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Pael-R (GPR37) Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP6410b

Specification

Pael-R (GPR37) Antibody (C-term) Blocking Peptide - Product Information

Primary Accession	O15354
Other Accession	NP_005293

Pael-R (GPR37) Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 2861

Other Names
Prosaposin receptor GPR37, Endothelin B receptor-like protein 1, ETBR-LP-1, G-protein coupled receptor 37, Parkin-associated endothelin receptor-like receptor, PAELR, GPR37

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP6410b was selected from the GPR37 region of human Pael-R (GPR37). A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Pael-R (GPR37) Antibody (C-term) Blocking Peptide - Protein Information

Name GPR37

Function
G-protein-coupled receptor that plays a role in several physiological pathways such as resolution of inflammatory pain and oligodendrocyte differentiation (By similarity). Acts as a receptor for several ligands including prosaposin, osteocalcin or neuroprotectin D1. Ligand binding induces endocytosis, followed by an ERK phosphorylation cascade (PubMed:11439185, PubMed:23690594). Acts as a receptor for osteocalcin (OCN) to regulate oligodendrocyte differentiation and central nervous system myelination. Mechanistically, plays a negative role in oligodendrocyte differentiation and myelination during development via activation of the ERK1/2 signaling pathway. Therefore, regulates the stability of myelin or resistance of myelin itself to demyelination. Upon activation by

neuroprotectin D1 (NPD1), promotes the activation of phagocytosis in macrophages as well as the shift in cytokine release toward an anti-inflammatory profile, and thus helps to reverse inflammatory pain. In addition, the increased macrophage phagocytosis mediates protection against sepsis upon pathogen infection. Additionally, extracellular vesicles derived from efferocyte express prosaposin, which binds to macrophage GPR37 to increase expression of the efferocytosis receptor TIM4 via an ERK-AP1-dependent signaling axis, leading to increased macrophage efferocytosis efficiency and accelerated resolution of inflammation (By similarity). May also act as a maturation factor of LRP6, protecting LRP6 from the endoplasmic reticulum (ER)-associated protein degradation (ERAD) and thereby promoting the Wnt/beta-catenin signaling pathway (PubMed:28341812).

Cellular Location
Cell projection, dendrite. Synapse Cell membrane; Multi-pass membrane protein. Endoplasmic reticulum membrane; Multi-pass membrane protein

Tissue Location
Expressed in brain and spinal cord, and at lower levels in testis, placenta and liver, but no detectable expression observed in any other tissue. When overexpressed in cells, tends to become insoluble and unfolded. Accumulation of the unfolded protein may lead to dopaminergic neuronal death in juvenile Parkinson disease (PDJ).

Pael-R (GPR37) Antibody (C-term) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

Pael-R (GPR37) Antibody (C-term) Blocking Peptide - Images

Pael-R (GPR37) Antibody (C-term) Blocking Peptide - Background

Parkinson is the second most common neurodegenerative disease after Alzheimers. About 1 percent of people over the age of 65 and 3 percent of people over the age of 75 are affected by the disease. The mutation is the most common cause of Parkinson disease identified to date. The function of Park2 is not well-known; however, it may play a role in the ubiquitin-mediated proteolytic pathway. Mutations in this gene are known to cause autosomal recessive juvenile parkinsonism. Alternative splicing of this gene produces three known products of undetermined function. Panneuronal expression of Parkin substrate Pael-R causes age-dependent selective degeneration of Drosophila dopaminergic (DA) neurons; coexpression of Parkin degrades Pael-R and suppresses its toxicity.

Pael-R (GPR37) Antibody (C-term) Blocking Peptide - References

Yang,Y., et al. Neuron 37 (6), 911-924 (2003)Imai,Y., et al. Mol. Cell 10 (1), 55-67 (2002)Imai,Y., et al. Cell 105 (7), 891-902 (2001)Marazziti,D., et al. Genomics 45 (1), 68-77 (1997)Zeng,Z., et al. BBRC 233 (2), 559-567 (1997)