Protein thiol oxidoreductas and allergic airways disease

Statements
regarding the dissertation

Protein thiol oxidoreductases and allergic airways disease

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Wednesday March 2, 2016

1. ER stress is induced in human nasal and bronchial epithelial cells and inhibition of ER stress responses may provide a potential therapeutic avenue in chronic asthma and sub-epithelial fibrosis (this thesis).

2. Activation of classical and alternative NF-κB pathways occurs within the airway epithelium and may coordinately contribute to allergic inflammation, AHR and fibrotic airway remodeling (this thesis).

3. The Grx1/*S*-glutathionylation redox status in mice is a critical regulator of airways function, suggesting that avenues to increase *S*-glutathionylation of specific target proteins may be beneficial to attenuate airways hyperresponsiveness (this thesis).

4. Grx1 and the protein thiol redox environment influences the nature of house dust mite-induced adaptive immune responses by promoting T_H2 driven inflammation and restricting IL-17A (this thesis).

5. Endoplasmic reticulum stress, oxidative stress, and inflammatory processes are intimately linked, highlighting the relevance of investigating the role of Grx1 and *S*-glutathionylation of PDIs in epithelial cell ER stress, apoptosis and fibrotic remodeling in allergic asthma (this thesis).

6. Therapeutic potential does not lie in distinguishing 'bad' from 'good' ROS, rather determining the extent to which aberrant oxidant production can be dampened while preserving physiologic function.

7. The balance of the pulmonary immune response is closely integrated with lung thiol redox status.

8. Asthma cannot always be categorized; the root causes and manifestations of the disease are often a sliding scale that incorporates multiple complex factors.

9. Take a breath to stay alive, but don't take more than needed.

10. Nature is so patient, why can we not be.