

# The Impact of Secondhand Smoke on Children: Respiratory and Other Medical Concerns

By Mark G. Lounsbury, DO, MA; Mark E. Bubak, MD

## Abstract:

Secondhand smoke (SHS) is commonplace in South Dakota. It is casually linked to an increased incidence and severity of childhood and adult asthma; lower respiratory tract infections; symptoms of cough, wheeze, phlegm; acute and chronic otitis media; sudden infant death syndrome; cancers; prematurity; and cardiovascular disease. At this time, the development of specific allergy cannot be causally linked. As there is no risk-free level of secondhand smoke, tobacco smoking cessation is the only effective treatment and should be actively pursued at every opportunity.

## Introduction

Secondhand smoke (SHS) regularly affects 45,000 South Dakota children.<sup>5</sup> This comes from the smoke of 119,700 smokers in South Dakota, who make up 20.4 percent of the state's population.

This article is to review the effect of this secondhand smoke on these children's respiratory and immunologic systems.

As early as 1969, Cameron, et. al. felt children exposed to SHS at home had a higher incidence of general illness, primarily respiratory illness.<sup>1</sup> Two Lancet articles in 1974 furthered these concerns to include a link between parental tobacco usage and acute lower respiratory illness in infants.<sup>2,3</sup> Studies over the next decade led to the 1986 Surgeon General's report of illnesses associated with SHS<sup>4</sup> that has been updated in the 2006 report.

Secondhand smoke contains over 4,000 chemicals that include many toxins, carcinogens and irritants. There is no safe level of SHS. Only smoke-free environments protect nonsmokers from the dangers of secondhand smoke.

## Disease States

Many illnesses are linked to secondhand smoke, with adverse respiratory symptoms and illnesses perhaps being the best-known.

Multiple studies demonstrate an increased frequency of the respiratory symptoms cough, phlegm and wheeze in the children of smokers.<sup>4,6,7</sup> Exposure to SHS has been linked to

increased asthma prevalence and severity.<sup>6,8,9</sup> The mechanism of this boost in asthma is yet to be fully enlightened, but appears to start in utero. Young and colleagues article reported that in utero exposure to maternal tobacco smoke increased airway responsiveness in newborn infants.<sup>10</sup>

Is the effect simply due to prenatal factors? Cook and Strachan in a 1997 Thorax article<sup>7</sup> displayed a postnatal effect with an ongoing significant risk of respiratory symptoms and asthma in households where only the father smoked. This study found that maternal smoking had an even greater impact than paternal smoking, raising a question of SHS dose dependent effect. Thus, both prenatal and postnatal SHS exposures are linked to an increased prevalence of asthma, cough, wheeze and phlegm. Thoughts as to why postnatal SHS exposure would boost asthma are focused on genetic modifications and increases in respiratory infections in early childhood, as well as other pathophysiology, such as inflammation of the respiratory epithelium.<sup>11</sup> More studies are needed to fully elucidate the physiologic mechanism.

Increased asthma severity and poorer outcomes are associated with secondhand smoke. A 1986 study found that asthmatic children who were exposed to SHS had decreased levels of lung function, increased symptom frequency and a fourfold increased responsiveness to inhaled histamine.<sup>12</sup> Asthmatic children with SHS exposure use more asthma medications. Weitzman, et. al. from Boston City Hospital analyzed data from the 1981 Child Health Supplement to

the National Health Interview Survey and found that asthmatic children subjected to SHS were more likely than non-exposed children to require asthma medications.<sup>13</sup> SHS exposure also increases the number of emergency department visits and hospitalizations of asthmatic children.<sup>9,14</sup> The etiology and severity of childhood asthma is an interplay between genetic and environmental factors. Exposing children genetically programmed for asthma to SHS is clearly an avoidable dangerous risk.

Lung growth and development are adversely impacted by SHS. Parental smoking has been shown to negatively impact their children's lung function.<sup>4,6,9</sup> A study of 193 high school athletes found a four-fold increase in the presence of low forced expiratory flow and cough in athletes exposed to SHS as opposed to their unexposed counterparts.<sup>15</sup> Secondhand smoke-exposed children have been shown to enter adulthood with less pulmonary reserve.<sup>16</sup>

The lower respiratory tract is not the only part of a child's body negatively impacted by SHS. A link between SHS and otitis media has long been hypothesized and was first suggested in the literature in 1983.<sup>17</sup> Over the past 25 years, the EPA and the Surgeon General's office both have concluded there is a casual relationship between SHS and otitis media in children.<sup>4,9,18</sup> This includes acute and recurrent otitis media and chronic middle ear effusion. The etiology of this association continues to be under investigation. Health care providers can stress this avoidable trigger to smoking parents and care-givers.

