Product Datasheet

CHREBP Antibody - BSA Free
NB400-135

Unit Size: 0.1 ml

Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.

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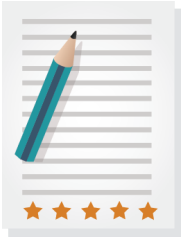
Updated 5/1/2024 v.20.1



NB400-135

CHREBP Antibody - BSA Free

Product Information

Unit Size	0.1 ml
Concentration	1 mg/ml
Storage	Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.
Clonality	Polyclonal
Preservative	0.02% Sodium Azide
Isotype	IgG
Purity	Immunogen affinity purified
Buffer	PBS
Target Molecular Weight	95 kDa

Product Description

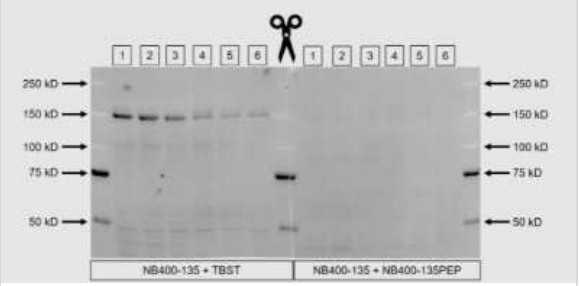
Host	Rabbit
Gene ID	51085
Gene Symbol	MLXIPL
Species	Human, Mouse, Rat
Reactivity Notes	Use in Mouse reported in scientific literature (PMID: 33812059).
Immunogen	A C-terminal synthetic peptide made to the human CHREBP protein sequence (between residues 800-852). [UniProt# Q9NP71, Isoform 1/Alpha]

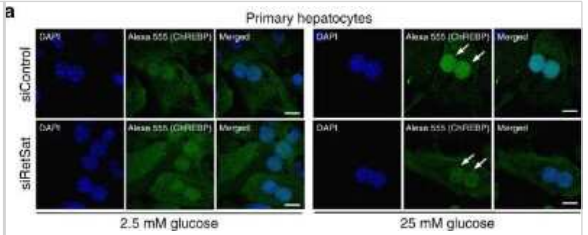
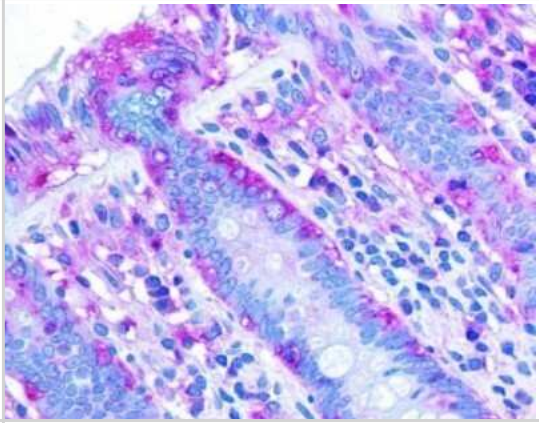
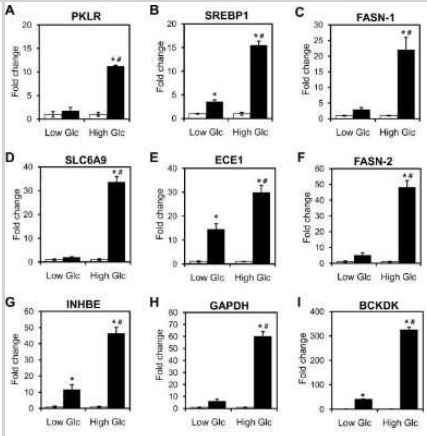
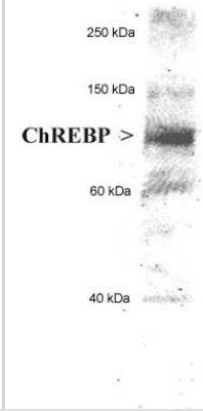
Product Application Details

Applications	Western Blot, Chromatin Immunoprecipitation, Gel Super Shift Assays, Immunoblotting, Immunocytochemistry/ Immunofluorescence, Immunohistochemistry, Immunohistochemistry-Paraffin, Immunoprecipitation, SDS-Page, Chromatin Immunoprecipitation (ChIP), Knockout Validated
Recommended Dilutions	Western Blot 1:1000, Chromatin Immunoprecipitation 1:10 - 1:500. Use reported in scientific literature (PMID 21282101), Immunohistochemistry 1:100, Immunocytochemistry/ Immunofluorescence 1:100 - 1:500, Immunoprecipitation 1:10 - 1:500, Immunohistochemistry-Paraffin 1:100, Immunoblotting reported in scientific literature (PMID 26181104), Gel Super Shift Assays reported in scientific literature (PMID 20025850), SDS-Page reported in scientific literature (PMID 35041621), Chromatin Immunoprecipitation (ChIP) 1:10-1:500, Knockout Validated reported in scientific literature (PMID 31668386)

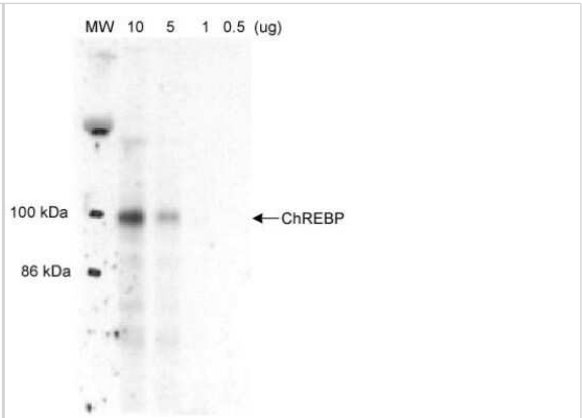
Images

Western Blot: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - Mouse liver tissue lysate, 10 ug total protein per lane, probed with CHREBP antibody. Specific bands at ~150 kDa (some wells as doublets). Weaker bands at ~45 kDa, very faint bands at ~100 kDa. These bands were absent after exposure to ChREBP blocking peptide, although some faint non-specific banding remained at ~90, ~60, and ~40 kDa. WB image submitted by a verified customer review.

