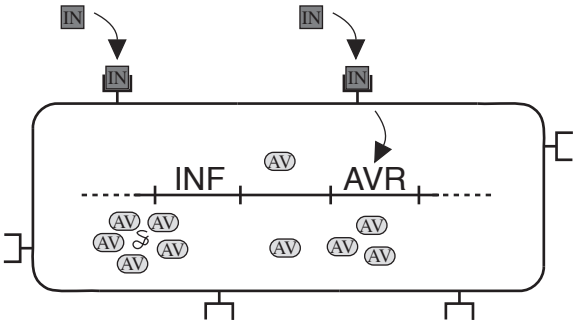
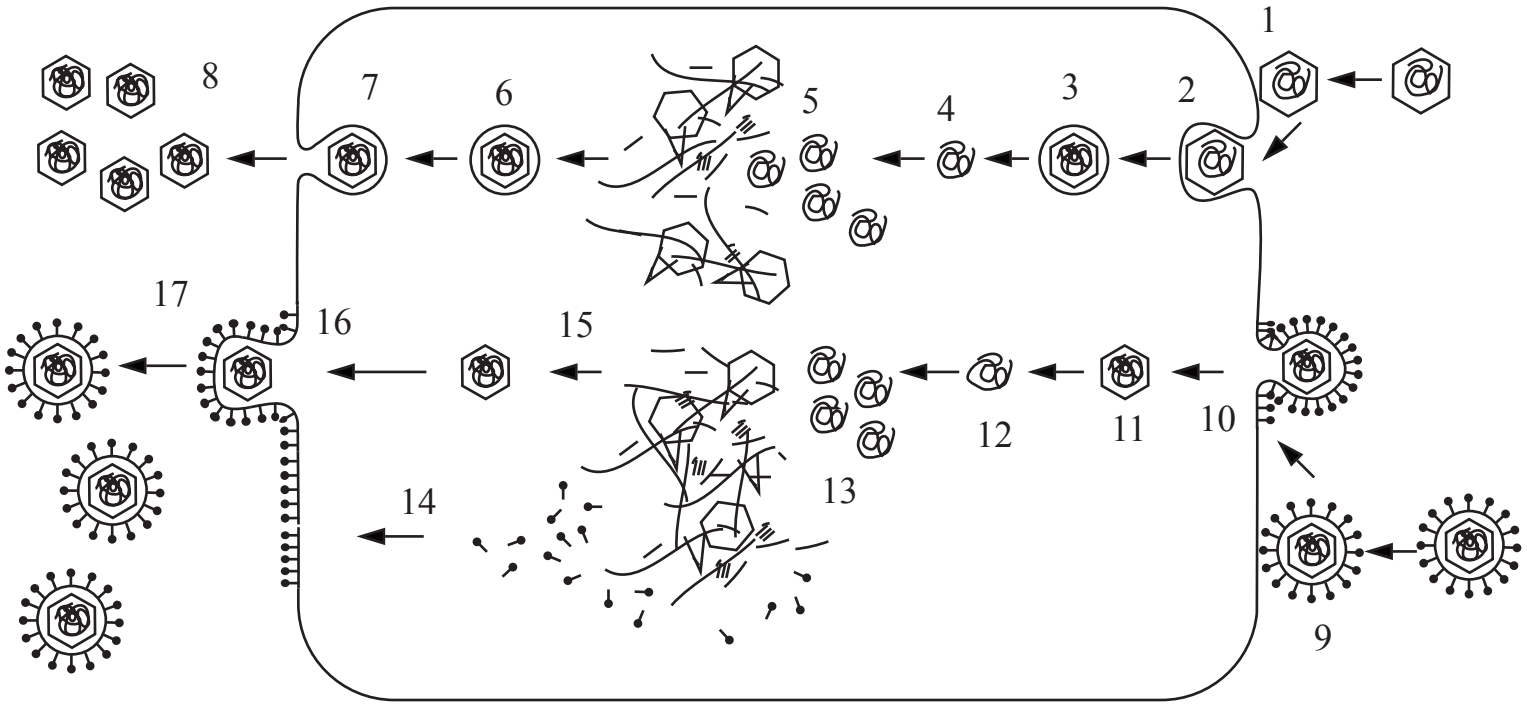
INTERFERON

