American journal of Rhinology & Otolaryngology
ISSN 2688-6243

Research Article Open Access

Respiratory diseases in the hands of air pollution.

Jose Luis Treviño Gonzalez[1], MD. PhD, Nadia G. Jasso Ramirez[1], MD, Amalia Gonzalez Larios[1], MD, Eber A. Medina Estrada[1]MD.

[1] Department of Otolaryngology and Head and Neck Surgery, University Hospital "Dr. Jose Eleuterio Gonzalez", University of Nuevo Leon, Mexico.

***Corresponding Author:** Jose Luis Treviño-Gonzalez, Department of Otolaryngology and Head and Neck Surgery, University Hospital "Dr. Jose Eleuterio Gonzalez", University of Nuevo Leon, Francisco I. Madero and Jose E. Gonzalez N/N, Colonia Mitras Centro Monterrey, Nuevo Leon, Mexico, 64460 Mexico. Phone: +52 8183332917, Fax: +52 8183334299, Email: jose.trevinog@uanl.mx

Citation: Respiratory diseases in the hands of air pollution: Am J Rhin and Otolo. 2019; 2(2): 0107.

Submitted: 17 October 2019; **Approved:** 20 Occtober 2019; **Published:** 22 October 2019

Abstract:

Ambient air pollution has been intensifying for decades, which is one of the most common worldwide environmental problems, especially in developing countries. Air pollution with high ambient fine particulate matter (PM2.5) concentration has become an urgent public health issue in recent years(1). Previous studies have indicated that increases in ambient PM2.5 concentration corresponded to adverse health impacts, being cardiovascular and respiratory diseases the more commonly related to PM2.5 exposure (3,4).

Children are the main vulnerable groups to air pollution, particularly, due to their high breath rate, undeveloped lung, and more time exposure outdoors. Those exposed to traffic-related air pollutants are at increased risks of asthma, allergic rhinitis, allergic sensitization, and reduced lung function (9,10).

The World Health Organization estimated that approximately 250,000 deaths annually between 2030 and 2050 could be due to climate change as a consequence of the pollution we are actually living with (7). The predominant sources of ambient air pollution vary according to country. A better understanding of the mechanisms of air pollution and their links with upper airway disease will contribute to disease prevention and pollution control action.

Keywords: Air pollution, particulate matter, health, airway, disease, children.

Highlights:

- Air pollution is a common worldwide environmental problem in developing countries.
- PM2.5 is an air pollutant that is a concern for people's health when levels are high.
- The development of infectious airway pathologies are influenced by climate variables.
- Children are the main vulnerable group to air pollution.
- Otitis media, allergic rhinitis, and asthma are the most prevalent diseases among children.
- Those exposed to traffic-related air pollutants have an increased risk of airway diseases.

1. Introduction

Ambient air pollution has been intensifying for decades, which is one of the most common worldwide environmental problems, especially in developing countries. Air pollution with high PM2.5 (fine particulate matter, of aerodynamic diameter ≤ 2.5 μm) concentration has become an urgent public health issue in recent years.

The principal pollutants include particulate matter, ozone (O3), sulfur dioxide (SO2), and nitrogen dioxide (NO2). Air pollutants constitute a toxic mixture of particles and gases

emitted in large quantities from many different combustion sources, including vehicles and factories(1). Indoor pollutants include smoke from tobacco, cooking, and the burning of wood and other material in stoves and fireplaces; dust particles resuspended during cleaning; and outdoor particles that infiltrate the indoor environment(2).

Previous studies have indicated that increases in ambient PM2.5 concentration corresponded to adverse health impacts, including hospital visits for respiratory diseases and cause-specific disease, development of upper airway disease and respiratory symptoms complains, such as increased mortality and morbidity(3,4). Climate change can affect human health, especially when infectious diseases are concerned(5). The present review describes air pollutants that contribute to airway disease, the diseases of concern, and the findings and of prior studies.

