Review

Mycoplasma infection of ducks and geese

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ABSTRACT Production of ducks and geese in certain parts of the world is very important. Mycoplasma diseases cause significant losses to the duck and goose industry. This review summarizes the epidemiological, clinical, and pathomorphological characteristics of mycoplasma diseases of ducks and geese and the involvement of the various mycoplasma species in their pathogenesis. The role of mycoplasma infections in the development of clinical signs, pathological lesions, and mortality of challenged birds is demonstrated in challenge experiments. Transmission of mycoplasma in the ovary and eggs resulting in the reduction of egg production and an increase of embryo mortality has been shown in challenge experiments as well as in field studies. The susceptibility of many mycoplasma isolates of the most important mycoplasma species of duck and goose origin were tested and showed relatively high average minimum inhibitory concentrations of lincomycin, tilosin, oxytetracycline, chlortetracycline, and enrofloxacin but not for tiamulin. The successful treatment of mycoplasma infections with antibiotics in ducks and geese should be selected based on the minimum inhibitory concentration values against the mycoplasmas isolated from the flock.

Key words: duck, goose, mycoplasma, antibiotic treatment

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INTRODUCTION

The production of ducks and geese can contribute to the improvement of the nutritional standards of the world's human population. Although most poultry meat and eggs come from chickens, significant amounts of meat are produced from ducks and geese in certain parts of the world. Meat and eggs of waterfowl are foods with high nutritional quality, unique, delicious flavor, and are marketed at relatively low prices that even the poor can afford. Waterfowl are also widely used as a source for down and feathers. Duck and goose production accounts for about 7.5% of the total world poultry meat production. China leads in duck and goose meat production by a wide margin and produces 66% of the duck and 93% of the goose meat in the world. Almost 30% of poultry meat in China is from ducks and geese. France, Thailand, Taiwan, the Ukraine, and Vietnam are also major countries in duck production after China (Pingel, 2004). In some countries duck and goose meat production plays an important role in large-scale farms having several thousand birds in one flock. In other countries, especially France, Hungary, and China, geese and ducks were force fed to produce fatty livers (European Union, 1998). Fatty liver production is the process of forced feeding (cramming) geese, normally between 9 and 25 wk of age, for a period of 14 to 21 d. During this period, the weight of the liver will increase from an initial weight of about 80 g to a final weight of between 600 and 1,000 g, which can be sold at a very high value.

This is a review of the available literature on geese/duck mycoplasmosis combined with some of our new research data.

MYCOPLASMA INFECTION OF DUCKS

Studies of mycoplasma infection in ducks goes back to the second part of 1960 when Roberts (1964) reported that in a case with mixed infections of influenza virus and mycoplasma, sinusitis was observed. Subsequently, more data about mycoplasma infections were published (Amin, 1977; Amin and Jordan, 1978; Gaillard-Perrin et al., 1983; Yamada and Matsuo, 1983; El-Ebeedy et al., 1987; Ivanics et al., 1988; Kempf, 1990; Lo et al., 1994; Abdul-Wahab et al., 1996). There are many data demonstrating that mycoplasma species are not always restricted to one animal species. Therefore it was not a surprise that mycoplasma of chicken origin have been cultured from ducks kept for several months on multiple-age chicken farms. In large studies (Bencina et al., 1987a,b) where 6 avian species was tested for mycoplasma, it was noted that *M. anatis* was found only in ducks but not in other avian species. *Mycoplasma anatis* together with other agents was associated with

