viruses in the epitherial cells lining their upper respiratory tract. One of these reassortants, possessing new subtype of the hemagglutinin (HA) derived from avian virus, that infect, transmit to, and cause pandemic influenza in humans, is defined as a new pandemic strain. Since the HA is the target antigen for neutralizing antibodies, prediction of or immediate identification of the HA subtype of a new pandemic strain is important for the prevention and control of emerging influenza.

The phage display system was employed for the detection of influenza A virus proteins. In the present study, phage clones that specifically bind to purified influenza virus were selected from the library. The phage clones that bound to the nucleoprotein (NP) or the matrix protein were obtained. However, none of the clones bound to the HA. Some clones recognized both influenza A and B virus NPs. Such phage clones may be useful for the detection both of influenza A and B viruses from nasopharyngeal samples of patients and animals for rapid diagnosis of influenza virus infection.

Amino Terminus of Bovine Herpesvirus 1 Glycoprotein gB is Responsible for Membrane Fusion Activity

Sanae Fujii

Laboratory of Microbiology,
Department of Disease Control,
School of Veterinary Medicine,
Hokkaido University, Sapporo 060-0818, Japan

Herpesviruses penetrate into host cells by fusion of virus envelope with cell membrane. Since the glycoprotein gB homologues are conserved among all herpesviruses, the glycoprotein is believed to play an important role in the process of virus infection.

To provide information on the functional domain of bovine herpesvirus 1 (BHV1) gB, escape mutant resistant to anti-gB monoclonal antibody 185/1, which required complement for virus neutralization, was selected. Nucleotide sequencing analysis revealed that amino acid substitution from Arg to Gln occured at the amino terminal of the mutant gB. This substitution did not influence binding of virus to heparin, analog of viral receptor, kinetics of adsorption to and penetration into host cells, or yield of progeny viruses. On the other hand, the mutant virus formed smaller plaques than those of wild type virus. This finging indicates that the amino acid substitution at the amino terminal of gB reduced efficiency of cell-to-cell spread of the virus.

The gB-specific complement-independent neutralizing monoclonal antibody 118E inhibited the enlargement of plaques of the wild type virus. Since antibody 118E lacked reactivity to the escape mutant, the antibody should bind to the epitope overlapping that of antibody 185/1. These results indicate that cell-to-cell spread as well as initiation of infection of BHV1 is interfered with by binding of the antibody to the amino terminal of gB.

Taken together, the present results suggest that the amino terminal region of gB are responsible for membrane fusion activity of the glycoprotein which play roles in penetration and cell-to-cell spread of BHV1.