

## DATA ON THE EPIDEMIOLOGY AND PATHOLOGY OF ANATIPESTIFER DISEASE IN HUNGARY (2010–2014)

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Anatipestifer disease is a contagious disease caused by *Riemerella anatipestifer*, affecting primarily ducks, geese and turkeys, and characterised by listlessness, diarrhoea, sneezing, nasal discharge, and nervous signs. Sporadically, it occurs in a wide range of other domesticated and wild birds as well. The incidence and characteristics of the disease seen in the three main host species are summarised based on birds submitted for routine laboratory investigation in Hungary over the period 2010–2014. The infection was diagnosed in a higher percentage in geese (9.9%) and ducks (7.5%). It occurred in 5-day-old to 17-week-old geese and 3- to 6.5-week-old ducks, respectively. The pathological lesions were comparable in these two species: enlarged spleen, serofibrinous pericarditis, perihepatitis, airsacculitis, catarrhal enteritis, subcutaneous oedema and hyperaemia over the cranium, mucopurulent exudate in the nasal cavity and occasionally pneumonia, conjunctivitis, purulent arthritis and caseous salpingitis. In some cases, *R. anatipestifer* produced only secondary lesions, which complicated other diseases such as circovirus infection, mycotoxicosis, mycoplasmosis, or Derzsy's disease. In turkeys, the disease occurred rarely (0.5%) and at an older age (12 to 19 weeks). The lesions most frequently seen were purulent osteomyelitis of the cranium and seropurulent meningitis. Purulent osteomyelitis in the cranium caused by *R. anatipestifer* infection had not been reported in turkeys previously. To various extents, other local lesions such as serofibrinous pericarditis, airsacculitis, arthritis, and in one case septicaemia were also observed. The high incidence of the disease in waterfowl underlines the importance of appropriate treatment and prevention that should be based on accurate diagnosis and antimicrobial susceptibility testing, proper biosecurity and vaccination with regard to the serotype(s) present on the farm.

**Key words:** Anatipestifer disease, *Riemerella anatipestifer*, goose, duck, turkey

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Anatipestifer disease was first described in geese as ‘septicaemia anserum exsudativa’ in Germany by Riemer (1904). In ducks, the disease was first diagnosed by Hendrickson and Hilbert (1932). They also isolated and characterised the causative bacterium, and called it *Pfeifferella anatipestifer*. It is a Gram-negative, rod-shaped, non-motile, non-spore-forming bacterium that had been reclassified several times (*Moraxella*, *Pasteurella*) until finally it was moved to the family Flavobacteriaceae and, in honour of Riemer, ultimately renamed as *Riemerella anatipestifer* (Segers et al., 1993). In Hungary, anatipestifer disease was first reported in ducks (Bitay et al., 1979), and almost twenty years later in geese as well (Ivanics et al., 1996).

*Riemerella anatipestifer* infection occurs all over the world where intensive duck or goose production is practised. It causes severe economic losses through high mortality rate (estimated to vary from 10% to 75%), weight loss, the costs of antibiotic treatment, prevention, and condemnation at the slaughterhouse (Ruiz and Sandhu, 2013).

Anatipestifer disease is observed most frequently in young (2- to 8-week-old) ducklings and goslings, characterised by listlessness, diarrhoea, upper respiratory signs and nervous signs such as head tremor, torticollis and lameness (Pickrell, 1966; Jortner et al., 1969; Pierce and Vorhies, 1973; Bisgaard et al., 2008; Fulton and Rimler, 2010). Birds are infected via the respiratory route or through skin wounds (Ruiz and Sandhu, 2013). Natural outbreaks have been described in 6- to 15-week-old turkeys as well (Zehr and Ostendorf, 1970; Smith et al., 1987; Frommer et al., 1990). Furthermore, sporadic cases of *R. anatipestifer* infections have also been reported in chickens, guinea fowl, pheasants, gulls, quails, budgerigars, and wild waterfowl (Bruner et al., 1970; Karstad et al., 1970; Rosenfeld, 1973; Pascucci et al., 1989; Hinz et al., 1998). Adverse environmental conditions (hot or cold weather, inadequate ventilation, stress caused by overcrowding) or concomitant diseases can predispose birds to disease outbreaks (Ruiz and Sandhu, 2013).

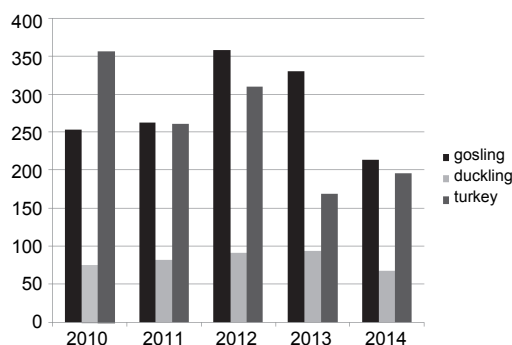
To date, no data have been published on the occurrence and characteristics of anatipestifer disease in different host species in Hungary. The aim of this study was to summarise the incidence and the lesions of anatipestifer disease observed in geese, ducks and turkeys in Hungary in the period of 2010–2014 based on the findings obtained in birds submitted for routine laboratory investigation.

