Peculiarities of a respiratory mycoplasmosis course of birds

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Abstract. The authors of the article consider an avian disease -the respiratory mycoplasmosis of birds, its etiology, pathogenesis, description of clinical and pathoanatomical and pathohistological features of the course of the disease. In our country, despite the fact that there is a developed system against respiratory mycoplasmosis of birds, based on protection of farms from the introduction of infection from the outside, compliance with veterinary and sanitary rules, zoohygienic and technological standards, as well as measures aimed at timely detection of the disease, the problem of the spread of this disease is quite acute. Currently, the topic of respiratory mycoplasmosis does not lose its relevance precisely because of the high percentage of infection in poultry farms. For example, as a result of research conducted by the ARRIAH over the past 5 years, it was revealed that 218 out of 250 poultry farms in Russia were diagnosed with respiratory mycoplasmosis.

1 Introduction

Avian respiratory mycoplasmosis (Latin: Mycoplasmosisrespiratoria) is a chronic contagious infectious disease, the causative agent of which is Mycoplasma gallisepticum, characterized by respiratory damage, exhaustion and reduced productivity [3, 5, 7, 11, 12, 17].

The causative agent of avian respiratory mycoplamosis is Mycoplasma gallisepticum. The causative agent belongs to Mycoplasmataceae family, Mycoplasma genus, Mycoplasma gallisepticum species. There are many serological types of M. gallisepticum. Mycoplasma gallisepticum has a spherical shape, adapts easily to adverse conditions, persists at low temperatures, is small in size and is a non-spore-forming bacterium.

Among domestic birds, chickens and turkeys are the most susceptible to respiratory mycoplasmosis, but other species of birds of the chicken family – partridges, pheasants, guinea fowls - also may have it. The sources of the pathogen are sick birds. There are 3 ways of spreading the infection: transovarial, aerogenic and contact.

Many factors contribute to the spread of the disease, such as:

- the lack of normal ventilation of the premises, which leads to dustiness, accumulation of the pathogen in the room, which means that the probability of infection increases;

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- the concentration higher than the permissible norms of ammonia, which irritates the respiratory tract, while reducing the protective barriers of the upper respiratory tract, which facilitates the penetration of the pathogen into the body;
- constant dust in the air;
- violation of the air humidity regime;
- the density of the population;
- poor nutrition, lack of essential vitamins, minerals, unbalanced proteins, fats and carbohydrates;
- vaccination of chickens with live vaccines against infectious diseases reduces immunity, during this period chickens are most easily infected with respiratory mycoplasmosis [6].

Infection of birds in natural conditions occurs through the respiratory system and infected eggs. The causative agent enters the body and shows its pathogenic properties. Weakened birds with reduced body resistance are particularly susceptible to the infection.

Mycoplasma gallisepticum enters the upper respiratory tract, but may not cause disease for a long time. However, in a certain stressful situation, the pathogen is activated and begins to actively multiply on the surface of the mucous membranes of the upper respiratory tract. Then it penetrates the epithelial cells and the underlying tissues. An inflammatory process begins in the cells, causing a violation of the tissue function and dystrophic processes. After that Mycoplasma gallisepticum enters the blood vessels and is carried throughout the body to all organs and tissues, causing also inflammatory and dystrophic processes, affecting the blood and lymphatic vessels.

As a result of pathological changes in the tissues of birds the microflora changes: the gram-negative one becomes dominant, where Escherichia coli bacteria predominate. The developing conditionally pathogenic microflora aggravates local pathological processes in the respiratory organs, and when it gets into the blood, it causes severe septicemia and general intoxication of the body. E. coli is layered on mycoplasmic infection, causing fibrinous serositis, severe dystrophic and necrotic changes in the parenchymal organs and the nervous system.

During studying the localization of the pathogen in the body of birds, mycoplasmas were found in a trachea in 62.5% of cases, in brain - in 51%, in a liver - in 35%, in kidneys - in 25%, in lungs and air sacs - in 17%, in a spleen - in 10.5% , in a heart - in 6%, in ovaries and in a spleen - in 5% of cases [1].

Purpose and objectives: To identify the features of the disease course of birds in private farming and to determine the pathomorphological and pathohistological changes in the organs of respiratory mycoplasmosis in fallen birds.

The disease is acute in 10-40%, maximum in 60-70% of birds. The disease can occur covertly and can only be detected during the study of blood serum of birds. The greatest incidence is observed in young animals at the age of 3-7 months and in chickens at the beginning of egg laying, as it was written above. The incubation period for a natural disease is approximately from 4 to 23 days.

