

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human Hepcidin in direct ELISAs
Source	Monoclonal Rabbit IgG Clone # 1033A
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Synthetic peptide containing human Hepcidin Accession # P81172
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

Hepcidin, also known as Liver Expressed Antimicrobial Protein 1 (LEAP-1), is a peptide hormone that is involved in the regulation of iron metabolism (1, 2). It is synthesized as a prohormone that is cleaved intracellularly and secreted as a mature 25 amino acid peptide (1, 3, 4). Hepcidin contains eight cysteine residues that form four disulfide bonds which appear to be important for stability in biological fluids (5). It is predominantly expressed, processed, and secreted by hepatocytes (2, 6). Hepcidin expression is positively regulated by inflammation via IL-6/JAK2/ STAT3 signaling, endoplasmic reticulum stress, and BMP-6 (7-11). BMP-6-dependent Hepcidin induction involves RGM-C/Hemojuvelin, which acts as a co-receptor for BMP-6 (11-13). Conversely, Hepcidin expression is negatively regulated by MMP-15/MT2-MMP and multiple erythropoietic stimuli, including anemia, hypoxia, and Erythropoietin (14-18). MMP-15 downregulates Hepcidin expression by interacting with and cleaving RGM-C (19). Hepcidin was originally identified in human blood and urine as an antimicrobial peptide (1, 3). It has since been shown to regulate iron metabolism. Hepcidin binds the cellular iron exporter Ferroportin, and this interaction results in Ubiquitin-mediated degradation of both Hepcidin and Ferroportin (20-22). Degradation of Ferroportin results in reduced iron release from macrophages, hepatocytes, and duodenal enterocytes, suggesting that Hepcidin may be an effector of inflammatory hypoferremia (20).

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