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: 234 View Online »

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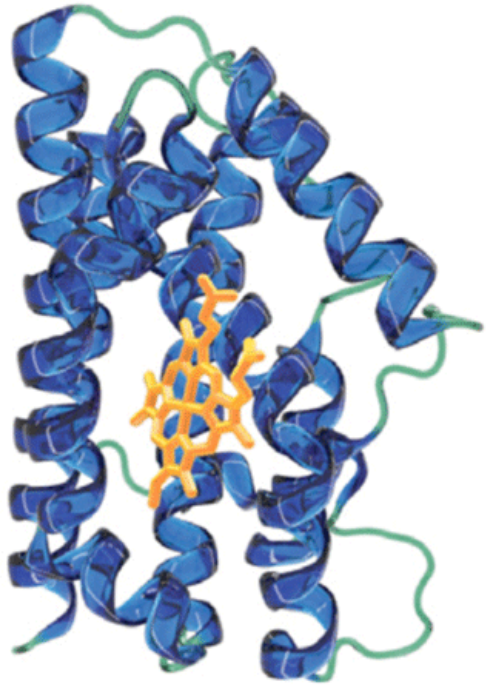
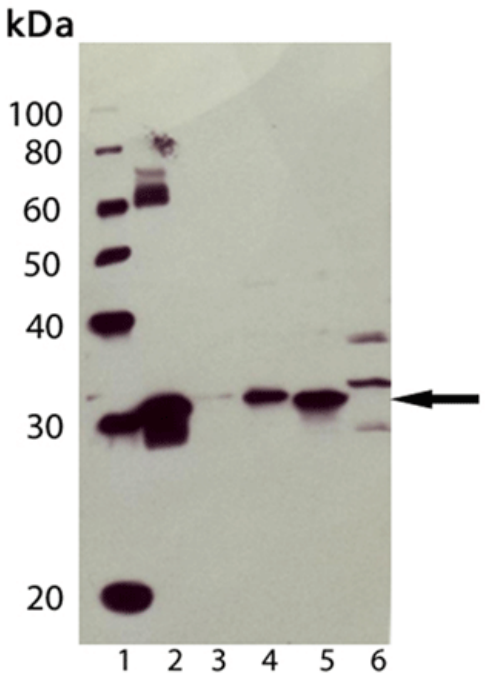
ADI-SPA-895-J	1mg
ADI-SPA-895-D	50µg
ADI-SPA-895-F	200µg

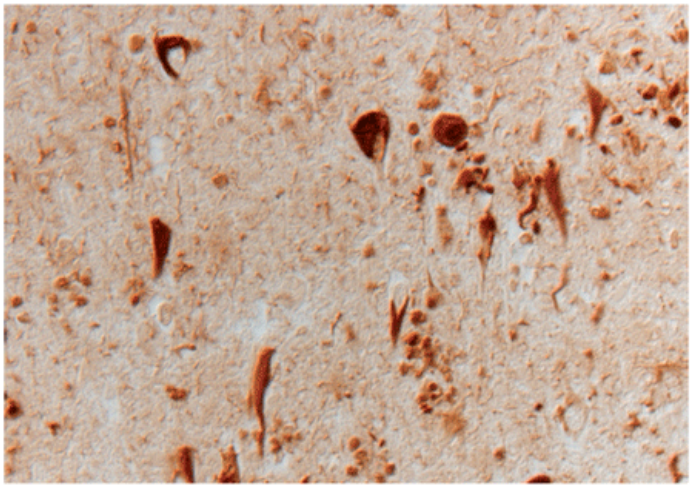
Manuals, SDS & CofA View Online »

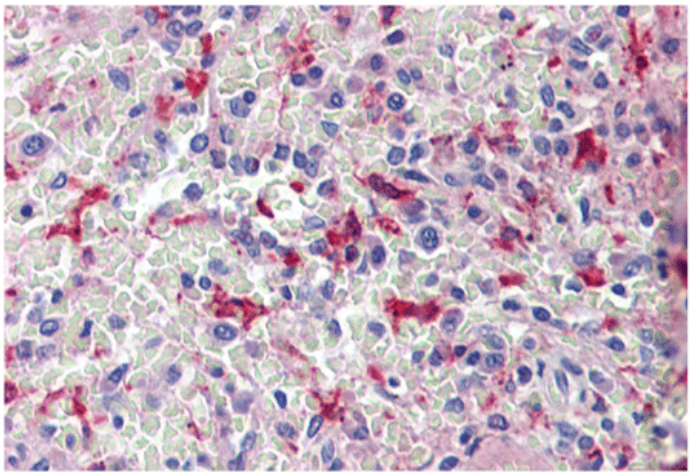


Crystal structure of human HO-1 (Hsp32) in complex with its substrate heme.