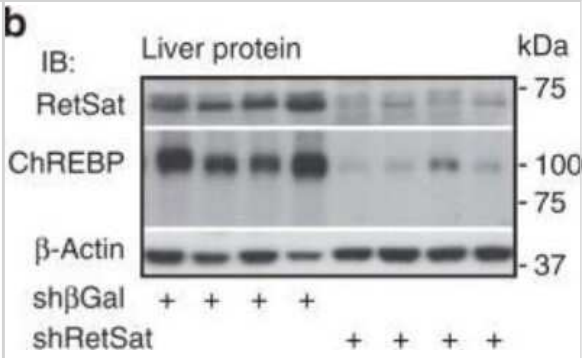


Immunocytochemistry/Immunofluorescence: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - RetSat depletion prevents the glucose-induced nuclear accumulation of ChREBP independent of 13,14-dihydroretinol generation. Hepatocytes were treated with Control or RetSat siRNA overnight. The next morning, cells were incubated with vehicle (DMSO) or 1 uM 13,14-dhretinol for 24 h at the indicated glucose concentrations and mRNA expression of Txnip determined by qPCR. Data are shown as mean +/- s.d., n = 4. Two-way ANOVA with Bonferroni post test revealed significances between low and high glucose (#P < 0.05) and between siControl and siRetSat (*P < 0.05), treatment with 13,14-dhretinol had no effect. Image collected and cropped by CiteAb from the following publication (https://www.nature.com/articles/s41467-017-00430-w), licensed under a CC-BY license.	
Immunohistochemistry-Paraffin: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - Staining of human colon, epithelium at a 40X magnification.	
Chromatin Immunoprecipitation: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - Effects of glucose on ChREBP binding.HepG2 cells were treated with low (2.7 mM) and high (25 mM) glucose for 8 h. Chromatin was isolated and fragmented, and ChIP was performed with control IgG or anti-ChREBP antibody. Validated primers for each gene were used for quantitative real-time PCR. The data presented as fold increase for the signal from anti-ChREBP relative to control IgG. The negative control, Cyclo, showed no enrichment (data not shown). Values represent the mean +/- S.D. of three independent samples. *p<0.005 vs. IgG, #p<0.0001 vs. 2.7 mM glucose with anti-ChREBP. Image collected and cropped by CiteAb from the following publication (https://dx.plos.org/10.1371/journal.pone.0022544), licensed under a CC-BY license.	
Western Blot: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - Detection of ChREBP in 20 ug of human hepatocyte lysate using NB400-135. 5-10 second film exposure.	

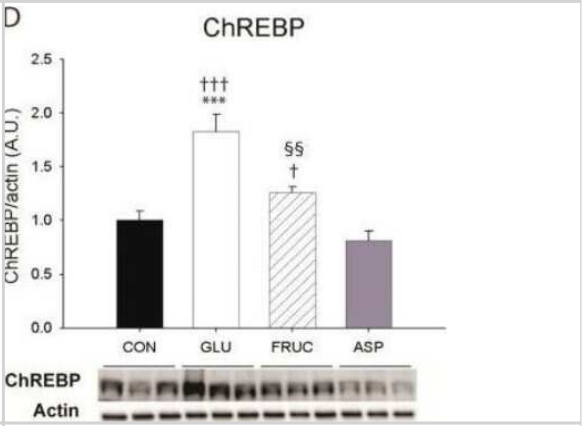
Western Blot: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - Detection of ChREBP in liver nuclear extracts from well-fed rats. 7% SDS-PAGE gel, 1:1000 dilution of NB400-135. Photo courtesy of Dr. Uyeda, UT Southwestern University.



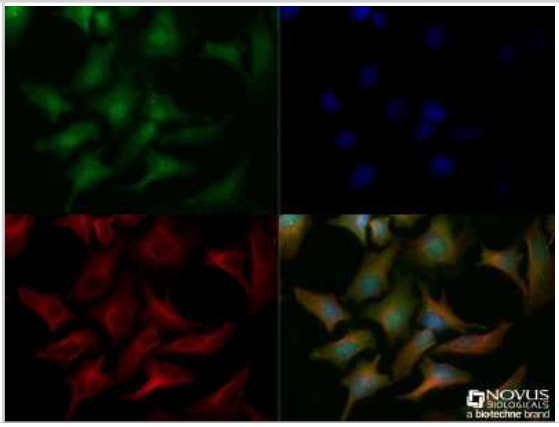
Western Blot: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - RetSat depletion in mouse liver reduces protein levels and target gene expression of ChREBP. RetSat and ChREBP protein expression by immunoblotting. Image collected and cropped by CiteAb from the following publication (https://www.nature.com/articles/s41467-017-00430-w), licensed under a CC-BY license.