## Materials and methods

### Cases

Birds from goose, duck and turkey flocks submitted for routine laboratory investigation to the Veterinary Diagnostic Directorate (Budapest, Kaposvár, Debrecen, Hungary) in the period 2010–2014 were included in the study. In total, the

case number was 1418 for goslings, 411 for ducklings, and 1292 for turkeys (Fig. 1). Each case represented 4 to 10 birds that were submitted in one group from the same flock. The age of the birds ranged from 5 days up to 36 weeks (geese), up to 22 weeks (ducks), and up to 28 weeks (turkeys). Cases diagnosed with anapitestifer disease were the subject of this study. The goose cases originated from 84 flocks, and from 19 flocks more than one bird was submitted. The duck cases originated from 19 flocks, and from three flocks birds were received on several occasions. The turkey cases originated from four flocks, and samples from two flocks were obtained several times. The presence of other diseases was examined according to routine procedures; however, the details are not shown here. Only the diagnoses were used for calculating the incidence of concomitant infections in cases positive for anapitestifer disease.



\*The age of the birds ranged from 5 days to 36 weeks (geese), to 22 weeks (ducks) and to 28 weeks (turkeys)

Fig. 1. Number of gosling, duckling and turkey cases\* submitted for routine laboratory investigation to the National Food Chain Safety Office – Veterinary Diagnostic Directorate (NÉBIH-ÁDI, Budapest, Debrecen, Kaposvár) in the years 2010–2014

### Postmortem examination

After detailed gross pathological examination of the birds, representative tissue samples were collected for histopathological examination in each case. Specimens were generally taken from the brain and liver, occasionally from the heart, bursa of Fabricius, lung, spleen, kidney, spine, and cranium. Tissue samples were fixed in 10% neutral-buffered formalin, embedded in paraffin, sectioned at 4  $\mu$ m and stained with haematoxylin and eosin.

### Isolation of *R. anapitestifer*

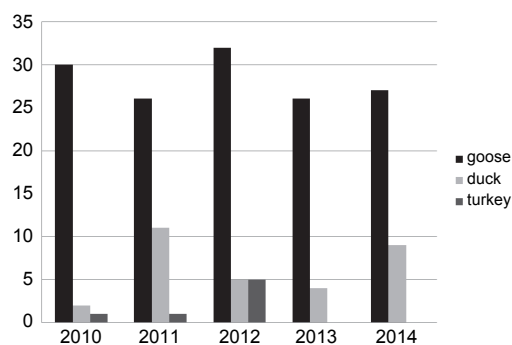
Various tissues, including the liver, pericardium, brain, lung, oviduct, joint and occasionally the trachea, conjunctiva, cranium, air sac and peritoneum were sampled, and all samples were cultured on Columbia agar plates (Biolab, Buda-

pest, Hungary) supplemented with 5% sheep blood at 37 °C for 24 h in an atmosphere containing 5% CO<sub>2</sub>. *Riemerella anatipestifer* suspect strains were first identified by colony morphology, oxidase reaction and Gram staining, and their identity was confirmed with a species-specific polymerase chain reaction (PCR) (Kardos et al., 2007).

## Results

### *Incidence and epidemiology*

Anatipestifer disease was diagnosed in 141 cases (9.9%) in geese, and in 31 cases (7.5%) in ducks. The disease was rather rare among turkeys. It was diagnosed only in seven cases (0.5%) (Fig. 2).



\*Each case contained 4–10 birds submitted together for routine laboratory investigation

Fig. 2. Number of cases\* of anatipestifer disease diagnosed in geese, ducks and turkeys at the National Food Chain Safety Office – Veterinary Diagnostic Directorate (NÉBIH-ÁDI, Budapest, Debrecen, Kaposvár) in the years 2010–2014

In geese, the incidence of anatipestifer disease was comparable to that of Derzsy's disease, circovirus infection, haemorrhagic nephritis-enteritis (polyomavirus infection), colibacillosis, streptococcosis, and mycotoxicosis (indicated by the presence of tubulonephrosis and liver dystrophy). The case number of fowl cholera and paratyphoid infection was about half of the anatipestifer disease cases (Table 1).

In ducks, the incidence of anatipestifer disease, showing some variation in different years, was similar to that of fowl cholera, circovirus infection, colibacillosis, and mycotoxicosis (Table 2).