1.1 Particulate matter (PM)

Air pollution is the presence in the air of material that has the capacity to affect living beings in some way, it has become an important situation due to the increasing levels and the high and inevitable exposure to which we humans are in contact. Globally, millions of deaths are associated with the complications of diseases in which pollution took a fundamental role, most often they were associated with fine particulate material PM2.5 and O3 (6). The presence of PM2.5 has a greater impact on health than O3 or more common pollutants such as carbon monoxide (CO). PM2.5 consists of a heterogeneous mixture of liquid and solid particles suspended in the air that continuously vary in concentration, size, and composition. The main components of these particles include inorganic compounds (sulfates, nitrates), organic compounds (hydrocarbons), biological compounds (allergens, endotoxins) and metals (iron, copper, nickel)(7). The main classification of PM consists of its aerodynamic diameter who is the one who confers the ability to transport by air, pass through the filters and continue to deposit in the airways; This is categorized in coarse particulate material with a diameter of up to 10 μm or in fine particulate material with a diameter smaller than 2.5 μm. Outdoors the source of these compounds comes from road and agricultural dust, construction sites, mining, related activities and mainly from vehicle emissions. Regarding indoor air, they are obtained from activities such as cooking, interior remodeling, cleaning, tobacco consumption, etc.

The effects of exposure to particulate matter directly depend on the characteristics of individuals such as developing organs, type of breathing, frequency, and respiratory capacity. For a better understanding we have that the smaller the particles, the easier it will be to inhale and deposit in the airway, during nasal breathing, the villi and mucus have the function of filtering the air and trapping particles larger than 10μm, thanks to the fact that the PM10 tends to settle quickly only reaches structures such as nose, larynx, trachea, and bronchi, to which our body reacts with defense mechanisms such as coughing and sneezing; while PM2.5 tend to settle at the level of bronchioles and alveoli, interfering with the gas exchange and probably leading its way to the bloodstream, giving them even greater capacity to cause disease(8).

1.2 Allergic response

Schoolchildren aged 6–14 years who are exposed to traffic-associated air pollutants (TAP) are at increased risks of asthma, allergic rhinitis, allergic sensitization, and reduced lung function. Relative humidity exhibits a strongly negative correlation with rhinitis symptoms (r.0.91) whereas the monthly mean daily concentrations of inhalable particles and NO2 are positively correlated with this condition (r.0.89 and 0.88, respectively)(9,10).

Exposure of children (6–7 years of age) and adolescents (13–14 years of age) to indoor risk factors (including smoke), secondhand smoke, and mold aggravates preexisting allergic rhinitis and asthma(11).

In a systematic review of 97 studies on allergic rhinitis, the condition was not associated with active smoking in adults but was associated with passive exposure to smoking. O3 exposure was also associated with allergic rhinitis in children living in industrial areas, the rate of sensitization to outdoor allergens, and both current wheezing and allergic rhinitis(12). In Kang et al. (13), relationships were evident between a high level of exposure to

photochemical or gaseous pollutants (O3, carbon monoxide, NO2, and sulfur dioxide) and both the risk and prevalence of symptomatic asthma, rhinitis, and atopic eczema.

Elevated ambient O3 concentrations have been epidemiologically associated with nasal activation of neutrophils and eosinophils. Ong et al. (14) found that acute exposure to O3 (over 1–2 days) caused nasal epithelial injury, resulting in cell death and neutrophilic rhinitis, whereas repeated daily exposure to O3 (for 9 days) triggered eosinophilic rhinitis and lymphoid cell-dependent epithelial remodeling.

Although O3 is not considered an aeroallergen, exposure to this irritant has been reported to trigger or exacerbate preexisting allergic airway diseases including allergic rhinitis and asthma in both laboratory animals and humans(15).

1.3 Otitis media

Middle ear inflammation, commonly known as otitis media (OM), is a multifactorial disease of the middle ear, with a high prevalence among young children. Acute OM is characterized by infection of the middle ear with associated symptoms(16).

Young children are particularly vulnerable to OM globally affecting over 80% of children below the age of 3 years, many of them, (30–45%), have suffered two or more episodes. Their susceptibility is due to their smaller and shorter middle ear anatomical features than in adults, more horizontally aligned Eustachian tubes, and frequent upper respiratory tract infections. When inflammatory agents such as viral or bacterial pathogens, allergens, pollutants, and other irritants interact with the nasal mucosa, results inflammation that can narrow or block the Eustachian tube. Dysfunction in the Eustachian tube can lead to middle ear fluid stasis and subsequent middle ear infection. The sequelae of OM include hearing loss, language delay, impaired cognitive development and social difficulties in later life.

