Pasteurellosis and other respiratory bacterial infections

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Published in:
Diseases of poultry

Publication date:
2013

Document version
Publisher's PDF, also known as Version of record

Diseases of Poultry

13th EDITION

Editor in Chief: David E. Swayne

Associate Editors: John R. Glisson, Larry R. McDougald, Lisa K. Nolan, David L. Suarez, and Venugopal Nair

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AMERICAN ASSOCIATION OF AVIAN PATHOLOGISTS

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Third edition, © 1952 Iowa State University Press
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Twelfth edition, © 2008 Blackwell Publishing Ltd.

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Library of Congress Cataloging-in-Publication Data

Diseases of poultry. – 13th ed. / editor-in-chief, David E. Swayne ; associate editors, John R. Glisson ... [et al.].
p. ; cm.
Includes bibliographical references and index.
ISBN 978-0-470-95899-5 (hardback : alk. paper)
I. Swayne, David E.
[DNLM: 1. Poultry Diseases. SF 995]
SF995
636.5'0896 dc23

2013007104

A catalogue record for this book is available from the British Library.

Wiley also publishes its books in a variety of electronic formats. Some content that appears in print may not be available in electronic books.

Cover design by Modern Alchemy LLC

Set in 9.5/12pt Times New Roman by SPi Publisher Services, Pondicherry, India

Printed and bound in Singapore by Markono Print Media Pte Ltd

1 2013

Dedicated to

Dr. Y.M. Saif, Associate Editor,
Diseases of Poultry, 10th edition
Editor-in-Chief, 11th and 12th editions

Dr. A.M. Fadly, Associate Editor,
Diseases of Poultry, 11th and 12th editions

Chapter 19

Pasteurellosis and Other Respiratory Bacterial Infections

Introduction

John R. Glisson

A number of distinct respiratory diseases are caused by small Gram-negative bacteria in commercial poultry. These diseases may have very similar clinical presentations. Many of the etiologic agents of bacterial respiratory diseases are classified as members of the family Pasteurellaceae but have in recent years been redesignated to new genera. Recent reclassifications included changing the name of *Pasteurella haemolytica* to *Gallibacterium anatis* biovar *haemolytica*, *Pasteurella gallinarum* to *Avibacterium gallinarum*, and *Haemophilus paragallinarum* to *Avibacterium paragallinarum*. The current taxonomy reflects advancements in techniques for determining genetic relatedness among bacteria. The new taxonomy is used in this text.

Four distinct diseases are included in this chapter: fowl cholera caused by *Pasteurella multocida*, *Riemerella anatipestifer* infection, *Ornithobacterium rhinotracheale* infection, and bordetellosis. These diseases are grouped together because they are caused by organisms that are genotypically and phenotypically related and because they induce diseases in commercial poultry that may present in a clinically similar way. Other diseases of poultry caused by members of the family Pasteurellaceae, such as fowl coryza caused by Avibacterium paragallinarum, are presented elsewhere in this text because the disease produced by infection with these organisms presents distinctly differently from the diseases included in this chapter.

In diagnostic poultry medicine, definitive diagnosis of fowl cholera, *Riemerella anatipestifer* infection, *Ornithobacterium rhinotracheale* infection, and bordetellosis depends upon the isolation and identification of the causative organism. Several organisms, such as *Avibacterium gallinarum*, which are less important as disease agents, may be isolated and must be differentiated from the more important disease agents included in this chapter. A clinical diagnostic text will be helpful in this regard (1).

Fowl Cholera

John R. Glisson, Charles L. Hofacre, and Jens P. Christensen

Introduction

Fowl cholera (FC) (avian cholera, avian pasteurellosis, or avian hemorrhagic septicemia) is a contagious disease affecting domesticated and wild birds. It usually appears as a septicemic disease associated with high morbidity and mortality, but chronic or benign conditions often occur. This disease historically important because of its role in the early development of bacteriology and because it was 1 of 4 diseases the Veterinary Division of the United States Department of Agriculture (USDA) was created to investigate.

History

Several epornitics among fowl occurred in Europe during the latter half of the 18th century. The disease was studied in France in 1782 by Chabert and in 1836 by Mailet, who first used the term fowl cholera. Huppe in 1886 referred to hemorrhagic septicemia, and Lignieres in 1900 used the term avian pasteurellosis. Benjamin in 1851 gave a good description of the disease and demonstrated that it could be spread by cohabitation. With this knowledge of the disease, he formulated procedures for its prevention. At about the same time,

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Renault, Reynal, and Delafond demonstrated its transmissibility to various species by inoculation. In 1877 and 1878, Perroncito of Italy and Semmer of Russia observed in tissues of affected birds a bacterium that had a rounded form and occurred singly or in pairs. In 1879, Toussaint isolated the bacterium and proved it was the sole cause of the disease (52).

