

DESCRIPTION

Species Reactivity	Human/Mouse
Specificity	Detects human and mouse RGM-C in Western blots. In this format, approximately 10% cross-reactivity with recombinant human (rh) RGM-B is observed and less than 5% cross-reactivity with rhRGM-A is observed.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant human RGM-C isoform a (R&D Systems, Catalog # 3720-RG) Gln36-Asp400 Accession # Q6ZVN8
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

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
Western Blot	0.1 µg/mL	Recombinant Human RGM-C (Catalog # 3720-RG) Recombinant Mouse RGM-C (Catalog # 3634-RG)

PREPARATION AND STORAGE

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

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, 2). RGM-C is expressed in striated muscle and periportal hepatocytes (3 - 5). The protein undergoes partial cleavage intracellularly, resulting in a disulfide-linked dimer of the 14 kDa N-terminal and 33 kDa C-terminal portions (4, 6, 7). The N-terminal fragment contains an RGD motif, while the C-terminal fragment carries the GPI attachment site (4, 7). Two alternatively spliced isoforms lack either approximately half or the entire N-terminal fragment. Full length RGM-C can also be released from the cell and circulates in the blood (6, 8). RGM-C is disrupted in type 2A juvenile hemochromatosis, a hereditary iron homeostasis disorder characterized by excessive iron accumulation (5). In mouse, loss of RGM-C function results in decreased expression of the iron regulatory hormone hepcidin and increased iron deposition in liver, pancreas, and heart (5, 9). Membrane associated RGM-C upregulates hepcidin while soluble RGM-C downregulates hepcidin expression (8). This appears to be an iron-responsive regulatory system, as high blood iron levels reduce the amount of soluble RGM-C produced (8). RGM-C, similar to RGM-A, associates with neogenin (7). Disease-related point mutations can prevent internal RGM-C cleavage or its ability to interact with neogenin (6, 7). Experimental inflammatory conditions result in decreased RGM-C expression and increased hepcidin expression, although the two effects occur independently (5, 10). RGM-C also functions as a BMP coreceptor and enhances BMP-2 and BMP-4 signaling (11). In this context, RGM-C enhances the BMP-2 upregulation of hepatic hepcidin (11). Mature human RGM-C shares 89% amino acid (aa) sequence identity with mouse and rat RGM-C. It shares 49% and 44% aa sequence identity with human RGM-A and RGM-B, respectively.

References:

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