Recombinant Receptor Interacting Serine Threonine Kinase 3 (RIPK3)(N-6His)

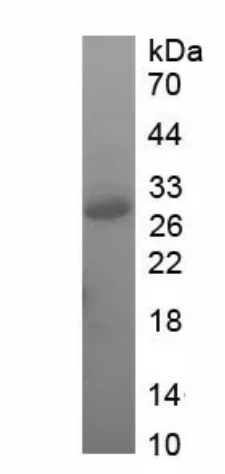
Catalog#:PEH22192

概述（Summary）

别称（Alternative Names）	RIP3; RIP-like protein kinase 3; Receptor-interacting protein 3
纯度（Purity）	Greater than 95% 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-【Met1-Ala216】
Accession #	Q9Y572
表达宿主（Host）	E.coli
种属（Species）	Homo sapiens (Human)
预测分子量（Predicted MW）	27.4 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）	Serine/threonine-protein kinase that activates necroptosis and apoptosis, two parallel forms of cell death. Necroptosis, a programmed cell death process in response to death-inducing TNF-alpha family members, is triggered by RIPK3 following activation by ZBP1. Activated RIPK3 forms a necrosis-inducing complex and mediates phosphorylation of MLKL, promoting MLKL localization to the plasma membrane and execution of programmed necrosis characterized by calcium influx and plasma membrane damage. In addition to TNF-induced necroptosis, necroptosis can also take place in the nucleus in response to orthomyxoviruses infection: following ZBP1 activation, which senses double-

stranded Z-RNA structures, nuclear RIPK3 catalyzes phosphorylation and activation of MLKL, promoting disruption of the nuclear envelope and leakage of cellular DNA into the cytosol. Also regulates apoptosis: apoptosis depends on RIPK1, FADD and CASP8, and is independent of MLKL and RIPK3 kinase activity. Phosphorylates RIPK1: RIPK1 and RIPK3 undergo reciprocal auto- and trans-phosphorylation. In some cell types, also able to restrict viral replication by promoting cell death-independent responses. In response to Zika virus infection in neurons, promotes a cell death-independent pathway that restricts viral replication: together with ZBP1, promotes a death-independent transcriptional program that modifies the cellular metabolism via up-regulation expression of the enzyme ACOD1/IRG1 and production of the metabolite itaconate. Itaconate inhibits the activity of succinate dehydrogenase, generating a metabolic state in neurons that suppresses replication of viral genomes. RIPK3 binds to and enhances the activity of three metabolic enzymes: GLUL, GLUD1, and PYGL. These metabolic enzymes may eventually stimulate the tricarboxylic acid cycle and oxidative phosphorylation, which could result in enhanced ROS production.; (Microbial infection) In case of herpes simplex virus 1/HHV-1 infection, forms heteromeric amyloid structures with HHV-1 protein RIR1/ICP6 which may inhibit RIPK3-mediated necroptosis, thereby preventing host cell death pathway and allowing viral evasion.

电泳图（SDS-PAGE image）



Greater than 95% as determined by reducing SDS-PAGE.

注意事项（Note）

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