The main sign of respiratory mycoplasmosis is a disorder of the function of respiratory organs. There are symptoms such as shortness of breath, cough, tracheal wheezing, a bird breathes with its beak open. Chickens often stretch their necks and yawn. Sick birds try to find a quiet place, huddle in a corner, and when they are scared away, they remain motionless, while a healthy bird runs away.

They often have rhinitis accompanied by the discharge of serous or serous-fibrinous exudate from a nose, this is especially noticeable when pressing on the nasal openings. Around the nasal openings crusts form due to the drying of the exudate. Rarely, a sick bird has conjunctivitis, some swelling in the area of the interdigital space. When infected with

mycoplasma, joints are affected, paws are deformed, wings sag, and movements become abnormal.

The bird becomes sluggish, loses appetite, there is a decrease in body weight, as well as in egg-laying, the plumage is ruffled, the crest is pale, wrinkled.

The disease usually occurs with a normal temperature, sometimes there is a slight increase, but not more than 1°C.

Young animals have more pronounced clinical symptoms. The initial sign of respiratory mycoplasmosis in chickens is the suspension of growth. In this case the cull reaches 75%. The waste of little chickens in the acute course is 10-25%, the waste of adult birds is 4-6%, the waste of embryos is 10-30%. Mortality is most often associated with the pathogenic effect of another bacterial or viral infection that has joined, for example, E. coli, viral infectious bronchitis, laryngotracheitis. This is the danger of respiratory mycoplasmosis. In farms that have problems with mycoplasmosis and infectious laryngotracheitis, there are two peaks of clinical disease: at the age of 35-90 and 130-170 days.

The disease of ducks is asymptomatic, except the oviposition period. During a clinical examination a duck's lower abdominal wall hangs down, and a bird aquired a penguin's gait. Ducklings lag behind in growth and development, during clinical examination we may note mucosal and mucosal-serous rhinitis, swelling of supraorbital sinuses, conjunctivitis.

Geese with this disease have a decrease in egg production during last 2 months of egg laying. The unfertilization of eggs reaches 30%. During the clinical examination of sick geese we may note the damage of a phallus and a cloaca, which become edematous, as weel as the hyperemia of amucous membrane. There is the formation of abscesses, the accumulation of fibrous exudate, from which an unpleasant smell emanates. Sick goslings have shortness of breath, rhinitis, lameness and lag in growth and development [4].

2 Materials and methods

For the study and analysis of pathological changes in bodies of birds with mycoplasmosis a fallen bird (20 heads) with clinical and erased signs of the disease was taken from a private farm. Birds (chicken) were of different ages and sexes. All birds were subjected to autopsy. During the autopsy of a bird the pathological material was taken (pieces of a liver, lungs, a trachea, a heart, kidneys, Fabricium bursa and a spleen) for histological examination. Histological examination included fixation, in a 10% neutral buffered formalin solution, paraffin. The sections were stained with hematoxylin and eosin. Photos of histological preparations were obtained using a Leica microscope.

Pathoanatomic changes. During the external examination of corpses of birds that have fallen from respiratory mycoplasmosis, exhaustion is found, and in cases of young animals - underdevelopment and stunting. Skin of the metatarsals and a beak are usually pale in color. Crusts of dried gray-brown exudate are found around the nostrils. Eye damage is rare, with hyperemia and swelling of the conjunctiva with serous discharge, but this is more associated with the addition of other pathogenic microflora. Some birds have swelling of infraorbital sinuses, usually unilateral.

In a nasal cavity, in sinuses and trachea at the beginning of the disease there is a transparent serous exudate. Later this exudate becomes sticky, with fibrin and caseous masses. These masses can clog a nasal cavity and sinuses. Mucous membrane of a nasal cavity, trachea is hyperemic and edematous with spot hemorrhages. In the chronic course a mucous membrane is pale and unevenly thickened with a weakly granular surface.

Lungs are blood-filled, and when they are pressed, a foamy catarrhal or catarrhal-fibrinous exudate is released. Sometimes small-focal or large-focal pneumonia can be detected. In the affected areas necrosis foci, ranging in size from a poppy seed to the size of a bean, are easily found.

