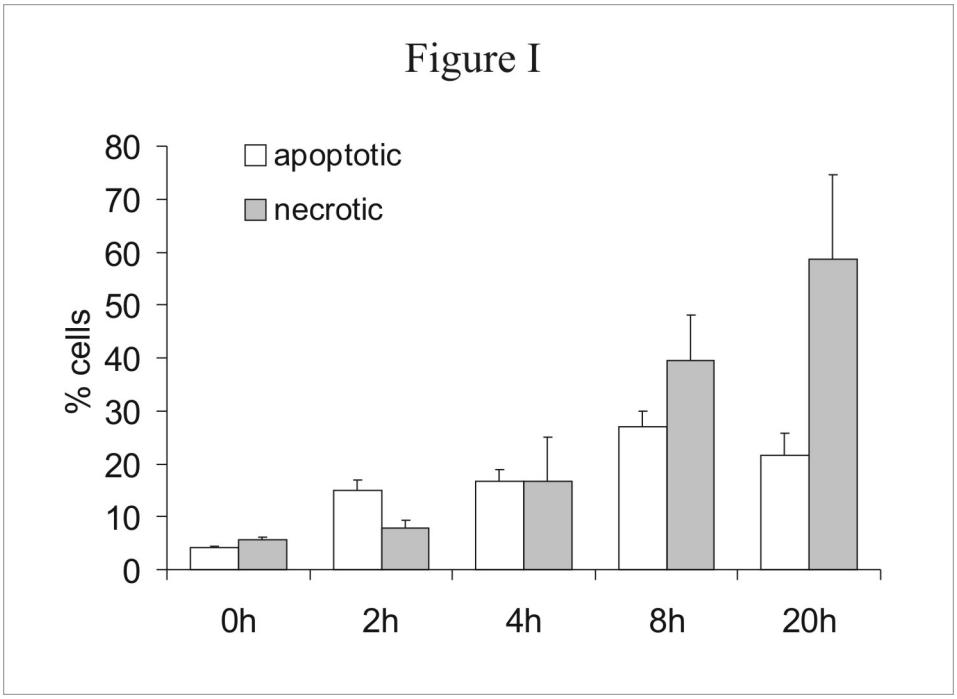
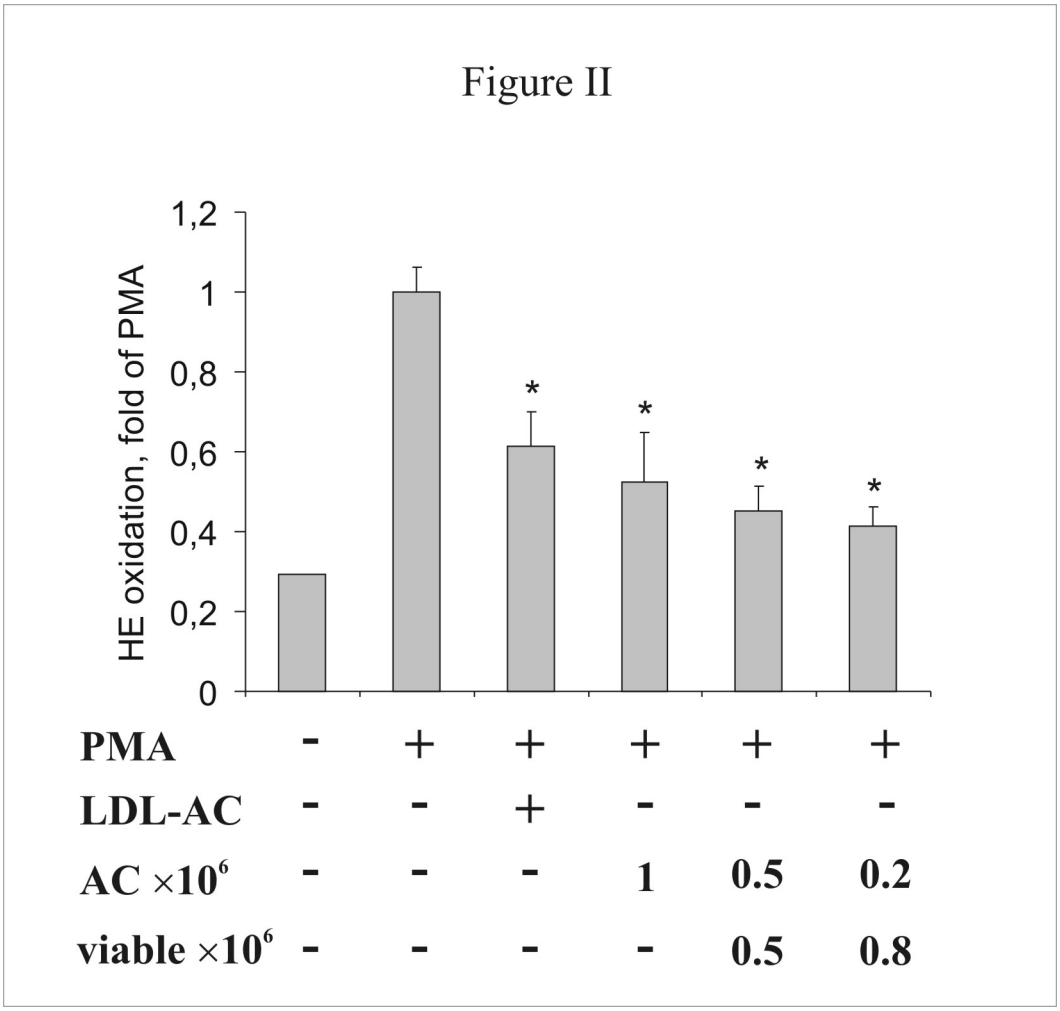
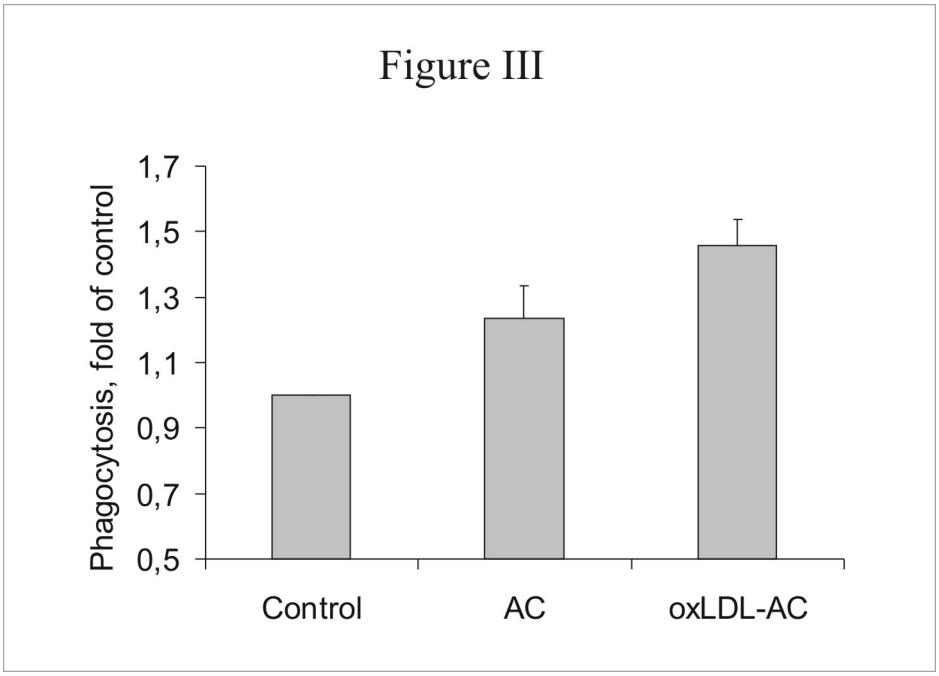


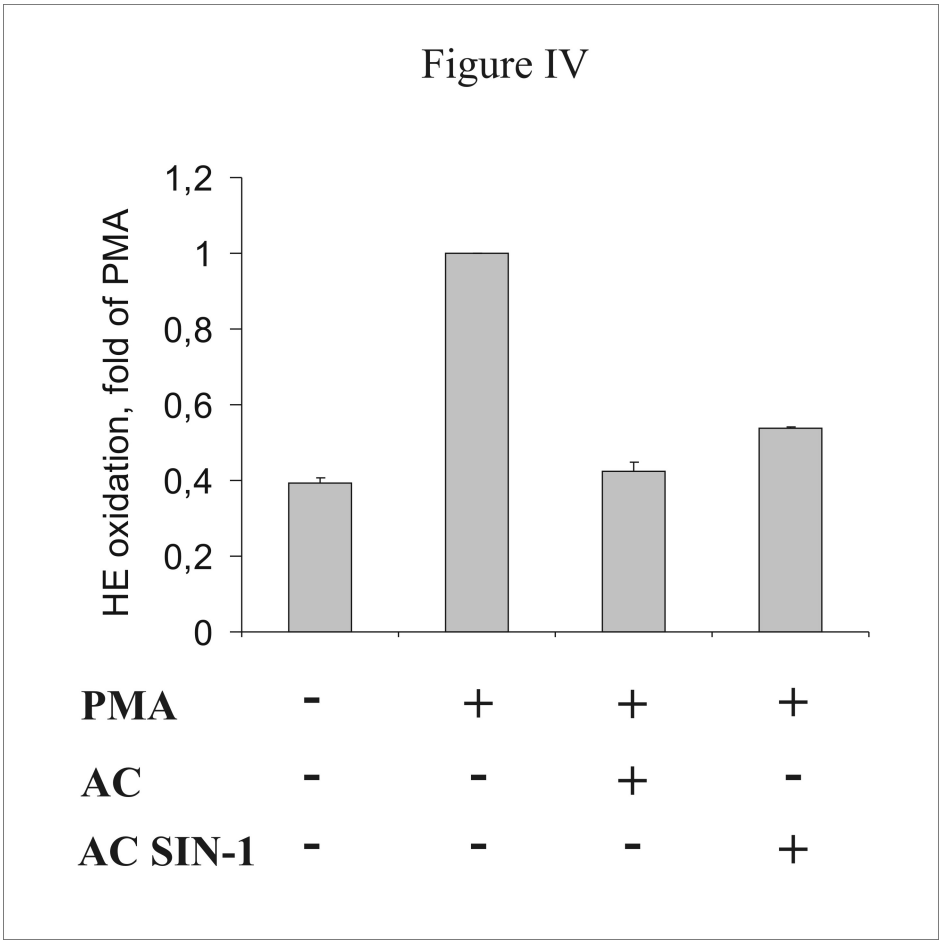
Supplementary Figure I. Time course of oxLDL-induced death of THP-1 cells. THP-1 cells were treated with 75 μg/ml oxLDL for indicated times and percentages of apoptotic/necrotic cells was analyzed by Annexin V/ propidium iodide staining and flow cytometry.



Supplementary Figure II. Predominant effect of apoptotic cells over viable cells in attenuating the oxidative burst, a lack of unmodified LDL effect. The oxidative burst was measured in RAW 264.7 macrophages incubated for 1 hour with indicated numbers of apoptotic (AC) and viable Jurkat cells as well as with 10^6 apoptotic cells generated in the presence of 75 μg/ml LDL (LDL-AC). *, P<0.05 vs. PMA.



Supplementary Figure III. Phagocytosis of apoptotic cells by RAW 264.7 macrophages. Jurkat cells were pre-labelled with 0.5 μM 5-chloromethylfluorescein diacetate (CMF-DA) prior to the treatment with 0.5 μg/ml staurosporine in the absence (AC) or in the presence of 25 μg/ml oxLDL (oxLDL-AC). Resulting apoptotic cells were incubated for 1 h with RAW264.7 cells labelled with 5 μM CellTracker Orange and phagocytosis was analyzed by flow cytometry as indicated in Methods. Data are presented as the fold increase of median FITC fluorescence.



Supplementary Figure IV. Apoptotic cells subjected to oxidative stress still inhibit the oxidative burst. The oxidative burst was measured in RAW 264.7 macrophages incubated for 1 hour with apoptotic Jurkat cells generated in the absence (AC) or in the presence (AC SIN-1) of 1 mM SIN-1. *, P<0.05 vs. PMA.