A Review on Health Effects of Ambient Air Pollution on Children

Nisarg Patel[*1], Louis Hemple[*2], Ralph Tripp[*3]

[*1]College of Engineering, University of Georgia, Athens, GA, 30606, USA

[2]Children's Medical Group, 6918 McGinnis Ferry Rd, Suwanee, GA, USA

[3]InfectiousDiseases, University of Georgia,Animal Health Research Center, Athens, GA, 30602, USA

[*1]1nisarg@uga.edu;[2]4hempels@comcast.net;[3]ratripp@uga.edu

***Abstract*-Children seem to be particularly vulnerable to harmful effects of ambient pollution due to higher inhalation ratio than adults as well as due to their lungs not having a fully developed defense system against ambient pollution. Increased prevalence of respiratory track symptoms, allergen sensitization, worsening of asthma, and increased use of anti-asthma medications has been linked to high ambient air pollution episodes. Lung growth and development has also been shown to be impaired in children due to high exposures. The harmful effects of ambient pollution can be seen in fetuses from the exposures of their mothers. A study has even linked neonatal morbidity to the concentration of pollutants in the atmosphere.**

Keywords-Ambient Air Pollution; Children; Health; Development

I. INTRODUCTION

With the start of the industrial revolution, the western world embarked on an unexpected global experiment lasting the past two centuries. Since the industrial revolution, the density of primary ambient air polluters such as Carbon Dioxide, Carbon Monoxide, and Sulfur Dioxide has drastically increased. Post-industrial value of CO2 levels has increased from 280ppm in the 1800s to about 390ppm in about 200 years [1].

The respiratory track comes into close contact with approximately 10,000 liters of ambient air and its components on a daily bases [2]. Urbanization and industrialization of our society combined with increased vehicular emissions, both in developed and developing nations, have led to a significant decrease in the quality of air we breathe. The pollutants enter along with air into

Table I NATIONAL AMBIANT AIR QUALITY STADARDS, USA

Pollutant	Primary Standards
Ozone	
1 hr average	0.12 ppm (235 μg/m^3)
8 hr average	0.008 ppm (157 μg/m^3)
Lead	
Quarterly average	1.5 μg/m^3
Carbon Monoxide	
8 hr average	9 ppm (10 μg/m^3)
1 hr average	35 ppm (40 mg/m^3)
Nitrogen Dioxide	
Annual mean	0.053 ppm (100 μg/m^3)
PM_{10}	
Annual mean	50 μg/m^3
$PM_{2.5}$	
Annual mean	15 μg/m^3
Sulfur Dioxide	
Annual mean	0.003 ppm (80 μg/m^3)

our lungs and get retained in the lungs. These substances not only cause localized damage to the lungs but also enter the systemic circulation through the alveolar capillary basement membrane to cause wide spread problems[2]. Although it has been over 4 decades since the publication of the Clean Air Act in 1970, the air in many parts of the United States is far from clean. Since the Clean Air Act, the ambient air quality has greatly improved; however, progress has slowed in recent years. This air pollution has been shown to have adverse health effects on humans. When the Clean Air Act was last amended in 1990, it required the EPA to set National Ambient Air Quality Standards for pollutants considered harmful to public health or the environment. In 2002, approximately 146 million Americans where living in areas were monitored air failed to meet the 1997 National Ambient Air Quality Standards for at least 1 of the 6 criteria air pollutants shown in Table I [3].

Infants and children are amongst the most susceptible groups to quality of the air. Eighty percent of alveoli isformed postnatally, and changes in the lungs continue through adolescence [4].During the early post neonatal period, the developing lung is highly susceptible to damage from exposure to environmental toxicants. Studies have also showed significant damage to the fetus from mother's exposure to low quality ambient air [5-9]. Ambient air quality has become an issue of great concern for child health professionals, epidemiologists, environmental policy makers, and families.

