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ISOLATION OF MYCOPLASMA SPP. FROM RACING PIGEONS (COLUMBA LIVIA)

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SUMMARY

Live and dead racing pigeons (Columba livia) from five lofts in Norfolk and Suffolk were examined clinically and cultured for Mycoplasma spp. Both clinically healthy birds and those showing signs of mild respiratory disease were included. The oropharynx was the culture site for 130 live birds, the nasal sinuses and other tissues for 58 carcases. Mycoplasma columbinum, M. columborale and M. columbinasale were isolated from the oropharynges and nasal sinuses; M. columbinum and M. columbinasale from the brain and M. columbinum and M. columborale from lungs and air sacs. One or more of these three Mycoplasma spp. was isolated at necropsy from 28% of 58 pigeons. Only 11% of 37 pigeons reacted serologically by the metabolism inhibition test to M. columbinum and none to M. columborale. Twenty-five birds examined for M. gallisepticum antibody by the haemagglutination-inhibition test were negative. No sex or age predilection to infection with Mycoplasma was apparent. About 10% of pigeons in all five lofts showed clinical signs of the respiratory disease sometimes described as 'mycoplasmosis catarrh', but most dead birds from which Mycoplasma spp. were isolated also had concomitant infections of various kinds. Although suggestive, the results of these investigations provide no clear evidence that Mycoplasma spp. are aetiologically involved in natural respiratory disease of pigeons. No conclusive satisfactory treatment was found for the elimination of mycoplasmas.

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INTRODUCTION

Respiratory problems appear to be common in racing pigeons and are difficult to diagnose, prevent and treat. Although pigeon fanciers recognise diseases such as “one-eyed cold”, “colds”, “ophthalmia”, “big-eye”, “coryza”, “roup” and “diphtheria” (Levi, 1974), there is considerable confusion regarding their aetiology. However, various diseases may be involved. These include ornithosis (Chlamydia psittaci infection), pigeon pox, Escherichia coli infection, aspergillosis (Aspergillus fumigatus infection) and vitamin A deficiency (Arnall and Keymer, 1975). Pigeon Herpesvirus I (PHV I) infection (Vindevogel and Pastoret, 1981) and lentogenic strains of Newcastle disease virus (Vindevogel et al., 1982) also cause respiratory signs, but the role of mycoplasmas in pigeons is debatable. Although mycoplasmosis is regarded as a clinical entity by many pigeon breeders and some veterinarians (Schrag et al., 1974), there is little documentary basis for this view.

Many strains of M. columbinum and M. columborale have been isolated from the trachea and oropharynx of clinically healthy feral pigeons in Japan (Shimizu et al., 1978) and from the respiratory tract and oesophagus of apparently healthy feral pigeons in Britain (Jordan et al., 1981). Sinclair (1980) isolated M. columbinasale (serotype L; Jordan et al., 1982) from pigeons showing respiratory disease. MacOwan et al., (1981) isolated M. columborale from pigeons in Britain also affected with a similar syndrome and demonstrated the potential pathogenicity of the organism by artificially infecting pathogen-free chickens.

This communication records the isolation of Mycoplasma spp. from racing pigeons in lofts with a history of mild respiratory disease similar to that referred to as ‘mycoplasmosis catarrh’ by Schrag et al. (1974).

MATERIALS AND METHODS

Between June 1976 and June 1982 racing pigeons with a history of mild respiratory disease from five different lofts in Norfolk and Suffolk were examined for mycoplasmas.

Birds from five lofts were submitted for post mortem examination, either as carcases or pigeons showing clinical signs (Table 1). Those received alive were killed by chloroform inhalation. Fifty-eight necropsies were carried out. The gross pathology was recorded and histopathological examinations made on a variety of tissues from 42 birds, using haematoxylin and eosin and other stains when indicated. The liver, intestinal tract and, sometimes, other organs of all dead birds were cultured for the usual range of pathogenic bacteria. This involved the use of MacConkey, sheep blood and deoxycholate citrate agar plates and selenite broth as appropriate. In addition, 130 apparently healthy and affected live birds from Lofts 3, 4 and 5 were clinically examined and swabs taken from the oropharynx for mycoplasma culturing.

