HO-1 polyclonal antibody

Heme Oxygenase-1 (HO-1) also known as Hsp32, is the inducible isoform of heme oxygenase that catalyzes the NADPH, oxygen, and cytochrome P450 reductase dependent oxidation of heme to carbon monoxide, ferrous iron and biliverdin which is rapidly reduced to bilirubin. These products of the HO reaction have important physiological effects: carbon monoxide is a potent vasodilator and has been implicated to be a physiological regulator of cGMP and vascular tone; biliverdin and its product bilirubin are potent antioxidants; "free" iron increases oxidative stress and regulates the expression of many mRNAs (e.g., DCT-1, ferritin and transferring receptor) by affecting the conformation of iron regulatory protein (IRP)-1 and its binding to iron regulatory elements (IREs) in the 5'- or 3'- UTRs of the mRNAs. To date, three identified heme oxygenase isoforms are part of the HO system that catalyze heme into biliverdin and carbon monoxide. These are inducible HO-1 or Hsp32, constitutive HO-2 that is abundant in the brain and testis, and HO-3 which is related to HO-2 but is the product of a different gene. The HO system is the rate-limiting step in heme degradation and HO activity decreases the levels of heme which is a well known potent catalyst of lipid peroxidation and oxygen radical formation.

This antibody is covered by our Worry-Free Guarantee.

Citations: 133

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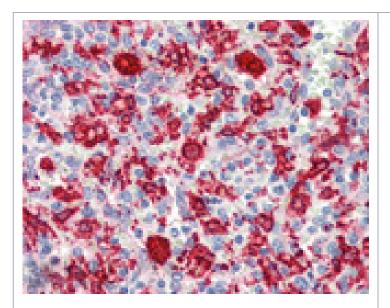
Ordering Information

Order Online »

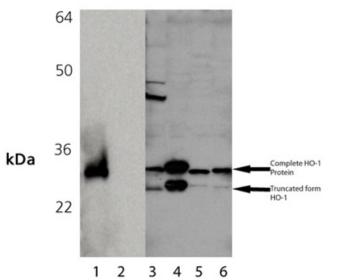
ADI-SPA-896-J	1mg
ADI-SPA-896-D	50µg
ADI-SPA-896-F	200µg

Manuals, SDS & CofA

View Online »



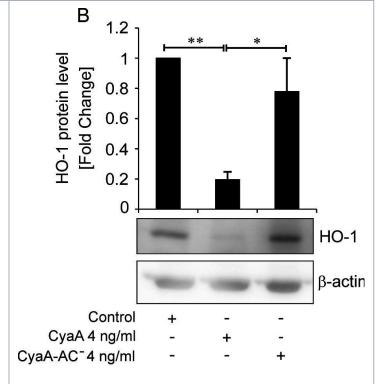
Immunohistochemistry analysis of human spleen tissue stained with HO-1, pAb at 10µg/ml.



Western blot analysis of HO-1 (HSP32) pAb: Lane 1: HO-1 (HSP32) Recombinant Human Protein, Lane 2:HO-2 Recombinant Human Protein (negative control), Lane 3: Human Liver Microsome Extract, Lane 4: Dog Liver Microsome Extract, Lane 5: Rat Liver Microsome Extract, Lane 6: Mouse Liver Microsome Extract.

