SANTA CRUZ BIOTECHNOLOGY, INC.

hepcidin (56-Z): sc-100277

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Hepcidin, also known as HAMP, HEPC, LEAP1 or HFE2B, is an 84 amino acid secreted protein that regulates iron-related signaling events. Highly expressed in liver with lower expression in heart, brain, lung, prostate and thyroid, hepcidin is thought to maintain iron homeostasis and, in conjunction with the HFE protein (a protein that is defective in hereditary hemochromatosis), may mediate both iron storage in macrophages and intestinal iron absorption. Additionally, hepcidin has strong antimicrobial activity against Gram-positive and Gram-negative bacteria, as well as certain yeast strains, suggesting that hepcidin may play a crucial role in staving off bacterial infections. Defects in the gene encoding hepcidin are the cause of hemochromatosis type 2B (also known as juvenile hemochromatosis), an early-onset autosomal recessive disorder that results in severe iron overload and is characterized by hepatic fibrosis, hypogonadotrophic hypogonadism and cardiomyopathy.

REFERENCES

1. Krause, A., et al. 2000. LEAP-1, a novel highly disulfide-bonded human peptide, exhibits antimicrobial activity. FEBS Lett. 480: 147-150.
2. Park, C.H., et al. 2001. Hepcidin, a urinary antimicrobial peptide synthesized in the liver. J. Biol. Chem. 276: 7806-7810.
3. Klüver, E., et al. 2002. Chemical synthesis of β-defensins and LEAP-1/hepcidin. J. Pept. Res. 59: 241-248.
4. Merryweather-Clarke, A.T., et al. 2003. Digenic inheritance of mutations in HAMP and HFE results in different types of haemochromatosis. Hum. Mol. Genet. 12: 2241-2247.
5. Roetto, A., et al. 2003. Mutant antimicrobial peptide hepcidin is associated with severe juvenile hemochromatosis. Nat. Genet. 33: 21-22.
6. Nemeth, E., et al. 2004. Hepcidin regulates cellular iron efflux by binding to ferroportin and inducing its internalization. Science 306: 2090-2093.
7. Robson, K.J., et al. 2004. Recent advances in understanding haemochromatosis: a transition state. J. Med. Genet. 41: 721-730.

CHROMOSOMAL LOCATION

Genetic locus: HAMP (human) mapping to 19q13.12.

SOURCE

hepcidin (56-Z) is a mouse monoclonal antibody raised against recombinant hepcidin of human origin.

PRODUCT

Each vial contains 100 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

hepcidin (56-Z) is recommended for detection of hepcidin of human origin by immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000)

Suitable for use as control antibody for hepcidin siRNA (h): sc-97892, hepcidin shRNA Plasmid (h): sc-97892-SH and hepcidin shRNA (h) Lentiviral Particles: sc-97892-V.

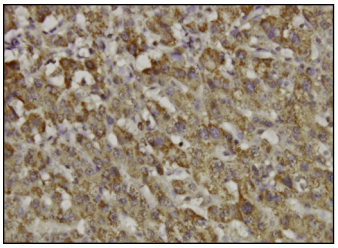
Molecular Weight of hepcidin: 9 kDa.

Positive Controls: human liver extract: sc-363766.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850. 2) Immunohistochemistry: use m-IgGκ BP-HRP: sc-516102 with DAB, 50X: sc-24982 and Immunohistomount: sc-45086, or Organo/Limonene Mount: sc-45087.

DATA



hepcidin (56-Z): sc-100277. Immunoperoxidase staining of formalin-fixed, paraffin-embedded human liver tissue showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Nieuwenhuizen, L., et al. 2013. Identification and expression of iron regulators in human synovium: evidence for upregulation in haemophilic arthropathy compared to rheumatoid arthritis, osteoarthritis, and healthy controls. Haemophilia 19: e218-e227.
2. Koeppen, A.H., et al. 2015. The pathogenesis of cardiomyopathy in Friedreich ataxia. PLoS ONE 10: e0116396.
3. Mancinelli, R., et al. 2020. Different iron-handling in inflamed small and large cholangiocytes and in small and large-duct type intrahepatic cholangiocarcinoma. Eur. J. Histochem. 64: 3156.

RESEARCH USE

For research use only, not for use in diagnostic procedures.