The development of specific allergies to certain pollens, danders, molds, etc., has at times been thought to be increased with SHS exposures. The Surgeon General's 2006 extensive review with meta analysis did not show causality.

Atherogenesis has been linked to childhood SHS exposure. A 2007 study examined changes in endothelial dependent flow-mediated vasodilatory responses of the brachial artery in children exposed to SHS, based on serum cotinine concentrations.<sup>19</sup> Exposure to SHS significantly decreased endothelial function in a dose dependent manner and, thus, potentially increased risk of atherogenesis in the children in this study.

Childhood SHS exposure has been associated with an increased risk of forming dental caries.<sup>20</sup> Prenatal and postnatal SHS exposure also has been suggested to confer an increased chance of developing some childhood cancers, particularly childhood lymphoma, childhood leukemia and childhood brain tumors.<sup>20</sup>

Fetal prematurity and perinatal mortality are increased by maternal direct and secondhand smoke. A 1999 World Health Organization consultative report noted a significant reduction in birth weight as well as an increase in congenital anomalies in infants born to mothers who smoked or were exposed to tobacco smoke.<sup>8</sup> Sudden infant death syndrome (SIDS) is increased in households with mothers who smoke and with SHS from others in the household.<sup>9,21,22</sup> The 2006 Surgeon General's report states that "tobacco smoke exposure is one of the major preventable risk factors for SIDS, and that all measures should be taken to protect infants from exposure to secondhand smoke."<sup>9</sup>

Prenatal and postnatal tobacco smoking and SHS exposure have a vast number of negative effects on children.

### **Biological Markers**

Extensive scientific inquiry has worked to clarify the mechanism of these symptoms and diseases caused by secondhand smoke. Though further study is needed, some interesting information already is available.

Immune responses may play an important role in these negative events. The risk of developing asthma is considered to be heavily influenced by lung immunology. The T-Helper 1 (Th1) pathway, which mediates cellular immunity, and the T-Helper 2 (Th2) pathway, which mediates allergic responses, play critical roles in asthma risk. SHS may advance immunologic development through the Th2 pathway, thus enhancing the genetic development of asthma and chronic respiratory disease.<sup>9</sup>

A recent article linked a specific variant called a single-nucleotide polymorphism (SNP) regulating the gene ORMDL3 on chromosome 17q21 as conferring an increased rate of childhood asthma.<sup>25</sup> An October 2008 article further explored these SNPs and their association with asthma.<sup>26</sup> The authors of this study indicated that these SNPs on chromosome 17q21 increased early-onset asthma, defined as onset of disease prior to four years of age. In addition, exposure to SHS further increased the rate of early-onset asthma. In particular, several SNPs that were not expressed in non-SHS subjected children were exhibited in their SHS exposed counterparts. This study demonstrated that exposure to SHS in a child's youth is associated with changes in genetic expression.

T-cells impact gene regulation, inflammatory cell function, cytokine production and immunoglobulin E (IgE) production. The consequence of SHS on T-cells has been conjectured to increase function and, thus, worsen allergic and respiratory disease. A 2006 UCLA study found an increase

in Th2 cytokine milieu (increase in IL-4, IL-5, IL-13 and decrease in IFN- $\gamma$  production) observed on nasal lavage following human subjects exposure to two hours of SHS.<sup>27</sup> In addition, IgE production was increased in the nasal lavage of human subjects exposed to SHS. These findings support a role for SHS on human allergic phenotypes.

Atopy is often characterized as an IgE mediated allergy test or an increased IgE serum level. Atopy is recognized as a significant risk factor for asthma. An elevation in IgE levels has a correlation with increased bronchial responsiveness. Thus, IgE is felt to play a central role in the pathophysiology of certain forms of asthma.<sup>28</sup>

Several studies have shown that exposure to SHS increases IgE levels. A 1990 study depicted an increase in both IgE and eosinophil counts in boys exposed to SHS.<sup>29</sup> Eosinophil counts in exposed boys were three times higher than in their unexposed counterparts. Bronchial responsiveness, the hallmark of asthma, has long been known to be negatively impacted by SHS.<sup>4</sup> A 2000 study depicted a direct and immediate decrease in FEV1 in smokers with asthma as opposed to their unexposed counterparts.<sup>22</sup> Given the qualitative similarities between mainstream smoke and SHS, these findings are relative to individuals subjected to secondhand smoke.<sup>4</sup> Avoidance studies support this.