Pasteur (126) isolated the organism and grew pure cultures in chicken broth. In further studies, Pasteur (127, 128) used the FC organism to perform his classic experiments in attenuation of bacteria for use in producing immunity. Salmon (153) appears to have been the first to study the disease in the United States. A good description of disease signs was reported, however, as early as 1867 in Iowa, where losses of chickens, turkeys, and geese had occurred (4).

Etiology

Classification

Pasteurella multocida is the causative agent of FC. When pronouncing multocida, the accent should be on the "ci" (16) rather than on the "to" as given in the 7th and 8th editions of *Bergey's Manual*. In the past, the bacterium has been given many names, including *Micrococcus gallicidus*, 1883; *M. cholerae gallinarum*, 1885; *Octopsis cholerae gallinarum*, 1885; *Bacterium cholerae gallinarum*, 1886; *Bacillus cholerae gallinarum*, 1886; *P. cholerae-gallinarum*, 1887; *Coccobacillus avicidus*, 1888; *P. avicida*, 1889; *Bacterium multicidum*, 1899; *P. avium*, 1903; *Bacillus avisepticus*, 1903; *Bacterium avisepticum*, 1903; *Bacterium avisepticus*, 1912; and *P. aviseptica*, 1920 (16, 19).

For a while, each isolate of *P. multocida* was named according to the animal from which it was isolated, such as *P. avicida* or *P. aviseptica*, *P. muricida* or *P. muriseptica*. In 1929, it was suggested that all isolates be referred to as *P. septica* (170). This name was used mainly in the United Kingdom and can be found in recent literature. *Pasteurella multocida*, proposed by Rosenbusch and Merchant (151), is now accepted as the official name in *Bergey's Manual* and is used exclusively throughout the world.

Morphology and Staining

P. multocida is a Gram-negative, nonmotile, nonspore-forming rod occurring singly, in pairs, and occasionally as chains or filaments. It measures 0.2–0.4 × 0.6–2.5 mm but tends to become pleomorphic after repeated subculture. A capsule can be demonstrated in recently isolated cultures using indirect methods of staining (Figure 19.1). In tissues, blood, and recently isolated cultures, the organism stains bipolar (Figure 19.2). Pili have been reported (49, 135).

Growth Requirements

P. multocida grows aerobically or anaerobically. The optimal growth temperature is 37°C. The optimal pH range is 7.2–7.8, but growth can occur in the range 6.2–9.0, depending upon composition of the medium. In liquid media, maximum growth is obtained in 16–24 hours. The broth becomes cloudy,

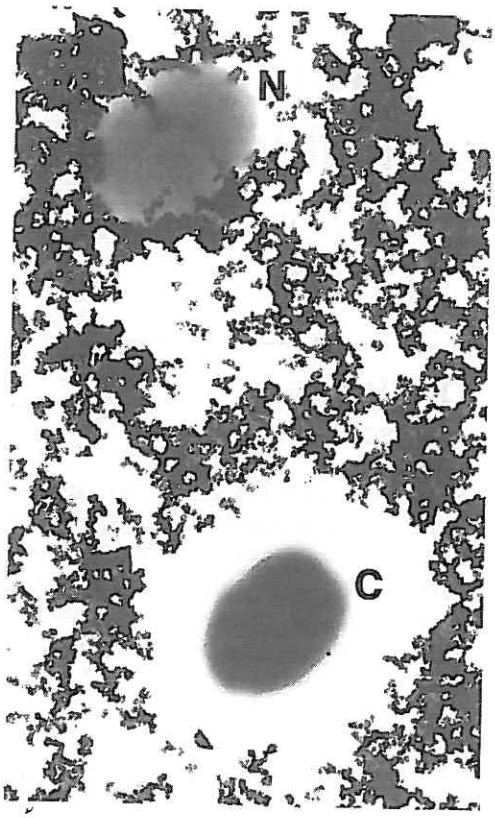

Figure 19.1. Electron photomicrograph of *Pasteurella multocida*-encapsulated cell (C) and nonencapsulated cell (N) suspended in India ink. ×19,000.

and in a few days, a sticky sediment collects. With some isolates, a flocculent precipitate occurs.

The bacterium will grow on meat infusion media; growth is enhanced when the medium is enriched with peptone, casein hydrolysate, or avian serum. Blood or serum from some animals inhibits growth of *P. multocida*. Inhibition is greatest from blood of horses, cattle, sheep, and goats; blood of chickens, ducks, swine, and water buffalo has little or no inhibitory action (152). Several selective media for isolation have been described (27, 28, 44, 99, 111, 160). Chemically defined media have been described by Jordan (92), Watko (167), Wessman and Wessman (169), and Flossmann et al. (42). Berkman (8) found that pantothenic acid and nicotinamide are essential for growth. Dextrose starch agar with 5% avian serum is an excellent medium for isolating and growing *P. multocida*.

