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An overview of viral immune evasion strategies

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Abstract
Viruses have evolved an extensive repertoire of immune evasion strategies that enable them to persist within hosts despite robust innate and adaptive immune responses. These mechanisms target nearly every stage of immune recognition and effector function, including pathogen sensing, interferon signaling, antigen presentation, antibody neutralization, and immune cell activation. Viral immune evasion not only facilitates viral replication and transmission but also contributes to chronic infection, immune dysregulation, and disease severity. This review provides an overview of the major strategies employed by viruses to evade host immunity, highlighting molecular mechanisms, representative viral examples, and their implications for antiviral therapy and vaccine development.

Keywords: Virus, Immune evasion, immune response, latency

Introduction
Viruses are obligate intracellular pathogens that rely entirely on host cellular machinery for replication and survival. Upon infection, host organisms activate a complex and multilayered immune response designed to recognize viral components, limit viral spread, and ultimately eliminate infected cells. The innate immune system provides the first line of defense through germline-encoded pattern recognition receptors (PRRs), such as Toll-like receptors, RIG-I–like receptors, and cytosolic DNA sensors, which detect conserved viral nucleic acids and proteins. Activation of these receptors initiates signaling cascades that culminate in the production of type I and III interferons (IFNs) and proinflammatory cytokines, establishing an antiviral state in both infected and neighboring cells (Randall & Goodbourn, 2008) [25].

Despite the effectiveness of these early responses, viruses have evolved sophisticated immune evasion mechanisms that enable them to circumvent host defenses. Viral immune evasion is a fundamental determinant of viral fitness, transmission, and pathogenicity, and it reflects a long-standing evolutionary arms race between host immunity and viral countermeasures. Many viruses encode proteins that directly antagonize PRR signaling pathways, suppress IFN induction, or inhibit IFN-stimulated gene expression. By targeting key adaptor molecules such as MAVS, STING, and IRF3, viruses can prevent the initiation of antiviral transcriptional programs, allowing replication to proceed largely unchecked during the early stages of infection (Alcami & Koszinowski, 2000; Finlay & McFadden, 2006) [1, 11].

Beyond innate immune suppression, viruses also interfere extensively with adaptive immune responses. Antigen presentation via major histocompatibility complex (MHC) class I and II molecules is essential for the activation of cytotoxic T lymphocytes and helper T cells. Numerous viruses, including herpesviruses and poxviruses, encode immune-modulatory proteins that disrupt antigen processing, retain MHC molecules in intracellular compartments, or promote their degradation, thereby preventing recognition of infected cells by T cells (Hansen & Bouvier, 2009) [12]. In addition, viruses may induce T cell exhaustion, skew cytokine responses, or interfere with costimulatory signaling to dampen effective adaptive immunity. Humoral immune responses are likewise targeted by viral evasion strategies. Neutralizing antibodies play a crucial role in preventing viral entry and reinfection, however, many viruses evade antibody-mediated immunity through rapid genetic variation, glycan shielding of surface proteins, or conformational masking of neutralizing epitopes.

RNA viruses such as influenza virus and human immunodeficiency virus (HIV) exhibit high mutation rates that facilitate antigenic drift, allowing escape from pre-existing antibody responses and contributing to persistent infection and vaccine challenges (Burton & Hangartner, 2016) [4].

In addition to direct immune antagonism, some viruses exploit host immune regulatory pathways to their advantage. Viral homologs of cytokines, chemokines, and their receptors can modulate immune cell recruitment and activation, creating a local environment conducive to viral persistence. Others manipulate apoptosis and autophagy pathways to prolong cell survival or evade immune-mediated cell death. Collectively, these strategies enable viruses to balance immune evasion with host viability, optimizing conditions for replication and transmission (Murphy *et al*., 2022) [22].

Understanding viral immune evasion strategies is of critical importance for public health and biomedical research. Immune evasion not only underlies viral persistence and chronic infection but also contributes to immune dysregulation, immunopathology, and disease severity. Detailed insights into virus–host interactions have informed the development of antiviral drugs, immune-based therapies, and vaccine platforms. As emerging and re-emerging viral pathogens continue to pose significant global threats, elucidating the mechanisms by which viruses evade immune surveillance remains a central focus in virology and immunology research.

