

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**Background:**

The PKR-like endoplasmic reticulum kinase (PERK, also known 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 in 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.

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

Gene Name:	Eif2ak3
Reactivity:	Mouse
Immunogen Type:	Recombinant Protein
Immunogen:	This whole rabbit serum was prepared by repeated immunizations with a recombinant fusion protein from amino acids 601-1115 of mouse deltaN PERK.
Purity/Specificity:	This antiserum is directed against PERK and reacts with the PERK from mouse tissues. Reactivity to other species is unknown.
Relevant Links:	• NCBI - 32451785 • UniProtKB - Q9Z2B5 • GenelD - 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

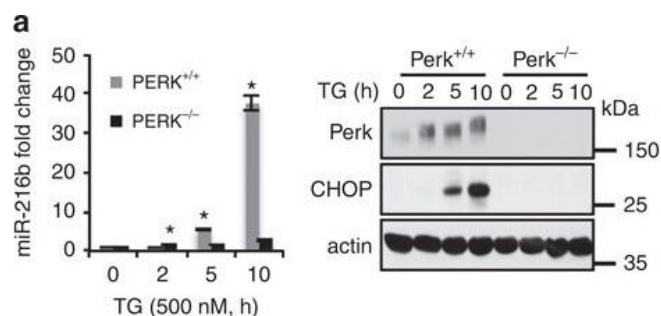
Formulation

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

Shipping & Handling

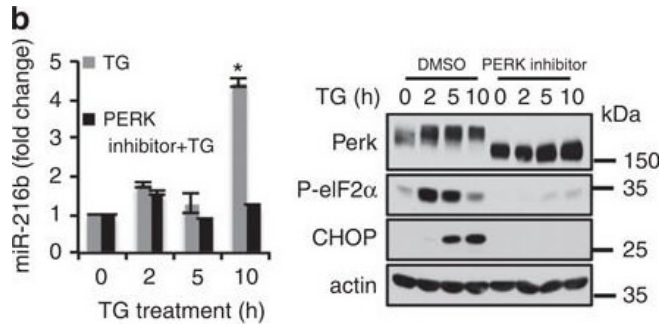
Shipping Condition:	Dry Ice
Storage Condition:	Store vial at -20° C prior to opening. Aliquot contents and freeze at -20° C or below for extended storage. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several 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



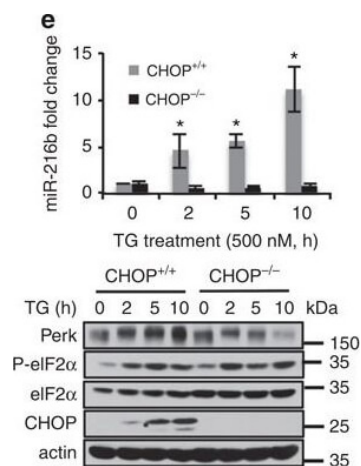
Western Blot

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 $\pm$ s.d. of three independent observations. Statistical significance was analysed by Student's t-test. (*P<0.05, WT versus -/-). Figure provided by CiteAb. Source: Nat Commun, PMID: 27173017.



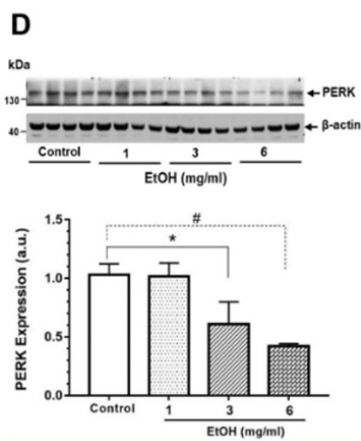
Western Blot

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 $\pm$ s.d. of three independent observations. Statistical significance was analysed by Student's t-test. (*P<0.05, WT versus -/-). Figure provided by CiteAb. Source: Nat Commun, PMID: 27173017.



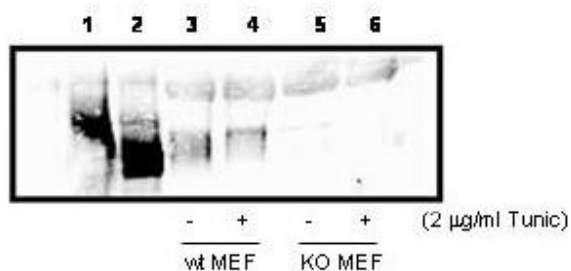
Western Blot

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 $\pm$ s.d. of three independent observations. Statistical significance was analysed by Student's t-test. (*P<0.05, WT versus -/-). Figure provided by CiteAb. Source: Nat Commun, PMID: 27173017.



Western Blot

EtOH-induced ER stress and UPR signaling in hPACs. Immunoblots along with respective bar diagram show expression levels of PERK (D.) Intensities were normalized to β -actin (loading control). Values are expressed as Mean $\pm$ SEM (n =16 replicates). *, #, † p-value $\leq$ 0.05. Fig 5. PMID: 32717227.



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 800ug wild type (Lanes 3 & 4), and PERK knock out (lanes 5 & 6) MEF cell lysate were immunoprecipitated 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.

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

- Goyon V et al. MAPL loss dysregulates bile and liver metabolism in mice. *EMBO Rep.* (2023)
- Srinivasan MP et al. Exposure to binge ethanol and fatty acid ethyl esters exacerbates chronic ethanol-induced pancreatic injury in hepatic alcohol dehydrogenase deficient deer mice. *Am J Physiol Gastrointest Liver Physiol.* (2022)
- Srinivasan MP et al. Activation of AMP-activated protein kinase attenuates ethanol-induced ER/oxidative stress and lipid phenotype in human pancreatic acinar cells. *Biochem Pharmacol.* (2020)
- Kaphalia L et al. Ethanol exposure impairs AMPK signaling and phagocytosis in human alveolar macrophages: Role of ethanol metabolism. *Alcohol Clin Exp Res.* (2019)
- Xu Z et al. Linking dysregulated AMPK signaling and ER stress in ethanol-induced liver injury in hepatic alcohol dehydrogenase deficient deer mice. *Nat Commun.* (2019)
- Maekawa et al. ATF7 mediates TNF- α -induced telomere shortening. *Nucleic Acids Research* (2018)
- Howley et al. A CREB3-regulated ER-Golgi trafficking signature promotes metastatic progression in breast cancer. *Oncogene* (2018)
- Xu et al. miR-216b regulation of c-Jun mediates GADD153/CHOP-dependent apoptosis. *Nature Communications* (2016)
- Mukhopadhyay et al. Casitas B-cell lymphoma (Cbl) proteins protect mammary epithelial cells from proteotoxicity of active c-Src accumulation. *Proc. Natl. Acad. Sci. U.S.A* (2016)

Disclaimer

This product is for research use only and is not intended for therapeutic or diagnostic applications. Please contact a technical service representative for more information. All products of animal origin manufactured by Rockland Immunochemicals are derived from starting materials of North American origin. Collection was performed in United States Department of Agriculture (USDA) inspected facilities and all materials have been inspected and certified to be free of disease and suitable for exportation. All properties listed are typical characteristics and are not specifications. All suggestions and data are offered in good faith but without guarantee as conditions and methods of use of our products are beyond our control. All claims must be made within 30 days following the date of delivery. The prospective user must determine the suitability of our materials before adopting them on a commercial scale. Suggested uses of our products are not recommendations to use our products in violation of any patent or as a license under any patent of Rockland Immunochemicals, Inc. If you require a commercial license to use this material and do not have one, then return this material, unopened to: Rockland Inc., P.O. BOX 5199, Limerick, Pennsylvania, USA.