds. The recommended duration of therapy is 18 months, provided that there were no adverse side effects [36].

2.5. Avian chlamydiosis

Chlamydiosis alludes to an infection with microorganisms of the genus *Chlamydia* sp., which is a microbe that lives within animal cells. *Chlamydia psittaci* is the species generally associated with this disease in birds. Avian chlamydiosis is caused by the bacterium *Chlamydia psittaci*. Avian chlamydiosis is a respiratory disease, usually systemic and occasionally fatal. *Chlamydia psittaci* can be transmitted to humans. The disease in birds and humans originally was called psittacosis or parrot fever because it was first recognized in psittacine birds and in humans associated with psittacine birds [3].

2.5.1. Transmission

Infection usually occurs with the inhalation of bacteria that are released into the air from birds' feather patches. Contagion also develops via beak-to-beak feeding from mother bird to juveniles. Since the chlamydia is not completely eliminated, reinfection and damage to host tissues continue. In persistent infections, the inflammatory response increases, chronic inflammatory advances continue in the focal areas, and the etiologic agent is shed from the damaged tissues. Vertical transmission has been demonstrated in ducks, parakeets, seagulls, and snow geese [37, 38].

2.5.2. Clinical aspects

Fowls frequently end up noticeably feeble, quit eating, and create purulent (liquid containing discharge) releases of the eyes and nares. Birds have a tendency to wind up plainly still, stay in a settled position, and crouched up with unsettled feathers [39]. Signs of chlamydiosis in turkeys infected with virulent strains are cachexia, anorexia, elevated body temperature, conjunctivitis, and respiratory distress. Diseased birds excrete yellow-green, gelatinous droppings. Egg production of severely affected hens declines rapidly to 10–20% and may temporarily cease or remain at a very low rate until complete recovery. Disease signs in a flock infected with strains of low virulence are usually anorexia and loose, green droppings in some birds, with less effect on egg production. In overwhelming infections with virulent strains, lungs show diffuse congestion, and the pleural cavity may contain fibrinous exudate. In fatal cases, a dark transudate may fill the thoracic cavity. The pericardial membrane is thickened, congested, and coated with fibrinous exudate. The liver is enlarged and discolored and may be coated with thick fibrin in birds that survive infection with a strain of low virulence, the

lungs may not be seriously affected. However, multiplication of organisms on the epicardium may result in the formation of one or more fibrin plaques [3].

2.5.3. Diagnosis

The recommended medium for chlamydiae consists of SPG buffer. For isolation, the following samples should be preferably collected: pharyngeal/choanal slit swabs in live birds. Cloacal swabs or fresh feces are less optimal because chlamydial shedding is intermittent. In dead birds, lungs, spleen, and liver can be sampled. The specimen should be stored at −80°C if it will not be sent to laboratory immediately [40]. This methodology would be the same or comparative for migratory birds.

2.5.4. Treatment and prevention

Chlamydiosis treatment for poultry has not changed over the years. The drug of choice varies from country to country. Among tetracylines, which are the drugs of choice, chlortetracycline and doxycycline are most often used. Enrofloxacin (fluoroquinolone antibiotic) can also be used. Contact with potential reservoirs or vectors such as pet birds, rodents, arthropods, and wild and feral birds should also be prevented. General sanitation must be practiced diligently. Movement of people should be restricted so that visitors do not have free access to premises holding birds. This is easier to accomplish if birds are confined in houses and if the "all-in-all-out" principle is used on the farm [41].

2.6. Mycoplasmosis

Mycoplasma phylogeny and taxonomy continue to be re-examined by the application of molecular tools such as DNADNA hybridization DNA sequence analysis of the 16S rRNA gene, 16S rRNA PCR and denaturing gradient gel electrophoresis, and tRNA gene PCR. The complete genome sequence has been determined for MG strains Rlow, Rhigh, and F [42], and a database dedicated to the comparative genomics of Mollicutes, including MG, has been established [3].

2.6.1. Transmission

Experimental intra-crop inoculation of house finches resulted in infection, disease, and a serological response [43]. *M. gallisepticum* seldom survives for more than a few days outside of a host, so clinical or subclinical carrier birds are essential to the epidemiology of MG diseases. However, additional transmission and more widespread disease outbreaks may occur via fomites-contaminated airborne dust and droplets, or feathers, coupled with suboptimal biosecurity and personnel practices. In house finches, experimentally infected birds were demonstrated to indirectly infect naive birds through contacted bird feeders and support the possible transmission of MG by fomites. Experimental research show that transmission happens between sick grown-up house finches and their posterity [2]. Avian mycoplasmosis can happen whenever of the year however for the most part a higher commonness has in the winter [44, 45].

2.6.2. Clinical aspects

In house finches, MG causes mild-to-severe eyelid swelling, conjunctivitis, and watery discharge from one or both eyes as well as nares. Air sacs frequently contain caseous exudate that may be focal, multifocal, or diffused. Conjunctivitis with periocular swelling and inflammation are characteristics of MG in house finches and other songbirds [46].

