

## DESCRIPTION

<b>Species Reactivity</b>	Mouse
<b>Specificity</b>	Detects mouse RGM-C in direct ELISAs and Western blots. In direct ELISAs, approximately 50% cross-reactivity with recombinant human RGM-C is observed and less than 5% cross-reactivity with recombinant mouse (rm) RGM-A and rmRGM-B is observed.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	Mouse myeloma cell line NS0-derived recombinant mouse RGM-C isoform 1 (R&D Systems, Catalog # 3634-RG) Gln33-Asp393 (Ile379Val) Accession # Q7TQ32
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

## APPLICATIONS

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. *General Protocols* are available in the *Technical Information* section on our website.

	Recommended Concentration	Sample
<b>Western Blot</b>	0.1 µg/mL	Recombinant Mouse RGM-C (Catalog # 3634-RG)

## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

## BACKGROUND

RGM-C, also known as hemojuvelin, is a member of the repulsive guidance molecule (RGM) family of GPI-linked neuronal and muscle membrane glycoproteins (1). RGM-C is expressed in striated muscle and periportal hepatocytes (2-4). The protein undergoes partial cleavage intracellularly, resulting in a disulfide-linked dimer of the 14 kDa N-terminal and 33 kDa C-terminal portions (3, 5, 6). The N-terminal fragment contains an RGD motif, while the C-terminal fragment carries the GPI attachment site (3, 6). An alternatively spliced isoform lacks the N-terminal fragment. Full length RGM-C can also be released from the cell and circulates in the blood (5, 7). RGM-C is disrupted in type 2A juvenile hemochromatosis, a hereditary iron homeostasis disorder characterized by excessive iron accumulation (4). Loss of RGM-C function results in decreased expression of the iron regulatory hormone hepcidin and increased iron deposition in liver, pancreas, and heart (4, 8). Membrane associated RGM-C upregulates hepcidin while soluble RGM-C downregulates hepcidin expression (7). This appears to be an iron-responsive regulatory system, as high blood iron levels reduce the amount of soluble RGM-C produced (7). RGM-C, similar to RGM-A, associates with neogenin (6). Disease-related point mutations can prevent internal RGM-C cleavage or its ability to interact with neogenin (5, 6). Experimental inflammatory conditions result in decreased RGM-C expression and increased hepcidin expression, although the two effects occur independently (4, 9). RGM-C also functions as a BMP co-receptor and enhances BMP-2 and BMP-4 signaling (10). In this context, RGM-C enhances the BMP-2 upregulation of hepatic hepcidin (10). Mature mouse RGM-C shares 89% and 97% amino acid (aa) sequence identity with human and rat RGM-C, respectively. It shares 51% and 44% aa sequence identity with mouse RGM-A and RGM-B, respectively.

## References:

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4. Niederkofler, V. *et al.* (2005) *J. Clin. Invest.* **115**:2180.
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9. Krijt, J. *et al.* (2004) *Blood* **104**:4308.
10. Babitt, J.L. *et al.* (2006) *Nat. Genet.* **38**:531.