**Obesity Impact on Airway & Lung: Value of TNFα Receptor Blocker**

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### BACKGROUND

- In the US, more than 1/3 of adults are obese (1)
- Obesity has been linked to asthma, with abdominal obesity restricting lung volumes, lowering chest wall compliance and decreasing respiratory muscle efficiency (2)
- However, less is known about pulmonary inflammatory responses associated with raised levels of systemic inflammation often seen in the obese (2)
- Previous studies in lab animals have shown that lung inflammatory responses induce pro-inflammatory mediators (Leptin, IL6) from adipose tissue (2)
- This study investigates the role of obesity in pulmonary inflammation and aims to determine if obesity alone is related to greater lung inflammation
- We will compare degree of inflammation in control ob/ob mice (genetically obese, leptin knockout mice) and TNFα receptor blocker ob/ob mice
- We hypothesized that there would be a decrease in inflammation systemically and that both fat and lung would show lower inflammation

### METHODS

- TNFα receptor blocker was administered 2x weekly X 8wks at a dose/kg (body weight)
- Abdominal fat and lung were paraffin embedded, sectioned and stained with H&E and with labeled with F4/80 antibody.
- 9 high power fields were counted on each tissue section (400x)
- Sera from both groups were tested by ELISA for IL6 and TNFα
- Statistics utilized the Statistica program

### RESULTS

#### RESULTS I

- Without intervention, inflammatory macrophages were found at high levels in lung as well as abdominal fat
- Mice receiving treatment had significant reduction of inflammatory macrophages in abdominal fat (p < 0.01)
- Treatment of mice did not lead to significant reduction of inflammatory macrophages in lung (p = 0.32)

**Further Research Questions**

- Do inflammatory macrophages originate in adipose tissue and distribute to other organ/sites?
- Since our intervention decreased only the abdominal inflammatory macrophages, can systemic effects of obesity be limited

**Strengths and Weaknesses**

- Use of appropriate genetically obese model
- Use of pictures to demonstrate procedures
- Size of group had been determined by a previous study and was adequate to find significant differences
- Count of inflammatory macrophages in fat and lungs of normal strain of mice would have furnished interesting data, but these would not be on obese mice

### References