Viral protein involved in the evasion of host innate defense by inhibiting the pathway leading to the triggering of interferon-mediated response. This pathway usually starts with the recognition of viral RNA or DNA by host proteins including DDX58 or IFIH1. Then, the signal is transmitted through MAVS and TRAFs leading to the activation and nuclear localization of transcription factors IRF3 and IRF7 to induce IFNa/beta transcription and protein production. Many viruses interact with components of this pathway to inhibit production of interferons and establishment of the antiviral state.

Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host innate immune response initiation by virus.

Biological process.
DE has evolved to provide a broader and more finely tuned repertoire of DE recognition for both self- and nonself-antigens. A lot of viruses DE escape the adaptive immune response by different mechanisms including DE interference with the presentation of antigenic peptides at the DE surface of infected cells.

HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host adaptive immune response by virus.

CA Biological process.

ID Inhibition of host DDX58/RIG-I by virus.
AC KW-xxxx
DE Viral protein involved in the evasion of host innate defense by DE inhibiting the DDX58/RIG-I protein. Upon recognition of viral RNA, the DE cytosolic receptor DDX58/RIG-I initiates an antiviral signaling DE cascade by interacting with downstream partners. Several viral DE proteins inhibit DDX58/RIG-I via direct interaction while others via DE proteolytic cleavage.

HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of IFN-mediated immune response initiation by virus; Inhibition of host DDX58/RIG-I by virus.

CA Biological process.

ID Inhibition of host IFIH1/MDA5 by virus.
AC KW-xxxx
DE Viral protein involved in the evasion of host innate defense by DE inhibiting the IFIH1/MDA5 protein. Upon recognition of long viral DE dsRNAs, IFIH1/MDA5 initiates an antiviral signaling cascade by DE interacting with downstream partners. Some viral proteins including DE paramyxovirus V proteins interact with IFIH1/MDA5 and blocks its DE binding with its downstream partner MAVS.

HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of IFN-mediated immune response initiation by virus; Inhibition of host IFIH1/MDA5 by virus.

CA Biological process.

ID Inhibition of host TRAFs by virus.
AC KW-xxxx
DE Viral protein involved in the evasion of host innate defense by DE inhibiting TRAF proteins. After viral infection, the cellular DE signaling pathway leading to production of interferons is activated DE and several TRAF family members including TRAF2, TRAF3, and TRAF5 DE participate in this cascade. Many viruses encode protein able to DE interact with TRAF members to inhibit their antiviral activity.
HI  Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of IFN-mediated immune response initiation by virus; Inhibition of host TRAFs by virus.
CA  Biological process.

ID  Inhibition of host MAVS by virus.
AC  KW-XXXX
DE  Viral protein involved in the evasion of host innate defense by inhibiting the MAVS protein. During viral replication, dsRNA is produced and detected by DDX58/RIG-I or IFIH1/MDA5 that will activate MAVS to coordinate pathways leading to induction of antiviral cytokines. Several viral proteins including NS3/4A from Hepatitis C, or protease 3C from hepatitis A virus, cleave MAVS to abrogate its activity.

HI  Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of IFN-mediated immune response initiation by virus; Inhibition of host MAVS by virus.
CA  Biological process.

ID  Inhibition of host IRF3 by virus.
AC  KW-XXXX
DE  Viral protein involved in the evasion of host innate defense by inhibiting the interferon regulatory factor-3 (IRF3) protein. Viral infection triggers the phosphorylation and activation of IRF3. The activated IRF3 migrates to the nucleus, where it complexes with the transcription coactivator CREBBP/EP300, leading to the transcriptional activation of the IFN-alpha and IFN-beta genes. Several viral proteins directly bind to IRF3 and inhibit its transcriptional activity while others target it to the proteasome for degradation.

HI  Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of IFN-mediated immune response initiation by virus; Inhibition of host IRF3 by virus.
CA  Biological process.

