

**F2RL1 / PAR2 Antibody (N-Terminus)**  
**Rabbit Polyclonal Antibody**  
**Catalog # ALS10078****Specification**

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**F2RL1 / PAR2 Antibody (N-Terminus) - Product Information**

Application	IHC-P
Primary Accession	<a href="#">P55085</a>
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	44kDa KDa
Dilution	IHC-P~~N/A

**F2RL1 / PAR2 Antibody (N-Terminus) - Additional Information****Gene ID** 2150**Other Names**

Proteinase-activated receptor 2, PAR-2, Coagulation factor II receptor-like 1, G-protein coupled receptor 11, Thrombin receptor-like 1, Proteinase-activated receptor 2, alternate cleaved 1, Proteinase-activated receptor 2, alternate cleaved 2, F2RL1, GPR11, PAR2

**Target/Specificity**

Human F2RL1. BLAST analysis of the peptide immunogen showed no homology with other human proteins.

**Reconstitution & Storage**

Long term: -70°C; Short term: +4°C

**Precautions**

F2RL1 / PAR2 Antibody (N-Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

**F2RL1 / PAR2 Antibody (N-Terminus) - Protein Information****Name** F2RL1**Synonyms** GPR11, PAR2**Function**

Receptor for trypsin and trypsin-like enzymes coupled to G proteins (PubMed:<a href="http://www.uniprot.org/citations/28445455" target="\_blank">28445455</a>). Its function is mediated through the activation of several signaling pathways including phospholipase C (PLC), intracellular calcium, mitogen-activated protein kinase (MAPK), I-kappaB kinase/NF-kappaB and Rho (PubMed:<a href="http://www.uniprot.org/citations/28445455" target="\_blank">28445455</a>). Can also be transactivated by cleaved F2R/PAR1. Involved in modulation of inflammatory responses and regulation of innate and adaptive immunity, and acts

as a sensor for proteolytic enzymes generated during infection. Generally is promoting inflammation. Can signal synergistically with TLR4 and probably TLR2 in inflammatory responses and modulates TLR3 signaling. Has a protective role in establishing the endothelial barrier; the activity involves coagulation factor X. Regulates endothelial cell barrier integrity during neutrophil extravasation, probably following proteolytic cleavage by PRTN3 (PubMed:<a href="http://www.uniprot.org/citations/23202369" target="\_blank">23202369</a>). Proposed to have a bronchoprotective role in airway epithelium, but also shown to compromise the airway epithelial barrier by interrupting E-cadherin adhesion (PubMed:<a href="http://www.uniprot.org/citations/10086357" target="\_blank">10086357</a>). Involved in the regulation of vascular tone; activation results in hypotension presumably mediated by vasodilation. Associates with a subset of G proteins alpha subunits such as GNAQ, GNA11, GNA14, GNA12 and GNA13, but probably not with G(o)-alpha, G(i) subunit alpha-1 and G(i) subunit alpha-2. However, according to PubMed:<a href="http://www.uniprot.org/citations/21627585" target="\_blank">21627585</a> can signal through G(i) subunit alpha. Believed to be a class B receptor which internalizes as a complex with arrestin and traffic with it to endosomal vesicles, presumably as desensitized receptor, for extended periods of time. Mediates inhibition of TNF-alpha stimulated JNK phosphorylation via coupling to GNAQ and GNA11; the function involves dissociation of RIPK1 and TRADD from TNFR1. Mediates phosphorylation of nuclear factor NF-kappa-B RELA subunit at 'Ser-536'; the function involves IKBKB and is predominantly independent of G proteins. Involved in cellular migration. Involved in cytoskeletal rearrangement and chemotaxis through beta-arrestin-promoted scaffolds; the function is independent of GNAQ and GNA11 and involves promotion of cofilin dephosphorylation and actin filament severing. Induces redistribution of COPS5 from the plasma membrane to the cytosol and activation of the JNK cascade is mediated by COPS5. Involved in the recruitment of leukocytes to the sites of inflammation and is the major PAR receptor capable of modulating eosinophil function such as pro-inflammatory cytokine secretion, superoxide production and degranulation. During inflammation promotes dendritic cell maturation, trafficking to the lymph nodes and subsequent T-cell activation. Involved in antimicrobial response of innate immune cells; activation enhances phagocytosis of Gram-positive and killing of Gram-negative bacteria. Acts synergistically with interferon-gamma in enhancing antiviral responses. Implicated in a number of acute and chronic inflammatory diseases such as of the joints, lungs, brain, gastrointestinal tract, periodontium, skin, and vascular systems, and in autoimmune disorders. Probably mediates activation of pro-inflammatory and pro-fibrotic responses in fibroblasts, triggered by coagulation factor Xa (F10) (By similarity). Mediates activation of barrier protective signaling responses in endothelial cells, triggered by coagulation factor Xa (F10) (PubMed:<a href="http://www.uniprot.org/citations/22409427" target="\_blank">22409427</a>).

#### **Cellular Location**

Cell membrane; Multi-pass membrane protein.

