Monocytes/macrophages enhance surface expression and release of HSP60 following activation by 27-hydroxycholesterol

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Investigating differentially expressed proteins in a milieu rich in cholesterol oxidation products, we found via mass spectrometry-based proteomics that surface levels of heat shock protein 60 (HSP60) were upregulated on monocytic cells in the presence of 27-hydroxycholesterol (27OHChol). The elevated levels of cytoplasmic membrane HSP60 were verified via Western blot analysis and visualized by confocal microscopy. Treatment with 27OHChol also resulted in increased levels of cellular HSP60 without altering its transcription. Cholesterol, however, did not affect cell-surface levels and cellular amount of HSP60. GSK 2033, an LXR antagonist, inhibited expression of live X receptor α , but not of HSP60, induced by 27OHChol. Treatment with 27OHChol also resulted in increased release of HSP60 from monocytic cells, but the release was significantly reduced by inhibitors of endoplasmic reticulum-Golgi protein trafficking, brefeldin A and monensin. Results of the current study indicate that 27OHChol upregulates not only cell-surface and cellular levels of HSP60 but also its release from monocytic cells, thereby contributing to activation of the immune system.