SANTA CRUZ BIOTECHNOLOGY, INC.

# cathepsin K (E-7): sc-48353

SANTA CRUZ BIOTECHNOLOGY

*The Power to Question*

## BACKGROUND

The cathepsin family of proteolytic enzymes contains several diverse classes of proteases. The cysteine protease class comprises cathepsins B, L, H, K, S, and O. The aspartyl protease class is composed of cathepsins D and E. Cathepsin G is in the serine protease class. Most cathepsins are lysosomal and each is involved in cellular metabolism, participating in various events such as peptide biosynthesis and protein degradation. Cathepsin K expression is highest in bone, cartilage and skeletal muscle. The strongest mRNA levels are revealed in osteoclasts.

## CHROMOSOMAL LOCATION

Genetic locus: CTSK (human) mapping to 1q21.3; Ctsk (mouse) mapping to 3 F2.1.

## SOURCE

cathepsin K (E-7) is a mouse monoclonal antibody raised against amino acids 191-240 mapping within an internal region of cathepsin K of human origin.

## PRODUCT

Each vial contains 200 µg $IgG_3$ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

cathepsin K (E-7) is available conjugated to agarose (sc-48353 AC), 500 µg/ 0.25 ml agarose in 1 ml, for IP; to HRP (sc-48353 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; and to either phycoerythrin (sc-48353 PE), fluorescein (sc-48353 FITC), Alexa Fluor® 488 (sc-48353 AF488) or Alexa Fluor® 647 (sc-48353 AF647), 200 µg/ml, for IF, IHC(P) and FCM.

Alexa Fluor® is a trademark of Molecular Probes, Inc., Oregon, USA

## APPLICATIONS

cathepsin K (E-7) is recommended for detection of cathepsin K of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for cathepsin K siRNA (h): sc-29936, cathepsin K siRNA (m): sc-29937, cathepsin K shRNA Plasmid (h): sc-29936-SH, cathepsin K shRNA Plasmid (m): sc-29937-SH, cathepsin K shRNA (h) Lentiviral Particles: sc-29936-V and cathepsin K shRNA (m) Lentiviral Particles: sc-29937-V.

Molecular Weight of cathepsin K: 39 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204, NAMALWA cell lysate: sc-2234 or 3611-RF whole cell lysate: sc-2215.

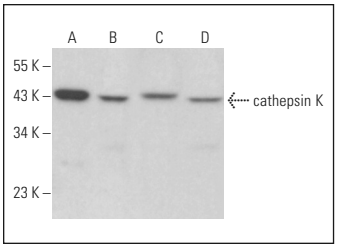
## STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

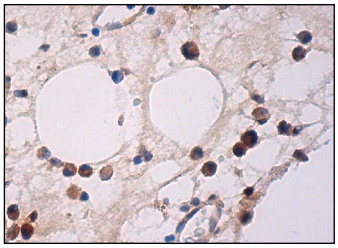
## RESEARCH USE

For research use only, not for use in diagnostic procedures.

## DATA




cathepsin K (E-7): sc-48353. Western blot analysis of cathepsin K expression in Jurkat (**A**), NAMALWA (**B**), 3611-RF (**C**) and K-562 (**D**) whole cell lysates.



cathepsin K (E-7): sc-48353. Immunoperoxidase staining of formalin fixed, paraffin-embedded human bone marrow tissue showing cytoplasmic staining of hematopoietic cells.

## SELECT PRODUCT CITATIONS


1. Kitami, S., et al. 2010. IL-17A suppresses the expression of bone resorption-related proteinases and osteoclast differentiation via IL-17RA or IL-17RC receptors in RAW 264.7 cells. Biochimie 92: 398-404.
2. Szewczyk, K.A., et al. 2013. Distinctive subdomains in the resorbing surface of osteoclasts. PLoS ONE 8: e60285.
3. Touaitahuata, H., et al. 2014. The mineral dissolution function of osteoclasts is dispensable for hypertrophic cartilage degradation during long bone development and growth. Dev. Biol. 393: 57-70.
4. Dapunt, U., et al. 2015. Neutrophil-derived MRP-14 is up-regulated in infectious osteomyelitis and stimulates osteoclast generation. J. Leukoc. Biol. 98: 575-582.
5. Bian, Q., et al. 2016. Excessive activation of TGFβ by spinal instability causes vertebral endplate sclerosis. Sci. Rep. 6: 27093.
6. Wu, M., et al. 2017. $G_{\alpha\ 13}$ negatively controls osteoclastogenesis through inhibition of the Akt-GSK3β-NFATc1 signalling pathway. Nat. Commun. 8: 13700.
7. Zach, F., et al. 2018. p62/sequestosome 1 deficiency accelerates osteoclastogenesis *in vitro* and leads to Paget's disease-like bone phenotypes in mice. J. Biol. Chem. 293: 9530-9541.
8. Bai, M., et al. 2019. Targeted genetic screening in mice through haploid embryonic stem cells identifies critical genes in bone development. PLoS Biol. 17: e3000350.
9. Zhang, Y., et al. 2020. SENP3 suppresses osteoclastogenesis by de-conjugating SUMO2/3 from IRF8 in bone marrow-derived monocytes. Cell Rep. 30: 1951-1963.e4.


## PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.