abcam

Product datasheet

Hsp27 peptide ab204861

Description

Product name	Hsp27 peptide
Purity	> 98 % HPLC.
Animal free	No
Nature	Synthetic
Sequence	RRLNRQLSVA-amide
Amino acids	80 to 85

Specifications

Our **Abpromise guarantee** covers the use of **ab204861** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications	HPLC Functional Studies
Form	Lyophilized
Additional notes	ab204861 (Hsp27 peptide) can be utilized as a substrate for the following active protein kinases: **ab85758** (Active human DCAMKL2 protein fragment) **ab60307** (Active MAPKAP Kinase 2 protein fragment) **ab60308** (Active human MAPKAP Kinase 3 full length protein) **ab125743** (Active human PRAK full length protein)

Preparation and Storage

Stability and Storage	Shipped at 4°C. Store at -20°C. Avoid freeze / thaw cycle.
Reconstitution	Dilute peptide in distilled water to a final concentration of 1 mg/ml. For optimal storage, aliquot diluted product into smaller quantities and store at recommended temperature.

General Info

Function	Involved in stress resistance and actin organization.
Tissue specificity	Detected in all tissues tested: skeletal muscle, heart, aorta, large intestine, small intestine, stomach, esophagus, bladder, adrenal gland, thyroid, pancreas, testis, adipose tissue, kidney, liver, spleen, cerebral cortex, blood serum and cerebrospinal fluid. Highest levels are found in the heart and in tissues composed of striated and smooth muscle.
Involvement in disease	Defects in HSPB1 are the cause of Charcot-Marie-Tooth disease type 2F (CMT2F) [MIM:606595]. CMT2F is a form of Charcot-Marie-Tooth disease, the most common inherited disorder of the peripheral nervous system. Charcot-Marie-Tooth disease is classified in two main groups on the basis of electrophysiologic properties and histopathology: primary peripheral demyelinating neuropathy or CMT1, and primary peripheral axonal neuropathy or CMT2. Neuropathies of the CMT2 group are characterized by signs of axonal regeneration in the absence of obvious myelin alterations, normal or slightly reduced nerve conduction velocities, and progressive distal muscle weakness and atrophy. Nerve conduction velocities are normal or slightly reduced. CMT2F onset is between 15 and 25 years with muscle weakness and atrophy usually beginning in feet and legs (peroneal distribution). Upper limb involvement occurs later. CMT2F inheritance is autosomal dominant. Defects in HSPB1 are a cause of distal hereditary motor neuronopathy type 2B (HMN2B) [MIM:608634]. Distal hereditary motor neuronopathies constitute a heterogeneous group of neuromuscular disorders caused by selective impairment of motor neurons in the anterior horn of the spinal cord, without sensory deficit in the posterior horn. The overall clinical picture consists of a classical distal muscular atrophy syndrome in the legs without clinical sensory loss. The disease starts with weakness and wasting of distal muscles of the anterior tibial and peroneal compartments of the legs. Later on, weakness and atrophy may expand to the proximal muscles of the lower limbs and/or to the distal upper limbs.
Sequence similarities	Belongs to the small heat shock protein (HSP20) family.
Post-translational modifications	Phosphorylated in MCF-7 cells on exposure to protein kinase C activators and heat shock.
Cellular localization	Cytoplasm. Nucleus. Cytoplasm > cytoskeleton > spindle. Cytoplasmic in interphase cells. Colocalizes with mitotic spindles in mitotic cells. Translocates to the nucleus during heat shock and resides in sub-nuclear structures known as SC35 speckles or nuclear splicing speckles.

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