Clinical Research

A systematic review of respiratory infection due to air pollution during natural disasters

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ABSTRACT

BACKGROUND Wildfire and volcano eruption occurred in Indonesia due to its geographical location, climate change, global warming, and human behavior. Various substances produced an increased risk of experiencing health problems, including respiratory infection. Evidence about the effect of pulmonary infection during natural disasters is still limited. This study was aimed to review and elaborate on previous studies to determine the effect of air pollution exposure during natural disasters and respiratory infection.

METHODS Literature searches were conducted on PubMed, EBSCOhost, and Google Scholar, and was limited to the 10 last years, human studies, and the English language. Inclusion criteria were articles with representation for clinical questions, review articles, population studies, and the full-text article was available. Exclusion criteria were articles that only discussed the exposure to and not the association with the effect of the respiratory infection. The Oxford Center for Evidence-Based Medicine tools appraised six relevant articles.

RESULTS Air pollution during a natural disaster enhances particulate matter to 10–70 µg/m³ and more than 5 times the aerosol optical depth measurement compared with the tolerated air concentration. The air level was consistently related to acute respiratory infection, pneumonia, bronchitis, and bronchiolitis admissions in wildfire smoke and volcanic eruption in this review. Nevertheless, there was a diverse result for upper respiratory infection cases.

CONCLUSIONS Natural disasters increased the level of ambient air pollution that exceeded the levels recommended by the World Health Organization air quality guideline. Air pollution may play an important role in respiratory tract infection, especially among population with high exposure.

KEYWORDS air pollution, disasters, respiratory tract infections, volcanic eruptions, wildfires

Nowadays, unhealthy air quality has become an environmental and health issue in Indonesia. Air pollution is a condition in which air is contaminated by a mixture of complex particles and gases that are chemically modified in the atmosphere. The sources of air pollution are divided into anthropogenic and natural causes. Anthropogenic sources of air pollution include vehicle exhaust gas, industrial processes, and the household sector when burning biomass and cigarettes. Meanwhile, natural causes of air pollution are wildfires, volcanic eruptions, storms, and similar events. Considering that Indonesia's geographical location is between three earth plates and is located in the Ring of Fire, its position increases its probability of being in close proximity to volcanic eruptions. Besides, climate change, global warming, drought,

deforestation by humans increases the risk of being overcome by wildfires. Data from the Indonesian National Board for Disaster Management states that there were 775 incidences of wildfires and 62 volcanic eruptions in 2018–2019.[1]

Volcanic eruptions produce various gases that are potentially harmful to health status, such as carbon dioxide, sulfur dioxide (SO_2), hydrogen sulfide, carbon monoxide (CO), radon, hydrogen chloride, and metals weight including mercury and lead.[2,3] Volcanic ash produced from volcanic eruptions is different from other natural dust because the surface of the particles is not weathered and is not oxidized or dissolved. Therefore, it can carry condensed volatiles, such as acids, polycyclic hydrocarbons, and various types of metal.[2] Wildfire smoke also contain various toxic substances as well as volcanic eruption, such as CO, SO_2, nitrogen dioxide, ozone, cyanide, fine particulates, and combustion products that are difficult to predict; causing asphyxia and lung damage.[3]

Ash exposure decreased the development of autophagosome-lysosome fusion, thereby disrupting the homeostasis process and cell differentiation in human alveolar macrophages. This exposure also decreases the mitogen-activated protein kinase (MAPK)-APK 2/3 pathways (extracellular-signal-regulated kinase, Jun N-terminal kinase) that is needed for the optimal cytokine response in cells, as well as decreased production of tumor necrosis factor-alpha mRNA by lipopolysaccharide in human alveolar macrophages. Ash exposure impairs macrophage function, causing a decrease in the cell's ability to kill bacteria. Ash also impairs the bactericidal and bacteriostatic activity of the airway system by inhibiting antimicrobial peptide activity. Antimicrobial peptides in the lung are located in the airway surface liquid, containing a lactoferrin to inhibit microbial respiration, a lysozyme to destroy bacterial cell walls, and defensin that has broad antibacterial activity.[4,5] A study in the rhesus macaque showed that early exposure to smoke affects the immune system, as measured by the reduced cytokine synthesis in peripheral blood cells, and it could be a possible mechanism that plays a role in the increase pulmonary infection.[3]

