Grp94 monoclonal antibody (9G10)

Grp94 (Glucose-regulated protein 94) is an abundant resident endoplasmic reticulum (ER) lumenal stress protein, which together with cytosolic Hsp90 belongs to the Hsp90 family of molecular chaperones. Grp94 expression is upregulated by stress conditions such as glucose starvation and heat shock, which promote protein misfolding or unfolding. In addition to a homeostatic role in protein folding and assembly, Grp94 can function in the intracellular trafficking of peptides from the extracellular space to the MHC class I antigen processing pathway of antigen presentation cells.

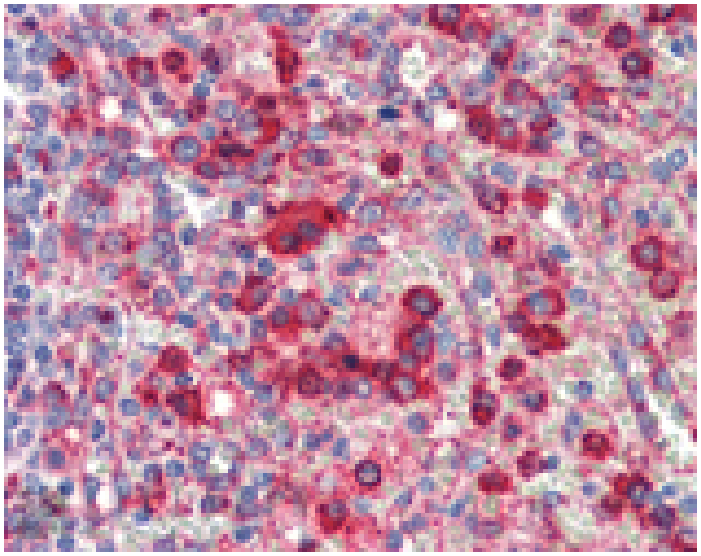
This antibody is covered by our Worry-Free Guarantee.

Citations: 98 View Online »

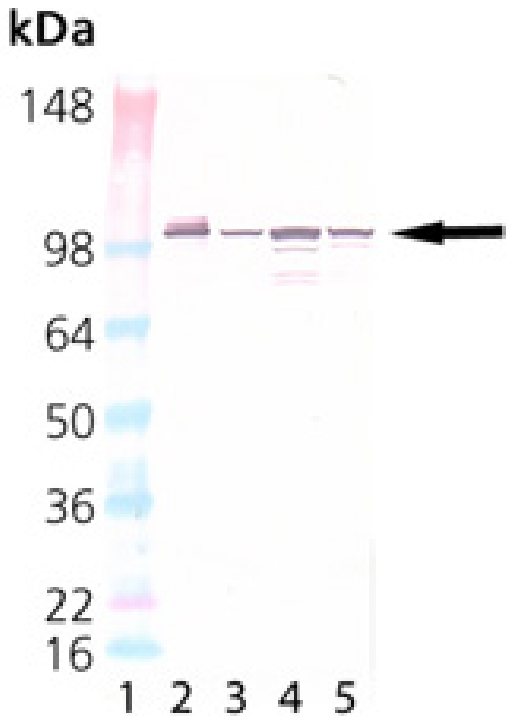
Ordering Information Order Online »

ADI-SPA-850-D	50µg
ADI-SPA-850-F	200µg

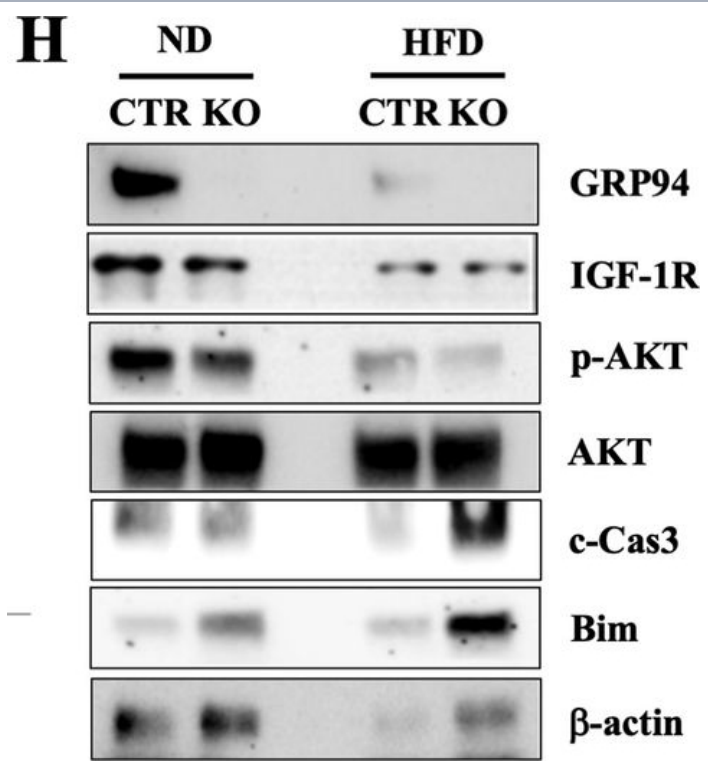
Manuals, SDS & CofA View Online »



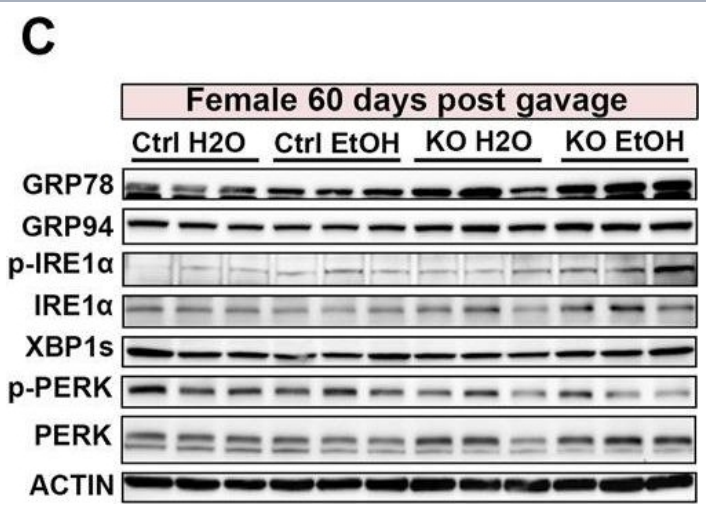
Immunohistochemistry analysis of human spleen tissue stained with Grp94, mAb (9G10) at 10µg/ml.