ID  Inhibition of host IRF7 by virus.
AC  KW-XXXX
DE  Viral protein involved in the evasion of host innate defense by inhibiting the interferon regulatory factor-7 (IRF7) protein. Viral infection triggers the phosphorylation and activation of IRF7. The activated IRF7 migrates to the nucleus leading to the transcriptional activation of the IFN-alpha and IFN-beta genes. Some viral proteins prevent IRF7 phosphorylation and nuclear activation. Ebola virus VP35 interacts with IRF7 and hijacks the cellular SUMOylation machinery for its advantage to increase IRF7 SUMOylation thereby disabling its
DE activity.
HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of IFN-mediated immune response initiation by virus; Inhibition of host IRF7 by virus.
CA Biological process.

ID Inhibition of host IRF9 by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of the type I and III interferon pathway by inhibiting the interferon regulatory factor-9 (IRF9) protein. Viral infection triggers the phosphorylation and activation of IRF9. The activated IRF9 migrates to the nucleus leading to the transcriptional activation of several hundred IFN-responsive genes. Some viral proteins inhibit IRF9 activation by preventing its nuclear localization upon infection or by sending it to the nucleus in an inactive state.
HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host IRF9 by virus.
CA Biological process.

ID Inhibition of host TBK1/IKBKE/DDX3 complex by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of host innate defenses by inhibiting the TBK1/IKBKE/DDX3 complex. Upon infection, the virus is recognized and the signal is transmitted to TBK1 and IKBKE that in turn phosphorylate and activate IRF3 and IRF7. Once phosphorylated, IRF3 and IRF7 homodimerize and translocate into the nucleus to drive transcription of interferons. Several viruses including Ebolavirus and Bornavirus interact directly with and inhibit TBK1 to prevent IRFs activation. Other viruses such as vaccinia virus inhibit host DDX3 to block the signaling pathway.
HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of IFN-mediated immune response initiation by virus; Inhibition of host TBK1/IKBKE/DDX3 complex by virus.
CA Biological process.

ID Inhibition of host STAT1 by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of the type I, II and III interferon pathways by inhibiting the STAT1 protein. Upon viral infection, STAT1 is activated by IFN-gamma, IFN-alpha/beta, or IFN-lambda that bind to specific cell surface receptors. While IFN-gamma induces STAT1 homodimerization, IFN-alpha/beta and IFN-lambda stimulate heterodimerization of STAT1 and STAT2, both leading to STAT1
DE nuclear localization and subsequent induction of IFN-stimulated genes.
DE Many viruses interfere with STAT1 activation, often by preventing
DE STAT1 phosphorylation and nuclear localization.
HI Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host STAT1 by virus.
CA Biological process.

ID Inhibition of host STAT2 by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of type I and III interferon
DE pathways by inhibiting STAT2 protein. Upon viral infection, STAT2 is
DE activated by IFN-alpha/beta or IFN-lambda that bind to specific cell
DE surface receptors. In turn, IFN-alpha/beta (or IFN-lambda) induces
DE heterodimerization of STAT1 and STAT2 by phosphorylation, leading to
DE STAT2 nuclear localization and subsequent induction of IFN-stimulated
DE genes. Many viruses interfere with STAT2 activation, often by
DE preventing STAT2 phosphorylation and nuclear localization.
HI Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host STAT2 by virus.
CA Biological process.

ID Inhibition of host NF-kappa-B by virus.
AC KW-XXXX
DE Viral protein involved in the inhibition of host NF-kappa-B. This
DE protein is a pleiotropic transcription factor which is present in
DE almost all cell types and is involved in many biological processes
DE such as inflammation, immunity, differentiation, cell growth,
DE tumorigenesis and apoptosis. Many viruses have developed strategies to
DE inhibit the NF-kappa-B pathway in order to evade host immunity and
DE inhibit production of proinflammatory cytokines.
HI Biological process: Host-virus interaction; Inhibition of host NF-
DE kappa-B by virus.
CA Biological process.

ID Activation of host NF-kappa-B by virus.
AC KW-XXXX
DE Viral protein involved in the activation of host NF-kappa-B. This
DE protein is a pleiotropic transcription factor which is present in
DE almost all cell types and is involved in many biological processes
DE such as inflammation, immunity, differentiation, cell growth,
DE tumorigenesis and apoptosis. Several viruses have developed strategies
DE to activate the NF-kappa-B pathway in order to promote viral
DE replication and prevent virus-induced apoptosis.
HI Biological process: Host-virus interaction; Activation of host NF-
DE kappa-B by virus.
CA Biological process.

