Particulate Matter 2.5 with Impaired Lung Function on Children: A Systematic Review

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Abstract. Particulate Matter 2.5 (PM$_{2.5}$) is generated from the combustion process, including exhaust fumes from motor vehicles, industrial activity, and forest fires. This review aims to find out PM$_{2.5}$ as one of the risk factors of impaired lung function on children through a critical review of the article journals. A systematic review strategy was conducted by searching for articles related to PM$_{2.5}$ with impaired lung function on children that already published in accredited journals from 2012-2017. The literature search was conducted in April 2017 through the online database, Pubmed, ProQuest, and Science Direct database. Seven article journals selected based on the inclusion criteria and the exclusion criteria that conducted with the critical review process. This systematic review showed the significant result that PM$_{2.5}$ is one of the risk factors for impaired lung function on children. The higher the average concentration of PM$_{2.5}$, the more decrease of lung function on children. Children, as one of the susceptible groups of PM$_{2.5}$, need more protection and preventive actions, especially with forest fires that happened in Indonesia.

Keywords: PM$_{2.5}$, Impaired Lung Function, Children

1 Introduction

PM$_{2.5}$ is a particulate with a diameter of 1-2.5 microns. PM$_{2.5}$ is generated from the combustion process, including exhausting fumes from motor vehicles. PM$_{2.5}$ is also produced from chemical reactions between various gases such as sulfur dioxide, nitrogen dioxide, and VOCs (Volatile Organic Compounds). PM$_{2.5}$ may exacerbate heart disease and lung disease and has been linked to several effects such as cardiovascular disease, heart attack, respiratory disorders, asthma attacks, emphysema, and bronchitis. Community groups susceptible to PM$_{2.5}$ exposure include having a history of heart disease or lung disease, the elderly, and children [1]. PM$_{2.5}$ can be in the atmosphere indefinitely. Currently, PM$_{2.5}$ is believed to be a major contributor to various human health problems, because the particulate can enter and block the smallest tubes contained in the lungs. Some studies have found that particulates smaller than 2.5 $\mu$m give worse effects on the respiratory system than larger particles. PM$_{2.5}$ can reach the bronchioles and alveoli. Then, it will accumulate in the respiratory tract, then it increases the risk of respiratory problems, inhibit the development and function of the lungs, and the most severe is the increased mortality due to respiratory and cardiovascular diseases [2].
PM concentrations are higher in urban areas than in rural areas, which may be caused by more motor vehicle emissions in cities than in villages. The PM concentration in the room depends on the source of the combustion from the room [3]. The study by Choo et al. found that kindergarten schools (TK) in urban areas had higher concentrations of PM$_{2.5}$ than rural areas. The prevalence of cough and high-frequency breathing sounds that were heard at the end of expiration (wheezing) was significantly higher among urban kindergarten children than in the village. This study proves that the increase in air pollution has adverse effects, especially on the development of respiratory function in childhood. Statistically, it was found that there was a significant relationship between exposure to high PM$_{2.5}$ indoors with an increased risk of respiratory distress in children [4].

Exposure to PM$_{2.5}$ can already occur since prenatal time. PM$_{2.5}$ concentrations indoors and exposure to prenatal cigarette smoke, affecting bronchiolitis and lower respiratory tract infections [5]. In preschool-aged children, there was an effect of increased PM$_{2.5}$ concentration with the progression of bronchitis [6]. A study by Martinez et al. found a link between the Forced Expiratory Volume in the first second (FEV1) and Forced Vital Capacity (FVC) with air pollution [7], especially particulates with a small size of 2.5 µm [8].

2 Materials and Methods

This review is a systematic review with the implementation of a literature search strategy to ensure all relevant literature is covered in this search. Furthermore, the articles will be assessed to get a conclusion in accordance with the purpose of this study; to know the exposure of PM$_{2.5}$ with impaired lung function in children. The search for articles is done by searching for articles related to PM$_{2.5}$ with impaired lung function in children that already published in accredited journals. My literature search was conducted in April 2017 through the online database, Pubmed, ProQuest, and Science Direct database. In addition, I also searched for the Environmental Health Perspectives journal because it relates to the theme of my review. The keywords used were "PM$_{2.5}$ and Impaired Lung Function" and the search year is limited from 2012 to 2017. Journals and articles obtained were filtered by title and abstract. The selected articles were based on the desired criteria, while the journals that were not relevant to the research topic were issued. The selected journals were evaluated using inclusion and exclusion criteria, based on the criteria separated from which the journal was eligible for review.