Immune Evasion Strategies

Evasion or targeting of PRRs

Viruses have developed several ways to evade detection by PRRs. Many RNA viruses replicate in the cytoplasm where they are sensed by the cytoplasmic PRRs, MDA5 and RIG-I.

Thus, to avoid detection by the host innate immune system at their sites of replication, viruses have evolved several evasion strategies.

Bovine viral diarrhoea virus (BVDV) encoded E^{rns} released from the infected cells interfere with immune response by the degradation of the circulating nucleic acids. E^{rns} shows RNase activity in the intracellular compartment, thus preventing IFN production by degrading RNA and removing the resistant pathogen-associated molecular pattern (PAMP), thus maintaining the appropriate milieu for persistent infection. As an RNAse protein, E^{rns} is a glycosylated protein carrying several N-acetyl glucosamine molecules. The E^{rns} activity is not confined to bovine cells. Extracellularly added E^{rns} was shown to be uptaken into bovine turbinate cells, probably by clathrin-dependent endocytosis, and to remain active for a long time after been engulfed. Degradation of viral RNA takes place in endosomal compartments before reaching cytosol. The E^{rns} protein belongs to the T2 family of endoribonucleases that preferably cleave ssRNAs. However, monomeric E^{rns} showed the ability to cleave dsRNA and RNA in DNA/RNA, methylated RNA/RNA hybrid (Al-Kubati *et al.*, 2021) [2]. Porcine reproductive and respiratory syndrome virus (PRRSV) Nsp11 contains endoribonuclease activity and is highly conserved in nidoviruses. Nonstructural protein-1 (NSP-1) of influenza viruses is the most important IFNs antagonist protein, acting on multiple targets and suppressing the host IFN response. The viral NS1 protein binds to TRIM25. This prevents the activation of RIG-I (Chen *et al*., 2018) [5]. These enteroviruses encode two proteases, $2A^{pro}$ and $3C^{pro}$, required for viral polyprotein processing. However, 2Apro and 3Cpro have also been shown to cleave MDA5 and RIG-I, respectively (Feng *et al*., 2014) [10].

Targeting of adaptor proteins and their kinases

African swine fever virus (ASFV) I329L Inhibiting the crucial adaptor protein TRIF. A276R could inhibit IFN-β production by targeting IRF3 (Wu *et al.*, 2021) [28]. Hepatitis c virus (HCV) NS3/4A protease prevents activation of the transcription factor IRF3 and induction of IFN by cleaving the signaling adaptor protein MAVS. Positive-sense RNA viruses, porcine reproductive and respiratory syndrome virus (PRRSV) cleaves MAVS during infection (Dong *et al*., 2015). Adenovirus E1A and human papilloma virus 18 (HPV18) E7 proteins bind to STING to prevent induction of type I IFN upon DNA transfection (Lau *et al*., 2015) [18]. ASFV A528R protein distinctly downregulates the activities of both reporters stimulated by IFN-β and IFN-γ. However, the underlying precise mechanism of how these viral proteins inhibit IFN production remains largely unclear. A recent study showed that A528R can negatively regulate the cGAS-STING-mediated IFN signaling pathway by promoting the expression of autophagy-related protein ULK1 to degrade STING (Wu *et al*., 2021) [28].

Targeting transcription factors

Porcine deltacoronavirus nsp5 Antagonizes Type I Interferon Signaling by Cleaving STAT2. Bluetongue virus non-structural protein 3 (NS3) and NS4 coordinatively antagonize type I interferon signaling by targeting STAT1. Bovine alphaherpesvirus (BoAHV1) bICP0 inhibits IFN-β promoter activity in transient transfection studies by reducing IRF3 (interferon regulatory factor 3) protein levels. The RING finger of bICP0 is an E3 ubiquitin ligase suggesting it mediates IRF3 degradation in a proteasome dependent manner. BICP0 also interacts with IRF7 and impairs activation of IFN-β promoter activity, but does not reduce IRF7 protein levels. IRF3 and IRF7 are transcription factors that stimulate IFN-β promoter activity. IRF3 directly binds several consensus DNA binding sites, including an ISRE (IFN response elements), interacts with STAT1 (Signal transducer and activator of transcription 1) and prevents STAT1 from entering the nucleus (Jones, 2019) [16]. BVDV Npro induces degradation of the IRF3 (essential IFN activation factor) during the virus replication in cell culture. (Al-Kubati *et al.*, 2021) [2]. Enterovirus 68 (EV-D68) 3Cpro cleaves IRF7 during infection. ASFV A238L is an analog of the inhibitory subunit of NF-κB α (IκB-α) that can inhibit the activation of the NF-κB pathway (Wu *et al.*, 2021) [28]. The dengue virus (DenV) NS5 protein targets STAT2 for degradation, resulting in the ubiquitination and degradation of STAT2 (Morrison *et al*., 2013) [21].

