SUPPLEMENTARY DATA

Myeloperoxidase modulates hydrogen peroxide mediated cellular

damage in murine macrophages.

Chaorui Guo, Inga Sileikaite, Michael J. Davies and Clare L. Hawkins*

Department of Biomedical Sciences, University of Copenhagen, Panum, Blegdamsvej 3B,

Copenhagen N, DK-2200, Denmark

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* To whom correspondence should be addressed: Prof. Clare Hawkins, Department of Biomedical Sciences, University of Copenhagen, Panum, Blegdamsvej 3B, Copenhagen N, DK-2200, Denmark. Email: clare.hawkins@sund.ku.dk

Table S1. Mus primer sequences used for qPCR.

Gene	Forward sequence	Reverse sequence
	(5'-3')	(5'-3')
TATA-box binding protein		
(ТВР)		
18S ribosomal RNA (18S)	GTAACCCGTTGAACCCCATT	CCATCCAATCGGTAGTAGCG
Mus beta-2 microglobulin	TGGTCTTTCTGGTGCTTGTC	GGATTTCAATGTGAGGCGGGT
(B2M)		
Heme oxygenase 1	ACAGCCCCACCAAGTTCAAA	TCTGCAGGGGCAGTATCTTG
(HMOX1)		
Superoxide dismutase 2,	ACAACTCAGGTCGCTCCTCAG	GATAGCCTCCAGCAACTCTCC
mitochondrial (SOD2)		
Glutathione Peroxidase 1	CCACCGTGTATGCCTTCTCC	AGAGAGACGCGACATTCTCAAT
(GPx1)		
Glutathione S-	ATGCCACCATACACCATTGTC	GGGAGCTGCCCATACAGAC
Transferase Pi 1 (GSTP1)		
Glutamate-Cysteine		
Ligase Catalytic Subunit	GGACAAACCCCAACCATCC	GTTGAACTCAGACATCGTTCCT
(GCLc)		
Glutamate-Cysteine		
Ligase Modifier Subunit	CTTCGCCTCCGATTGAAGATG	AAAGGCAGTCAAATCTGGTGG
(GCLm)		
NAD(P)H Quinone	AGGATGGGAGGTACTCGAATC	TGCTAGAGATGACTCGGAAGG
Dehydrogenase 1 (NQO1)		
Glutathione synthetase	GGTATCTTCCCTCAGCAGCCTT	GCTTCCATTCCCACACTCCAAA
(GS)		





J774A.1 cells (1×10^5) were treated with GO (0-200 mU mL⁻¹) in HBSS with or without glucose (5.6 mM), in the absence and presence of MPO (0-100 nM) for either (a) 1 h or (b) 4 h before reincubation in MTS containing cell media for 4 h. Data are expressed as the percentage of metabolic activity compared to the non-treated group and represent the mean ± S.E.M from 3 independent experiments. * shows a significant difference (p < 0.05) compared to the non-treated cells; # shows a significant difference (p < 0.05) compared to the glucose/GO group without MPO, by a 2-way ANOVA with a Tukey's multiple comparison test.



Figure S2: Effect of the glucose/GO/MPO enzymatic system on intracellular thiols in J774A.1 cells.

J774A.1 cells (5×10^5) were treated with GO (50 mU mL^{-1}) in the absence and presence of MPO (20 and 50 nM) in HBSS containing glucose (5.6 mM) for 1 h before re-incubation in cell media for 24 h. Intracellular thiols were quantified using ThioGlo 1 and normalised to the total protein concentration measured by BCA assay. Data are shown as the fold change of thiols compared to non-treated group and represent mean ± S.E.M from three independent experiments. Analysis by 1-way ANOVA with Dunnett's multiple comparison test showed no significant changes compared to the non-treated control (HBSS).



Figure S3. Cytoplasmic Nrf2 and c-JUN are not altered in J774A.1 cells on exposure to the glucose/GO/MPO enzymatic system.

J774A.1 cells (2 × 10⁶) were incubated in HBSS containing glucose (5.6 mM) (black bars), with GO (50 mU mL⁻¹), GO/MPO (50 nM) or GO/MPO/SCN⁻ (200 μ M) for 1 h at 37 °C before re-incubation in cell media for 24 h. The cytoplasmic protein was extracted using a commercial kit and 10 μ g protein was loaded. β -actin was used as a loading control. Images are representative of 3 independent experiments (**A**). Panels **B** and **C** show the densitometry analysis of Nrf2 (**B**) and c-JUN (**C**) following normalization to β -actin. Data are expressed as the fold change compared to the respective non-treated group.