Bronchial mucosa is also hyperemic and swollen. Bronchi connect a lung and the affected air sacs, so a lumen of bronchi contains fibrinous-caseous masses. In places where bronchi come into contact with a lung parenchyma, necrotization of the lung tissue occurs, which can then be encapsulated or sequestered.

At the beginning of the disease the wall of air sacs thickens and becomes cloudy. Serous exudate with a small amount of fibrin is exudated into the cavity, then its exudation increases. With a chronic course the wall of air sacs thickens and becomes completely opaque. The cavity is filled with fibrinous-caseous masses. The outcome of this pathological process can be almost complete overgrowth of the cavity of air sacs with connective tissue. Most often changes occur in the chest air sacs (Fig. 1, Fig. 2).

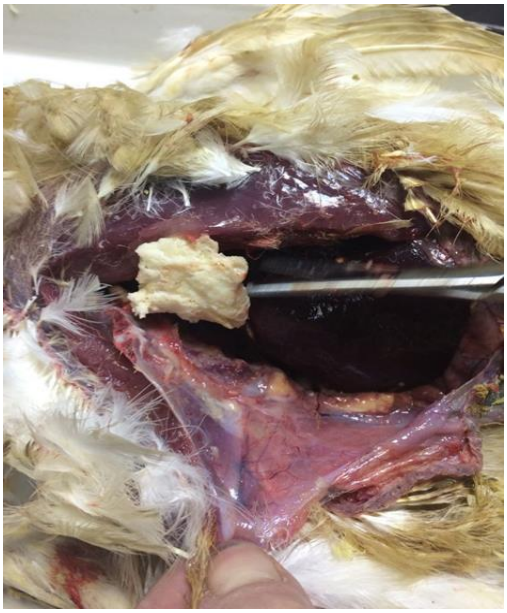

Fig. 1. Autopsy of a bird, detection of fibrin on the air sacs and in the bronchi.

Fig. 2. Autopsy of a bird, detection of fibrin on the air sacs and in the bronchi.

Changes in a heart are more often limited to increased blood filling and a slight increase in the amount of fluid in the pericardial cavity. With a more severe course of the disease adhesive periepicarditis is detected — a heart wall is strongly stretched, filled with fibrinous-caseous masses, sometimes fused with the myocardium. With a chronic course there is mainly fibrinous inflammation of the pleura and peritoneum. Since respiratory mycoplasmosis is quite frequently complicated by colisepticemia, in this case the fallen bird is found to have fibrinous-caseous masses in a heart sac. The consequence of fibrinous epicarditis can be foci of necrosis on the epicardium. In the myocardium itself no changes are detected, but there may be signs of weak granular dystrophy.

Changes in other organs are less pronounced. Congestive hyperemia of a liver and kidneys, as well as dystrophic changes in them, are established more often.

However, in severe cases, some birds show perigepatitis with abundant fibrin deposition on the surface of a liver in the form of gelatinous easily separated masses. A liver itself is enlarged, with a flabby consistency. Kidneys are enlarged, a spleen is often hyperplastic, and there is also inflammation and aplasia of the ovary with a complete absence of maturation of the follicles. In the thoracic cavity an increase in the amount of liquid of a light yellow color is sometimes detected. Some birds may have catarrhal inflammation of the mucous

membranes of the stomach and small intestine. The brain is usually not changed, only sometimes excessively blood-filled and swollen [13].

Thus, the main pathoanatomical signs of avian respiratory mycoplasmosis are:

1. Acute catarrhal-fibrinous rhinitis and tracheitis;
2. Catarrhal or croup-necrotic pneumonia;
3. Fibrinous aeroscout;
4. Acute catarrhal gastroenteritis;
5. Granular dystrophy of a liver, kidneys and myocardium, focal necrosis in a liver.

3 Pathohistological changes

Histological examination reveals congestive vascular hyperemia, mucus hypersecretion, proliferation of lymphoid, plasma cells and histiocytes, hyperplasia and the formation of new lymphatic follicles, mucosal dystrophy and desquamation of secretory and respiratory epithelial cells, sometimes with the formation of erosions.

In the chronic course the cells of the respiratory epithelium proliferate and form a cell layer that resembles a flat epithelium. The mucous glands assume a tubular structure due to the sharp thickening of the mucous membrane and constant pressure on them. Some glandular cells die, and some are replaced by proliferating cells. The mucous membrane thickens unevenly with the formation of outgrowths resembling papillomas.

With fibrinous inflammation in the upper respiratory tract it is found with the deposition of fibrin and caseous masses on the surface of the mucous membrane. Rhinitis or sinusitis are often found.