ID Inhibition of host TYK2 by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of the type I and III interferon pathways by inhibiting the host TYK2 protein. Upon viral infection, the TYK2 protein is activated by IFN-alpha/beta or IFN-lambda stimulation leading to a series of phosphorylation events that induce transcription of several hundred IFN-responsive genes. Several viruses have evolved mechanisms to inhibit TYK2 activity thereby preventing the subsequent activation of downstream partners STAT1 and STAT2.
HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host TYK2 by virus.
CA Biological process.

ID Inhibition of host JAK1 by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of the type I, II and III interferon pathways by inhibiting the JAK1 protein. Upon viral infection, JAK1 is activated by the interferon-alpha/beta, -gamma, and -lambda signal transduction pathways. Several viral proteins can directly interact with JAK1 to prevent its ability to phosphorylate the downstream partners STAT1 or STAT2.
HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host JAK1 by virus.
CA Biological process.

ID Inhibition of host ISG15 by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of host immune defense by inhibiting the ISG15 protein, an ubiquitin-like modifier playing important roles in the innate immune response. Like ubiquitin, ISG15 is conjugated to lysines on numerous target proteins through its conserved C-terminal region. Viruses escape from the antiviral activity of ISG15 by direct interaction or by cleavage of ISG15 derivatives.
HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host ISG15 by virus.
CA Biological process.

ID Inhibition of host interferon receptors by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of the interferon pathway by
inhibiting interferon receptors. Interferon signaling exerts antiviral effects through cell surface receptors termed interferon receptors. In response to binding of extracellular interferons, they activate the JAK/STAT pathway causing transcriptional activation of IFN-regulated genes. To avoid this antiviral response, several viruses target the interferon receptors and send them to degradation via the proteasome.

**Biological process:** Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host interferon receptors by virus.

Viral protein involved in the evasion of the interferon pathway by inhibiting the interferon induced PKR/EIF2AK2 protein. During viral replication of RNA viruses, dsRNA is produced leading to the activation of the PKR/EIF2AK2 kinase. Once activated, PKR/EIF2AK2 autophosphorylates and catalyzes the phosphorylation of many substrates including the translation initiation factor EIF2S1, leading to the inhibition of the initiation of protein synthesis. Several viral proteins prevent PKR/EIF2AK2 activation by direct interaction while others target PKR/EIF2AK2 to degradation.

**Biological process:** Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Inhibition of host PKR/EIF2AK2 by virus.

Protein phosphatase-1 (PP1) is a member of the Serine/Threonine phosphatases. The enzyme regulates many important physiological processes, including gene transcription, translation, metabolism, cell growth and division. Different viruses including asfivirus, herpes simplex virus or papillomavirus interact with and modulate PPP1 phosphatase activity to dephosphorylate specific cellular substrates including EIF2S1. Upon viral infection, the host PKR/EIF2AK2 triggers the phosphorylation of EIF2S1 leading to a complete translational shut-off. By dephosphorylating EIF2S1 with PPP1CA, viruses manage to circumvent this antiviral response and prevent translational shut-off.

**Biological process:** Host-virus interaction; Viral immunoevasion; Inhibition of host innate immune response by virus; Inhibition of interferon signaling pathway by virus; Modulation of host PP1 activity by virus.

**Biological process.**
ID  Inhibition of host BST2/Tetherin by virus.
AC  KW-XXXX
DE  Viral protein involved in the evasion of host immune defense by
DE  inhibiting the BST2/Tetherin protein. BST2/Tetherin is an alpha
DE  interferon-inducible cellular factor that impairs the release of many
DE  enveloped viruses, including human immunodeficiency virus type 1 (HIV-
DE  1), HIV-2, as well as other retroviruses. Several viruses manage to
DE  circumvent the antiviral activity of BST2/tetherin either by sending
DE  BST2/Tetherin to degradation (HIV-1) or by lowering the presence of
DE  BST2 on cell surfaces (HIV-2).
HI  Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host innate immune response by virus; Inhibition of
interferon signaling pathway by virus; Inhibition of host BST2/Tetherin by
virus.
CA  Biological process.