2.6.3. Diagnosis

The gold standard for MG diagnosis is isolation and identification of the organism. In some cases, the isolation of MG in culture is impaired by the overgrowth of saprophytic mycoplasmas that inhabit the upper respiratory tract of avian species and contaminant bacteria and fungi that may not be successfully inhibited by mycoplasma-selective media. To culture MG, fluid sinus exudate should be inoculated directly to mycoplasma broth and/or agar media [47]. Swabs can also be taken from the trachea or choanal cleft (palatine fissure) for MG culture. *M. gallisepticum* may also be present in oviducts [48] and has been isolated from the cloaca of turkeys and chickens [49]. Detection of MG using DNA and ribosomal RNA gene probes has been described, but for most applications these methods have been superseded by various PCR-based procedures that are relatively less complex and more rapid, sensitive, and specific. Multiplex PCR protocols have been described, which allow for the simultaneous detection of different organisms [3]. A test based on amplification of the 16S rRNA gene with "Mycoplasma specific" primers and separation of the PCR product by denaturing gradient gel electrophoresis has been described [50]. Detection of MG-specific DNA by PCR has become the frontline approach at diagnostic and institutional laboratories using commercial conventional PCR kits or established protocols [51]. More rapid and highly specific detection by quantitative PCR methods has also been described [3]. Detection of MG DNA by PCR compared to isolation of the organism in culture provides a negative or positive result in hours instead of days, does not rely on the presence of viable organisms, and is not susceptible to saprophytic mycoplasmas and microbial contaminants. However, culture and isolation of MG organisms remain essential for further studies such as experimental infections, pathogenicity studies, and intra-species (strain) identification. An inoculated mycoplasma broth can be divided and processed for both culture and PCR. When culture and isolation of viable organisms are not necessary or possible, FTA filter paper may be used for the inactivation and storage of MG suspensions or field specimens prior to PCR or other DNA-dependent assays [52]. A positive serologic test together with history and clinical signs typical of MG disease allows a presumptive diagnosis pending isolation and/or identification of the organisms [3].

2.6.4. Treatment and prevention

M. gallisepticum is inherently resistant to penicillins or other antibiotics, which act by inhibiting cell wall biosynthesis [53]. Tylosin and tilmicosin are reported to be effective in house finches [54, 55]. Close perception of birds and prompt detailing of outbreaks to experts will give the chance to early intercession in view of convenient determination and for starting a proper disease control technique particular to the area and populace included [3].

2.7. Borreliosis

Borrelia sp. is highly motile, helical spirochetes that stain well with aniline dyes, hematologic stains, and silver impregnation. Spirochetes can be readily identified in wet smears of blood or tissues by dark-field or phase microscopy [3]. *Borrelia anserina* causes nonrelapsing, tick-borne spirochetosis in avian species, including chickens, turkeys, pheasants, geese, and ducks, in tropical and subtropical areas. Occasional outbreaks have been identified in the southwestern United States in chickens, turkeys, and pheasants [56]. Extensively reared free-range flocks are more likely to be affected than confined flocks, and indigenous breeds of chickens are generally more resistant than exotic breeds. The disease is usually an acute septicemia characterized by high morbidity and mortality, but may be mild if birds are infected with low-virulent strains [57]. Birds can also develop asymptomatic infections with *B. burgdorferi*, the cause of Lyme disease in people, and serve as hosts for ticks capable of spreading the spirochete to mammals. Wild turkeys are also hosts for *B. lonestari* and *B. miyamotoi*. No clinical disease has been recognized in birds infected with Borrelia species other than *B. anserina*. Occurrence of spirochetosis corresponds with the distribution of fowl ticks in the genus Argas, which serve as both the reservoir and primary vector. Attempts to transmit *B. anserina* with the tick *Amblyomma cajennense* were unsuccessful. In addition to ticks and other biting arthropods (mosquitoes, mites), infection can result from cannibalism; scavenging on carcasses; multiple use of syringes and needles; or ingestion of infective blood, droppings, or infected ticks. Virulent strains are capable of penetrating unbroken skin. *B. anserina* is not resistant outside of the host. Recovered birds are not carriers; organisms disappear from tissues at or shortly after they disappear from the circulation. Birds infected with virulent strains of *B. anserina* are visibly sick, with cyanosis or pallor of the comb and wattles, ruffled feathers, dehydration, inactivity, and anorexia. A marked elevation in body temperature begins shortly after infection accompanied by rapid weight loss. Affected birds pass fluid green droppings containing excess bile and urates and have increased water consumption. Late in the disease, birds develop paresis or paralysis, become anemic, and are somnolent to comatose. Body temperatures are subnormal just prior to death. Birds recovering from the disease are often emaciated and have temporary residual weakness or paralysis. Infection with low-virulent strains may be mild or inapparent. Marked enlargement and mottling of the spleen is typical of spirochetosis but may not be evident when birds are infected with low-virulent strains or early in the disease [58]. Livers are often enlarged and contain small hemorrhages, pale foci, or marginal infarcts. Kidneys are swollen and pale with excess urates distending the ureters. Green, mucoid intestinal contents are usually present, and often there are variable amounts of hemorrhage, especially at the proventriculus-ventriculus junction. Fibrinous pericarditis occurs infrequently. Extensive hemorrhage and muscle necrosis occur in naturally infected pheasants. Splenic lesions result from macrophage and lymphoid hyperplasia, erythrophagocytosis, and hemosiderin deposition. Multifocal necrosis and hyalinization of white pulp and/or extensive hemorrhage may be present in some birds. The liver is congested with increased periportal infiltrates of mixed lymphocytes, hemocytoblasts, and phagocytic cells with vacuolated cytoplasm. Erythrophagocytosis and hemosiderin are seen in Kupffer cells. Extramedullary hematopoiesis may be present. Lymphoplasmacytic infiltrates occur in kidneys and intestinal lamina propria of some birds. Occasionally, there is mild-to-moderate

