


Product datasheet

Anti-Artemis antibody ab87271

1 Image

Overview

<b>Product name</b>	Anti-Artemis antibody
<b>Description</b>	Rabbit polyclonal to Artemis
<b>Host species</b>	Rabbit
<b>Tested applications</b>	<b>Suitable for:</b> IHC-P
<b>Species reactivity</b>	<b>Reacts with:</b> Human <b>Predicted to work with:</b> Chimpanzee, Orangutan 
<b>Immunogen</b>	Synthetic peptide, corresponding to a region within C-terminal amino acids 350 - 692 of Human Artemis (TrEMBL entry Q96SD1) (Peptide available as <a href="#">ab113540</a> .)
<b>Positive control</b>	Anaplastic thyroid carcinoma, breast carcinoma, colon carcinoma, prostate carcinoma, stomach adenocarcinoma, testicular seminoma.

Properties

<b>Form</b>	Liquid
<b>Storage instructions</b>	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
<b>Storage buffer</b>	Preservative: 0.09% Sodium Azide Constituents: 0.1% BSA, Tris buffered saline
<b>Purity</b>	Immunogen affinity purified
<b>Clonality</b>	Polyclonal
<b>Isotype</b>	IgG

Applications

Our [Abpromise guarantee](#) covers the use of **ab87271** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
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IHC-P

**Application notes** IHC-P: 1/100 - 1/500. Antigen retrieval with tris-EDTA buffer pH 9.0 recommended.

Likely to work with frozen sections.

Not yet tested in other applications.

Optimal dilutions/concentrations should be determined by the end user.

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## Target

### 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.

### 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.

### Involvement in 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 (RSCID) [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.

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.

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+).

### Sequence similarities

Belongs to the DNA repair metallo-beta-lactamase (DRMBL) family.

### Post-translational modifications

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.

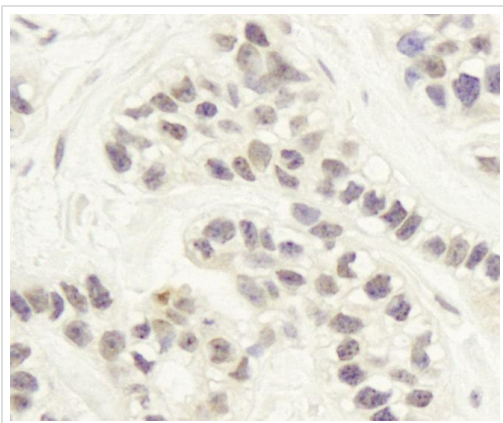
### Cellular localization

Nucleus.

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## Images

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ab87271 at 1/250 dilution staining Artemis in formalin-fixed paraffin-embedded Human breast carcinoma tissue.  
Detection: DAB staining.

Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Artemis antibody (ab87271)

**Please note:** All products are "FOR RESEARCH USE ONLY AND ARE NOT INTENDED FOR DIAGNOSTIC OR THERAPEUTIC USE"

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