Evasion of ISGs

Some viruses have evolved ways to cap their RNA to evade IFIT1 recognition. For example, Lassa fever virus and influenza virus snatch caps from host mRNAs. Additionally, many viruses encode proteins that can perform these capping functions. In particular, the West Nile virus NS5 protein contains 20-Omethyltransferase (20 O-MT) activity to generate a cap 1 structure. This particular cap structure is not sensed by IFIT1 during infection therefore this allows the virus to evade restriction by IFIT1. Coronaviruses, positive-sense ssRNA viruses, also encode a 20 O-MT protein, nsp16. Similar to the MT activity of WNV NS5A, the MT activity of nsp16 is required for evasion of IFIT sensing during both

murine hepatitis virus and severe acute respiratory syndrome coronavirus infection. SARS-CoV-2 RNAs are capped at the 5' end and escape recognition from PRRs (Encinar and Menendez, 2020) [9]. This virus encodes two proteins, pTRS1 and pIRS1, that antagonize PKR to prevent its autophosphorylation and subsequent phosphorylation of eIF2a. Importantly, deletion of the viral pTRS1 and pIRS1 proteins leads to decreased expression of viral early and late proteins, resulting in decreased viral replication. This suggests that these proteins are critical for HCMV to prevent the antiviral activity of PKR for its replication.

