Trends in Clinical and Medical Sciences

PSR Press

Article

Air pollution and Otitis media among children: A systematic review and meta-analysis

Ancy Anthony Vithayathil[1,*], Aniketa Sharma[2], Anupama Arora[3] and Nagarathna H K[4]

1 Al Azhar Medical College, Ezhalloor, Thodupuzha.
2 Department of Medicine, Dr. Y. S. Parmar Govt. Medical College, Nahan, H.P. India.
3 Department of Psychiatry, Dr. Y. S. Parmar Govt. Medical College, Nahan, H.P. India.
4 Department of ENT, Akash Institute of Medical Sciences and Research Centre, Bangalore.
* Correspondence: anzav123@yahoo.com

Received: 2 March 2023; Accepted: 10 May 2023; Published: 15 May 2023.

Abstract: Background: Otitis media (OM), a common ear infection, affects children. Most pediatric medical appointments are for otitis media. Otitis media (OM) is the major cause of permanent hearing loss. OM has three primary subtypes: acute, OM with effusion, and chronic suppurative OM (CSOM).
Material and Methods: This study is a meta-analysis on the link between air pollution and middle ear infections in Indian children. The PRISMA (Preferred Reporting Items for Systematic reviews and Meta-analyses) guidelines for reporting systematic reviews and meta-analyses were followed throughout this investigation. This study was about otitis media, which is also called glue ear, middle ear infection, OM, AOM, OME, CSOM, or middle ear infection. It is important because it affects so many people. We were exposed to air pollution from both inside (from heating and cooking) and outside (from cars and trucks) sources (from factories or cars).
Results: 1246 references were taken out because there were fewer of them. After the first round of evaluation, 661 out of the 743 citations were found to be unnecessary. The full texts of 82 publications were looked at to see if they could be included. There was a total of eleven investigations: three in the US, two in Canada, and one in the both Netherland and Germany, the other studies were conducted in Italy, Czech Republic, South Korea, China and Spain.
Conclusion: This comprehensive review found that air pollution increases the risk of OM in babies and children. Exposure to higher amounts of NO2, PM, SO2, PAH, and wood smoke increases OM infections, however the extent is unclear. PM10, NO2, O3, SO2, and CO enhanced OM risk immediately and for 1-4 weeks.

Keywords: Otitis Media; Air Pollution; Ear Infection; Ear Discharge; Children; Chronic suppurative otitis media; Acute otitis media.

1. Introduction

Children frequently suffer from otitis media (OM), a common ear infection [1]. When it comes to pediatric medical care, otitis media is the most common reason for visits [2]. Infections in the middle ear, collectively known as otitis media (OM), are a leading cause of permanent hearing loss. Acute OM, OM with effusion, and chronic suppurative otitis media (CSOM) are the three main subtypes of OM [3]. Otalgia, fever, and tympanic membrane erythema are classic markers of an ear infection, and their rapid onset characterizes acute otitis media (AOM). CSOM comprise a wide range of surgical procedures and account for a sizable percentage of patients referred to ear, nose, and throat clinics. [4,5].

Middle ear infections happen to about two-thirds of all children [6]. Studies put its incidence anywhere from 15% to 40% [7,8]. More than 42 million people over the age of three suffer permanent hearing loss from otitis media each year, according to the World Health Organization [9]. This disorder affects around 90% of children before they start school. Many otitis media bouts cure spontaneously within three months, although the illness recurs in 30% to 40% of youngsters [10,11]. Throughout the first three years of life, 75% of children experience otitis media at least three times [12].

Living in densely populated places, having a big family, prolonged nursing, and smoking all increase the likelihood of developing ear infections. Craniofacial abnormalities, such as Down syndrome and cleft palate, can be inherited, and so can susceptibilities to certain diseases and nutritional deficiencies (e.g., age, gender, race, health status, history of several acute otitis episodes, rhinorrhea, allergic rhinitis, seasonal rhinitis, snoring, upper respiratory tract infections, and adenoid hypertrophy) [13–15].

Many environmental and meteorological characteristics, such as temperature and air humidity, and their effects on OM have been studied [16,17]. A large body of epidemiological research from both Europe and the United States has linked OM in children to exposure to polluted outdoor air. In 1993, researchers Sperm and Branica established a link between aridity and OM. Outbreaks of OM were most common in the winter and least common in the summer [18]. In conducting a cross-sectional study of 393 children in three different regions of São Paulo, Brazil, researchers Ribeiro and Cardoso found a favorable correlation between air pollution levels and the occurrence of respiratory symptoms such ear infections [19].

