

## Product Information

**Anti-Cathepsin B antibody, Mouse monoclonal**  
clone CB59-4B11, purified from hybridoma cell culture

Product Number **C6243**

### Product Description

Anti-Cathepsin B antibody, Mouse monoclonal (mouse IgG1 isotype) is derived from the hybridoma CB59-4B11 produced by the fusion of mouse myeloma cells (P3X63AG8.653) and splenocytes from BALB/c mice immunized with recombinant human cathepsin B (Gene ID: 1508). The isotype is determined using a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents, Product Number ISO2. The antibody is purified from culture supernatant of hybridoma cells grown in a bioreactor.

Monoclonal Anti-Cathepsin B specifically recognizes human<sup>1</sup> and rat<sup>1</sup> cathepsin B (~ 25 kDa). The antibody epitope resides within the peptide sequence EPGYSP which corresponds to amino acids 212-217 of human cathepsin B. The antibody does not cross react with the closely related lysosomal cysteine proteases: cathepsins L, H, K, S, V, and W. Applications include ELISA, immunoblotting,<sup>1</sup> and immunohistology.

Cathepsins are lysosomal proteases that play an important role in the intracellular degradation of exogenous and endogenous proteins, activation of enzyme precursors, and tumor invasion and metastasis. They are normally localized in lysosomes of almost all mammalian cells, but under certain conditions they can be secreted from the cell.<sup>2-5</sup>

Cathepsin B is both an endo- and exopeptidase, composed of a dimer of disulfide-linked heavy and light chains, both produced from a single protein precursor. It is involved in the proteolysis of many proteins involved in different physiological and pathological pathways.<sup>4</sup> In the brain, this protease, also known as amyloid precursor protein secretase, is responsible for the proteolytic processing of amyloid precursor protein (APP). An incomplete proteolytic processing of APP has been found as a causative factor in Alzheimer disease. Furthermore, animals with ablated genes for cathepsin D or simultaneous ablation of genes for cathepsin B and L were found to die due to massive neuronal cell death, presumably as a consequence of defects in the terminal steps of autophagy.<sup>6</sup> Cathepsin B has been implicated in tumor progression by virtue of its increased mRNA and protein levels, as well as its

localization at the invading front of the tumor. For example, blocking cathepsin B expression in human glioblastoma SNB19 cells by a stable transfection of an expression vector expressing antisense cathepsin B, caused suppression of glioblastoma-induced neovascularization.<sup>7</sup>

### Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody concentration: ~1.5 mg/mL

### Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

### Storage/Stability

For extended storage, freeze at -20 °C in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

### Product Profile

**Immunoblotting:** a working concentration of 4-8 µg/mL is recommended using total cell extracts of HCT-116 cells.

**Note:** In order to obtain the best results using various techniques and preparations, we recommend determining optimal working dilutions by titration.

### References

1. Di, Piazza., et al., *J. Virol.*, **81**, 4186-4198 (2007).
2. Weber, E., et al., *Hybridoma*, **16**, 159-165 (1997).
3. Tolosa, E., et al., *J. Clin. Invest.*, **112**, 517-526 (2003).
4. Turk, B., and Stoka, V., *FEBS Lett.*, **581**, 2761-2767 (2007).

5. Igdoura, S.A., et al., *J. Histochem. Cytochem.*, **43**, 545-557 (1995).
6. Koike, M., et al., *Am. J. Pathol.*, **167**, 1713-1728 (2005).

7. Yanamandra, N., et al., *Oncogene*, **23**, 2224-2230 (2004).

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