Mycoplasma isolations from dead pigeons were carried out at the Veterinary Investigation Centre (VIC) Norwich. The nasal sinuses were cultured in all 58 carcases: other sites were also sometimes used, especially lung and cerebrum but occasionally trachea, air sac, cerebellum, ovary, oviduct, yolk in abdominal cavity, eye, liver or spleen. Swabs or suspensions from tissues were inoculated into 3 ml volumes of a modified Hayflick (1965) broth. The broths were subcultured to a modified Hayflick agar medium after incubation for 1 to 3 days. All media were incubated at 37°C in an atmosphere of 5 to 10% carbon dioxide in air and the plates enclosed in a plastic bag sealed with a cotton-wool plug. Swabs from live pigeons were examined.
Mycoplasma spp. in pigeons

mainly at the Mycoplasma Reference Laboratory (MRL), by means of a more complex isolation procedure involving initial inoculation of both agar and agar/broth overlay media plus additional cultures inoculated with diluted swab-wash fluids. The overlay media were subcultured to agar after appropriate incubation periods at 37°C. All agar plates were incubated in 5% CO\textsubscript{2} in nitrogen at 37°C.

The isolates were examined further at the MRL, where they were characterised as glucose-fermenting and/or arginine-utilising Mycoplasma spp. (Leach, 1973) and identified with specific antisera in colony immunofluorescence tests (Rosendal and Black, 1972), occasionally supplemented with metabolism inhibition (MI) tests (Leach, 1973). Some strains were lost, either during incubation or after storage, before being fully examined and these are recorded in Table 2 as unidentified Mycoplasma spp. A few isolates were identified at the Central Veterinary Laboratory (CVL), Weybridge, Surrey.

Fourteen attempts were made to isolate herpesviruses and pox viruses from the respiratory tract of pigeons housed in Loft 1 by the inoculation of tissues onto dropped chorioallantoic membranes of 9 to 10-day-old embryonated fowls’ eggs. Nine attempts were carried out by Mr. R.V. Barling, Houghton Poultry Research Station (HPRS) and five at the VIC, Norwich. At the CVL six cloacal swabs were examined for paramyxoviruses and influenza viruses by inoculation of the chorioallantoic cavity of 9 or 10-day-old embryonated fowls’ eggs.

Tissue culture (mouse fibroblast cell line NCTC 929 clone L cells) was used on eight occasions in an attempt to isolate Chlamydia psittaci from the spleen and respiratory tract.

Examination for parasites was made by searching the plumage of dead birds for ectoparasites and the alimentary tract for endoparasites. Wet smears of scrapings from the epithelium of the crop, duodenum and ileum were examined microscopically for protozoa and helminths.

Forth-five pigeons (Lofts 1, 2, 4 and 5), including 37 that had been killed prior to necropsy, were bled for serological examinations: 37 serum samples were examined at the MRL for *M. columbinum* and *M. columborale* antibodies by the MI test and 25 (Loft 1) for *M. gallisepticum* antibody at the VIC, using the haemagglutination-inhibition (HI) test.

Three anti-mycoplasmal antibiotics were used for treatment, namely:

1. Lincomycin hydrochloride BP (Lincomycin. Upjohn Ltd.) given continuously for 14 days at a dosage rate of 1.2 g/litre of the drinking water, calculated to provide each pigeon with 5 to 10 mg of the drug/day.
2. Tiamulin hydrogen fumarate (12.5%) (Dynamutalin solution, E.R. Squibb & Sons Ltd.) at 8 ml/litre of the drinking water for 7 days.
3. Spiramycin (Rovamycin, May & Baker Ltd.) at 300 mg/litre of the drinking water. This was administered for a week and repeated after an interval of a week.