Complement evasion strategies

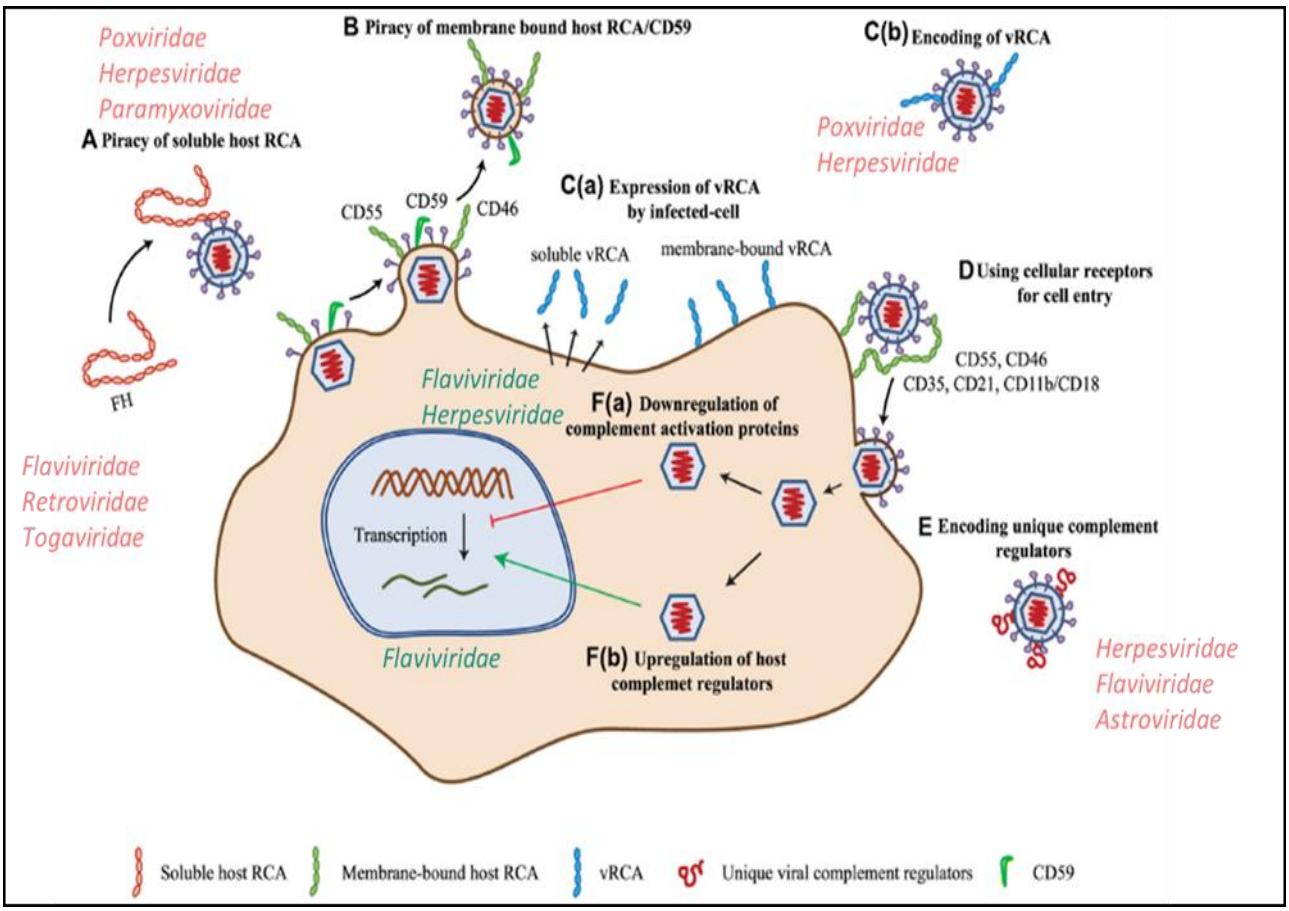

Fig 1: Complement evasion strategies of viruses, (A) Piracy of soluble host RCA.

Viruses evade complement attack by recruiting soluble complement regulator, *viz*. complement factor H (FH) by members of the families Flaviviridae, Retroviridae, and Togaviridae. (B) Piracy of membrane-bound host RCA. During budding, many enveloped viruses (*viz*. members of Poxviridae, Herpesviridae, Flaviviridae, Retroviridae, Orthomyxoviridae, and Paramyxoviridae) recruit membrane-bound regulators like CD55, CD46, and CD59. (C) Encoding homologs of RCA (vRCA). Viruses belonging to the families Poxviridae and Herpesviridae have been shown to encode regulators which are homologs of the human RCA gene family proteins. These are expressed as soluble [C(a)] as well as membrane-bound [C(b)] proteins. (D) Use of complement regulators and receptors for cellular entry. Viruses of the families Herpesviridae, Adenoviridae, Flaviviridae, Retroviridae, Picornaviridae, and Paramyxoviridae are known to use complement receptors and regulators for cellular entry (e.g., CD35, CD21, CD11b/CD18, CD55, and CD46). (E) Encoding of unique complement regulatory proteins. Apart from vRCA, members of some virus families namely, Herpesviridae, Flaviviridae, and Astroviridae encode unique complement regulatory proteins for evading the complement system. (F) Modulation of complement protein expression. Viruses are also known to modulate complement proteins for their benefit. These include down-regulation of complement activation proteins [F(a)] and up-regulation of complement regulatory proteins [F(b)]. Members of Herpesviridae, Flaviviridae, and Paramyxoviridae are involved in up-regulation of host complement regulators, while that of Flaviviridae are known to down-regulate the expression of complement activation proteins. Key: CD55, decay-accelerating factor; CD46, membrane cofactor protein; vRCA, viral regulators of complement activation; CD35, CD21, CD11b/CD18, complement receptor-1, -2 and -3.

Interference with antigen presentation via MHC class I and induction of antiviral immune responses

The Tat protein encoded by HIV-1 is a transcriptional activator of the viral long terminal repeat. However, it can also repress several cellular gene promoters. The activating and repressing functions reside in distinct domains of the protein. The repressive domain (at the C terminus) can associate with the transcription factor IID complex and inhibit the histone acetyl transferase activity of the TFII250 factor, causing repression of several genes involved in the induction of immune response, e.g., MHC class I and B_2 microglobulin. The E5 and E7 proteins of the bovine and human papillomaviruses are oncoproteins, which are expressed early in the viral life cycle in the Golgi complex (GC) and ER.

