Artemis (Phospho Ser516) Rabbit pAb

CatalogNo: YP0643 Orthogonal Validated

Key Features

Host Species
- Rabbit

Reactivity
- Human,Mouse

Applications
- WB,IHC,IF,ELISA

MW
- 78kD (Observed)

Isotype
- IgG

Recommended Dilution Ratios

WB 1:500-1:2000
IHC 1:100-1:300
ELISA 1:5000
IF 1:50-200

Storage

Storage*	-15°C to -25°C/1 year(Do not lower than -25°C)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	The antiserum was produced against synthesized peptide derived from human Artemis around the phosphorylation site of Ser516. AA range:482-531

Specificity	Phospho-Artemis (S516) Polyclonal Antibody detects endogenous levels of Artemis protein only when phosphorylated at S516.The name of modified sites may be influenced by many factors, such as species (the modified site was not originally found in human samples) and the change of protein sequence (the previous protein sequence is incomplete, and the protein sequence may be prolonged with the development of protein sequencing technology). When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports. The antibody binds to the following modification sequence (lowercase letters are modification sites):GGsQS

Target Information

Gene name	DCLRE1C
Protein Name	Protein artemis

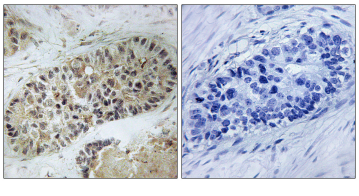
Organism	Gene ID	UniProt ID
Human	64421;	Q96SD1;
Mouse	227525;	Q8K4J0;

Cellular Localization	Nucleus .
Tissue specificity	Ubiquitously expressed, with highest levels in the kidney, lung, pancreas and placenta (at the mRNA level). Expression is not increased in thymus or bone marrow, sites of V(D)J recombination.

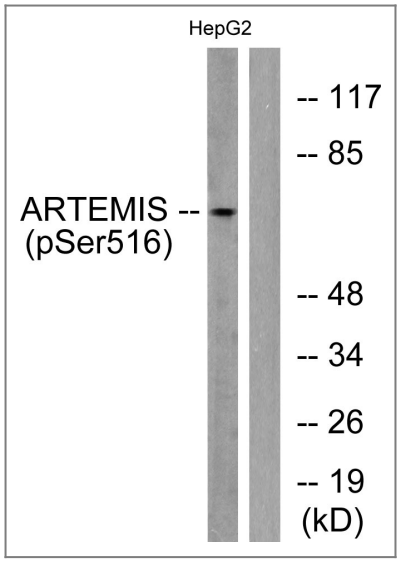
Function

Disease:Defects in DCLRE1C are a cause of Omenn syndrome (OS) [MIM:603554]. OS is characterized by severe combined immunodeficiency associated with erythrodermia, hepatosplenomegaly, lymphadenopathy and alopecia. Affected individuals have elevated T-lymphocyte counts with a restricted T-cell receptor (TCR) repertoire. They also generally lack B-lymphocytes, but have normal natural killer (NK) cell function (T+ B- NK+).,Disease:Defects in DCLRE1C are a cause of severe combined immunodeficiency autosomal recessive T-cell-negative/B-cell-negative/NK-cell-positive with sensitivity to ionizing radiation (RSSCID) [MIM:602450]. SCID refers to a genetically and clinically heterogeneous group of rare congenital disorders characterized by impairment of both humoral and cell-mediated immunity, leukopenia, and low or absent antibody levels. Patients with SCID present in infancy with recurrent, persistent infections by opportunistic organisms. The common characteristic of all types of SCID is absence of T-cell-mediated cellular immunity due to a defect in T-cell development. Individuals affected by RS-SCID show defects in the DNA repair machinery necessary for coding joint formation and the completion of V(D)J recombination. A subset of cells from such patients show increased radiosensitivity.,Disease:Defects in DCLRE1C are the cause of severe combined immunodeficiency Athabaskan type (SCIDA) [MIM:602450]. SCIDA is a variety of RS-SCID caused by a founder mutation in Athabascan-speaking native Americans, being inherited as an autosomal recessive trait with an estimated gene frequency of 2.1% in the Navajo population. Affected individuals exhibit clinical symptoms and defects in DNA repair comparable to those seen in RS-SCID.,Function:Required for V(D)J recombination, the process by which exons encoding the antigen-binding domains of immunoglobulins and T-cell receptor proteins are assembled from individual V, (D), and J gene segments. V(D)J recombination is initiated by the lymphoid specific RAG endonuclease complex, which generates site specific DNA double strand breaks (DSBs). These DSBs present two types of DNA end structures: hairpin sealed coding ends and phosphorylated blunt signal ends. These ends are independently repaired by the non homologous end joining (NHEJ) pathway to form coding and signal joints respectively. This protein exhibits single-strand specific 5'-3' exonuclease activity in isolation and acquires endonucleolytic activity on 5' and 3' hairpins and overhangs when in a complex with PRKDC. The latter activity is required specifically for the resolution of closed hairpins prior to the formation of the coding joint. May also be required for the repair of complex DSBs induced by ionizing radiation, which require substantial end-processing prior to religation by NHEJ.,online information:DCLRE1C mutation db,PTM:Phosphorylation on undefined residues by PRKDC may stimulate endonucleolytic activity on 5' and 3' hairpins and overhangs. PRKDC must remain present, even after phosphorylation, for efficient hairpin opening. Also phosphorylated by ATM in response to ionizing radiation (IR) and by ATR in response to ultraviolet (UV) radiation.,similarity:Belongs to the DNA repair metallo-beta-lactamase (DRMBL) family.,subunit:Interacts with ATM, BRCA1, PRKDC and TP53BP1. Also exhibits ATM- and phosphorylation-dependent interaction with the MRN complex, composed of MRE11A/MRE11, RAD50, and NBN.,tissue specificity:Ubiquitously expressed, with highest levels in the kidney, lung, pancreas and placenta (at the mRNA level). Expression is not increased in thymus or bone marrow, sites of V(D)J recombination.,

Validation Data



Immunohistochemistry analysis of paraffin-embedded human lung carcinoma, using Artemis (Phospho-Ser516) Antibody. The picture on the right is blocked with the phospho peptide.



Western blot analysis of lysates from HepG2 cells treated with EGF 200ng/ml 30', using Artemis (Phospho-Ser516) Antibody. The lane on the right is blocked with the phospho peptide.

Contact information

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Please scan the QR code to access additional product information: **Artemis (Phospho Ser516) Rabbit pAb**