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a nervous disease of ducks in a large farm (Ivanics et al., 1988). A disease associated with central nervous system signs was observed in 2 duck flocks, each consisting of 6,000 3-wk-old ducklings, a week after their arrival to the receiving farms. The clinical signs included leg weakness, dyspnea, diarrhea, and in many cases disturbance of equilibrium, retrograde movement, torticollis, and twisting of the neck. Pathological and histopathological examinations revealed lympho-histiocytic meningitis and cerebroventriculitis, airsacculitis with moderate amounts of fibrinous exudate, bronchitis associated with formation of lymphoid follicles, focal or interstitial pneumonia, in some cases with fibrinous serositis, interstitial catarrh, and splenomegaly. Large numbers of mycoplasmas were seen by electron microscopy and were isolated from the affected meninges and pericardium. Other diseases were excluded by laboratory examinations. The isolates were identified as *M. anatis.* The pathogenicity and immunogenicity of the strain was investigated in an artificial infection experiment in ducks. During this experiment no clinical signs or deaths occurred, but birds infected into the air sacs or intracranially exhibited pathological lesions similar to, but milder than, those seen in the field outbreak. No immune response was detectable by the serological and cellular tests applied. These data confirm the presence of other factors (probably virus or bacteria) in the development of the diseases of the nervous system. The most frequently isolated mycoplasma species were *M. synoviae* and *M. gallisepticum*, but *M. lipofaciens* and *M. cloacale* (Bradbury and Forrest, 1984; Bradbury et al., 1987) were also detected. In other studies (Bencina et al., 1988a), *M. gallisepticum*, *M. synoviae*, *M. cloacale*, and *M. anatis* were isolated from ducks kept in a yard in close contact with chickens. *M. gallisepticum*, *M. synoviae*, and *M. cloacale* were isolated also from embryonated duck eggs and from infertile duck eggs laid during the first 4 wk of egg production. Infected ducks did not show clinical signs of *M. gallisepticum* or *M. synoviae* infection as in chicken. Detectable *M. gallisepticum* or *M. synoviae* agglutinating antibodies were not present in duck sera. However, they were found in 2 yolks of 10 embryonated eggs. Using the hemagglutination-inhibition (**HI**) tests, yolks from embryonated eggs yielded significantly higher ($P < 0.01$) titers of *M. synoviae* antibodies than duck sera. Geometric mean value of *M. synoviae* HI titers in tested duck sera was 20, whereas those of yolks from embryonated eggs was 333 (Bencina et al., 1988a).

Similar results were obtained a few years later by Tiong (1990) by examination of 263 cases of clinically diseased ducks of all ages for the presence of mycoplasmas. Mycoplasmas and acholeplasmas belonging to more than 8 species were cultured from 68 of them, and comprised 12 *M. anatis*, 1 *M. columbinasale*, 2 *M. gallinaceum*, 2 *M. gallinarum*, 9 *M. synoviae*, 3 unidentified mycoplasma species, and 37 *Acholeplasma laidlawii*. There was a correlation between the severity of infections with *M. anatis* and *P. multocida* (Tiong, 1990).

The pathogenicity of mycoplasma of chicken origin was studied by infecting 1-d-old ducklings with *M. gallisepticum* and *M. anatis*, respectively, by air sac inoculation. Clinical disease was not produced but airsacculitis was produced by both infections. *Mycoplasma gallisepticum* could be recovered readily from respiratory tissue 10 and 30 d after infection, but rapid-slide agglutination (**RSA**) tests were negative. *Mycoplasma anatis* was not reisolated from these tissues or from the cloaca, but a positive RSA test was observed with the serum of a few ducklings (Amin, 1977; Amin and Jordan, 1978). In other studies specific-pathogen-free (**SPF**) ducks 24 and 180 d old were inoculated intranasally with *M. gallisepticum*. No significant gross lesions were found in trachea, lung, or air sacs at 7 or 28 d postinfection (**PI**). *Mycoplasma gallisepticum* was recovered from the infraorbital sinus and trachea but not from the air sacs 7 and 28 d PI. A few ducks responded serologically by developing agglutinating antibody. *Mycoplasma gallisepticum* multiplied in embryonated duck eggs but at lower titers than in embryonated chicken eggs (Yamada and Matsuo, 1983). Similar results were observed by inoculating SPF ducks, 24 and 180 d old, intranasally with the WVU 1853 strain of *M. synoviae*. No significant gross lesions were found in the infraorbital sinuses, tracheas, or air sacs at 7 or 28 d PI, although *M. synoviae* was recovered from all these organs. A few ducks responded serologically by developing agglutinating antibodies. *Mycoplasma synoviae* multiplied in embryonated duck eggs but at lower titers than in embryonated chicken eggs (Yamada and Matsuo, 1983).

In some laboratory studies, it was demonstrated that *M. imitans* proliferated on the epithelial surface and adhered to the respiratory epithelium of duck embryos by means of its terminal tip structure in the same manner as *M. gallisepticum*. These observations endorse the striking phenotypic similarities between *M. imitans* and *M. gallisepticum* and suggest that *M. imitans* may have pathogenic potential in vivo (Abdul-Wahab et al., 1996).