*Riemerella anatipestifer* infections were diagnosed most frequently in young, 1- to 8-week-old or more often in 2- to 5-week-old goslings, 3- to 6-week-old ducklings and 12- to 19-week-old turkeys. The chronic form of anatipestifer disease accompanied by severe weight loss was rarely observed.

**Table 1**

Incidence of diseases most commonly diagnosed in geese at the National Food Chain Safety Office – Veterinary Diagnostic Directorate (NÉBIH-ÁDI, Budapest, Debrecen, Kaposvár) in the years 2010–2014

Diseases	Years				
	2010	2011	2012	2013	2014
Derzsy's disease	39*	44	64	53	29
Streptococcosis	20	25	43	40	37
Colibacillosis	19	16	22	33	46
Pneumomycosis	12	34	25	33	14
Circovirus infection	16	24	34	41	19
<b>Anatipestifer disease</b>	<b>30</b>	<b>26</b>	<b>32</b>	<b>26</b>	<b>27</b>
Polyomavirus infection	10	13	38	42	7

\*The number of positive cases

**Table 2**

Incidence of diseases most commonly diagnosed in ducks at the National Food Chain Safety Office – Veterinary Diagnostic Directorate (NÉBIH-ÁDI, Budapest, Debrecen, Kaposvár) in the years 2010–2014

Diseases	Years				
	2010	2011	2012	2013	2014
Circovirus infection	8*	13	14	28	9
Mycotoxycosis	10	9	8	6	11
Fowl cholera	10	9	7	9	5
Colibacillosis	5	10	9	7	9
Pneumomycosis	2	3	1	2	7
<b>Anatipestifer disease</b>	<b>2</b>	<b>11</b>	<b>5</b>	<b>4</b>	<b>9</b>

\*The number of positive cases

In geese, anatipestifer disease occurred at the age of 5 days to 17 weeks. In some cases, concurrent diseases were also observed in addition to anatipestifer disease in some of the birds belonging to the case in question. In 19% of the cases circovirus infection, while in 5% of the cases Derzsy's disease was the primary diagnosis. In 21% of the cases some of the birds died of colibacillosis and in 12% of the cases of pneumomycosis, while in 8% of the cases each paratyphoid and streptococcosis occurred as concomitant diseases; there was only one case in which the majority of the birds died of anatipestifer disease.

In ducks, anatipestifer disease occurred at the age of 3 to 6.5 weeks. In some cases, concurrent diseases were diagnosed as well. In 46%, 13% and 8% of the cases circovirus infection, mycotoxycosis and mycoplasmosis was the primary diagnosis, respectively. In 38% of the cases some birds died of colibacillosis,

but the majority of them succumbed to anatipestifer disease. Paratyphoid, staphylococcosis, pasteurellosis, amyloidosis, and visceral gout also occurred in a few birds within some cases.

In turkeys, anatipestifer disease occurred at the age of 12–19 weeks. *Riemerella anatipestifer* could be isolated in pure culture from local lesions of the cranium, meninges and joints. In one case, besides the afore-mentioned localisations, *R. anatipestifer* pericarditis and peritonitis were also seen. In another case, *R. anatipestifer* septicaemia was also observed. In 72% of the cases, local *R. anatipestifer* infection was accompanied by concurrent *E. coli* septicaemia. In some of these cases, *R. anatipestifer* could be isolated together with *E. coli* from certain locations such as the pericardium, air sacs and airways. Interestingly, although most of these cases occurred at different time points and at various geographic locations, all of the affected flocks belonged to the same owner.

