SunLong Biotech Co.,LTD
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[Primary Antibody]

Rabbit Anti-MNX1/HLXB9 antibody

Catalog Number: SL22522R

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	MNX1/HLXB9
Immunogen:	KLH conjugated synthetic peptide derived from human MNX1/HLXB9 : 201-300/401
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:	WB=1:500-2000 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human,Mouse(predicted:Rat,Chicken,Dog,Pig,Cow,Rabbit)
Predicted MW.:	41kDa
Subcellular Location:	The nucleus
Background:	The HB9 homeobox transcription factor regulates gene expression during embryonic development and also in specific adult tissues. HB9 gene mutations are implicated in Curriano syndrome, which is characterized by a triad consisting of a presacral tumor, sacral agenesis and anorectal malformation. In human bone marrow cells, HB9 expression directly correlates with CD34 expression. Furthermore, HB9 expression increases in CD34+ cells that are treated with IL-3 and granulocyte macrophage-colony-stimulating factor. Early in murine development, HB9 is expressed in pancreatic buds (dorsal and ventral) with subsequent expression in differentiating beta cells in the islets of Langerhans. The dorsal lobe of the pancreas fails to form in HB9(-) mice; the resultant pancreas has smaller islets of Langerhans and less beta cells than normal pancreas. The HB9 gene is expressed in the human adult pancreas. In the developing

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	vertebrate embryo, the HB9 gene plays an essential role in motor neuron differentiation. The motor columns of HB9(-) mice are disorganized, lacking phrenic and abducens nerves and exhibiting intercostal nerve defects. **Function:** Putative transcription factor involved in pancreas development and function. **Subcellular Location:** Nucleus. **Tissue Specificity:** Expressed in lymphoid and pancreatic tissues. DISEASE: Defects in MNX1 are a cause of Currarino syndrome (CURRAS) [MIM:176450]. The triad of a presacral tumor, sacral agenesis and anorectal malformation constitutes the Currarino syndrome which is caused by dorsal-ventral patterning defects during embryonic development. The syndrome occurs in the majority of patients as an autosomal dominant trait. **Similarity:** Contains 1 homeobox DNA-binding domain. **Database links:** **Entrez Gene:** 3110 Human **Entrez Gene:** 15285 Mouse Omim: 142994 Human **SwissProt:** P50219 Human **SwissProt:** Q9QZW9 Mouse **Unigene:** 37035 Human **Unigene:** 103760 Mouse

[VALIDATION IMAGES]

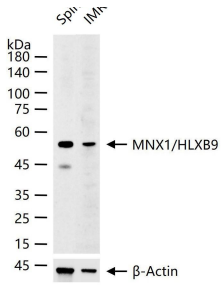


Figure 1. 25 ug total protein per lane of various lysates (see on figure) probed with MNX1/HLXB9 polyclonal antibody, unconjugated (SL22522R) at 1:1000 dilution and 4°C overnight incubation. Followed by conjugated secondary antibody incubation at r.t. for 60 min

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