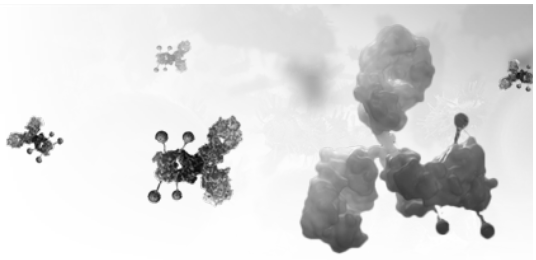

Rabbit Anti-RUNX1 / AML1 + RUNX3 + RUNX2 monoclonal antibody, clone TE1914 (DCABH-2632)

This product is for research use only and is not intended for diagnostic use.

PRODUCT INFORMATION

Target	RUNX1+RUNX2+RUNX3
Immunogen	Recombinant protein
Isotype	IgG
Source/Host	Rabbit
Species Reactivity	Human, Mouse, Rat
Clone	TE1914
Purification	Protein A purified.
Conjugate	Unconjugated
Applications	WB, ICC/IF, IHC, IP, FC
Molecular Weight	49 kDa
Cellular Localization	Nucleus, Cytoplasm.
Positive Control	Jurkat, MCF-7, N2A, SHG-44, human tonsil tissue, mouse testis tissue.
Format	Liquid
Size	100 µl
Buffer	1×TBS (pH7.4), 1% BSA, 40% Glycerol.

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Preservative	0.05% Sodium Azide
Storage	Store at +4°C after thawing. Aliquot store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.

BACKGROUND

Introduction

The mammalian Runt-related transcription factor (RUNX) family comprises three members, RUNX1 (also designated AML-1, PEBP2αB, CBFA2), RUNX2 (also designated AML-3, PEBP2 αA, CBFA1, Osf2) and RUNX3 (also designated AML-2, PEBPαC, CBFA3). RUNX family members are DNA-binding proteins that regulate the expression of genes involved in cellular differentiation and cell cycle progression. RUNX1 is involved in hematopoiesis and is frequently targeted in human leukemia by chromosomal translocations that fuse the DNA-binding domain of RUNX1 to other transcription factors and corepressor molecules. In addition to its role in leukemogenesis, RUNX1 is also involved in sensory neuron diversification. RUNX1 promotes axonal growth, is selectively expressed in neural crest-derived TrkA+ sensory neurons and mediates TrkA transactivation in migratory neural crest cells. RUNX2 is essential for skeletal mineralization in that it stimulates osteoblast differentiation of mesenchymal stem cells, promotes chondrocyte hypertrophy and contributes to endothelial cell migration and vascular invasion of developing bones. Regulating RUNX2 expression may be a useful therapeutic tool for promoting bone formation. Mutations in the C-terminus of RUNX2 are associated with cleidocranial dysplasia syndrome, an autosomal-dominant skeletal dysplasia syndrome that is characterized by widely patent calvarial sutures, clavicular hypoplasia, supernumerary teeth, and short stature. RUNX3 is expressed in cells of hematopoietic origin, including myeloid and B-cell lines and spleen. By playing a role in controlling the growth and differentiation of gastric epithelial cells, RUNX3 is a strong candidate as a gastric cancer tumor suppressor. Specifically, hypermethylation inactivates the gene encoding RUNX3. The detection of hypermethylation at multiple regions within the RUNX3 CpG island may aid in the diagnosis and risk assessment of gastric cancer.

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