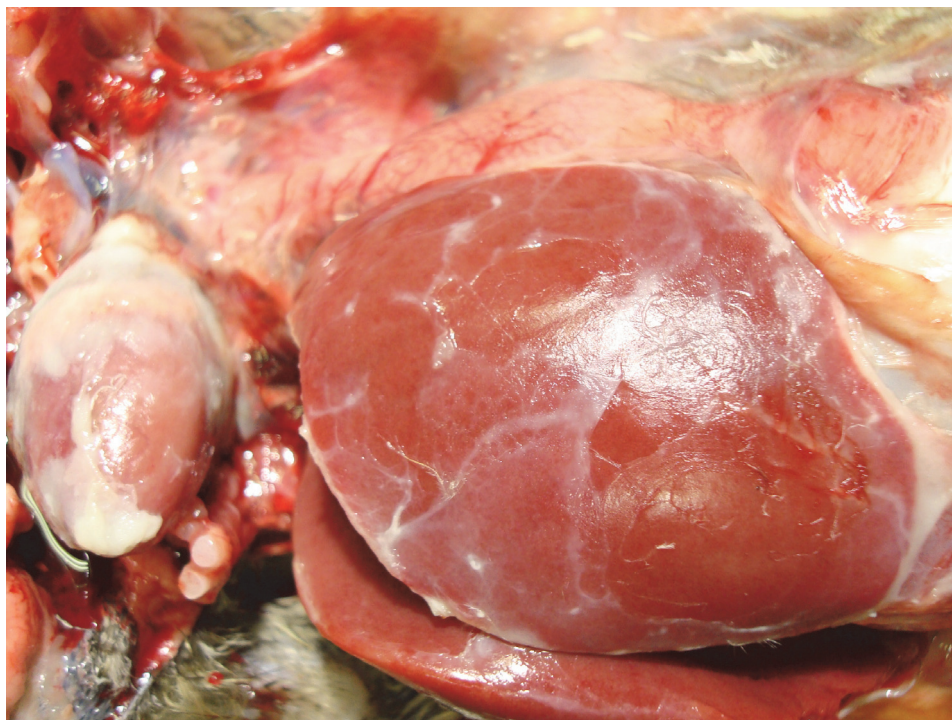


Fig. 3. Fibrinous exudate on the surface of the liver and on the pericardium in a goose

#### *Pathological lesions*

In geese and ducks, serofibrinous pericarditis, perihepatitis and airsaccu-  
litis were the most obvious gross lesions of *R. anatipestifer* infection. Fibrinous  
exudate accumulated in the pericardial sac, and on the surface of the liver (Fig. 3).

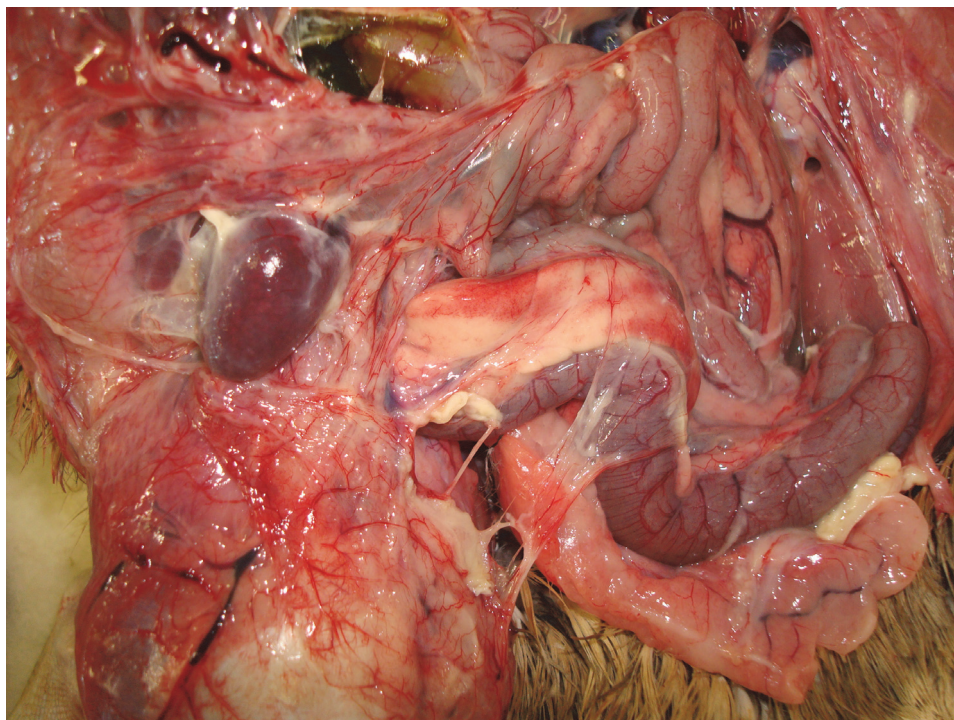
The spleen was generally enlarged (Fig. 4), occasionally mottled, and congestion occurred in the liver. Mucopurulent exudate was seen in the nasal cavity and sinuses, and sporadic catarrhal pneumonia was also noticed. Acute catarrhal enteritis was seen in the small intestine. Usually some birds showed oedema and hyperaemia of the subcutaneous connective tissue over the cranium within each case (Fig. 5). Occasionally, inflammation of the joints or caseous salpingitis also occurred (Fig. 6). Serofibrinous arthritis occurred in 6% of the goose cases, caseous exudate was observed in the oviduct in 5% of the goose cases and in 13% of the duck cases. Conjunctivitis was observed in 4% of the duck cases.

In most of the cases, *R. anatipestifer* infections caused only local lesions in turkeys. The most prominent lesion was cranial suppurative osteomyelitis and consequent seropurulent leptomeningitis. Rarely, seropurulent arthritis and pericarditis associated with *R. anatipestifer* infection were also observed. In a single case, *R. anatipestifer* could be isolated not only from local lesions but also from the heart blood (septicaemia). In several cases, although *E. coli* septicaemia/bacteraemia could be determined from local lesions, *R. anatipestifer* was grown in pure culture.