There is an increasing body of evidence supporting an association between higher ambient air pollution exposure and a higher risk of OM in children, while NO2 and SO2 showed the most consistent association(17).

Girguis et al(18). reported that increased risk of otitis media among preterm infants was associated with increased PM2.5 exposures 4 or 7 days prior to clinical encounter corresponding to the viral replication period, indicating that PM2.5 exposure may increase susceptibility to otitis media infection. There is evidence that PM2.5 exposure may further decrease mucus clearance by the cilia, causing inflammation leading to increased susceptibility to otitis media. Exposure to environmental tobacco smoke especially from parents who smoke in the home environment and the development of OM in children is well established. A systematic review by Jones et al. reported in 2012 that living with a smoker increased the risk of children's OM by 62%(19).

Deng et al. (20) found that industry-related air pollutants (SO2) significantly increased the OM risk in children. Particularly in preschooler females, was associated with prenatal exposure to outdoor air pollutant SO2 emitted from industries.

1.4 Rhinitis

As a typical respiratory illness, allergic rhinitis (AR) affects 20%–40% of the population worldwide, although the prevalence varies with age and region(21). Although it is usually a minor respiratory disease, AR frequently presented with symptoms that affect work performance, quality of daily life and consumes health resources(22).

In the respiratory system, allergic rhinitis is a common cause of other health conditions including sinusitis, otitis media, asthma, and hypertension. Particulate matters can induce the inflammation of nasal airway by mediating the response to nasal allergen. Teng et al. (22) also found that particulate matters (PM10 and PM2.5) were positively associated with allergic rhinitis risk development (including children and adults). In fact, relative to adult individuals, children are more susceptible to develop respiratory symptoms, when exposed to air particulate matters. Haiyan Chu et al. (23) study stratified their results by sex and observed that females p0.005 (1.63, 9.61) had higher allergic rhinitis risk associated with PM10 and PM2.5 pollution versus males p0.021 (0.61, 7.71). Kim et al. (12) reported a significant correlation with O3 exposure to the risk of AR as well as Graham and Koren(15) who observed that O3 exposure was associated with increased neutrophil content of nasal lavage fluid from

nonallergic individuals, indicating that O3 also induces nasal inflammation.

An experimental trial on in vitro systems, animals and human volunteers found that exposure to diesel exhaust particles leads to an increase in the total IgE level and produced inflammatory reactions on the airway at low concentrations and short time exposure(20). Living alongside busy roads is associated with an increased risk of allergic sensitization, and exposure of infants and school children to air pollution from traffic decreased their lung function(24,25).

1.5 Laryngitis

Voice disorders are most commonly related to environmental factors(15).

Damage from moisture is common and can initiate microbial growth and / or diffusion of chemicals from building materials. Problems due to moisture damage are common and are associated with a number of health symptoms, for example, rhinitis, cough and asthma symptoms, in addition, it predisposes the individual to bacterial growth and the diffusion of chemicals from the materials of the building. The microbes can be fungi, yeast, and bacteria, and they can each have different effects on health(26-28). Kallvik et al(29) studied nursery, preschool and school children and demonstrated the association between the frequency of hoarseness and the degree of indoor air problems due to moisture damage and laryngitis. In this study, 9.3% of children (n = 1857) in the group that had attended spaces with indoor air problems due to severe moisture damage were hoarse every week or more frequently. This was lower in a previous study by Meklin et al(30), where 28-30% of children who attended a school with microbial damage were hoarse. Participants in this study were between 6 and 17 years old and, therefore, children whose voices were probably affected by changes in puberty were included. The results indicate that these symptoms due to moisture damage should be included in voice anamnesis.

1.6 Asthma

Asthma and allergic rhinitis are the most prevalent chronic diseases in the pediatric age group. The prevalence of current asthma, 24.3% and 19% for school children and adolescents, respectively. Prevalence can vary from 10 – 20 fold in countries at different latitudes, suggesting the important role of indoor and outdoor environmental factors in their pathogenesis(31).

