

# **AXL Blocking Peptide (C-term)**

Synthetic peptide Catalog # BP21364b

## **Specification**

## **AXL Blocking Peptide (C-term) - Product Information**

**Primary Accession** 

P30530

# **AXL Blocking Peptide (C-term) - Additional Information**

Gene ID 558

### **Other Names**

Tyrosine-protein kinase receptor UFO, AXL oncogene, AXL, UFO

## Target/Specificity

The synthetic peptide sequence is selected from aa 838-852 of HUMAN AXL

### **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.

## **AXL Blocking Peptide (C-term) - Protein Information**

**Name AXL** 

**Synonyms UFO** 

### **Function**

Receptor tyrosine kinase that transduces signals from the extracellular matrix into the cytoplasm by binding growth factor GAS6 and which is thus regulating many physiological processes including cell survival, cell proliferation, migration and differentiation. Ligand binding at the cell surface induces dimerization and autophosphorylation of AXL. Following activation by ligand, AXL binds and induces tyrosine phosphorylation of PI3-kinase subunits PIK3R1, PIK3R2 and PIK3R3; but also GRB2, PLCG1, LCK and PTPN11. Other downstream substrate candidates for AXL are CBL, NCK2, SOCS1 and TNS2. Recruitment of GRB2 and phosphatidylinositol 3 kinase regulatory subunits by AXL leads to the downstream activation of the AKT kinase. GAS6/AXL signaling plays a role in various processes such as endothelial cell survival during acidification by preventing apoptosis, optimal cytokine signaling during human natural killer cell development, hepatic regeneration, gonadotropin-releasing hormone neuron survival and migration, platelet activation, or regulation of thrombotic responses. Also plays an important role in inhibition of Toll-like receptors (TLRs)-mediated innate immune response.



#### **Cellular Location**

Cell membrane; Single-pass type I membrane protein

#### **Tissue Location**

Highly expressed in metastatic colon tumors. Expressed in primary colon tumors. Weakly expressed in normal colon tissue.

# **AXL Blocking Peptide (C-term) - Protocols**

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

# Blocking Peptides

**AXL Blocking Peptide (C-term) - Images** 

## AXL Blocking Peptide (C-term) - Background

Receptor tyrosine kinase that transduces signals from the extracellular matrix into the cytoplasm by binding growth factor GAS6 and which is thus regulating many physiological processes including cell survival, cell proliferation, migration and differentiation. Ligand binding at the cell surface induces dimerization and autophosphorylation of AXL. Following activation by ligand, ALX binds and induces tyrosine phosphorylation of PI3- kinase subunits PIK3R1, PIK3R2 and PIK3R3; but also GRB2, PLCG1, LCK and PTPN11. Other downstream substrate candidates for AXL are CBL, NCK2, SOCS1 and TENC1. Recruitment of GRB2 and phosphatidylinositol 3 kinase regulatory subunits by AXL leads to the downstream activation of the AKT kinase. GAS6/AXL signaling plays a role in various processes such as endothelial cell survival during acidification by preventing apoptosis, optimal cytokine signaling during human natural killer cell development, hepatic regeneration, gonadotropin-releasing hormone neuron survival and migration, platelet activation, or regulation of thrombotic responses. Plays also an important role in inhibition of Toll-like receptors (TLRs)-mediated innate immune response. In case of filovirus infection, seems to function as a cell entry factor.

## **AXL Blocking Peptide (C-term) - References**

Partanen J., et al. Proc. Natl. Acad. Sci. U.S.A. 87:8913-8917(1990). O'Bryan J.P., et al. Mol. Cell. Biol. 11:5016-5031(1991). Janssen J.W.G., et al. Oncogene 6:2113-2120(1991). Grimwood I., et al. Nature 428:529-535(2004). Lee S.-T., et al. Oncogene 8:3403-3410(1993).