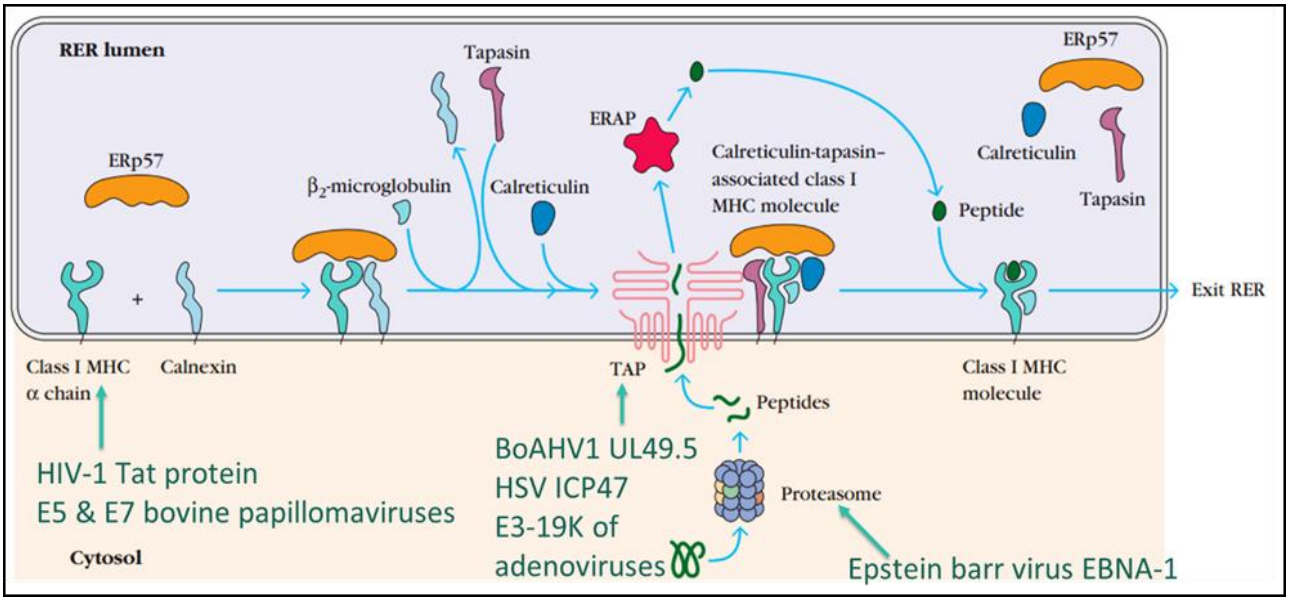

Fig 2: Assembly and stabilization of class I MHC molecules

They reduce MHC class I mRNA levels with a certain degree of specificity as well as retain MHC antigens in the GC and ER. The E1A early protein of the oncogenic adenovirus Ad12 also inhibits transcription of all components of the MHC class I pathway. The Epstein Barr virus protein EBNA-1, which is essential for replication of the viral episome in dividing virus-infected/transformed cells. The protein contains a glycine-alanine-rich (GAr) domain, which inhibits its degradation by the 26S proteasome, thus reducing the pool of EBNA-1-derived peptides that could be presented with MHC class I antigens on the cell surface. For example, the EBV encodes a nuclear protein, Blocking TAP functions

The bovine herpesvirus-1-encoded protein UL49.5 is a potent inhibitor of TAP. It inhibits TAP by inducing a conformational arrest of the transporter as well as by targeting TAP to proteasomal degradation. It is noteworthy that UL49.5 homologues are found in two other varicelloviruses: pseudorabies virus and equine herpesvirus-1.

The adenovirus early transcription unit-3 (E3)-19K and the HSV-1 protein infected cell peptide 47 (ICP47) can also inhibit peptide translocation into ER by blocking functions of TAP leading to a decrease in cell surface expression of MHC class I antigens. The ICP47 binds to the cytosolic side of TAP and blocks its function, whereas E3-19K binds TAP and MHC and acts as a competitive inhibitor of tapasin. The disruption of TAP function, however, does not affect expression of HLA-E, a neo-classical MHC class I molecule, which binds peptides derived from MHC class I signal sequences and confers protection from NK cell-mediated lysis.

