Urban dust cytotoxicity in human alveolar epithelial cells (A549) with depleted glutathione

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INTRODUCTION

Exposure to air pollutants causes many health problems including asthma, COPD, cardiopulmonary diseases, lung cancer, and birth defects. The consequences of exposure are highly unpredictable, but the exposure brings about persistent inflammation of the airways. Short-term exposures to air particulate matter (PM) usually exacerbate pre-existing diseases, especially in respiratory and cardiovascular system, depending on the individual age and health status and increase hospital admissions. Long-term exposures, on the other side, increase the rate of disease progression and significantly reduce life expectancy. Due to high inconsistency of clinical and experimental data in this field, it is crucial to establish some biomarkers for PM exposure, which may unravel the complexity of relationship between exposure and respiratory health.

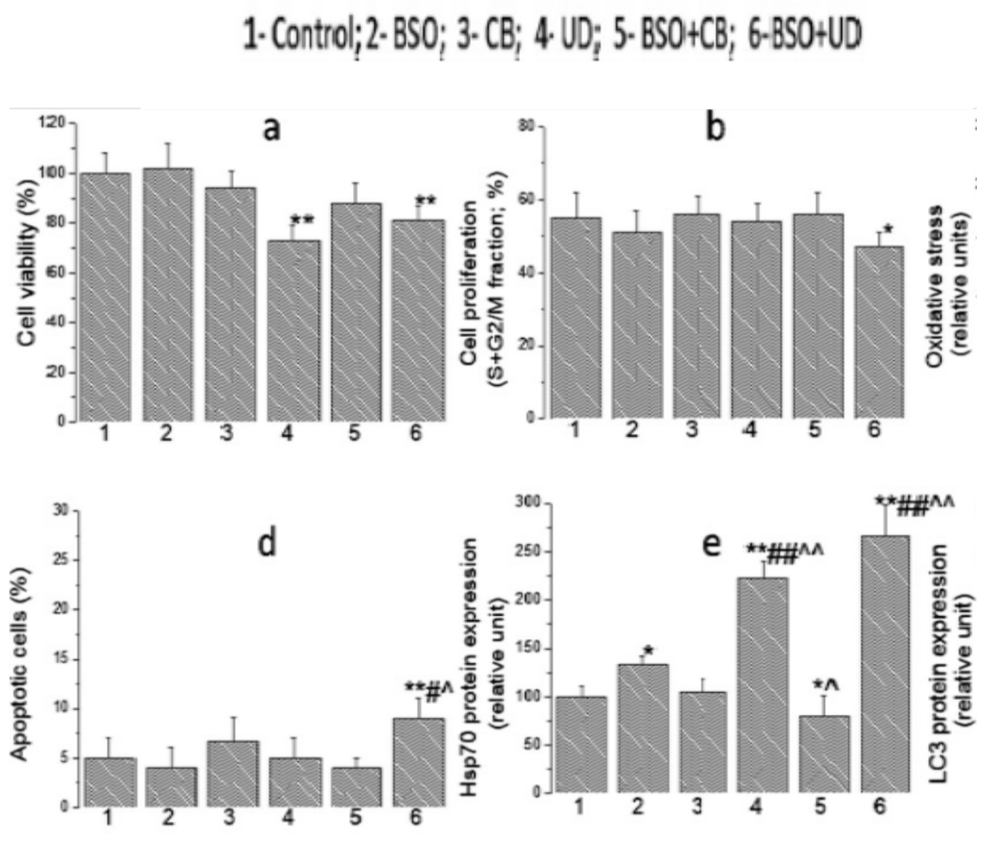
AIM

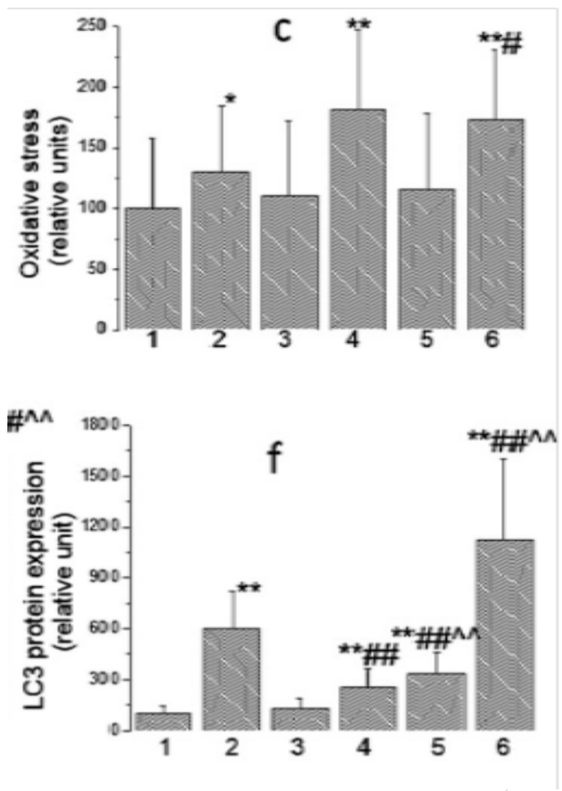
In recent years, evidence accumulates in support of a notion that PM-induced ROS are important to autophagy, but it is still unclear of how ROS and inflammation drives autophagy in PM-exposed cells. Therefore, in this study we seek to explore the mechanisms of PM-induced ROS and autophagy using standardized urban dust (UD) and the human alveolar epithelial A549 cells.

METHODS

- Cell Culture
  - Cell Treatment
  - Cell Viability and Proliferation
  - Oxidative Stress
  - Autophagy and Heat Shock Protein 70
  - Apoptosis
  - Proteins and Glutathione (GSH) Levels
  - Statistics

RESULTS





CONCLUSIONS

-In the present experimental model, UD decreased intra- cellular GSH by about 30% and diminished cell viability by about 27%. However, a similar decrease in GSH by BSO was without a significant effect on cell survival. Moreover, when both GSH-depleting compounds, BSO and UD, were sequentially applied to the cells, UD toxicity was not enhanced. It is, therefore, possible that GSH depletion is not a prerequisite for acute UD toxic- ity in A549 cells. Cell proliferation was unaf- fected by CB and UD. However, when A549 cells were pre-treated with BSO and then treated with UD, a significant reduction in the number of dividing cells was noticed.

-In the present study, pro-oxidative alterations were detected in cells treated with BSO, in cells grown with UD, and in cells treated successively with both compounds. However, there was no additive effect of BSO and UD on oxidative stress.