ID  Inhibition of host complement factors by virus.
AC  KW-XXXX
DE  Viral protein involved in the evasion of host humoral response by
DE  inhibiting the complement factors. The activation of complement
DE  involves the sequential proteolysis of proteins to generate enzymes
DE  with catalytic activities. The biological functions of the complement
DE  include opsonization, inflammation, lysis of immune complexes, or
DE  enhancement of the humoral immune response. Some herpesviruses,
DE  poxviruses and retroviruses mimic or interact with complement
DE  regulatory proteins to block complement activation and neutralization
DE  of virus particles.
HI  Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host complement factors by virus.
CA  Biological process.

ID  Inhibition of host chemokines by virus.
AC  KW-XXXX
DE  Viral protein involved in the evasion of host immune response by
DE  inhibiting chemokines. Chemokines have several roles including Th1/Th2
DE  differentiation, T_cell costimulation, or promotion of leukocyte
DE  migration. Due to the importance of chemokines in immunity, viruses
DE  have evolved mechanisms to counter the chemokine network. They encode
DE  chemokine-like proteins, chemokine receptors, or chemokine-binding
DE  proteins to inhibit cellular chemokines.
HI  Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host chemokines by virus.
CA  Biological process.

ID  Modulation of host immune response by viral IgG Fc receptor-like
protein.
Viral protein acting as an IgG Fc receptors, able to bind IgG and inhibit host Fc-dependent immune activation. Fc receptors are proteins found at the surface of certain cells of the immune system including macrophages, monocytes, natural killer cells or B-cells. They allow these cells to bind to antibodies that are attached to the surface of infected cells or pathogens, helping these cells to identify and eliminate pathogens.

Biological process: Host-virus interaction; Viral immunoevasion; Modulation of host immune response by viral IgG Fc receptor-like protein.

Viral protein sharing sequence homology with host interleukins. Interleukins are produced by immune system cells such as lymphocytes, macrophages and monocytes, and modulate inflammation and immunity by regulating growth, mobility and differentiation of lymphoid and other cells. Several viruses encode interleukin-like proteins playing a role in immune evasion. Additionally, viral interleukins have been shown to activate cellular signaling cascades that enhance virus replication.

Biological process: Host-virus interaction; Viral immunoevasion; Modulation of host immune response by viral interleukin-like protein.

Viral protein involved in the evasion of host adaptive immune response by inhibiting MHC class I peptide antigen generation by the proteasome. The processing of foreign proteins leads to the presentation of viral peptides by MHC class I molecules to cytotoxic T lymphocytes and triggers immune response. Several viral proteins have evolved mechanisms to avoid synthesis of antigenic peptide by the proteasome. Epstein-Barr virus EBNA-1 for example contains an internal repeat exclusively composed of glycines and alanines that inhibits its proteasomal degradation.

Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host adaptive immune response by virus; Inhibition of proteasome antigen processing by virus.

Viral protein involved in the evasion of host adaptive immune response by inhibiting the TAP complex. Transporter associated with antigen (TAP),
composed of two subunits TAP1 and TAP2, is required for the translocation of peptides into the ER, where they are loaded onto MHC class I. Thereafter, the viral peptides are presented to cytotoxic T lymphocytes at the cell surface and trigger immune response. The loading of peptide on MHC by TAP is targeted by several viruses including herpesviruses and retroviruses.

Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host adaptive immune response by virus; Inhibition of host TAP by virus.

Inhibition of host tapasin/TAPBP by virus.
Viral protein involved in the evasion of host adaptive immune response by inhibiting the tapasin/TAPBP protein. Tapasin/TAPBP is a type I transmembrane protein essential for the optimal expression of stable MHC class I molecules on host cell surface. It helps the MHC class I molecules to remain in a peptide receptive state, avoiding irreversible denaturation. Several retroviruses and DNA viruses encode proteins interacting with tapasin/TAPBP and inhibiting its activity.

Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host adaptive immune response by virus; Inhibition of host tapasin/TAPBP by virus.

Inhibition of MHC class I molecule presentation by virus.
Viral protein involved in the evasion of host adaptive immune response by inhibiting the presentation of loaded MHC class I molecules at the cell surface. Many viruses intercept the loaded MHC class I molecules and retain them in the endoplasmic reticulum or target them to degradation in order to prevent presentation of the peptides at the cell surface.

Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host adaptive immune response by virus; Inhibition of MHC class I molecule presentation by virus.

Inhibition of MHC class II molecule presentation by virus.
Viral protein involved in the evasion of host adaptive immune response by inhibiting the presentation of loaded MHC class II molecules at the cell surface. MHC class II molecules are found only on a few specialized cells termed professional antigen-presenting cells (APCs). This group includes macrophages, dendritic cells and B-cells. Many
DE viruses intercept the loaded MHC class II molecules and retain them in the endoplasmic reticulum or target them to degradation in order to prevent presentation of the peptides at the cell surface.

HI Biological process: Host-virus interaction; Viral immunoevasion; Inhibition of host adaptive immune response by virus; Inhibition of MHC class II molecule presentation by virus.

CA Biological process.

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ID Inhibition of host autophagy by virus.
AC KW-XXXX
DE Viral protein involved in the inhibition of host autophagy. Autophagy is a major intracellular pathway in the delivery of cytoplasmic material to lysosomes for degradation. It is also essential for the removal of pathogenic protein aggregates from the cell during infection. Several viruses including influenza and HIV-1 block autophagosome maturation by interacting with and inhibiting host Beclin-1, an essential protein playing a central role in autophagy.

HI Biological process: Host-virus interaction; Inhibition of host autophagy by virus.
CA Biological process.

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ID Activation of host autophagy by virus.
AC KW-XXXX
DE Viral protein involved in the activation of host autophagy. Autophagy is a major intracellular pathway in the delivery of cytoplasmic material to lysosomes for degradation. It is also essential for the removal of pathogenic protein aggregates from the cell during infection. Although autophagy is clearly important for antiviral immune response, it can also be activated by viruses and serves as a platform for viral replication. Some viruses such as poliovirus, use the autophagic pathway as a nonlytic mechanism for viral release.

HI Biological process: Host-virus interaction; Activation of host autophagy by virus.
CA Biological process.

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ID Modulation of dendritic cell activity by virus.
AC KW-XXXX
DE Viral protein involved in the modulation of host dendritic cell activity. Dendritic cells operate at the interface between the innate and adaptive immune response by their ability to sample their environment for pathogenic products, to process them, and to present viral antigens to T-cells. This results in T cell proliferation and the induction of virus-specific adaptive immune responses. Therefore impairing dendritic cell function by viruses is an effective strategy to disrupt the host immune response.

HI Biological process: Host-virus interaction; Viral immunoevasion; Modulation of dendritic cell activity by virus.
CA Biological process.

ID Modulation of NK-cell activity by virus.
AC KW-XXXX
DE Viral protein involved in the modulation of host NK-cell activity.
DE Natural killer (NK) cells are critical in defense against viral
DE infections, since they provide host protection by releasing cytokines
DE such as IFN-gamma or by direct lysis of infected targets. Therefore,
DE during viral infections, viruses and NK cells are in a constant battle
DE and many viruses have developed a variety of strategies to modulate NK
DE cell activity.
HI Biological process: Host-virus interaction; Viral immunoevasion;
Modulation of NK-cell activity by virus.
CA Biological process.

ID Modulation of host cell cycle by virus.
AC KW-XXXX
DE Viral protein involved in the modulation of host cell cycle. The cell
DE cycle can be divided into four stages: G1, S, G2 and mitosis, while
DE cells resting are termed quiescent cells (G0). Viruses have evolved
DE strategies to modulate cell cycle progression including stimulation of
DE S phase entry from G1 or G0 or cell cycle arrest at G2/M for example.
DE This regulation allows viruses to maximize their own replication.
HI Biological process: Host-virus interaction; Modulation of host cell
cycle by virus.
CA Biological process.