It is commonly known that children who are exposed to ambient tobacco smoke, particularly from smoking parents, have a higher risk of developing OM. The prevalence of OM in children is increased by 62% when they are exposed to secondhand smoke, according to a thorough review done in 2012 by Jones et al. [20]. Animal studies have shown how prolonged exposure to ambient tobacco smoke affects the middle ear and the mucosa of the Eustachian tube [21]. Exposure to environmental tobacco smoke has been linked to OM in children, although the relationship with exposure to ambient air pollution is unknown at this time.

The nasopharynx is the opening between the nasal cavity and the oral cavity via which inspired air is taken in. The eustachian tube is a passageway that runs from the back of the nasopharynx to the middle ear. This passageway connects the middle ear directly to the air that is inhaled. Larger airborne particles, such as PM 10 and formaldehydes, are engulfed or expectorated after being dissolved or trapped by the nasal mucosa and transported to the back of the nasopharynx. The nasopharynx is a major entry point for gases and PM 2.5 particles into the respiratory system.[22] The connection between the nasal cavity and the middle ear could mean that these toxins can go down the nasal cavity and harm the epithelium lining the Eustachian tube. A columnar ciliated cell's motile, hair-like cilia beat repeatedly in the direction of the nasopharynx to clear mucociliary debris and drain middle ear fluid. The Eustachian tube controls air pressure and volume in the middle ear. Eustachian tube inflammation caused by viruses, allergies, pollution, and other irritants can cause it to become smaller or clogged. Disruption of the Eustachian tube can lead to a middle ear infection.

Furthermore, epidemiological research has linked OM to breathing in dirty air, but this link hasn't been looked at in a systematic way recently, especially in children. We decided to do a systematic review of all the studies on the subject to see how strong the evidence was for a link between children's short-term and long-term exposure to ambient air pollution and OM.

2. Material and methods

2.1. Study protocol

This study is a meta-analysis on the link between air pollution and middle ear infections in Indian children. The PRISMA (Preferred Reporting Items for Systematic reviews and Meta-analyses) guidelines for reporting systematic reviews and meta-analyses were followed throughout this investigation. [23] Researchers in this study handled every step on their own.

2.2. Inclusion criteria

This systematic review looks at research on otitis media, air pollution, acute OM, and chronic suppurative OM. Only studies with full English descriptions of cohort, case-control, case-crossover, cross-sectional, or time-series epidemiologic study designs were taken into account. All children younger than 18 years old are in the data set. This study was about otitis media, which is also called glue ear, middle ear infection, OM, AOM, OME, CSOM, or middle ear infection. It is important because it affects so many people. We were exposed to air pollution from both inside (from heating and cooking) and outside (from cars and trucks) sources (from factories or cars). Since our focus was on air pollution in the environment, we left out studies that looked at the effects of cigarette smoke and OM in the environment. The study was made to be open-ended so that any relevant research from the past could be added.

2.3. Source of information

We searched for relevant studies on the association between air pollution and otitis media in children from the time they were originally published until the first week of January 2022 and found them all. "Air pollution", "otitis media", "Acute otitis media", "Chronic suppurative otitis media", "children", "ear infection", "environmental variables", "hearing loss", "causality" "Middle ear" "etiology", and "randomized controlled trial" were the search terms utilized. We broadened our search to include papers from PSYCHInfo, Scopus, Medlib, Google Scholar, PubMed, Web of Sciences, PROQUEST, Medline, Embase, Google, and the Cochrane library, despite the fact that the review contained all pertinent material.

As grey literature was included in the PROQUEST database with e-books, we did not have to do a separate search for it. In order to find other publications that might be relevant, we meticulously looked through the references of a select number of journals. To conduct this search, we used a systematic review approach and a comprehensive set of keywords.

Before retrieving the whole papers, one author reviewed the titles and abstracts of all potentially relevant articles. All of the authors agreed with the final selection of papers to be included. We restricted our analysis to randomized controlled trials examining air pollution and otitis media among Childrens. When meta-analysis-suitable raw data was necessary, the authors were contacted. Section 4 presents a summary and ranking of the selected studies based on the quality of their supporting evidence.

2.4. Data extractions

We developed and standardized our method of data collecting in response to the Cochrane data collection grid. The review included all studies that met the inclusion criteria. Two reviewers independently decided which articles should be included from the retrieved literature and which studies were acceptable for inclusion. Co-evidence was used to screen all of the papers for potential conflicts of interest, and the authors initially reviewed the titles and abstracts independently. As disagreements arose, they were discussed and ultimately resolved. This similar method was used during the stage of the study in which we screened each text in its entirety. While this method added time to the screening process, it also allowed both reviewers to double-check that the study's goals were accomplished by referring back to the protocol.