In the lungs the vessels are sharply filled with blood, there is serous edema of perivascular, interstitial, and less often peribronchial connective tissue, weak cell-proliferative and cell-infiltrative reactions and effusion of serous exudate into the lumen of the bronchi and parabronchi.

With catarrhal inflammation the proliferation of lymphoid, plasma cells and histiocytes is significantly increased. At the same time, the cells of the respiratory epithelium of the bronchial mucosa and parabronchial complexes proliferate and desquamate. Catarrhal exudate accumulates in the lumen of the bronchi, parabronchi and vestibules. In addition, there is an infiltration of the lung tissue by pseudoeosinophilic leukocytes, they are much contained in the exudate.

In the bronchial mucosa the continuous fields of lymphoid, plasma cells and histiocytes are found. The lumen of the bronchi and most of the vestibules are filled with proliferating cells of the respiratory epithelium.

With croup inflammation the vessels are sharply blood-filled, infiltration of serous-fibrinous exudate of perivascular and interstitial connective tissue is observed, as well as exudate effusion into the lumen of the bronchi. The exudate contains numerous lymphoid cells, pseudosinella, desquamating cells of respiratory epithelium and separate red blood cells. In the mucous membrane lymphoid and plasma cells dominate, there are few pseudoadenoviral.

In the inflamed areas the foci of dry necrosis with giant-cell sanitation along the periphery are found. Giant cell granulomas and encapsulated sequesters are found in the areas of granulation tissue development.

In the lungs the lymph follicles hyperplasize. They are formed again in large quantities, in places where they are normally rare and in small quantities, such as interstitial tissue, parabronchial complexes.

In the air sacks the vessels are strongly blood-filled, there are serous and serous-fibrinous edema of the sac wall, proliferation in a large number of histiocytes, lymphoid, plasma cells and fibroblasts, and sometimes infiltration of the wall with pseudoeosinophils, proliferation

and desquamation of respiratory epithelial cells. The cavity of the air sacs is filled with serous or fibrinous exudate. Fibrin, which has got into the cavity, quickly hardens and, together with numerous decaying pseudoeosinophils and other cells, forms caseous masses.

With diphtheria inflammation necrosis of the inner surface of a highly altered and thickened wall develops. On the periphery of the necrotic masses a leukocyte shaft with giant cells is formed. Necrosis and giant cell granulomas may also be of a similar nature in the wall thickness of air sacs [2, 8, 9, 15].

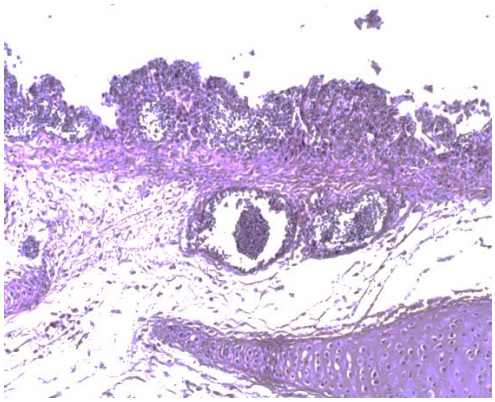

Fig. 3. Trachea. Edema of tracheal tissues and desquamation of epithelial cells. Zoom x400.Stained with hematoxylin and eosin.

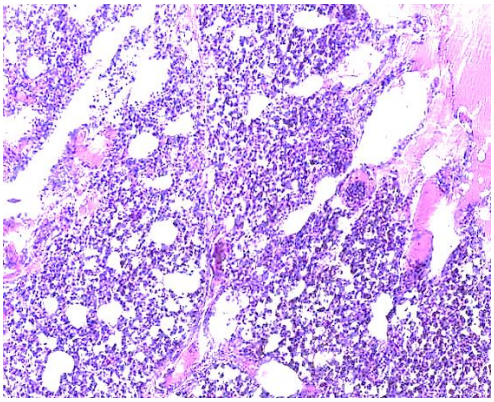

Fig. 4. Lung. Edema of interstitial tissue, alveolitis. Zoom x200. Stained with hematoxilin and eosin.

