Human RGM-C/Hemojuvelin Alexa Fluor® 532-conjugated Antibody

Monoclonal Mouse IgG_{2B} Clone # 751741

Catalog Number: FAB3720X

100 µg

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human RGM-C/Hemojuvelin in direct ELISAs.
Source	Monoclonal Mouse IgG_{2B} Clone # 751741
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Mouse myeloma cell line, NS0-derived human RGM-C/Hemojuvelin Gln36-Asp400 Accession # Q8ZVN8
Conjugate	Alexa Fluor 532 Excitation Wavelength: 534 nm Emission Wavelength: 553 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.*

ELISA	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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 hepicidin and increased iron deposition in liver, pancreas, and heart (5, 9). Membrane associated RGM-C upregulates hepicidin while soluble RGM-C downregulates hepicidin 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 hepicidin 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 hepicidin (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.

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