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[Primary Antibody]

Rabbit Anti-RGMC/Repulsive Guidance Molecule C antibody

Catalog Number: SL11475R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	RGMC/Repulsive Guidance Molecule C
Immunogen:	KLH conjugated synthetic peptide derived from human Repulsive Guidance Molecule C: 205-255/426
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol. Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Applications:	ELISA=1:5000-10000 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	(predicted: Human, Mouse, Rat, Pig, Cow, Rabbit, Sheep)
Predicted MW.:	39kDa
Subcellular Location:	The cell membrane
Background:	The repulsive guidance molecule (RGM) family of proteins are important in the guidance of growth cones of developing neurons. They are repulsive for a group of axons, those from the temporal half of the retina. RGM have been implicated in both axonal guidance and neural tube closure but as opposed to for ephrins, semaphorins, netrins and slits, no receptor mechanism for RGM activation has been defined. Dorsal root ganglion axons do not respond to RGM but neogenin (a netrin-binding protein which can function as an RGM receptor) expression can spur RGM responsiveness. The RGM proteins are attached to the membrane by a GPI-anchor. Two members of this family, RGMa and RGMb, are expressed in the nervous system. RGMc, also known as Hemojuvelin, is a part of the signaling pathway activating hepcidin and works together with hepcidin to restrict iron absorption in the gut. Defects in the gene encoding for RGMc causes the autosomal

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	recessive disorder juvenile hemochromatosis (JH). **Function:** Mutations in Repulsive Guidance Molecule (also known as RGM-C; HJV; hemojuvelin; Hfe2) cause juvenile hemochromatosis, a severe iron overload disease. RGM-C gene expression has been characterized in the developing mouse and found to be exclusively expressed in all striated muscle and in the myocardium. **Subunit:** Interacts with BMP2 and BMP4. Interacts with BMPR1B. Interacts with TMPRSS6. **Subcellular Location:** Cell membrane; Lipid-anchor, GPI-anchor **Tissue Specificity:** Adult and fetal liver, heart, and skeletal muscle. DISEASE: Defects in HFE2 are the cause of hemochromatosis type 2A (HFE2A) [MIM:602390]; also known as juvenile hemochromatosis (JH). HFE2A is an early-onset autosomal recessive disorder due to severe iron overload resulting in hypogonadotrophic hypogonadism, hepatic fibrosis or cirrhosis and cardiomyopathy, occurring typically before age of 30. It is the consequence of intestinal iron hyperabsorption associated with macrophages that do not load iron. Deleterious mutations of HFE2 reduced HAMP (hepcidin) levels despite iron overload, which normally induces HAMP expression. **Similarity:** Belongs to the repulsive guidance molecule (RGM) family. **Database links:** **Entrez Gene:** 148738 Human **Entrez Gene:** 69585 Mouse **Entrez Gene:** 310681 Rat Omim: 608374 Human **SwissProt:** Q6ZVN8 Human **SwissProt:** Q7TQ32 Mouse **SwissProt:** Q8N7M5 Rat **Unigene:** 632436 Human

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