RESULTS

Microbiological isolations

*Mycoplasma* spp. Three recognised species were isolated: *M. columbinum*, *M. columborale* and *M. columbinasale* (serotype L). *M. columbinasale* was first isolated in June 1976, *M. columbinum* in November 1978 and *M. columborale* in April 1979 (Table 1,
Nos. 1, 3 and 4), from dead birds examined during episodes of respiratory disease in both young and old birds. All three species were also isolated from 130 oropharyngeal swabs from live pigeons (Table 2), taken mainly from apparently healthy birds but also from a few reputed to be showing respiratory signs or with apparent excess catarhal material or other lesions in the oropharynx. The exact percentage of infected birds was not ascertained because some of the tested swabs were pooled (Table 2).

*Table 1. Mycoplasma isolations from pigeons* post mortem.

<table>
<thead>
<tr>
<th>No.</th>
<th>Month/year</th>
<th>Age and sex</th>
<th>Organs cultured</th>
<th>Culture results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6/76 (Loft 1)</td>
<td>Unrecorded</td>
<td>&quot;Brain&quot;</td>
<td><em>M. columbicasale</em></td>
</tr>
<tr>
<td>2</td>
<td>6/77 (Loft 1)</td>
<td>Unrecorded</td>
<td>Nasal sinus</td>
<td><em>M. columbicasale</em></td>
</tr>
<tr>
<td>3</td>
<td>11/78 (Loft 1)</td>
<td>&lt;1 year</td>
<td>Nasal sinus</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td>4</td>
<td>4/79 (Loft 1)</td>
<td>&lt;1 year</td>
<td>Nasal sinus</td>
<td><em>M. columbinum</em> and <em>M. columborale</em></td>
</tr>
<tr>
<td>5</td>
<td>6/79 (Loft 1)</td>
<td>1 month</td>
<td>Nasal sinus</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cerebrum</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td>6</td>
<td>6/79 (Loft 1)</td>
<td>&lt;3 months</td>
<td>Nasal sinus</td>
<td><em>M. columbinum</em> and <em>M. columborale</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Air sac</td>
<td><em>M. columbinum</em> and <em>M. columborale</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cerebrum</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td>7</td>
<td>7/79 (Loft 1)</td>
<td>&lt;3 months</td>
<td>Lung</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cerebrum</td>
<td>Negative</td>
</tr>
<tr>
<td>8</td>
<td>1/80 (Loft 1)</td>
<td>6 years</td>
<td>Oropharynx</td>
<td><em>M. columbinum</em> and <em>M. columborale</em></td>
</tr>
<tr>
<td>9</td>
<td>3/80 (Loft 1)</td>
<td>2 years</td>
<td>Air sac</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Nasal sinus</td>
<td>Negative</td>
</tr>
<tr>
<td>10</td>
<td>3/80 (Loft 1)</td>
<td>8 years</td>
<td>Cerebrum</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Nasal sinus</td>
<td>Negative</td>
</tr>
<tr>
<td>11</td>
<td>3/80 (Loft 1)</td>
<td>&lt;1 year</td>
<td>Cerebrum</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Nasal sinus</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Yolk in peritoneal cavity</td>
<td>Negative</td>
</tr>
<tr>
<td>12</td>
<td>4/80 (Loft 1)</td>
<td>18 days</td>
<td>Nasal sinus</td>
<td><em>M. columborale</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cerebrum</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lung</td>
<td>Negative</td>
</tr>
<tr>
<td>13</td>
<td>8/79 (Loft 2)</td>
<td>1 year</td>
<td>Nasal sinus</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cerebrum</td>
<td>Negative</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lung</td>
<td>Negative</td>
</tr>
<tr>
<td>14</td>
<td>10/80 (Loft 3)</td>
<td>2 years</td>
<td>Nasal sinus</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lung</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td>15</td>
<td>10/80 (Loft 4)</td>
<td>5 years</td>
<td>Nasal sinus</td>
<td><em>M. columbinum</em></td>
</tr>
<tr>
<td>16</td>
<td>3/82 (Loft 5)</td>
<td>1 year</td>
<td>Nasal sinus</td>
<td><em>M. columborale</em></td>
</tr>
</tbody>
</table>
Table 2. Mycoplasma isolations from oropharynges of 130 live pigeons (healthy and otherwise).