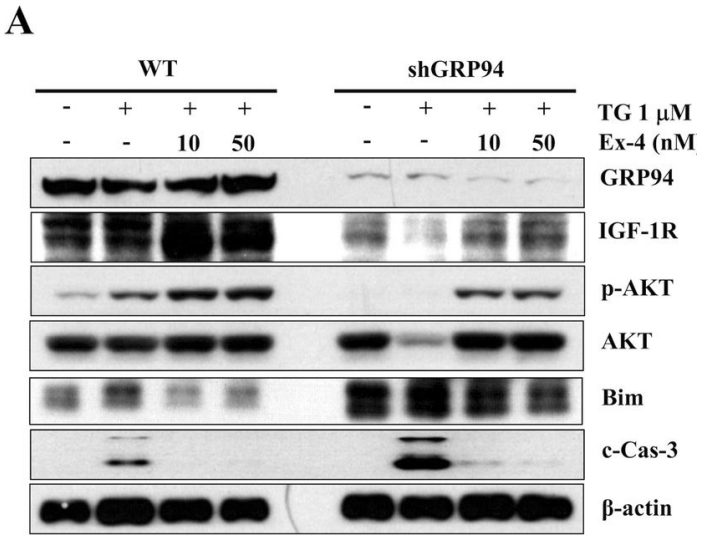
Western Blot Analysis of Grp94: Lane 1: MW Marker, Lane 2: Grp94 (canine), (recombinant) (Prod No. ADI-SPP-766), Lane 3: HeLa, (cell lysate) (Prod No. ADI-LYC-HL100), Lane 4: Mouse Liver Lysate, Lane 5: Vero Cell Lysate



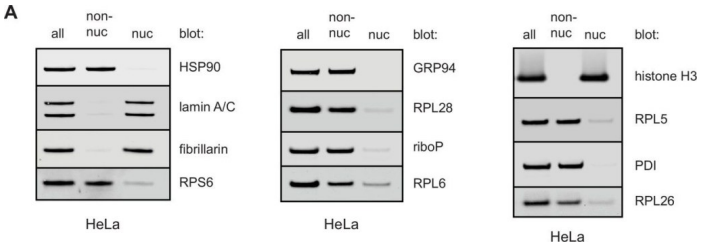
GRP94 deletion increases β cell susceptibility to HFD-induced β cell death and diabetes progression.GRP94 KO (n = 12) and Cre control (n = 12) mice fed normal diet (ND) or HFD for 20 weeks. Random-fed blood glucose levels (A), blood glucose levels during an IPGTT (B), and area under the curve (AUC) during an IPGTT after HFD (C). *p < 0.05 versus control-ND, #p < 0.05 versus KO-ND, $p < 0.05 versus control-HFD, one-way ANOVA. D C-peptide secretion during an IPGTT measured before (0 min), 15 min, and 30 min after glucose injection. *p < 0.05 versus control-ND, #p < 0.05 versus KO-ND, $p < 0.05 versus control-HFD, one-way ANOVA. E β cell mass was analyzed in ND-fed and HFD-fed mice. Ten pancreatic sections from each individual mouse (N = 4 per group) were analyzed. *p < 0.05 versus control-ND, #p < 0.05 versus KO-ND, $p < 0.05 versus control-HFD, one-way ANOVA. F Fluorescence analysis from triple staining for TUNEL, insulin, and DAPI. White arrows point to TUNEL+ cells. G Histogram shows percentages of TUNEL-positive β cells in each group. Scale bar, 50 μm. *p < 0.05 versus control-ND, #p < 0.05 versus KO-ND, $p < 0.05 versus control-HFD; one-way ANOVA. H Protein expression in mouse islets isolated from all 4 treatment groups at week 21. Immunoblot shows relative protein expression of GRP94, IGF-1R, p-AKT, AKT, c-Cas-3, and β-actin. *P < 0.05.



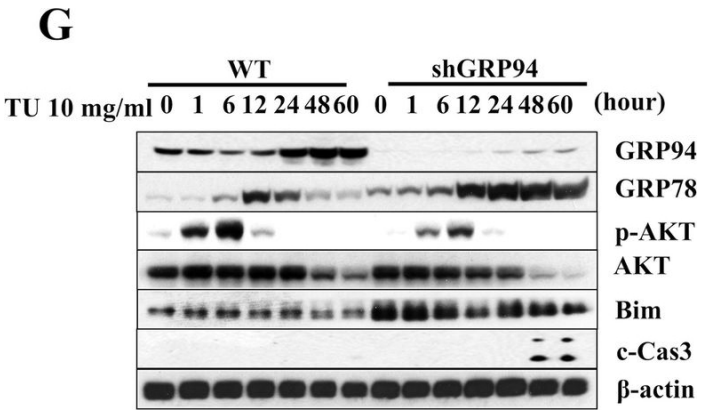
Effects of binge alcohol exposure and neuronal MANF deficiency on neuronal ER homeostasis. (A–D) Representative immunoblots (A, C) and quantification (B, D) of ER stress markers in female control and MANF KO cerebral cortex 1 day (A, B) or 60 days (C–D) post H2O or EtOH treatment. (E, H) Representative immunoblots (E, G) and quantification (F, H) of ER stress markers in male control and MANF KO cerebral cortex 1 day (E, F) or 60 days (G, H) post H2O or EtOH treatment. All data were expressed as mean ± SEM. n = 3 per group. Two-way ANOVA followed by Tukey's post hoc test. *p < 0.05, **p < 0.01, ***p < 0.001 when compared to H2O treated control. #p < 0.05, ##p < 0.01, ###p < 0.001 when compared to EtOH treated control. @p < 0.05, @@p < 0.01 when compared to H2O treated KO.



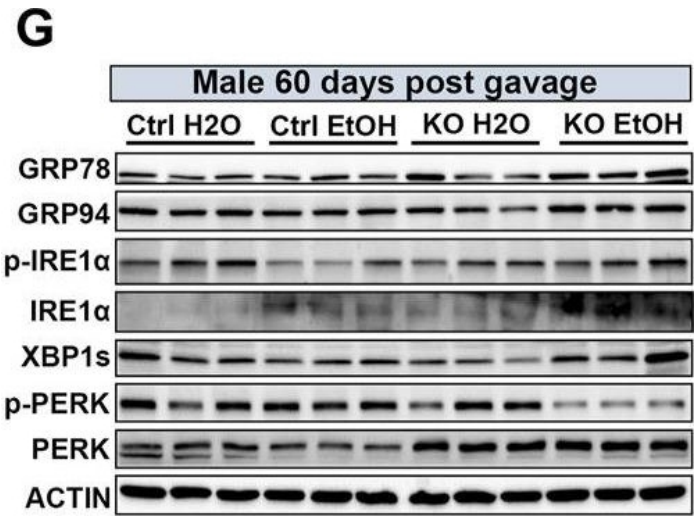
Treatment with Exendin-4 or overexpression of IGF-1R or GRP94 protects β cells from TG-induced apoptosis.A WT and GRP94 KD cells were treated with 1 μM TG in the absence or presence of 10 nM or 50 nM Exendin-4 for 6 h. Total cell extracts were analyzed by immunoblot for GRP94, IGF-1R, p-AKT, AKT, Bim, c-Cas-3, and β-actin. B WT and GRP94 KD cells were transfected with control plasmid (p.babe plasmid) or IGF-1R overexpression (p.babe-IGF-1R) plasmid and then treated with TG for 6 h or TU for 48 h. Total cell extracts were then analyzed by immunoblot for GRP94, IGF-1R, p-AKT, AKT, Bim, c-Cas-3, and β-actin. C GRP94 KD cells were transfected with GRP94 WT plasmid. Total cell extracts were then analyzed by immunoblot for GRP94, IGF-1R, p-AKT, Bim, and β-actin.