For instance, in Brazil, Rios et al. (32) concluded that asthma prevalence in adolescents was directly related to air pollution (p=0.002), and similar results in Holland associated with prevalence (OR=1.3) and asthma exacerbations (OR=1.2). However, there was no association between the concentration of outdoor pollutants and an increase in asthma prevalence (OR=0.7). Specific pollutants can induce airway inflammation (eg, O3, nitrogen dioxide, and PM2.5) and airway hyperresponsiveness (O3 and NO2), two characteristic features of asthma. Oxidative stress (a feature of severe asthma) has been associated with pollutant exposures (O3, NO2, and PM2.5). This mechanism has been suggested to be the major underlying mechanism behind many of the toxic reactions induced by air pollutants. Reactive oxygen species and free radicals are generated in response to TAP and can overwhelm the redox system and damage cell wall lipid, protein, and DNA, thereby leading to airway inflammation and airway hyperreactivity. Variation in the genes that regulate these mechanisms could confer increased susceptibility to the development of new-onset asthma or exacerbations of existing disease with exposure to air pollution.
(33,34).

A framework for how air pollution might contribute to the development and exacerbation of asthma proposed by the UK's Committee on the Medical Effects of Air Pollutants identified four main mechanisms: oxidative stress and damage, airway remodeling, inflammatory pathways, and immunological responses, and enhancement of respiratory sensitization to aeroallergens(35). Short-term exposure to ambient PM2.5 and PM10 in prospective cohorts of asthmatic children and adults has been associated with asthma symptoms, especially in children with allergic sensitization. Long-term exposure to PM is associated with poorly controlled asthma and decrements in lung function in children and

adults. Studies of asthmatic children and adults in the past 5 years have identified associations between nitrogen dioxide and symptoms of asthma, reduced response to bronchodilators, decrements in lung function, and exacerbation of asthma(36).

Young children with asthma have been found the most susceptible to adverse effects of air pollution because of their developing lungs, immature metabolic pathways, high ventilation rates per body weight, and increased time exercising outdoors. Even exposures in utero might affect the postnatal risk of asthma and asthma exacerbation. Low birth weight, which might be associated with narrow airways during early childhood, is a risk factor for symptoms of asthma-related to air pollution(37).

No consistent difference in air pollution effects on asthma has been evident between boys and girls, although asthma exacerbations are more prevalent and severe in young boys than in girls. In adults, active asthma is more prevalent among women than men. Elderly individuals with asthma are likely to be at increased risk for adverse effects related to air pollution, but scant literature is available to support this point(38).

1.7 Pneumonia

Children are the main vulnerable groups to air pollution being cardiovascular and respiratory diseases the more commonly related to PM2.5 exposure(1).

Several pieces of research explored the impacts of air pollution on human respiratory diseases including pneumonia, Girguis et al. (18) found that clinical encounters for infant bronchiolitis were positively associated with increases in PM2.5. For infant bronchiolitis, the elevated risk was observed when exposed to high levels were 1 or 4 days before the presentation of symptoms, corresponding to the estimated viral incubation period, indicating the possible influence of exposure on bronchiolitis susceptibility, also found that preterm infants are at a significantly greater risk of contracting the infection.

Lv et al. (39) reported that an increase of 1.06% of admissions for pneumonia in pediatric hospitals was associated with a 10 μg/m3 increase of PM2.5 concentrations in 2014 in Jinan, China. Consistently with previous studies mentioned, Jinyue Liu et al.(40) described that an increase of 10 μg/m3 in PM2.5 was associated with a 0.19% increase in the rates of pneumonia, student groups (age 6–17 years) and males were more vulnerable to PM2.5 exposure than other age and gender groups, and that the impacts on respiratory hospital admissions of air pollution was increased with the age of the child increases. In addition, the hospital admission for upper infection was significantly associated with the increase of PM2.5 concentration. Vahedian et al. found in adults that the risk of PM2.5 on the respiratory hospitalization was higher also in males than females. These results might be attributed to men's jobs, such as taxi driving, industry, and other outdoor activities, which make them exposed to more air pollution(41).