2.5. Prisma Flow Chart

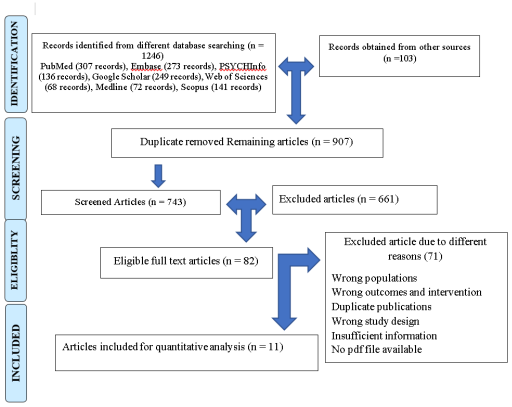

Figure 1. PRISMA technique were used for the selection mechanism schedule of articles in this study

3. Results

Based on the Preferred Reporting Items for Systematic Reviews (PRISMA) statement, the systematic review was done (Figure 1). A meta-analysis of randomized controlled trials was used to look at air pollution and otitis media among Childrens. We looked for RCTs on this topic by searching PubMed, Web of Science, Scopus, Google Scholar, Copernicus, Embase, PSYCHInfo, and the Cochrane Library from the time they opened until now (language obstacles permitting). There were links to papers that defined the terms air pollution, middle ear infection, acute middle ear infection, chronic suppurative middle ear infection, children,

ear infection, environmental factors, hearing loss, causation, the middle ear, the etiology, and randomized controlled trials. Next, we looked through the works that were cited in a small number of journals by hand to see if there was any more relevant research. 1246 references were taken out because there were fewer of them. After the first round of evaluation, 661 out of the 743 citations were found to be unnecessary. The full texts of 82 publications were looked at to see if they could be included. Eleven of them met the minimum requirements, and they were all pretty good (Figure 1). In Section 4, all of the above information is put together in one place. Each study compared more than one group by using what the researchers already knew. There was not a single study where people were picked at random. There was a total of eleven investigations: three in the US, two in Canada, and one in the both Netherland and Germany, the other studies were conducted in Italy, Czech Republic, South Korea, China and Spain.

Changsha preschoolers had 7.3% lifetime doctor-diagnosed OM. Childhood OM was solely linked with prenatal industrial air pollution with adjusted OR (95% CI) = 1.44 (1.09–1.88) for a 27 μg/m3 increase in SO2, mainly in the first trimester. Prenatal SO2 exposure was linked with OM but not repeated episodes, adjusted OR (95% CI) = 1.47 (1.10–1.96).

160,875 OM-related hospitalizations among kids younger than 15 years old. Most time lags exhibited odds ratios (ORs) that were larger for correlations with higher concentrations of the five pollutants than with the reference values. Exposed to CO for 2 days before to an OM ED visit, PM10 had the greatest impact on OM incidence at a time lag of 0 weeks, while NO2 and O3 had the greatest affects at time lags of 1 and 4 weeks, respectively [24].

Depending on lag, there were different non-significant associations with middle ear infections. Preterm babies had a much higher chance of getting bronchiolitis one day before they saw a doctor (OR = 1.17, 95% CI = 1.08–1.28) and otitis media four and seven days before they saw a doctor (OR = 1.09, 95% CI = 1.02–1.16 and OR = 1.08, 95% CI = 1.02–1.15) [25]. LRTI and ear infections were related with a 10-μg/m3 rise in average NO2. The RRs for an interquartile range (IQR) rise in NO2 were 1.08 for LRTI and 1.31 for ear infections. Relative to NO2, an IQR increase in average benzene exposure was similar for LRTI (RR = 1.06; 95% CI: 0.94, 1.19) and slightly lower for ear infections (RR = 1.17; 95%: 0.93, 1.46). Infants whose mothers spent more time at home throughout pregnancy had slightly stronger associations [26].

With a lag time of 0-8 hours per 10 gm3 of NO2, there is a weak connection between elevated NO2 and visits to the ED for OM. The RR was 1.03 (95% CI 1.01,1.05) [27]. Air pollution caused by vehicular traffic made the odds of getting otitis media higher. An increase of 3 g/m3 PM2.5, 0.5 g/m3 elemental carbon, and 10 g/m3 NO2 was linked to odds ratios of 1.13 (1.00–1.27), 1.10 (1.00–1.22), and 1.14 (1.03–1.27) in the Netherlands and 1.24 (0.84–1.83), 1.10 (0.86–1.41), and 1.14 (0.87–1.49) in Germany. [28] 14,527 ED visits for OM in children 1–3 years old over 10 years were analyzed. ED visits for OM were associated with interquartile increases in CO and NO2 after controlling for ambient temperature and relative humidity. In the warmer months (April–September), females and all patients were exposed to CO and NO2, and boys to CO, two days before an OM ED visit [33].