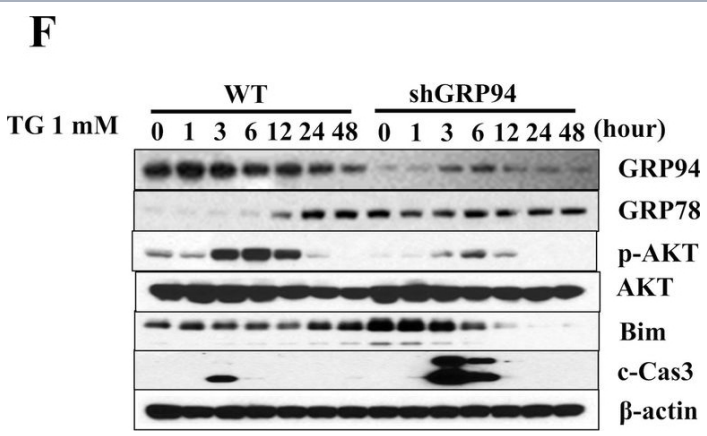
Fractionation of HeLa or T cells reveals few ribosomal components in nuclear lysates.HeLa cells (A), freshly isolated resting OT-I T cells (B), or OT-I T cells stimulated with PMA/ionomycin and IL-2 in vitro for 2 days (C) were either lysed directly in sodium dodecyl sulfate (SDS) extraction buffer (all) or subjected to a hypotonic lysis procedure to isolate non-nuclear lysates and nuclear lysates. Equal amounts of each fraction were subjected to immunoblotting for markers typical of the cytosol, ER, and nucleus. Antibodies against ribosomal proteins were used to determine where the majority of ribosomal proteins (and therefore ribosomes) fractionated. Controls with antibodies specific for nucleolar located fibrillarin, histone H3, and lamin A/C establish lack of nuclear contamination in non-nuclear fractions. ER and cytoplasmic proteins HSP90, GRP94, PDI, and actin indicate lack of contamination in the nuclear fraction. Representative of two experiments.Figure 6—figure supplement 1—source data 1.Uncropped and outlined immunoblot images related to Figure 6—figure supplement 1.Figure 6—figure supplement 1—source data 2.Uncropped immunoblot images related to Figure 6—figure supplement 1.Uncropped and outlined immunoblot images related to Figure 6—figure supplement 1.Uncropped immunoblot images related to Figure 6—figure supplement 1.



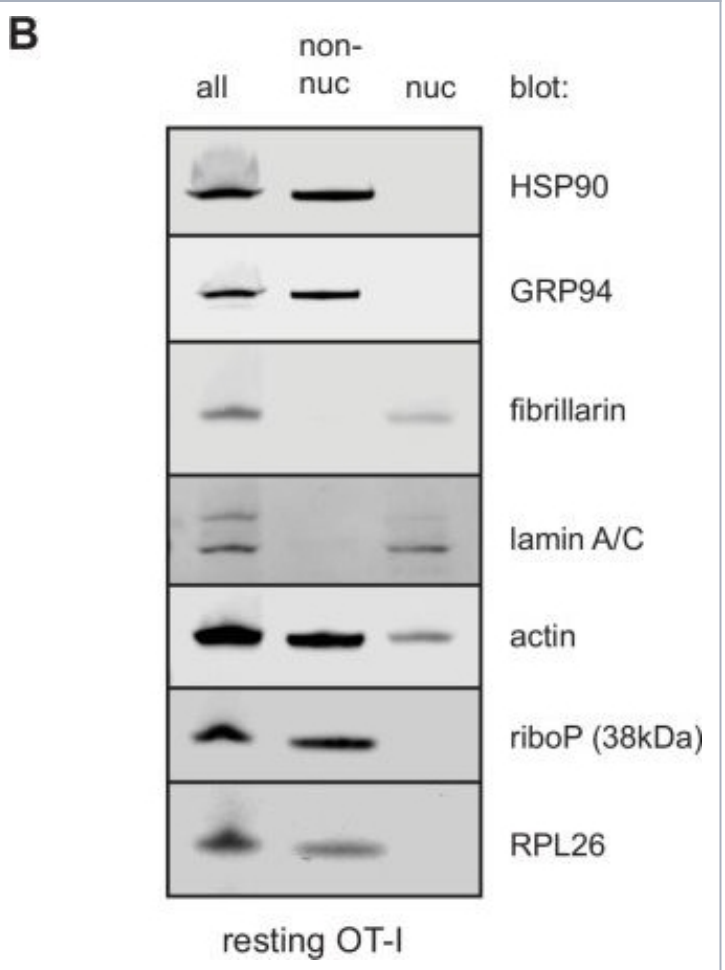
GRP94 KD cells are more susceptible to stress-induced cell death.A Relative GRP94 mRNA expression in WT control and Knockdown cells (shGRP94). B Protein expression of GRP94 in WT and KD cells. C Viability of WT control (white columns) and KD (black columns) cells after TG (1 μM)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 24 h WT, %p < 0.05 versus 24 h shGRP94, ^p < 0.05 versus 48 h WT, one-way ANOVA, D TU (10 μg/ml)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 48 h WT, %p < 0.05 versus 48 h shGRP94, ^p < 0.05 versus 72 h WT, one-way ANOVA, or E Pal (200 μM)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 24 h WT, %p < 0.05 versus 24 h shGRP94, ^p < 0.05 versus 48 h WT, one-way ANOVA, at different time after treatment. Cell death was measured by trypan blue (n = 3). F–H Protein expression of GRP94, GRP78, p-AKT, AKT, Bim, cleaved Caspase-3 (c-Cas-3), and β-actin in WT control and GRP94 KD cells at indicated times after TG, TU, or Pal treatment as analyzed by immunoblot.



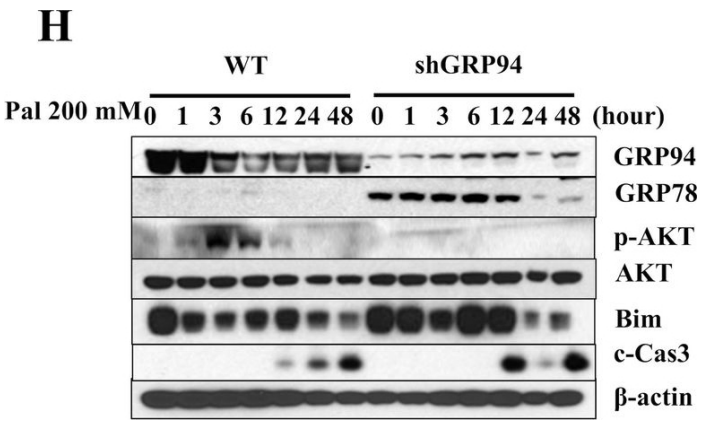
Effects of binge alcohol exposure and neuronal MANF deficiency on neuronal ER homeostasis. (A–D) Representative immunoblots (A, C) and quantification (B, D) of ER stress markers in female control and MANF KO cerebral cortex 1 day (A, B) or 60 days (C–D) post H2O or EtOH treatment. (E, H) Representative immunoblots (E, G) and quantification (F, H) of ER stress markers in male control and MANF KO cerebral cortex 1 day (E, F) or 60 days (G, H) post H2O or EtOH treatment. All data were expressed as mean ± SEM. n = 3 per group. Two-way ANOVA followed by Tukey's post hoc test. *p < 0.05, **p < 0.01, ***p < 0.001 when compared to H2O treated control. #p < 0.05, ##p < 0.01, ###p < 0.001 when compared to EtOH treated control. @p < 0.05, @@p < 0.01 when compared to H2O treated KO.



