

IRS-1 (Phospho Ser636/639) Rabbit pAb

CatalogNo: YP1370

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat

Applications

- WB, IHC

MW

- 170kD (Observed)

Isotype

- IgG

Recommended Dilution Ratios

WB 1:500-2000

IHC 1:50-300

Storage

Storage*

-15°C to -25°C/1 year (Do not lower than -25°C)

Formulation

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality

Polyclonal

Immunogen Information

Immunogen

Synthesized phospho peptide around human IRS-1 (Ser636 and 639)

Specificity

This antibody detects endogenous levels of IRS-1 only when phosphorylated at Ser636 or Ser639, and dually phosphorylated at two sites

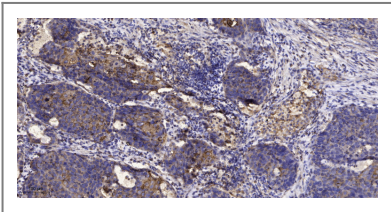
Target Information

Gene name

IRS1

Protein Name	IRS-1 (Ser636/639)		
	Organism	Gene ID	UniProt ID
	Human	3667 ;	P35568 ;
	Mouse	16367 ;	P35569 ;
	Rat	25467 ;	P35570 ;
Cellular Localization	nucleus,cytoplasm,cytosol,plasma membrane,insulin receptor complex,caveola,intracellular membrane-bounded organelle,		
Tissue specificity	Epithelium,Eye,Skeletal muscle,		
Function	<p>Disease:Polymorphisms in IRS1 may be involved in the etiology of non-insulin-dependent diabetes mellitus (NIDDM) [MIM:125853].,Function:May mediate the control of various cellular processes by insulin. When phosphorylated by the insulin receptor binds specifically to various cellular proteins containing SH2 domains such as phosphatidylinositol 3-kinase p85 subunit or GRB2. Activates phosphatidylinositol 3-kinase when bound to the regulatory p85 subunit.,polymorphism:The Arg-971 polymorphism impairs the ability of insulin to stimulate glucose transport, glucose transporter translocation, and glycogen synthesis by affecting the PI3K/AKT1/GSK3 signaling pathway. The polymorphism at Arg-971 may contribute to the in vivo insulin resistance observed in carriers of this variant. Arg-971 could contribute to the risk for atherosclerotic cardiovascular diseases associated with non-insulin-dependent diabetes mellitus (NIDDM) by producing a cluster of insulin resistance-related metabolic abnormalities. In insulin-stimulated human endothelial cells from carriers of the Arg-971 polymorphism, genetic impairment of the IRS1/PI3K/PDPK1/AKT1 insulin signaling cascade results in impaired insulin-stimulated nitric oxide (NO) release and suggested that this may be a mechanism through which the Arg-971 polymorphism contributes to the genetic predisposition to develop endothelial dysfunction and cardiovascular disease. The Arg-971 polymorphism not only reduces phosphorylation of the substrate but allows IRS1 to act as an inhibitor of PI3K, producing global insulin resistance.,PTM:Phosphorylation of Tyr-896 is required for GRB2-binding.,PTM:Serine phosphorylation of IRS1 is a mechanism for insulin resistance. Ser-312 phosphorylation inhibits insulin action through disruption of IRS1 interaction with the insulin receptor.,similarity:Contains 1 IRS-type PTB domain.,similarity:Contains 1 PH domain.,subunit:Interacts with the NPXY motif of tyrosine-phosphorylated IGF1R and INSR via the PTB domain. Binds to phosphatidylinositol 3-kinase p85 subunit via the phosphorylated YXXM motifs. Binds ROCK1. Binds to UBTF and PIK3CA in nuclear extracts (By similarity). Interacts with SOCS7.,</p>		

| Validation Data



Immunohistochemical analysis of paraffin-embedded human Squamous cell carcinoma of lung. 1, Antibody was diluted at 1:200(4° overnight). 2, Tris-EDTA,pH9.0 was used for antigen retrieval. 3,Secondary antibody was diluted at 1:200(room temperature, 45min).

| Contact information

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Ser636/639) Rabbit
pAb**

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