<table>
<thead>
<tr>
<th>No.</th>
<th>Month/year (loft)</th>
<th>Number of swabs examined</th>
<th>Number positive</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10/80 (Loft 3)</td>
<td>8</td>
<td>5</td>
<td>M. columbinum 3; and M. columborale 1; M. columbinum and M. columborale 1.</td>
</tr>
<tr>
<td>2</td>
<td>10/80 (Loft 4)</td>
<td>6</td>
<td>3</td>
<td>M. columbinum 1; M. columborale 1; M. columbinum and M. columborale 1.</td>
</tr>
<tr>
<td>3a</td>
<td>4/82 (Loft 5)</td>
<td>8</td>
<td>7</td>
<td>M. columbinum 1; M. columborale 1; M. columbinum and M. columborale 1; M. columborale and M. columbisale 1.</td>
</tr>
<tr>
<td>3b</td>
<td>4/82 (Loft 5)</td>
<td>10 (2 pools)</td>
<td>(2 pools)</td>
<td>Pool 1: M. columbinum and M. columbinasale Pool 2: M. columbinum.</td>
</tr>
<tr>
<td>3d</td>
<td>4/82 (Loft 5)</td>
<td>19 (3 pools)</td>
<td>(3 pools)</td>
<td>Pool 1: M. columborale Pool 2: M. columbinum and M. columborale Pool 3: Mycoplasma sp. (unidentified)a</td>
</tr>
<tr>
<td>4a</td>
<td>6/82 (Loft 5)</td>
<td>10</td>
<td>10</td>
<td>M. columborale 5; M. columborale and M. columbinasale 3; M. columbinum and M. columborale 1; M. columbinum and Mycoplasma sp. (unidentified)a 1</td>
</tr>
<tr>
<td>4b</td>
<td>6/82 (Loft 5)</td>
<td>29 (6 pools)</td>
<td>(5 pools)</td>
<td>Pools 1–5: M. columborale plus either M. columbinum or M. columbinasale Pool 6: Negative</td>
</tr>
<tr>
<td>4c</td>
<td>6/82 (Loft 5)</td>
<td>9 (2 pools)</td>
<td>(2 pools)</td>
<td>Pool 1: M. columbinum Pool 2: Mycoplasma sp. (unidentified)a</td>
</tr>
</tbody>
</table>

a Mycoplasma sp. (unidentified) = strain lost before identification was completed (see Materials and Methods).

Mycoplasma isolations were made from 16 (28%) of the 58 dead pigeons examined (Table 1). One or more Mycoplasma spp. were identified in birds of both sexes, varying in age from 18 days to 8 years. M. columbinum only was isolated from nine birds, M. columborale only from two and both species from three. M. columbinasale (identified at the CVL) was cultured from two birds in one loft. All M. columborale isolates were from the respiratory tract except one from the oropharynx, which probably indicated infection of the internal nares and therefore sinuses. M. columbinum and M. columbinasale were isolated from the respiratory tracts and brains. No mycoplasmas were cultured from other tissues.

Mycoplasmas were isolated in pigeons from all five lofts (Tables 1 and 2) between June 1976 and June 1982). M. columbinum or M. columborale or both were detected in all and M. columbinasale in two lofts. In four lofts there was evidence of disease caused by a variety of other organisms (see below).

Bacteria. Salmonellosis (Salmonella typhimurium phage type 99 infection) was diagnosed in 10 pigeons from Loft 1 (Table 1, Nos. 1, 2, 4-7, 9-12). All these birds were
also infected with *Mycoplasma* spp. However, although no other dual infections with *Salmonella* were diagnosed, *S. typhimurium* was isolated from one pigeon in Loft 2 and from another in Loft 4: phage type 99 and 2 respectively being present. Salpingitis associated with *Escherichia coli* infection was seen in one bird from Loft 2.

