
Product Name: Artemis Rabbit Polyclonal Antibody**Catalog #: APRab07177**

For research use only.

Summary

Description	Rabbit polyclonal Antibody
Host	Rabbit
Application	IHC, ICC/IF, ELISA
Reactivity	Human, Rat, Mouse
Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Concentration	1mg/ml
Storage	Aliquot and store at -20°C (valid for 12 months). Avoid freeze/thaw cycles.
Shipping	Ice bags
Buffer	Liquid in PBS containing 50% glycerol, 0.5% protective protein and 0.02% New type preservative N.
Purification	Affinity purification

Application

Dilution Ratio IHC 1:100-1:300, ICC/IF 1:200-1:1000, ELISA 1:5000-1:20000

Molecular Weight

Antigen Information

Gene Name	DCLRE1C
Alternative Names	DCLRE1C; ARTEMIS; ASCID; SCIDA; SNM1C; Protein artemis; DNA cross-link repair 1C protein; Protein A-SCID; SNM1 homolog C; hSNM1C; SNM1-like protein
Gene ID	64421.0
SwissProt ID	Q96SD1
Immunogen	The antiserum was produced against synthesized peptide derived from human Artemis. AA range:482-531

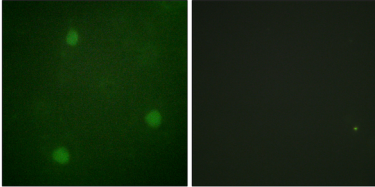
Background

This gene encodes a nuclear protein that is involved in V(D)J recombination and DNA repair. The encoded protein has single-strand-specific 5'-3' exonuclease activity; it also exhibits endonuclease activity on 5' and 3' overhangs and hairpins. The protein also functions in the regulation of the cell cycle in response to DNA damage. Mutations in this gene can cause Athabaskan-type severe combined immunodeficiency (SCIDA) and Omenn syndrome. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Jan 2014],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 Athabaskan-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.,

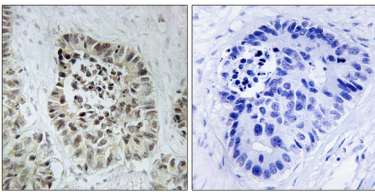
Research Area

Non-homologous end-joining;Primary immunodeficiency;

Image Data



Immunofluorescence analysis of NIH/3T3 cells, using Artemis Antibody. The picture on the right is blocked with the synthesized peptide.



Immunohistochemistry analysis of paraffin-embedded human lung carcinoma tissue, using Artemis Antibody. The picture on the right is blocked with the synthesized peptide.