ID Host G2/M cell cycle arrest by virus.
AC KW-XXXX
DE Viral protein involved in the modulation of host cell cycle by
DE inhibiting the G2/M transition. A variety of viruses have been
DE associated with G2/M arrest, including some DNA viruses, some RNA
DE viruses and retroviruses but the mechanisms by which arrest is
DE achieved greatly differs between those viruses.
HI Biological process: Host-virus interaction; Modulation of host cell
cycle by virus; Host G2/M cell cycle arrest by virus.
CA Biological process.

ID Inhibition of host mitotic exit by virus.
AC KW-XXXX
DE Viral protein involved in the inhibition of host cell cycle
DE progression by preventing cells to exit mitosis.
HI Biological process: Host-virus interaction; Modulation of host cell
cycle by virus; Inhibition of host mitotic exit by virus.
CA Biological process.
**ID**  G1/S cell checkpoint dysregulation by virus.
**AC**  KW-XXXX
**DE**  Viral protein involved in the modulation of host cell cycle
dysregulation by dysregulating the G1/S transition. Some viruses benefit
from an arrest in G1 to S phase transition, while others force through
S phase to favor their own replication.
**HI**  Biological process: Host-virus interaction; Modulation of host cell
cycle by virus; G1/S cell checkpoint dysregulation by virus.
**CA**  Biological process.

**ID**  Modulation of host cell cycle by viral cyclin-like protein.
**AC**  KW-XXXX
**DE**  Viral protein sharing sequence homology with cellular cyclins. Most
dehomologues are closely related in sequence to the
cellular D-type cyclins, which are implicated in regulating the
transit of cells from G1 into S and are thought to operate via the
inactivation of the retinoblastoma tumour suppressor protein.
**HI**  Biological process: Host-virus interaction; Modulation of host cell
cycle by virus; Modulation of host cell cycle by viral cyclin-like protein.
**CA**  Biological process.

**ID**  G0/G1 cell checkpoint dysregulation by virus.
**AC**  KW-XXXX
**DE**  Viral protein involved in the modulation of host cell cycle
dysregulation by dysregulating the G0/G1 transition. Some viruses
benefit from keeping cells in resting state (G0), while others favor
entry through G1 and subsequent cell division to replicate more
efficiently.
**HI**  Biological process: Host-virus interaction; Modulation of host cell
cycle by virus; G0/G1 cell checkpoint dysregulation by virus.
**CA**  Biological process.

**ID**  Virus-mediated host mRNA decay by hyperadenylation.
**AC**  KW-XXXX
**DE**  Viral protein involved in the degradation of host mRNA by
hyperadenylation. Viruses have evolved ways of interacting with the
cellular RNA decay machinery to favor their survival and maximize the
expression of their own mRNAs. Proper 3' end formation and
polyadenylation are required for mRNA export to the cytoplasm.
Therefore, hyperadenylation by viruses triggers host mRNA nuclear
degradation by quality control pathways.
**HI**  Biological process: Host-virus interaction; Virus-mediated host mRNA
decay by hyperadenylation.
**CA**  Biological process.
ID Inhibition of host mRNA nuclear export by virus.
AC KW-XXXX
DE Viral protein involved in the disruption of the mRNA nuclear export machinery. Viruses have evolved ways of interacting with the nuclear export machinery to inhibit host translation. This global inhibition of cellular protein synthesis serves to ensure maximal viral gene expression and to evade host immune response.
HI Biological process: Host-virus interaction; Inhibition of host mRNA nuclear export by virus.
CA Biological process.

ID Inhibition of host pre-mRNA processing by virus.
AC KW-XXXX
DE Viral protein involved in the disruption of host pre-mRNA processing. Viruses have evolved ways of interacting with the host cell RNA splicing machinery and regulate splicing of cellular pre-mRNAs as a part of the mechanism for shutting down the synthesis of host proteins.
HI Biological process: Host-virus interaction; Inhibition of host pre-mRNA processing by virus.
CA Biological process.