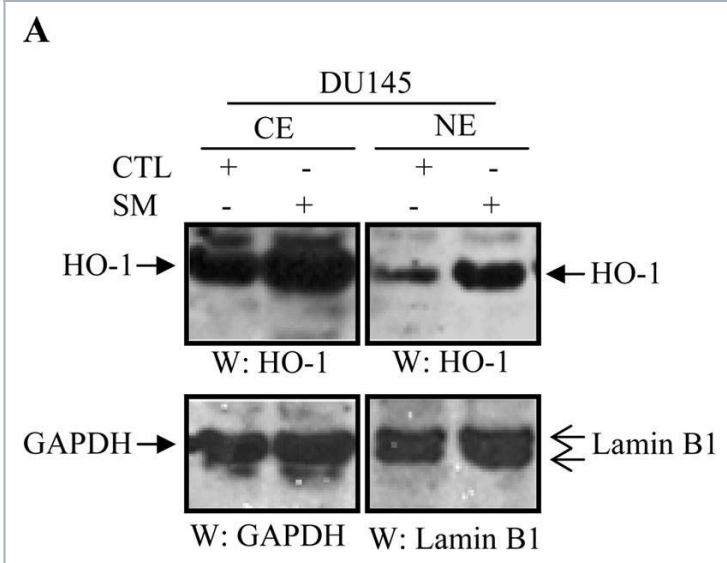


Western blot analysis of HO-1 pAb: Lane 1: MW Marker, Lane 2: HO-1 (rat), Recombinant Protein, Lane 3: Human Liver Microsomes, Lane 4: Mouse Liver Microsomes, Lane 5: Rat Liver Microsomes, Lane 6: Canine Liver Microsomes.

