

The Respiratory Effects of Tobacco Smoke Exposure on the Fetus and Child

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The adverse effects of environmental tobacco smoke (ETS) on health, both for the active and passive smoker, have been recognized and documented for decades with regards to heart and lung disease and risk of malignancies. The effect this exposure has on the developing respiratory system with respect to lung growth and function of the pediatric patient will be the topic of this article. This review will also discuss the respiratory related effects of ETS with regard to Sudden Infant Death Syndrome (SIDS), asthma and nasal/sinus disease.

Means of Exposure

Children are at risk from tobacco exposure on multiple fronts. The earliest risk occurs in utero with maternal smoking. The effects on the developing fetus may be secondary to direct effects of nicotine or carbon monoxide, both of which cross the placenta, or to other chemical constituents including cadmium, which accumulate in the placenta and may have a role in intrauterine growth retardation.¹ Postnatal exposure to the products of ETS includes transmission through breast milk and passive exposure. This may occur either as the result of contact with smoke directly exhaled by the active smoker or by inhalation of sidestream smoke, which results from the combustion of the tobacco along with its delivery device (e.g. cigarette, cigar, pipe, etc.) Finally, children may actively smoke. One must bear in mind that it may, at times, be

difficult to separate out the prenatal effects of exposure from the postnatal effects, since exposure frequently occurs spanning both timeframes.

ETS and Sudden Infant Death Syndrome (SIDS)

The incidence of SIDS is increased significantly in infants exposed both prenatally and in those who are exposed to ETS postnatally. The exact mechanism that accounts for this increase is not known; however, effects on cardiorespiratory control, including abnormal responses of the autonomic nervous system (ANS) along with an abnormal arousal response to hypoxia, secondary to abnormalities in brainstem development and/or peripheral chemoreceptors have been postulated.

Nicotine appears to exert effects both within the brainstem and peripherally on baroreceptors located within the aorta and carotid arteries. Some animal models have documented abnormalities in auto-resuscitation in response to hypoxia² and delayed arousal from quiet sleep following exposure to nicotine.³ Possible prenatal effects of nicotine exposure on the ANS are suggested by studies that show reductions in heart rate variability (HRV) in term males of mothers who smoked during pregnancy.⁴ A second study by Dahlstrom also linked decreased HRV in male infants to nicotine exposure in breast milk, independent of in utero exposure.⁵ Other effects noted that suggest an alteration in cardiovascular regulation include elevation in the blood pressures of neonates and infants exposed to ETS in utero.⁶

Abnormalities in arousal responses also have been noted in infants exposed to ETS. Cutz, et. al. studied peripheral receptors in the lungs that sense airway hypoxia. Irritants such as nicotine and products of inflammation that occur with viral infections may reduce the response of these sensors, leading to a blunted arousal response.⁷ An alteration

in the auditory arousal response during sleep also has been noted.⁸

ETS and Nasal and Sinus Disease

An effect of ETS exposure on the development of disease in the nose and sinuses occurs either as the result of enhancement of allergic reactions or secondary to changes in the immune response.

An increased risk of sensitization to dust mites has been noted. This appears to be impacted more by exposure to maternal than paternal smoking.⁹ Enhanced allergic reactions to inhaled allergens secondary to an increase in epithelial permeability to environmental allergens has been noted following secondhand smoke exposure.^{10,11}

Immune dysfunction also has been noted. Diminished nasal mucociliary clearance has been demonstrated following secondhand smoke exposure.¹² Other abnormalities in the immune system include alterations in cytokine production, abnormalities in fetal mononuclear cell responses and alterations in Toll-like receptors. These latter receptors play a role in microbial responses and in the inhibition of allergic immune responses.¹³

ETS and Lung Growth, Function and Bronchial Hyperreactivity

Multiple factors secondary to ETS exposure in utero appear to be responsible for the abnormalities noted in lung size, reactivity and elasticity. These include preterm birth, nutrient deficiency, hypoxia and direct toxic effects of nicotine.¹⁴

Infant pulmonary function testing has documented impairments in fetal airway development manifested as a decrease in expiratory flow rates¹⁵⁻¹⁷ and an elevation in airway resistance.^{18,19} These changes may be secondary to abnormalities in the thickness of the walls of the airway, resulting in a decrease in the caliber of the airway lumen.²⁰ Animal models would suggest this may be a direct effect of nicotine exposure.²¹

Postnatal exposure studies reveal a reduction of forced expiratory volume in the first second (FEV1) of 1.4 percent. A greater reduction is noted in mid-expiratory flow rates, including forced expiratory flow (FEF) 25 percent to 75 percent where a 5 percent reduction was noted, along with a 4.3 percent reduction in end-expiratory flow rates.²²

Bronchial reactivity has been noted to be increased in infants exposed to ETS. There is an increased incidence of wheezing illness in infants and children exposed to parental smoking. This increase appears to continue to the age of six years but not beyond.²³ Causes of increased bronchial hyperreactivity include an elevation in cells in the airways that synthesize and release mediators that cause bronchospasm and stimulation of bronchopulmonary C fibers.²⁴ These C

fibers, upon stimulation, cause bronchoconstriction, increase in microvascular leakage and an increase in mucus production. An increase in IgE has also been described in response to ETS.²⁵

ETS and Adolescents, Adults and Parents

The impact of parental smoking on adolescent and adult behavior also has been documented. The model that parents who smoke present to their children can have a lifelong effect on the smoking behavior of their impressionable offspring. Paul, et al. have documented that the children of parents who smoke have an increased risk of becoming smokers as adults.²⁶

The effects of active smoking in adolescents with asthma include more severe symptoms, a decline in lung function and impairment in response to short-term courses of corticosteroids.²⁷

Finally, one recent study²⁸ found that parents who smoked underestimated the harm ETS exposure could have on their child. On a positive note, this study also noted that most of these same parents were receptive to programs that would reduce this exposure.

Conclusion

ETS can have a profound effect on the developing respiratory system of the fetus, newborn and child. For readers who wish a more extensive review, the Surgeon General's Report on the Health Consequences of Involuntary Exposure to Tobacco Smoke, 2006,²⁹ is recommended.

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