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Rabbit Anti-ARFGEF2 antibody

SL11804R

Product Name:	ARFGEF2
Chinese Name:	二磷酸腺苷核糖基化因子鸟嘌呤核苷酸交换因子2抗体
Alias:	ADP ribosylation factor guanine nucleotide exchange factor 2 (brefeldin A inhibited); ADP ribosylation factor guanine nucleotide exchange factor 2; ARFGEF 2; ARFGEF2; ARFGEP2; BIG 2; BIG2; Brefeldin A inhibited 2; Brefeldin A inhibited GEP 2; Brefeldin A inhibited guanine nucleotide exchange protein 2; dJ1164I10.1; BIG2_HUMAN.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Chicken,Dog,Pig,Cow,Horse,Sheep,
Applications:	WB=1:500-2000ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800ICC=1:100-500IF=1:100-500（Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	202kDa
Cellular localization:	cytoplasmicThe cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from human ARFGEF2/BIG2:761-860/1785
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
PubMed:	PubMed
Product Detail:	Guanine nucleotide-exchange proteins (GEPs) accelerate replacement of bound GDP with GTP and thereby activate ADP-ribosylation factors (ARFs), a family of guanine nucleotide-binding proteins that play an important role in intracellular vesicular

	trafficking. GEPs comprise two major families, large GEPs that are inhibited by brefeldin A (BFA), a protein that effects golgi structure, and a group of smaller GEPs that are insenstive to BFA. Two genes for GEPs found on human chromosomes 8 and 20 encode BFA sensitive GEPs designated BIG1 and BIG2. Both GEPS contain a sec7 domain that is responsible for their brefeldin inhibition and also their catalytic activity. In vivo, BIG1 and BIG2 exist in macromolecular complexes that move between the golgi membranes and cytosol. BIG2 associates with PKA regulatory subunits, implying that BIG2 may act as an A kinase-anchoring protein (AKAP) that could coordinate the cAMP and ARF regulatory pathways. **Function:** ADP-ribosylation factors (ARFs) play an important role in intracellular vesicular trafficking. ARFGEF2 promotes guanine-nucleotide exchange on ARF1, ARF5 and ARF6 and the activation of ARF1/ARF5/ARF6 through replacement of GDP with GTP. It contains a Sec7 domain, which may be responsible for its guanine-nucleotide exchange activity and also brefeldin A inhibition. **Subunit:** Homodimer (Probable). Interacts with BIG1; both proteins are probably part of the same or very similar macromolecular complexes. Interacts with PRKAR1A, PRKAR2A, PRKAR1B, PRKAR2B, PPP1CC, PDE3A, TNFRSF1A, MYCBP and EXOC7. Interacts with GABRB1, GABRB2 and GABRB3 **Subcellular Location:** Cytoplasm. Membrane. Golgi apparatus. Cytoplasm, perinuclear region. Golgi apparatus, trans-Golgi network (By similarity). Endosome (By similarity). Cytoplasm, cytoskeleton, centrosome. Cell projection, dendrite (By similarity). Cytoplasmic vesicle (By similarity). Cell junction, synapse (By similarity). Cytoplasm, cytoskeleton (By similarity). Note=Translocates from cytoplasm to membranes upon cAMP treatment. Localized in recycling endosomes. **Tissue Specificity:** Expressed in placenta, lung, heart, brain, kidney and pancreas. **Post-translational modifications:** Phosphorylated upon DNA damage, probably by ATM or ATR. In vitro phosphorylated by PKA reducing its GEF activity and dephosphorylated by phosphatase PP1. **DISEASE:** Defects in ARFGEF2 are the cause of autosomal recessive periventricular nodular heterotopia type 2 (PVNH2) [MIM:608097]; also known as periventricular heterotopia with microcephaly autosomal recessive. PVNH is a developmental disorder characterized by the presence of periventricular nodules of cerebral gray matter, resulting from a failure of neurons to migrate normally from the lateral ventricular proliferative zone, where they are formed, to the cerebral cortex. PVNH2 is an autosomal recessive form characterized by microcephaly (small brain), severe

	developmental delay and recurrent infections. No anomalies extrinsic to the central nervous system, such as dysmorphic features or grossly abnormal endocrine or other conditions, are associated with PVNH2. **Similarity:** Contains 1 SEC7 domain. **SWISS:** Q9Y6D5 **Gene ID:** 10564 **Database links:** Entrez Gene: 10564 Human Entrez Gene: 99371 Mouse Entrez Gene: 296380 Rat Omim: 605371 Human SwissProt: Q9Y6D5 Human **Important Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.