1.8 Chronic obstructive pulmonary disease (COPD)

COPD affects 12-16 million people in the United States, constituting the third leading cause of death(42).

This chronic disease is characterized by symptoms of wheezing, shortness of breath and cough. Intermittent exacerbations of the disease often change the disease course, leading to a decrease in quality of life, decreased lung function, reduced functional capacity and an increased risk of death (43). The World Health Organization (WHO) estimates that ambient air pollution is responsible for 3.7 million premature deaths worldwide in 2012 and 14% of these deaths were due to COPD or acute respiratory lower tract infections(44). Particularly, PM, NO2, and O3 have been related to COPD. Several longitudinal studies have shown that outdoor air pollution has longterm effects on lung function and that increased exposure to pollution leads to a rapid decrease in lung function in general population cohorts (44). This accelerated loss of lung function may contribute to the development of COPD and, according to literature, although studies are not definitive, the results suggest that exposure to outdoor air pollution is related to the incidence and prevalence of COPD. A recent meta-analysis showed that there was a tendency towards a higher prevalence of COPD, defined as chronic bronchitis or bronchitic symptoms, related to increased exposure to PM, but this relationship did not reach statistical significance(45). Also,

Andersen et al. investigated the average annual NO2 levels for 35 years and found an increase in the incidence of COPD associated with cumulative exposure in more than 57,000 subjects(46). A study of PM10 and hospital admissions for COPD found a 2.5% increase in income for each increase of 10 μg / m3 in PM10(47). In addition, Faustini et al. meta-analysis of 18 studies of PM10 and exacerbations found that an increase of 10 μg / m3 in daily PM10 was associated with a 2.7% increase in hospitalizations for COPD(48).

A study of PM10 and hospital admissions for COPD found a 2.5% increase in income for each increase of 10 μg / m3 in PM10(47). In addition, Faustini et al. meta-analysis of 18 studies of PM10 and exacerbations found that an increase of 10 μg / m3 in daily PM10 was associated with a 2.7% increase in hospitalizations for COPD(48). Higher levels of outdoor pollution have been linked to increased mortality in the general population, with higher figures for deaths of respiratory origin. In particular, several studies have suggested a link between outdoor particle concentrations and increased mortality in patients with COPD. There has been a significant association between PM10 and mortality using Medicare data of subjects discharged with a diagnosis of COPD between 1985 and 1999 (49).

Zhu et al. performed a meta-analysis of PM10 and mortality in patients with COPD and found a higher probability of mortality from COPD with exposure to PM10 with an increase of 10 μg / m3 in daily PM10 associated with an increase of 1.1% (95% CI 0.8-1.4) COPD mortality risk (48). Regarding the NO2 ratio, studies have suggested that the increase in their concentrations are associated with a higher COPD mortality(50). Overall, the results suggest that people with COPD are more susceptible to air pollution, even at lower levels than the general population.

2. Conclusion

In conclusion, this study indicated that short-term exposure to PM2.5 was associated with an increased of airway disease in any age patients. The World Health Organization estimated that approximately 250,000 deaths annual between 2030 and 2050 could be due to climate change as a result of the pollution in which we currently live. This is a public health issue that entails a cost of millions of dollars per year resulting from the costs of assistance to the health and productivity decline mainly due to absenteeism.

The reason for this review was the need to know the adverse effects that the current environmental situation may lead. We know that the main source of pollutants is vehicle emissions, as well as every year the number of circulating vehicles increases without promoting awareness of the responsible use of these.

The present work demonstrates the findings of countries in conditions similar to ours and it is our goal to emphasize the importance and urgency of air pollution control. In addition to taking preventive measures for the protection of the most vulnerable groups.

We encourage government authorities to implement actions to control the problem we face and is increasing; to the health system to raise awareness and promote preventive measures; to the researchers to keep on further studies about the effects of air particulate matter on the respiratory system.