#### *Histopathological lesions*

In geese, ducks and in two turkey cases, serofibrinous inflammation was observed on the epicardium (Fig. 7), on the Glisson's capsule of the liver, on the pleura, and in the wall of the air sacs. The exudate contained lymphocytes, histiocytes and heterophil granulocytes. Serous hepatitis was observed, the sinusoids of the liver were distended, and white blood cells accumulated between the liver plates. In some parenchymal cells of the liver hydropic degeneration was observed. Mild periportal lympho-histiocytic infiltration was seen as well. Occasionally in the lower respiratory tracts, in the parabronchi of the lung serous or catarrhal exudate was observed. Lymphoid depletion occurred in the medulla of the follicles in the bursa of Fabricius. Serofibrinous meningitis and ventriculitis were seen in the brain. The leptomeninges of the brain and spine were diffusely widened, serofibrinous inflammation and lymphocytic, histiocytic and heterophil granulocytic infiltration were seen (Fig. 8). Similar cellular infiltration occurred in the ventricles of brain, in the central canal of the spine and under the ependyma as well. In the majority of the turkey cases signs of purulent osteomyelitis with heterophil granulocytic infiltration were observed in the cranium (Fig. 9), and this lesion was regularly associated with seropurulent meningitis. The leptomeninx of the brain was widened, seropurulent inflammation was seen with mostly heterophil granulocytic and also lymphocytic and histiocytic infiltration.



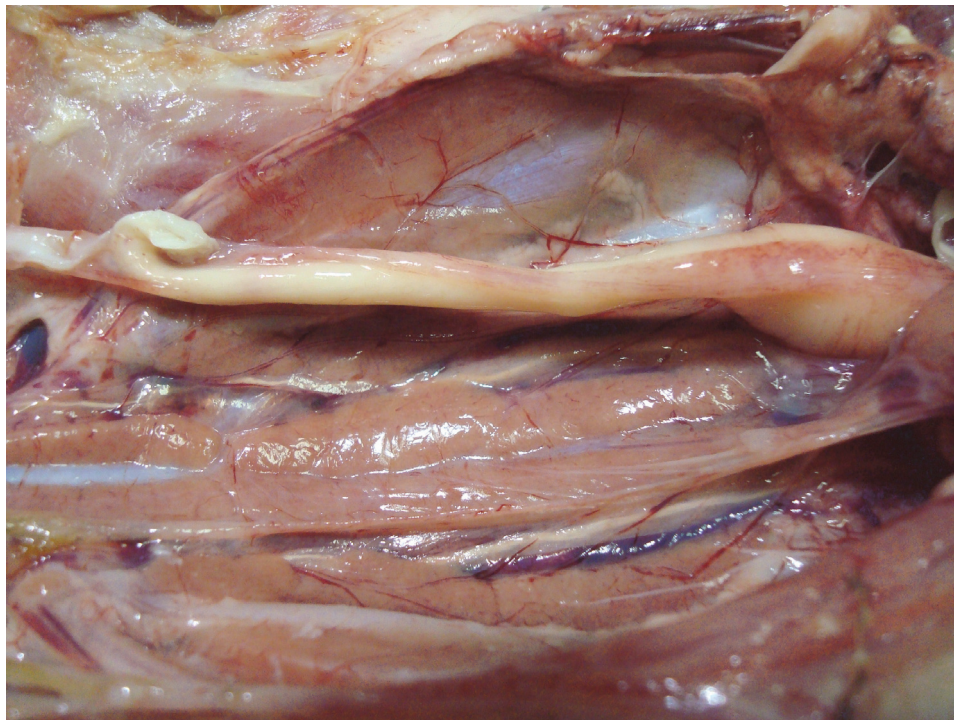


*Fig. 4.* Enlargement of the spleen and serofibrinous inflammation in the body cavity of a goose

