Anti-TBC1D4 antibody

Description	Rabbit polyclonal to TBC1D4.

Model	STJ95925
Host	Rabbit
Reactivity	Human, Mouse
Applications	ELISA, IF, IHC
Immunogen	Synthesized peptide derived from human TBC1D4 around the non-phosphorylation site of T642.
Immunogen Region	580-660 aa
Gene ID	9882
Gene Symbol	TBC1D4
Dilution range	IHC 1:100-1:300IF 1:200-1:1000ELISA 1:10000
Specificity	TBC1D4 Polyclonal Antibody detects endogenous levels of TBC1D4 protein.
Tissue Specificity	Widely expressed. Isoform 2 is the highest overexpressed in most tissues. Isoform 1 is highly expressed in skeletal muscle and heart, but was not detectable in the liver nor in adipose tissue. Isoform 2 is strongly expressed in adrenal and thyroid gland, and also in lung, kidney, colon, brain and adipose tissue. Isoform 2 is moderately expressed in skeletal muscle. Expressed in pancreatic Langerhans islets, including beta cells (at protein level). Expression is decreased by twofold in pancreatic islets in
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.

Note	For Research Use Only (RUO).
Protein Name	TBC1 domain family member 4 Akt substrate of 160 kDa AS160
Molecular Weight	146.563 kDa
Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:19165OMIM:612465
Alternative Names	TBC1 domain family member 4 Akt substrate of 160 kDa AS160
Function	May act as a GTPase-activating protein for RAB2A, RAB8A, RAB10 and RAB14. Isoform 2 promotes insulin-induced glucose transporter SLC2A4/GLUT4 translocation at the plasma membrane, thus increasing glucose uptake.
Cellular Localization	Cytoplasm. Isoform 2 shows a cytoplasmic perinuclear localization in a myoblastic cell line in resting and insulin-stimulated cells.
Post-translational Modifications	Phosphorylated by AKT1; insulin-induced. Also phosphorylated by AMPK in response to insulin. Insulin-stimulated phosphorylation is required for SLC2A4/GLUT4 translocation. Has no effect on SLC2A4/GLUT4 internalization. Physiological hyperinsulinemia increases phosphorylation in skeletal muscle. Insulin-stimulated phosphorylation is reduced by 39% in type 2 diabetic patients.