lymphocytic meningoencephalitis. Spirochetosis can be tentatively diagnosed by finding characteristic lesions in birds with signs consistent with the disease. Diagnosis is confirmed by demonstrating *B. anserina* in blood or tissue sections. In chickens exposed to ticks (*Argas miniatus*) infected with *B. anserina*, spirochetes were found in blood smears prepared from the exposed birds between day 5 and day 12 post exposure, with the peak number of spirochetemic birds occurring between days 7 and 9. Spirochetes were not found in blood smears from any of the exposed birds after day 13 [59].

Borrelia sp. cannot be cultured on routine bacteriologic media but will grow in chick embryos following yolk sac inoculation or in susceptible young chicks or poults. It can be grown in liquid medium but loses virulence [60]. Bursectomy or dexamethasone treatment of chicks may be necessary to detect low-virulent strains. Isolates are usually maintained in ticks, day-old chicks, and chicken or turkey embryos or by cryopreservation (−70°C or in liquid nitrogen) in 5% glycerol or dimethylsulfoxide added to infective blood [58]. Several serologic methods have been used to detect antibodies in immune birds. Spirochetal antibodies occur in yolk of eggs from immune hens [60]. Arsenicals and most antibiotics, including penicillin, chloramphenicol, kanamycin, streptomycin, tylosin, and tetracyclines, are effective in treating infected birds. Intramuscular injections of penicillin at 20,000 IU/bird given three times in 24 h or 20 mg oxytetracycline given daily for 2 days represent current treatment regimens. Active immunity follows recovery or immunization. Immunity is serotype-specific; infection with other *B. anserina* serotypes can occur in recovered or vaccinated birds. An autogenous or polyvalent vaccine containing multiple serotypes may be necessary to provide full protection. Controlled infection followed by antibiotic treatment 3 days later has also been used to induce active immunity. Passive maternal immunity provides protection for 5–6 weeks. Preventing fowl tick infestation is the best method to control spirochetosis in endemic areas. Young chickens in dense poultry areas during the summer are more likely to be infested with fowl ticks. Adult ticks can remain alive without feeding and carry the spirochete for as long as 3 years [61].

2.8. Tularemia

Tularemia is essentially a malady of mammals, yet normal diseases by *Francisella tularensis* have caused deaths of ruffed grouse and other grouse species. An assortment of avian animal categories has been observed to be vulnerable to contamination because of serological studies that have identified counter antibody against tularemia, test concentrates to decide susceptibility, and by reason for death evaluations for birds submitted for necropsy. Ticks are responsible of disease outbreaks in birds [1].

There are no reports of the clinical course in normally infected birds, and clinical signs in wild animals are inadequately archived, principally because of the acute character of tularemia in many species [2].

The bacteria can also be identified in culture or specimens by hybridization with probes specific to the 16S rRNA gene of *F. tularensis,* and types A and B can be distinguished by this method [62].

3. Conclusion

Avian cholera has become the most important emerging bacterial disease of waterbirds and geese. The vast majority of the geographic extension and expanded recurrence of outbreaks of avian cholera has happened since 1970. The high prevalence of avian tuberculosis disease that has happened since 1982 has challenged the survival of crane populations. Salmonellosis has turned into a noteworthy source of mortality at poultry breeders all through the world, and mycoplasmosis in house finches has turned into the most quickly spreading infection at any point found in free living birds. Avian botulism has likewise extended in geographic circulation and has increased expanded noticeable prominence as a disease of waterbirds. It is, without a doubt, the most critical malady of waterbirds around the world. The geographic expansion of avian botulism has mostly occurred during the past quarter century. Of the diseases addressed in this section, chlamydiosis and tick-borne pathogens pose the greatest risk to public health and other livestock, especially poultry production. Avian tuberculosis can be a significant risk for humans who are immunocompromised. Salmonellosis is a common, but seldom fatal, human infection that can be acquired from infected wild birds. Convenient and exact identification of mortality is expected to appropriately guide disease control procedures. Keeping in mind the main scope to precisely identify which diseases are emerging and zoonotic in the field, specimen should be sent to avian research laboratories, which know about the wide assortment of conceivable diseases that may infect wild birds and domestic livestock.

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