3050 Spruce Street
Saint Louis, Missouri 63103 USA
Telephone 800-325-5832 • (314) 771-5765
Fax (314) 286-7828
email: techserv@sial.com
sigma-aldrich.com

ProductInformation

Monoclonal Anti-Rit1
Clone 14G7
Purified Mouse Immunoglobulin

Product Number **R 1527**

Product Description

Monoclonal Anti-Human Rit1 (mouse IgG1 isotype) is derived from the hybridoma produced by the fusion of a mouse proprietary cell line and splenocytes from a BALB/c mouse immunized with full-length recombinant human Rit1 protein. Anti-Rit1 recognizes full-length human and murine recombinant or native Rit1 protein (approx. 25 kDa) [1] by immunoblotting. It does not cross-react with Rit2 protein.

The Ras super-family comprises a group of structurally related low molecular weight (20-30 kDa) proteins, which bind GTP or GDP and exhibit a low intrinsic GTPase activity. They are involved in signal transduction and the regulation of a variety of cellular processes, including cell growth, transformation, differentiation and morphogenesis, nucleocytoplasmic transport, and apoptosis. Ras proteins promote cancer by disrupting the normal controls of cell proliferation and differentiation. All Ras subgroups (Ras, Rho, Rab, Ran, and ADP-ribosylation factor) contain five highly conserved domains (G1-G5) and act as molecular switches by alternating between an active GTP-bound form and an inactive GDP-bound form.

Rit2 (Ras-like protein in neurons) and Rit1 (Ras-like protein in tissues) share 50% homology to Ras. Both contain the five highly conserved domains (G1-G5), bind GTP *in vitro,* and are membrane-associated. However, both lack a CAAX box and their mechanism of membrane association is distinct from the typical Ras protein. Rit is a non-lipid-modified Ras-related protein, which lacks any known recognition signal for C-terminal lipidation. Rit1 and Rit2 share a unique effector domain (DPTIEDAYK) that is 100% conserved between murine and human proteins. Rit1 is expressed ubiquitously, but Rit2 is expressed only in subsets of neurons and may be involved in calcium-mediated signaling within neurons.

Rit uses novel effector pathways to regulate proliferation and transformation. It has been suggested that Rit1 and Rit2 play important role in the regulation of signaling pathways, distinct from those controlled by Ras. Ras and associated pathways have also been implicated in the development and maintenance of the mammalian retina.[1-3]

Reagent

Monoclonal Anti-Human Rit1 is provided at approx. 100 µg in 100 µl of sterile filtered solution pH 7.5, containing 20 mM sodium phosphate, 150 mM sodium chloride, 50% glycerol and 3 mM sodium azide.

Precautions and Disclaimer

Due to the sodium azide content a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling practices.

Storage/Stability

Store at –20 °C. For extended storage, freeze in working aliquots. Avoid repeated freezing and thawing to prevent denaturing of the antibody. Do not store in a frost-free freezer. The antibody is stable for at least 12 months when stored appropriately.

Product Profile

A recommended working concentration of 0.5 to 1.0 µg/ml was determined by immunoblotting using HEK293 cells transfected with full-length human Rit1 protein. Untransfected HEK293 cells are used as a negative control to determine the extent of non-specific binding.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

References

1. Lee, C.H., et al., Rin, a neuron-specific and calmodulin-binding small G-protein, and Rit define a novel subfamily of ras proteins. J. Neurosci., **16**, 6784-6794 (1996).
2. Shao, H., et al., Biochemical characterization of the Ras-related GTPases Rit and Rin. Arch. Biochem. Biophys., **371,** 207-219, (1999).
3. Rusyn, E.V., et al., Rit, a non-lipid-modified Ras-related protein, transforms NIH3T3 cells. Oncogene, **41,** 4685-4694 (2000).

AHrev 1/03