ID Inhibition of host transcription initiation by virus.
AC KW-XXXX
DE Viral protein involved in the disruption of the host transcriptional machinery. Viruses have evolved ways of interacting with the host preinitiation complex (PIC) to shutoff host transcription initiation. For example, the TATA binding protein and TFIIH are targeted by some viral proteins and thus cannot assemble properly to form a functional PIC.
HI Biological process: Host-virus interaction; Inhibition of host transcription initiation by virus.
CA Biological process.

ID Inhibition of host RNA polymerase II by virus.
AC KW-XXXX
DE Viral protein involved in the disruption of the host RNA polymerase II. Many viruses induce alterations in the host cell gene expression. Among these, shutoff of host transcription by targeting RNA polymerase II is commonly used. Indeed, many viruses are able to modify RNAP II CTD including Herpes virus, HIV, Epstein-Barr virus or Bunyamwera virus.
HI Biological process: Host-virus interaction; Inhibition of host RNA polymerase II by virus.
CA Biological process.
ID Modulation of host chromatin by virus.
AC KW-XXXX
DE Viral protein involved in the regulation of host chromatin structure.
DE Chromatin has a major role in life cycle of many viruses, and a lot of
DE them have evolved mechanisms to modulate chromatin-related processes.
DE For example, histone acetyltransferases, histone deacetylases or
DE histones are common targets of viruses.
HI Biological process: Host-virus interaction; Modulation of host
chromatin by virus.
CA Biological process.

ID Cleavage of host translation initiation factors by virus.
AC KW-XXXX
DE Viral protein responsible for the cleavage of host translation
DE initiation factor(s). Viruses have evolved ways of interacting with
DE the host translational machinery to shutoff host gene expression
DE without affecting viral translation.
HI Biological process: Host-virus interaction; Cleavage of host
translation initiation factors by virus.
CA Biological process.

ID Dephosphorylation of host translation initiation factors by virus.
AC KW-XXXX
DE Viral protein responsible for the dephosphorylation of host
DE translation initiation factor(s). Viruses have evolved strategies to
DE rapidly inhibit protein synthesis from host mRNA and, at the same
DE time, promote protein synthesis from its own mRNA.
HI Biological process: Host-virus interaction; Dephosphorylation of host
translation initiation factors by virus.
CA Biological process.

ID Inhibition of host PABPC1 protein by virus.
AC KW-XXXX
DE Viral protein involved in the inhibition of host translation by
DE inhibiting the poly(A)-binding protein. Many viruses target the host
DE translational machinery either to evade cellular defense mechanisms or
DE to subvert the host translational machinery. One common target is the
DE translation initiation factor PABPC1. For example, picornavirus viral
DE proteases are able to cleave PABPC1, while rotavirus displace PABPC1 from
DE EIF4G1.
HI Biological process: Host-virus interaction; Inhibition of PABPC1
protein by virus.
CA Biological process.
ID   Modulation of host ubiquitin pathway by virus.
AC   KW-XXXX
DE   Viral protein involved in the modulation of the host ubiquitin
DE   pathway. The ubiquitination pathway comprises E1, E2, and E3 ligases
DE   that conjugate ubiquitin to protein substrate. Usually, the host E3
DE   ligase determines the substrate specificity. Some viruses encode E3
DE   ligases that modulate the substrate specificity of host E3 ligases.
DE   Alternatively, some viruses encode deubiquitinases able to remove
DE   ubiquitin or ubiquitin-like proteins from their substrate.
HI   Biological process: Host-virus interaction; Modulation of host
HI   ubiquitin pathway by virus.
CA   Biological process.

ID   Modulation of host ubiquitin pathway by viral E3 ligase.
AC   KW-XXXX
DE   Viral protein functioning as a cellular E3 ubiquitin ligase. These
DE   viral
DE   proteins usually target several host proteins for proteasomal
DE   degradation.
HI   Biological process: Host-virus interaction; Modulation of host
HI   ubiquitin pathway by virus; Modulation of host ubiquitin pathway by viral
HI   E3 ligase.
CA   Biological process.

