

**RHAMM/CD168 Antibody**

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**For Research Use Only. Not for Use in Diagnostic Procedures.**

<b>Applications:</b> W, IP	<b>Reactivity:</b> H	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 85	<b>Source/Isotype:</b> Rabbit	<b>UniProt ID:</b> #O75330	<b>Entrez-Gene Id:</b> 3161
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**Product Usage Information****Application**

Western Blotting  
Immunoprecipitation

**Dilution**

1:1000  
1:100

**Storage**

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

**Specificity/Sensitivity**

RHAMM/CD168 Antibody recognizes endogenous levels of total RHAMM/CD168 protein.

**Source / Purification**

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the carboxy terminus of human RHAMM/CD168 protein. The antigenic peptide spans a region that is 100% conserved among the four isoforms of RHAMM/CD168 reported in Uniprot. Antibodies are purified by protein A and peptide affinity chromatography.

**Background**

Receptor for Hyaluronic acid-Mediated Motility (RHAMM, known also as CD168 or HMMR) was first identified as a putative receptor for hyaluronic acid (HA) that modulated HA-mediated cell motility (1). RHAMM/CD168 is functionally similar to the HA receptor CD44; however in contrast to CD44, RHAMM/CD168 does not contain a transmembrane domain or a signal peptide leader sequence, and so is not targeted exclusively to the cell membrane (1). RHAMM/CD168 has multiple isoforms; some are reportedly exported to the cell membrane in response to signaling by growth factors and cytokines (e.g., TGF-β) (2, 3), whereas others have been implicated in intracellular functions including mitotic spindle regulation (4). Cell surface RHAMM/CD168 is localized to membrane ruffles, consistent with proteins that regulate cell motility (1). Numerous research studies have reported that the expression of RHAMM/CD168 is positively associated with cancer cell growth, motility and/or metastasis (5-7), in addition to HA-mediated inflammation (8), suggesting the potential for therapeutic approaches that target HA-receptor mediated signaling (9,10).

**Background References**

1. Hardwick, C. et al. (1992) *J Cell Biol* 117, 1343-50.
2. Samuel, S.K. et al. (1993) *J Cell Biol* 123, 749-58.
3. Naor, D. (2016) *Front Immunol* 7, 39.
4. Tolg, C. et al. (2010) *J Biol Chem* 285, 26461-74.
5. Mele, V. et al. (2017) *Oncotarget* 8, 70617-29.
6. Morera, D.S. et al. (2017) *Br J Cancer* 117, 1507-17.
7. Wang, D. et al. (2016) *Oncotarget* 7, 39957-69.
8. Hauser-Kawaguchi, A. et al. (2018) *Matrix Biol* 78-79, 346-56.
9. Wong, K.M. et al. (2017) *Curr Oncol Rep* 19, 47.
10. Yang, C. et al. (2017) *Theranostics* 7, 1719-34.

**Species Reactivity**

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

**Western Blot Buffer**

**IMPORTANT:** For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting **IP:** Immunoprecipitation

**Cross-Reactivity Key**

**H:** Human

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