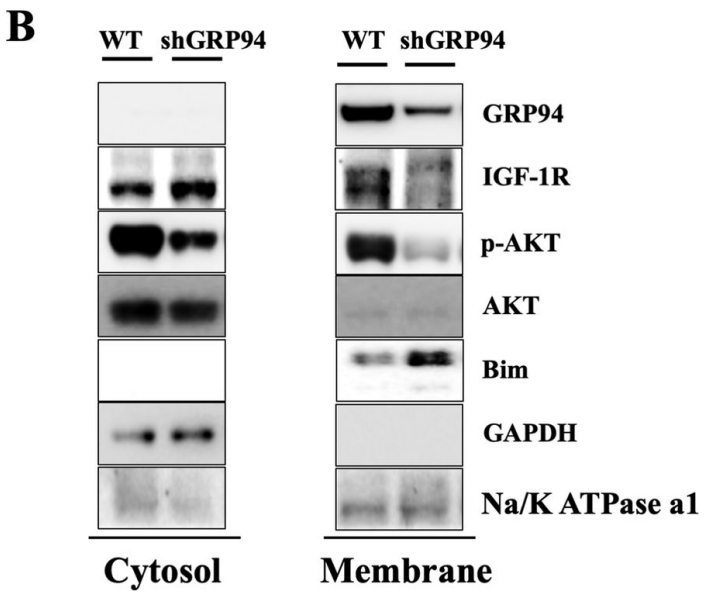
GRP94 KD cells are more susceptible to stress-induced cell death.A Relative GRP94 mRNA expression in WT control and Knockdown cells (shGRP94). B Protein expression of GRP94 in WT and KD cells. C Viability of WT control (white columns) and KD (black columns) cells after TG (1 μM)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 24 h WT, %p < 0.05 versus 24 h shGRP94, ^p < 0.05 versus 48 h WT, one-way ANOVA, D TU (10 μg/ml)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 48 h WT, %p < 0.05 versus 48 h shGRP94, ^p < 0.05 versus 72 h WT, one-way ANOVA, or E Pal (200 μM)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 24 h WT, %p < 0.05 versus 24 h shGRP94, ^p < 0.05 versus 48 h WT, one-way ANOVA, at different time after treatment. Cell death was measured by trypan blue (n = 3). F–H Protein expression of GRP94, GRP78, p-AKT, AKT, Bim, cleaved Caspase-3 (c-Cas-3), and β-actin in WT control and GRP94 KD cells at indicated times after TG, TU, or Pal treatment as analyzed by immunoblot.



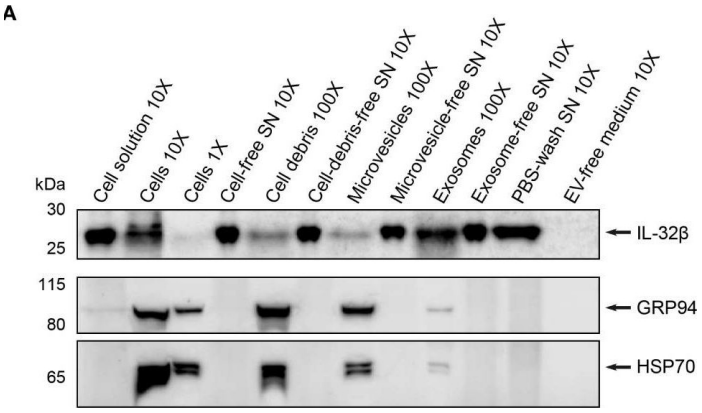
Fractionation of HeLa or T cells reveals few ribosomal components in nuclear lysates.HeLa cells (A), freshly isolated resting OT-I T cells (B), or OT-I T cells stimulated with PMA/ionomycin and IL-2 in vitro for 2 days (C) were either lysed directly in sodium dodecyl sulfate (SDS) extraction buffer (all) or subjected to a hypotonic lysis procedure to isolate non-nuclear lysates and nuclear lysates. Equal amounts of each fraction were subjected to immunoblotting for markers typical of the cytosol, ER, and nucleus. Antibodies against ribosomal proteins were used to determine where the majority of ribosomal proteins (and therefore ribosomes) fractionated. Controls with antibodies specific for nucleolar located fibrillarin, histone H3, and lamin A/C establish lack of nuclear contamination in non-nuclear fractions. ER and cytoplasmic proteins HSP90, GRP94, PDI, and actin indicate lack of contamination in the nuclear fraction. Representative of two experiments.Figure 6—figure supplement 1—source data 1.Uncropped and outlined immunoblot images related to Figure 6—figure supplement 1.Figure 6—figure supplement 1—source data 2.Uncropped immunoblot images related to Figure 6—figure supplement 1.Uncropped and outlined immunoblot images related to Figure 6—figure supplement 1.Uncropped immunoblot images related to Figure 6—figure supplement 1.



GRP94 KD cells are more susceptible to stress-induced cell death.A Relative GRP94 mRNA expression in WT control and Knockdown cells (shGRP94). B Protein expression of GRP94 in WT and KD cells. C Viability of WT control (white columns) and KD (black columns) cells after TG (1 μM)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 24 h WT, %p < 0.05 versus 24 h shGRP94, ^p < 0.05 versus 48 h WT, one-way ANOVA, D TU (10 μg/ml)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 48 h WT, %p < 0.05 versus 48 h shGRP94, ^p < 0.05 versus 72 h WT, one-way ANOVA, or E Pal (200 μM)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 24 h WT, %p < 0.05 versus 24 h shGRP94, ^p < 0.05 versus 48 h WT, one-way ANOVA, at different time after treatment. Cell death was measured by trypan blue (n = 3). F–H Protein expression of GRP94, GRP78, p-AKT, AKT, Bim, cleaved Caspase-3 (c-Cas-3), and β-actin in WT control and GRP94 KD cells at indicated times after TG, TU, or Pal treatment as analyzed by immunoblot.

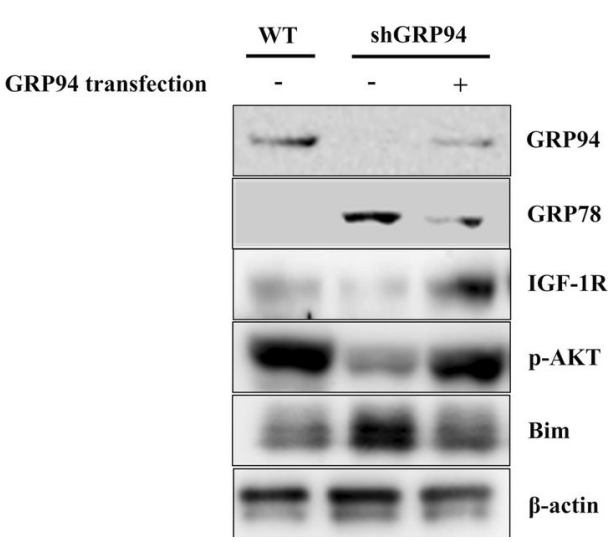


GRP94 is required for membrane expression/maturation of IGF-1R.A Immunoblot analysis of GRP94, IGF-1R, IR, p-AKT, AKT, Bim, and β-actin from total lysates of WT and GRP94 KD cells. B Immunoblot analysis of GRP94, IGF-1R, p-AKT, AKT, Bim, and GAPDH from cytosol or membrane of WT and GRP94 KD cells. C Immunoblot analysis of GRP94, IGF-1R, p-AKT, AKT, Bim, and β-actin in islets harvested from 8-weeks old control or GRP94 KO KO mice. D Immunofluorescence analysis of GRP94 (red), IGF-1R (green), and nucleus (blue) in WT or KO mouse islets. Scale bar, 25 μm.



