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


ID   Inhibition of interferon signaling pathway by virus.
AC   KW-XXXX
DE   Viral protein involved in the evasion of host innate defense by
DE   inhibiting the interferon signaling pathway leading to the production
DE   of interferon-induced genes. Interferons bind to the IFN receptors
DE   (IFNAR) on the cell surface and activate Jak/Tyk kinases. These
DE   kinases phosphorylate STAT1 and STAT2 that translocate to the nucleus
DE   and induce the expression of interferon stimulated genes (ISGs). Many
DE   viruses interact with components of this pathway to prevent expression
DE   of ISGs and inhibit the host immune response.
HI   Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host innate immune response by virus; Inhibition of
interferon signaling pathway by virus.
CA   Biological process.


ID   Inhibition of host innate immune response by virus.
AC   KW-XXXX
DE   Viral protein involved in the evasion of host innate immune response.
DE   Upon viral infection, the innate immune system initially defends the
DE   host in a non-specific manner. Many viral proteins interact with and
DE   inhibit components the host innate system to replicate more
DE   efficiently.
HI   Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host innate immune response by virus.
CA   Biological process.


ID   Inhibition of host adaptive immune response by virus.
AC   KW-XXXX
DE   Viral protein involved in the evasion of host adaptive immune
DE   response. Upon infection, the innate immune system provides mechanisms
DE   for the rapid sensing and elimination of viruses. Adaptive immunity
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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
DE inhibiting the MAVS protein. During viral replication, dsRNA is
DE produced and detected by DDX58/RIG-I or IFIH1/MDA5 that will activate
DE MAVS to coordinate pathways leading to induction of antiviral
DE cytokines. Several viral proteins including NS3/4A from Hepatitis C,
DE or protease 3C from hepatitis A virus, cleave MAVS to abrogate 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 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
DE inhibiting the interferon regulatory factor-3 (IRF3) protein. Viral
DE infection triggers the phosphorylation and activation of IRF3. The
DE activated IRF3 migrates to the nucleus, where it complexes with the
DE transcription coactivator CREBBP/EP300, leading to the transcriptional
DE activation of the IFN-alpha and IFN-beta genes. Several viral proteins
DE directly bind to IRF3 and inhibit its transcriptional activity while
DE 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
DE inhibiting the interferon regulatory factor-7 (IRF7) protein. Viral
DE infection triggers the phosphorylation and activation of IRF7. The
DE activated IRF7 migrates to the nucleus leading to the transcriptional
DE activation of the IFN-alpha and IFN-beta genes. Some viral proteins
DE prevent IRF7 phosphorylation and nuclear activation. Ebola virus VP35
DE interacts with IRF7 and hijacks the cellular SUMOylation machinery for
DE 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
DE   pathway by inhibiting the interferon regulatory factor-9 (IRF9)
DE   protein. Viral infection triggers the phosphorylation and activation
DE   of IRF9. The activated IRF9 migrates to the nucleus leading to the
DE   transcriptional activation of several hundred IFN-responsive genes.
DE   Some viral proteins inhibit IRF9 activation by preventing its nuclear
DE   localization upon infection or by sending it to the nucleus in an
DE   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
DE   inhibiting the TBK1/IKBKE/DDX3 complex. Upon infection, the virus is
DE   recognized and the signal is transmitted to TBK1 and IKBKE that in
DE   turn phosphorylate and activate IRF3 and IRF7. Once phosphorylated,
DE   IRF3 and IRF7 homodimerize and translocate into the nucleus to drive
DE   transcription of interferons. Several viruses including Ebolavirus and
DE   Bornavirus interact directly with and inhibit TBK1 to prevent IRFs
DE   activation. Other viruses such as vaccinia virus inhibit host DDX3 to
DE   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
DE   interferon pathways by inhibiting the STAT1 protein. Upon viral
DE   infection, STAT1 is activated by IFN-gamma, IFN-alpha/beta, or IFN-
DE   lambda that bind to specific cell surface receptors. While IFN-gamma
DE   induces STAT1 homodimerization, IFN-alpha/beta and IFN-lambda
DE   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-
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 processed
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-
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
DE   pathways by inhibiting the host TYK2 protein. Upon viral infection,
DE   the TYK2 protein is activated by IFNalpha/beta or IFN-lambda
DE   stimulation leading to a series of phosphorylation events that induce
DE   transcription of several hundred IFN-responsive genes. Several viruses
DE   have evolved mechanisms to inhibit TYK2 activity thereby preventing
DE   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
DE   interferon pathways by inhibiting the JAK1 protein. Upon viral
DE   infection, JAK1 is activated by the interferon-alpha/beta, -gamma, and
DE   -lambda signal transduction pathways. Several viral proteins can
DE   directly interact with JAK1 to prevent its ability to phosphorylate
DE   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
DE   inhibiting the ISG15 protein, an ubiquitin-like modifier playing
DE   important roles in the innate immune response. Like ubiquitin, ISG15
DE   is conjugated to lysines on numerous target proteins through its
DE   conserved C-terminal region. Viruses escape from the antiviral
DE   activity of ISG15 by direct interaction or by cleavage of ISG15
DE   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