3. Acknowledgements

We want to thank all the Medical Doctors from our Otolaryngology Department, Jennifer Mistica Zafiro Garcia Villanueva, Luis Angel Garza Montelongo, Mauricio Moreyra Villarreal, Julian Antonio Contreras Lopez, Adriana Miroslava Perez Peña, Fernando Balderas Elizondo and Jacinto Eduardo Treviño Cardenas for their kind support and help to the accomplishment of our paper. **4 . Funding sources**

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. **5 . Declaration of Competing Interest**

No conflict of interest was declared by the authors. **References**

1. Zhang D, Tian Y, Zhang Y, Cao Y, Wang Q, Hu Y. Fine particulate air pollution and hospital utilization for upper respiratory tract infections in Beijing, China. Int J Environ Res Public Health. 2019;16(4).

2. Jang AS, Jun YJ, Park MK. Effects of air pollutants on upper airway disease. Curr Opin Allergy Clin Immunol. 2016;16(1):13–7.

3. Guo Y, Barnett AG, Zhang Y, Tong S, Yu W, Pan X. The short-term effect of air pollution on cardiovascular mortality in Tianjin, China:

Comparison of time series and case-crossover analyses. Sci Total Environ [Internet]. 2010;409(2):300–6. Available from: http://dx.doi.org/10.1016/j.scitotenv.2010.10.013

4. Zanobetti A, Schwartz J, Samoli E, Gryparis A, Touloumi G, Peacock J, et al. The temporal pattern of respiratory and heart disease mortality in response to air pollution. Environ Health Perspect. 2003;111(9):1188–93.

5. Wu X, Lu Y, Zhou S, Chen L, Xu B. Impact of climate change on human infectious diseases: Empirical evidence and human adaptation. Environ Int [Internet]. 2016;86:14–23. Available from: http:// dx.doi.org/10.1016/j.envint.2015.09.007

6. Kim KH, Kabir E, Kabir S. A review on the human health impact of airborne particulate matter. Environ Int [Internet]. 2015;74:136–43. Available from: http://dx.doi.org/10.1016/j.envint.2014.10.005

7. (WHO) WHO. Health effects of particulate matter. Policy implications for countries in eastern Europe, Caucasus and central Asia. Copenhagen: WHO Regional Office for Europe. 2013.

8. Fu M, Zheng F, Xu X NL. Fu M, Zheng F, Xu X, Niu L. Advances of study on monitoring and evaluation of PM2.5 pollution. Meteorol Disaster Reduc Res. 20011;34:1–6.

9. Jung DY, Leem JH, Kim HC, Kim JH, Hwang SS, Lee JY, et al. Effect of traffic-related air pollution on allergic disease: Results of the Children's Health and Environmental Research. Allergy, Asthma Immunol Res. 2015;7(4):359–66.

10. Nicolussi FH, dos Santos APM, André SC da S, Veiga TB, Takayanagui AMM. Air pollution and respiratory allergic diseases in schoolchildren. Rev Saude Publica. 2014;48(2):326–30.

11. Azalim SP, Camargos P, Alves AL, Senna MIB, Sakurai E, Schwabe Keller W. Exposure to environmental factors and relationship to allergic rhinitis and/or asthma. Ann Agric Environ Med. 2014;21(1):59–63.

12. Kim BJ, Kwon JW, Seo JH, Kim H Bin, Lee SY, Park KS, et al. Association of ozone exposure with asthma, allergic rhinitis, and allergic sensitization. Ann Allergy, Asthma Immunol. 2011;107(3):214219.e1.

13. Kang IG, Ju YH, Jung JH, Ko KP, Oh DK, Kim JH, et al. The effect of PM10 on allergy symptoms in allergic rhinitis patients during spring season. Int J Environ Res Public Health. 2015;12(1):735–45.

14. Articles A. Page 1 of 39 Hepatology. Hepatology. 2014;(979):1–39.

15. Graham, D. E. and HSK. Biomarkers of inflammation in ozone-exposed humans. Comparison of the nasal and bronchoalveolar lavage. Am Rev Respir Dis. 1990;142:152–61.