#### **Tissue Location**

Widely expressed in tissues with especially high levels in pancreas, liver, kidney, small intestine, and colon (PubMed:7556175, PubMed:8615752). Moderate expression is detected in many organs, but none in brain or skeletal muscle (PubMed:7556175, PubMed:8615752). Expressed in endothelial cells (PubMed:23202369)

#### **Volume**

50 µl

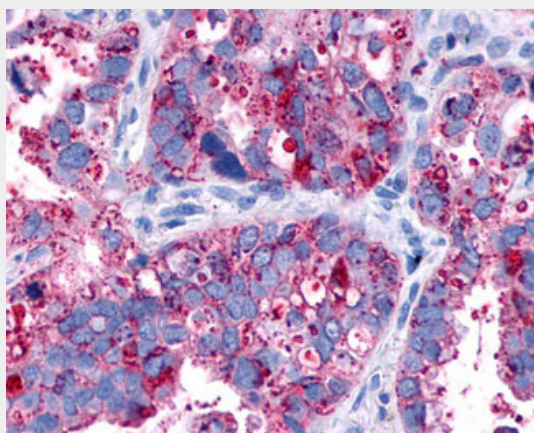
#### **F2RL1 / PAR2 Antibody (N-Terminus) - Protocols**

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

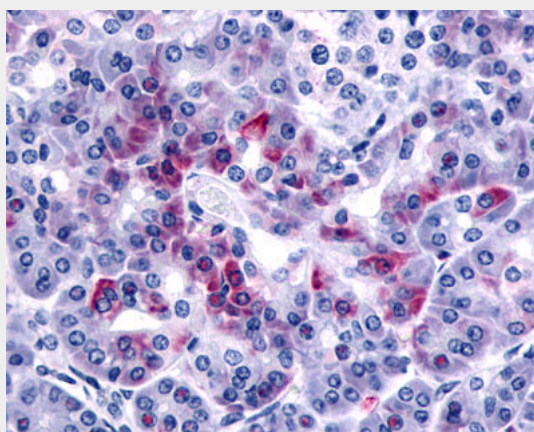
- [Western Blot](#)
- [Blocking Peptides](#)

- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

## **F2RL1 / PAR2 Antibody (N-Terminus) - Images**



Anti-F2RL1 / PAR2 antibody IHC of human Lung, Adenocarcinoma.



Anti-F2RL1 antibody ALS10078 IHC of human pancreas.

## **F2RL1 / PAR2 Antibody (N-Terminus) - Background**

Receptor for trypsin and trypsin-like enzymes coupled to G proteins. Its function is mediated through the activation of several signaling pathways including phospholipase C (PLC), intracellular calcium, mitogen-activated protein kinase (MAPK), I- kappaB kinase/NF-kappaB and Rho. Can also be transactivated by cleaved F2R/PAR1. Involved in modulation of inflammatory responses and regulation of innate and adaptive immunity, and acts as a sensor for proteolytic enzymes generated during infection. Generally is promoting inflammation. Can signal synergistically with TLR4 and probably TLR2 in inflammatory responses and modulates TLR3 signaling. Has a protective role in establishing the endothelial barrier; the activity involves coagulation factor X. Proposed to have a bronchoprotective role in airway epithelium, but also shown to compromise the airway epithelial barrier by interrupting E-cadherin adhesion. Involved in the regulation of vascular tone; activation results in hypotension presumably mediated by vasodilation. Associates with a subset of G proteins alpha subunits such as G alpha-q, G alpha-11, G alpha-14, G alpha-12 and G alpha-13, but probably not with G(o) alpha, G(i) subunit alpha-1 and G(i) subunit alpha-2. However, according to

PubMed:21627585 can signal through G(i) subunit alpha. Believed to be a class B receptor which internalizes as a complex with arrestin and traffic with it to endosomal vesicles, presumably as desensitized receptor, for extended periods of time. Mediates inhibition of TNF-alpha stimulated JNK phosphorylation via coupling to G alpha-q/11; the function involves dissociation of RIPK1 and TRADD from TNFR1. Mediates phosphorylation of nuclear factor NF-kappa-B RELA subunit at 'Ser-536'; the function involves IKBKB and is predominantly independent of G proteins. Involved in cellular migration. Involved in cytoskeletal rearrangement and chemotaxis through beta-arrestin-promoted scaffolds; the function is independent of G alpha-q/11 and involves promotion of cofilin dephosphorylation and actin filament severing. Induces redistribution of COPS5 from the plasma membrane to the cytosol and activation of the JNK cascade is mediated by COPS5. Involved in the recruitment of leukocytes to the sites of inflammation and is the major PAR receptor capable of modulating eosinophil function such as proinflammatory cytokine secretion, superoxide production and degranulation. During inflammation promotes dendritic cell maturation, trafficking to the lymph nodes and subsequent T-cell activation. Involved in antimicrobial response of innate immune cells; activation enhances phagocytosis of Gram- positive and killing of Gram-negative bacteria. Acts synergistically with interferon-gamma in enhancing antiviral responses. Implicated in a number of acute and chronic inflammatory diseases such as of the joints, lungs, brain, gastrointestinal tract, periodontium, skin, and vascular systems, and in autoimmune disorders.

#### **F2RL1 / PAR2 Antibody (N-Terminus) - References**

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Kalnina N.,et al.Submitted (AUG-2003) to the EMBL/GenBank/DDBJ databases.  
Mural R.J.,et al.Submitted (JUL-2005) to the EMBL/GenBank/DDBJ databases.