Effects of antioxidants on CSE-induced cell death in human asthmatic primary bronchial epithelial cells

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The link between cigarette smoke (CS) and lung inflammation is quite strong, however relatively little is still known on the effects of CS on human bronchial epithelial cells survival during asthma. In this study we focused our attention on the apoptotic effects of CS on healthy (HC) and asthmatic (AS) primary bronchial epithelial cells (PBEC) and on the role of antioxidants to protect epithelial cells from CSE-induced apoptosis.

Twenty subjects (10 HC and 10 AS) were recruited for this study and PBEC were obtained by bronchoscopy. PBEC were treated with oxidants (H_2O), anti-oxidants (GSH and AA) and cigarette smoke extracts (CSE). Early apoptosis (EA) and necrosis were measured by flow cytometry using Annexin-V and propidium iodide.

After treatment with CSE 20%, AS showed an increased susceptibility to the CSE treatment compared to HC (24.34+/-9.61 vs 48.45+/-11.91, p=0.003). Similarly, when EA was taken into consideration, there was a significant increase of EA cells in the AS group treated with CSE compared to HC (33.12+/-10.38 vs 16.73+/-6.92, $p<0.05$).

AA failed to protect both HS and AS PBEC from CSE-induced cell death. GSH instead was able to protect significantly both HS and AS from CSE-induced cell death. In particular, the association between GSH and CSE 20% determined a significant (p=0.005 in HC and p=0.003 in AS) increase of viability when compared to CSE alone and at the same time EA levels dropped considerably ($p<0.05$ in HC and p=0.003 in AS) down in the presence of this antioxidant Moreover, GSH treatment determined a significantly bigger (p=0.002) overall increase in viability in the AS group when compared to the HC group.

In view of this data it could be possible to hypothesise that the typical imbalance in oxidants-antioxidants levels of asthmatic bronchial epithelial cells might be responsible for their increased susceptibility to oxidative stress.

Key words
Bronchial epithelium, asthma, cigarette smoke, oxidative stress