

Produktinformation



Forschungsprodukte & Biochemikalien



Zellkultur & Verbrauchsmaterial



Diagnostik & molekulare Diagnostik



Laborgeräte & Service

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Lieferung & Zahlungsart

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Datasheet for 100-401-962 PERK Antibody

Overview

Description:	Anti-PKR-like Endoplasmic Reticulum Kinase (PERK) (RABBIT) Antibody - 100-401-962
Item No.:	100-401-962
Size:	100 μL
Applications:	IHC, IP, WB, Other
Reactivity:	Mouse
Host Species:	Rabbit

Product Details

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Bac	Kgro	und:	

The PKR-like endoplasmic reticulum kinase (PERK, also know as Eukaryotic translation initiation factor 2-alpha kinase 3) is a type I transmembrane protein localized to the endoplasmic reticulum (ER). PERK consists of an N-terminal ER luminal domain, a membrane-spanning region, and a cytosolic C-terminal serine/threonine kinase domain (1). The luminal domain of PERK is bound to the ER chaperone GRP78 in unstressed cells (2). PERK activation occurs upon accumulation of misfolded proteins and the ER lumen, which triggers GRP78 dissociation from PERK thereby allowing PERK dimerization and autophosphorylation (3, 4). PERK phosphorylates two established targets: the eukaryotic translation initiation factor 2 alpha (eIF2, (1)) and the Nrf2 transcription factor (5). Phosphorylation of eIF2 results in attenuation of translation initiation (6). The translational block also contributes to cell cycle arrest due to loss of the G1 regulatory protein, cyclin D1 (7). PERK-dependent phosphorylation of Nrf2 promotes transcription of phase II detoxifying enzymes which is critically important for elimination of intracellular reactive oxygen species (8). Thus, while inhibiting new protein synthesis and thereby decreasing the ER protein load PERK simultaneously induces expression of genes that help restore cellular redox homeostasis and promote survival.

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Synonyms:	rabbit anti-PKR-like Endoplasmic Reticulum Kinase Antibody, rabbit anti-PERK Antibody, PKR-like endoplasmic reticulum kinase, PERK, Eukaryotic translation initiation factor 2-alpha kinase 3
Host Species:	Rabbit
Clonality:	Polyclonal
Format:	Antiserum

Target Details

www.rockland.com Page 1 of 7



Eif2ak3
Mouse
Recombinant Protein
This whole rabbit serum was prepared by repeated immunizations with a recombinant fusion protein from amino acids 601-1115 of mouse deltaN PERK.
This antiserum is directed against PERK and reacts with the PERK from mouse tissues. Reactivity to other species is unknown.
 NCBI - 32451785 UniProtKB - Q9Z2B5 GeneID - 13666

Application Details

Tested Applications:	IHC, IP, WB
Suggested Applications:	Other (Based on references)
Application Note:	This antiserum has been tested for use in western blotting, immunoprecipitation and immunohistochemistry. Specific conditions for reactivity should be optimized by the end user. Expect bands approximately 150kDa by western blotting in the appropriate cell lysate or extract.
Assay Dilutions:	All assays should be optimized by the user. Recommended dilutions (if any) may be listed below.
ELISA:	1:4,000 - 1:20,000
IF:	User Optimized
IHC:	1:1,000
IP:	10-30ul
WB:	1:500 - 1:3000

Formulation

Physical State:	Liquid (sterile filtered)
Concentration:	80 mg/mL by Refractometry
Buffer:	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Preservative:	0.01% (w/v) Sodium Azide
Stabilizer:	None

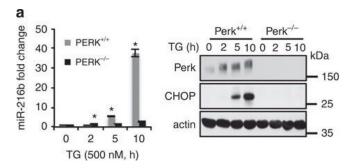
www.rockland.com Page 2 of 7



Shipping & Handling

Shipping Condition:	Dry Ice
Storage Condition:	Store vial at -20° C prior to opening. Aliquot contents and freeze at -20° C or below for extended storage. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several weeks at 4° C as an undiluted liquid. Dilute only prior to immediate use.
Expiration:	Expiration date is one (1) year from date of receipt.

Images



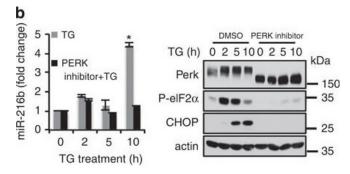
Western Blot

PERK-dependent miR-216b induction.(a) PERK+/+ and PERK-/- MEFs were treated with 500 nM TG for indicated times. MiR-216b was assessed by qPCR (left graph); PERK and CHOP were assessed by immunoblot (right). (b) MiR-216b levels were quantified by qPCR following exposure of cells to thapsigargin and a small-molecule PERK inhibitor (left). PERK, eIF2 α -p and CHOP induction were assessed by immunoblot (right). (c-e) MEFs of the indicated genotype were treated with TG (500 nM) for indicated intervals. Protein extracts from these cells were immunoblotted for the proteins as indicated (lower panels) and miR-216b levels were quantified by qPCR (upper panels; n=3). (f) CHOP-/-MEFs were transfected with vector or CHOP and 2 days later treated with TG (500 nM) for indicated intervals. Protein extracts from these cells were immunoblotted for CHOP and miR-216b levels quantified by qPCR (n=3). (g) MiR-216b expression and (h) c-Jun mRNA levels were analysed in MMTV-Neu tumours from either PERK+/+ or a PERK-/background. Data represent mean±s.d. of three independent observations. Statistical significance was analysed analysed by Student's t-test. (*P<0.05, WT versus -/-). Figure provided by CiteAb. Source: Nat Commun, PMID: 27173017.

www.rockland.com Page 3 of 7





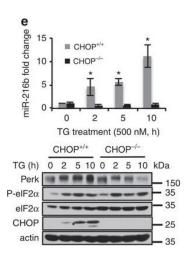


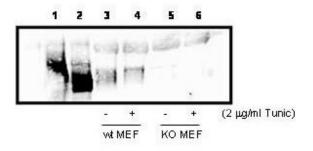
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www.rockland.com Page 4 of 7







Western Blot

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Western Blot

Western blot analysis using Rockland Immunochemical's anti-PERK to detect PERK in cell lysates. 300µg PERK over-expressing 293T cell lysate (lanes 1 & 2), or 800µg wild type (Lanes 3 & 4), and PERK knock out (lanes 5 & 6) MEF cell lysate were immunoprecipated with 15µl anti-PERK, followed by western blotting with 1:1000 dilution of anti-PERK in 5% milk/TBST buffer. Lane 1, 293T cells over-expressing Myc-PERK wt, Lane 2, 293T cells over-expressing Myc-PERK K618A. Personal Communication. A, Diehl, Univ. of Pennsylvania, Philadelphia, PA.

www.rockland.com Page 5 of 7





Immunohistochemistry

Immunohistochemistry staining of mouse mammary gland samples from lactating mice (L10) with Rockland Immunochemical's anti-PERK. Positive staining signal observed in wild type mouse sample with anti-PERK staining only (middle image), but not in the knock out mouse sample (right image) and pre-immune serum staining (left image) The anti-PERK was diluted 1:1,000 in 5% goat serum in PBS and allowed to incubate for 2h at room temperature in a humidified chamber. Personal Communication. A, Diehl, Univ. of Pennsylvania, Philadelphia, PA.



References

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www.rockland.com Page 6 of 7





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www.rockland.com Page 7 of 7