Respiratory Effects in Children from Exposure to Second-Hand Smoke

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Abstract

Background: The effect of exposure to air pollution during childhood on the development of lung disease in adulthood remains to be defined. A common component of air pollution from fossil fuels, environmental tobacco smoke, and burning of solid fuels such as biomass is particulate matter. The detrimental effects of tobacco on children's health are well known. Nonetheless, the prevalence of secondhand cigarette smoke exposure in the pediatric population has not significantly decreased over time. As early as1974, two articles published in the journal Lancet alerted readers to a possible link between parental smoking & the risk of a lower respiratory illness among infants[1]. Although adverse effects on children from exposure to secondhand tobacco smoke had already been suggested, the association with early episodes of acute chest illness was of immediate and continuing interest because of the suspected long- term consequences for lung growth, chronic respiratory morbidity in childhood, and adult chronic obstructive lung disease [2].

Keywords: Chronic obstructive pulmonary disease (COPD), passive smoking, respiratory disease, asthma, pneumonia.

INTRODUCTION

It is now established beyond doubt that inhaling secondhand smoke as a result of smoking is harmful. There is no safe level of exposure. According to World Bank exposure to secondhand smoke causes an estimated 5% of the global burden of disease, slightly higher than the burden from direct use of tobacco (4%) [3]. Also called passive smoking, environmental tobacco smoking or second-hand smoke (SHS), worldwide exposure to it caused nearly [6], 03,000 premature deaths of non-smokers estimated in 2004. The associated effects include heart disease, lung cancer, severe asthma attacks, sudden infant death syndrome and many others[4]. Early childhood (usually defined as a new born baby until the age of 8 years) is the phase of incredible growth in several aspects: physical, cognitive, social-emotional, and language skills. During the early years, the brain develops quickly and has a high capacity for change, with the foundation set for health and wellbeing throughout life. Therefore, this period is critical. Protecting children from threat, including secondhand smoke exposure, is part of nurturing care that is sensitive to children's health and nutrition needs[5].

Mechanisms of Health Effects from Secondhand Tobacco Smoke

This reviews the biologic impact of secondhand smoke on the respiratory system of the child.

Developmental Vulnerabilities

Pregnant women who smoke expose the fetus to tobacco smoke components during a critical window of lung development, with consequences may be persistent. In infancy and early childhood, the contributions of prenatal vs. postnatal exposure to secondhand smoke are difficult to separate. For children, exposure to secondhand smoke may lead to respiratory illness as a result of adverse effects on immune system and on lung growth and development.

Postnatal lung growth is divided into the first 3 years of life where new alveoli are developed, and later childhood where lung growth occurs by expansion. The effect of very early environmental exposures may therefore be more damaging, or at least qualitatively different, to exposures in later childhood6. The higher breathing rate in children also increases risk of particulate matter-induced lung damage. Young children may also be more vulnerable to oxidative stress–mediated injury to the airway. Oxidative stress is a putative mechanism for both PM-induced lung injury [7], and the development of COPD [8]. Exposure to Secondhand smoke is associated with increased oxidative damage to DNA and lipids. As noted above, MDA can be used as a measure of lipid peroxidation, and children exposed to SHS have been found to have significantly higher circulating levels of MDA and also significantly lower levels of glutathione peroxidase. Concerning antioxidant micronutrients, the evidence for SHS exposure mirrors the evidence for smoking. Compared to non-smokers not exposed to SHS, non-smokers exposed to SHS have significantly reduced circulating concentrations of vitamin C and provitamin A carotenoids, indicating that even low-dose cigarette smoke exposures lower circulating antioxidant micronutrient concentrations. Evidence of lowered circulating antioxidant micronutrient concentrations has also been observed in children of smokers [9, 10, 11]. Data on developmental changes in antioxidant defenses in human airway cells are limited. One of the very few studies that compared mRNA and activity levels of superoxide dismutases (SOD), catalase (CAT), and glutathione peroxidase (GPx) in human adult, neonatal, and fetal lung tissue found conflicting results. Whether a functionally relevant immaturity in pulmonary oxidant defenses is present in young children therefore remains unclear.

Lung Gowth and PM

Entering adulthood with impaired lung function is a nonspecific risk factor for respiratory disease in adulthood. Lower lung function per se is also a risk factor for diseases in childhood that may cause further structural damage to the developing lung. Studies show that older children whose parents smoke get sick more often. Their lungs grow less than children who do not breathe secondhand smoke, and they get more bronchitis and pneumonia. For example, infants with lower lung function in the first weeks of life (i.e., before their first respiratory infection) are at increased risk of developing respiratory syncytial virus (RSV)-bronchiolitis [12]. This primary infection of the bronchioles triggers persistent wheezing, and presumably structural changes in the lung, in a subgroup of infants. Secondhand smoke can trigger an asthma attack in a child. Children with asthma who are around secondhand smoke have more severe and frequent asthma attacks [13]. There is convincing evidence that exposure to PM increases the prevalence of respiratory symptoms in young children.