Various hazardous substances contained in wildfires or volcanic eruption smoke, cause a high risk of experiencing health problems.[2] Many studies documented the association between smoke exposure and the effects on general respiratory health, specifically asthma exacerbation or chronic obstructive pulmonary disease. Nevertheless, the evidence of whether there is an increased risk of pulmonary infections and air pollution exposure during natural disasters remains limited.[3] Therefore, this article will review and elaborate on related-previous studies to determine the effect of air pollution exposure during natural disasters (volcanic eruption or wildfires) on the respiratory infection.

METHODS

A literature search was performed on electronic databases, including PubMed (https://www.ncbi.nlm.nih.gov/pubmed), EBSCO (http://search.ebscohost.com), and Google Scholar (https://scholar.google.com). A literature search was conducted on November 1, 2019 and the last data were accessed on December 23, 2019, using the keywords listed in Table 1. The results obtained from the database corresponded to the clinical questions using the Boolean system presented in Table 1.

Table 1. Literature search strategy

Database	Keyword	Result
PubMed	((((((("wildfire"[Title/Abstract]) OR ("volcano eruption"[Title/Abstract] OR "volcanic "[Title/Abstract] OR "disaster"[Title/Abstract])) OR ("forest fire"[Title/Abstract] OR "forest fire event"[Title/Abstract] OR "forest fire emissions"[Title/Abstract] OR "forest fire episode"[Title/Abstract] OR "forest fire incidents"[Title/Abstract] OR "forest fire occurrence"[Title/Abstract] OR "forest fire period"[Title/Abstract]))) AND free full text[sb] AND English[lang])) AND (((((respiratory infection[Title/Abstract]) OR morbidity[Title/Abstract]) OR mortality[Title/Abstract]) OR pulmonary infection[Title/Abstract]) OR acute respiratory infection[Title/Abstract]))	381
EBSCO	TX (forest fires or wildfire) OR volcano eruptions) AND TX respiratory infection	269
Google Scholar	Volcanic eruption exposure OR wildfire OR forest fire AND effect on respiratory infections OR pneumonia	2,170

The literature search process was continued using the limits of the literature research and then the titles and abstracts were selected from each database. Studies were included in this review if met the following inclusion criteria: representation for clinical questions (P: person who was affected or lived in wildfire or volcanic area; I: natural disaster's smoke [volcano eruption or wildfire]; C: no exposure to natural disaster smoke; O: respiratory infection), type of study was a review article or population study (cohort, case-control, case-crossover, or time-series study), and if the full-text article was available. The studies were excluded if the association between the exposure and the respiratory infection effect was not discussed. Multiple article checks were performed on the three databases. The appropriate study was then read in full paper and appraised. A critical appraisal was made based on the Oxford's Center for Evidence-Based Medicine which assesses the validity, importance, and applicability of each article. A flow diagram describing the study selection process is shown in Figure 1.

The Cochrane risk of a bias assessment tool for non-randomized studies of interventions (ACROBAT-NRSI) was used to measure the risk of bias in this systematic review.[6] The measurement results for cross-sectional, cohort, and case-crossover study designs will be presented using relative risk/risk ratios, and for the case-control study design using odds ratio. The method of data extraction from the study reports was done independently.

