AVIAN PARAMYXOVIRUS TYPE 1 INFECTIONS IN PIGEONS - SPREAD TO DOMESTIC POULTRY IN GREAT BRITAIN IN 1984

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I. DISEASE IN PIGEONS
a) Clinical signs

Although pigeons (Columba livia) and other members of the dove family, Columbidae, have long been known to be susceptible to infection with avian paramyxovirus type 1 (PMV-1) viruses, reports of natural infections have been rare and usually occurred when Newcastle disease has been prevalent in domestic poultry (reviewed by Lancaster and Alexander, 1975).

In 1981 a disease of racing and show pigeons was first seen in Europe and shown to be caused by a PMV-1 virus (Vindevogel et al, 1982). Respiratory disease signs were notably absent in affected pigeons but in all other respects the disease resembled the neurotropic form of Newcastle disease in chickens (Hanson, 1978). Clinical signs in pigeons consisted of combinations of: loss of condition, anorexia, excessive drinking, diarrhoea (frequently green), torticollis, dropping of wings, leg paralysis, alighting difficulties, tremors, inco-ordination and abnormal flying. The number of signs seen and their severity varied considerably but within these parameters the disease changed little over the years 1981-1985. Mortality and even morbidity was often difficult to assess due to culling by the owners and the very slow spread within a loft. However in more severely affected lofts up to 80% of the birds showed signs of disease and mortality as high as 50% was often reported. The disease appeared to be more severe in young birds.

b) Spread of disease

Reports of the disease suggest it first reached pigeons in Europe in 1981 when it was seen in Italy (Perini et al, 1982). However there is some evidence to suggest that the virus responsible was present in Iraq in 1977 and that the clinical disease was seen in Egypt in early
1981 (Kaleta, personal communication).

Due to the mixing of birds from widespread geographical locations at races and shows and trading of such birds, domesticated pigeons offer an efficient means of propagation and spread of virus diseases. Between 1981 and 1984 virtually every European country reported the disease in pigeons. In a collaborative study Alexander et al (1985a) examined isolates from 15 different countries and confirmed their identity using monoclonal antibodies (Table 1). In addition, reports from at least 9 other countries indicated the presence of disease. By 1984 the disease of pigeons had reached panzootic proportions being reported in countries representing Asia (Japan), Middle East (Iraq, Egypt, Israel), Africa (Sudan) and North America (USA) in addition to the European countries.

The disease was first reported in Great Britain in July 1983, and up to December 1983 disease was confirmed in 192 lofts (Alexander et al, 1984a). Vaccine was made available in September 1983, but there was a marked reluctance to vaccinate susceptible pigeons. During 1984 further spread of the disease was seen in racing and show pigeons and a total of 866 outbreaks were confirmed (Alexander et al, 1985c).

By the end of 1983 there was evidence in Great Britain (and other European countries) that the disease had spread to feral pigeons. Although evidence of such infections was rare, a focus of disease at Liverpool docks where large numbers of pigeons appeared to be severely affected caused some concern. The pigeon population at Liverpool and other docks in the Merseyside area was estimated as in excess of 30,000 birds and considerable numbers were seen to exhibit neurological signs with associated high mortality. Virus was isolated from a carcase submitted from this source in February 1984.

c) Characterisation of isolates

Use of conventional polyclonal antiserum confirmed that the causative virus of the disease in pigeons was a PMV-1 (or Newcastle disease virus, NDV) which showed some variation from more classical strains and could be regarded as a variant. Alexander et al (1984b) used mouse monoclonal antibodies to further characterize pigeon isolates and demonstrate their distinctiveness. Russell and Alexander (1983) had used monoclonal antibodies prepared against NDV-Ulster 2C to divide 40 NDV isolates into eight groups on the basis of their ability to cause binding of the monoclonal antibodies to infected MDBK cells which they