II. WHY INFANTS AND CHILDREN ARE MORE SUSCEPTIBLE TO AMBIENT AIR POLLUTION

Infants and Children seem to be affected more harshly by air pollution than adults. Biologically, the peripheral airways are more susceptible to polluted air, which leads to greater airway obstruction than in adults [11]. At birth, we have approximately 24 million alveoli, which increases to 267 million by 4 years and 600 million by adulthood [12]. During this growth period, complex and precisely timed sequences of chemical messages are exchanged in the body to facilitate lung growth. Many of the pollutants found in our air chemically interfere with the natural processes in the body. Infants and children, at this stage in life, have not acquired the same ability to metabolize and detoxify their body from harmful air pollutants that adults possess. Along with different detoxification abilities, the permeability of growing alveolar epithelium is much higher than adults. This leads to a higher concentration of harmful substances making their way into the circulatory system for children. In addition, children have a lower surface area to body weight ratio leading to higher oxygen consumption. This means more air will have to pass through a smaller surface area causing the concentration of particles trapped in the lung tissue to be much higher for children.

Along with being chemically different, children are physically different from adults. Children perform a much greater level of physical activity, thereby increasing the intake of ambient air pollutants [12]. During exercise, there is almost a five-fold increase in partials deposited in the lungs compared to during rest [12]. Higher activity levels coupled with more time spent outside, especially during summer, greatly increase the susceptibility of children to ambient air pollution.

III. RELATIVE CONTRIBUTION OF VARIOUS AIR POLLUTANTS ON CHILDREN

Evidence on the implications of adverse health effects comes from a wide array of sources, including observational epidemiology, controlled human exposures to pollutants and animal toxicology. Overall analysis demonstrated that CO_2, NO_2, and CO seem to have very similar health effects with NO_2 having the most severe health effect due to its extent of further transformations [13]. NO_2 is subjected to further atmospheric transformations that can lead to the formation of O3 and other strong oxidants. Interpretation of evidence on NO_2 exposures is further complicated by the fact that most in most urban locations, nitrogen oxide (the precursor of nitrogen dioxide) is emitted primarily by motor vehicles [13]. Meta-analysis on mortality showed a strong (7.6% increase) relationship between exposures of NO2 to mortality [14].

Growing evidence seems to demonstrate that exposure to ambient particulate matter that is 2.5 micrometers or less is associated with increases in respiratory symptoms and prevalence of asthma in children. Research suggests that PM seems to have more of a long term effect such as asthma compared to CO_2, CO, or NO_2 which seem to have a shore term impact such as shortness of breath, and coughing [15]. When dealing with high concentrations, the chemical based pollutants have acute effect such as death while PM can lead to lung cancer.

IV. EFFECTS OF AMBIENT AIR POLLUTION ON CHILDREN'S LUNG FUNCTION AND OTHER CHANGES

Ambient air pollution is known to have a significant effect on the lung function of children, more so than on adult's lung function due to the still developing stage that the children's lungs are in. A study conducted in Taiwan to assess the association between air pollutants exposure and lung function of junior high school students in a mass screening program had a sample size of 10,396 students [16]. During the study, forced vital capacity (FVC) and forced expiratory flow in 1second (FEV_1) in association with daily ambient concentrations were assessed by regression models [16]. Measurements were taken eight times for each student and the best results were chosen for further analysis. Measured values for the five pollutants analyzed were as follows: SO_2 (1.3-5.2ppb), CO (0.6-1.4ppb), O_3 (5.4-27.4ppb), PM_{10} (28-81μg/m^3), and NO_2 (19.3-41.7ppb). The lower range represents Q25th while the upper range represents Q75th. The results showed FVC had a significant negative association with short-term exposure to O_3 and PM_{10} measured on the day of spirometry testing. An increase in 1-ppm CO was associated with the reduction in FVC for 69.8mL or in FEV_1 for 73.7mL [16]. The study concluded that the short-term exposure to O_3 and PM_{10} was associated with reducing FVC and FEV_1. The study also found that CO and SO_2 exposure had a strong one day lag effect on FVC and FEV_1.

One of the earliest studies ever conducted to look for an association between air pollution episodes and pulmonary function in children was conducted by the Harvard University School of Public Health in Ohio in 1978 and 1979. The results show a 2% decrease in the FVC following a total suspended particulates (TSP) and SO_2 concentrations exceeding the standards [17]. The Forced Expired Volume in .75s ($FEV_{0.75}$) also showed a 4% decrease [17]. Following studies that looked at asthmatic children over a period of several months studied 71 children (aged 5 to 7 yrs) with mild asthma who resided in the northern part of Mexico City [18]. In Mexico City, PM10 and ozone frequently exceeded the standards set and therefore they were the main pollutants analyzed [18]. An increase 20μg/m^3 of PM10 from the standards showed an 8% increase in lower respiratory illness (LRI) among children on the same day (95% confidence interval = 1.04-1.15) [18]. A 50 ppb increase in ozone was correlated with a 9% increase in LRI on the same day. Other experiments have demonstrated similar decrease in the FEV and LRI in correlation to pollutant increase over the standards set. Current levels of ambient air pollution have shown to have adverse effects on lung development leading to a chronic decrease in lung function of children.