MYCOPLASMA INFECTION OF GEESE

Numerous mycoplasmas were isolated from adult geese in association with reproductive disorders. Mycoplasma infection of geese suffering from salpingitis was first reported by Kosovac and Djurisic (1970); the isolated mycoplasmas were not further identified. The first publication about occurrence of identified members of the order Mycoplasmatales from materials of goose origin appeared in 1975. *Acholeplasma laidlawii* strains were isolated from 2- to 8-d-old goslings with heavy fibrinous airsacculitis, peritonitis, and perihepatitis. Losses reached 30% of the flock by the end of the 8th week of age. *Acholeplasma axanthum* strains were detected in goose embryos that died on d 13 of incubation. A significant loss (up to 60%) of embryos was observed in the flock and some layers died showing fibrinous peritonitis, salpingitis, and abdominal airsacculitis (Stipkovits et al., 1975b).

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Table 1. Pathological lesions in goslings originated from goose breeders challenged with *Mycoplasma anseris*[1]

Hatch	Breeders	Airsacculitis positive/tested	Peritonitis positive/tested	Omphalitis positive/tested
1	Challenged	6/13***	4/13***	5/13***
1	Nonchallenged	0/12	0/12	1/12
2	Challenged	3/4***	3/6***	3/7***
2	Nonchallenged	0/4	0/4	0/4
3	Challenged	5/6***	3/6	3/6***
3	Nonchallenged	0/3	1/3	0/6

[1]Comparison of incidence of airsacculitis, peritonitis, and omphalitis in the challenged and nonchallenged group in each hatch by chi-squared test.

***Difference at the level of $P < 0.001$.

In challenge studies *A. laidlawii* killed only goose embryos, whereas *M. gallinarum* failed to kill them (Kisary and Stipkovits, 1975). Infection of 3-d-old goslings with these mycoplasmas resulted in no mortality, but lesions were produced with *A. axanthum* in 9 of 10 birds. Dual infection of 3-d-old goslings with maternal antibody to goose parvovirus, with *M. gallinarum* or *A. axanthum* and a virulent parvovirus, resulted in some deaths and all birds showed lesions (Stipkovits et al., 1975a, 1976; Kisary et al., 1976a,b).

Similarly to mycoplasma infection of ducks, it was not a surprise that from geese kept on a multiple-age chicken farm, *M. gallisepticum* and *M. synoviae* were isolated (Dupiellet, 1984, 1988; Bencina et al., 1988b,c; Depuillet et al., 1990). Agglutinating antibodies against *M. gallisepticum* and *M. synoviae* were found in the sera of some geese, which were positive also in the hemagglutination-inhibition tests. The isolation of *M. gallisepticum* and *M. synoviae* from several organs of goose embryos indicates that egg transmission occurs. Mycoplasmas of chicken origin were detected in 2-yr-old geese flocks in the Landes region of Southwest France. In one flock of 134 birds, *M. gallisepticum* was isolated from 3 individuals, from a different site in each bird (i.e., esophagus, trachea, and cloaca). *Mycoplasma gallisepticum* was also isolated from the semen of one goose in the other flock of 70 birds (Buntz et al., 1986; Buntz, 1987).

At the end of the 1970s, extensive cloaca and phallus inflammation was observed in ganders. Originally, several bacteria species and gram-negative micrococci were cultured from the surface of mucosal membrane of the phallus and disease was mostly associated with the presence of gram-negative micrococci (Szép et al., 1974; Fadin et al., 1976). These micrococci were considered gonorrhea-like bacteria, similar to human infections. However, it has been proved that these micrococci were mycoplasma, namely the same as *Mycoplasma* sp. 1220 strain (Dobos-Kovács et al., 1985; Stipkovits et al., 1978, 1984a,b,c, 1986a,b). The spread of inflammation of the cloaca and phallus in goose flocks has been intensively investigated in Hungary. In the flocks surveyed, before starting the laying period, about 15 to 20% had mycoplasma in the phallus lymph, but they did not show any clinical signs of cloaca and phallus inflammation. However, after 2 mo of the laying period, 57.5 to 71.8% of the ganders included originally in the flock became clinically diseased. These affected ganders were removed from the flock and a new group of clinically healthy ganders were mixed with the layers and 39.8 to 100% of the replacement ganders became affected during 2 subsequent months. Based on the results of several hundred attempts to isolate mycoplasmas, a relationship has been established between mycoplasma infection and the occurrence of inflammation of the cloaca and phallus in goose flocks. The strains that were isolated were rarely *M. cloacale* or *M. imitans* (Bradbury et al., 1993), more frequently *M. anseris* and very frequently *Mycoplasma* sp. 1220 type of strains (Dobos-Kovács et al., 1985; Varga et al., 1986, 1989, 1990; Bradbury et al., 1988). At the same time in the diseased flocks, antibodies against *Mycoplasma* sp. 1220 strain were very frequently demonstrated. At the peak of egg production, mycoplasmas were isolated

