

**NISCH Blocking Peptide (N-Term)**  
**Synthetic peptide**  
**Catalog # BP22144a****Specification**

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**NISCH Blocking Peptide (N-Term) - Product Information**Primary Accession [Q9Y2I1](#)**NISCH Blocking Peptide (N-Term) - Additional Information****Gene ID** 11188**Other Names**

Nischarin, Imidazoline receptor 1, I-1, IR1, Imidazoline receptor antisera-selected protein, hIRAS, Imidazoline-1 receptor, I1R, Imidazoline-1 receptor candidate protein, I-1 receptor candidate protein, I1R candidate protein, NISCH, IRAS, KIAA0975

**Target/Specificity**

The synthetic peptide sequence is selected from aa 5-18 of HUMAN NISCH

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**NISCH Blocking Peptide (N-Term) - Protein Information****Name** NISCH**Synonyms** IRAS, KIAA0975**Function**

Acts either as the functional imidazoline-1 receptor (I1R) candidate or as a membrane-associated mediator of the I1R signaling. Binds numerous imidazoline ligands that induces initiation of cell-signaling cascades triggering to cell survival, growth and migration. Its activation by the agonist rilmenidine induces an increase in phosphorylation of mitogen-activated protein kinases MAPK1 and MAPK3 in rostral ventrolateral medulla (RVLM) neurons that exhibited rilmenidine-evoked hypotension (By similarity). Blocking its activation with efaroxan abolished rilmenidine-induced mitogen-activated protein kinase phosphorylation in RVLM neurons (By similarity). Acts as a modulator of Rac-regulated signal transduction pathways (By similarity). Suppresses Rac1-stimulated cell migration by interacting with PAK1 and inhibiting its kinase activity (By similarity). Also blocks Pak-independent Rac signaling by interacting with RAC1 and inhibiting Rac1-stimulated NF-kB response element and cyclin D1 promoter activation (By similarity). Also

inhibits LIMK1 kinase activity by reducing LIMK1 'Tyr-508' phosphorylation (By similarity). Inhibits Rac-induced cell migration and invasion in breast and colon epithelial cells (By similarity). Inhibits lamellipodia formation, when overexpressed (By similarity). Plays a role in protection against apoptosis. Involved in association with IRS4 in the enhancement of insulin activation of MAPK1 and MAPK3. When overexpressed, induces a redistribution of cell surface ITGA5 integrin to intracellular endosomal structures.

#### **Cellular Location**

Cell membrane. Cytoplasm. Early endosome. Recycling endosome. Note=Enriched in the early/sorting and recycling endosomes. Colocalized in early/sorting endosomes with EEA1 and SNX2 and in recycling endosomes with transferrin receptor. Detected in the perinuclear region partially associated with punctate structures (By similarity). Colocalizes with PAK1 in cytoplasm, vesicular structures in the perinuclear area and membrane ruffles (By similarity) Colocalizes with RAC1 in the cytoplasm and vesicles structures (By similarity). Colocalized with MAPK1 and MAPK3 in RVLM neurons (By similarity).

#### **Tissue Location**

Isoform 1, isoform 3 and isoform 4 are expressed in brain. Isoform 1 is expressed in endocrine tissues

### **NISCH Blocking Peptide (N-Term) - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

### **NISCH Blocking Peptide (N-Term) - Images**

### **NISCH Blocking Peptide (N-Term) - Background**

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### **NISCH Blocking Peptide (N-Term) - References**

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Bechtel S.,et al.BMC Genomics 8:399-399(2007).