Children living in areas of high ambient levels of air pollutants show evidence of ulta-structural changes in airway mucosa. Nasal biopsies taken from children from Mexico city, a city where children are exposed to high levels of ambient pollution(0.15 to 0.30ppm for 6 to 90d), were compared to children from Veracruz, a city with comparatively low ambient air pollution (0.06-0.12ppm for 6 to 90d) [19]. The biopsies from children in Mexico City show histopathology alterations: marked changes in ciliated and goblet cell populations, basal cell hyperplasia, squamous metaplasia, and mild dysplasia [19]. The biopsies also showed presence of particulate matter in the intercellular spaces [19]. These changes are responsible for weaker mucociliary defense mechanisms and lower ability to protect lower respiratory tract.

V. EFFECTS OF AMBIENT AIR POLLUTION ON THE FETUS AND NEONATES

Ambient air pollution has been demonstrated to increase health complications even before birth. The placenta is the site of exchange of oxygen and nutrients between the mother and fetus. Pregnant mothers exposed to high levels of ambient air pollutants show presence of pollutants in their blood, which then enters the circulatory system of the fetus through the placenta and umbilical cord [5]. Exposure to high levels of ambient air pollutants has been associated with low birth weight, premature birth, intrauterine growth retardation, and increased perinatal morbidity.

Results of a study conducted to investigate whether an allergen to which mothers are exposed to throughout pregnancy, house dust mite antigen Der P1, is exposed to the fetus show that Der p 1 was detectable in 56% of fetal blood from women undergoing amniocentesis at 16-17 weeks' gestation [5]. This study demonstrates just how susceptible unborn children can be to the mother's environment.

A nationwide study conducted to evaluate the association of maternal air pollution exposure with markers of placental growth and function was carried out among 7,801 pregnant women in Netherlands [20]. Modeling techniques were used to estimate the concentration of different pollutants at the home address of each pregnant woman. Various markers such as soluble fms-like tyrosine kinase 1 (sFlt-1), a protein that decreases the placental growth factors (PLGF), were analyzed in maternal blood during first and second trimester and in fetal cord blood after delivery [20]. Results indicated high PM10 (>180ug/m^3, 24hr) and NO (>0.07 ppm) concentrations are associated with an increase in sFlt-1 (which means lower PLGF) [20].

A study conducted between 1988 and 1991 followed all pregnant women living in four residential areas of Beijing from early pregnancy until delivery [7]. Multiple liner regression and logistic regression were used to estimate the effects of air pollution on birth weight. The results show a significant exposure response relationship between maternal exposures to sulfur dioxide and total suspended particles during the third trimester of pregnancy and infant birth weight [7]. The estimated reduction in birth weight was 7.3g for each 100 mg/m^3 increase in sulfur dioxide and 6.9g reduction for each 100 mg/m^3 increase in total suspended particles. Similar results of increased preterm delivery, low infant birth weights, increased perinatal morbidity have been shown in Korea[9], Canada[18], and USA[12].

Neonates, 0-28 days of age, have a unique spectrum of morbidity including presentations such as acute life-threatening events, and respiratory distress syndrome. To better understand the contribution of pollution to neonatal morbidity, a study measured the association between daily respiratory hospitalizations and daily concentrations of ambient air pollution. The results show an increase in the ambient air pollutants resulted in ~10% increase in hospitalizations of neonates [21]. The study concludes that neonates are experiencing adverse effects of air pollution at current levels in Canada, and that accounts for a significant portion of hospitalizations in this subgroup [21].

