5252

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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 examining potential effects of antenatal SHS exposure suggest that antenatal exposure to SHS impacts cardiovascular and respiratory function through childhood and may have lifelong ramifications. In the present study, we sought to determine effects of antenatal SHS exposure on 4-week-old mice. C57/Bl6 mice (n=8) were exposed to SHS or room air via a nose-only delivery system (Scireq) beginning on gestational day (dGA) 14.5 to 17.5 dGA. At 4 weeks of age, mice were weighted and blood pressure and heart rate was determined with a tail occlusion cuff (Kent Scientific). Whole body, heart/body and liver/body weights were determined. Lastly, organ-specific mitochondrial function tests were performed. At 4 weeks of age, antenatal exposure of SHS caused: 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) no differences in the animal's heart rate; 4) significantly decreased heart (p<0.004) and kidney (p<0.0006) weights when indexed to body weight; and 5) significantly decreased oxygen consumption related to cellular respiration in SHS-exposed hearts and lungs when compared to room air exposed controls. Our results 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. These results may be beneficial in understanding the long-term effects of antenatal SHS exposure.

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