abcam

Product datasheet

Anti-AK2 antibody ab71729

2 Images

Overview

Product name	Anti-AK2 antibody
Description	Rabbit polyclonal to AK2
Tested applications	**Suitable for:** IHC-P, WB
Species reactivity	**Reacts with:** Human
Immunogen	KLH conjugated synthetic peptide selected from the C terminal region of human AK2.
Positive control	Jurkat cell lysate and human cancer tissue (hepatocarcinoma)

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.
Storage buffer	Preservative: 0.09% Sodium Azide Constituents: PBS
Purity	Ammonium Sulphate Precipitation
Purification notes	This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Clonality	Polyclonal
Isotype	IgG

Applications

Our Abpromise guarantee covers the use of **ab71729** in the following tested applications.

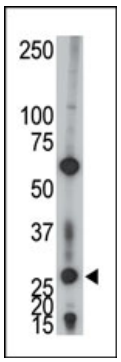
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
IHC-P		1/50 - 1/100.
WB		1/1000. Detects a band of approximately 26 kDa (predicted molecular weight: 26 kDa).

Target

Function	Catalyzes the reversible transfer of the terminal phosphate group between ATP and AMP. This small ubiquitous enzyme involved in energy metabolism and nucleotide synthesis that is essential for maintenance and cell growth. Plays a key role in hematopoiesis.
Tissue specificity	Present in most tissues. Present at high level in heart, liver and kidney, and at low level in brain, skeletal muscle and skin. Present in thrombocytes but not in erythrocytes, which lack mitochondria. Present in all nucleated cell populations from blood, while AK1 is mostly absent. In spleen and lymph nodes, mononuclear cells lack AK1, whereas AK2 is readily detectable. These results indicate that leukocytes may be susceptible to defects caused by the lack of AK2, as they do not express AK1 in sufficient amounts to compensate for the AK2 functional deficits (at protein level).
Involvement in disease	Defects in AK2 are the cause of reticular dysgenesis (RDYS) [MIM:267500]; also known as aleukocytosis. RDYS is the most severe form of inborn severe combined immunodeficiencies (SCID) and is characterized by absence of granulocytes and almost complete deficiency of lymphocytes in peripheral blood, hypoplasia of the thymus and secondary lymphoid organs, and lack of innate and adaptive humoral and cellular immune functions, leading to fatal septicemia within days after birth. In bone marrow of individuals with reticular dysgenesis, myeloid differentiation is blocked at the promyelocytic stage, whereas erythro- and megakaryocytic maturation is generally normal.In addition, affected newborns have bilateral sensorineural deafness. Defects may be due to its absence in leukocytes and inner ear, in which its absence can not be compensated by AK1.
Sequence similarities	Belongs to the adenylate kinase family. AK2 subfamily.
Cellular localization	Mitochondrion intermembrane space.

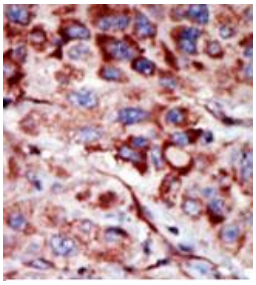
Images



Western blot - AK2 antibody - Carboxyterminal end (ab71729)

Anti-AK2 antibody (ab71729) at 1/100 dilution
+ Jurkat cell lysate at 12.5 µg

Predicted band size : 26 kDa
Observed band size : 26 kDa
Additional bands at : 15 kDa,62 kDa. We are unsure as to the identity of these extra bands.



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - AK2 antibody - Carboxyterminal end (ab71729)

ab71729, at a 1/50 dilution, staining AK2 in formalin fixed, paraffin embedded human cancer tissue (hepatocarcinoma) by Immunohistochemistry. ab71729 was peroxidase conjugated to the secondary antibody, followed by AEC staining.

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