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LEF1 Antibody

Purified Mouse Monoclonal Antibody (Mab)
Catalog # AM8648b

Product Information

Application	WB, E
Primary Accession	Q9UJU2
Reactivity	Human
Predicted	Mouse, Rat, Bovine, Canine, Rabbit, Chicken
Host	Mouse
Clonality	monoclonal
Isotype	IgG1,κ
Clone Names	1866CT561.78.3
Calculated MW	44201

Additional Information

Gene ID	51176
Other Names	Lymphoid enhancer-binding factor 1, LEF-1, T cell-specific transcription factor 1-alpha, TCF1-alpha, LEF1
Target/Specificity	This antibody is generated from a mouse immunized with a recombinant protein between 95-283 amino acids from human.
Dilution	WB~~1:4000 E~~Use at an assay dependent concentration.
Format	Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	LEF1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	LEF1 (HGNC:6551)
Function	Transcription factor that binds DNA in a sequence-specific manner (PubMed:2010090). Participates in the Wnt signaling pathway (By similarity). Activates transcription of target genes in the presence of CTNNB1 and EP300 (By similarity). PIAG antagonizes both Wnt-dependent and Wnt-independent activation by LEF1 (By similarity). TLE1, TLE2, TLE3 and TLE4 repress

	transactivation mediated by LEF1 and CTNNB1 (PubMed:11266540). Regulates T-cell receptor alpha enhancer function (PubMed:19653274). Required for IL17A expressing gamma-delta T-cell maturation and development, via binding to regulator loci of BLK to modulate expression (By similarity). Acts as a positive regulator of odontoblast differentiation during mesenchymal tooth germ formation, expression is repressed during the bell stage by MSX1-mediated inhibition of CTNNB1 signaling (By similarity). May play a role in hair cell differentiation and follicle morphogenesis (By similarity).
Cellular Location	Nucleus {ECO:0000255|PROSITE-ProRule:PRU00267}. Note=Found in nuclear bodies upon PIASG binding.
Tissue Location	Detected in thymus. Not detected in normal colon, but highly expressed in colon cancer biopsies and colon cancer cell lines. Expressed in several pancreatic tumors and weakly expressed in normal pancreatic tissue. Isoforms 1 and 5 are detected in several pancreatic cell lines.

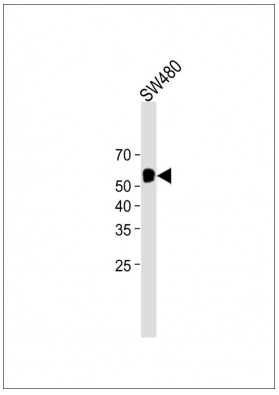
Background

Participates in the Wnt signaling pathway. Activates transcription of target genes in the presence of CTNNB1 and EP300. May play a role in hair cell differentiation and follicle morphogenesis. TLE1, TLE2, TLE3 and TLE4 repress transactivation mediated by LEF1 and CTNNB1. Regulates T-cell receptor alpha enhancer function. Binds DNA in a sequence-specific manner. PIAG antagonizes both Wnt-dependent and Wnt-independent activation by LEF1 (By similarity). Isoform 3 lacks the CTNNB1 interaction domain and may be an antagonist for Wnt signaling. Isoform 5 transcriptionally activates the fibronectin promoter, binds to and represses transcription from the E-cadherin promoter in a CTNNB1- independent manner, and is involved in reducing cellular aggregation and increasing cell migration of pancreatic cancer cells. Isoform 1 transcriptionally activates MYC and CCND1 expression and enhances proliferation of pancreatic tumor cells.

References

Waterman M.L.,et al.Genes Dev. 5:656-669(1991).
Hovanes K.,et al.Nucleic Acids Res. 28:1994-2003(2000).
Jesse S.,et al.Int. J. Cancer 126:1109-1120(2010).
Kobielak A.,et al.Submitted (AUG-2000) to the EMBL/GenBank/DDBJ databases.
Ota T.,et al.Nat. Genet. 36:40-45(2004).

Images



All lanes: Anti-LEF1 at 1:1000 dilution + SW480 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary: Goat Anti-Mouse IgG, (H+L), Peroxidase conjugated (ASP1613) at 1/8000 dilution. Observed band size: 58 KDa Blocking/Dilution buffer: 5% NFDM/TBST.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.