ABSTRACT

Pigeon herpesvirus 1 (PHV1) infection induces lesions in the upper digestive and respiratory tracts. It is the most common infection of pigeons in the E.E.C.

After experimental inoculation of squabs free of the infection, animals excrete high titres of infectious particles of PHV1 very soon and this excretion lasts a minimum of 7 to 10 days. The typical lesions appear 1 to 3 days after inoculation when viral excretion reaches its maximum. Mild episodes of recurrence (re-excretion of the virus) may occur spontaneously without clinical signs. High titres of antibodies do not prevent these recurrences and, conversely, recurrent episodes are not more frequent when the animals are nearly devoid of specific antibodies.

If experimentally infected pigeons are treated with cyclophosphamide a few months later, a viral excretion episode, nearly equivalent to that following primary infection, is provoked.

In a flock of pigeons naturally infected with PHV1, mature birds are asymptomatic carriers of the virus and they shed it from time to time. They may therefore transmit it to their offspring. Squabs become infected when they are protected from the disease by passive immunity of parental origin. Indeed, parental passive immunity, as in the other avian species, is conferred to the squabs through the egg yolk and protects it from the worst effects of infection. Therefore most of the squabs become latent carriers themselves after this initial infection although they are very soon devoid of detectable antibodies; nevertheless infection can be unmasked by cyclophosphamide treatment.

Clinical disease is therefore mainly observed during primary infection of young pigeons born to parents free of the infection or in carriers of the virus with the help of debilitating factors.

A strain of PHV1 has been attenuated by 100 passages on chicken embryo fibroblasts. The resulting attenuated strain multiplies in the animal to the same extent as the original wild strain and persists also after vaccination.

Previous infection of the pigeon with a wild strain of PHV1 prevents the occurrence of symptoms when they are re-infected.

Vaccination of pigeons either with the attenuated strain or with inactivated wild virus in oil adjuvant reduces viral excretion and symptoms after challenge with a virulent strain.
but is unable to prevent the establishment of latency as demonstrated by cyclophosphamide treatment of the animals. However, vaccination diminishes spontaneous viral re-excretion and therefore viral dissemination.

In conclusion, immunity, either passive or active, does not prevent the establishment of latent infection but helps to control the dissemination of the virus and protects from the disease resulting from infection. Under natural conditions, there is a sophisticated equilibrium between the virus and its host that prevents the occurrence of the disease.

INTRODUCTION

Since 1967, a herpesvirus (Pigeon herpesvirus 1, PHV1) has been isolated from pigeons affected with respiratory illness in numerous countries including Great-Britain, Belgium, France, Germany and Italy (Vindevogel, 1981).

Pigeon herpesvirus 1 infection is widespread in Belgium. Indeed, specific antibodies were detected by indirect immunofluorescence in the sera of 84% of clinically normal pigeons and by counter-immuno-electro-osmophoresis (C.I.E.O.P.) in 63% of the sera of pigeons affected with acute respiratory illness. PHV1 has been isolated in 60% of dove-cots permanently affected with respiratory troubles (Vindevogel et al., 1981; Vindevogel, 1981). A similar situation has been described in France and Germany (Heffels et al., 1981; Landré et al., 1982). PHV1 seems therefore to be frequently associated with respiratory distress in pigeons and we were able to reproduce the natural disease by experimental exposure of pigeons to the virus (Vindevogel et al., 1975; Vindevogel and Pastoret, 1981).

Classical signs of PHV1 infection are conjunctivitis, rhinitis and focal necrosis in mouth, pharynx and larynx. In addition, during natural outbreaks, lesions may be observed in the trachea, liver, spleen, kidney and pancreas (Vindevogel and Pastoret, 1981).

PHV1 has also been isolated from budgerigars where it provokes, naturally or experimentally, fatal hepatitis (Vindevogel and Duchatel, 1977; Vindevogel et al., 1980b).

PHV1 cannot be antigenically distinguished from Falcon herpesvirus and Owl herpesvirus but is antigenically dissimilar to Turkey herpesvirus (Turkey herpesvirus 1), Marek's disease virus (Phasianid herpesvirus 2), infectious laryngotracheitis