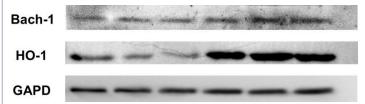
FLVCR1 mutations impair heme export in patientderived cells.(A) gRT-PCR analysis of FLVCR1a mRNA in patient 1 compared to control fibroblasts (black). Values represent mean \pm SEM. n = 6. * = P<0.05. (B) gRT-PCR analysis of FLVCR1a mRNA in patient 2 compared to control LCLs (grey). Values represent mean FLVCR1a mRNA levels in patient 2 compared to the mean FLVCR1a mRNA levels of 4 different control LCLs. (C) Immunoprecipitation and western blotting of FLVCR1a in patient 1 compared to control fibroblasts. A representative blot is shown. The antibody against SUMO was used as control. (D) Immunoprecipitation and western blotting of FLVCR1a in patient 2 compared to control LCLs. A representative blot is shown. The antibody against SUMO was used as control. (E) gRT-PCR analysis of ALAS1, HO1, FT-L, FT-H and FPN1 mRNA in patient compared to control fibroblasts. Values represent mean ± SEM. n = 6. * = P<0.05; *** = P<0.001. (F) qRT-PCR analysis of ALAS1, HO1, FT-L, FT-H and FPN1 mRNA in patient compared to control LCLs. Values represent mean mRNA levels in patient 2 compared to the mean mRNA levels of 4 different control LCLs. (G) Western blot analysis of HO1 and ALAS1 protein in patient 1 compared to control fibroblasts. A representative blot is shown. (H) Western blot analysis of ALAS1 and HO1 protein levels in patient 2 compared to 4 different control LCLs. A representative blot is shown. (I) Measurement of heme content in patient 1 compared to control fibroblasts. Values represent mean ± SEM. n = 6. Two-way ANOVA. *** = P<0.001. (J) Measurement of heme content in patient 1 compared to control LCLs. Values represent heme content of patient 2 LCLs compared to the mean heme content of 4 different control LCLs. P1 = patient 1, P2 = patient 2, C = control.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Mutations in the Heme Exporter FLVCR1 Cause Sensory Neurodegeneration with Loss of Pain Perception. *PLoS Genet* (2016)



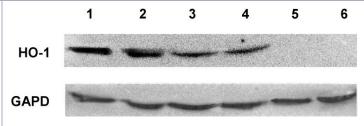
CyaA downregulates HO-1 expression and HO-1 induction does not relieve the inhibition of monocyte differentiation imposed by CyaA. (A) HO-1 transcript levels in CD14+ monocytes cultured for 5 days with 4 ng/mL of CyaA. HO-1 expression was normalized to β2microglobulin and β-actin transcript level and the mean ± SD of values from three independent determinations on cells of different donors are shown. (B) HO-1 protein levels are determined in cell lysates by densitometry of immunoblot signals, using β-actin detection for normalization of the signal. (C) Immunoblot detection of HO-1 induction by CoPP, using β-actin detection as a loading control. (D) HO-1 induction by 0.5 µM CoPP does not relieve the CyaA-imposed block of monocyte differentiation, CD14+ monocytes were cultured with 4 ng/mL of CyaA for 5 days in the presence or absence of 0.5 µM CoPP, and cell surface expression of the macrophage markers CD11b, CD204, and CD206 was analyzed by flow cytometry. (E) The alternative heme importer protein SLCO2B1 levels were probed by immunoblot of cell lysates using specific antibody and βactin levels serving as loading control. ***P < 0.0005, **P < 0.005, *P < 0.05; NS, not significant.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: cAMP signaling of Bordetella adenylate cyclase toxin blocks M-CSF triggered upregulation of iron acquisition receptors on differentiating CD14+ monocytes. *mSphere* (2024)



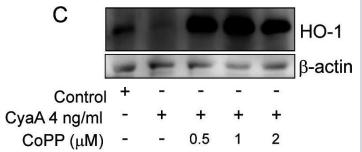
Positive correlation between HO-1 and Bach-1 protein level (Western Blot analysis).

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Hepatic HMOX1 expression positively correlates with Bach-1 and miR-122 in patients with HCV mono and HIV/HCV coinfection. *PLoS One* (2014)



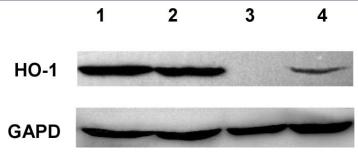
Presence of HO-1 protein levels for representative patients (Western Blot analysis).

