**Polyclonal Antibody** Catalog #: APRab09002



# **Summary**

Cleaved-Kininogen-1 HC (K380) Rabbit Polyclonal Antibody **Production Name** 

Description Rabbit Polyclonal Antibody

Rabbit Host **Application** WB

Reactivity Human, Rat, Mouse

### **Performance**

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

# **Immunogen**

**Gene Name** KNG1

KNG1; BDK; KNG; Kininogen-1; Alpha-2-thiol proteinase inhibitor; Fitzgerald factor; **Alternative Names** 

High molecular weight kininogen; HMWK; Williams-Fitzgerald-Flaujeac factor

**Gene ID** 3827.0

P01042.Synthesized peptide derived from Cleaved-Kininogen-1 HC (K380) . at AA

range: 300-380

# **Application**

SwissProt ID

**Dilution Ratio** WB 1:500-1:2000. ELISA: 1:10000.

**Molecular Weight** 41kD

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# **Background**

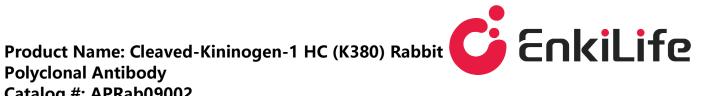
This gene uses alternative splicing to generate two different proteins- high molecular weight kiningeen (HMWK) and low molecular weight kiningen (LMWK). HMWK is essential for blood coagulation and assembly of the kallikrein-kinin system. Also, bradykinin, a peptide causing numerous physiological effects, is released from HMWK. Bradykinin also functions as an antimicrobial peptide with antibacterial and antifungal activity. In contrast to HMWK, LMWK is not involved in blood coagulation. Three transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Nov 2014], disease: Defects in KNG1 are the cause of high molecular weight kiningen deficiency (HMWK deficiency) [MIM:228960]. HMWK deficiency is an autosomal recessive coagulation defect. Patients with HWMK deficiency do not have a hemorrhagic tendency, but they exhibit abnormal surface-mediated activation of fibrinolysis., function:(1) Kininogens are inhibitors of thiol proteases; (2) HMW-kiningen plays an important role in blood coagulation by helping to position optimally prekallikrein and factor XI next to factor XII; (3) HMW-kiningen inhibits the thrombin- and plasmin-induced aggregation of thrombocytes; (4) the active peptide bradykinin that is released from HMW-kininogen shows a variety of physiological effects: (4A) influence in smooth muscle contraction, (4B) induction of hypotension, (4C) natriuresis and diuresis, (4D) decrease in blood glucose level, (4E) it is a mediator of inflammation and causes (4E1) increase in vascular permeability, (4E2) stimulation of nociceptors (4E3) release of other mediators of inflammation (e.g. prostaglandins), (4F) it has a cardioprotective effect (directly via bradykinin action, indirectly via endothelium-derived relaxing factor action); (5) LMW-kiningen inhibits the aggregation of thrombocytes; (6) LMW-kiningen is in contrast to HMW-kiningen not involved in blood clotting, online information: High molecular weight kiningen entry, polymorphism: The T-kinin peptide is missing residues 378 to 380, probably as a result of a naturally occurring variant. The complete sequence of the T-kinin peptide is therefore ISRPPGFSPFR. This peptide is associated with malignant tumors but not with benign ones., PTM:Bradykinin is released from kininogen by plasma kallikrein., PTM:Hydroxylation of Pro-383 occurs prior to the release of bradykinin., similarity: Contains 3 cystatin domains, tissue specificity: Plasma. T-kinin is detected in malignant ovarian, colon and breast carcinomas, but not in benign tumors.,

## **Research Area**

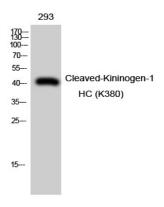
Complement and coagulation cascades;

#### **Image Data**

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Western Blot analysis of 293 cells using Cleaved-Kininogen-1 HC (K380) Polyclonal Antibody

### Note

For research use only.

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