RESULTS

Based on the literature search strategy in three databases (PubMed, EBSCO, and Google Scholar), 1,436 studies were screened by title and abstract. Most articles in the literature search discussed the general effects of air pollution on the respiratory tract, but did not specifically discuss respiratory infection. Six articles were selected for an appraisal (Table 2) that met the eligibility criteria. Four articles discussed wildfires (Tinling et al,[7] Rapold et al,[8] Delfino et al,[9] Johnston et al[10]), and the last two articles discussed the volcano eruption effect (Carlsen et al,[11] Tyas et al[12]). The appropriate clinical questions of each study in this systematic review that were asked included, if the sample size was large enough, and if the time to follow up was long enough. However, there was no randomization and blinding for each study. The characteristics and results of each study are summarized in Table 2.
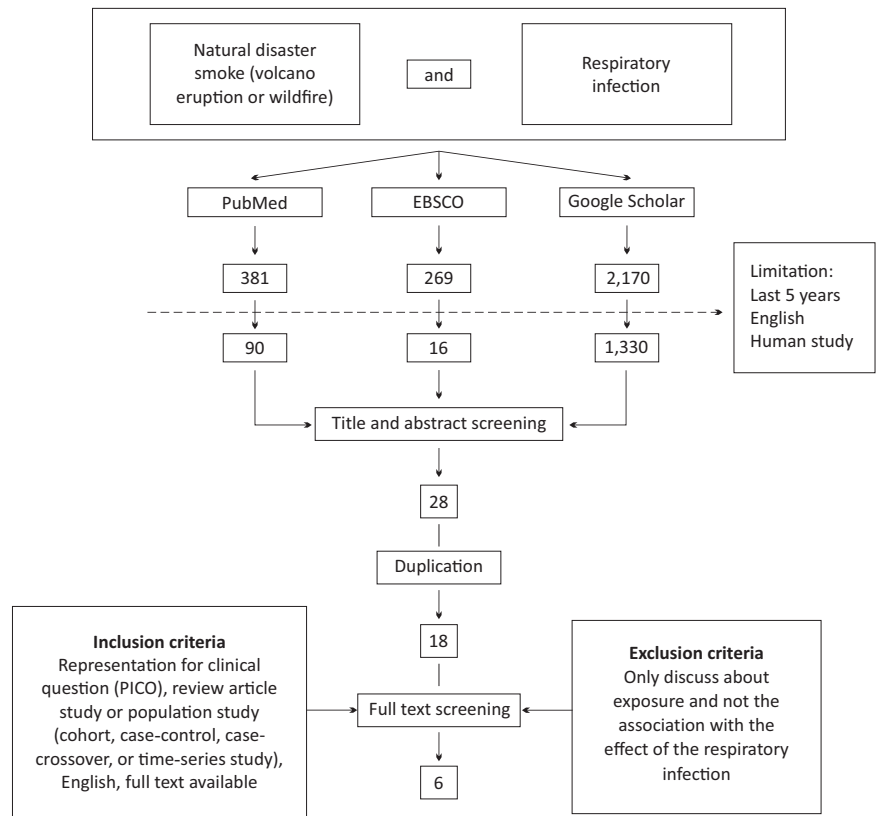

Figure 1. Study selection process

Table 2. Characteristics and critical appraisal of articles

Article	PICO	Study size	Study period	Validity				Relevance			
				Sample size	Randomization	Similarity of case– and control group	Blinding	Domain	Determinant	Result measurement	Level of evidence
Tinling et al[7]	P: resident of Eastern North Carolina affected by wildfire I: experienced at least one 24 hours average smoke-attributable PM2.5 concentration exceeding 20 μg/m³ during wildfire C: none O: cardiopulmonary health	Resident of 28 contiguous counties in Eastern North Carolina (1,841,372 population)	May 5–June 19, 2011 (45 days)	+	–	–	–	+	+	+	2c
Rapold et al[8]	P: resident of Eastern North Carolina I: exposed to peat bog wildfire smoke C: not exposed O: cardiopulmonary emergency department visit	Resident of 42 contiguous counties in Eastern North Carolina	June–July 2018	+	–	+	–	+	+	+	2c
Carlsen et al[11]	P: all resident in the municipalities closest to Eyjafjallajökull volcano I: adult eruption exposed South Icelanders C: resident in non-exposed area in Northern Iceland. O: health effect (respiratory symptom and disease)	1,658 resident eruption exposed	November 2010–March 2011	+	–	+	–	+	+	+	2b
Delfino et al[9]	P: Southern California children and adults I: California wildfires exposure C: before exposed to wildfires smoke O: respiratory and cardiovascular hospital admissions	40,856 hospital admission	October 1–November 15, 2003	+	–	+	–	+	+	+	2b
Johnston et al[10]	P: people living in Sydney I: air pollution events from wildfire C: non-event days from wildfire O: emergency department admissions due to non-trauma condition, heart disease and respiratory condition	4.06 million population (based on Sydney 2001 census)	1996–2007	+	–	+	–	+	+	+	2c
Tyas et al[12]	P: patients who visited Kepohbaru Health Center in February 2013 and February 2014 I: after exposed to volcanic ash C: before exposed to volcanic ash O: ARI	938 cases (448 cases before and 490 cases after volcanic exposure)	February 2013 and February 2014	+	–	+	–	+	+	+	2c