4. Literature Review: Air Pollution and its Impact on Otitis Media

Park et al. [24], focused on a case-control study conducted in South Korea. The study included a total of 160,875 children aged 0-14 years. The researchers utilized generalized estimating equations to examine the correlations between OM occurrence on a weekly basis and five air contaminants. The exposure assessment involved evaluating the effects of air pollutants on OM hospitalizations. The study found that most time lags had greater odds ratios (ORs) for associations with higher concentrations of the five pollutants compared to the reference values. Specifically, exposure to carbon monoxide (CO) two days before an OM emergency department visit, particulate matter with a diameter of 10 micrometers or less (PM10) had the largest effect on OM incidence at 0 weeks, nitrogen dioxide (NO2) at 1 week, and ozone (O3) at 4 weeks.

The study, conducted by Girguis et al. [25], was a case-control study conducted in the USA. The study included a total of 42,336 infants aged 0-3 years. The researchers examined the association between acute air pollution exposure and the risk of bronchiolitis and otitis media in preterm and term infants. Air pollution measurements were obtained using fixed monitoring stations and special monitors set up for the study. The study found varied relationships between middle ear infections and lag periods. However, preterm infants

were more likely to have bronchiolitis one day prior to visiting a doctor, as well as otitis media four and seven days before seeking medical attention.

Aguilera et al. [26] conducted a cohort study in Spain to investigate the association between early-life exposure to outdoor air pollution and respiratory health, ear infections, and eczema in infants. The study included 2,199 participants aged 0-2 years from the INMA study. Parents' reports of otitis media (OM) were most commonly observed in the first two years of life. The researchers examined the impact of exposure to nitrogen dioxide (NO2) and benzene on the occurrence of OM and lower respiratory tract infections (LRTIs). The study found that an increase in average NO2 levels of 10 μg/m3 was associated with a higher risk of OM (RR = 1.31) and LRTIs (RR = 1.08). The study also noted a similar risk of LRTIs with average benzene exposure (RR = 1.06) and a slightly lower risk of ear infections (RR = 1.17) compared to NO2. These associations were marginally stronger for babies whose mothers stayed at home more often during pregnancy.

Gestro, M. et al. [27] conducted a retrospective observational study in Italy to explore the relationship between meteorological factors, air pollutants, and emergency department (ED) visits for otitis media. The study analyzed data from a fixed-site monitoring station and examined the effects of particulate matter with a diameter of 10 micrometers or less (PM10), nitrogen dioxide (NO2), ozone (O3), and carbon monoxide (CO). The study found that there was not a substantial link between high NO2 levels and ED visits for otitis media with a delay of 0-8 hours per 10 μg/m3 of NO2. The 95% confidence interval for the relative risk (RR) was 1.01-1.05.

Brauer M et al. [28] conducted a cohort study in the Netherlands and Germany to investigate the association between traffic-related air pollution and otitis media (OM). The study included 4,146 participants aged 0-1 year. Parent-reported doctor-diagnosed infectious OM in the first two years of life was used as the outcome measure. The study found that traffic-related air pollution, including PM2.5, elemental carbon, and NO2, was associated with increased odds ratios for OM. In the Netherlands, an increase of 3 μg/m3 in PM2.5, 0.5 μg/m3 in elemental carbon, and 10 μg/m3 in NO2 were linked to odds ratios of 1.13, 1.10, and 1.14, respectively. In Germany, the corresponding odds ratios were 1.24, 1.10, and 1.14. These findings suggest that exposure to traffic-related air pollution may contribute to the development of otitis media in early childhood.

Deng et al. [16] conducted a prospective cohort study in China to explore the association between prenatal exposure to industrial air pollution and the onset of early childhood ear infections. The study included 1,617 participants aged 4 to 7 years. Parent-reported doctor-diagnosed lifelong prevalence of infectious otitis media at 3-4 years was used as the outcome measure. The study found that prenatal exposure to industrial air pollution, specifically sulfur dioxide (SO2), was associated with an increased risk of childhood otitis media. For every 27 μg/m3 increase in SO2 exposure during pregnancy, there was an adjusted odds ratio of 1.44 for childhood otitis media. The association was mainly observed for exposures occurring in the first trimester. Prenatal SO2 exposure was associated with otitis media but not with repeated episodes. The adjusted odds ratio was 1.47. These findings suggest that prenatal exposure to industrial air pollution, particularly SO2, may be a risk factor for the development of early childhood otitis media.