Table 2. Body weights (g; mean ± SD) of goslings originated from goose layers challenged with *Mycoplasma anseris*[1]

Hatch	Challenge	d 1	d 30	d 60	d 75
1	Challenged	85.8 ± 5.4**	—	—	—
1	Nonchallenged	90.4 ± 3.5	—	—	—
2	Challenged	74.5 ± 8.4***	2.321 ± 121***	3.223 ± 189***	4.522 ± 136***
2	Nonchallenged	84.3 ± 5.5	2.498 ± 145	3.687 ± 174	4.9 ± 154
3	Challenged	85.2 ± 8.2*	1.431 ± 156*	3.323 ± 189***	4.2 ± 125***
3	Nonchallenged	91.6 ± 5.4	1.689 ± 143	3.778 ± 198	4.8 ± 134

[1]Comparison of BW of offspring goslings in the challenged and nonchallenged group in each hatch by Student *t*-test.

*, **, and *** = difference at the level of $P < 0.05$, 0.01, and 0.001, respectively.

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Table 3. Goose embryo pathogenicity (no., % in parentheses) of *Mycoplasma* sp. 1200 strain inoculated into yolk sac[1]

Group	Died after 5 d	Died after 10 d	Hatched and died
Inoculated at age 10 d	8	12	7
(40 embryos)	(20.0)***	(30.0)***	(17.5)*
Inoculated at age 20 d	8	10	6
(40 embryos)	(20.0)***	(25.0)***	(15.0)*
Control	0	0	1
			(2.5)

[1]Comparison of embryo pathogenicity in the challenged and nonchallenged groups by Student *t*-test.
* and *** = difference at the level of $P < 0.05$ and 0.001, respectively.

from 92.1% of the phallus lymph samples and 94.6% of the test sera were positive for antibodies to strain 1220 (Stipkovits et al., 1986b,c, 1987b,c).

Similar observations were made in Germany. Cloaca swabs from adult breeding geese of both sexes from 6 separate farms were culturally examined for mycoplasmas. The results revealed the presence of mycoplasmas (*M. cloacale*, *M. anseris*, and *Mycoplasma* sp. 1220) in all the flocks tested. More than one mycoplasma species was simultaneously isolated from 14 out of 37 geese (Behr and Hinz, 1989; Hinz et al., 1994). Mycoplasma infection was diagnosed in Taiwan also (Lin et al., 1995).

Several mycoplasma isolation trials have been performed on infertile goose eggs and goose embryos, which died during incubation, as well as on geese of different ages in France. A total of 43 out of 110 goose eggs proved to be contaminated by mycoplasmas. Upon autopsy of layers, which laid mycoplasma-infected eggs, lesions were observed in the air sacs. Mycoplasmas could be isolated from their air sacs and oviduct. Some of the isolated strains caused 50 to 80% mortality among embryos inoculated into the yolk-sac at 12 d of age. In goslings inoculated at the age of 3 d with these strains, fibrinous airsacculitis and peritonitis were observed. By inoculating laying geese with one of these strains, a decrease in egg production, an increase in early-embryo mortality, and egg transmission of mycoplasmas was demonstrated (Stipkovits et al., 1984c). Dead embryos originated from infected goose layers showed frequently airsacculitis, peritonitis, and omphalitis (Table 1). Body weights of newly hatched embryos from eggs originated from mycoplasma-challenged layers had 15 to 20% lower BW than embryos hatched at the same time but from eggs of nonchallenged layers. At the age of 2 mo, the average of BW of goslings in the former group was 0.4 kg lower than the second group (Table 2).

MYCOPLASMA DISEASES OF DUCKS AND GEESE

Based on literature data, disease of ducks and geese is associated with the presence of several mycoplasma and acholeplasma species. These species definitely have an important role on the immune system of the birds together with other infectious and environmental factors. The role of a single species in the pathogenesis of the disease might be different, but they determine clinical signs and pathological lesions at a certain point. Generally, the disease in both ducks and geese appears as described below.