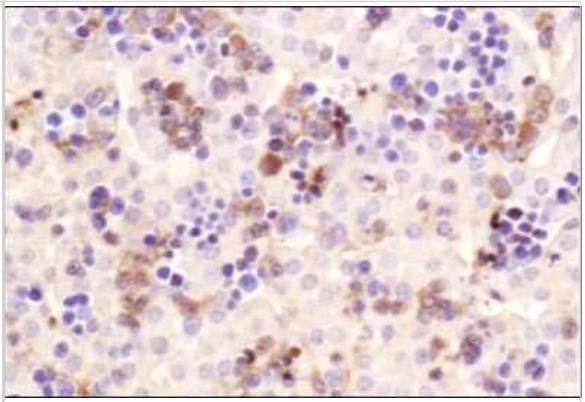
Western Blot: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - Protein expression levels of carbohydrate-responsive element-binding protein (ChREBP), in livers of rats receiving normal water (CON), a 13% (w/v) glucose solution (GLU), a 13% (w/v) fructose solution (FRUC), or a 0.4% (w/v) aspartame solution (n = 6 per diet group). All data were normalized to beta-actin expression levels and are expressed relative to the controls (CON). Data are expressed as means +/- SEM. * p < 0.05, ** p < 0.01, *** p < 0.001 vs. CON; +p < 0.05, ++p < 0.01, +++p < 0.001 vs. ASP; p < 0.01, p < 0.001 vs. GLU. Image collected and cropped by CiteAb from the following publication (https://www.mdpi.com/2072-6643/9/5/476), licensed under a CC-BY license.



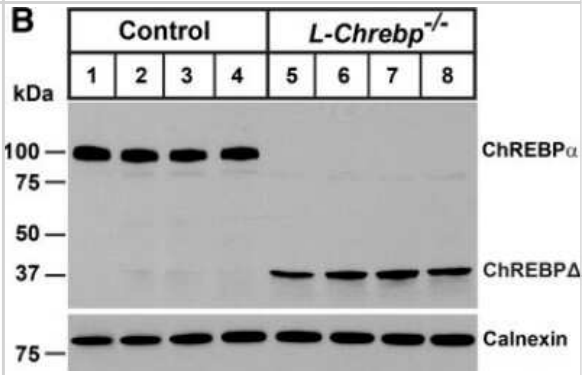
Immunocytochemistry/Immunofluorescence: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - HeLa cells were fixed for 10 minutes using 10% formalin and permeabilized for 5 minutes using 1X TBS + 0.5% Triton X-100. The cells were incubated with anti-ChREBP (NB400-135) at a 1:200 dilution overnight at 4C and detected with an anti-rabbit DyLight 488 (Green) at a 1:500 dilution. Alpha tubulin was used as a co-stained at 1:1000 and detected with an anti-mouse DyLight 550 (Red) at a 1:500. Nuclei were counterstained with DAPI (Blue). Cells were imaged using a 40X objective.



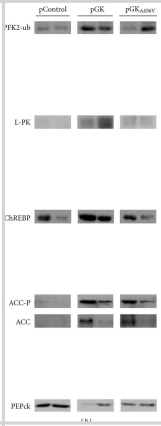
Immunohistochemistry: CHREBP Antibody - BSA Free [NB400-135] - CHREBP Antibody [NB400-135] - Analysis of CHREBP in mouse liver using DAB with hematoxylin counterstain.



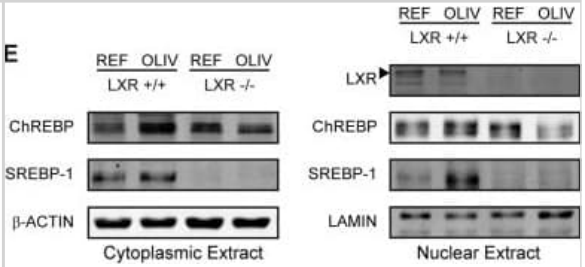
Knockout Validated: CHREBP Antibody - BSA Free [NB400-135] - Immunoblot analysis of ChREBP in liver lysates of control and L-Chrebp -/- mice. Aliquots (60 ug of protein) of liver whole-cell lysates were subjected to SDS-PAGE and immunoblot analysis with anti-ChREBP and anti-calnexin antibodies. ChREBP delta denotes a truncated aberrant ChREBP protein present only in lysates prepared from L-Chrebp -/- livers. Image collected and cropped by CiteAb from the following publication (//pubmed.ncbi.nlm.nih.gov/29335275/) licensed under a CC-BY license.



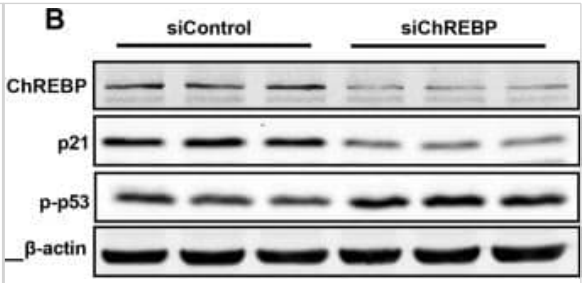
Analysis of protein levels in livers transfected with GK and GKA456V. Livers excised from mice in postabsorptive state were homogenized and resolved by Western blot. (a, b) Representative blots from three independent experiments. The densitometric analysis is presented (N = 4, *P < 0.05 versus pControl, ***P < 0.001 versus pGKA456V). (c) Liver sections of 5-hour fasted mice injected with pControl, pGK, or pGKA456V were immunostained with Glc6Pase antibody. TO-PRO-3 was used to visualize nuclei.