In a retrospective study conducted by Kousha et al. [29] in Canada in 2016, the researchers investigated the relationship between the air quality health index and emergency department (ED) visits for otitis media (OM) in children aged 0-3. The study involved 4,815 patients, and the exposure assessment was based on the air quality health index. The OM diagnoses were obtained from medical records, and there was a time lag of up to 15 days between air pollution monitoring and OM ED presentations. The findings revealed that three days after exposure, the discharge diagnosis of OM increased by 5% to 6% for every 1 unit increase in the air quality health index. Nonlinear time series analysis further strengthened the observed effects. Moreover, during the first 15 days following an increase in the air quality health index, the risk of OM was 1.22 times higher compared to days without such exposures. The study received a quality rating of 8.

Another relevant study by Strickland et al. [30] in the USA in 2016 examined the association between short-term changes in PM2.5 concentrations and pediatric emergency department visits for OM. This cross-sectional study included 237,833 participants aged 0-18. The exposure assessment relied on the MODIS satellite data to measure aerosol optical depth as a proxy for PM2.5 concentrations. Simultaneous measurements of air pollution and OM were taken into account. The results, expressed as odds ratios, showed that for same-day OM ED visits, a 10 g/m3 increase in PM2.5 concentration had a Lag 0 odds ratio of 1.005 (95% CI 0.996, 1.014) and a Lag 1 odds ratio of 0.995 (95% CI 0.985, 1.004).

In a case-crossover study conducted by Xiao et al. [31] in the USA in 2016, the researchers investigated the relationship between pediatric emergency department (ED) visits and ambient air pollution in the state of Georgia. The study included 422,268 participants aged 0-18 and employed a case-control design. The researchers utilized simulations from the CMAQ model to estimate daily concentrations of various air pollutants. Medical records were used to identify ED reports for otitis media (OM), with a time lag of up to 3 days between measuring air pollution and ED reports. The findings showed that a rise in the 3-day moving average ambient air pollutant interquartile range increased the risk of OM. Specifically, traffic pollutants such as carbon monoxide (CO), nitrogen dioxide (NO2), elemental carbon (EC), and organic carbon (OC) were found to be associated with an increased risk of OM. When considering interactions, the odds ratio (OR) for OM with interactions was 1.025 (95% CI 1.012, 1.039), while the OR without interactions was 1.018 (95% CI 1.010, 1.026). The study received a quality rating of 7.

In a retrospective cohort study by Dostal et al. [32] in the Czech Republic in 2014, the researchers examined the differences in the spectra of respiratory illnesses between children living in urban and rural environments. The study included 960 participants aged 0-10 and relied on medical records coded with ICD-10 to identify cases of serous and infectious otitis media, mastoiditis, and perforation. The findings revealed that in the first two years of life, children living in urban and industrial areas in Teplice had a significantly higher incidence of otitis media compared to rural children in Prachatice. The rate ratio was 2.3 (95% CI 1.7-4.1), indicating a considerably greater risk of otitis media in urban and industrial environments. The study received a quality rating of 8.

Zemek et al. [33] conducted a case-crossover study in Edmonton, Canada in 2010 to explore the association between air pollution and emergency department (ED) visits for otitis media (OM). The study included 14,527 ED visits for OM in children aged 1-3 years over a 10-year period. The researchers utilized data from fixed-site monitoring stations to obtain measurements of carbon monoxide (CO), nitrogen dioxide (NO2), ozone (O3), sulfur dioxide (SO2), particulate matter with a diameter of 10 μm or less (PM10), and particulate matter with a diameter of 2.5 μm or less (PM2.5). Medical records were used to identify OM ED visits, with a time lag of up to 4 days between air pollution monitoring and ED presentations.

The findings showed that ED visits for OM were associated with increases in CO and NO2 levels, even after controlling for ambient temperature and relative humidity. In the warmer months (April-September), females and all patients were exposed to elevated levels of CO and NO2 two days prior to an OM ED visit. Boys, specifically, were exposed to increased CO levels. This suggests that there is a relationship between air pollution, specifically CO and NO2, and the occurrence of OM requiring ED visits in young children. The study received a quality rating of 7.