<table>
<thead>
<tr>
<th>Inclusion</th>
<th>Exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Published from 2012-2017</td>
<td>Published before 2012</td>
</tr>
<tr>
<td>PM$_{2.5}$</td>
<td>Didn’t explain about PM$_{2.5}$</td>
</tr>
<tr>
<td>Outcome: Impaired Lung Function</td>
<td>Outcome didn’t explain about Impaired Lung Function</td>
</tr>
<tr>
<td>Focused on children</td>
<td>Focused on Adult or older people</td>
</tr>
</tbody>
</table>
3 Result

There were 2033 articles in the Pubmed, ProQuest, and Science Direct database plus 77 articles from the Environmental Health Perspectives journal that found after the author searched using the keyword. After that, the articles' title was scanned based on the inclusion criteria. There were 2047 articles issued because of incompatibility with the research criteria. Then, there were 63 articles abstracts left to be reviewed. After that, 38 remaining articles still compatible with the criteria and then reviewed by the full script. Finally, seven articles appropriated for the final full script review.

![Flowchart of the systematic review process](image)

Fig. 1. Systematic review

From 7 articles that had been reviewed, there were four articles used prospective cohort design, two articles used retrospective cohorts, and one article used a cross-sectional design. Table
Table 2. Critical appraisal

<table>
<thead>
<tr>
<th>Authors, Research’s year, Location</th>
<th>Aim of the study</th>
<th>Study design</th>
<th>Sample</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hwang et al. 2007-2009, Taiwan</td>
<td>Assess the relationship between exposure to air pollution and the development of lung function and to examine the potential differences in sex susceptibility to air pollution</td>
<td>Prospective Cohort</td>
<td>2941 children who didn’t smoke</td>
<td>Decreases in FVC, FEV1, and FEF were associated with increased PM$_{2.5}$ exposure. Decreased lung function was greater in boys than girls.</td>
</tr>
<tr>
<td>Chen et al., 2011, Taiwan</td>
<td>To recognize the chronic and subchronic effects of ambient air pollution on lung function and compare it among children of different ages</td>
<td>Cross-sectional</td>
<td>1494 schoolchildren 6-15 years old at 44 schools on 24 sub-districts in Taiwan</td>
<td>The increase in PM$<em>{2.5}$ was associated with a decrease in FEV1 / FVC and MMEF / FVC. On children age 6-15 years old that got chronic exposure to PM$</em>{2.5}$ will lead to decreased lung function and it founded that there were some damages to the texture of the children respiratory tracts that associated with PM$_{2.5}$.</td>
</tr>
<tr>
<td>Gehring et al. 2012, Germany, Sweden, Netherlands, UK</td>
<td>To determine the relationship between exposure to air pollution in the population with lung function in five cohort studies in Europe.</td>
<td>Retrospective Cohort</td>
<td>5921 Children aged 6-8 years old.</td>
<td>Air pollution exposure reduces lung function in school-aged children. FEV1 changed between $-1.77%$ (95% CI: $-3.34$, $-0.18%$) for increased of 5-µg/m3 PM$_{2.5}$.</td>
</tr>
<tr>
<td>Meng Wang et al., 2014, Netherlands</td>
<td>Evaluate the relationship between long-term air pollution exposure using two modeling techniques of exposure and to compare estimates of</td>
<td>Retrospective Cohort</td>
<td>1,058 children</td>
<td>Predictions of LUR and dispersion models were positively associated with PM$<em>{2.5}$ and NO$</em>{2}$.</td>
</tr>
</tbody>
</table>
the relationship between long-term air pollution exposure and lung function in children using the modeling technique

Watanabe et al., 2015. Japan
To know the effects of PM on the lungs of school-aged children and their potential for an inflammatory response
Prospective cohort
339 children aged 10-12 years old
The increase in PM$_{2.5}$ was associated with a decrease in PEF (-1.72 L / min). There were significant relationships between PEF, SPM, and PM$_{2.5}$.