ID   Modulation of host E3 ubiquitin ligases by virus.
AC   KW-XXXX
DE   Viral protein involved in the modulation of cellular E3 ubiquitin
DE   ligases. In general, viral proteins redirect cellular E3 ubiquitin
DE   ligases to select specific host proteins for proteasomal degradation.
DE   The aim of this subversion is the creation of a favorable environment
DE   for virus replication and dissemination.
HI   Biological process: Host-virus interaction; Modulation of host
HI   ubiquitin pathway by virus; Modulation of host E3 ubiquitin ligases by
HI   virus.
CA   Biological process.

ID   Modulation of host ubiquitin pathway by viral ubiquitin-like protein.
AC   KW-XXXX
DE   Viral protein sharing sequence similarity with host ubiquitin. Several
DE   of these homologues are present in large DNA viruses such as
DE   entomopoxvirus or canarypoxvirus and are thought to modulate host
DE   ubiquitin pathway.
HI   Biological process: Host-virus interaction; Modulation of host
HI   ubiquitin pathway by virus; Modulation of host ubiquitin pathway by viral
HI   ubiquitin-like protein.
CA Biological process.

ID Modulation of host ubiquitin pathway by viral deubiquitinase.
AC KW-XXXX
DE Viral protein possessing deubiquitinating activity. Hijacking the
DE ubiquitin system plays an essential role during viral replication.
DE Therefore, several viruses including EBV or HCMV encode for proteins
DE able to remove ubiquitin or ubiquitin-like proteins from their
DE substrate.
HI Biological process: Host-virus interaction; Modulation of host
ubiquitin pathway by virus; Modulation of host ubiquitin pathway by viral
deubiquitinase.
CA Biological process.

ID Modulation of host cell apoptosis by virus.
AC KW-XXXX
DE Viral protein involved in the modulation of host cell apoptosis by
DE acting different steps of the process. Several viruses encode proteins
DE that inhibit apoptosis while other viruses use apoptosis to their
DE advantage to suppress immune response or to disseminate.
HI Biological process: Host-virus interaction; Modulation of host cell
apoptosis by virus.
CA Biological process.

ID Activation of host caspases by virus.
AC KW-XXXX
DE Viral protein involved in the activation of host cell apoptosis by
DE acting on host caspases. While many viruses encode protein that
DE inhibit apoptosis, viruses can also use apoptosis to their advantage
DE to suppress immune response or to disseminate. Therefore, some viral
DE proteins are able to cleave or activate caspases in order to promote
DE apoptosis.
HI Biological process: Host-virus interaction; Modulation of host cell
apoptosis by virus; Activation of host caspases by virus.
CA Biological process.

ID Inhibition of host caspases by virus.
AC KW-XXXX
DE Viral protein involved in the evasion of host cell apoptosis by
DE inhibiting host caspases. Many viruses from diverse families have
DE evolved mechanisms to evade or delay cell death by suppressing the
DE activity of cytoplasmic proteases termed caspases which have a central
DE role in apoptosis induction.
HI Biological process: Host-virus interaction; Modulation of host cell
apoptosis by virus; Inhibition of host caspases by virus.
CA Biological process.
ID   Inhibition of host apoptosis by viral FLIP-like protein.
AC   KW-XXXX
DE   Viral protein sharing sequence similarity with host FLIPS (FLICE-inhibitory proteins). Cellular FLIPs play an essential role in apoptosis functioning as a link between cell survival and cell death pathways. Viral FLIPs inhibit apoptosis by interfering with death receptor signaling.
HI   Biological process: Host-virus interaction; Modulation of host cell apoptosis by virus; Inhibition of host apoptosis by viral FLIP-like protein.
CA   Biological process.

ID   Inhibition of host apoptosis by viral BCL2-like protein.
AC   KW-XXXX
DE   Viral protein sharing sequence similarity with host BCL2 protein. Cellular BCL2 family members are divided in two groups, some having anti-apoptotic activity (such as BCL2 itself) while others have pro-apoptotic function (such as BAX). If the level of proapoptotic members are higher than inhibitors, then the cell undergoes apoptosis. So far, all viral homologues display anti-apoptotic activity.
HI   Biological process: Host-virus interaction; Modulation of host cell apoptosis by virus; Inhibition of host apoptosis by viral BCL2-like protein.
CA   Biological process.