

Product name:	Fibulin-5 Rabbit Polyclonal Antibody
Cat number:	ABN10981
Conjugate:	Unconjugated
Size:	100µL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human FBLN5. AA range:171-220
Reactivity:	Human,Mouse,Rat
Applications:	WB 1:500-1:2000,ELISA 1:5000-1:10000
Molecular Weight:	50kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Storage:	Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

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],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 (ARMD3) [MIM:608895]. ARMD is a multifactorial disease and the most common cause of irreversible vision loss in the developed world. In most patients, the disease is manifest as ophthalmoscopically visible yellowish accumulations of protein and lipid (known as drusen) that lie beneath the retinal pigment epithelium and within an elastin-containing structure known as Bruch membrane.,function:Promotes adhesion of endothelial cells through interaction of integrins and the RGD motif. Could be a vascular ligand for integrin receptors and may play a role in vascular development and remodeling.,similarity:Belongs to the fibulin family.,similarity:Contains 6 EGF-like domains.,tissue specificity: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.,