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Viruses cause numerous medically important diseases, affecting developing, developed, rich and poor alike. The diseases vary in severity, including chickenpox, smallpox, influenza, shingles, herpes, rabies, polio, Ebola, hanta fever, AIDS and the common cold, amongst others. Regardless of the type of tissue or organ affected, all viruses follow the same basic steps to infect host cells. Once in contact with host cells viruses release their genetic material into the cell followed by genome replication, production of viral proteins, assembly of the virus particle and egress from the infected cell. Viruses disrupt normal host cell processes in order to facilitate their own replication/assembly by re-directing cellular machinery for viral transcription, translation, assembly, release and by inhibiting antiviral responses. Regulated nuclear transport of macromolecules through the nuclear pore complex, the only means of transport across the nuclear membrane, is essential for normal cell function and an effective antiviral response. Many viruses disrupt or exploit the nucleocytoplasmic trafficking pathways in host cells. Cytoplasmic viruses exploit the host cell nucleocytoplasmic trafficking machinery to access nuclear functions and/or disrupt nuclear transport, while several DNA viruses use the trafficking pathways to enable export of their components into the cytoplasm; yet others complete their assembly within the nucleus and use nuclear export pathways to access the cytoplasm. Indeed, the many and varied interactions of viruses and viral proteins with

nucleocytoplasmic trafficking components have been invaluable in pathway discovery. Importantly, mounting evidence suggests that these interactions play essential roles in virus replication/assembly and hence may be key to understanding pathophysiology of viral diseases. This Frontiers Research Topic is dedicated to the importance of nucleocytoplasmic trafficking to viral pathogenesis.