

## APEG1 Protein, Human, Recombinant (His)

### General Information

Synonyms:	SPEG complex locus;BPEG;SPEGalpha;SPEGβ;APEG-1;SPEGbeta;APEG1;SPEGα;CNM5
Protein Construction:	A DNA sequence encoding the human SPEG isoform 3 (Q15772-4) (Met 1-Glu 113) was fused with a polyhistidine tag at the C-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	E. coli
Accession:	Q15772-4
Molecular Weight:	14 kDa (predicted); 19 kDa (reducing conditions)

### QC Testing

Biological Activity:	Kinase activity untested
Purity:	> 85 % as determined by SDS-PAGE
Endotoxin:	Please contact us for more information.
Formulation:	Supplied as sterile PBS, pH 7.4.

### Preparation and Storage

#### Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

#### Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

*Actual storage temperature shall be subject to the COA.*

#### Shipping:

Proteins are shipped with blue ice.

### Protein Background

Striated muscle preferentially expressed protein kinase, also known as aortic preferentially expressed protein 1, APEG-1, SPEG and KIAA1297, is a protein that belongs to the protein kinase superfamily and CAMK Ser/Thr protein kinase family. SPEG / APEG-1 contains two fibronectin type-III domains, nine Ig-like (immunoglobulin-like) domains, two protein kinase domains. Isoform 1 of SPEG is preferentially expressed in striated muscle. Non-kinase form such as isoform 3 of SPEG is predominantly expressed in the aorta. Isoform 3 of SPEG appears to be expressed only in highly differentiated ASMC in normal vessel walls and down-regulated in dedifferentiated ASMC. Isoform 3 of SPEG may have a role in regulating the growth and differentiation of arterial smooth muscle cells. Isoform 3 of SPEG is quickly down-regulated in response to vascular injury, when ASMC cells change from a quiescent to a proliferative phenotype.

Reference

- Hsieh C.-M., et al., 1996, J. Biol. Chem. 271:17354-17359.  
Manjasetty B.A., et al., 2005, BMC Struct. Biol. 5:21-21.  
Zhou,Y. et al., 2007, Anal Chem. 79 (15): 5826-37.  
Greenman C., et al., 2007, Nature. 446:153-158.

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