Multiple components of SHS may impact a child's health negatively. One example is a bacterial toxin known as lipopolysaccharide. Lipopolysaccharide is a bacterial endotoxin that can cause chronic bronchitis in nonsmoking individuals exposed to respirable agricultural dusts.<sup>30</sup> Hasday, et. al. in 1999 demonstrated that lipopolysaccharide can be found in both mainstream and secondhand tobacco smoke.<sup>30</sup> These authors suggested that lipopolysaccharide may contribute to chronic bronchitis. Another study concluded that lipopolysaccharide may alter allergen challenge responses in rats.<sup>31</sup>

#### **Long Term Effects of Childhood SHS Exposure**

The effects of childhood secondhand smoke also can manifest in adulthood. A 2001 article concluded that childhood contact with SHS confers an increased prevalence of asthma among adult never-smokers.<sup>24</sup> The inhabitants of Orebro, Sweden, were evaluated by questionnaire for exposures, airway symptoms and respiratory history. In never-smokers with childhood SHS exposure, the prevalence of physician-diagnosed asthma was 7.6 percent, versus 5.9 percent in unexposed subjects ( $p=0.036$ ). Thus, SHS increases both childhood and adult asthma.

It has been documented that SHS is a cause of lung cancer

in adulthood.<sup>9</sup> Rates of lung cancer also are higher in children with significant SHS contact. Nonsmoking children and adolescents exposed to 25 or more smoker-years doubled their risk of lung cancer. It has been estimated that 17 percent of lung cancers in nonsmokers can be attributed to childhood SHS exposure.<sup>32</sup> The Surgeon General's report attributes a 20 to 30 percent increase risk of lung cancer from living with smokers.<sup>9</sup> The long-term cancer risk for children subjected to SHS in their youth is a troubling issue.

It has long been demonstrated that active smoking has a casual effect on coronary artery disease.<sup>34</sup> SHS exposure also has been shown to have an association with cardiovascular disease.<sup>9</sup> The effect of childhood SHS exposure on cardiovascular disease has not been as evident as spouses exposed to SHS, but several studies have depicted a link between childhood SHS and increased cardiovascular risk factors.<sup>35-37</sup> Though more research focusing on childhood SHS exposure and development of coronary heart disease is needed, the American Heart Association's Council on Cardiopulmonary and Critical Care has concluded that SHS both increases the risk of heart disease and is a major preventable cause of disease and death.<sup>38</sup> The 2006 report of the Surgeon General reiterates this conclusion, stating a 25 to 30 percent increase in the risk of coronary heart disease.<sup>9</sup>

#### **Impact of Reducing Exposure**

What positive impact can be achieved by decreased SHS exposure? Multiple studies have shown improvement in lung function, FEV1 and symptoms in active smokers that quit smoking.<sup>39</sup> A 2006 study found more specifically that by six weeks of smoking cessation by asthmatic smokers, subjects had improved lung function (measured by FEV1) and a fall in sputum neutrophil counts.<sup>40</sup> Thus, if active smokers can display these changes from reduced usage, what effects are found with reduced SHS contact?

An early case control trial found that in 265 children with asthma exposed to SHS in their homes, symptoms improved by 90 percent when their parents stopped smoking.<sup>41</sup> It also has been shown that biologic markers are affected when smoke inhalation is decreased. A 2000 study found that IgE levels were increased in both active and passive smokers, but, interestingly, found that ex-smokers' levels of IgE were similar to those of non-smokers, regardless of duration of abstinence.<sup>42</sup> Thus, discontinuation or reduction of SHS exposure has multiple and long-lasting benefits.

How is the best way to guide patients to reduce their children's exposure to SHS? Educating patients to the risks

of SHS exposure is paramount. Smokers often know of the negative ramifications of smoking on their health but are unaware of the specifics of the impact of SHS on their children and family members. Education of parents can best be done at specific “teachable moments.”<sup>43,44</sup> A few of these teachable moments include:

- Pregnancy;
- The birth of a child;
- Early childhood (e.g., a child begins to imitate smoking behavior or asks about smoking);
- Acute illness of the child that is related to smoking (e.g., otitis media, pneumonia); and
- Onset or exacerbations of asthma.

A 2003 study used intervention at one of these specific “teachable moments.”<sup>43</sup> Patients were given smoking cessation counseling at the time of a child’s admission to a teaching hospital for a respiratory illness and followed for two months. At the two-month follow-up, results of this study found 49 percent of subjects attempted to quit smoking for at least 24 hours. Twenty-one percent of subjects had not smoked a cigarette in the last 7 days, 38 percent of subjects visited their primary physician and an increased proportion of parents (29 percent to 71 percent) implemented rules prohibiting smoking in the house.<sup>43</sup>

Once a smoker has been educated, counseled and has undergone smoking cessation intervention, the final step must be follow-up. This often is done with return office visits to their health care provider or through patient telephone support, as described in the Task Force on Community Preventative Services’ recommendations regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke.<sup>44</sup> It is imperative to continue to support patients following smoking cessation and to reinforce the positives of continued abstinence from tobacco.

