

TBC1D4 Antibody

Catalog # ASC10621

Product Information

Application	E, IHC-P
Primary Accession	O60343
Other Accession	NP_055647 , 114688046
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	146563
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	TBC1D4 antibody can be used for immunohistochemistry starting at 10 μ g/mL.

Additional Information

Gene ID	9882
Other Names	TBC1 domain family member 4, Akt substrate of 160 kDa, AS160, TBC1D4, AS160, KIAA0603
Target/Specificity	TBC1D4;
Reconstitution & Storage	TBC1D4 antibody can be stored at 4°C for three months and -20°C, stable for up to one year.
Precautions	TBC1D4 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	TBC1D4
Synonyms	AS160, KIAA0603
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 Location	Cytoplasm. Note=Isoform 2 shows a cytoplasmic perinuclear localization in a myoblastic cell line in resting and insulin-stimulated cells
Tissue Location	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 type 2 diabetes patients compared to control subjects. Up-regulated in T-cells from patients with atopic dermatitis.

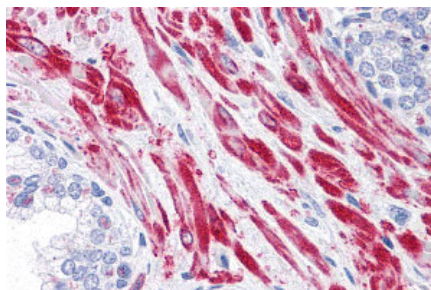
Background

TBC1D4 Antibody: TBC1D4, also known as the Akt substrate AS160, was initially identified as an Akt substrate containing a GTPase-activating domain that regulates GLUT4 trafficking, with activation following insulin stimulation. TBC1D4 truncations in humans is a major cause of dominant inherited insulin resistance. The loss of TBC1D4 results in the accumulation of GLUT4 in compartments that are primed for fusion in basal adipocytes.

References

- Kane S, Sano H, Liu SCH, et al. Akt phosphorylates a novel adipocyte protein with a Rab GTPase-activating protein (GAP) domain. *J. Biol. Chem.* 2002; 277:22115-8.
- Sano H, Kane S, Sano E, et al. Insulin-stimulated phosphorylation of a Rab GTPase-activating protein regulates GLUT4 translocation. *J. Biol. Chem.* 2003; 278:14599-602.
- Dash S, Sano H, Rochford JJ, et al. A truncation mutation in TBC1D4 in a family with acanthosis nigricans and postprandial hyperinsulinemia. *Proc. Natl. Acad. Sci. U.S.A.* 2009; 106:9350-5.
- Brewer PD, Romenskaia I, Kanow MA, et al. Loss of AS160 Akt substrate causes Glut4 protein to accumulate in compartments that are primed for fusion in basal adipocytes. *J. Biol. Chem.* 2011; 286:26287-97.

Images



Immunohistochemistry of TBC1D4 in human prostate tissue with TBC1D4 antibody at 10 µg/mL.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.