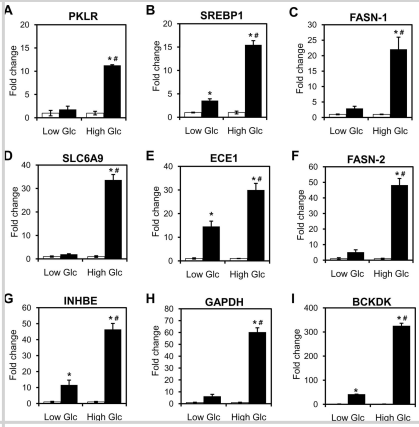
LXR mediate the induction of lipogenesis induced by an oleic acid-rich diet. (E) Cytoplasmic and nuclear expression levels of LXR, SREBP-1c and ChREBP assayed by Western Blotting. Data are the mean+/-SEM of values measured in LXR+/+ and LXR-/- mice fed REF or OLIV diet. a Significant genotype effect. b Significant difference versus REF diet (n = 6 mice per group). Image collected and cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/28732092), licensed under a CC-BY licence.



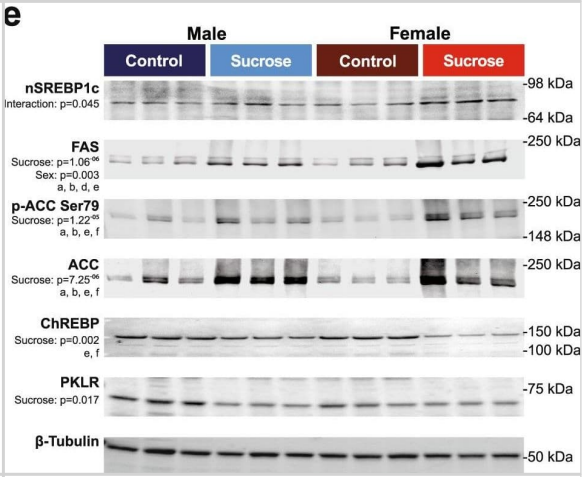
ChREBP knockdown inhibited glycolysis, lipogenesis and p21 in HT29 cells. (B) Western-blot of ChREBP, phospho-p53 and p21 and their quantification on the right. beta-actin was served as a loading control. Proteins were extracted from cells transfected with sicontrol and siChREBP after 48 hours. The quantification of western blot was normalized to beta-actin. Image collected and cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/32144313), licensed under a CC-BY licence.



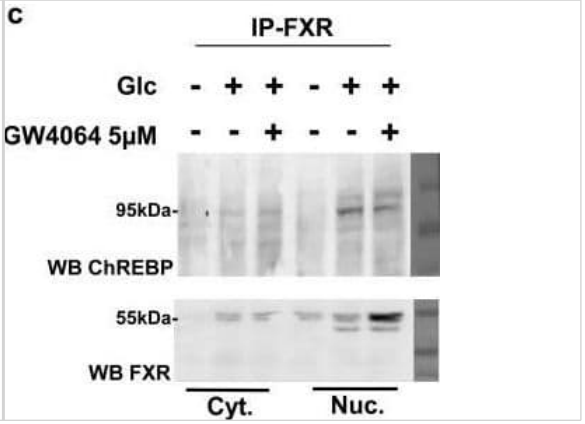
Effects of glucose on ChREBP binding.HepG2 cells were treated with low (2.7 mM) and high (25 mM) glucose for 8 h. Chromatin was isolated and fragmented, and ChIP was performed with control IgG or anti-ChREBP antibody. Validated primers for each gene were used for quantitative real-time PCR. The data presented as fold increase for the signal from anti-ChREBP relative to control IgG. The negative control, Cyclo, showed no enrichment (data not shown). Values represent the mean +/- S.D. of three independent samples. *p<0.005 vs. IgG, #p<0.0001 vs. 2.7 mM glucose with anti-ChREBP. Image collected and cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/21811631), licensed under a CC-BY licence.



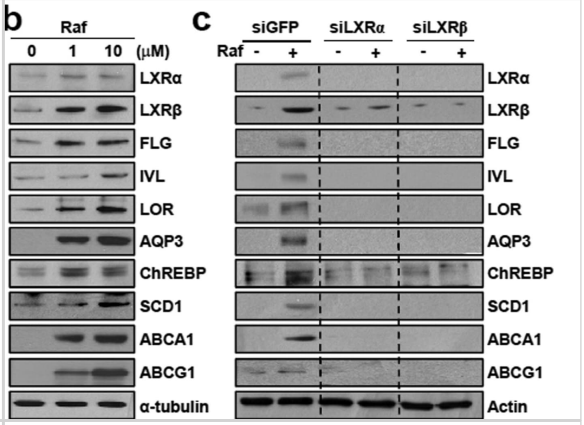
Sucrose intake increases hepatic triglyceride content via sex-specific mechanisms. e) Representative western blots demonstrating that the abundance of proteins encoded by the transcripts differentially expressed (as well as ChREBP and its target PKLR) between males and female mice in response to sucrose intake. Statistics were derived following densitometric quantification of bands from samples run on a single gel that were normalized to total protein and then log transformed and compared using two-way ANOVA with post hoc Tukey's tests (FAS: ap= 1.43-4, bp = 1.55-4, dp = 0.044, ep = 2.80-6; pACC: ap = 0.001, bp = 0.007, ep = 2.8-4, fp = 0.041; ACC: ap = 0.001, bp = 0.003, ep = 2.99-4, fp = 0.014; ChREBP: ep = 0.024, fp = 0.010; MC: n = 6, MS: n = 6, FC: n = 6, FS: n = 6). Image collected and cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/36229459), licensed under a CC-BY licence.