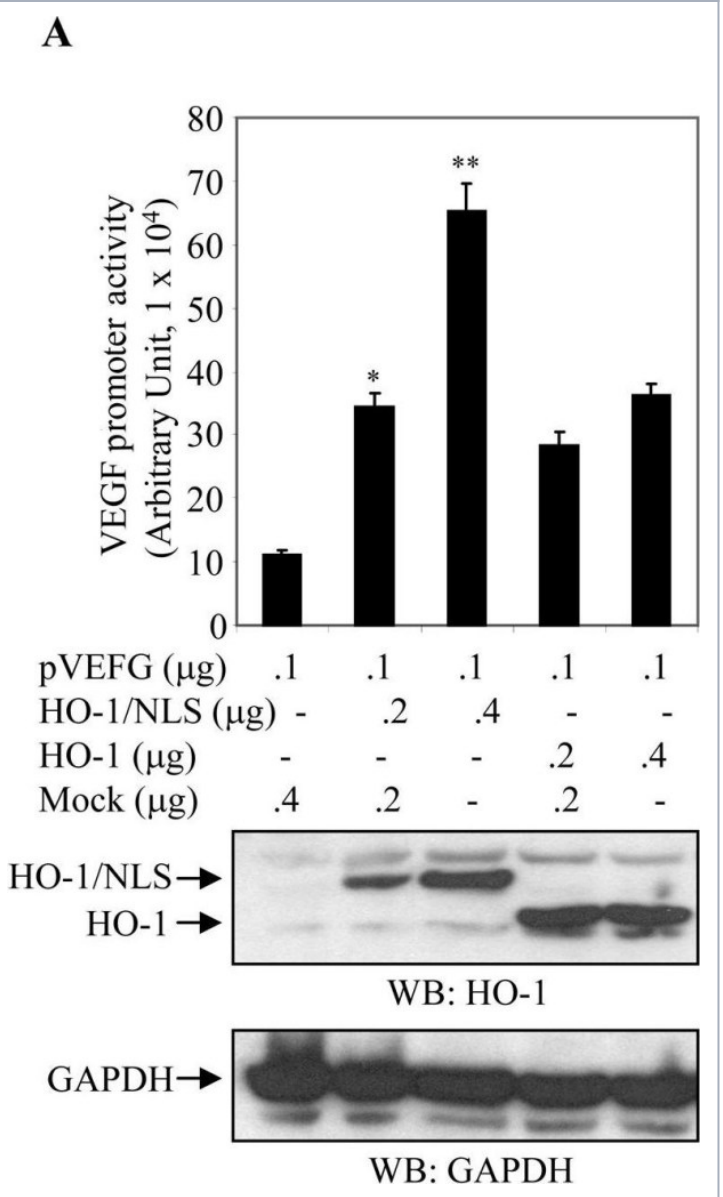




Immunohistochemistry analysis of human spleen tissue stained with HO-1, pAb at 10µg/ml.

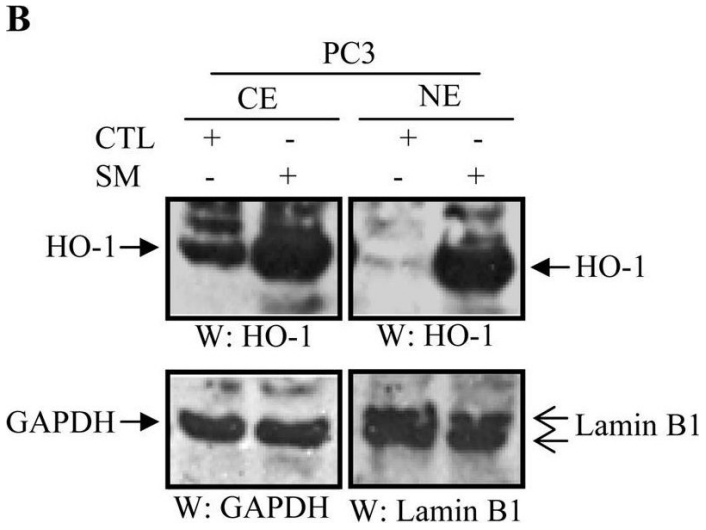


Cigarette smoke induced nuclear translocation of HO-1 in prostate cancer cells. (A) DU145 and (B) PC3 cells were grown on 6-well-plates and treated with SM. After 24 h, cellular fractionation was performed, and the cytoplasmic and nuclear fractions were analyzed by western blotting using an anti-HO-1 antibody. The blots were re-probed with anti-GAPDH or anti-Lamin B1 antibodies. (C) DU145 and (D) PC3 cells were treated with SM. Nuclear extracts were probed with anti-HO1 and Lamin B1 antibodies. Nuclear expression of HO-1 was normalized to that of the nuclear marker Lamin B1, and relative expression of nuclear HO-1 was expressed in arbitrary units. Data shown in micrographs were derived from three individual experiments. Columns, mean; bars, SD; **p<0.01. CTL, control; CE, cytoplasmic extract; NE, nuclear extract.



Nuclear localization of HO-1 promoted transcriptional activity of VEGF. (A) Differential activation of VEGF transcriptional activity by cytoplasmic and nuclear HO-1. HEK293 cells were co-transfected with the VEGF promoter and HO-1 or HO-1/NLS in a dose-dependent fashion, as indicated. After 24 h, VEGF promoter activity (luciferase activity) was measured and normalized to β-Gal activity. Relative VEGF promoter activity derived from three experiments was expressed in arbitrary units. Cell extracts were also blotted with anti-HO-1 and anti-GAPDH antibodies. Columns, mean; bars, SD; *p<0.05; **p<0.01. pVEGF, VEGF promoter; HO-1, plasmid expressing HO-1 in cytosol; HO-1/NLS, plasmid expressing nuclear HO-1; NLS, nuclear localization signal. (B) Differential activation of VEGF transcriptional activity by heat shock proteins. HEK293 and COS7 cells were co-transfected with the VEGF promoter and pNuc-HO-1/NLS or EGFP-HSP72 in a dose-dependent manner as indicated. After 24 h, luciferase activity was measured and normalized to β-galactosidase (β-Gal) activity to quantify VEGF promoter activity. Relative VEGF promoter activity was expressed in arbitrary units. Micrograph is representative of three independent experiments. Cell extracts were also blotted with anti-

