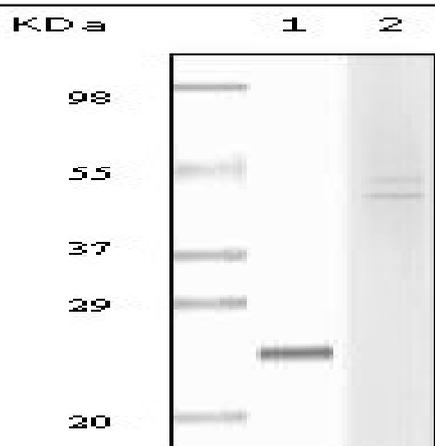


Fibulin-5 Monoclonal Antibody

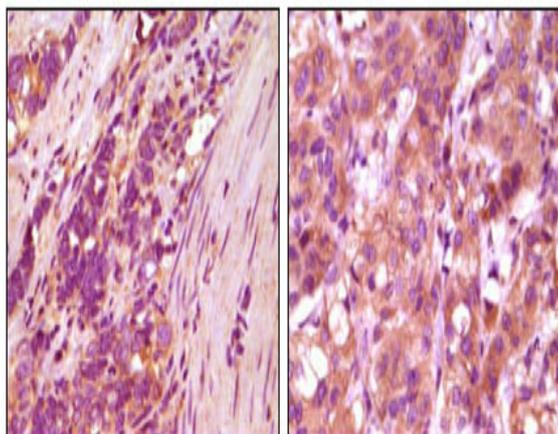
Catalog No :	YM0272
Reactivity :	Human
Applications :	WB;IHC;IF;ELISA
Target :	Fibulin-5
Gene Name :	FBLN5
Protein Name :	Fibulin-5
Human Gene Id :	10516
Human Swiss Prot No :	Q9UBX5
Mouse Swiss Prot No :	Q9WVH9
Immunogen :	Purified recombinant fragment of Fibulin-5 expressed in E. Coli.
Specificity :	Fibulin-5 Monoclonal Antibody detects endogenous levels of Fibulin-5 protein.
Formulation :	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source :	Monoclonal, Mouse
Dilution :	WB 1:500 - 1:2000. IHC 1:200 - 1:1000. ELISA: 1:10000.. IF 1:50-200
Purification :	Affinity purification
Storage Stability :	-15°C to -25°C/1 year(Do not lower than -25°C)
Molecularweight :	50kD
P References :	<ol style="list-style-type: none">1. Wen Wang , Scott A. LeMaire, Li Chen. Surgery. 2005 Aug;138(2):352-92. K. Kadoya, T. Sasaki, G. Kostka. Br J Dermatol. 2005 Sep;153(3):607-123.3. Michael J. Lee, Nakshatra K. Roy, Jon E.

Background :	The protein encoded by this gene is a secreted, extracellular matrix protein containing an Arg-Gly-Asp (RGD) motif and calcium-binding EGF-like domains. It promotes adhesion of endothelial cells through interaction of integrins and the RGD motif. It is prominently expressed in developing arteries but less so in adult vessels. However, its expression is reinduced in balloon-injured vessels and atherosclerotic lesions, notably in intimal vascular smooth muscle cells and endothelial cells. Therefore, the protein encoded by this gene may play a role in vascular development and remodeling. Defects in this gene are a cause of autosomal dominant cutis laxa, autosomal recessive cutis laxa type I (CL type I), and age-related macular degeneration type 3 (ARMD3). [provided by RefSeq, Jul 2008],
Function :	disease:Defects in FBLN5 are a cause of autosomal dominant cutis laxa [MIM:123700]. Hereditary cutis laxa refers to a heterogeneous group of connective tissue disorders characterized by cutaneous abnormalities and variable systemic manifestations. The most constant clinical feature is loose skin, sagging over the face and trunk. Hereditary cutis laxa is inherited in both autosomal dominant and autosomal recessive modes. Autosomal dominant cutis laxa is a relatively benign inherited and acquired connective tissue disorder.,disease:Defects in FBLN5 are a cause of autosomal recessive cutis laxa type I (CL type I) [MIM:219100]. CL type I shows the most severe phenotype and has the poorest prognosis. In addition to the skin, internal organs enriched in elastic fibers, such as the lung and arteries, are affected.,disease:Defects in FBLN5 are the cause of age-related macular degeneration type 3
Subcellular Location :	Secreted . Secreted, extracellular space, extracellular matrix . co-localizes with ELN in elastic fibers. .
Expression :	Expressed in skin fibroblasts (at protein level)(PubMed:17035250). Expressed predominantly in heart, ovary, and colon but also in kidney, pancreas, testis, lung and placenta. Not detectable in brain, liver, thymus, prostate, or peripheral blood leukocytes (PubMed:10428823).
Sort :	6059
No4 :	1
Host :	Mouse
Modifications :	Unmodified

Products Images



Western Blot analysis using Fibulin-5 Monoclonal Antibody against truncated fibulin5 recombinant protein (1) and HeLa cell lysate (2).



Immunohistochemistry analysis of paraffin-embedded human stomach carcinoma (left) and breast carcinoma (right), showing cytoplasmic localization with DAB staining using Fibulin-5 Monoclonal Antibody.