

Kallikrein 6 Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP6325a**Specification**

Kallikrein 6 Antibody (N-term) Blocking peptide - Product InformationPrimary Accession [Q92876](#)**Kallikrein 6 Antibody (N-term) Blocking peptide - Additional Information****Gene ID** 5653**Other Names**

Kallikrein-6, 3421-, Neurosin, Protease M, SP59, Serine protease 18, Serine protease 9, Zyme, KLK6, PRSS18, PRSS9

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP6325a](/product/products/AP6325a) was selected from the N-term region of human KLK6. 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.

Kallikrein 6 Antibody (N-term) Blocking peptide - Protein Information**Name** KLK6**Synonyms** PRSS18, PRSS9**Function**

Serine protease which exhibits a preference for Arg over Lys in the substrate P1 position and for Ser or Pro in the P2 position. Shows activity against amyloid precursor protein, myelin basic protein, gelatin, casein and extracellular matrix proteins such as fibronectin, laminin, vitronectin and collagen. Degrades alpha-synuclein and prevents its polymerization, indicating that it may be involved in the pathogenesis of Parkinson disease and other synucleinopathies. May be involved in regulation of axon outgrowth following spinal cord injury. Tumor cells treated with a neutralizing KLK6 antibody migrate less than control cells, suggesting a role in invasion and metastasis.

Cellular Location

Secreted. Nucleus, nucleolus. Cytoplasm. Mitochondrion. Microsome. Note=In brain, detected in the nucleus of glial cells and in the nucleus and cytoplasm of neurons. Detected in the mitochondrial and microsomal fractions of HEK-293 cells and released into the cytoplasm following cell stress

Tissue Location

In fluids, highest levels found in milk of lactating women followed by cerebrospinal fluid, nipple aspirate fluid and breast cyst fluid. Also found in serum, seminal plasma and some amniotic fluids and breast tumor cytosolic extracts. Not detected in urine. At the tissue level, highest concentrations found in glandular tissues such as salivary glands followed by lung, colon, fallopian tube, placenta, breast, pituitary and kidney. Not detected in skin, spleen, bone, thyroid, heart, ureter, liver, muscle, endometrium, testis, pancreas, seminal vesicle, ovary, adrenals and prostate. In brain, detected in gray matter neurons (at protein level). Colocalizes with pathological inclusions such as Lewy bodies and glial cytoplasmic inclusions. Overexpressed in primary breast tumors but not expressed in metastatic tumors.

Kallikrein 6 Antibody (N-term) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

Kallikrein 6 Antibody (N-term) Blocking peptide - Images

Kallikrein 6 Antibody (N-term) Blocking peptide - Background

Kallikreins are a subgroup of serine proteases having diverse physiological functions. Growing evidence suggests that many kallikreins are implicated in carcinogenesis and some have potential as novel cancer and other disease biomarkers. The KLK6 enzyme is regulated by steroid hormones. In tissue culture, the enzyme has been found to generate amyloidogenic fragments from the amyloid precursor protein, suggesting a potential for involvement in Alzheimer's disease.

Kallikrein 6 Antibody (N-term) Blocking peptide - References

Christophi, G.P., et al., J. Neurochem. 91(6):1439-1449 (2004). Bayes, A., et al., Biol. Chem. 385(6):517-524 (2004). Pampalakis, G., et al., Biochem. Biophys. Res. Commun. 320(1):54-61 (2004). Ghosh, M.C., et al., Tumour Biol. 25(4):193-199 (2004). Sauter, E.R., et al., Int. J. Cancer 108(4):588-591 (2004).