It is understood that exposure to wildfire smoke can exacerbate respiratory disease, however the long-term effects of exposure during development is understudied. Our research objective is to determine how early life wildfire exposures precede adult chronic lung disease. We hypothesize that neonatal lung epithelium is more susceptible to inflammation and injury as a result of wildfire smoke $PM_{2.5}$ exposure. Furthermore we propose that wildfire smoke $PM_{2.5}$ exposure can lead to ER stress in the lung epithelium. I will conduct *in vitro* exposures using primary rhesus monkey epithelial cultures derived from infant and adult monkeys at Air-Liquid interface. Exposures will include acute and prolonged timepoints, with measurements including analysis of cell proliferation, ER stress markers, and innate immune function. Additionally, I will conduct *in vitro* exposures using neonatal and young adult wild type C57BL/6 mice. Mice will be dosed intratracheally with wood smoke particles, timepoints will be at 24 hours and 8 weeks, as a control a 16 week unexposed neonatal group will be used. Lung injury will be measured using lavage samples analyzing cell counts, BCA and LDH assays. Mouse lungs will also be fixed and examined for injury, and SP-C positive cells will be quantitated.