16. Bowatte G, Tham R, Perret JL, Bloom MS, Dong G, Waidyatillake N, et al. Air pollution and otitis media in children: A systematic review of literature. Int J Environ Res Public Health. 2018;15(2):1–19. 17. Leung, A.K.C.; Wong AH. Acute otitis media in children. Recent Pat. Inflamm. Allergy Drug Discov. 2017;11:32–40.

18. Girguis MS, Bartell SM, Vieira VM, Strickland MJ, Hu X, Liu Y, et al. Exposure to acute air pollution and risk of bronchiolitis and otitis media for preterm and term infants article. J Expo Sci Environ Epidemiol. 2018;28(4):348–57.

19. Jones LL, Hassanien A, Cook DG, Britton J, Leonardi-Bee J. Parental smoking and the risk of middle ear disease in children: A systematic review and meta-analysis. Arch Pediatr Adolesc Med. 2012;166(1):18–27.

20. Deng Q, Lu C, Jiang W, Zhao J, Deng L, Xiang Y. Association of outdoor air pollution and indoor renovation with early childhood ear infection in China. Chemosphere. 2017;169:288–96.

21. Greiner, A.N.; Meltzer EO. Overview of the treatment of allergic rhinitis and nonallergic rhinopathy. Proc Am Thorac Soc. 2011;8:121–31. 22. Bo Teng 1,*, Xuelei Zhang 2, 3,*, Chunhui Yi 4, Yan Zhang 5, Shufeng Ye 1, Yafang Wang 1, Daniel Q. Tong 3, 6 and Binfeng Lu 1 7. The Association between Ambient Air Pollution and Allergic Rhinitis: Further Epidemiological Evidence from Changchun, Northeastern China. Int J Environ Res Public Heal 2017, 14, 226; 2017;14(226).

23. Chu H, Xin J, Yuan Q, Wang M, Cheng L, Zhang Z, et al. The effects of particulate matters on allergic rhinitis in Nanjing, China. Environ Sci Pollut Res. 2019;26(11):11452–7.

24. Nordling E, Berglind N ME. Traffic-related air pollution and childhood respiratory symptoms, function and allergies. Epidemiology. 2008;19:401–8.

25. Rosenlund M, Forastiere F PD. Traffic-related air pollution in relation to respiratory symptoms, allergic sensitization and lung function in school children. Thorax. 2009;64:573–80. 26. Vardoulakis S, Dimitroulopoulou C, Thornes J, Lai KM, Taylor J, Myers I, et al. Occupational safety and health aspects of voice and speech professions.

Cite this article: Respiratory diseases in the hands of air pollution: Am J Rhin and Otolo. 2019; 2(2): 01-07.

Environ Int [Internet]. 2016;6(1):28–33. Available from: http://www.who.int/mediacentre/news/releases/2014/air-pollution/es/

27. McKinnon DH, McLeod S, Reilly S. The prevalence of stuttering, voice, and speech-sound disorders in primary school students in Australia. Lang Speech Hear Serv Sch. 2007;38(1):5–15.

28. Vardoulakis S, Dimitroulopoulou C, Thornes J, Lai KM, Taylor J, Myers I, et al. Impact of climate change on the domestic indoor environment and associated health risks in the UK. Environ Int [Internet]. 2015;85:299–313. Available from: http:// dx.doi.org/10.1016/j.envint.2015.09.010

29. Kallvik E, Putus T, Simberg S. Indoor Air Problems and Hoarseness in Children. J Voice [Internet]. 2016;30(1):109–13. Available from: http:// dx.doi.org/10.1016/j.jvoice.2015.02.012 30. Meklin T, Potus T, Pekkanen J, Hyvärinen A, Hirvonen MR, Nevalainen A. Effects of moisture-damage repairs on microbial exposure and symptoms in schoolchildren. Indoor Air, Suppl.

2005;15(10):40–7.

31. Solé D, Wandalsen GF, Nunes ICC, Naspitz CK I– GB. Prevalence of symptoms of asthma, rhinitis, and atopic eczema among Brazilian children and adolescents identified by the International Study of Asthma and Allergies in Childhood (ISAAC) – Phase 3. Pediatr (Rio J). 2006;82:341–6.