In young 1- to 2-wk-old ducks and goslings, respiratory signs with lacrimation and a slight nasal discharge can be seen. The growth of the birds is uneven. In this period the mortality rate varies between 5 to 9% in the first 2 wk of life. At necropsy of the dead birds, the accumulation of mucus can be seen in the very hemorrhagic tracheas and accumulation of serous-fibrinous masses can be detected in the thoracic and abdominal air sacs. Pneumonia is always present (Stipkovits et al., 1975a, 1976). If the infection is extensive, severe arthritis develops in about 30% of the 3- to 4-wk-old birds (Stipkovits et al., 1993). The diseased birds show significant retardation in growth. The joints are swollen. Accumulations of yellowish fluid in joints can be observed, from which mycoplasma can be cultured. During this period mortality increases to 12 to 15%.

At a later age, clinical signs of respiratory disease slow down when birds are kept in the open air, and mortality reduces to 1 to 2% month per month. If the duck or geese are used for forced feeding, severe respiratory disease starts to develop in few days. During 3 to 4 wk of forced feeding, 10 to 15% of birds die due to severe airsacculitis and peritonitis. Mycoplasma can be detected in severely affected organs.

When ducks and geese reach the laying period, clinical signs start to develop. Depression, decreased feed consumption (up to 50%), and the appearance of clinical signs of respiratory disease can be observed. Mortality increases monthly up to 5% during the laying period. Egg production is delayed for about 3 wk. Production of eggs with abnormal shells can reach 25%. Egg production remains about 25 to 30% lower in the laying

Table 4. Infertility (%) of eggs in goose flocks infected with *Mycoplasma* sp. 1220 mycoplasma

Date	Flock 1/1	Flock 1/2	Flock 2/1	Flock 2/2
January	—	—	—	—
February	22.2	17.8	14.4	15.7
March	31.5	58.8	78.3	6.1
April	84.1	77.8	31.5	30.8
May	98.8	96.9	71.4	71.4

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Table 5. Occurrence of phallus inflammation of ganders in flocks infected with *Mycoplasma* sp. 1220 mycoplasma

Item	Spring season	Autumn season
Initial no. of ganders	320	418
No. of replaced ganders	64	127
No. of diseased ganders (%)	184 (57.5)	300 (71.8)
No. of disease ganders in replaced group (%)	30 (46.9)	51 (39.8)
No. of ganders that died	16	20

period compared with the control flock (Stipkovits et al., 1984a,c). During the first 2 mo of egg production, the rate of egg infertility is about 8 to 10% and the rate of embryo mortality is 5 to 10%. Embryos die after 3 to 4 wk of incubation. Later, the embryo mortality reaches as high as 60 to 80% on the 3rd or 4th month of the laying period. At this time most embryos die during the first 10 to 15 d of incubation. From 60 to 70% of the dead embryos, mycoplasmas can be isolated (Stipkovits et al., 1986c). The pathogenicity of mycoplasmas in goose embryos was demonstrated by the artificial challenge of embryos (Tables 3 and 4) (Kisary et al., 1976a,b; Glávits et al., 1987; Stipkovits et al., 1987a).

In ducks, but mostly in goose flocks, phallus inflammation of males starts to develop after 3 wk of the laying period. The number of affected males increases rapidly, reaching 50% at the second month of laying period. After another 2 mo it reached 80 to 100%. If affected males were replaced in the flock, the newcomers became affected by 50% also similarly to the first 1- to 2-mo period. Mycoplasmas could be isolated in high proportion from the phallus lymph of affected males. During the first month of laying period, the isolation rate of mycoplasma from phallus lymph is about 15 to 20%, then every month the mycoplasma isolation rate gradually increased, reaching 85 to 90% at the 4th month of the laying period (Table 5; Stipkovits et al., 1986b,c, 1987b; Stipkovits and Kempf, 1996).

In the affected dead or killed breeders, vaginitis (Dobos-Kovács et al., 1987, 2009), serous-fibrinous airsacculitis, granulocytic peritonitis and salpingitis, infiltration of the lamina propria in the uterus, and heterophil granulocytes in the isthmus and magnum of the oviduct could be observed. Mycoplasmas could be isolated from the air sac, liver, spleen liver, ovary, magnum, and peritoneum of affected layers (Varga et al., 1990; Dobos-Kovács et al., 2009). The affected males had similar airsacculitis and peritonitis as the layers and mycoplasmas could be cultured from air sacs, liver, and spleen of affected males. Beside these lesions, fibrinous inflammations of the phallus and accessory organs could be recorded. Mycoplasmas could be isolated from accessory the organs of the phallus.