5. Discussion

After being exposed to air pollution for a long time, your lungs may start to get sick. Pollutants in the air tend to build up in cities because there are so many things that burn fuel there. Since at least 2004, air pollution in the environment has been linked to a higher risk of OM in children. So far, the best information we have comes from studies that look at whole groups of people, such as newborns. Researchers may get different results because of differences in how they measure exposure, the air pollutants they study (like traffic, PAHs, benzene, wood smoke, etc.), the timing of exposure windows (prenatal and postnatal), and geographical factors. Even so, every study that looked at the link between OM and pollution found that pollution made the risk of getting OM higher. In case-crossover and time-series studies, there were also significant links found between OM exposure to outdoor air pollution and trips to the emergency room [16,24–33].

It is well known that OM is common in very young children and that young children are more likely to get sick from pollution in the environment. But different studies have different ideas of what it means to be "very young." We first thought about ages 2 and 3, but in the end, we chose age 2 as the criterion because study results were more different around that age. This study found something interesting: kids older than 2 were more likely to get OM when they were exposed to more PM10, but babies and toddlers were more likely to get OM when they were exposed to more PM2.5. Notably, the study by Park et al. (2018) [24], which only looked at children over the age of 5, had a big effect on the PM10 results and gave information that was different from what the other studies showed. We need to do more research to confirm this finding, because this could be a big source of bias.

Possible delays were also investigated for their impact. There was little change in the frequency of OM across investigations with delays of 0-7 days for PM2.5. However, there was little consistency in the findings across studies when looking at this particular subset. It's worth noting that while using the same lag time, Zemek et al. [33] reported the lowest odds ratio and Kousha et al. [29] reported the greatest odds ratio (i.e., 3 days after exposure to PM 2.5). The time interval between PM10 exposure and the onset of symptoms was also investigated by Zemek et al. [33], who similarly found no significant differences between the 0- and 4-day time intervals. It is not possible to compare the findings of Xiao et al. [31] and Park et al. [24], who both discovered considerably positive risk ratios, due to the differing lag times. Since every study in the PM 10 cohort had a different lag, it was impossible to conduct a comprehensive analysis of the lags employed by these investigations.

Women in low-income nations perform most of the cooking, and young children often help out. This increases the likelihood that pregnant women and children would be exposed to indoor biomass smoke. Two recent Chinese studies added to the body of evidence linking indoor air pollution from home renovations and new furnishings to the onset of OM in children [16]. Children's exposure to PAH and OM was investigated in one study. For their study of benzene levels in the environment, researchers in Spain employed computational models based on data from the INMA birth cohort. Children diagnosed with OM between 12 and 18 months of age were found to have been exposed to benzene during the first two trimesters of pregnancy [26]. Since PAHs are immunotoxic, they may lower the fetal immune system, increasing the risk of childhood OM.

The combined evidence from these research points to a connection between OM in young children and both outdoor and interior air pollution. From a biological perspective, this makes sense in light of previous papers detailing the disorders that might result in OM. This evaluation did not investigate ETS exposure; however, a recent systematic review and meta-analysis demonstrated a robust association between ETS exposure and OM in children [34,35].

Different ways of measuring air pollution that are used in epidemiological research have their pros and cons. From stationary monitoring stations, we know a lot about how air pollution changes over time, but we don't know much about how it changes over space. Distance to roads and other measures of closeness don't take into account things like the direction of the wind, the amount of traffic, or the type of building. LUR methods are used to figure out how much air pollution a person is exposed to. These methods take into account things like traffic, population density, land use, and vegetation. There were many different OM case definitions in the studies we looked at. Parents' reports of how OM was diagnosed by a doctor, which may be prone to recall bias, have been used in a number of studies.

6. Conclusion

This study concluded that the data suggests infants and children who are exposed to air pollution are at increased risk for developing OM, as reported in this systematic review. Exposure to greater levels of NO2, PM, SO2, PAH, and wood smoke is associated with an increase in OM infections, however the extent to which this is true was not universally agreed upon. After being exposed to PM10, NO2, O3, SO2, and CO, a person's risk of OM increased immediately and persisted for 1-4 weeks. What we can glean from these findings regarding how OM.

Author Contributions: All authors contributed equally to the writing of this paper. All authors read and approved the final manuscript.

Conflicts of Interest: The authors declare that they do not have any conflict of interests.

References

[1] BAuinger, P., Lanphear, B. P., Kalkwarf, H. J., & Mansour, M. E. (2003). Trends in otitis media among children in the United States. *Pediatrics*, *112*(3), 514-520.

