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Title: Downregulation of IL-27 bronchial epithelial expression by heat-shock protein-60

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Body: Background Heat-shock protein 60 (Hsp60) is ubiquitous and highly conserved Molecular chaperone. Hsp60 plays an important role in protein folding, inflammation, and tissue repair. We previously reported increased levels of HSP60 in COPD patients, suggesting a role for HSP60 in the inflammatory response in COPD. This study is aimed to evaluate the HSP60 immunomodulatory activity and Th1/Tc1 cytokines (IL-27/INFγ) production in Human Bronchial Epithelial cells line (16HBE). Methods 16HBE were plated in 6 wells plate with Dulbecco's modified Minimum Essential Medium (DMEM) and exposed to various concentrations of HSP60 protein (1 ng/mL, 100 ng/mL and 1 µg/mL) for 8 and 24 hours. Induction of HSP60 and inflammatory molecules mRNA was evaluated after oxidative stress and inflammatory stimuli (H2O2, 100 µM; cytomix: IL-1β, 1ng/ml + TNFα, 10ng/ml + IFNγ, 10ng/ml). The effect of oxidative, inflammatory stimuli and of HSP60 was investigated by qRT-PCR using Rotor gene SYBER Green RT-PCR Kit (Qiagen) for detection of mRNA. Results Exposure of 16HBE to oxidative stress (H2O2) up-regulated HSP60 mRNA at 24h ($p<0.001$). Conversely, exposure of 16HBE to HSP60 protein down-regulated the Th1/Tc1 IL-27/IFNγ mRNA expression at 8h and 24h: IL-27 at 8h (mean ± SD: 20.59 ± 0.52 vs: 17.52 ±1.44 ΔΔ Ct (NT vs 1 µg/mL), $p=0.0256$) and 24h (mean ± SD: 20.90 ± 0.56 vs: 17.28 ± 0.26 ΔΔ Ct (NT vs 1 µg/mL), $p=0.0005$); INFγ was significantly reduced only at 24h (mean ± SD: 16.74 ±0.32 vs: 14.26 ± 0.20 ΔΔ Ct (NT vs 1 µg/mL), $p=0.0003$). Conclusion On the basis of these results we hypothesize a protective effect of HSP60 on Human Bronchial Epithelial cells, particularly after exposure to oxidative stress.