10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

GPD1L Rabbit pAb

Catalog # AP99276

Product Information

Application	IHC-P, IHC-F, IF
Primary Accession	Q8N335
Other Accession	Q8N335
Reactivity	Rat
Predicted	Human, Mouse, Chicken, Dog, Sheep
Host	Rabbit
Clonality	Polyclonal
Calculated MW	38419
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human GPD1L
Epitope Specificity	201-300/351
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the NAD-dependent glycerol-3-phosphate dehydrogenase family.
DISEASE	Defects in GPD1L are the cause of Brugada syndrome type 2 (BRS2) [MIM:611777]. BRS2 is an autosomal dominant tachyarrhythmia characterized by right bundle branch block and ST segment elevation on an electrocardiogram (ECG). It can cause the ventricles to beat so fast that the blood is prevented from circulating efficiently in the body. When this situation occurs (called ventricular fibrillation), the individual will faint and may die in a few minutes if the heart is not reset. Defects in GPD1L are a cause of sudden infant death syndrome (SIDS) [MIM:272120]. SIDS is the sudden death of an infant younger than 1 year that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of clinical history. Pathophysiologic mechanisms for SIDS may include respiratory dysfunction, cardiac dysrhythmias, cardiorespiratory instability, and inborn errors of metabolism, but definitive pathogenic mechanisms precipitating an infant sudden death remain elusive.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	The protein encoded by this gene catalyzes the conversion of sn-glycerol 3-phosphate to glycerone phosphate. The encoded protein is found in the cytoplasm, associated with the plasma membrane, where it binds the sodium channel, voltage-gated, type V, alpha subunit (SCN5A). Defects in this gene are a cause of Brugada syndrome type 2 (BRS2) as well as sudden infant death syndrome (SIDS). [provided by RefSeq, Jul 2010]

Additional Information

Gene ID	23171
Other Names	GPD1-L; 2210409H23Rik; D9Ertd660e; GPD1L_HUMAN; GPD1L; 1.1.1.8;

	KIAA0089; GPD1L_MOUSE; glycerol-3-phosphate dehydrogenase 1 like
Dilution	IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. 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.

Protein Information

Name	GPD1L (HGNC:28956)
Synonyms	KIAA0089
Function	Plays a role in regulating cardiac sodium current; decreased enzymatic activity with resulting increased levels of glycerol 3- phosphate activating the DPD1L-dependent SCN5A phosphorylation pathway, may ultimately lead to decreased sodium current; cardiac sodium current may also be reduced due to alterations of NAD(H) balance induced by DPD1L.
Cellular Location	Cytoplasm. Note=Localized to the region of the plasma membrane
Tissue Location	Most highly expressed in heart tissue, with lower levels in the skeletal muscle, kidney, lung and other organs

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