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Hepatic HMOX1 expression positively correlates with Bach-1 and miR-122 in patients with HCV mono and HIV/HCV coinfection. *PLoS One* (2014)



CyaA downregulates HO-1 expression and HO-1 induction does not relieve the inhibition of monocyte differentiation imposed by CyaA. (A) HO-1 transcript levels in CD14+ monocytes cultured for 5 days with 4 ng/mL of CyaA. HO-1 expression was normalized to β2microglobulin and β -actin transcript level and the mean ± SD of values from three independent determinations on cells of different donors are shown. (B) HO-1 protein levels are determined in cell lysates by densitometry of immunoblot signals, using β-actin detection for normalization of the signal. (C) Immunoblot detection of HO-1 induction by CoPP, using β-actin detection as a loading control. (D) HO-1 induction by 0.5 µM CoPP does not relieve the CyaA-imposed block of monocyte differentiation, CD14+ monocytes were cultured with 4 ng/mL of CyaA for 5 days in the presence or absence of 0.5 µM CoPP, and cell surface expression of the macrophage markers CD11b, CD204, and CD206 was analyzed by flow cytometry. (E) The alternative heme importer protein SLCO2B1 levels were probed by immunoblot of cell lysates using specific antibody and βactin levels serving as loading control. ***P < 0.0005, **P < 0.005, *P < 0.05; NS, not significant.

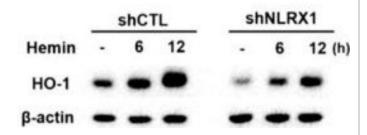
Image collected and cropped by CiteAb under a CC-BY license from the following publication: cAMP signaling of Bordetella adenylate cyclase toxin blocks M-CSF triggered upregulation of iron acquisition receptors on differentiating CD14+ monocytes. *mSphere* (2024)



HO-1 protein levels in selected patients with HIV/HCV co-infection (Western Blot analysis).

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Hepatic HMOX1 expression positively correlates with Bach-1 and miR-122 in patients with HCV mono and HIV/HCV coinfection. *PLoS One* (2014)





LPS-induced protective HO-1 expression is attenuated by NLRX1 silencing. shCTL and shNLRX1 SM826 cells were treated with LPS (100 ng/mL), olaparib (10 µM), hemin (20 μ M) and/or ZnPP (10 μ M) for the indicated times. (A,C) Total cell lysates were analyzed by immunoblotting to determine HO-1. (B) Quantitative real-time PCR was used to determine the gene expression of HO-1. (D,E) After drug treatment for 24 h, cell viability was determined. The data were presented as the mean ± S.E.M from 3 independent experiments. *, p < 0.05 indicates a significant effect of LPS (100 ng/mL) compared to the untreated shCTL group. #, p < 0.05 indicates the significant effects of olaparib, hemin and ZnPP on LPS-induced cell death. †, p < 0.05 indicates a significant difference in the LPS response between the shCTL and shNLRX1 groups. In (A), it is the marker indicated as "M" in lane 6.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: NLRX1 Inhibits LPS-Induced Microglial Death via Inducing p62-Dependent HO-1 Expression, Inhibiting MLKL and Activating PARP-1. *Antioxidants (Basel)* (2024)

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 HMOX1, Hsp32, Heat shock protein 32, Heme oxygenase 1

Application IHC (PS), IP, WB

Application Notes Detects a band of ~32kDa by Western blot.

Formulation Liquid. In PBS containing 50% glycerol and 0.09% sodium azide.

GenBank ID X14782

Gene/Protein Identifier 3162 (Entrez GeneID), 141250 (OMIM)

Host Rabbit

Immunogen Synthetic peptide corresponding to the sequence near the N-terminus of human HO-1.

Purity Detail Protein A 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 rabbit serum.

Species Reactivity Dog, Hamster, Human, Monkey, Mouse, Rabbit, Rat

nzolifesciences.com

UniProt ID P09601



European Sales Office Belgium, The Netherlands France Germany

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