VI. EFFECTS OF AMBIENT AIR POLLUTION ON CHILDREN

Many studies have shown a relationship between exposures to high levels of air pollution and to respiratory symptoms like asthma in children [22-29]. A Dutch study was conducted to see the relationship between air pollution from living close to heavy traffic and respiratory symptoms in schoolchildren [25]. The study found that children living close to motorways with high truck traffic showed increased respiratory symptoms [25]. The study also found a higher sensitization to pollen in relation to high truck traffic measured by serum immunoglobulin E and skin prick tests [25].

Another study in Germany investigated symptoms of wheezing and allergy in association with traffic density in 13-14 year old school children [27]. The study analyzed children who lived in areas with rare, frequent and constant flow of truck traffic. The results showed 29%, 58%, and 57% increased prevalence of wheezing respectively [27]. Also, the prevalence of allergic rhinitis increased by 20%, 35%, and 69% respectively [27]. These data suggest that living in an area with high levels of ambient pollution increases the risk of developing asthma and allergic rhinitis.

VII. CONCLUSION

A growing amount of researches suggest a connection between exposure to ambient air pollution and an increased health risks in children. The pollutants most particularly harmful seem to be NO_2, ozone, PM_{10}, and $PM_{2.5}$. Children seem to be particularly vulnerable to harmful effects of ambient pollution due to higher inhalation ratio than adults as well as due to their lungs not having a fully developed defense system against ambient pollution. Increased prevalence of respiratory track symptoms, allergen sensitization, worsening of asthma, and increased use of anti-asthma medications has been linked to high ambient air pollution episodes. Lung growth and development has also been showed to be impaired in children due to high exposures. The harmful effects of ambient pollution can be seen in fetuses from the exposures of their mothers. A study has even linked neonatal morbidity to the concentration of pollutants in the atmosphere. Physicians can significantly reduce child morbidity as well as improve children's health by advising families to minimize the exposure of children to ambient air pollution. Preventive steps like living 2-5 miles from heavily used highways or living away from densely populated cities could play a significant role in a child's health. Also, supplements of antioxidants such as vitamin E and Vitamin C, have been shown to have beneficial role in preventing deterioration in lung function. Broader changes could be achieved by educating policymakers about the health effects of ambient air pollution on children.

REFERENCE

[1] Hofmann, D. J., Butler, J. H., & Tans, P. P. "A new look at atmospheric carbon dioxide". *Atmospheric Environment*, *43*(12), 2084-2086; 2009.

[2] S. Sundeep, "Health Effects of Ambient Air Pollution in Children"; *Pediatric Respiratory Reviews*; Vol. 8; pp. 275-280; 2007.

[3] Committee on Environmental Health. "Ambient Air Pollution: Health Hazards to Children"; *Pediatrics*; Vol. 114; pp. 1699-1707; 2004.

[4] RR Dietert, RA Etzel, D Chen, et al.; "Workshop to identify critical windows of exposure for children's health: Immune and respiratory systems work group summary"; *Environmental Health Prospect*; Vol. 108; pp. 483-490; 2000.

[5] JA Holloway, JO Warner, GH Vance; "Detection of House-dust-mite allergen in amniotic fluid and umbilical-cord blood"; *The Lancet*; Vol. 356; pp. 1900-1902; 2000.

[6] X Xu, H Ding, X Wang; "Acute effects of total suspended particles and sulfur dioxides on preterm delivery: a community-based cohort study"; *Arch Environ Health*; Vol. 50; pp. 407-415; 1995.

[7] X Wang, H Ding, L Ryan, X Xu; "Association between air pollution and low birth weight: A community-based study"; *Environ Health Perspect*; Vol. 105; pp. 514-520; 1997.

[8] M Bobak, DA Leon; The effect of air pollution on infant mortality appears specific for respiratory causes in the post-neonatal period; *Epidemiology;* Vol. 10; pp. 666-670; 1999.

[9] EH Ha, YC Hong, BE Lee, et al. ; Is air pollution a risk factor for low birth weight in Seoul?; *Epidemiology*; Vol. 12; pp. 643-648; 2001.

[10] L. Trasande, GD. Thurston; The role of air pollution in asthma and other pediatric morbidities; *Journal of Epidemiology and Community Health*; Vol. 115; pp. 869-699; 2005.

[11] Yap, P., Gilbreath, S., Garcia, C., Jareen, N., & Goodrich, B. The Influence of Socioeconomic Markers on the Association between Fine Particulate Matter and Hospital Admissions for Respiratory Conditions Among Children. *American Journal of Public Health*, *103*(4), 945-e8. 2013.

