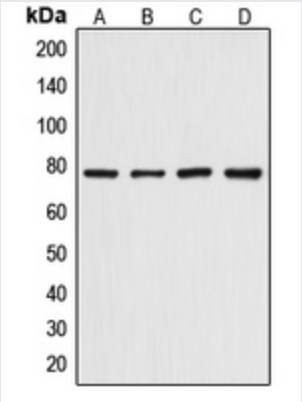
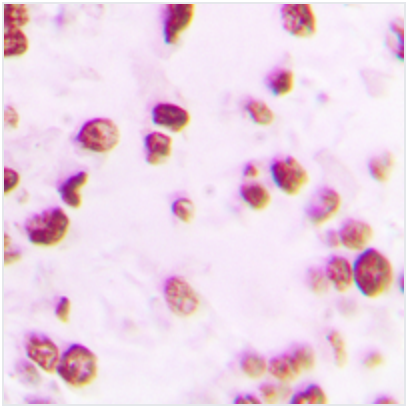
DCLRE1C Antibody – Rabbit Polyclonal

PRODUCT OVERVIEW

Product Name	Artemis (DCLRE1C), Polyclonal Antibody
Full Product Name	Rabbit anti-Artemis (pS516) Antibody
Product Gene Name	anti-DCLRE1C antibody
Product Synonym Names	ARTEMIS; ASCID; SCIDA; SNM1C; Protein artemis; DNA cross-link repair 1C protein; Protein A-SCID; SNM1 homolog C; hSNM1C; SNM1-like protein
Clonality	Polyclonal
Host	Rabbit
Species Reactivity	Human, Mouse, Rat
Form/Format	Liquid in 0.42% Potassium phosphate, 0.87% Sodium chloride, pH 7.3, 30% glycerol, and 0.01% sodium azide.
Purity/Purification	The antibody was purified by immunogen affinity chromatography.
Concentration	1mg/mL (lot specific)
Preparation and Storage	Store at -20 degree C. Avoid repeated freeze/thaw cycles.
Molecular Weight	78,436 Da
Chromosome Location	Chromosome: 10; NC_000010.10 (14948870..14996094, complement). Location: 10p13
OMIM	602450
3D Structure	ModBase 3D Structure for Q96SD1
Immunogen	KLH-conjugated synthetic peptide encompassing a sequence within the center region of human Artemis. The exact sequence is proprietary.
Product Categories/Family for anti-DCLRE1C antibody	Primary antibodies
ISO Certification	Manufactured in an ISO 13485:2003 and EN ISO 13485:2012 Certified Laboratory.
Applications Tested/Suitable for anti-DCLRE1C antibody	Western Blot (WB), Immunohistochemistry (IHC)
Application Notes for anti-DCLRE1C antibody	WB: 1:500-1:1000 IHC: 1:100-1:200
Related Product Information for anti-DCLRE1C antibody	MBS4509364 is an antibody targeting DCLRE1C. Also known as Rabbit anti-Artemis (pS516) Antibody. Reactive in Human, Mouse, Rat. Host: Rabbit.
Other Notes	Small volumes of anti-DCLRE1C antibody vial(s) may occasionally become entrapped in the seal of the product vial during shipment and storage. If necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the container`s cap. Certain products may require to ship with dry ice and additional dry ice fee may apply.



Western Blot (WB) of anti-DCLRE1C antibody
Western blot analysis of Artemis (pS516) expression in HepG2 (A), HeLa (B), mouse skeletal muscle (C), rat skeletal muscle (D) whole cell lysates.



Immunohistochemistry (IHC) of anti-DCLRE1C antibody
Immunohistochemical analysis of Artemis (pS516) staining in human lung cancer formalin fixed paraffin embedded tissue section. The section was pre-treated using heat mediated antigen retrieval with sodium citrate buffer (pH 6.0). The section was then incubated with the antibody at room temperature and detected using an HRP conjugated compact polymer system. DAB was used as the chromogen. The section was then counterstained with haematoxylin and mounted with DPX.

ADDITIONAL DETAILS

NCBI/Uniprot data below describe general gene information for DCLRE1C. It may not necessarily be applicable to this product.

NCBI GI #	76496497
NCBI GeneID	64421
NCBI Accession #	NP_001029027.1
UniProt Primary Accession #	Q96SD1
UniProt Secondary Accession #	Q8K4J0; Q5XIX3
UniProt Related Accession #	Q96SD1
GenBank Nucleotide #	NM_001033855.1
NCBI Official Full Name	protein artemis isoform a
NCBI Official Synonym Full Names	DNA cross-link repair 1C
NCBI Official Symbol	DCLRE1C
NCBI Official Synonym Symbols	SCIDA; SNM1C; A-SCID; hSNM1C; RS-SCID; DCLREC1C
UniProt Protein Name	Protein artemis
UniProt Synonym Protein Names	DNA cross-link repair 1C protein; Protein A-SCID; SNM1 homolog C; hSNM1C; SNM1-like protein
Protein Family	Protein artemis
UniProt Gene Name	DCLRE1C
UniProt Synonym Gene Names	ARTEMIS; ASCID; SCIDA; SNM1C; hSNM1C
UniProt Entry Name	DCR1C_HUMAN
NCBI Protein Information	protein artemis; PSO2 homolog; SNM1 homolog C; SNM1-like protein; DNA cross-link repair 1C protein; severe combined immunodeficiency, type a (Athabascan); DNA cross-link repair 1C (PSO2 homolog, S. cerevisiae)
NCBI Summary for DCLRE1C	This gene encodes a nuclear protein that is involved in V(D)J recombination and DNA repair. The protein has single-strand-specific 5'-3' exonuclease activity; it also exhibits endonuclease activity on 5' and 3' overhangs and hairpins when complexed with protein kinase, DNA-activated, catalytic polypeptide. Mutations in this gene cause Athabascan-type severe combined immunodeficiency (SCIDA). [provided by RefSeq, Jul 2008]

UniProt Comments for DCLRE1C	Artemis: 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. 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). 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. Defects in DCLRE1C are the cause of severe combined immunodeficiency Athabaskan type (SCIDA). 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. Defects in DCLRE1C are a cause of Omenn syndrome (OS). 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+). Belongs to the DNA repair metallo-beta-lactamase (DRMBL) family. 4 isoforms of the human protein are produced by alternative splicing. Protein type: DNA repair, damage; EC 3.1.-.-; Deoxyribonuclease Chromosomal Location of Human Ortholog: 10p13 Cellular Component: nucleus Molecular Function: 5'-3' exonuclease activity; single-stranded DNA specific endodeoxyribonuclease activity Biological Process: V(D)J recombination; B cell differentiation; double-strand break repair; response to ionizing radiation; telomere maintenance; DNA catabolic process, endonucleolytic Disease: Omenn Syndrome; Severe Combined Immunodeficiency With Sensitivity To Ionizing Radiation
Research Articles on DCLRE1C	**1.** Structural basis of DNA ligase IV-Artemis interaction in nonhomologous end-joining.
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