The tracking of lung function from infancy to early adulthood suggests that damaging exposures in the first years of life may have a disproportionate influence on attainment of maximal lung function in early adulthood [14]. But measuring lung function in infants is difficult, and the association between environmental PM and infant's lung function remains unknown. The most convincing evidence that PM impairs lung growth comes from studies of school-age children, in whom spirometry is easier to perform. The mechanism for PM-mediated effects remains unknown.

Bacterial infection

Increased vulnerability to bacterial infection of the lower respiratory tract is a hallmark of COPD[15]. Recent data from human bronchial epithelial cells exposed to cigarette smoke suggest that this may, in part, be due to suppression of antibacterial host defense [16]. Similarly, in children, there is good

evidence that exposure to PM increases vulnerability to bacterial infection. This association between PM and bacterial infection in children is important because (1) exposure to PM is ubiquitous and (2) infection is common, with 156 million new episodes of pneumonia per year in young children worldwide (151 million of these in the developing world) [17]. Ten percent of these episodes are life-threatening. Also, there is increase in hospital admission in doctor-diagnosed "pneumonia or bronchitis" in children less than 5 years of age. A putative mechanism whereby environmental factors increase vulnerability to pneumococcal pneumonia is via increased nasopharyngeal carriage [18].

Chronic obstructive pulmonary disease (COPD):

Long-term exposure to secondhand smoke (SHS) during childhood increases the risk of chronic obstructive pulmonary disease (COPD) in adulthood, according to a new study. Offspring exposed to parental smoking in childhood had approximately twice the risk of having a carotid atherosclerotic plaque in adulthood than did those with non-smoking parents. However, among offspring of parents who smoked and had a detectable serum cotinine level, which was indicative of poor parental smoking hygiene (e.g., smoking in the presence of the child), the risk of plaque was more than doubled compared with those with no detectable cotinine. These data add to the growing body of evidence proposing that exposure to parental smoking early in life has an irreversible effect on arterial health in adulthood [19, 20]. A putative sequence is that chronic exposure to PM (1) reduces attainment of maximal lung function in childhood, (2) accelerates lung function decline in adulthood, (3) stimulates airway mucus production, and (4) impairs pulmonary innate immunity. If exposure to PM during childhood is high, then symptoms suggestive of COPD will develop early.

Immunologic effects and Inflammation

The development of lung immunophenotype (i.e., the pattern of immunologic response in the lung) is considered to have a key role in determining the risk for asthma, particularly in regard to the T-helper 1 (Th1) pathway (which mediates cellular immunity) and the Th2 pathway (which mediates allergic responses). Secondhand smoke exposure may promote immunologic development along Th2 pathways, thus contributing to the intermediate phenotypes associated with asthma and with a predilection to chronic respiratory disease.

Secondhand smoke effects on T cells may influence gene regulation, inflammatory cell function, cytokine production, and immunoglobulin E (IgE) synthesis. These effects are particularly important to consider in regard to immune system ontogeny and for the subsequent development of allergies in childhood. Researchers have demonstrated that mainstream and side stream smoke condensates selectively suppress the interferon gamma induction of several macrophage functions, including phagocytosis of Ig-opsonized sheep red blood cells, class II major histocompatibility complex expression, and nitric oxide synthesis, which are all representative of effects on immunity [21]. Alterations in antigen presentation may occur not only in the respiratory tract but also in the rest of the body where absorbed toxicants are distributed. Macrophages are potent effector cells for immune responsiveness; suppression of their ability to respond to environmental challenges could have lifelong consequences on immune function.

There are many specific components of secondhand smoke that may adversely affect a child's lung. For example, a bacterial endotoxin known as lipopolysaccharide (LPS) can be detected in both mainstream and side stream tobacco smoke. Some suggested that chronic LPS exposure from cigarette smoke may contribute to the inflammatory effects of secondhand smoke. Other studies show that LPS exposure may alter responses to allergen challenge[22].

DISCUSSION

Children whose parents are smokers are at increased risk of SHS exposure in the home. There was also some evidence that children whose parents held more negative attitudes towards SHS were less likely to be exposed. Associations were strongest for parental cigarette smoking status; compared to children of non-smokers, those whose mothers or both parents smoked were between two and 13 times more likely to be exposed to SHS at home. The best way to prevent child SHS exposure in the home is by encouraging smoking parents to quit.