PM=particulate matter; ARI=acute respiratory infection
Based on the Center for Evidence-Based Medicine Oxford University criteria

DISCUSSION

In this systematic review, we examined the evidence of wildfire and volcanic eruption smoke's effect on respiratory infection cases, as almost all the studies presented concurrent infection and non-infectious conditions of various cardiopulmonary problems. Respiratory infection in this review was based on the ICD 10 code list for respiratory tract infections, that consisted of upper respiratory infection, acute respiratory infection, pneumonia, bronchitis, and bronchiolitis. Respiratory infections in the studies included were observed in various durations, with the shortest duration lasting about one month and the longest duration persisting for 11 years. This length of time could present representative data about the effect of air quality due to smoke of disaster, a wide range of study populations, and various diseases available for analysis. However, the data available on respiratory infection cases were quite limited because most studies were more interested in asthma and chronic obstructive pulmonary disease cases.

Our review on the effect of wildfire smoke and volcanic eruptions in respiratory infection found that bad air quality (measured by exceeding the level of aerosol optical depth, particulate matter [PM]2.5 and/ or PM10) related to respiratory infection cases (Tinling et al, Rapold et al, Delfino et al, Johnston et al, Carlsen et al) in an affected population.[7–11] The average level of PM varied in each study. PM is a pollution particle and liquid droplets in the atmosphere with a diameter of <10 μm (PM10) and <2.5 μm (PM2.5). Particles <4 μm can penetrate the alveolar region of the lung and have a high toxic potential to the lower respiratory tract.[2] The smoke during wildfires and volcanic eruptions dramatically increased air pollutants and local PM2.5 concentrations, resulted in admissions for most respiratory outcomes.[2] Possible mechanisms that caused respiratory system damage due to PM exposure were injured by organic components of PM induced neutrophils, T cell and eosinophil migration to the lungs and other tissues. Also, the inhibition of inflammation promotes the expression of a high level of an inflammation inhibitor of macrophages. This inhibitor, in turn, promotes free radical production to oxidize lung cells, consume antioxidant ingredients, and produced hydroxyl radicals. These radicals cause oxidative damage to DNA, suppress DNA repair, and promote replication of damaged DNA fragments.[13]

The result of this review supports the evidence that respiratory health, especially respiratory infection, should be considered when evaluating public health cases related to smoke in disaster settings. Tinling et al[7] analyzed that PM2.5 levels greater than 20 μg/m³ statistically increased the frequency of ED visits due to URI, but Rapold et al[8] found that wildfire smoke was related to increased bronchitis and pneumonia visits, but not in URI cases. The difference in the results of both studies could be caused by different amounts of exposure, different comparisons made by each study, and differences in the characteristics of population that were associated with an increased risk of infection in each study. Individuals residing in the same area might also experience different exposure, caused by lifestyle factors, occupation, air conditioning use, housing quality, duration spent outdoors, and protective tools usage.[7]

Delfino et al[9] assessed pneumonia, bronchitis, and bronchiolitis admissions significantly higher in PM2.5 per 10 μg/m³, while Johnston et al[10] found that PM2.5 and PM10 levels reach 39.1 and 60.5 μg/m³, a four times increase during a smoke event compared with a non-smoke event related to pneumonia or acute bronchitis ED visits. PM levels in the studies had exceeded the World Health Organization (WHO) recommendation for air quality threshold. WHO recommended air quality guideline (AQG) values in averaging time one year and 24 hours for PM2.5 and PM10 were 10, 25, 20, and 50 respectively.[14]

One interesting finding in this systematic review was that respiratory infection cases were not higher in elderly subjects during wildfire. It is different from Zhang et al[15] that elderly individuals were more susceptible to be hospitalized and visit the ED after PM exposure in air pollution. The reason for this might be because elderly individuals were more aware of the risk and practiced proper prevention, such as limited their exposure, using masks, and other preventive measures.