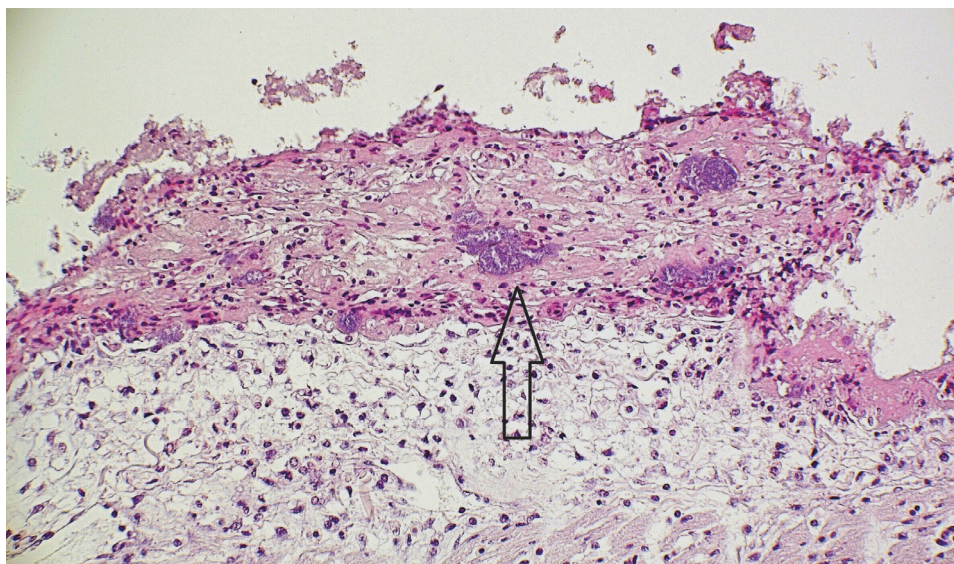


*Fig. 5.* Oedema and hyperaemia of the subcutaneous connective tissue over the cranium in a goose



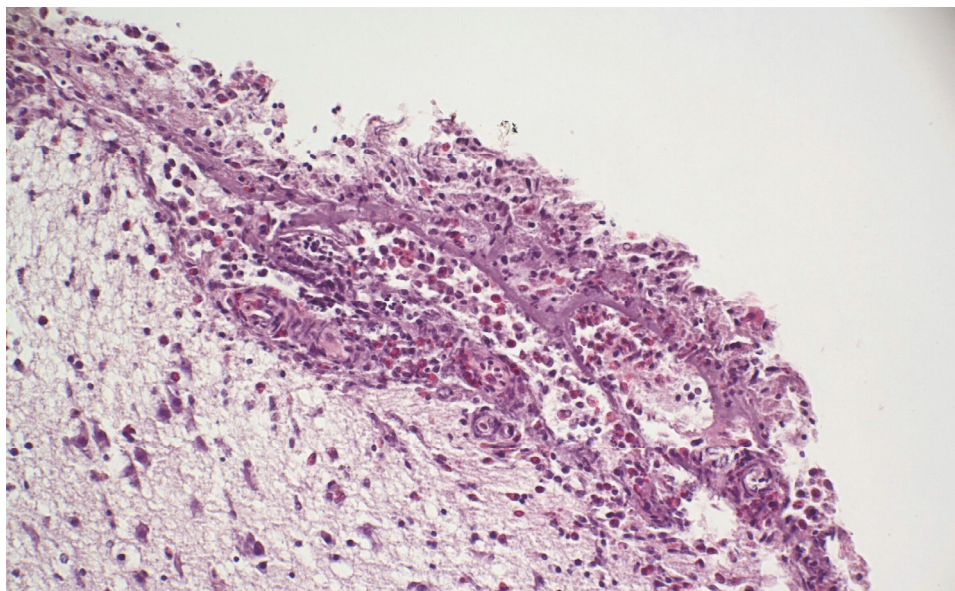


*Fig. 6.* Caseous salpingitis in a goose

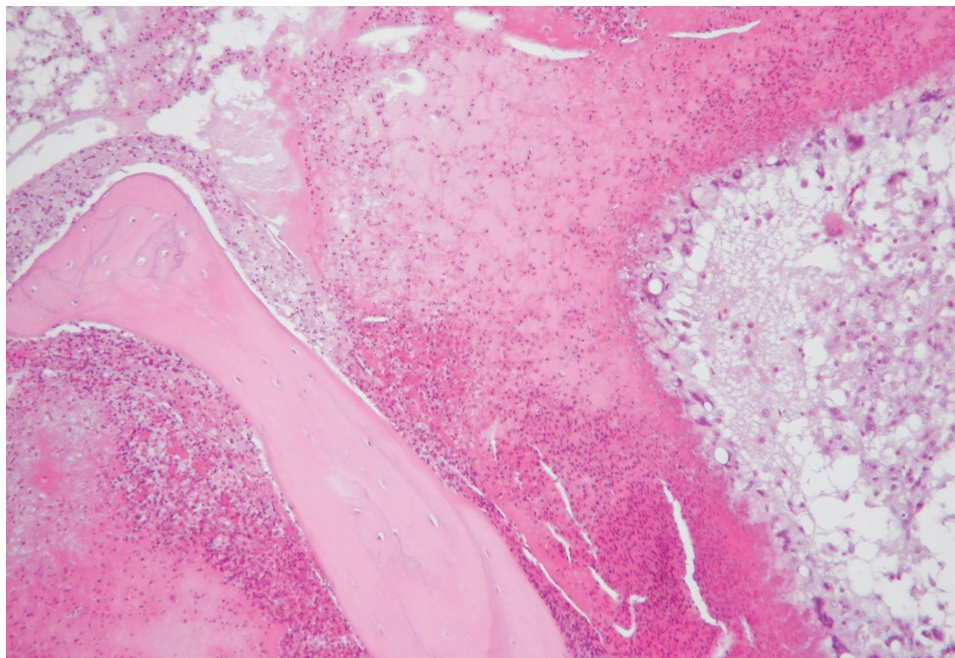


*Fig. 7.* Serofibrinous inflammation in the epicardium with clusters of bacteria (↑) in a goose.  
Haematoxylin and eosin, × 400