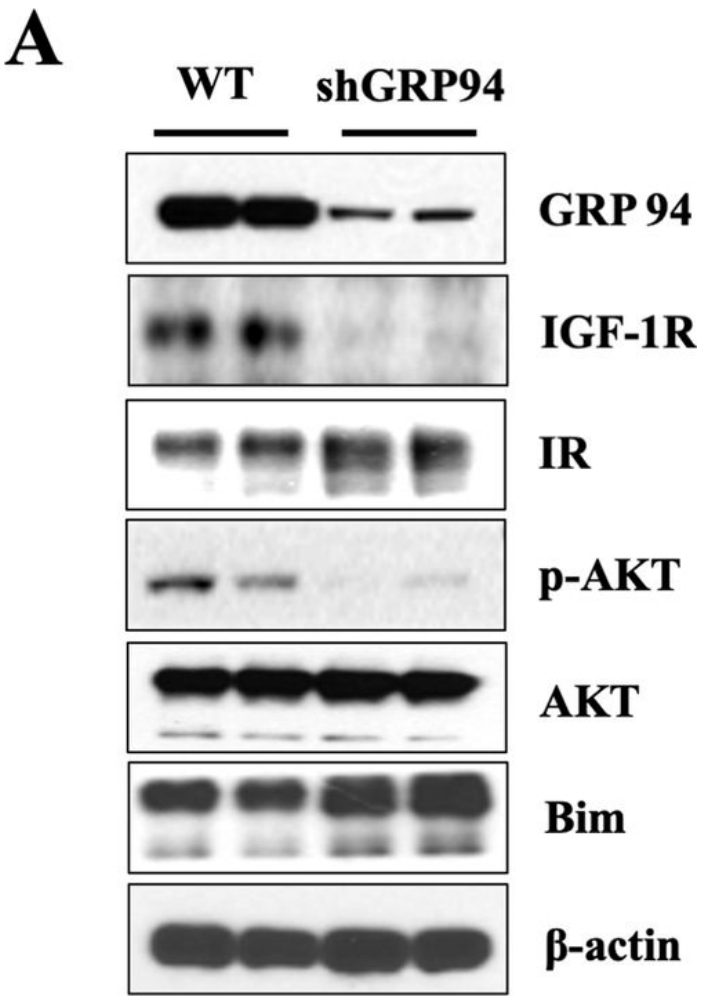
IL-32 is secreted by T cells predominantly as a free protein. (A) Representative WB analyses of IL-32β and the microvesicle/exosome markers GRP94 and HSP70 in lysates of cells, cell-debris, microvesicles and exosomes, into the respective cell solution and cell-free, cell debris-free, microvesicle-free and exosome-free supernatant (SN), the PBS-wash SN, which was used to wash the exosome pellet, and in the EV-free medium used to culture Survivin-specific T cells for 4 h with anti-CD3/CD28 antibodies. The loaded quantity of each lysate and SN sample corresponds to 0.16×106 (1X), 1.6×106 (10X) and 16×106 (100X) Survivin-specific T cells and the amount of PBS-wash SN derived from this cell number or the volume of EV-free medium used to culture this cell number. MW in kDa (IL-32β: 23.1, GRP94: 98, HSP70: 70). IL-32β, GRP94, HSP70: n=4. n represents independent experiments. (B) Cumulative data of quantified IL-32β expression in lysates and supernatants displayed by the Area Under the Curve (AUC) for n=4 independent experiments normalized to 0.16×106 Survivin-specific T cells. Dots depict data from individual experiments, Student's paired t-test, *p ≤ 0.05.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: IL-2 and TCR stimulation induce expression and secretion of IL-32β by human T cells. *Front Immunol* (2024)

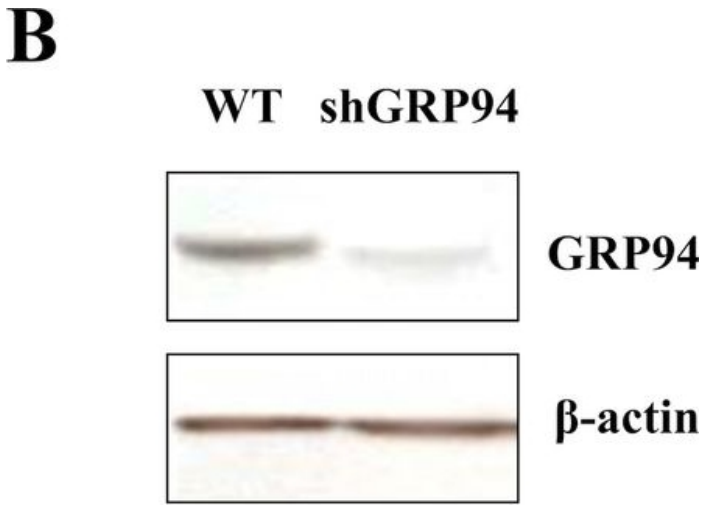


Treatment with Exendin-4 or overexpression of IGF-1R or GRP94 protects β cells from TG-induced apoptosis.A WT and GRP94 KD cells were treated with 1 μM TG in the absence or presence of 10 nM or 50 nM Exendin-4 for 6 h. Total cell extracts were analyzed by immunoblot for GRP94, IGF-1R, p-AKT, AKT, Bim, c-Cas-3, and β-actin. B WT and GRP94 KD cells were transfected with control plasmid (p.babe plasmid) or IGF-1R overexpression (p.babe-IGF-1R) plasmid and then treated with TG for 6 h or TU for 48 h. Total cell extracts were then analyzed by immunoblot for GRP94, IGF-1R, p-AKT, AKT, Bim, c-Cas-3, and β-actin. C GRP94 KD cells were transfected with GRP94 WT plasmid. Total cell extracts were then analyzed by immunoblot for GRP94, IGF-1R, p-AKT, Bim, and β-actin.

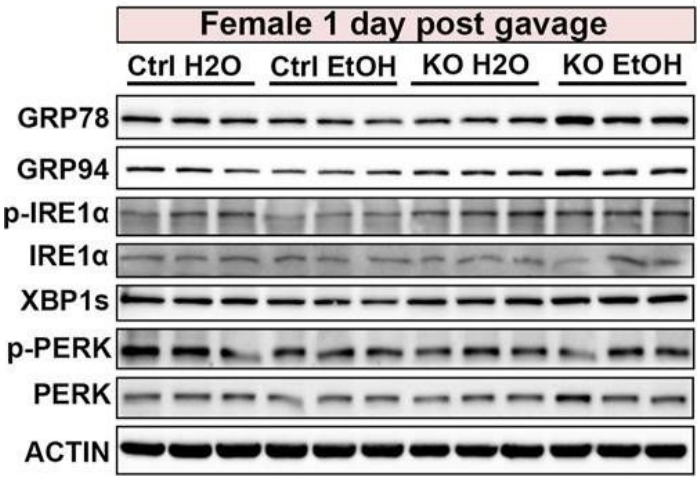
Image collected and cropped by CiteAb under a CC-BY license from the following publication: GRP94 is an IGF-1R chaperone and regulates beta cell death in diabetes. *Cell Death Dis* (2024)



