ATS 2018 Highlights

Respiratory Structure and Function Early Career Professionals

Get to know members of the RSF Assembly

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Is your research clinical, basic science or translational?

Basic Science

Tell us about your research?

Smoking is long thought to be the only cause of COPD, but many COPD patients are non-smokers. Pollution, and in particular particulate matter (PM) exposure, is considered as a major risk factor for non-smoking COPD. To date animal models have used acute high dose exposure. My research aims to create a more realistic long term low dose PM exposure model. The next step is to identify possibly pathways how PM exposure might lead to COPD in adulthood.

Where do you see yourself in 5 years?

Research, academia and teaching. I want to establish a research group that focuses on studying various possible causes of lung fibrosis.

What do you find is the major benefit of RSF Assembly Membership?

As an early career researcher, the RSF assembly will provide me opportunity to network with top physiologist. More people knowing my work can help establish future collaborations

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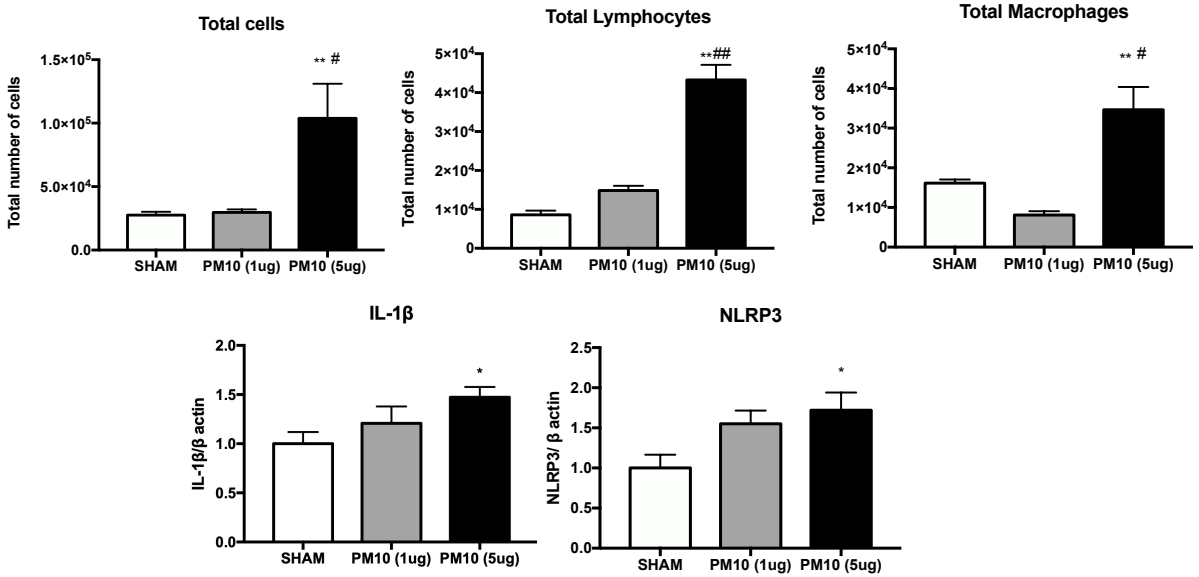

Figure 1. This figure shows that a 5ug PM exposure for 3 weeks can already elicit inflammatory response in Balb/c mice. This include increase in number of lymphocytes, macrophages in BALF. This is accompanied by increased IL-1β and NLRP3 (inflammasome) protein level. Results are expressed as mean ± SEM, n = 8. *p<0.05, **p<0.01, compared with SHAM; #P<0.05, ##p<0.01, compared with PM_{10} (1ug). PM_{10}: particulate matter exposure.

Impact of traffic related air pollutant exposure on lung inflammation and mitochondrial wellbeing in mouse lungs

It is well accepted that traffic related air pollution (TRAP) is detrimental for respiratory health, causing inflammation. One of the core components of TRAP is particulate matter (PM). However, there is no consensus in animal models, and most models used high dose of PM acutely which might not be physiologically relevant.

Objective: In this study, we aimed to develop a mouse model of subchronic low dose PM exposure representing similar environmental exposure as the humans.

Methods: Balb/c mice (7 weeks old, male) were exposed to saline or roadside PM10 (<10 micron; particle removed from Teflon filter) 1µg or 5µg daily for three weeks (n=10). Inflammatory response was assessed in bronchoalveolar lavage (BAL), and mitophagy markers (to measure recycling mechanism of mitochondria) was measured by Western blotting (n=8).

Results:. Exposure to 1µg of PM10 did not affect inflammatory and mitochondrial markers in the lung, while 5µg of PM10 exposure increased lymphocytes and macrophages in BAL. Inflammatory cytokine IL-1β was increased and mediated through upstream NLRP3 in mice exposed to 5µg PM10 exposure (P<0.05). IL-6 protein level was not changed. PM10 reduced mitochondrial antioxidant manganese superoxide and mitochondrial fusion marker OPA-1 and increased mitochondrial fission marker Drp-1(P<0.05). Autophagy marker LC3-II and AMPK were reduced with increased apoptosis marker caspase-3 (P<0.05). Fibrotic markers fibronectin, TGF-β1 and Collagen-III were not changed by PM10 exposure at either 1µg or 5µg.

Conclusion: Low level of PM10 exposure can elicit inflammation and alter mitochondrial fission, fusion and autophagy in the subchronic setting. This model can be used to study other TRAP exposure related conditions, such as asthma.

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