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SUCNR1 / GPR91 Antibody (Cytoplasmic Domain)
Rabbit Polyclonal Antibody
Catalog # ALS10645

Specification

SUCNR1 / GPR91 Antibody (Cytoplasmic Domain) - Product Information

Application	**IHC**
Primary Accession	Q9BXA5
Reactivity	**Human, Monkey**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**39kDa KDa**

SUCNR1 / GPR91 Antibody (Cytoplasmic Domain) - Additional Information

Gene ID 56670

Other Names
Succinate receptor 1, G-protein coupled receptor 91, P2Y purinoceptor 1-like, SUCNR1, GPR91

Target/Specificity
Human GPR91. BLAST analysis of the peptide immunogen showed no homology with other human proteins.

Reconstitution & Storage
Long term: -70°C; Short term: +4°C

Precautions
SUCNR1 / GPR91 Antibody (Cytoplasmic Domain) is for research use only and not for use in diagnostic or therapeutic procedures.

SUCNR1 / GPR91 Antibody (Cytoplasmic Domain) - Protein Information

Name SUCNR1 (HGNC:4542)

Synonyms GPR91

Function
G protein-coupled receptor for succinate able to mediate signaling through Gq/GNAQ or Gi/GNAI second messengers depending on the cell type and the processes regulated (By similarity) (PubMed:15141213, PubMed:23770096, PubMed:34133934). Succinate-SUCNR1 signaling serves as a link between metabolic stress, inflammation and energy homeostasis (PubMed:18820681, PubMed:34133934). In macrophages, plays a range of immune-regulatory roles. During inflammation, succinate-SUCNR1 signaling may act as an anti-inflammatory mediator or

boost inflammation depending on the inflammatory status of cells (By similarity). Hyperpolarizes M2 macrophages versus M1 phenotype through Gq signaling by regulating the transcription of genes involved in immune function (PubMed:34133934). In activated M1 macrophages, plays a pro-inflammatory role in response to LPS (By similarity). Expressed in dendritic cells, where it is involved in the sensing of immunological danger and enhances immunity. Mediates succinate triggered intracelleular calcium mobilization, induces migratory responses and acts in synergy with Toll-like receptor ligands for the production of proinflammatory cytokines as well as an enhancement of antigen-specific activation of helper T cells (PubMed:18820681). In the small intestine, mediates the activation of tuft cells by dietary succinate and triggers type 2 immunity (By similarity). In adipocytes, plays an important role in the control of energy metabolism. In response to succinate, controls leptin expression in an AMPK-JNK-CEBPA-dependent as well as circadian clock-regulated manner (By similarity). In muscle tissue, is expressed in non-muscle cells and coordinates muscle remodeling in response to the succinate produced during exercise training in a paracrine manner (By similarity). In retina, acts as a mediator of vessel growth during retinal development. In response to succinate, regulates the production of angiogenic factors, including VEGF, by retinal ganglion neurons (By similarity).

Cellular Location
Cell membrane; Multi-pass membrane protein

Tissue Location
Expressed specifically in kidney (PubMed:11273702). Highly expressed in immature dendritic cells, expression rapidly downregulates after maturation. Also expressed in macrophages (PubMed:18820681).

Volume
50 µl

SUCNR1 / GPR91 Antibody (Cytoplasmic Domain) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

SUCNR1 / GPR91 Antibody (Cytoplasmic Domain) - Images

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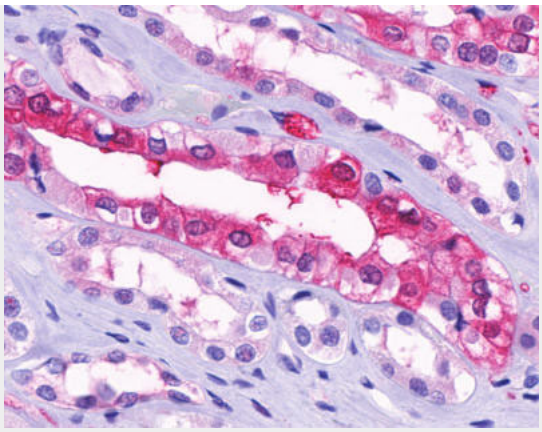

Anti-GPR91 antibody ALS10645 IHC of human kidney, medulla.

SUCNR1 / GPR91 Antibody (Cytoplasmic Domain) - Background

Receptor for succinate.

SUCNR1 / GPR91 Antibody (Cytoplasmic Domain) - References

Wittenberger T.,et al.J. Mol. Biol. 307:799-813(2001).
Zhang W.,et al.Submitted (MAR-2000) to the EMBL/GenBank/DDBJ databases.
Kaighin V.A.,et al.Submitted (DEC-2007) to the EMBL/GenBank/DDBJ databases.
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Muzny D.M.,et al.Nature 440:1194-1198(2006).