Picornaviruses

Family: *Picornaviridae*

Genus	Type species
Enterovirus	Poliovirus
Rhinovirus	Human rhinovirus A
Cardiovirus	Encephalomyocarditis virus
Aphthovirus	Foot-and-mouth disease virus
Hepatovirus	Hepatitis A virus
Parechovirus	Human parechovirus
Erbovirus	Equine rhinitis B virus
Kobuvirus	Aichi virus
Teschovirus	Porcine teschovirus 1

The family *Picornaviridae* includes many important human and animal pathogens, such as poliovirus, hepatitis A virus, foot-and-mouth disease virus, and rhinovirus. Because they cause serious disease, poliovirus and foot-and-mouth disease viruses have been the best-studied picornaviruses. Study of these two viruses has had important roles in the development of virology: the first animal virus discovered, in 1898, was foot-and-mouth disease virus. The plaque assay was developed using poliovirus, and the first RNA-dependent RNA polymerase discovered was poliovirus $3D^{pol}$. Polyprotein synthesis was discovered in poliovirus-infected cells, as was translation by internal ribosome entry. The first infectious DNA clone of an animal RNA virus was that of the poliovirus genome, and the first three-dimensional structures of animal viruses determined by X-ray crystallography were those of poliovirus and rhinovirus.

Figure 13 Structure and genomic organization. (A) The virion. (Left) Electron micrograph of negatively stained poliovirus. Courtesy of N. Cheng and D. M. Belnap, National Institutes of Health. (Right) Diagram of the virion, showing the names and locations of component proteins and genomic RNA. The capsid consists of 60 structural units (each made up of a single copy of VP1, VP2, VP3, and VP4, colored blue, yellow, red, and green, respectively) arranged in 12 pentamers. One of the icosahedral faces has been removed to illustrate the locations of VP4 and the viral RNA. **(B) Genome organization.** Polioviral RNA is shown with the VPg protein covalently attached to the 5′ end. The genome is of (+) polarity and encodes a polyprotein precursor. The polyprotein is cleaved during translation by two virus-encoded proteases, $2A^{pro}$ and $3C^{pro}$, to produce structural and nonstructural proteins. The P1 protein is cleaved into the virion capsid proteins, while the P2 and P3 proteins are cleaved to form the proteases and the proteins that participate in viral RNA synthesis.

Figure 14 Single-cell reproductive cycle. The virion binds to a cellular receptor **(1)**; the mechanism and site of uncoating of the RNA genome are unknown **(2)**. The VPg protein, depicted as a small orange circle at the 5′ end of the virion RNA, is removed, and the resulting RNA associates with ribosomes **(3)**. Translation is initiated at an internal site 741 nucleotides from the 5′ end of the viral mRNA, and a polyprotein precursor is synthesized **(4)**. The polyprotein is cleaved during and after its synthesis to yield the individual viral proteins **(5)**. Only the initial cleavages are shown here. The proteins that participate in viral RNA synthesis are transported to membrane vesicles **(6)**. RNA synthesis occurs on the surfaces of these infected-cell-specific membrane vesicles. The (+) strand RNA is transported to these membrane vesicles **(7)**, where it is copied into (–) strands carrying VPg at their 5′ ends **(8)**. These (–) strands serve as templates for the synthesis of (+) strand genomic RNAs **(9)**. Some of the newly synthesized (+) strand RNA molecules enter the translational system after the removal of VPg **(10)**. Structural proteins formed by partial cleavage of the P1 precursor **(11)** associate with (+) strand RNA molecules that retain VPg to form progeny virions **(12)**, which are released from the cell upon lysis **(13)**.