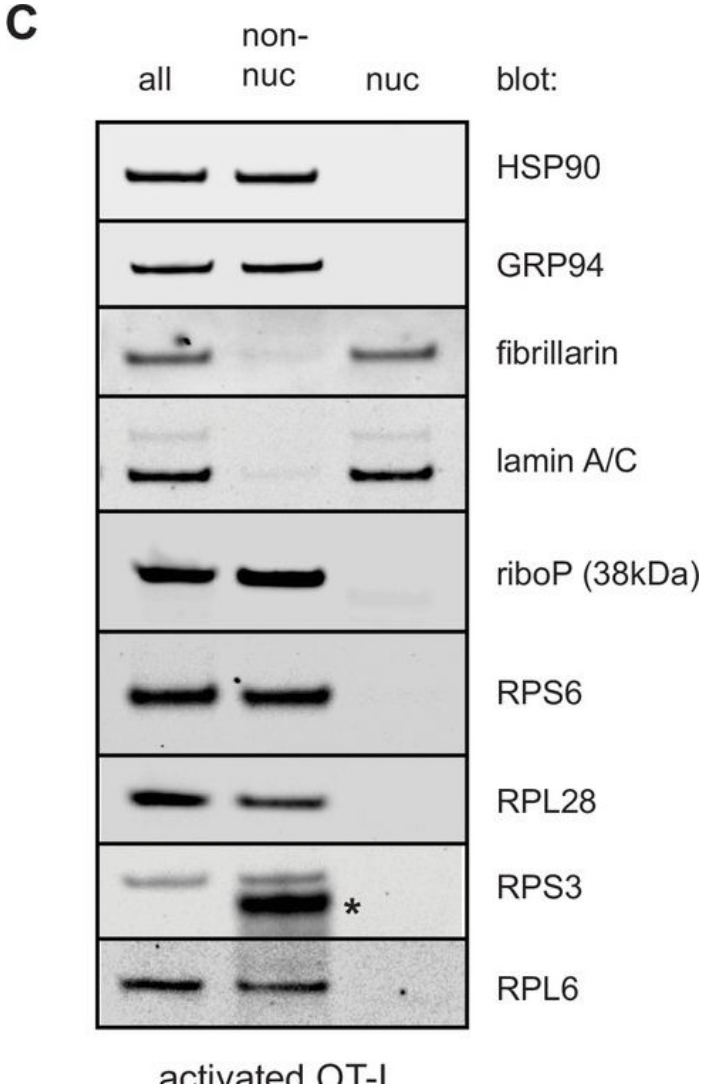
GRP94 is required for membrane expression/maturation of IGF-1R.A Immunoblot analysis of GRP94, IGF-1R, IR, p-AKT, AKT, Bim, and β-actin from total lysates of WT and GRP94 KD cells. B Immunoblot analysis of GRP94, IGF-1R, p-AKT, AKT, Bim, and GAPDH from cytosol or membrane of WT and GRP94 KD cells. C Immunoblot analysis of GRP94, IGF-1R, p-AKT, AKT, Bim, and β-actin in islets harvested from 8-weeks old control or GRP94 KO KO mice. D Immunofluorescence analysis of GRP94 (red), IGF-1R (green), and nucleus (blue) in WT or KO mouse islets. Scale bar, 25 μm.



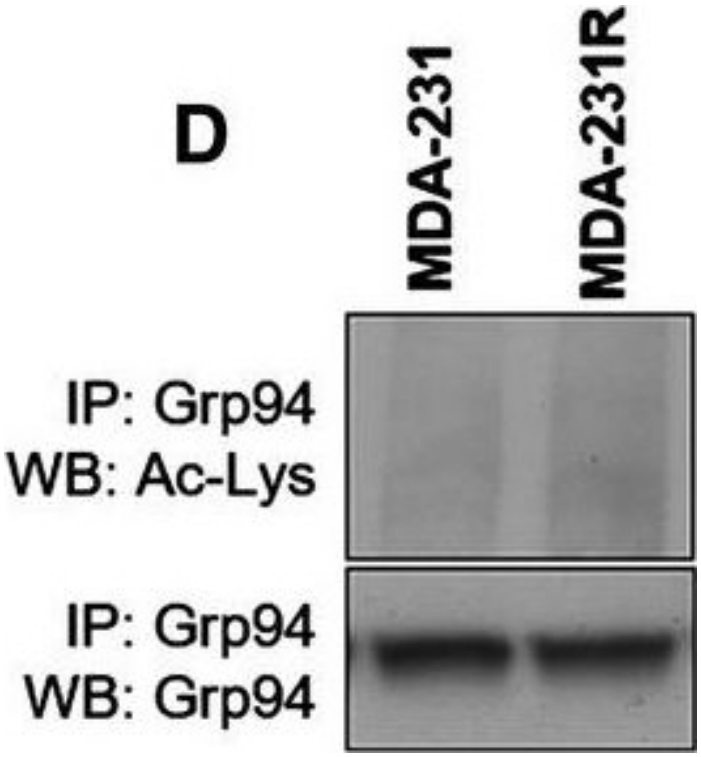
GRP94 KD cells are more susceptible to stress-induced cell death.A Relative GRP94 mRNA expression in WT control and Knockdown cells (shGRP94). B Protein expression of GRP94 in WT and KD cells. C Viability of WT control (white columns) and KD (black columns) cells after TG (1 μM)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 24 h WT, %p < 0.05 versus 24 h shGRP94, ^p < 0.05 versus 48 h WT, one-way ANOVA, D TU (10 μg/ml)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 48 h WT, %p < 0.05 versus 48 h shGRP94, ^p < 0.05 versus 72 h WT, one-way ANOVA, or E Pal (200 μM)*p < 0.05 versus con WT, #p < 0.05 versus con shGRP94, $p < 0.05 versus 24 h WT, %p < 0.05 versus 24 h shGRP94, ^p < 0.05 versus 48 h WT, one-way ANOVA, at different time after treatment. Cell death was measured by trypan blue (n = 3). F–H Protein expression of GRP94, GRP78, p-AKT, AKT, Bim, cleaved Caspase-3 (c-Cas-3), and β-actin in WT control and GRP94 KD cells at indicated times after TG, TU, or Pal treatment as analyzed by immunoblot.



