

**IL-36G Antibody**  
**Catalog # ASC11712****Specification**

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**IL-36G Antibody - Product Information**

Application	WB, IHC-P, IF, E
Primary Accession	<a href="#">Q9NZH8</a>
Other Accession	<a href="#">NP_062564</a> , <a href="#">9665234</a>
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	Predicted: 19 kDa

Application Notes	<b>Observed: 22 kDa KDa</b> <b>IL-36G antibody can be used for detection of IL-36G by Western blot at 1 - 2 µg/ml.</b>
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**IL-36G Antibody - Additional Information**

Gene ID **56300**

**Target/Specificity**

IL36G; IL-36G antibody is human specific. IL-36G antibody will not cross-react with IL-36A or IL-36B.

**Reconstitution & Storage**

IL-36G antibody can be stored at 4°C for three months and -20°C, stable for up to one year.

**Precautions**

IL-36G Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**IL-36G Antibody - Protein Information**

**Name** IL36G ([HGNC:15741](#))

**Function**

Cytokine that binds to and signals through the IL1RL2/IL-36R receptor which in turn activates NF-kappa-B and MAPK signaling pathways in target cells. Part of the IL-36 signaling system that is thought to be present in epithelial barriers and to take part in local inflammatory response; similar to the IL-1 system with which it shares the coreceptor IL1RAP. Seems to be involved in skin inflammatory response by acting on keratinocytes, dendritic cells and indirectly on T-cells to drive tissue infiltration, cell maturation and cell proliferation. In cultured keratinocytes induces the expression of macrophage, T-cell, and neutrophil chemokines, such as CCL3, CCL4, CCL5, CCL2, CCL17, CCL22, CCL20, CCL5, CCL2, CCL17, CCL22, CXCL8, CCL20 and CXCL1; also stimulates its own expression and that of the prototypic cutaneous pro-inflammatory parameters TNF-alpha, S100A7/psoriasin and inducible NOS. May play a role in pro-inflammatory responses during particular neutrophilic airway inflammation: activates mitogen-activated protein kinases and NF-kappa B in primary lung fibroblasts, and stimulates the expression of IL-8 and CXCL3 and Th17

chemokine CCL20 in lung fibroblasts. May be involved in the innate immune response to fungal pathogens, such as *Aspergillus fumigatus*.

#### **Cellular Location**

Cytoplasm. Secreted. Note=The secretion is dependent on protein unfolding and facilitated by the cargo receptor TMED10; it results in protein translocation from the cytoplasm into the ERGIC (endoplasmic reticulum-Golgi intermediate compartment) followed by vesicle entry and secretion.

#### **Tissue Location**

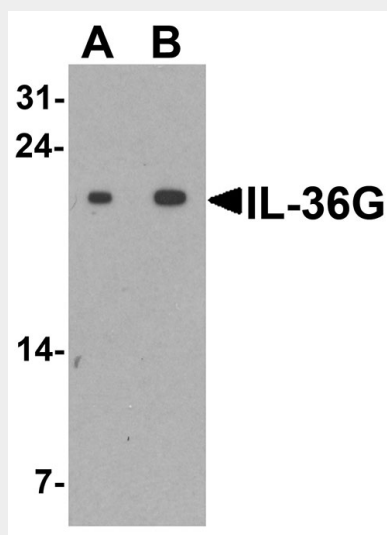
Highly expressed in tissues containing epithelial cells: skin, lung, stomach and esophagus. Expressed in bronchial epithelial. In skin is expressed only in keratinocytes but not in fibroblasts, endothelial cells or melanocytes. Up-regulated in lesional psoriasis skin. Expressed in monocyte-derived dendritic cells and M1 macrophages.

### **IL-36G Antibody - Protocols**

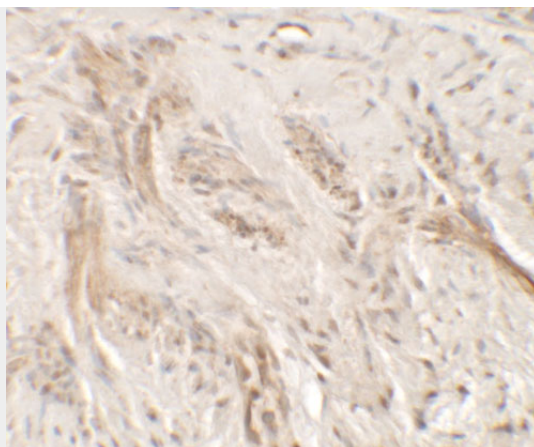
Provided below are standard protocols that you may find useful for product applications.

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

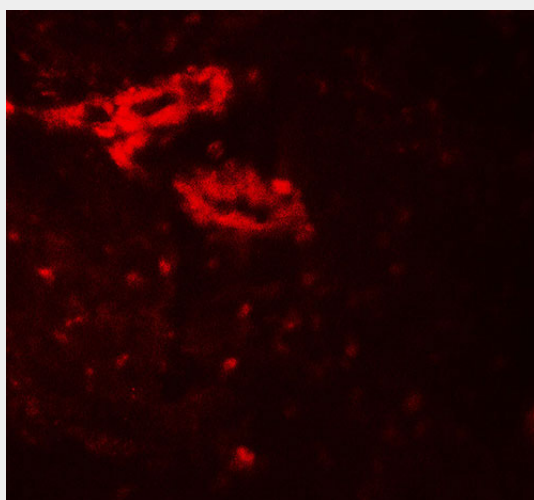
### **IL-36G Antibody - Images**



Western blot analysis of IL-36G in human spleen tissue lysate with IL-36G antibody at (A) 1 and (B) 2 µg/ml.



Immunohistochemistry of IL-36G in human bladder tissue with IL-36G antibody at 5 µg/mL.



Immunofluorescence of IL-36G in human bladder tissue with IL-36G antibody at 20 µg/mL.

### **IL-36G Antibody - Background**

IL-36G is a member of the interleukin 1 cytokine family whose gene and eight other interleukin 1 family genes form a cytokine gene cluster on chromosome 2 (1). IL-36G is thought to activate the NF-kappaB pathway through IL-1 receptor family members IL-1RL2 and IL-1RAcP (2). Like the related proteins IL-36A and IL-36B, IL-36G requires post-translational processing for full agonist activity, but the cleavage mechanism is currently unknown (3). IL-36G expression is stimulated by interferon-gamma, tumor necrosis factor-alpha and interleukin 1 (4). The IL-36 cytokines have been suggested to amplify Th1 responses by enhancing proliferation and Th1 polarization of naive CD4+ T cells (5).

### **IL-36G Antibody - References**

- Smith DE, Renshaw BR, Ketchem RR, et al. Four new members expand the interleukin-1 superfamily. *J. Biol. Chem.* 2000; 275:1169-75.
- Towne JE, Garka KE, Renshaw BR, et al. Interleukin (IL)-1F6, IL-1F8, and IL-1F9 signal through IL-1Rrp2 and IL-1RAcP to activate the pathway leading to NF-kappaB and MAPKs. *J. Biol. Chem.* 2004; 279:13677-88.
- Towne JE, Renshaw BR, Douangpanya J, et al. Interleukin-36 (IL-36) ligands require processing for full agonist (IL-36a, IL-36b, and IL-36g) or antagonist (IL-36Ra) activity. *J. Biol. Chem.* 2011; 286:42594-602.
- Chustz RT, Nagarkar DR, Popowski JA, et al. Regulation and function of the IL-1 family cytokine

IL-1F9 in human bronchial epithelial cells. Am. J. Respir. Cell Mol. Biol. 2011; 45:145-53.