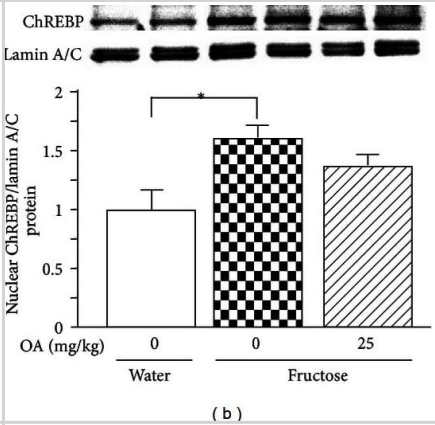
FXR inhibits glucose-induced proglucagon expression. (c) ChREBP and FXR western-blots after FXR immunoprecipitation on lysates from cytoplasm and nucleus of GLUTag cells treated or not with GW4064 (5 umol L-1) in presence or not of glucose (5.6 mmol L-1) (performed 2 times). Image collected and cropped by CiteAb from the following publication (https://www.nature.com/articles/ncomms8629), licensed under a CC-BY licence.



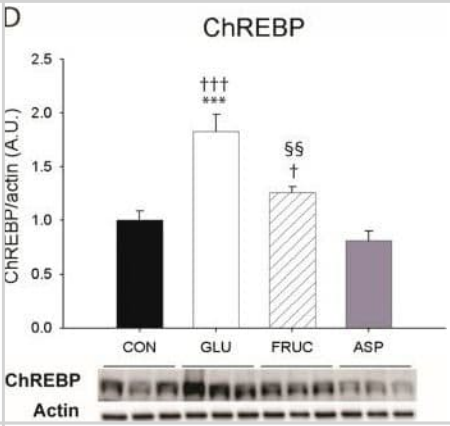
Raffinose stimulates transcription of genes involved in epidermal barrier function in HaCaT cells.(a and b) HaCaT cells were treated with vehicle, 1 uM or 10 uM raffinose (Raf), or 1 uM TO901317 (T17) for 24 h. Expressions of transcripts (a) and proteins (b) were analyzed by qRT-PCR or western blotting, respectively. FLG; filaggrin. IVL; involucrin, LOR; loricrin, AQP3; aquaporin3. (c) HaCaT cells were transfected with siGFP control, siLXRalpha, or siLXRbeta, and then treated with 1 uM raffinose for 24 h. Expression of proteins was analyzed by western blotting. The original blots are shown in Supplementary Fig. S9. Image collected and cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/28266648), licensed under a CC-BY licence.



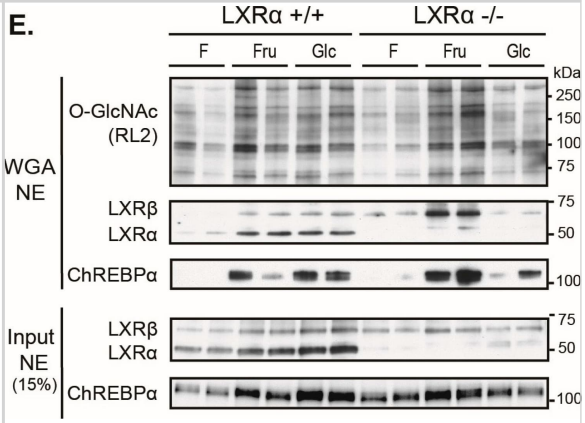
Hepatic expression of mRNAs encoding carbohydrate response element-binding protein (ChREBP) (a), liver pyruvate kinase (LPK) (c), and microsomal triglyceride transfer protein (MTTP) (d), and nuclear ChREBP protein (b) in water-control, 10% fructose solution-control, and fructose pair-fed oleanolic acid- (OA-) treated rats at week 10. Animals were administered with OA (25 mg/kg/day) or vehicle (OA: 0 mg/kg, 5% Gum Arabic) by oral gavage daily for 10 weeks. mRNA was determined by real-time PCR. Protein expression was determined by Western blot. Data are means +/- SEM (n = 6 each group). *P < 0.05. Image collected and cropped by CiteAb from the following publication (https://pubmed.ncbi.nlm.nih.gov/23737835), licensed under a CC-BY licence.



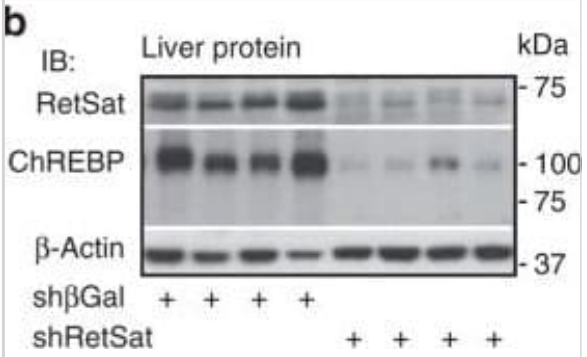
Protein expression levels of (D) carbohydrate-responsive element-binding protein (ChREBP); (E) 128 kDa precursor sterol regulatory element-binding protein-1c (SREBP-1c); and (F) 65 kDa cleaved SREBP-1c, in livers of rats receiving normal water (CON), a 13% (w/v) glucose solution (GLU), a 13% (w/v) fructose solution (FRUC), or a 0.4% (w/v) aspartame solution (n = 6 per diet group). All data were normalized to beta-actin expression levels and are expressed relative to the controls (CON). Data are expressed as means +/- SEM. * p < 0.05, ** p < 0.01, *** p < 0.001 vs. CON; +p < 0.05, ++p < 0.01, +++p < 0.001 vs. ASP; **p < 0.01, ***p < 0.001 vs. GLU. Image collected and cropped by CiteAb from the following publication (https://www.mdpi.com/2072-6643/9/5/476), licensed under a CC-BY licence.



