Effect of Fine Particulate Matter (PM2.5) Collected in Hong Kong on Mitochondrial Permeability Transition Pore (mPTP) in Human Airway Epithelial Cells

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Background

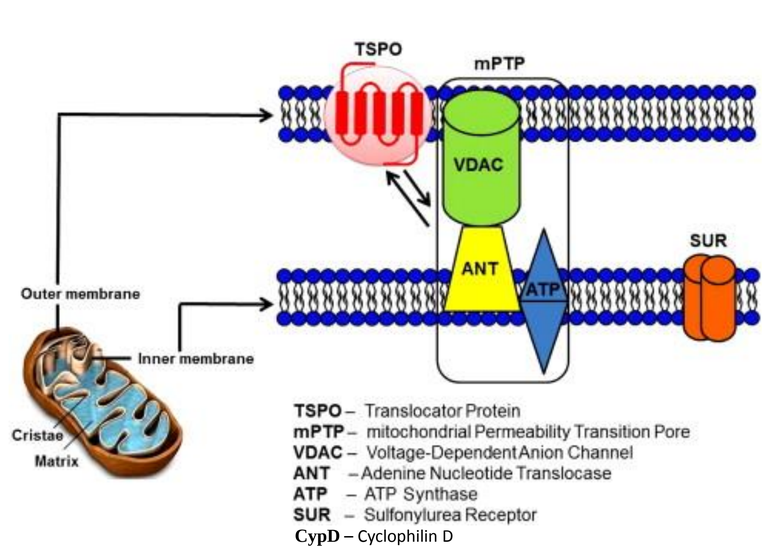
- **Air pollution was the 4th leading risk factor for early death worldwide in 2019.**
- **PM2.5, also called fine particulate matter, has the an aerodynamic diameter of less than 2.5 μm.**
  - ***PM2.5 could penetrate deep in the lungs reaching the large and small airways and even alveoli.***
- **PM2.5 is closely related to the mortality rate and the incidence of human respiratory diseases, such as asthma and chronic obstructive pulmonary disease (COPD).**
- **Mitochondria are highly sensitive to environmental toxicants and their roles on respiratory diseases have been well-documented. However, studies on the relationship between PM2.5 and mitochondria in respiratory tract are limited.**
- **Recently, strong evidence is emerging that mitochondrial permeability transition pore (mPTP) may be important in certain physiological conditions and in the processes of cell damage and death.**

Hypothesis

- **PM2.5 collected in Hong Kong may affect mPTP and cause apoptosis in human airway epithelial cells.**

Aims

- **To investigate the role of PM2.5 collected in Hong Kong on mitochondrial function in human bronchial epithelial cells (BEAS-2B);**
- **To study the role of PM2.5 on mPTP in BEAS-2B cells;**
- **To explore the potential mechanism by which PM2.5 might regulate lung disease.**



Methods and Results

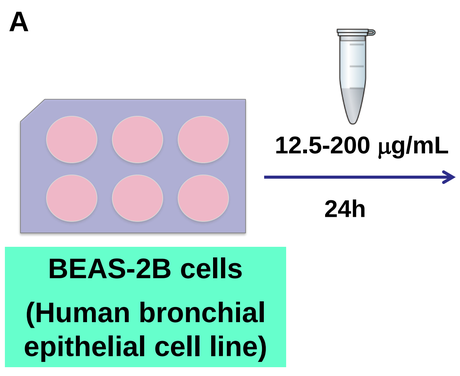


Figure 1. Experimental design.

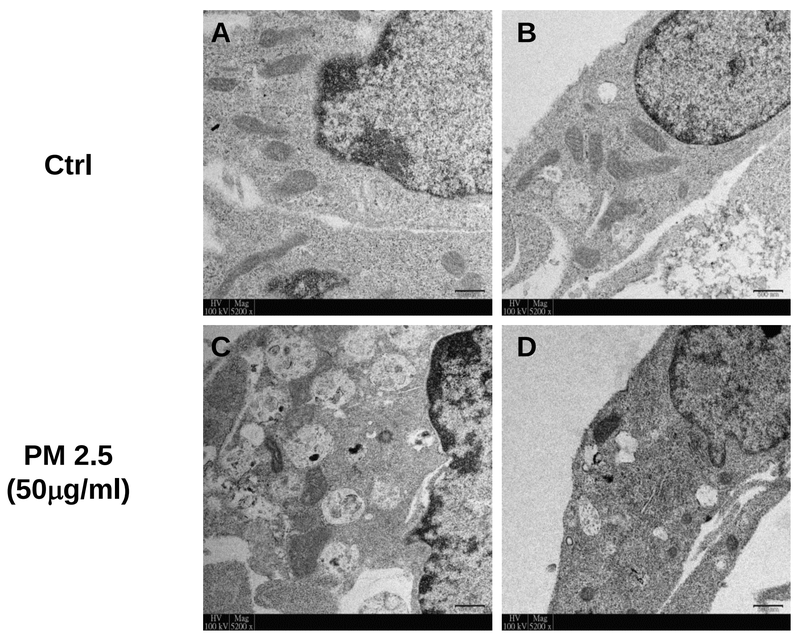


Figure 2. Morphological alterations on mitochondria visualized by TEM images.

