

Monoclonal Anti-Human Heparanase 1 (HPA1) Antibody Clone HP3/17

Catalog Number: *INS-26-1-0000-10* (50 µg)
INS-26-1-0000-11 (100 µg)
INS-26-1-0000-12 (150 µg)

BACKGROUND

Heparanase is an endo-β-D-glucuronidase, which degrades heparan sulfate side chains of heparan sulfate proteoglycans (HSPGs) in the extracellular matrix. Heparanase plays an important role in ECM degradation, facilitating the migration and extravasation of tumor cells and inflammatory leukocytes (1,2,3). Upon degradation, heparanase releases growth factors and cytokines that stimulate cell proliferation and chemotaxis (4,5).

Heparanase is a heterodimer comprised of a 50 kDa subunit harboring the active site and a 8 kDa subunit. It is produced as a latent 65 kDa precursor and proteolytically processed to its active form (1,6).

Heparanase is highly expressed in myeloid leukocytes (i.e. neutrophils) in platelets and in human placenta. Human heparanase was found to be upregulated in various types of primary tumors, correlating in some cases with increased tumor invasiveness and vascularity and with poor prospective survival (7,8).

SOURCE

Mab HP3/17 is a Protein G affinity purified monoclonal antibody raised against a polypeptide from the 50 kDa subunit of Heparanase.

Ig CLASS

Mouse IgG_{2Bκ}

PRODUCT

Each vial contains 50, 100 or 150 µg in 12.5, 25 or 37.5 µl respectively, of 0.22 micron filtered solution of 20 mM Sodium Phosphate; 150 mM NaCl; pH 7.2, containing 0.01% Thimerosal.

APPLICATIONS

Western blot
Immunohistochemistry

SPECIFICITY

HP3/17 reacts with the 50 kDa subunit and with the 65 kDa precursor of human or mouse Heparanase by Western blotting and immunohistochemistry.

Recommended dilution range for Western blot analysis: 1:4000.

Recommended dilution range for immunohistochemistry: 1:40.

PURITY

>98% on SDS-PAGE when loaded 50 µg/lane.

STORAGE

Store at 4°C. Stable for six months from the date of shipment. For extended storage, freeze in working aliquots at -20°C. Avoid repeated freeze-thaw cycles.

RESEARCH USE

For *in vitro* research use only. Not for use in diagnostic procedures.

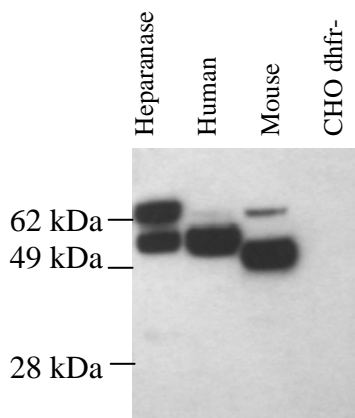
PATENTS

Anti-heparanase antibodies and their uses, including HP3/17 and its uses, are protected by US. Patents No. 6,177,545; 6,531,129, additional US patent applications and patents and patent applications worldwide.

REFERENCES

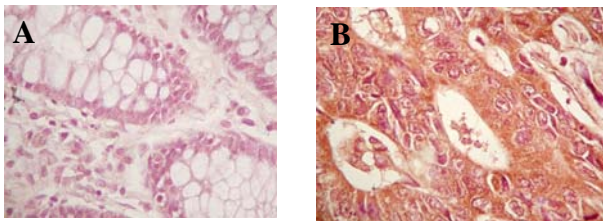
1. I. Vlodayvsky, Y. Friedmann, M. Elkin, H. Aingorn, R. Atzmon, R. Ishai-Michaeli, M. Bitan, O. Pappo, T. Peretz, I. Michal, L. Spector, I. Pecker. 1999. Mammalian heparanase: gene cloning, expression and function in tumor progression and metastasis. *Nat. Med.* **5**: 793–802.
2. I. Vlodayvsky, Y. Friedman. 2001. Molecular properties and involvement of heparanase in cancer metastasis and angiogenesis. *J. Clin. Invest.* **108**: 341–347.
3. C.R. Parish, C. Freeman, M.D. Hulett. 2001. Heparanase: a key enzyme involved in cell invasion. *Biochem. Biophys. Acta* **1471**: M99–M108.
4. I. Vlodayvsky, G. Korner, R. Ishai-Michaeli, P. Bashkin, R. Bar-Shavit, Z. Fuks, 1990. Extracellular matrix-resident growth factors and enzyme: Possible involvement in tumor metastasis and angiogenesis. *Cancer Metastasis Rev.* **9**: 203–226.
5. P. Bashkin, S. Doctrow, M. Klagsbrun, C.M. Svahn, J. Folkman, I. Vlodayvsky. 1989. Basic fibroblast growth factor binds to subendothelial extracellular matrix and is released by heparitinase and heparin-like molecules. *Biochemistry* **28**: 1737–1743.
6. M.B. Fairbanks, A.M. Mildner, J.W. Leone, G.S. Cavey, W.R. Mathews, R.F. Drong, J.L. Slightom, M.J. Bienkowski, C.W. Smith, C.A. Bannow, R.L. Heinrikson. 1999. Processing of the human heparanase precursor and evidence that the active enzyme is a heterodimer. *J. Biol. Chem.* **274**: 29587–29590.
7. A. Koliopanos, H. Friess, J. Klee., X. Shi, Q. Liao, I. Pecker, I. Vlodayvsky, A. Zimmermann, M.W. Buchler. 1992. Heparanase expression in primary and metastatic pancreatic cancer. *Cancer Res.* **61**: 4655–4659.
8. K. Gohji, H. Hirano, M. Okamoto, S. Kitazawa, M. Toyoshima, J. Dong, Y. Katsuoka, M. Nakajima. 2001. Expression of three extracellular matrix degradative enzymes in bladder cancer. *Int. J. Cancer* **95**: 295–301.

