**Product datasheet**

**Anti-Tropoelastin antibody ab21600**

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### 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 aldon, 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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