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Antenatal exposure to secondhand smoke impacts growth and cardiopulmonary energetics in 4-week-old mice

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Secondhand smoke (SHS) is a well-established cause of respiratory illness in infants and children who live in environments where exposure is common. Recent studies have been performed to examine the potential effects of SHS exposure on fetus’s before they are born1. The data collected suggests that antenatal exposure to SHS impacts cardiovascular and respiratory function through childhood and may even have lifelong ramifications, even if the exposure is terminated shortly after birth. In our study, we sought to determine the impact of SHS exposure during development in the womb would have on 4 week-old mice, or the equivalent of an early adolescent human. We sought to look especially at deficits Cardiovascular and Respiratory function.

Four pregnant mothers were chosen from a common strain of mice2, and exposed to SHS via a nose-only delivery system3 beginning on gestational day 14.5 through gestational day 17.5. This system takes puffs alternately from two cigarettes and pumps the second hand smoke from the surround chamber to the apparatus containing the mice. Four similar pregnant mothers were restrained and exposed to room air as a control. After exposure to SHS for four days, the mice were permitted to give birth as normal. It should be noted that the typical gestational period for mice is 19-21 days, thus we minimized smoking to 4 days in order to ensure consistent exposure to SHS amongst subjects. At 4 weeks of age, the pups were taken and tests were performed. The mice were weighed, and blood pressure and heart rate were determined using a tail occlusion cuff4. Whole body weights were compared, as were heart/whole body and liver/whole body weights. Lastly, organ specific mitochondrial function tests were performed to determine the efficiency of oxygen use by the specific organ.

Our results show that at 4 weeks of age the pups whose mothers were exposed to SHS had 1) a significant reduction (p<0.03) in total body weight; 2) significantly elevated systolic (p<0.0002) and diastolic (P<0.004) blood pressure; 3) significantly decreased heart (p<0.004) and kidney (p<0.0006) weights when indexed to body weight; and 4) significantly decreased oxygen consumption related to cellular respiration in SHS-exposed hearts and lungs when compared to room air exposed controls. However, no significant differences were seen in heart rate.

Our data indicate that antenatal exposure to SHS has a detrimental effect on growth rates and cardiopulmonary energetics through the 4th week of age, or the equivalent of early adolescence in humans. These results may be beneficial in understanding the long-term effects of antenatal SHS exposure. Further studies would be required to determine if these negative effects continue into late adulthood, and to what degree older mice may be effected by the SHS smoke exposure when they were in the womb. We speculate that these subjects would be at a greater risk for developing other lung related diseases such as Chronic Obstructive Pulmonary Disease.