A global indiscriminate down-regulation of MHC class I molecules on the surface of virus-infected cells may prevent their recognition from virus-specific CTL. However, this strategy also renders the infected cells susceptible to NK cell-mediated killing. As stated earlier, MHC class I molecules, particularly HLA-C, act as ligands for inhibitory NK cell receptors, e.g., KIR. A loss or a decreased expression of these HLA alleles on the surface of virus-infected cells results in a loss of inhibition of NK cells. To evade killing by NK cells and virus-specific CTL, many viruses have evolved strategies to differentially down-regulate MHC class I molecules. More specifically, they down-regulate expression of HLA-A and -B, which mainly present viral epitopes to CTL, but not the expression of HLA-C and HLA-E, which act as ligands for inhibitory NK cell receptors.

HIV-1 uses this strategy via Nef protein, which binds hypophosphorylated cytoplasmic tails in early forms of the MHC class I antigens in the ER and redirects them from the trans-Golgi network (TGN) to endosomal degradation. Indeed, studies have shown that all Nef domains (the N terminal alpha helix, polyproline, acidic, and oligomerization domains) are involved in this association. Nef interacts selectively with the intracellular tyrosine motifs of different HLA-A and HLA-B allotypes. However, the HLA-C and HLA-E do not have these tyrosine motifs and are not targeted by Nef, which interacts with the subunit of the cellular adaptor protein (AP) complex and recruits it to the MHC cytoplasmic tails (Tolstrup *et al*., 2004) [26]. Certain viruses may in fact increase the expression of these molecules on the surface of the infected cells, at least in the early phase of the infection, when NK cells are activated. For example, flaviviruses stimulate TAP activity by up to 50%. More specifically, by down-regulating the expression of co-stimulating molecules (Herzer *et al*., 2003) [13].

Evasion of NK cell responses

In humans the viruses may also evade NK cell responses by increasing the expression of HLA-E. The HCMV protein UL-40 acts as a source of the peptides that can bind HLA-E. Thus, by supplying a source of HLA-E-specific peptides, UL-40 stabilizes the expression of HLA-E on the surface of HCMV-infected cells. HLA-E inhibits NK cell activation by interacting with the inhibitory receptor CD94/NKG2A (Patel *et al*., 2018) [23]. It has been demonstrated recently that the HCMV UL141 gene product blocks the surface expression of CD155, which is known as a ligand for the activating NK cell receptors DNAM-1 (CD226) and TACTILE (CD96), (Jackson *et al*., 2011) [15].

By down-regulating the expression of co-stimulating molecules

Stimulation of CD4+ T cells via antigen alone (MHC class II molecules loaded with the receptor specific peptides) would not proliferate and produce IFN-unless co-stimulated via B7.1 and CD28 interactions. Instead, they would rather become anergic or undergo apoptosis. Kaposis sarcoma herpesvirus -

K5 down-regulates ICAM-1 and B7.2 on the surface of virus-infected cells. Myxomavirus homologue of the K5, M153R, is a Ub ligase. It targets MHC class I antigens and CD4 and internalizes and redirects them to proteasomal degradation (Coscoy *et al*., 2001) [7]. The adenovirus oncoprotein E1A decreases the expression of another adhesion molecule lymphocyte function-associated antigen-3 on the surface of Ad5- and Ad12-transformed cells. Nef, Vpu, and Gp160 of HIV-1 reduce surface expression of CD4 and CD28 on the virus-infected cells. Therefore, HIV infected cells cannot provide proper costimulation when they interact with virus-specific T cells (Bottley *et al*., 2005) [3].

Evasion from CTL by antigenic variation

One of the simplest mechanisms of viral immune evasion involves antigenic variation of RNA viruses. Rapidly occurring point mutations accompanied by poor editing functions of RNA polymerases permit the generation of large numbers of closely related but distinct viruses. A minor change to an influenza virus is known as antigenic drift. Both influenza A and B viruses undergo antigenic drift. Due to its segmented nature, influenza viruses can swap whole sections of their genome. If the segment swapped encodes an influenza antigen (such as HA or NA) which is targeted by the host immune system, this is termed antigenic shift and can radically alter a host immune system's ability to recognize the virus. The antigenic variability of viruses is a great hurdle in developing effective antiviral vaccines.