Fuertes et al., 2015. Germany
To determine the relationship between exposure to air pollution at residence and lung function in 15 years of life in two German birth cohort studies
Prospective cohort
2266 children aged 15 years old that live in city and village
Exposure to long-term air pollution is not associated with lung function

To evaluate the short-term effects of PM$_{2.5}$ exposure on lung function and see the potential role of CC16 on this effect
Prospective cohort
36 healthy students
PM$_{2.5}$ exposure is negatively related to lung function

4 Discussion

More than half of the studies show that there was a significant relationship between PM$_{2.5}$ exposure and impaired lung function in children. The results of research by Hwang et al. from 2007 to 2009 in Taiwan on 2941 non-smoked children found that there was a decrease in Forced Vital Capacity (FVC), Forced Expiration Volume in 1 second (FEV1), and Forced Expiratory Flow (FEF) if there was an increase in exposure to PM$_{2.5}$. For example, the higher PM$_{2.5}$ exposure (IQR, 17.92 µg / m$^3$) was associated with 75 mL FVC decline in boys and 61mL in girls (p <0.05). The decline is more common in boys than girls [9]. The results of the study by Chen et al. 2011 in Taiwan on 1494 schoolchildren 6-15 years old at 44 schools in 24 sub-districts in Taiwan found that lung function changed every average increase of PM$_{2.5}$ for two months (12 µg / m3, 32-44) was -103 on FVC, -86 on FEV1 and -102 ml / s at MMEF. PM$_{2.5}$ was associated with a decrease in the ratio of FEV1 / FVC and MMEF / FVC. On children aged 6-10 years, damage to respiratory tract structures in the lung may be associated with PM$_{2.5}$ exposure [10]. Then, the results of research by Gehring et al. in 2012, in Germany, Sweden, the Netherlands, and the UK, on 5921
children aged 6-8 years old also found a decrease in maximal expiratory expression in 1 second (FEV1) of -1.77% (95% CI: -3.34, -0.18%) for 5-µg / m3 escalation of PM2.5 [11]. Meng Wang et al. in 2014 in the Netherlands studied 1,058 children using two exposure modeling techniques named LUR and dispersion-modeled. Concentrations were found to be high for NO2 and PM2.5 (R = 0.86-0.90) [12]. Watanabe et al. in 2015 conducted a study of 339 children aged 10-12 in Japan. He found that an increase of 10 mg/m3 PM2.5 was associated with a decrease in PEF (-1.72 L / min) [13].

Many other epidemiological studies have proven significant associations between PM2.5 and pulmonary function. However, not all studies have found it because it depends on climatic conditions and mixtures of other pollutants. Fuertes et al. in 2015 conducted a study of 2266 15-year-olds living in urban and rural Germany. Fuertes found that long-term air pollution was not related to lung function [14]. Cuicui Wang et al. research from 2014 to 2015 in Shanghai on 36 healthy and non-smoking students found that PM2.5 exposure was negatively associated with lung function. However, Wang found that the acute effect on PM2.5 could be an immediate decrease in lung function because it impairs the integrity of the pulmonary epithelium [15]. Children are more susceptible to the effects of air pollution. The development of the lungs of children is still not perfect at birth. Lung development occurs by the proliferation of the alveoli and capillaries up to two years of age. Then, alveolus expansion will develop until the age of 5-8 years. The lungs will continue to develop during adolescence and will be perfect as adults [16, 17]. Infants and children have higher levels of metabolic fatigue and higher levels of oxygen consumption than adults. This is because children are still in a period of rapid growth and development and have a very active physical activity. It causes exposure to air pollution to be riskier to health. Another cause is the need for higher children's oxygen, and children have narrower airways than adults, so air pollution that provides a small response in adults will have a more severe effect on the breathing of children [18].

5 Conclusion

Half of the studies have found that PM2.5 results in impaired lung function in children. The higher the average concentration of PM2.5, the more decrease in lung function. However, some studies didn’t find the PM2.5 relationship with lung function. It is due to climatic factors at the site and the presence of a mixture of other contaminants. It needs further research that shows the difference in the outcome of lung function disorder with PM2.5 on children, especially because of forest fires that happened in Indonesia.

References