[12] JF Rogers, SJ Thompson, CL Addy, et al.; Association of very low birth weight with exposures to environmental sulfur dioxide and total suspended particulates; *American Journal of Epidemiology*; Vol. 151; pp. 602-613; 2000.

[13] World Health Organization. Health Aspects of Air Pollution. *World Health Organization*. Bonn, Germany. 2003.

[14] Shang, Y., Sun, Z., Cao, J., Wang, X., Zhong, L., Bi, X., & ... Huang, W. Systematic review of Chinese studies of short-term exposure to air pollution and daily mortality. *Environment International*, *54*100-111. 2013.

[15] Yap, P., Gilbreath, S., Garcia, C., Jareen, N., & Goodrich, B. The Influence of Socioeconomic Markers on the Association between Fine Particulate Matter and Hospital Admissions for Respiratory Conditions Among Children. *American Journal of Public Health*, *103*(4), 945-e8. 2013.

[16] YK. Chang, CC. Wu, LT. Lee, RS. Lin, YH. Yu, YC. Chen; The short-term effects of air pollution on adolescent lung function in Taiwan; *Chemosphere*; Vol. 87; pp. 26-30; 2011.

[17] DW. Dockery, JH. Ware, BG. Ferris, et al. Effects of air pollution on the respiratory health of asthmatic children living in Mexico City; *Journal of the air pollution control association*; Vol. 32; pp. 937-942; 1982.

[18] I. Romieu, F. Meneses, S. Ruiz, et al.; Effects of air pollution on the respiratory health of asthmatic children living in Mexico City; *American Journal of Respiratory and Critical Care Medicine*; Vol. 32; pp. 937-942; 1982.

[19] Caldero´n-Garciduen˜as L, Valencia-Salazar G, Rodrı´guez-Alcaraz A, et al; Ultrastructural nasal pathology in children chronically and sequentially exposed to air pollutants; *American Journal of Respiratory Cell and Molecular Biology*; Vol. 24; pp. 132-138; 2001.

[20] Van den Hooven, E. H., Pierik, F. H., de Kluizenaar, Y., Hofman, A., van Ratingen, S. W., Zandveld, P. J., & ... Jaddoe1, 3. V. Air Pollution Exposure and Markers of Placental Growth and Function: The Generation R Study. *Environmental Health Perspectives*, *120*(12), 1753-1759. 2012.

[21] RE. Dales, S. Cakmek, MS. Doiron; Gaseous Air pollutants and Hospitalization for Respiratory Disease in the Neonatal Period; *Environ Health Prospect*; Vol. 114; 1751-1754; 2006.

[22] M. Wjst, P. Reitmeir, S. Dold, et al; Road traffic and adverse effects on respiratory health in children; *British Medical Journal*; Vol. 307; pp. 596-600; 1993.

[23] SK. Weiland, KA. Mundt, A. Ruchkmann, et al; Self-reported wheezing and allergic rhinitis in children and traffic density on street of residence*; Annals of Epidemiology*; Vol. 4; pp. 243-247; 1994.

[24] G. Ciccone, F. Forastiere, N. Agabiti, et al. Road traffic and adverse respiratory effects in children; *Occupational & Environmental Medicine*; Vol. 55; 771-778; 1998.

[25] J. Edwards, S. Walters, RK. Griffiths; Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom; *Arch Environ Health*; Vol. 49; pp. 223–227; 1994.

[26] B. Brunekreef, NA. Janssen, J. de Hartog , et al; Air pollution from truck traffic and lung function in children living near motorways; *Epidemiology*; Vol. 8: pp. 298–303; 1997.

[27] NA Janssen, BBrunekreef, van Vliet P et al; The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren; *Environ Health prospect*; Vol. 111: pp. 1512–1518; 2003.

[28] SL Lee, WH Wong, Lau YL; Association between air pollution and asthma admission among children in Hong Kong; *Clinical and Experimental Allergy*; Vol. 36; Vol. 1138–1146; 2006.

[29] T Behrens, DTaeger, WMaziak, et al.; Self-reported traffic density and atopic disease in children: Results of the ISAAC Phase III survey in Muenster, Germany; *Pediatric Allergy and Immunology*; Vol. 15; pp.331–339; 2004.