Colonial Morphology and Related Properties

Colonial morphology observed with obliquely transmitted light is one of the most useful characteristics in the study of *P. multocida*. On primary isolation from birds with FC, colonies may be iridescent, sectored with various intensities of iridescence, or blue with little or no iridescence (Figure 19.3). Iridescence is related to the presence of a capsule. The term

FC and fowl typhoid or fowlpox were concurrently present, chloramphenicol treatment was not successful (81). A chloramphenicol-dexamethasone-pyribenzamine combination was used successfully with vaccination in treatment of FC in breeding turkeys. Respiratory problems, which occurred 1 week after the initial outbreak, responded readily to IM administration of this drug combination (51). Water-soluble erythromycin at the rate of 1 lb/50 gallon of drinking water halted mortality in t2wo flocks of Muscovy ducklings infected with *P. multocida* (62). Fluoroquinolones are used successfully to treat FC. *Pasteurella multocida* isolates from poultry are typically highly susceptible to fluoroquinolones (47).

Antibiotics used in rations at very low levels for promotion of growth, according to the experiments of Dorsey and Harshfield (36), did not significantly influence the course of FC infection in inoculated birds. At therapeutic levels, birds that received penicillin and streptomycin in feed died at about the same rate as controls. No deaths occurred in groups that received sulfaquinoxaline or sulfamerazine. These workers also found oxytetracycline and chlortetracycline effective in preventing mortality in experimental FC in a small flock of laying birds; mortality was 80% in an untreated group compared with 12% in a group receiving mash containing oxytetracycline at the level of 500 g/ton. In 6 naturally occurring outbreaks, oxytetracycline at this level in feed checked mortality, but losses returned in 3 flocks after withdrawal of the antibiotic.

Prevention and Control

Management Procedures

Prevention of FC can be effected by eliminating reservoirs of *P. multocida* or by preventing their access to poultry flocks. Good management practices, with emphasis on sanitation, are the best means of preventing FC. Unlike many bacterial diseases, FC is not a disease of the hatchery. Therefore, infection occurs after birds are in the hands of the producer, and consideration must be given to the many ways that infection might be introduced into a flock.

The primary source of infection is usually sick birds or those that have recovered and still carry the causative organism. Only young birds should be introduced as new stock; they should be raised in a clean environment completely isolated from other birds. Isolation should be extended to housing. Unless separate houses can be provided for first- and second-year layer flocks, the older flock should be marketed in its entirety. Different species of birds should not be raised on the same premises. The danger of mixing birds from different flocks cannot be overemphasized. Farm animals (particularly pigs, dogs, and cats) should not have access to the poultry area. Water fountains should be self-cleaning, and feeders should be covered to prevent contamination as much as possible.

P. multocida has been recovered from many species of free-flying birds and warrants consideration as source of bacteria to poultry; wit measures should be taken to prevent their association with the flock. Raising turkeys in areas where FC is a serious problem may warrant their confinement in houses from which free-flying birds, rodents, and other animals can be excluded. If an outbreak of FC occurs, the flock should be quarantined and disposed of as soon as economically feasible. All housing and equipment should be cleaned and disinfected before repopulation.

Vaccination

Vaccination should be considered in areas where FC is prevalent, but it should not be substituted for good sanitary practice. Commercially produced bacterins and live vaccines are available. Bacterins usually contain whole cells of serotypes 1, 3, and 4 emulsified in an oil adjuvant. Because a bacterin will not provide protection against an FC challenge from a serotype not contained in that bacterin, an autogenous whole-cell bacterin containing a locally isolated strain other than serotypes 1, 3, or 4 may be used (63). The choice of adjuvant for an autogenous vaccine can be water-in-oil emulsion or aluminum hydroxide (9). 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. This response can result in significant declines in egg production. The negative effect on egg production is less with aluminum hydroxide adjuvant whole-cell FC bacterins. It has been well documented that aluminum hydroxide bacterins do not stimulate the immune response as well as water-in-oil bacterins (69, 107). Therefore, if an aluminum hydroxide bacterin is used, revaccination may be required to afford immunity to a flock for an entire laying cycle.

Three live vaccines available for use in the United States are CU, a strain of low virulence; M-9, a mutant of CU with very low virulence; and PM-1, a mutant of CU intermediate in virulence between CU and M-9. Vaccination of chickens and turkeys with these live *P. multocida* vaccines induces protection against heterologous serotype challenge. The use of live FC vaccines stimulates an effective immune response but has the disadvantage of potentially resulting in mortality in the vaccinated birds (11). 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 (123).