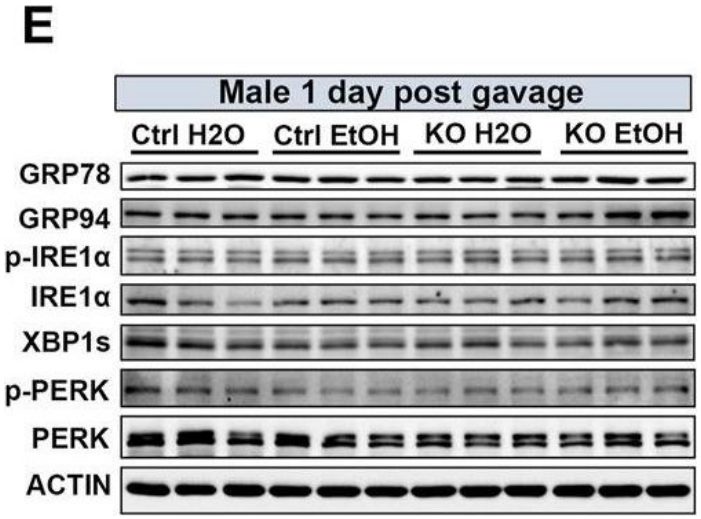
Effects of binge alcohol exposure and neuronal MANF deficiency on neuronal ER homeostasis. (A–D) Representative immunoblots (A, C) and quantification (B, D) of ER stress markers in female control and MANF KO cerebral cortex 1 day (A, B) or 60 days (C–D) post H2O or EtOH treatment. (E, H) Representative immunoblots (E, G) and quantification (F, H) of ER stress markers in male control and MANF KO cerebral cortex 1 day (E, F) or 60 days (G, H) post H2O or EtOH treatment. All data were expressed as mean ± SEM. n = 3 per group. Two-way ANOVA followed by Tukey's post hoc test. *p < 0.05, **p < 0.01, ***p < 0.001 when compared to H2O treated control. #p < 0.05, ##p < 0.01, ###p < 0.001 when compared to EtOH treated control. @p < 0.05, @@p < 0.01 when compared to H2O treated KO.



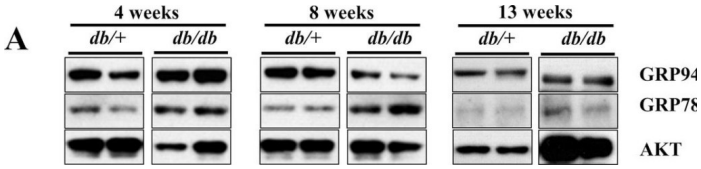
Fractionation of HeLa or T cells reveals few ribosomal components in nuclear lysates.HeLa cells (A), freshly isolated resting OT-I T cells (B), or OT-I T cells stimulated with PMA/ionomycin and IL-2 in vitro for 2 days (C) were either lysed directly in sodium dodecyl sulfate (SDS) extraction buffer (all) or subjected to a hypotonic lysis procedure to isolate non-nuclear lysates and nuclear lysates. Equal amounts of each fraction were subjected to immunoblotting for markers typical of the cytosol, ER, and nucleus. Antibodies against ribosomal proteins were used to determine where the majority of ribosomal proteins (and therefore ribosomes) fractionated. Controls with antibodies specific for nucleolar located fibrillarin, histone H3, and lamin A/C establish lack of nuclear contamination in non-nuclear fractions. ER and cytoplasmic proteins HSP90, GRP94, PDI, and actin indicate lack of contamination in the nuclear fraction. Representative of two experiments.Figure 6—figure supplement 1—source data 1.Uncropped and outlined immunoblot images related to Figure 6—figure supplement 1.Figure 6—figure supplement 1—source data 2.Uncropped immunoblot images related to Figure 6—figure supplement 1.Uncropped and outlined immunoblot images related to Figure 6—figure supplement 1.Uncropped immunoblot images related to Figure



Altered NQO1 levels, HDAC family member expression and altered acetylation status in 17□AAG□resistant cell lines. Analysis of parental and resistant cell lines demonstrated altered expression levels of a number of molecules. Semiquantitative PCR demonstrated that the expression levels of NQO1 in resistant MDA□435 cells were decreased when compared with parental cells, while no alteration was noted between MDA□231 and MDA□231R cell lines (A). Western blot analysis of parental and resistant MDA□231 total cell lysates examining levels of HDAC family members in the presence and absence of 17□AAG for a period of 24 h (B). Analysis of acetylated HSP90 by immunoprecipitation of HSP90 and western blot analysis with antiacetylated lysine antibody of total cell lysates of parental and resistant MDA□231 cells treated with and without 17□AAG demonstrated increased acetylated HSP90 (C). Analysis of acetylation of Grp94 (D) and Trap1 (E) by immunoprecipitation and western blot analysis of MDA□231 and MDA□231R total cell lysates demonstrated no alteration in acetylation status. Acetylated lysine residue was detected by western blotting. Western blot analysis of acetylated histone 3 in parental and resistant MDA□231 cells treated with and without 17□AAG demonstrated decreased nuclear acetylation (F).

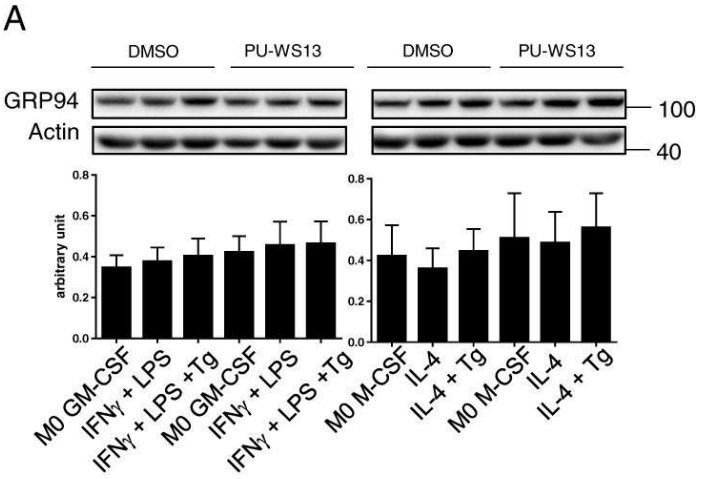


Effects of binge alcohol exposure and neuronal MANF deficiency on neuronal ER homeostasis. (A–D) Representative immunoblots (A, C) and quantification (B, D) of ER stress markers in female control and MANF KO cerebral cortex 1 day (A, B) or 60 days (C–D) post H2O or EtOH treatment. (E, H) Representative immunoblots (E, G) and quantification (F, H) of ER stress markers in male control and MANF KO cerebral cortex 1 day (E, F) or 60 days (G, H) post H2O or EtOH treatment. All data were expressed as mean ± SEM. n = 3 per group. Two-way ANOVA followed by Tukey's post hoc test. *p < 0.05, **p < 0.01, ***p < 0.001 when compared to H2O treated control. #p < 0.05, ##p < 0.01, ###p < 0.001 when compared to EtOH treated control. @p < 0.05, @@p < 0.01 when compared to H2O treated KO.



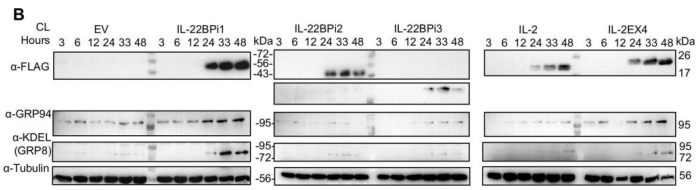
Dynamic change of GRP94 expression in db/db mice at different ages.Expression of GRP94 and GRP78 in mouse islets isolated from 4, 8, and 13-week-old diabetic db/db mice and their heterozygous littermates controls by Western blot (A) and quantification (B). Immunoblots of GRP78, GRP94, and densitometry analyses are shown. *p < 0.05 versus 4wks db/+, #p < 0.05 versus 4wks db/db, $p < 0.05 versus 8wks db/+, %p < 0.05 versus 8wks db/db, ^p < 0.05 versus 4wks db/+, one-way ANOVA. C Immunostaining for GRP94 (green), insulin (red), and nucleus (blue) in sections from 4 and 13-week-old diabetic db/db mice. Scale bar, 50 µm. D Histograms show corrected total cell fluorescence (CTCF) of GRP94 fluorescence quantified by ImageJ software. The CTCF = Integrated Density – (Area of selected cell x Mean fluorescence of background readings). *p < 0.05, Student's t-test.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: GRP94 is an IGF-1R chaperone and regulates beta cell death in diabetes. *Cell Death Dis* (2024)