*Fig. 8.* The leptomeninx of the brain is diffusely thickened and infiltrated with inflammatory cells (lymphocytes, histiocytes and heterophil granulocytes) in a goose. Haematoxylin and eosin,  $\times 400$



*Fig. 9.* Seropurulent osteomyelitis with heterophil granulocyte infiltration in the cranium of a turkey. Haematoxylin and eosin,  $\times 200$

### *Isolation of R. anatipestifer*

The diagnosis of anatipestifer disease was always confirmed by the isolation of *R. anatipestifer* from the birds. The majority of strains from geese were isolated from the pericardium, brain, liver and occasionally from the joints, oviduct, lung, and in a few cases from the trachea, air sacs and peritoneum. The strains from ducks were most often isolated from the brain, pericardium or occasionally from the liver, oviduct, lung, trachea, and in one case from the conjunctiva. Almost all of the strains originating from turkeys were isolated from the cranium. In one case, *R. anatipestifer* was isolated from several organ samples in pure culture from the brain, air sac, trachea, pericardium, heart blood, liver, joint, and in mixed culture from the lungs.

### **Discussion**

Over a period of five years, we examined 1418 cases of goslings, 411 cases of ducklings, and 1292 cases of turkeys. Our data show that anatipestifer disease was one of the most frequently diagnosed diseases in geese and ducks, while in turkeys it was found only occasionally.

In geese, *R. anatipestifer* infection typically occurs at the age of 5 days to 6 weeks (Riemer, 1904; Pierce and Vorhies, 1973; Ivanics et al., 1996; Hess et al., 2013). We have diagnosed the disease in older, up to 17-week-old birds as well. In other parts of the world, anatipestifer disease has been seldom reported in geese (Hess et al., 2013); however, in Hungary it is rather common, which may be explained by the large number and high density of geese flocks in certain areas of the country. In young birds, the gross lesions and histopathological findings obtained in our cases were generally similar to those previously reported elsewhere (Riemer, 1904; Pierce and Vorhies, 1973; Ivanics et al., 1996; Hess et al., 2013). Nevertheless, we have observed some differences. When lesions in the brain occurred, birds showed oedema and hyperaemia of the subcutaneous connective tissue over the cranium. These lesions were seen in 2- to 6-week-old geese, usually accompanied by serofibrinous meningitis and ventriculitis of the brain. We found the septicaemic form of the disease typically in 1- to 6-week-old geese. *Riemerella anatipestifer* was regularly isolated from the pericardium, brain, and liver.

At the age of 8 to 17 weeks, which is the usual force-feeding period of geese, generally catarrhal pneumonia was the only finding. *Riemerella anatipestifer* was isolated from the lung and occasionally from the air sacs. In waterfowl over 8 weeks of age, in conformity with data of the literature (Ruiz and Sandhu, 2013), *R. anatipestifer* infection occurred sporadically and produced only local lesions. In some rare cases, however, we observed the acute septicaemic form of anatipestifer disease even in 9- to 17-week old geese. The bacterium was

isolated mostly from the liver, and occasionally from the pericardium, brain, lung and air sacs. Some of these birds had been force fed, which may have acted as a predisposing factor, or other immunosuppressive influences might have occurred. In about half of these cases, concurrent diseases were also observed.

In ducks, anatipestifer disease usually occurs at the age of 1 to 8 weeks (Pickrell, 1966; Jortner et al., 1969; Bitay et al., 1979). In our case, the disease was observed in 3- to 6.5-week-old ducks, which falls within this time interval. The gross and histopathological lesions were similar to those reported previously (Dougherty et al., 1955; Pickrell, 1966; Jortner et al., 1969; Bitay et al., 1979; Sarver et al., 2005), although we did not detect severe sinusitis, which had been described earlier in Hungary by Bitay et al. (1979). Oedema and hyperaemia of the connective tissue over the cranium were observed in the case of lesions in the brain, in the same way as found in geese. In one case, severe conjunctivitis was noticed in 3-week-old ducks, caused by *R. anatipestifer* infection in addition to mycoplasmosis suspected on the basis of the histopathological findings. The lack of septicaemia in ducks could possibly be explained by the lower pathogenicity of the bacterium or the different routes of exposure (Ruiz and Sandhu, 2013).

