Recombinant Junctophilin 2 (JPH2)(N-6His)

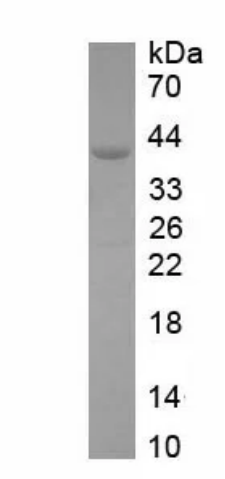
Catalog#:PEH22867

概述（Summary）

别称（Alternative Names）	JP2; Junctophilin type 2
纯度（Purity）	Greater than 90% as determined by reducing SDS-PAGE.
内毒素（Endotoxin level）	<1 EU/μg as determined by LAL test.
蛋白构建（Construction）	6His-【Arg5-Ser301】
Accession #	Q9BR39
表达宿主（Host）	E.coli
种属（Species）	Homo sapiens (Human)
预测分子量（Predicted MW）	35 kDa
制剂（Form）	Lyophilized from a 0.2 μm filtered solution of PBS, pH7.4, containing 0.01% SKL, 1mM DTT, 5% Trehalose and Proclin300.
运输方式（Shipping）	The product is shipped with ice bag. Upon receipt, store it immediately at the temperature listed below.
稳定性&储存（Stability &Storage）	12 months at -20°C to -80°C in lyophilized state.6 months at -20°C to -80°C under sterile conditions after reconstitution.7-10 days at 2°C to 8°C under sterile conditions after reconstitution.Please minimize freeze-thaw cycles.
复溶（Reconstitution）	Reconstitute in 10mM PBS (pH7.4) to a concentration of 0.1-1.0 mg/mL. Do not vortex.
背景介绍（References）	Membrane-binding protein that provides a structural bridge between the plasma membrane and the sarcoplasmic reticulum and is required for normal excitation-contraction coupling in cardiomyocytes. Provides a structural foundation for functional cross-talk between the cell surface and intracellular Ca(2+) release channels by maintaining the 12-15 nm gap between the sarcolemma and the sarcoplasmic reticulum membranes in the cardiac dyads. Necessary for proper intracellular Ca(2+) signaling in cardiac myocytes via its involvement in ryanodine receptor-mediated calcium ion release. Contributes to the construction of skeletal muscle triad junctions.; Transcription repressor required to safeguard

against the deleterious effects of cardiac stress. Generated following cleavage of the Junctophilin-2 chain by calpain in response to cardiac stress in cardiomyocytes. Following cleavage and release from the membrane, translocates to the nucleus, binds DNA and represses expression of genes implicated in cell growth and differentiation, hypertrophy, inflammation and fibrosis. Modifies the transcription profile and thereby attenuates pathological remodeling in response to cardiac stress. Probably acts by competing with MEF2 transcription factors and TATA-binding proteins.

电泳图（SDS-PAGE image）



Greater than 90% as determined by reducing SDS-PAGE.

注意事项（Note）

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