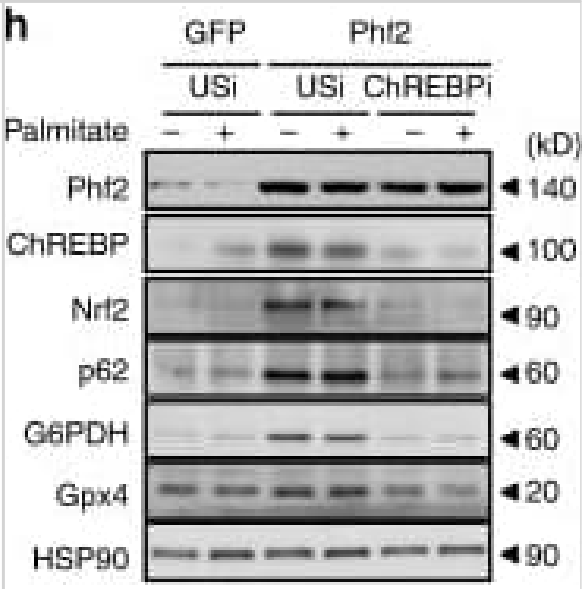
Optical redox ratio (ORR) demonstrates reduced glycolytic phenotype following 24 hours of YC-1 treatment. ORR of A549 and A549RR cells before, 12 and 24 hours following 50 μM YC-1 treatment to inhibit HIF-1 (a,b). Immunoblotting for HIF-1α (c) and PDK-1 (d) before and 24 hours following YC-1 treatment to verify in of HIF-1α. Representative immunoblots were horizontally cropped at the molecular weight indicated (e). Uncropped exposures can be seen in Supplemental Fig. 1. Experiments were performed in triplicate across 3 independent experiments. *p < 0.05 compared to A549 0 hr control, †p < 0.05 compared to A549RR 0hrs. Scale bar in images represents 50 μm. ME – Main effect of cell type and time; however, no significant interactions were found between the two main effects. Image collected and cropped by CiteAb from the following open publication (https://www.nature.com/articles/s41598-018-27262-y), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



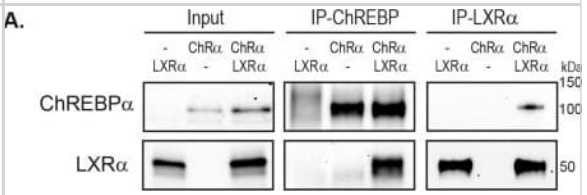
Excessive AP-endonuclease activity in cells with p53 impairment leads to accumulation of genomic instability. (A) Representative western blotting analysis on WI38 (SV40) cells shows rescue of γH2AX staining upon co-depletion of XRCC1 and APE1. (B) Neutral Comet assay on WI38 (SV40) fibroblasts shows accumulation of DSBs upon XRCC1-depletion and rescue after co-depletion of XRCC1 and APE1. Results are expressed as mean ± SD of three independent experiments. (C) Representative western blotting analysis on WI38 (SV40) cells shows rescue of γH2AX staining after depletion of XRCC1 in presence of APE1 inhibitor III (APE1i III, 5 μM for 24 h), or AR03 (2.5 μM for 24 h). Actin was used as loading control in all western blotting experiments. Image collected and cropped by CiteAb from the following open publication (https://academic.oup.com/nar/article-lookup/doi/10.1093/nar/gkw015), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



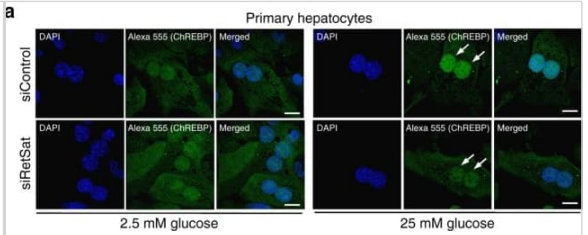
Effects of resveratrol on the expression of SIRT1, PGC-1α, HIF-1α and apoptosis proteins in vitro. (A) The expression of SIRT1, PGC-1α, and Bcl-2 proteins were lower in iohexol treated HK-2 cells compared with control cells, which was reversed by treatment with resveratrol. The expression of HIF-1α, Bax, cleaved caspase-3, and Cyt-C were markedly increased after iohexol injection, which was reversed by treatment with resveratrol. Furthermore, this effect was enhanced by HIF-1α inhibitor, 2-MeOE2. (B–H) Relative densitometry of SIRT1–PGC-1α–HIF-1α signaling proteins expressed as mean ± standard error. *P < 0.05 vs. Cont; #P < 0.05 vs. PC–AKI. Cont, control; Res, resveratrol; PC–AKI, post-contrast acute kidney injury; SIRT1, silent information regulator l; HIF-1α, hypoxia-inducible transcription factor-1α; Cyt-C, cytochrome C; PGC-1α, peroxisome proliferator-activated receptor gamma coactivator-1 alpha; Ex527, SIRT1 specific inhibitor; LG, low glucose. Groups: Res, HG+res+iohexol; Ex527, HG+Ex527+iohexol; PC–AKI, HG+iohexol; 2-MeOE2, HG+res+2-MeOE2+iohexol; Cont, LG. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/31402864), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