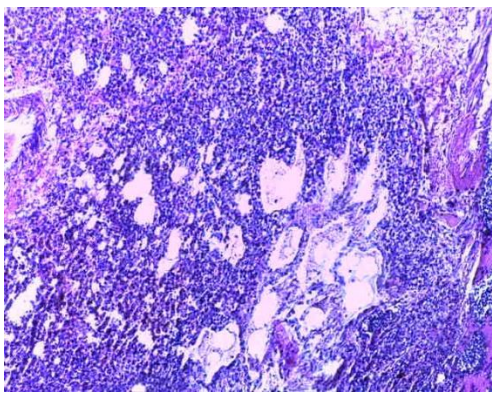

Fig. 5. Lung. Interstitial edema, alveolitis and focal pneumonia. Zoom x200. Stained with hematoxilin and eosin.

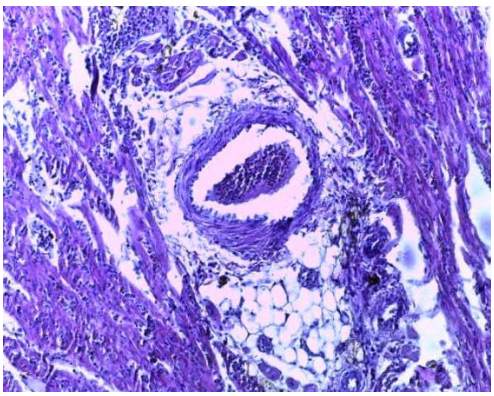

Fig. 6. Heart. Myocarditis and perivascular edema. Zoom x200. Stained with hematoxilin and eosin.

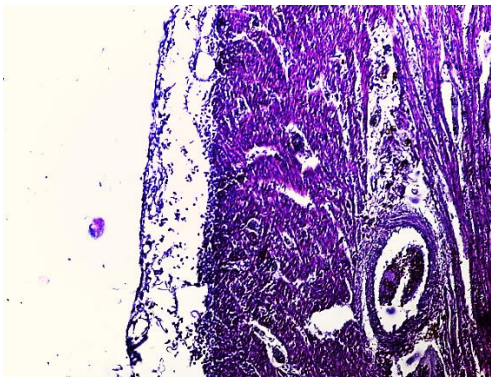

Fig. 7. Parry's disease and epicarditis. Zoom x200. Stained with hematoxilin and eosin.

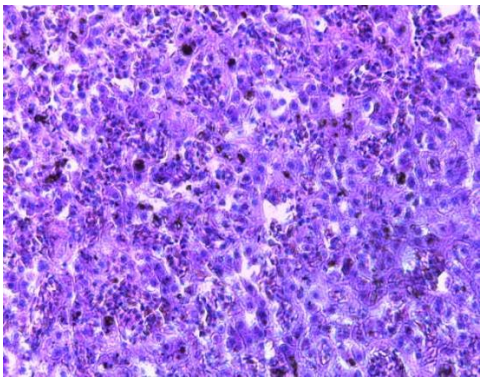

Fig. 8. Liver. Granular dystrophy of hepatocytes and deposition of hemosiderin. Zoom x200. Stained with hematoxilin and eosin.

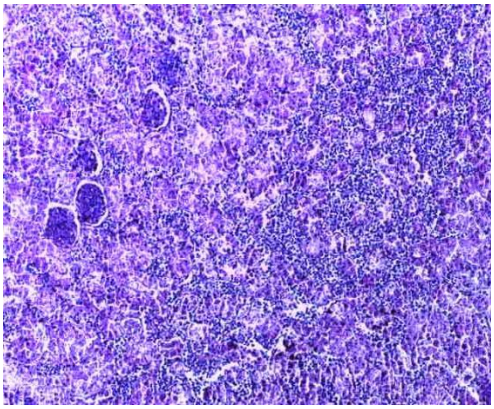

Fig. 9. Kidney. Interstitial nephritis. Zoom x200. Stained with hematoxilin and eosin.

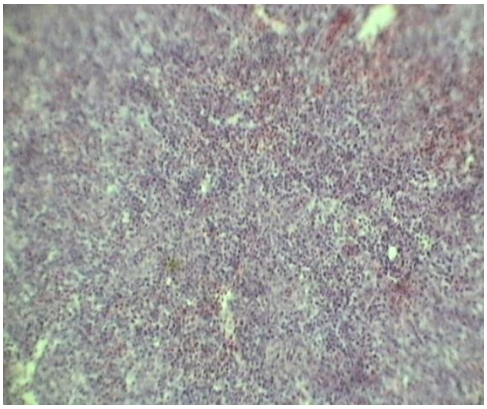

Fig. 10. Spleen. Reduction of lymphoid follicles Zoom x200. Stained with hematoxilin and eosin.