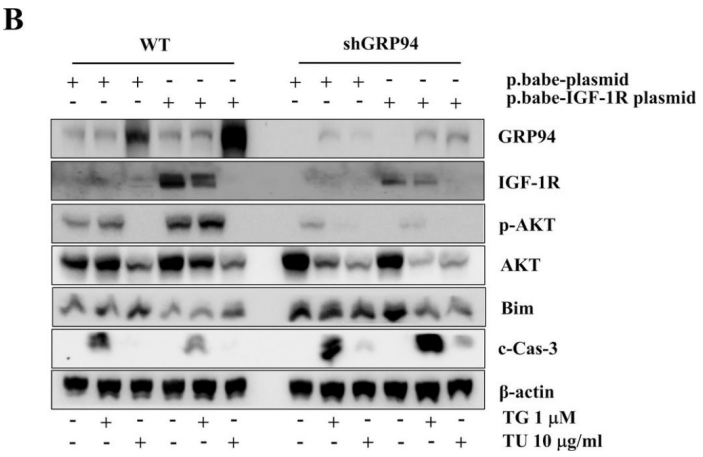


Inhibition of GRP94 by PU-WS13 impairs the effect of Tg-induced ER stress on M2 macrophages.M1 and M2 macrophages from PBMC healthy volunteers were activated in the presence of GM-CSF and M-CSF, respectively. Tg was added to cell cultures during the 48 h activation period. PU-WS13 or DMSO as vehicle was added in cell culture medium at the concentration of 25 µM 24 h before and during all the activation period. A Intracellular GRP94 western-blot analysis (n = 3). B FACS analysis of membrane GRP94 expression (n = 6). C GRP94 quantification by ELISA in activated M2 cell culture supernatants (n = 6). D CD80 and CD206 analysis by flow cytometry (n = 4). E, F Western blot analysis of MMP9 in activated M2 macrophages (n = 4) (E) and of the signalization proteins pSTAT1/STAT1, pSTAT6/STAT6 and pIκB/ IκB (n = 4) (F). G Analysis of pro- and anti-inflammatory cytokines in M2 cell culture supernatants (n = 5–7). IL-1β, IL-6, IFNγ and TNFα were quantified by the Multiplex™ method and TGF-β was quantified by ELISA (*p < 0.05; **p < 0.01; ***p < 0.005; ****p < 0.001).

Image collected and cropped by CiteAb under a CC-BY license from the following publication: The HSP GRP94 interacts with macrophage intracellular complement C3 and impacts M2 profile during ER stress. *Cell Death Dis* (2021)



IL-22BPi1 and IL-2EX4 induce unfolded protein response (UPR) genes. HEK293 cells were transiently transfected with IL-22BPi1, IL-22BPi2, IL-22BPi3, IL-2, IL-2EX4 or empty vector (EV) as control. Cells were collected at the indicated hours after transfection. (A) Expression of different genes related to ER function or UPR were analyzed by RT-qPCR. Each gene expression value is represented as fold change relative to the same time-point expression value of the EV condition and relative to the housekeeping gene GAPDH. Mean ± SEM of three independent experiments. All primers are listed in Supplementary Table 2. (B) GRP78 and GRP94 protein levels correlate with mRNA levels observed in (A). Cell lysates (CL) were immunoblotted for FLAG, GRP94, KDEL and tubulin as loading control. (C) IL-22BPi1 and IL-2EX4 cause XPB1 splicing. XBP1 splicing was detected with conventional PCR for the indicated conditions and times. Un-spliced and spliced XBP1 are indicated as XBP1-u or XBP-1s respectively. (D) IL-22BPi2 secretion was not increased when co-expressed with different ratios of IL-22BPi1. HEK293 cells were co-expressed with different ratios of EV:IL-22BPi1:IL-22BPi2 expression plasmids. 48 h later, secreted IL-22BP in conditioned media (CM) was quantified by ELISA (mean ± SEM; n = 3). (E) Cell viability measured with alamarBlue was not compromised by any of the conditions in two different cell lines. Reduction of alamarBlue was measured after 48 h of transfection and assayed for the indicated times and cell lines. Values are represented as percentage of reduction in each condition relative to EV (mean ± SEM; n = 3).



Treatment with Exendin-4 or overexpression of IGF-1R or GRP94 protects β cells from TG-induced apoptosis.A WT and GRP94 KD cells were treated with 1 μM TG in the absence or presence of 10 nM or 50 nM Exendin-4 for 6 h. Total cell extracts were analyzed by immunoblot for GRP94, IGF-1R, p-AKT, AKT, Bim, c-Cas-3, and β-actin. B WT and GRP94 KD cells were transfected with control plasmid (p.babe plasmid) or IGF-1R overexpression (p.babe-IGF-1R) plasmid and then treated with TG for 6 h or TU for 48 h. Total cell extracts were then analyzed by immunoblot for GRP94, IGF-1R, p-AKT, AKT, Bim, c-Cas-3, and β-actin. C GRP94 KD cells were transfected with GRP94 WT plasmid. Total cell extracts were then analyzed by immunoblot for GRP94, IGF-1R, p-AKT, Bim, and β-actin.

Handling & Storage

Handling	Avoid freeze/thaw cycles.
Long Term Storage	-20°C
Shipping	Blue Ice

Regulatory Status

RUO - Research Use Only

Product Details

Alternative Name	Endoplasmin, Tra1, Hsp90B1, Gp96
Application	Electron microscopy, ICC, IF, IHC (PS), IP, WB
Application Notes	Detects a band of ~98kDa by Western blot.
Clone	9G10
Formulation	Liquid. In PBS containing 50% glycerol and 0.09% sodium azide.
GenBank ID	M14772
Host	Rat
Immunogen	Native chicken Grp94.
Isotype	IgG2a
Purity Detail	Protein G affinity purified.
Recommendation Dilutions/Conditions	Immunoprecipitation (1:100)Western Blot (1:1,000, ECL)Suggested dilutions/conditions may not be available for all applications.Optimal conditions must be determined individually for each application.
Source	Purified from ascites.
Species Reactivity	Bovine, Chicken, Dog, Guinea pig, Hamster, Human, Monkey, Mouse, Porcine, Rabbit, Rat, Sheep, Xenopus

UniProt ID P08110

Worry-free Guarantee **This antibody is covered by our Worry-Free Guarantee .**

Last modified: May 29, 2024

ENZO LIFE SCIENCES, INC.
Phone: 800.942.0430
info-usa@enzolifesciences.com

European Sales Office
ENZO LIFE SCIENCES (ELS) AG
Phone: +41 61 926 8989
info-eu@enzolifesciences.com

Belgium, The Netherlands & Luxembourg
Phone: +32 3 466 0420
info-be@enzolifesciences.com

France
Phone: +33 472 440 655
info-fr@enzolifesciences.com

Germany
Phone: +49 7621 5500 526
info-de@enzolifesciences.com

UK & Ireland
Phone (UK customers): 0845 601 1488
Phone: +44 1392 825900
info-uk@enzolifesciences.com