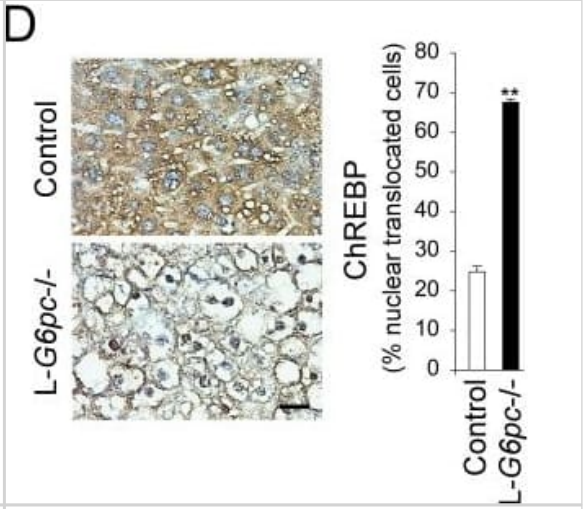
EpEX and EpCAM activate a STAT3-HIF2α signal for EpEX/EpCAM-mediated iPSC formation.(A) iPSCs were infected with two different EpCAM shRNAs (two clones, #1 and #2). The protein expressions of EpCAM and HIF2α were detected by Western blotting. (B) MEFs were stimulated by EpEX (1 μg/mL) at the indicated times. Nuclear-translocation was detected with a specific antibody against HIF2α (n = 3). (C) Immunofluorescence staining was performed to detect subcellular localization of HIF2α. Nuclei were stained with DAPI. Scale bar: 10 μm. (D) MEFs were treated with STAT3 inhibitor (WP1066, 10 μM), and then stimulated with EpEX for 30 min. The nuclear-translocation of HIF2α was detected by Western blotting with anti-HIF2α antibody (n = 3). (E) iPSC morphology was observed at day 20 after induction. Reprogramming of Oct4-GFP MEFs was induced by transfection of OSKM, OE + EpEX, and KE + EpEX with or without STAT3 inhibitor WP1066, or HIF2α shRNA (n = 3). Scale bar: 50 μm. Image collected and cropped by CiteAb from the following open publication (https://www.nature.com/articles/srep41852), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



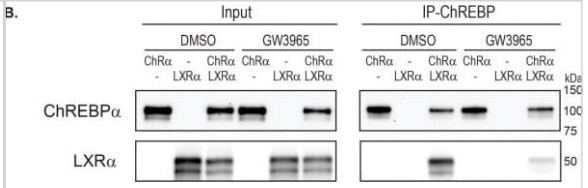
Cell cycle block under PHD3 depletion is accompanied by p27 induction. a PHD3 depletion induces a cell cycle block in G0/G1. HeLa and renal cell adenocarcinoma cells (786-O) were transfected with control (siScr) or PHD3 targeted (siPHD3) siRNA followed by synchronization at G0 and 24-h hypoxic exposure. Cell cycle progression was monitored by FACS analysis 8 h after cell cycle release. The combined means of three independent experiments are presented (±SEM) shown in the tables below. b PHD3 depletion induces p27 expression in HeLa cells and in 786-O cells under hypoxia (1 % O2) and normoxia (21 % O2) by siPHD3 and independent adenoviral shRNA against PHD3. p21 or p16 expression is not elevated by PHD3 knockdown. c Depletion of either PHD1 or PHD2 by siRNA does not increase p27 expression in 786-O cells Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/26223520), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



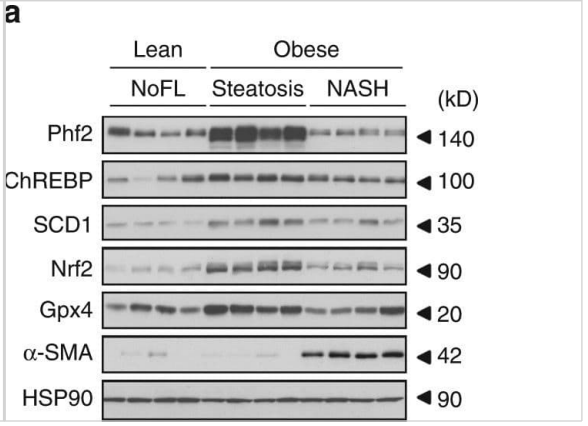
GAD65/67-positive neurons expressed HIF-1α under hypoxic conditions. A) HIF-1α expression co-localized with GAD65/67-ir in neurons exposed to hypoxia when compared to normoxia (upper panel) or GAD65/67-negative neurons in hypoxia (bottom panel, open arrow). B) Quantification shows the percentage of HIF-1α-expressing GAD65/67-positive neurons after hypoxia in vitro (mean ± SD; from n = 6 cultures). C)In vivo immunostaining illustrates HIF-1α-positive (bottom panel, solid arrow) and HIF-1α-negative (bottom panel, open arrow) in GAD65/67-ir neurons in the ipsilateral region, whereas the contralateral region shows no HIF-1α staining in GAD65/67-ir neurons (see Figure 1 for region selection). Scale bars, 10 µm (A); 20 µm (B). Image collected and cropped by CiteAb from the following open publication (https://actaneurocomms.biomedcentral.com/articles/10.1186/2051-5960-2-51), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Absence of Zizimin proteins, but normal numbers of hematopoietic cells in KO mice. (A-B) Western blotting for Ziz2 (A) or Ziz3 (B) Protein lysates (30 µg/lane) were loaded and designated antibodies were reacted with the membranes. Replicated membranes were used for all antibodies. Zizimin proteins were absent in KO mice (8 weeks old, male). (C-F) The numbers of hematopoietic cells were counted after the hemolytic reaction (C-E) or flow cytometric analysis (F). No significant difference was observed among the groups (C-F). Three mice (8 weeks old, female) per group from three independent experiments (one mouse per group per experiment) were used. 2KO: Ziz2 KO. 3KO: Ziz3 KO. Image collected and cropped by CiteAb from the following open publication (https://immunityageing.biomedcentral.com/articles/10.1186/s12979-015-0028-x), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Immunocytochemical analysis of CNHCs with antibodies against the RCC marker CAIX. Clusters of CNHCs cytomorphologically classified as uncertain malignant (−UMF) with cytoplasmic positive staining with antibodies against the RCC marker CAIX (A). Clusters of CNHC-UMF and -BF without reactivity for CAIX antibodies (B and C, respectively). A single CNHC-MF with positive cytoplasmic (D) and without staining for CAIX (E). Single CAIX-negative CNHC-UMF and -BF (F and G, respectively). Image collected and cropped by CiteAb from the following open publication (https://translational-medicine.biomedcentral.com/articles/10.1186/1479-5876-11-214), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Publications