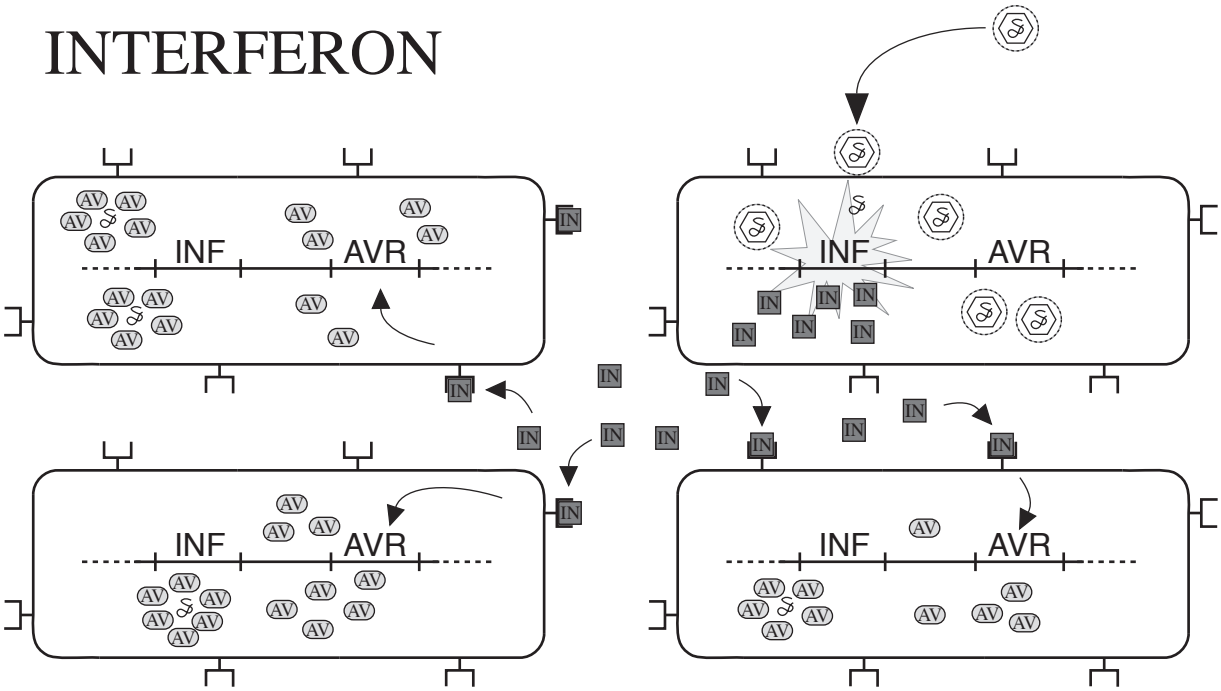


By Noel Ways



	Nonenveloped Viruses	Enveloped Viruses
Adsorption	1. Virus adsorbs (attaches) onto the host cell membrane by receptors on capsid.	9. Virus adsorbs (attaches) onto the host cell membrane by receptors on the envelope (in this case, spikes).
Penetration	2. The host cell is induced to engulf the virus. 3. The nucleocapsid is in vesicles derived from the cell membrane.	10. The viral envelope and host cell membrane fuse. 11. The nucleocapsid is released in the cytoplasm.
Uncoating	4. Envelope and capsid have been removed, and genetic material is released into the cytoplasm.	12. The capsid has been removed, and the virus' genetic material is released into the
Replication	5. Genetic material is duplicated and also used to make new viral components and enzymes.	13. Genetic material is duplicated and also used to make new viral components and enzymes. 14. Also, spikes will have formed and will embed in one region of the cell.
Assembly	6. New viral components self assemble. In this case, the virus is in a vesicle (details not covered here).	15. New viral components self assemble.
Release	7. The vesicle fuses with the host cell membrane. 8. The virus is released. Note, the host cell is not destroyed.	17. New viral nucleocapsid migrates to the region where spikes have been inserted. The host cell membrane now “buds” out and releases a new virus. Note, the host cell is not destroyed.

INTERFERON



INF — Interferon Gene

AVR — Anti Viral Replication Protein Gene

IN — Interferon

AV — Anti Viral Replication Proteins

Mature Viral Particles

Viral Genetic Material

Viral Genetic Material activity disrupted by Anti Viral Replication Proteins

The body cells above have two genes that work together to stop the viral life cycle with host cells. The first gene is the interferon gene (INF), and the anti-viral replication protein gene (AVR). The INF gene is expressed when a cell is virally infected. Although the infected cell will likely die as a result of the infection, the doomed cell will produce interferon (IN), which will leave the cell and bind to interferon receptors on the cell surface of neighboring cells. The neighboring cells respond by producing anti-viral replication proteins (AV). The presence of these proteins within uninfected cells will interfere with viral replication should a viral particle penetrate it. Therefore, the spread of the virus within the body is halted.

Eventually, the initially infected cell bursts, releasing hundreds of mature viral particles. When these particles infect the neighboring cells, and the viral particles' genetic material is released into the cells, the anti-viral replication proteins interfere with the viral life cycle by inhibiting RNA synthesis.

Interestingly, once someone has had a viral infection, there is a "window of time" where some viruses can not virally infect them for a short period of time. This is where the name "interferon" originally came from, where the process interfered with new potential infections. Obviously, there is much interest in exploiting interferon for medicinal purposes. Unfortunately, the utility of using interferon for therapeutic or prophylactic purposes is limited, although use against inhibiting one form of leukemia, genital warts, and hepatitis B have proven effective. Research continues!