SIRT6 is required for NRF2-depedent HO-1 expression in hMSCs. (A) Volcano plot showing significantly altered genes (q-value < 0.05, FC[SIRT6-deficient/WT] 2) between WT and SIRT6-deficient hMSCs. Representative NRF2 target genes were highlighted (indicated by arrows). FC, fold change. (B) Gene ontology (GO) analysis (biological process) of significantly downregulated genes in hMSCs upon SIRT6 depletion. (C) Venn diagram showing that early passage (EP, passage 6) and late passage (LP, passage 9) hMSCs shared 183 significantly downregulated genes in SIRT6-deficient hMSCs compared with WT hMSCs. NRF2 target genes shared in EP and LP were indicated. (D) RT-qPCR analysis of NRF2 target genes in WT and SIRT6-deficient hMSCs. Values were normalized against 18S rRNA. Data were presented as mean ± SEM, n = 3, *P < 0.05, **P < 0.01, ***P < 0.001. (E) Average profile of the H3K4me3 histone modification around the gene body regions of NRF2 target genes in SIRT6-deficient and WT hMSCs. TSS, transcription start site; TTS, transcription termination site. (F, G) RT-qPCR (F) and western blotting (G) analyses of HO-1 expression in WT and SIRT6-deficient hMSCs treated with 25 μM PX-12 for the indicated times. Relative mRNA and protein expressions were presented as fold induction. For RT-qPCR (F), values were normalized against 18S rRNA. Data were presented as mean ± SEM, n = 3, *P < 0.05, **P < 0.01. (H) Overexpression of SIRT6 (WT), not SIRT6 (HY), in SIRT6-deficient hMSCs partially restored HO-1 transcript. Values were normalized against 18S rRNA. Data were presented as mean ± SEM, n = 3, **P < 0.01, ***P < 0.001.



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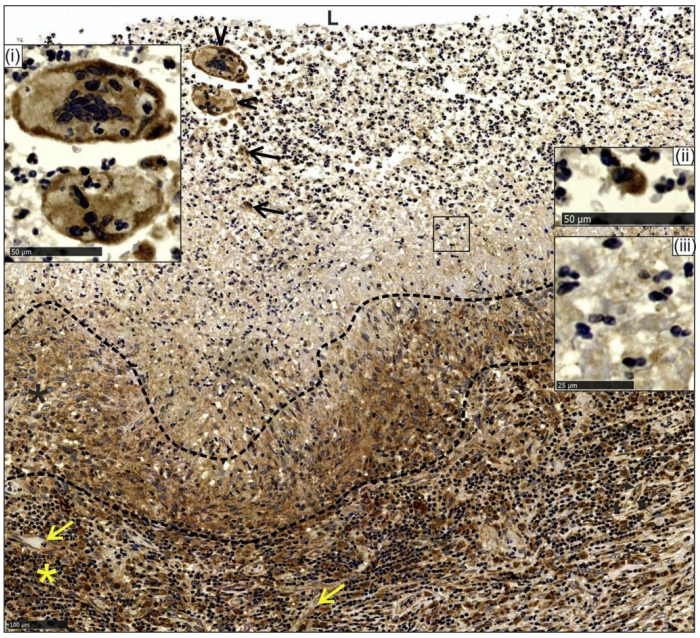

HO-1 Staining Profile in the Cavity WallHO-1 staining of cellular component in adluminal cells with bright staining of phagocytic giant cells (arrowheads, inset i), bright staining of neutrophils (arrows, inset ii), and negative karyorrhectic neutrophil staining (rectangle, inset iii) (L = Lumen). Shown is bright staining of histiocytes in the granulomatous layer (black asterisks) and bright staining of inflammatory cells (lymphocytes, plasma cells, and histiocytes) and endothelial cells (yellow arrows) in the granulation tissue layer (asterisk).

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	Flow Cytometry, ICC, IHC (PS), 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	J02722
Host	Rabbit
Immunogen	Recombinant rat HO-1 (Hsp32) lacking the membrane spanning region.
Purity Detail	Protein A affinity purified.
Recommendation Dilutions/Conditions	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, Human, Mouse, Rabbit, Rat, Sheep
Technical Info / Product Notes	**Cited samples:** *For an overview on cited samples please click here.*
UniProt ID	P06762

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