Isolation and Serological Differentiation of a Herpesvirus from Bobwhite Quail (*Colinus virginianus*, L. 1758)

Brief Report

By

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With 1 Figure

Accepted July 10, 1980

Summary

An infectious agent was isolated from the liver of bobwhite quails (*Colinus virginianus*, L. 1758). The agent was sensitive to chloroform and its multiplication was inhibited by 5-iodine-2-deoxy-uridine. It passed filters with a pore diameter of 220 nm and more but not 100 nm filters. Electron microscopic examination revealed numerous nucleocapsids with hollow capsomeres and few enveloped particles in the supernatant fluids of infected cultures. The nucleocapsids were calculated to have 162 capsomeres on their surface. Using the plaque reduction method for neutralization tests no serological cross reactions could be detected between the quail isolate and sera against Marek’s disease virus, turkey herpesvirus (HV), duck enteritis HV, infectious laryngotracheitis HV, amazon parrot HV, great horned owl HV, eagle owl HV, snowy owl HV, falcon HV, pigeon HV, Lake Victoria Cormorant HV, and stork HV. The isolate from bobwhite quail did only cross-react with antiserum against crane HV. It is concluded that the isolated virus is a member of the avian herpesvirus group and it is proposed to tentatively term it herpesvirus colinum (from *Colinus virginianus* = bobwhite quail).

Many different avian species may harbour economically and ecologically important herpesviruses (HV). Isolates were obtained hitherto from several species of owls, psittacine birds, cormorant, falcon, crane, black stork and domesticated birds such as chickens, turkeys, ducks and pigeons (2, 9, 11). This brief report describes the isolation and serological characterisation of a herpesvirus from bobwhite quail (*Colinus virginianus*, L. 1758) in Germany.
In December 1979 two dead bobwhite quails were submitted to the Clinic of Poultry, Hannover, for postmortem diagnosis. They originated from a group of 19 quails. According to the owner, all birds showed ruffled plumage, anorexia and wet droppings for two to three days. All birds died within the first 4 weeks of life. Both necropsied birds were in moderate to good muscular condition, the livers and spleens were enlarged, the parenchyma contained numerous yellowish to white foci of different sizes. The small intestines showed a catarrhal enteritis and several ulcers. The histological examination of the livers revealed multiple necrotic areas which contained in their centres rod-like bacteria. In addition, perivascular lymphocytic infiltrations could be noted. The bacteriological examination of heart, liver, spleen, lung and intestines of both birds yielded anaerobically growing bacteria which were identified as *Clostridium* spec. The course of the disease, the pathological and histopathological lesions as well as the isolation of *Clostridium* spec. are indicative for the so-called quail disease, a condition caused by clostridia in different species of quails. *Trichomonas* infection as well as salmonellosis and pseudotuberculosis might be accompanied with lesions similar to the ones described. It was not possible, however, to isolate any of these agents.

For virus isolation primary chicken embryo fibroblast (CEF) cultures were inoculated of affected livers. Details for virus isolation and characterization have been described elsewhere (7—10). Developing plaques consisted of few round refractile cells. Plaques were clearly visible on the tenth day p.i. and consisted of roundish cells in the cell sheet. Passages to fresh CEF cultures with cell-free infectious supernatant fluids and with cells dispersed with trypsin-versene buffer resulted in the development of a similar cytopathic effect five days p.i. as seen in the first cultures. Intranuclear inclusions with a distinct halo were visible on some cells.

If inoculated CEF cultures were maintained under agar overlay, distinct plaques were visible already after 6 days of incubation.

Inoculation of five days old SPF chicken embryos via yolk sac with infectious supernatant fluid resulted in no mortality but reduced growth, necrobiotic lesions in the livers, and numerous withish focal lesions on the chorioallantoic membranes at the 14th day of incubation. The titre of the quail virus in embryos was $10^{4.1}$ EID$_{50}$ in comparison to $10^{6.0}$ PFU$_{50}$ per ml in CEF cultures.

The chloroform treated quail virus yielded no plaques when inoculated into primary CEF cultures. Replicate cultures inoculated with untreated quail virus had a mean titre of $10^6$ PFU/ml.

Filtration through 450 and 300 nm membrane filters resulted in no definite drop of infectivity when compared with the titre of the unfiltered control. Most of infectivity was retained in the 220 nm filter. No infectivity was detected after filtration through 100 and 50 nm membranes. The concentrations of $100$ and $50$ µg/ml of 5-iodine-2-deoxy-uridine prevented plaque formation entirely. Only few plaques were visible in cultures treated with 10 µg IUDR/ml. Numerous particles with herpesvirus-like morphology were observed in the examined fraction. Only few particles had a tight envelope (Fig. 1), whereas the majority of them were naked nucleocapsids with 162 typical capsomeres (Fig. 1). The arithmetic mean of the diameter of 26 measured nucleocapsids was 101 nm. Projections on the surface of the viral envelope could not be detected.