DE   inhibiting interferon receptors. Interferon signaling exerts antiviral
DE   effects through cell surface receptors termed interferon receptors. In
DE   response to binding of extracellular interferons, they activate the
DE   JAK/STAT pathway causing transcriptional activation of IFN-regulated
DE   genes. To avoid this antiviral response, several viruses target the
DE   interferon receptors and send them to degradation via the proteasome.
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 interferon
receptors by virus.
CA   Biological process.

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

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

AC   KW-XXXX
DE   Viral protein acting as an IgG Fc receptors, able to bind IgG and
DE   inhibit host Fc-dependent immune activation. Fc receptors are proteins
DE   found at the surface of certain cells of the immune system including
DE   macrophages, monocytes, natural killer cells or B-cells. They allow
DE   these cells to bind to antibodies that are attached to the surface of
DE   infected cells or pathogens, helping these cells to identify and
DE   eliminate pathogens.
HI   Biological process: Host-virus interaction; Viral immunoevasion;
Modulation of host immune response by viral IgG Fc receptor-like protein.
CA   Biological process.

ID   Modulation of host immune response by viral interleukin-like protein.
AC   KW-XXXX
DE   Viral protein sharing sequence homology with host interleukins.
DE   Interleukins are produced by immune system cells such as lymphocytes,
DE   macrophages and monocytes, and modulate inflammation and immunity by
DE   regulating growth, mobility and differentiation of lymphoid and other
DE   cells. Several viruses encode interleukin-like proteins playing a role
DE   in immune evasion. Additionally, viral interleukins have been shown to
DE   activate cellular signaling cascades that enhance virus replication.
HI   Biological process: Host-virus interaction; Viral immunoevasion;
Modulation of host immune response by viral interleukin-like protein.
CA   Biological process.

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

ID   Inhibition of host TAP by virus.
AC   KW-XXXX
DE   Viral protein involved in the evasion of host adaptive immune response
by
DE   inhibiting the TAP complex. Transporter associated with antigen (TAP),

DE   composed of two subunits TAP1 and TAP2, is required for the
DE   translocation of peptides into the ER, where they are loaded onto MHC
DE   class I. Thereafter, the viral peptides are presented to cytotoxic T
DE   lymphocytes at the cell surface and trigger immune response. The
DE   loading of peptide on MHC by TAP is targeted by several viruses
DE   including herpesviruses and retroviruses.
HI   Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host adaptive immune response by virus; Inhibition of host
TAP by virus.
CA   Biological process.

ID   Inhibition of host tapasin/TAPBP by virus.
AC   KW-XXXX
DE   Viral protein involved in the evasion of host adaptive immune response
DE   by inhibiting the tapasin/TAPBP protein. tapasin/TAPBP is a type I
DE   transmembrane protein essential for the optimal expression of stable
DE   MHC class I molecules on host cell surface. Its helps the MHC class I
DE   molecules to remain in a peptide receptive state, avoiding
DE   irreversible denaturation. Several retroviruses and DNA viruses encode
DE   proteins interacting with tapasin/TAPBP and inhibiting its activity.
HI   Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host adaptive immune response by virus; Inhibition of host
tapasin/TAPBP by virus.
CA   Biological process.

ID   Inhibition of MHC class I molecule presentation by virus.
AC   KW-XXXX
DE   Viral protein involved in the evasion of host adaptive immune response
by
DE   inhibiting the presentation of loaded MHC class I molecules at the
DE   cell surface. Many viruses ìinterceptî the loaded MHC class I
DE   molecules and retain them in the endoplasmic reticulum or target them
DE   to degradation in order to prevent presentation of the peptides at the
DE   cell surface.
HI   Biological process: Host-virus interaction; Viral immunoevasion;
Inhibition of host adaptive immune response by virus; Inhibition of MHC
class I molecule presentation by virus.
CA   Biological process.

