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Product Information Sheet

Monoclonal Anti-Actin (Sepharose Bead Conjugate)

Catalogue No. MA1000-S Immunogen

Synthetic actin C-terminal peptide

Lot No. 08A12 Ser-Gly-Pro-Ser-Ile-Val-His-Arg-Lys-Cys-Phe,

attached to a Multiple Antigen Peptide (MAP) backbone.

Clone: AC-40

Purification

Ig type: mouse IgG2a Purified by the goat anti-mouse IgG affinity chromatography.

Size: 200µl Formulation

50% slurry in PBS pH 7.2 with 0.01mg NaN₃a₃ preservative.

Specificity

Human, mouse, rat, chicken. Storage

No cross reactivity with other Store at 4°C for frequent use.

proteins.

Description:

Recommended applicationThis Antagene antibody is immobilized via covalent binding of primary amino groups

Immunoprecipitation(IP) to N-hydroxysuccinimide (NHS)-activated sepharose beads. It is useful for

immunoprecipitation assays.

BACKGROUND

Actin, a highly conserved protein, is a major component of both the cytoskeletal and contractile structures in the cell types. It varies in amount, being related to the type of differentiation and to the functional state of cells and tissues. The actins exhibit over 90% sequence homology, but each isoform has a unique NH2-terminal sequence. The isoforms are comprised of three alpha-actin, one beta-actin, two gamma-actin. Because the amino acid sequence of the C-terminal is the same for almost all actins, this antibody has been raised using a synthetic peptide corresponding to the C-terminal 11 residues.

REFERENCE

1. Gunning, P., Ponte, P., Okayama, H., Engel, J., Blau, H. and Kedes, L. Isolation and characterization of full-length cDNA clones for human alpha-, beta-, and gamma-actin mRNAs: skeletal but not cytoplasmic actins have an amino-terminal cysteine that is subsequently removed.

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2.Goebel, H.H., Brockmann, K., Bonnemann, C.G., Warlo, I.A., Hanefeld, F., Labeit, S., Durling, H.J. and Laing, N.G. Actin-related myopathy without any missense mutation in the ACTA1 Gene.

J. Child Neurol.2004; 19 (2), 149-153.

3.Laing, N.G., Clarke, N.F., Dye, D.E., Liyanage, K., Walker, K.R., Kobayashi, Y., Shimakawa, S., Hagiwara, T., Ouvrier, R., Sparrow, J.C., Nishino, I., North, K.N. and Nonaka, I. Actin mutations are one cause of congenital fibre type disproportion. Ann. Neurol. 2004; 56 (5), 689-694.