Joan Sanchez-Gurmaches, Camila Martinez Calejman, Su Myung Jung, Huawei Li, David A. Guertin Brown fat organogenesis and maintenance requires AKT1 and AKT2 Molecular Metabolism 2019-02-20 [PMID: 30833219]

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Li L, Sakiyama H, Eguchi H et al. Activation of the mitogen-activated protein kinase ERK1/2 signaling pathway suppresses the expression of ChREBP? and ? in HepG2 cells FEBS Open Bio 2021-07-01 [PMID: 34051057] (WB)

Zhao S, Jang C, Liu J et al. Dietary fructose feeds hepatic lipogenesis via microbiota-derived acetate Nature 2020-03-26 [PMID: 32214246] (B/N)

Li, L;Long, J;Mise, K;Poungavrin, N;Lorenzi, PL;Mahmud, I;Tan, L;Saha, PK;Kanwar, YS;Chang, BH;Danesh, FR; The Transcription Factor ChREBP Links Mitochondrial Lipidomes to Mitochondrial Morphology and Progression of Diabetic Kidney Disease The Journal of biological chemistry 2023-08-21 [PMID: 37611830]

More publications at http://www.novusbio.com/NB400-135

Procedures

Western Blot Protocol for CHREBP Antibody (NB400-135)
Western Blot Protocol

1. Perform SDS-PAGE on samples to be analyzed, loading 10-25 ug of total protein per lane.
2. Transfer proteins to PVDF membrane according to the instructions provided by the manufacturer of the membrane and transfer apparatus.
3. Stain the membrane with Ponceau S (or similar product) to assess transfer success, and mark molecular weight standards where appropriate.
4. Rinse the blot TBS -0.05% Tween 20 (TBST).
5. Block the membrane in 5% Non-fat milk in TBST (blocking buffer) for at least 1 hour.
6. Wash the membrane in TBST three times for 10 minutes each.
7. Dilute primary antibody in blocking buffer and incubate overnight at 4C with gentle rocking.
8. Wash the membrane in TBST three times for 10 minutes each.
9. Incubate the membrane in diluted HRP conjugated secondary antibody in blocking buffer (as per manufacturer's instructions) for 1 hour at room temperature.
10. Wash the blot in TBST three times for 10 minutes each (this step can be repeated as required to reduce background).
11. Apply the detection reagent of choice in accordance with the manufacturers instructions.

Immunocytochemistry/ Immunofluorescence Protocol for CHREBP Antibody (NB400-135)
Immunocytochemistry Protocol

Culture cells to appropriate density in 35 mm culture dishes or 6-well plates.

1. Remove culture medium and wash the cells briefly in PBS. Add 10% formalin to the dish and fix at room temperature for 10 minutes.
2. Remove the formalin and wash the cells in PBS.
3. Permeablize the cells with 0.1% Triton X100 or other suitable detergent for 10 min.
4. Remove the permeablization buffer and wash three times for 10 minutes each in PBS. Be sure to not let the specimen dry out.
5. To block nonspecific antibody binding, incubate in 10% normal goat serum from 1 hour to overnight at room temperature.
6. Add primary antibody at appropriate dilution and incubate overnight at 4C.
7. Remove primary antibody and replace with PBS. Wash three times for 10 minutes each.
8. Add secondary antibody at appropriate dilution. Incubate for 1 hour at room temperature.
9. Remove secondary antibody and replace with PBS. Wash three times for 10 minutes each.
10. Counter stain DNA with DAPi if required.

Immunohistochemistry Whole-Mount Protocol for CHREBP Antibody (NB400-135)
Immunohistochemistry-Paraffin Embedded Sections

Antigen Unmasking:
Bring slides to a boil in 10 mM sodium citrate buffer (pH 6.0) then maintain at a sub-boiling temperature for 10 minutes. Cool slides on bench-top for 30 minutes (keep slides in the sodium citrate buffer all the time).

Staining:
1. Wash sections in deionized water three times for 5 minutes each.
2. Wash sections in PBS for 5 minutes.
3. Block each section with 100-400 ul blocking solution (1% BSA in PBS) for 1 hour at room temperature.
4. Remove blocking solution and add 100-400 ul diluted primary antibody. Incubate overnight at 4 C.
5. Remove antibody solution and wash sections in wash buffer three times for 5 minutes each.
6. Add 100-400 ul HRP polymer conjugated secondary antibody. Incubate 30 minutes at room temperature.
7. Wash sections three times in wash buffer for 5 minutes each.
8. Add 100-400 ul DAB substrate to each section and monitor staining closely.
9. As soon as the sections develop, immerse slides in deionized water.
10. Counterstain sections in hematoxylin.
11. Wash sections in deionized water two times for 5 minutes each.
12. Dehydrate sections.
13. Mount coverslips.

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NB400-135PEP	CHREBP Antibody Blocking Peptide
HAF008	Goat anti-Rabbit IgG Secondary Antibody [HRP]
NB7160	Goat anti-Rabbit IgG (H+L) Secondary Antibody [HRP]
NBP2-24891	Rabbit IgG Isotype Control

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