When considering the most appropriate vaccination program for FC, the following should be taken into consideration: prevalence of FC in the area, most prevalent serotypes of *P. multocida* in area, age of birds to be vaccinated, and the value of the birds to be vaccinated (i.e., breeder turkeys vs. commercial turkeys or parent chicken breeders vs. grandparent chicken breeders). There have been many successful vaccination protocols for chicken breeders against FC. Bacterins, live vaccines, or both are used, and usually 2 doses are given: the first at 8–10 weeks of age and the second at 18–20 weeks of age. Protection occurs only against serotypes contained in the

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One of the most successful programs for vaccination of both breeder turkeys and commercial meat turkeys is the use of a live vaccine in the drinking water every 4 weeks beginning at 6–8 weeks of age and continuing for the life of the flock. Bacterins also can be used in breeder turkeys. They are vaccinated 2–5 times before the onset of egg production, with the first vaccination beginning at 6–8 weeks.

Riemerella anatipestifer Infection

Jaime A. Ruiz and Tirath S. Sandhu

Introduction

Definition and Synonyms

Riemerella anatipestifer (RA) infection is a contagious disease of domestic ducks, geese, turkeys, and various other domestic and wild birds. It is also known as new duck disease, duck septicemia, anatipestifer syndrome, anatipestifer septicemia, and infectious serositis. In geese, RA infection has been called goose influenza or septicemia anserum exsudativa (49). It occurs as an acute or chronic septicemia characterized by fibrinous pericarditis, perihepatitis, airsacculitis, caseous salpingitis, and meningitis. *Riemerella columbina* (RC), a similar organism to RA, has been isolated from clinically diseased pigeons (69).

Economic Significance

R. anatipestifer infection is a major disease confronting the duck industry worldwide. It accounts for significant economic losses due to high mortality, weight loss, condemnations, downgrading, and salvage. Prevention and control programs consist of diagnosing the infection, vaccinating at-risk flocks, and treating the disease, which all add to the production cost.

Public Health Significance

The disease has no public health significance.

History

R. anatipestifer infection was first described in 1932 in Pekin ducks from 3 farms on Long Island, New York (33). The report referred to a new disease which became known in the area as the "new duck disease". The disease was first observed in 7- to 10-week-old ducks with about 10% mortality and later spread to younger ducklings of about 3 weeks of age. Six years later, the disease was observed in ducks from a commercial farm in Illinois and was reported as "duck septicemia" (23). Dougherty and colleagues (18) gave the disease the designation "infectious serositis" after a comprehensive pathologic study. Leibovitz (48) recommended the term *R. anatipestifer* infection to identify the disease as being caused specifically by *R. anatipestifer* and to differentiate it from other infections with similar pathology. Riemer (66) described a similar disease, septicemia anserum exsudativa, found in geese. The causative agent, *Pasteurella septicaemiae*, is identical to RA based on reported characteristics (35, 81).

Etiology

Classification

In 1932, Hendrickson and Hilbert (33) isolated and characterized the causative bacterium of RA and called it *Pfeifferella anatipestifer*. Bruner and Fabricant (10) studied and compared its characteristics with those of *Brucella, Pasteurella, Moraxella, Actinobacillus*, and *Haemophilus*, concluding that the organism had more in common with *Moraxella sp*. They subsequently suggested the name *Moraxella anatipestifer*. Thereafter, it was listed in the 7th edition of *Bergey's Manual of Determinative Bacteriology* as *Pasteurella anatipestifer* (8). Based on its uncertain taxonomic status, RA was placed as species *incertae sedis* in the 8th (81) and 9th (52) editions of *Bergey's Manual of Systematic Bacteriology*.

Comparing its DNA-base composition, DNA-DNA homology, and cellular fatty-acid profile indicated it should be excluded from the genus *Moraxella* as well as *Pasteurella* (5, 52). Piechulla et al. (63) suggested that RA be transferred to the *Flavobacterium/Cytophaga* group on the basis of its low but significant DNA binding and ability to produce menaquinones and branched-chain fatty acids. Subsequently, Segers et al. (80) reported significant differences between RA and its close genotypic relatives *Flavobacterium* and *Weeksella*. They suggested placing this organism in a separate genus, *Riemerella*, in honor of Riemer (66), who first described the disease "septicemia anserum exsudativa" in geese in 1904. They ultimately named it *Riemerella anatipestifer* on the basis of a DNA-ribosomal RNA hybridization analysis, its protein and fatty acid methylester (FAME) profiles, and its phenotypic characteristics such as lack of pigment production and presence of respiratory quinone, menaquinone 7.

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ISBN 978-0-4709-5899-5

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