To improve child health, we therefore need interventions targeted at adults: preventing them

from taking up smoking, or helping them quit. One approach is that of individual prevention, which attempts to change parents' attitudes and educate them through individual counselling, education or smoking cessation programs. A second approach is structural, which depends on changing the environment and organizational structures by methods such as economic incentives, reducing the availability of cigarettes, tobacco-free advertising, or smoke-free public spaces 23.

Individual prevention remains important in clinical practice. Combining medication and counselling by a physician doubles the chance that individual smokers will quit, and is more cost-effective than other clinical interventions [19]. However, absolute rates of quitting remain low and smoking cessation programs, including nicotine replacement therapy; electronic cigarettes and nicotine vaccines have small effects 24, 25.

In line with the findings of this review, sociodemographic characteristics are often linked to health inequalities. Low SES is frequently reported to be associated with poorer health outcomes, health morbidity and mortality. There was also some evidence that children whose parents were single, separated or divorced were at increased risk of SHS exposure in the home. The greatest observed risks in this review were for children whose mothers 26 or both parents were smokers, which strongly suggests that the best way to reduce child SHS exposure in the home is for parents who smoke to quit. This finding has implications for younger children of pre-school age, who spends an increased proportion of their time at home with parents compared to older, school-aged children. In a recent review 27, the effectiveness of any one interventional approach to reduce children's SHS exposure was not conclusively demonstrated and as such there remains a need for novel, evidence-based interventions which are sensitive to both the context in which smokers live and smokers' environments. The Theory of Reasoned Action argues that interventions designed to change beliefs and attitudes can influence intentions and subsequent behavior across a range of health behaviors 28. Interventions targeting attitudes towards SHS by supporting parents to recognize the benefits of protecting their children from SHS may therefore be useful to promote smoke-free homes.

Changing attitudes alone may not be sufficient to change behavior. A combined approach that targets attitudinal change and provides practical context specific advice to parents, for example balancing child safeguarding with smoking outside of the home or negotiation with other household smokers, may be helpful.

CONCLUSION

Childhood respiratory disease covers a spectrum of diseases and underlying pathogenic mechanisms that include infection, prenatal alterations in lung structure, inflammation, and allergic responses. There is a potential for secondhand smoke to contribute over the long term to the development of respiratory disease through altered organ maturation and immune function. Mechanisms underlying the adverse health effects of secondhand smoke vary across the phases of lung growth and development, extending from the in utero period to the completion of lung growth in late adolescence. The long-term effects of secondhand smoke are a field of ongoing research. These effects may vary among individuals because of individual genetic susceptibilities and gene-environment interactions.

BIBLIOGRAPHY

1. Colley JRT, Holland WW, Corkhill RT. Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. Lancet. 1974;2(7888):1031–4. [PubMed]
2. SAMET JM; TAGET IB; SPEIZER FE AMERICAN (THE) REVIEW OF RESPIRATORY DISEASES; ISSN 0003-0805; USA; DA. 1983; VOL. 127; NO 4; PP. 508-523; BIBL. 166 REF.
3. Economic of tobacco control. The World Bank Group. 1999.
4. Öberg M, Jaakkola MS, Woodward A, Peruga A, Prüss-Ustün A. Worldwide burden of disease from exposure to second-hand smoke: A retrospective analysis of data from 192 countries. Lancet 2011;377:139-46.

