

DESCRIPTION

Species Reactivity	Human/Mouse
Specificity	Detects human and mouse RUNX3/CBFA3 in Western blots.
Source	Monoclonal Mouse IgG _{2A} Clone # 527327
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human RUNX3/CBFA3 Lys186-Tyr415 Accession # Q13761
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
Intracellular Staining by Flow Cytometry	0.25-1 µg/10 ⁶ cells	Human PBMC treated with PMA and mouse splenocytes fixed with paraformaldehyde and permeabilized with saponin

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. <ul style="list-style-type: none"> 12 months from date of receipt, 2 to 8 °C as supplied.

BACKGROUND

RUNX3, also called CBFA3, AML-2 or PEBP2-αC, is a member of the Runt domain family of nuclear transcriptional regulators. All of the RUNX proteins form dimers with CBF-β. The runt domain (aa 54-186) is required for DNA binding, while a pro/ser/thr-rich region (aa 191-415) transcriptionally activates target genes. Isoform 2 has an alternate 19 aa in place of the N-terminal 5 aa of isoform 1. The 415 aa Human RUNX3 shares 91% aa identity with mouse or rat RUNX3. RUNX3 is necessary for growth control of gastric epithelium, neurogenesis of dorsal root ganglia, and T cell differentiation. RUNX3 expression is frequently mutated in tumors and appears to be silenced by methylation.

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