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Product Information

Monoclonal Anti-HAVCR1 antibody produced in mouse
clone KM-23, purified from hybridoma cell culture

Product Number **SAB4200677**

Product Description

Monoclonal Anti-HAVCR1 (mouse IgG2a isotype) is derived from the KM-23 hybridoma produced by the fusion of mouse myeloma cells and splenocytes from mouse BALB/c mice immunized with a synthetic peptide corresponding to a sequence at the internal region of human HAVCR1 (GeneID: 26762), conjugated to KLH. The isotype is determined by ELISA using Mouse Monoclonal Antibody Isotyping Reagents, Product Number ISO2. The antibody is purified from cell culture supernatant of hybridoma cells.

Monoclonal Anti-HAVCR1 recognizes human and rat HAVCR1. The antibody may be used in various immunochemical techniques including Immunoblotting (~38kDa), Immunofluorescence and Immunohistochemistry. Detection of the HAVCR1 band by Immunoblotting is specifically inhibited by the immunogen.

HAVCR1 (Hepatitis A virus cellular receptor 1) also known as KIM1 (Kidney injury molecule 1) and TIM-1 (T-cell immunoglobulin mucin receptor 1), belongs to the TIM-family proteins. The latter are classical type I transmembrane proteins, with an extracellular N terminus containing the variable Ig-like (IgV) domain, a highly conserved PS binding site and cytosolic C-terminal tail.[1] Human HAVCR1 was initially discovered as the receptor for hepatitis A virus (HAV). HAVCR1 enhances infection of several virus types such as filoviruses, alphaviruses, flaviviruses and arenaviruses due to its involvement in glycoproteins dependent and independent attachment of viral particles to cell membrane. This is carried by direct interaction of HAVCR1 with phosphatidylserine (PS) on the viral envelope.[2] Conversely, HAVCR1 was also reported to inhibit HIV-1 replication in $CD4^+$ T cells and inhibit additional viruses release, including murine leukemia (MLV) virus and Ebola virus (EBOV) through its PS receptor.[1,2]

Human HAVCR1 is predominantly expressed in epithelial and T helper 2 (T_H2) cells and is involved in cell proliferation and apoptotic body uptake.[1] HAVCR1 expression is increased in proximal tubule epithelial cells in the kidney during ischemic acute renal failure.[3-4]

Following exposure to nephrotoxicants, HAVCR1 presence in the urine is upregulated thus it has a potential role as general biomarker for tubular injury and repair processes.[4-5]

Specific antibody to HAVCR1 may prevent membrane fusion and inhibit viral cellular entry[2] but needs to take in mind HAVCR1 role in blocking viral release.[1]

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

This product is 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 in working aliquots. Repeated freezing and thawing 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 extract of human 769-P cells.

Immunohistochemistry: a working concentration of 10 μg/mL is recommended using heat-retrieved formalin-fixed, paraffin-embedded human kidney sections and Biotin/ExtrAvidin®-Peroxidase staining system.

Immunofluorescence: a working concentration of 1-2 μg/mL is recommended using human 769-P cells.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

References

1. Li M., et al., *Proc Natl Acad Sci U S A*., **111**, E3699-707 (2014).
2. Kuroda M., et al., *J Virol.*, **89**, 6481-93 (2015).
3. Bailly V., et al., *J Biol Chem.*, **277**, 39739-48 (2002).
4. Ichimura T., et al., *Am J Physiol Renal Physiol.*, **286**, F552-63 (2004).
5. Vaidya VS., et al., *Kidney Int.*, **76**, 108-14 (2009).

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