[2] Vergison, A., Dagan, R., Arguedas, A., Bonhoeffer, J., Cohen, R., DHooge, I., ... & Pelton, S. I. (2010). Otitis media and its consequences: beyond the earache. *The Lancet infectious diseases*, *10*(3), 195-203.

[3] Gates, G. A., Klein, J. O., Lim, D. J., Mogi, G., Ogra, P. L., Pararella, M. M., ... & Tos, M. (2002). Recent advances in otitis media. 1. Definitions, terminology, and classification of otitis media. *The Annals of otology, rhinology & laryngology. Supplement*, *188*, 8-18.

[4] Chole, R. A., & Sudhoff, H. H. (1998). Chronic otitis media, mastoiditis, and petrositis. Otolaryngology Head and Neck Surgery. *Third Ed. Mosby-Year Book, Inc*, 3026-46.

[5] Wang, H. M., Lin, J. C., Lee, K. W., Tai, C. F., Wang, L. F., Chang, H. M., ... & Ho, K. Y. (2006). Analysis of mastoid findings at surgery to treat middle ear cholesteatoma. *Archives of Otolaryngology–Head & Neck Surgery, 132*(12), 1307-1310.

[6] Todberg, T., Koch, A., Andersson, M., Olsen, S. F., Lous, J., & Homøe, P. (2014). Incidence of otitis media in a contemporary Danish National Birth Cohort. *PLoS One, 9*(12), e111732.

[7] DeAntonio, R., Yarzabal, J. P., Cruz, J. P., Schmidt, J. E., & Kleijnen, J. (2016). Epidemiology of otitis media in children from developing countries: A systematic review. *International Journal of Pediatric Otorhinolaryngology, 85*, 65-74.

[8] Abrahams, S. W., & Labbok, M. H. (2011). Breastfeeding and otitis media: a review of recent evidence. C*urrent allergy and asthma reports, 11*, 508-512.

[9] Kumari, M. S., Madhavi, J., Krishna, N. B., Meghanadh, K. R., & Jyothy, A. (2016). Prevalence and associated risk factors of otitis media and its subtypes in South Indian population. *Egyptian Journal of Ear, Nose, Throat and Allied Sciences, 17*(2), 57-62.

[10] Teele, D. W., Klein, J. O., Rosner, B., & Greater Boston Otitis Media Study Group. (1989). Epidemiology of otitis media during the first seven years of life in children in greater Boston: a prospective, cohort study. *Journal of infectious diseases, 160*(1), 83-94.

[11] Sophia, A., Isaac, R., Rebekah, G., Brahmadathan, K., & Rupa, V. (2010). Risk factors for otitis media among preschool, rural Indian children. *International journal of pediatric otorhinolaryngology, 74*(6), 677-683.

[12] Pau, B. C., & Ng, D. K. (2016). Prevalence of otitis media with effusion in children with allergic rhinitis, a cross sectional study. *International journal of pediatric otorhinolaryngology, 84*, 156-160.

[13] Kørvel-Hanquist, A., Koch, A., Lous, J., Olsen, S. F., & Homøe, P. (2018). Risk of childhood otitis media with focus on potentially modifiable factors: A Danish follow-up cohort study. *International journal of pediatric otorhinolaryngology, 106*, 1-9.

[14] Homøe, P., Kværner, K., Casey, J. R., Damoiseaux, R. A., van Dongen, T. M., Gunasekera, H., ... & Weinreich, H. M. (2017). Panel 1: epidemiology and diagnosis. *Otolaryngology–Head and Neck Surgery, 156*, S1-S21.

[15] Jones, L. L., Hassanien, A., Cook, D. G., Britton, J., & Leonardi-Bee, J. (2012). Parental smoking and the risk of middle ear disease in children: a systematic review and meta-analysis. *Archives of pediatrics & adolescent medicine, 166*(1), 18-27.

[16] Deng, Q., Lu, C., Li, Y., Chen, L., He, Y., Sundell, J., & Norbäck, D. (2017). Association between prenatal exposure to industrial air pollution and onset of early childhood ear infection in China. *Atmospheric environment, 157*, 18-26.

[17] MacIntyre, E. A., Karr, C. J., Koehoorn, M., Demers, P. A., Tamburic, L., Lencar, C., & Brauer, M. (2011). Residential air pollution and otitis media during the first two years of life. *Epidemiology*, 81-89.

[18] Šprem, N., & Branica, S. (1993). Effect of climatic elements on the frequency of secretory otitis media. *European archives of oto-rhino-laryngology, 250*, 286-288.

