Product name:	IRS-1 (phospho Tyr896) Rabbit Polyclonal Antibody
Cat number:	ABN04884
Conjugate:	Unconjugated
Size:	100µL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human IRS-1 around the phosphorylation site of Tyr896. AA range:862-911
Reactivity:	Human,Mouse,Rat
Applications:	WB 1:500-1:2000,IHC 1:50-1:300
Molecular Weight:	170kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

Background:

This gene encodes a protein which is phosphorylated by insulin receptor tyrosine kinase. Mutations in this gene are associated with type II diabetes and susceptibility to insulin resistance. [provided by RefSeq, Nov 2009],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.,