PERFORMANCE

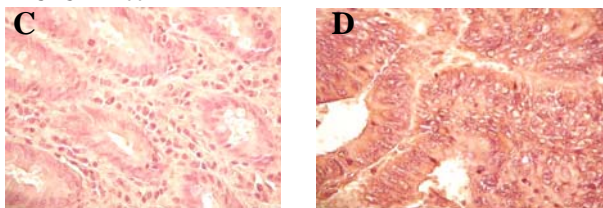


An extract from 2×10^4 CHO cells, transfected with the human or the mouse Heparanases, and an extract from 5×10^5 non-transfected CHO dhfr⁻ cells, were loaded onto 4-12% SDS-PAGE. The proteins were transferred to a PVDF membrane and subjected to Western blot analysis using 1 µg/ml of HP3/17. Purified recombinant human heparanase served as a control (left lane), where the 65 kDa precursor and the 50 kDa subunits are clearly detected.

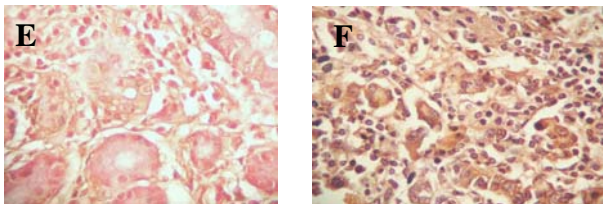
COLON x400



RECTUM x200

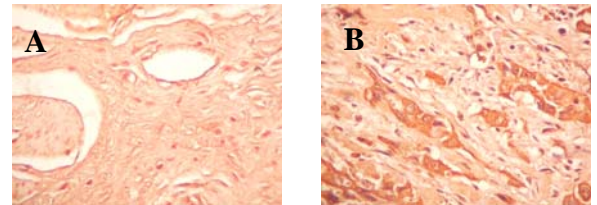


STOMACH x400

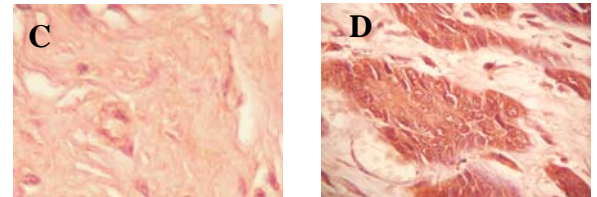


Paraffin sections of human colon (B), rectal (D) and stomach (E) adenocarcinoma, and normal counter-tissues from the same patient (A, C, E) were immuno-stained with 100 µg/ml of HP3/17.

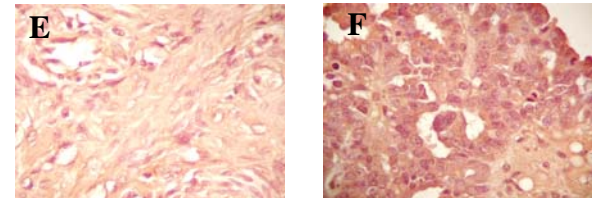
ESOPHAGUS x200



ESOPHAGUS x400

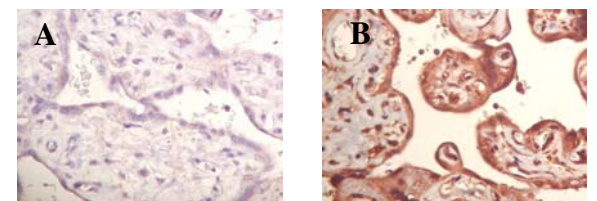


OVARY x400



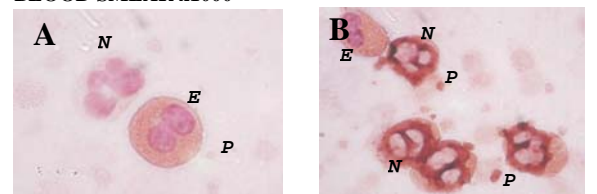
Paraffin sections of human esophageal squamous cell carcinoma (B and D), ovarian cystadenocarcinoma (F) and normal counter-tissues from the same patient (A, C, E) were immuno-stained with 100 µg/ml of HP3/17.

HUMAN PLACENTA x400



Paraffin sections of human placenta were stained with monoclonal human anti mouse IgG3 (A) or with HP3/17 (B) (x400).

BLOOD SMEAR x1000



Human blood smears were stained with 10 µg/ml of HP3/17 (B) or without 1^o mAb (A). Note the strong staining of neutrophils (N) and platelets (P), while eosinophils (E) and lymphocytes are not stained.