Volcanic eruptions affect respiratory health. Carlsen et al[11] found that residents exposed to eruptions had a higher prevalence of respiratory infection and respiratory symptoms compared with the non-exposed group regarding long-term effect. This result is in concordance with Gudmundsson G,[16] who found that there were acute respiratory symptoms after volcanic ash exposure, but no long-term effects have been found. The different results of this study could be caused by different materials emitted from

Table 3. Risk of pulmonary infection due to natural disaster exposure

Article	Statistical method	Location	Exposure assessment	Finding
Tinling et al[7]	Cross-sectional	North Carolina	PM2.5	Total frequency of ED admission caused by URI due to wildfire was 6,457 cases, most common in 18–64 years (3,428 cases). PM2.5 concentration exceeding 20 µg/m³ during wildfire increased risk of ED visit among age <18 (cRR = 1.14 [95% CI = 1.04–1.24]), 18–64 (cRR = 1.15 [95% CI = 1.07–1.24]), >65 (cRR = 0.64 [95% CI = 0.39–1.05]), all adults (cRR = 1.13 [95% CI = 1.05–1.22]), female (cRR = 1.14 [95% CI = 1.05–1.24]), and male (cRR = 1.13 [95% CI = 0.99–1.29]) due to URI in exposure over 0–2 lag days.
Rapold et al[8]	Cross-sectional	North Carolina	AOD measurement	AOD level ≥ 1.25 categorized as high-density plume of smoke. Wildfire smoke in exposed counties increased risk of ED visit associated with bronchitis and pneumonia among all subject (cRR = 1.59 [95% CI = 1.07–2.34]), <65 (cRR = 1.87 [95% CI = 1.13–3.1]), ≥65 (cRR = 1.15 [95% CI = 0.61–2.16]), female. Risk of ED visit due to wildfire smoke in exposed counties related to URI (cRR = 1.68 [95% CI = 0.94–3.00]) in all ages, <65 (cRR = 1.44 [95% CI = 0.77–2.71]), and ≥65 (cRR = 1.43 [95% CI = 0.11–19.16]). Pneumonia & bronchitis (cRR = 1.94 [95% CI = 1.16–3.25]) and URI (cRR = 1.83 [95% CI = 0.92–3.67]) cases increased among women in exposed county compared with man.
Carlsen et al[11]	Cohort, with non-exposed control group	Southern Iceland	PM10	PM10 level in study population was surpassed limit more than 50% of official tolerated daily average level (50 µg/m³). Allergic rhinitis among non-exposed population and exposed population were 19.1% (96/502) and 19.1% (213/1,116), respectively (OR = 1.1 [95% CI = 0.8–1.4]). Chronic bronchitis during last 12 months of volcanic eruption in exposed population were 7% (78/1,107), compared with non-exposed 4.2% (21/503) (OR = 1.9 [95% CI = 1.1–3.1], p = 0.02).
Delfino et al[9]	Cohort retrospective	Southern California	PM2.5 and PM10	Average PM2.5 increased during heavy smoke condition is 70 g/m³. Per 10 g/m³ wildfire-related PM2.5, pneumonia admission increased 1.318 times (95% CI = 1.17–1.48) in overall cases after period of fires. Pneumonia admission for ages 0–4, 20–64, and 65–99 years were 46%, 30%, and 27% higher after the period of fires, respectively. Admission due to acute bronchitis and bronchiolitis increased by 48% (RR = 1.58 [95% CI = 1.09–2.29]) after the fires.
Johnston et al[10]	Time-stratified case-crossover design	Sydney, Australia	PM2.5 and PM10	Mean PM10 and PM2.5 level during smoke event days is 60,5 g/m³ and 39.1 g/m³ compared with 17.8g/m³ and 9.9 g/m³ during non-smoke event days. Pneumonia or acute bronchitis attendance during study period is 130,915 cases, mean value was 33 cases/day with range 3–88 cases. Most of them are <15 year (49%), 15–64 years (22%), and >65 years (29%). The 46 validated wildfire smoke event days happened during study period, which increased the risk of ED admission in the same day related to respiratory condition (OR = 1.07 [95% CI = 1.04–1.10]) and pneumonia/bronchitis cases (OR = 1.02 [95% CI = 0.95–1.10]).
Tyas et al[12]	Cross-sectional	Indonesia		ARI cases were higher in population after volcanic ash (490 cases) compared with before exposure (448 cases). Characteristics of majority after volcanic ash were more frequent in females (53.67%), the prevalence increases 11.09% cases in group age 26–55 years, and without history of ARI in 58.37%. Compared with before exposure, the characteristics of ARI cases were majority in males (52.01%), most cases in group age 0–25 years (56.7%), and no history of ARI in 58.37% cases.

