Recombinant Mouse RGM-C/Hemojuvelin

Catalog Number: 3634-RG

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Gln33-Asp393 (Ile379Val) (mature) & Pro166-Asp393 (Ile379Val) (C-terminus peptide), both with a C-terminal 6-His tag & Gln33-Asp165 (N-terminus peptide) Accession # Q7TQ32
N-terminal Sequence Analysis	Gln33 (mature & N-terminus peptide) & Pro166 (C-terminus peptide)
Predicted Molecular Mass	40 kDa (mature), 14 kDa (N-terminus peptide), 26 kDa (C-terminus peptide)

SPECIFICATIONS

SDS-PAGE	57 kDa, 20 kDa and 38 kDa, reducing conditions
Activity	Measured by its binding ability in a functional ELISA. Immobilized rhBMP-4 at 1 µg/mL (100 µL/well) can bind rmRGM-C with a linear range of 0.016-1 µg/mL.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 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). 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 hepicidin and increased iron deposition in liver, pancreas, and heart (4, 8). Membrane associated RGM-C up-regulates hepicidin while soluble RGM-C down-regulates hepicidin 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 hepicidin 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 up-regulation of hepatic hepicidin (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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