32. Rios JLM, Boechat JL, Sant´Anna CS FA. Atmospheric pollution and the prevalence of asthma: study among schoolchildren of 2 areas in Rio de Janeiro, Brazil. Ann Allergy Asthma Immunol. 2004;92:629–34.

33. Gehring U, Wijga AH, Brauer M, Fischer P, de Jongste JC KM. Traffic-related air pollution and the development of asthma and allergies during the first 8 years of life. Am J Respir Crit Care Med. 2010;181:596–603.

34. Seltzer J, Bigby BG SM. O3-induced change in bronchial reactivity to methacholine and airway infl ammation in humans. J Appl Physiol. 1986;(60):1321–6.

35. Gowers AM, Cullinan P AJ. Does outdoor air pollution induce new cases of asthma? Biological plausibility and evidence; a review. Respirology.

2012;17:887–98.

36. Samoli E, Nastos PT, Paliatsos AG, Katsouyanni K PK. Acute eff ects of air pollution on pediatric asthma exacerbation: evidence of association and eff ect modifi cation. Environ Res. 2011;(111):418– 24.

37. Miller MD MM. Impact of environmental chemicals on lung development. Environ Health Perspect. 2010;118:1155–64.

38. Johnston NW SM. Asthma exacerbations. 1: epidemiology. Thorax. 2006;61:722–8.

39. Lv C, Wang X, Pang N, Wang L, Wang Y, Xu T, et al. The impact of airborne particulate matter on pediatric hospital admissions for pneumonia among children in Jinan, China: A case-crossover study. J Air Waste Manag Assoc. 2017;67(6):669–76.

40. Liu J, Li Y, Li J, Liu Y, Tao N, Song W, et al. Association between ambient PM2.5 and children's hospital admissions for respiratory diseases in Jinan, China. Environ Sci Pollut Res. 2019; 41. Dastoorpoor M, Masoumi K, Vahedian M, Aghababaeian H, Sekhavatpour Z, Khanjani N, et al. Associations of short-term exposure to air pollution with respiratory hospital admissions in Ahvaz, Iran. Process Saf Environ Prot. 2019;150–60.

42. Nadia N. Hansel MCM& VK. The Effects of Air Pollution and Temperature on COPD. COPD J Chronic Obstr Pulm Dis. 2015;0:1–8.

43. Donaldson GC WJ. COPD exacerbations: Epidemiology. Thorax. 2006;61(2):164–8.

44. World Health Organization. Ambient

(outdoor) air quality and health. Geneva World Heal Organ. 2014;Fact sheet.

45. Song Q, Christiani DC, Xiaorong Wang RJ. The global contribu- tion of outdoor air pollution to the incidence, prevalence, mortality and hospital admission for chronic obstructive pulmonary disease: a systematic review and meta-analysis. Int J Environ Res Public Health. 2014;11(11):11822–32.

46. Andersen ZJ, Hvidberg M, Jensen SS, Ketzel M, Loft S SM. Chronic obstructive pulmonary disease and long-termexposure to traffic-related air pollution: a cohort study. Am J Respir Crit Care Med.

2011;183(4):455–61.

47. Ji M, Cohan DS BM. Meta-analysis of the Association between Short-Term Exposure to Ambient Ozone and Respiratory Hospital Admissions. Environ Res Lett. 2011;6(2).

48. Zhu R, Chen Y, Wu S, Deng F, Liu Y YW. The relationship between particulate matter (PM10) and hospitalizations and mortal- ity ofchronic obstructive pulmonary disease: ameta-analysis. COPD J Chronic Obstr Pulm Dis. 2013;10(3):307–15.

Cite this article: Respiratory diseases in the hands of air pollution: Am J Rhin and Otolo. 2019; 2(2): 01-07.

49. Zanobetti A, Bind MA SJ. Particulate air pollution and sur- vival in a COPD cohort. Environ Health Perspect. 2008;7:48.

50. Faustini A, Stafoggia M, Cappai G FF. Shortterm effects of air pollution in a cohort of patients with chronic obstructive pulmonary disease. Epidemiology. 2012;23(6):861–79.

Cite this article: Respiratory diseases in the hands of air pollution: Am J Rhin and Otolo. 2019; 2(2): 01-07.