Mycoplasmas cultured from ducks are a mixture of mycoplasma species, such as *M. anatis*, *M. synoviae*, *M. gallisepticum*, and various acholeplasmas, whereas isolates from geese consist mostly of *M. anseris*, *M. cloacale*, and *A. axanthum* but most frequently, *Mycoplasma* sp. strain 1220, which according to detailed biochemical and serological examinations, is expected to represent a new avian species within the genus *Mycoplasma* (Dobos-Kovacs et al., 2009; Volokhov et al., 2010).

Four sets of challenge experiments were performed with various *Mycoplasma* sp. 1220 strains on 1-yr-old goose layers, free of mycoplasma infection. The challenge of the layers was performed by inoculating 1 mL of a culture inoculum intravaginally through a catheter of 2-mm diameter. The concentration of the mycoplasma cultures varied between 1×10^5 and 2.9×10^8 cfu/mL. Ganders kept with infected layers were not challenged, so they served as in-contact control birds. At the same time we placed groups of layers with ganders without challenge as control, which were kept isolated from the challenged geese. Experimental geese in the challenged groups were kept for 4- to 39-d postchallenge and afterward they were euthanized and examined for the presence of pathological lesions and mycoplasma in organs (Table 6).

All challenged goose layers and ganders kept with them remained clinically healthy, although depression was noticed in layers. Egg production decreased or stopped. In geese necropsied at the end of the experiment, accumulation of serous-fibrinous exudates was observed in the uterus, and sometimes in the magnum and infundibulum. The mucosal membrane of the oviduct was pink in color and edematous. Development of follicles in the ovary stopped. In some cases exudate

Table 6. Salpingitis in goose layers caused by *Mycoplasma* sp. 1220 inoculated in cloaca

Day of examination	Number of challenged layers	Number of layers with salpingitis	Reisolation of mycoplasmas
d 10	8	2	3
d 20	7	5	6
d 30	10	6	6
d 39	7	4	4
Total (%)	32 (100)	17 (53.1)	19 (59.4)

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Table 7. Average (Av.) minimum inhibitory concentration (MIC) values (μg/mL) of mycoplasma (*M.*) strains cultured from geese and ducks[1]

	M. anseris (86)[2,3]			*M.* sp. 1220 (157)			*M. cloacale* (19)			*M. anantis* (65)		
Item	MIC50%	MIC90%	Av. MIC	MIC50%	MIC90%	Av. MIC	MIC50%	MIC90%	Av. MIC	MIC50%	MIC90%	Av. MIC
En	2.00	4.00	2.97	2.00	4.00	3.25	1.00	4.00	2.12	1.00	4.00	1.87
Ty	2.00	8.00	3.98	2.00	8.00	4.27	2.00	8.00	4.79	2.00	4.0	3.56
CTC	2.00	8.00	3.97	1.00	4.00	2.00	4.00	3.67	3.22	2.00	4.00	3.51
OTC	4.00	8.00	4.56	2.00	8.00	3.90	4.00	8.00	4.12	2.00	8.00	4.12
DOX	2.00	4.00	2.67	1.00	4.00	2.50	4.00	3.66	3.11	2.00	4.00	3.02
Ti	0.125	1.00	0.89	0.06	0.25	0.125	1.00	1.32	0.91	0.25	1.00	0.34
Li	2.00	8.00	3.97	2.00	4.00	3.90	8.00	4.87	3.96	2.00	4.00	3.67

[1]En = enrofloxacin, Ty = tylosin, CTC = chlortetracycline, OTC = oxytetracycline, DOX = doxycycline, Ti = tiamulin, Li = lincomycin.

[2]Number of tested strains in parentheses.

[3]MIC50% and MIC90% = at given concentration of antibiotics, 50 or 90% of tested strains are inhibited, respectively.

from the oviduct flowed through the infundibulum and into the peritoneal cavity. In such cases exudates was observed between the follicles in the ovary. In addition, exudates in the peritoneum caused serous-fibrinous-purulent peritonitis of varying severity. Sometimes degeneration of follicles was observed. The incidence of gross lesions in the oviduct (salpingitis) of 32 challenged layers was 53.1%.

