Isolation of mycoplasmas from pigeons suffering eye lesions and respiratory disease

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**Isolation of mycoplasmas from pigeons suffering eye lesions and respiratory disease**

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Mycoplasmas cause a range of diseases in birds, in particular sinusitis, respiratory disease and eye disorders (Bradbury 1998). The most well-known pathogens are *Mycoplasma gallisepticum*, *Mycoplasma synoviae* and *Mycoplasma meleagridis*, but little is known of the pathogenic potential of other mycoplasmas that are often isolated from diseased birds. A few reports have described respiratory distress in racing pigeons (*Columba livia*), from which *Mycoplasma columborale*, *Mycoplasma columbinum* and *Mycoplasma columbinasale* have been identified, but definitive evidence for their role in disease is lacking (MacOwan and others 1981, Esposito 2000). This short communication describes an outbreak of respiratory and eye disease affecting a large number of racing and show pigeons.

The birds were of the white pavoncello, black cappuccio, triganino, gozzuto and torraiolo breeds; all were kept in a single, large cage 15 m long, 3 m wide and 2·2 m high, in Palermo, Italy, over a two-month period (July and August) in the summer of 2004 (Fig 1). In total there were 320 birds, of which 10 died and 24 presented with severe ocular lesions (Fig 2); 15 of these cases were unilateral, and in the other nine both eyes were affected. Permanent loss of vision in one or both eyes was evident in a number of birds. Approximately 60, mostly young, birds also showed respiratory distress. Small numbers of additional birds showed lesions daily.

Ocular and cloacal swabs were taken from 20 affected birds and cultured for mycoplasmas using standard procedures in Eaton's medium containing glucose and arginine (Nicholas and Baker 1998). Mycoplasmas were isolated from the eyes of the majority of birds showing eye lesions; mycoplasmas were also found in a small number of cloacal samples. The mycoplasmas were identified by growth inhibition tests using hyperimmune rabbit antiserum (Poveda and Nicholas 1998). Zones of inhibition exceeded 5 mm with antiserum to *M. columborale* but to no other avian mycoplasma. Two euthanased birds were examined postmortem; both showed small foci of pneumonia but no mycoplasmas could be isolated from the lung, spleen or liver using standard cultural procedures as described above (Nicholas and Baker 1998). Apart from a few opportunistic bacteria, no other pathogens, including viruses, were isolated.

During the early part of the episode, poor results were achieved with treatment of clinical cases with topical oxytetracycline (Xantervit; SIFI), so following discussions with G. R. L., a daily course of tylosin (Tylan 200 soluble; Elanco Animal Health), 250 to 300 mg/l in the drinking water, and erythromycin (Erythrocin; Abbott) 125 mg/l in the drinking water, was recommended and continued for 15 days. Despite this, recovery was slow, and a further 30 birds developed ocular lesions up to the end of September. However, by mid-October no further cases were seen and the majority of birds appeared healthy.

It is difficult to know why the birds became so severely affected, but stress of confinement in the cage for two months without release could have exacerbated any existing infection as the stocking density was high, which enabled the rapid spread of the mycoplasma to many of the birds. The owner stated that some bought-in birds had had contact with sick, captive falcons. These falcons had been fed on live chicks and had themselves experienced high levels of mortality; however, there are no reports of the isolation of *M. columborale* from chicks or birds of prey. There was no serological or cultural evidence of *M. gallisepticum* in the affected pigeons.

This report provides strong but not conclusive evidence that *M. columborale* was the causative or synergistic agent in the present outbreak. Further work is needed to confirm these observations.
References