[19] Ribeiro, H., & Cardoso, M. R. A. (2003). Air pollution and children's health in São Paulo (1986–1998). *Social science & medicine, 57*(11), 2013-2022.

[20] Jones, L. L., Hassanien, A., Cook, D. G., Britton, J., & Leonardi-Bee, J. (2012). Parental smoking and the risk of middle ear disease in children: a systematic review and meta-analysis. *Archives of pediatrics & adolescent medicine, 166*(1), 18-27.

[21] Kong, S. K., Chon, K. M., Goh, E. K., Lee, I. W., Lee, J. W., & Wang, S. G. (2009). Histologic changes in the auditory tube mucosa of rats after long-term exposure to cigarette smoke. *American journal of otolaryngology, 30*(6), 376-382.

[22] Jones, L. L., Hassanien, A., Cook, D. G., Britton, J., & Leonardi-Bee, J. (2012). Parental smoking and the risk of middle ear disease in children: a systematic review and meta-analysis. *Archives of pediatrics & adolescent medicine, 166*(1), 18-27.

[23] Moher, D., Shamseer, L., Clarke, M., Ghersi, D., Liberati, A., Petticrew, M., ... & Stewart, L. A. (2015). Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. *Systematic reviews, 4*(1), 1-9.

[24] Park, M., Han, J., Jang, M. J., Suh, M. W., Lee, J. H., Oh, S. H., & Park, M. K. (2018). Air pollution influences the incidence of otitis media in children: A national population-based study. *PLoS One, 13*(6), e0199296.

[25] Girguis, M. S., Strickland, M. J., Hu, X., Liu, Y., Chang, H. H., Kloog, I., ... & Vieira, V. M. (2018). Exposure to acute air pollution and risk of bronchiolitis and otitis media for preterm and term infants. *Journal of exposure science & environmental epidemiology, 28*(4), 348-357.

[26] Aguilera, I., Pedersen, M., Garcia-Esteban, R., Ballester, F., Basterrechea, M., Esplugues, A., ... & Sunyer, J. (2013). Early-life exposure to outdoor air pollution and respiratory health, ear infections, and eczema in infants from the INMA study. *Environmental health perspectives, 121*(3), 387-392.

[27] Gestro, M., Condemi, V., Bardi, L., Fantino, C., & Solimene, U. (2017). Meteorological factors, air pollutants, and emergency department visits for otitis media: a time series study. *International journal of biometeorology, 61*, 1749-1764.

[28] Brauer, M., Gehring, U., Brunekreef, B., de Jongste, J., Gerritsen, J., Rovers, M., ... & Heinrich, J. (2006). Traffic-related air pollution and otitis media. *Environmental health perspectives, 114*(9), 1414-1418.

[29] Kousha, T., & Castner, J. (2016). The air quality health index and emergency department visits for otitis media. *Journal of Nursing Scholarship, 48*(2), 163-171.

[30] Strickland, M. J., Hao, H., Hu, X., Chang, H. H., Darrow, L. A., & Liu, Y. (2016). Pediatric emergency visits and short-term changes in PM2. 5 concentrations in the US State of Georgia. *Environmental health perspectives, 124*(5), 690-696.

[31] Xiao, Q., Liu, Y., Mulholland, J. A., Russell, A. G., Darrow, L. A., Tolbert, P. E., & Strickland, M. J. (2016). Pediatric emergency department visits and ambient Air pollution in the US State of Georgia: a case-crossover study. *Environmental Health, 15*(1), 1-8.

[32] Dostál, M., Prucha, M., Rychlíková, E., Pastorková, A., & Srám, R. J. (2014). Differences between the spectra of respiratory illnesses in children living in urban and rural environments. *Central European Journal of Public Health, 22*(1), 3.

[33] Zemek, R., Szyszkowicz, M., & Rowe, B. H. (2010). Air pollution and emergency department visits for otitis media: a case-crossover study in Edmonton, Canada. *Environmental health perspectives, 118*(11), 1631-1636.

[34] Song, J. J., Kwon, J. Y., Park, M. K., & Seo, Y. R. (2013). Microarray analysis of gene expression alteration in human middle ear epithelial cells induced by micro particle. *International Journal of Pediatric Otorhinolaryngology, 77*(10), 1760-1764.

[35] Song, J. J., Lee, J. D., Lee, B. D., Chae, S. W., & Park, M. K. (2012). Effect of diesel exhaust particles on human middle ear epithelial cells. *International journal of pediatric otorhinolaryngology, 76*(3), 334-338.

© 2023 by the authors; licensee PSRP, Lahore, Pakistan. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license (http://creativecommons.org/licenses/by/4.0/).