

Product Information

Monoclonal Anti-Calreticulin, Clone TO-11 produced in mouse, tissue culture supernatant

Product Number **C7492**

Product Description

Monoclonal Anti-Calreticulin (mouse IgG1 isotype) is derived from the hybridoma TO-11 produced by the fusion of mouse myeloma cells (P3-X63-AG8.653 cells) and splenocytes from BALB/c mice immunized with a synthetic peptide corresponding to a fragment of human calreticulin, conjugated to KLH.¹ The isotype is determined using a double diffusion immunoassay using Mouse Monoclonal Antibody Isotyping Reagents, Product Number ISO2.

Monoclonal Anti-Calreticulin recognizes human^{1,2} calreticulin. The antibody may be used in ELISA,¹ immunoblotting (~50 kDa),¹ immunohistochemistry,^{1,2} flow cytometry,¹ and immunocytochemistry.¹

Newly synthesized cellular and extracellular proteins must be correctly folded and assembled in the ER before they progress to the cytosol or the cell surface. This process is facilitated by transient interaction with a specific set of molecular chaperones that reside in the ER lumen including calnexin, calreticulin, protein disulfide isomerase (PDI), and molecular chaperones of the Hsp60, Hsp70, and Hsp90 families.

Calreticulin acts as a lectin-like chaperone binding oligosaccharide residues of newly synthesized N-linked glycoproteins, and misfolded proteins.^{3,4} Calreticulin (Calregulin) is a 60 kDa, calcium-binding chaperone, that is localized primarily in the lumen of the endoplasmic reticulum (ER).¹⁻⁴ It is believed to play a critical role in quality control processes during protein synthesis and folding. The lectin specificity of calreticulin has been identified as high mannose oligosaccharides terminating in monoglucosyl residues linked through $\alpha 1 \rightarrow 3$.³⁻⁵ Increased expression of calreticulin increases Ca^{2+} storage capacity of the ER. It also appears to modulate store-operated Ca^{2+} -influx, and to alter Ca^{2+} transport by the sarcoplasmic/ER Ca^{2+} -ATPase (SERCA).^{4,6} Overexpression of calreticulin results in increased sensitivity of HeLa cells to drug-induced apoptosis.⁴ In contrast, calreticulin-deficient cells show increased resistance to apoptosis.

Calreticulin gene disruption in mice (*crt*^{-/-}), is embryonic lethal showing a marked impairment of cardiac development, indicating that calreticulin is essential for proper cardiac development.^{7,8} In *crt*^{-/-} cells, agonist induced Ca^{2+} release via the inositol 1,4,5-triphosphate (InsP3) pathway is also inhibited, and the ER has lower capacity for Ca^{2+} storage^{6,7} indicating that calreticulin, in addition to its chaperone activity, plays a critical role in Ca^{2+} homeostasis. Calreticulin loss of function has also been shown to enhance the ubiquitin-proteasome activity, which could function as a compensatory mechanism for cell survival.⁹

Reagent

Supplied as a tissue culture supernatant containing 15 mM sodium azide as a preservative.

Precautions and Disclaimer

For R&D use only. Not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2–8 °C for up to one month. 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 dilution of 1:50-1:100 is recommended using total cell extract of HeLa cells.

Note: In order to obtain best results in various techniques and preparations, it is recommended to determine optimal working dilutions by titration.

Exposure to sensitive film is recommended.

References

1. Ogino, T., et al., *Tissue Ant.*, **62**, 385-393 (2003).
2. Ogino, T., et al., *Clin. Can. Res.*, **9**, 4043-4051 (2003).
3. Gelbert, P., et al., *Int. J. Biochem. Cell Biol.*, **37**, 260-266 (2005).
4. Johnson, S., et al., *Trends Cell Biol.*, **11**, 122-129 (2001).
5. Helenius, A., and Aebi, M., *Ann. Rev. Biochem.*, **73**, 1019-1049 (2004).
6. Bedard, K., et al., *Int. Rev. Cytol.*, **245**, 91-121 (2005).
7. John, L.M., et al., *J. Cell Biol.*, **142**, 963-973 (1998).
8. Nakamura, K., et al., *J. Cell Biol.*, **150**, 731-740 (2000).
9. Uvarov, A.V., and Mesaeli, N., *Biochem. Biophys. Acta*, **1783**, 1237-1247 (2008).

VS,EK,GG,KAA,PHC,MAAM 08/19-1