**Chlamydia.** All attempts to isolate *Chlamydia psittaci* were unsuccessful.

**Viruses.** A herpesvirus was isolated in Loft 1 from the respiratory tract of three pigeons which were infected with mycoplasmas. Pigeon pox was diagnosed in Loft 2.

**Parasites.** Trichomoniasis of the upper digestive tract was diagnosed in pigeons from Lofts 2 and 4, intestinal coccidiosis in Loft 5, and intestinal hexamitiasis in one bird from Loft 2. No parasites were found in association with *Mycoplasma* infections in Loft 1 and 3.

Helminth infestations were found in Lofts 4 and 5. In the former, several pigeons were affected with intestinal ascaridiasis (*Ascaridia columbae* infestation) and in Loft 5 with *Capillaria obsignata* infestation of the intestine.

**Serological results**

Using the MI test, only 4 (11%) of 37 tested pigeons reacted to *M. columbinum*. These were from Lofts 1 and 5. They reacted with only slight titres of 10 to 40 and showed some variability in repeat tests. No sera reacted to *M. columborale* and *M. columbina*-

**Histological results**

The value of histological examinations in diagnosing possible mycoplasma infections was limited, because of the presence in many birds of other infectious agents which could have been responsible for microscopic lesions. However, microscopic lesions were found in the trachea of one pigeon (Table 1, No. 16) in which *M. columborale* infection was considered to be unassociated with other potential pathogens. There appeared to be hyperplasia of the mucus glands and thickening of the tunica propria with mononuclear cell infiltration. Similar tracheal lesions have been seen since the end of this survey in another pigeon (not tabulated), from which staphylococci, as well as *M. columborale* and *M. columbinum*, were isolated from the nasal sinuses and oropharynx.

**Clinical aspects and therapy**

In all the affected lofts except No. 2, a mild respiratory syndrome was noticed in about 10% of the pigeons. "Coughing" occurred and respiratory rales could be heard, especially when birds were disturbed. Two owners reported failure of some birds to return after a race; one owner being convinced that the homing ability of infected pigeons was impaired. Most birds showed slight yellowish-white discharge around and within the external nares, but usually this was only visible on close examination. A few pigeons also exhibited unilateral or bilateral, slight, watery, ocular discharge. However, except in those with salmonellosis, gross lesions in the respiratory tract were very difficult to detect at necropsy, only a slight excess of catarrhal material being observed in the nasal sinuses. One pigeon with meningo-encephalitis (Table 1, No. 3) also showed air-sacculitis, with yellowish material in the abdominal air sacs.

**Loft 1.** The clinical signs and post-mortem lesions attributed to salmonellosis could not be distinguished from those suspected of being due to mycoplasma infection (Schrag *et al.*, 1974). However, pigeons in this loft responded to treatment with lincomycin hydrochloride BP, followed by a multivitamin preparation in the drinking water for a week. Housing, hygiene and ventilation were improved at the
same time and these may well have been important contributory factors, especially as respiratory signs have not recurred up to early 1983.

**Loft 2.** A pigeon was submitted for necropsy (Table 1, No. 13) because it showed so-called “one-eyed cold”, i.e. unilateral, ocular and nasal discharge. However, the problems in this loft were not primarily of a respiratory nature but were multifactorial and apparently associated with over-crowding and poor hygiene.

**Loft 3.** Birds recently purchased to set up a loft of racing pigeons developed respiratory problems. Either or both *M. columbinum* and *M. columborale* were isolated from five of eight oropharyngeal swabs taken from affected pigeons (Table 2, No. 1). One bird was received for necropsy (Table 1, No. 14). It showed no definite clinical signs or gross lesions but *M. columbinum* was isolated from its respiratory tract. It is not known if any treatment was given to the birds following these isolations and no subsequent history could be ascertained.

