## **Product Name: TBC1D4 Rabbit Polyclonal Antibody**

Catalog #: APRab18677



#### **Summary**

Production Name TBC1D4 Rabbit Polyclonal Antibody

**Description** Rabbit Polyclonal Antibody

Host Rabbit
Application IF,ELISA

**Reactivity** Human, Mouse

#### **Performance**

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

#### **Immunogen**

Gene Name TBC1D4

TBC1D4; AS160; KIAA0603; TBC1 domain family member 4; Akt substrate of 160 kDa;

AS160

**Gene ID** 9882.0

O60343.The antiserum was produced against synthesized peptide derived from human **SwissProt ID** 

AS160. AA range:611-660

### **Application**

**Dilution Ratio** IF 1:200-1:1000. ELISA: 1:10000.

**Molecular Weight** 

**Alternative Names** 

#### **Background**

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This gene is a member of the Tre-2/BUB2/CDC16 domain family. The protein encoded by this gene is a Rab-GTPaseactivating protein, and contains two phopshotyrosine-binding domains (PTB1 and PTB2), a calmodulin-binding domain (CBD), a Rab-GTPase domain, and multiple AKT phosphomotifs. This protein is thought to play an important role in glucose homeostasis by regulating the insulin-dependent trafficking of the glucose transporter 4 (GLUT4), important for removing glucose from the bloodstream into skeletal muscle and fat tissues. Reduced expression of this gene results in an increase in GLUT4 levels at the plasma membrane, suggesting that this protein is important in intracellular retention of GLUT4 under basal conditions. When exposed to insulin, this protein is phosphorylated, dissociates from GLUT4 vesicles, resulting in increased GLUT4 at the cell surface, and enhanced glucose transport. Phdisease: May be involved in atopic dermatitis (AD), function: May act as a GTPase-activating protein for RAB2A, RAB8A, RAB10 and RAB14. Isoform 2 promotes insulininduced glucose transporter SLC2A4/GLUT4 translocation at the plasma membrane, thus increasing glucose uptake, PTM: Insulin-stimulated phosphorylation is required for SLC2A4/GLUT4 translocation., PTM: Phosphorylated by AKT1; insulin-induced., PTM: Physiological hyperinsulinemia increases phosphorylation in skeletal muscle. Insulin-stimulated phosphorylation is reduced by 39% in type 2 diabetic patients,,similarity:Contains 1 Rab-GAP TBC domain, similarity: Contains 2 PID domains, subcellular location: Isoform 2 shows a cytoplasmic perinuclear localization in a myoblastic cell line in resting and insulin-stimulated cells., tissue specificity: Widely expressed, but differential expression for isoforms 1 and 2, with highest overall expression of isoform 2 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 strongly expressed in adrenal and thyroid gland, and also in lung, kidney, colon, brain and adipose tissue. Moderate isoform 2 expression 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.,

#### **Research Area**

Insulin Receptor

#### **Image Data**



Immunofluorescence analysis of HeLa cells, using AS160 Antibody. The picture on the right is blocked with the synthesized peptide.

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#### Note

For research use only.