Product datasheet

Anti-Tropoelastin antibody ab21600

Overview

Product name  Anti-Tropoelastin antibody
Description  Rabbit polyclonal to Tropoelastin
Host species  Rabbit
Specificity  Interacts with tropoelastin and insoluble elastin (there are many shared epitopes between the two forms of the protein).
Species reactivity  Reacts with: Mouse, Human
                  Predicted to work with: Rat, Cow
Immunogen  Synthetic peptide corresponding to Mouse Tropoelastin. ab21600 was generated to the N-terminal portion of mouse tropoelastin, the soluble precursor form of elastin.

Properties

Form  Liquid
Storage instructions  Shipped at 4°C. Store at +4°C short term (1-2 weeks). Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Storage buffer  Constituents: Whole serum, 50% Glycerol
Purity  Whole antiserum
Clonality  Polyclonal
Isotype  IgG

Target

Function  Major structural protein of tissues such as aorta and nuchal ligament, which must expand rapidly and recover completely. Molecular determinant of the late arterial morphogenesis, stabilizing arterial structure by regulating proliferation and organization of vascular smooth muscle.
Tissue specificity  Expressed within the outer myometrial smooth muscle and throughout the arteriolar tree of uterus (at protein level). Also expressed in the large arteries, lung and skin.
Involvement in disease  Defects in ELN are a cause of autosomal dominant cutis laxa (ADCL) [MIM:123700]. Cutis laxa is a rare connective tissue disorder characterized by loose, hyperextensible skin with decreased resilience and elasticity leading to a premature aged appearance. The skin changes are often
accompanied by extracutaneous manifestations, including pulmonary emphysema, bladder diverticula, pulmonary artery stenosis and pyloric stenosis. Defects in ELN are the cause of supravalvular aortic stenosis (SVAS) [MIM:185500]. SVAS is a congenital narrowing of the ascending aorta which can occur sporadically, as an autosomal dominant condition, or as one component of Williams-Beuren syndrome. Note=ELN is located in the Williams-Beuren syndrome (WBS) critical region. WBS results from a hemizygous deletion of several genes on chromosome 7q11.23, thought to arise as a consequence of unequal crossing over between highly homologous low-copy repeat sequences flanking the deleted region. Haploinsufficiency of ELN may be the cause of certain cardiovascular and musculo-skeletal abnormalities observed in the disease.

Sequence similarities
Belongs to the elastin family.

Post-translational modifications
Elastin is formed through the cross-linking of its soluble precursor tropoelastin. Cross-linking is initiated through the action of lysyl oxidase on exposed lysines to form allysine. Subsequent spontaneous condensation reactions with other allysine or unmodified lysine residues result in various bi-, tri-, and tetrafunctional cross-links. The most abundant cross-links in mature elastin fibers are lysinonorleucine, allysine aldol, desmosine, and isodesmosine. Hydroxylation on proline residues within the sequence motif, GXPG, is most likely 4-hydroxy as this fits the requirement for 4-hydroxylation in vertebrates.

Cellular localization
Secreted > extracellular space > extracellular matrix. Extracellular matrix of elastic fibers.

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