**Loft 4.** Initial respiratory signs shown by pigeons in this loft subsided temporarily following cessation of racing, improvement of the loft ventilation and treatment for worms and trichomoniasis. However, respiratory signs associated with excess mucus in the oropharynx then recurred and treatment with tiamulin hydrogen fumarate appeared not to be effective because respiratory signs persisted. *M. columbinum* was isolated from a sacrificed, affected bird (Table 1, No. 15) and both *M. columbinum* and *M. columborale* from three of six oropharyngeal swabs taken from six live birds (Table 2, No. 2) about 2 months after this treatment. Spiramycin was then administered and the clinical signs subsided. Attempts to isolate *Mycoplasma* spp. from the oropharynx of six pigeons chosen at random as soon as the treatment was finished were unsuccessful. The owner reported no further indication of respiratory disease in 1½ years following this investigation.

**Loft 5.** The owner reported mild respiratory signs, although the standard of hygiene and management appeared to be good and loft ventilation apparently satisfactory. After the isolation of *M. columborale* from the nasal sinuses of one typically affected killed bird (Table 1, No. 16), the oropharynges of all 60 pigeons (Table 2, Nos. 3a-d) in the loft were examined and swabs taken for culture. In eight pigeons there appeared to be excess catarrhal material in the oropharyngeal region. Swabs from these birds were cultured separately and *Mycoplasma* spp. were isolated from seven (Table 2, No. 3a). Swabs taken from the remaining apparently healthy 52 birds in the loft were pooled and *M. columbinum* and *M. columbinasale* were isolated (Table 2, Nos. 3b-d). After the results of these cultures were known, all birds in the loft were treated with spiramycin. Following this treatment, however, mild respiratory signs still persisted in some birds. Swabs were again taken from all the pigeons (then 48) in the loft and the cultures still revealed widespread presence of the three *Mycoplasma* spp. (Table 2, Nos. 4a-c), both in birds which appeared to have excess catarrhal material in their oropharynx and in pooled samples from apparently normal birds.

**DISCUSSION**

The results of the present investigations indicate that mycoplasmas are commonly present in racing pigeons, at least in Norfolk and Suffolk. Although suggestive, the results provide no clear evidence that the organisms are involved in natural respiratory disease. Both clinically healthy pigeons and those showing respiratory or other types of disease were infected with *M. columbinum* and *M. columborale*. The low prevalence of *M. columbinasale* isolations (Tables 1 and 2) might reflect merely its less vigorous cultural growth in standard mycoplasma media.
It is possible that the serological response, albeit slight, shown by a few birds to *M. columbinum* might point to some pathogenic potential for this organism. The MI test, although relatively specific, is not particularly sensitive for detecting natural mycoplasma infections and is inclined to give rather varying titres. The serological results therefore probably give no more than a preliminary indication that *M. columbinum* antibodies exist in some groups of pigeons infected with this organism. It is not yet clear whether the negative MI results for *M. columborale* also reflect the insensitivity of the method or simply a lack of pathogenicity of this organism. However, MacOwan *et al.* (1981) provided evidence of pathogenicity of *M. columborale*, by experimentally infecting specific pathogen-free chickens and similar tracheal lesions to those recorded by them in the chickens were also found by one of us (IFK) in clinically affected pigeons naturally infected with *M. columborale*. Serological examination of affected birds by other serological methods may prove useful, but it should be noted that Gerlach (1978) also found the occurrence of mycoplasma "humoral antibodies" to be "exceedingly rare" in pigeons.

In Loft 4, treatment with 12.5% tiamulin hydrogen fumarate was unsuccessful. This is contrary to the experience of Sinclair (1980), but agrees with the findings of Howse and Jordan (1983). Spiramycin treatment, however, appeared to be effective in this loft, although not in Loft 5. There are three possible explanations for this discrepancy, the most likely being that the owner of Loft 5 may not have carried out strictly the treatment regime with the result that not all the pigeons received an adequate dose. The possibility that all three spp. of *Mycoplasma* in Loft 5 were resistant to spiramycin cannot be entirely excluded, especially as the actual antibiotic-sensitivity spectrum of such isolates remains to be determined. The absence of detectable mycoplasmas in Loft 4 after treatment might be attributed to cultivation difficulties, but this seems to be the least likely explanation, especially as the same cultural methods were used for both lofts and that the pigeons in Loft 4, unlike those in Loft 5, responded clinically to the therapy. Since both the clinical respiratory disease and the mycoplasmas themselves disappeared from Lofts 1 and 4 after treatment with lincomycin and spiramycin, it remains possible that *M. columbinum* and *M. columborale* may have been clinically involved in these episodes.

