

# IRS1 Rabbit mAb

Catalog # AP78980

#### **Product Information**

**Application** WB, IHC-P, ICC

Primary Accession P35568
Reactivity Human
Rabbit

**Clonality** Monoclonal Antibody

Calculated MW 131591

### **Additional Information**

**Gene ID** 3667

Other Names IRS1

**Dilution** WB~~1/500-1/1000 IHC-P~~N/A ICC~~N/A

Format 10mM PBS, pH 7.4, 150mM sodium chloride, 0.05% BSA, 0.02% sodium azide

and 50% glycerol.

**Storage** Store at 4°C short term. Aliquot and store at -20°C long term. Avoid

freeze/thaw cycles.

### **Protein Information**

Name IRS1

**Function** Signaling adapter protein that participates in the signal transduction from

two prominent receptor tyrosine kinases, insulin receptor/INSR and

insulin-like growth factor I receptor/IGF1R (PubMed: 7541045,

PubMed:33991522, PubMed:38625937). Plays therefore an important role in development, growth, glucose homeostasis as well as lipid metabolism (PubMed:19639489). Upon phosphorylation by the insulin receptor, functions as a signaling scaffold that propagates insulin action through binding to SH2 domain-containing proteins including the p85 regulatory subunit of PI3K, NCK1, NCK2, GRB2 or SHP2 (PubMed:11171109, PubMed:8265614). Recruitment of GRB2 leads to the activation of the guanine nucleotide exchange factor SOS1 which in turn triggers the Ras/Raf/MEK/MAPK signaling

exchange factor SOS1 which in turn triggers the Ras/Raf/MEK/MAPK signaling cascade (By similarity). Activation of the PI3K/AKT pathway is responsible for most of insulin metabolic effects in the cell, and the Ras/Raf/MEK/MAPK is involved in the regulation of gene expression and in cooperation with the PI3K pathway regulates cell growth and differentiation. Acts a positive regulator of the Wnt/beta-catenin signaling pathway through suppression of DVL2 autophagy-mediated degradation leading to cell proliferation

(PubMed:<u>24616100</u>).

### **Cellular Location**

Cytoplasm. Nucleus. Note=Nuclear or cytoplasmic localization of IRS1 correlates with the transition from proliferation to chondrogenic differentiation.

# **Images**



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