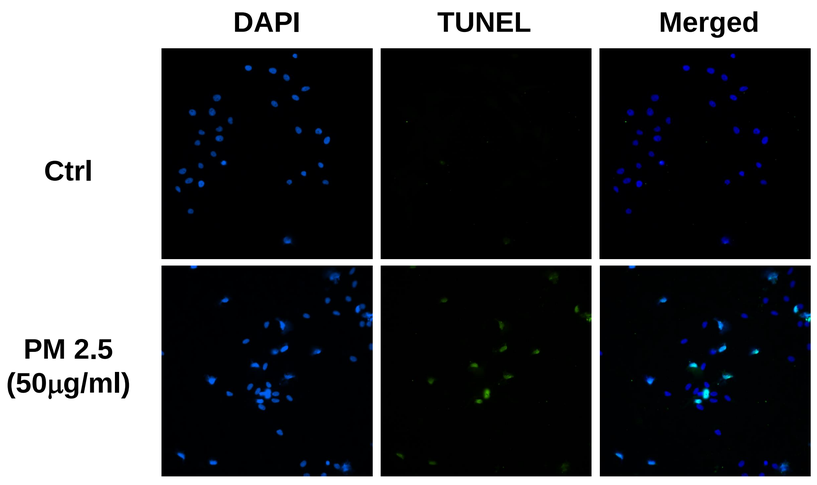


Figure 6. Cell apoptosis detected by TUNEL staining.

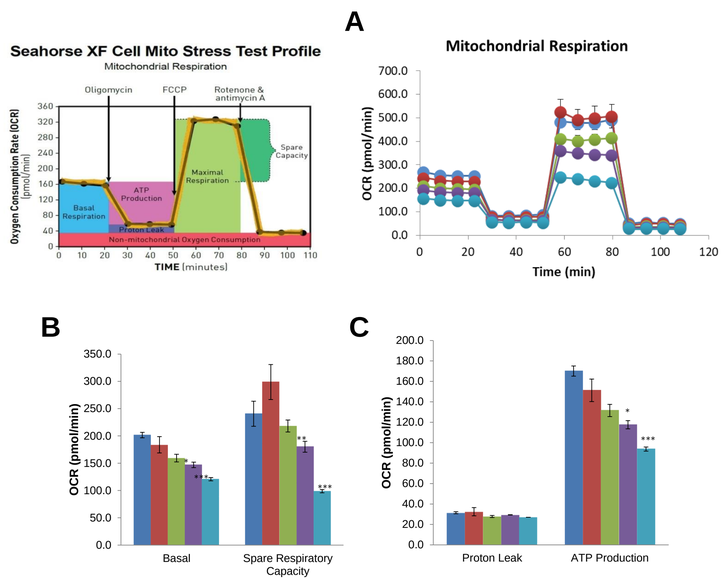


Figure 3. Mitochondrial respiratory function after PM2.5 exposure (n=3).

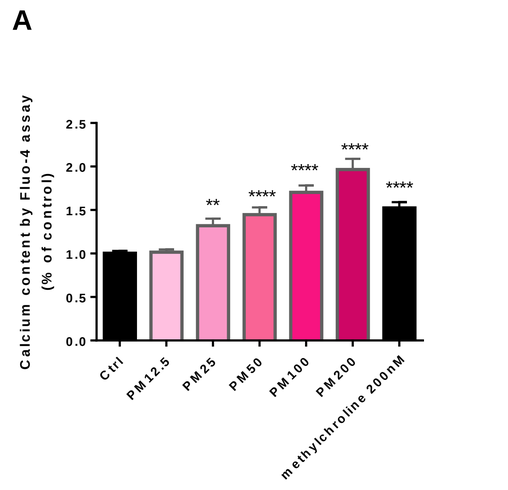


Figure 4. Intracellular Ca^{2+} content after PM2.5 exposure (n=5).