Immune evasion through latency

The state of a reversible, nonproductive viral infection in the host cells is called latency. Viruses may evade immune responses of the host by becoming "latent" and invisible to the immune system. During latency, viruses may infect nonpermissive or semipermissive cells of the host and express only a minimum number of viral genes, which are just necessary to maintain the virus in the cells. Some viruses may persist in immuneprivileged tissues of the host, e.g., brain, retina, and kidney (Khanna *et al*., 2004) [17]. HIV-1 is known to persist as a latent transcriptionally inactive provirus in the host cell's genome in long-lived, resting CD4+ memory T cells. These cells may lack virus-needed transcription factors. The virus may also persist in the brain, which is protected by blood brain barrier from infiltration of lymphocytes. These cells and tissues serve as reservoirs of the virus, which are resistant to chemotherapy and represent a real challenge for a complete elimination of the virus from the infected host. BoAHV1 is known to become latent in sensory ganglia (Jones, 2019) [16].

Targeting cytokines of the host

The poxviruses and herpesviruses modulate host's cytokine responses by producing proteins, which act as mimics for cytokines or their receptors. The BCRF-1 open-reading frame (ORF) of EBV encodes a protein (vIL-10), which is a homologue of the human IL-10. The HCMV UL111a gene also encodes an IL-10 homologue, which shares 27% sequence homology with human IL-10. Both the vIL-10s are highly immunosuppressive and can inhibit production of IFN- and TNF-from monocytes. The encoding of an IL-10 homologue is not restricted to herpesviruses; a poxvirus-encoded protein Y134R was also recently shown to have IL-10-like activities (Prichard *et al*., 2005) [24].

The certain poxviruses such as cowpox virus, ectromelia virus, and vaccinia virus encode a soluble protein vIL-18BP, which like its cellular homologue, binds and neutralizes the biological activity of IL-18. It is noteworthy that in concert with IL-12, IL-18 strongly stimulates antiviral cellular immunity. The cowpox virus CrmA inhibits caspase-1, also called IL-1-converting enzyme, which is needed to cleave precursor, immature IL-1and IL-18 into biologically active, mature cytokines (Iannello *et al*., 2006) [14].

Interference with apoptosis of the virus-infected host cells

All poxvirus genomes encode vIAP to inhibit apoptosis. The cowpox virus protein, the CrmA, can inhibit several caspases, probably via covalent modification of caspase 8, and prevents or delays apoptosis mediated by CTL, NK cells, TNF-alpha, and FasL (Iannello *et al.*, 2006) [14]. Adenoviruses protect virus-infected cells from apoptosis by inhibiting the expression of DR on the cell surface. The E3 region of all adenoviruses encodes three integral membrane viral proteins: E3-10.4K, E3-14.5K, and E3-6.7K. They are expressed as heteromeric complexes, receptor internalization and degradation (RID) complexes, which reduce the membrane expression of Fas and receptors for TRAIL and epithelial growth factor. The loss of these receptors leads to protection of the virus-infected cells from the cytototoxic activity exerted by CTL and NK cells. The RID complexes, however, do not target the transferrin receptor or MHC class I antigens. The complexes redirect intracellular trafficking of the DR to late endosomes for degradation (Windheim *et al*., 2004) [27].

Conclusions

Viruses have evolved different mechanisms to escape from almost all the possible immunological pathways. Evasion of the host antiviral innate immune response is critical for virus replication and spread. Viruses have several strategies to evade IFN induction and signaling to avoid the antiviral mechanisms of the host innate immune system. BVDV E^{rns} and PRRSV Nsp11 have endonuclease activity which remove the PAMP. 3Cpro and 2Apro of enteroviruses cleave RIG-I and MDA5 respectively to inhibit IFN production. PRRSV nsp4 and HCV NS3/4A cleave MAVS. BVDV Npro and BoAHV degrade the IRF3 protein. BTV NS3, PDCoV nsp5 and DenV NS5 target STAT molecule for inhibition of interferon stimulated gens production. WNV NS5 and coronaviruses generate cap structure for their RNA to evade PRR recognition. The E5 and E7 proteins of the bovine and human papillomaviruses are oncoproteins reduce MHC class I mRNA levels. BoAHV1 encoded protein UL49.5 is a potent inhibitor of TAP. The antigenic variability of influenza viruses and HIV is a great hurdle in developing effective antiviral vaccines. All poxvirus genomes encode vIAP to inhibit apoptosis. The cowpox virus protein, the CrmA, can inhibit several caspases and apoptosis.

Conflict of Interest

Not available

Financial Support

Not available

Reference

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