RUNX1 (Phospho Ser276) Rabbit pAb

CatalogNo: YP0622

Key Features

Host Species
- Rabbit

Reactivity
- Human,Mouse,Rat

Applications
- WB,IHC,IF,ELISA

MW
- 55kD (Observed)

Isotype
- IgG

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.

Recommended Dilution Ratios

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

Basic Information

Clonality	Polyclonal

Immunogen Information

Immunogen	The antiserum was produced against synthesized peptide derived from human AML1 around the phosphorylation site of Ser303. AA range:269-318
Specificity	Phospho-RUNX1 (S276) Polyclonal Antibody detects endogenous levels of RUNX1 protein only when phosphorylated at S276.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 When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports.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):PIsPG

Target Information

Gene name RUNX1

Protein Name Runt-related transcription factor 1

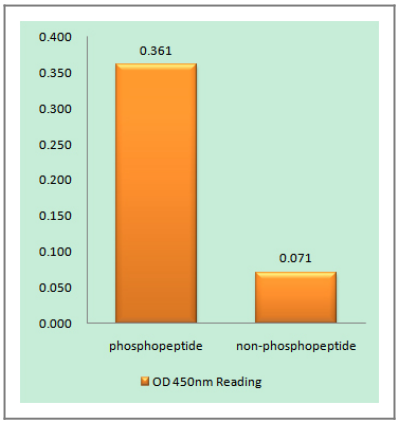
Organism	Gene ID	UniProt ID
Human	861;	Q01196;
Mouse	12394;	Q03347;
Rat	50662;	Q63046;

Cellular Localization Nucleus.

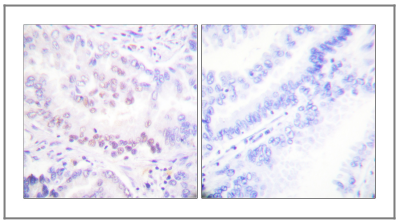
Tissue specificity Expressed in all tissues examined except brain and heart. Highest levels in thymus, bone marrow and peripheral blood.

Function Alternative products:Additional isoforms seem to exist,Caution:The fusion of AML1 with EAP in T-MDS induces a change of reading frame in the latter resulting in 17 AA unrelated to those of EAP.,Disease:A chromosomal aberration involving RUNX1/AML1 is a cause of chronic myelogenous leukemia (CML). Translocation t(3;21)(q26;q22) with EAP, MSD1 or EVI1.,Disease:A chromosomal aberration involving RUNX1/AML1 is a cause of chronic myelomonocytic leukemia. Inversion inv(21)(q21;q22) with USP16.,Disease:A chromosomal aberration involving RUNX1/AML1 is a cause of M2 type acute myeloid leukemia (AML-M2). Translocation t(8;21)(q22;q22) with RUNX1T1/MTG8/ETO.,Disease:A chromosomal aberration involving RUNX1/AML1 is a cause of therapy-related myelodysplastic syndrome (T-MDS). Translocation t(3;21)(q26;q22) with EAP, MSD1 or EVI1.,Disease:A chromosomal aberration involving RUNX1/AML1 is found in childhood acute lymphoblastic leukemia (ALL). Translocation t(12;21)(p13;q22) with TEL. The translocation fuses the 3'-end of TEL to the alternate 5'-exon of AML-1H.,Disease:A chromosomal aberration involving RUNX1/AML1 is found in therapy-related myeloid malignancies. Translocation t(16;21)(q24;q22) that forms a RUNX1-CBFA2T3 fusion protein.,Disease:Defects in RUNX1 are the cause of familial platelet disorder with associated myeloid malignancy (FPDMM) [MIM:601399]. FPDMM is an autosomal dominant disease characterized by qualitative and quantitative platelet defects, and propensity to develop acute myelogenous leukemia.,Domain:A proline/serine/threonine rich region at the C-terminus is necessary for transcriptional activation of target genes.,Function:CBF binds to the core site, 5'-PYGPYGGT-3', of a number of enhancers and promoters, including murine leukemia virus, polyomavirus enhancer, T-cell receptor enhancers, LCK, IL-3 and GM-CSF promoters. The alpha subunit binds DNA and appears to have a role in the development of normal hematopoiesis. Isoform AML-1L interferes with the transactivation activity of RUNX1. Acts synergistically with ELF4 to transactivate the IL-3 promoter and with ELF2 to transactivate the mouse BLK promoter. Inhibits MYST4-dependent transcriptional activation.,PTM:Methylated.,PTM:Phosphorylated in its C-terminus upon IL-6 treatment. Phosphorylation enhances interaction with MYST3.,similarity:Contains 1 Runt domain.,subunit:Heterodimer with CBFB. RUNX1 binds DNA as a monomer and through the Runt domain. DNA-binding is increased by heterodimerization. Isoform AML-1L can neither bind DNA nor heterodimerize. Interacts with TLE1 and THOC4. Interacts with ELF1, ELF2 and SPI1. Interacts via its Runt domain with the ELF4 N-terminal region. Interaction with ELF2 isoform 2 (NERF-1a) may act to repress RUNX1-mediated transactivation. Interacts with MYST3 and MYST4. Interacts with SUV39H1, leading to abrogate the transactivating and DNA-binding properties of RUNX1.,tissue specificity:Expressed in all tissues examined except brain and heart. Highest levels in thymus, bone marrow and peripheral blood.,

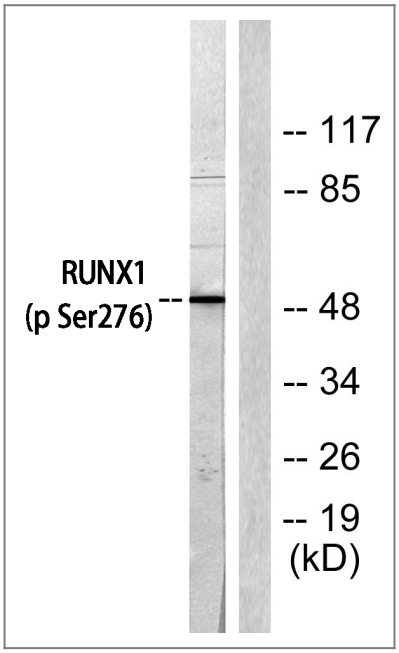
Validation Data



Enzyme-Linked Immunosorbent Assay (Phospho-ELISA) for Immunogen Phosphopeptide (Phospho-left) and Non-Phosphopeptide (Phospho-right), using AML1 (Phospho-Ser303) Antibody



Immunohistochemistry analysis of paraffin-embedded human lung carcinoma, using AML1 (Phospho-Ser303) Antibody. The picture on the right is blocked with the phospho peptide.



Western blot analysis of lysates from Jurkat cells, using AML1 (Phospho-Ser303) 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: **RUNX1 (Phospho Ser276) Rabbit pAb**