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Product Information

Anti-HRD1/SYVN1 antibody, Mouse monoclonal
clone HRD1-5, purified from hybridoma cell culture

Catalog Number **SAB4200423**

Product Description

Monoclonal Anti-HRD1/SYVN1 (mouse IgG1 isotype) is derived from the hybridoma HRD1-5 produced by the fusion of mouse myeloma cells and splenocytes from BALB/c mice immunized with a synthetic peptide corresponding to the C-terminal region of human HRD1/SYVN1 (GeneID: 84447), conjugated to KLH. The corresponding sequence differs by two amino acids in rat and mouse. The isotype is determined by ELISA using Mouse Monoclonal Antibody Isotyping Reagents, Catalog Number ISO2. The antibody is purified from culture supernatant of hybridoma cells grown in a bioreactor.

Monoclonal Anti-HRD1/SYVN1 recognizes human HRD1/SYVN1. The antibody may be used in several immunochemical techniques including immunoblotting (~70 kDa) and immunoprecipitation. Detection of the HRD1/SYVN1 band by immunoblotting is specifically inhibited by the immunizing peptide.

The mammalian homolog of yeast Hrd1p/Der3p, HRD1, also named synoviolin (SYVN1), is an ER-membrane resident E3 ubiquitin ligase. HRD1 protects against ER stress-induced apoptosis through ER stress-associated protein degradation (ERAD).[1,2] Quality control in the ER is regulated by productive folding and ERAD mechanisms. During ERAD, misfolded proteins accumulated in the ER are transferred to the cytosol, where they are destroyed by the ubiquitin-proteasome system.[3] Accelerated refolding and degradation of unfolded proteins are induced in response to ER stress by the UPR transcriptional program.[4]

HRD1 expression is strongly induced by ER stress.[5] HRD1 has a five-transmembrane domain, a RING-finger domain that mediates the transfer of ubiquitin from E2 to substrates, and a proline-rich domain.[1] HRD1 interacts with Pael-R, a substrate of Parkin, through its proline-rich domain, promoting Pael-R degradation, and thus protects neurons from cell death caused by the accumulation of Pael-R.[6] HRD1 was also found to enhance the degradation and to suppress the toxicity of polyglutamine-expanded huntingtin.[7]

In addition to its role in ERAD, HRD1 targets the p53 tumor suppressor gene for proteasomal degradation.[8] Overexpression of HRD1 is implicated in the pathogenesis of rheumatoid arthritis.[9]

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: ~1.0 mg/mL

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 concentration of 2–4 µg/mL is recommended using whole extracts of HEK-293T cells overexpressing human HRD1/SYVN1.

Immunoprecipitation: a working amount of 5–10 µg is recommended using lysates of human HEK-293T cells.

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

References

1. Kaneko, M. et al., *FEBS Lett*., **532**, 147-152 (2002).
2. Kaneko, M., and Nomura, Y., *Life Sci.*, **74**, 199-205 (2003).
3. Meusser, B. et al., *Nature Cell Biol.*, **7**, 766-772 (2005).
4. Kostova, Z., and Wolf, D.H., *EMBO J*., **22**, 2309-2317 (2003).
5. Kaneko, M. et al., *FEBS Lett*., **581**, 5355-5360 (2007).
6. Omura, T. et al., *J. Neurosci. Res.*, **86**, 1577-1587 (2008).
7. Yang, H. et al., *Exp. Cell Res*., **313**, 538-550 (2006).
8. Yamasaki, S. et al., *EMBO J*., **26**, 113-122 (2007).
9. Amano, T. et al., *Genes Dev*., **17**, 2436-2449 (2003).

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