The Effect of Environmental Tobacco Smoke (ETS) on Immune Function and Susceptibility to Bacterial Infection in BalbC Mice
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Objective:

To characterize the bacterial susceptibility of Balb/c mice exposed to ETS.

Proposed research to accomplish:

Secondary bacterial infection during or shortly after recovery from an influenza virus infection is a common cause of pneumonia and influenza-related death. One of the most common pathogens involved in influenza-associated bacterial pneumonia is *Staphylococcus aureus* which is found frequently in the upper respiratory tract of healthy children as well as in those with disease. Studies show that mice are also susceptible to this pathogen, and there is research to support that mice can be superinfected with these bacteria after a primary influenza infection. The objective of this study is to characterize the infectivity of the bacterial strain *S. aureus* ATCC 29253 in a naturally susceptible animal host, BALB/c mice. To establish the optimal bacterial dose and to determine the infectivity of this bacterial strain will allow us to most effectively conduct experiments to investigate the hazardous effects of perinatal exposure to environmental tobacco smoke (ETS) on the susceptibility of bacterial infection after primary infection with influenza. This project is a contribution to the research efforts detailing how exposure to ETS during the perinatal period increases the incidence and severity of respiratory influenza infection in infants and young children and how early exposure to ETS increases the morbidity and mortality arising from secondary bacterial infection following influenza infection. We hypothesize that ETS negatively affects phagocytic ability of alveolar macrophages in the lung despite an increase in opsonizing ability of antibodies in bronchoalveolar lavage fluid (BALF).

Brief discussion of results:

We saw that infecting mice with concentrations of *S. aureus* below 1x10^8 CFU per 100 μl was ineffective at producing an adequate cellular response; however, an increase in total cell count in mice administered concentrations of 2x10^8 and 1x10^8 CFU per 100 μl was observed. The total cell counts and cell differentials were similar between the groups given 2x10^8 and 1x10^8 CFU per 100 μl, suggesting that at 24 hours post-infection a maximum cellular response had been achieved. Data showed that an adequate macrophage response occurred in mice inoculated with the saline control. Mice challenged with bacterial suspensions required a neutrophilic response to overcome acute infection. Lymphocytes were recruited to the site of inflammation by the time the one week necropsies were performed, indicating an antibody-mediated response in the mice. The inflammation in response to bacterial inoculation resolved within one week with cell counts returning to control levels; however, ETS appeared to significantly enhance delayed lymphocyte influx to the lungs in response to bacterial infection. We concluded that *S. aureus* infection in Balb/c mice overwhelms alveolar macrophages and as a result infection exhibits a dose response with significant influx of neutrophils into the lungs instead.

Now that we have characterized the response of Balb/C mice to infection with *S. aureus*, we are eager to observe the difference in the immune response of mice inoculated with the bacteria following a primary influenza challenge.