PM=particulate matter; ED=emergency department; URI=upper respiratory infection; cRR=cumulative relative risk; CI=confidence interval; AOD=aerosol optical depth; OR=odds ratio; RR=relative risk; ARI=acute respiratory infection

volcanoes, the type, intensity and magnitude of the eruption, duration of the eruption, and external factors, such as wind strength, wind direction, and change in the wind direction during eruption.[16] The health effects of volcanic ash depend on particle size, mineral composition, and physicochemical properties of ash particle's surface.[16]

The effect of volcanic eruption on respiratory health status was shown in the study of Tyas et al,[12] that ARI cases were increased after volcanic ash exposure and Trisnawati et al that analyzed Merapi eruption composition consisted of crystalline silica in ash eruption.[17,18] Inhalation of these particles induced respiratory tract inflammation including bronchitis, asthma, and chronic exposure leads to pneumonoultramicroscopicsilicovolcanoconiosis.[17] Furthermore, the size of the particles will affect their penetration ability. Particles >15 mm diameter will not penetrate to respiratory tract beyond the nose. The particles <10 mm may cause lung irritation when entering the bronchioles. Respirable particles are fine particles <4 mm, and have a role in alveolar health because they can penetrate the alveolar region. Inhalation of large quantities of these particles over the long-term will worsen the condition of the respiratory tract.[17] Other factors that contributed to health status was type of occupational. Tyas et al[12] found that farmers or outdoor jobs rarely use personal equipment tools, such as masks, and are exposed to ash continuously during work. Therefore, the risk of respiratory infection was increased.

The increasing incidence of respiratory infections after an eruption illustrates that volcanic ash might influence bacterial growth due to innate immunity. Study of Monick et al[19] showed that exposure to ash disrupts the function of macrophages, including autophagy, the ability of bacterial killing, and the inflammatory response. Also, exposure to volcanic ash causes a bacterial growth spurt, which statistically increased bacterial culture growth.[3] Well-known pathogens in respiratory infections related to a disaster setting include bacterial (*Streptococcus pneumoniae*, *Bordetella pertussis*, *Mycobacterium tuberculosis*, *Legionella*, *Mycoplasma pneumoniae*) and viral (influenza, respiratory syncytial virus, adenovirus).[20] The ability to differentiate and identify the etiology of pneumonia was important to determine the need for antibiotic therapy for prompt treatment in bacterial infection.

This study had several limitations. First, there was no randomization and no blinding for each study. Thus, the risk of bias could not be avoided. Second, the study population in each study came from a different race, used different criteria, had different exposure, and had a different duration of the disaster. There was no specification or standardized criteria for each type of respiratory infection. Its variation could affect the results of studies. Third, the confounding factors that could affect the results could not be excluded.

In conclusion, we found that natural disasters, such as wildfires and volcanic eruptions, increased levels of ambient air pollution, measured by PM level, which exceeded the recommended WHO AQG level. It may play an important role in respiratory tract infection, especially among population with high exposure. Knowledge about groups at risk that had an effect on respiratory infection is important to prevent new cases increased disease burden. Comparing the health impacts of air pollution effect due to volcanic eruptions or wildfires in different regions and durations are difficult. There will be variation in fuel type, population, combustion intensity, material, and many factors that might contribute to the results. Additional studies are needed to assess the risk factors that contribute to respiratory infection in affected populations with minimal confounding factors.

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