### Summary

Childhood secondhand smoke exposure has significant and long-lasting consequences on the health of many citizens of our state. Nationally, a number of bans and restrictions on tobacco smoking in public and in the workplace have been set in place over the past 30 years. Some states and municipalities have chosen to ban smoking in all public places. Unfortunately, most children are exposed in their own homes. Even with these modifications in laws and attitudes, 45,000 children in the state of South Dakota are exposed to secondhand smoke regularly. As healthcare providers, we have a responsibility to promote a smoke-free environment for all children (and adults) in the state of South Dakota

### REFERENCES

1. Cameron P, Kostin J, Zaks J, Wolfe J, Tighe G, Oselett B, Stocker R, Winton J. The health of smokers’ and non-smokers’ children. *Journal of Allergy* 1969;43(6):336–41.
2. Colley J. Respiratory symptoms in children and parental smoking and phlegm production. *British Medical Journal* 1974;2(912):201–4.
3. Harlap S, Davies A. Infant admissions to hospital and maternal smoking. *Lancet* 1974;1(7857):529–32.
4. U.S. Department of Health and Human Services. The Health Consequences of Involuntary Smoking. A Report of the Surgeon General. Rockville (MD): U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Health Promotion and Education, Office on Smoking and Health, 1986. DHHS Publication No. (CDC) 87-8398.
5. [http://www.cdc.gov/tobacco/data\\_statistics/state\\_data/data\\_highlights/2006/00\\_pdfs/DataHighlights06rev.pdf](http://www.cdc.gov/tobacco/data_statistics/state_data/data_highlights/2006/00_pdfs/DataHighlights06rev.pdf)
6. California Environmental Protection Agency (Cal EPA), Office of Environmental Health Hazard Assessment. Health Effects of Exposure to Environmental Tobacco Smoke. 1997.
7. Cook DG, Strachan DP. Health effects of passive smoking. 3. Parental smoking and prevalence of respiratory symptoms and asthma in school age children. *Thorax* 1997;52:1081-94.
8. WHO. International Consultation on Environmental Tobacco Smoke (ETS) and Child Health. Consultation Report. 1999.
9. U.S. Department of Health and Human Services. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 2006.
10. Young, S, Le Souef, PN, Geelhoed, GC, et al. The influence of a family history of asthma and parental smoking on airway responsiveness in early infancy. *N Engl J Med* 1991; 324:1168-73.
11. Tager IB. Passive smoking-bronchial responsiveness and atopy. *Am Rev Respir Dis* 1988; 138:507-9.
12. Murray AB, Morrison BJ. The effect of cigarette smoke from the mother on bronchial responsiveness and severity of symptoms in children with asthma. *J Allergy Clin Immunol* 1986; 77:575-81.
13. Weitzman M, Gortmaker S, Walker DK, Sobol, A. Maternal smoking and childhood asthma. *Pediatrics* 1990; 85:505-11.
14. Evans D, Levison MJ, Feldman CH, et al. The impact of passive smoking on emergency room visits of urban children with asthma. *Am Rev Respir Dis* 1987; 135:567-72.
15. Tsimoyianis GV, Jacobson MS, Feldman JG, et al. Reduction in pulmonary function and increased frequency of cough associated with passive smoking in teenage athletes. *Pediatrics* 1987; 80:32-6.
16. Cook, DG, Strachan, DP. Health effects of passive smoking-10: Summary of effects of parental smoking on the respiratory health of children and implications for research. *Thorax* 1999; 54:357-66.
17. Kraemer MJ, Richardson MA, Weiss NS, Furukawa CT, Shapiro GG, Pierson WE, Bierman CW. Risk factors for persistent middle-ear effusions: otitis media, catarrh, cigarette smoke exposure, and atopy. *JAMA* 1983;249(8):1022–5.
18. US Environmental Protection Agency (EPA). Respiratory health effects of passive smoking: Lung cancer and other disorders. 1992; EPA/600/006F.
19. Kallio K, Jokinen E, Raitakari OT, et al. Tobacco smoke exposure is associated with attenuated endothelial function in 11-year-old healthy children. *Circulation* 2007; 115:3205-12.
20. Aligne, CA, Moss, ME, Auinger, P, Weitzman, M. Association of pediatric dental caries with passive smoking. *JAMA* 2003; 289:1258-64.
21. Pollack HA. Sudden infant death syndrome, maternal smoking during pregnancy, and the cost-effectiveness of smoking cessation intervention. *Am J Public Health* 2001; 91:432-6.
22. Jensen EJ, Dahl R, Steffensen F. Bronchial reactivity to cigarette smoke; relation to lung function, respiratory symptoms, serum-immunoglobulin E and blood eosinophil and leukocyte counts. *Resp Med* 2000;94:119-127.

Please note: Due to limited space, we are unable to list all 58 references. You may contact South Dakota Medicine at 605.336.1965 for a complete listing.