4 Differential diagnosis

To make an accurate diagnosis, it is necessary to conduct a differential diagnosis to exclude diseases that have similar symptoms. Thus, it is necessary to exclude such diseases as xerophthalmia, infectious laryngotracheitis, infectious bronchitis, avian pox, aspergillosis, pasteurellosis, infectious nasal catarrh and colisepticemia.

Xerophthalmia is characterized by the presence of white dense nodules on the mucous membrane of the pharynx and esophagus; keratization and metaplasia of the mucous membrane of the upper respiratory tract, esophagus, bronchi; gouty nephritis. At the same time, there is no croup pneumonia and serous-fibrinous aerosacculitis.

Infectious laryngotracheitis is characterized by the presence of fibrinous plugs with blood clots in the larynx and trachea; hemorrhage in the mucous membrane of the larynx, trachea; the presence of specific intracranial inclusions in the cells of the respiratory epithelium.

In infectious bronchitis there is a sharp edema of the tracheal mucosa and mononuclear cell infiltration; damage to the kidneys and reproductive organs. There are no or very minor changes in the lungs. Aeroscout flows in a less severe form of serous inflammation.

Avian pox differs from respiratory mycoplasmosis by specific exanthema on the skin, diphtheria overlays on the mucous membrane of the oral cavity, esophagus, larynx and less

often trachea. A characteristic feature of smallpox is intraplasmic inclusions in the epithelial cells - the Bollinger corpuscles.

Aspergillosis is characterized by epithelioid granulomas, as well as the presence of fungi in the sections.

An infectious nasal catarrh spreads very quickly, infecting all poultry on the farm. It is characterized by profuse discharge from the nose, sharp swelling of the mucous membrane, especially the shells. And it proceeds without a pronounced cellular reaction, unlike respiratory mycoplasmosis.

Pasteurellosis is characterized by pronounced hemorrhagic diathesis, foci of necrosis in the liver, hemorrhagic gastroenteritis, necrosis of the walls of blood vessels and interstitial lung tissue in places of accumulation of pasteurellosis, capillary thrombosis. There are no changes in the upper respiratory tract, which are characteristic of respiratory mycoplasmosis.

Colisepticemia, as an independent disease, is difficult to diagnose in this case due to the fact that respiratory mycoplasmosis is often complicated by E. Coli. It is necessary to slaughter a sick bird for diagnostic purposes to determine the presence of pure colisepticemia or layered infection. Colisepticemia is characterized by fibrinous pericarditis and epicarditis, aerosacculitis, dystrophic changes in parenchymal organs, a sharp depletion of the lymphoid cells of the fabricium sac, spleen. There are no changes in the nasal cavity, in the larynx and trachea. With respiratory mycoplasmosis mainly the respiratory organs are affected. In contrast to colisepticemia, a lymphofollicular reaction in the respiratory system and weak dystrophic changes in the parenchymal organs are characteristic.

After excluding diseases with similar symptoms and identifying the pathogen Mycoplasma gallisepticum, you can finally make a diagnosis – avian respiratory mycoplasmosis [8, 16].

5 Conclusion

Scientists in the Russian Federation have developed a strategy to combat respiratory mycoplasmosis, which is based on: preventing the introduction of infection in safe farms; on carrying out preventive measures aimed at identifying infected herds, establishing quarantine in them; on strict compliance with veterinary and sanitary rules, zoohygienic and technological standards; on mandatory drug therapy and vaccination in disadvantaged farms. In Russia All-Russian Scientific Research Veterinary Institute of Poultry developed inactivated vaccine against respiratory mycoplasmosis of birds. Its immunological effectiveness is up to 80%, immunity lasts 6-8 months. Russian scientists have developed a plan of veterinary and preventive measures for respiratory mycoplasmosis of chickens, which includes three main stages: prevention of the disease in the parent herd and prevention of transmission of mycoplasmas from laying hens and roosters to offspring; disinfection of hatching eggs from mycoplasmas; prevention and treatment of mycoplasmosis in young animals. Only a comprehensive application of veterinary and preventive measures will effectively combat respiratory mycoplasmosis in chickens.

Respiratory mycoplasmosis of birds causes great losses to the industrial poultry industry in Russia. Monitoring the spread of this disease, carrying out veterinary and preventive measures is a necessary condition for creating a healthy poultry population [10, 14].

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