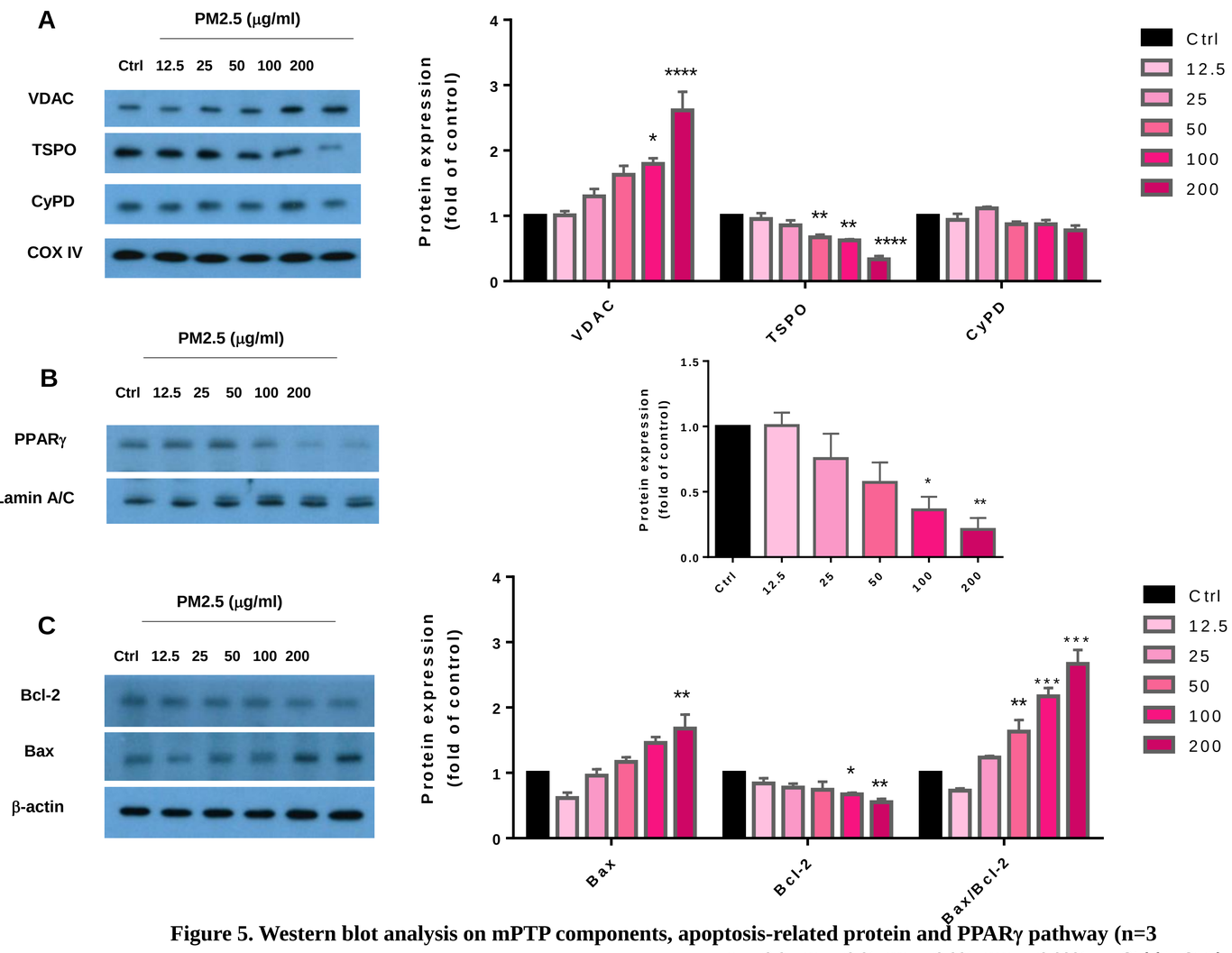
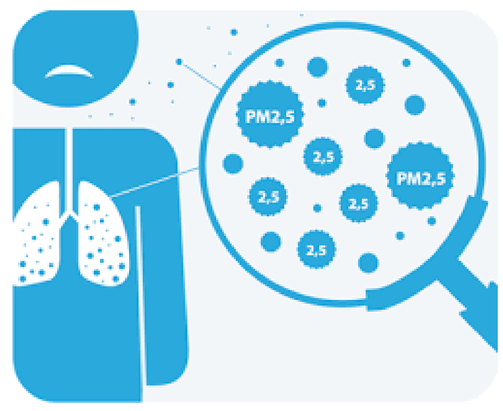


Figure 5. Western blot analysis on mPTP components, apoptosis-related protein and PPARγ pathway (n=3

*** p <0.05, ** p <0.01, *** p <0.001, **** p <0.0001 vs. Ctrl (ANOVA)**

Summary and Conclusions

- **Our data has successfully demonstrated that exposure to PM2.5 collected from Hong Kong area caused mitochondrial swelling and structural damage on cristae from TEM images.**
- **PM2.5 exposure reduced mitochondrial basal respiration and ATP production.**
- **PM2.5 exposure significantly increased intracellular calcium content in a dose-dependent manner, which was accompanied by the upregulation of the major mPTP component VDAC expression.**
- **PM2.5 caused mitochondrial injury via PPARγ pathway, eventually led to cell apoptosis.**



Acknowledgement : This study is supported by Hong Kong Lung Foundation (HKLF) Research Grant Award and National Natural Science Grant of China Grant (NSFC 81870038).