ID   Inhibition of MHC class II molecule presentation by virus.
AC   KW-XXXX
DE   Viral protein involved in the evasion of host adaptive immune response
by
DE   inhibiting the presentation of loaded MHC class II molecules at the
DE   cell surface. MHC class II molecules are found only on a few
DE   specialized cells termed professional antigen-presenting cells (APCs).
DE   This group includes macrophages, dendritic cells and B-cells. Many

DE   viruses ìinterceptî the loaded MHC class II molecules and retain them
DE   in the endoplasmic reticulum or target them to degradation in order to
DE   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.

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

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

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

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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.
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ID   G1/S cell checkpoint dysregulation by virus.
AC   KW-XXXX
DE   Viral protein involved in the modulation of host cell cycle
DE   progression by dysregulating the G1/S transition. Some viruses benefit
DE   from an arrest in G1 to S phase transition, while others force through
DE   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
DE   viral cyclin homologues are closely related in sequence to the
DE   cellular D-type cyclins, which are implicated in regulating the
DE   transit of cells from G1 into S and are thought to operate via the
DE   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
DE   progression by dysregulating the G0/G1 transition. Some viruses
DE   benefit from keeping cells in resting state (G0), while others favor
DE   entry through G1 and subsequent cell division to replicate more
DE   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
DE   hyperadenylation. Viruses have evolved ways of interacting with the
DE   cellular RNA decay machinery to favor their survival and maximize the
DE   expression of their own mRNAs. Proper 3' end formation and
DE   polyadenylation are required for mRNA export to the cytoplasm.
DE   Therefore, hyperadenylation by viruses triggers host mRNA nuclear
DE   retention and subsequent degradation by quality control pathways.
HI   Biological process: Host-virus interaction; Virus-mediated host mRNA
decay by hyperadenylation.
CA   Biological process.
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ID   Inhibition of host mRNA nuclear export by virus.
AC   KW-XXXX
DE   Viral protein involved in the disruption of the mRNA nuclear export
DE   machinery. Viruses have evolved ways of interacting with the nuclear
DE   export machinery to inhibit host translation. This global inhibition
DE   of cellular protein synthesis serves to ensure maximal viral gene
DE   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.
DE   Viruses have evolved ways of interacting with the host cell RNA
DE   splicing machinery and regulate splicing of cellular pre-mRNAs as a
DE   part of the mechanism for shutting down the synthesis of host
DE   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
DE   machinery. Viruses have evolved ways of interacting with the host
DE   preinitiation complex (PIC) to shutoff host transcription initiation.
DE   For example, the TATA binding protein and TFIIH are targeted by some
DE   viral proteins and thus cannot assemble properly to form a functional
DE   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
DE   II. Many viruses induce alterations in the host cell gene expression.
DE   Among these, shutoff of host transcription by targeting RNA polymerase
DE   II is commonly used. Indeed, many viruses are able to modify RNAP II
DE   CTD including Herpes virus, HIV, Epstein-Barr virus or Bunyamwera
DE   virus.
HI   Biological process: Host-virus interaction; Inhibition of host RNA
polymerase II by virus.
CA   Biological process.
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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.
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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
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
viral
DE   proteins usually target several host proteins for proteasomal
DE   degradation.
HI   Biological process: Host-virus interaction; Modulation of host
ubiquitin pathway by virus; Modulation of host ubiquitin pathway by viral
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
ubiquitin pathway by virus; Modulation of host E3 ubiquitin ligases by
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
ubiquitin pathway by virus; Modulation of host ubiquitin pathway by viral
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-
DE   inhibitory proteins). Cellular FLIPs play an essential role in
DE   apoptosis functioning as a link between cell survival and cell death
DE   pathways . Viral FLIPs inhibit apoptosis by interfering with death
DE   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.
DE   Cellular BCL2 family members are divided in two groups, some having
DE   anti-apoptotic activity (such as BCL2 itself) while others have pro-
DE   apoptotic function (such as BAX). If the level of proapoptotic members
DE   are higher than inhibitors, then the cell undergoes apoptosis. So far,
DE   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.