By histological examination, serous exudate and heterophil granulocyte infiltration of lamina propria was recorded in the uterine parts of the oviduct. Presence of heterophil granulocytes was detected mostly in the epithelial layer. Sometimes infiltration of the mucosal membrane by granulocytes was also observed in the isthmus and magnum. In the folds of the oviduct mucosal membrane, purulent exudate from serum and heterophil granulocytes was noticed. Diffuse heterophil granulocyte and lymphocyte infiltration was recorded around the portal blood vessels of the liver in all infected geese.

No bacteria were detected on blood or Drigalski agar from samples of liver, spleen, oviduct, and from peritoneal exudate. No chlamydias were detected in touch preparations made from exudates in peritoneum or oviduct stained by Stamp or Giemsa. From different organs of 32 challenged birds, mycoplasmas could be cultured and 59.4% of oviduct samples proved to be positive. Mycoplasma was cultured from most of the inner organs of the geese. Mycoplasmas could be reisolated also from all 9 unchallenged contact control ganders. All isolates proved to be biochemically and serologically identical with the mycoplasma strain *M.* sp. 1220. In contrast, no mycoplasmas could be detected from the 14 uninfected control layers and 4 control ganders.

Testing of antibiotic sensitivity of a relatively high number of the strains of mycoplasma species isolated during last 10 yr from geese and ducks, considered as strains associated with the diseases of geese and ducks, revealed that most antimicrobial drugs, such as tylosin, enrofloxacin, lincomycin, oxytetracycline, and chlortetracycline, being used in practice for treatment of mycoplasma diseases of chickens or turkeys (Jordan et al., 1989) had high and variable average minimum inhibitory concentration (**MIC**) values (Table 7). Therefore, these antibiotics are unlikely to reach such MIC values that will be enough to prevent mycoplasma propagation and cure the mycoplasma disease of ducks and geese. In comparison, tiamulin showed reasonable average MIC values. Using tiamulin for the treatment of birds (100 mg/kg of feed for 10 d) during the first month of life resulted in a significantly reduced number of clinically diseased birds and mortality to 1 to 2%.

During the laying period antibiotic treatment of a flock (2,600 breeders and 650 ganders) in feed every month for 10 d, egg production was increased by 12.9 to 18.5 eggs/breeder and embryo mortality was reduced by 12.2 to 12.8%, infertility rate of eggs by 11.1 to 14.0% in comparison with those parameters observed in the untreated group. At the same time, the number of males showing phallus inflammation and mortality of adult birds had decreased by 36.1% (Tables 8 and 9).

In conclusion, it can be stated that mycoplasma diseases of ducks and geese are very important economically. Mycoplasma infection of the birds can aggravate

Table 8. Efficacy of treatment of goose flocks with antibiotic on occurrence of phallus inflammation of ganders[1]

Item	Flock no. 1 (treated, %)	Flock no. 2 (nontreated, %)
October: no. of diseased ganders	24 (5.7)*	98 (14.6)
November: no. of diseased ganders	27 (6.7)*	122 (18.1)
December: no. of diseased ganders	38 (9.0)*	131 (19.5)
Total no. of diseased ganders	148 (34.9)***	478 (71.0)

[1]Remarks: Comparison of number of diseased ganders in the treated and nontreated groups by chi-squared test.

* and *** = difference at the level of $P < 0.05$ and 0.001, respectively.

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Table 9. Effect of antibiotic treatment of geese layers on infertility, hatchability, and production of eggs[1]

	% of infertility of eggs		% of hatchability		Number of eggs/breeder	
Date	Treated	Nontreated	Treated	Nontreated	Treated	Nontreated
April	38.6	38.7	54.5	53.9	33.3	25.5
May	24.0**	35.7	68.4**	55.6	22.5*	12.0
June	34.0**	48.0	560**	43.8	25.8**	12.9

[1]Comparison of percentage of infertility of eggs and hatchability as well as number of eggs per breeder in the treated and nontreated groups by chi-squared test.

* and ** = difference at the level of $P < 0.05$ and 0.01, respectively.

diseases associated with other agents such as viruses or pasteurellas. Economic losses due to mycoplasma disease can be significantly reduced by treatment of birds with affective antibiotics (Czifra et al., 1986; Dobos-Kovács et al., 1988a,b; Stipkovits et al., 1986a, 1987b), which should be selected based on previous antibiotic sensitivity testing of the mycoplasma strain present in the affected flock.

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