Schrag *et al.* (1974) considered mycoplasmosis to be a cause of nasal catarrh in racing pigeons, especially when *Mycoplasma* (species not named) were associated with other infections. They stated that "it must be assumed that virtually all pigeons are infected with mycoplasma organisms". Gerlach (1977) considered that "the pathogenicity of all types of mycoplasmas was negligible". Subsequently, *Mycoplasma* spp. have been isolated from both healthy pigeons (Gerlach, 1978; Shimizu *et al.*, 1978; Jordan *et al.*, 1981) and those showing respiratory disease (Sinclair, 1980; MacOwan *et al.*, 1981). The aetiological situation in the field therefore remains uncertain.

It is evident that much more cultural and serological investigation of the rôle of mycoplasmas in both natural and experimental infection of pigeons is needed. Future pathogenicity studies should concentrate initially upon the three recognised pigeon *Mycoplasma* spp. and also on the possible rôle of concurrent viral infections such as *Herpesvirus* and Paramyxoviruses. More continuous monitoring is required for mycoplasmas and mycoplasma antibodies in lofts where mild respiratory disease is present, exploring the use of more varied serological methods. If possible, related studies should also be carried out under controlled conditions with experimentally-inoculated birds. Also needed is more knowledge of drug sensitivities of the various *Mycoplasma* spp. concerned.
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REFERENCES

RESUME
Isolation de Mycoplasma spp. à partir de pigeons voyageurs (Colomba livia)
Des pigeons voyageurs vivants et morts (Colomba livia) provenant de cinq pigeonniers du Norfolk et du Suffolk ont été examinés cliniquement et des cultures de mycoplasmes ont été réalisées, incluant à la fois les oiseaux sains et les oiseaux montrant des signes respiratoires. L'oropharynx a été le site de cultures pour 130 oiseaux.
vivants, les sinus nasaux et les autres tissus pour 58 carcasses. *Mycoplasma columbinum*, *M. columborale* et *M. columbinasale* ont été isolés de l'oropharynx et des sinus nasaux; *M. columbinum* et *M. columbinasale* du cerveau et *M. columbinum* et *M. columborale* du poumon et des sacs aériens. Un ou plus de ces trois *Mycoplasmas* spp. ont été isolés à l'autopsie de 28% des 58 pigeons. Seulement 11% des 37 pigeons ont réagi sérologiquement par le test d'inhibition métabolique vis-à-vis de *M. columbinum* et non de *M. columborale*. 25 oiseaux examinés en vue de la recherche des anticorps par le test d'inhibition hémagglutinante vis-à-vis de *M. gallisepticum* ont été négatifs. Aucune relation entre le sexe et l'âge et l'infection par *Mycoplasma* n'a été établie. Environ 10% des pigeons des cinq pigeoniers ont montré des signes cliniques de la maladie respiratoire quelquefois décrite comme une mycoplasmosse catarrhale et la plupart des oiseaux morts à partir desquels *Mycoplasma* spp. ont été isolés avaient des infections concomitantes de différentes sortes. Malgré le caractère suggestif des résultats de ces observations, aucune preuve n'a été apportée sur le rôle de *Mycoplasma* spp. dans l'étiologie des infections respiratoires naturelles du pigeon. Aucun traitement satisfaisant n'a été trouvé pour l'élimination des mycoplasmes.

**ZUSAMMENFASSUNG**

Die Isolierung von *Mycoplasma* spp. aus Reisetauben

(*Columba livia*)