5. World Health Organization Early child development. https://www.who.int/topics/early-child-development/en/. Accessed January 26, 2020.
6. Silverman M, Kuehni CE. Early lung development and COPD. Lancet 2007;370:717–719.
7. Donaldson K, Stone V, Borm PJ, Jimenez LA, Gilmour PS, Schins RP, Knaapen AM, Rahman I, Faux SP, Brown DM, et al. Oxidative stress and calcium signaling in the adverse effects of environmental particles (PM10). Free RadicBiol Med 2003;34:1369–1382.
8. Ciencewicki J, Trivedi S, Kleeberger SR. Oxidants and the pathogenesis of lung diseases. J Allergy ClinImmunol 2008;122:456–468, quiz 69–70.
9. Wilson KM, Finkelstein JN, Blumkin AK, Best D, Klein JD. Micronutrient levels in children exposed to secondhand tobacco smoke. Nicotine & Tobacco Research. 2011;13(9):800–808. [PubMed]
10. Yilmaz G, IsikAgras P, Hizli S, Karacan C, Besler HT, Yurdakok K, Coskun T. The effect of passive smoking and breast feeding on serum antioxidant vitamin (A, C, E) levels in infants. ActaPaediatrica. 2009;98(3):531–536. [PubMed]
11. Zalata A, Yahia S, El-Bakary A, Elsheikha HM. Increased DNA damage in children caused by passive smoking as assessed by comet assay and oxidative stress. Mutation Research. 2007;629(2):140–147. [PubMed]
12. U.S. Department of Health and Human Services. The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon Generalexternal icon. Atlanta: 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 [accessed 2017 Jan 11]
13. U.S. Department of Health and Human Services. The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General. Atlanta: 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, 2014 [accessed 2017 Jan 11].
14. Stern DA, Morgan WJ, Wright AL, Guerra S, Martinez FD. Poor airway function in early infancy and lung function by age 22 years: a non-selective longitudinal cohort study. Lancet 2007;370:758–764.
15. Sethi S, Murphy TF. Infection in the pathogenesis and course of chronic obstructive pulmonary disease. N Engl J Med 2008;359:2355–2365.
16. Herr C, Beisswenger C, Hess C, Kandler K, Suttorp N, Welte T, Schroeder JM, Vogelmeier C. Suppression of pulmonary innate host defence in smokers. Thorax 2009;64:144–149.
17. Rudan I, Boschi-Pinto C, Biloglav Z, Mulholland K, Campbell H. Epidemiology and etiology of childhood pneumonia. Bull World Health Organ 2008;86:408–416.
18. Cardozo DM, Nascimento-Carvalho CM, Andrade AL, Silvany-Neto AM, Daltro CH, Brandao MA, Brandao AP, Brandileone MC. Prevalence and risk factors for nasopharyngeal carriage of Streptococcus pneumoniae among adolescents. J Med Microbiol 2008;57:185–189.
19. Juonala M, Magnussen CG, Venn A, Gall S, Kähönen M, Laitinen T, Taittonen L, Lehtimäki T, Jokinen E, Sun C, Viikari JS, Dwyer T, Raitakari OT. Parental smoking in childhood and brachial artery flow-mediated dilatation in young adults: the Cardiovascular Risk in Young Finns study and the Childhood Determinants of Adult Health study.ArteriosclerThrombVasc Biol. 2012; 32:1024–1031. doi: 10.1161/ATVBAHA.111.243261.
20. Gall S, Huynh QL, Magnussen CG, Juonala M, Viikari JS, Kähönen M, Dwyer T, Raitakari OT, Venn A. Exposure to parental smoking in childhood or adolescence is associated with increased carotid intima-media thickness in young adults: evidence from the Cardiovascular Risk in Young Finns study and the Childhood Determinants of Adult Health study.Eur Heart J. 2014; 35:2484–2491. doi: 10.1093/eurheartj/ehu049
21. Braun KM, Cornish T, Valm A, Cundiff J, Pauly JL, Fan S. Immunotoxicology of cigarette smoke condensates: suppression of macrophage responsiveness to interferon γ Toxicology and Applied Pharmacology. 1998;149(2):136–43. [PubMed]
22. Tulić MK, Wale JL, Holt PG, Sly PD. Modification of the inflammatory response to allergen challenge after exposure to bacterial lipopolysaccharide. American Journal of Respiratory Cell and Molecular Biology. 2000;22(5):604–12. [PubMed]
23. Frieden TR, Bloomberg MR How to prevent 100 million deaths from tobacco. Lancet 2007; 369: 1758–1761
24. Hartmann-Boyce J, Cahill K, Hatsukami D, et al.Nicotine vaccines for smoking cessation. Cochrane Database Syst Rev 2012; 8: CD00
25. Stead LF, Perera R, Bullen C, et al.Nicotine replacement therapy for smoking cessation. Cochrane Database Syst Rev 2012; 11: CD000146.
26. Raisamo SU, Doku DT, Heloma A, Rimpelä AH (2013) Persistence of socioeconomic differences in adolescents' environmental tobacco smoke exposure in

Finland: 1991–2009. Scandinavian journal of public health: 1403494813514301.
27. Family and carer smoking control programmes for reducing children's exposure to environmental tobacco smoke. Cochrane Database of Systematic Reviews.2014:Issue 3
28. Ajzen I, Albarracin D (2007) Chapter 1. Predicting and Changing Behavior: A Reasoned Action Approach. In: Ajzen I, Albarracin D, Hornik R, editors. Prediction and Change of Health Behaviour, Applying the Reasoned Action Approach. New Jersey: Lawrence Erlbaum.