*Riemerella anatipestifer* is a facultative pathogen, and therefore anatipestifer disease occasionally occurs in combination with concurrent diseases both in waterfowl and turkeys (Ivanics et al., 1996; Banda et al., 2007; Rubbenstroth et al., 2009). In a certain part of the cases, *R. anatipestifer* infection complicated other primary diseases, such as circovirus infection, mycotoxicosis, mycoplasmosis, or Derzsy's disease. Circovirus causes immunosuppression in waterfowl; therefore, the presence of circovirus infection can facilitate several other infections. In our cases, the presence of circovirus infection, colibacillosis, paratyphoid, streptococcosis and pneumomycosis was detected within one case in addition to anatipestifer disease, which is consistent with the results of other authors (Ivanics et al., 1996; Banda et al., 2007; Rubbenstroth et al., 2009).

In turkeys, anatipestifer disease usually occurs at the age of 6 to 15 weeks (Zehr and Ostendorf, 1970; Helfer and Helmboldt, 1977; Smith et al., 1987). We have observed anatipestifer disease in turkeys at a slightly older age, at 12 to 19 weeks. Earlier studies reported that *R. anatipestifer* causes septicaemia, serofibrinous pericarditis, perihepatitis, airsacculitis, and occasionally pneumonia, sinusitis, arthritis and meningitis in turkeys. The isolation of this bacterium from the liver or spleen was usually successful (Zehr and Ostendorf, 1970; Helfer and Helmboldt, 1977; Smith et al., 1987). In contrast, we have diagnosed *R. anatipestifer* septicaemia in only one of the examined turkey cases. Generally, we observed local lesions such as purulent osteomyelitis in the cranium and seropurulent meningitis, arthritis and pericarditis mainly together with *E. coli* septicaemia or bacteraemia. American authors also observed *E. coli* infections that commonly followed *R. anatipestifer* infections in one to two weeks' time (Smith et al., 1987). In another case, serofibrinous pericarditis, peritonitis, purulent cranial os-

teomyelitis, purulent meningitis, ventriculitis in the brain, and arthritis were seen in 15- to 18-week-old turkeys. Septicaemia was not observed in these birds, since *R. anatipestifer* was isolated from various organs but neither from the liver nor from the heart blood. Presumably, some immunosuppressive effects might have predisposed these birds to *R. anatipestifer* infection. Israeli authors also reported *R. anatipestifer* infection in turkeys without septicaemia (Frommer et al., 1990). They observed neurological signs besides slightly increased mortality, and *R. anatipestifer* was isolated from the brain of the diseased birds, but no gross pathological lesions could be found. American authors reported spondylitis in turkeys as a consequence of experimental infection with *R. anatipestifer* via the intravenous route performed one week after vaccination (Cooper and Charlton, 1992). Vertebral osteomyelitis (spondylitis) was found in the sixth thoracic vertebra; however, purulent osteomyelitis in the cranium in association with *R. anatipestifer* infection has not been reported in the literature so far. The explanation could be a previous bacteraemia or an initiated antibiotic therapy. The origin of *R. anatipestifer* infection could not be thoroughly traced; however, transmission from duck populations kept in the region cannot be excluded. Previous studies reported nearby duck farms or duck manure as a source of *R. anatipestifer* infection in turkeys (Zehr and Ostendorf, 1970; Helfer and Helmboldt, 1977; Smith et al., 1987; Frommer et al., 1990).

The high incidence of the disease in waterfowl underlines the importance of appropriate treatment and prevention. Antibiotics are important tools for controlling the disease. On the basis of the antibiotic susceptibility testing of 185 *R. anatipestifer* strains isolated in Hungary, the most effective antibiotics were florfenicol, ampicillin, penicillin and sulphamethoxazole–trimethoprim with 97.9%, 95.1%, 93% and 92.4% sensitivity, respectively, although the resistance patterns showed some variation depending on the geographical origin and the year of isolation (Gyuris et al., 2017). *Riemerella anatipestifer* is a facultative pathogen, and therefore predisposing factors such as unfavourable environmental conditions should be avoided and concomitant diseases should be treated. Proper management procedures such as good sanitation practices, high level of biosecurity (separation of flocks on multiple-age farms, keeping away wild waterfowl and rodents) and all-in/all-out production should be implemented (Ruiz and Sandhu, 2013). Inactivated autogenous vaccines can be used for prevention (Ivanics et al., 1996). This approach seems to be the most effective as no cross-protection has been observed between the 21 serotypes of *R. anatipestifer* (Ruiz and Sandhu, 2013). Regular monitoring of the actual serotypes in a flock is recommended because more than one serotype can be present or the serotypes can change over time in the same farm (Bisgaard et al., 2008; Ryll et al., 2001).

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