



Fibulin-5 Polyclonal Antibody

Catalog No	BYab-17023
Isotype	IgG
Reactivity	Human;Mouse;Rat
Applications	WB;ELISA
Gene Name	FBLN5
Protein Name	Fibulin-5
Immunogen	The antiserum was produced against synthesized peptide derived from human FBLN5. AA range:171-220
Specificity	Fibulin-5 Polyclonal Antibody detects endogenous levels of Fibulin-5 protein.
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source	Polyclonal, Rabbit,IgG
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Dilution	Western Blot: 1/500 - 1/2000. ELISA: 1/5000. Not yet tested in other applications.
Concentration	1 mg/ml
Purity	≥90%
Storage Stability	-20°C/1 year
Synonyms	FBLN5; DANCE; Fibulin-5; FIBL-5; Developmental arteries and neural crest EGF-like protein; Dance; Urine p50 protein; UP50
Observed Band	50kD
Cell Pathway	Secreted . Secreted, extracellular space, extracellular matrix . co-localizes with ELN in elastic fibers. .
Tissue Specificity	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).
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

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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. Defects in FBLN5 are the cause of age-related macular degeneration type 3

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],

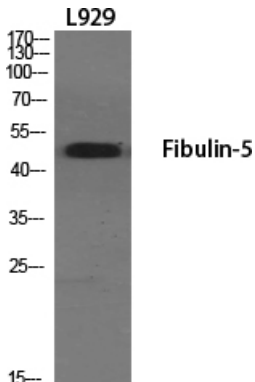
matters needing attention

Avoid repeated freezing and thawing!

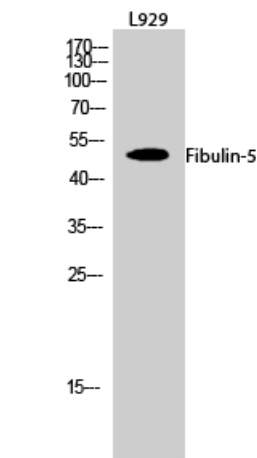
Usage suggestions

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

Products Images

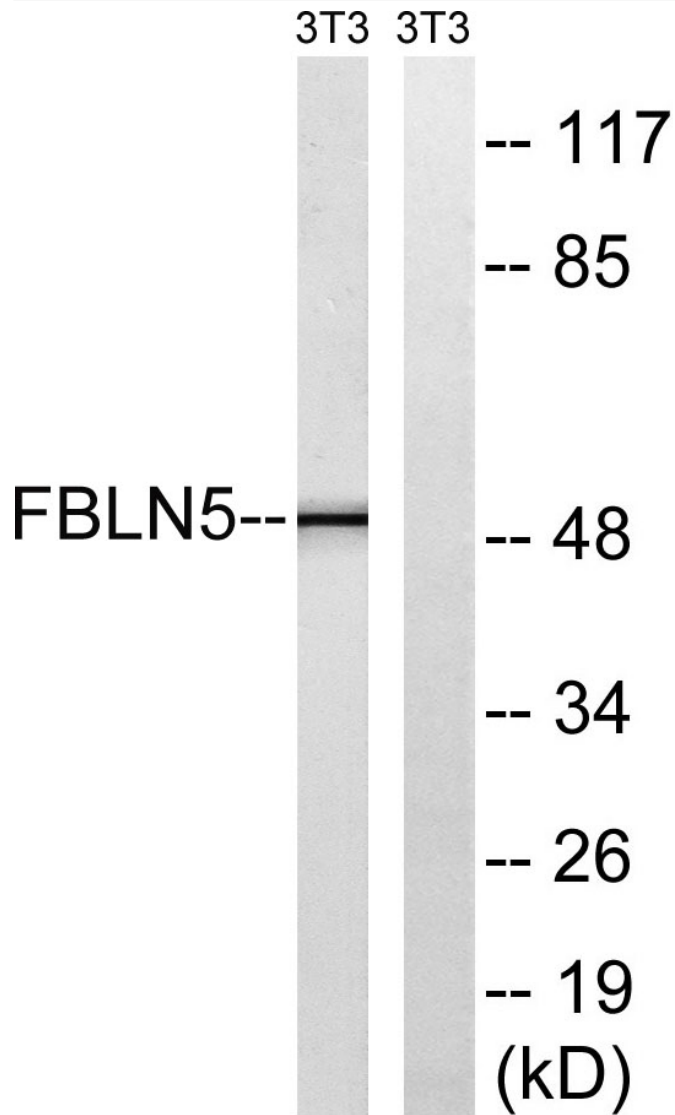


Western Blot analysis of various cells using Fibulin-5 Polyclonal Antibody diluted at 1:1000



Western Blot analysis of L929 cells using Fibulin-5 Polyclonal Antibody diluted at 1:1000

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Western blot analysis of lysates from NIH/3T3 cells, using FBLN5 Antibody. The lane on the right is blocked with the synthesized peptide.